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Neurovascular lesions and mechanisms in suicidal hanging: an anatomical, physiological and pathological study

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"Every man has leaned upon the past. Every liberty we enjoy has been brought at an incredible cost.
There is not a privilege nor an opportunity that is not the product of other men's labours. We drink every day from the wells we have not dug; we live by liberties we have not won; we are protected by institutions we have not set up.
No man lives by himself alone. All the past is invested in the lives of others."

Dr. Thomas Gibbs

NEUROVASCULAR LESIONS AND MECHANISMS IN SUICIDAL HANGING: AN ANATOMICAL, PHYSIOLOGICAL AND PATHOLOGICAL STUDY

‘ A thesis submitted to the Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, in fulfilment of the requirements for the degree of Doctor of Philosophy in Medicine’

Jacob Joseph Moar

Johannesburg, June 2012

DEDICATION

To my supervisor

Professor J.C. Allan

without whose immense support and encouragement this thesis would not have been possible.

ABSTRACT

Background and Purposes

Suicide by hanging is a relatively common occurrence. The actual cause of death in suicidal hanging is, however, controversial, having been attributed variously to asphyxia, carotid artery compression and vagal nerve stimulation.

The aim of this Ph.D thesis was to determine the possible neurovascular cause of death in suicides by hanging by careful study of the anatomy and physiology of the neck region in relation to the ensuing pathology. The study was, therefore, approached from an anatomical, physiological, histological and pathological pespective. It therefore comprised a detailed exploration of the anatomy and physiology of the neck structures to match these with the underlying traumatised neurovascular structures, the latter trauma being brought about by the suicidal hanging process.

Methods

The methods used in the study included an investigation of the ligature and position of the ligature in relation to the level of the neck and the physical effects of the ligature on the skin and underlying anatomical structures. A careful and detailed dissection of the neck was undertaken and samples of the vessels and nerves were processed for histological study. Fifty consecutive cases of suicidal hanging and five "non-hanging" cases which served as controls were used in the study. In addition, ten cases of suicidal hanging not included in the study were subjected to occlusion

studies by means of probe exploration. This technique and procedure was not carried out or applied to the cases included in the study for fear that the probe itself might produce artefactual damage to the delicate **endothelium** lining the inner layer of the vessel wall.

The study was classified into various components such as:

1. Examination of the type and structure of the ligature material;
2. The position of the ligature on the neck, i.e. whether involving upper, middle, or lower third of neck and to correlate this position with the underlying anatomical structures subjected to the accompanying tensile, compressive and haemodynamic forces;
3. The physical effects of the ligature upon the skin and the underlying deeper neurovascular structures of the neck;
4. Meticulous “bloodless” dissection of the neck structures to corroborate any pathology noted with the above three criteria. Currently, all putative causes of death remain speculative;
5. Particular attention was paid to those structures most vulnerable to the compressive forces, tensile forces and haemodynamic forces operative in hanging. These comprise the neurovascular structures contained within the fibrous carotid sheath and the phrenic nerves in the neck, in particular with regard to the anatomical relationship of these structures to the positioning of the ligature. As far as analysing the forces involved, the engineering principles pertaining to these were interpreted in consultation with the Faculty of Engineering at the University of the Witwatersrand.

Results

The main findings of the study showed damage to **vascular**, **neural** (including **phrenic nerve**), **carotid bodies** and **accessory glomal bodies**. The **vascular** findings emerged following an examination of the total number of **arteries** in the study, namely, 300, the figure derived as follows: six arteries in each of the fifty hanging subjects, viz., the **left common carotid artery**, the **right common carotid artery**, the **left internal carotid artery**, the **right internal carotid artery**, the **left external carotid artery** and the **right external carotid artery** (6 x 50 = 300). The damage shown was particularly the case with regard to the finding of **tears** in the various layers of the vessel wall. These extended from the **intima** through to the **adventitia** or outermost layer of the vessel wall and these were further subdivided into being either single or multiple.

The **tears** found ranged from those involving the **intima** alone (single **tears** being found in 17 (5.6%) of the 300 arteries examined and multiple **tears** in 37 (12.3%) of the 300 arteries examined., the **intima** extending to the **internal elastic lamina** (single **tears** being found in 20 (6.6%) of the 300 arteries examined and multiple **tears** in 8 (2.6%) of the 300 arteries examined), **tears** involving the **intima** and extending through to involve the **media**, i.e. **intimo-medial tears** and whether these latter **tears** involved the inner-, middle-, or outer-thirds of the **media** (single or multiple). Single **intimo-medial tears** extending through the **intima** to involve the **inner-third of the media** comprised 6 (2.0%) of the arteries examined, those extending from the **intima** to involve the **middle-third of the media** comprised 3 (1.0%) of the 300 arteries examined and single **intimo-medial tears** extending through the **intima** to involve the **outer-**

third of the media similarly comprised 3 (1%) of the arteries examined. Multiple intimo-medial tears extending through from intima to inner-, middle-, and **outer-thirds of the media** respectively, comprised 3 (1.0%), 5 (1.6%) and 1 (0.3%) of the arteries examined.

Single **tears** involving the **inner-third of the media** alone comrised 6 (2.0%) of the 300 arteries examined, single **tears** involving the **middle-third of the media** comprised 9 (3.0%) of the arteries examined and single **tears** involving the **outer-third of the media** alone comprised 8 (2.6%) of the arteries examined. Multiple **tears** involving the **inner-, middle** and **outer-thirds of the media** respectively comprised 6 (2.0%), 13 (4.3%) and 16 (5.3%) of the arteries examined.

Single **tears** involving both **adventitia** and **media**, i.e. **adventitio-medial tears** extending through the **inner-, middle-,** or **outer-thirds of the media** to involve the **adventitia** comprised 1 (0.3%), 2 (0.6%) and 6 (2.0%) respectively of the 300 arteries examined. Multiple **adventitio-medial tears** of the **inner-, middle-,** and **outer-thirds of the media**, respectively, comprised 0 (0.0%), 3 (1.0%) and 2 (0.6%) of the 300 arteries examined.

Single **tears** of the **adventitia** alone comprised 21 (7.0%) of the arteries examined while multiple **tears** comprised 7 (2.3%). **Complete circumferential transverse rupture** of the vessel wall was found in 3 (1.0%) of the arteries examined while **adventitial haemorrhage** was found in 103 (34.3%) of the 300 arteries examined.

The **vascular** findings were represented numerically in tabular form in the 50 hanging subjects in Table III and were further analysed and compared with regard to either unilateral or bilateral vessel involvement in the fifty (50) suicidal

hanging subjects and the findings represented in Tables IIIa (unilateral involvement) and IIIb (bilateral involvement).

Additional **vascular** findings comprised **endothelial elevation/avulsion**, **internal elastic lamina dehiscence**, **subendothelial clefts**, **multiple medial fenestrations**, **adventitio-medial separation**, **vascular congestion** and a **vascular plane of cleavage**. These were similarly represented in Table IV and analysed with regard to unilateral or bilateral involvement in Tables IVa and IVb. **Endothelial elevation/avulsion** was found in 295 (98.3%) of the 300 arteries examined, **internal elastic lamina dehiscence** in 290 (96.6%) of the arteries examined, **subendothelial clefts** in 289 (96.3%) of the arteries examined, **multiple medial fenestrations** in 17 (5.6%) of the arteries examined, **adventitio-medial separation** in 273 (91.0%) of the arteries examined, **vascular congestion** in 224 (74.6%) of the arteries examined and a **vascular plane of cleavage** in 98 (32.6%) of the arteries examined. These findings, unexpected, showed the extreme fragility and vulnerability of the **intima** and **adventitia** to the compressive and tensile forces acting on the vessel wall during hanging, being explicable not only on the basis of the various complex forces interacting simultaneously during hanging but on the magnitude of forces applied. A mathematical analysis, found at the end of the Discussion chapter, conducted in order to estimate the minimum peak pressure applied and exerted on the vessel wall during hanging, in collaboration with the School of Mechanical, Industrial and Aeronautical Engineering at the University of the Witwatersrand, confirmed the magnitude of these forces.

The **neural** findings (Table V) were divided into **neural congestion**, **neural haemorrhage**, **neural internal dehiscence**, **neural tearing** and **perineural**

separation and these were similarly analysed with regard to either unilateral or bilateral involvement in the fifty hanging subjects (Tables Va and Vb). **Neural congestion** was found in association with 20 (6.6%) of the 300 arteries examined, **neural haemorrhage** in14 (4.6%), **neural internal dehiscence** in 54 (18.0%), **neural tearing** in 35 (11.6%) and **perineural separation** in 112 (37.3%). **Neural ganglionic** findings were similarly divided into **ganglionic congestion**, **ganglionic haemorrhage**, **ganglionic internal dehiscence** and **ganglionic tearing**. **Ganglionic congestion**, in association with the 300 arteries examined, was found in 20 (6.6%), **ganglionic haemorrhage** in 8 (2.6%), **ganglionic internal dehiscence** in 15 (5.0%) and **ganglionic tearing** in 6 (2.0%).

The findings in the **carotid bodies** were divided into **carotid body congestion**, **carotid body haemorrhage**, **carotid body internal dehiscence** and **carotid body tearing**. **Carotid body congestion**, in association with the 300 arteries examined, was found in 8 (2.6%), **carotid body haemorrhage** in 2 (0.6%), **carotid body internal dehiscence** in 4 (1.3%) and **carotid body tearing** in 2 (0.6%).

Accessory glomal body findings were, once again, divided into **accessory glomal congestion**, **accessory glomal haemorrhage**, **accessory glomal internal dehiscence** and **accessory glomal tearing**. However, in view of the close anatomical association between the **accessory glomal bodies** and the **adventitia** of the arterial walls, an additional pathological finding of **accessory glomal adventitial separation** emerged. **Accessory glomal congestion**, in association with the 300 arteries examined, was found in 20 (6.6%), **accessory glomal haemorrhage** in 7 (2.3%), **accessory glomal internal dehiscence** in 50 (16.6%),

accessory glomal tearing in 18 (6.0%) and **accessory glomal adventitial separation** in 124 (41.3%). This latter finding once again demonstrated the vulnerability of the adventitial layer of the vessel wall to tensile forces, separating it from its associated structures.

Damage to the **phrenic nerves** and surrounding **muscles**, underlying the site of ligature application, was similarly found, suggesting a role for **phrenic nerve** stimulation with consequent diaphragmatic paralysis in contributing to death in the hanging process.

Discussion and Conclusion

In this Ph.D thesis the principles of **dimensional analysis** i.e., the breaking down of a complex phenomenon into its component parts, have been applied. However, in view of the complexity and proximity of structures to one another in the neck, consisting not only of the rigid hyoid-larynx complex and vertebral column but also the integrated **vascular** and **neural** structures, it appears that not one single biological mechanism can be ascribed and attributed to the cause of death in suicidal hanging. Rather, it appears that unconsciousness and death causation appears to be multifactorial. Both the sympathetic and parasympathetic arms of the autonomic nervous system are involved, often with antagonistic and therefore paradoxical effects. In a ddition, pressure to the **phrenic nerve**, not previously considered in playing a role in death causation in hanging, may, it is suggested, be a major contributory factor in death causation. This **nerve**, the innervation to the major muscle of respiration, i.e. the diaphragm, in a neural response to the compressive and tensile forces in hanging, fixes the diaphragm in a state of

inspiratory paralysis. This latter effect would be further augmented by neural stimulation of the accessory muscles of respiration, i.e. the **sternocleidomastoid** and **scaleni muscles**, similarly lying deep to the site of ligature application, contributing to the thoracic cage becoming fixed in a state of inspiratory paralysis. This latter effect, as described in that section of the Discussion chapter dealing with an analysis of the physiological functions at play, is brought about by initiation of the **dynamic and static stretch reflexes** occurring in these muscles on application of a compressive or tensile stimulus.

Compression of the **carotid arteries**, on the other hand, results, as shown, not only in major damage to these vessels and their accompanying veins, but, in addition, must produce a dramatic element of cerebral ischaemia with ensuing loss of consciousness. It thus appears that loss of consciousness is the critical factor for it is the state when the victim is no longer able to save himself or herself. Without loss of consciousness survival may occur, but with it, death becomes inevitable.

The question then arises :- what is the cause of unconsciousness? In physiological terms, carotid artery occlusion induces rapid unconsciousness, i.e. within 11 to 12 seconds, resulting ultimately in death. In other words, the sudden application and unremitting pressure of the ligature must inevitably result in death. On the other hand, the sudden application of a ligature with consequent vagal nerve compression may produce instantaneous cardiac arrest with cessation of blood flow to the brain and resultant loss of consciousness. This event would produce unconsciousness in less than the time period of 11 seconds of carotid artery occlusion (although the brain continues to survive for several minutes thereafter despite cessation of heart beat). If, however, unconsciousness is

contributed to by **phrenic nerve** compression, it would not be instantaneous as shown by the fact that one can normally hold one's breath for several minutes (as underwater swimmers do) and unconsciousness does not supervene either instantaneously or within 11 seconds. In short, unconsciousness would not occur within 11 seconds in the case of compression of the **phrenic nerve** unless a more critical factor supervenes.Thus, the rapidity of onset of unconsciousness appears to be the critical factor in determining the progression to ultimate (and inevitable) death. Moreover, as pointed out in the Materials and Methods chapter, the **carotid arteries** in several tested cases would not allow the passage of a probe through the obstructed arteries beneath a tightly applied ligature. This obstruction would, therefore, appear to be the initiator of the deadly unconsciousness factor, although respiratory arrest would be compounded by **neural** and **muscular** factors.

While in this thesis the principles of **dimensional analysis** i.e., the breaking down of a complex phenomenon into its component parts have been applied, the principles of **integrated analysis** i.e., the combining and synthesis of separate parts into a whole have also been attempted. In essence, while it is suggested that the **neural elements** play a pivotal role in the hanging process due to the neural effect on both brain and heart as a result of autonomic nervous system stimulation and the function of the **phrenic nerve** in respiration, it appears that multiple factors, acting in concert, simultaneously or in rapid sequence to one another, all play a role in contributing to death causation in the hanging process.

DECLARATION

I declare that this is my own, unaided work. It is being submitted for the degree of Doctor of Philosophy in Medicine in the University of the Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination in any other University.

Jacob Joseph Moar

May, 2011.

DEDICATION

To my supervisor, Professor J. C. Allan, without whose support and encouragement this thesis would not have been possible.

PREFACE

A GUIDE TO THE READER

Originally, the author, a forensic pathologist, performing numerous autopsies in the context of medico-legal death investigation, noticed that in cases of suicidal hanging, the cause of death could seldom be attributed to fracture of the hyoid bone or hyoid-larynx complex, the latter having been extensively studied and reported on in the forensic literature. Although the general view amongst forensic pathologists is that the cause of death in suicidal hanging is asphyxial in origin, this seemed to run counter to common observation being, therefore, counter-intuitive.

This simple observation prompted the author to seek for possible, more subtle, causes for the rapid onset of unconsciousness and subsequent death. Clearly, since the hanging ligature was always placed around the neck, it seemed that the onset of the deadly events (unconsciousness and death) would originate either in an anatomical structure in the neck or be initiated by some physiological event in the neck.

In the absence of fracture of the hyoid bone (in the present series not a single case showed damage to the hyoid bone or hyoid-larynx complex) as evidence of airway compression which could, in large measure, account for the victim's death, the author set about looking for more subtle or obscure reasons for the death of the victim in these cases. While this does not negate the possibility that airway obstruction could occur, but, if it does, it must, unlike the case in manual strangulation, be the exception rather than the rule. The possibility of causes other than asphyxia causing the death of the victim thus necessitated that the neck region

of the hanging victim be examined and dissected at medico-legal autopsy with the aim of finding a possible neurovascular cause of death.

The dissection of the neck region was performed in a very careful and delicate manner lest subtle patho-anatomical changes be overlooked. Dissection was further performed in this manner so that possible procedural damage or dissection technique could not be confused or mistaken with pathology which could have occurred as a result of the application of a ligature. The main objective of the dissection was, therefore, to preserve 'as clean' a field of action as possible.

In the so-called 'bloodless dissection' of the neck, and especially of the carotid artery region, performed in as gentle a manner as possible, it soon became evident that there was haemorrhagic infiltration of the surrounding tissues. This led to the careful examination of the carotid vessels themselves.

To be sure of the type of injury which the carotid vessels might sustain, the carotid sheaths and their contents were removed on both sides of the neck, and fixed in buffered formalin. After fixation, the specimens were dissected and divided so that the three carotid vessels were isolated. These were then dehydrated, embedded in paraffin wax and sections cut at a thickness of 5 microns. The mounted sections were then stained with haematoxylin and counter-stained with eosin.

Since the examination of the arteries was to be microscopical, the author decided to attend a six-week's course in the technology of processing tissues and in section cutting. Prior to the examination of the 'hanging' arteries, it was also decided to examine the arteries of five 'non-hanging' cases to establish clearly the histological features of uninjured carotid arteries.

The reason for this was two-fold. Firstly, it was felt necessary for the investigator to have a clear idea of the histology of a normal carotid artery as it

appears in a ‘non-hanging’ case for comparison with a damaged artery. Secondly, it was done to determine if any pathological changes could have occurred, simply from the fact of death and from the time between death and the post-mortem examination, i.e. the post-mortem interval.

Each of the 30 slides of the so-called ‘normal’ arteries and the 300 slides of the ‘hanging’ cases were then carefully examined and detailed notes kept of the findings. To provide standardisation and eliminate observer bias, all slides were examined in conjunction with the supervisor. In this way, any pathological findings noted in the tissues could, in all likelihhod, be attributed to the presence of a ligature and the attendant compressive and tensile forces taking place during the hanging process, and not to extraneous or ‘man-made’, i.e. artefactual factors.

While the author was originally alerted to the arteries within the carotid sheath, it became inevitable that with so detailed an anatomical examination of the tissues, other, possibly relevant, structures would be revealed. These comprised the nerves of the region and their ganglia, as well as the carotid bodies and their surrounding accessory ‘mini-glomi’. Accordingly, these structures were also studied and the possibility of their causing unconsciousness and death by various physiological mechanisms was considered in some detail.

Finally, after reviewing the general and specific anatomy of the neck in relation to suicidal hanging, it became clear that the position of the phrenic nerves in the neck could be affected by the constricting ligature. A review of the literature revealed that the phrenic nerves had received no attention up to the present. The author thus felt that it would be unwise to neglect this factor as there was a possibility that constriction or damage to these nerves could cause, or at least initiate, a previously unconsidered respiratory reason for the unconsciousness and death of

the victim. This promoted the study of the phrenic nerves in five additional cases of suicidal hanging.

During the course of these studies, a number of pertinent engineering issues arose and which the author had the opportunity and was able to discuss with senior members of the Faculty of Engineering at the University.

Despite the original evidence that the most likely cause of unconsciousness and death was due to compression and damage to the main blood vessels supplying the brain and the cerebral ischaemia attendant theron, a number of ancillary, neural factors emerged during the study. Since it seems possible that these latter factors may have had a role to play in death causation in victims of suicidal hanging, these additional neural factors were also considered in the present study.

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LIST OF ABBREVIATIONS IN FIGURES AND TABLES

A= adventitia

AA = adventitial avulsion

AF = avulsed fragment

AG = accessory glomus

AGAS = accessory glomal adventitial separation

AGH = accessory glomal haemorrhage

AGID = accessory glomal internal dehiscence

AGVC = accessory glomal vascular congestion

AH = adventitial haemorrhage

AM = adventitio-medial

AM= arterial media

A-MS = adventitio-medial separation

AS= adventitial separation

AT = adventitial tear

AT-mt-M = adventitial tear extending to the middle-third of the media

AT-ot-M = adventitial tear extending to the outer-third of the media

ATF = avulsed tissue fragments

AW = arterial wall

B = belt (Table II)

BL = bootlace (Table II)

CB = carotid body

CBID = carotid body internal dehiscence

CBH = carotid body haemorrhage

CBH = carotid body haemorrhage

CL = cloth (Table II)

CBT = carotid body tearing

CCTR = complete circumferential transverse rupture

CTD = connective tissue disruption

CTH = connective tissue haemorrhage

CTT = connective tissue tearing

CTTD = connective tissue tearing and disruption

DSCM = disrupted sternocleidomastoid muscle

EA = endothelial elevation

EAV = empty arterial vessel

EE = endothelial elevation

EL = empty lumen

F = fixed knot (Table II)

FA = firm apposition

G = ganglion

GC = ganglion cells

GH = ganglionic haemorrhage

GID = ganglionic internal dehiscence

GT = ganglionic tearing

H = haemorrhage

HI = haemorrhagic infiltration

I = intima

I = indentation

IAP = intimal atheromatous plaque

ID = internal dehiscence

IE = intimal elevation

IEL = internal elastic lamina

IELD = internal elastic lamina dehiscence

IF = intimal fragment

IF = intimal folds

IGC = intraganglionic congestion

IH = interstitial haemorrhage

IS = intimal split

ISA = Indigenous South African

IT= intimal tear

IT-it-M = intimal tear extending to the inner-third of the media

IT-mt-M = intimal tear extending to the middle-third of the media

IT-ot-M = intimal tear extending to the outer-third of the media

ITD = interstitial tissue disruption

IW = insulated wire (Table II)

L = ligature (Table II)

L = lumen

L = left (Table II)

LCC = left common carotid artery (Tables III, IV and V)

LEC = left external carotid artery (Tables III, IV and V)

LIC = left internal carotid artery (Tables III, IV and V)

LIJV = left internal jugular vein

LMI = linear microtome incision

LMT = linear microtome tear

LN = lymph node

LT = lower third of neck (Table II)

M = media

mT = microTear

MF = fenestration of the media

MMF = multiple fenestrations of the media

MT= tear of the media (muscular layer) of the vessel wall

MT = middle-third of the neck (Table II)

N = nerve

NC = neural congestion

ND = neural dehiscence

NE = neural element

NH = neural haemorrhage

NID = neural internal dehiscence

NT = neural tearing

O = occiput (Table II)

PAT = periadventitial tissues

PN = phrenic nerve

PNS = perineural separation

R= right (Table II)

RC = red cells

RCC = right common carotid artery

REC = right external carotid artery

RIC = right internal carotid artery

RIJV = right internal jugular vein

RP = rope (Table II)

S = slip knot (Table II)

S = single (Tables III, IV and V)

SAM = scalenus anterior muscle

SEC = subendothelial cleft

SAED = South Africans of European Descent (Table I)

SAID = South Africans of Indian Descent (Table I)

SAMD = South Africans of Mixed Descent (Table I)

SCM = sternocleidomastoid muscle

SV = subclavian vein

T = tear

TA = tear of the adventitia

T-it-M = tear extending to the inner-third of the media

T-mt-M = tear extending to the middle-third of the media

T-ot-M =tear extending to the outer-third of the media

TA-mt-M = tear of the adventitia extending to the middle-third of the media

TA-ot-M = tear of the adventitia extending to the outer-third of the media

TB = tissue bridge

TD = tissue disruption

TF = tissue fragment

TFSAM = torn fragments of scalenus anterior muscle

TI-mt-M = tear of the intima extending to the middle-third of the media

TT = tissue tear

U = unknown (Table II)

UT = upper third of the neck (Table II)

V = vein

VC = vascular congestion

VPC = vascular plane of cleavage

VR = vascular rupture

VV = venous valve

X = multiple (Tables III, IV and V)

CHAPTER 1

THE HISTORICAL, RELIGIOUS AND SOCIOLOGICAL ANTECEDENTS OF LEGAL MEDICINE AND ITS INFLUENCE ON JUDICIAL AND SUICIDAL HANGING

Introduction and Definitions of Terms

Legal medicine may appropriately be defined as the application of medical knowledge to the administration of justice and the furtherance of law. [1] Various alternative terms are also applicable in this context. These include forensic medicine, (the medicine of the forum, or the law courts), and medical jurisprudence, the general legal aspects of medicine. These latter terms are sometimes used in a more restrictive sense, forensic medicine being the medical aspects of law while medical jurisprudence refers to the legal aspects of medicine. On the other hand, the two terms are sometimes used interchangeably and this practice extends to the varying ways in which different authors express themselves.

For example, Vanezis, [2] a noted British author, very succinctly and concisely defines hanging (suspension), in pathological terms as a compressive neck injury in which the weight of the body constricts a ligature around the neck. This definition, having the great merit of both brevity, clarity and simplicity, contrasts with the broader and more cumbersome definition by that of an earlier British author Polson, [3] who defined hanging as: "Constriction of the neck as a result of suspension in such a manner that the weight of the body, or a part of the body, of the victim pulls upon the ligature."

This more encompassing definition by Polson does not, of course, make reference to modification of the mechanism of hanging and its subsequent effects on the pathology according to the position of the knot (if there is a knot) or the ligature. The ligature itself may lie over the lower, middle, or upper thirds of the neck, i.e. zones I, II, or II with corresponding effects on the underlying anatomical structures involved. As regards the knot, this may be either a fixed or sliding one, the latter being one where no tied knot is present but rather the rope running through a looped segment of itself. In this case, that part of the neck on the side opposite to the “knot” is subjected to greater compressive force than that on the side of the” knot”, with a gap between the sliding loop and the underlying neck. In other words, the compressive force is exerted in a more unilateral manner and, in this latter instance, one would expect to find confirmatory pathological evidence of this in the form of haemorrhage and/or micro-lacerations confined to or more prominently evident on one side of the neck than on the other.

It is, furthermore, possible to classify the type of ligature e.g. cloth, belt, rope etc. and whether a knot or buckle was present and its position, providing these have been submitted with the body to the mortuary. In not a few instances, this was not possible in the present study due to the ligature having been removed at the scene. This was effected either by the paramedics, who are unaware of the medico-legal significance of the ligature, or by the immediate family, the latter in the occasional attempt to hide the true manner of death in order to negate non-payment of an insurance policy in the event of the death being suicidal.

This inclusion by Polson of "a part of the body" in his definition of hanging carries forensic significance in the context of suicidal hangings in prison where the victim will use any available point of suspension at hand such as a doorknob or other

such object in order to carry out the hanging. What Polson means is that from a low point of suspension such as a doorknob or bedpost only the upper part of the body or part of the trunk and upper limbs are suspended rather than including the whole body with complete suspension of the lower limbs as well. In this case, only a part of the mass of the body brings pressure to bear on the ligature. It is important to realise, therefore, that contrary to the layman's conception of hanging, the point of suspension need not necessarily be one high enough to include lifting of the feet from the floor.

By contrast, Knight, [4] defines hanging as: "a form of ligature strangulation in which the force applied to the neck is derived from the gravitational drag of the weight of the body or a part of the body." This latter definition is of interest not only in that Knight similarly includes the phrases "part of the body" and "weight of the body" in his definition but that it opens the door to an exploration of the biomechanical, tensile and compressive forces acting on the neck, its contained neurovascular bundles and, more specifically, its vascular channels.

Twisting of a ligature with the body in a recumbent or sitting position will produce similar effects, the most famous example perhaps being that of the dancer Isadora Duncan, whose long scarf became caught and entangled in the rear wheel of the motor vehicle in which she was sitting. Rotation of the wheel then exerted both a compressive and tensile force on the neck, with, in addition, an element of hyperextension of the cervical spine and its attendant dislocation. This latter element of hyperextension of the cervical spine would occur as a consequence of the rotational force exerted by the spinning wheel of a motor car but would not normally be expected to occur in the usual suicidal hanging, unless, of course, by convulsions and thrashing of the limbs, the body rotates.

Twisting, as such, is not, however, an essential element of the hanging process. Rotation of the body as a consequence of the drop with gravitational force as applied in judicial hangings, where the object appears to be to “break the neck”, will certainly add an element of bruising to the soft tissues of the neck, its ligamentous structures and its contained neurovascular bundles. Keelhauling, a punishment practice employed in the British and Dutch navies in the seventeenth and eighteenth centuries in which the body of the offender was dragged under the keel of the ship, also probably constituted the application of both tensile and rotational forces (if the neck became entangled in the rope), together with an element of aspiration of water and drowning.

The Historical Antecedents of Legal Medicine

Legal medicine, [1] a fascinating study in its own right, contains several, although infrequent, references to both hanging and suicide over the course of its long history. While the oldest known legal codes, such as those of Babylon, Egypt, Persia, China and India make no specific references to hanging, they do, however, deal clearly with malpractice, setting out the criminal and civil liabilities of the "healer" as well as the obligations incumbent upon the witness in court. These “healers” were often priests.

Although well-intentioned and obviously constituting early attempts at administration of justice, some of these provisions are inadvertently and unintentionally humourous. In India, for example, the drunk, fatigued, insane or those with defective senses of vision and hearing were not permitted to be witnesses in court.

Later tracts, however, such as the Visigothic Code of the fifth century, itself bearing resemblance to the earliest known law code, i.e. that of Hammurabi of Babylon, dating from about 2200 B.C. (although a Chinese Materia Medica providing information on poisons dates from about 3000 B.C.) imposed restrictions on the medical man, highlighting the medical mores of the time. Thus, a medical practitioner might not bleed a married woman except in the presence of her relatives lest he commit adultery with her nor might he visit a prisoner lest he give him poison thereby defeating the ends of justice. These latter might be interpreted as a reflection on the ethics and public perception of the healing profession at the time.

In classical antiquity both Greece and Rome maintained and continued the earlier traditions of imposing restrictions on the medical man. As was the preceding legal practice in Persia, the procuring of an abortion in Greece carried criminal liability while both Greece and Rome classified wounds and discussed their lethal nature.

Herophilus of Chalcedon, a Greek, drew attention to the hyoid bone. He did not, however, mention the medico-legal significance of this bone, fractures to which play so pivotal a role in the diagnosis of strangulation. Of course, no reference to Greek medicine is adequate without allusion to Hippocrates (460-355 B.C.), whose Hippocratic Oath imposed restrictions on the practitioner including the promise not to administer poisons or procure an abortion. The philosopher Aristotle (384-322 B.C.), also pronounced on medical matters, not only fixing the viability of the foetus at 40 days (of legal relevance in determining whether an induced abortion had occurred before or after this date), but also advocated abortion before 40 days as a means of population limitation.

In Rome, the first matter of medico-legal interest dates from the time of Numa Pompilius at about 600 B.C. This document prescribed the performance of caesarean section on all women dying during childbirth in an attempt to save the life of the child and burial was expressly forbidden unless this was done. Both the great Roman generals Scipio Africanus and Julius Caesar were delivered by caesarean section, the latter lending his name to the procedure. Caesar thus perpetuated and immortalised his name not only in the annals of military history by flouting the authority of the Roman Senate in crossing the Rubicon, but also in obstetrics.

The Lex Aquillia of 572 B.C. specifically dealt with the lethal nature of wounds. The next most important promulgation was found in the XII Tables of 449 B.C. which constituted a binding legal code. These Tables set a period of 300 days as the extreme limit of pregnancy, thus barring legitimacy if the father had been dead or absent for a longer period. Interestingly, this period is identical with that specified in the Code Napoleon, formulated more than 2000 years later.

The promulgations of the XII Tables were followed by the Lex Cornelia of Sulla (138-78 B.C.), which not only heavily punished the inducement of an abortion but also decreed that a physician causing the death of a patient was to be exiled or executed. More specifically, Pliny the Elder (A.D. 23-79) wrote of suicide, as well as of sudden natural death. Hanging, however, appears to have been an infrequent occurrence, otherwise reference to it would have appeared in the law. With regard to suicide, in Rome, a highly legalistic, organised and militaristic society, this was, in fact, not only honourable, but expected of a defeated general falling on his sword as occurred to Anthony after his defeat by Octavian.

With the conquest of Rome by the Visigoths and the establishment of the Eastern Empire centred on Constantinople, the Emperor Justinian unified the various

laws into a codex with Justinian law, specifically dealing with a number of medico-legal issues including poisoning and, once again, abortion.

Leges Barbarorum[1]

The Roman Empire, and Rome itself, was overrun in the fifth century by the Visigoths who continued moving west, establishing one capital at Rennes le Chateau in France and another at Cartagena in Spain. The former, Rennes le Chateau, is a site prominently associated with modern historical speculation of whether Jesus of Nazareth survived the crucifixion, a form of death by suspension.[5] This controversial theory postulates that the Holy Grail in fact, refers to the blood of Jesus, which gave origin to the French Merovingian dynasty and who are therefore purported to be blood descendants of Jesus.[6] Be that as it may, these Germanic tribes, regarded as barbarians and the destroyers of Roman civilization, were, in fact, the first to clearly lay down in statute that medical experts (according to the standards of the time), be used to give opinions in matters pertaining to wounding arising in the Visigothic courts. These laws, the Lex Alemannorum, laid down between the fifth and tenth centuries, classified injuries in great detail giving precise descriptions and with clear reference being made to using medical opinions in court.

Charlemagne (742-814 A.D.), in trying to restore the Roman Empire, attempted to reintroduce uniformity to the disparate laws of his widespread dominion. He thus instructed his bishops to draw up the Capitularies, these latter giving specific orders to judges to rely on medical evidence in court. This dealt particularly with wounds, blows and, once again, questions of suicide

The Assizes of Jerusalem[1]

This was a code of laws promulgated for the Kingdom of Jerusalem at the behest of the crusader, Godfrey de Bouillon, in 1100 A.D. Because the crusaders were drawn from the ranks of the aristocracy and their followers from the Christian countries, these Assizes were based on those laws already extant in Europe and, in particular, France. These laws specifically provided for the courts to order a medical examination by a court-ordered physician. In view of their European and, in particular, their French derivation, it is likely that such medico-legal examinations also prevailed in Europe at that time.

Early Italian, German and French Developments[1]

It was in Italy that legal medicine first appeared as an entity in its own right. Roger the Second, of Sicily, placed medical practice in 1140 A.D. under the law, requiring physicians to take examinations. This was followed in 1224 A.D. by the edicts of Frederick the Second, who ordered that physicians take a public examination based on the teachings of Hippocrates, Galen and Avicenna after having studied for seven years and serving one year's apprenticeship. Not only did Frederick prescribe structured courses of teaching, examinations, a qualification, and a high standard of medical ethics,but he also ordered that every five years a human body should be dissected in public. This practice was subsequently followed throughout Italy. Probably the most famous city in Italy associated with legal medicine was Bologna. From the end of the thirteenth century, many reports by doctors from this city dealt with the examination of mortal wounds and poisonings. As in previous centuries,

these laws and practices spread to other cities and, interestingly, some of these reports are still extant. Other than Germany, however, there appears to be no specific mention made of "hanging".

Italy

In Rome, Pope Gregory IX in 1234 A.D., ordered the compilation of the Nova Compilatio Decretalium, a collection of the decisions of the Popes and Councils up to that time. Their medico-legal relevance lies in their dealing with caesarean section, abortion, issues of medical evidence and crimes against the person but, however, with no mention of hanging.

A particularly intrusive issue offered by medical evidence was that of the Proof of Congress in cases of alleged impotence. In this dubious practice, an experienced matron was instructed to observe a husband and wife who attempted to perform sexual intercourse. The matron then reported on what had taken place. At a later date, three physicians, three surgeons and three midwives replaced the matron in observing the proceedings. Following condemnation by theologians, lawyers and physicians, this scandalous practice was gradually phased out, but was only abolished in France, a country with a longstanding reputation of interest in matters carnal, in 1677 A.D.

Further statutes followed in subsequent centuries. For example, Pope John XXII in the fourteenth century reminded the ecclesiastical courts of the need for medical expertise. These reminders were contained in many medico- legal documents dating from the end of the fourteenth century. Many of the documents are still in existence. Other popes, notably Pope Gregory XII, in 1582 A.D. re-

established the doctrines of Gregory IX in making medico-legal expertise obligatory in matters such as wounding, abortion, and poisoning.

France

The bishops of Maine and of Anjou were reputed to have had medical experts in their service from the eleventh century with “surgeons-general” in Paris from the twelfth century. Norman laws of 1207 A.D. onwards similarly made reference to such surgeons-general as did the book of common law of St Louis in 1260 A.D. with attested surgeons being appointed in the major French towns. Despite the presence of these sworn surgeons, no autopsies were performed. Only external examinations were made, calling into question the value of such examinations. The right of autopsy was, however, granted to the Faculty of medicine at Montpellier by the Pope in 1374 A.D. Again, information relating to suicide by hanging is singularly absent.

The enactment of Francois the First provided a link between medicine and law although this had been preceded by a number of years by the Caroline Code of 1532 A.D. in Germany. Successive kings added further provisions with, for example, Henry the Second of England in 1556 A.D. condemning to death any woman who had concealed her pregnancy and the fetus subsequently being found dead. This was, however, condoned if the surgeon's report showed the fetus to have been either pre-term or stillborn.

Germany

Under the auspices of George, Prince Bishop of Bamberg, a comprehensive and systematic code of legal procedure was drawn up at Mainz in 1507 A.D. This laid down that medical evidence be called in all cases of apparent non-natural death in which violence appeared to be instrumental. A quarter of a century later, in 1532 A.D., almost all its provisions were incorporated in the Constitutio Criminalis Carolina, the code of the Emperor Charles the Fifth of Germany. This comprehensive code incorporated statutes dealing with hanging, suicide, murder, infanticide, procurement of abortion, malpractice, severity of wounds and poisoning. Further ordinances followed in 1535 A.D. and 1582 A.D. but it was not until 1580 A.D. that specific laws were passed in Germany prohibiting shepherds and herdsmen from practicing obstetrics on human females. This doubtless contributed to enhancing the standing and image of this speciality at the time.

China

The "Instructions to Coroners", published circa 1250 A.D., was the classic Chinese work in the field. Far ahead of anything else existing in the world at the time, it was republished with amendments up to the end of the nineteenth century. Its five volumes dealt with hanging, suicide, strangulation, drowning, burning, inquests, abortions, poisoning and the signs of death. Inquests were obligatory in all cases where the cause of death was suspect.

Late Sixteenth Century Europe and beyond

In 1575 A.D., the famous surgeon, Ambroise Pare, produced a book dealing with hanging, drowning, lightning, abortion, ante- and post-mortem wounds and poisoning by carbon monoxide and corrosives. In addition, the last five years of the sixteenth century saw the emergence of notable works dealing with legal medicine. A work of great importance, published in 1597 A.D., was the Methodus Testificandi (Method of Testifying), of Codronchius, a physician of Imola. This dealt with wounds, poisoning and sexual matters. An even greater work, published in the following year, was that of Fortunatus Fidelis, of Palermo, entitled De Relationibus Medicorum (About the Relations of Physicians), this being the first great systematic treatise on legal medicine. It dealt with suffocation, wounds, lightning, poisoning and medical errors. Fortunatus Fidelis advocated complete medico-legal autopsies and interestingly, described a modern patho-physiological concept of great significance in forensic pathology at present, namely, that of the sudden cardiac death syndrome.[7]

From the seventeenth century onwards, numerous tracts dealing with wounds, poisoning and other issues of medico-legal relevance were published. Two in particular are worthy of mention, namely, that of Johannes Bohn in Germany and that of Antoine Louis in France.

Bohn produced two notable works. The first, De Renunciatione Vulnerum, was published in 1689 A.D., dealt with the differentiation of ante- from post-mortem wounds (an issue which sometimes arises in hanging when an attempt is made to conceal the true manner of death), as well as strangulation (always a possibility in a simulated hanging). The second, more comprehensive work, published in 1704 A.D., advocated complete medico-legal autopsies as well as objecting to untutored

midwives giving expert opinions. Bohn thus pursued scientific objectivity rather than following the collective conventions and practices of his time. This required no little measure of courage considering the fact that Giordano Bruno was burned at the stake for suggesting that the earth revolved around the sun rather than keeping to the conventional viewpoint that the sun revolved around the earth.

In France, the most notable figure during this period was Antoine Louis and who was the first to teach legal medicine in that country. He wrote on drowning in 1748 A.D., on the signs of death in 1752 A.D., and on the examination of the hanged person in 1763 A.D., including the differentiation between suicidal and homicidal hanging, in which crucial differences exist to the alert pathologist. During his time as a medico-legal expert to the courts for over 30 years, he was able to show that a young man's death had been suicidal rather than homicidal. Unfortunately, the father had already been wrongfully executed for the death. Louis's contribution, however, together with the intervention of the famous writer Voltaire, made it that much more difficult for such a miscarriage of justice to ever occur again.

Finally, no description of nineteenth century German and British forensic medicine is complete without mentioning the writings of Johann Ludwig Casper and Alfred Swain Taylor.

Casper (1796-1864 A.D.), published his classic work Praktisches Handbuch der Gerichtlichen Medizin in 1856 A.D. and which went through nine editions being translated into English in 1860 A.D. under the auspices of the New Sydenham Society. [8] In an analogous manner, the writings of Taylor on legal medicine were similarly prolific. His first work appeared in 1836 A.D., while his Principles and Practice of Medical Jurisprudence, [9] updated and going through several revisions, contains a wealth of illustrative case histories and knowledge

although, of course, superseded by other British authors such as Polson, Vanezis, Knight and, of course, Sir Sydney Smith whose book, **Mostly Murder**, similarly contains numerous case histories, all related in a delightfully humorous manner. Lastly, one should, perhaps, mention Camps, Simpson and the famous Bernard Spilsbury, the latter taking his own life by means of carbon monoxide intoxication.

The Religious Views of Suicide

Christianity

The New Testament documents seven suicides, most notably in Mathew 27: 3-5, describing the suicide of Judas Iscariot, who betrayed Jesus.

According to the theology of the Catholic Church, death by suicide is considered a grievous sin, the chief Catholic argument being that one's life is the property of God and a gift to the world. Hence, to destroy that life is to wrongly assert dominion over what is God's and is a tragic loss of hope. Conservative Protestants (Evangelicals, Charismatics and Pentecostals) have, in addition, often argued that because suicide involves self-murder, anyone who commits it is sinning and is the same as if the person murdered another human being.

Islam

Islam, like other Abrahamic religions, views suicide as one of the greatest sins, utterly detrimental to one's spiritual journey. A verse in the fourth chapter of the

Qur'an, An Nissa (The Women) instructs: "And do not kill yourselves, surely God is most Merciful to you." (4:29).

Most Muslim scholars and clerics consider suicide haraam, i.e. forbidden, including suicide bombings and often cite the aforementioned verse in the Qur'an as a clear commandment forbidding suicide. Some Islamic scholars even classify suicide as an unpardonable sin, the equivalent of eternal sin in Christianity.

The prohibition of suicide has also been recorded in authentic statements of Hadith, the sayings of Muhammad. For example: "He who commits suicide by throttling shall keep on throttling himself in the Fire (Hell), and he who commits suicide by stabbing himself, he shall keep stabbing himself in the Fire (Hell)." This allusion to throttling in all likelihood refers to ligature strangulation by oneself, i.e. hanging.

Judaism

Suicide is forbidden by Jewish law, Judaism traditionally viewing suicide as a serious sin. It is not seen as an acceptable alternative, even if one is being forced to commit certain cardinal sins for which one must give up one's life rather than sin. Assisting in suicide and requesting such assistance (thereby creating an accomplice to a sinful act) is also forbidden. Just as in Hinduism, it is accepted that the souls of individuals who commit suicide are denied entry to the afterlife, remaining wandering in this world.

Hinduism

In Hinduism, committing suicide is often considered equally sinful as murdering another, with the possible exception of suttee, a now defunct Hindu practice whereby a widow immolated herself on her husband's funeral pyre. Hindu scriptures hold that to die by suicide (and any type of violent death) results in becoming a ghost, wandering the earth until the time one would have otherwise died, had one not committed suicide.

Buddhism

According to Buddhism, the past acts of an individual heavily influence what the person experiences in the present. Present acts, in turn, become the background influence for future experiences constituting the doctrine of karma. Intentional action by mind, body or speech have a reaction or repercussion which is the cause of differences and conditions one comes across in the world.

For Buddhists, the first precept is to abstain from the destruction of life, including oneself. Thus, suicide is clearly seen as a negative form of action. However, unlike Christianity and other Western religions, Buddhism does not condemn suicide but rather states what the reasons for it are. An Asian ideology similar to the Japanese seppuku (hara-kiri), continues to influence oppressed Buddhists who then choose the act of honourable suicide as a form of protest. As a latter-day example, Tibetan monks have chosen suicide in order to protest the Chinese occupation of Tibet and China's human rights violations against Tibetans.

Jainism

Jainism is one of the few religions that does permit suicide. Adherents have been known to starve themselves to death although there is no record of application of any other violent means due to heavy insistence on non-violence.

Religious cults

In contrast, some religious cults not only permit suicide but actually encourage their members to commit suicide. This is rooted in their belief that suicide is an escape pathway for a soul to another, better world.

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CHAPTER 2

THE EPIDEMIOLOGY OF SUICIDE BY HANGING

A REVIEW OF THE LITERATURE

Introduction

Hanging is one of the most frequently used methods of suicide worldwide,[1] with an estimated 815,000 people having killed themselves in the year 2000 alone.[2] During the last 45 years, suicide rates have increased by 60% globally, making suicide amongst the three leading methods of death of those aged between 15-44 years.[3] On average, 2233 people commit suicide daily i.e. roughly one person every 40 seconds,[4] with age- and gender-specific cultures of suicidal behaviour.[5-7] For example, in Hungary, Norway, Ireland and Finland, an increase in suicide has been observed in the younger age-groups (10 to 14 and 15 to 19) while amongst the indigenous native American Indian cultures of Canada and the Maori culture of New Zealand, a similar increase has been observed. In particular, the last 40 years have seen an increase in hanging suicides amongst young males in Australia, [8] New Zealand, [9] South Africa, [10] and elsewhere.

England and Wales

In England, there occur around 2000 suicides by hanging annually, with this modality being the most commonly used method. The prevalence of hanging as a means of suicide in England dates back at least to medieval times, when it accounted

for about half of all suicides.[11] While suicide by hanging has increased markedly in males under the age of 65 years,[12, 13] increases have also been seen amongst young (under 45 years) females. However, rates amongst older females have been declining since the mid-1980s. In all other age and gender groups, hanging accounts for 80-90% of deaths in the so-called "asphyxial" categories such as strangulation, choking, or suffocation by means of a plastic bag.

While national suicide prevention strategies in England and internationally place emphasis on restricting access to commonly used methods of suicide as a means of reducing suicide rates, [14] most authors agree that this approach is generally not possible for hanging suicides. This is because the ligature points and ligatures commonly used are universally available. These comprise rope, belts or nylon cord while the most common ligature points are beams, banisters, hooks, door knobs, and trees. An exception to this is suicide in institutional settings such as police custody, prisons and hospitals, where great effort has gone into the construction of so-called "safer cells", designed to be ligature-free. [15] The Safer Custody Group of HM Prisons in Great Britain have sought to strike a balance between the need for removal of ligature points and the construction of a humane environment. Specific criteria have been laid down for furniture and sanitary ware so that ligatures cannot be attached. Bars in "safer cells" are covered with transparent polycarbonate sheeting and a ventilator inserted. Lights are recessed, pipes covered, and the bed base is fixed. Garment hanging space is constructed so that the rail is collapsible or a shelf lip is provided for hanging garments using cardboard hangers. Shelving is fitted flush against walls, button water controls are used on washbasins instead of taps and a button flush is used on toilets. Toilet seats, a potential ligature point, are not used but the pedestal is covered with an acrylic resin. In addition, fixed plugs on washbasins

have been removed as it was found that these could be used as a ligature point. Individual cells are tested with a fishing line and if the line snags behind shelves or pipes, gaps are filled with an anti-pick filler material. The upper hinge of the cell door has also been modified to prevent its use as a ligature point. Prison deaths, however, comprise only about 10% of all hanging suicides.[16, 17]

General population studies in England have shown the place of death in approximately three-quarters of the cases to be the person's own home with deaths in custody or hospital accounting, as indicated, for about 10% and the rest occurring in public places. While there were 172 prison suicide deaths in England and Wales in 1999/2000, 159 (92%) of these were by hanging, [18] indicating the great availability of means to effect this method of suicide. Suicide by hanging accounts for a similarly high proportion of deaths in custody in the United States, Australia, Canada, and Holland. [19-25] Deaths by suicide in police custody in England and Wales is much lower than those in prisons. [26]

With regard to suicide by hanging in psychiatric wards, 234 suicides occurred in the four years from 1April, 1996 with three-quarters of these (175/234) being due to hanging. Such deaths accounted for around 3% of the 6554 suicides by hanging in England between 1997 and 2001. [27] Hanging makes a similar contribution to "in hospital" suicides in other countries. [28, 29]

South Africa

In South Africa, about 10,000 people commit suicide yearly, most of these being young people leading economically active lives. Studies carried out have indicated that the problem of suicide is on the increase with major reasons for committing this

act being mental illness and inability to solve problems.[30] A National Injury Mortality Surveillance System (NIMSS) report showed that hanging accounted for 42.3% of suicidal deaths while firearm injuries accounted for 29.4%, poisoning by drugs and pesticides for 13.6% and carbon monoxide intoxication for 7.1%. More than half of all suicide victims were between 20 and 39 years of age. The ratio of male to female suicides was 4,7: 1,[31] (i.e. approximately 5:1).

A comprehensive survey of the incidence of suicide by hanging was conducted in the Transkei region. This formerly constituted one of the so-called "black homelands" but now comprises part of the Eastern Cape Province. This study showed an increasing trend of hangings by almost threefold from 5,2 per 100,000 to 16,2. [10] The highest incidence of hanging was in the 20- to 29-year age group (32,8 per 100,000) and lowest in those over 70 years of age (2,2 per 100,000). Black males comprised 86.4% far outnumbering females with the incidence for males being 7,8 per 100,000 and that for females 1,2 per 100,000. The ratio of male to female suicide was 6,4:1, (i.e. approximately 6:1). In those less than 19 years of age, the incidence of hanging was 23,8 per 100,000 with the two youngest suicide victims being males aged 9 years. Hangings in the Eastern Cape region are thus equivalent to the global suicide rate of 16 per 100,000. [32]

A particularly interesting feature emerging from this study was the disparity between males and females in suicide rates. One of the most consistent findings in suicides was the lower rate amongst women. [33] The suicide rate in males below 40 years is 7,3 times higher than those over 40 years with hanging being 6.5 times more common in males (7,8 per 100,000) than females (1,2 per 100,000). While the gender ratio for suicide in Sub-Saharan Africa as a whole is 4,7:1, it is higher in this latter study, being 6,4:1. The corresponding gender ratios in other parts of the world are:

Eastern Europe: 4,3:1; United States: 4,3:1; India: 1,2:1, and China: 0,9:1. [34] China is thus the only region where the suicide rate is higher in females.

Australia

The suicide rate for Australia as a whole has remained remarkably constant over the last century (20,6 and 5,5 per 100,000, respectively, for males and females in 1897, and 21 and 5,5 per 100,000 in 1995). What has changed, and dramatically so, is the rate for young males. This has increased from about 10 to 25 per 100,000 since the 1960s. Indeed, the increase for young adult males in Australia is largely accounted for by increasing rates of suicide due to hanging which is the most common method (approximately two-thirds) of suicide amongst Indigenous Australians. In Queensland, for example, while the suicide rate for the period 1990-1995 was 14,5 per 100,000 for whites, that for Aborigines was 23,6 per 100,000. This elevated rate was entirely accounted for by the increased indigenous male suicides which are concentrated in the 15 to 24 year (112,5 per 100,000) and 25 to 34 year (72,5 per 100,000) age-groups. These constitute 3,6 and 2,2 times the rates of these male age-groups for the State of Queensland as a whole, comprising 84% of all indigenous suicides. [35]

Review of the Queensland government reports for the period 1990-1996, for example, indicates that while the number of overall female deaths remained steady, male deaths increased from 10 in 1990 to 36 in 1996. [36] Of all general suicide deaths, 88% were male, with a mean age at death of 26 years. Of these male suicides, 78% were by hanging, which accounted for the overall increase in suicide deaths over this time. Hanging thus constitutes the most common means of suicide in Australia. [37]

New Zealand

Maori male suicide rates for males aged 15 to 24 years peaked in 1989 at 49 per 100,000, and for females in 1986 at 17,3 per 100,000. [38] For the 15 to 19-year-old-male age-group, Maori suicide rates are 35 per 100,000 compared to 27 per 100,000 for non-Maori, the proportions reversing for those aged 20 to 24 years. The rates for Maori females versus non-Maori females aged 15 to 24 are 6 and 4 per 100,000, respectively, the proportions reversing as for males in the subsequent age-group. As in Australia, the disproportionate number of Maori suicides occur in custody. This is accounted for by much higher rates of arrest and incarceration. [39]

Canada

Data from the United States and Canada show a marked increase in suicide rates for males aged 15 to 34 during the 1970s. [40] The National Task Force on Suicide in Canada reported that in the early 1980s, 60% of suicides occurred in the 15 to 24 year age-group with a male to female ratio of 3:1. [41] The proportion of deaths due to hanging has been increasing in Canada in recent decades for both males and females. In a retrospective review of deaths for the period 1987 to 1992 from British Columbia, suicide rates amongst the native North American Indian First Nations people were three-to five-fold higher than for the non-indigenous population. [42] In a similar review for 1988 to 1994 in Manitoba, overall indigenous rates of suicide were 2,3 times higher than for the non-indigenous population, with rates for those aged 15 to 19 years 6,5 times higher. In this study, hanging accounted for over half the deaths. [43]

Hungary

In Europe, [44] the suicide rate amongst children and adolescents is the highest in Hungary, a country where the rate of suicide amongst different socio-democratic groups is traditionally high . While the overall suicide rate in Hungary has shown a dramatic decline decreasing from 45,3 per 100,000 in 1983 to about 30 per 100,000 a decade later, this has not been reflected amongst children and adolescents. In younger age-groups (10 to 14 and 15 to 19 years), no decline has been experienced. On the contrary, the ratio of suicide in young people appears to be increasing.

An Hungarian study has indicated that while a continuous decrease in the number of suicides committed by girls in the younger age-groups can be observed from the mid-1980s, this has not been shown amongst boys and young men. [45] Studies from other European countries, notably Norway, [46, 47] Ireland [48] and Finland, [49] have similarly shown a marked increase in suicide rates among young men.

A further study conducted in the Department of Forensic Medicine in Budapest between 1996 and 1998 comprising 72 cases of suicide involving children and adolescents consisting of 59 (82%) males and 13 (18%) females. [50] The significant finding emerging from this study was not only the overwhelming male preponderance, but that the most frequent modalities of suicide in this gender group were either hanging or leaping from a height into deep water. As indicated by Moar, [51] this latter manner of death carries major medico-legal implications in the context of any associated injuries which may have been incurred. Should a person incur injuries during the fall into the water, i.e. from an abutting rock in a river bed, the question arises as to whether the injuries subsequently noted at medico-legal autopsy

actually occurred prior to, rather than as a consequence of the fall into the water, i.e. is one dealing with a genuine suicide or with homicide. Furthermore, such a person may survive the fall long enough to be washed ashore from a river or lake yet still die of the injuries sustained. Alternatively, sufficient water may be aspirated in this weakened state to cause the person to drown. The question then becomes: what is the true cause of death? This involves a cardinal point in law. While the immediate cause of death may be drowning, the prior cause which initiated the train of events is the original injury, provided that a direct relationship can be shown between the two. What is meant by this? If, for example, a person sustains a gunshot wound of the spine which renders him paraplegic but he nevertheless survives for several years to die of a terminating complication such as pneumonia, the immediate cause of death is the terminating complication. However, the original injury resulting in the terminal complication is the gunshot wound, which thus becomes the proximate cause of death. Once again, this involves a crucial point in law with regard to the apportioning of culpability. The outcome of a court case sometimes depends on this, and it then falls to the medical witness to assess the anatomical, physiological, and haemodynamic consequences of the original injury.

Latter-day South Africa

South Africa as of 2009 is a complex mix of the third and the first world.[52] Between 1994 and 2009 the country changed from being an apartheid state to a democracy in which a wide spectrum of cultural groupings established their place in the nascent society. As with all such radical changes, crime has flourished, and continues to flourish, in an environment where oppressive, but efficient, policing had previously

suppressed much of the criminal activity, at least that affecting the minority population. Hence, in contrast to more stable societies, crime and violence with its unfortunate preponderance of homicide continues to play a major role in death causation.

Over the same period, urbanization has accelerated as part of government policy. In particular, the conurbation of Johannesburg and Pretoria, has grown exponentially in population with a large influx of immigrants, legal and illegal, from neighbouring countries into these urban areas. Many of these urban dwellers live in conditions of extreme poverty. This, coupled with the ready availability of weapons from civil wars in the surrounding countries, specifically Mozambique and Angola, has lead to a society where both homicide and suicide are rife. Homicide by means of both 9 mm handguns and 7.62 mm AK-47 Kalashnikov high-kinetic energy, high-velocity military assault rifles has burgeoned exponentially, while the depressant effects of poverty, unemployment, and personal problems has led to a marked increase in the rate of suicide. [53]

As if the latter were not sufficient cause for concern, the open borders of the country, and its early failure to police imports of illegal narcotic drugs, has made the country vulnerable to a flood of such illicit substances. These have replaced the previously dominant, locally manufactured, drugs of abuse- mandrax and cyclizine-dipinanone. [54, 55] South Africa has thus become a major role player in international drug syndicates, the country forming part of the transportation conduit of illegal narcotics grown, harvested, and manufactured in South and Central America through to South Africa and Nigeria, thence on to Spain (which shares a common language with South America) and the rest of Europe. [56]

ADDENDUM

The Role of the Forensic Pathologist in the South African Legal System and the Legal Statutes Applying

The major role of the forensic pathologist lies in the elucidation of sudden, unexpected, suspicious or overtly criminal deaths or deaths following anaesthesia. In terms of South African law, the medico-legal authorities require notification of any death occurring during the administration of a local anaesthetic, regional anaesthetic, or general anaesthetic or under circumstances where administration of the anaesthetic has been completed, but in which administration of the anaesthetic may be regarded as a contributory factor to death. Hence, any death which is reported under this section of South African law is regarded as a death due to non-natural causes, to be followed by an Inquest. The attending clinicians are not allowed to issue a death certificate in these cases and the case must be referred to the regional medico-legal laboratories for medico-legal autopsy. In addition, no fixed period of time after the administration of the anaesthetic following which an anaesthetic-associated death is no longer considered to be anaesthetic-related has been laid down in law.

The following **legal statutes** apply to the practice of forensic pathology in South Africa:

Section 56 of the Medical, Dental and Supplementary Health Services Act No 56 of 1974 vol. 3 (i.e. the death of a person while under the influence of an anaesthetic or to which the administration of an anaesthetic has been a contributory cause shall not be deemed to be a death from natural causes as outlined in the Inquests Act No 58 of 1959 or the Births, Marriages and Deaths Registration Act No 81 of 1963). Several

other Acts, apart from those already mentioned, affect the discretion of the medical practitioner as to what constitutes a “natural” or “non-natural” death. These are the Human Tissue Act No 65 of 1983, in terms of which the medical practitioner is required, prior to the removal of donated tissue, to decide whether a person is dead or not; the Abortion and Sterilization Act No 2 of 1975, in terms of which the medical practitioner may take a decision regarding the termination of a pregnancy under given circumstances, and the Criminal Procedure Act No 51 of 1977, in terms of which certain evidence from a medical practitioner may be accepted in court even if it is not in the form of an affidavit or solemn declaration.

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CHAPTER 3

THE REGIONAL AND PHYSIOLOGIC ANATOMY OF THE NEURO-VASCULAR BUNDLES AND VASCULAR CONDUITS OF THE NECK

Introduction

Why do the Precordium and Neck constitute Sites of Election, i.e. "Target Sites" in both Homicide and Suicide"

Data indicating the thorax and precordium as a preferential site of election, i.e. a target site for infliction of injury, has emerged from a detailed study of wounding produced by sharp-edged and pointed instruments conducted by the author in Johannesburg.[1, 2] This study, awarded a Master's degree at the University of the Witwatersrand, was subsequently abstracted in Cambridge Scientific Abstracts in association with the University of Southern California.[3] Hard data emerging from the author's study indicate the chest and, in particular, the precordium, to constitute a target area. This is as a result of the natural tendency on the part of the assailant to strike at the heart as his target organ of choice. However, neurological factors in addition to behavioural and instinctual factors, also come into play. In particular, the issue of cerebral dominance plays a major role. Right-handed persons comprise 93% of the population.[4] In these persons, left cerebral dominance occurs in 99%, while in left-handed people left cerebral dominance still predominates in 60%. Hence, more than 95% of the population have left cerebral dominance and, with this preponderance of right-handed persons in the general population, it would be

expected that a knife wielded by a right-handed assailant would tend to strike the left anterior chest wall when assailant and victim are facing one another. [5] However, other factors, such as percentage of total body surface area, also come into play. The chest constitutes 18% of the total body surface area, i.e. almost one-fifth and is, in addition, easily accessible.

Why should the neck, however, constitute such a focal point, comprising as it does only 1% of the body surface area? Putting aside the rhetorical statement that without a neck there would be neither strangulation nor hanging,(except in the suspension of crucifixion), the answer must necessarily lie in recourse to the evolution of man, his anatomy, and his instinctual nature.

Probably the greatest development in mammalian evolution was the long series of adaptations which allowed vertebrates to spread from an aquatic to a terrestrial environment. The ancestors of mammals were primitive amniotes which separated at a very early stage (at least 300 million years ago) from the lineage which gave rise to modern reptiles and birds. In a non-aquatic environment in the absence of a surrounding fluid milieu, a streamlined body permitting undulation no longer carries advantages in locomotion. On land, however, a neck becomes advantageous because the head can now turn to facilitate vision and feeding, without affecting the mechanics of locomotion. In addition, deprived of the buoyancy of water and being subject therefore to greater forces of gravity, the head and body must be supported by the limbs and the development of a neck. In this regard, the nuchal ligament, extending from the anterior thoracic neural spines to the back of the skull and neural spines of anterior cervical vertebrae, is an excellent example of an antigravity mechanism, particularly prominent in large mammals.

In Darwinian terms, man has evolved from the primates, an upright or semi-upright bimanual species. What distinguishes and differentiates man from subhuman primates, however, is that unlike the apes, man possesses a so-called saddle arch. This enables his two opposable thumbs to not only make fine adjustments to gripping, but, of crucial importance in the context of strangulation, to grasp the neck of the victim and produce bimanual compression, accounting for the oval finger pad contusions seen on the victim's neck. Fine finger movement and adjustment, on the other hand, permits the tying of a knot in hanging. As an interesting empirical finding, historical evidence exists of ligature strangulation and ligature preservation. This involves Tolund man, whose preserved remains are on exhibit in the Department of Forensic Medicine in Copenhagen. This involved a case of ligature strangulation 2000 years ago, where the body of the victim was disposed of in a bog in which the acidic milieu enabled preservation, not only of the tissues, but also of the encircling ligature.

Anatomical considerations alone, however, cannot account for the neck being a potential site of election in suicide and homicide. Some, and possibly major, significance must be attached to man's instinctual nature. This latter not only renders grasping as an instinctive reaction, but inadvertently renders the neck, a vulnerable area, an obvious and inviting target.

Unlike the thorax, which may be encased in animal fur (as with Neanderthal man), armour (as in classical Greece, Rome, medieval knights and the Spanish conquistadores), the neck is unprotected. This unprotected area comprises neurovascular bundles, vascular conduits and an airway. These anatomical structures moreover, lie at a height which in an upright creature such as man, is easily reachable

by a bimanual species with the ability to extend the upper limbs in order to instinctually grasp an unprotected and inviting object that presents itself. Compounding the vulnerability of the neck, is the fact that the vital structures such as trachea, nerves and vessels lie both anteriorly (trachea) and anterolaterally (neurovascular bundles and vascular channels). Minimal intervening soft tissue lies between these structures affording protection against the compressive force of an encircling ligature.

Rationale

In view of the fact that this study entails a detailed analysis of the arterial lesions ensuing on hanging, emphasis will be predominantly directed to the anatomy of these vessels, their cervical anatomical relations, their neural innervation and the fascial compartments enclosing them.

The Fascial Planes and Compartments of the Neck

The Superficial Cervical Fascia

This is usually a thin lamina covering the platysma muscle and may contain considerable adipose tissue, more so in females. Like all superficial fascia, it is not a separate stratum but a zone of loose connective tissue between the dermis and deep fascia, and continuous with both.

The Deep Cervical Fascia

This consists of fibro-areolar tissue lying internal to platysma and investing the nuchal muscles. It forms well-defined fibrous sheaths which are condensed around blood vessels. Its superficial (investing) lamina is continuous with the ligamentum nuchae and periosteum of the seventh cervical spine. It thinly covers the trapezius muscle and continues from the anterior border of this muscle as a loose areolar layer to the posterior border of sternocleidomastoid muscle, where it becomes denser. It encloses this muscle and at its anterior margin unites as a single lamina to the midline and so to the opposite side. Adherent to the symphysis menti and the body of the hyoid bone, it is fused with periosteum along the superior nuchal line, the mastoid process and the entire base of the mandible. Between the mandible and mastoid process it ensheaths the parotid gland.

Inferiorly, the deep fascia is attached to the acromion, clavicle and manubrium sterni, fusing with their periostea. A little above the manubrium, it splits into superficial and deep layers which are attached to the anterior and posterior manubrial borders and the interclavicular ligament. Inferiorly between trapezius and sternocleidomastoid, the fascia also has superficial and deep layers. The superficial layer is attached to the superior clavicular border while the deep layer surrounds the inferior belly of omohyoid. Deep to the sternocleidomastoid, it blends with the fascia around subclavius and the periosteum on the posterior aspect of both the clavicle and anterior end of the first rib.

The Carotid Sheath

This, a condensation of cervical fascia, encloses the common and internal carotid arteries, internal jugular vein, vagus nerve and constituents of the ansa cervicalis. Thicker around the arteries, it is peripherally continuous with adjacent loose areolar tissue.

The Prevertebral Lamina

This covers the anterior vertebral muscles, extending laterally on scalene anterior and medius and levator scapulae as a fascial floor. Laterally, the lamina is thin and areolar, ceasing to be a definite layer under trapezius. Superiorly it is attached to the cranial base while inferiorly it descends in front of longus colli into the superior mediastinum, blending with the anterior longitudinal ligament. Anteriorly the prevertebral lamina is separated from the pharynx and buccopharyngeal fascia by an areolar zone, the so-called retropharyngeal space. Laterally this tissue connects the lamina to the carotid sheath and fascia on the deep aspect of sternocleidomastoid.

The Pretracheal Lamina

This very thin lamina, ensheathing the thyroid gland, is attached to the arch of the cricoid cartilage and continues into the superior mediastinum with the inferior thyroid veins.

Surgical Anatomy

The superficial lamina of deep cervical fascia opposes the superficial extension of abscesses, and pus therefore extends laterally. Hence, pus may reach the mediastinum anterior to the pretracheal lamina. However, due to the thinness of the fascia here, it more often "points" above the sternum. Pus behind the prevertebral lamina may extend laterally into the posterior triangle. Alternatively, pus may perforate the lamina and buccopharyngeal fascia, bulging into the pharynx as a retro-pharyngeal abscess.

The Arteries of the Neck

The common carotid, internal carotid, and external carotid arteries provide the major source of blood to the head and neck. These arteries, and their accompanying veins and nerves, all lie in a cleft that is bound posteriorly by the transverse processes of cervical vertebrae and attached muscles and medially by the trachea, oesophagus, thyroid gland, larynx and pharyngeal constrictors. Anterolaterally, the cleft is bound by the sternocleidomastoid muscle and, at different levels, by omohyoid, sternohyoid, sternothyroid, digastric and stylohyoid muscles. The common and internal carotid arteries lie within the carotid sheath, accompanied by the internal jugular vein and the vagus nerve.

The Common Carotid Arteries

The common carotid arteries differ on the right and left sides with respect to their origins. On the right, the common carotid arises from the brachiocephalic artery as it passes posterior to the sternoclavicular joint. On the left side, the common carotid artery comes directly from the arch of the aorta in the superior mediastinum. The right common carotid has, therefore, only a cervical part whereas the left common carotid has cervical and thoracic parts.

Following a similar course on both sides, the common carotid artery ascends, diverging laterally from behind the sternoclavicular joint to the level of the upper border of the thyroid cartilage of the larynx (cervical 3-4 vertebral junction), where it divides into the external and internal carotid arteries. This bifurcation may sometimes be at a slightly higher cervical level. The common carotid artery may be compressed against the carotid tubercle of the transverse process of the sixth cervical vertebra. Above this level it is more superficial, covered merely by skin, superficial fascia, platysma, deep cervical fascia and the medial margin of the sternocleidomastoid muscle.

In the lower part of the neck below the cricoid cartilage, the common carotid arteries are separated by the trachea. Above this, and between them, lie the thyroid gland, larynx and pharynx. On the right side, the recurrent laryngeal nerve crosses obliquely behind the artery. The right internal jugular vein diverges from it below, but the left vein approaches and often overlaps its artery. As indicated, each artery is contained within the carotid sheath of deep cervical fascia which also encloses the internal jugular vein and the vagus nerve. The vein lies lateral to the artery, and the nerve lies between them and posterior to both.

Of paramount importance in terms both of physiology and forensic significance is that at its division into the internal and external carotid arteries the vessel has a dilatation, the **carotid sinus.** This dilatation usually involves the bifurcation and extends to, or is restricted to, the beginning of the internal carotid artery. Histologically, the tunica media is thinner at the dilatation but the tunica adventitia is relatively thick, containing many receptor endings of the glossopharyngeal nerve. There are also small contributions from the cervical sympathetic trunk and the vagus nerve. These receptor endings tend to a degree of degeneration and regeneration, suggesting a replacement of the nervous elements.

The Functional Anatomy of the Carotid Sinus

The carotid sinus is responsive to changes in arterial blood pressure, leading to reflex haemodynamic modification. Phylogenetically (or is it by intelligent design?), its strategically situated position on the brain's main artery renders it appropriate to its role as a baroreceptor in control of intracranial blood pressure. However, in order to understand the function of this baroreceptor in possible relation to hanging, it is necessary to examine its role in the overall interrelated systems for arterial pressure regulation.

Interrelated Systems of Arterial Pressure Regulation

Since in hanging there is pressure on the structures (muscles, arteries, veins, nerves), it is of interest to speculate on what may occur in the early phases of hanging. It is

thus important to know and understand the normal physiological reactions which occur under pressure.

Arterial pressure is not regulated by a single pressure controlling system but instead by several interrelated systems that perform specific functions. When the blood pressure falls suddenly following injury, two problems immediately confront the pressure control system. The first is to return the arterial pressure immediately to a high enough level so that the person can survive the acute traumatic incident. The second, in the case of an acute haemorrhagic episode, is to return the blood volume eventually to as normal a level as possible. This includes the return of the circulatory system to full normality, not merely back to a pressure level required for survival. These two problems and their solution thus characterise the two major types of arterial pressure control systems in the body:

1) a system of rapidly acting pressure control mechanisms involved with immediate survival, and;
2) a system for long-term control of the basic arterial pressure level.

Long-term regulators of arterial pressure are required because the rapidly acting mechanisms for arterial pressure regulation generally lose their power to control arterial pressure after a few hours to a few days. This is because the pressure receptors “adapt”, i.e. they lose their responsiveness. Long-term regulation, instead, is vested mainly in a renal-body fluid-pressure control mechanism. Part of this mechanism involves control of renal function by several different hormonal systems. This includes particularly the juxta-glomerular apparatus with its renin-angiotensin system and the hormone aldosterone secreted by the adrenal cortex. These will not be further discussed.

What is the situation with regard to the application of pressure to the neck and the resultant pathophysiological and psychological stress to which the individual is subjected? [6] Cardiovascular function is under the regulatory control of the sympathetic nervous system, with sympathetic stimulation increasing the rate and force of cardiac contraction, conduction velocity within the myocardium, and blood pressure. Levels of both adrenaline and noradrenaline increase, with noradrenaline levels increasing 7 to 10 times above baseline levels, and adrenaline 3 to 8 times.[7,8] The heart rate increases to a maximum of 180-200 beats per minute and the systolic blood pressure increases to between 162 and 216 mm Hg. [9] Simultaneously, noradrenaline acts on the alpha-$_1$ receptors in the coronary arteries producing vasoconstriction. This results in myocardial ischaemia on the basis of reduced blood flow and oxygen supply to the myocardium. Ischaemia, in turn, sensitizes the myocardium to the effect of catecholamines at the time of increased myocardial oxygen demand. This lethal and paradoxical combination at the time of greatest myocardial oxygen demand renders the heart vulnerable to the arrhythmia-producing properties of catecholamines.

What is the situation with regard to the loss of consciousness experienced by SCUBA (self-contained underwater breathing apparatus) divers who descend to depths at which they are subjected to pressure? The loss of consciousness experienced is more the result of occlusive arterial nitrogen gas emboli within the vascular system rather than to any pressure applied from outside. The reason why external pressure has no effect on the diver is because he derives his oxygen supply from a compressed gas cylinder containing a mixture of helium, nitrogen and oxygen. The re-breathing equipment incorporated regulates the oxygen concentration to that appropriate to the depth of descent of the dive (as well as having a carbon

dioxide absorbing chemical). The oxygen concentration is thus calibrated to a level appropriate to the depth of the dive and the gas pressures maintain oxygen delivery to the brain appropriate to the depth of descent. The intravascular gas pressures are thus higher than those at sea surface levels. While, however, the external pressures are neutralized, should rapid ascent to the surface have occurred, the diver must undergo decompression on return to the surface in order to allow the tissues to rid themselves of the gases which were formerly contained in solution under pressure.

Rapidly acting Nervous Mechanisms for Arterial Pressure Control

Three different pressure control mechanisms begin to react within seconds after an acute abnormal change in the arterial pressure. All are nervous pressure control mechanisms: a) the baroreceptor feedback mechanism; b) the central nervous system vasomotor centre ischaemic mechanism via the glossopharyngeal nerve (sinus nerve), and; c) the chemoreceptor mechanism.

The Baroreceptor Control System

By far the best known of the mechanisms for arterial pressure control is the baroreceptor reflex. This is initiated when baroreceptors are subjected to pressure or stretch as in the compressive and tensile forces occurring in hanging. While a few baroreceptors are located in the wall of almost every large artery of the thoracic and neck regions, they are particularly abundant in the carotid sinus and the wall of the aortic arch. On stimulus induction, signals are transmitted from each carotid sinus through the very small Hering's nerve (sinus nerve) to the glossopharyngeal nerve

and thence to the tractus solitarius in the medullary vital centres of the brainstem. The carotid sinus baroreceptors are not stimulated at all by pressures between 0 and 60 mm Hg, Above 60 mm Hg, however, they respond progressively more and more rapidly, reaching a maximum at about 180 mm Hg.

An interesting feature of the pressure response is that the increase in number of impulses for each unit change in arterial pressure is greatest at a pressure level near the normal mean arterial pressure. In other words, in the normal operating range of arterial pressure, even a slight change in pressure causes strong autonomic reflexes to readjust the arterial pressure back toward normal. Hence, the baroreceptor feedback mechanism functions most effectively in the very pressure range at which it is most needed. Furthermore, not only do the baroreceptors respond extremely rapidly to changes in arterial pressure, but they respond much more to a rapidly changing pressure than to a stationary pressure. This latter point is of relevance in the context of the rapidly occurring compressive and tensile forces of hanging.

After the baroreceptor signals have entered the tractus solitarius of the medulla, secondary signals inhibit the vasoconstrictor centre of the medulla and excite the vagal centre. The net effects are: a) vasodilatation throughout the peripheral circulatory system, and; b) decreased heart rate (bradycardia) and strength of cardiac contraction. Therefore, excitation of the baroreceptors by pressure in the arteries reflexly causes the arterial pressure to decrease both by a decrease in peripheral resistance and a decrease in cardiac output.

The Carotid Body

Near the common carotid bifurcation and having bilateral representation lies a 5-7 mm in height and 2.5-4 mm in width reddish-brown oval structure which functions as a chemoreceptor and is known as the **carotid body.** First described in 1743 by von Haller,[10] the **carotid glomus** develops from mesenchyme in the third pharyngeal arch. It lies either posterior to the carotid bifurcation or between its branches, and is attached to, or sometimes partly embedded in, their adventitia. Occasionally it takes the form of a group of separate nodules. Aberrant miniature carotid bodies, micro-structurally similar but with diameters of 600 micron or less, may appear in the adventitia and adipose tissue near the carotid sinus. [11]

The carotid body is surrounded by a fibrous capsule from which septa divide the enclosed tissue into lobules. Each lobule contains glomus (Type I) cells which are separated from an extensive network of fenestrated sinusoids by sustentacular (Type II) cells. [12-14] Glomus cells store a number of peptides, particularly encephalins, bombesin and neurotensin, and amines including dopamine, serotonin, adrenaline (epinephrine) and noradrenaline (norepinephrine), and are therefore regarded as paraneurones. [15,16] Unmyelinated axons lie in a collagenous matrix between the sustentacular cells and the sinusoidal endothelium, and many synapse on the glomus cells. They are visceral afferents which travel in the carotid sinus nerve to join the glossopharyngeal nerve. Preganglionic sympathetic axons and fibres from the carotid sinus synapse on parasympathetic and sympathetic ganglion cells. These lie either in isolation or in small groups near the surface of each carotid body. Postganglionic axons travel to local blood vessels, the parasympathetic efferent fibres being vasodilatory and the sympathetic ones vasoconstrictor.

The carotid body receives a rich blood supply from branches of the adjacent external carotid artery, consistent with its role as an arterial chemoreceptor. When stimulated by hypoxia, hypercapnia or increased hydrogen ion concentration (low pH) in the blood flowing through it, it elicits reflex increases in the rate and volume of ventilation via connections with brain stem respiratory centres. Most prominent in children, the bodies normally involute in older age, when they are infiltrated by lymphocytes and fibrous tissue.

Other small bodies, resembling carotid bodies, and also considered to be chemoreceptors, occur near the arteries of the fourth and sixth pharyngeal arches. Hence, they are found near the aortic arch, ligamentum arteriosum and right subclavian artery, and are supplied by branches from the vagus nerve.

The blood flow through the carotid bodies is extremely high, as high as that for almost any tissue in the body. Because of this, the arterio-venous oxygen difference is less than 1 volume per cent, which means that the venous blood leaving the carotid bodies still has an oxygen concentration nearly equal to that of the arterial blood. Therefore, it is the arterial oxygen concentration, not the venous oxygen concentration, that normally determines the degree of chemoreceptor stimulation. Thus, as occurs in hanging where compressive force is exerted on the arterial walls, blood flow is reduced, the oxygen concentration falls, and the chemoreceptors become stimulated. The impulse rate in the chemoreceptors is particularly sensitive to changes in arterial oxygen concentration in the range between 60 and 30 mm Hg, which is the range in which the arterial haemoglobin saturation with oxygen decreases rapidly.

As indicated, each carotid artery is contained in a **carotid sheath**, continuous with the deep cervical fascia and of loose texture, though that actually around the

artery is denser. This sheath encloses also the internal jugular vein and vagus nerve, the vein lateral to the artery, the nerve between them and posterior to both. The superior root of the ansa cervicalis is embedded in the anterior wall of the sheath. From the point of view of the nervous responses to not only control of pressure but of response to compressive force exerted on the vessel wall, it is important to appreciate that the veins participate in all the reactions and reflexes to which the arteries are subject.

The External Carotid Artery

This artery begins lateral to the upper border of the thyroid cartilage, level with the intervertebral disc between the third and fourth cervical vertebrae. A little curved, and with a gentle spiral, it first ascends slightly forwards and then inclines backwards and a little laterally, to pass midway between the mastoid tip and mandibular angle. Here, in the substance of the parotid gland behind the neck of the mandible, it divides into the superficial temporal and maxillary arteries. This latter vessel, the larger of the two terminal vessels, is of interest in that it gives rise to the middle meningeal artery, a vessel of great forensic significance not only in the context of extradural haematoma formation following head injury, but also through its anastomosis with the lacrimal artery. This vessel (which may be derived from the middle meningeal), usually arises from the ophthalmic artery, itself arising from the internal carotid. An anastomosis is thus established between the internal and external carotid arterial circulations.

Due to its many large branches, the external carotid artery diminishes rapidly in calibre. In children, it is smaller than the internal carotid, but in adults the two are

of almost equal size. At its origin, it lies in the carotid triangle, which, in the living subject, may be seen as a triangular depression in the neck. In the triangle, the vessel lies anteromedial to the internal carotid, but then becomes lateral to this vessel as it ascends. Finally, at mandibular levels, the styloid process and its attached structures intervene between the vessels, the internal carotid being deep and the external carotid superficial to the styloid process.

The Anatomical Relations of the Arteries to their Surrounding Structures

Superficial to the artery in the carotid triangle are the skin, superficial fascia, the loop between the facial nerve's cervical branch and the transverse cutaneous nerve of the neck, deep fascia and the anterior margin of the sternocleidomastoid muscle. The artery is crossed by the hypoglossal nerve and its vena comitans and by the lingual (common), facial and sometimes by the superior thyroid veins.

Leaving the carotid triangle, the artery is crossed by the posterior belly of the digastric and stylohyoid muscles. It then ascends between this latter muscle and the posteromedial surface of the parotid gland. It then enters the parotid, lying medial to the facial nerve and the junction of the superficial temporal and maxillary veins.

Medial to the artery are at first the pharyngeal wall, superior laryngeal nerve and ascending pharyngeal artery. At a higher level, however, the internal carotid artery is separated from the external by the styloid process, styloglossus and stylopharyngeus muscles, the glossopharyngeal nerve, the pharyngeal branch of the vagus nerve and part of the parotid gland. While not of direct relevance to the current study, the relation of the artery to the parotid gland is controversial, many clinicians

asserting that the artery is often medial to the gland rather than in it. Both relations, however, occur at about equal frequency.

The Internal Carotid Artery

The internal carotid artery may conveniently be divided into cervical, petrous, cavernous and cerebral parts. The cervical part begins at the carotid bifurcation and ascends in front of the upper three cervical transverse processes to the inferior aperture of the carotid canal in the petrous temporal bone. Superficial at first in the carotid triangle, it then passes deeper, medial to the posterior belly of the digastric muscle. Except near the skull, the internal jugular vein and vagus nerve are lateral, the external carotid being first anteromedial but then curving back to become superficial.

The artery has many other relations. Posteriorly, it adjoins the longus capitis, with the superior cervical sympathetic ganglion between them and the superior laryngeal nerve crossing obliquely behind it. Medial lies the pharyngeal wall separated by fat and pharyngeal veins from the ascending pharyngeal artery and superior laryngeal nerve, the latter a branch of the vagus and of paramount forensic importance in the context of pressure to the neck with its attendant bradycardia.

Anterolaterally, the artery is covered by the sternocleidomastoid muscle. Below the level of the digastric, the hypoglossal nerve, superior root of the ansa cervicalis, and the lingual and facial veins are superficial. At the level of the digastric, the vessel is crossed by the stylohyoid muscle and the occipital and posterior auricular arteries. Above the digastric, the vessel is separated from the external carotid by the styloid process, styloglossus and stylopharyngeus,

glossopharyngeal nerve, vagal pharyngeal branch and the deeper part of the parotid gland. This proximity to the glossopharyngeal nerve similarly carries forensic implications in view of the role played by this nerve in conduction of impulses originating from the carotid baroreceptors. Finally, at the base of the skull, the glossopharyngeal, vagus, accessory and hypoglossal nerves lie between the internal carotid artery and the internal jugular vein, which here has become posterior.

The Vertebral Artery

This artery arises from the superoposterior aspect of the first part of the subclavian artery. It ascends through the foramina of all cervical transverse processes except the seventh, these foramina therefore affording it protection against the compressive force of an encircling ligature. However, its *first part* ascends posteriorly between the **longus colli muscle** and the **scalenus anterior muscle**, the latter of interest in view of the fact that the **scalenus anterior muscle** forms the 'cushion' on whose anterior surface runs the **phrenic nerve**, the nerve to the diaphragm, the major muscle of respiration. The **scalenus anterior muscle** itself constitutes one of the accessory muscles of respiration. Furthermore, the **vertebral artery** is related anteriorly to the **common carotid artery** and **vertebral vein**, the former of central interest and significance to this study. Posteriorly, on the other hand, lie the seventh cervical transverse process, the **cervicothoracic ganglion** and ventral rami of the seventh and eighth cervical spinal nerves. These anatomical features serve to highlight the complexity of arrangement of the anatomical structures surrounded by, and compressed by, the encircling ligature in the victim of suicidal hanging.

The Vertebral Vein and Vertebral Venous Plexus

The **vertebral vein** is formed by many small tributaries from internal vertebral plexuses which leave the vertebral canal. These then join small veins from local deep muscles to form a vessel which descends as a **plexus** around the **vertebral artery**. It then descends behind and in proximity to the **internal jugular vein** where it too may thus be subjected to the compressive forces of an encircling ligature. This not only highlights once again the multiplicity and complexity of arrangement of the anatomical structures in the neck but that this arrangment may also contribute to the damming up of blood flow from the head and neck by the encircling ligature.

The Origins, General Architecture and Cellular Composition of Arteries

The circulatory system is the first of all organ systems to become functional during development. Adult tetrapods (vertebrates having four legs in their ancestry) lack both first and second aortic arches. The common carotid arteries are derived from segments of the ancestral (and embryonic) paired ventral aortae between the ventral roots of the third and fourth arterial arches. The external carotid arteries possibly represent the paired ventral aortae anterior to the ventral roots of the third arch. The internal carotid arteries are derived from the ancestral internal carotids, plus the paired dorsal aortae between the dorsal roots of the first and third arches, together with the third arch distal to its junction with the external carotid.

The general architecture and cellular composition of blood vessels are the same throughout the cardiovascular system but certain features of the vasculature vary with and reflect distinct functional requirements at different locations. To

withstand the pulsatile flow and higher blood pressures in arteries, arterial walls are generally thicker than the walls of veins. Arterial wall thickness gradually decreases as the vessels become smaller, but the ratio of wall thickness to lumen diameter becomes greater.

Arteries, veins, and capillaries are at first indistinguishable histologically. Each is a tube formed from thin, flat endothelial cells which are loosely wrapped on the outside in a meshwork of connective tissue. This is the definitive structure of capillaries. Arteries and veins each retain these tissues as a **tunica interna** but add more peripheral tissues as they mature. Arteries develop a thick **tunica media** which usually consists of circularly arranged smooth muscle fibres but which in the largest arteries consists instead of elastic fibres. Arteries are completed by a thinner **tunica externa** of longitudinally oriented connective tissue.

Veins are usually larger in diameter and thinner-walled than corresponding arteries. The **tunica media** is typically thin and may be indistinct. The **tunica externa**, by contrast, is as thick or thicker than that of arteries.

Based on their size and structural features, arteries are divided into three types: 1) large or elastic arteries such as the aorta and its large branches i.e. the common carotid arteries; 2) medium-sized or muscular arteries comprising other branches of the aorta such as the coronary and renal arteries, and; 3) small arteries (< approximately 2 mm in diameter) and arterioles (20 to 100 mm in diameter). These latter lie within the substance of tissues and organs.

The basic constituents of the walls of blood vessels are endothelial cells and smooth muscle cells, and extracellular matrix (ECM), including elastin, collagen, and glycosoaminoglycans. Vascular walls comprise three concentric layers: intima, media, and adventitia, which are most clearly defined in the larger vessels,

particularly arteries. In normal arteries, the intima consists of a single layer of endothelial cells with minimal underlying subendothelial connective tissue. It is separated from the media by a dense elastic membrane called the **internal elastic lamina**. The smooth muscle cell layers of the media near the vessel lumen receive oxygen and nutrients by direct diffusion from the vessel lumen, facilitated by holes in the internal elastic membrane. However, diffusion from the lumen is inadequate for the outer portions of the media in large and medium-sized vessels. These areas are therefore nourished by small arterioles arising from outside the vessel and coursing into the outer one half to two thirds of the media. They are known as the **vasa vasorum**, literally "vessels of the vessels". This micro-anatomical and structural configuration carries practical implications in the context of hanging where stretching and compression of the vascular wall with rupture of these nutrient vessels results in haemorrhage into the adventitial layer.

The outer limit of the media of most arteries is a well-defined **external elastic lamina**. External to the media is the adventitia, consisting of connective tissue with nerve fibres and the **vasa vasorum**.

The relative amount and configuration of the basic constituents vary along the arterial system owing to local adaptations to mechanical or metabolic needs. These structural changes lie principally in the media and in the ECM. In the elastic arteries, such as the carotids, the media is rich in elastic fibres. These are arranged in fairly compact layers separated by and alternating with layers of smooth muscle cells. The mechanical function of the elastic components of the vessel wall allows expansion during cardiac contraction with elastic recoil during relaxation propelling blood through the vascular system. This layered configuration of elastic fibres in the media carries, however, an inherent and fatal flaw in that vascular rupture permits

extension of blood along the laminar planes of the media. This is, in part, due to the natural tendency of haemorrhage acting under systolic pressure to propagate along the path of least resistance such as that offered to it by a laminated structure.

The Vagus Nerve

This large mixed nerve has a more extensive course and distribution (vagus = wandering) than any other cranial nerve, traversing the neck, thorax and abdomen. Of crucial pathophysiological significance in the context of pressure to the neck, this nerve exits the skull through the jugular foramen accompanied by the accessory nerve, with which it shares an arachnoid and a dural sheath. Both nerves lie anterior to a fibrous septum that separates them from the glossopharyngeal nerve. The vagus descends vertically in the neck in the carotid sheath, between the internal jugular vein and the internal carotid artery, to the upper border of the thyroid cartilage. The nerve thus lies directly in the area of force application in hanging. It then passes between the vein and the common carotid artery to the root of the neck. Its relationships in this part of its course are therefore similar to those described for these structures.

Its further course differs on the two sides. The right vagus descends posterior to the internal jugular vein to cross the first part of the subclavian artery and enter the thorax. The left vagus enters the thorax between the left common carotid and subclavian arteries and behind the left brachiocephalic vein.

The Recurrent Laryngeal Nerve

This branch of the vagus merits separate mention in the context of hanging for several reasons:

1) It carries afferent fibres from laryngeal stretch receptors;

2) On both sides it ascends in or near a groove between the trachea and oesophagus with this proximity to trachea similarly rendering it vulnerable to the same forces producing tracheal stretch, and;

3) As it curves round the subclavian artery, or the aortic arch, it gives cardiac filaments to the deep cardiac plexus thus contributing to the bradycardia and cardiac arrest following vagal stimulation.

Anatomically, it is important to realize that the nerve does not always lie in an ostensibly protected position in the tracheo-oesophageal groove (but still subject to the same stretch forces to which larynx and trachea are susceptible). The nerve may lie slightly anterior to the groove (more often on the right), and it may lie markedly lateral to the trachea (but is still subject to the same forces exerted by an encircling ligature) at the level of the lower part of the thyroid gland.

A further anatomical point of interest is that on the right side the nerve is as often anterior to, posterior to, or intermingled with, the terminal branches of the inferior thyroid artery. On the left the nerve is usually posterior to the artery, though occasionally anterior to it. The point is that the trachea, which is subject to the compressive and tensile forces of hanging, is supplied with blood mainly by branches of the inferior thyroid arteries amongst which the nerve is intermingled. Furthermore, the trachea is innervated by this nerve as well as by other branches of the vagi and sympathetic trunks. The forensic and pathophysiological implications of this blood

supply and innervation is that traction involving the trachea must, of necessity, produce the vagal cardioinhibitory effects associated with stimulation of the vagus and/or its branches.

The Phrenic Nerve

This nerve, the sole motor supply to the diaphragm, the major muscle of respiration, arises chiefly from the fourth cervical ramus (C.4) but also has contributions from the third (C.3) and fifth (C.5). It starts deep to the prevertebral fascia at the upper part of the lateral border of the obliquely placed **Scalenus Anterior,** crossing it from posterior to anterior border. It descends almost vertically on the anterior surface of this muscle and posterior to the sternocleidomastoid. This anatomical positioning and course of the nerve thus places it immediately beneath an encircling ligature making it subject to any compressive forces applied carrying major implications for diaphragmatic paralysis and hence respiratory failure.

The Trachea and its Cervical Relations

The trachea is a tube formed of cartilage and fibromuscular membrane, lined internally by mucosa. The anterolateral portion is made up of incomplete rings of cartilage, and the posterior aspect by a flat muscular wall. It is about 10-11 cm long, and descends from the larynx from the level of the sixth cervical vertebra to the upper border of the fifth thoracic vertebra, where it divides into right and left principal (pulmonary) bronchi. It lies approximately in the sagittal plane, but its point of bifurcation is usually a little to the right. Its external transverse diameter is about 2

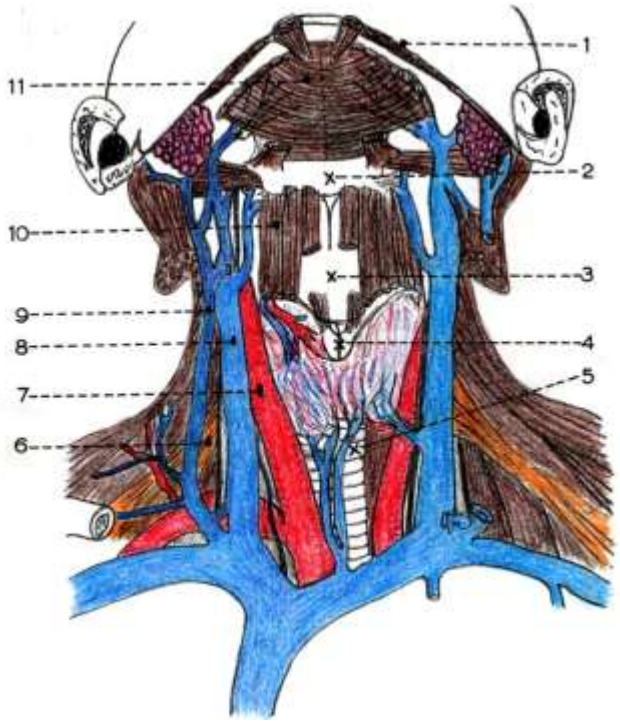
cm in adult males, and 1.5 cm in adult females. The lumen in live adults is about 12 mm in transverse diameter, although this increases after death as the smooth muscle making up its posterior aspect relaxes.

Anteriorly, the trachea is crossed by skin and by the superficial and deep cervical fasciae. It is also crossed by the jugular arch and overlapped by sternohyoid and sternothyroid. The second to fourth tracheal cartilages are crossed by the isthmus of the thyroid gland, above which an anastomotic artery connects the bilateral superior thyroid arteries. Below this and in front are the pretracheal fascia, inferior thyroid veins, thymic remnants and the thyroid ima artery (when it exists).

Posteriorly lies the oesophagus, which runs between the trachea and the vertebral column. As indicated, the recurrent laryngeal nerves ascend on each side, in or near the grooves between the sides of the trachea and oesophagus.

Laterally lie the paired lobes of the thyroid gland, which descend to the fifth or sixth tracheal cartilage, and the common carotid and inferior thyroid arteries.

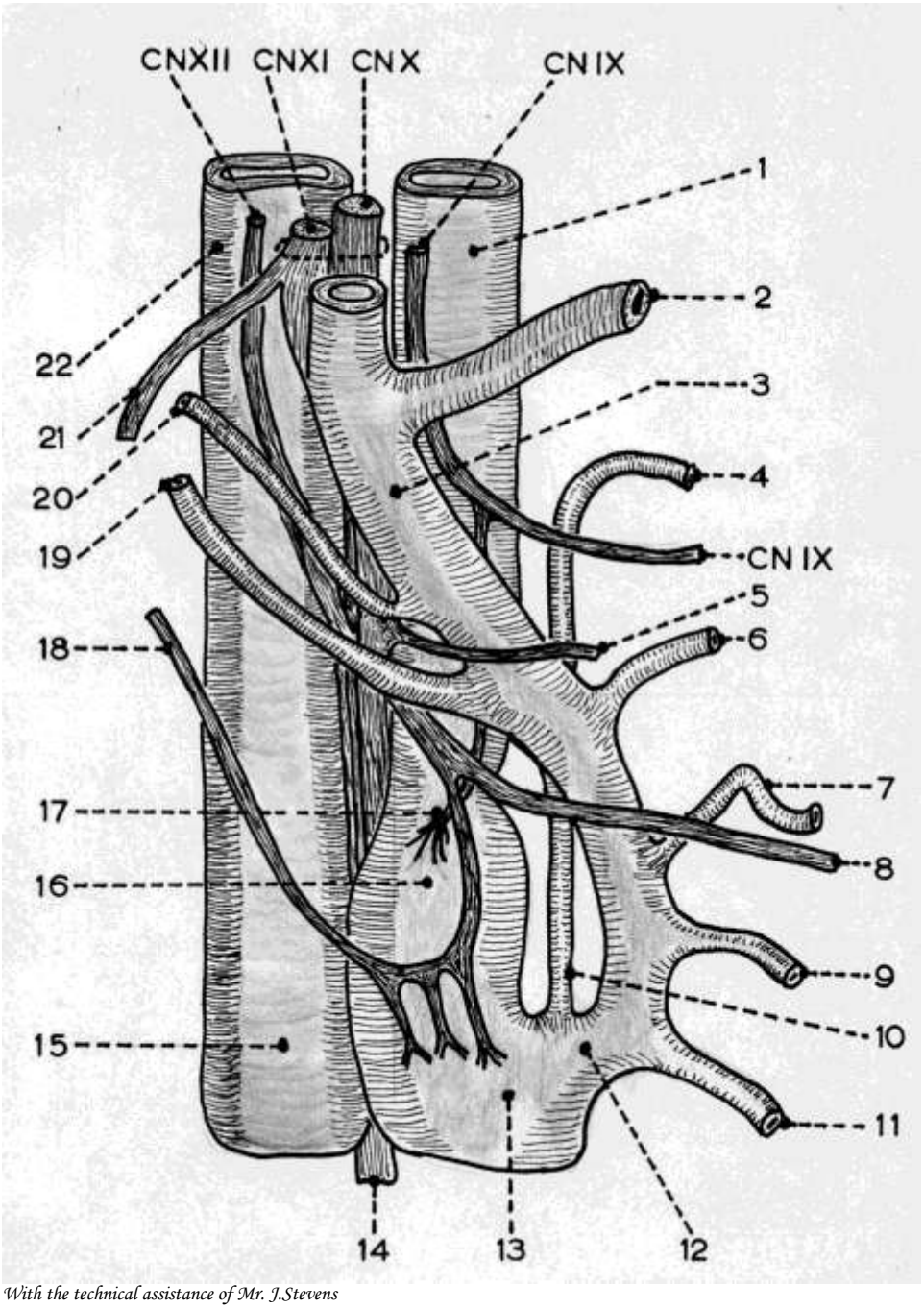
There follow a series of anatomical sketches of the structures of the neck and radiological arterial infusions of the **common carotid artery** and its bifurcation into its respective **internal carotid artery** and **external carotid artery** branches.



With the technical assistance of Mr. J.Stevens

Fig.3.1. A diagrammatic representation of an anterior view of a neck dissection.

1. **Subcutaneous fat underlying the body of the mandible**
2. **Hyoid bone**
3. **Thyro-hyoid membrane**
4. **Laryngeal prominence**
5. **Trachea**
6. **Scalenus anterior muscle**
7. **Common carotid artery**
8. **Internal jugular vein**
9. **External jugular vein**
10. **Thyro-hyoid muscle**
11. **Mylo-hyoid muscle**



With the technical assistance of Mr. J.Stevens

Fig. 3.2. A diagrammatic representation of the arteries, veins and nerves of the neck

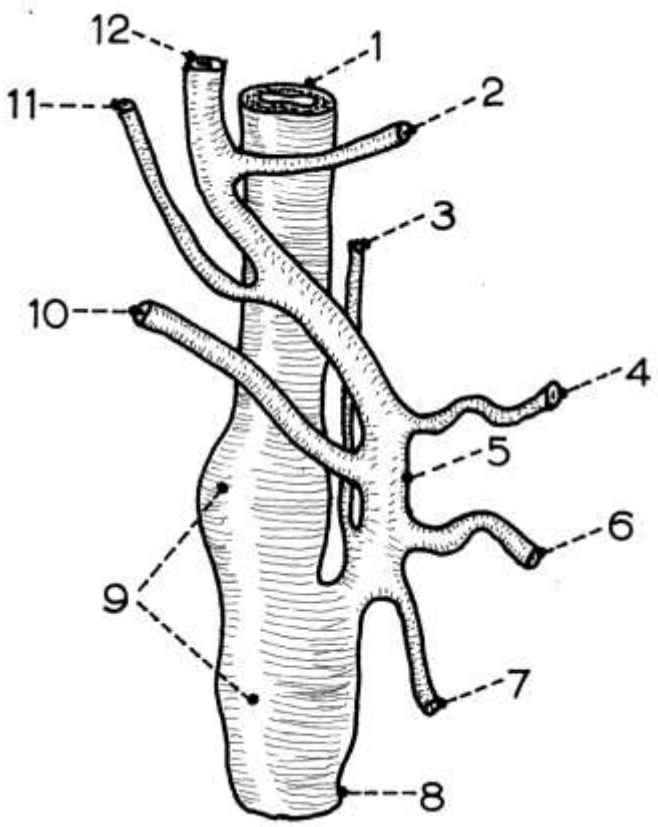
CNXII = hypoglossal nerve

CNXI = accessory nerve

CNX = vagus nerve

CNIX = glossopharyngeal nerve

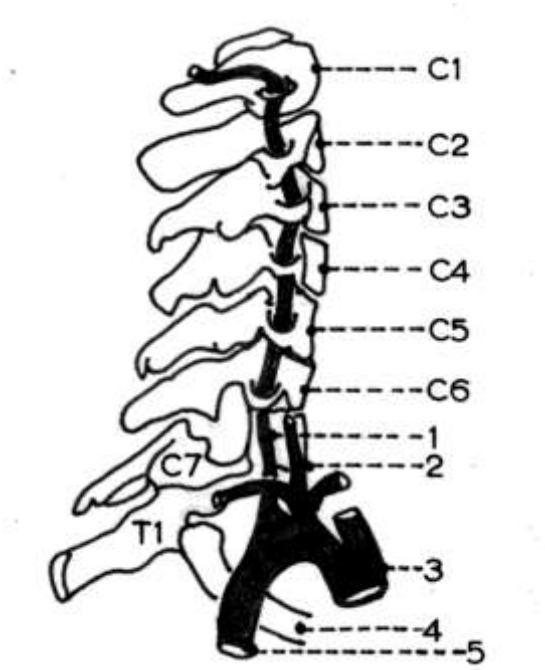
1. **Internal carotid artery**
2. **Maxillary artery**
3. **External carotid artery**
4. **Ascending pharyngeal artery**
5. **Superior laryngeal nerve (vagus branch to pharyngeal plexus)**
6. **Sternomastoid branch**
7. **Facial artery**
8. **Hypoglossal nerve (CNXII)**
9. **Lingual artery**
10. **Ascending pharyngeal artery**
11. **Superior thyroid artery**
12. **External carotid artery**
13. **Bifurcation of common carotid artery**
14. **Vagus nerve (CNX)**
15. **Internal jugular vein**
16. **Carotid sinus**
17. **Sinus nerve to glossopharyngeal nerve (CNIX)**
18. **Ascending loop of ansa cervicalis**
19. **Occipital artery**
20. **Posterior auricular artery**
21. **Spinal branch of accessory nerve (CNXI)**
22. **Internal jugular vein**



With the technical assistance of Mr. J.Stevens

Fig. 3.3. A diagrammatic representation of the carotid system of arteries.

1. **Internal Carotid artery**
2. **Maxillary artery**
3. **Ascending pharyngeal artery**
4. **Facial artery**
5. **External carotid artery**
6. **Lingual artery**
7. **Superior thyroid artery**
8. **Common carotid artery**
9. **Carotid sinus**
10. **Occipital artery**
11. **Posterior auricular artery**
12. **Superficial temporal artery**

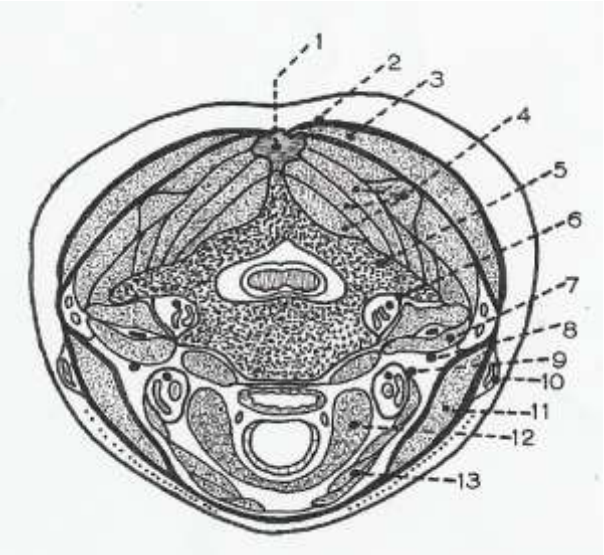


With the technical assistance of Mr. J.Stevens

Fig. 3.4. A diagrammatic representation of the vertebral system of arteries: C1 to T1

1. **Vertebral artery**
2. **Superior thyroid artery**
3. **External carotid artery**
4. **First rib**
5. **Axillary artery**

Posterior

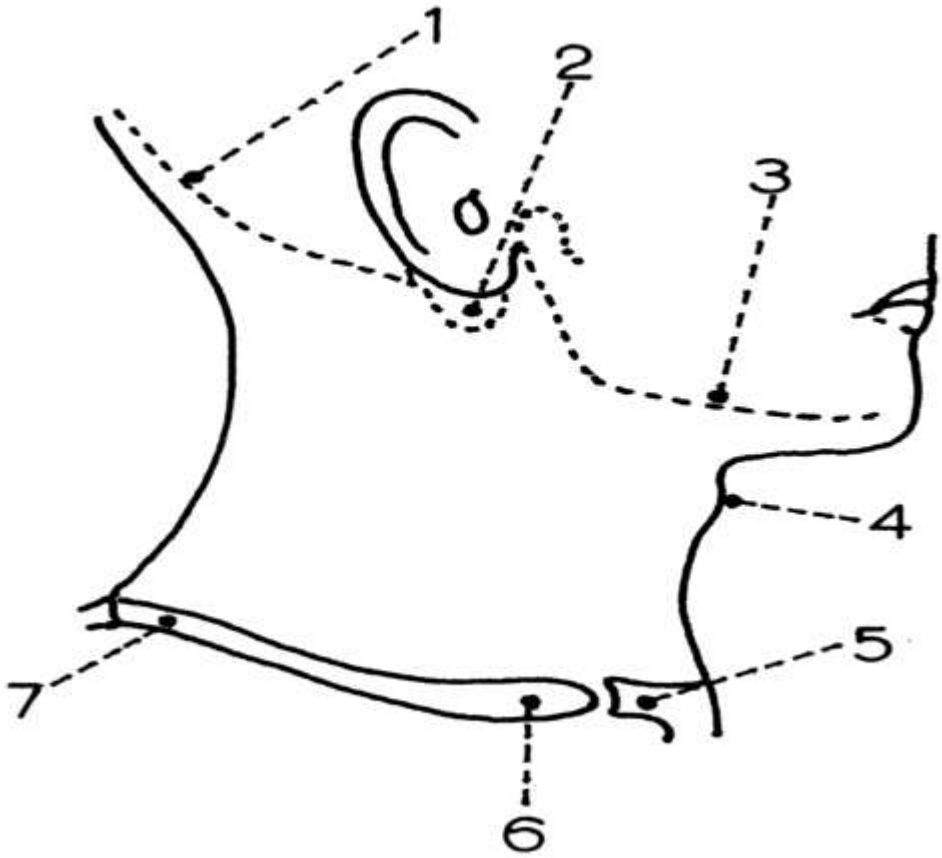


Anterior

With the technical assistance of Mr. J.Stevens

Fig. 3.5. A diagrammatic representation of a cross-section of the neck at the level of C6 seen from below.

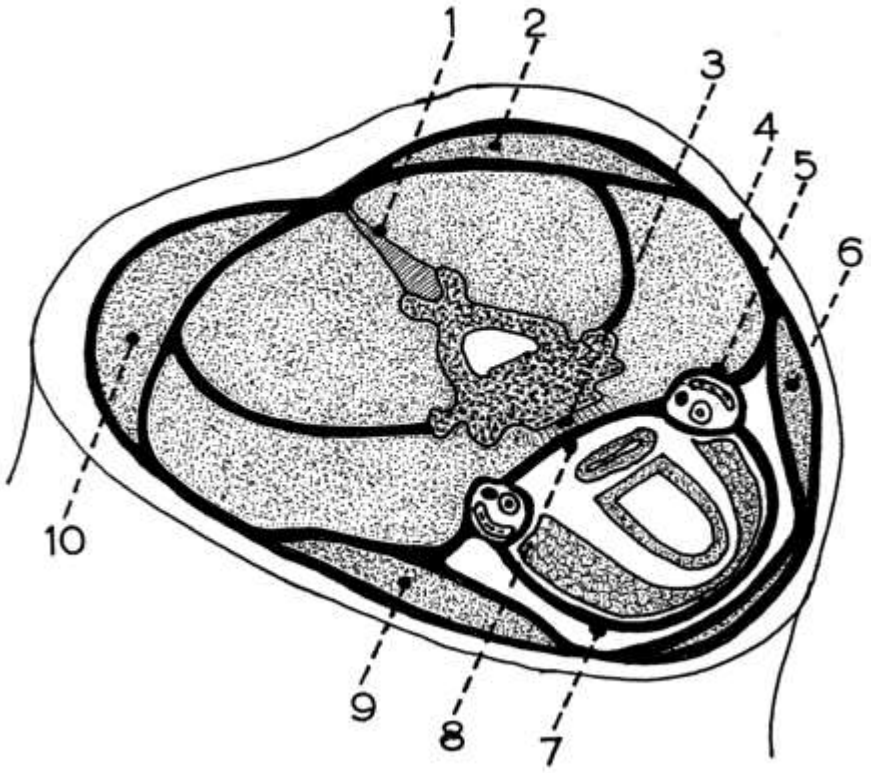
1. **Ligamentum nuchae.**
2. **General investing layer of fascia.**
3. **Trapezius muscle.**
4. **Deep neck muscles.**
5. **Cervical vertebra.**
6. **Vertebral vessels and sympathetic plexus.**
7. **Scalenus anterior.**
8. **Phrenic nerve.**
9. **Carotid sheath and contents.**
10. **External jugular vein.**
11. **Sternocleidomastoid muscle.**
12. **Lobe of thyroid overlying trachea, oesophagus and recurrent laryngeal nerve.**
13. **'Strap muscles' of neck.**



With the technical assistance of Mr. J.Stevens

Fig. 3.6. A diagrammatic representation of a lateral view of the neck.

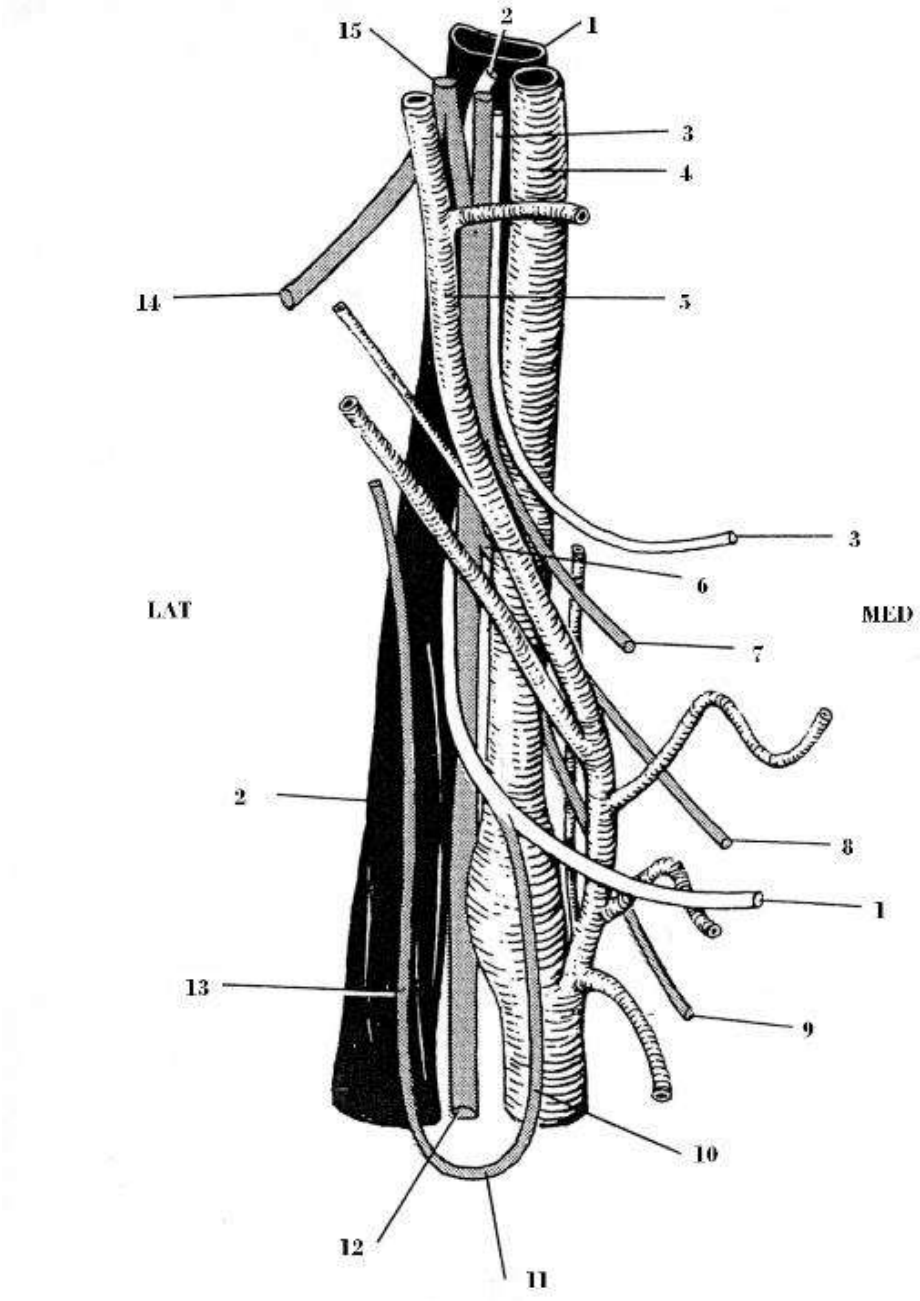
1. **Position of occipital bone**
2. **Position of mastoid process**
3. **Position of body of mandible**
4. **Laryngeal prominence**
5. **Manubrium of sternum**
6. **Medial end of clavicle**
7. **Lateral end of clavicle**



With the technical assistance of Mr. J.Stevens

Fig. 3.7. A diagrammatic representation of an oblique view of the neck.

1. **Vertebral spinous process**
2. **Trapezius muscle**
3. **Anterior longitudinal ligament**
4. **General investing layer of fascia**
5. **Carotid sheath**
6. **Sternocleidomastoid muscle**
7. **Pretracheal fascia**
8. **Prevertebral fascia**
9. **Sternocleidomastoid muscle**
10. **Trapezius muscle**

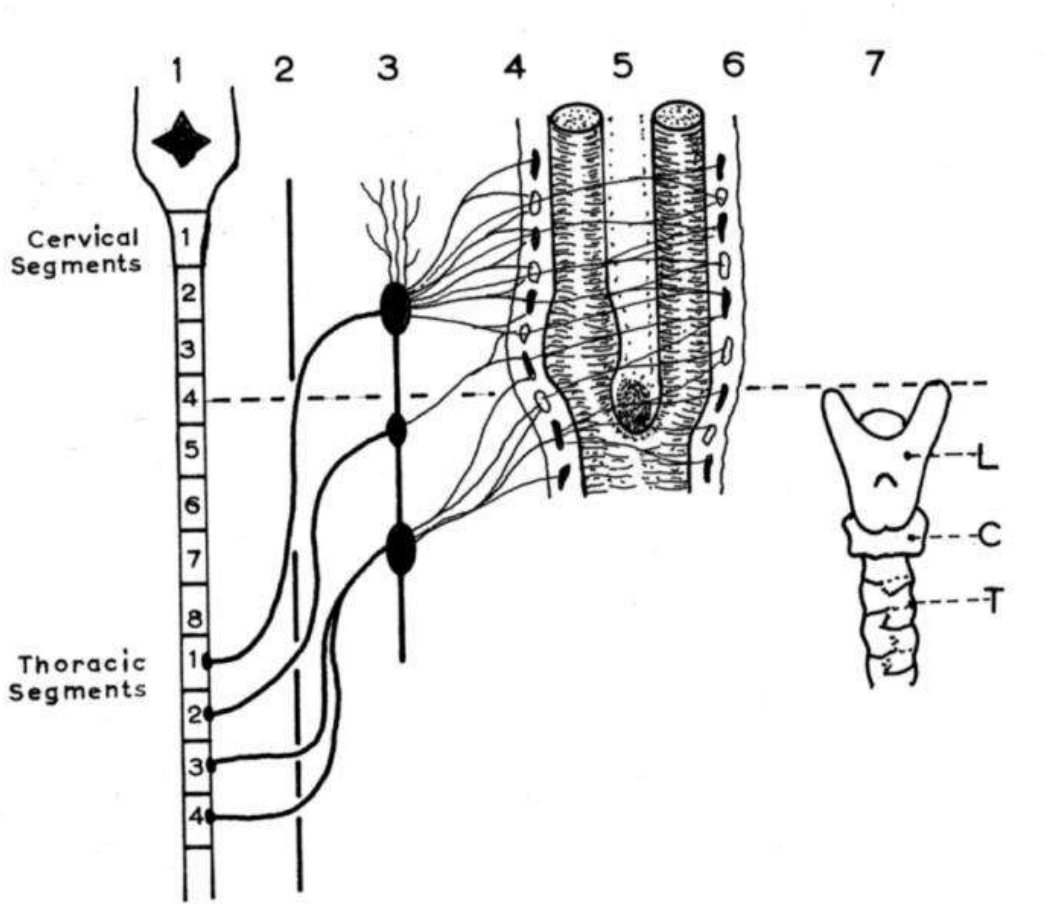


With the technical assistance of Mr. J.Stevens

Fig.3.8. A diagrammatic representation of the carotid arteries and last four cranial nerves.

1. **Hypoglossal nerve (CNXII)**
2. **Internal jugular vein**
3. **Glossopharyngeal nerve (CNIX)**
4. **Internal carotid artery**
5. **External carotid artery**
6. **Superior laryngeal nerve**
7. **Pharyngeal branch(es) of vagus nerve (CNX)**
8. **Internal branch of superior laryngeal nerve**

9. External branch of superior laryngeal nerve
10. Superior root of ansa cervicalis
11. Ansa cervicalis
12. Vagus nerve (CNX)
13. Inferior root of ansa cervicalis
14. External branch of accessory nerve (CNXI)
15. Accessory nerve (CNXI)



With the technical assistance of Mr. J.Stevens

Fig. 3.9. A diagramatic representation of the sympathetic supply to the carotid sheath.

1. **Spinal cord and segments.**
2. **Prevertebral fascia.**
3. **Sympathetic ganglion trunk.**
4. **Layer of carotid sheath containing ganglia and accessory glomi.**
5. **Bifurcation of common carotid artery and carotid body.**
6. **Carotid sheath with ganglia and accessory glomi.**

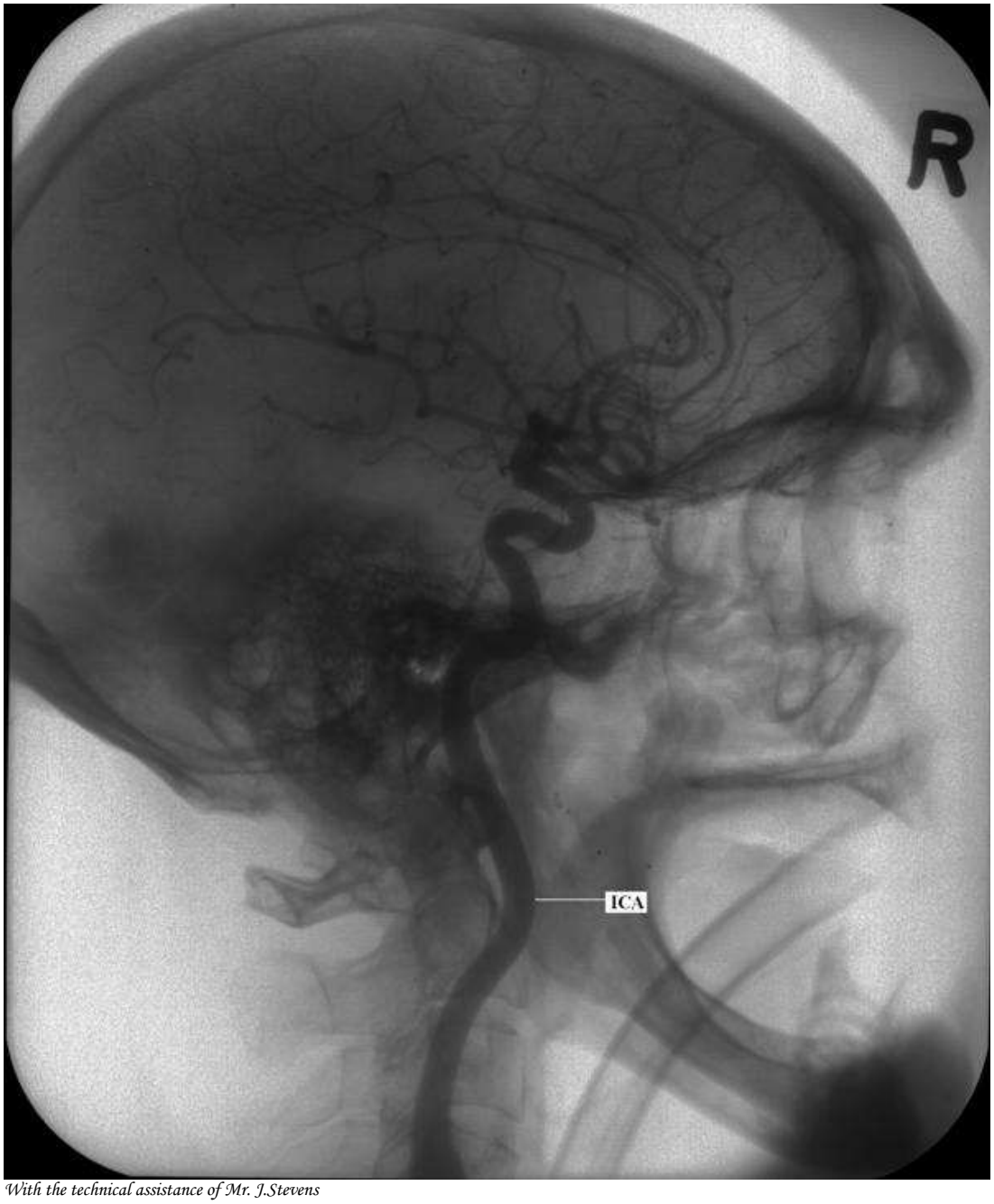
Level of bifurcation: L = larynx; C = cricoid; T = trachea.



With the technical assistance of Mr. J.Stevens

Fig. 3.10. An arteriogram showing the common carotid artery at its bifurcation into its respective internal carotid artery and external carotid artery branches. The carotid sinus is well demonstrated with the infusion needle beneath the bifurcation.

ICA = internal carotid artery.
ECA = external carotid artery.
CCA = common carotid artery.
CS = carotid sinus.
B = bifurcation.
IN = infusion needle.



With the technical assistance of Mr. J.Stevens

Fig. 3.11. An arteriogram showing the further course and continuation of the cervical internal carotid artery in the neck beneath the base of the skull as well as its intracranial part.

ICA = internal carotid artery

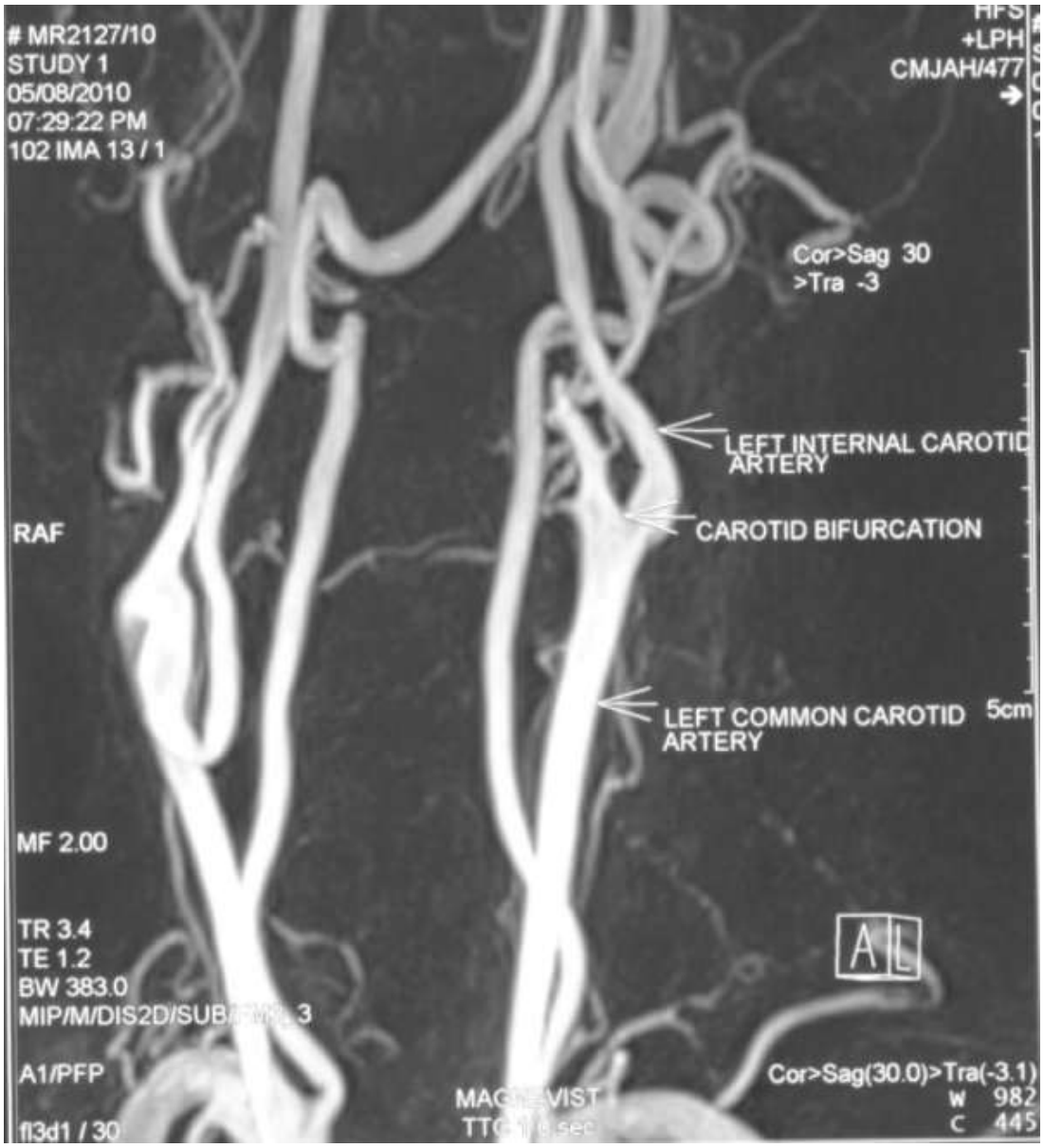


Fig. 3.12. An MRI of the neck depicting the arteries. The **left common carotid artery**, the **left internal carotid artery** and the **carotid bifurcation** have been labelled. Note the complexity of vessels.

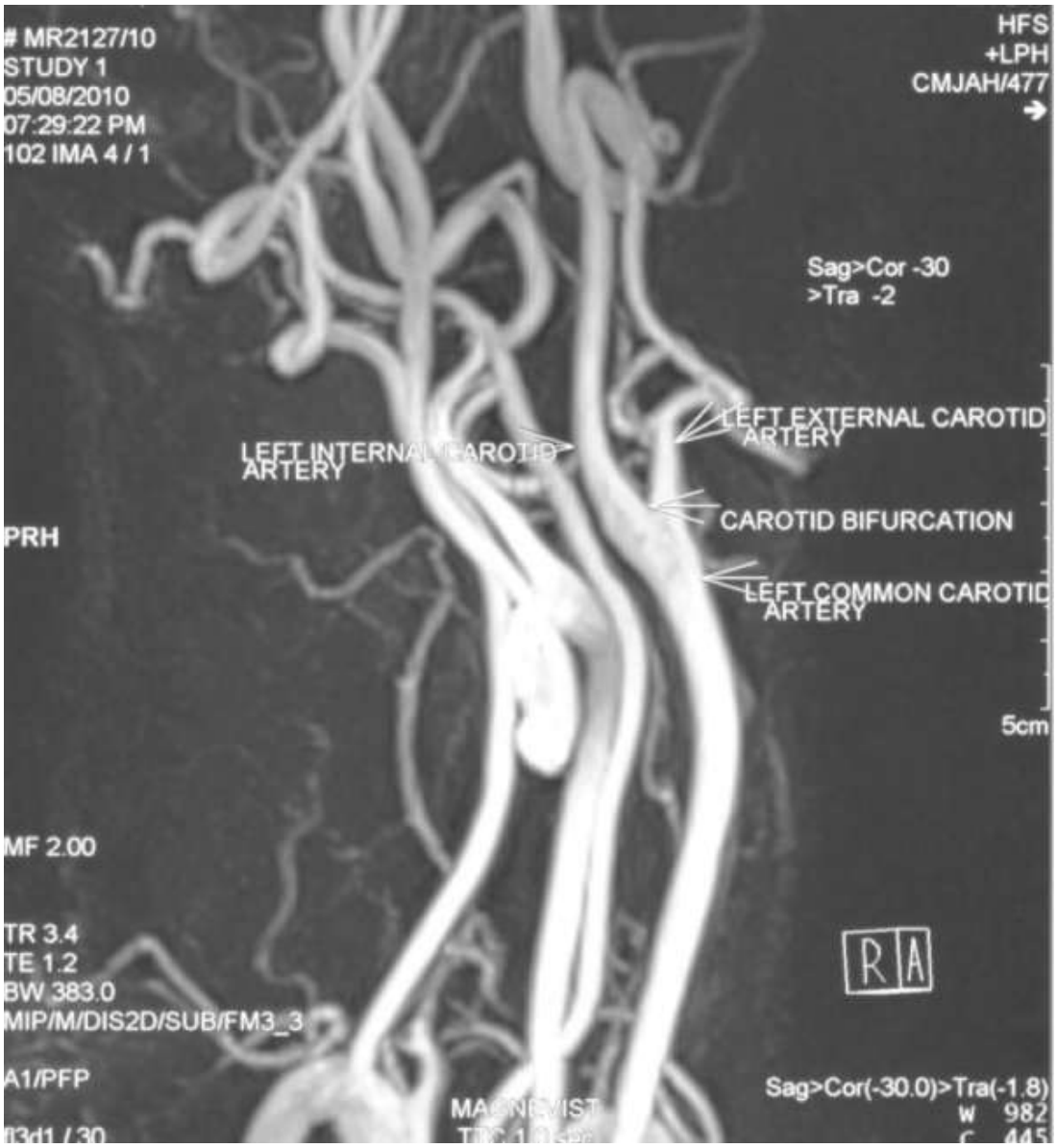


Fig. 3.13. An MRI of the neck depicting the arteries. The **left common carotid artery**, the **left internal carotid artery**, the **left external carotid artery** and the **carotid bifurcation** have been labelled. Note once again the complexity of vessels.

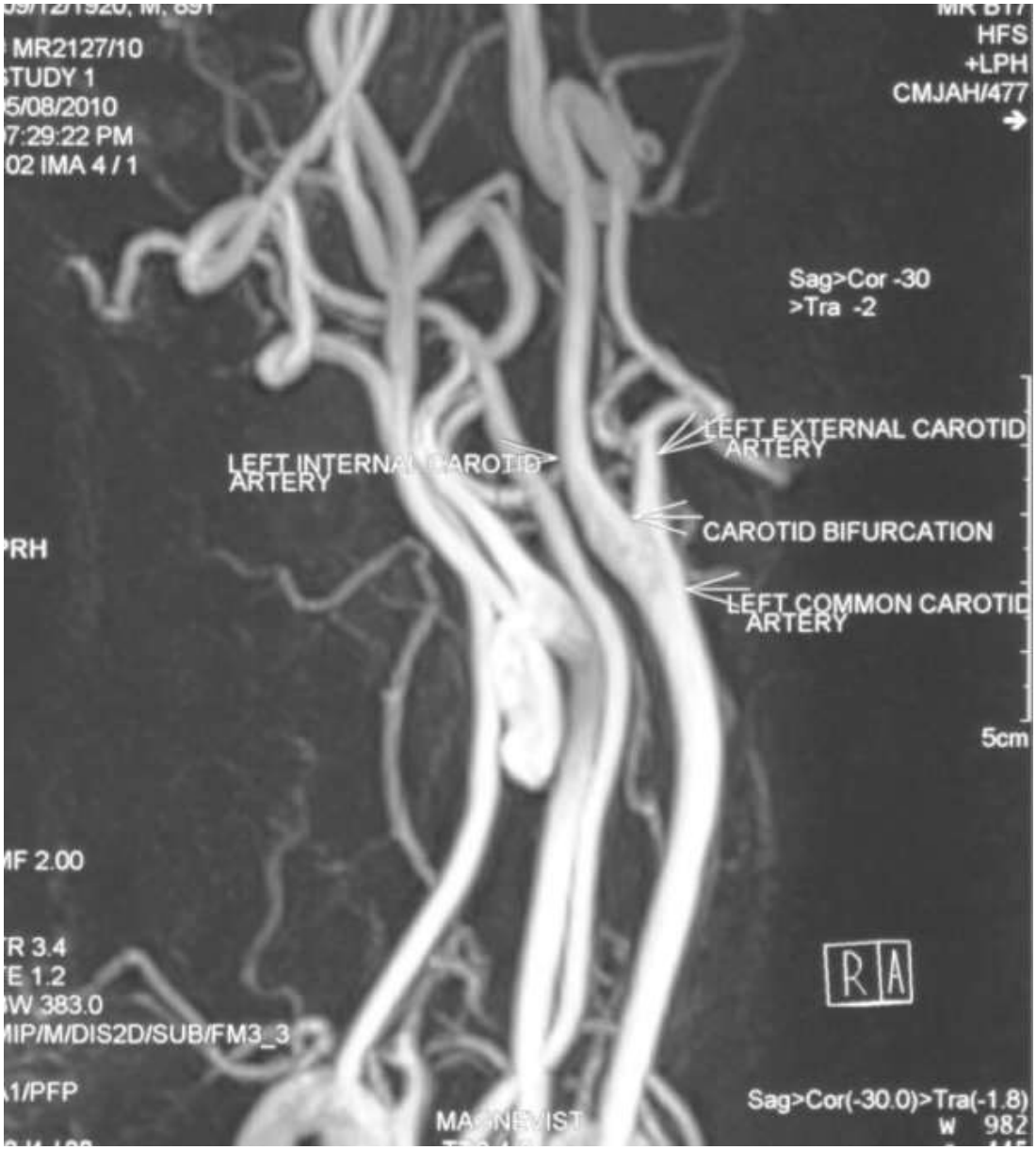


Fig. 3.14. An MRI of the neck depicting the arteries. The **left common carotid artery**, the **left internal carotid artery**, the **left external carotid artery** and the **carotid bifurcation** have been labelled. Note the extreme complexity of vessels.



Fig. 3.15. An MRI of the neck depicting the arteries. The **left vertebral artery** has been labelled.



Fig. 3.16. An MRI of the neck. The **left common carotid artery** and the **left internal jugular vein** have been labelled.

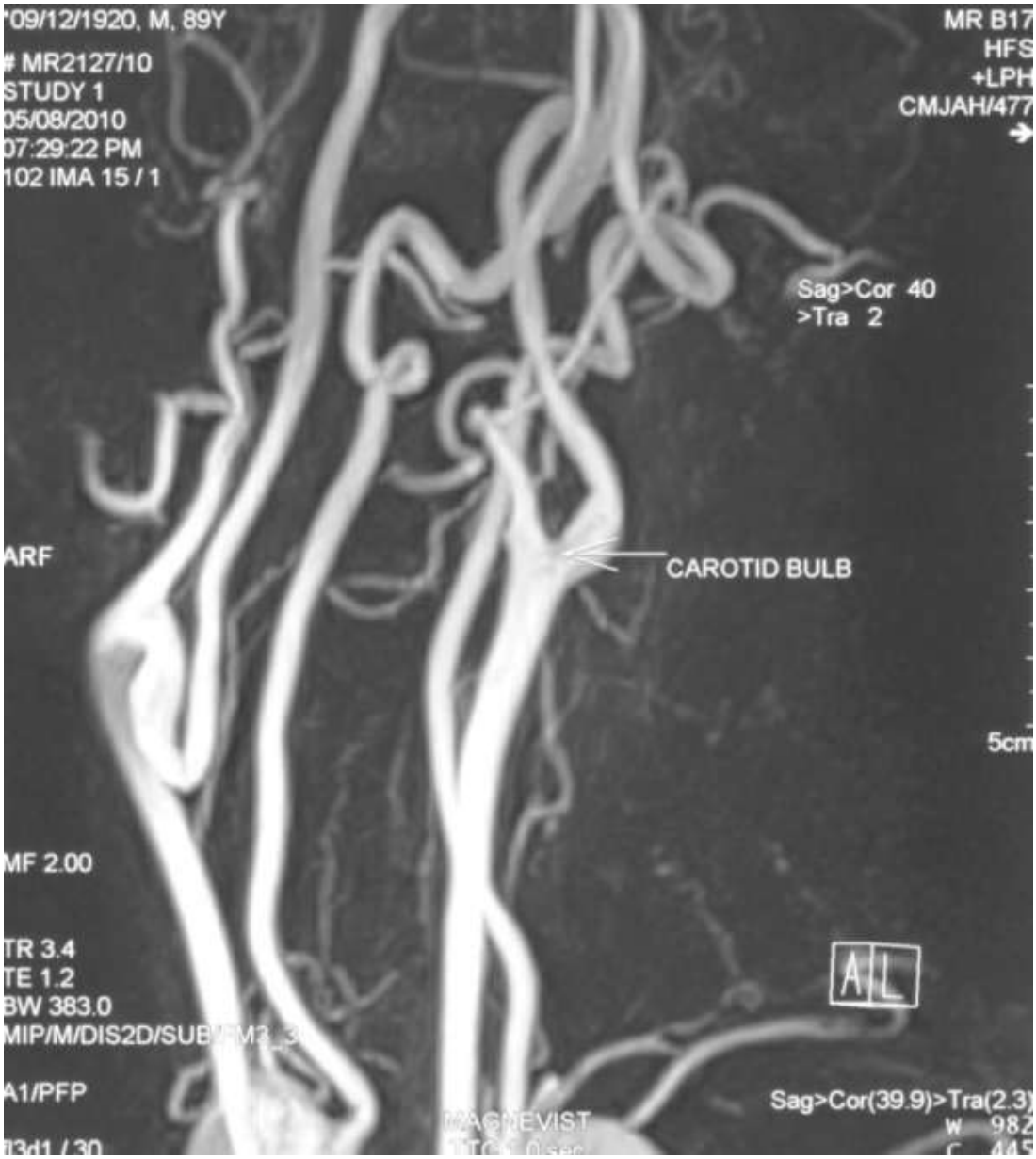


Fig. 3.17. An MRI of the neck depicting the arteries and veins. The **carotid bulb** has been labelled.

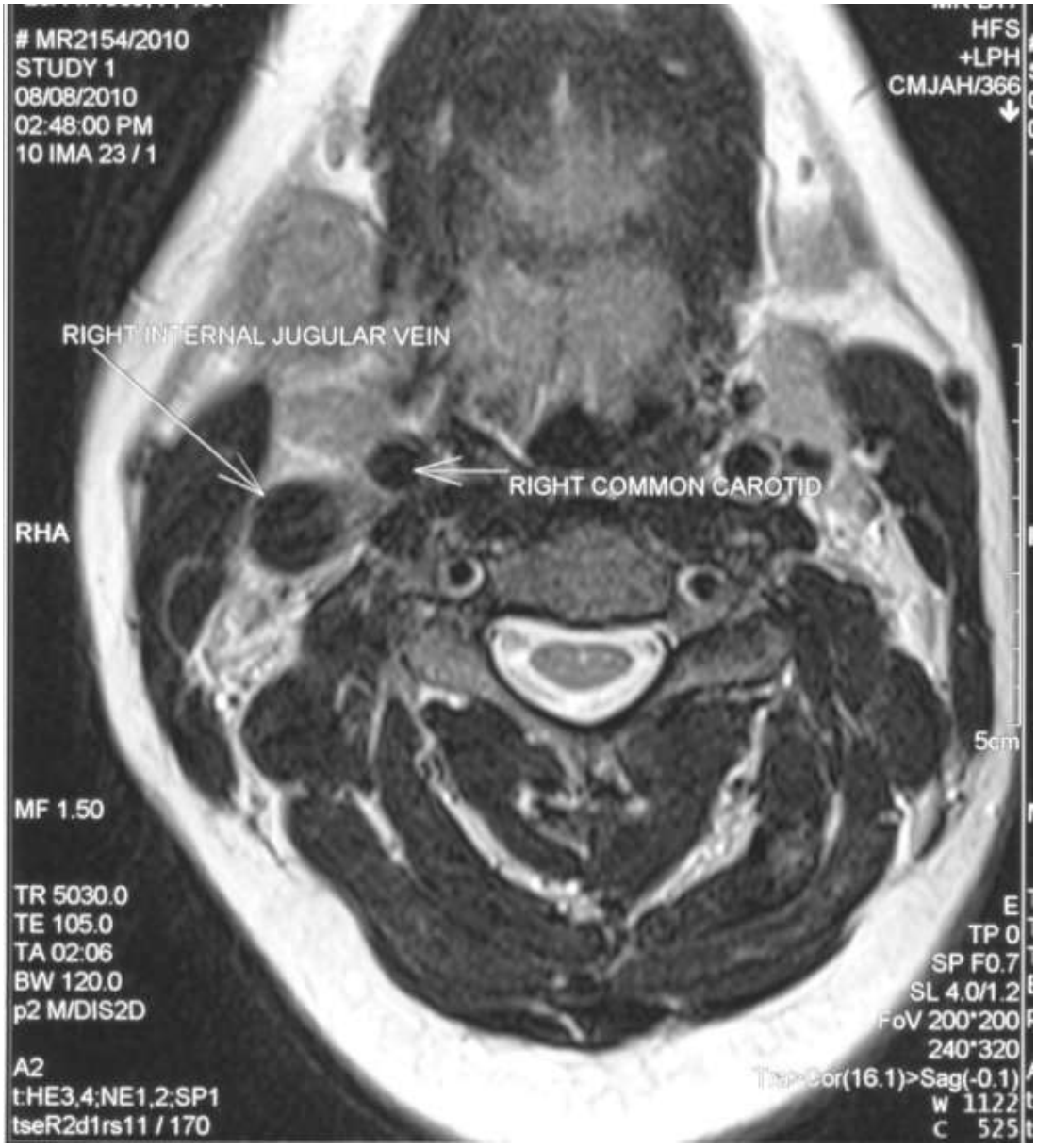


Fig. 3.18. An MRI of the neck in transverse section. The **right common carotid artery** and the **right internal jugular vein** have been labelled.

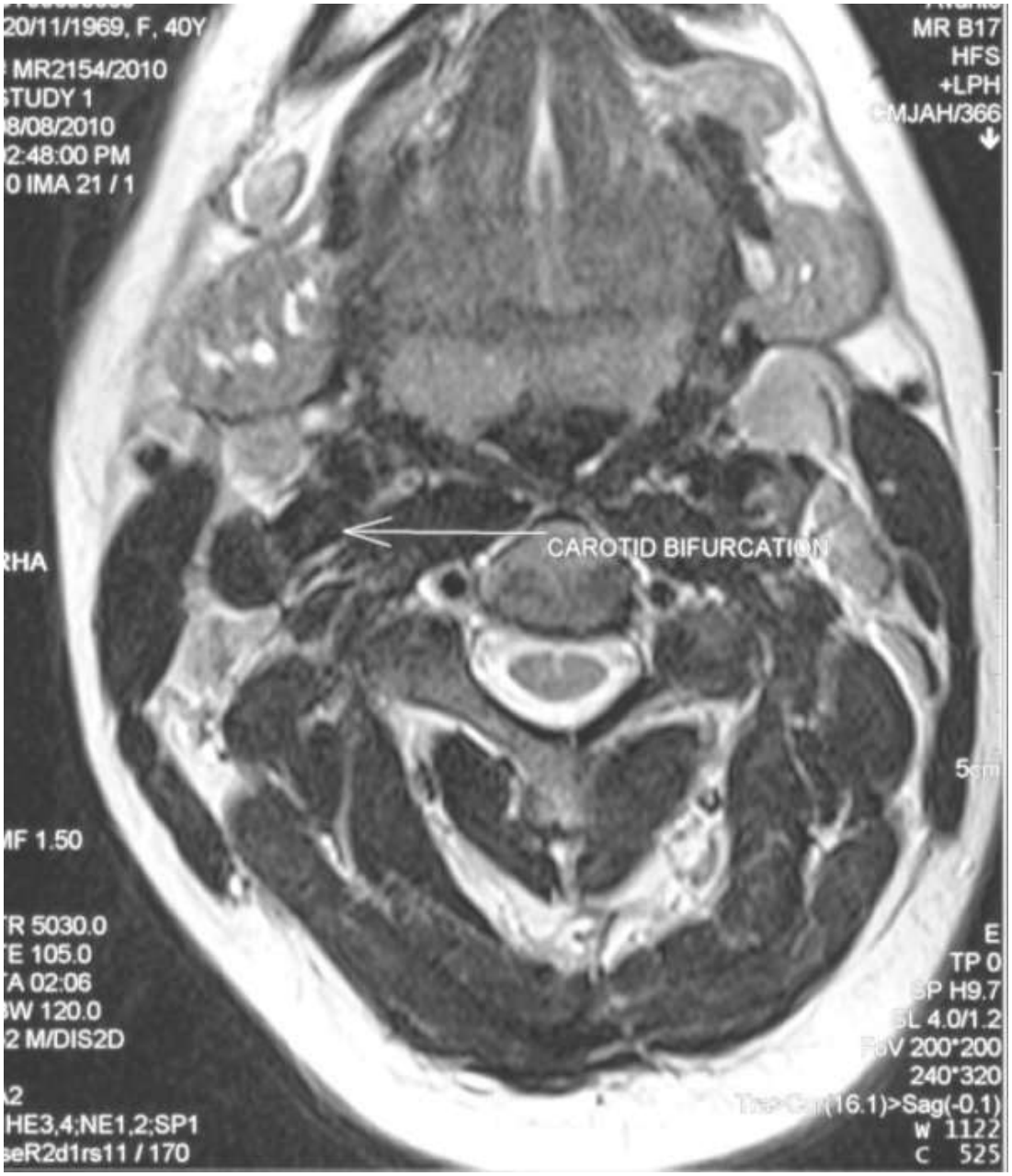


Fig. 3.19. An MRI of the neck in transverse section. The **carotid bifurcation** has been labelled.

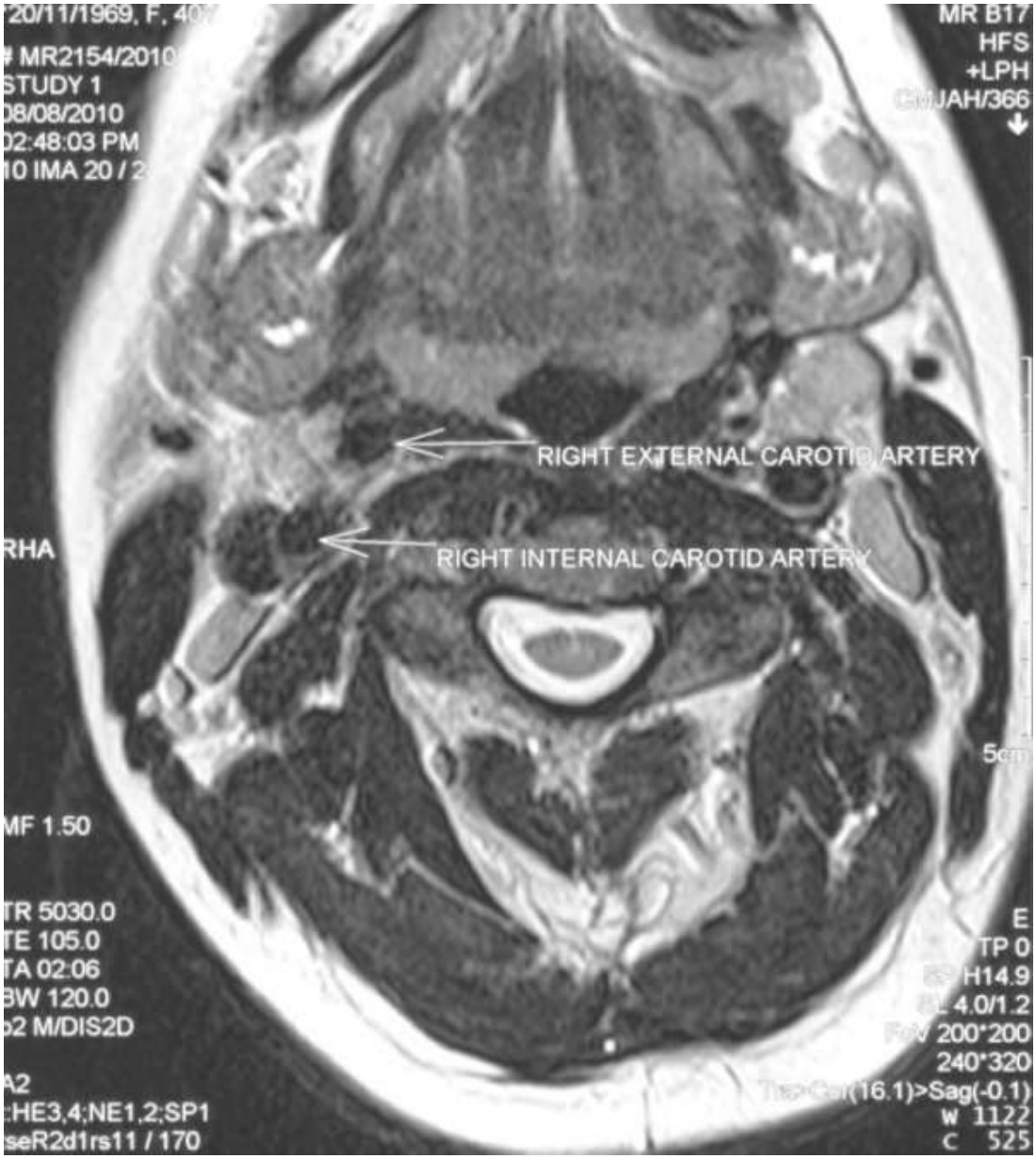


Fig. 3.20. An MRI of the neck in transverse section. The **right internal carotid artery** and the **right external carotid artery** have been labelled.

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CHAPTER 4

THE FUNCTIONAL MECHANISMS AND CLINICAL SYMPTOMATOLOGY OF DEATH BY HANGING

THE EARLY RESEARCH FINDINGS

Hanging has traditionally been considered and classified as an "asphyxial" mode of death. The term "asphyxia", however, has acquired a broad definition, indeed quite different from its etymological derivation of "pulselessness". Adelson, a noted American forensic pathologist, has provided a very broad-based but rather awkward definition of asphyxia as: "the physiologic and chemical state in a living organism in which acute lack of oxygen available for cell metabolism is associated with inability to eliminate excess carbon dioxide." [1] In terms of Adelson's statement, therefore, this definition would include cerebral ischaemia on the basis of carotid artery compression with decreased blood flow to the brain. Critical consideration of this definition, however, indicates that it is common to almost all modes of death.

According to Stedman's Dictionary, asphyxia is defined as: " impaired exchange of oxygen and carbon dioxide, usually on a ventilatory, *not* (italics are mine) circulatory basis; combined hypercarbia and hypoxia". Stedman gives 8 types of asphyxia. Dorland's Medical Dictionary, on the other hand, defines asphyxia as: " stopping of the pulse: a condition due to lack of oxygen in respired air resulting in impending or actual cessation of life". The emphasis in Dorland, therefore, is on pulselessness which, while indicating absence of circulation, appears to relate rather

to the final physiological outcome rather than to any specific preceding condition which resulted in absence of circulation.

Epidemiologists, unfamiliar with the precise terminology employed by forensic pathologists, necessitated by the latter's frequent subjection to cross-examination in court, have gone so far as to state that:" the usual cause of death in hanging is asphyxia". [2] This loose use of the term is to be deprecated in view of the popular association of the term "asphyxia" in the mind of the layman (such as judges and lawyers) and not a few medical men, with airway occlusion as the primary and predominant mode of death in hanging. This practice is, therefore, to be decried, with a further American pathologist categorically stating that:" one should refrain from the use of the term "asphyxia" except where one is obliged to speak in broad generalities". [3]

Asphyxia, thus, either inadvertently or by design, really implies interference with adequate delivery of oxygen to the lungs and eventually to the brain. This may occur in several ways: either by a noxious gas or airway occlusion however caused, e.g. a secretion soaked gag in the mouth or choking by a food bolus obstruction. The end result is, inevitably, cerebral ischaemia and unconsciousness and, possibly, death. Because of the vagueness of the definition, as expressed in a number of medical dictionaries, as well as its divergence amongst different authors with only a "partial application" to hanging, it is recommended that the term be expunged from the "hanging literature" as the sole cause of death and probably <u>not</u> the cause of death. Asphyxia is but one component of the pathophysiology of hanging, and, in the light of both early and more recent experimental and pathological findings, of lesser significance than, in the opinion of the present author, other mechanisms of death

operative in the pathology of hanging and to which, perhaps, undue prominence has been given and attention directed.

The Early Experimental Observations

The effects of applying the weight of the body to a ligature around the neck are complex (a purist, such as a physicist, would query the term "weight" rather than "mass". By definition, "weight" is the physical attraction of the effect of gravity on a "mass"). While the effects are modified by the kind of ligature used, whether it be a fixed or running noose and, if fixed, by the position of the knot, the effects depend more upon the degree of suspension, whether complete or incomplete. Most crucially from the point of view of pathology, however, as observed and determined by the morphological and histopathological findings, is the force exerted and damage inflicted on the underlying vascular and nervous structures whether unilateral or bilateral.

Few people appreciate the ease with which it is possible for hanging to take place from a low point of suspension, no more than a few inches from the floor. This, however, is readily understood when it is appreciated how little force is required to occlude the vessels of the neck nor how much force is applied when, in a semi-reclining position, only a fraction of the total body weight is exerted upon the ligature.

Experiments conducted by Hofmann on cadavers, confirmed in 1897 by Brouardel, [4] showed that the jugular veins are closed by a tension in the rope of 2 kg (4.4 lb) and when this was raised to 5 kg (11 lb), the carotid arteries were closed. An increase to 15 kg (33 lb) closed the trachea and at 30 kg (66 lb) the vertebral arteries

were closed. Since the latter lie within the bony canals of the transverse processes from C_6 to C_1, this compression must take place below C_6. This would be entirely in keeping with the anatomy, as the vertebral arteries do not traverse the transverse foramina of C_7, creating a potentially vulnerable locus for compressive force. It is only with the downward drop in judicial hangings that sufficient force is routinely exerted to produce significant vertebral artery and cervical spinal injury. In view of the fact that judicial hangings were routinely employed as a mode of execution in Victorian England, it is not surprising that cervical spinal injuries would figure so prominently.

Judicial Hanging

Knight has provided an excellent description of judicial hangings in Britain and the mechanisms operative. [5] Knight emphasizes that the modern form of judicial execution is unrelated to the usual suicidal hangings seen in routine forensic practice, as the former depends upon severe mechanical disruption of the neck structures. In Britain, until the nineteenth century, judicial hanging was carried out by "ordinary" hanging, where the victim was strangled at the end of a rope by his own weight. Before this, in Britain as well as in the West Indies, hanging by means of suspension by chains, although not around the neck, was used as a method of torture and execution, suspension being effected by an iron hook through an incision made in an intercostal space.[6] The victim was then left to die of thirst, starvation and exposure. Once this had occurred, the practice in the West Indies was to place the body on view in an iron cage, usually at the entrance to a harbour, one harbour in particular being graced with the name "Gallows Point".

At Tyburn docks, near Marble Arch in London, thousands of people (and not only for the crime of piracy), were put to death. The usual method employed was the placing of a rope noose around the neck of the condemned person, who stood on a cart. Following removal of this support, the victim was left suspended. While many died without further movement, many did not, and it was then left to the victim's relatives to pull on the victim's legs in order to shorten the agony.

It was in an attempt to increase the rapidity with which death occurred that certain innovations were introduced, the main one being the introduction of a drop brought about suddenly by means of a trapdoor. The provision of a drop required the placing of the "knot", in reality a brass eyelet and rubber washer, under the left ear, where it was less likely to be pulled over the head, than at the traditional site, e.g. the occiput. The object was to "break the neck" when the falling body was suddenly arrested, the executioner relying on the empirical observation that this manner of execution would result in death. It is unlikely that in the light of seventeenth century knowledge of physiologic anatomy, an awareness would exist that arresting the drop in this way would result in dislocation of the odontoid process of the axis vertebra with crushing of the spinal cord at the lower end of the medulla. As the vital centres are situated at this latter site, crushing injury would result in immediate cessation of respiration and cardiac arrest. Interestingly, exhumation of persons executed by this method showed that cervical spinal fracture was unusual. However, post-mortem findings indicated that atlanto-occipital fracture-dislocation and spinal cord transection was common, a not unexpected finding in view of the sudden traction exerted.

The effects of hanging by this method appeared to have been variable, with some accounts describing decapitation of the victim while other victims appeared to

not even have lost consciousness. Despite the cord or brainstem damage sustained, the heart continued to beat, sometimes for up to 20 minutes. Again, this is a not surprising finding in view of the heart's ability to switch from aerobic to anaerobic metabolism. In addition, physiologic experiments have shown that the isolated heart muscle preparation has an "idiopathic rhythm" of about 50 beats/minute accounting for the continued beating despite brainstem/cervical spinal cord severance.

While Knight provides an excellent description of judicial hangings as they were carried out in Britain, these procedures were also carried out in the American colonies, most notably on those convicted of the crime of piracy. According to the Maritime laws extant in Boston in the sixteenth and seventeenth centuries, a condemned pirate was to be hanged within ten days of being found guilty. The time between the sentencing and the hanging was to allow the condemned man to repent and thus save his immortal soul. Interestingly, this is somewhat analogous to the practice of the Holy Inquisition in Spain in which repentance, while not saving the person from the death sentence, allowed hanging rather than burning at the stake as the mode of execution.

The ten day period between pronouncement of the death sentence and its execution was not only to allow the deceased time to repent, but had a practical application as well. As in Britain, hangings were public spectacles designed as a deterrence to those who might consider carrying out similar crimes. The ten day period not only allowed the condemned time to repent, but also gave time for people from neighbouring towns to travel to the place of execution to view this "enlightening" and chastening spectacle.

As in Britain, the condemned man would stand on a cart, and, once this was pulled away, the victim would be left suspended in the air. Alternatively, a rope

would be passed through a pulley with a noose at one end and a few men or a horse at the other. When it was time to carry out the sentence, the condemned would be hoisted into the air and the rope tied off. Either way, contemporary descriptions indicated that convulsions would take place, so much so that hangings were also referred to as the "Devil's Jig", or "Gallows' Dance". The important point is that the convulsions occurring were indicative of the cerebral ischaemia which took place.

Keelhauling, previously alluded to briefly in Chapter 1, was a form of corporal punishment originating in the Dutch Navy. The term derives from the Dutch word *kielhalen* meaning: "to drag along the keel". This was legally permitted in the Dutch Navy, the earliest official mention being a Dutch ordinance of 1560, and was not formally abolished until 1853. While not an official punishment in the British Navy, it was nevertheless used by some British Royal Navy and merchant marine captains. In this context, it should be remembered that in the Royal Navy on the high seas, the Captain was the absolute master of his ship and could hand out punishments as he saw fit.

In keelhauling, the sailor had several loops of rope wound around the body, the rope then being looped beneath the vessel. The sailor was then thrown overboard on one side of the ship, and dragged under the ship's keel to the other side. Apart from the risk of the neck becoming entangled in the rope, as well as concomitant drowning, the underside of the keel was usually covered in barnacles which could produce cuts and abrasive injuries. The latter would result not only in haemorrhage, but also in wound infection from marine organisms.

The figures quoted by Brouardel appear to have been accepted and quoted by all modern authorities who have, in addition, stated that the weight of the head (4.5-5.4 kg; 10-12 lb) against a noose is sufficient to occlude the carotid arteries.[7] This is

one of the factors which accounts for death occurring from a low point of suspension with only a fraction of the total body weight exerted upon the ligature. From a biomechanical point of view, the body can be divided into different segments, each with its own mass. [8-10] In incomplete hanging therefore, each of these continue to exert pressure greater than that required for carotid occlusion. [11, 12] With regard to the question as to the pressures initiated within the carotid arteries on application of compressive force, and in view of the importance of the biomechanical and pathological issues (such as vessel fracture and rupture) which then arise and ensue, this is more comprehensively dealt with below. To these issues a separate chapter (Chapter 5) has been devoted.

Reuter in 1901 repeated these experiments and found that even less tension was required to produce carotid and vertebral arterial occlusion, tension in this context meaning the "pull" on the ligature. The carotid arteries, for example, were closed by a tension of only 3.5 kg (7.7 lb) while the vertebral arteries were closed by a tension of 16.6 kg (36.5 lb).[11] It should, however, be borne in mind that these experiments were conducted on cadavers with no systemic systolic pressure being present within the vascular system. Bearing this in mind, Polson himself confirmed experimentally that the carotid artery is appreciably obstructed by a ligature under low tension. Having first established free flow of fluid between the common carotid artery, exposed in the upper chest, and the internal carotid artery, seen inside the skull after removal of the calvarium, Polson then applied a running noose (i.e. with a sliding knot) around the neck. Weights were added and injection was repeated below the level of the ligature. The experiment showed that a pull of as little as 3.2 kg (7 lb) was sufficient to reduce free flow through the artery to a mere trickle. [12] What Polson failed to take into account, however, was the arterial counter- pressure within the

carotid arteries which, in life, is equivalent to that within the aorta itself, as well as the sympathetic vasoconstrictor tone which maintains a partial state of contraction in the blood vessels all of which factors act to resist compression and maintain the intravascular pressure (fluid is incompressible) and patency of the vessels during life. This experiment, therefore, cannot truly reflect the situation in a living vessel (unless a willing subject subjected himself to this procedure, which appears unlikely) or the experiment was carried out on an unwilling subject, in which case the ethics of the profession would have been breached with litigation following.

An interesting clinical point made by Polson is that severe obstruction of the carotid arteries, requiring only a tension of about 3 kg (6.6 lb, i.e. slightly more than the weight of the head), will rapidly induce cerebral anoxia and unconsciousness. A simple but interesting calculation follows: 3 ÷ 13.6 = 0.220 mm Hg; 1 kg = 1000 mg. This would therefore be equivalent to a pressure of 220 mm Hg in a narrow tube, i.e. equivalent to severe hypertension.

Consciousness is lost within about 10-12 seconds, thus accounting for the failure of suicides to save themselves should they change their minds. It appears that once launched upon suicide by hanging there is no retreat. Of course, in the intervening 10-12 second interval between initiation of pressure and resulting unconsciousness, the self-induced victim of hanging does have sufficient time to make abortive attempts at loosening the ligature. These grasping attempts by the victim would then result in self-induced fingernail abrasions over the front and sides of the neck. This accounts for the occasional finding of fingernail abrasions on the neck surrounding the ligature in victims of suicidal hanging, giving rise to suspicion of either ligature strangulation or homicidal hanging, latter instances of which have been described. [13-15]

Interesting research was also conducted in the nineteenth century on hanging in dead bodies. Between the years 1817 and 1855, Casper (previously alluded to in Chapter 1) performed 23 experiments to determine the effect of hanging on a cadaver. [16] These experiments lead Casper to conclude that the ligature impressions seen on the neck in hanging during life could also be produced by a ligature applied to the neck within 2 hours, if not later, after death. This point is of great medicolegal significance in the context of simulated hanging with death being due to other causes.

Modification of the Mechanism by Alteration of the Position of the Knot

When a fixed loop is used with the knot positioned posteriorly, the weight of the body loads mainly on the front of the neck with a force of almost equal amount being applied also to the sides of the neck. Experiments conducted by Langreuter in 1886 showed that the loop then draws the base of the tongue upwards against the posterior pharyngeal wall.[17] This folds the epiglottis over the entrance to the larynx thus occluding the airway. Using the bodies of persons who had died of natural causes, Langreuter cut away part of the skull and prepared the parts in a manner which allowed direct inspection of the effects of applying a ligature as in hanging. He then found that only moderate tension was required to close the air passages and this was due to the ascent of the tongue, with displacement folding back the epiglottis. In contrast, when the ligature was applied over or below the thyroid cartilage, the vocal cords were not closed by an upward and backward pull of great force. Experiments conducted by Dixon Mann in 1908 using a rolled handkerchief yielded similar results, but they required greater force than did a cord probably due to diffusion of

the force. [18] This is understandable in view of the fact that a handkerchief consists of a softer and more broad-based material than a cord which is narrower, the latter resulting in a less diffuse and more focal application of force.

Ecker in 1870 conducted empirical observations on the frozen body of a man found hanging from a tree in winter. [19] When Ecker sectioned the head and neck in the vertical plane, it was shown that the tongue was folded upon itself and had ascended to the naso-pharynx. Hofmann (cited by Tidy in 1883) confirmed this observation by section of bodies which were first frozen to preserve the position of the parts. [20] Nevertheless, it was the opinion of Hofmann that large vessel occlusion was the more important factor in hanging, the result being immediate arrest, or severe reduction of, cerebral circulation. Kalle, in 1933, found, in addition, in judicial hanging, when these were still carried out in Britain, that the vertebral arteries may remain patent and the heart-beat continues for from 5 to 20 minutes. [21] Once again, this represents an example of idiopathic rhythm as demonstrated in isolated heart muscle preparations with denervation of the heart.

While an added anatomical factor is that, with age, the thyroid and cricoid cartilages become calcified and even ossified, closure of the airway does not appear to be an essential component of the hanging process despite, as previously indicated, the prominence attributed to asphyxia in the mind of the layman (and non-forensic pathologists) in association with hanging. This misconception is probably due to the fact that asphyxial phenomena may dominate the clinical picture when hanging has taken place in the semi-reclining or sitting position, especially when a sliding knot is present. This allows continued flow of blood to the head above the level of the ligature with facial congestion and petechiae of the face. In contrast, when

suspension is complete, or in the standing position, the victim usually presents (but) slight evidence of these asphyxial signs.

Confirming the non-essential element of closure of the airway in hanging was an interesting case described by Reineboth in 1895 involving the suicide by hanging of a man who had had a tracheostomy for carcinoma of the larynx. [22] Although the victim had died from hanging, the ligature was above the level of the tracheostomy site with a patent airway therefore still being present. Bertelsmann, an early twentieth century author, described a similar example. [23]

The Clinical Symptoms of Hanging

Review of the early literature is particularly valuable in this regard, not least because of experimental observations as well as of inadvertant instances of hanging. Of course, in the so-called sexual asphyxias, the manner of death is accidental not suicidal, with the victim by and large not surviving to give an account of his symptoms. Perforce, attention must, therefore, be directed to those exceptional accounts of self-experimentation together with the accounts of stage performers who got into difficulties and were resuscitated as well as instances of attempted hanging in the context of suicide

Experimental hanging was described anonymously in a nineteenth century journal, the Medical Times and Gazette in 1882. [24] The symptoms then experienced were a feeling of heat in the head, visual flashes of light, a deafening sound in the ears, and numbness in the legs. Tidy, commenting on this in 1883, said that loss of consciousness had been sudden.[20] The event may apparently be either silent or noisy and there may be no convulsive phase. In this regard, Ogston in 1878 reported the

case of a man who had hanged himself in a sitting position in bed and yet two children who shared the bed with him were not aroused from sleep.[25] However, it is not known whether this incident occurred in a Victorian poorhouse where the occupants, including children, were notoriously provided with liberal amounts of gin before bedtime in order to ensure a quiet night for the caretakers. Had this indeed been the case, with the children falling asleep in a drunken stupor, it is unlikely that they would have responded to any auditory stimuli. Unfortunately, this incident remains in the realms of speculation.

An exceptional example of self-experimentation was that carried out by Minovici, a 79 kg man who underwent complete suspension, his feet being raised a metre above the ground.[26] From the first moments, until he descended, he experienced pain at the site of the knot, probably due to skin pain from pressure on and stretching of the underlying skin. The pain to the right of the hyoid bone was so intense, possibly due to the conglomerate mass of nerves in this region, that he could not continue the experiment. This is of interest in that it refutes the prevailing views extant in the Department of Forensic Medicine in Stockholm, as reported to the present author (Moar) by two exchange students from that department, that the process of hanging is essentially painless. This emphasizes the value of reviewing the early literature and the findings emanating therefrom and, most importantly, making a detailed study of the anatomy of the region.

Further findings emerged from Minovici's self-experiment. As soon as his feet left the ground, his eyelids contracted violently and closure of the airway was so complete that he found it impossible to breathe, in all probability as a consequence of upward displacement of the tongue into the nasopharynx. This may either confirm that any weight greater than 15 kg produces tracheal closure or that the diaphragm

and intercostal muscles have insufficient force to expand the thorax when the entire weight of the body bears upon them. In addition to respiratory difficulty, Minovici could not hear the voice of his assistant who was holding the cord, as he counted the number of seconds. This is unlikely to be a nervous phenomenon but rather due to blockage of the ostium of the pharyngo-tympanic tube by the previously mentioned upward displacement of the tongue into the nasopharynx. Whistling in his ears and inability to breathe necessitated an abrupt termination of the experiment.

When Minovici was released, he had watering of the eyes and increasing difficulty in swallowing due to pharyngeal congestion. The pain was especially severe in the region of the greater horns of the hyoid. This latter finding is not unexpected in view of the traction exerted on these latter structures and was probably due to trauma to the nerves and vessels specific to this region. These symptoms persisted for 10 to 12 days in all likelihood due to oedema and bruising (as found in the present study), accompanied by great thirst persisting for 1 or 2 days. In this regard, it should be borne in mind that two salivary glands are associated with the myelohyoid muscle and these could have been damaged either by the ligature in the upper part of the neck (zone III) or by displacement. The ligature impression presented as a groove associated with multiple confluent haemorrhages, especially to the right of the hyoid bone and near the mastoid process. These haemorrhages appeared at about 5 to10 minutes after his release and persisted for 8 to 11 days.

Loss of consciousness is rapid. Minovici became unconscious almost at once, when a maximum tension of only 5 kg was exerted on the ligature. When suspension was incomplete, i.e. when he leant on the cord, his face at once became suffused and his sight blurred. All this took place within 5 to 6 seconds. When the knot was at the nape (back) of the neck, the ligature rapidly closed the blood vessels and airway.

This would occur due to symmetrical or almost symmetrical compressive force being bilaterally exerted on the vessels with the trachea being compressed in an antero-posterior direction. Tolerance of the ligature when its knot was at the side of the neck exerting a more unilateral force was more prolonged, but only for about 8 or 9 seconds.

Minovici also tried the effects of a running noose, i.e. one with a sliding knot. Despite his courage, however, after experiencing effects similar to those described, he stopped the experiment within 5 seconds.

Recovery after attempted Hanging

With regard to recovery after attempted hanging, a woman who hanged herself when drunk was rescued within a few minutes. She was found to be unconscious, with pallor of the face and slow and laborious breathing. Despite suspension having been effected by a silk handkerchief, a dusky one-quarter inch (6.25 mm) ligature impression was present around the neck.[20] This empirical evidence slightly contradicts the conventional view that soft ligatures such as scarves tend to leave less prominent impressions than firmer ones such as electrical cords or belts.

Petrina in 1880 described the recovery of a victim of attempted suicide by hanging.[18] The man remained unconscious for 24 hours during which time he experienced violent clonic convulsions of the entire body, followed by generalised muscular rigidity. When he became conscious, he had a crossed paralysis involving the right side of the face and the left side of the body. This neurological state may have been contributed to by pre-existing arterial atheromatous occlusive disease of

the carotid arteries. This passed but was followed by a right sided ataxia, later extending to both sides.

Two further cases of recovery following unsuccessful attempts at hanging were described by Terrien in 1887.[27] In the first case, epileptiform convulsions were accompanied by tetanic spasm, the victim remaining unconscious for several days after the hanging while in the second case, the convulsions were accompanied by opisthotonus. When the victim recovered consciousness, he made movements as if ambulant. All these effects thus seem to be neurological in origin, in all probability due to cerebral ischaemia from arterial obstruction or reduced cardiac output.

Two years later, in 1889, Wagner reported a case where an acute dementia was attributed to an attempted suicide by hanging.[20] However, while a modern psychiatrist would probably argue that the dementia and its mental aberrations may have precipitated the attempt at hanging rather than be caused by it, this could contrarily be argued that the victim had exhibited no signs of mental illness prior to the hanging episode.

An interesting case of recovery from hanging occurred when one Harnshaw, a stage performer accustomed to suspending himself for public entertainment, misjudged his act. On recovery, he related that he had lost consciousness almost immediately. He could not get his breath and felt as if a great weight was attached to his feet. A significant point is that he had been unable to move his hands to save himself.

In summary, therefore, the great value of both these observations and “living experiments” by the early investigators of the nineteenth century lies in their having laid the foundations for the elucidation and understanding of the basic mechanisms involved in the hanging process. However, the apparent lack of knowledge of the

anatomy and physiology of the structures of the neck region seems to have escaped the investigators. This is unfortunate in view of the fact that the first edition of Gray's Anatomy was published in 1858.

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CHAPTER 5

SHOULD BIOMECHANICAL AND NEURAL FACTORS BE CONSIDERED AS PLAYING A PIVOTAL ROLE IN HANGING?

THE ROLE OF VAGAL INHIBITION, THE AUTONOMIC NERVOUS SYSTEM, VENOUS COMPLIANCE, AND VESSEL STRETCH, COMPRESSION AND HYDROSTATIC PRESSURE WAVES

Four basic mechanisms of death have traditionally been considered as operative in pressure to the neck which occurs in hanging. These comprise airway occlusion, occlusion of the neck veins, carotid artery compression, and vagal inhibition. These act either singly, or in combination, with the role of vagal inhibition continuing to intrigue forensic pathologists. While all British forensic pathologists of authority such as Vanezis and Knight, as well as their American counterparts, agree that vagal inhibition plays a role in death causation, argument still persists as to whether this role is merely ancillary, or whether it is central and pivotal.

Furthermore, insufficient attention appears to have been drawn to the contributory role of vessel stretch. In view of the suspension and traction exerted on the constricting ligature by the weight of the body (or part of the body, as previously indicated) which occurs in hanging, be it complete or incomplete, longitudinal vessel stretch must therefore constitute an integral part of the hanging process. This longitudinal stretching of the blood vessels would, in part, account for the tearing and disruption of the various layers of the wall of the blood vessels. Dissection of blood, in particular along the laminar planes of the media, and as noted in the present study,

would then occur due to the fact that a head of blood pressure is still present because the heart, as previously indicated, is still beating not least because of its inherent idiopathic rhythm.

In terms of hydraulics, it is known that significant hydrostatic pressure waves are initiated as a response to application of pressure to a compressible fluid-containing vessel. The fluid itself, being incompressible, responds to pressure application transmitted through the vessel wall in the form of a propagating wave of pressure and displacement of blood. The explanation for the finding of haemorrhage, therefore, in particular of the outer, adventitial, layer of the arterial wall in the present study, must accordingly lie in a detailed understanding of the anatomy, physiology and traumatic pathology of the structures of the neck. The explanation of the possible causes of unconsciousness and death, therefore, forms the basis of the present study.

The question of whether a differential of pressure exists if the ligature pressure is applied suddenly or slowly raises interesting issues. While no experiments have been performed on living subjects with the intravascular pressure being monitored and while animal experiments cannot directly be extrapolated to humans in view of differences in anatomy, logic dictates that the intravascular pressure must rise at the site of application of a ligature. The issue of intravascular pressure is dealt with below. However, the issue of sudden application of external pressure (not to be confused with a rise of intravascular pressure or initiation of a hydrostatic pressure wave) carries great physiological and medico-legal implications in the context of vagal inhibition with its bradycardia, cardiac arrest, and collapse of the victim. The fundamental mechanism of death in this context is neurological, not vascular and which has given rise to misunderstanding in the minds of lay people

such as lawyers and judges. This has formed the basis of more than one court case and its legal arguments as to intent is dealt with below.

An Overview of the Four Basic Mechanisms

Airway Occlusion

In essence, a strong constricting cervical force applied externally, as by a ligature to the neck, will result in compressive narrowing of laryngeal and tracheal lumina. It should be borne in mind, however, that these are air-containing tubes with an intraluminal pressure at sea level of 760 mm Hg. Pressure exerted from without does not alter the intraluminal pressure. However, as discussed in Chapter 8, disturbances in blood flow influence parameters at the wall. This should be differentiated from choking which is blockage of the airways internally, as by a food bolus or secretion-soaked gag. As indicated in the previous chapter (p. 92), a force of 15 kg is sufficient to occlude the trachea. If the force is directed in a cephalic direction as in hanging, the pharynx and posterior third of the tongue are elevated and forced backward, with consequent obstruction of the laryngeal opening. The net result is thus airway occlusion

Obstruction of Jugular Venous Return

Cervical anatomical relationships are such that any external force capable of narrowing or occluding the airway must necessarily compress the internal jugular veins lying in the neighbouring bilateral carotid sheaths. It has been indicated, in the previous chapter (p. 92), that a compressive force of only 2 kg is sufficient to occlude the jugular veins because of the low venous counter-pressure. Even so, this force carries serious physiological sequelae.

Two principal factors act to affect the function of the systemic circulation in relation to regulation of the cardiac output. These are: 1) the degree of filling of the systemic circulation, e.g. as found in haemorrhagic shock, and; 2) the resistance to blood flow in the different segments of the circulation. As indicated in greater detail below, in the context of hanging, a number of factors act in concert to cause damming up of blood in the large capacitance venous vessels such as the superior and inferior venae cavae. It should be borne in mind that the heart fills from both above and below via these two veins. The greater part of the superior vena caval inflow is from the two jugulars. Hence, blocking the jugulars would result in reducing the venous return thereby reducing the cardiac output and thus the stroke volume. It could be argued that a decreased cardiac output with decreased blood flow to the brain and its consequent ischaemic/anoxic effect might play a major contributory role in producing the rapid unconsciousness and other signs described in the "living experiments" in Chapter 4 (pp. 101-104). However, the normal blood flow through the brain of the adult averages 50 to 55 ml per 100 grams of brain tissue per minute. For the entire brain of the average adult, this translates to approximately 750 ml per minute, or 15 per cent (one-sixth) of the total resting cardiac output.

Therefore, even with complete obstruction to blood flow in the jugular veins produced by an encircling ligature, only one-sixth of the cardiac output would be affected. This is hardly enough to affect the CNS ischaemic response which only becomes activated when the systemic arterial pressure drops to half normal, e.g. 60 mm Hg, reaching its greatest degree of stimulation at a systolic pressure of 15 to 20 mm Hg, e.g. one-sixth of normal. Furthermore, as far as we know, the phenomenon of consciousness is located in the cerebral cortex (and having a separate existence to the brain in the view of those who believe in the near-death phenomenon where consciousness, on the basis of many empirical anecdotes, splits off from the brain at or near death and transcends physical demise) and the circulation to the cortex is large. As a counter-argument to the role of activation of the CNS ischaemic response under conditions of low arterial blood pressure, however, is that the sympathetic nervous system has already been activated, negating any need for initiation of this response. In essence, therefore, while counter-balancing and conflicting factors all come into play when a ligature constricts the neck, the fact remains that only one-sixth of the cardiac output would be affected.

Cervical compression which occludes the jugular venous system without simultaneously occluding carotid arteries results in acute passive hyperaemia above the obstruction with resultant overdistention of intracranial venous sinuses, cerebral, facial, palpebral and ocular bulbar veins as well as capillaries. This elevated back pressure, combined with anoxic endothelial damage, the latter occurring immediately after injury,[1] is responsible for the appearance of punctuate haemorrhages in the brain, subconjunctival regions of the eyelids and globes, and facial and neck skin above the level of constriction so typically seen in hanging. Adelson has made the point that the posterior laryngeal haemorrhages seen in some cases of cervical

compression is due to rupture of the thin-walled venous sinuses comprising the pharyngo-laryngeal plexus of vessels.[2]

Obstruction to Carotid Arterial Flow

The common carotid arteries are covered by skin, platysma muscle, deep cervical fascia, sternocleidomastoid muscle and carotid sheath from the level of the cricoid cartilage until their bifurcation at the upper border of the thyroid cartilage. During this part of their course, they rest on the unyielding surfaces of the transverse processes of the fourth, fifth and sixth cervical vertebrae. This anatomical configuration renders the patency of the vessels vulnerable to forces which can compress them against the subjacent vertebral transverse processes and cartilaginous larynx with any force greater than 3.5 to 5 kg causing carotid occlusion with consequent cerebral ischaemia, anoxia and loss of consciousness.

The vertebral arteries, on the other hand, are protected from this type of compressive pressure because they ascend through the transverse foramina in the cervical vertebrae, requiring a force of 16.6 kg for occlusion. However, they do not do so through the seventh cervical vertebra, creating a potentially vulnerable locus to compressive force. This site, however, lies below the usual point of suspension in suicidal hangings, tending to negate this vulnerable locus for compression. It is only with the drop in judicial hangings that injuries to the vertebral artery are commonly expected. A point of neurological, rather than anatomical, importance is that the vertebral arteries carry an insufficient supply of blood to the brain (2% of cerebral blood flow) to maintain neuronal viability and integrity in the face of complete bilateral carotid occlusion.

The Role of Carotid Sinus Stimulation and the Parasympathetic Nervous System in Vagal Inhibition

Reflexly induced vagal mechanisms play a crucial role in the production of cardiac irregularities, and bradycardia and cardiac arrest may be produced by stimulation of several functionally different receptors.[3] In particular, an important functional mechanism that controls reflex regulation of cardiovascular function originates from the carotid sinus, a fusiform enlargement of the internal carotid artery at the level of the carotid bifurcation. Specialized nervous receptors embedded in the sinus wall constitute the sensing element in a reflex arc whose afferent limb is formed by the glossopharyngeal and vagus nerves and whose efferent limb is the cardiovascular autonomic outflow. Stimulation of these receptors causes a profound vagus nerve mediated sinus bradycardia, elicited by an increase in intrasinus tension brought about either by pressure from within or pressure from without. This carotid sinus reflex carries not only physiological but clinical and forensic implications. Most intriguingly, the clinical manifestations of carotid sinus stimulation bear a marked resemblance to the symptoms and signs observed in those cases of attempted and experimental hanging described in the previous chapter.

What is the Position with regard to the Veins?

While great attention has been directed to the reaction of the carotid arteries to pressure, what would be the comparable position with regard to the neck veins? Because, anatomically, the walls of arteries are far stronger than those of veins, the latter, on average, are about six to ten times as distensible as the arteries. In other

words, a given rise in pressure will cause about six to ten times as much extra blood to fill a vein as an artery of comparable size. However, distensibility is quite different from compliance or capacitance, both of which are physical terms meaning the increase in volume that causes a given increase in pressure in any vascular area. The latter is of obvious relevance with regard to the compression of the neck veins (and, specifically, the internal jugular vein) which takes place in hanging.

The compliance of a vein is about 24 times that of its corresponding artery because it is about 8 times as distensible and it has a volume about 3 times as great (8 x 3 = 24).[4] This difference in compliance is important because it means that large amounts of blood can be stored in the veins with only slight changes in pressure. This is, however, counteracted by sympathetic stimulation which increases smooth muscle tone in the vascular walls, thus increasing the pressure at each volume of blood contained in the arteries or veins at that particular part of the circulation. Furthermore, the vascular effect of sympathetic stimulation with constriction of the cutaneous vessels expresses most of the blood out of the skin into deeper parts of the circulation. When this occurs, the skin assumes the colour of the subcutaneous connective tissue, which consists mainly of collagen fibres having a whitish hue. Thus, in conditions causing sympathetic stimulation with consequent vascular constriction, the skin takes on an ashen white pallor, accounting for the facial pallor which may be seen above the level of the ligature in hanging. This should not be confused with the mechanism of causation of petechiae which are small (one-tenth of a millimetre to about two millimetres) pin-point haemorrhages originating from the postcapillary venules as a result of venous obstruction with an acute rise in venous pressure. Thus, at the risk of overemphasis, two entirely separate mechanisms (and

findings) are in play, one due to an autonomic sympathetic response and the other to venous obstruction.

Counteracting this appearance, however, is the volume increase in the neck vessels due to their distensibility, which is, in turn, accompanied by slowing of venous blood flow due to the constrictive effect of the ligature. Slowing of blood flow allows more time for oxygen to be removed from the blood for uptake by the tissues before the blood can leave the capillaries. The practical consequence is that the capillaries and (distensible) veins then contain large amounts of dark, deoxygenated blood giving the skin a bluish hue in contrast to the ashen white pallor produced by a purely sympathetic effect.

A further factor complicating the above effects is the phenomenon of delayed compliance which occurs only slightly in arteries but to a much greater extent in veins. This phenomenon, known also as stress-relaxation, occurs when a vessel whose pressure is increased by increased volume then progressively stretches, gradually losing much of the volume-induced pressure increase. While the increased volume of blood in the neck veins above the level of the constricting ligature causes immediate elastic distension of the vein, the smooth muscle fibres of the vein begin to "creep" to longer lengths, and their tensions correspondingly decrease. Hence, the pressure which initially rose markedly then decreases.

While the function of arteries is to transport blood under high pressure to the tissues, the veins function as conduits (hence the inclusion of the term "conduits" in the title of chapter 3) for transport of blood from the tissues back to the heart. Approximately 84 per cent of the entire blood volume of the body is in the systemic circulation with 64 per cent of the systemic circulation in the veins, 15 per cent in the arteries, and 5 per cent in the capillaries (64 + 15 + 5 = 84). In addition, veins have a

much larger cross-sectional area than the arteries, averaging in total about four times those of the corresponding arteries. This is a further factor explaining the very large storage capacity of the venous system for blood in comparison with that in the arterial system. The actual cross-sectional areas of the arterial and venous vessels are as follows, the figures quoted being the average of many vessels:

Vessel	Cm^2
Aorta	2.5
Small arteries	20
Arterioles	40
Capillaries	2500
Venules	250
Small veins	80
Venae cavae	8

A further factor offering resistance to blood flow and hence compounding the effect of damming up of blood in the neck veins above the level of the ligature in hanging is the resistance caused by compression of the veins from the outside, which keeps many of them collapsed. For example, the veins from the arms are compressed by their sharp angulation over the first rib while the pressure in the neck veins often falls so low that the atmospheric pressure on the outside of the neck causes them to collapse. This collapse of the neck veins produced by the atmospheric pressure causes the pressure in these veins to remain essentially zero along their entire extent, all the way from the skull. Any tendency for the pressure to fall below this level collapses the veins still more, increasing their resistance and again returns the

pressure back to zero. This is reflected by the pressure at the beginning of the venous system, e.g., at the venules, which is about 10 mm Hg, decreasing to almost exactly 0 mm Hg at the right atrium. This large decrease in pressure in the veins indicates that the veins have far more resistance than would be expected for vessels of their large sizes. The practical implication of this resistance to blood flow following venous obstruction is that any mechanical obstruction of venous return to the heart, as by the ligature in hanging, results in venous engorgement with rupture of the thin-walled postcapillary venules and the production of petechial haemorrhages.

An important physiological factor of forensic significance is that the veins inside the skull are in a non-collapsible chamber and they will, therefore, not collapse. Consequently, negative pressure can exist in the dural sinuses of the head with the venous pressure in the sagittal sinus in the standing position being approximately -10 mm Hg because of the gravitational hydrostatic “suction” between the top of the skull and the base of the skull. Hence, if the sagittal sinus is opened during surgery, or the sinus sustains a penetrating gunshot or stab wound, air may immediately be sucked into this vein. The air may then travel downward as an embolus with obstruction of cardiac valve function and death.

Finally, as a clinical point, while pressure pulses enter the arteries intermittently with each heart beat, they are almost completely damped out before they pass through the capillaries into the systemic veins. However, pulsations are sometimes transmitted backward from the heart to cause pressure pulses in the large veins. This backward transmission of pulses does not occur to a significant extent in the normal circulation because, as indicated, most of the veins leading into the thoracic cavity are compressed by the surrounding tissues. This causes sufficient resistance to damp out the pulsations before they can be transmitted backward into

the peripheral veins. However, whenever the right atrial pressure is high, as in cardiac failure, the veins are well filled with blood and can then easily transmit the retrograde pulsations. Presumably, an attending doctor or paramedic who comes upon a hanging victim in cardiac failure and who is still alive in the very early stages of the hanging process should then be able to make the clinical observation of pressure pulses in the internal jugular vein below the level of constriction.

Autonomic Control of the Heart

Neural influences play a major role not only in cardiac adaptation to the functional demands of the body but also in the response to insults. For example, the hypothalamus, although comprising only 4 cm^3 of neural tissue, or 0.3 per cent of the total brain, can, through its actions on the vasomotor centre, produce every known type of neurogenic effect on the cardiovascular system.

Peripheral autonomic activity is integrated at higher levels in the brainstem and cerebrum. This includes various nuclei of the brainstem reticular formation, the thalamus, hypothalamus, limbic lobe and prefrontal neocortex, together with the ascending and descending pathways which interconnect these regions. It is recognized that central control of the cardiovascular system is exerted by a longitudinally arranged series of parallel pathways involving specific regions of the neuraxis extending from the cerebral cortex to the spinal cord.[5] The area that appears to be most intimately linked with the cardiovascular system is the orbital surface of the frontal lobe, followed by the anterior cingulate gyrus (area 24). The former is the main cortical representation of the parasympathetic nervous system and has a relay station in the anterior hypothalamus, with connections to the midbrain, pons, and

medulla. The autonomic nervous system, with both afferent and efferent components, thus acts on the nodal tissues and their prolongations, on coronary vessels, and on the working atrial and ventricular myocardium.

Rich plexuses of nerves containing cholinesterase, adrenergic transmitters, and other peptides such as neuropeptide Y (NY) are found in the subendocardial regions of all chambers and in the valve leaflets.[6] Complex endorgans have also been described in the subendocardial region of the left atrium.[7] These intrinsic ganglia thus act as sites for integration not only of extrinsic nervous inputs forming complex circuits for local neuronal control of the heart, but also for local reflexes which may develop after injury.[8]

These reflexes are mediated via a mechanism known as signal transduction which is the sum total of processes whereby an extracellular neural (or hormonal) stimulus is converted to an intracellular physiological change. In the case of the parasympathetic nervous system, signal transduction occurs via an extracellular first messenger (acetylcholine), a receptor system (the muscarinic receptor), and a sarcolemmal signalling system (the G-protein system). In the heart, the myocardial muscarinic receptor (M_2) is specifically associated with the activity of the vagal nerve endings so that muscarinic receptor stimulation produces a negative cardiac chronotropic (rate of conduction of the cardiac impulse) response.

The negative inotropic effects of vagal stimulation are multiple, and include:- 1) bradycardia; 2) inhibition of cyclic adenosine monophosphate (AMP) formation, and; 3) a direct negative inotropic (force of cardiac contraction) effect mediated by cyclic guanosine monophosphate (GMP).[9] This negative inotropic effect has best been observed in the presence of beta-adrenergic receptor stimulation, when vagal effects counteract those of prior beta-receptor stimulation.[10] A further mechanism

whereby vagal stimulation produces cardiac inhibitory effects lies in the sympathetic terminal neurons, where a presynaptic muscarinic M_2-receptor inhibits the release of norepinephrine. In this regard, it should be remembered that norepinephrine acts to:- 1) increase the force of cardiac contraction (increased inotropic effect); 2) increase the rate of cardiac contraction (increased chronotropic effect); 3) increase the velocity of conduction, and; 4) increase the systolic blood pressure. Thus, the net effect of vagal stimulation is to counteract the inotropic and chronotropic effects of beta-adrenergic stimulation with resultant "braking" of cardiac action. In essence, vagal stimulation results in bradycardia.

The Electrocardiographic and Electroencephalographic Abnormalities and Clinical Manifestations following Vagal Stimulation

Many experimental investigations have been carried out on the role played by both the sympathetic and parasympathetic nervous systems in the production of electrocardiographic abnormalities. More than 70 years ago, investigations of this nature indicated a distinct relationship between the nervous system and abnormal vagally-initiated cardiac electrical activity. [11] These primarily involved disturbances of rhythm and re-polarization.

Experiments conducted by Pool more than 50 years ago showed that stimuli applied to the circle of Willis were more likely to cause electrocardiographic abnormalities than when applied to any other of the cranial nerves, other than the glossopharyngeal and vagus.[12] This is understandable in view of the fact that the vessels of the circle of Willis derive their autonomic innervation from the glossopharyngeal and vagus nerves, as well as from a continuation of the cervical

sympathetic chain. Moreover, the circle of Willis is actually the blood supply of the hypothalamus, the "home" of the autonomic nervous system. A further early experiment, similarly conducted more than 50 years ago, involved embolism production by means of injection of paraffin oil into the carotid artery.[13] Shortly after injection of the paraffin, the experimental animal developed electrocardiographic changes consisting of bradycardia, prolonged Q-T interval, and deeply inverted T waves. Carotid sinus stimulation induced further electrocardiographic abnormalities, including sinoatrial slowing, atrial conduction defects, prolongation of the P-R interval, atrioventricular block, sinoatrial arrest, ventricular ectopic beats, and complete cardiac asystole.[14]

Electroencephalographic studies conducted more than 70 years ago have shown that during the initial 3 to 4 seconds of cardiac asystole, the electroencephalogram (EEG) is normal.[15-18] After transient flattening of electrical activity, during which the patient complains of dizziness and exhibits facial pallor (as in those cases of survival following attempted hanging), there is increased beta brain wave activity. With a longer period of asystole lasting 7 to 15 seconds, the EEG shows diffuse slowing of activity in the theta and delta ranges, followed by flattening of the recording. This is associated clinically with unconsciousness, dilated pupils, and unrecordable blood pressure. With resumption of cardiac activity, the physiologic EEG pattern returns to normal in 10-15 seconds.

The Role of Vessel Stretch, Compression and Production of Hydrostatic Pressure Waves in Carotid Vascular Injury

What are the biomechanics of the application of force to the human body and the response of organ systems to impact? [19, 20] The human body, from a mechanical viewpoint, is an extremely complex system which may be described as a heterogeneous viscoelastic mass within which run a number of channels filled with blood and other fluids. When such a system is subjected to impact forces, the immediately resulting disturbances are partly mechanical and partly thermal in nature. The most important changes are mechanical, and appear mainly as a deformation at the site of application of force with displacement of parts of the system relative to one another. This latter mechanism has been invoked to explain aortic rupture, [21] where the fixity of the ascending aorta, arch and descending aorta vary relative to one another. Hence, torsion and shearing stresses occur not only between these different segments but also between the aorta and other thoracic organs.

Deformation and its shearing stresses leads not only to displacement of parts of the system relative to one another, but also to the generation of a pressure pulse wave throughout the tissues. It has been experimentally demonstrated that the principal physiologic function of the vascular elastic tissues is to provide a hollow tube with considerable tensile strength. A prime example of this arrangement is the aorta and its major branches. Following impact with application of pressure, a pressure wave may be produced within the column of blood in the aorta. The magnitude of this effect increases with the degree of change of velocity of the blood within the artery.[22] Application of pressure to the contents of the aorta may thus,

result in a pressure wave being transmitted along the column of blood, and, in accordance with Pascal's law which states that the pressure is transmitted equally in all directions.[23] The carotid arteries, arising either directly from the aortic arch or its brachiocephalic branch, are immediately in continuity for transmission of both the systemic head of pressure and this pressure pulse wave. In the aorta, the pressure pulse wave has been considered as one of the main mechanisms for arterial rupture.

While modern observations indicate that carotid blood flow to the head decreases by 85% following pressure application to the carotid artery, reaching its lowest point in 6 seconds,[24] early investigations now assume greater importance in the light of modern well established laws of solid state physics. In 1893, Rindfleisch held the opinion that arterial rupture is produced by a sudden stretching of the vessel.[25] This conforms with present day concepts of the elasticity of arteries which has been described by von Gierke [20] and Nickerson.[26] These authors hold that while arterial tissues are endowed with considerable tensile strength, rapid dynamic stretching as occurs on sudden application of force, the resistance to the rate of stretching increases tremendously. As a consequence, the tissues become stiffer, more rigid, and fracture more readily. This provides one of the mechanisms of arterial rupture noted in the present study.

In 1918, Oppenheim ligated the branches of human aortas and filled the vessels with water at a pressure of up to 3000 mmHg, and noted that ruptures regularly occurred in the ascending part, the area from which the carotids arise.[27] While such pressures are unlikely ever to occur in the living, Oppenheim surmised that lower levels of increased intraluminal pressure may cause rupture *in vivo* because the resistance of the aorta and its major branches to stress imposed during life is lower than that after death. Klotz and Simpson conducted similar experiments

in 1932 on the aortas of young human subjects and concluded that, despite the vessels resisting an internal pressure of about 1000 mmHg without rupturing, a sudden rise in pressure with the generation of a pressure wave nevertheless occurred.[28] While these vessels did not rupture, it should be borne in mind that the experiments were carried out on non-living tissue.

More recent research has indicated that the first dramatic change that occurs in tissue on application of force, is the shock wave which is a special type of acoustic wave travelling through the medium at a velocity dependent upon the medium and the temperature.[29] For water at 20 degrees Celsius, this wave travels at approximately the speed of sound in water, i.e. 1483 m/second. In the case of gunshot wounds, which are extremely sudden phenomena (the muzzle velocity of a modern high-kinetic energy, high-velocity military assault rifle is of the order of 1200 m/second, i.e. 4320 kilometres/hr or roughly 2700 miles/hr) an "overpressure" as much as 60 atmospheres occurs. However, the speed of sound depends not only upon temperature but also upon pressure amplitude. The acoustic wave itself is a longitudinal compression wave where the particles of the medium oscillate around the rest position, the amplitude depending upon compressibility of the contained fluid. In the case of fluids, this is very low, meaning that the fluid oscillates at a low frequency. Hence, with the small amplitudes occurring with acoustic waves, oscillations are harmonic, i.e. positive and negative amplitudes are essentially equal. However, because of the increase in the speed of sound with increasing amplitude, the positive half-wave moves more quickly than the negative. The result is an increasing steepness at the front of the wave, and, if this increase develops so strongly that the pressure increases unsteadily from a minimum to a maximum, this would constitute a shock wave.

Thus, a shock wave is a form of acoustic wave produced by a sudden, aperiodic and intense stimulation. From the incremental increase in steepness, it follows that every shock wave turns into an acoustic wave after a certain amount of time, i.e. after covering a certain distance, this being only a question of the size of the amplitude.

Unlike a sinusoidal sound wave which has only one frequency, a shock wave is a non-recurring and very short event with an extremely steep front. This steep increase at the front of the shock wave is important because of the change from a state of rest to a pressure gradient in vascular structures. [30, 31] In vessels, a pressure wave is produced by the heart beat which is a sudden pressure application and which, according to haemodynamic laws, is propagated along the inside of the vessel and corresponding to a pulse wave as far as its physical behaviour is concerned. The velocity of the pulse wave in the aorta and its major branches in the young adult is about 4 m/second and that in the medium-sized arteries about 7 to 12 m/second. The pulse wave increases with age depending upon the elasticity of the vessels and their radius. While it appears that production of all these mechano-physiological changes are dependent on “sudden” application of force, most suicidal hangings in the present study indeed fall into this category, suspension having been effected from a height such as a wall, garage door, or tree branch.

Moar [32] has shown that a close relationship exists between lesions of the central nervous and cardiovascular systems, all mediated via the autonomic nervous system and its signal transduction messengers. Moreover, Moar has shown that following nervous system stimulation, a train of pathological events is set in motion, culminating in cardiomyocyte dropout, electrocardiographic abnormalities, and

myocardial fibrosis with long-term implications for myocardial contractile function.[33, 34]

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CHAPTER 6

MATERIALS AND METHODS

Fifty consecutive cases of suicidal hanging coming to medico-legal autopsy at the Forensic Pathology Service Mortuary, Johannesburg were examined on a prospective basis following ethical clearance given by the Ethics Committee of the Medical Faculty of the University of the Witwatersrand in accordance with Ethical Clearance Certificate M110462. The medico-legal autopsies themselves were performed in accordance with the instructions of the magistrate of the jurisdiction in which death had occurred, i.e. Johannesburg. They were therefore conducted under the legal decrees and statutes pertaining to the performance of medico-legal post-mortem examinations in South Africa.

To maintain subject confidentiality, all tissue samples removed at medico-legal autopsy in accordance with this protocol were processed and identified solely by means of the mortuary death register number, assigned on admission to the mortuary, as well as a sequential tissue sample number allocated to tissue specimens. The mortuary death register number is normally available only to the mortuary personnel, who are bound by the *sub judice* rule prohibiting discussion of the case or the disclosure of any details of the case to an extraneous person. The only persons having access to the records of the incident are:

i) The immediate relatives of the deceased for insurance payment purposes;

ii) The investigating officer on the case;

iii) The attending pathologist, and;

iv) The Inquest magistrate.

The histological tissue sample number is known only to the laboratory histopathology technologist involved in the processing of the tissue specimens and the pathologist carrying out that specific autopsy.

For the purpose of this study, the following items were documented in each case:

1) The age, gender, and ethnic group of the individual subject;
2) The weight and height of the subject;
3) The nature of the ligature encircling the neck;
4) The dimensions of the ligature impression in the skin and tissues of the neck, in terms of width and depth;
5) The position of the knot and vertical placement of the ligature, i.e. whether it overlay the upper, middle, or lower thirds of the neck, in an attempt to correlate these with the underlying injuries;

In addition to the above information about the 50 consecutive cases of suicidal hanging, there was information as to the circumstances of death which was provided by the police investigation report accompanying the body to the mortuary.
This information indicated that, in all but five cases, death had occurred in the preceding 12 hours. On discovery of the body, either the police or paramedical personnel were summoned, the body was cut down and then transported in the

recumbent position on its back in the mortuary vehicle to the mortuary. On admission to the mortuary, the body was subjected to immediate refrigeration until performance of an autopsy within 24 hours. Hence, no problems were encountered with regard to post-mortem decomposition producing tissue autolysis with effacement of the tissue architecture. Any pathological lesions noted on subsequent histopathological examination of the neck tissues could thus be attributed to ligature pressure and the forces occurring during the hanging process rather than to decompositional change occurring in the tissues. There were, however, five cases in which the body had been discovered several days after death with post-mortem decomposition having set in. While autopsies were conducted on these individuals, they were excluded from the present study.

In addition, five "non-hanging" cases to serve as controls were subjected to examination and bloodless neck dissection. These control cases constituted only those where no neck trauma had been sustained and involved focal trauma occurring away from the neck region. These comprised, for example, solitary gunshot wounds of an extremity, the thorax or the head, a penetrating incised wound of the thorax, and a case of poisoning. However, *vide infra.*

In those cases where the ligature had not been removed from the neck by either the family, the police or the paramedical personnel called to the scene with the ligature still being present in situ around the neck, great difficulty was encountered in inserting a pair of scissors underneath the ligature and away from the position of the knot in order to effect ligature removal due to the extreme tightness of ligature application.

Once the fifty hanging cases were dissected , yielding three hundred arteries, viz. the **left common carotid**, the **right common carotid**, the **left internal carotid**,

the **right internal carotid**, the **left external carotid** and the **right external carotid arteries**, i.e., **6** x **50** = **300**, 6 cm long specimens of the **carotid sheath** and its contents were taken from each side of the neck specifically at the area of the position of the ligature and the carotid bifurcation. This was done to obtain the anatomy of the **carotid arteries**, the **carotid body** and the **carotid sinus** in the tissue specimen as well as the **vagus nerve**.

To provide photographic documentation and comparison for demonstration, a number of examples of the common carotid, the internal carotid, and the external carotid arteries were photographed after formalin fixation and these comprised both "hanging" and "non-hanging" control cases. Furthermore, appropriate anatomical sketches as well as radiological demonstrations of the arterial distribution of the neck vessels were included as part of the anatomical foundation of the study.

In addition to the foregoing, an experiment was conducted to determine whether complete vascular occlusion occurs at the site of ligature application. To this end, ten cases of suicidal hanging not included in the study were subjected to probe exploration of the **carotid arteries** on each side of the neck. Prior to removal of the ligature, but after evisceration of the brain and intra-thoracic organs in order to create as 'bloodless' a field as possible, a blunt-ended probe was inserted from below upwards into each **common carotid artery** up to the point of ligature application. In all ten cases thus subjected to probe insertion, the blunt-ended point of the probe was arrested at the site of the ligature, no further upwards (i.e. distal and vertical to the heart) progression and passage being possible. It is suggested that probe impedance and arrest attested to the tightness of ligature application and the vascular occlusion attendant thereon. However, it must be emphasized that this technique and procedure was not carried out or applied to all the cases included in the present study for fear

that the probe itself might produce artefactual damage to the delicate **endothelium** lining the inner layer of the vessel wall. Any damage due to the probe may then have been falsely attributed to and misconstrued as being due to the ligature and the various forces ensuing therefrom.

Definitions and Systematic Dissection of the Neck

Definitions:

Cause of Death

Simply put, the cause of death is any injury or disease that produces an anatomical or physiological derangement in the body that results in the cessation of life of the individual. The mechanism of death, on the other hand, is the physiological derangement produced by the cause of death and which results in death. The manner of death (always and inevitably confused by lay persons with the cause of death) is how the cause of death came about and may be divided into five medicolegal categories: Natural, Accidental, Homicidal, Suicidal, and Undetermined.

Postmortem Examination

This simply refers to an examination conducted after death and may only comprise "viewing" of the body. Therein lies a problem, as South African law does not require a full or even partial autopsy to be performed, the law merely stating that a "postmortem examination" be performed. Needless to say, this has resulted in

numerous miscarriages of justice with inadequate or incomplete “autopsies” having been performed.

Necropsy

This is an old (circa 1881) somewhat outdated term referring to surgical investigation of a dead body, i.e. an autopsy.

Autopsy

This, on the other hand, encompasses a much more comprehensive definition. The term refers not only to seeing with one’s own eyes, i.e. personal observation, but, more specifically and to the point, dissection of a dead body, so as to ascertain by actual inspection, the seat or cause of disease. In other words, and in simple terms, this is a “proper” postmortem examination.

In a situation such as in hanging, where one is looking for a possibly obscure anatomical or pathological factor instrumental in causing the death of the victim, it is imperative that the dissection of the neck structures be delicate, painstaking and accurate. Coarse or brutal dissection, too often employed by those untutored in anatomy, may easily destroy or eliminate subtle clues vital to the discovery of the true underlying cause of death.

Dissection of the Neck Region (Fig. 6.1.)

The body is placed upon the autopsy table in the recumbent position. A wooden block measuring 20 x 15 x 7,5 cm is placed under the body at the mid-thoracic level. This enables the neck to undergo a degree of extension which not only facilitates dissection but, for the purposes of the present study, brings the carotid sheath and its enclosed structures into prominence. Unlike the situation in anatomical dissections where the body is embalmed and where a period of some 15 to 20 minutes is required to allow almost complete extension of the neck, this is not the case in forensic pathology autopsies where postmortem rigidity may have largely worn off and the neck extends easily. Furthermore, any untoward manipulation of the neck must be avoided in order not to create any artefactual injuries. After laying out the body and placing the block, examination of the ligature, its position around the neck, the accompanying knot (if present), and whether the knot is fixed or sliding is then undertaken. This is then followed by removal of the ligature by cutting through it away from the position of the knot. The ligature forms part of the body of evidence submitted to the police investigating officer. Following removal of the ligature, the underlying neck area and the ligature impression are carefully examined for the presence of abrasions or bruises surrounding the ligature mark. Record photographs are then taken to show the ligature impression and any accompanying marks. A "bloodless" dissection of the neck will then be performed.[1]

This latter is a procedure/method whereby the muscle layers of the neck are reflected layer by layer, from superficial to deep, in order to not only expose the underlying structures such as larynx and trachea, but, eventually, laying bare the carotid sheath. This carotid sheath is the fascial "tube" enclosing the common carotid

and internal carotid arteries, the internal jugular vein, the vagus nerve, and occasionally a branch of the ansa cervicalis of the cervical plexus. The purpose of this technique is that, while haemorrhage is indeed caused by the dissection procedure, the pathologist is immediately made aware of the artefactual nature of the haemorrhage, this having been caused by him or herself rather than having been produced by the encircling ligature. This spurious haemorrhage should not obscure possible haemorrhage due to the actual hanging.

The so-called “bloodless dissection” may be regarded as a special form of dissection which is performed slowly, carefully and meticulously. Its purpose is to seek for possible pathological details which may lead to the discovery of the cause of death in the hanging. Furthermore, it is standard practice in cases of hanging to first open the scalp and remove the calotte and brain to decompress the vasculature of the head and neck so as to provide as bloodless a field as possible.

The bloodless dissection is achieved by making a midline incision in the skin and underlying platysma muscle extending from the mandible (chin) to the suprasternal notch. Following this primary midline incision in the neck, the successive muscle layers of the neck beneath the investing layer of deep cervical fascia are exposed, taking care to avoid the external jugular vein and anterior jugular vein to prevent soiling with extraneous blood creating an artefactual haemorrhage. In practice, in order to perform a complete medico-legal autopsy, mandatory in all cases of non-natural or suspected non-natural death, this incision is continued downwards to the symphysis pubis.

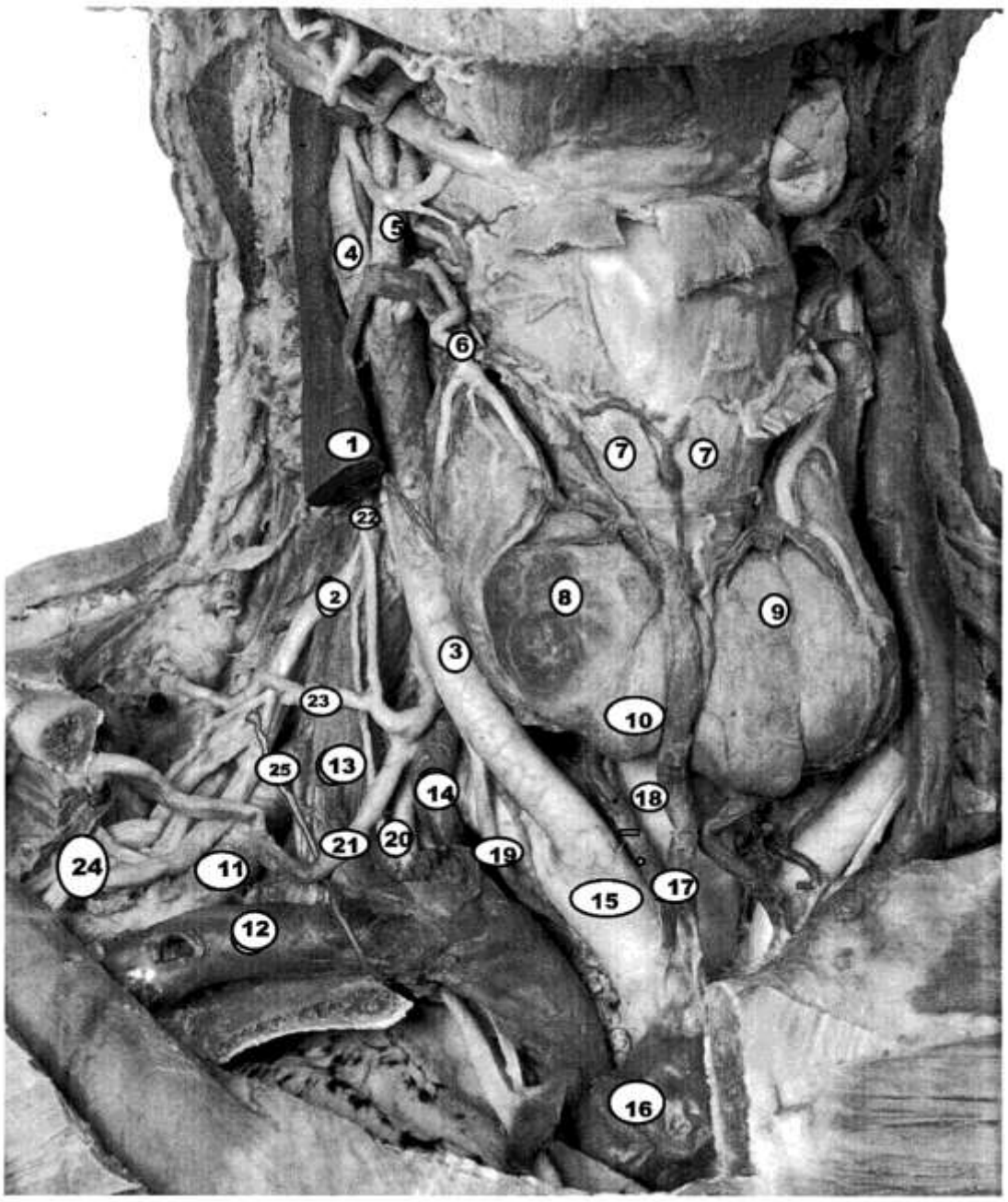
In sequential order, the layers of muscle from superficial to deep comprise the sternocleidomastoid in both its sternal and clavicular heads, the omo-hyoid muscle, and the sterno-hyoid muscle, the latter two lying beneath the level of the hyoid bone.

Division of the attachments of the sternocleidomastoid muscles then takes place, and these are retracted laterally. Between the sterno-hyoid muscles, which are bilateral, the thyroid cartilage and its investing pretracheal fascia lying in the midline are thus exposed. Deep to the sterno-hyoid muscles lie the thyro-hyoid muscles running from the oblique line of the thyroid cartilage to the hyoid bone and which is exposed on further dissection.

The hyoid bone itself divides the region of the front of the neck into a suprahyoid and an infrahyoid part. The suprahyoid part has the anterior bellies of the digastric muscles for its lateral sides, the hyoid bone for its base, and the mylo-hyoid muscle for its floor. The hyoid bone and mylo-hyoid muscle are examined to determine if superior displacement has occurred. In addition, the mouth is prised open to determine if the tongue has been displaced superiorly into the nasopharynx from the constricting pressure-effect of the ligature. Division of the suprahyoid and infrahyoid muscles then takes place to expose the body of the hyoid bone, the thyroid cartilage, cricoid cartilage and trachea. These latter structures lie within the infrahyoid region of the neck, which is bounded on each side by the exposed sterno-hyoid muscle above. Further neck dissection reveals the sterno-thyroid muscles below. Between thyroid cartilage above, and cricoid cartilage beneath it, the crico-thyroid membrane is exposed, being seen to run, with the crico-thyroid muscles extending postero-laterally from the cricoid cartilage, to the thyroid cartilage. Extending downwards from the cricoid cartilage lies the first tracheal ring beneath which and on either side of which is the carotid sheath with its contained common carotid artery, internal jugular vein and vagus nerve.

Delicate dissection of the carotid sheath then takes place. The hyoid bone and thyroid cartilages are separated by blunt dissection from the medial surface of the

sheath, preserving if possible as much of the surrounding connective tissue and fascia. In doing so, the branches of the external carotid artery are carefully exposed from below upwards. The lowest visible branch is usually the superior thyroid artery. This is followed superiorly by the lingual and facial arteries. If the small sternomastoid branches are present, they will have been divided by the lateral retraction of the sternocleidomastoid muscles. The dissection is then continued on the medial side of the carotid sheath superiorly until the fairly large maxillary artery is encountered. This is then divided.



With the technical assistance of Mr. J.Stevens

Fig. 6.1. A view of an anatomical dissection of the neck with the structures *in situ*.

1. **Internal jugular vein**
2. **5th cervical ventral spinal rami**
3. **Common carotid artery**
4. **Internal carotid artery**
5. **External carotid artery**
6. **Superior thyroid artery**
7. **Cricothyroid muscle**
8. **Right lobe of thyroid gland**

9. **Left lobe of thyroid gland**
10. **Isthmus of thyroid gland**
11. **Subclavian artery**
12. **Subclavian vein**
13. **Scalenus anterior muscle**
14. **Anterior vertebral vein**
15. **Brachiocephalic trunk**
16. **Brachiocephalic vein**
17. **Inferior thyroid veins**
18. **Tracheal cartilages**
19. **Vagus nerve (Cranial nerve X)**
20. **Phrenic nerve**
21. **Suprascapular artery**
22. **Ascending cervical artery**
23. **Superficial cervical artery**
24. **Posterior cords of the brachial plexus**
25. **Accessory phrenic nerve**

The possibility exists that the carotid sheath and its contents may be compressed between the hanging ligature and the underlying thyroid cartilage with compression of the vascular structures and vagus nerve in relation to the level of the ligature (whether upper, middle, or lower thirds of neck). Thus, a 6 cm segment of the sheath and its contents from its origin (right common carotid artery) or the beginning of its cervical portion (left common carotid artery) [2] up to 2 cm beyond the bifurcation into its respective internal and external branches were then excised and placed in 10% neutral buffered formalin (formaldehyde solution) for fixation.

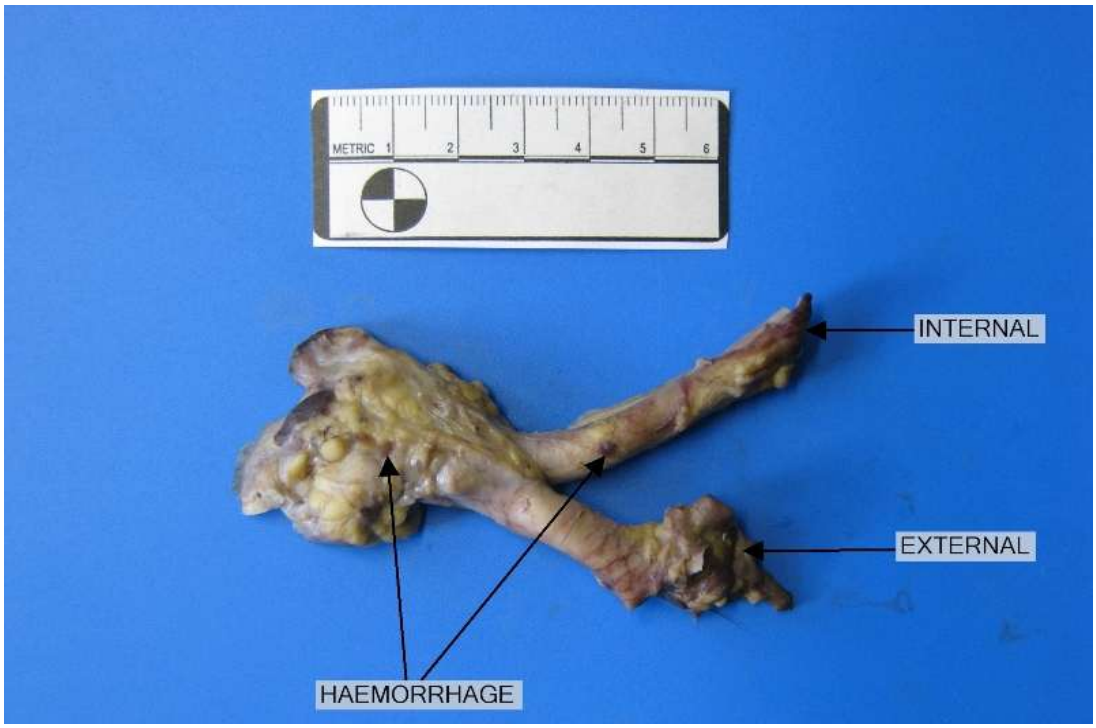


Fig. 6.2. A view of the removed, undissected and formalin fixed wide block of the carotid sheath and its contents, taken on one side of the neck at the bifurcation so as to include the **carotid body**, the **accessory glomi**, and the **vagus nerve**. The respective **internal and external carotid arteries** are seen (labels). Haemorrhage is noted (arrows). Magnification x 2.

No longitudinal sectioning of the vessel was performed, so as not to create any artefactual disruption of the vessel walls. Most importantly, longitudinal sectioning would have defeated the purpose of specimen collection as the anatomical and pathological aim was to obtain a complete cross-sectional view of the vessel wall and its surrounding structures. This technique/method of specimen removal was thoroughly explored and utilized in two pilot studies conducted by the present author involving trauma to the thoracic aorta and carotid arteries and subsequently published. [3, 4]

As far as the **carotid body** was concerned, this was identified on each side of the neck as a 5 – 7 mm in diameter reddish-brown ovoid structure situated near the bifurcation of the **common carotid artery** into its respective **internal carotid artery** and **external carotid artery** branches. Microscopically, similar but smaller bodies were histologically seen in the adventitia of the vessels near the **carotid sinus**, these latter being identified as **accessory glomi** of the **carotid body**.

In addition, tissue blocks of 5 additional cases of hanging were taken to include the **phrenic nerve** on each side. As indicated, the **phrenic nerve** lies deep to the sternocleidomastoid muscle but on the surface of the underlying scalenus anterior muscle, coursing diagonally medially and downwards over the latter's anterior aspect. Tissue blocks were taken on each side of the neck beneath the site of the overlying encircling and compressive ligature so as to include these two muscles and the intervening **phrenic nerve**. The same delicate dissection process as before was adopted in taking the **phrenic nerves** and their surroundings. To facilitate subsequent identification after formalin fixation, the upper, superolateral, border of each block was identified by means of a horizontal incision while the lower,

inferomedial, border of each block was identified by a V-shaped incision. Right and left sides were, of course, identified by being placed into the appropriately labelled specimen containers at the time of removal. Following formalin fixation, a transverse section was taken from each block so as to include the **sternocleidomastoid muscle**, the underlying **phrenic nerve** and the **scalenus anterior muscle**. However, in order to emphasize the widespread disruption of tissues which occur in hanging, Figs. 7.57, 7.58, 7.59, 7.60, 7.61 and 7.62 did not include the **phrenic nerve** in the photomicrographs.

To provide photographic evidence for demonstration purposes, a number of bilateral examples of the **common carotid**, the **internal carotid**, and the **external carotid arteries** were photographed (to scale) after formalin fixation. On completion of fixation for a period of three weeks in 10% neutral buffered formalin (formaldehyde solution), a cross-sectional segment of each artery was excised together with a segment of surrounding arterial tissue so as to include the vagus nerve (in the case of each common carotid artery) and sympathetic nerves.



Fig. 6a. A view of the longitudinal and as yet undissected segment of the **left common carotid artery** and its surrounding soft tissues together with a subsequent circular segment of the artery from ‘non-hanging’ control case no. 3. A small area of haemorrhage (arrow) of the soft tissues surrounding the longitudinal segment of the tissue is recognized as artefactual by the pathologist. This artefact has been produced during the course of the autopsy procedure.



Fig. 6b. A view of dissected longitudinal and circular segments of the **left internal carotid artery** from 'non-hanging' control case no. 3. The haemorrhage noted is artefactual.



Fig. 6c. A view of longitudinal and circular sections of the **left external carotid artery** from control case no. 3. The longitudinal segment of the artery is clearly seen, with artefactual haemorrhage and an adjacent, but still attached segment of soft tissue.



Fig. 6d. A view of longitudinal and circular sections of the **right common carotid artery** from control case no. 3. Minimal areas of purplish haemorrhage are noted (arrows), but recognized as artefactual by the prosector.



Fig. 6e. A view of small longitudinal and circular sections of the **right internal carotid artery** from 'non-hanging' control case no. 3. As before, slight areas of purplish-blue contusional discolouration (arrows) are noted.

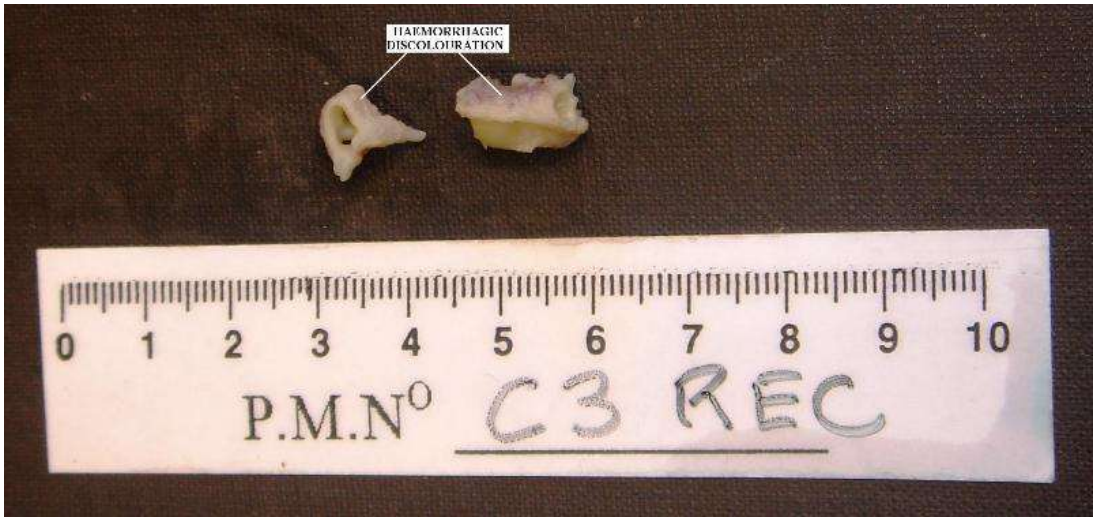


Fig. 6f. A view of small longitudinal and circular segments of the **right external carotid artery** from control case no. 3. Evident as before are the slight areas of purplish-blue contusional discolouration (arrows).

Because of the necessity of the author having to examine the microscopic sections derived from both the normal arteries, veins and nerves as well as from those of the cases of hanging, the author decided to undergo a course of training in the techniques of preparation and processing of microscopic sections. The main objects of the author undertaking this course was not only to familiarize himself with the methods and techniques used but also to be aware of the pitfalls which may occur in the processing of biological tissues. To misinterpret a processing fault for a pathological process would clearly, therefore, result in a misdiagnosis and the reaching of an erroneous conclusion.

Processing of the Tissue

A. Put-through

Cross-sections of the left common carotid artery, the left internal carotid artery, the left external carotid artery, the right common carotid artery, the right internal carotid artery and the right external carotid artery were taken after formalin fixation in 10% neutral buffered formalin. Cross-sectioned tissue sections, for histological processing, were taken using a scalpel, away from the cut edge of the excised segment of artery. This was to preclude the possibility of any artefactual trauma caused during sampling of the arteries at autopsy as well as to preserve the anatomical relationship of the various layers of the vessel wall to one another and to the internal jugular vein, the vagus nerve and the vagus (nodose) ganglion.

B. Tissue Processor

The cassetes containing the tissue sections were placed in the tissue processor and then passed through various solutions containing increasing concentrations of alcohol, the purpose of which was to dehydrate the tissues. Following tissue dehydration, the cassetes were cleared through agitation by being passed through chloroform beakers following which this clearing agent was replaced, also by agitation, in 2 x paraffin wax beakers at a temperature of 56 degrees to 58 degrees centigrade. Finally, in order to infiltrate all areas of the tissue section with wax, the wire basket containing the cassetes was placed in a vacuum infiltrator to remove all residual air bubbles.

C. The Embedding Process

The cassettes were then placed in a heated container containing a small amount of melted wax at 60 degrees centigrade. Following solidification on a cold plate at a temperature of +/- 4 degrees centigrade, the wax block containing the embedded tissue was removed from the wax mould, the excess wax surrounding the block being removed by scraping this wax off with a blunt knife.

D. Cutting by means of a Microtome

The wax block was placed in the block holder of a Microtome Leica RM 2125 RT and the excess wax trimmed away by means of the microtome knife in order to expose the surface area of the specific section. After fine trimming was performed sections were cut at +/- 3μ (microns) thickness. These were floated out on to water at 40 degrees centigrade. The effect of the floating was to flatten the sections and remove creases. The flattened sections were then lifted from the water bath on to microscope slides. The excess water was blotted away and the mounted sections were allowed to dry out in the air at room temperature (+/- 25 degrees centigrade).

E. Immersion and Staining

A Shandon Linistain automated machine was set for the standard Haematoxylin and Eosin method globally employed. The air-dried slides were placed by means of a plastic slide holder (clip) on a chain link of this machine, the slides then being passed

sequentially through a series of baths containing progressively increasing concentrations of alcohol to dehydrate the slide. This was then followed by immersion in a 1 x xylene bath in order to clear the alcohol so that the slide could then be mounted in DPX mounting medium.

F. Mounting Process

A few drops of DPX mounting fluid was syringed onto a cover slip measuring 2 cm x 2 cm. The slide of the tissue section was then inverted on top of the cover slip and its mounting fluid taking care to ensure that all air bubbles were removed from the mounted section. The slides so processed and produced were then ready for examination by conventional light microscopy.

Because it was necessary to examine the entire circumference of the vessel wall, transverse sections of each of the three arteries on each side were taken. The processing (dehydration, wax embedding, cutting, rehydration, staining and dehydration) resulted in multiple transverse folding of the **media**. This was in part due to the processing and in part due to the nature of the **media**, this being an elastic and muscular layer. The intima and adventitia were found not to be involved in folding and crease formation.

G. Slide Examination

The slides were examined by means of an **OLYMPUS BX41** clinical microscope.

To provide standardisation and to eliminate observer error, all sections were examined in conjunction with the supervisor at the University of the Witwatersrand,

Johannesburg. The five " non-hanging" control cases were reviewed after examining the 50 cases in order to compare any deviation from the findings observed in the hanging cases. It then became evident that these so-called controls could not be regarded as "normals" in view of the fact that they all constituted forensic cases comprising one or other kind of traumatic non-natural death. Indeed, in control case no. 2, comprising a gunshot wound of an upper extremity with perforation of the axillary artery at the lateral border of the 1st rib, the left external carotid artery showed evidence of multiple haemorrhagic splits of the medial layer of the vessel wall, explicable on the basis of vascular acoustic shock wave effects being propagated following missile impact.[5-10] Furthermore, in view of the great importance of the **carotid body** (glomus) as a component of the neurovascular mechanism of death causation, a glomus was removed from a non-traumatic control case in order to compare the normal histology with the histological findings observed in the non-natural hanging cases.[11-13]

All slides of the hanging cases were reviewed after examining each batch of some three to five cases so as to compare the findings observed with those noted on initial examination and whether any findings had been missed. Furthermore, where a slide/slides was deemed of insufficient standard to allow accurate and acceptable examination, recourse was had to resectioning of the tissue block with remounting, processing and staining.

To eliminate and prevent observer bias and provide standardisation, all samples of the respective three hundred arteries (see pages 132-133) of the fifty 'hanging cases', the 50 respective arteries of the five 'control cases' and the additional 5 cases taken to study the **phrenic nerve** were examined in conjunction with the supervisor rather than by the author of the present study alone.

Consultation with the two experienced laboratory technologists involved in the processing of some of the tissue specimens, however, indicated that the most 'perfect' of microtome blades carry areas of minute imperfection and combined with increasing bluntness of the blade as tissue cutting progresses, disruptions and teasing of fibres are often a result of non-pathological factors.

Sections were viewed at varying magnifications by conventional light microscopy and photomicrographs were taken of relevant sections for demonstration of tearing or disruption of the arterial wall. It should be noted that infiltration of blood into the wall of a vessel through an intimal tear is unlikely to occur post-mortem where there is no blood pressure and where dissection and processing are carefully performed. No difficulties were encountered in visualizing and defining either internal or external elastic laminae using the standard Haematoxylin and Eosin stain as both these laminae are clearly defined in medium sized muscular arteries such as the carotid arteries. This stain thus proved to be more than adequate to delineate the lesions encountered.

In view of the fact that the walls of the arterial vessels comprise three layers, namely, **adventitia**, **media**, and **intima**, all findings noted were classified into those categories. The classification system employed was thus based on the actual layer and elastic lamina of arterial wall involved extending in a logical and step-wise manner on depth of involvement from superficial to deep, i.e., from **intima** through to **adventitia**. These were classified and defined as follows:

Classification and Definitions

In the following classification, a '**tear**' is defined as a violent or forcible rending or tearing apart of the tissues.

1) superficial intimal tears, these being defined as those **tears** involving the **intima** or innermost layer of the vessel wall only;

2) intimal tears extending to or along the internal elastic lamina, defined as those **tears of the intima** which extend to or along the defining membrane separating the **intima** from the **media**;

3) intimo-medial tears, these being those **tears** which extend through the full thickness of the **intima** to involve the deeper **media**, i.e. the **muscular** (and middle) **layer of the vessel wall**.These **tears** were further subdivided according to depth/extent of involvement of the **media** into:

i) intima to the inner-third of the arterial media;

ii) intima to the middle-third of the arterial media;

iii) intima to the outer-third of the arterial media;

4) tears of the media (muscular layer) of the vessel wall, this term being self-explanatory, indicating those **tears** involving only this layer of the vessel wall;

5) tears of the adjacent adventitio-medial layer of the vessel wall. This refers to those **tears** which extend fro or through the outermost layer of the vessel wall, i.e. the **adventitia** to involve the underlying **media (muscular layer) of the vessel wall**;

6) tears of the arterial adventitia. This refers to those **tears** involving the outermost layer of the vessel wall;

7) adventitial haemorrhage. This refers to **haemorrhage** (as a consequence of vessel rupture) within the **adventitial layer**;

8) complete circumferential transverse rupture. This refers to those **tears** extending throughout the full thickness of the vessel wall involving all layers of the wall, i.e. a through-and-through breach.

Any adventitial haemorrhage noted was deemed to be non-artefactual and hence genuine if it was away and removed from the plane of section. In this regard, it should be borne in mind that the tissue block is fixed and embedded in solid wax. In addition, the microtome blade is fixed thus preventing tissue movement with consequent displacement of red blood cells, tissue disruption and factitious haemorrhage.

With regard to the **neural findings**, i.e. those involving the **carotid body**, its **accessory glomi**, the **ganglia**, and the **autonomic nerves**, these were classified and analysed according to the pattern of findings which emerged on microscopic examination. On this basis, **dehiscence**, i.e. 'splits' within a nerve, with residual strands of tissue bridging, were regarded as 'true splits' of a pathological nature and most likely due to the presence of the ligature, its compressive forces and the tensile and stretch forces of the weight of the body and the pull of gravity.

Following analysis of these arterial disruptions, an attempt was made to determine whether the lesions noted could be correlated to:

i) the degree of trauma;

ii) the nature of trauma;

iii) level of ligature;

iv) nature and structure of ligature, and;

v) position of knot (if present).

It was further noted if a vessel had sustained a single tear only or multiple tears and, if the latter, the extent of each tear. In addition, the number of vessels involved was noted, the anatomical distribution of vessel involvement, and whether, in those cases exhibiting multi-vessel injury, such injury was unilateral or bilateral.

Statistical Analysis

Data pertaining to the study such as the epidemiological parameters of age, gender and ethnic group would be analysed as well as the weight of the individual, the type of ligature and the type and position of the knot to determine whether a correlation could be made between any of these factors and the arterial and neural lesions observed. Percentages mainly would be applied, but should the differences found be sufficiently small to lend themselves to analysis by statistical methods, the Mann-Whitney U-test and t-test would be employed. For comparative purposes, as well as ease of reading and reference, the arterial and neural lesions in particular would be subjected to percentage analysis to determine whether any of the lesions found, single or multiple, unilateral or bilateral, their particular vessel or neural involvement or the type, extent or depth of vascular lesion observed carried numerical and percentage preponderance and hence of pathological significance.

Background Review of the Literature and Foundation of the Study

These were extensively researched and the rationale for, and underlying foundations of, this study were presented under the appropriate headings in Chapters 1 to 5 with numerous specific and appropriate references provided. In addition, appropriate sketches pertaining to the anatomy as well as radiological demonstrations of the arterial distribution of the head and neck were included as part of the anatomical foundation of the study.

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ADDENDUM

HAEMATOXYLIN AND EOSIN TECHNIQUE (MANUAL)

PRINCIPLE:

HAEMATOXYLIN IS A BASIC DYE WHICH BINDS WITH ACIDIC COMPONENTS (i.e. NUCLEI).
EOSIN/PHLOXINE IS AN ACIDIC DYE BINDING WITH BASIC COMPONENTS (i.e. CYTOPLASM).

FIXATION:

PHOSPHATE BUFFERED FORMALIN.

SECTION:

THIN (THREE MICRONS) PARAFFIN SECTIONS.

CONTROL:

NONE NECESSARY.

SOLUTIONS:

1. HARRIS'S HAEMATOXYLIN WORKING SOLUTION.
2. EOSIN/PHLOXINE WORKING SOLUTION.

METHOD:

1. SECTIONS TO RUNNING TAP WATER.
2. STAIN IN HARRIS'S HAEMATOXYLIN 5 Mins.
3. WASH IN R.T.W.
4. DIFFERENTIATE IN 1% Aq. HYDROCHLORIC ACID.
5. WASH IN R.T.W.
6. STAIN IN EOSIN/PHLOXINE SOLUTION 2 Mins.
7. RINSE IN R.T.W.
8. DEHYDRATE, CLEAR AND MOUNT.

RESULTS:

NUCLEI	– BLUE.
CYTOPLASM	– SHADES OF PINK.
RED BLOOD CELLS	– RED.
ELASTIN	– REFRACTILE RED.

NOTES:

RUN A TEST SLIDE AND CHECK MICROSCOPICALLY BEFORE STAINING BATCHES.
FILTER HAEMATOXYLIN ONCE A WEEK.
CHANGE SOLUTIONS WHEN DIRTY OR WHEN STAINING REACTION WEAKENS.

REFERENCES:

THE THEORY AND PRACTICE OF HISTOLOGICAL TECHNIQUES: BANCROFT AND STEVENS, 2nd EDITION.

CHAPTER 7

RESULTS

Introduction

It should be borne in mind that in a busy medicolegal jurisdiction such as Johannesburg with its high level of violence (as in South Africa as a whole where some 50 murders occur countrywide on a daily basis) almost 80 percent of cases are traumatic in origin and the term "normal" is thus used with circumspection. To obviate this difficulty, the five "non-hanging" cases serving as controls comprised only those subjects where no neck trauma had been sustained and involving only focal trauma away from the neck region. In view of the fact that subjects of South African Indigenous origin comprise the majority of cases coming to medico-legal autopsy in Johannesburg and comprise some 85% of cases, all five subjects were males of this ethnic origin ranging in age from 25 to 37 years.

The neurovascular structures examined comprised the common carotid arteries, the internal carotid arteries and the external carotid arteries (left and right sides), the vagus and phrenic nerves (left and right), the sympathetic nerves, the carotid bodies (left and right) and the accessory miniglomi contained within the surrounding adventitial tissues. In each case 6 cm segments of the carotid sheath, its contained arteries and vagus nerve and the surrounding adventitial tissues so as to include the carotid bodies and accessory glomi, were removed bilaterally. The **carotid body** was identified on each side of the neck as a 5 -7 mm in diameter reddish-brown ovoid structure situated near the bifurcation of the **common carotid**

artery into its **internal carotid artery** and **external carotid artery** branches. Microscopically similar but miniature smaller bodies were histologically seen in the adventitia of the vessels near the carotid sinus, these latter being identified as **accessory glomi** of the **carotid body**.

After fixation, cross-sectional segments of these structures, all at the area of bifurcation, were then taken for histological processing.

A. <u>Histological Examination of the "Control" Cases</u>

Based on their size and structural features, arteries may be divided into three types:

1) large or **elastic** arteries which comprise the aorta and its major branches such as the common carotid and common iliac arteries;

2) medium-sized or **muscular** arteries comprising other branches of the aorta such as the coronary and renal arteries, and;

3) small arteries (less than approximately 2 mm in diameter) and **arterioles** (20 to 100 nm in diameter), within the substance of tissues and organs.

An artery itself, as seen under the light microscope, consists of three concentric anatomical layers- **intima**, **media**, and **adventitia**. Beginning with the innermost layer, i.e. the **intima**, forms a lining within the lumen of the vessel. In the control specimens examined in the present study, the **intima** consisted of a single layer of endothelial cells, upon a basement membrane, with a minimal amount of underlying subendothelial connective tissue. However, the thickness of the **intima** varied from case to case depending on whether an atheromatous plaque was found to be present. This was not an entirely unexpected finding in view of the almost ubiquitous presence of atherosclerosis in present-day populations subsisting on the

so-called ‘Western diet’ with its high fat and cholesterol content. For example, in two of the control cases in particular (numbers: three and four,) in spite of the relatively young age of the subjects, widespread intimal thickening due to concentric atheromatous plaque deposition was noted. A factor that may have contributed to atheromatous deposition in these subjects, despite their relatively young age, is the genetic predilection for essential hypertension in the Indigenous South African population.

In these cases, despite the atheromatous process involving the carotid arteries on both the left and right sides of the neck, i.e. bilateral vessel involvement, the atheromatous plaque was noted to be thicker and of a greater degree of severity on the right as compared to the left. The right common carotid artery takes a slight degree of angulation and inclination (+/- 5 degrees) as it arises from the brachiocephalic artery in contrast to the left common carotid artery which takes a more vertical course at its origin from the arch of the aorta. Because, sites of arterial branching and angulation are known to predispose to atheromatous plaque deposition as well as to shearing stresses at these sites. These factors constitute focal points for the development of turbulence and eddy currents. While it is tempting, therefore, to attribute the greater degree of atheromatous plaque formation on the right side as opposed to the left, the +/- 5 degree of shift is very small and is, moreover, likely to vary anatomically in person to person.

Moving further outwards through the **intima** towards the **media**, in all cases a well-defined pink-staining dense elastic membrane, the **internal elastic lamina** was noted. This lamina has a wavy and serpentine configuration as it is followed circumferentially around the vessel wall. In no cases was reduplication of the lamina

noted, this latter being a well-recognized pathological and morphological change in long-standing hypertension.

Proceeding outwards to the **media** where circumferentially situated smooth muscle cells are found, occasional clefts (fenestra) were encountered between the smooth muscle cells. These contained no red blood cells and were predominantly confined to areas of 'wrinkling' distributed at circumferential intervals around the vessel. While the fenestra were, in fact, a normal feature of the **media,** the 'wrinkling' was attributed to unavoidable processing artifacts occurring in a tubular structure (the reasons for this have been described in the previous chapter dealing with Materials and Methods). Also observed were small arterioles coursing into the outer one- half to two- thirds of the **media**. These arterioles comprised the well-known **vasa vasorum** whose function is nourishment of the **media**. In the outer limit of the **media** was the **external elastic lamina** demarcating and separating the **media** from the overlying **adventitia**. Interestingly, in all the 'control' specimens, this lamina was less well defined than the **internal elastic lamina** and appeared in some areas to be discontinuous.

In three cases out of the 5 control cases examined, firm apposition was noted between **media** and overlying **adventitia**. However, in the remaining 2 cases, focal teasing and disruption of the connective tissue fibres of the **adventitia** was noted, with separation of these latter fibres from the **media** forming clefts between **media** and **adventitia**. These focal areas of separation and cleft formation were discontinuous as the vessel wall was followed circumferentially and contained no red blood cells or tissue bridges, and were therefore regarded as a dissection or processing artefact.

Also observed within the connective tissue fibres of the **media** were the small terminal fibrils of the sympathetic nerves, occuring either singly or in clusters, and often seen in close apposition to arterioles. Noted within these vessels were laked erythrocytes, the latter consistent with post mortem autolysis in autopsied bodies as opposed to tissue biopsies from living subjects. Also seen at the area of bifurcation of the common carotid artery was the **vagus nerve**, this being the major local representative of the parasympathetic nervous system. This nerve is of crucial importance in the context of the present study (see Chapters 3 and 5). With a (tight) ligature pressure to the neck and producing stimulation of the vagus nerve, it is possible that reflex "cardiac standstill" could occur as a result of the parasympathetic responses of the heart.

The Carotid Body

Lying near the bifurcation of the common carotid artery and on right and left sides is a 5 to 7 mm high and 2.5 to 4 mm wide reddish-brown oval structure known as the **carotid body** and which functions as a chemoreceptor. [1-3] First described in 1743 by von Haller, [4] the **carotid glomus** develops from mesenchyme in the third pharyngeal arch. [5] As seen under conventional light microscopy, it lies either posterior to the carotid bifurcation or between its branches being attached to, or sometimes partly embedded in, the surrounding adventitia. Occasionally, as seen in this study, the "body" adopts the form of a group of separate nodules. Aberrant miniature carotid bodies, microstructurally similar but with diameters of 600 μ or less, may appear in the adventitia and adipose tissue near to the carotid sinus. [6]

The **carotid body** is surrounded by a fibrous capsule from which septa divide the enclosed tissue into lobules. Each lobule contains glomus (Type I) cells which are separated from an extensive network of fenestrated sinusoids by sustentacular (Type II) cells. [7-9] Unmyelinated axons lie in a collagenous matrix between the sustentacular cells and the sinusoidal endothelium, many synapsing on the glomus cells. They comprise visceral afferents which travel in the carotid sinus nerve to join the glossopharyngeal nerve (cranial nerve IX). Of relevance and pathophysiological significance to the study, however, is that glomus cells store a number of peptides and amines, in particular adrenaline and noradrenaline, and are therefore regarded as paraneurones. [10-12] In addition, preganglionic sympathetic axons and fibres from the carotid sinus synapse on parasympathetic and sympathetic ganglion cells. These cells can be seen to lie either in isolation or in small groups near the surface of each **carotid body**. A view of the **carotid sheath** and its contents prior to dissection is depicted in Fig. **6.2** in the previous 'Materials and Methods' chapter.

In contrast to the fifty "hanging cases", the control group of five "non-hanging", so-called "normal" cases, showed no evidence of the systematic damage to the vascular, neural, accessory glomal and carotid body structures noted in the hanging cases. The following few illustrations exemplify briefly the histological appearance and structure of "normal" **carotid arteries** as obtained from autopsies. These are provided for comparative purposes with the "pathological" arteries of the "hanging" cases. What was observed, however, was a mild degree of vascular congestion in the tissues of the neck (Fig.7.1).

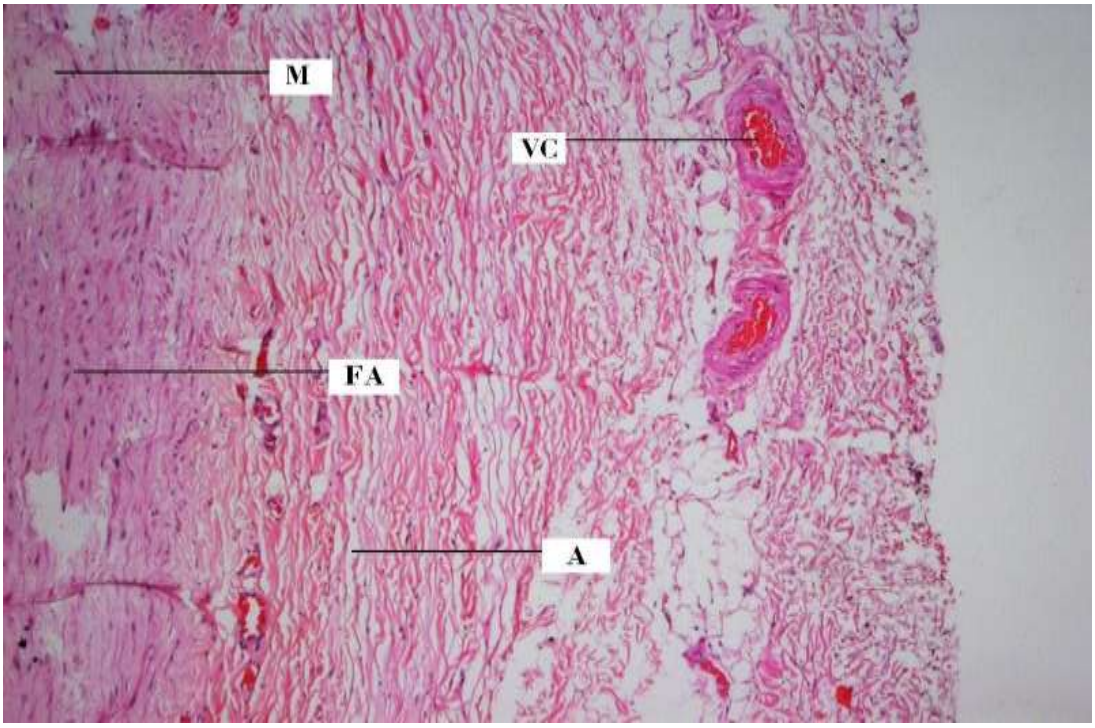


Fig. 7.1. Left common carotid artery from a 'non-hanging' control case showing the **adventitia** of the vessel wall and part of the underlying **media** or muscular layer. **Adventitio-medial separation** is absent, firm apposition of the **adventitia** to the underlying **media** being noted. Marked periadventitial **vascular congestion** is present, in keeping with decreased venous return as an element of terminal cardiac failure. The purpose of the figure is to illustrate the close attachment of **media** to **adventitia**. Control case no. 3 (Haematoxylin and Eosin x 100). **A** = adventitia. **M** = media or muscular layer. **VC** = vascular congestion. **FA** = firm apposition between adventitia and media.

An interesting exception, however, was that in one of the 30 control arteries examined (six arteries in each control case x five cases; 5 x 6 = 30) namely, control case number two, multiple **fenstrations of the media** of the arterial wall of the **right internal carotid artery** were noted (Fig.7.2).

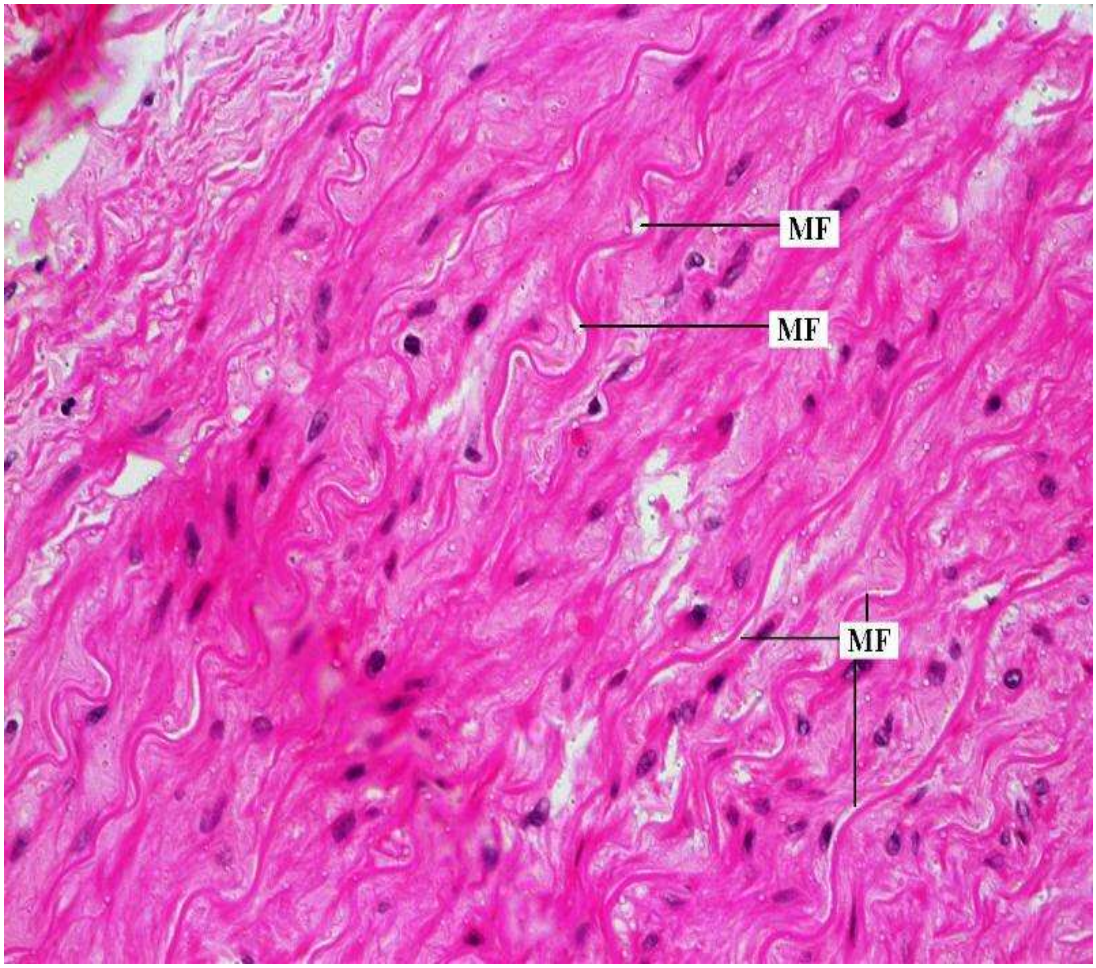


Fig. 7.2. Right internal carotid artery. A mild-to-moderate degree of **fenestration** along the laminar planes of the **media** is noted. Control case no. 2. Gunshot wound of the right axillary artery (Haematoxylin and Eosin x 400). **MF** = fenestration of the media.

This case was that of a young male with a perforating gunshot wound of the right upper limb involving the right axillary artery at the lateral border of the first rib.

Summary of microscopic findings of the arteries, nerves and carotid glomi of the 'non-hanging' cases

Apart from the finding of evidence of atherosclerosis (the latter feature also noted in several of the pathological arteries of the "hanging" cases) and a minimal degree of congestion, no other pathological features in any of the examined structures were noted.

B. Results of the Physical Observations and Microscopic Findings of Cases of Suicidal Hanging

The results of the physical observations on the individual victims and of the microscopic findings in the various structures of the neck have been set out as follows:

I. **Demographics and Epidemiology.**
II. **Macroscopic findings in relation to the ligature and position of the knot.**
III. **Vascular findings.**
IV. **Additional vascular findings.**
V. **Neural findings.**

I. Demographics and Epidemiology

The data regarding the 50 subjects in the present study are presented in Tables I, Ia, Ib and Ic which follow.

Table I : Demographics and Epidemiology (N = 50 Hanging Cases)									
	M = Male			F = Female		N/A = Not Applicable			
I SA= Indigenous South Africans				SAED = South Africans of European Descent					
SAID = South Africans of Indian Descent				SAMD = South Africans of Mixed Descent					
Case No	Age (Y)	Sex		Ethnic Origin				Height (M)	Weight (Kg)
		M	F	ISA	SAED	SAID	SAMD		
1	26		X		X			1.64	66
2	22	X		X				1.76	56
3	24	X		X				1.56	62
4	25	X		X				1.65	74
5	48	X		X				1.80	80
6	25	X		X				1.69	64
7	48	X		X				1.75	62
8	25	X		X				1.47	58
9	55	X		X				1.73	79
10	43	X		X				1.62	66
11	29	X		X				1.80	105
12	20		X	X				1.75	72
13	14	X		X				1.74	79
14	35	X		X				1.69	72
15	48	X		X				1.78	61
16	31	X		X				1.72	57
17	20	X				X		1.79	70
18	17	X		X				1.70	60
19	37	X		X				1.67	68
20	32	X		X				1.65	38
21	32	X		X				1.69	62
22	40	X		X				1.58	68
23	30	X		X				1.79	55
24	42	X				X		1.68	73
25	36	X			X			1.84	72
26	25	X		X				1.60	52
27	26	X		X				1.77	72
28	30	X		X				1.76	75
29	26	X		X				1.65	68
30	20		X	X				1.68	59
31	38	X		X				1.68	68
32	30	X		X				1.70	69
33	28		X	X				1.65	54
34	61	X			X			1.75	92
35	24	X		X				1.68	68
36	30	X		X				1.70	71
37	34	X		X				1.72	72
38	54	X			X			1.78	78
39	83	X		X				1.68	64
40	35		X	X				1.66	75
41	40	X		X				0.73	76
42	33	X			X			1.65	68
43	68	X			X			1.65	65
44	39		X		X			1.75	70
45	13	X		X				1.38	41
46	41	X					X	1.76	73
47	75	X			X			1.70	72
48	27	X			X			1.78	79
49	25	X		X				1.68	68
50	23	X		X				1.73	66
TOTAL		44	6	38	9	2	1	1.68	68
%		88	12	76	18	4	2	N/A	N/A

¶ 44 (88%) were male and 6 (12%) were female (Table Ia);

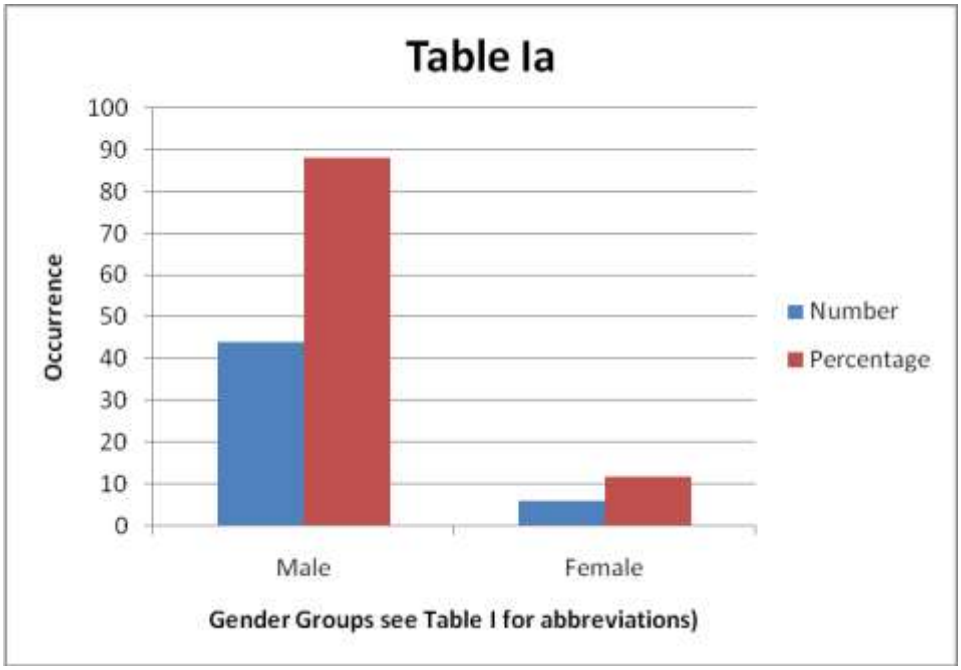


Table Ia : Gender Groups: (N = 50)

	Male	Female
TOTAL	44	6
%	88.0	12.0

¶ 38 (76%) were Indigenous South Africans (ISA);

¶ 9 (18%) were South Africans of European descent (SAED);

¶ 2 (4%) were South Africans of Indian descent (SAID);

¶ 1 (2%) was a South African of Mixed descent (SAMD) (Table Ib).

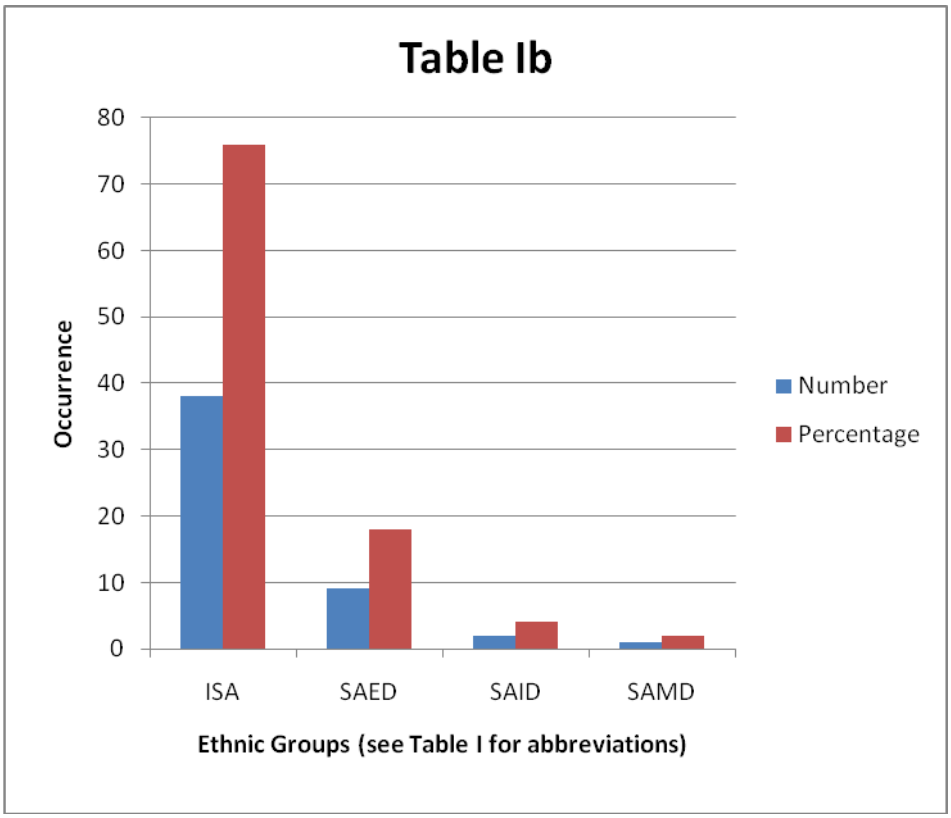


Table Ib: Ethnic Groups: (N = 50)

	ISA	SAED	SAID	SAMD
TOTAL	38	9	2	1
%	76.0	18.0	4.0	2.0

The subjects ranged in age from 13 to 83 years with the greatest number of subjects, 18 (36%) in the 21-30 year age group. In descending order of frequency, the remaining age distribution was as follows:

¶ 31- 40 years, 13 subjects (26%);

¶ 11- 20 and 41-50 years, 6 subjects each (12%);

¶ 51- 60 and 61-70 years, 2 subjects each (4%);

¶ 71- 80 and 81-90 years, 1 subject each (2%) (Table Ic).

11 - 20 = 12%

31 - 40 = 26%

51 - 60 = 4%

61 - 70 = 4%

71 - 80 = 2%

81 - 90 = 2%

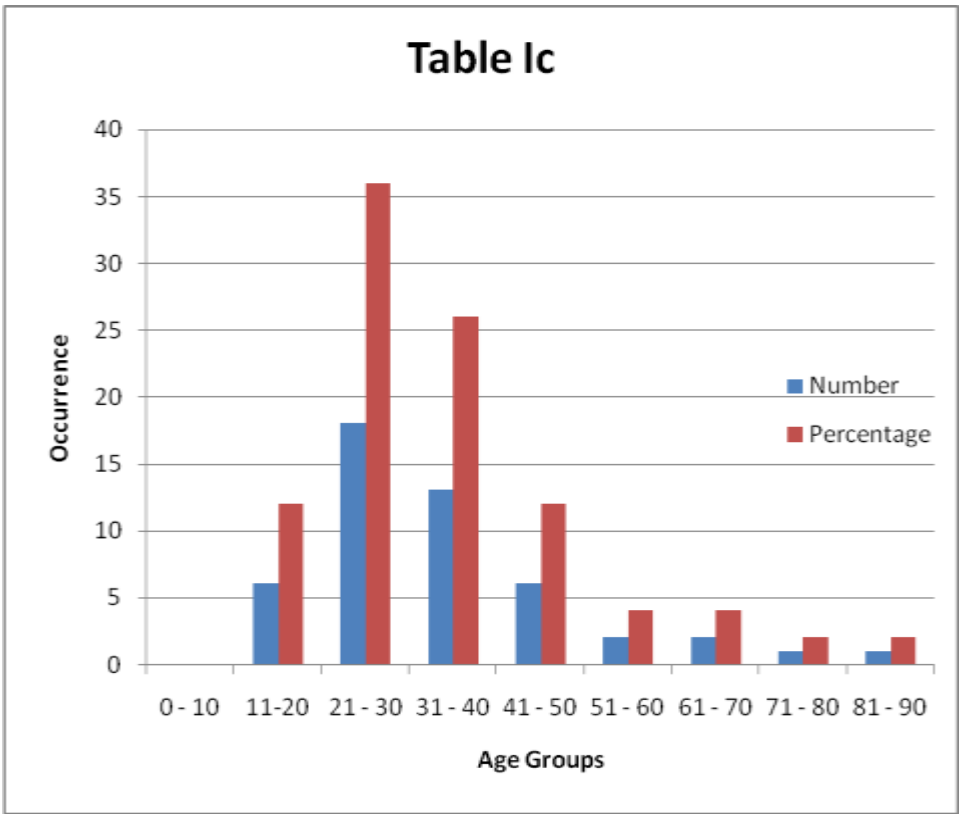


Table Ic : Age Groups: (N = 50)

	0 - 10	11 – 20	21 – 30	31 - 40	41 - 50	51 – 60	61 - 70	71 – 80	81 – 90
TOTAL	0	6	18	13	6	2	2	1	1
%	0.0	12.0	36.0	26.0	12.0	4.0	4.0	2.0	2.0

As indicated in Chapter 2 dealing with the epidemiology of hanging, in Hungary,[13,14] Norway,[15,16] Finland [17] and Ireland,[18] an increase in suicide has been observed in the younger age groups 10 to 19, while amongst the indigenous native American Indian indigenous cultures of Canada and the Maori culture of New Zealand, a similar increase has been observed. In particular, the last 40 years have seen an increase in hanging suicides amongst young males in Australia,[19] New Zealand,[20] South Africa,[21] and elsewhere.

The commonest ages for suicidal hanging in the present study therefore lie in the 21 to 40 year age groups. This distribution is markedly skewed, hence, the best average is the median value.

Median = **30.5** years (i.e. sort into numerical order and apply n + 1 ÷ 2 = 51 ÷ 2 = **25.5** = **30.5**.

Statistical Analysis

Age

Mean = **34.64** years

Standard deviation = **14.75**

Standard Error = **2.087**

Minimum = **13**

Maximum = **83**

Median = **30.5** years

Weight

Mean = **67.88** kg

Standard deviation = **10.96**

Sample size = **50**

Standard error of mean = **1.55**

Lower 95% confidence limit = **64.76**

Upper 95% confidence limit = **70.99**

Minimum = **38** kg

Median = **68.00** kg

Maximum = **105** kg

Normality = +

Height

Mean = **1.6762 m.**

Standard deviation = **0.1595 m.**

Sample size = **50**

Standard error of mean = **0.02256 m.**

Lower 95% confidence limit = **1.631 m.**

Upper 95% confidence limit = **1.722 m.**

Median = **1.695 m.**

Minimum = **0.7300 m.**

Maximum = **1.840 m.**

The distribution is not symmetrical. In other words, with reference to Table Ia: Age Groups, the following findings pertained:

¶ 36% = common

¶ 26% = fairly common

¶ 12% = fairly unusual

¶ 4% = uncommon

¶ 2% = rare

¶ 0% = very rare

II. Macroscopic Findings:

1. Level of Ligature around the Neck

2. Position of the Knot

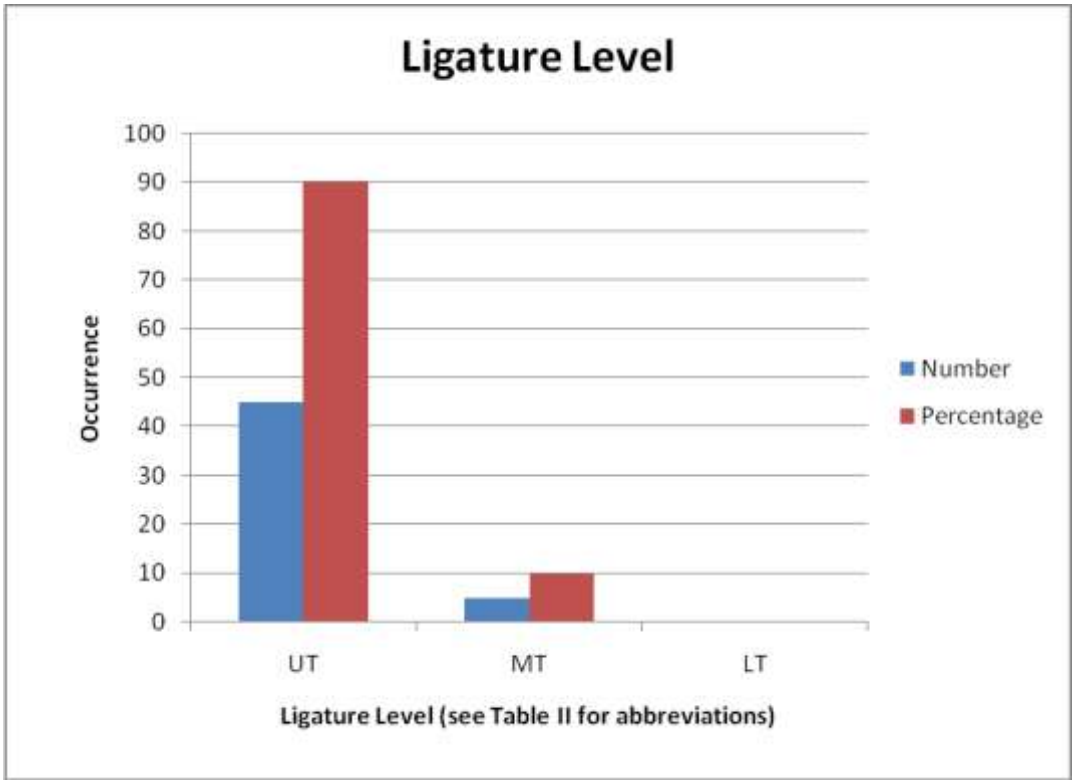
3. Type of Ligature Material

1. <u>Position (level) of the Ligature</u>

The physical post-mortem findings were analysed and classified with regard to the level of the ligature around the neck, i.e. whether upper third, middle third or lower third, as well as the position of the knot and the type of ligature material used. Analysis showed the level of the ligature around the neck in the following distribution:

a. <u>upper third of the neck</u>: 45 subjects (90%);
b. <u>middle third of the neck: 5 subjects (10%);</u>
c. <u>lower third of the neck</u>: none of the subjects (0%).

The high preponderance of ligature positioning in the upper third of the neck of ligature positioning is understandable since with the pull of gravity upon the body, the ligature would tend to slide upwards. However, an additional factor that should be considered, and which may have contributed to this 90% incidence, is that the ligature may have been so placed originally by the victim.



2. <u>Position of the Knot or Sliding Loop</u>

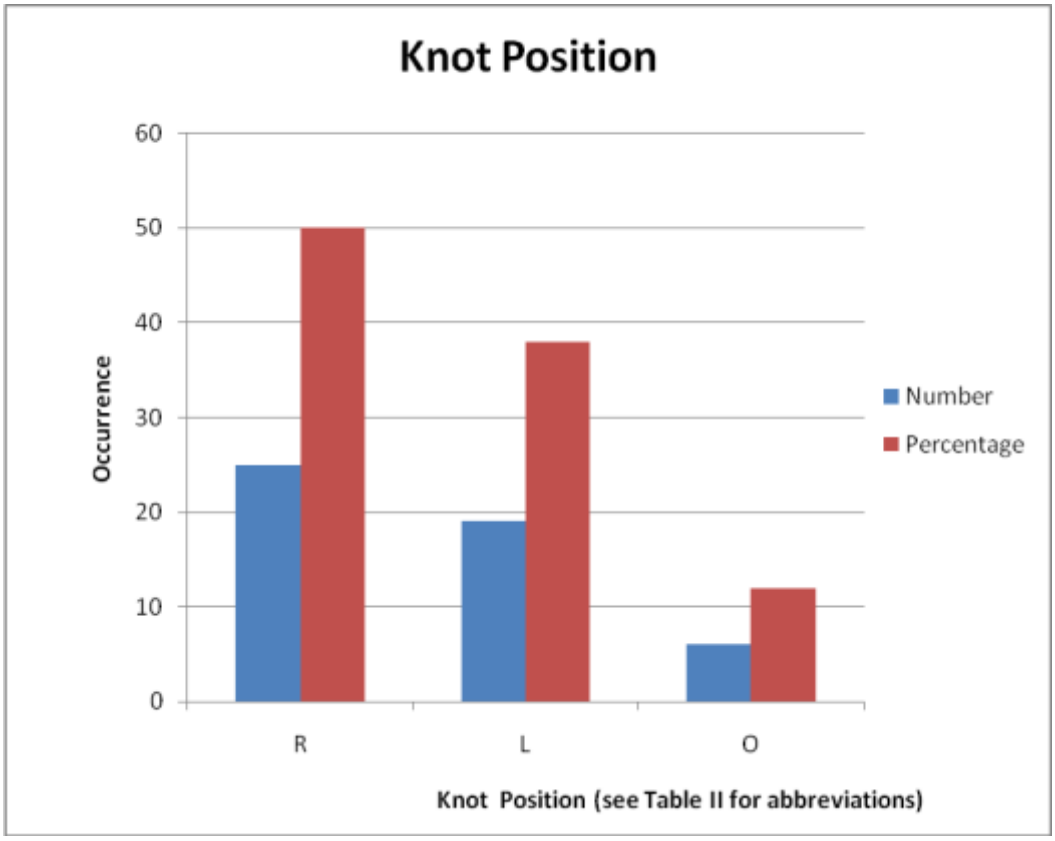
With regard to the position of the knot (when present) or sliding loop, this was found (or deduced to be found) over the right side of the neck in 25 subjects (50%), over

the left side of the neck in 19 subjects (38%), and at the occiput in 6 subjects (12%), i.e.:

a. right side of neck: 25 subjects (50%);

b. left side of neck: 19 subjects (38%);

c. occipital position: 6 subjects (12%).



3. Type of Ligature Material

The ligature itself consisted of the following materials:

¶ Rope in 6 subjects (12%);

¶ Nylon cord in 12 subjects (24%);

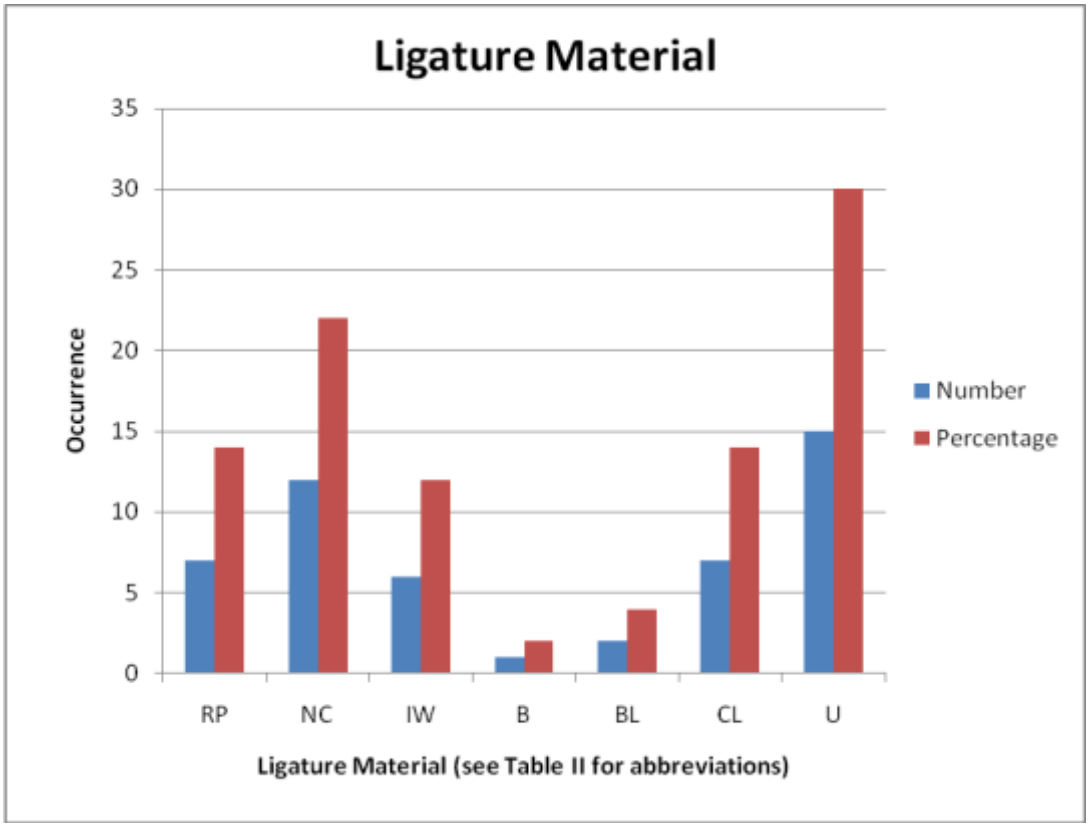
¶ Insulated wire in 6 subjects (12%);

¶ Leather belt in 1 subject (2%);

¶ Boot lace in 2 subjects (4%);

¶ Cloth in 7 subjects (14%);

¶ Unknown (due to removal of ligature by paramedical personnel or relatives) in 15 subjects (30%).



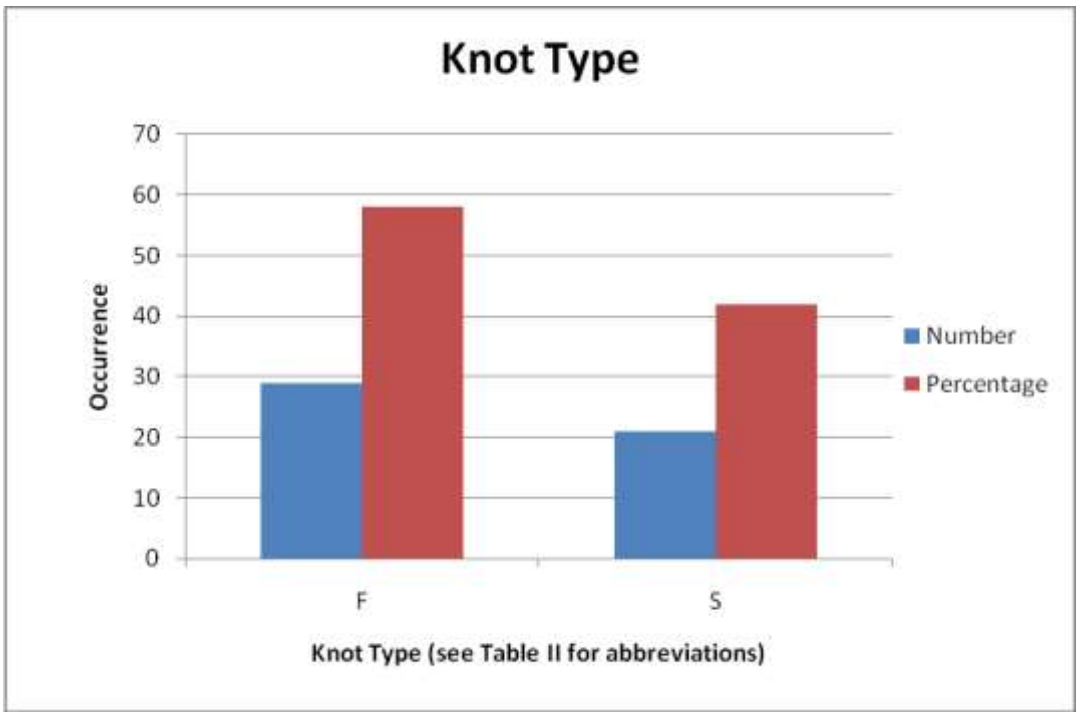
The finding of boot laces in the 2 subjects requires a measure of explanation. These were of the foot-ball, i.e. athletic type and were presumably used because an ordinary shoe lace is too short to go around the neck and be tied or attached to a fixed point. In 15 of the subjects (30%), the nature of the ligature was unknown due to the ligature having been removed at the scene by either the paramedical personnel or the relatives of the deceased and, therefore, not submitted with the

body to the mortuary. Despite the ligature being absent in these subjects, whether a fixed knot or sliding loop had been present could, nevertheless, be deduced by the presence of an irregularly oval area of abrasion overlying the ligature impression at a fixed point along its circumference. This was interpreted as having been produced by a fixed knot. Conversely, a slightly absent segment of skin marking along the circumference of the ligature impression was interpreted as indicating the presence of a sliding loop. (As an addendum, the force required to produce an abrasion in the living human subject is not precisely quantifiable, ethical considerations precluding this determination. However, by strict definition, an abrasion involves the superficial epidermal layers only, the dermis remaining uninvolved, suggesting insufficient force to involve the underlying deeper dermis. The best probable definition of an abrasion would be that of a superficial lesion produced by a lateral or tangential scraping force rubbing away or avulsing the epidermis. In the context of hanging, this force would be accompanied by movement, either of the ligature, the body itself, or a combination of the two. In other words, a friction abrasion results).

With these in mind:

¶ a sliding loop had been present in 29 subjects (58%)

¶ a fixed knot had been present in 21 subjects (42%)



These findings are further summarised in Table II which follows.

Table II: Ligature Findings (N = 50)

	Level of Ligature			**Position of Knot**			**Type of Knot**								
	UT = Upper Third			**R = Right**					**RP = Rope**				**B = Belt**		
	MT = Middle Third			**L = Left**					**NC= Nylon Cord**				**BL = Boot Lace**		
	LT = Lower Third			**O = Occiput**					**IW = Insulated Wire**				**CL = Cloth**		
	U = Unknown					**F = Fixed Knot**						**S = Slip Knot**			
	Ligature Level			**Knot Position**			**Knot Type**		**Type of Ligature Material**						
Case No	**UT**	**MT**	**LT**	**R**	**L**	**O**	**F**	**S**	**RP**	**NC**	**IW**	**B**	**BL**	**CL**	**U**
1	X					X	X								X
2	X				X		X				X				
3	X				X		X				X				
4	X			X			X			X					
5	X				X		X								X
6		X				X	X								X
7	X				X		X								X
8	X				X			X							X
9	X			X				X						X	
10		X			X		X			X					
11	X					X	X								X
12	X				X		X								X
13	X				X			X							X
14		X			X		X								X
15	X			X			X		X						
16		X		X				X		X					
17		X		X			X		X						
18	X			X				X							
19	X				X		X			X					
20	X			X				X		X					
21	X					X	X						X		
22	X					X	X								X
23	X				X			X		X					
24	X				X			X		X					
25	X			X				X		X					
26	X			X				X						X	
27	X			X			X				X				
28	X			X				X	X						
29	X			X				X							X
30	X					X	X				X				
31	X				X		X							X	
32	X				X		X							X	
33	X				X			X						X	
34	X				X			X			X				
35	X			X				X	X						
36	X			X				X		X					
37	X			X				X	X						
38	X			X			X			X					
39	X			X				X		X					
40	X				X			X	X						
41	X			X			X			X					
42	X			X				X	X						
43	X				X		X								X
44	X			X			X							X	
45	X			X			X						X		
46	X				X			X						X	
47	X			X			X								X
48	X			X			X								X
49	X			X			X				X				
50	X			X			X					X			

TOTAL	45	5	0	25	19	6	29	21	7	12	6	1	2	7	15
%	90.0	10.0	0.0	50.0	38.0	12.0	58.0	42.0	14.0	22.0	12.0	2.0	4.0	14.0	30.0

However, a rider must be added. Conventional wisdom dictates that at the meeting of the two ends of the loop, a gap is usually present between the overlying ligature and the underlying neck. In 15 of the 50 subjects (30%) no ligature had been submitted with the body while in the remaining 36 subjects (72%) the ligature had been submitted. In these 36 cases, i.e. nearly three-quarters of the total, the constricting ligature was found to be very tightly applied around the neck, regardless of whether a fixed knot or sliding loop was present. This tightness of ligature application was attested to by the very great difficulty encountered in inserting the point of a pair of scissors underneath the ligature in order to cut it. A factor contributing to the tightness of ligature application would be, in the case of a sliding loop, the weight of the body. In those subjects with a fixed knot, the contributing factor to tightness of ligature application would be the weight of the body but compounded by the victim him-or-herself applying the ligature tightly.

To reiterate, this tightness of ligature application included those subjects with a sliding loop, no gap being present between the ligature and the underlying neck tissues. This finding in the present study therefore tends to refute the conventional wisdom that a gap is always present where a sliding loop has been employed. In other words, due to the tightness of ligature application with no gap being present, the pressure applied around the neck would be expected to be equally or almost equally distributed around and throughout the tissues of the neck. This tends to lend support to the inference that the bilateral incidence of **arterial** and **neural** damage encountered in the present study was, in many cases (*vide infra*), either identical or very similar, indicating an equal or almost equal application of circumferential force. In those cases where differences in bilateral

incidence existed, it is suggested with reservation that these could be attributed to slight differences in vessel angulation brought about either by anatomical variation, the 5 – 6 kilogram weight of the head falling to one or other side, or differences in posture of the body where full suspension had not taken place.

Illustration of Selected Hanging Cases from the Present Study

The following illustrations (photographs) are included to illustrate some of the features in a few of the cases in the present series. Relevant comments are included in each case.

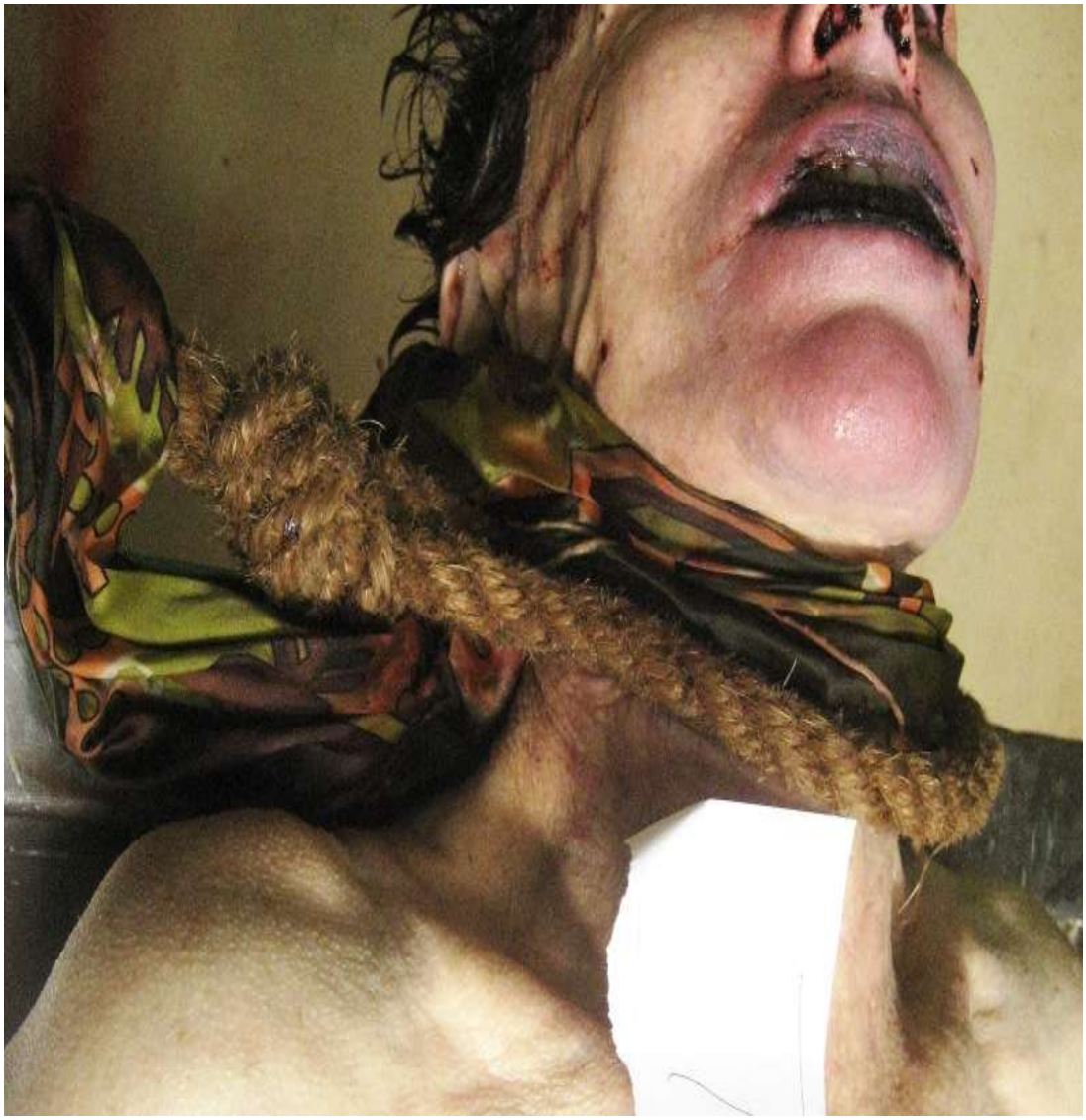


Fig. 7a. Rope and scarf ligature. (the scarf was tightly applied beneath the overlying loosely applied rope). Note the haemorrhage from the mouth and nose. Case no. 44.



Fig. 7b. Nylon cord ligature with a fixed knot over the right side of the neck. Case no. 38.

The dried blood seen extending from the nostrils and over the lips represents a combination of blood derived from two sources, namely, lung and nasal mucosa. Haemorrhage from the lungs may occur for three reasons, acting singly or in combination:

i) purging of fluids of decomposition;

ii) ruptured small pulmonary vessels and alveolar septal capillaries as a result of the neurogenic pulmonary oedema consequent on sympathetic stimulation, and;

iii) ruptured alveolar septal capillaries from increased intra-alveolar pressure.

It is suggested that, as a mechanism for the above, the latter may possibly occur as a result of diaphragmatic paralysis, with the diaphragm becoming fixed in a state of inspiration due to pressure on the **phrenic nerve** (the major neural innervation of the

diaphragm), the **sternocleidomastoid muscle** and the **scalenus anterior muscle** (an accessory muscle of inspiration). As a consequence of action potentials being initiated in these latter muscles, the thoracic cage, i.e. chest wall becomes fixed in a state of inspiratory paralysis due to initiation of both **dynamic and static stretch reflexes**. These are discussed in greater detail in the Discussion chapter which follows.

It should be borne in mind that when the ligature lies over the upper-third of the neck, the **phrenic nerve**, the **scalenus anterior muscle** and the **sternocleidomastoid muscle** lie beneath the encircling compressive ligature.

On the other hand, haemorrhage from the nose and mouth may also be due to rupture of nasal mucosal vessels following their congestion due to increased pressure as a consequence of ligature compression of the pharyngeal venous plexus as well as vessels derived from the external carotid arteries and veins.



Fig. 7c. Insulated wire ligature. The ligature is seen encircling the neck with a further diagonal ligature impression above the ligature. Despite the tightness of ligature application, the ligature has been displaced downwards by the weight of the body when the suspension was cut. Case no. 2.

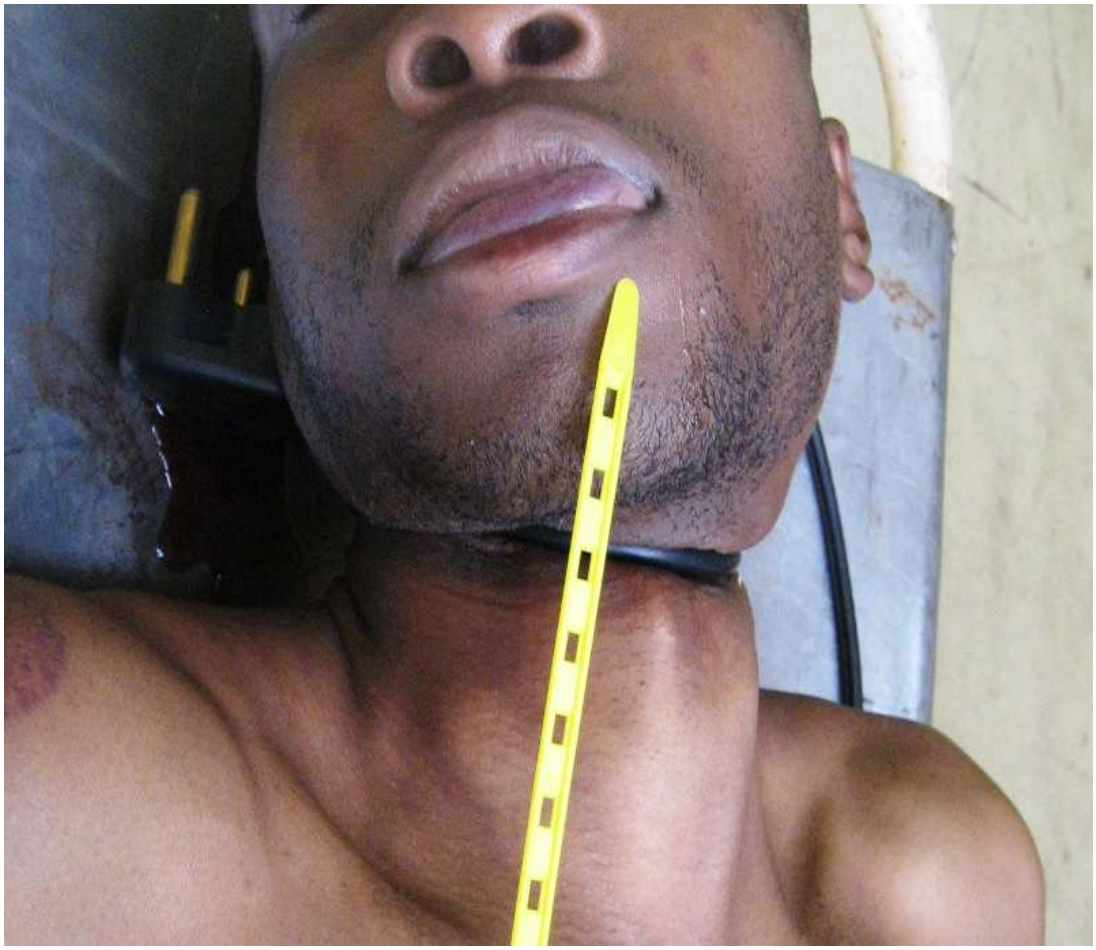


Fig. 7d. Insulated electrical cord ligature. The ligature is seen encircling the upper third of the neck with a fixed knot on the right. Note the tightness of ligature application with protrusion of the tongue. Case no. 27.



Fig. 7e. Belt ligature. The ligature in this case has been displaced downwards from its original position over the upper third of the neck to a lower position in order to reveal the ligature abrasion over the upper third of the neck, slightly better seen on the right which is not in shadow. The ligature in this case was loosened by the paramedical personnel. Case no. 50.



Fig. 7f. Cloth ligature. The ligature consists of a necktie with a fixed knot on the left side of the neck. The ligature has been moved downwards and backwards to allow visualization of the well-marked ligature impression slanting diagonally upwards at the lower border of the mandible. The ligature mark does not reflect the true width of the ligature as is often the case with soft cloth ligatures. The ligature is broader but the depth and well-marked impression reflects the original tightness of ligature application. Case. No. 32.



Fig. 7g. Cloth ligature. A sliding knot was present in this case over the occiput but the doubled ligature has been loosened and moved forwards and over to the right side of the neck to emphasize how cloth ligatures are sometimes wound several times around the neck by the victim. Presumably, this type of ligature application is to enhance the hanging effect and hasten the demise. Note the protrusion and drying of the tip of the tongue. Case no. 26.



Fig. 7h. Ligature Unknown. A problem encountered in several cases where the ligature had been removed at the scene either by the relatives of the deceased, the paramedical personnel or the police and not submitted with the body to the mortuary. However, a well-marked ligature impression is seen anteriorly with linear abrasions and areas of bruising slanting diagonally upwards, backwards and to the right over the upper third of the neck. Case no. 48.



Fig. 7i. Ligature Unknown. A further example of a body submitted to the mortuary without the ligature. However, a well-marked ligature impression is seen slanting diagonally upwards, backwards and to the left side over the upper third of the neck. The wider more irregular area on the left neck along the circumference of the ligature acts as a pointer to the position of the original knot. Despite the absence of the ligature, the well-marked ligature impression bears witness to the original tightness of ligature application as well as illustrating how, in some cases, the ligature rises to as high a level around the neck as possible (and only prevented of sliding higher by the prominent angle of the mandible. Case no. 12.

There follow a series of photographs of suicidal hanging that illustrate the complexity of the ligature and the complete encirclement of the neck. The photographs are purely to emphasise these features and did not form part of the present study, as the subjects underwent medicolegal autopsy prior to inception of the study.



Fig. 7j. Nylon cord ligature. A good example of a sliding loop.The ligature is seen twice encircling the upper third of the neck with a sliding loop on the left. The ligature is prevented from sliding higher by the angle of the mandible. (Non-series case).



Fig. 7k. Belt ligature. The ligature is seen encircling the upper third of the neck. Note the tightness of ligature application with constriction of the underlying tissues. The ligature has been applied so tightly as to ‘cut’ into the tissues of the neck. (Non-series case).



Fig. 7l. Rope ligature. The ligature is seen encircling the upper third of the neck. Note the complexity of ligature application, the ligature being wound several times around the neck with multiple interwoven knots on the right. Note also the tight application of the ligature with constriction of the underlying tissues. It appears as though the tissues underneath the ligature have been 'cut' by the pressure. An inverted 'U'-shaped ligature abrasion is seen beneath the ligature corresponding to one of the loops of the rope. (Non-series case).



Fig. 7m. Rope ligature. A further view of the ligature seen in Fig. 7l showing the complexity of ligature application and constriction of the tissues. (Non-series case).



Fig. 7n. Nylon strap ligature. The ligature is seen encircling the neck several times with a partial knot beneath the mandible on the left. A well-marked ligature impression is seen slanting diagonally upwards to the lower border of the pinna of the ear and mastoid process on the left. Note the tightness of ligature application.
(Non-series case).

As pointed out in the Introduction and Materials and Methods, the major thrust of this work was to study in detail the **vascular** and **neural** structures of the neck in an attempt to determine the actual cause of unconsciousness and death in suicidal hanging.

This involved the careful and meticulous dissection of the neck structures and their careful removal, followed by a microscopic study of the damage produced by a constricting ligature.

It should be pointed out that in none of the hanging cases examined was any damage found to the **hyoid-larynx complex**, its associated **cartilages**, or **vertebral column**, although, as shown in **Figs. 7.55a.**, **7.55b.**, **7.56.**, **7.57.**, **7.58.**, **7.59.**, **7.60.**, **7.61.**, **7.62.**, **7.63.**, **7.64.**, **7.65a.**, **7.65b.**, **7.66a.**, **7.66b.**, **7.67a.**, **7.67b.** and **7.68.** and their accompanying legends (*vide infra*) and text, clear-cut damage to the surrounding **nerves**, **veins**, **connective tissues** and **lymph nodes** deep to the site of ligature application was noted.

In the light of these stringent requirements, the histopathological findings have been classified into three major groups to facilitate appreciation and understanding of the effects of a constrictive pressure to the neck.

The groups are related to the complexity of the anatomical and physiological structures present in the neck. These are numbered in series as set out previously, i.e. the **Vascular findings (III)**, the **Additional vascular findings (IV)**, and the **Neural findings (V)**. (Group **I** refers to the **Demographics and Epidemiology** while Group **II** refers to the **Ligature findings** previously alluded to and fully described above).

III. Vascular Findings

These comprised the first of the 3 histopathological findings emerging from the study The **vascular findings** were assessed by the microscopic examination of the three **carotid** arteries on each side of the neck. These were performed in detail in an

attempt to discover specific lesions of the vascular structures of the neck which could be related to the presence or action of:

a) a circumferential tightly compressive and constricting force applied to the neck by a ligature, and;

b) lesions occurring as a consequence of tensile stretching forces acting on the vascular structures due to the gravitational pull of the weight of the body in the hanging process.

In this section, to lend emphasis and avoid confusion, the three layers of the vessel wall, namely, **intima**, **media**, and **adventitia** have been highlighted in bold script. In this regard, the term **media** refers to the middle laminated muscle layer of the vessel wall. In addition, for ease of reference and for purposes of comparison, a summary of the different findings is provided at the end of each section with a general summary at the end of the entire section dealing with the **Vascular Findings**. These latter are presented both in the text and in the simplified form of Table IIIa. In the description which follows, it should be noted that the Concise Oxford Dictionary defines '**tear**' as a violent or forcible rending or pulling apart of the tissues. On this basis, the **vascular findings** noted were classified and divided into the following categories:

(1) **superficial intimal tears**, single or multiple, involving the **intima** or inner layer of the vessel wall only;

(2) **intimal tears extending to or along the internal elastic lamina**, single or multiple, being defined as those **tears of the intima** which extend to or along the **internal elastic lamina**. The **internal elastic lamina** is a dense elastic membrane which separates the **intima** from the **media,** as defined in standard text books of anatomy and histology; (3) **intimo-medial tears**, being those **tears** which extend

through the full-thickness of the **intima** to involve the deeper, middle or **muscular layer of the vessel wall**. These **tears** were further divided according to depth or extent of involvement of the **media** into:

i) **tears** extending through **intima to the inner-third of the muscular layer;**

ii) **tears** extending through **intima to the middle-third of the muscular layer;**

iii) **tears** extending through **intima to the outer-third of the muscular layer**;

(4) **tears of the media (muscular layer of the vessel wall)** only, involving either inner-, middle- or outer- thirds of the **layer**, and being single or multiple;

(5) **tears of the adjacent adventitio-medial layer of the vessel wall**. This refers to those **tears** which extend from or through the outermost layer of the vessel wall, i.e. the **adventitia** to involve the underlying **media**, either to the latter's inner-, middle- or outer- thirds, and being single or multiple;

(6) **tears of the arterial adventitia**. This refers to **tear** or **tears** involving only the outermost layer of the vessel wall, being single or multiple; the phrase 'single or multiple' within the specific context of the present study refers to a solitary **tear**, i.e. 'single' with more than one **tear** being 'multiple', involving that particular layer of the vessel wall as seen at the level of the bifurcation. This latter point cannot be stressed strongly enough and is, in fact, indicated in the 'Material and Methods' chapter. The **tear/tears** of that particular layer of the vessel wall observed have, as indicated in both the text and in Tables IIIa and IVa, been classified in accordance with each individual artery involved, namely, the **left common carotid artery**, the **left internal carotid artery**, the **left external carotid artery**, the **right common carotid artery**, the **right internal carotid artery**, and the **right external carotid artery**. The numerical representation of each **tear**, i.e. whether single or multiple

seen at the level of bifurcation is, at the risk of overemphasis, documented both in the text which follows and in Table IIIb and IVb which deal with the numerical representation of each lesion in that particular artery.

(7) **adventitial haemorrhage**: this refers to **haemorrhage** (as a consequence of small vessel rupture) within the **adventitial layer**;

(8) **complete circumferential transverse rupture** which refers to those **tears** extending throughout the full-thickness of the vessel wall, i.e. a through-and-through breach involving all layers of the arterial wall (Table III; see Appendix).

Each classified finding is illustrated by a photomicrograph of a vessel accompanied by labels and descriptions shown in the photograph. All arterial specimens for histopathological examination were taken at the level of bifurcation in view of the tendency of arteries to sustain damage at points of branching due to the tangential shearing stresses applied. The lesions observed and documented refer, of course, to the specific artery, i.e. **left common carotid**, **left internal carotid**, **left external carotid**, **right common carotid**, **right internal carotid** and **right external carotid** in which the lesion/lesions were found.

1. Superficial Intimal Tears

An analysis of the anatomical distribution of arterial involvement by each type of histopathological change as well as the numerical distribution of involvement showed the **left common carotid artery** specifically to be involved by single **intimal tears** (at the level of bifurcation) in 5 subjects (10%) and multiple **intimal tears** (at the level of bifurcation) in 7 subjects (14%). In contrast, the **left internal carotid artery** showed single **intimal tears** (at the level of bifurcation) in 4 subjects

(8%) and multiple **intimal tears** (at the level of bifurcation) in 4 subjects (8%) (Tables III and IIIa) (Figs.7.3a and 7.3b).

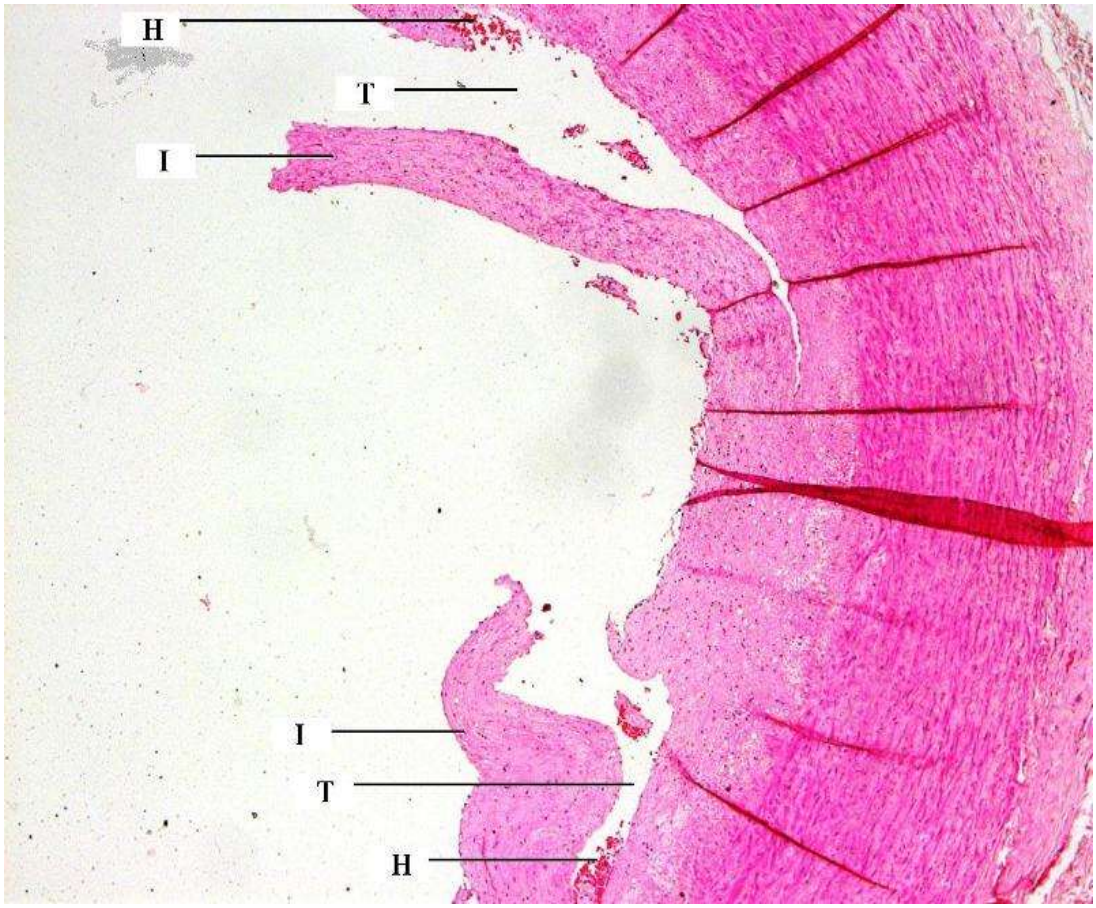


Fig. 7. 3a. Left internal carotid artery. Multiple **tears** of an intimal atheromatous plaque are noted. One **tear** extends circumferentially within the plaque while the other extends to the **internal elastic lamina**. Small accumulations of red blood cells are noted within the tear clefts. Case no. 6 (Haematoxylin and Eosin x 50). **H** = haemorrhage. **T** = tear. **I** = intima.

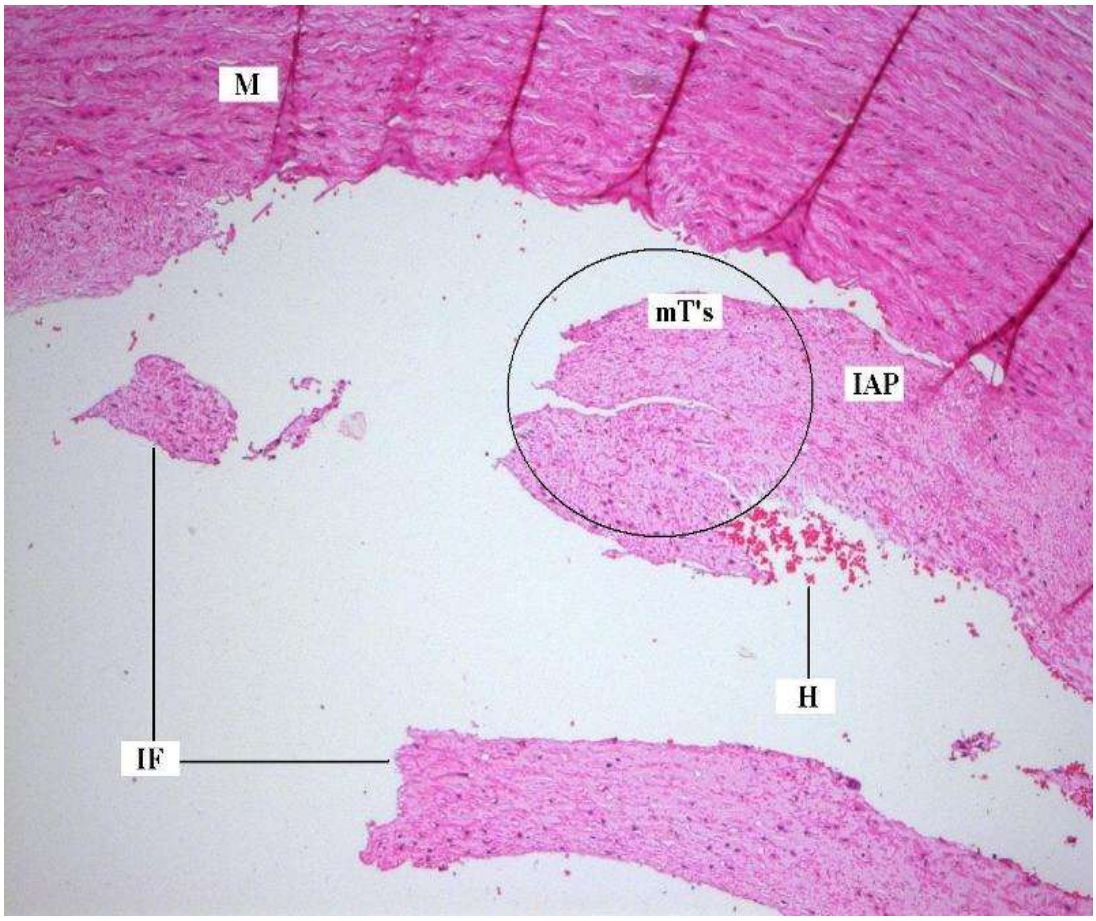


Fig. 7. 3b. Left internal carotid artery. A high-power view of the **tears** of the intimal atheromatous plaque seen in Fig. 7. 3a. Several **microtears** are, in addition, noted. Scattered accumulations of red cells are seen. Case no. 6 (Haematoxylin and Eosin x 100). **I** = intima. **IF** = intimal fragment. **IAP** = intimal atheromatous plaque. **H** = haemorrhage. **mT =** microtear. **M =** media.

The **left external carotid artery** showed no single **intimal tears** in any of the subjects (0%) but multiple **intimal tears** in 8 subjects (16%). The **right common carotid artery** showed single **intimal tears** in 4 subjects (8%), multiple **intimal tears** in 4 subjects (8%), the **right internal carotid artery**, single **tears** of the **intima** in 3 subjects (6%) and multiple **tears** in 7 subjects (14%), and the **right**

external carotid artery single **tears** of the **intima** (at the level of bifurcation) in 1 subject (2%) and multiple **intimal tears** (at the level of bifurcation) in 7 subjects (14%) .

N. B. At this juncture, it is important to reiterate that the histological section examined is at a specific level, i.e. at the bifurcation, the focal point of interest and emphasis in the present study in view of the tendency of arteries to split or sustain damage at points of arterial branching due to tangential shearing stresses.

Summary of Superficial Intimal Tears

In summary, in the 50 subjects examined, the incidence of positive findings (damage) in each artery was as follows:

Left common carotid artery: single tears = 10%
multiple tears = 14%

Left internal carotid artery: single tears = 8%
multiple tears = 8%

Left external carotid artery: single tears = 0%
multiple tears = 16%

Right common carotid artery: single tears = 8%
multiple tears = 8%

Right internal carotid artery: single tears = 6%
multiple tears = 14%

Right external carotid artery: single tears = 2%
multiple tears = 14%

Analysis of Findings

Left: Single tears occurred in 18% (out of 50 cases)

Multiple tears occurred in 38% (out of 50 cases)

Right: Single tears occurred in 16% (out of 50 cases)

Multiple tears occurred in 36% (out of 50 cases)

There is a remarkable correspondence of **Left and Right single tears (18 and 16%)** and of **multiple tears Left and Right (38% and 36%)**.

In general, and as previously indicated, it would appear that equivalent degrees of damage occur on **Left** and **Right** sides. While there is no correspondence with other factors such as age, height and weight, there appears to be some degree of equivalence with regard to the position of the knot and whether a fixed knot or sliding loop had been present. A right-sided knot (or sliding loop) was present in 25 out of the 50 cases and a left-sided knot or loop in 19 out of the 50 cases with, in 6 cases, the knot or loop lying in an occipital position. A fixed knot had been present in 29 cases and a sliding loop in 21 cases. These findings are interpreted in greater detail in the Discussion chapter which follows.

2. Intimal Tears extending to or along the Internal Elastic Lamina

Single **intimal tears extending to or along the internal elastic lamina** were noted involving the **left common carotid artery** in 5 subjects (10%) and multiple **tears** in 3 subjects (6%) (Figs.7.4a,7.4b,7.4c, 7.5, 7.6, 7.7a and 7.7b).

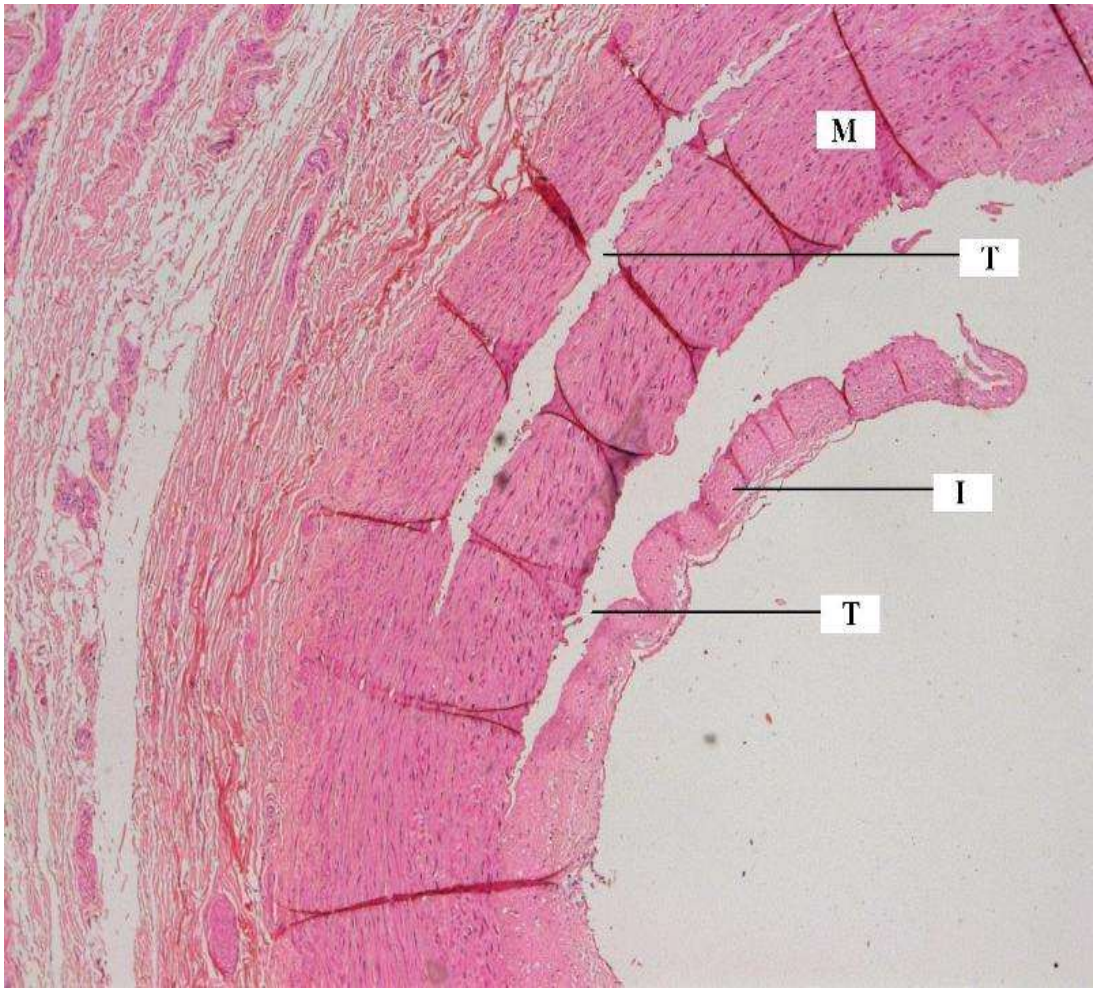


Fig. 7.4a. Left common carotid artery. An **intimal tear** with haemorrhage extending along the **internal elastic lamina** is noted together with an overlying **tear** involving the **middle-third of the media**. **Endothelial elevation** and **subendothelial clefts** are, in addition, noted. Case no. 10 (Haematoxylin and Eosin x 50). **I** = intima. **M** = media (muscle layer). **T** = tear.

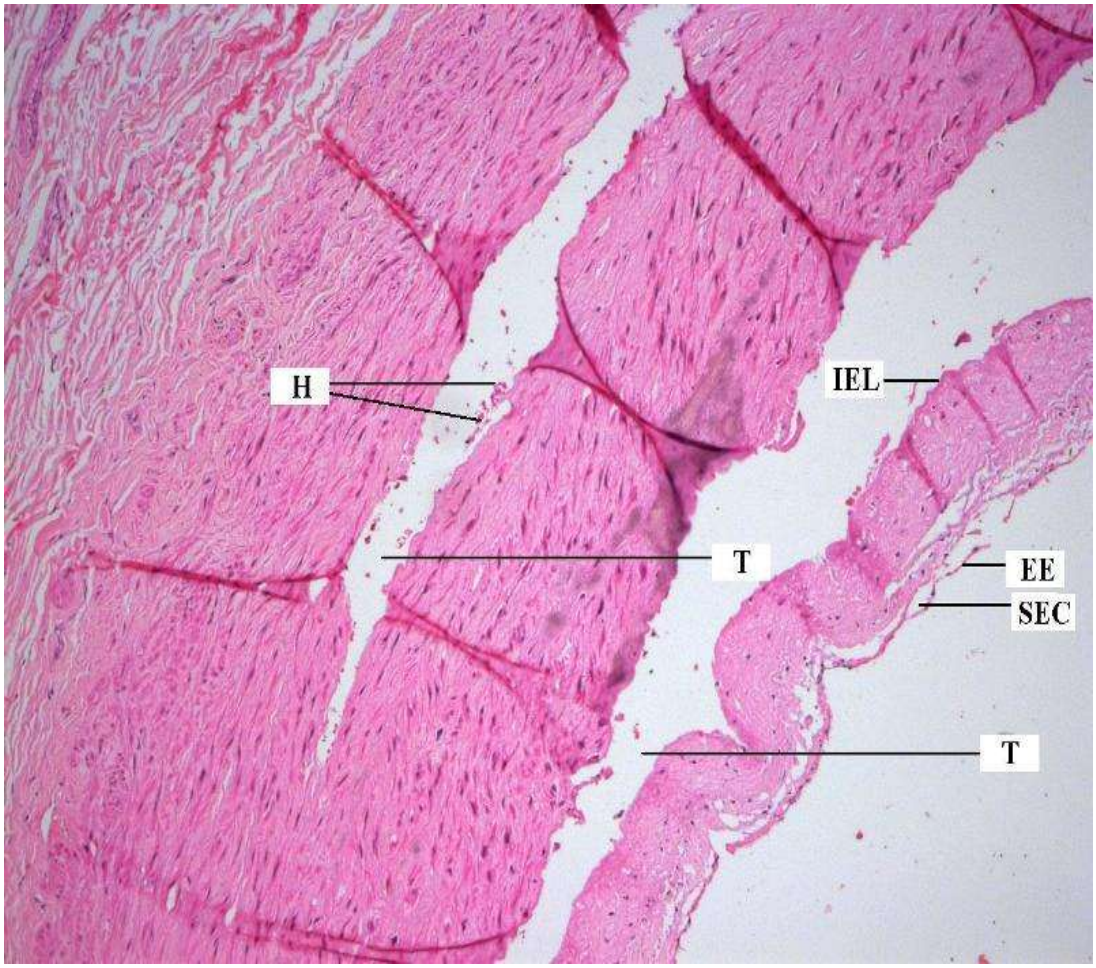


Fig. 7. 4b. Left common carotid artery. A high-power view of the **intimal tear** with haemorrhage extending along the **internal elastic lamina** noted in Fig. 7.4a is seen together with the overlying **tear** and its contained haemorrhage involving the **middle-third of the media**. **Endothelial elevation** and **subendothelial clefts** are, in addition, noted. Case no. 10 (Haematoxylin and Eosin x 100). **T** = tear. **H** = haemorrhage. **SEC** = subendothelial cleft. **EE** = endothelial elevation. **IEL =** internal elastic lamina.

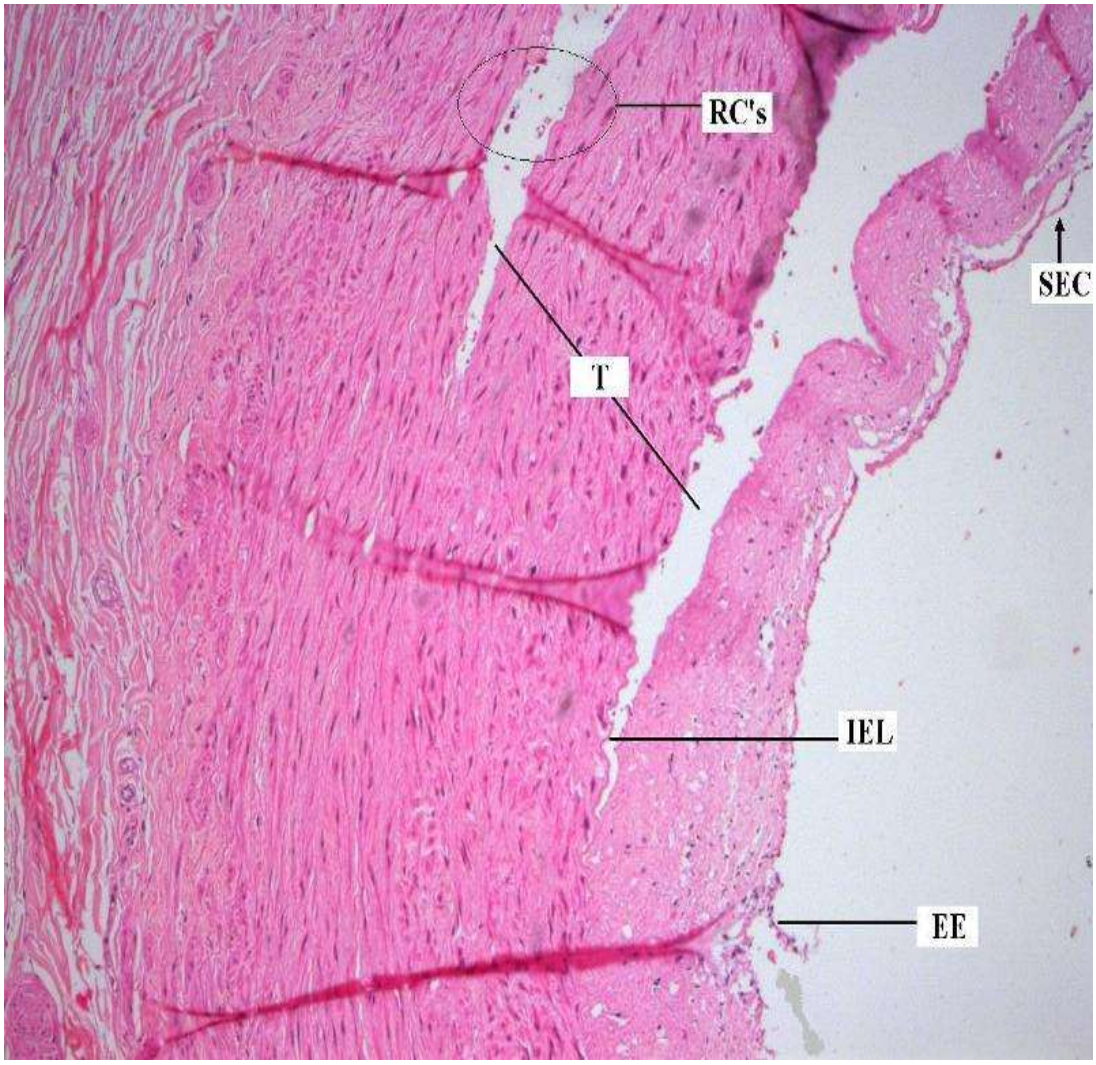


Fig 7.4c. Left common carotid artery. The **tear** along the **internal elastic lamina** and the **tear of the media** seen in Figs. 7.4a and 7.4b are shown but continued to their points of origin and inception. The **endothelial elevation** and **subendothelial clefts** seen in the previous figures are highlighted by an arrow. Case no. 10 (Haematoxylin and Eosin x 100). **T** = tear. **RC's** = red blood cells. **SEC** = subendothelial cleft. **EE** = endothelial elevation. **IEL=** internal elastic lamina.

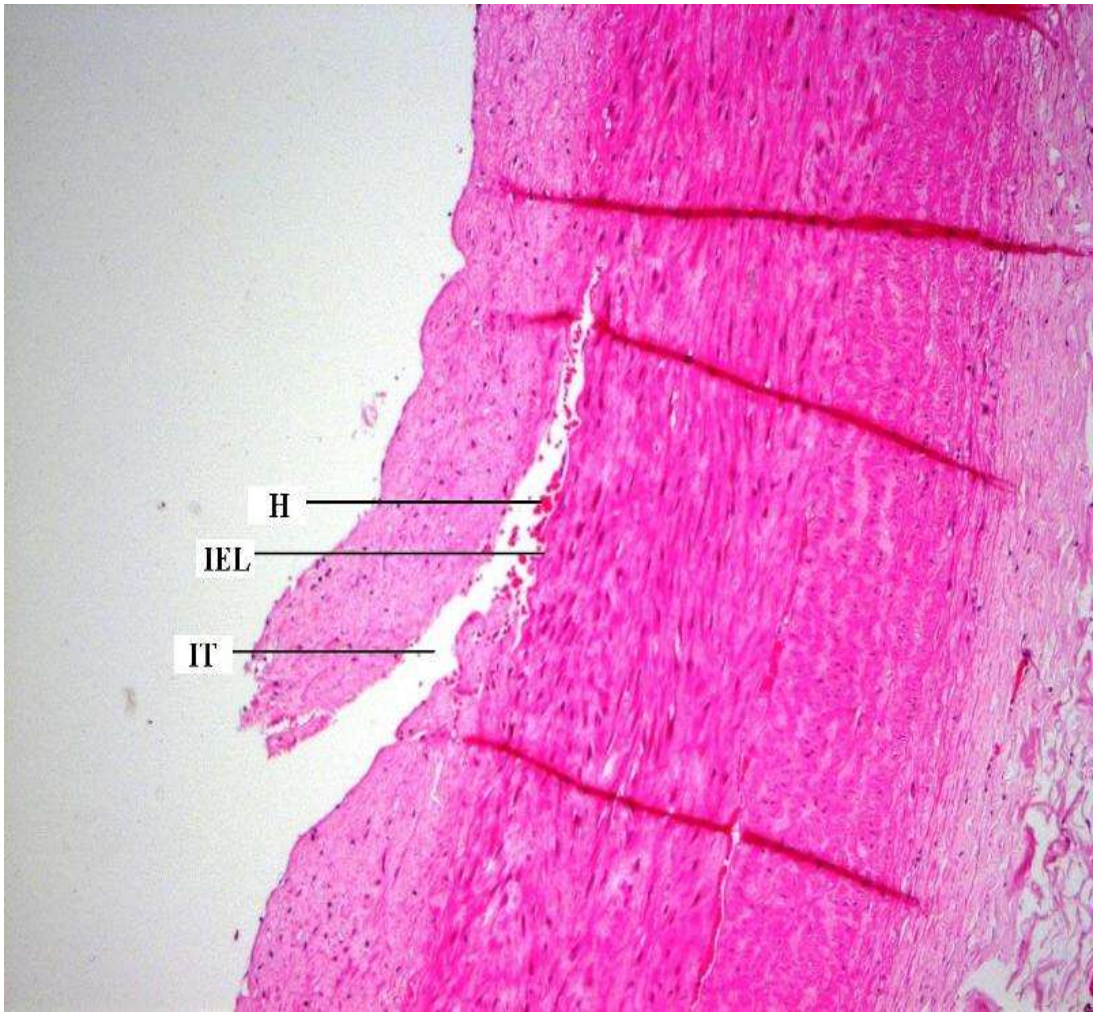


Fig. 7. 5. Left common carotid artery. An **intimal tear** with **haemorrhage** along the **internal elastic lamina** is noted. Case no. 31 (Haematoxylin and Eosin x 100). **IT** = intimal tear. **H** = haemorrhage. **IEL** = internal elastic lamina.

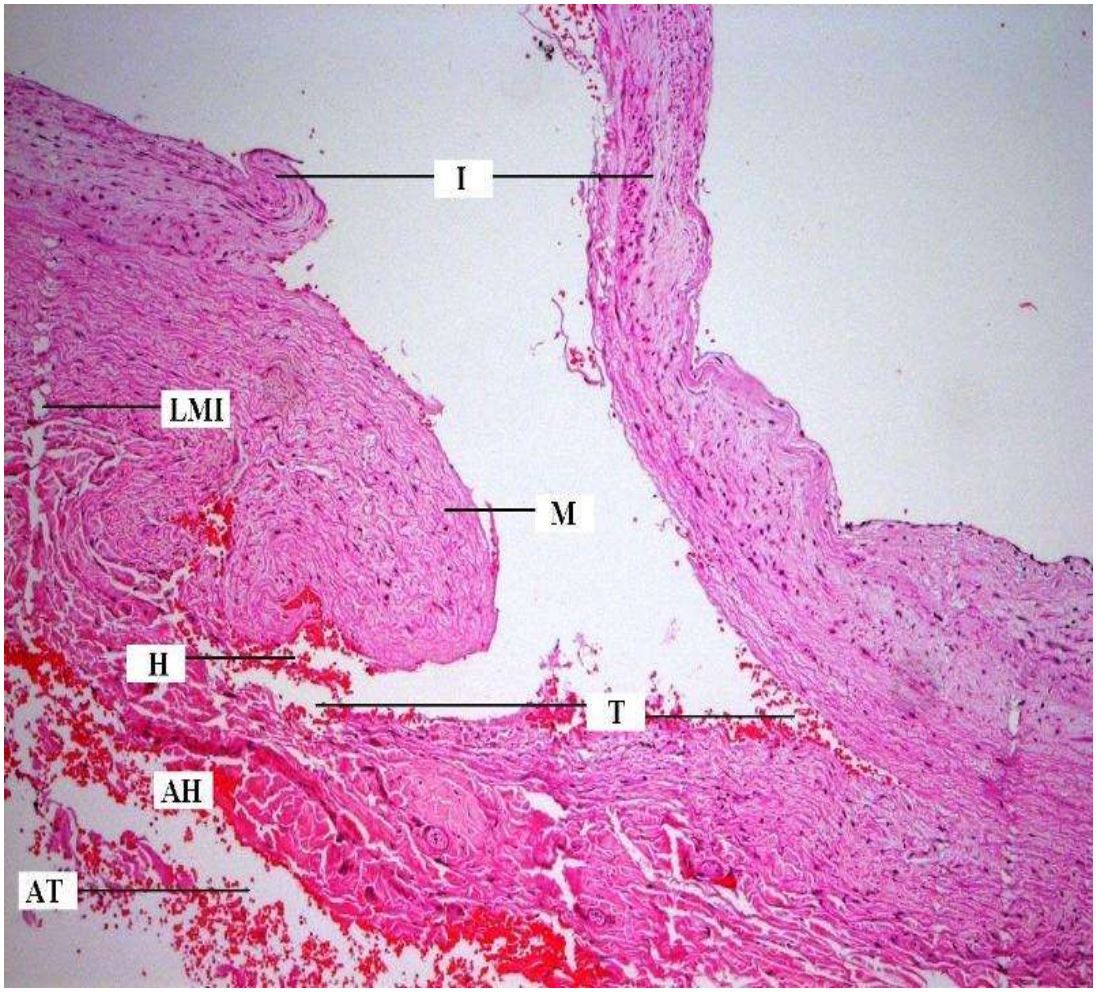


Fig. 7. 6. Right common carotid artery. A **tear** extending through the **intima** and along the **internal elastic lamina** with **tears** of the **inner-, middle-, and outer-thirds of the media** with **haemorrhage** is noted, together with **adventitial tearing**, **disruption** and **haemorrhage**. At the left border of the field, a vertical linear microtome incision is noted. Case no. 25 (Haematoxylin and Eosin x 100). **I** = intima. **LMI** = linear microtome incision. **M** = media. **T** = tear. **H** = haemorrhage. **AH** = adventitial haemorrhage. **AT** = adventitial tear.

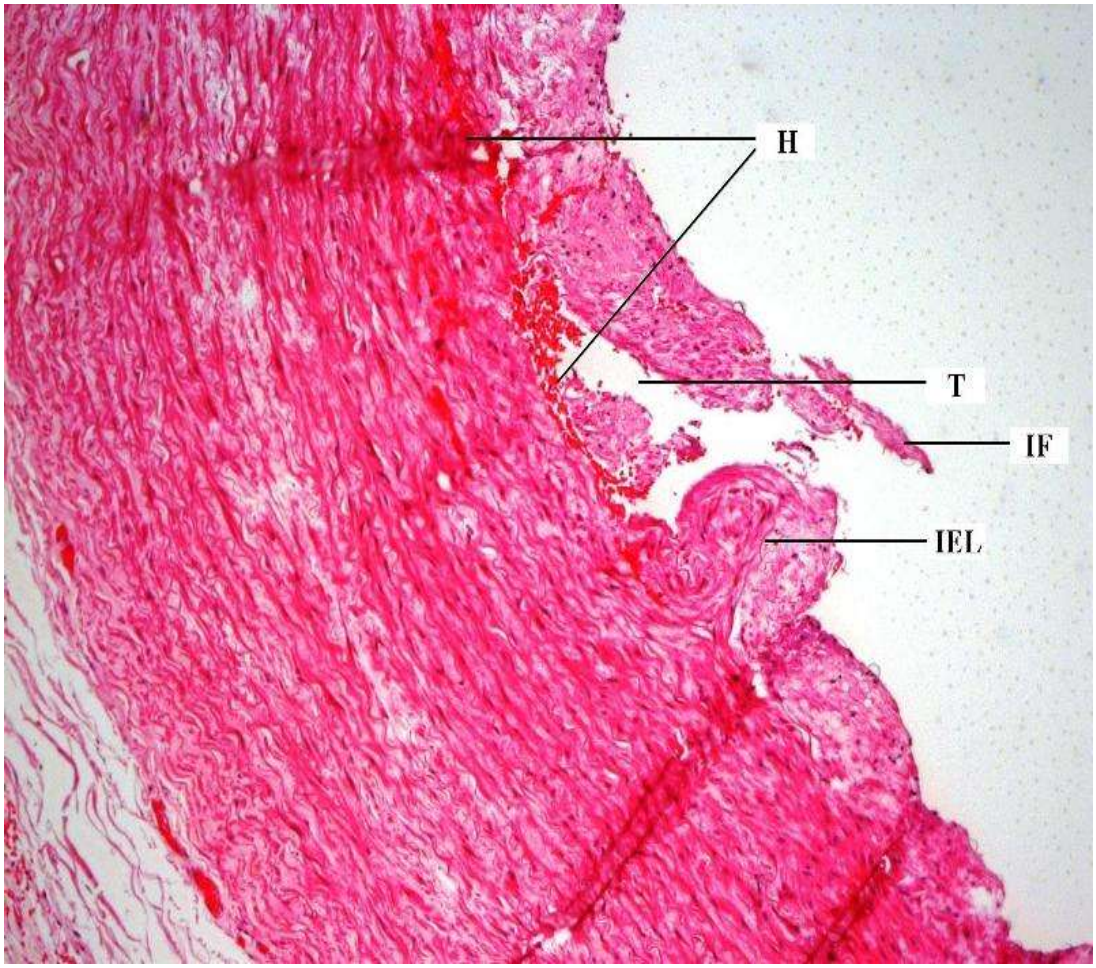


Fig 7. 7a. Left common carotid artery. Endothelial elevation and avulsion, an **intimal tear** with infiltrating haemorrhage extending to and along the **internal elastic lamina** and **tears of the inner-third of the media** with **haemorrhage** are noted. A fragment of the **intima** has been avulsed into the lumen of the vessel. Case no. 38 (Haematoxylin and Eosin x 80). **H** = haemorrhage. **T** = tear. **IEL** = internal elastic lamina. **IF** = intimal fragment.

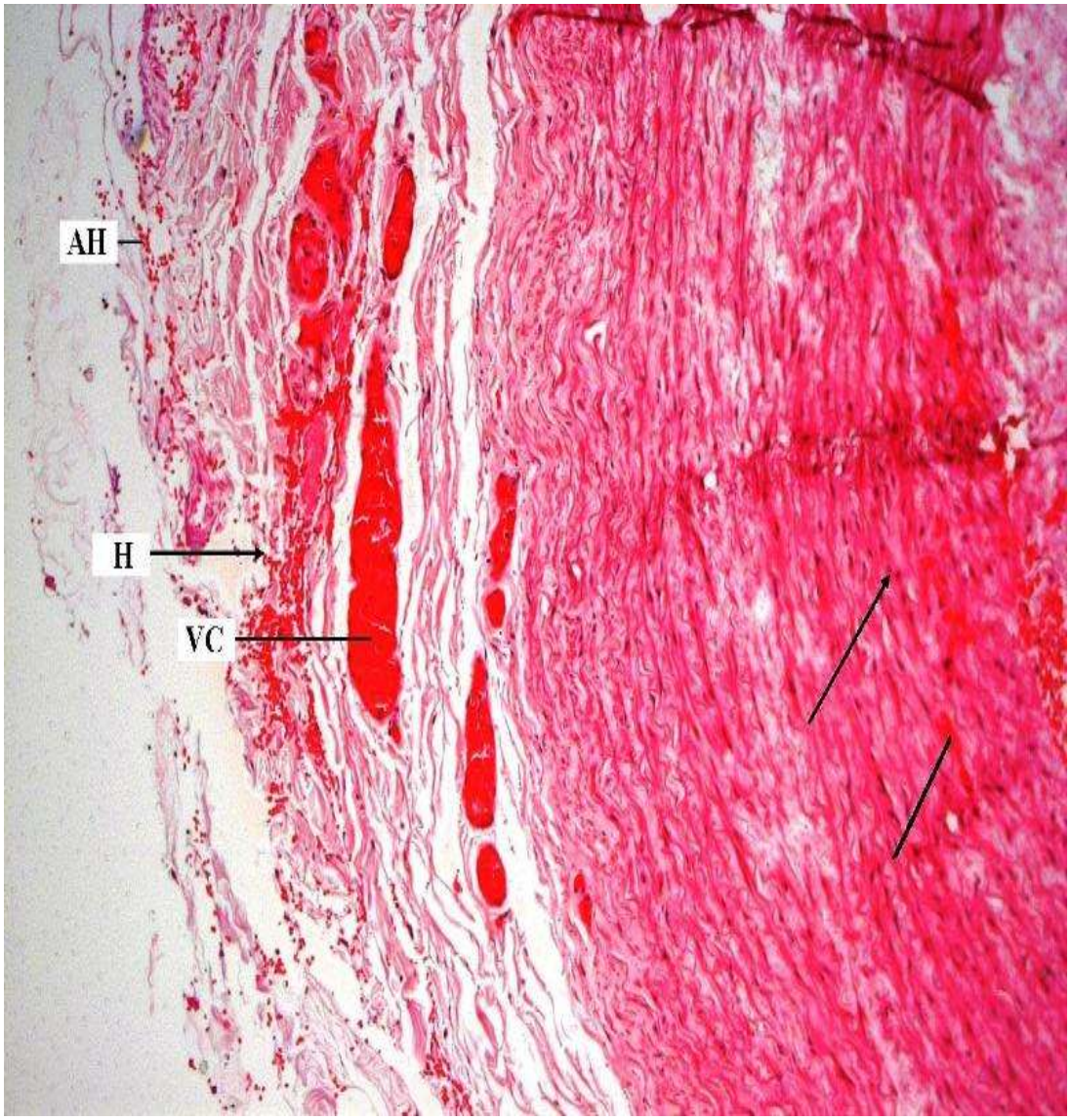


Fig. 7.7b. Left common carotid artery. A view of part of the outer circumference of the vessel wall showing **haemorrhage** into the **inner-third of the media** (arrows) as well as congested adventitial blood vessels together with **adventitial haemorrhage** (arrow). Case no. 38 (Haematoxylin and Eosin x 100). **AH** = adventitial haemorrhage. **VC** = vascular congestion. **H** = haemorrhage.

Intimal tears extending to or along the Internal Elastic Lamina (continuation)

The **left internal carotid artery** showed single involvement in 4 subjects (8.0%) (Fig. 7.3).

No multiple involvement of this latter artery ('multiple' meaning involvement by more than one lesion of the particular type) was noted in any of the subjects (0%). The **left external carotid artery** showed single involvement in 2 subjects (4%) and multiple involvement in 1 subject (2%), while the **right common carotid artery** showed single involvement in 4 subjects (8%) (Figs. 7.15a and 7.15b).

Multiple involvement was noted in this latter artery in 2 subjects (4%). The **right internal carotid artery** showed single involvement in 3 subjects (6%) and multiple involvement in 1 subject (2%), and the **right external carotid artery** showed single involvement in 2 subjects (4%) and multiple involvement in 1 subject (2%).

Summary of Intimal Tears extending to or along the Internal Elastic Lamina

In summary, in the 50 subjects examined, the incidence of positive findings was as follows:

Left common carotid artery: single tears = 10%
multiple tears = 6%
Left internal carotid artery: single tears = 8%
multiple tears = 0%
Left external carotid artery: single tears = 4%

multiple tears = 2%

Right common carotid artery: single tears = 8%

multiple tears = 4%

Right internal carotid artery: single tears = 6%

multiple tears = 2%

Right external carotid artery: single tears = 4%

multiple tears = 2%

Analysis of Findings

		Single	Multiple
		%	%
Left:	**Common carotid artery**	**10**	**6**
	Internal carotid artery	**8**	**0**
	External carotid artery	**4**	**2**
		22%	**8%**
Right:	**Common carotid artery**	**8**	**4**
	Internal carotid artery	**6**	**2**
	External carotid artery	**4**	**2**
		18%	**8%**

Clearly, in this instance, single tears are more common than multiple tears, being almost three times as common on the left and twice as common on the right.

Thus, in comparison with **superficial intimal tears**, the incidence of **intimal tears extending to or along the internal elastic lamina** appears to be less than the superficial, i.e. the incidence of deeper **tears** is generally less than the more superficial ones. This would accord with the circumstances in that greater force would be required to produce a deep breach rather than a more superficial one. Furthermore, extension of the **tear extending along the internal elastic lamina** would appear to indicate that the **internal lamina** acts as a limiting plate preventing further breach of the vessel wall. However, where the forces applied are great enough to overcome the resistance of the **internal lamina**, further breaching would then occur.

3. <u>Intimo-Medial Tears</u>

These, as previously indicated, refer to those **tears** which extend through the full-thickness of the **intima** to involve the **media**. These **tears** were further subdivided according to depth/extent of involvement of the **media** into:

a. **intima to the inner-third of the media;**
b. **intima to the middle-third of the media;**
c. **intima to the outer-third of the media.**

With regard to arterial involvement by **intimo-medial tears**, the **left common carotid artery** showed no single **tears** extending to the **inner-third of the media** in any of the subjects (0%) but multiple **tears** in 2 subjects (4%), no single **tears** extending to the **middle-third of the media** again in any of the subjects (0%) but multiple **tears** in 3 subjects (6%), and no single **tears** extending to the **outer-third of the media** in any of the subjects (0%) but multiple **tears** in 1 subject (2%). The **left**

internal carotid artery showed no involvement by **intimo-medial tears** either single or multiple extending to **inner, middle or outer-thirds of the media** in any of the subjects (0%), the **left external carotid artery** similarly showed no involvement in any of the subjects except for a single **intimo-medial tear** extending to the **outer-third of the media** in 1 subject (2%).

The **right common carotid artery** showed a single **intimo-medial tear** extending to the **inner-third of the media** in 4 subjects (8%) and multiple **tears** in 1 subject (2%) (Figs. 7.15a and 7.15b).

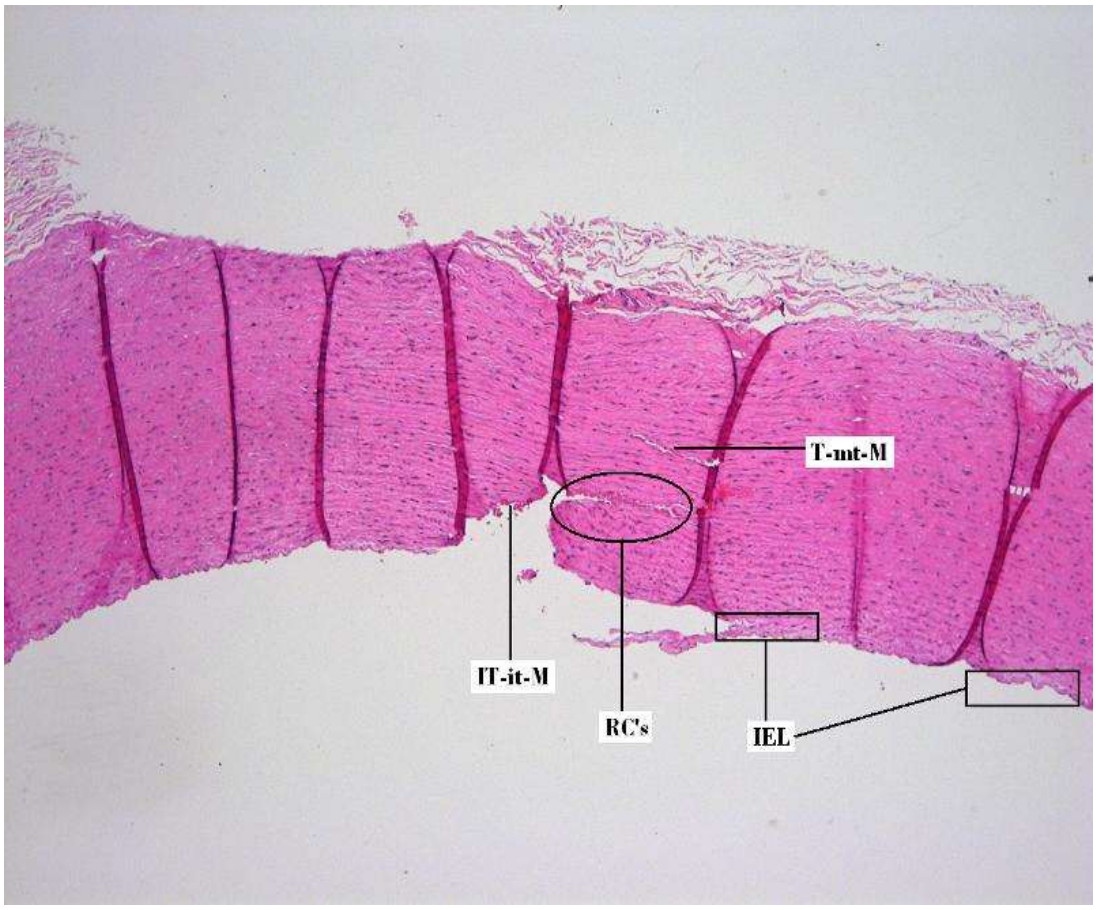


Fig. 7.15a. Right common carotid artery. An **intimal tear** extending through to the **inner-third of the media** is noted. Scattered red cells are noted within the **tear**. A separate **tear** with **haemorrhage** involving the **middle-third of the media** is, in

addition, noted although better seen in the higher magnification in **Fig. 7.15b**. The **intima** has been avulsed. Case no. 33 (Haematoxylin and Eosin x 50). **IT-it-M** = intimal tear extending to the inner-third of the media. **T-mt-M** = tear of the middle-third of the media. **RC's** = red blood cells. **IEL** = internal elastic lamina.

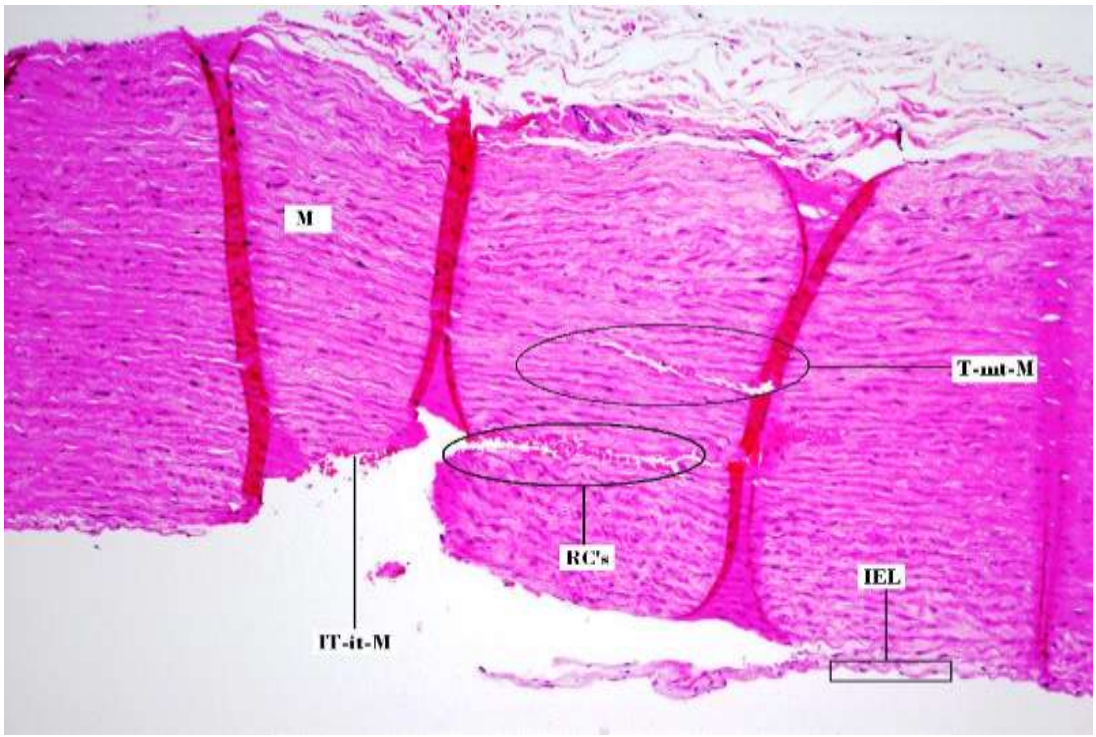


Fig. 7.15b. Right common carotid artery. A high-power view of the **intimal tear** extending to the **inner-third of the media** is noted. The **tear** involving the **middle-third of the media** is better seen at this greater magnification. Case no. 33 (Haematoxylin and Eosin x 100). **T-mt-M** = tear of the middle third of the media. **IT-it-M** = intimal tear extending to the inner-third of the media. **IEL**= internal elastic lamina. **RC's** = red blood cells. **M** = media.

With regard to deeper arterial damage, multiple **intimo-medial tears** extending to the **middle-third of the media** were noted in only 1 subject (2%), but no **intimo-**

medial tears extending to the **outer-third of the media** in any of the subjects (0%) were noted. The **right internal carotid artery,** on the other hand, showed a single **intimo-medial tear** extending to the **inner-third** and **outer-third of the media** in 1 subject each (2%) but no multiple involvement extending to **inner-, middle- or outer-thirds of the media** in any of the subjects (0%). The **right external carotid artery** showed a single **intimo-medial tear** extending to the **inner- and middle-thirds of the media** in 1 subject (2%) each, multiple **intimo-medial tears** extending to the **middle-third of the media** in 1 subject (2%) but no multiple **intimo-medial tears** extending to either the **inner- or outer-thirds of the media** in any of the subjects (0%).

Single **tears** extending to the **middle- and outer-thirds of the media** in 2 (4%) and 1 subjects (2%) respectively were noted (Figs.7.8a and 7.8b). It is noteworthy that these were of low incidence, being, therefore, uncommon.

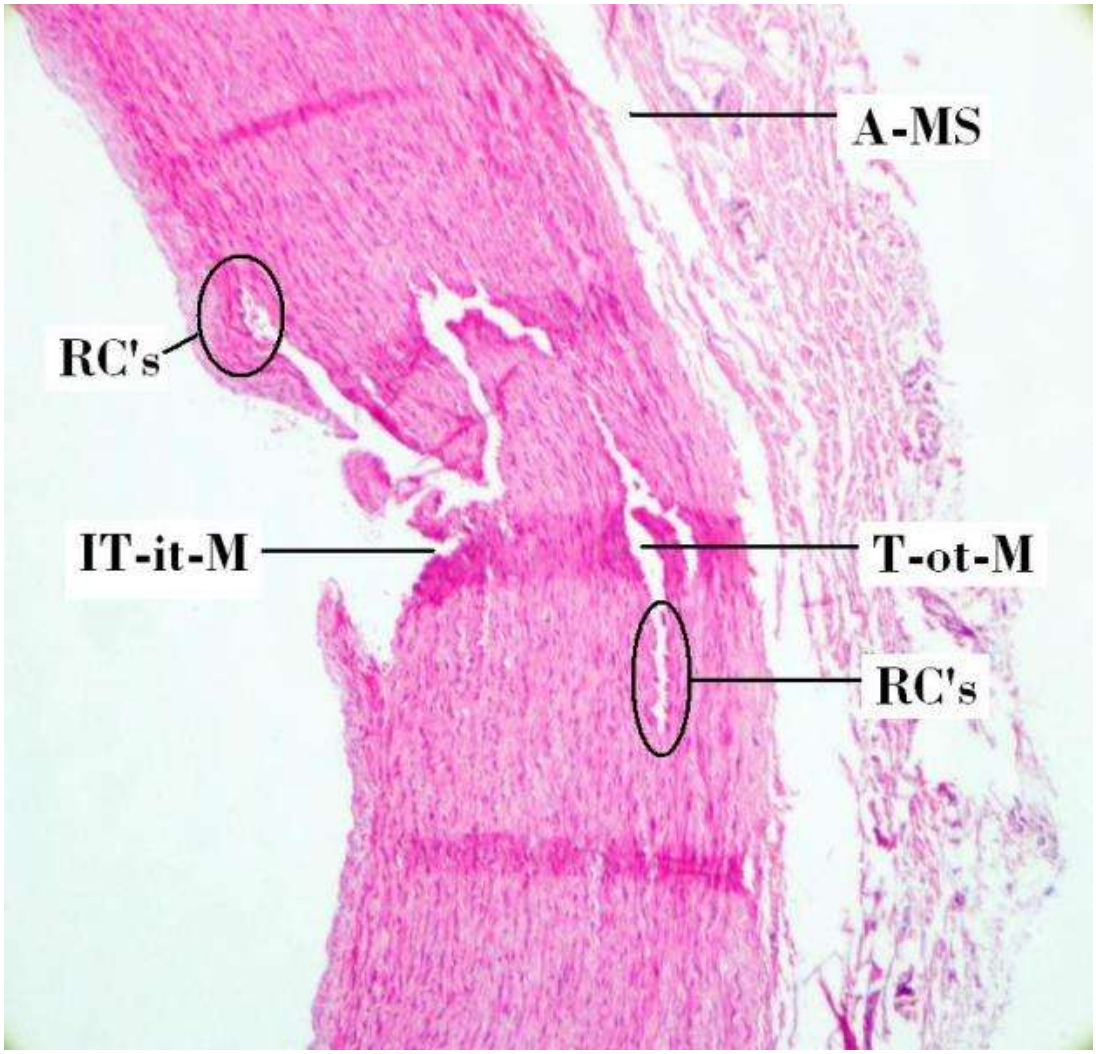


Fig. 7.8a. Right common carotid artery. An **intimal tear** with scattered red cells is seen extending to the **inner-third of the media**. The intima has been avulsed. **Tears** of the **middle- and outer-thirds of the media** are, in addition, noted as well as **adventitio-medial separation**. Case no. 21 (Haematoxylin and Eosin x 50). **IT-it-M** = intimal tear extending to the inner-third of the media. **T-ot-M =** tear of the outer-third of the media. **A-MS** = adventitio-medial separation. **RC's** = red blood cells.

The victim was of average height and weight (Table I). While there was no indication in the history of whether a jump from a height or a jerk of the ligature had occurred to account for the extensive nature of the arterial wall disruption. In this

case the ligature consisted of a **boot lace**. This conforms to the engineering principle that, where a narrow object is applied, it results in the focal application of force rather than a diffusion of force by a wider ligature such as a tie or a cloth.

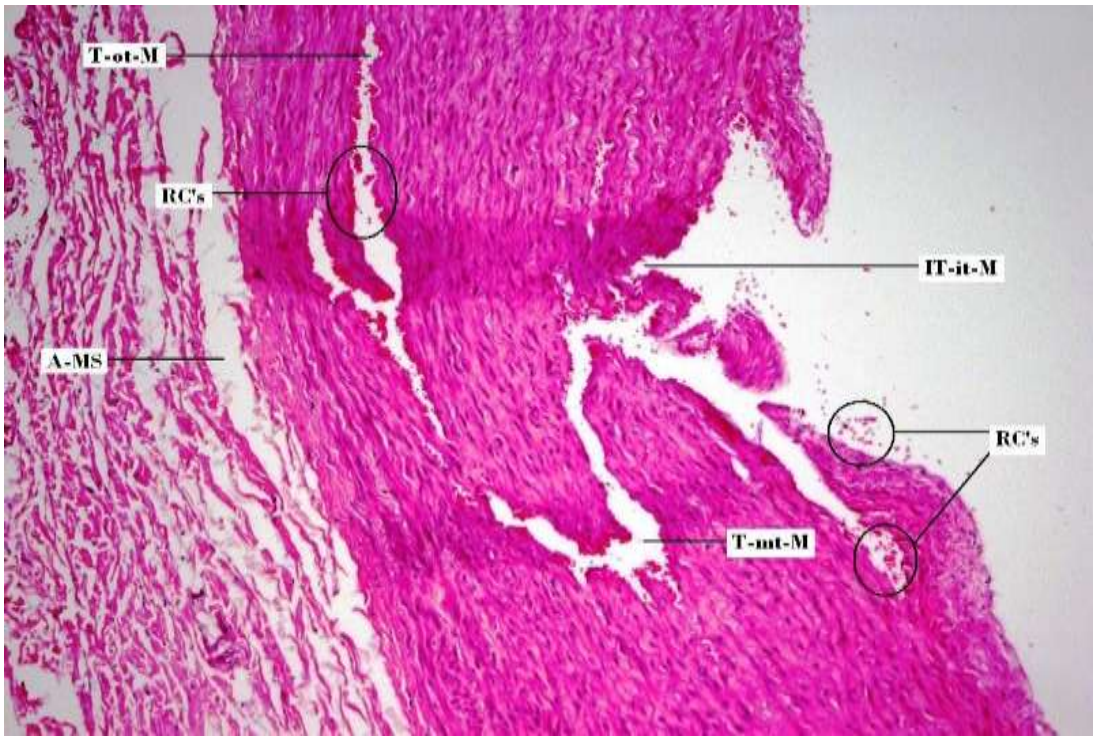


Fig. 7. 8b. Right common carotid artery. A high-power view of the **tear** seen extending from the **intima** to the **inner-third of the media** with infiltration of red cells better seen at the higher magnification. **Tears** of the **middle- and outer-thirds of the media** are, in addition, noted with infiltration by erythrocytes. The **adventitio-medial separation** shows, at this magnification, the residual tissue bridges so characteristic of a tearing or separation effect. Case no. 21 (Haematoxylin and Eosin x 100). **IT-it-M** = intimal tear extending to the inner-third of the media. **T-ot-M** = tear of the outer-third of the media. **T-mt-M** = tear of the middle-third of the media. **A-MS** = adventitio-medial separation. **RC's** = red blood cells.

Summary of Intimo-Medial Tears

In summary, in the 50 subjects examined, the incidence of positive findings was as follows:

a. Intimal Tears extending to the inner-third of the Media

Left common carotid artery: single tears = 0%
multiple tears = 4%

Left internal carotid artery: single tears = 0%
multiple tears = 0%

Left external carotid artery: single tears = 0%
multiple tears = 0%

Right common carotid artery: single tears = 8%
multiple tears = 2%

Right internal carotid artery: single tears = 2%
multiple tears = 0%

Right external carotid artery: single tears = 2%
multiple tears = 0%

Analysis of Findings

	Single	Multiple
	%	%
Left: Common carotid artery	**0**	**4**
Internal carotid artery	**0**	**0**
External carotid artery	**0**	**0**
	0%	**4%**
Right: Common carotid artery	**8**	**2**
Internal carotid artery	**2**	**0**
External carotid artery	**2**	**0**
	12%	**2%**

An increased incidence is seen on the right. The possible reasons for this are discussed at the end of the section..

b. Intimal Tears extending to the middle-third of the Media

Left common carotid artery: single tears = 0%

multiple tears = 6%

Left internal carotid artery: single tears = 0%

multiple tears = 0%

Left external carotid artery: single tears = 0%

multiple tears = 0%

Right common carotid artery: single tears = 4%

multiple tears = 2%

Right internal carotid artery: single tears = 0%

multiple tears = 0%

Right external carotid artery: single tears = 2%

multiple tears = 2%

Analysis of Findings

		Single %	Multiple %
Left:	**Common carotid artery**	**0**	**6**
	Internal carotid artery	**0**	**0**
	External carotid artery	**0**	**0**
		0%	**6%**
Right:	**Common carotid artery**	**4**	**2**
	Internal carotid artery	**0**	**0**
	External carotid artery	**2**	**2**
		6%	**4%**

Once again, an increased incidence on the right is noted.

c. Intimal Tears extending to the outer-third of the Media

Left common carotid artery: single tears = 0%

multiple tears = 2%

Left internal carotid artery: single tears = 0%

multiple tears = 0%

Left external carotid artery: single tears = 2%

multiple tears = 0%

Right common carotid artery: single tears = 2%

multiple tears = 0%

Right internal carotid artery: single tears = 2%

multiple tears = 0%

Right external carotid artery: single tears = 0%

multiple tears = 0%

Analysis of Findings

	Single %	Multiple %
Left: Common carotid artery	**0**	**2**
Internal carotid artery	**0**	**0**
External carotid artery	**2**	**0**
	2%	**2%**
Right: Common carotid artery	**2**	**0**

Internal carotid artery	**2**	**0**
External carotid artery	**0**	**0**
	4%	**0%**

Putting all the former analyses (a, b and c) together, two findings emerge:

i) in contrast to the findings with regard to **superficial intimal tears** and **intimal tears extending to or along the internal elastic lamina** where an equal or almost equal incidence was found between left-sided and right-sided lesions, **intimo-medial tears** extending to either the **inner-third, middle-third or outer-third of the media** showed a slight right-sided preponderance, and;

ii) a lesser incidence of damage occurs as soon as the **media** is reached.

It is suggested that two different factors may account for these findings:

i) the equal or almost equal incidence of lesions of the **intima** may be attributed to an equivalent force acting from within the lumen of the vessel. This force consists of the systemic head of blood pressure acting equally against the innermost layer of the vessel wall, i.e. the **intima**. Put more simply, the systemic head of pressure, acting from within, impacts against the **intima** at the site of vessel constriction due to the externally applied ligature, and;

ii) the relative resistance of the **media** to disruptive damage as a consequence of its laminated muscular structure.

It is thus suggested that the systemic head of pressure itself acts centrifugally, applying force outwards in all directions. Bearing in mind that fluid itself is incompressible, the force exerted would tend to distribute the pressure not only outwards at the point of vessel constriction but equally along the innermost layer of the vessel wall, i.e. the **intima**, resulting in an equal incidence of left-sided and right-sided disruptive damage. This issue is fully explored in the Discussion chapter.

4. Tears of the Media (muscular layer)

This refers to those **tears** involving the middle layer of the vessel wall. However, due to the multiplicity of disruptive lesions of the **intima**, **media** or **adventitia** encountered as one proceeded along the circumference of the vessel wall, the photomicrographs may show, in addition to the illustrative lesions of the **media**, lesions of the **intima** or **adventitia**. These latter lesions have no bearing on the classification of the **tears of the media** as each lesion of the vessel wall has been accorded its individual category and classification.

A single **tear** of the **inner-third of the media** involving the **left common carotid artery** was noted in 1 subject (2%) as was a single **tear** of the **outer-third of the media** in 1 subject (2%). No single **tears** of the **middle-third of the media** were noted in this artery.

Multiple **tears** of the **inner-, middle- and outer-thirds of the media** of the **left common carotid artery** were noted involving the **inner-third** in 2 subjects (4%), the **middle-third** in 3 subjects (6%), and the **outer-third** in 3 subjects (6%) (Figs.7.4 and 7.7).

By contrast, the **left internal carotid artery** showed a single **medial tear** involving the **inner-third of the media** in 2 subjects (4%), and multiple **medial tears** involving this layer of the vessel wall in 1 subject (2%) (Figs.7.9a and 7.9b).

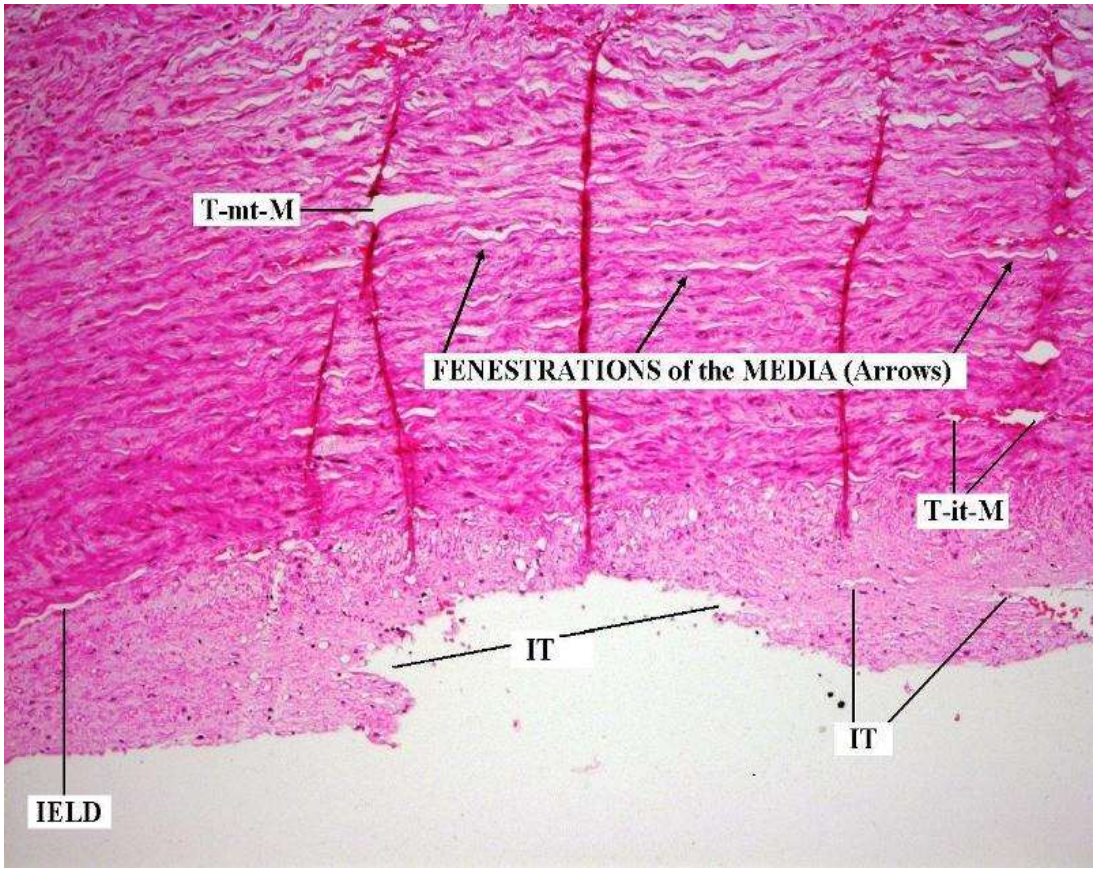


Fig. 7.9a. Left internal carotid artery. A view of the artery showing atheromatous **intimal tears** with partial avulsion of this layer of the vessel wall, **dehiscence** of the **internal elastic lamina** seen to the left of the field, multiple small **tears** with haemorrhage involving the **inner- and middle-thirds of the media** and **multiple fenestrations of the media** (arrows). Case no. 6 (Haematoxylin and Eosin x 50). **IT** = intimal tear. **IELD** = internal elastic lamina dehiscence. **T-mt-M =** tear of the middle-third of the media.

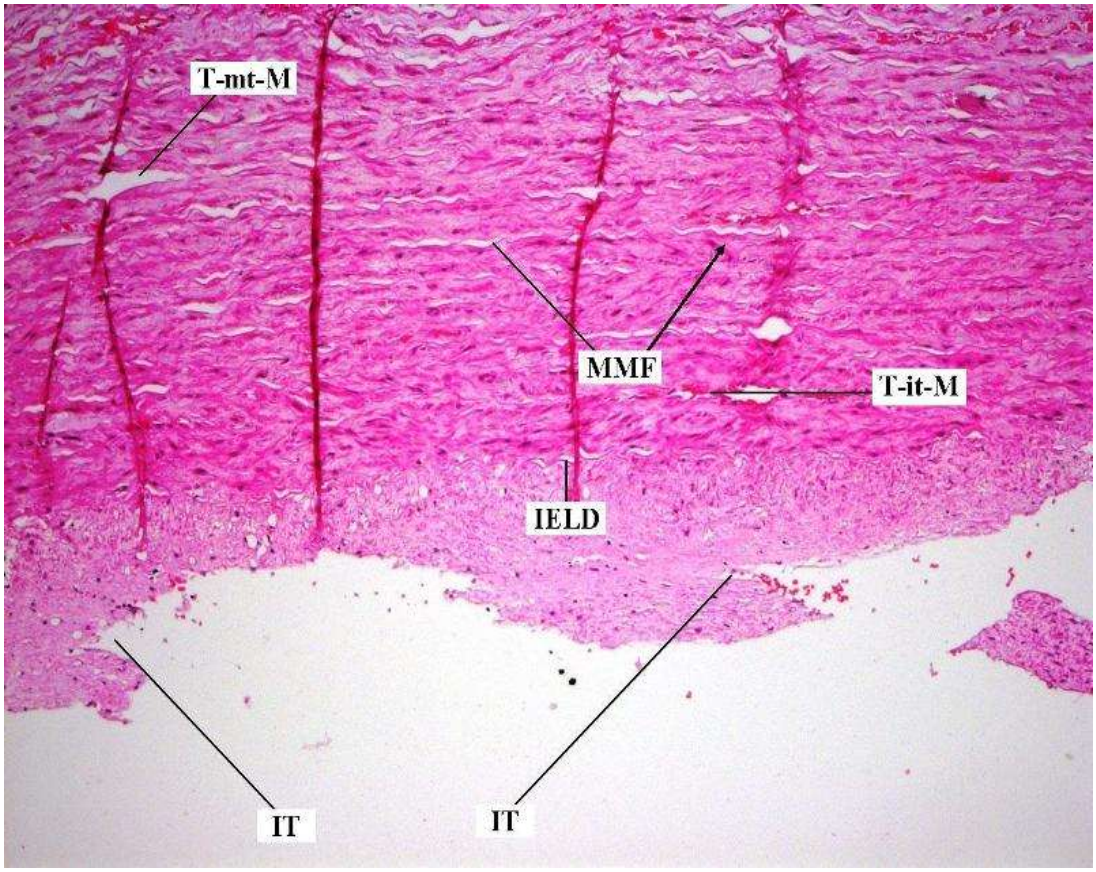


Fig. 7. 9b. Left internal carotid artery. A high-power view of the artery showing the **intimal atheromatous tears**, **dehiscence** of the **internal elastic lamina**, small **tears** with haemorrhage involving the **inner- and middle-thirds of the media** and **multiple fenestrations of the media** (arrow). Case no. 6 (Haematoxylin and Eosin x 100).

IT = intimal tear. **IELD** = internal elastic lamina dehiscence. **T-it-M** = tear of the inner-third of the media. **MMF** = multiple fenestrations of the media.

Single **tears of the media** involving the **middle- and outer-thirds** were noted in 1 subject (2%) each, and multiple **tears** of the **middle- and outer-thirds** in 1 subject (2%) and in 2 subjects (4%) each, respectively (Figs. 10a and 7.10b). As previously indicated in the introductory remarks with regard to **tears of the media**, where the illustrative photomicrographs show disruptive lesions of the **intima** or **adventitia** in addition to the **tears of the media** encountered, these **intimal** or **adventitial** lesions have been classified and included in their separate categories. The presence of lesions of other layers of the vessel wall in addition to **tears of the media** emphasizes the sheer multiplicity of lesions involving multiple layers of the vessel wall as one proceeded along the circumference.

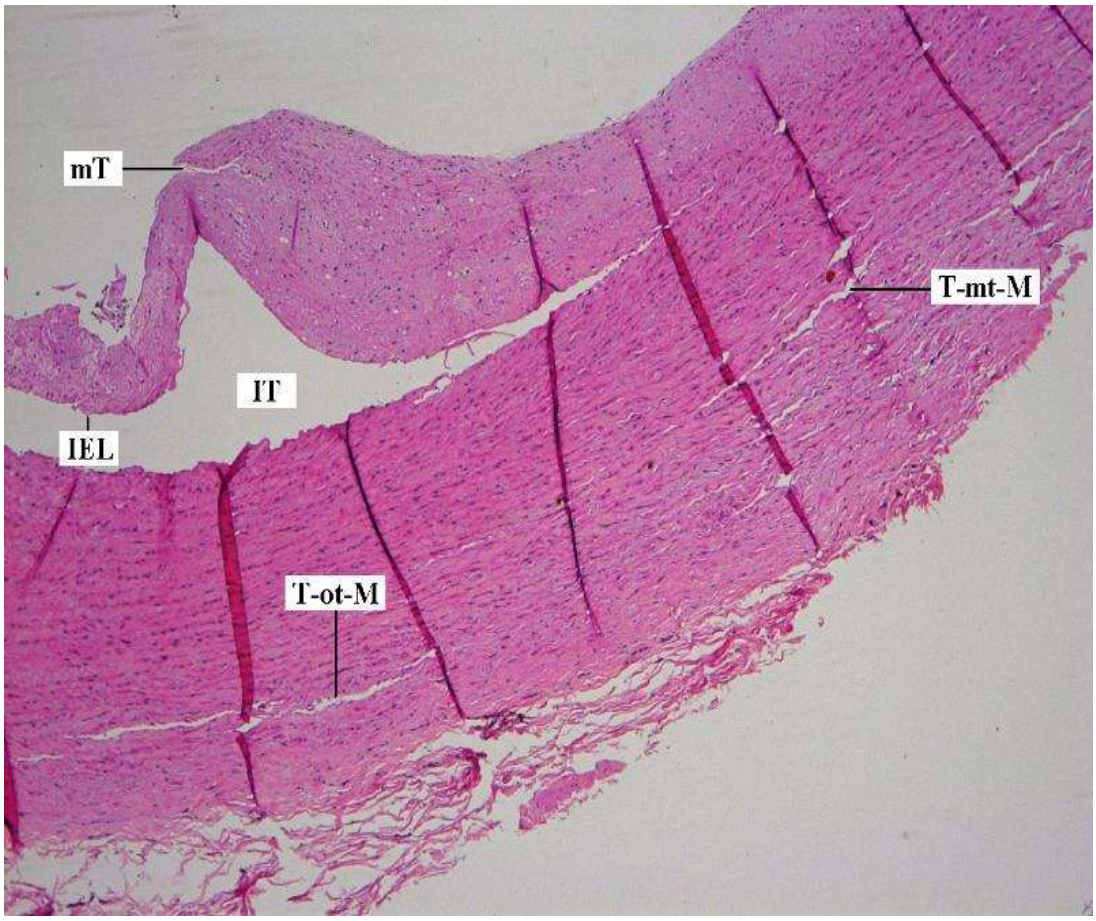


Fig. 7.10a. Left internal carotid artery. A view of the artery showing, in addition to an **intimal tear extending along the internal elastic lamina** with elevation of this segment of the **intima** into the lumen of the vessel, multiple **tears of the middle- and outer-thirds of the media**. These latter **tears of the media** extend through and beyond the areas of 'wrinkling' produced by tissue processing of a tube-like structure, suggesting the **tears** to be primary in origin preceding the secondary and subsequent processing artefact. A **microtear** of the **intima** is, in addition, noted. Case no. 6 (Haematoxylin and Eosin x 50). **IT** = intimal tear. **IEL =** internal elastic lamina. **T-mt-M** = tear of the middle-third of the media **T-ot-M** = tear of the outer-third of the media. **mT** = microtear of the intima.

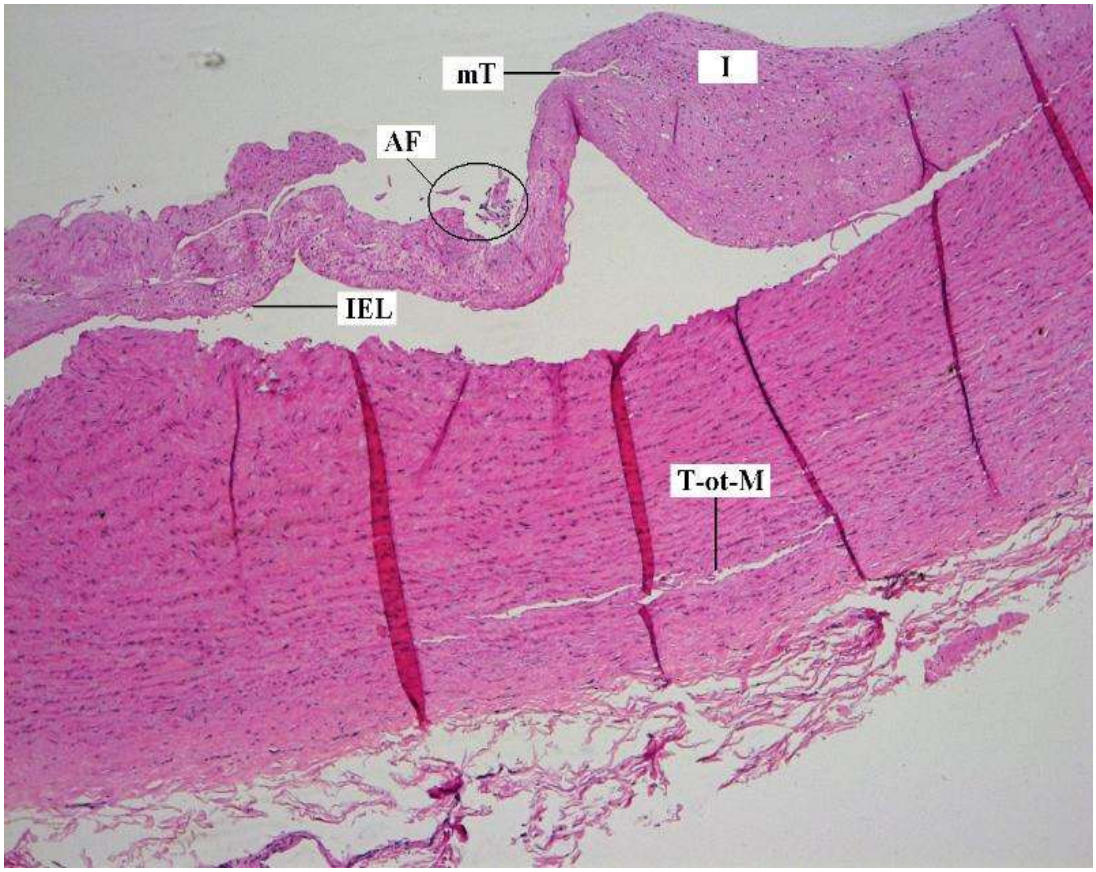


Fig. 7.10b. Left internal carotid artery. An additional view of the artery depicted in **Fig. 7.10a.** further along the circumference of the wall. In addition to the **intimal tear extending along the internal elastic lamina** with elevation of the **intima** into the lumen of the vessel seen in **Fig. 7.10a.**, a **tear of the outer-third of the media** is seen. A **microtear** of the **intima** is, in addition, noted. Case no. 6 (Haematoxylin and Eosin x 50). **I** = intima. **IEL** = internal elastic lamina. **T-ot-M** = tear of the outer-third of the media (muscular layer). **mT** = microtear of the intima. **AF** = avulsed fragment.

The **left external carotid artery**, showing a single **tear** of the **inner-third of the media** in 1 subject (2%), a single tear of the **middle-third of the media** in 2 subjects (4%), no multiple tears of the **inner-third of the media** in any of the subjects (0%),

but multiple tears of the **middle- and outer-thirds of the media** in 2 subjects (4%) each, respectively (Figs.7.11).

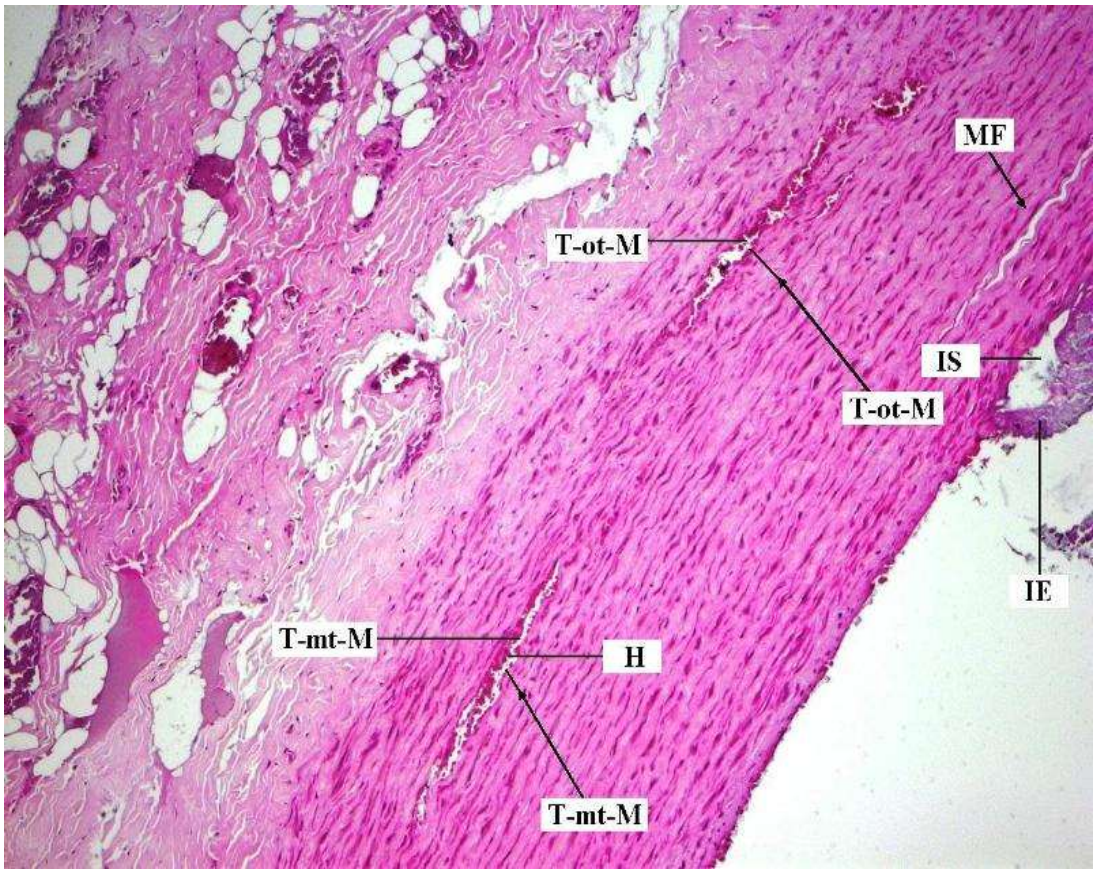


Fig. 7. 11. Left external carotid artery. Multiple **tears** with **haemorrhage** involving the **middle- and outer-thirds of the media** are noted (arrows) together with a **fenestration of the media** (arrow) and **intimal separation and elevation**. Case no. 33 (Haematoxylin and Eosin x 100). **IS** = intimal split. **IE** = intimal elevation. **T-ot-M** = tear of the outer-third of the media (muscular layer). **T-mt-M** = tear of the middle-third of the media. **MF** = fenestration of the media. **H** = haemorrhage.

The **right common carotid artery** showed a single **tear** of the **inner-third of the media** in 1 subject (2%), a single **tear** of the **middle-third of the media** in 4 subjects (8%), and a single **tear** of the **outer-third of the media** in 4 subjects (8%). Multiple **tears** of the **inner-, middle- and outer-thirds of the media** were noted in 1 subject (2%), 3 subjects (6%), and 3 subjects (6%) each, respectively (Figs. 7.8, 7.12a, 7.12b and 7.13).

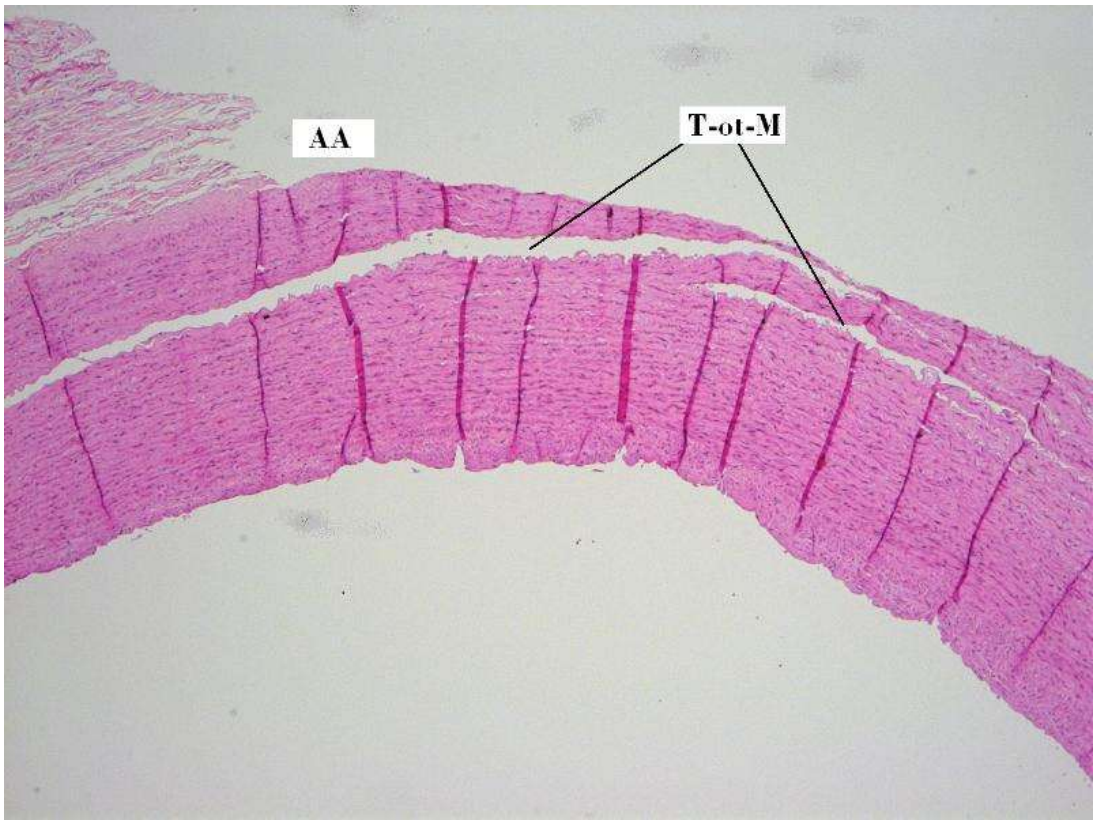


Fig. 7.12a. Right common carotid artery. Two **tears** of the **outer-third of the media** are noted. The larger **outer-third tear** appears to extend more deeply to the **middle-third of the media**, better seen at the higher magnification in Fig.7.12b. The **adventitial layer** overlying the **tears** has been completely avulsed, a feature not seen

in the control cases. Case no. 10 (Haematoxylin and Eosin x 50).**T-ot-M** = tear of the outer-third of the media. **AA** = adventitial avulsion.

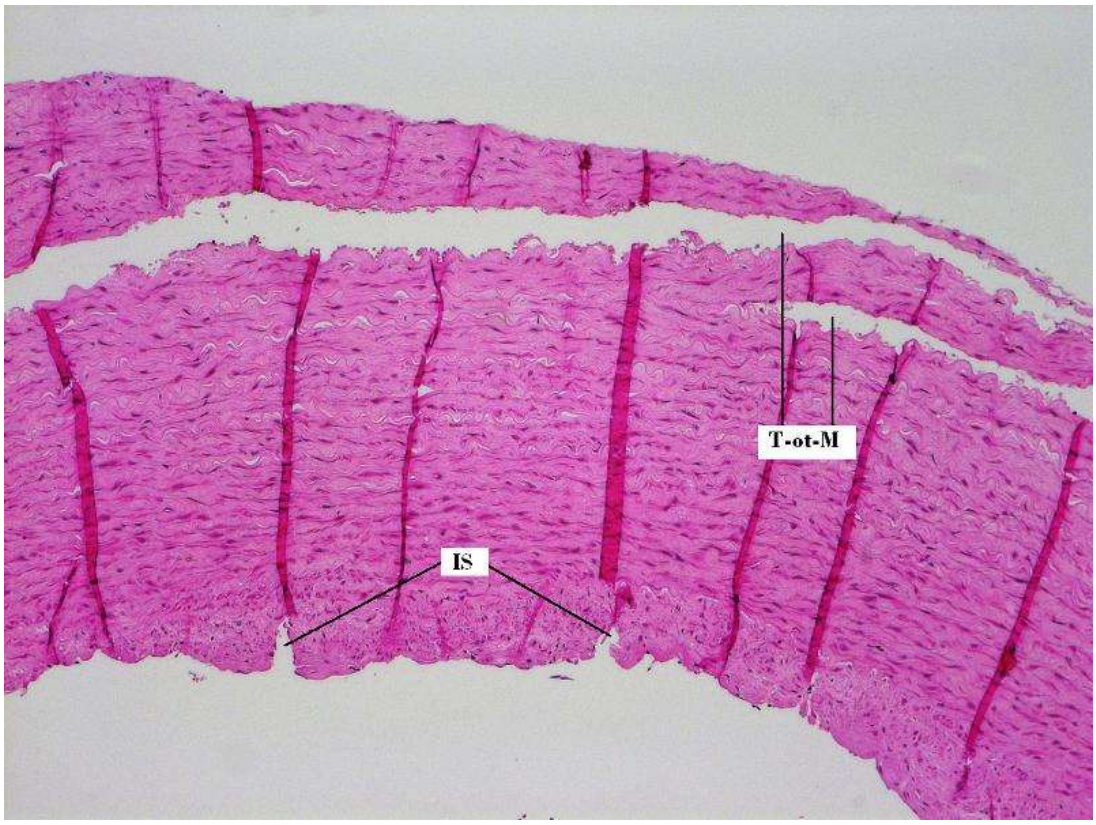


Fig. 7. 12b. Right common carotid artery. A high-power view of the two **tears** of the **outer-third of the media** is noted. The larger **outer-third tear** appears to extend more deeply towards the **middle-third of the media**.The **adventitia** has been completely avulsed, a feature not seen in the control cases. The intimal separations seen appear to be a processing artefact occurring in a tubular structure in view of their position directly beneath the wrinkling and folding of the **media**. Case no. 10 (Haematoxylin and Eosin x 100). **T-ot-M** = tear of the outer-third of the media. **IS** = intimal separation.

While the person was of average height and weight as in case. No. 21 (Fig. 7.8b; Table I), the encircling ligature consisted of a **nylon cord**. In other words, the

encircling ligature was narrow, suggesting a more focal application of force rather than a diffusion of force, which would result in greater damage to the vessel wall.

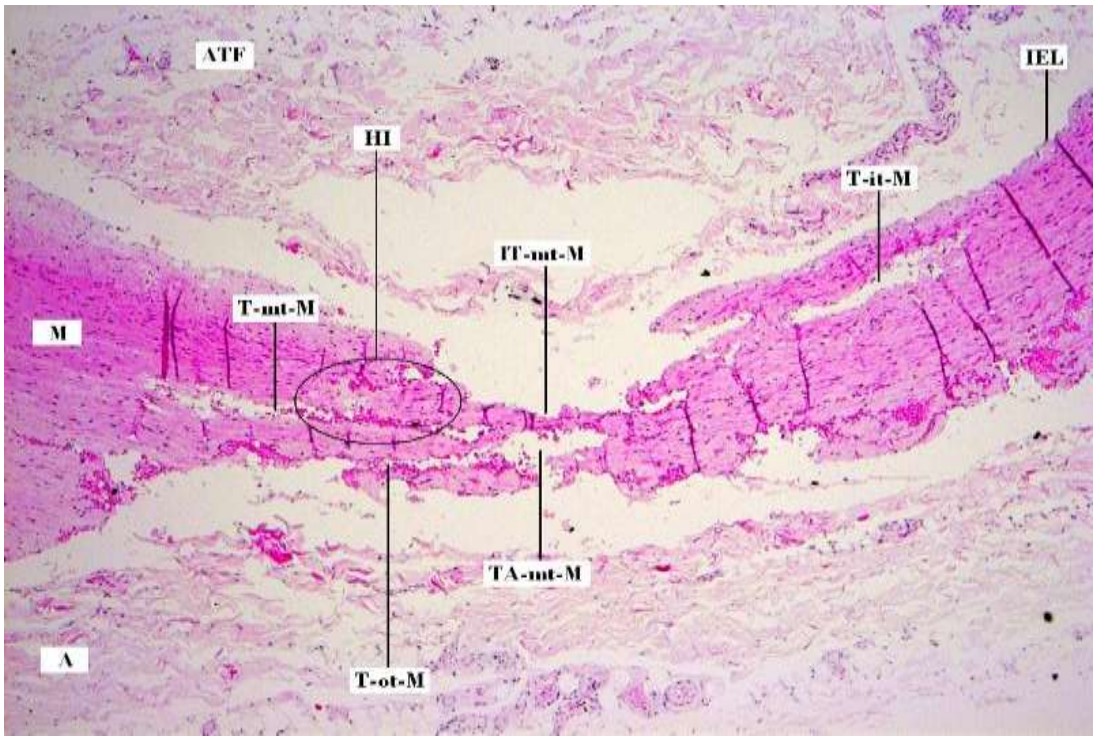


Fig. 7. 13. Right common carotid artery. Multiple **tears** of the **inner-, middle- and outer-thirds of the media** with haemorrhagic infiltration are noted. In addition, haemorrhagic **intima** to **tears of the middle-third of the media** and **adventitia** to **tears of the middle-third of the media** are together with **haemorrhage**. Case no. 22 (Haematoxylin and Eosin x 50). **IT-mt-M** = intimal tear extending to the middle-third of the media. **T-it-M** = tear of the inner-third of the media. **T-mt-M** = tear of the middle-third of the media. **T-ot-M** = tear of the outer-third of the media. **TA-mt-M** = tear of the adventitia extending to the middle-third of the media. **HI** = haemorrhagic infiltration. **I** = intima. **M** = media. **A** = adventitia. **ATF** = avulsed tissue fragments.

The **right internal carotid artery** showed single **tears** of the **inner- and middle-thirds of the media** in 1 subject (2%) each, respectively (Fig. 7.14).

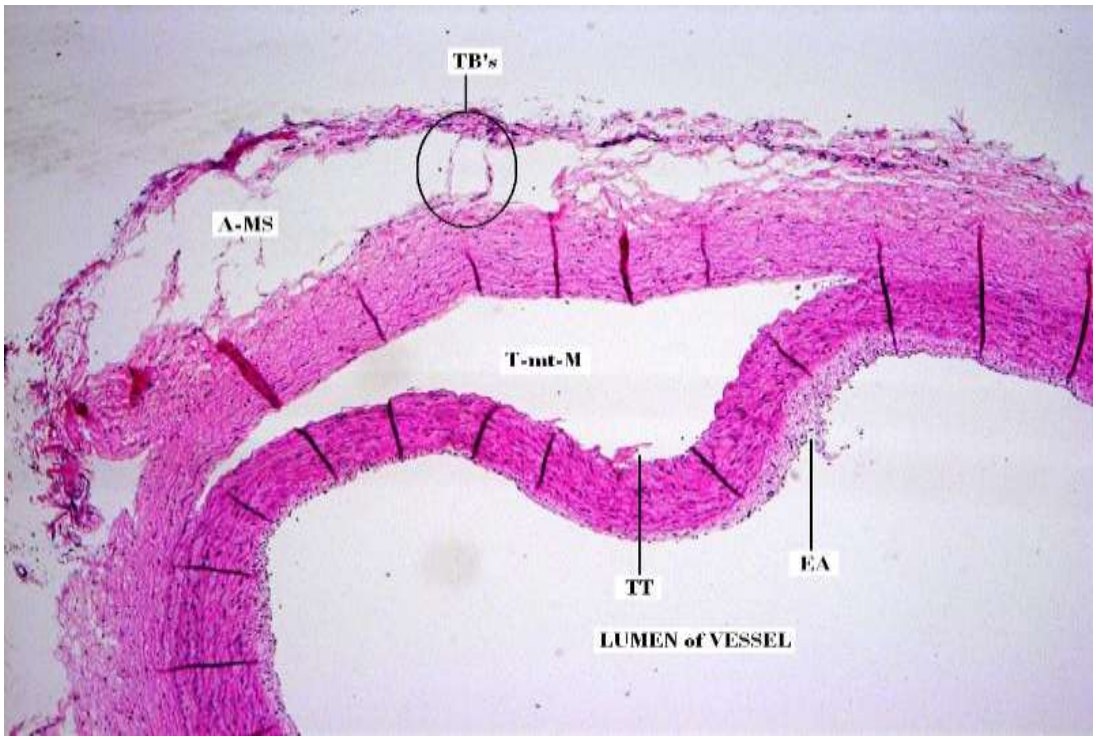


Fig. 7. 14. Right internal carotid artery. A **tear** of the **middle-third of the media** showing marked separation of the **media** with tissue tags protruding into the area of separation. **Endothelial avulsion** at one point beneath the area of separation is noted and extending into the lumen of the vessel. The overlying **adventitia** is widely separated from the underlying **media** with residual tissue bridges being present. Case no. 8 (Haematoxylin and Eosin x 50). **T-mt-M** = tear of the middle-third of the media. **TT** = tissue tags. **A-MS** = adventitial- media separation. **TB's** = tissue bridges. **EA** = endothelial avulsion.

No single **tears** of the **outer-third of the media** were noted in any of the subjects (0%). Multiple **tears** of the **inner-, middle- and outer-thirds of the media** were noted in 1 (2%), 2 (4%), and 4 subjects (8%) each, respectively. The **right external carotid artery** showed no single **tears** of the **inner-third of the media** in any of the subjects (0%), but single **tears** of the **middle- and outer-thirds of the media** were noted in 1 (2%) and 2 (4%) subjects, respectively. Multiple **tears** of the **inner-, middle- and outer-thirds** of this layer of the vessel wall were noted in 1 subject (2%), 2 subjects (4%) and 2 subjects (4%) each, respectively.

Summary of Tears of the Media (muscular layer)

In summary, of the 50 subjects examined, the incidence of positive findings was as follows:

a. Tears of the inner-third of the Media (To determine the number of cases:- divide **100** by **2**).

Left common carotid artery: single tears = 2% = 1
multiple tears = 4% = 2

Left internal carotid artery: single tears = 4% = 2
multiple tears = 2% = 1

Left external carotid artery: single tears = 2% = 1
multiple tears = 0% = 0

Right common carotid artery: single tears = 2% = 1
multiple tears = 2% = 1

Right internal carotid artery: single tears = 2% = 1

multiple tears = 2% = 1

Right external carotid artery: single tears = 0%

multiple tears = 2%

Analysis of Findings

		Single %	Multiple %
Left:	**Common carotid artery**	**2**	**4**
	Internal carotid artery	**4**	**2**
	External carotid artery	**2**	**0**
		8%	**6%**
Right:	**Common carotid artery**	**2**	**2**
	Internal carotid artery	**2**	**2**
	External carotid artery	**0**	**2**
		4%	**6%**

b. Tears of the middle-third of the Media

Left common carotid artery: single tears = 0%

multiple tears = 6%

Left internal carotid artery: single tears = 2%

multiple tears = 2%

Left external carotid artery: single tears = 4%

multiple tears = 4%

Right common carotid artery: single tears = 8%

multiple tears = 6%

Right internal carotid artery: single tears = 2%

multiple tears = 4%

Right external carotid artery: single tears = 2%

multiple tears = 4%

Analysis of Findings

		Single	Multiple
		%	%
Left:	**Common carotid artery**	**0**	**6**
	Internal carotid artery	**2**	**2**
	External carotid artery	**4**	**4**
		6%	**12%**
Right:	**Common carotid artery**	**8**	**6**
	Internal carotid artery	**2**	**4**
	External carotid artery	**2**	**4**
		12%	**14%**

Comment: While the incidence of multiple **tears of the middle-third of the media** was double that of single **tears** on the left, the incidence was almost equal on the right. Moreover, the incidence of multiple **tears** on the left equalled that of single **tears** on the right and almost equalled that of multiple **tears** on the right. In other words, other than the incidence of single **tears** on the left, the incidence was equal or almost equal overall.

c. Tears of the outer-third of the Media

Left common carotid artery: single tears = 2%
multiple tears = 6%

Left internal carotid artery: single tears = 2%
multiple tears = 4%

Left external carotid artery: single tears = 0%
multiple tears = 4%

Right common carotid artery: single tears = 8%
multiple tears = 6%

Right internal carotid artery: single tears = 0%
multiple tears = 8%

Right external carotid artery: single tears = 4%
multiple tears = 4%

Analysis of Findings

		Single	Multiple
		%	%
Left:	**Common carotid artery**	**2**	**6**
	Internal carotid artery	**2**	**4**
	External carotid artery	**0**	**4**
		4%	**14%**
Right:	**Common carotid artery**	**8**	**6**
	Internal carotid artery	**0**	**8**
	External carotid artery	**4**	**4**
		12%	**18%**

Comment: As with **tears of the middle-third of the media**, a greater incidence of **tears of the outer-third of the media** was noted on the right as compared to the left. Similarly, as with **tears of the middle-third of the media**, the incidence of multiple tears on the left almost mirrored the incidence of **tears**, both single and multiple, on the right.

5. Tears of the adjacent Adventitio-Medial layers of the Vessel Wall

As indicated, this refers to those **tears** which extend from or through the outermost layer of the vessel wall, i.e. from the **adventitia** to involve the underlying **media** or muscular (middle layer) of the vessel wall. In accordance with the classification adopted with regard to all other disruptive arterial lesions noted, this category refers specifically to **tears of the adjacent adventitio-medial layer of the vessel wall**. As previously indicated, due to multiplicity of lesions encountered as one proceeded circumferentially along the vessel wall, these lesions were then accorded their specific categories. In addition, the reversal of direction from outwards to inwards in classifying those lesions extending from the **adventitia** to the **media** is intentional, as logic dictates that an encircling compressive ligature would exert pressure first, on the outermost layer of the vessel wall. A greater amount of force would then be expected to extend a **tear** of the **adventitia** to its immediately underlying layer, i.e. the **media**. However, in keeping with the general trend of direction in classifying the lesions observed, regardless of the direction of compressive forces exerted, the categorization begins with the **inner-third of the media** proceeding outwards.

An interesting ancillary point arises. Following vessel transection as, for example, after a penetrating incised wound, vascular constriction occurs as a result of contraction of the **media** or muscular layer of the vessel wall in a physiological attempt to stem the haemorrhage. In hanging, however, the artery is constricted by the external pressure applied, i.e. in a pathophysiological manner.

Neither single nor multiple **adventitio-medial tears** extending to involve the **inner- and middle-thirds** of the **media** of the **left common carotid artery** were

noted in any of the subjects (0%), but single **tears** of the **adventitia** extending to the **outer-third** of the **media** were noted in 2 subjects (4.%) and multiple **tears** in 1 subject (2%), respectively. The **left internal carotid artery** showed no involvement by **tears** involving this region of the vessel wall in any of the subjects (0%), while the **left external carotid artery**, on the other hand, showed neither single nor multiple **adventitial tears** extending to the **inner-third** of the **media** in any of the subjects (0%). Single and multiple **adventitial tears** extending to the **middle-third** of the **media** were noted in 1 subject (2%) each, single **tears** extending to the **outer-third** of the **media** in 2 subjects (4%), and multiple **tears** extending to the **outer-third** of the **media** in 1 subject (2%) (Fig. 7.16).

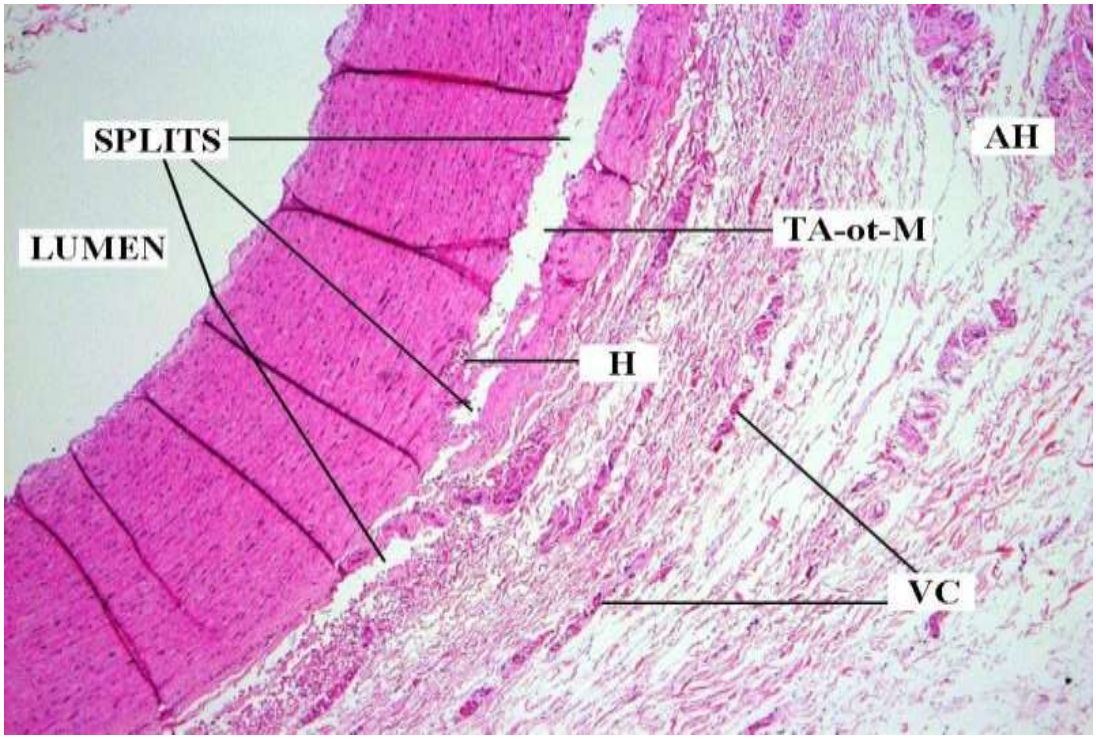


Fig. 7. 16. Left external carotid artery. A **tear** extending inwards from the **adventitia** to the **outer-third of the media** in which residual tissue tags are present is noted. **Adventitial haemorrhage** is present together with **vascular congestion**. The adventitial separation and **adventitia** to **outer-third media tear** appear to be in continuity suggesting causation by a common factor. Unfortunately, the type of ligature used in this case was unknown, although the tensile force of stretch of the artery would tend to produce tearing and separation of the tissues in the same plane. Case no. 29 (Haematoxylin and Eosin x 50). **TA-ot M** = tear of the adventitia to outer-third of the media. **H** = haemorrhage. **VC** = vascular congestion. **AH** = adventitial haemorrhage.

The **right common carotid artery** showed neither single nor multiple **adventitial tears** extending to involve the **inner-third** of the **media** in any of the subjects (0%),

but single and multiple **tears** extending to involve the **middle-third** of the **media** were noted in 1 subject (2%) each (Fig. 7.13).

Single **adventitial tears** extending to involve the **outer-third** of the **media** were noted in 2 subjects (4%) but no multiple **tears** extending to the **outer-third** of the **media** were noted in any of the subjects (0%). The **right internal carotid artery** proved interesting in that the distribution of **tears** involving these two layers of the vessel wall exactly mirrored that seen in the **left internal carotid artery** with no **tears** extending from the **adventitia** to involve either **inner-, middle- or outer-thirds** of the **media** in any of the subjects (0%). (A possible anatomical explanation for this could lie in the fact that due to the overlying nature of the **external carotid artery** with the consequent protected position of the two **internal carotid arteries** relative to their respective **external carotid arteries**, the latter provide a buffering and cushioning effect to the more internally situated vessels). The **right external carotid artery** showed a single **tear** extending to the **inner-third** of the **media** in 1 subject (2%), no multiple **tears** extending to the **inner-third** of the **media** in any of the subjects (0%), and no single **tears** extending to the **middle-third** of the **media** in any of the subjects (0%). Multiple involvement of the **middle-third** of the **media** was noted in 1 subject (2.0%) (Fig. 7.17).



Fig. 7. 17. Right external carotid artery. Adventitial tears extending to the **middle- and outer-thirds of the media** are noted together with **adventitial haemorrhage**. A separate **intima** to **middle-third media tear** with **haemorrhage** is present. The **adventitial tears** thus extend from the outer limit of the **media**, damaging its entire extent. Case no. 36 (Haematoxylin and Eosin x 50). **AT-mt-M** = adventitial tear extending to the middle-third of the media. **AT-ot-M** = adventitial tear extending to the outer-third of the media. **AH** = adventitial haemorrhage. **IT-mt-M** = intima to middle-third media tear. **TF** = tissue fragment. **H** = haemorrhage. **VC** = vascular congestion. **L** = lumen.

As in cases 10 and 21 (Figs. 7.8b and 7.12b), a narrow ligature, in this case a **nylon cord**, was noted, the latter being present over the upper-third of the neck on the right. As previously indicated in Table II, the position of the ligature lay over the upper-third of the neck in 45 (90%) of the 50 cases examined with 19 (38%) of the 50 cases

having a narrow ligature in the form of either a nylon cord, an insulated wire or a bootlace. Wide ligatures such as belts or scarves were only present in 8 (16%) of the 50 cases examined, while in 14 (28%) of the cases the nature of the ligature was unknown. Of the 36 known cases, therefore, narrow ligatures comprised 19 (52.8%) and, if this general trend was followed in the 14 unknown cases, the majority of subjects had hung themselves using a narrow ligature. This would account for the universal finding of some form of arterial damage in all the cases and would be in keeping with the engineering principle of force multiplication. The more focal the application of force (as with the points of stiletto heels of women's shoes where the focal pressure exerted is sufficient to indent a metal escalator) the greater is the concentration of force and pressure exerted. Soft, wide ligatures such as scarves, on the other hand, tend to produce faint, sometimes barely discernable, ligature impressions due to the softness of the material and the wider area of force application resulting in force diffusion.

Summary of Adventitio-Medial Tears

In summary, of the 50 subjects examined, the incidence of these findings was as follows:

a. Tears extending from the Adventitia to the inner-third of the Media

Left common carotid artery: single tears = 0%

multiple tears = 0%

Left internal carotid artery: single tears = 0%

multiple tears = 0%

<u>Left external carotid artery</u>: single tears = 0%

multiple tears = 0%

<u>Right common carotid artery</u>: single tears = 0%

multiple tears = 0%

<u>Right internal carotid artery</u>: single tears = 0%

multiple tears = 0%

<u>Right external carotid artery</u>: single tears = 2%

multiple tears = 0%

In other words, **tears extending from the adventitia to the inner-third of the media** were only found in the **right external carotid artery** and in only 4 (2%) of the 50 subjects examined.

<u>Analysis of Findings</u>

		<u>Single</u>	**<u>Multiple</u>**
		<u>%</u>	**<u>%</u>**
<u>Left:</u>	**Common carotid artery**	**0**	**0**
	Internal carotid artery	**0**	**0**
	External carotid artery	**<u>0</u>**	**<u>0</u>**
		0%	**0%**
<u>Right:</u>	**Common carotid artery**	**0**	**0**
	Internal carotid artery	**0**	**0**

External carotid artery	**2**	**0**
	2%	**0%**

In essence, **tears** extending from the **adventitia** to the **inner-third of the media** were virtually non-existent in this series of cases.

b. Tears extending from the Adventitia to the middle-third of the Media

Left common carotid artery: single tears = 0%
multiple tears = 0%

Left internal carotid artery: single tears = 0%
multiple tears = 0%

Left external carotid artery: single tears = 2%
multiple tears = 2%

Right common carotid artery: single tears = 2%
multiple tears = 2%

Right internal carotid artery: single tears = 0%
multiple tears = 0%

Right external carotid artery: single tears = 0%
multiple tears = 2%

Analysis of Findings

		Single	Multiple
		%	%
Left:	**Common carotid artery**	**0**	**0**
	Internal carotid artery	**0**	**0**
	External carotid artery	**2**	**2**
		2%	**2%**
Right:	**Common carotid artery**	**2**	**2**
	Internal carotid artery	**0**	**0**
	External carotid artery	**0**	**2**
		2%	**4%**

In essence, **tears** extending from the **adventitia** to the **middle-third of the media** show a very low incidence.

c. Tears extending from the Adventitia to the outer-third of the Media

Left common carotid artery: single tears = 4%
multiple tears = 2%

Left internal carotid artery: single tears = 0%
multiple tears = 0%

Left external carotid artery: single tears = 4%
multiple tears = 2%

Right common carotid artery: single tears = 4%

multiple tears = 0%

Right internal carotid artery: single tears = 0%

multiple tears = 0%

Right external carotid artery: single tears = 0%

multiple tears = 0%

Analysis of Findings

		Single %	Multiple %
Left:	**Common carotid artery**	**4**	**2**
	Internal carotid artery	**0**	**0**
	External carotid artery	**4**	**2**
		8%	**4%**
Right:	**Common carotid artery**	**4**	**0**
	Internal carotid artery	**0**	**0**
	External carotid artery	**0**	**0**
		4%	**0%**

An interesting result. **Tears** extending from the **adventitia** to the **outer-third of the media** show a greater incidence than **tears** extending to either the **middle-third** or **outer-third of the media**. This confirms the view that the force exerted by an encircling, compressive ligature tends to lessen and decrease as one proceeds from

outwards to inwards. This may possibly be contributed to by an anatomical plane of cleavage between **adventitia** and **media** (although normally in firm apposition) with differing tissue densities enhancing force diffusion.

6. Tears of the Arterial Adventitia alone

These refer to **tears** involving the outermost layer of the vessel wall. The **left common carotid artery** showed single **tears** of the arterial **adventitia** in 5 subjects (10%) and multiple **tears** in 1 subject (2%), the **left internal carotid artery** single **tears** in 2 subjects (4%) with no multiple **tears** in any of the subjects (0%), and the **left external carotid artery** single **tears** in 2 subjects (4%), and multiple **tears** in 1 subject (2%).

The **right common carotid artery** showed an identical distribution to that of the **left** with single **tears** in 5 subjects (10%), and multiple **tears** in 1 subject (2%) with the **right internal carotid artery** manifesting the same distribution. The **right external carotid artery** showed single **tears** in 2 subjects (4%), and multiple **tears** in 4 subjects (8%) (Figs. 7.17, 7.18 and 7.19).

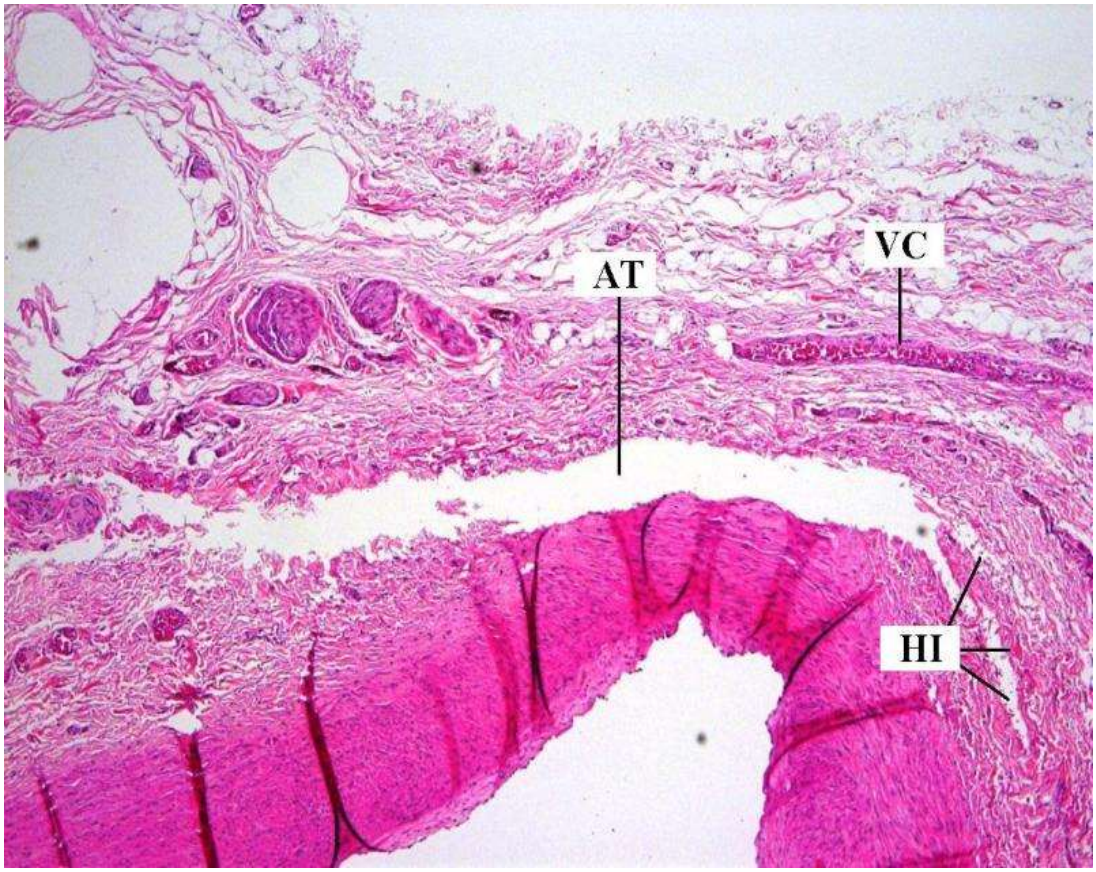


Fig. 7. 18. Right external carotid artery. An **adventitial tear** with haemorrhagic infiltration is noted. **Vascular congestion** within the periadventitial tissues is, in addition, noted. Case no. 3 (Haematoxylin and Eosin x 100). **AT** = adventitial tear. **HI** = haemorrhagic infiltration. **VC** = vascular congestion.

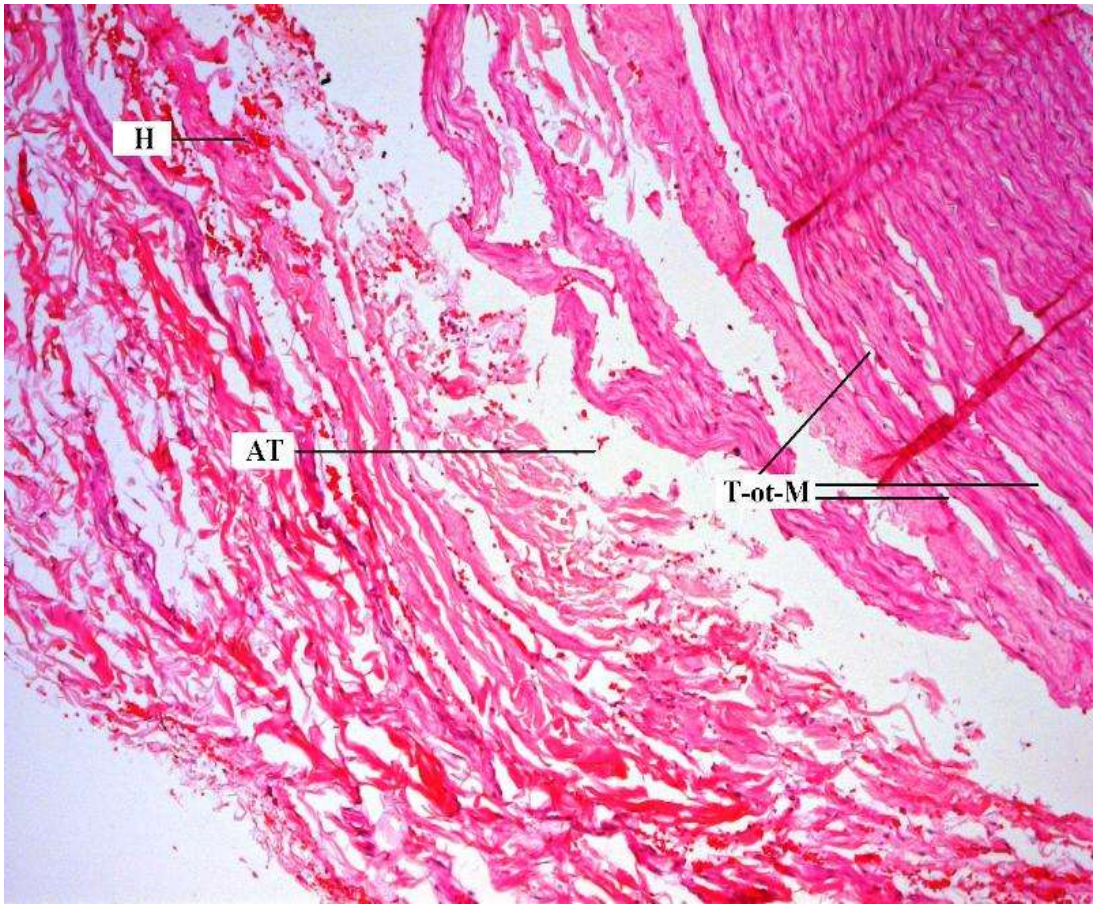


Fig. 7. 19. Right external carotid artery. An **adventitial tear** with haemorrhagic infiltration is noted. Lying immediately beneath this **tear** are multiple **tears of the outer-third of the media**, similarly with haemorrhagic infiltration. In view of the fact that the adventitia-muscular arterial plane is also a neurovascular plane, **haemorrhage** is to be expected, the latter occurring at the time of vascular rupture but only becoming evident on ligature removal allowing extravasation of erythrocytes into the surrounding damaged tissues to then take place. Case no. 36 (Haematoxylin and Eosin x 100). **AT** = adventitial tear. **H** = haemorrhagic infiltration. **T-ot-M** = tears of the outer-third of the media.

Summary of Tears of the Arterial Adventitia alone

In summary, of the 50 subjects examined, the incidence of **tears of the arterial adventitia** was as follows:

Around the left common carotid artery: single tears = 10%
multiple tears = 2%

Around the left internal carotid artery: single tears = 4%
multiple tears = 0%

Around the left external carotid artery: single tears = 4%
multiple tears = 2%

Around the right common carotid artery: single tears = 10%
multiple tears = 2%

Around the right internal carotid artery: single tears = 10%
multiple tears = 2%

Around the right external carotid artery: single tears = 4%
multiple tears = 8%

In general, there is a tendency for multiple **tears** to be fewer than single **tears**.

Analysis of Findings

		Single	Multiple
		%	%
Left:	**Common carotid artery**	**10**	**2**
	Internal carotid artery	**4**	**0**
	External carotid artery	**4**	**2**
		18%	**4%**
Right:	**Common carotid artery**	**10**	**2**
	Internal carotid artery	**10**	**2**
	External carotid artery	**4**	**8**
		24%	**12%**

Comment: A total of **18%** and **24%** are relatively common although multiple damage is less common.

7. Adventitial Haemorrhage

This refers to **haemorrhage** (as a consequence of vessel rupture) within the **adventitial layer of the vessel wall**. This **haemorrhage**, while occurring at the time of vascular rupture, would probably only become evident following removal of the compressive ligature allowing extravasation of red blood cells into the surrounding tissues. Post-mortem decomposition of the clotting factors would produce cellular and tissue dissolution and liquefaction allowing liquid blood to extravasate through a

breached vessel. Of course, in walled chambers such as the heart, post-mortem clots do occur.

Adventitial haemorrhage was noted in the **left common carotid artery** in 21 subjects (42%) (Fig. 7.7). In the **left internal carotid artery adventitial haemorrhage** was noted in 18 subjects (36%), and in the **left external carotid artery** in 14 subjects (28%) (Figs. 7.16 and 7.20).

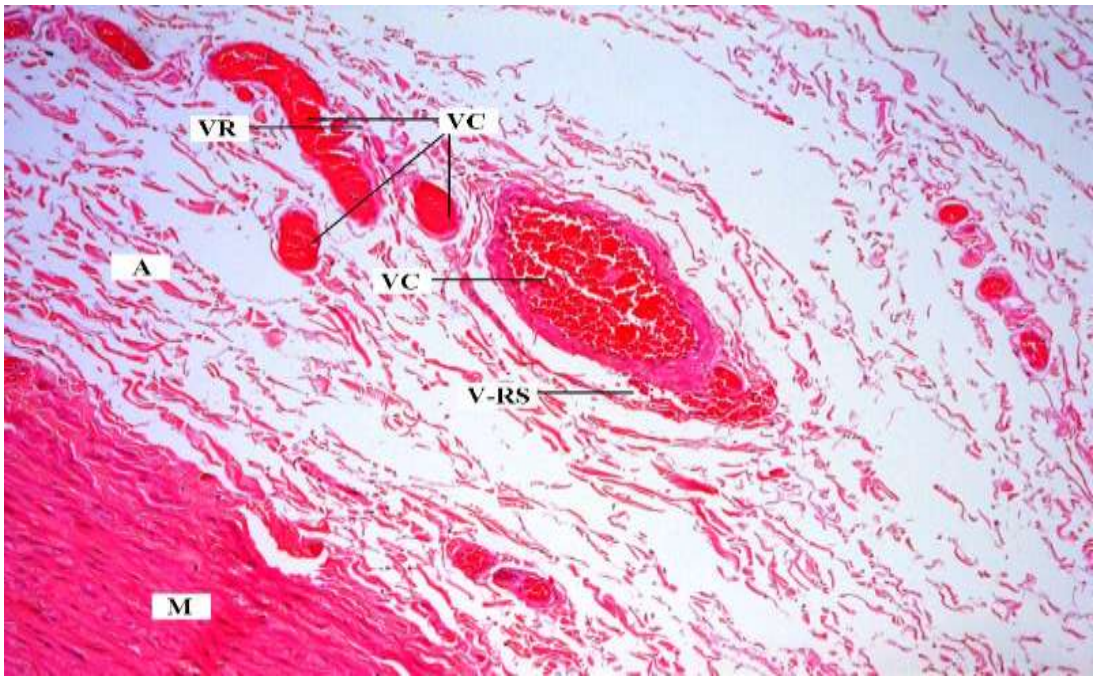


Fig. 7. 20. Left external carotid artery. Congested ruptured adventitial blood vessels are noted with, following removal of the encircling ligature, expected haemorrhagic extravasation into the surrounding perivascular spaces, similar to the Virchow Robin spaces surrounding cortical vessels as an extension of the arachnoid and pia. Case no. 5 (Haematoxylin and Eosin x 100). **A** = adventitia. **VC** = vascular congestion. **VR** = vascular rupture. **M** = media. **V-RS** = Virchow-Robin space.

The **right common carotid artery** showed **adventitial haemorrhage** in 20 subjects (40%), the **right internal carotid artery** in 15 subjects (30%) and the **right external carotid artery** similarly in 15 subjects (30%) (Figs. 7.17, 7.18 and 7.21).

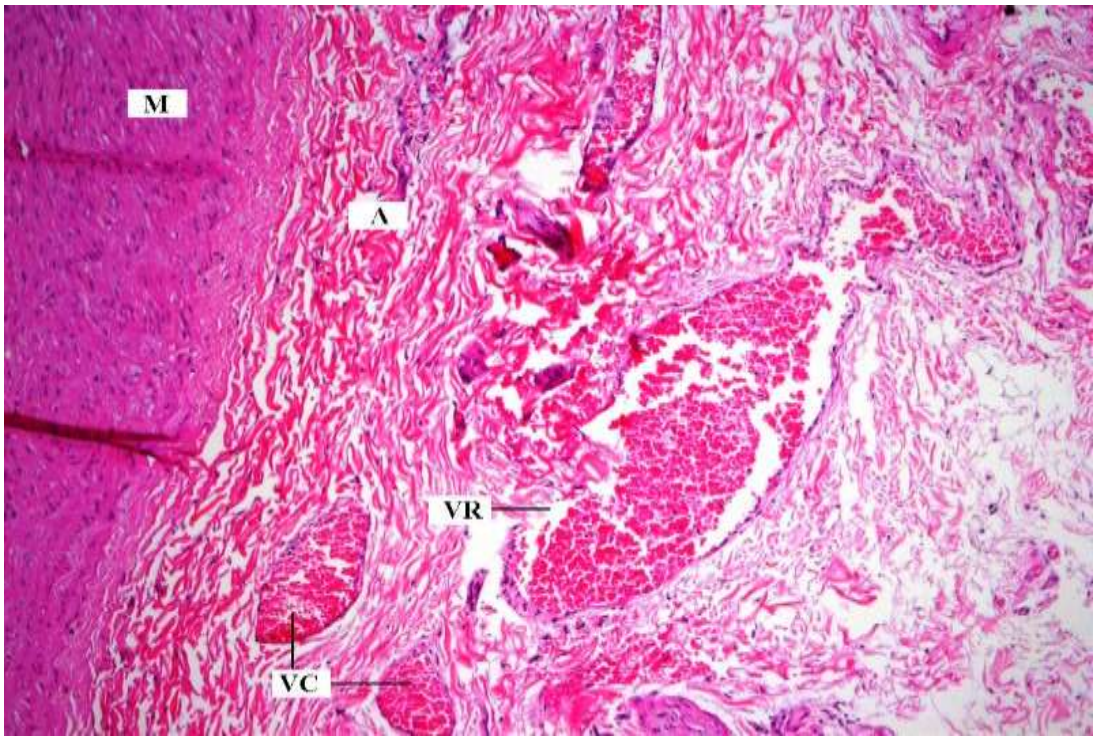


Fig. 7. 21. Right external carotid artery. A ruptured congested adventitial blood vessel with haemorrhagic extravasation into the surrounding perivascular adventitial tissues is noted. Case no. 3 (Haematoxylin and Eosin x 100). **A** = adventitia. **M** = media. **VC** = vascular congestion. **VR** = vascular rupture.

Once again, it must be emphasized that 'bloodless' dissection and removal of tissues was carried out in a gentle and careful manner in an attempt to avoid vascular rupture produced by rough handling of the tissues.

The **adventitial haemorrhage** noted was presumably due to small vessel damage, since other than in 3 cases (6%) (*vide infra)*, the large vessels were neither completely ruptured nor perforated. The findings, however, would seem to imply bilateral and equally applied internally directed force by an external circumferential constricting ligature producing, and resulting, in small vessel rupture, as clearly seen in Figures 7.20 and 7.21.

As far as **adventitial haemorrhages** of the **thoracic** and **abdominal aortae** are concerned, as part of the findings relating to the complete autopsy procedure performed, and where all the organs are examined, these **haemorrhages** were found to be present to a greater or lesser degree in all the cases of suicidal hanging. The possible reasons for these findings are discussed in detail in the Discussion chapter which follows.

Summary of Adventitial Haemorrhage

In summary, of the 50 subjects examined, the incidence of **adventitial haemorrhage** was as follows:

Around the left common carotid artery: 42%

Around the left internal carotid artery: 36%

Around the left external carotid artery: 28%

Around the right common carotid artery: 40%

Around the right internal carotid artery: 30%

Around the right external carotid artery: 30%

Thus, **adventitial haemorrhage** was noted in between one-third to two-fifths of the subjects, a fairly high incidence, being commonest around the **left common carotid artery** and the **right common carotid artery**. This emphasizes the findings previously noted with regard to **adventitial tears** extending to either the **inner-, middle-, or outer-third of the media** in that the layers of the vessel wall in closest proximity to the overlying ligature, or the **arteries** in closest proximity to the ligature bear the brunt of the compressive forces exerted. However, as indicated in the analysis of the **Additional Vascular Findings** (*vide infra* pp. 297-299), additional forces such as the systemic head of pressure, contribute to the very high incidence of lesions involving the innermost and most delicate layer of the vessel wall, namely, the **endothelium**.

The question arises as to whether the fairly high incidence of **adventitial haemorrhage** could possibly be contributed to by instrumental damage, the sheer tightness of the ligature creating great difficulty in passing the blade of a scissors or scalpel blade between the ligature and the underlying skin. In this regard, analysing further the above figures where 100 represents the total number of cases as a percentage and the adjacent figure indicates the number of cases with haemorrhage.

100 – 42 = 58% had no haemorrhage

100 – 36 = 64% had no haemorrhage

100 – 28 = 72% had no haemorrhage

100 – 40 = 60% had no haemorrhage

100 – 30 = 70% had no haemorrhage

100 – 30 = 70% had no haemorrhage

In view of the considerable percentage with no haemorrhage, **adventitial haemorrhage** is unlikely to be caused by instrumental damage, i.e, due to scissors or scalpel trauma.

8. Complete Circumferential Transverse Rupture

By definition, **complete circumferential transverse rupture** refers to those **tears** extending throughout the full-thickness of the vessel wall involving all layers of the vessel wall, i.e. a through-and-through breach. This lesion was found to be present in the **left external carotid artery** in only 1 subject (2%) (Fig. 7.22).

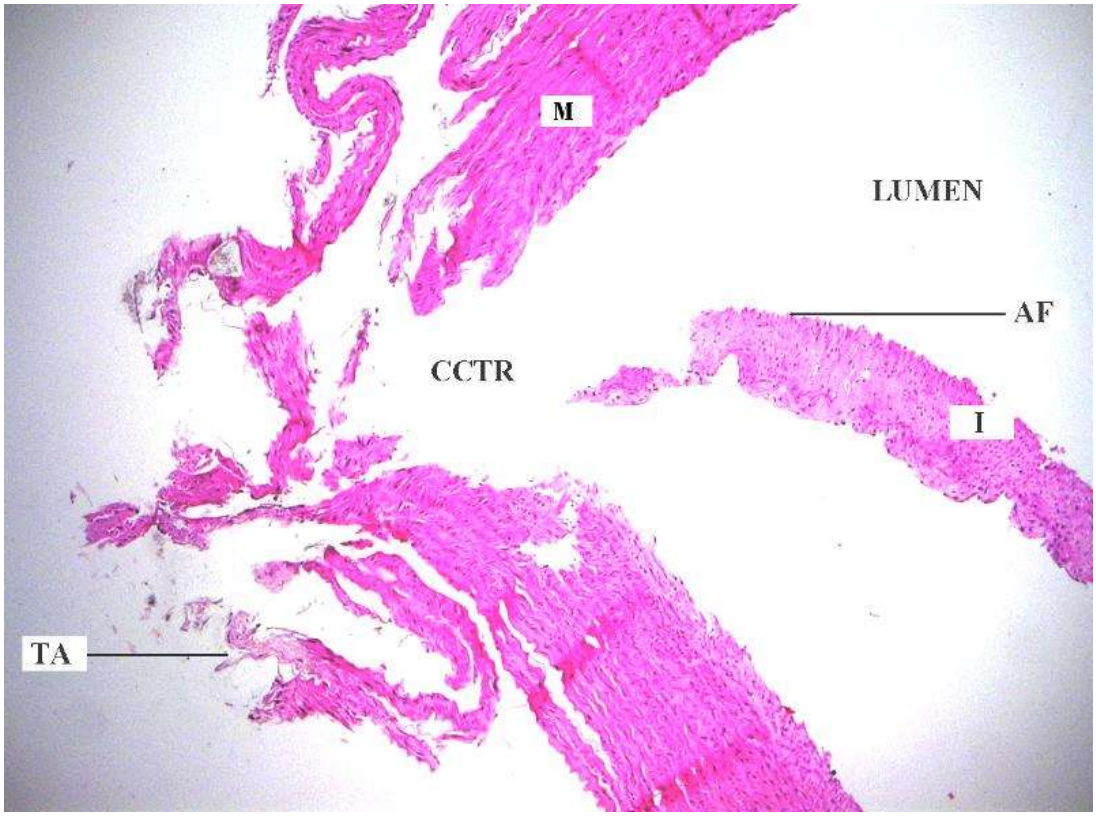


Fig. 7. 22. Left external carotid artery. A **complete circumferential transverse rupture** of the vessel wall is noted extending from the **intima** through to the **adventitia**. An avulsed fragment of the vessel wall lies within the lumen. An interesting feature is that most of the avulsed fragments appear to be extruded outwards suggesting application of an outwardly directed force rather than an inwardly directed compressive force. A further interesting finding in this slide is the total absence of red blood cells. Case no. 20 (Haematoxylin and Eosin x 50). **CCTR** = complete circumferential transverse rupture. **AF** = avulsed fragment. **TA** = torn adventitia. **I** = intima. **M** = media.

As indicated in Table II, the ligature in this case lay over the upper-third of the neck, the possibility being that the position of the ligature was above the point of rupture. The rupture could then be due to pressure from below, i.e. vis-a-tergo, raising the

inference that the impacting systemic head of pressure may, in fact, be sufficient to 'blow-out' the vessel wall when the vessel is compressed from outside, an intriguing thought. As pointed out in the legend to Fig. 7.22, another interesting feature is the total absence of red blood cells. In this case a **nylon cord**, tightly applied, had been present over the upper-third of the neck lending credence to the view that the ligature had compressed and obstructed the vessel completely.

As in cases 10, 21 and 36 (Figs. 7.8b, 7.12b and 7.17, a narrow ligature, i.e. a **nylon cord**, had been applied. However, the person's body weight in this case, although an adult, was only 38 kg. The extreme weight loss found is suggestive of the acquired immunodeficiency syndrome prevalent in the South African Indigenous population. However, testing for the human immunodeficiency virus in terms of South African law is illegal without permission of the surviving relatives. This permission could not be obtained in this case. The general weight loss and loss in body mass, especially muscle and fat, with decrease in the subcutaneous adipose tissue in the neck, as in the other body regions, might result not only in closer proximity of the carotid vessels to the overlying encircling ligature, but might also result in a lesser cushioning and buffering protective effect to the underlying vessels. While no vascular lesions associated with the acquired immune deficiency syndrome were specifically noted in this case, an argument could be made that the general debility associated with this syndrome may have contributed to a weakening of the vessel wall, increasing its susceptibility to any compressive, tensile or other force exerted. Case no. 20 (Haematoxylin and Eosin x 50).

In the **right common carotid artery**, **complete circumferential transverse rupture** was noted in 2 subjects (4%) with none of the other arteries of these cases

showing this lesion. These positive findings therefore, comprise only 3 (6%) of the 50 cases of hanging and 1% of the total number of 300 arteries examined.

General Summary

A simplified Table, i.e. Table IIIa (p. 279), incorporated for ease of reference, follows at the end of the summary section. For emphasis, however, the original terms in use for describing and classifying the lesions noted have, in the main, been retained. A small comment with regard to 'commonness' is, perhaps, appropriate at this juncture. While it is difficult to precisely categorise 'commonness' as a percentage, a reasonable classification may be as follows:

100% = extremely common

75% = common

50% = relatively common

25% = fairly uncommon.

In summary (Table IIIa), single **intimal tears** were found in 17 (5.6%) of the total number of 300 arteries examined, multiple **intimal tears** in 37 (12.3%) of the 300 arteries examined, single **intimal tears extending to or along the internal elastic lamina** were found in 20 (6.6%) of the total number of arteries examined, and multiple **tears** of this kind in 8 (2.6%) of the 300 arteries examined. Single inner-, middle-, and outer- third **intimo-medial tears** were found in 6 (2% of the total number of arteries examined), 3 (1%), and 3 (1%), respectively, while multiple **intimo-medial tears** extending to either the inner-, middle-, and outer- thirds of the **media** were found in 3 (1%), 5 (1.6%), and 1 (0.3%) of the arteries, respectively. Single inner-, middle-, and outer- third **tears** of the **media** were found in 6 (2%), 9

(3%), and 8 (2.6%) of the arteries, respectively, while multiple inner-, middle-, and outer- third **tears** of the **media** or muscular layer of the artery were found in 6 (2%), 13 (4.3%), and 16 (5.3%) of the arteries, respectively.

Single **adventitio-medial tears** extending from the adventitia to the inner-, middle-, and outer- thirds of the media were found in 1 (0.3%), 2 (0.6%), and 6 (2%) of the arteries, respectively, while multiple **tears** of this kind were found in 0 (0%), 3 (1%), and 2 (0.6%) of the arteries respectively. Single **adventitial tears** were found in 21 (7%) of the arteries, while multiple **adventitial tears** were found in 8 (2.6%) of the arteries. **Adventitial haemorrhage** was found in 103 (34.3%) of the arteries examined, while **complete circumferential transverse rupture** was found in 3 (1.%) of the arteries examined.

The numerical values and percentages are presented in the consolidated **Table N** = **300 (Total Number of Arteries examined)**, as follows:

	Single		**Multiple**
Superficial intimal tears:	**17**		**37 = 54 (18%)**
Intimal tears extending to or along the internal elastic lamina:	**20**		**8 = 28 (9.3%)**
Intimo-medial tears extending to the inner-, middle-, and outer-thirds of the media:	**6, 3, 3 (12)**		**3, 5, 1 (9) = 21 (7%)**
Medial tears:			
Inner-third media tears:	**6**	**+**	**6 = 12 (4%)**

Middle-third media tears:	**9**	**+**	**13 = 22 (7.3%)**
Outer-third media tears:	**8**	**+**	**16 = 24 (8%)**
Adventitio-medial tears extending to the inner-, middle-, and outer-thirds of the media:	**1, 2, 6 (9)**	**+**	**0, 3, 2 (5) = 14 (4.6%)**
Adventitial tears extending to the inner-third of the media:	**1**	**+**	**0 = 1 (0.3%)**
Adventitial tears extending to the middle-third of the media:	**2**	**+**	**3 = 5 (1.6%)**
Adventitial tears extending to the outer-third of the media:	**6**	**+**	**2 = 8 (2.6%)**
Adventitial tears:	**21**	**+**	**8 = 29 (9.6%)**
Adventitial haemorrhage:			**103 (34%)**
Complete circumferential transverse rupture:	**3**		**(1%)**
<u>Total Damage of All Kinds:</u>			**(106%)**

This latter figure of **106%** means that some degree of arterial damage occurs in <u>all</u> cases of suicidal hanging and therefore being ‘extremely common’. The reason for the final total being greater than 100% is due to the multiplicity of vascular involvement by the various lesions.

To reiterate, the results are summarised in the consolidated **Table** below, where **N** = **300 (Total Number of Arteries examined)**. The derivation of this number, as indicated in the Materials and Methods chapter, was as follows:

Each of the fifty suicidal hanging cases yielded six arteries from each case, namely, the **left common carotid artery**, the **right common carotid artery**, the **left internal carotid artery**, the **right internal carotid artery**, the **left external carotid artery** and the **right external carotid artery**, i.e., **6** x **50** = **300**. This latter N = 300 Table, therefore, is a consolidated Table constituting a summary of the data shown in Table IIIa. The entire Table IIIa is found in the Appendix.

Table IIIa: Vascular Findings: Unilateral Involvement (Total number of hanging cases examined (N = 50))

LCC = Left Common Carotid	X = Multiple	T = Tear
LIC = Left Internal Carotid	S = Single	IT = Intimal Tear
LEC = Left External Carotid	I = Intimal	MT = Medial Tear
RCC = Right Common Carotid	A = Adventitial	AT = Adventitial Tear
RIC = Right Internal Carotid	M = Medial	IMT = Intimo-Medial Tear
REC = Right External Carotid	AM = Adventitio-Medial	AH = Adventitial Haemorrhage
IEL = Internal Elastic Lamin..	CCTR = Complete Circumferential Transverse Rupt..re	
i = inner ⅓ media	m = middle ⅓ media	o = outer ⅓ media	AMT = Adventitio-Medial Tear

	IT		IT ext. to IEL		IMT						MT						AMT						AT		AH	CCTR
					i		m		o		i		m		o		i		m		o					
Site	S	X	S	X	S	X	S	X	S	X	S	X	S	X	S	X	S	X	S	X	S	X	S	X		
LCC	5	7	5	3	0	2	0	3	0	1	1	2	0	3	1	3	0	0	0	0	2	1	5	1	21	0
%	10.0	14.0	10.0	6.0	0.0	4.0	0.0	6.0	0.0	2.0	2.0	4.0	0.0	6.0	2.0	6.0	0.0	0.0	0.0	0.0	4.0	2.0	10.0	2.0	42.0	0.0
LIC	4	4	4	0	0	0	0	0	0	0	2	1	1	1	1	2	0	0	0	0	0	0	2	0	18	0
%	8.0	8.0	8.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	4.0	2.0	2.0	2.0	2.0	4.0	0.0	0.0	0.0	0.0	0.0	0.0	4.0	0.0	36.0	0.0
LEC	0	8	2	1	0	0	0	0	1	0	1	0	2	2	0	2	0	0	1	1	2	1	2	1	14	1
%	0.0	16.0	4.0	2.0	0.0	0.0	0.0	0.0	2.0	0.0	2.0	0.0	4.0	4.0	0.0	4.0	0.0	0.0	2.0	2.0	4.0	2.0	4.0	2.0	28.0	2.0
RCC	4	4	4	2	4	1	2	1	1	0	1	1	4	3	4	3	0	0	1	1	2	0	5	1	20	2
%	8.0	8.0	8.0	4.0	8.0	2.0	4.0	2.0	2.0	0.0	2.0	2.0	8.0	6.0	8.0	6.0	0.0	0.0	2.0	2.0	4.0	0.0	10.0	2.0	40.0	4.0
RIC	3	7	3	1	1	0	0	0	1	0	1	1	1	2	0	4	0	0	0	0	0	0	5	1	15	0
%	6.0	14.0	6.0	2.0	2.0	0.0	0.0	0.0	2.0	0.0	2.0	2.0	2.0	4.0	0.0	8.0	0.0	0.0	0.0	0.0	0.0	0.0	10.0	2.0	30.0	0.0
REC	1	7	2	1	1	0	1	1	0	0	0	1	1	2	2	2	1	0	0	1	0	0	2	4	15	0
%	2.0	14.0	4.0	2.0	2.0	0.0	2.0	2.0	0.0	0.0	0.0	2.0	2.0	4.0	4.0	4.0	2.0	0.0	0.0	2.0	0.0	0.0	4.0	8.0	30.0	0.0

N = 300 (Total Number of Arteries examined)

TOTAL	17	37	20	8	6	3	3	5	3	1	6	6	9	13	8	16	1	0	2	3	6	2	21	8	103	3
%	5.6	12.3	6.6	2.6	2.0	1.0	1.0	1.6	1.0	0.3	2.0	2.0	3.0	4.3	2.6	5.3	0.3	0.0	0.6	1.0	2.0	0.6	7.0	2.6	34.3	1.0

Bilateral Vessel Involvement

Since the ligature encircles the neck, the possibility of bilateral damage to the **arteries** was considered and the findings are listed below. For ease of description, the right and left vessels have been paired, i.e. the **left common and right common carotid arteries**, the **left internal and right internal carotid arteries**, and the **left external and right external carotid arteries**. This was felt necessary in case bilaterality of trauma might provide a pointer to the possible causation of unconsciousness and death. By means of bilateral grouping for purposes of comparison and analysis, the total number of 300 arteries examined were thus halved and comprised 150 groups. **Bilateral vessel involvement** refers to involvement of both a left-sided and right-sided artery by the same pathological lesion. Furthermore, it should be pointed out that "bilaterality" does not imply and should not be confused with "multiplicity" although, of course, paired arterial groups may show multiplicity of involvement by a particular arterial lesion.

Could any conclusions be drawn from the findings based upon comparative analysis of the groups? Surprisingly, for a circumferentially applied ligature, bilateral vessel involvement was found in this study to be present in only a minority of subjects (**Table IIIb**).

Table IIIb: Vascular Findings: Bilateral Involvement (N = 50)

LCC = Left Common Carotid	X = Multiple	T = Tear
LIC = Left Internal Carotid	S = Single	IT = Intimal Tear
LEC = Left External Carotid	I = Intimal	MT = Medial Tear
RCC = Right Common Carotid	A = Adventitial	AT = Adventitial Tear
RIC = Right Internal Carotid	M = Medial	IMT = Intimo-Medial Tear
REC = Right External Carotid	AM = Adventitio-Medial	AH = Adventitial Haemorrhage
IEL = Internal Elastic Lamina	CCTR = Complete Circumferential Transverse Rupture	
i = inner ½ media	m = middle ⅓ media	o = outer ½ media	AMT = Adventitio-Medial Tear

	IT		IT ext. to IEL		IMT						MT						AMT						AT		AH	CCTR
					i		m		o		i		m		o		i		m		o					
Site	S	X	S	X	S	X	S	X	S	X	S	X	S	X	S	X	S	X	S	X	S	X	S	X		
LCC + RCC		2	2																				2		10	
LIC + RIC	1	2	1																						9	
LEC + REC		1																							6	
TOTAL	1	5	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	25	0
%	2.0	10.0	6.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	4.0	0.0	52.0	0.0

Number of Arteries with Bilateral Involvement: (N = 150)

TOTAL	3	15	9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	6	0	78	0
%	2.0	10.0	6.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	4.0	0.0	52.0	0.0

Thus, of the number of arteries showing bilateral involvement, only 3 of the 150 bilaterally grouped arteries (2%) showed single **intimal tears**, 15 of the bilaterally grouped arteries (10%) showed multiple **intimal tears**, 9 of the bilaterally grouped arteries (6%) showed **intimal tears extending to or along the internal elastic lamina**, 6 of the bilaterally grouped arteries (4%) showed single **adventitial tears**, and 78 of the 150 bilaterally grouped arteries (52%) showed **adventitial haemorrhages**.

Thus:

Single **intimal tears:**	**3 (2%)**
Multiple **intimal tears:**	**15 (10%)**
Intimal tears extending to or along the internal elastic lamina:	**9 (6%)**
Adventitial tears:	**6 (4%)**
Adventitial haemorrhage:	**78 (52%)**

Unfortunately, the number of cases was too small to enable a meaningful comparison and analysis of a predominant dual distribution by each particular pathological change to be made. However, according to the macroscopic pictures of the hanging cases and the finding of very tight ligature application around the neck to the extent that great difficulty was encountered in passing an instrument between the ligature and the underlying skin, the impression gained is that the pressure should have been the same all the way around the neck. However, subtle differences in force application manifesting as differences in involvement of the respective layers of the vessel wall, i.e. the **intima**, **media** and **adventitia** by the pathological processes encountered must have been present. Furthermore, it could be proposed that differences in force application and response to these forces occurs between the

layers of the neck tissues in comparison with the layers of arterial tissues. The reasons for this are enumerated and analysed in the Discussion chapter which follows.

However, the universal finding of empty (empty in this context meaning fluid which has been displaced) **carotid arteries** and empty **internal jugular veins** throughout this study provides additional and further evidence of an externally applied constricting element forcing fluid (i.e. blood, which is incompressible) out of the vessel/s, contributed to by continued venous outflow from the **vertebral vein** and the **vertebral venous plexus** (Figs 7.23 and 7.24). It should be recalled that the **vertebral venous plexus** is a massive network with multiple drainage sites to the **caval system** and especially by the **azygos** and **hemiazygous veins**.

There follow two examples of **internal jugular veins**, one on the right and the other on the left, illustrating not only the emptiness, i.e. devoid of blood within the lumen, of the vein but also the concomitant damage to the venous wall, analogous to the findings observed in the **arteries**. While **endothelial elevation/avulsion** is absent, it should be borne in mind that the **veins**, unlike the **arteries**, are, as discussed in Chapter 4, capacitance vessels and, therefore, not directly exposed to the systemic head of blood pressure as are the **arteries**.

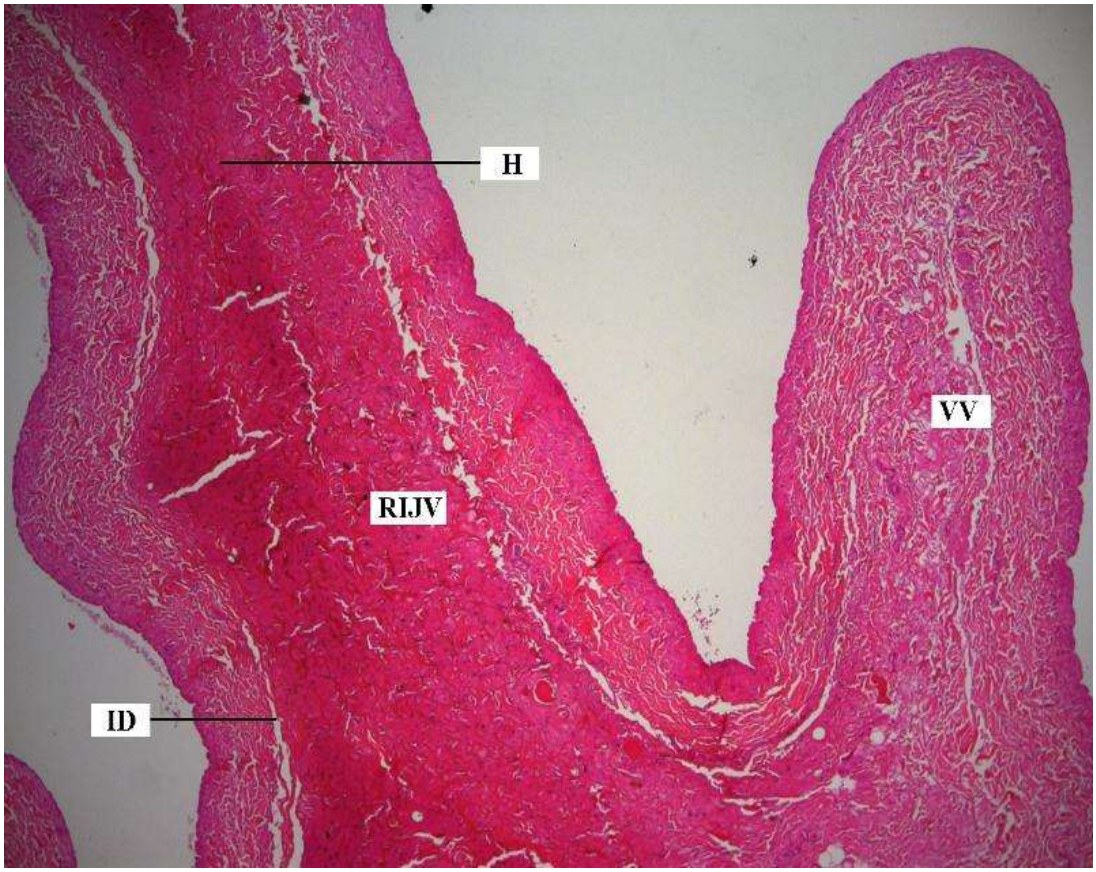


Fig. 7. 23. Right internal jugular vein. A view of the empty vein showing a venous valve with **haemorrhage** into the wall of the vein and with **internal dehiscence.** Case no. 12 (Haematoxylin and Eosin x 50). **RIJV** = right internal jugular vein. **VV** = venous valve. **H** = haemorrhage. **ID** = internal dehiscence.

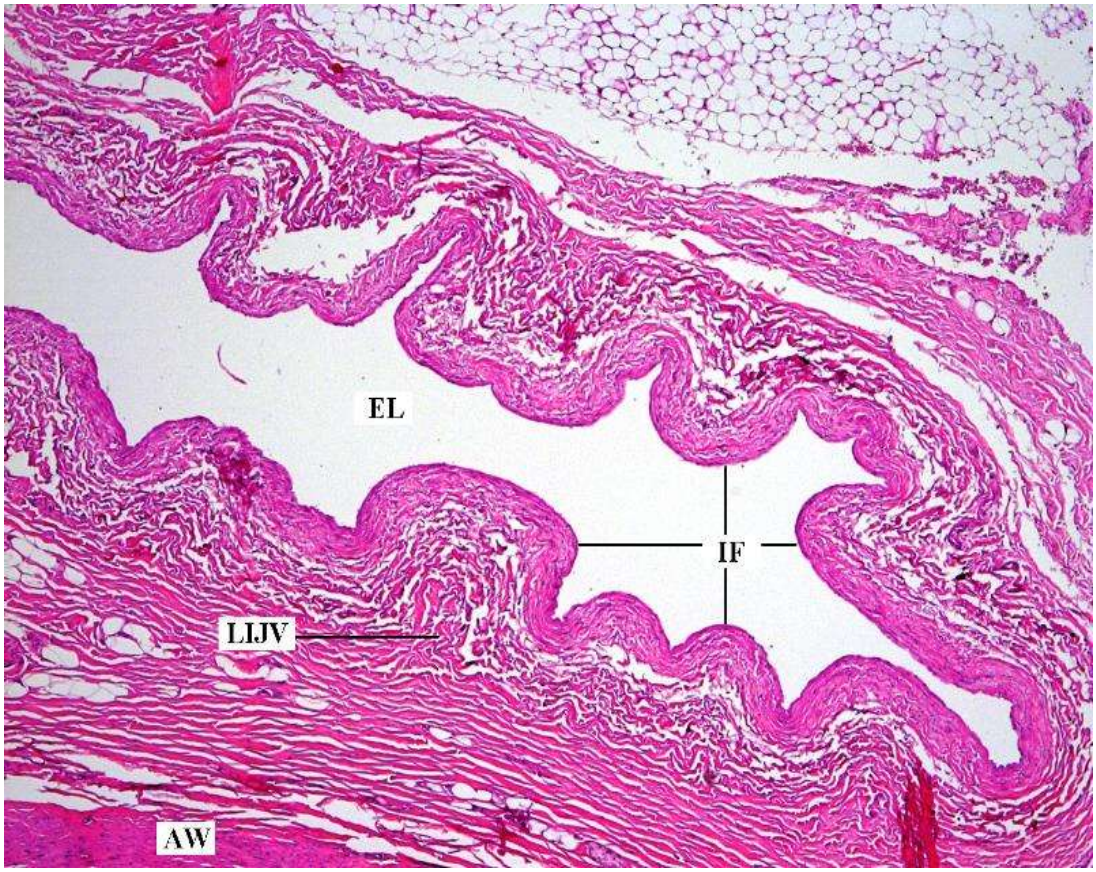


Fig. 7. 24. Left internal jugular vein. The empty vein is seen together with its surrounding adventitial tissues and part of the wall circumference of the adjacent artery. Note the folds of the intima and the lack (or diminution) of the muscular layer. Both these factors indicate the ability of the vein to undergo massive dilatation. Case no. 25 (Haematoxylin and Eosin x 50). **LIJV** = left internal jugular vein. **EL** = empty lumen. **AW** = artery wall. **IF** = intimal folds.

The **haemorrhage** into the wall of the **internal jugular vein** noted in Fig.7.23. with **internal dehiscence** (the latter dealt with under "**Additional Vascular Findings**"; Tables IV, IVa, and IVb) in the next section (**IV**) provides further evidence of both externally and internally applied pressure. The possible reasons for the empty lumen of the vein is discussed in the Discussion chapter which follows.

IV. Additional Vascular Findings

As previously indicated, these involved a second group of histopathological findings emerging from this study. In view of the detail emerging, as well as the fact that some of these findings have gone previously unreported in the literature, they were included as a separate group. These findings comprised **intimal endothelial elevation/avulsion**, **subendothelial clefts**, **internal elastic lamina dehiscence/ separation**, **adventitio-medial separation**, **vascular congestion**, a **vascular plane of cleavage** involving adventitial vessels, and **multiple fenestrations of the media** (muscular layer) of the vessel wall (Tables IV and IVa).

1. Intimal Endothelial Elevation or Avulsion

Intimal endothelial elevation or avulsion was noted in the **left common carotid artery** in 49 subjects (98%), i.e. very common (Figs. 7.4 and 7.7).
In the **left internal carotid artery** this finding was noted in 48 subjects (96%), i.e. very common. In the **left external carotid artery** the finding was noted in 49 subjects (98%), i.e. very common, and in the **right common carotid artery** in 50 subjects (100%), i.e. very common (Fig. 7.25). It should be emphasized that none of these findings were present in the 'control' cases lending support to the view that they constitute genuine pathology. The reason for and pathogenesis of these findings is discussed in some detail in the Discussion chapter which follows.

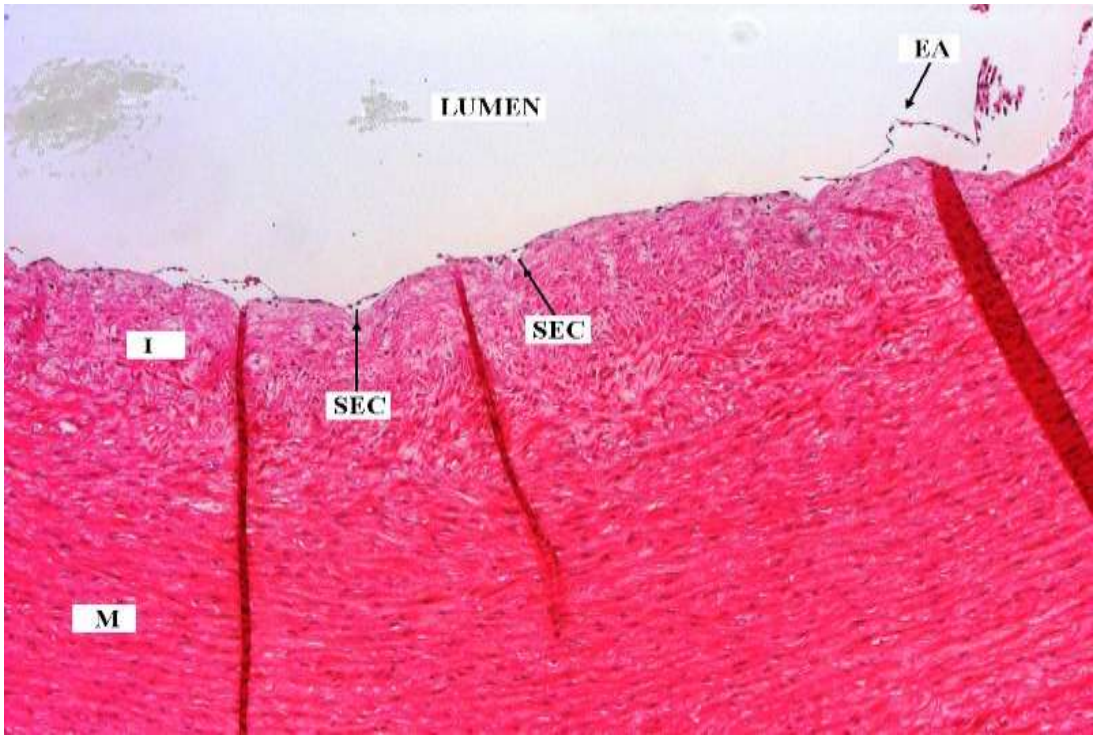


Fig. 7. 25. Right common carotid artery. Endothelial avulsion, **endothelial elevation** and **subendothelial clefts** (arrows) are noted. These would appear to be true and non-artefactual as they are away from the folds. Case no. 38 (Haematoxylin and Eosin x 100). **I** = intima. **M** = media. **EA** = endothelial avulsion. **SEC** = subendothelial cleft.

In the **right internal carotid artery** this finding was noted in 49 subjects (98%), and in the **right external carotid artery** in 50 subjects (100%).

In essence and summary, therefore, **intimal endothelial elevation or avulsion** is a very common form of damage, a not unexpected finding in view of the thinness and delicacy of the endothelial lining highlighting the vulnerability of the **endothelium** to the forces at play during the hanging process.

As previously indicated in the Materials and Methods chapter, death in all cases in the present study had occurred in the 12 hours preceding discovery of the

body. On admission to the mortuary, immediate refrigeration had taken place with medico-legal autopsy being performed within the next 24 hours. Hence, no problems were encountered with regard to post-mortem decomposition producing tissue autolysis with effacement of tissue architecture. While cases of suicidal hanging with post-mortem decomposition were received at the mortuary, these were not included in this study. Any pathological changes observed in the **arterial** and **neural** tissues of the neck on subsequent histopathological examination could not, therefore, be attributed to decompositional change.

2. <u>Subendothelial Clefts</u>

These comprised focal clefts occurring between the **endothelium** and the underlying **intima** as the former was 'lifted off' its underlying attachment to the **intima** producing focal spaces.

Subendothelial clefts were noted in the **left common carotid artery** in 48 subjects (96%) (Figs. 7.4 and 7.7).

In the **left internal** and **left external carotid arteries** this finding was noted in 47 subjects (94%), each, and in the **right common carotid artery** in 49 subjects (98%) (Fig. 7.25).

In the **right internal** and **right external carotid arteries** this finding was noted in 49 subjects (98%), each, respectively.

In summary, therefore, as with **intimal endothelial elevation/avulsion**, **subendothelial clefts** proved to be an <u>extremely common finding</u>.

3. <u>Internal Elastic Lamina Dehiscence/Separation</u>

Internal elastic lamina dehiscence was found in the **left common carotid artery** in 50 subjects (100%) and in the **left internal carotid artery** in 49 subjects (98%) (Figs. 7.9a and 7.9b).

In the **left external carotid artery** this finding was noted in 48 subjects (96%), in the **right common carotid artery** in 49 subjects (98%), and in the **right internal carotid artery** in 48 subjects (96%) (Fig. 7.26).

Again, this was a <u>very common finding</u> with <u>none</u> of these findings being encountered in the so-called normal or 'control-cases', the inference being that these

very common occurrences are, in some manner, due to the hanging process and the presence of a ligature since that too is present in 100% of cases. A further inference that could be made is that **dehiscence** constitutes the pathological response of the **internal elastic lamina** to the forces taking place during the hanging process.

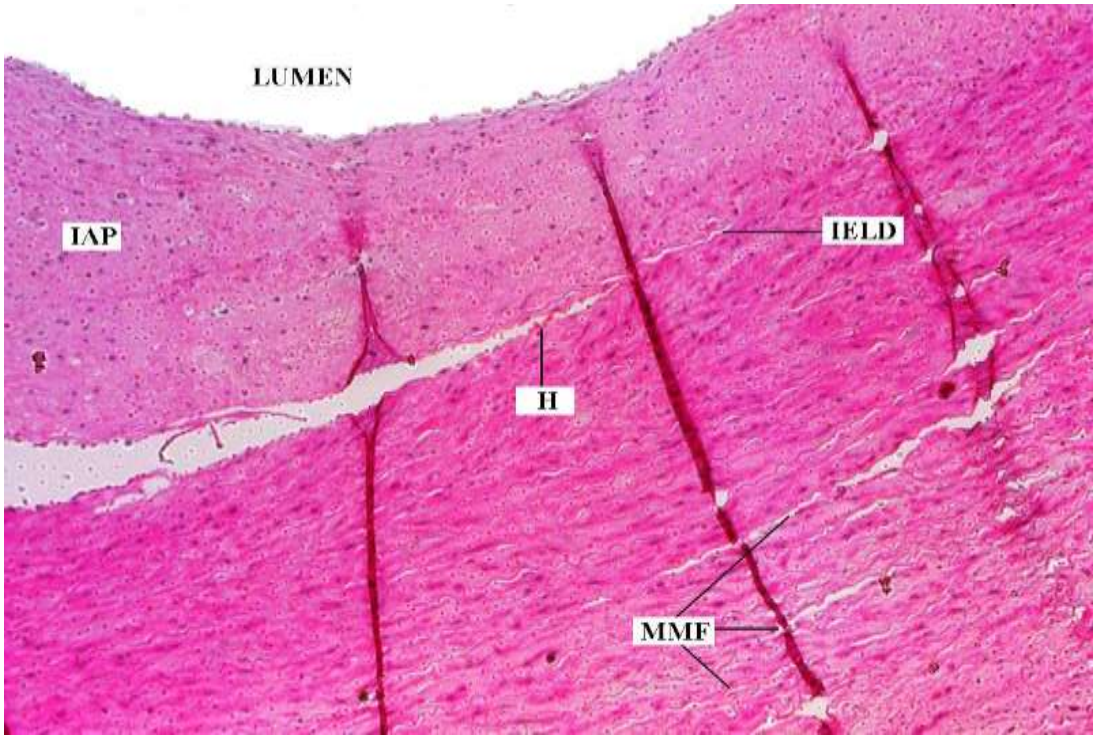


Fig. 7. 26. Right internal carotid artery. A view of the vessel wall showing **dehiscence** and **haemorrhage** extending along the **internal elastic lamina**. Multiple widely separated **fenestrations of the media** are noted in the same segment of the vessel wall. Note the thickness of the intimal atheromatous plaque. Case no. 6 (Haematoxylin and Eosin x 100). **IELD** = internal elastic lamina dehiscence. **H** = haemorrhage. **MMF** = multiple fenestrations (separations) of the media. **IAP =** intimal atheromatous plaque.

In the **right external carotid artery** the finding was noted in 46 subjects (92%).

In summary, therefore, **internal elastic lamina dehiscence/separation**, like **intimal endothelial elevation/avulsion** and **subendothelial clefts** thus comprised an extremely common finding, not surprising in the light of the tightness of ligature application with constriction of the underlying tissues, as evident in Figs. 7a, 7c, 7d, 7k, 7l, 7m and7n.

4. <u>Adventitio-Medial Separation</u>

Adventitio-medial separation was noted in the **left common carotid artery** in 48 subjects (96%) (Fig.7.27).

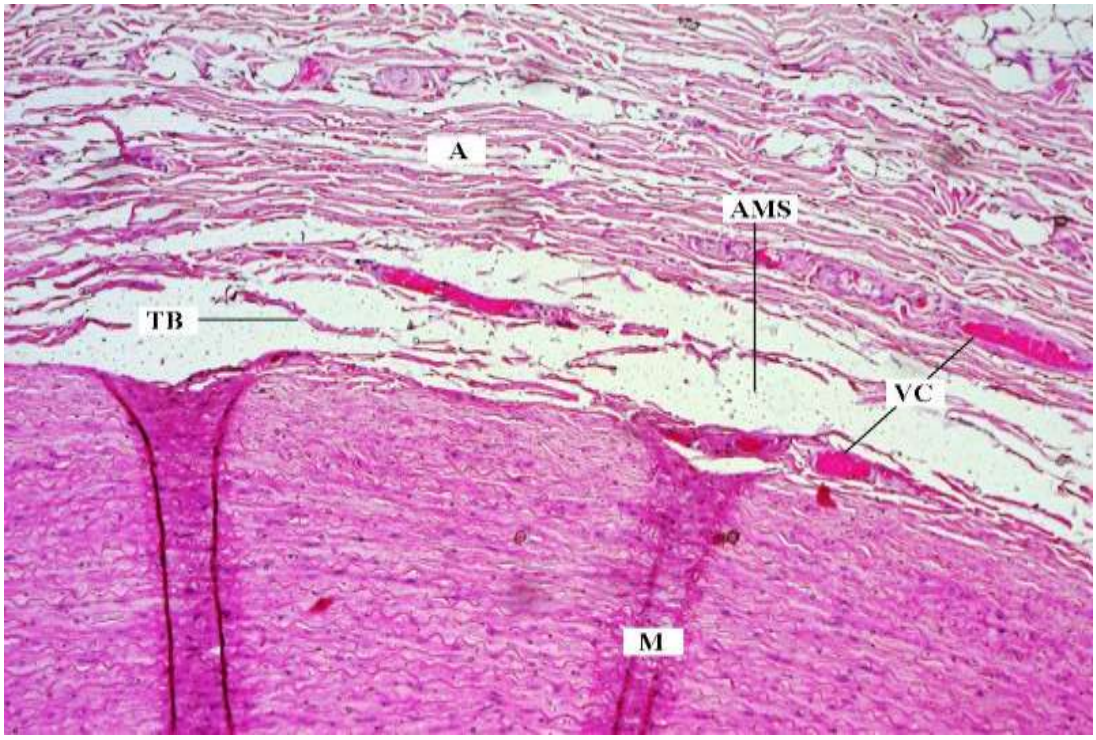


Fig. 7. 27. Left common carotid artery. Adventitio-medial (muscular) **separation** and tissue fragmentation with haemorrhagic infiltration and residual tissue bridges are noted, the latter characteristic of a tensile stretching effect with separation. Case no. 1 (Haematoxylin and Eosin x 100). **A** = adventitia. **A-MS** = adventitio-medial separation. **TB** = tissue bridge. **M** = media. **VC** = vascular congestion.

In the **left internal carotid artery**, **adventitio-medial separation** was noted in 44 subjects (88%), in the **left external carotid artery** in 41 subjects (82%), and in the **right common carotid artery** in 47 subjects (94%) (Fig. 7.12).

In the **right internal carotid artery**, **adventitio-medial separation** was noted in 46 subjects (92%) (Fig. 7.28).

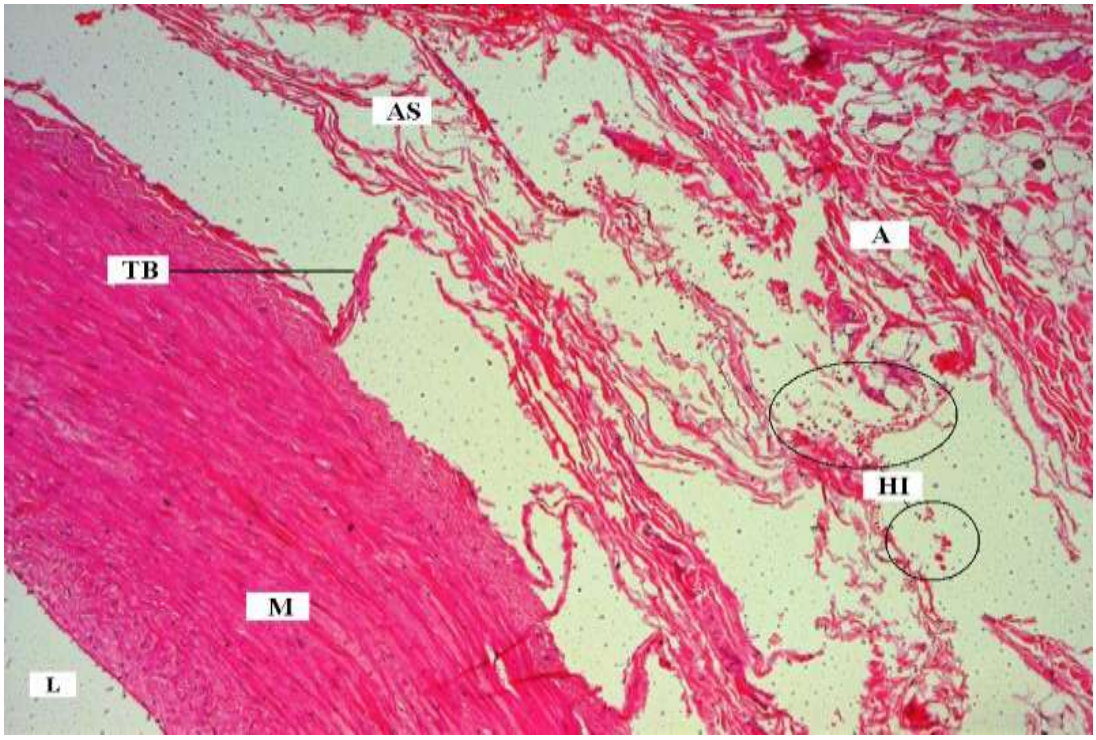


Fig. 7. 28. Right internal carotid artery. Marked separation of the **media** from the overlying **adventitia** is noted. Residual tissue bridges are present. Haemorrhagic infiltration is noted. Case no 25 (Haematoxylin and Eosin x 100. **HI** = haemorrhagic infiltration. **L** = lumen of vessel. **A** = adventitia. **M** = muscularis. **AS** = adventitial sepration. **TB** = tissue bridge.

In the **right external carotid artery**, **adventitio-medial separation** was noted in 47 subjects (94%) (Fig. 7. 29).

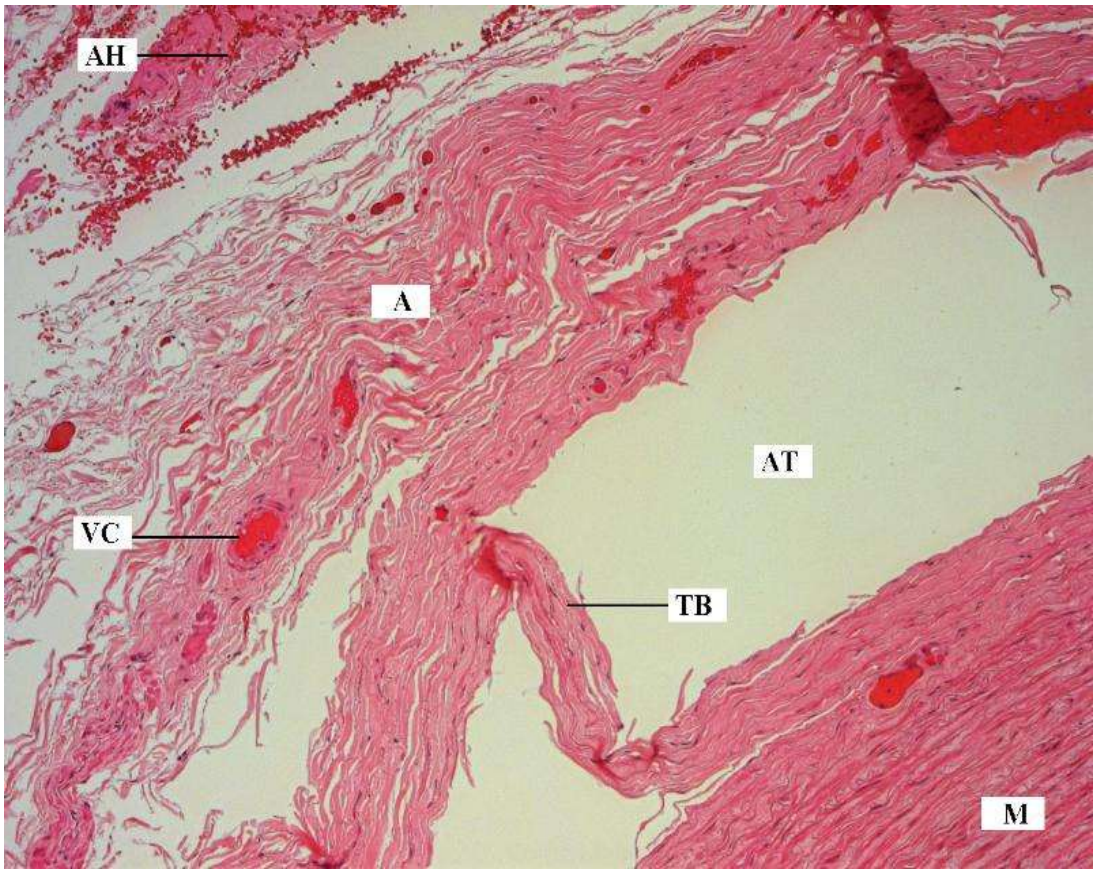


Fig. 7. 29. Right external carotid artery. Marked **adventitial tearing and separation** is noted with residual tissue bridging, overlying **adventitial haemorrhage** and **vascular congestion**. Case no. 38 (Haematoxylin and Eosin x 100). **A** = adventitia. **AT** = adventitial tear. **AH** = adventitial haemorrhage. **TB** = tissue bridge. **VC** = vascular congestion. **M** = media.

In summary, therefore, **adventitio-medial separation** is found extremely commonly in this series of cases. It should be pointed out that arterial dissection was carried out

with great care and gentleness to reduce to a minimum and obviate the possibility of artefactual separation of the tissues.

5. Vascular Congestion

Vascular congestion within the adventitia and periadventitial tissues was noted in the **left common carotid artery** in 42 subjects (84%) (Fig. 7.7).
In the **left internal carotid artery** the finding was noted in 38 subjects (76%), in the **left external carotid artery** in 37 subjects (74%), in the **right common carotid artery** similarly in 37 subjects (74%), in the **right internal carotid artery** in 35 subjects (70%), and in the **right external carotid artery** similarly in 35 subjects (70%) (Figs. 7.18 and 7.21).
In summary, therefore, **vascular congestion** constituted a relatively common finding.

6. Vascular Plane of Cleavage

This refers to an anatomical plane of cleavage between **adventitia** and **media**. An anatomical **vascular plane of cleavage**, identified by the **vascular congestion** which brought the blood vessels into relief, was found in the **left common carotid artery** in 19 subjects (38%) (Fig. 7.7).
In the **left internal carotid artery** the finding was noted in 18 subjects (36%), in the **left external carotid artery** in 15 subjects (30%), in the **right common carotid artery** in 19 subjects (38%), in the **right internal carotid artery** in 14 subjects (28%), and in the **right external carotid artery** in 13 subjects (26%).

In summary, an anatomical **vascular plane of cleavage** between the **adventitia** and **media** was evident in about one-third of the cases. It is possible that this latter finding is as a consequence of the stretch and shear forces taking place during the hanging process. It further emphasizes the tendency of tissues to 'split' along tissue interfaces.

It is, perhaps, pertinent at this juncture to reiterate and recall the finding, previously indicated, of the extreme tightness of ligature application around the neck. This tightness of ligature application occurred to the extent of creating great difficulty in inserting an instrument between the ligature and the underlying skin in order to effect ligature removal.

7. Multiple Fenestrations of the Media (muscular layer)

Multiple fenestrations (i.e. excessive separation) of the **media** were found in the **left common carotid artery** in 5 subjects (10%), and in the **left internal carotid artery** in 1 subject (2%) (Fig. 7.9). In the **left external carotid artery** the finding was noted in 4 subjects (8%) (Figs. 7.10a, 7.10b and 7.22).

In the **right common carotid artery** the finding was noted in 3 subjects (6%), and in the **right internal carotid artery** in 2 subjects (4%) (Fig. 7.26).

In the **right external carotid artery** the finding was noted in 2 subjects (4%).

In summary, this finding was relatively uncommon and may not always be pathological.

Summary of the Additional Vascular Findings

In summary, therefore, of the total number of 300 arteries examined (Table IVa), 295 arteries (98.3%) showed **intimal endothelial elevation or avulsion**, i.e. an extremely frequent finding; 289 arteries (96.3%) showed **subendothelial clefts**, a similarly frequent finding; 290 arteries (96.6%) showed **internal elastic lamina dehiscence/separation**, an extremely frequent finding; 273 arteries (91%) showed **adventitio-medial separation**, a similarly frequent finding; 224 arteries (74.6%) showed **vascular congestion** within the adventitia and periadventitial tissues, a frequent finding; 98 arteries (32.6%) showed an anatomical **vascular plane of cleavage**, a common finding, and 17 arteries (5.6%) showed **multiple fenestrations** of the **media**, an unusual finding. Other than the above findings, all of which are part and parcel of the application of a tight ligature to the neck, the possible reasons for the frequency of findings involving the innermost, i.e. the **intima** and the outermost, i.e. the **adventitia**, layers of the vessel wall are discussed and interpreted in the Discussion chapter which follows.

.

The results are summarised in Table IVa below, where N = Total number of Arteries examined.

Table IVa: Additional Vascular Findings: Unilateral Involvement (Total number of hanging cases examined (N = 50))

LCC = Left Common Carotid	**RCC = Right Common Carotid**
LIC = Left Internal Carotid	**RIC = Right Internal Carotid**
LEC = Left External Carotid	**REC = Right External Carotid**
EE/A = Endothelial Elevation/Avulsion	**SC = Subendothelial Clefts**
IELD = Internal Elastic Lamina Dehiscence	**A-MS = Adventitio-Medial Separation**
VC = Vascular Congestion	**VPC = Vascular Plane of Cleavage**
MMF = Multiple Medial Fenestrations	

Site	EE/A	SC	IELD	A-MS	VC	VPC	MMF
LCC	49	48	50	48	42	19	5
%	98.0	96.0	100.0	96.0	84.0	38.0	10.0
LIC	48	47	49	44	38	18	1
%	96.0	94.0	98.0	88.0	76.0	36.0	2.0
LEC	49	47	48	41	37	15	4
%	98.0	94.0	96.0	82.0	74.0	30.0	8.0
RCC	50	49	49	47	37	19	3
%	100.0	98.0	98.0	94.0	74.0	38.0	6.0
RIC	49	49	48	46	35	14	2
%	98.0	98.0	96.0	92.0	70.0	28.0	4.0
REC	50	49	46	47	35	13	2
%	100.0	98.0	92.0	94.0	70.0	26.0	4.0

N = 300 (Total Number of Arteries examined)							
Site	**EE/A**	**SC**	**IELD**	**A-MS**	**VC**	**VPC**	**MMF**
TOTAL	**295**	**289**	**290**	**273**	**224**	**98**	**17**
TOTAL %	**98.3**	**96.3**	**96.6**	**91.0**	**74.6**	**32.6**	**5.6**

Several striking features emerge from the above distribution. The first is the high incidence of these additional vascular findings, being more than 90% with regard to **endothelial elevation or avulsion**, **subendothelial clefts**, **internal elastic lamina dehiscence**, and **adventitio-medial separation**. The second is the remarkable similarity of lesion incidence and distribution amongst the various arteries. Even with regard to the remaining additional vascular findings, i.e. **vascular congestion**, a **vascular plane of cleavage**, and **multiple fenestrations** of the arterial **media,** where a much lower incidence of lesions was found, the incidence amongst the various arteries was remarkably similar. Thirdly, a notable feature was the very low incidence of **multiple fenestrations** of the **media** amounting to no more than 5.6% of the total number of arteries examined. This parallels and mirrors the observations made with regard to **tears** of the arterial **media** noted above (Tables III and IIIa; Appendix and p. 279), where, while these are indeed present and most certainly do occur, **intima** to **media** and **adventitia** to **media tears** are of a far lesser frequency. In other words, the finding of a paucity of **multiple fenestrations** of the arterial **media** would seem to confirm the resistance of the tough laminated structural configuration of this muscular layer of the vessel wall to the tensile forces operating in the hanging process.

In essence, therefore, there appears little doubt that a tight ligature around the neck causes a wide spectrum and considerable degree of vascular trauma to the **carotid arteries** of the neck. As indicated above, the possible reasons for this are suggested in the Discussion chapter which follows.

Bilateral Vessel Involvement of the Additional Vascular Findings

One hundred and fifty bilateral arterial groups were examined comprising the **left and right common carotid arteries**, the **left and right internal carotid arteries** and the **left and right external carotid arteries**.

Bilateral **endothelial elevation or avulsion** was noted in 50 subjects (100%), comprising 145 arteries (96.6%) of the 150 arterial groups examined. Bilateral **subendothelial clefts** were noted in all 50 subjects (100%), comprising 139 arteries (92.6%) of the 150 bilateral arterial groups examined while **internal elastic lamina dehiscence** was noted in 49 of the 50 subjects (98%), comprising 137 arteries (91.3%) of the 150 bilateral arterial groups examined.

Bilateral **adventitio-medial separation** was noted in 48 of the 50 subjects (96%), comprising 124 arteries (82.6%) of the 150 bilateral arterial groups examined. Bilateral **vascular congestion** was noted in 43 of the 50 subjects (86%), comprising 91 arteries (60.6%) of the 150 bilateral arterial groups examined. A **vascular plane of cleavage**, identified by means of the **vascular congestion** which brought the blood vessels into relief, was noted in 18 of the 50 subjects (36%), comprising 24 arteries (16%) of the 150 bilateral arterial groups examined.

Multiple fenestrations of the media (middle or muscular layer) were noted in 3 of the 50 subjects (6%), comprising 4 arteries (2.6%) of the 150 bilateral arterial groups examined (Table IVb).

Table IVb: Additional Vascular Findings: Bilateral Involvement (N = 150)

EE/A = Endothelial Elevation/Avulsion	**SC = Subendothelial Clefts**
IELD = Internal Elastic Lamina Dehiscence	**A-MS = Adventitio-Medial Separation**
VC = Vascular Congestion	**VPC = Vascular Plane of Cleavage**

MMF = Multiple Medial Fenestrations

	EE/A	SC	IELD	A-MS	VC	VPC	MMF
TOTAL	50	50	49	48	43	18	3
%	100.0	100.0	98.0	96.0	86.0	36.0	6.0

Number of Arteries with Bilateral Involvement: (N = 150)

	EE/A	SC	IELD	A-MS	VC	VPC	MMF
TOTAL	145	139	137	124	91	24	4
%	96.6	92.6	91.3	82.6	60.6	16.0	2.6

What do these tabular figures mean and is it possible to draw any inferences or conclusions from this incidence and distribution? Comparison of the **additional vascular findings** (section **IV**; Tables IVa and IVb; *vide supra*) with the incidence and distribution of the **vascular findings** (section **III**; Tables IIIa and IIIb; *vide supra*) shows that, in contrast to the unilateral incidence of pathological changes noted in the **vascular findings** group (section **III**), the unilateral incidence noted of the **additional vascular findings** group (section **IV**) was very much higher. Furthermore, the incidence of bilateral arterial involvement of the **additional vascular findings** group (section **IV**) was extremely high. For example, in the case

of **endothelial elevation or avulsion** and **subendothelial clefts** the incidence was > 90%, while in the case of **internal elastic lamina dehiscence** and **adventitio-medial (muscular) separation** it exceeded 80%.

In addition to the foregoing, the **additional vascular findings** (Section IV) were compared with the **vascular findings** (Section III). Comparison showed that in both groups of **arterial findings**, the preponderance of pathological changes involved the **intima** and **adventitia**, i.e. the innermost and outermost layers of the vessel wall, highlighting the vulnerabilty of these two layers to the tensile and compressive forces taking place in hanging.

What **additional vascular findings** were noted in the **muscular layer** ? Not only was this layer of the vessel wall relatively spared by **fenestration**, i.e, excessive separation, but the latter lesion (**fenestration**) appears to be a rarity, being involved in only 17 (5.6%) of the total number of 300 arteries examined and in only 4 arteries (2.6%) of the 150 arterial groups showing bilateral involvement. This finding, therefore, provides additional evidence and confirmation of the relative resistance of the laminated planes of the **muscularis** to the tensile forces operating in the hanging process. On the other hand, some of the most severe trauma involving actual **tearing** was to be found in this layer of the vessel wall (Figs.7.4a. 7.4b, 7.4c, 7.6, 7.7a, 7.8a, 7.8b, 7.9a. 7.9b, 7.10a, 7.10b, 7.11, 7.12a, 7.12b, 7.13, 7.14, 7.15a, 7.15b, 7.16, 7.17, 7.19 and 7.22). This issue is fully discussed in the Discussion chapter which follows.

The following two tables (Table VI and Table VII) show the percentage and numerical representation of **intima**, **media** and **adventitia** involvement of vessel wall tears.

Table VI: Diagrammatic Representation of Percentage Involvement of Arterial Wall Tears in 50 Subjects. (Arteries: N = 300)

Intima	Media			Adventitia	Single	Multiple
	Inner ⅓	Middle ⅓	Outer ⅓			
*					40%	16%
---------					12%	6%
----------					6%	10%
----------					6%	2%
	*				12%	12%
		*			18%	26%
			*		16%	32%
	----------				2%	0%
		----------			4%	6%
			----------		12%	4%

Table VII: Numerical Representation of Percentage Involvement of Arterial Wall Tears in 50 Subjects. (Arteries: N = 300)

Site	Artery	Single	Multiple
Intima	LCC	10	6
	LIC	8	0
	LEC	4	2
	RCC	8	4
	RIC	6	2
	REC	4	2
	Total	**40%**	**16%**

Site	Artery	Single	Multiple	Site	Single	Multiple	Site	Single	Multiple
Intima to Inner ⅓ Media	LCC	0	4	Inner ⅓ Media	2	4	Adventitia to Inner ⅓ Media	0	0
	LIC	0	0		4	2		0	0
	LEC	0	0		2	0		0	0
	RCC	8	2		2	2		0	0
	RIC	2	0		2	2		0	0
	REC	2	0		0	2		2	0
	Total	**12%**	**6%**		**12%**	**12%**		**2%**	**0%**
Intima to Middle ⅓ Media	LCC	0	6	Middle ⅓ Media	0	6	Adventitia to Middle ⅓ Media	0	0
	LIC	0	0		2	2		0	0
	LEC	0	0		4	4		2	2
	RCC	4	2		8	6		2	2
	RIC	0	0		2	4		0	0
	REC	2	2		2	4		0	2
	Total	**6%**	**10%**		**18%**	**26%**		**4%**	**6%**
Intima to Outer ⅓ Media	LCC	2	6	Outer ⅓ Media	2	6	Adventitia to Outer ⅓ Media	11	2
	LIC	2	4		2	4		0	0
	LEC	0	4		0	4		11	2
	RCC	8	6		8	6		11	0
	RIC	0	8		0	8		0	0
	REC	4	4		4	4		0	0
	Total	**16%**	**32%**		**16%**	**32%**		**33%**	**4%**

In essence, all the arteries have some degree of anatomical damage but there is superimposed, physiological, damage, i.e. compression of and blockage of blood supply. Since the victim has not recovered, the anatomical and pathological damage is inconsequential if regarded in isolation but critical in light of the physiological

implications and consequences which ensue. In other words, anatomical and pathological damage is not merely academic but points to physiological factors which must be considered in causing the death of the victim.

V. Neural Findings

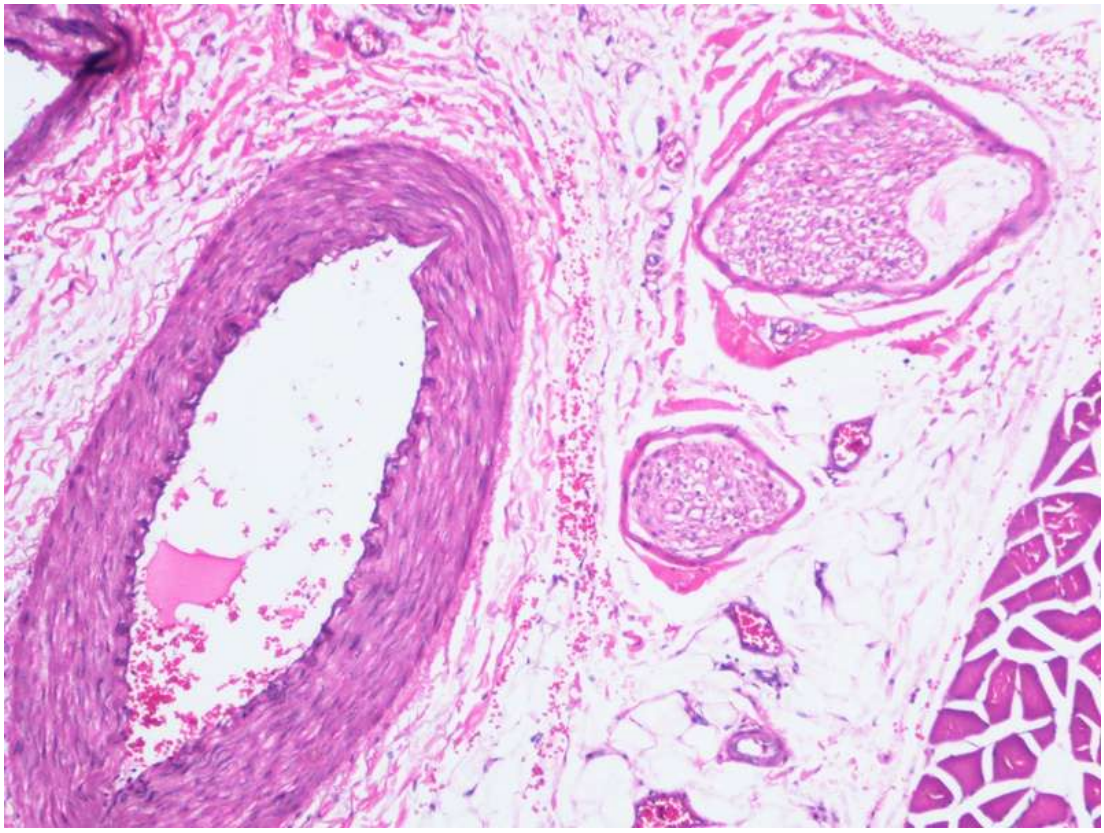
While it is not easily possible to differentiate microscopically between peripheral somatic nerve and autonomic nerve without recourse to special techniques, the nerves visualized probably comprise peripheral nerves, i.e. cervical plexus. However, when **ganglia** were visualized these were sympathetic nerve structures.
The **neural findings** comprised the third of the 3 major groups of histopathological findings emerging from this study.

Introduction

As shown in that part of this work dealing with the anatomy of the neck, with its accompanying anatomical sketches, the neck is a highly complex structure, incorporating not only vascular and neural elements, but elements in close apposition to and integrated with one another. Thus, a circumferentially applied, tightly constricting and compressive ligature, might be expected to produce multiple anatomical and physiological effects, often in sympathy with, or antagonistic to one another. These latter effects are, in addition, complicated by the gravitational effect of the weight of the hanging body. On the one hand, there is the vascular constriction produced by sympathetic stimulation (accounting for the facial pallor described in the literature in some victims), while, on the other hand, parasympathetic stimulation

may result in cardiac inhibition with consequent bradycardia and possible cardiac arrest. Thus, the **neural findings** emerging from this study, not previously considered or explored to such an extent should, perhaps, be considered as playing a major role and being of paramount importance in the pathophysiology of the hanging process in initiating unconsciousness. In these circumstances of ligature hanging, unremitting unconsciousness must, inevitably, result in death with the prodromal incident (unconsciousness) being the harbinger of death.

The **neural findings** emerging from this study have been subdivided into those involving the **nerves**, the **neural ganglia**, the **carotid bodies**, the **accessory glomi** of the **carotid bodies**, and, in addition, the **phrenic nerve** (Tables V, Va and Vb). For comparative purposes, a control case of a **nerve** from a non-hanging subject is reproduced overleaf.



Section shows normal nerve in proximity to a small arterial vessel and normal sternocleidomastoid muscle from non-hanging control case no.3. Note that no **intraneural congestion** or indentation of the overlying muscle is seen (Haematoxylin and Eosin X 10).

A. The Nerves

In all the arterial sections examined, the surrounding nerves showed some form of ligature damage.

1. Intraneural Congestion

Intraneural congestion was noted in the **neural elements** around the **left common carotid artery** in 2 subjects (4%), around the **left internal carotid artery** in 5 subjects (10%), around the **left external carotid artery** in 3 subjects (6%), around the **right common carotid artery** in 2 subjects (4%), around the **right internal carotid artery** in 3 subjects (6%), and around the **right external carotid artery** in 5 subjects (10%) (Fig. 7. 30a.).

Analysis of Findings

Left:	**Common carotid artery**	**2 (4%)**
	Internal carotid artery	**5 (10%)**
	External carotid artery	**3 (6%)**
		10
Right:	**Common carotid artery**	**2 (4%)**
	Internal carotid artery	**3 (6%)**
	External carotid artery	**5 (10%)**
		10

An equal incidence of findings was, therefore, noted.

While this finding will be discussed in greater detail in the Discussion chapter which follows, **intraneural congestion** appears to reflect, once again, the general damming up of blood as a consequence of: i) obstruction to blood vessels by the ligature, or; ii) a failing circulation. It should be noted that the veins are capacitance vessels acting physiologically as a blood reservoir. Furthermore, and this is of paramount importance in the context of this study, such **congestion** must, of necessity, produce some element of compression, and/or subsequent stimulation, of the adjoining and adjacent nerve fibres. This issue will be explored in greater detail in the next section.

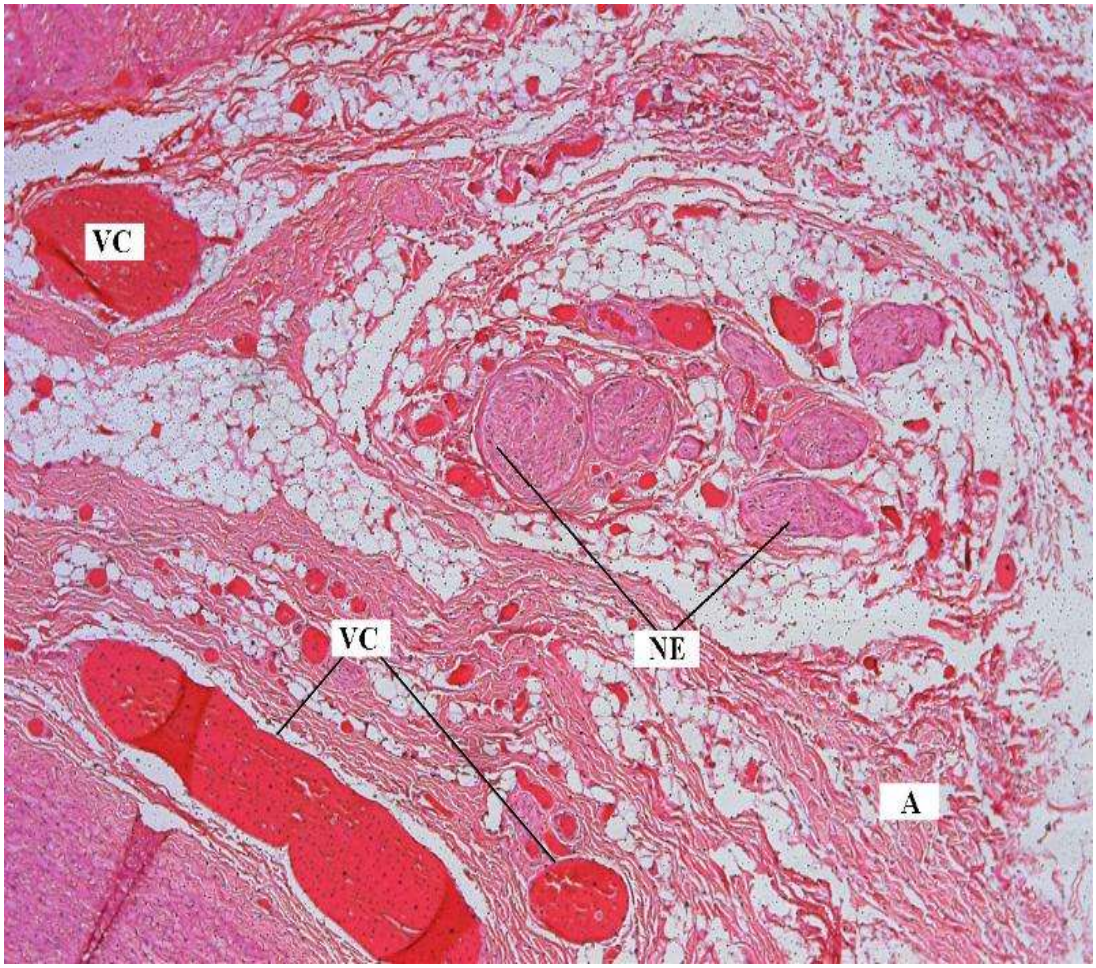


Fig. 7. 30a. A view of the tissues surounding the **right external carotid artery.** While the **vascular congestion** noted is predominantly perineural, with marked **vascular congestion** of the surrounding periadventitial tissues, **intraneural congestion** is indeed present in the neural bundle occupying the centre of the field. Case no. 50 (Haematoxylin and Eosin x 50). **NE** = neural elements. **VC** = vascular congestion. **A** = adventitia.

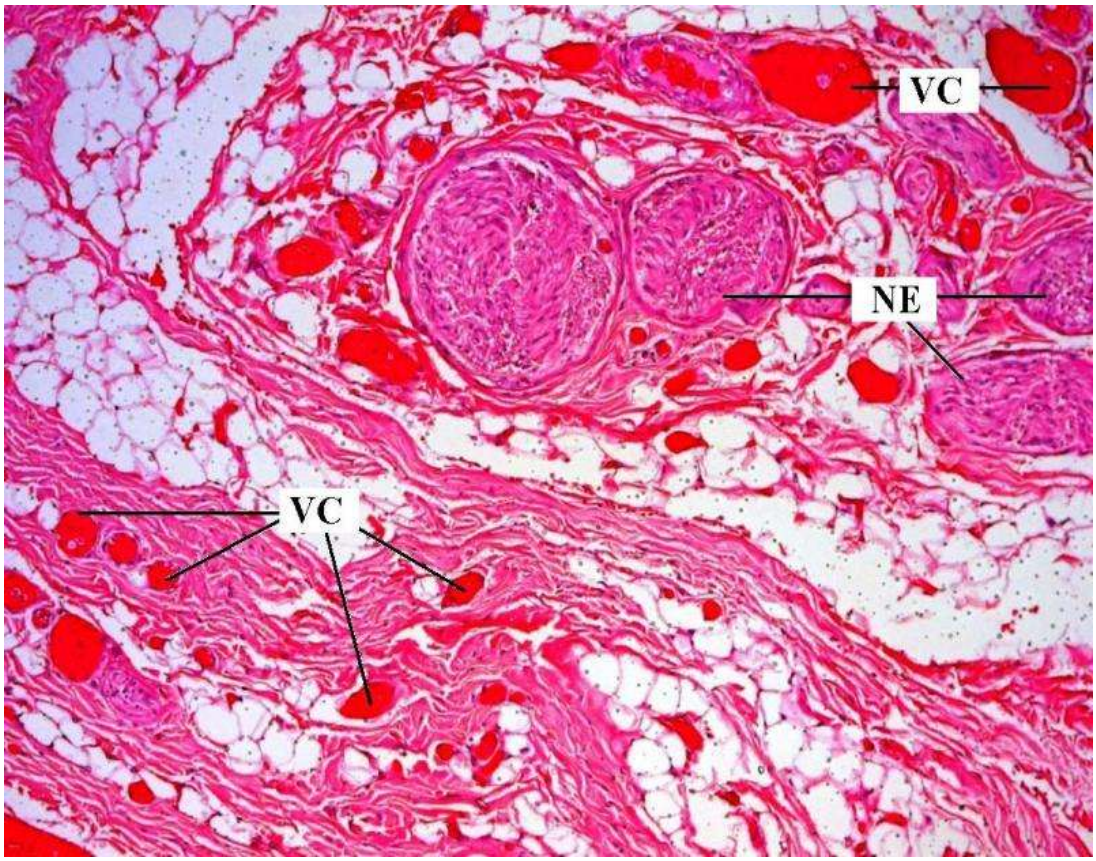


Fig. 7. 30b. A high-power view of the tissues surrounding the **right external carotid artery** with the features seen in Fig. 7. 30a. While marked perineural **vascular congestion** is noted with haemorrhagic infiltration of the surrounding perineural tissues, **intraneural congestion** is, in addition, seen Case no. 50 (Haematoxylin and Eosin x 100). **NE** = neural elements. **VC** = vascular congestion.

2. <u>Neural/ Perineural Haemorrhage</u>

Neural/perineural haemorrhage was noted in the **neural elements** around the **left common carotid artery** in 7 subjects (14%), around the **left internal carotid artery** in 1 subject (2%), around the **left external carotid artery** in 2 subjects (4%), around the **right common carotid artery** in 3 subjects (6%), around the **right internal**

carotid artery in none of the subjects (0%), and in the **neural elements** in the tissues around the **right external carotid artery** in 1 subject (2%).

Analysis of Findings

Left:	**Common carotid artery**	**7 (14%)**
	Internal carotid artery	**1 (2%)**
	External carotid artery	**2 (4%)**
		10

Right:	**Common carotid artery**	**3 (6%)**
	Internal carotid artery	**0 (0%)**
	External carotid artery	**1 (2%)**
		4

In other words, a greater incidence of **neural haemorrhage** was present on the left as compared with the right. It is suggested that this does not necessarily imply greater application of pressure to the blood vessels on the left by the position of the knot, resulting in consequent vascular rupture with **neural haemorrhage**. It may be equally as a result of the head falling to the right with a consequent greater degree of stretch and blood vessel deformation on the side contralateral to the head, i.e. the left.

Neural haemorrhage, like **intraneural and perineural congestion**, would be expected to exert pressure on adjacent neural fibres. In contrast with **congestion**, however, which is a pathophysiological change, **haemorrhage** represents a more

profound pathological alteration constituting evidence of rupture of blood vessels, whether due to vascular fragility or due to ‘ligature trauma’.

3. Neural Internal Dehiscence/Separation

Neural internal dehiscence/separation (i.e. separation) (**NID**) was noted in the **neural elements** around the **left common carotid artery** in 13 subjects (26%) (Fig. 7. 31).

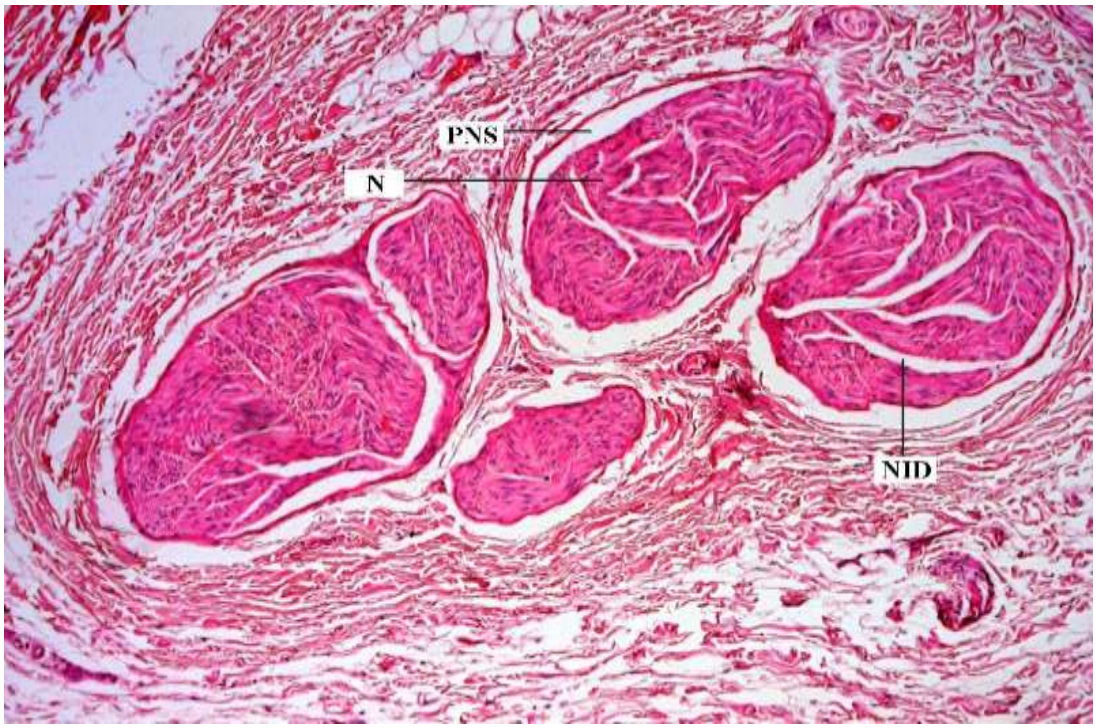


Fig. 7.31. A view of **neural elements** in the tissues surrounding the **left common carotid artery. Neural internal dehiscence** and **perineural separation** is noted. Case no. 19 (Haematoxylin and Eosin x 100). **N** = Nerve. **NID** = neural internal dehiscence. **PNS** = perineural separation.

Internal dehiscence was noted in the **neural elements** around the **left internal carotid artery** in 10 subjects (20%) (Fig. 7.32).

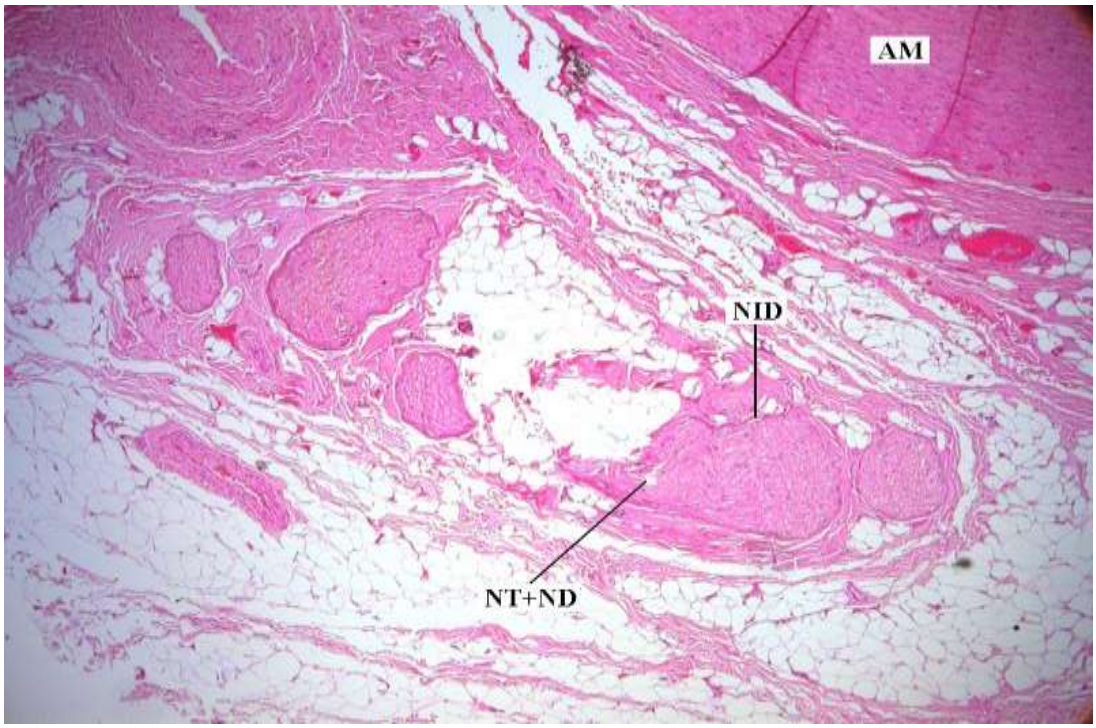


Fig. 7. 32. A view of the **neural elements** in the tissues surrounding the **left internal carotid artery. Neural tearing** and **neural disruption** with some degree of **neural internal dehiscence** is noted. Case no. 34 (Haematoxylin and Eosin x 50). **NT + ND** = neural tearing and neural disruption. **NID** = neural internal dehiscence. **AM** = arterial media.

In the **neural elements** around the **left external carotid artery**, **neural internal dehiscence** was noted in 11 subjects (22%) (Fig. 7. 33).

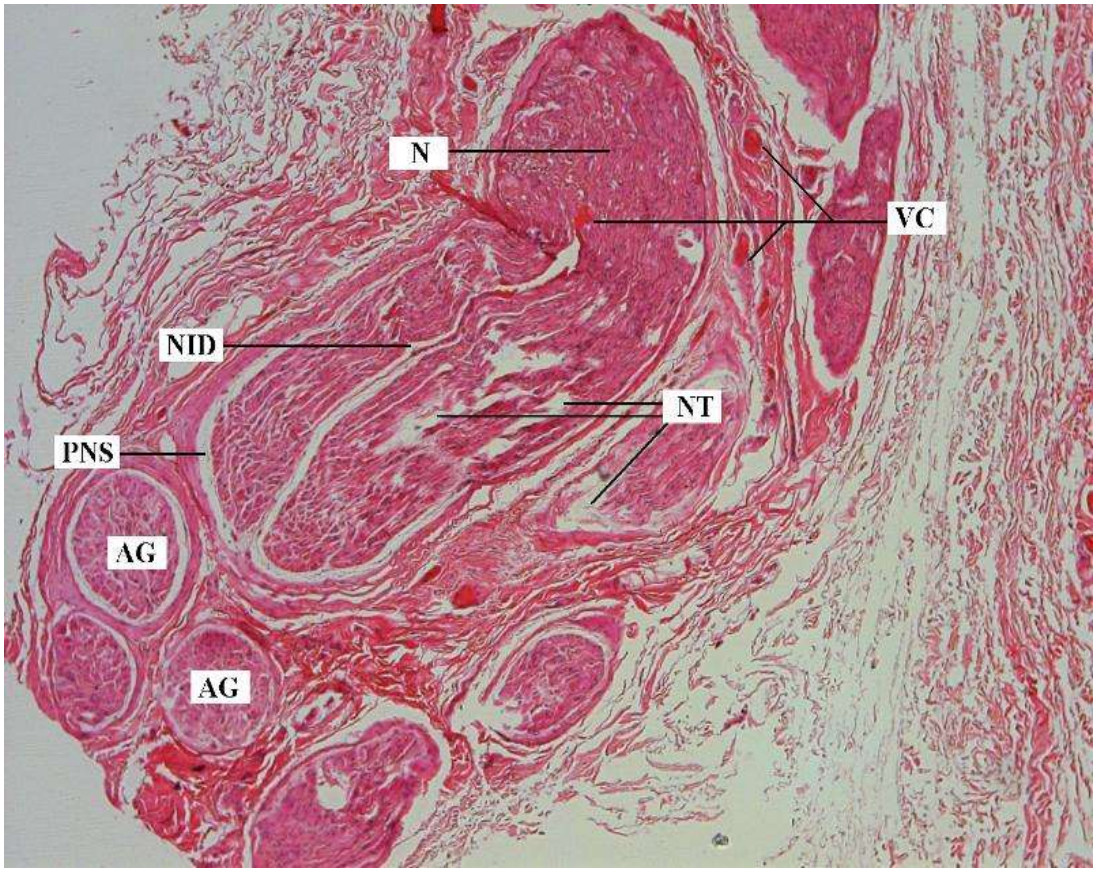


Fig. 7. 33. A view of the **neural elements** and perineural tissues around the **left external carotid artery. Neural tearing**, **neural internal dehiscence** and **perineural separation** is noted together with perineural **vascular congestion**. Case no. 43 (Haematoxylin and Eosin x 50). **N** = nerve. **NID** = neural internal dehiscence. **NT** = neural tearing. **PNS** = perineural separation. **VC** = vascular congestion. **AG** = accessory glomus.

In the **neural elements** around the **right common carotid artery**, **internal dehiscence** was noted in 8 subjects (16%), around the **right internal carotid artery** in 4 subjects (8%), and in the **neural elements** around the **right external carotid artery** in 8 subjects (16%) (Figs. 7.34 and 7.35).

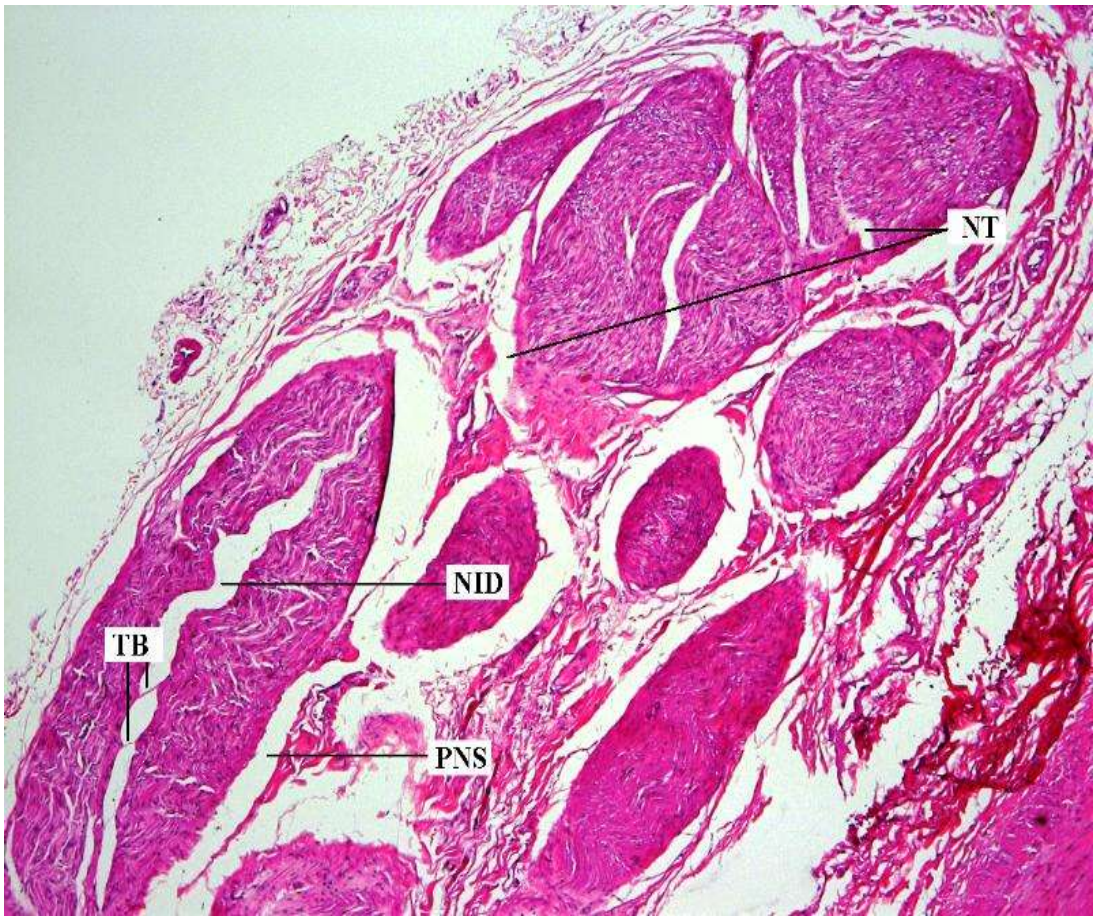


Fig. 7.34. A view of the **neural elements** around the **right external carotid artery**. **Neural tearing**, **neural internal dehiscence** and **perineural separation** are noted. Case no. 39 (Haematoxylin and Eosin x 50). **NT** = neural tearing. **NID** = neural internal dehiscence. **PNS** = perineural separation. **TB =** tissue bridge.

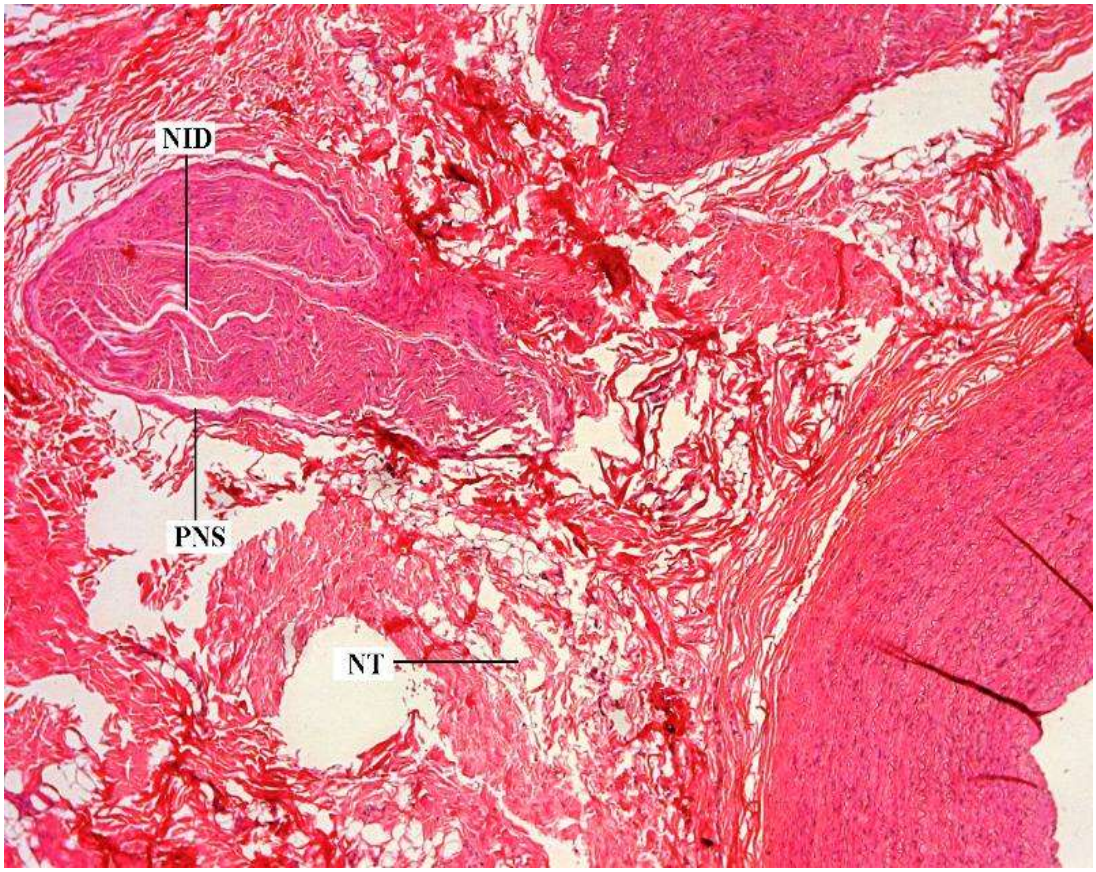


Fig. 7.35a. A view of the **neural elements** in the tissues around the **right external carotid artery. Neural tearing**, **neural internal dehiscence** and **perineural separation** are noted. Case no. 41 (Haematoxylin and Eosin x 50). **NT** = neural tearing. **NID** = neural internal dehiscence. **PNS** = perineural separation.

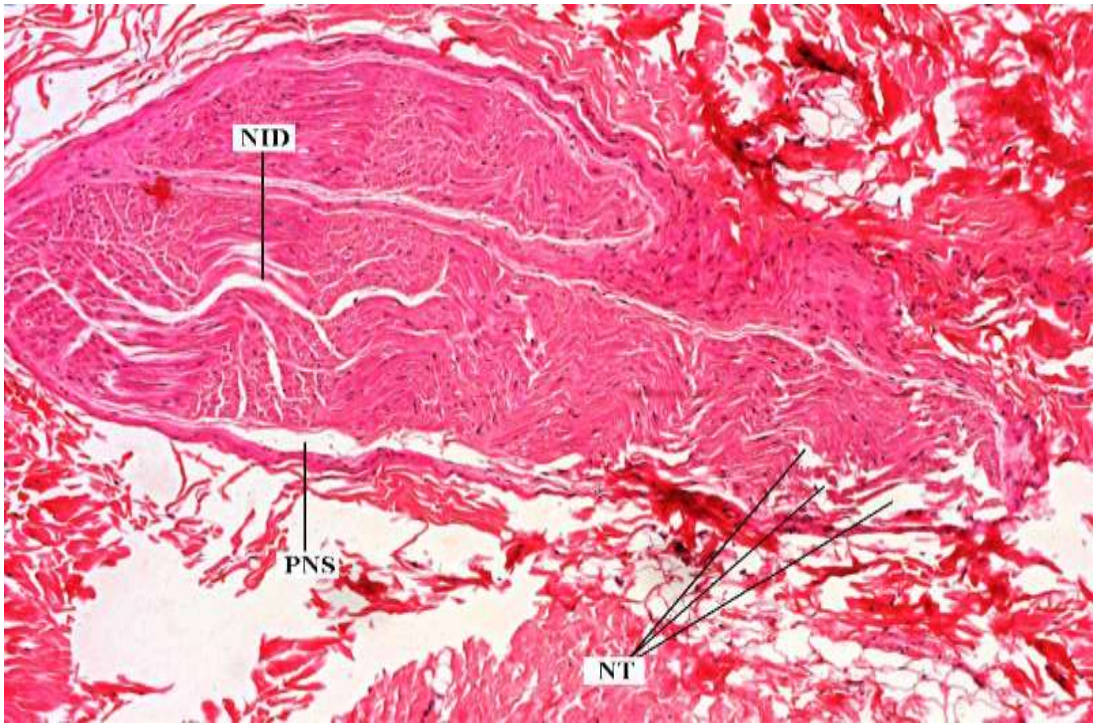


Fig. 7. 35b. A high-power view of the **neural element** in the tissues around the **right external carotid artery. Neural tearing**, **neural internal dehiscence** and **perineural separation** as seen in Fig. 7. 35a is noted. Note that there are two branches- the upper one with what appears to be a natural separation while the other appears to be a 'damage split'. Case no. 41 (Haematoxylin and Eosin x 100). **NT** = neural tearing. **NID =** neural internal dehiscence. **PNS =** perineural separation.

Analysis of Findings

Left:	**Common carotid artery**	**0 (0%)**
	Internal carotid artery	**10 (20%)**
	External carotid artery	**11 (22%)**
		21
Right:	**Common carotid artery**	**8 (16%)**

Internal carotid artery	**4 (8%)**
External carotid artery	**8 (16%)**
	20

In other words, an almost equal incidence.

4. <u>Neural Tearing</u>

Neural tearing was found in the **neural elements** around the **left common carotid artery** in 7 subjects (14%) (Figs. 7. 36a and 7.36b).

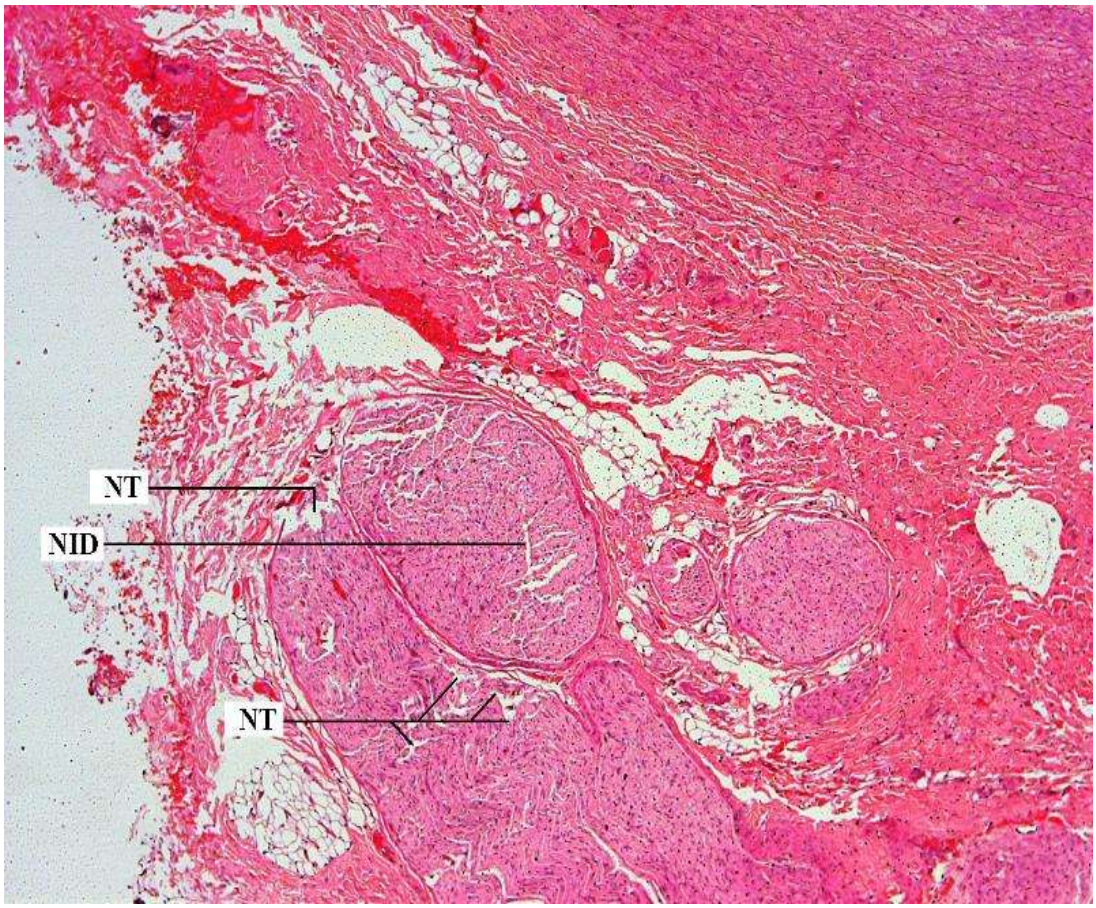


Fig. 7. 36a. A view of the **neural elements** in the tissues around the **left common carotid artery. Neural tearing** and **neural internal dehiscence** with haemorrhagic infiltration of the surrounding periadventitial tissues is noted. Again, multiple branches of the nerve are present. Case no. 45 (Haematoxylin and Eosin x 50). **NT** = neural tearing. **NID** = neural internal dehiscence.

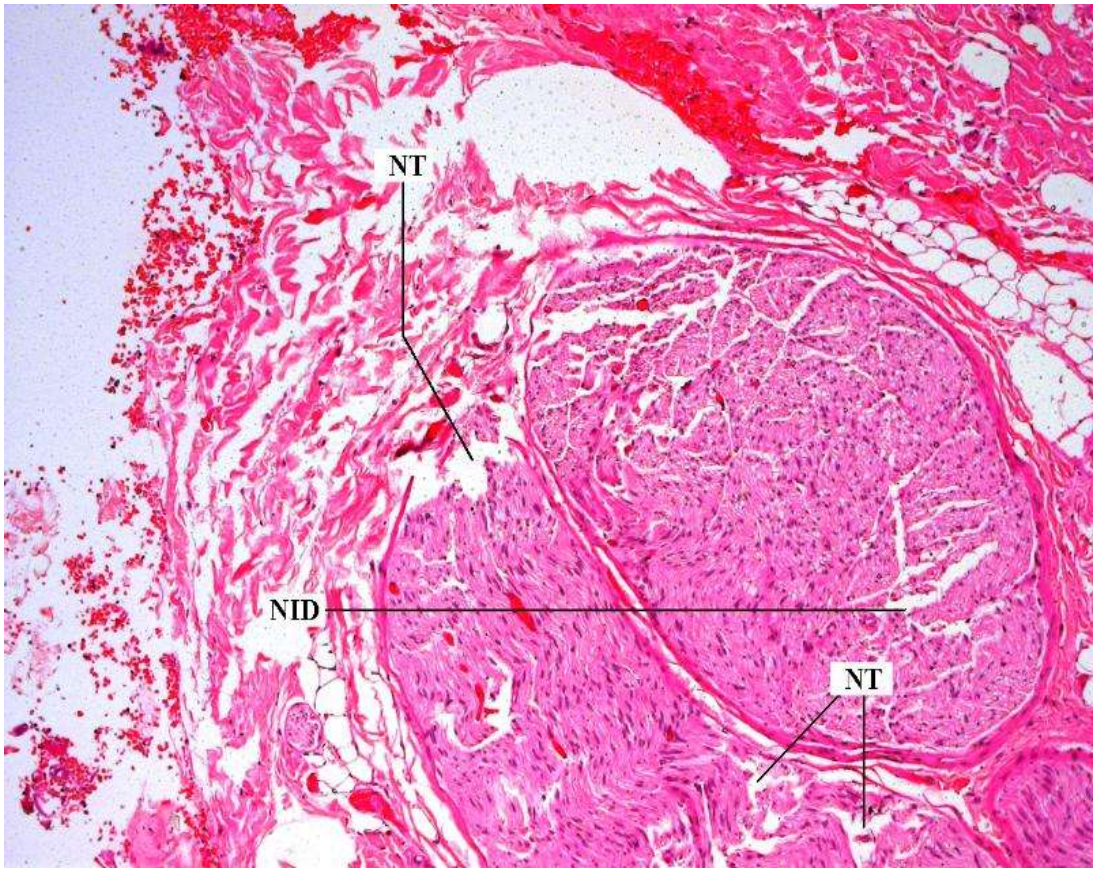


Fig. 7.36b. A high-power view of the **neural element** seen in Fig. 7.36a. **Neural tearing** and **neural internal dehiscence** are noted. Two branches of the nerve are noted. Case no. 45 (Haematoxylin and Eosin x 100). **NT** = neural tearing. **NID** = neural internal dehiscence.

Neural tearing of the **neural elements** around the **left internal carotid artery** was similarly noted in 7 subjects (14%) (Fig. 7. 32) while in the **neural elements** around the **left external carotid artery** it was noted in 4 subjects (8%) (Fig. 7. 33). The **neural elements** of the tissues around the **right common carotid artery** showed **neural tearing** in 7 subjects (14%) (Figs. 7.37 and 7. 38).

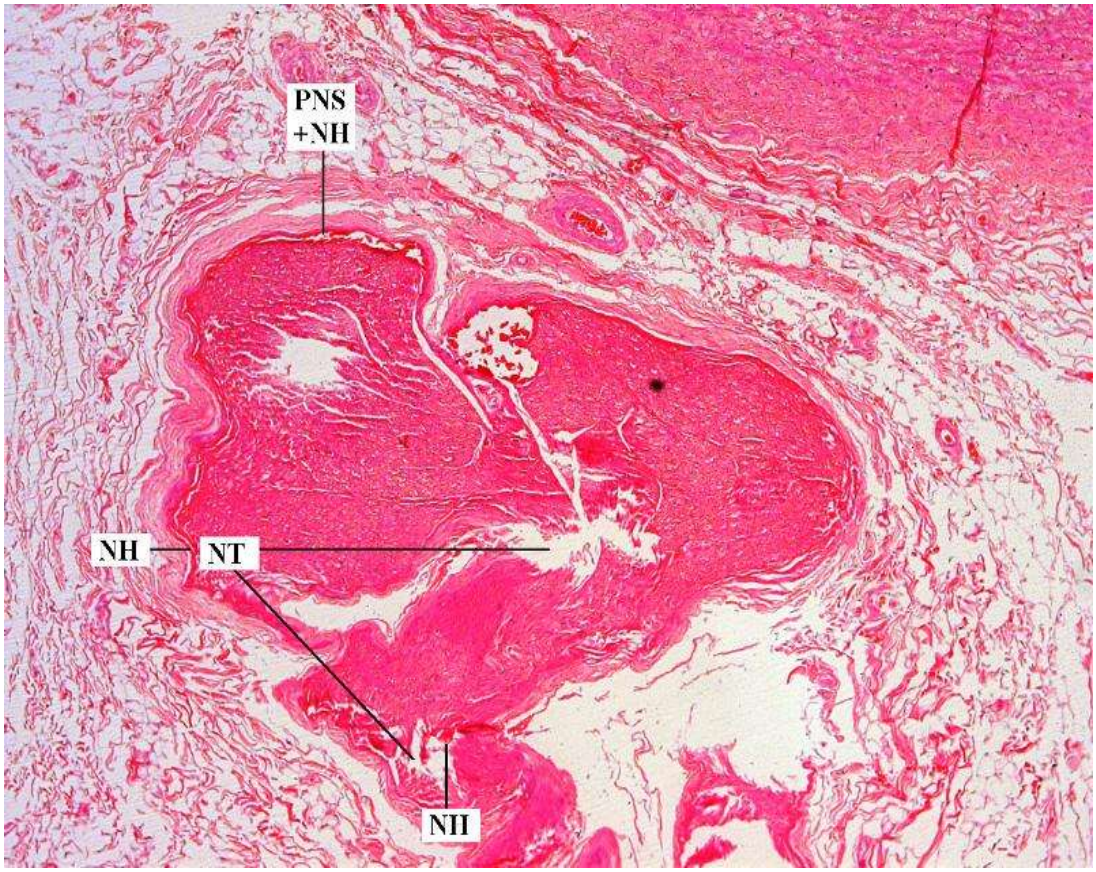


Fig. 7. 37. A view of a **neural element** in the tissues around the **right common carotid artery**, the latter seen in the upper right of the field. Severe **neural tearing**, **neural internal dehiscence**, **perineural separation** and **neural haemorrhage** are noted. Case no. 47 (Haematoxylin and Eosin x 50). **NT** = neural tearing. **NH** = neural haemorrhage. **PNS + NH** = perineural separation + neural haemorrhage.

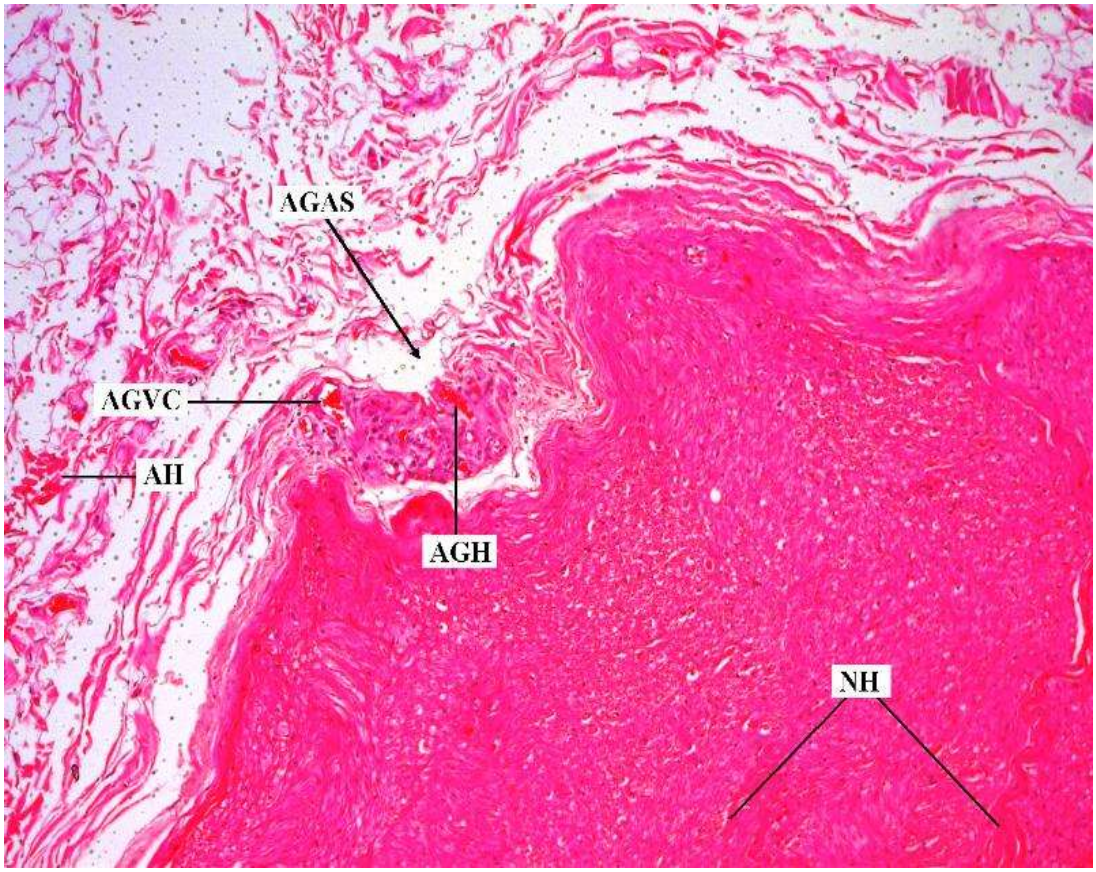


Fig. 7. 38. A view of the tissues around the **right internal carotid artery** showing what appears to be an **accessory glomus** in close apposition to but separate from a large **neural element** in which **neural haemorrhage** is noted. **Accessory glomal adventitial separation**, **glomal haemorrhage** and **glomal vascular congestion** are noted (arrow). **Adventitial haemorrhage** is, in addition, noted. Case no. 47 (Haematoxylin and Eosin x 100). **NH** = neural haemorrhage. **AGAS** = accessory glomal adventitial separation. **AGH** = accessory glomal haemorrhage. **AGVC** = accessory glomal vascular congestion. **AH** = adventitial haemorrhage.

The **neural elements** of the tissues around the **right internal carotid artery** showed **neural tearing** in 4 subjects (8%) (Fig. 7. 39).

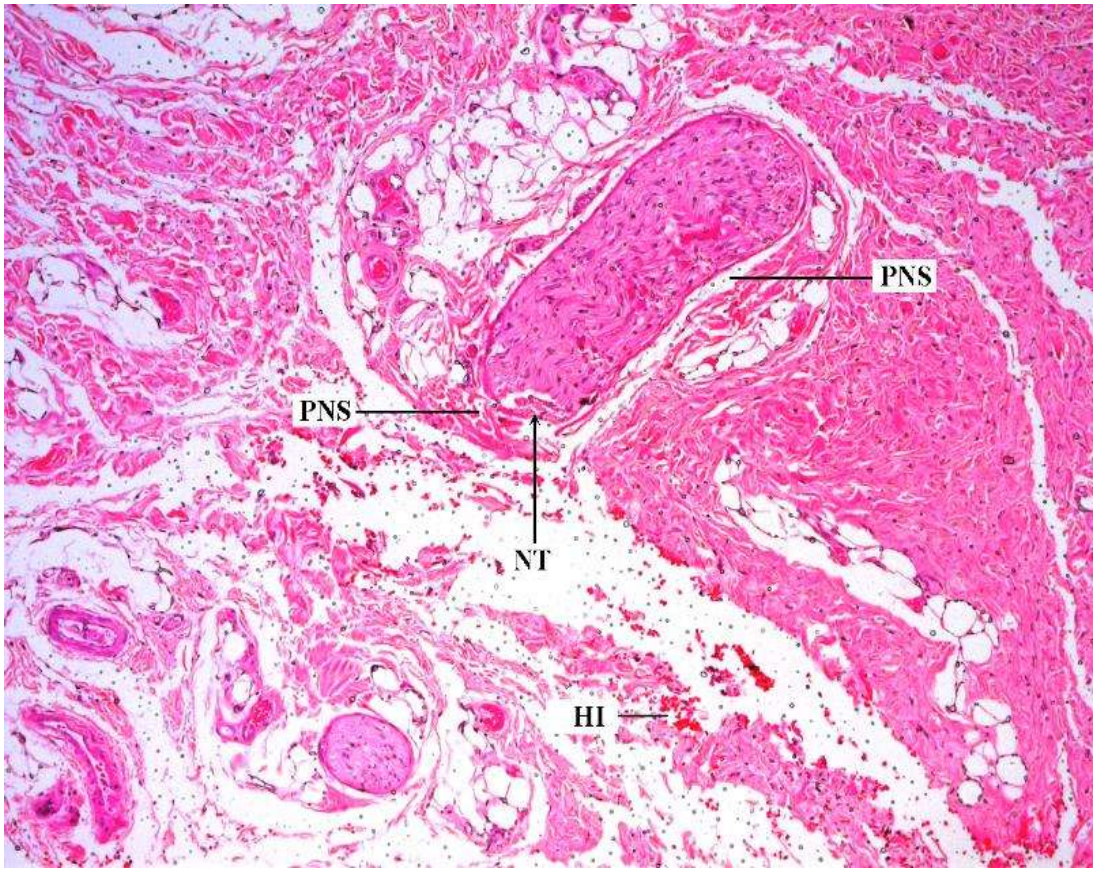


Fig. 7. 39. A view of a **neural element** in the tissues around the **right internal carotid artery.** Gross **neural tearing** is noted focally at the nerve periphery together with **perineural separation**. **Haemorrhage** is present within the periadventitial tissues. Case no. 45 (Haematoxylin and Eosin x 100). **NT** = neural tearing. **PNS** = perineural separation. **HI** = haemorrhagic infiltration.

The **neural elements** of the tissues around the **right external carotid artery** showed **neural tearing** in 5 subjects (10%) (Figs. 7.34 and 7.35) (*vide supra*).

Analysis of Findings

Left:	**Common carotid artery**	**7 (14%)**
	Internal carotid artery:	**7 (14%)**
	External carotid artery:	**4 (8%)**
		18

Right:	**Common carotid artery**	**7 (14%)**
	Internal carotid artery	**4 (8%)**
	External carotid artery	**5 (10%)**
		16

In other words, no real difference in incidence.

5. Perineural Separation

Perineural separation of the **neural elements** in the tissues around the **left common carotid artery** was noted in 21 subjects (42%) (Fig. 7. 31) while in the **neural elements** in the tissues around the **left internal carotid artery**, **perineural separation** was noted in 18 subjects (36%). In the **neural elements** of the tissues around the **left external carotid artery** it was noted in 24 subjects (48%) (Fig. 7.33). In the **neural elements** in the tissues around the **right common carotid artery**, **perineural separation** was noted in 22 subjects (44%), while in the **neural elements** in the tissues around the **right internal carotid artery**, **perineural separation** was noted in 11 subjects (22%) (Fig. 7.37). In the **neural elements** in the tissues around

the **right external carotid artery**, **perineural separation** was noted in 16 subjects (32%) (Figs. 7.34 and 7.35). This was not evident in any of the control sections examined.

Analysis of Findings

Left:	**Common carotid artery**	**21 (42%)**
	Internal carotid artery	**18 (36%)**
	External carotid artery	**24 (48%)**
		63

Right:	**Common carotid artery**	**22 (44%)**
	Internal carotid artery	**11 (22%)**
	External carotid artery	**16 (32%)**
		49

Comment. This common incidence, not noted in the control cases, highlights once again the tendency of tissues to split at interfaces in response to stretch. Tissue separation may be exaggerated by tensile forces experienced during the hanging process. Furthermore, if the ligature of hanging produces damage to arteries, it could reasonably be expected to produce damage to other structures. This issue is dealt with more fully in the Discussion chapter which follows.

B. The Accessory Glomi

As previously indicated, these were identified as smaller miniature bodies microscopically similar to the ‘traditional’ **carotid body** described in anatomical texts but seen histologically in the **adventitia** of the vessels near the carotid sinus.

1. Accessory Glomal Congestion

Accessory glomal congestion was noted in the **accessory glomi** in the adventitial tissues around the **left common carotid artery** in 3 subjects (6%), in the **accessory glomi** in the adventitial tissues around the **left internal carotid artery** in 2 subjects (4%), and in the **accessory glomi** in the adventitial tissues around the **left external carotid artery** in 4 subjects (8%). **Accessory glomal congestion** in the **accessory glomi** in the adventitial tissues around the **right common carotid artery** was noted in 4 subjects (8%) while the **accessory glomi** in the tissues around the **right internal carotid artery** showed **vascular congestion** in 1 subject (2%) (Fig. 7. 40).



Fig. 7. 40. For ease of reference, a view of the **accessory glomus** in close apposition to a large **neural element** in the adventitial tissues around the **right internal carotid artery** in Fig. 7. 38. **Accessory glomal haemorrhage** and **vascular congestion** are noted. Case no. 47 (Haematoxylin and Eosin x 100). **AGH** = accessory glomal haemorrhage. **AGVC =** accessory glomal vascular congestion.

In the **accessory glomi** in the adventitial tissues around the **right external carotid artery**, **vascular congestion** was noted in 16 subjects (32%)

(Figs. 7. 30a and 7. 30b).

2. <u>Accessory Glomal Haemorrhage</u>

Accessory glomal haemorrhage was noted in the **accessory glomi** in the adventitial tissues around the **left common carotid artery** in 1 subject (2%), but not in the **accessory glomi** in the adventitial tissues around the **left internal carotid artery** in any of the subjects (0%). **Haemorrhage** in the **accessory glomi** in the adventitial tissues around the **left external carotid artery** was noted in 3 subjects (6%). In the **accessory glomi** in the adventitial tissues around the **right common carotid artery**, **haemorrhage** was noted in 2 subjects (4%) while in the **accessory glomi** in the adventitial tissues around the **right internal carotid artery**, like the left, no **haemorrhage** was noted in any of the subjects (0%). The **accessory glomi** in the adventitial tissues around the **right external carotid artery** showed **haemorrhage** in 1 subject (2%).

In summary, a very low incidence of **accessory glomal haemorrhage** was noted. The possible reasons for this low incidence are discussed more fully in the Discussion chapter which follows.

3. <u>Accessory Glomal Internal Dehiscence</u>

Accessory glomal internal dehiscence was noted in the **accessory glomi** in the adventitial tissues around the **left common carotid artery** in 12 subjects (24%), in the **accessory glomi** in the adventitial tissues around the **left internal carotid artery** in 10 subjects (20%), and in the **accessory glomi** in the adventitial tissues around the **left external carotid artery** in 9 subjects (18%). The **accessory glomi** in the

adventitial tissues around the **right common carotid artery** showed **internal dehiscence** in 6 subjects (12%) (Fig.7. 41).



Fig. 7. 41. A view of a **neural element** and its incorporated **accessory glomus** in the adventitial tissues around the **right common carotid artery** showing marked **neural internal dehiscence**, **perineural separation** and **accessory glomal adventitial separation**. Case no. 29 (Haematoxylin and Eosin x 100). **N** = neural element. **NID** = neural internal dehiscence. **PNS** = perineural separation. **AG** = accessory glomus. **AGAS** = accessory glomal adventitial separation.

The **accessory glomi** in the adventitial tissues around the **right internal carotid artery** showed **internal dehiscence** in 3 subjects (6%), while those in the adventitial tissues around the **right external carotid artery** showed **internal dehiscence** in 10 subjects (20%). The reason for the greater incidence of **internal dehiscence** noted in

the **right external carotid artery** as compared with the **right internal carotid artery** may lie in the more exposed position of the **right external carotid artery** in comparison with the **right internal carotid artery**.

4. Accessory Glomal Tearing

Accessory glomal tearing was noted in the **accessory glomi** in the adventitial tissues around the **left common carotid artery** in 5 subjects (10%), in the **accessory glomi** in the adventitial tissues around the **left internal carotid artery** in 2 subjects (4%), and in the tissues around the **left external carotid artery** in 5 subjects (10%). The **accessory glomi** in the adventitial tissues around the **right common carotid artery** showed **tearing** in 3 subjects (6%). The **accessory glomi** in the tissues around the **right internal carotid artery** showed **tearing** in 2 subjects (4%), while those in the adventitial tissues around the **right external carotid artery** showed **tearing** in 1 subject (2%).

In summary, therefore, **accessory glomal tearing** was noted in about one-third of the cases.

Analysis of Findings

Left: Common carotid artery: 5 (10%)

Internal carotid artery: 2 (4%)

External carotid artery: 5 (10%)

12

Right: Common carotid artery: 3 (6%)

Internal carotid artery:	**2 (4%)**
External carotid artery:	**1 (2%)**
	6

In short, a considerable difference in incidence between the left and right sides. Interestingly, this correlates with the incidence of **adventitial haemorrhage** which showed a greater incidence on the left as compared with the right but not, however, with the incidence of **adventitial tearing**, which showed a greater preponderance on the right. Could this possibly be due to the greater resistance to tearing by stretch forces on the part of the spherical **accessory glomi** in comparison with the longitudinal **arteries**, as well as the fact that they are embedded in connective tissue and have on one side a flexible (fluid containing) structure, i.e. opposite the force applied by the ligature ? This issue is discussed in detail in the Discussion chapter which follows.

5. Accessory Glomal Adventitial Separation

Accessory glomal adventitial separation was noted in the adventitial tissues around the **left common carotid artery** in 19 subjects (38%) (Figs. 7.42a and 7.42b).

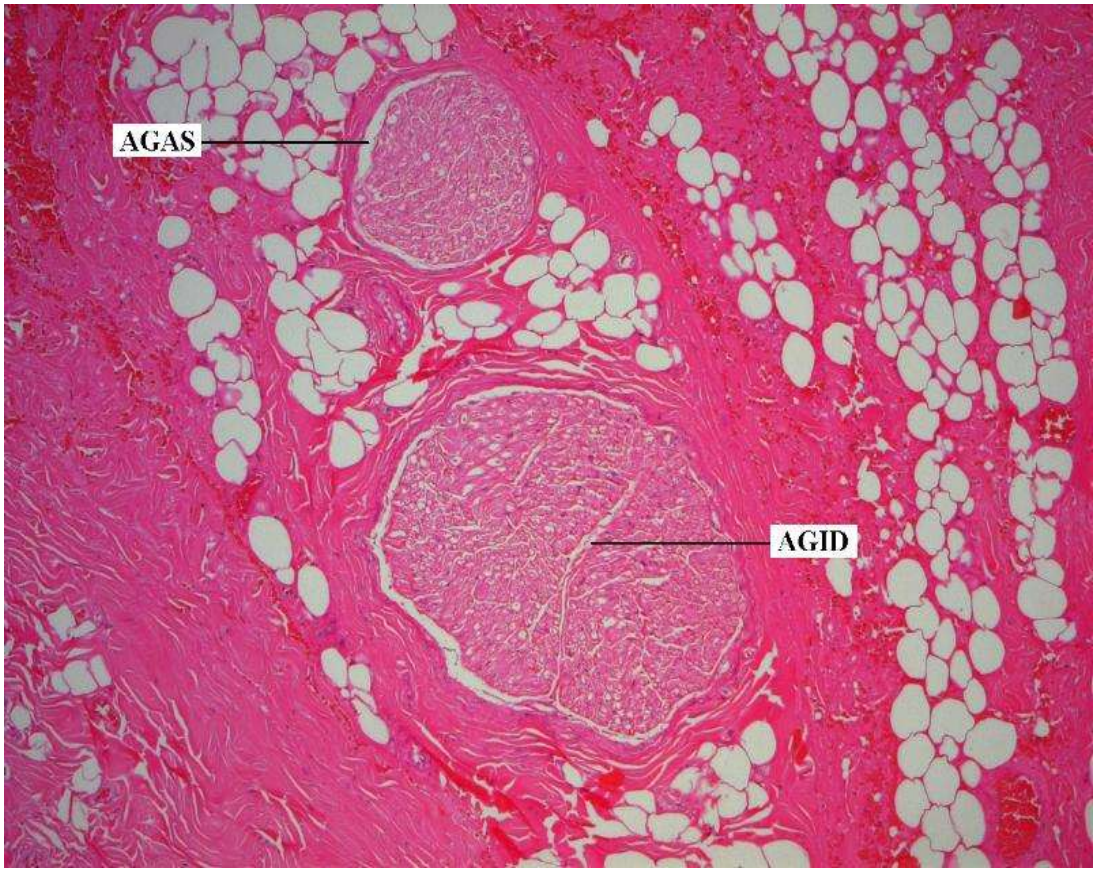


Fig. 7. 42a. A view of two **accessory glomi** in the tissues around the **left common carotid artery**. **Accessory glomal adventitial separation** is noted at their medial aspect together with some degree of **internal dehiscence**. Haemorrhagic infiltration of the surrounding fibro-fatty connective tissues is noted. Case no. 42 (Haematoxylin and Eosin x 50). **AGID** = accessory glomal internal dehiscence. **AGAS** = accessory glomal adventitial separation.

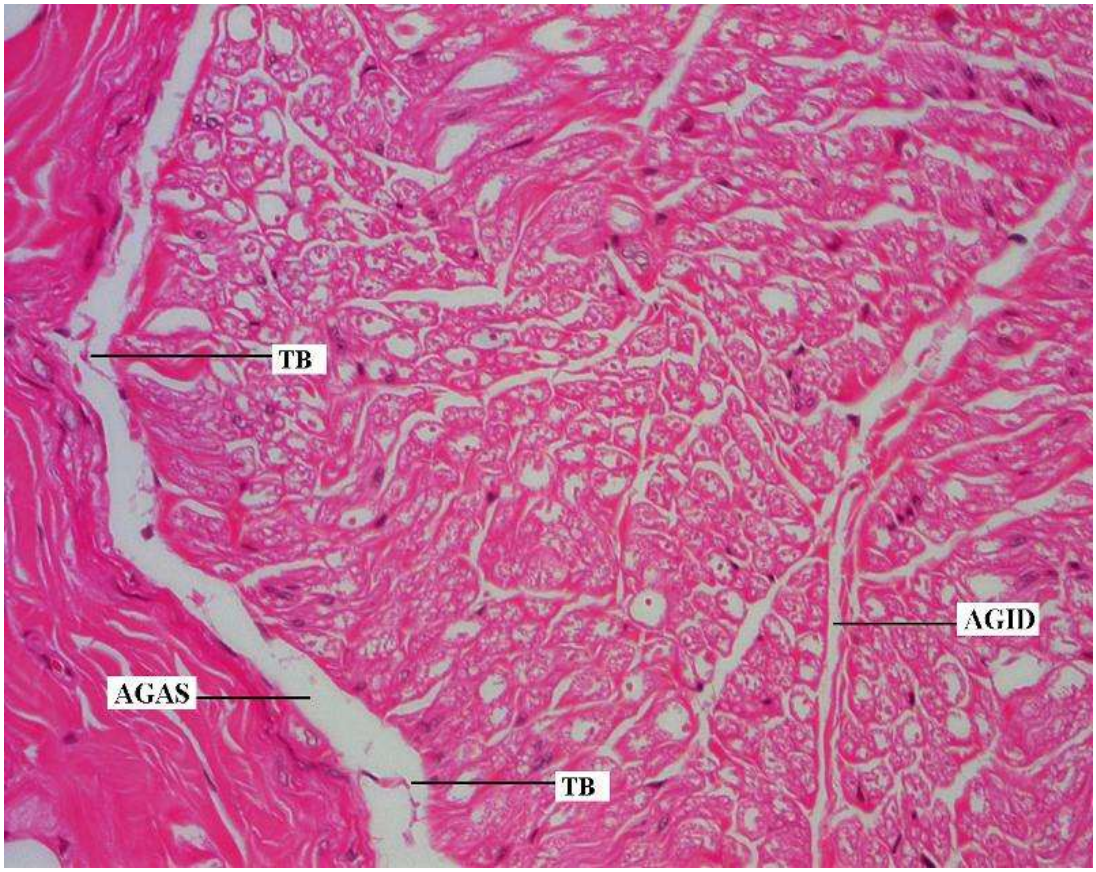


Fig. 7.42b. A high-power view of the larger of the two **accessory glomi** seen in the tissues noted around the **left common carotid artery** in Fig.7.42a. Marked **internal dehiscence** and **adventitial separation** is noted. Case no. 42 (Haematoxylin and Eosin x 100). **AGID** = accessory glomal internal dehiscence. **AGAS** = accessory glomal adventitial separation. **TB** = tissue bridge.

In the **accessory glomi** in the adventitial tissues around the **left internal carotid artery**, **adventitial separation** was noted in 26 subjects (52%), while those in the adventitial tissues around the **left external carotid artery** showed **adventitial separation** in 24 subjects (48%). The **accessory glomi** in the adventitial tissues around the **right common carotid artery** showed **adventitial separation** in 19 subjects (38%) (Fig. 7. 41), while those in the adventitial tissues around the **right**

internal carotid artery showed **adventitial separation** in 14 subjects (28%) (Figs. 7. 43a and 7. 43b).

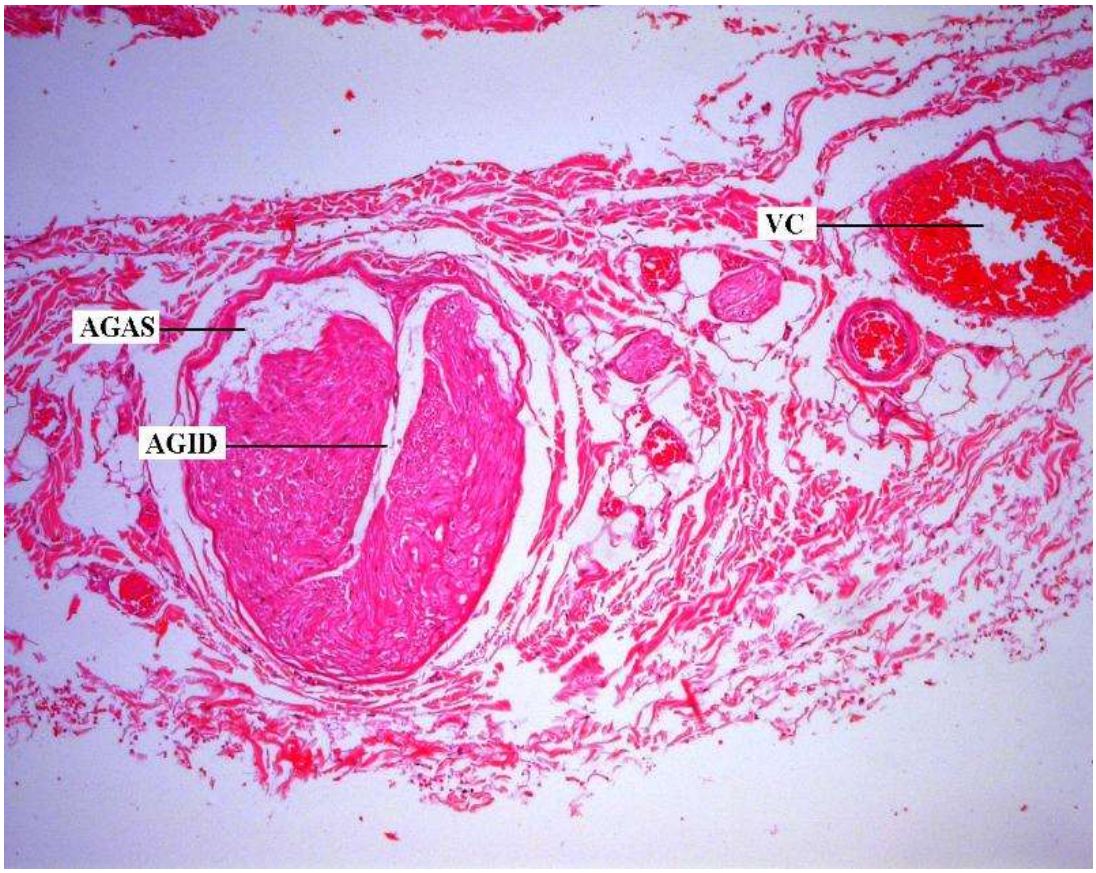


Fig. 7. 43a. An **accessory glomus** in the tissues around the **right internal carotid artery** showing marked **accessory glomal internal dehiscence** and **adventitial separation** is also noted. **Vascular congestion** within the surrounding connective tissues is present. Case no. 29 (Haematoxylin and Eosin x 100). **VC** = vascular congestion. **AGAS** = accessory glomal adventitial separation. **AGID** = accessory glomal internal dehiscence.

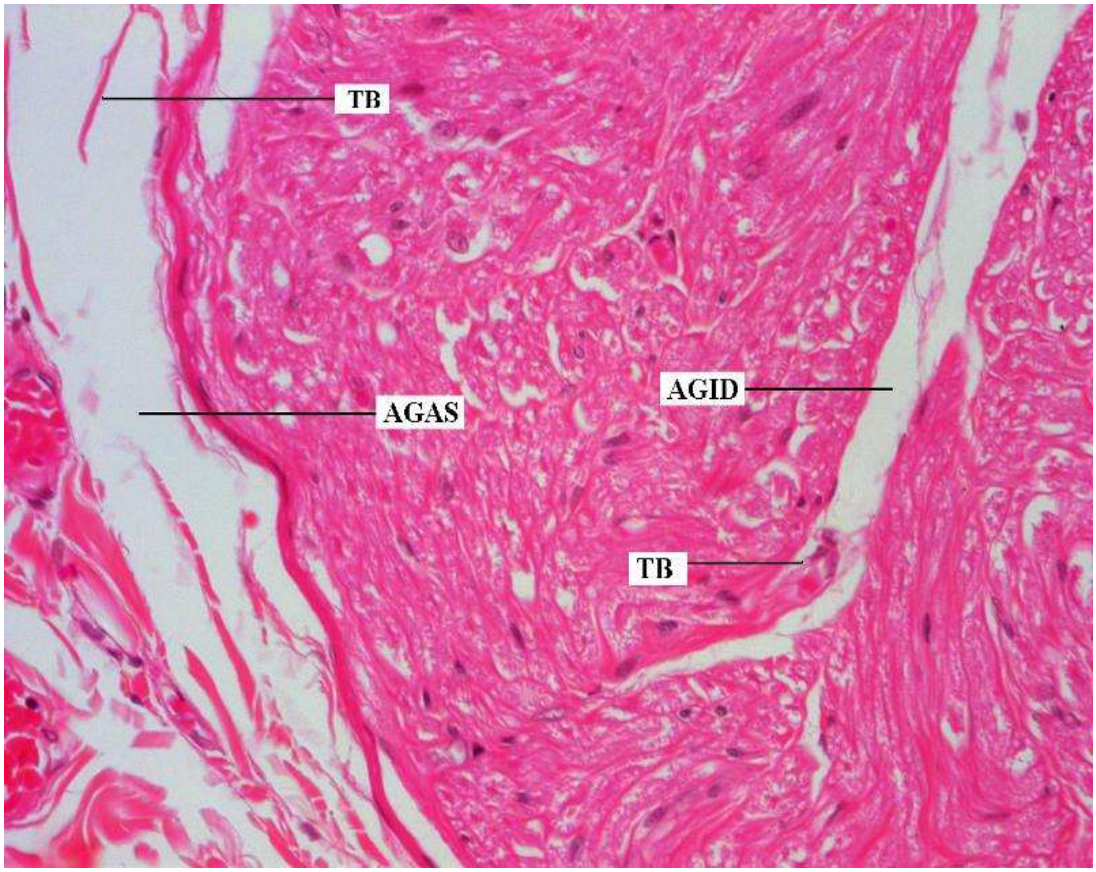


Fig. 7.43b. A high-power view of the **accessory glomus** seen in the tissues around the **right internal carotid artery** in Fig. 7.43a. Marked **internal dehiscence** and **adventitial separation** is noted. Case no 29 (Haematoxylin and Eosin x 400). **AGID** = accessory glomal internal dehiscence. **AGAS** = accessory glomal adventitial separation. **TB =** tissue bridge

The **accessory glomi** in the adventitial tissues around the **right external carotid artery** showed **adventitial separation** in 22 subjects (44%).

Analysis of Findings

Left:	**Common carotid artery:**	**19 (38%)**
	Internal carotid artery:	**26 (52%)**
	External carotid artery:	**24 (48%)**
		69

Right:	**Common carotid artery:**	**19 (38%)**
	Internal carotid artery:	**14 (28%)**
	External carotid artery:	**22 (44%)**
		55

As with **perineural separation**, these values are large but, as previously indicated, great care and gentleness was taken in the anatomical dissection with great effort being made to avoid teasing apart and 'technical damage' of the tissues. This would therefore tend to negate the possibility that the **accessory glomal adventitial separation** noted was artefactual. The basis for this **separation** and the **biomechanical factors** involved in producing this **separation** are discussed in detail in the Discussion chapter which follows.

C. The Neural Ganglia

1. Intraganglionic Congestion

Intraganglionic congestion was noted in the **ganglia** adjacent to the **left common carotid artery** in 1 subject (2%) (Fig. 7. 44a and 7.44b).

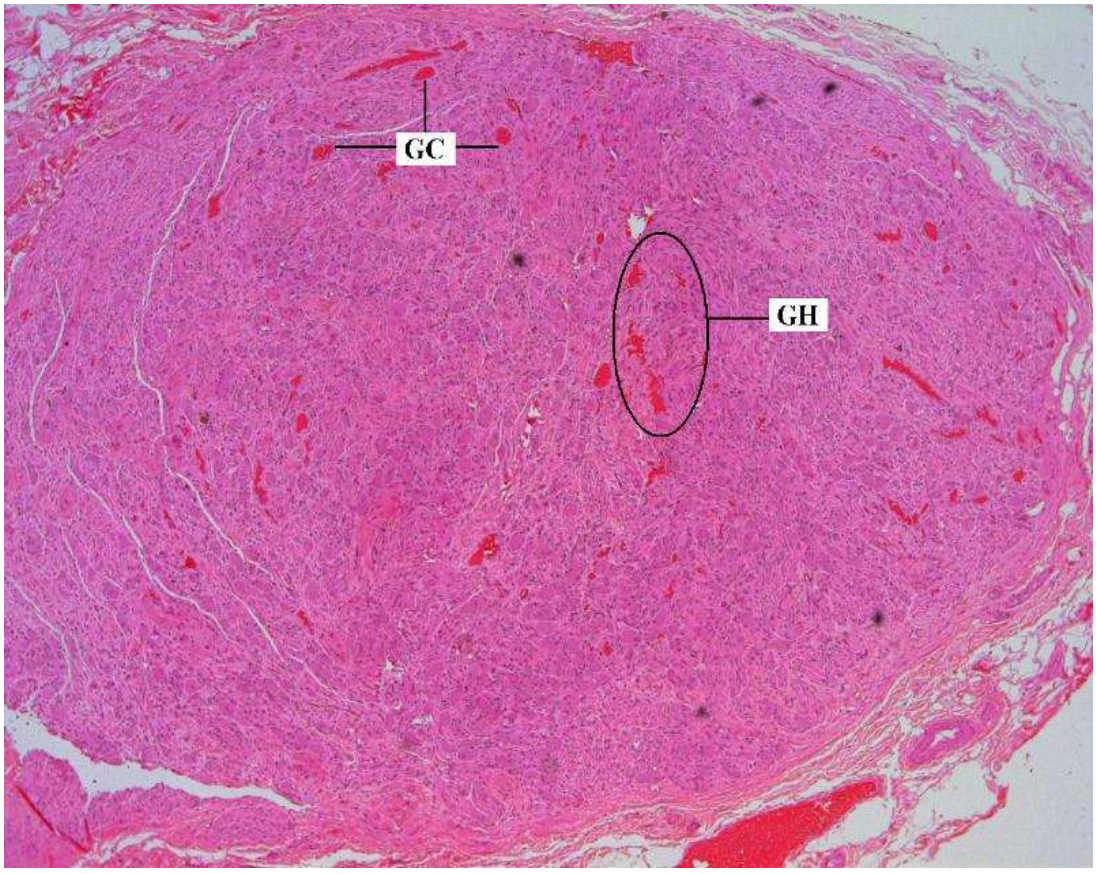


Fig. 7.44a. A view of a **ganglion** in the tissues adjacent to the **left common carotid artery**. **Ganglionic haemorrhage** and **intraganglionic congestion** are noted. Case no. 45 (Haematoxylin and Eosin x 50). **GH** = ganglionic haemorrhage. **GC** = ganglionic congestion.

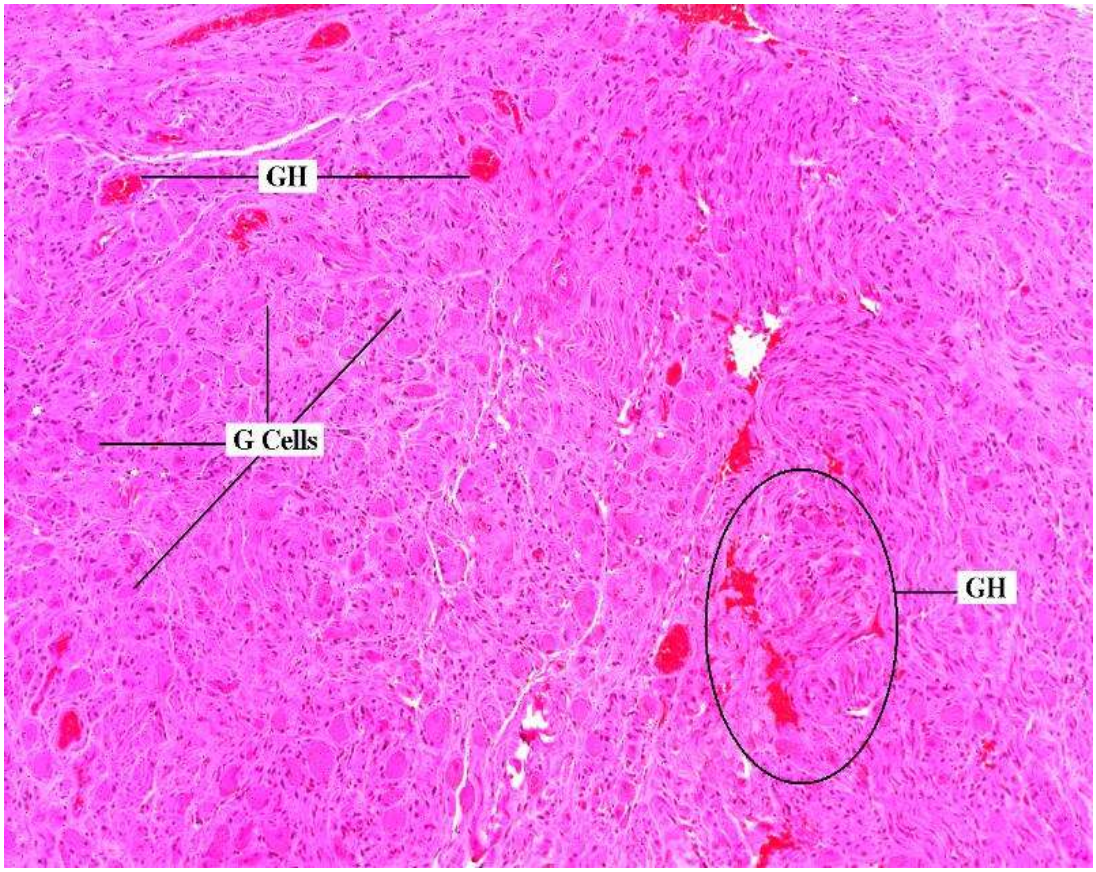


Fig. 7. 44b. A high-power view of the **ganglion** seen in the tissues adjacent to the **left common carotid artery** in Fig. 7.44a. **Intraganglionic congestion**, **ganglionic haemorrhage** and **ganglion cells** are noted. Case no.45 (Haematoxylin and Eosin x 100). **GH** = ganglionic haemorrhage. **G cells** = ganglion cells.

In the **ganglia** adjacent to the **left internal carotid artery**, **vascular congestion** was noted in 3 subjects (6%) (Fig. 7. 45).

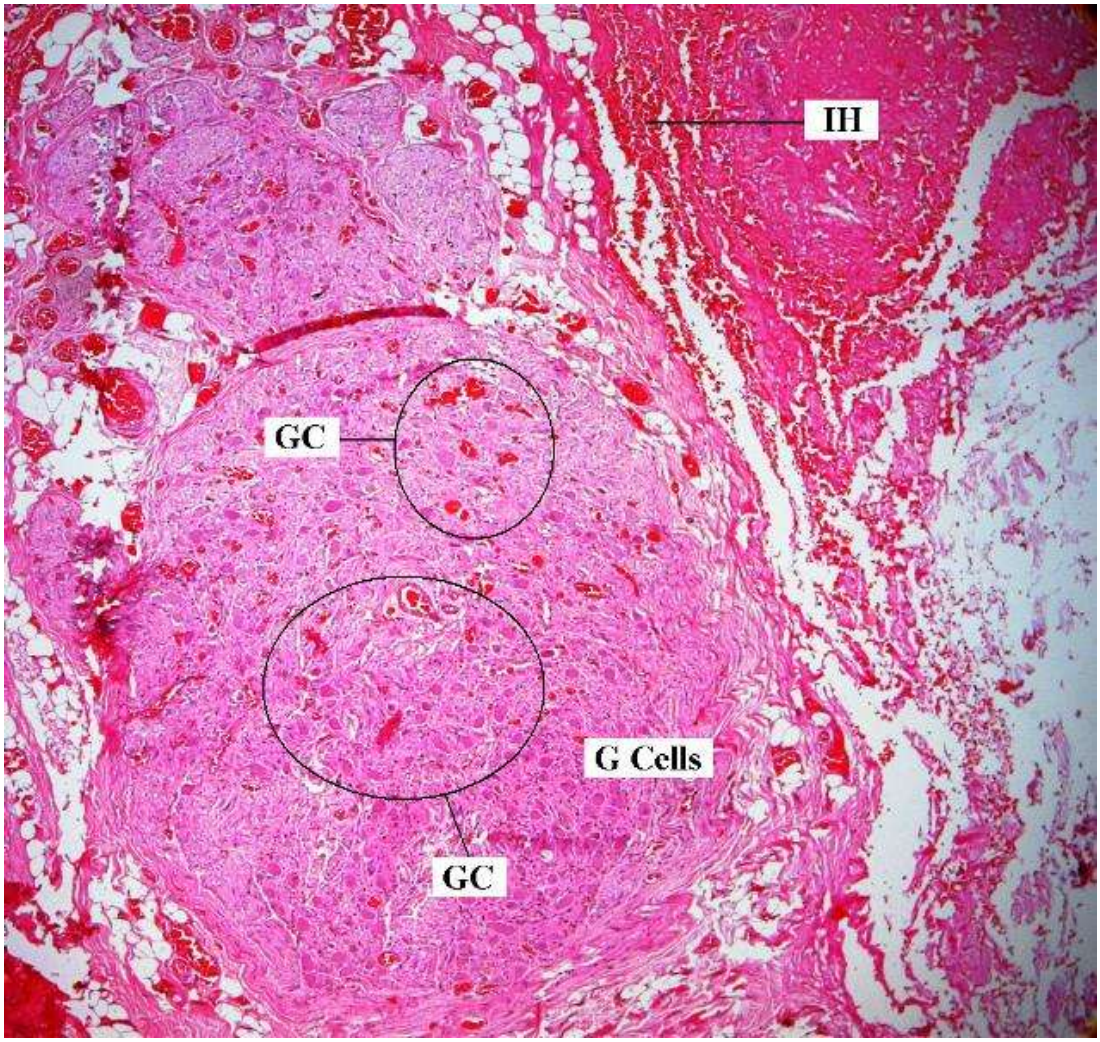


Fig. 7.45. A view of a **ganglion** in the tissues adjacent to the **left internal carotid artery** showing marked **intraganglionic vascular congestion**. Marked **haemorrhage** is noted in the surrounding interstitial connective tissues. Case no. 42 (Haematoxylin and Eosin x 100). **GC** = ganglionic congestion. **G Cells** = ganglion cells. **IH** = interstitial haemorrhage.

In the **ganglia** adjacent to the **left external carotid artery**, **vascular congestion** was noted in 7 subjects (14%) (Figs. 7. 46a and 7. 46b).

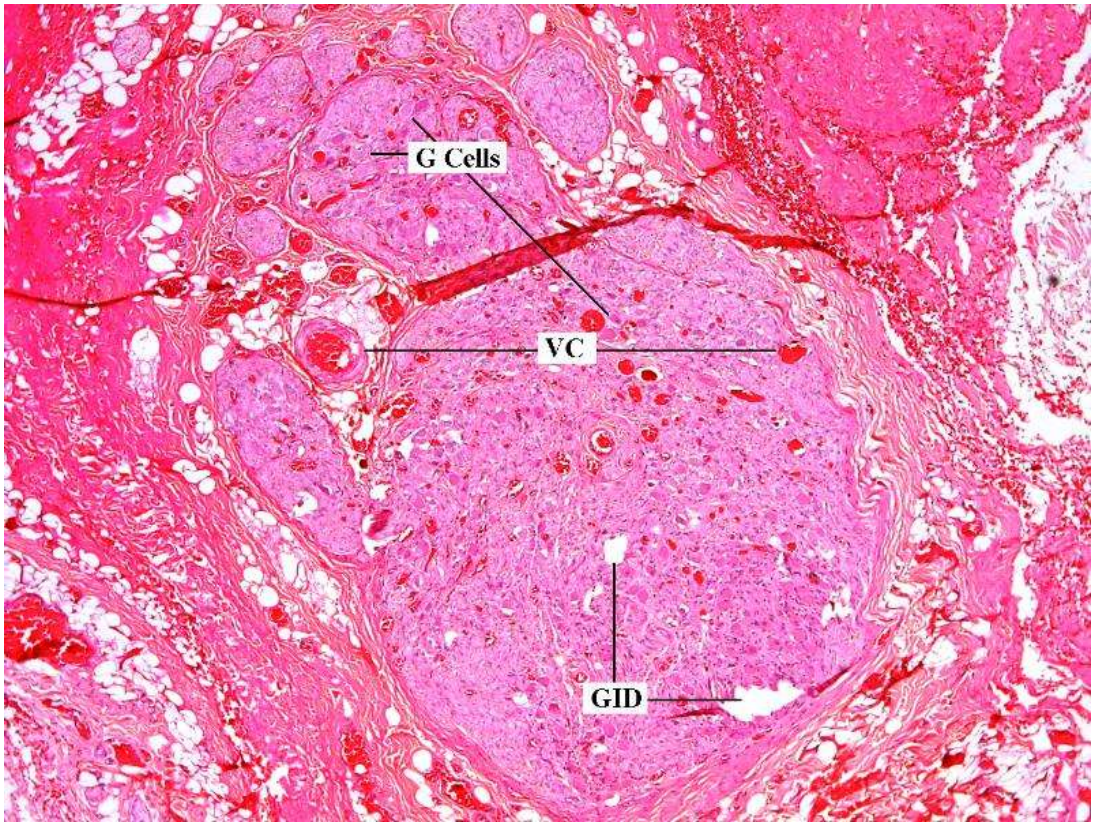


Fig. 7.46a. A view of a **ganglion** in the tissues adjacent to the **left external carotid artery.** Marked **haemorrhage** of the surrounding interstitial tissues is noted as well as **intraganglionic and periganglionic vascular congestion.** Areas of **ganglionic internal disruption** are, in addition, noted. Case no 42 (Haematoxylin and Eosin x 50).

VC = vascular congestion. **GID** = ganglionic internal disruption. **G Cells** = ganglion cells.

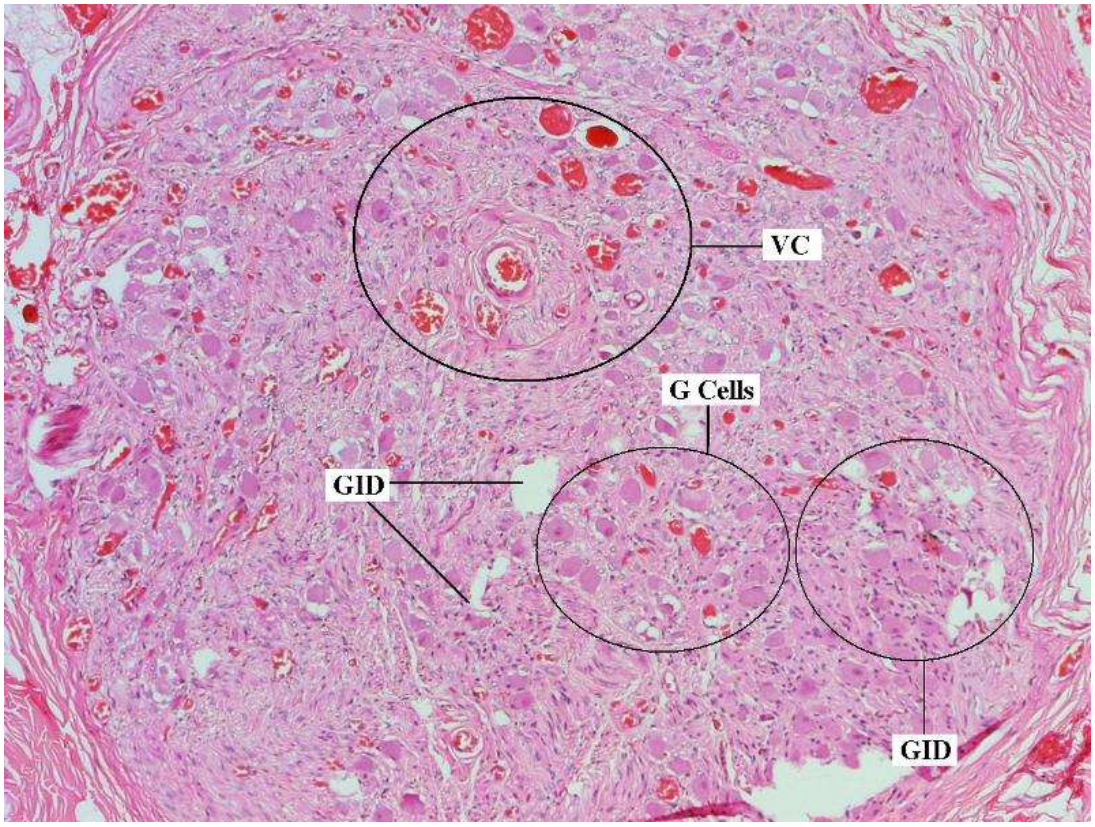


Fig. 7. 46b. A high-power view of the **ganglion** seen in the tissues adjacent to the **left external carotid artery** in Fig. 7.46a. Marked **intraganglionic vascular congestion** together with surrounding **periganglionic congestion** is noted. Areas of **ganglionic internal disruption** are noted. Case no 42 (Haematoxylin and Eosin x 100). **VC** = vascular congestion. **GID** = ganglionic internal disruption. **G Cells** = ganglion cells.

In the **ganglia** adjacent to the **right common carotid artery**, the **right internal carotid artery**, and the **right external carotid artery**, **vascular congestion** was noted in decreasing order of frequency, being seen in 4 subjects (8%), 3 subjects (6.0%), and 2 subjects (4%) each, respectively.

Analysis of Findings

Left: **Common carotid artery: 1 (2%)**

Internal carotid artery: 3 (6%)

External carotid artery: 7 (14%)

11

Right: **Common carotid artery: 4 (8%)**

Internal carotid artery: 3 (6%)

External carotid artery: 2 (4%)

9

In essence, no appreciable difference between the left and right sides

2. Ganglionic Haemorrhage

Ganglionic haemorrhage was noted in the **ganglia** adjacent to the **left common carotid artery** in 2 subjects (4%) (Fig. 7. 47).

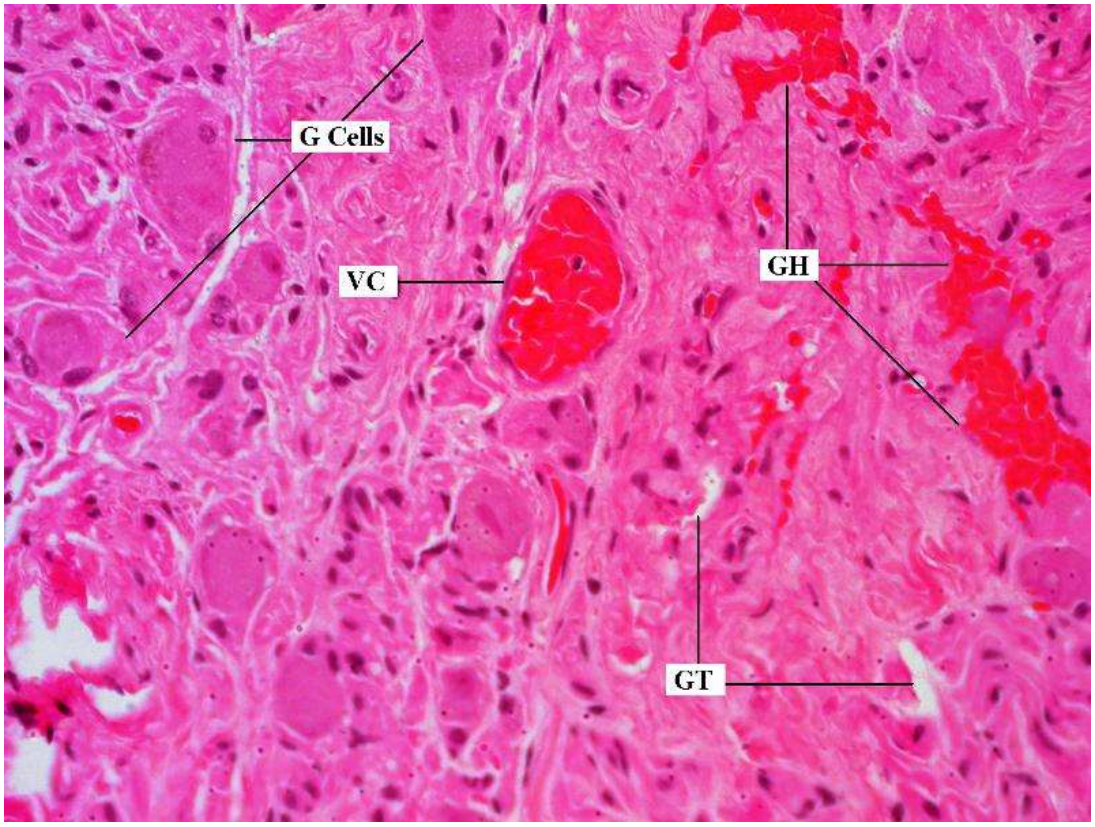


Fig. 7. 47. A high power view of the **ganglion** adjacent to the **left common carotid artery** in Fig. 7. 44 showing **vascular congestion, ganglionic tearing** and **ganglionic haemorrhage.** Numerous **ganglion cells** are noted.. Case no. 45 (Haematoxylin and Eosin x 400). **G Cells** = ganglion cells. **GH =** ganglionic haemorrhage. **GT =** ganglionic tearing. **VC** = vascular congestion.

Haemorrhage was noted in the **ganglia** around the **left internal carotid artery** in 2 subjects (4%), in the **ganglia** around the **right internal carotid artery** in 4 subjects (8%), but not around any of the other arteries.

Analysis of Findings

Left:	**Common carotid artery:**	**2 (4%)**
	Internal carotid artery:	**2 (4%)**
	External carotid artery:	**0 (0%)**
		4

Right:	**Common carotid artery:**	**0 (0%)**
	Internal carotid artery:	**4 (8%)**
	External carotid artery:	**0 (0%)**
		4

Thus, an equal incidence between the left and right sides.

3. Ganglionic Internal Dehiscence

Ganglionic internal dehiscence was noted in the **ganglia** adjacent to the **left common carotid**, the **left internal carotid** and the **right common carotid arteries** in 2 subjects (4%) each, in the **ganglia** adjacent to the **left external carotid artery** in 5 subjects (10%), the **right internal carotid artery** in 3 subjects (6%) (Fig. 7.48), and in the **ganglia** adjacent to the **right external carotid artery** in 1 subject (2%).

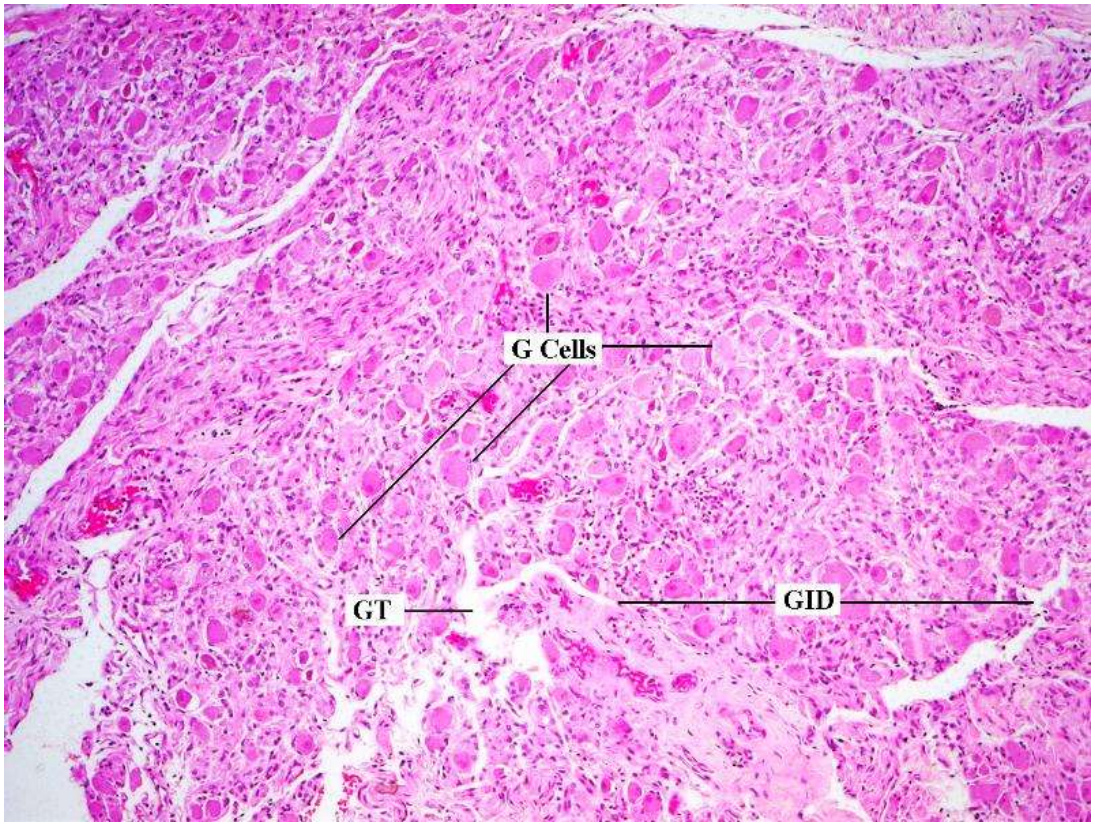


Fig. 7. 48. A view of a **ganglion** in the tissues adjacent to the **right internal carotid artery. Ganglionic internal dehiscence** and **ganglionic tearing** is noted. Case no. 4 (Haematoxylin and Eosin x 100). **G Cells** = ganglion cells. **GT** = ganglionic tearing. **GID** = ganglionic internal dehiscence

Analysis of Findings

Left:	**Common carotid artery:**	**2 (4%)**
	Internal carotid artery:	**2 (4%)**
	External carotid artery:	**5 (10%)**
		9

Right:	**Common carotid artery:**	**2 (4%)**
	Internal carotid artery:	**3 (6%)**
	External carotid artery:	**1 (2%)**
		6

4. Ganglionic Tearing

Tearing or damage to the **ganglia** adjacent to the **left common carotid artery** was noted in 2 subjects (4%) (Fig. 7. 47) while in the **ganglia** adjacent to the **left internal carotid**, the **left external carotid**, the **right common carotid** and the **right internal carotid arteries ganglionic tearing** was noted in 1 subject (2%), each (Fig. 7. 48). In the **ganglia** adjacent to the **right external carotid artery** no **tearing** was noted in any of the subjects (0%).

Analysis of Findings

Left:	**Common carotid artery:**	**2 (4%)**
	Internal carotid artery:	**1 (2%)**
	External carotid artery:	**1 (4%)**
		4

Right:	**Common carotid artery:**	**1 (2%)**
	Internal carotid artery:	**1 (2%)**
	External carotid artery:	**0 (0%)**
		2

A small incidence overall, although a greater incidence on the left side.

In general, it seems that no structures related to the **arteries**, **veins** and **nerves** in the upper part of the neck are immune to trauma in suicidal hanging.

D. The Carotid Body

As previously indicated in the Material and Methods chapter and as described in Chapter 3 dealing with the anatomy of the neck, the **carotid body** was identified on each side of the neck as a 5 – 7 mm in diameter reddish-brown ovoid structure situated near the bifurcation of the **common carotid artery** into its respective **internal carotid artery** and **external carotid artery** branches. It was further identified by its histological structure.

1. Carotid Body Congestion

Vascular congestion of the **carotid body** near the bifurcation of the **left common carotid artery** was not noted in any of the subjects (0%) (no glomi occur below the bifurcation), but was seen in the **carotid body** near the **left internal carotid artery** in 3 subjects (6%). The **carotid body** near the **left external carotid artery** showed **vascular congestion** in 1 subject (2%) (Fig. 7. 49).

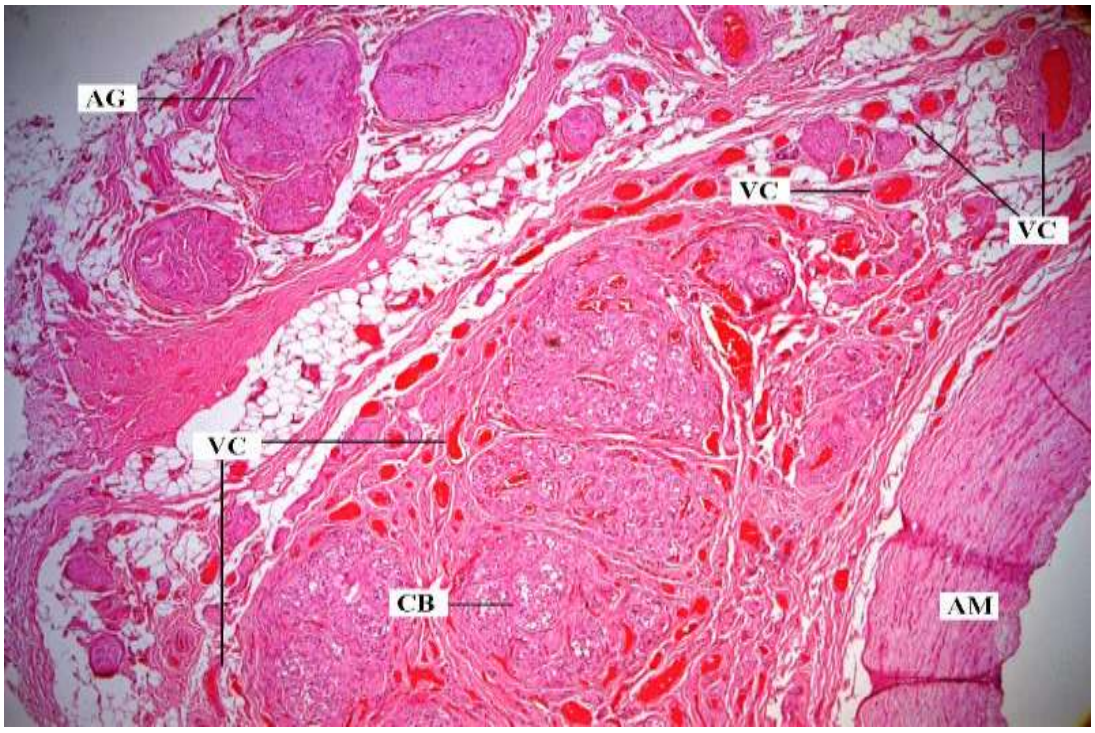


Fig. 7. 49. A view of the marked **vascular congestion** within and surrounding the multicentric **carotid body** near the **left external carotid artery** is noted. Several **accessory glomi** to the **carotid body** are present. Part of the circumference of the **arterial wall** is seen. Case no. 44 (Haematoxylin and Eosin x 50). **AG** = accessory glomus. **VC** = vascular congestion. **AM** = arterial media (muscularis). **CB** = multicentric carotid body.

A **carotid body** with a lesser degree of **vascular congestion** (Fig. 7.50) but highlighting the multicentric nature of the structure is shown for comparison with Fig. 7. 49.

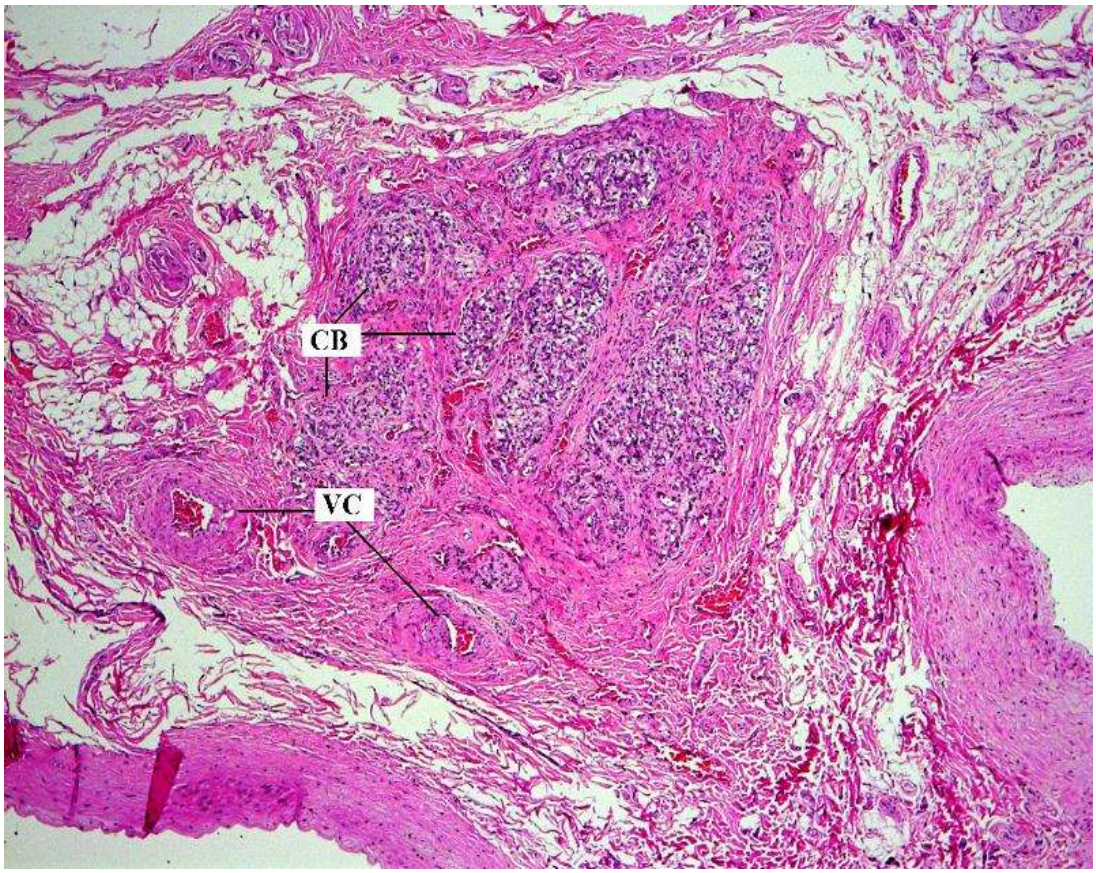


Fig. 7. 50. A **carotid body** with some degree of **vascular congestion** but showing the multicentric nature of the structure. Case no. 19 (Haematoxylin and Eosin x 100). **CB** = carotid body. **VC** = vascular congestion.

The **carotid body** near the **right common carotid artery** showed **vascular congestion** in 1 subject (2%) (Fig. 7. 51).

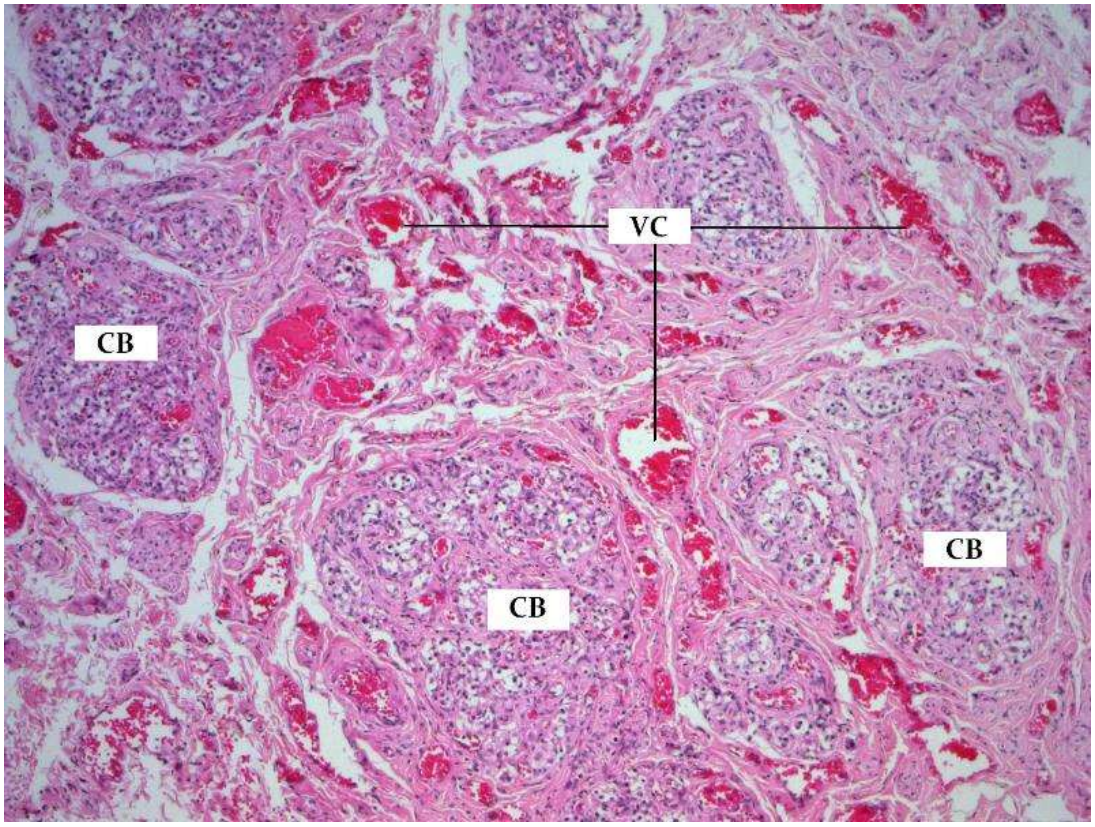


Fig. 7. 51. A multicentric **carotid body** near the bifurcation of the **right common carotid artery** showing **vascular congestion**. Case no. 14 (Haematoxylin and Eosin x 100). **CB** = carotid body. **VC** = vascular congestion.

The **carotid body** near the **right internal carotid artery** showed **vascular congestion** in 2 subjects (4%), while the **carotid body** near the **right external carotid artery** showed **congestion** in 1 subject (2%) (Figs. 7. 52a and 7.52b).

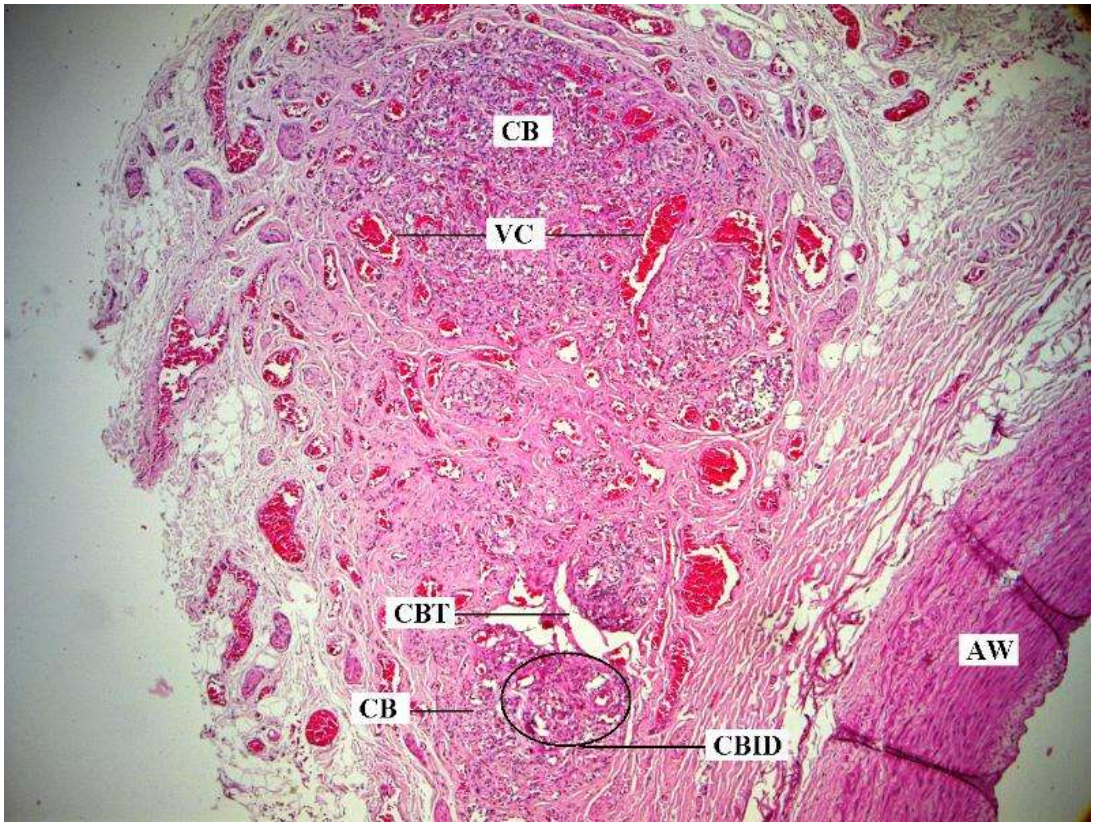


Fig. 7.52a. A view of the multicentric **carotid body** structure near the **right external carotid artery** (bottom right of field) is seen. **Vascular congestion**, **internal dehiscence** and **tearing** of the **carotid body** are noted. Case no. 3 (Haematoxylin and Eosin x 50). **CB** = carotid body. **VC** = vascular congestion. **AW** = arterial wall. **CBT** = carotid body tearing. **CBID** = carotid body internal dehiscence.

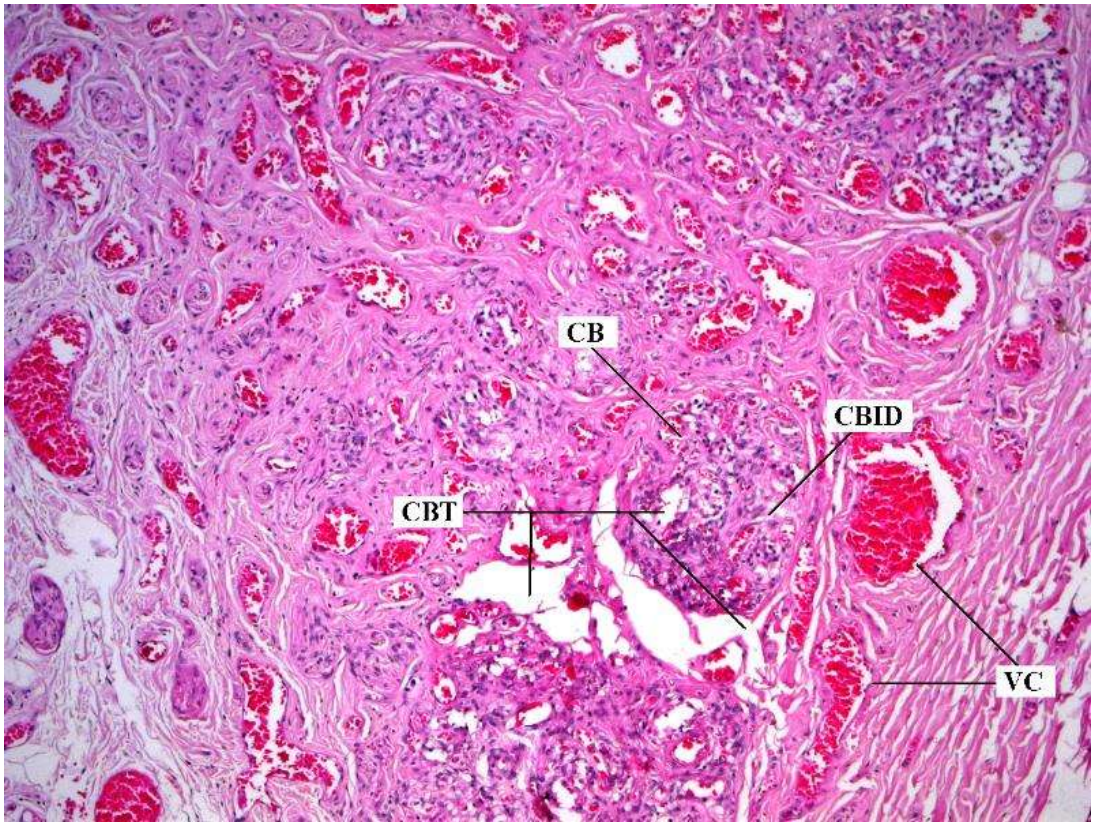


Fig. 7. 52b. A slightly higher-power view of the multicentric **carotid body** with satellite **accessory glomi** near the **right external carotid artery** seen in Fig. 7.52. Marked **vascular congestion**, **tearing** and **internal dehiscence** is noted. Case no. 3 (Haematoxylin and Eosin x 80). **CB** = carotid body. **VC** = vascular congestion. **CBT** = carotid body tearing. **CBID** = carotid body internal dehiscence.

These **accessory glomi** appear to lie in the adventitia which is continuous with the connective tissue of the **carotid sheath**. Thus, it would not be surprising if they were damaged by the pressure of the ligature.

2. Carotid Body Haemorrhage

Carotid body haemorrhage was an uncommon finding, being absent in the **carotid body** near the **left common carotid**, the **left internal carotid** and the **left external carotid artery** subjects (0%). However, in the **carotid body** near the **right common carotid artery**, **haemorrhage** was noted in 1 subject (2%) (Fig. 7.53).

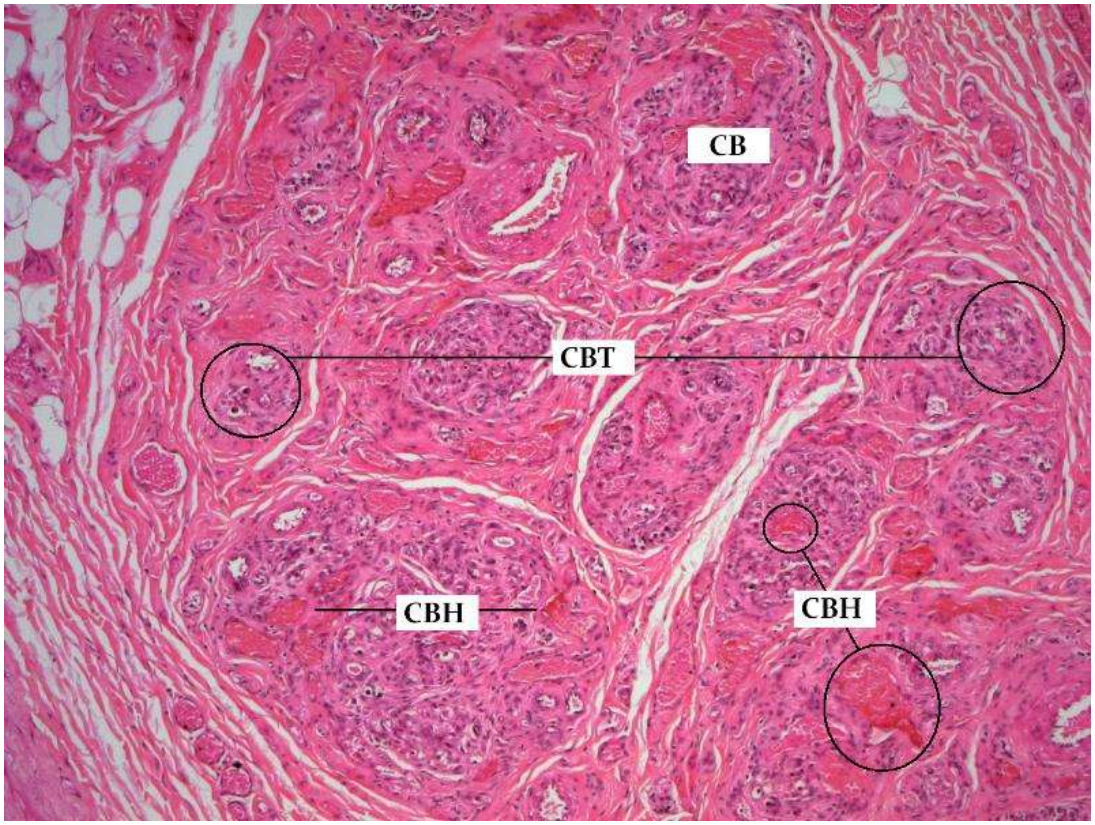


Fig. 7. 53. A view of a multicentric **carotid body** near the bifurcation of the **right common carotid artery**. In addition to highlighting the multicentric nature of the structure, **tearing** and **haemorrhage** are noted. Case no. 29 (Haematoxylin and Eosin x 100). **CB** = carotid body. **CBT** = carotid body tearing. **CBH** = carotid body haemorrhage.

The **carotid body** near the **right internal carotid artery** similarly showed **haemorrhage** in 1 subject (2%), while that near the **right external carotid artery** showed no haemorrhage in any of the subjects (0%).

3. Carotid Body Internal Dehiscence

Internal dehiscence in the **carotid body** near the bifurcation of the **left common carotid artery** was not noted in any of the subjects (0%), but was noted in the **carotid body** near the **left internal carotid artery** in 1 subject (2%) (Fig. 7.54).

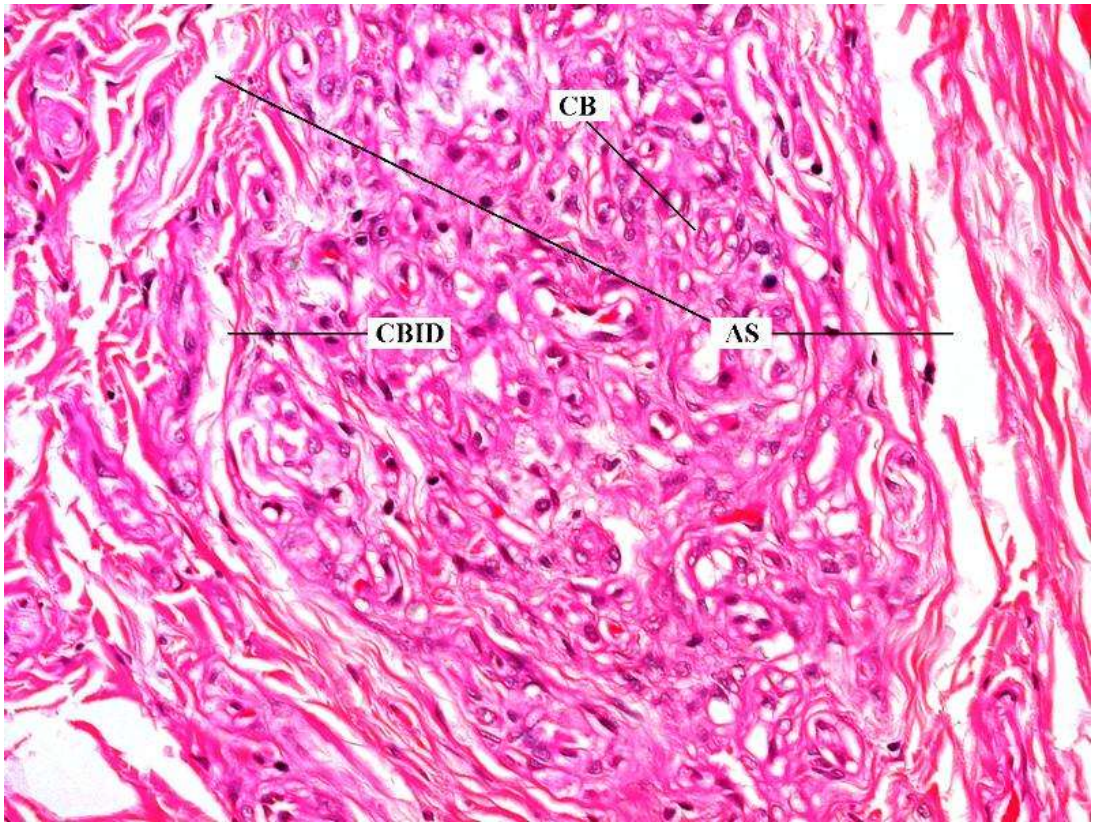


Fig. 7. 54. A high- power view of a **carotid body** near the **left internal carotid artery**. **Internal dehiscence** and **adventitial separation** are noted. Case no. 4 (Haematoxylin and Eosin x 400). **AS** = adventitial separation. **CB** = carotid body. **CBID** = carotid body internal dehiscence.

In the **carotid body** near the **left external carotid artery**, **internal dehiscence** was not noted in any of the subjects (0%), but in the **carotid body** near the **right common carotid**, the **right internal carotid**, and the **right external carotid arteries**, **internal dehiscence** was noted in 1 subject (2%) each, respectively.

4. Carotid Body Tearing

Carotid body tearing was not noted in the **carotid body** near the **left common carotid**, the **left internal carotid**, and the **left external carotid arteries** in any of the subjects (0%). In the **carotid body** near the bifurcation of the **right common carotid artery**, however, **tearing** was noted in 1 subject (2%) (Fig. 7.53) while in the **carotid body** near the **right internal carotid artery** no **tearing** was noted in any of the subjects (0%). In the **carotid body** near the **right external carotid artery**, **tearing** was noted in 1 subject (2%).

The presumed reasons for the very low incidence of **carotid body tearing** is dealt with in the Discussion chapter which follows.

Summary

In summary, the following changes were noted in the tissues surrounding the total number of 300 arteries examined:

The Nerves

¶ neural congestion: 20 (6%);

¶ neural/perineural haemorrhage: 14 (4%);

¶ neural internal dehiscence: 54 (18%);

¶ neural tearing: 34 (11.3%);

¶ perineural separation: 112 (37.3%.

The Accessory Glomi

In the **accessory glomi** in the tissues surrounding the total number of 300 arteries examined:

¶ congestion: 20 (6.6%);

¶ haemorrhage: 7 (2.3%);

¶ internal dehiscence: 50 (16.6%);

¶ tearing: 18 (6%);

¶ adventitial separation: 124 (41.3%).

The Ganglia

¶ congestion: 20 (6.6%);

¶ haemorrhage: 8 (2.6%);

¶ internal dehiscence: 15 (5%);

¶ tearing: 6 (2%).

The Carotid Body

¶ congestion: 8 (2.6%);

¶ haemorrhage: 2 (0.6%);

¶ internal dehiscence: 4 (1.3%);

¶ tearing: 2 (0.6%).

The results are summarised in Table Va below, where N= 300 (Total Number of Arteries examined).

Table Va: Neural Findings: Unilateral Involvement (Total number of hanging cases examined (N = 50))

NC = Neural Congestion	AGC = Accessory Glomal Congestion
NH = Neural Haemorrhage	AGH = Accessory Glomal Haemorrhage
NID = Neural Internal Dehiscence	AGID = Accessory Glomal Internal Dehiscence
NT = Neural Tearing	AGT = Accessory Glomal Tearing
PNS = Perineural Separation	AGAS = Accessory Glomal Adventitial Separation
GC = Ganglionic Congestion	CBC = Carotid Body Congestion
GH = Ganglionic Haemorrhage	CBH = Carotid Body Haemorrhage
GID = Ganglionic Internal Dehiscence	CBID = Carotid Body Internal Dehiscence
GT = Ganglionic Tearing	CBT = Carotid Body Tearing

Site	NC	NH	NID	NT	PNS	AGC	AGH	AGID	AGT	AGAS	GC	GH	GID	GT	CBC	CBH	CBID	CBT
LCC	2	7	13	7	21	3	1	12	5	19	1	2	2	2	0	0	0	0
%	4.0	14.0	26.0	14.0	42.0	6.0	2.0	24.0	10.0	38.0	2.0	4.0	4.0	4.0	0.0	0.0	0.0	0.0
LIC	5	1	10	8	18	2	0	10	2	26	3	2	2	1	3	0	1	0
%	10.0	2.0	20.0	16.0	36.0	4.0	0.0	20.0	4.0	52.0	6.0	4.0	4.0	2.0	6.0	0.0	2.0	0.0
LEC	3	2	11	4	24	4	3	9	5	24	7	0	5	1	1	0	0	0
%	6.0	4.0	22.0	8.0	48.0	8.0	6.0	18.0	10.0	48.0	14.0	0.0	10.0	2.0	2.0	0.0	0.0	0.0
RCC	2	3	8	7	22	4	2	6	3	19	4	0	2	1	1	1	1	1
%	4.0	6.0	16.0	14.0	44.0	8.0	4.0	12.0	6.0	38.0	8.0	0.0	4.0	2.0	2.0	2.0	2.0	2.0
RIC	3	0	4	4	11	1	0	3	2	14	3	4	3	1	2	1	1	0
%	6.0	0.0	8.0	8.0	22.0	2.0	0.0	6.0	4.0	28.0	6.0	8.0	6.0	2.0	4.0	2.0	2.0	0.0
REC	5	1	8	5	16	6	1	10	1	22	2	0	1	0	1	0	1	1
%	10.0	2.0	16.0	10.0	32.0	12.0	2.0	20.0	2.0	44.0	4.0	0.0	2.0	0.0	2.0	0.0	2.0	2.0

N = 300 (Total Number of Arteries examined)

Site	NC	NH	NID	NT	PNS	AGC	AGH	AGID	AGT	AGAS	GC	GH	GID	GT	CBC	CBH	CBID	CBT
TOTAL	20	14	54	35	112	20	7	50	18	124	20	8	15	6	8	2	4	2
%	6.6	4.6	18.0	11.6	37.3	6.6	2.3	16.6	6.0	41.3	6.6	2.6	5.0	2.0	2.6	0.6	1.3	0.6

What do these figures mean and can any conclusions be drawn from them? While there appears to be little, if any, correlation with either the vascular or additional vascular findings noted in Tables IIIa and IVa, there appears to be a remarkable similarity between the various **neural findings** themselves. The most striking example was that of **vascular congestion**, where **neural congestion**, **accessory glomal congestion**, and **ganglionic congestion** all showed the same incidence of involvement of 20 (6.6%) of the **nerves**, **accessory glomi** and **ganglia** of the tissues surrounding the total number of 300 arteries examined. The second similarity lay in the incidence of **internal dehiscence**, being 54 (18%) in the case of the **nerves**, and 50 (16.6%) in the case of the **accessory glomi**. The incidence of **ganglionic internal dehiscence** was very much lower, being only 15 (5%) of the **ganglia** of the tissues surrounding the total number of 300 arteries examined, while that of **carotid body dehiscence** was very low, comprising only 4 (1.3%) of the **carotid bodies** of the tissues surrounding the total number of 300 arteries examined.

The incidence of **accessory glomal haemorrhage** and **ganglionic haemorrhage** was almost identical, being 7 (2.3%) and 8 (2.6%) each, respectively. The incidence of **neural haemorrhage**, however, was double, or almost double, comprising 14 (4.6%) of the **nerves** of the tissues surrounding the total number of 300 arteries examined. As far as tissue separation and separation external to the **nerves** and **accessory glomi** was concerned, some similarity of incidence was present in that **perineural separation** was found to be present in the **nerves** of the tissues surrounding 112 (37.3%) of the 300 arteries examined, while **accessory glomal adventitial separation** was found in the **accessory glomi** of the tissues surrounding 124 (41.3%)

of the 300 arteries examined. Other than these comparative findings, therefore, very few comparisons could be made, although the incidence of traumatic disruption of the **carotid body** was noteworthy in being remarkably low.

Was this due to possible protection afforded by the **external** and **internal carotid arteries**, as well as the multicentric, globular and spherical structure of the **carotid body** itself? This issue will be dealt with more fully in the Discussion chapter which follows.

Bilateral Involvement of the Neural Findings

For purposes of comparison, and in an attempt to determine whether any correlation could be made between the bilateral **vascular findings** (Table IIIb)**,** the bilateral **additional vascular findings** (Table IVb) and the **neural findings**, the latter were similarly grouped bilaterally (Table Vb).

Table Vb: Neural Findings: Bilateral Involvement (N = 50)

NC = Neural Congestion	AGC = Accessory Glomal Congestion
NH = Neural Haemorrhage	AGH = Accessory Glomal Haemorrhage
NID = Neural Internal Dehiscence	AGID = Accessory Glomal Internal Dehiscence
NT = Neural Tearing	AGT = Accessory Glomal Tearing
PNS = Perineural Separation	AGAS = Accessory Glomal Adventitial Separation
GC = Ganglionic Congestion	CBC = Carotid Body Congestion
GH = Ganglionic Haemorrhage	CBH = Carotid Body Haemorrhage
GID = Ganglionic Internal Dehiscence	CBID = Carotid Body Internal Dehiscence
GT = Ganglionic Tearing	CBT = Carotid Body Tearing

	NC	NH	NID	NT	PNS	AGC	AGH	AGID	AGT	AGAS	GC	GH	GID	GT	CBC	CBH	CBID	CBT
Total	0	0	4	0	28	0	0	6	2	32	1	0	2	0	1	0	0	0
%	0.0	0.0	8.0	0.0	56.0	0.0	0.0	12.0	4.0	64.0	2.0	0.0	4.0	0.0	2.0	0.0	0.0	0.0

Number of Arteries with Bilateral Involvement: (N = 150)

	NC	NH	NID	NT	PNS	AGC	AGH	AGID	AGT	AGAS	GC	GH	GID	GT	CBC	CBH	CBID	CBT
Total	0	0	12	0	84	0	0	18	6	96	3	0	6	0	3	0	0	0
%	0.0	0.0	8.0	0.0	56.0	0.0	0.0	12.0	4.0	64.0	2.0	0.0	4.0	0.0	2.0	0.0	0.0	0.0

As indicated, the **vascular findings** (**section III**; **Table IIIb**) comprised **intimal tears, intimal tears extending to or along the internal elastic lamina, intimo-medial tears, medial tears, adventitio-medial tears, adventitial tears, adventitial haemorrhages** and **complete circumferential transverse ruptures**. These were grouped into 150 bilateral arterial groups, i.e. **left common carotid and right common carotid arteries, left internal and right internal carotid arteries** and **left external and right external carotid arteries**. The **additional vascular findings** (**section IV, Table IVb**), on the other hand, consisted of mainly previously unreported findings involving the arterial wall, namely, **endothelial elevation or avulsion, subendothelial clefts, internal elastic lamina dehiscence, adventitio-medial separation, vascular congestion, vascular plane of cleavage** and **multiple**

fenestrations of the media. These were similarly grouped into 150 bilateral arterial groups.

While the incidence of bilateral involvement of the **vascular findings** was very low, being 0% in most cases, the incidence of bilateral involvement of the **additional vascular findings**, on the other hand, was very high, reaching more than 96% in the case of **endothelial elevation or avulsion** and more than 90% in the cases of **subendothelial clefts** and **internal elastic lamina dehiscence**.

In contrast, however, the incidence of bilateral involvement of the **neural findings** lay between that of the **vascular findings** and the **additional vascular findings**. In some cases it was high, while in others it was either very low (with two exceptions, namely **accessory glomal adventitial separation**, where the incidence was 41.3% and **perineural separation**, where the incidence was 37.3%) or was 0%. In this latter instance of a low (0%) bilateral incidence of **neural findings**, therefore, the incidence mirrored that found in regard to that seen in the **vascular findings**, i.e. 0% in most cases. In other words, the bilateral response to injurious stimuli in the form of the compressive and tensile forces operative in the hanging process in these cases appeared to affect the **arteries** and **nerves** in an all-or-none manner.

Few correlations other than the above could, therefore, be made between the bilateral **vascular findings**, the bilateral **additional vascular findings**, and the bilateral **neural findings**. However, correlations could be made and conclusions drawn between the various **neural findings** themselves and the above **arterial findings**.

Looking at the figures in detail (Table Vb, page 361), **neural vascular congestion**, **neural haemorrhage**, **neural tearing**, **accessory glomal congestion**, **accessory glomal haemorrhage**, **ganglionic haemorrhage**, **ganglionic tearing**,

carotid body haemorrhage and **carotid body tearing** all mirrored and reflected one another in that, in these cases, the bilateral incidence was identical to that of the bilateral **arterial** incidence, being 0%. Once again, an all-or-none phenomenon appears to be operative. In two instances, however, i.e. that of **intraganglionic congestion** and **carotid body congestion**, the bilateral incidence of involvement was similarly identical, being 3 (2%) in each case, respectively, i.e. very small.

Perineural separation and **accessory glomal adventitial separation**, however, showed a fairly high incidence, comprising 84 (56%) and 96 (64%) of the **nerves** and **accessory glomi** of the tissues surrounding the 150 bilateral arterial groups examined, respectively. What inference can be drawn from this finding? These figures must indicate that loose adventitial tissues surrounding discrete structures such as **nerves** and **accessory glomi** tend to separate from the underlying respective anatomical structure at areas of tissue interfaces and where tissue densities differ.

What, however, of the bilateral findings of lesser incidence? These comprised **neural internal dehiscence**, **accessory glomal internal dehiscence** and **ganglionic internal dehiscence**. These showed a bilateral incidence of 12 (8%), 18 (12%), and 6 (4%), respectively. As evidence of the forces applied, **internal dehiscence** of the **carotid body**, on the other hand, showed an incidence of 0%. These findings would appear to reflect the relative tolerances of the different structures to tensile forces tending to stretch and pull the tissues apart. The 0% incidence of bilateral **carotid body internal dehiscence** would be in keeping with the observations noted above, i.e. that discrete multicentric structures, for the reasons enumerated, tend to be more resistant to tensile forces, even if these forces are equally, or almost equally, applied by a circumferentially placed constricting ligature. This greater resistance to the

forces applied, appears to hold despite the fact that the majority of the glomal structures lie in the tissues surrounding the vessels. This latter factor should, on the face of it, put them at greater risk of the surrounding compressive forces of the ligature. The suggested reasons for this apparent greater resistance to force application are put forward in the Discussion chapter which follows.

The Phrenic Nerve and its Anatomical Surroundings

It will be recalled that the **phrenic nerve** is composed of the anterior primary rami of cervical 3, 4 and 5 nerves (but mostly C4). It lies upon the anterior surface of the **scalenus anterior muscle**, running in a lateral-to-medial direction with the **sternocleidomastoid muscle** lying over it but with interposed fascial connective tissue. Thus, in hanging, where the ligature lies over the upper-third of the neck (as was found to be the case in 90% of the subjects in this study), the ligature will overlie the upper part of the nerve.

As indicated, the **phrenic nerves** and its surroundings were taken in an additional 5 cases of hanging. The same delicate dissection process was adopted in scrutinising and taking the **phrenic nerves** and its surroundings, as with the **carotid arteries**. However, to show the surroundings, not all the photomicrographs indicate the presence of the nerve.

The taking of the **phrenic nerves** was done for two reasons:

i) in view of the function of this nerve as the innervation to the diaphragm, the major muscle of respiration, and;

ii) in an attempt to determine whether or not any morphological findings emerging could be attributed to the compressive and tensile forces occurring during the hanging process.

The findings which emerged proved interesting in that they mirrored and reflected the **neural findings** noted in the present study. In addition to **neural congestion**, **neural internal dehiscence/separation**, **neural tearing**, and **perineural separation**, the ancillary as well as surrounding structures reflected the findings previously noted. These findings comprised **perineural** (although not **intraneural**) **haemorrhage**, **vascular congestion** within the surrounding tissues, **empty venous and arterial vessels**, **haemorrhagic**, **traumatised and disrupted interstitial connective tissues and skeletal muscles,** and **traumatised and disrupted lymph nodes**. Some of these findings are illustrated in the following photomicrographs (Figs. 7.55 to 7.68). A section of muscle from a non-hanging control case is shown overleaf for comparison.



A section of normal muscle from control case no. 3. No indentation of the muscle fibres is seen (Haematoxylin and Eosin x 10).



Fig. 7.55a. A view of the **sternocleidomastoid muscle** immediately beneath the point of ligature application showing grooving and indentation of the muscle underlying the position of the ligature. The overlying interstitial tissues are disrupted. Case no. 51 (Haematoxylin and Eosin x 50). **ITD** = interstitial tissue disruption. **SCM** = sternocleidomastoid muscle.

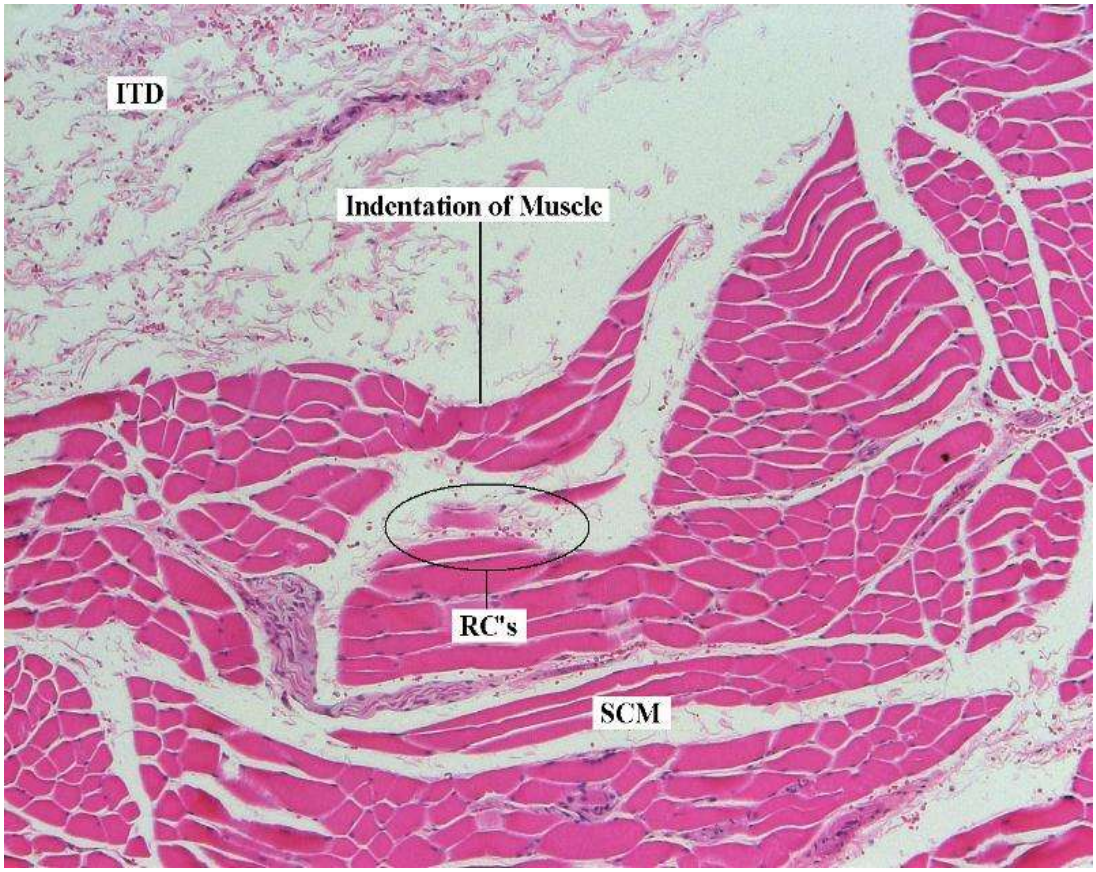


Fig. 7.55b. A high-power view of the indentation and grooving of the **sternocleidomastoid muscle** beneath the point of ligature application. The overlying connective tissues are disrupted and show the infiltration by **haemorrhage** seen at this higher magnification. Case no. 51 (Haematoxylin and Eosin x 100). **ITD** = interstitial tissue disruption. **SCM** = sternocleidomastoid muscle. **RC's** = red blood cells.



Fig. 7.56. A view of the disrupted, torn and fragmented **sternocleidomastoid muscle** beneath the site of ligature application. Does the paucity of haemorrhage in this case reflect the previously noted findings of empty arterial and venous vessels as a consequence of ligature compression? Case no. 52 (Haematoxylin and Eosin x 50). **DSCM** = disrupted sternocleidomastoid muscle.

Incidentally, the **external carotid artery** has a branch called the '**sternomastoid branch**' (not always present) but in this case possibly so, with pressure to the **external carotid** occluding blood flow to the muscle via its **sternomastoid branch**.

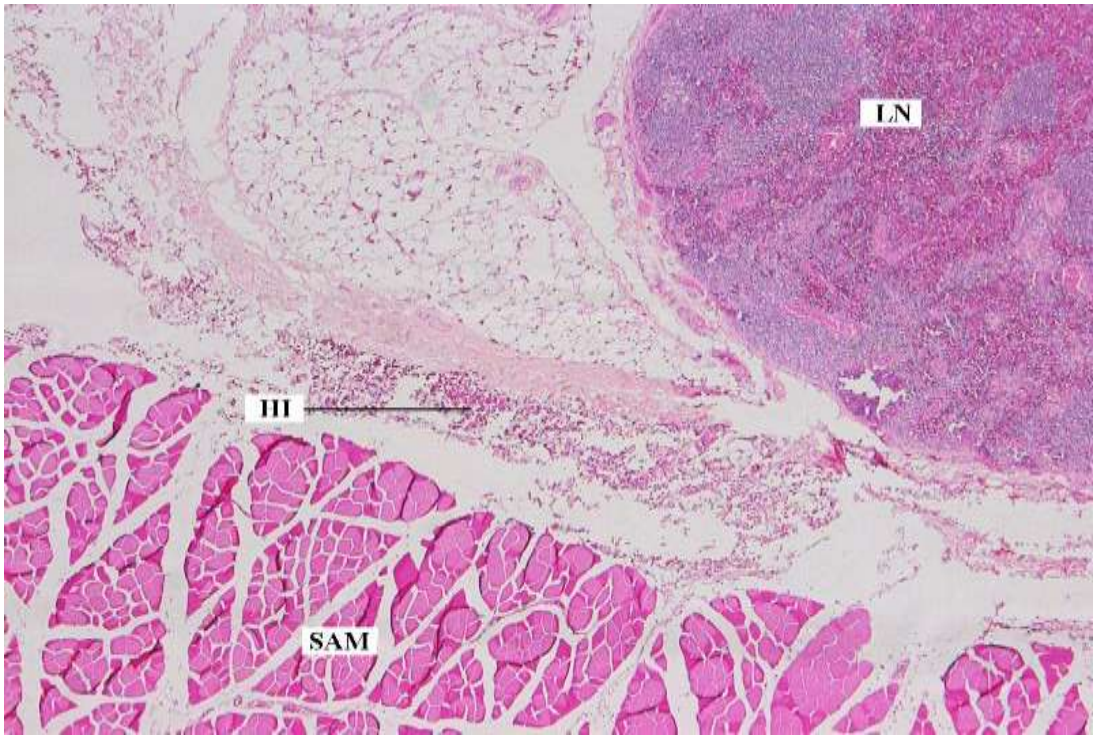


Fig. 7.57. A view of **scalenus anterior muscle** showing an adjacent lymph node and intervening connective tissue in which **haemorrhagic infiltration** is noted. **Haemorrhage** into the lymph node and surrounding connective tissues constitutes corroborative evidence of the widespread nature of the trauma. Case no. 52 (Haematoxylin and Eosin x 50). **LN** = lymph node. **HI** = haemorrhagic infiltration. **SAM** = scalenus anterior muscle.

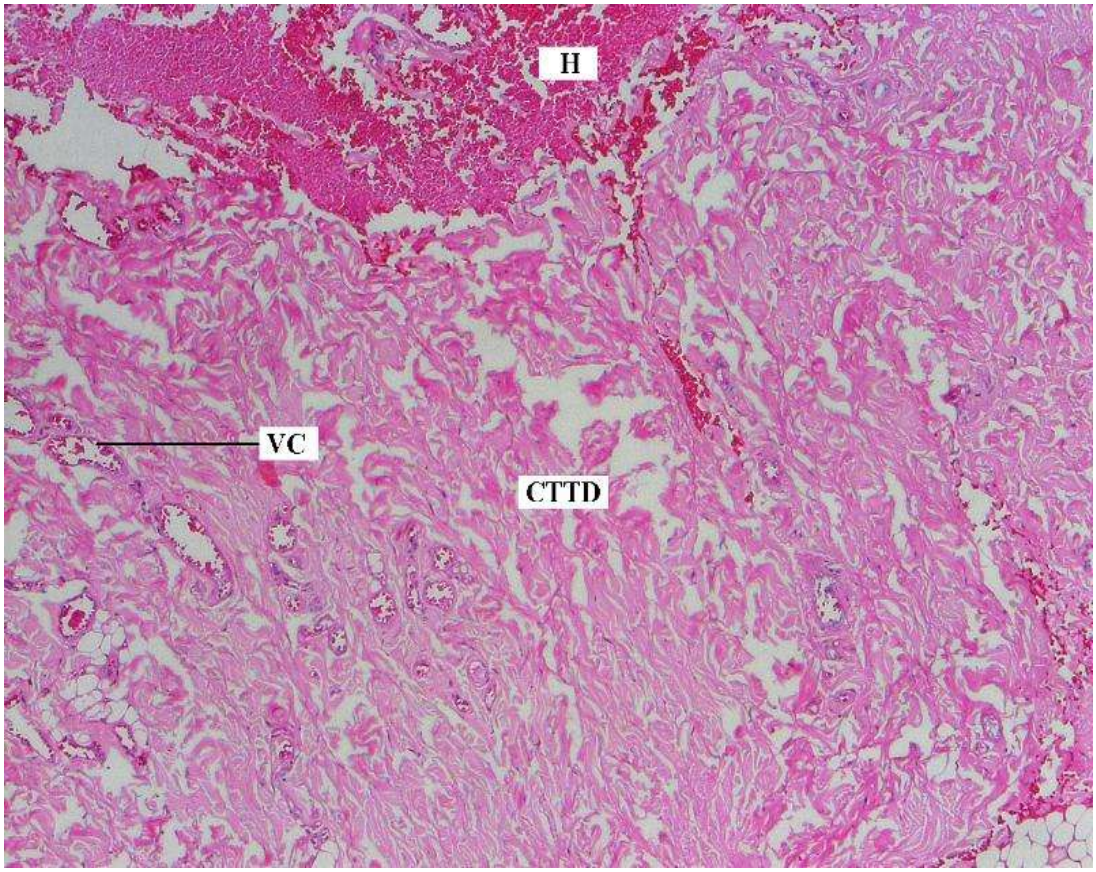


Fig. 7.58. A view of the connective tissues in proximity to the plane of section of the **phrenic nerve** (not shown) but deep to the site of ligature application showing **tearing**, **disruption**, **vascular congestion** and overlying **haemorrhage**. Case no 51 (Haematoxylin and Eosin x 50). **H** = haemorrhage. **VC** = vascular congestion. **CTTD** = connective tissue tearing and disruption.

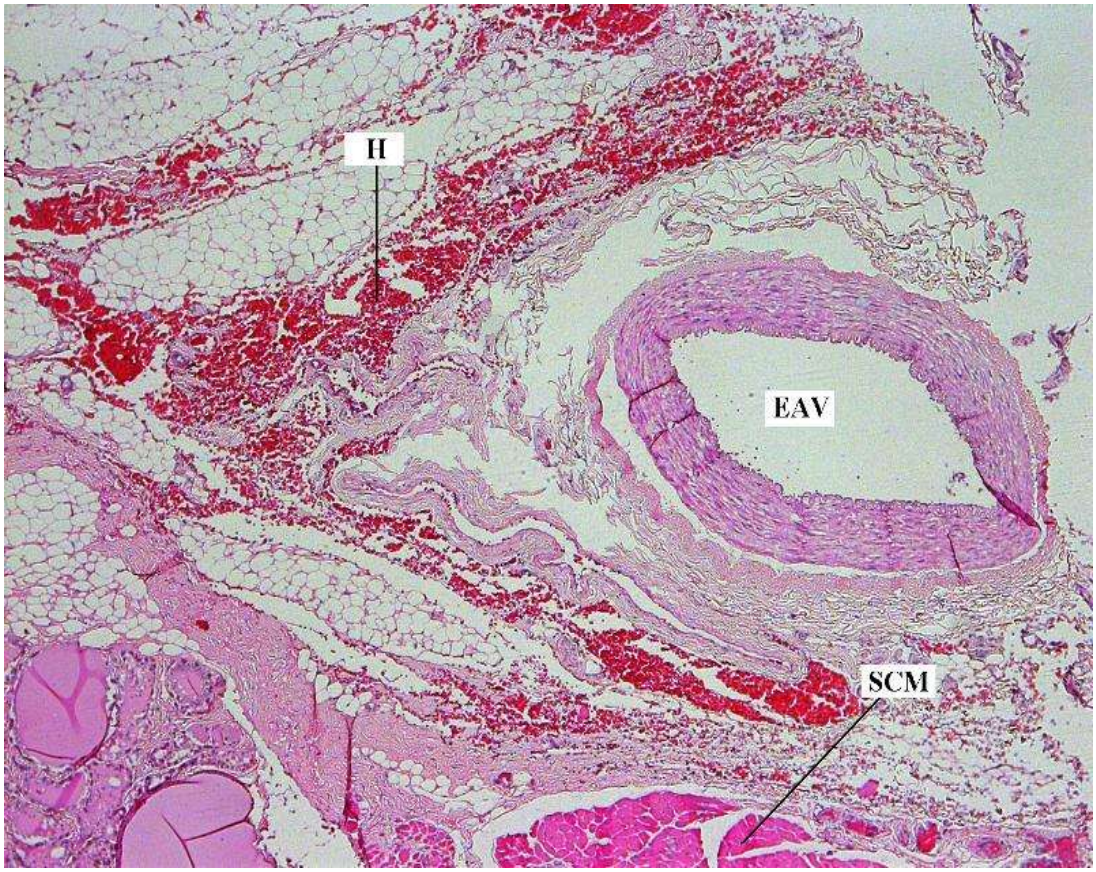


Fig. 7.59. A view of the tissues below the **phrenic nerve** (not included in the plane of section) showing an empty small arterial vessel, adjacent empty and collapsed venous vessel, and surrounding connective tissue in which severe **haemorrhage** is noted. Part of the **sternocleidomastoid muscle** is seen at the centre bottom of the field as well as two thyroid follicles at the left lower corner of the field. The presence of the two thyroid follicles indicates a lower position of the section than the upper-third of the neck. Case no. 53 (Haematoxylin and Eosin x 50). **H** = haemorrhage. **EAV** = empty arterial vessel. **SCM** = sternocleidomastoid muscle.

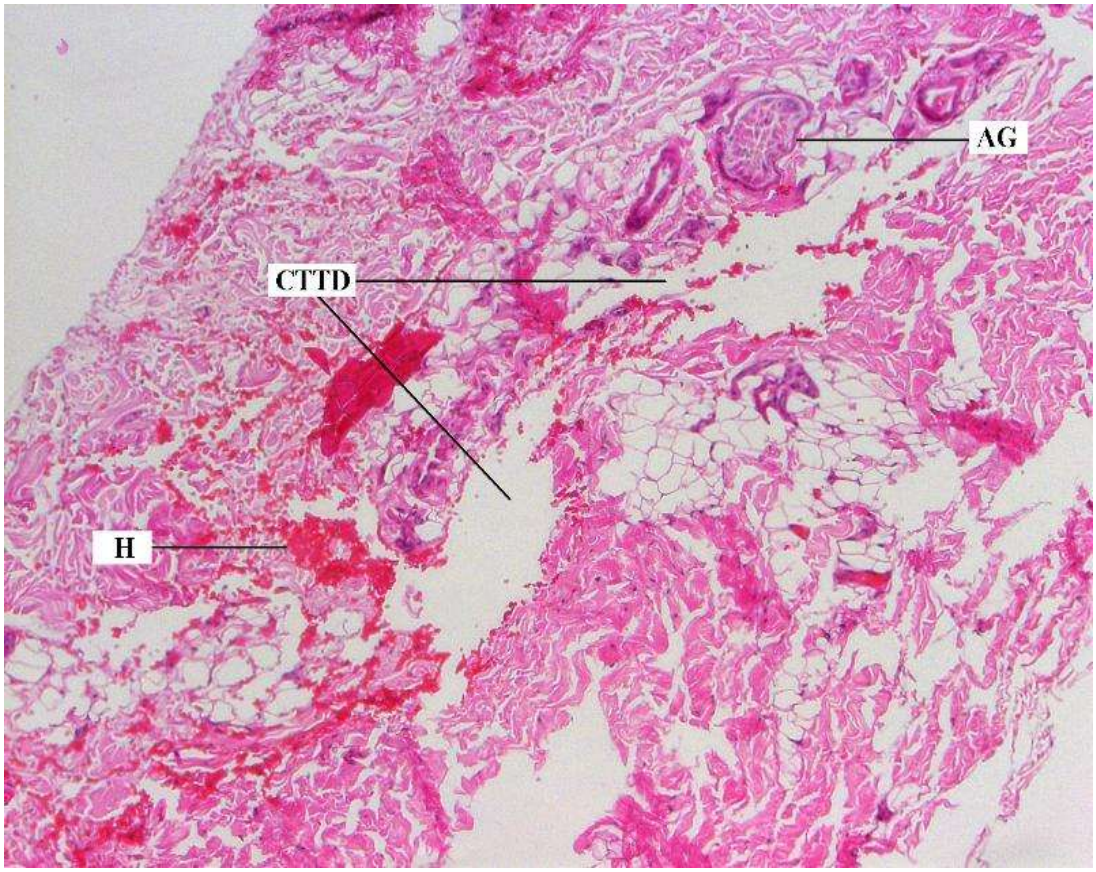


Fig. 7.60. A further view of the connective tissues below the **phrenic nerve** (not included in the plane of section) but deep to the site of ligature application. **Tearing**, **disruption** and **haemorrhage** are noted. An **accessory glomus** is seen at the top centre right of the field. Case no. 53 (Haematoxylin and Eosin x 100). **CTTD** = connective tissue tearing and disruption. **AG** = accessory glomus. **H =** haemorrhage.

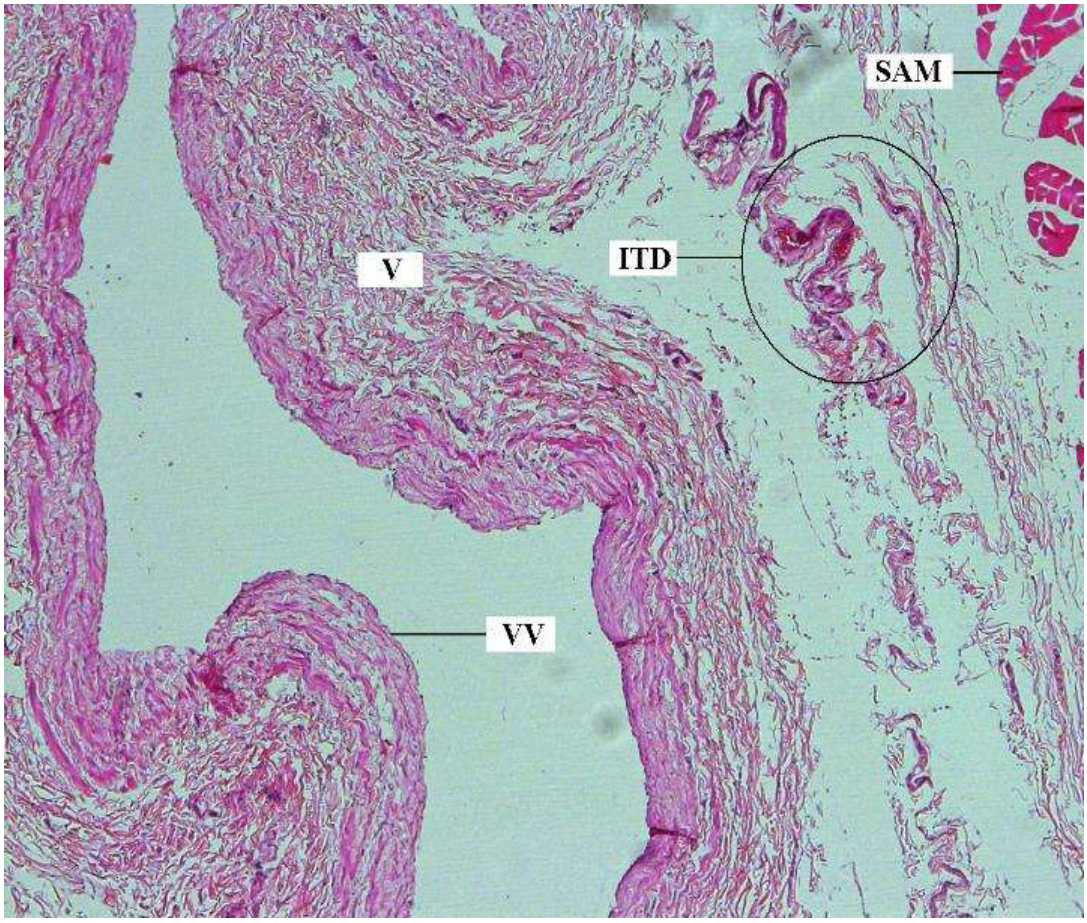


Fig. 7.61. A view of an empty **vein** and its venous valves (seen here on the left) lying anterior to part of the **scalenus anterior muscle** at the top right corner of the field. **Disruption** and teasing apart of the interstitial connective tissues is noted. Case no. 53 (Haematoxylin and Eosin x 100). **V** = vein. **SAM** = scalenus anterior muscle. **ITD** = interstitial tissue disruption. **VV** = part of a venous valve.

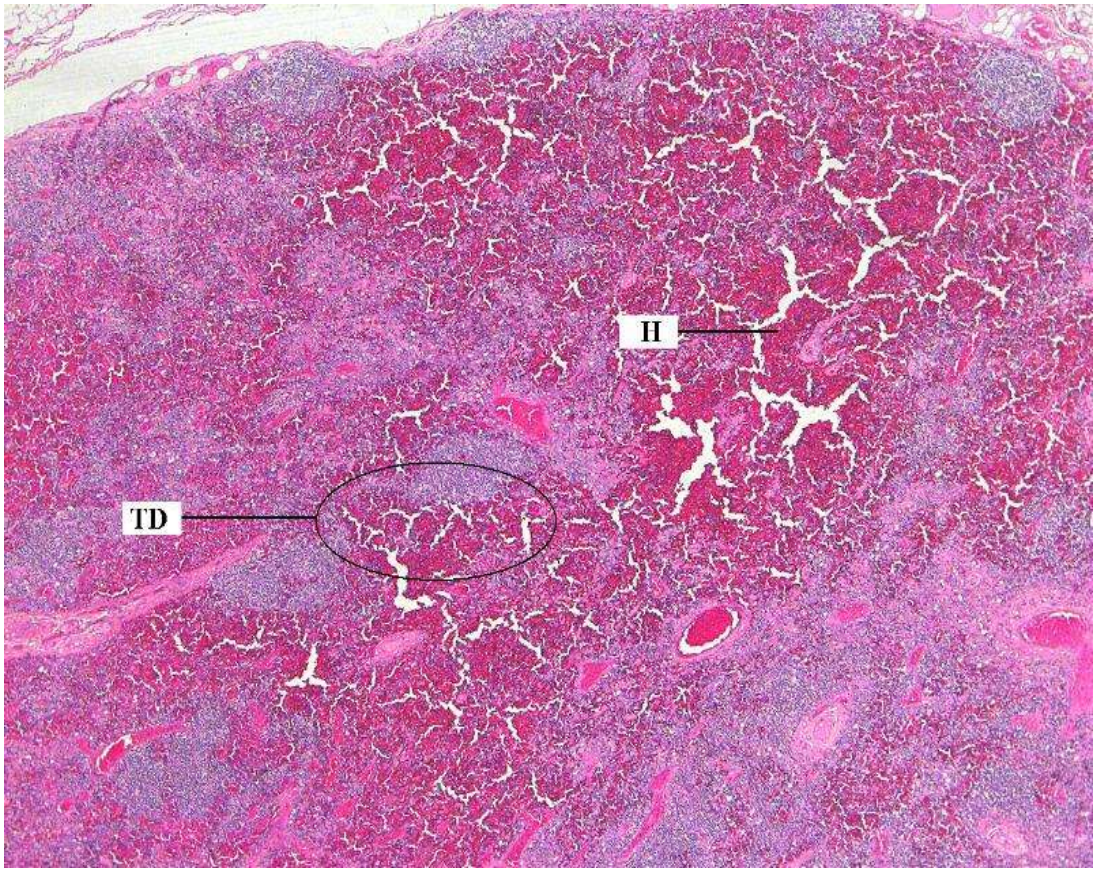


Fig. 7. 62. A high-power view of the **lymph node** and surrounding connective tissues seen in Fig. 7.57 constituting corroborative evidence of the widespread nature of the trauma. **Tearing**, **disruption** and **haemorrhage** are noted. Case no. 52 (Haematoxylin and Eosin x 100). **TD** = tearing and disruption. **H** = haemorrhage.

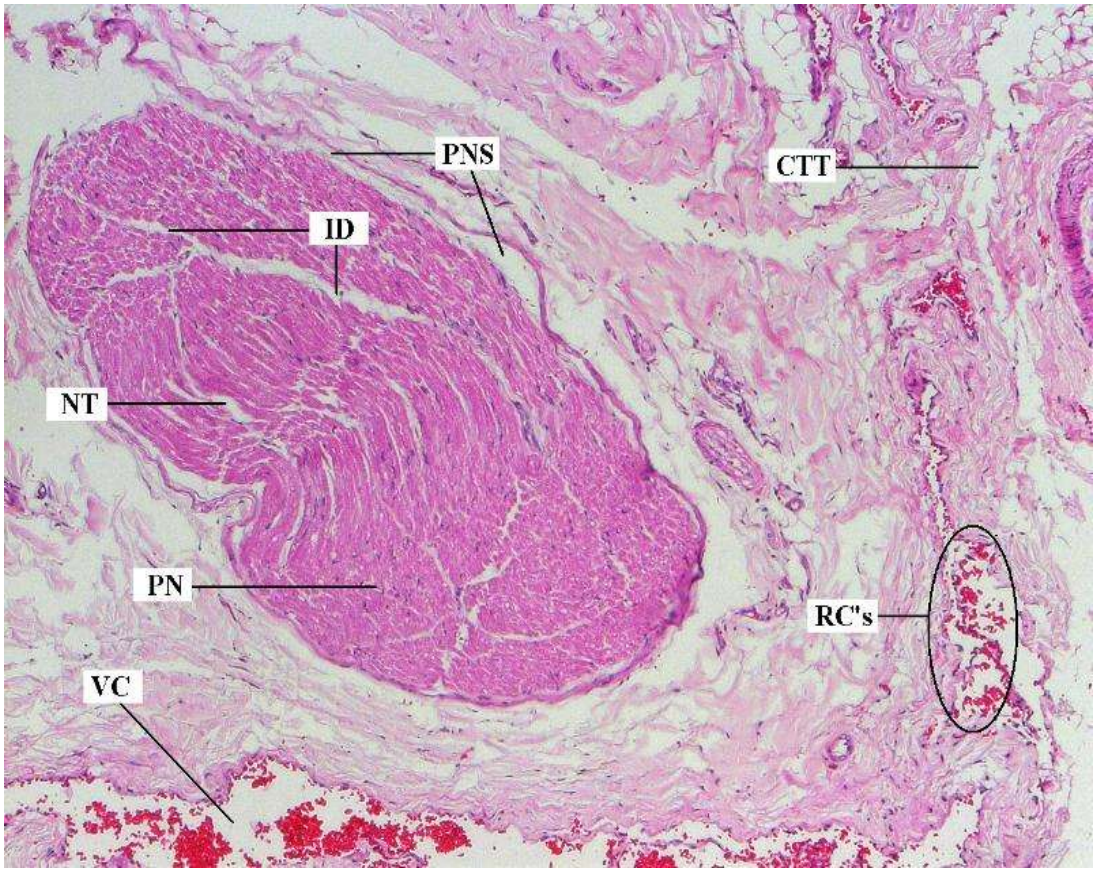


Fig. 7.63. A view of the **phrenic nerve** deep to the site of ligature application showing **internal dehiscence**, **neural tearing** and **perineural separation**. The surrounding connective tissues show **vascular congestion** as well as an area of **disruption** and **tearing** in which scattered **red cells** are noted. Case no. 51 (Haematoxylin and Eosin x 100). **PN** = phrenic nerve. **ID** = internal dehiscence. **NT**= neural tearing. **PNS** = perineural separation.**VC** = vascular congestion. **RC's** = red blood cells. **CTT** = connective tissue tearing.

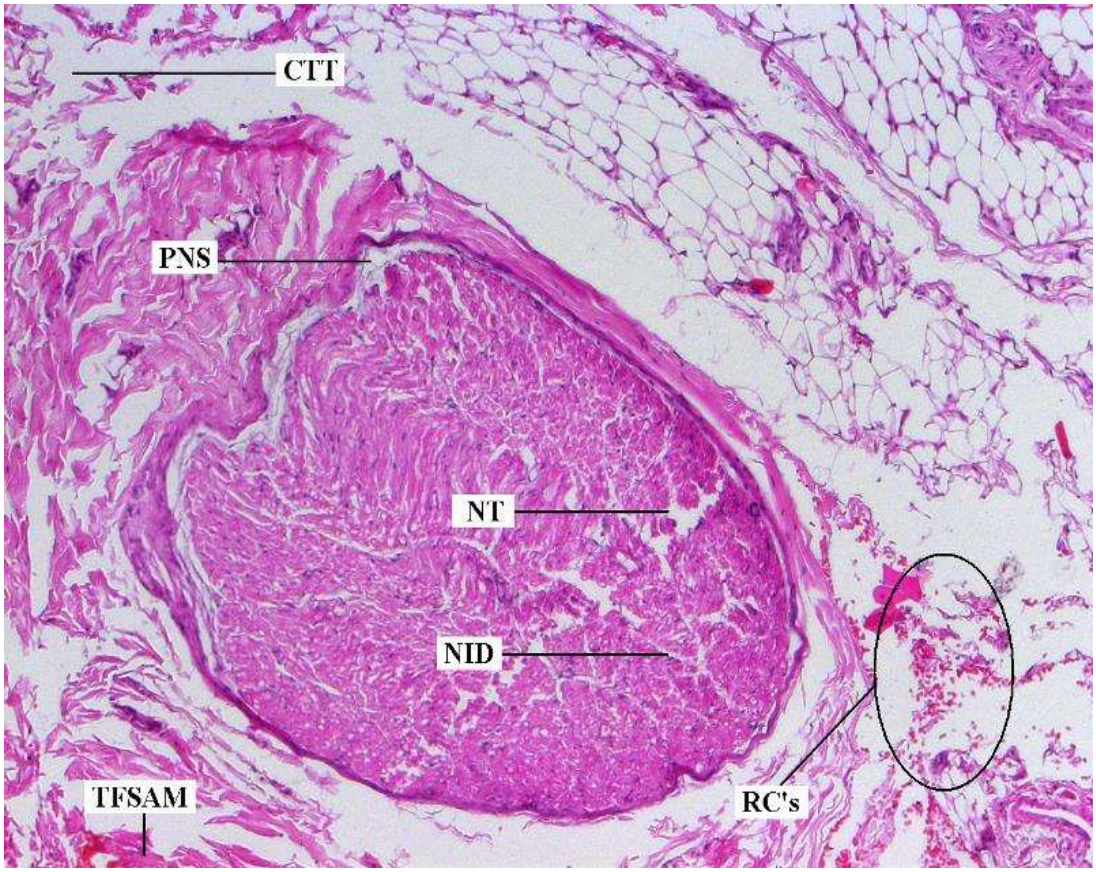


Fig. 7.64. A further view of a **phrenic nerve** showing **perineural separation**, **internal dehiscence** and **neural tearing**. The linear split which runs diagonally through the **nerve** is non-artefactual, constituting **neural internal dehiscence/separation** terminating at the nerve periphery. An artefactually-induced microtome split, on the other hand, would have included and continued beyond the nerve borders at each end of the split to involve the surrounding tissues. **Tearing** and **disruption** with **haemorrhage** into the surrounding connective tissues is noted. A few fragments of the underlying **scalenus anterior muscle** are noted at the bottom left corner of the field. Case no. 54 (Haematoxylin and Eosin x 100). **PNS** = perineural separation. **NID/S** = neural internal dehiscence/separation. **NT** = neural tearing. **CTT** = connective tissue tearing. **RC's** = red blood cells. **TFSAM** = a few torn fragments of scalenus anterior muscle with haemorrhage.

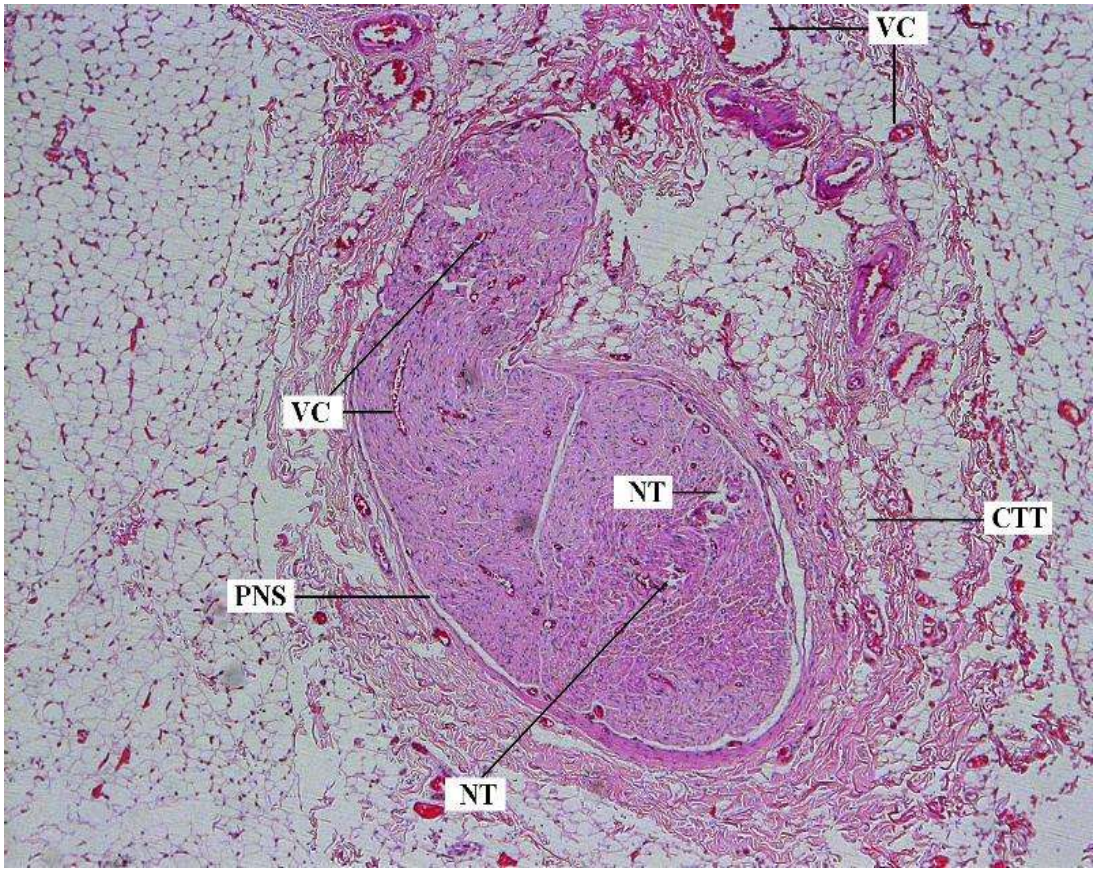


Fig. 7.65a. A further view of a **phrenic nerve** deep to the site of ligature application. **Vascular congestion**, **neural tearing** and **perineural separation** are noted. The surrounding tissues similarly show **tearing** but with some degree of **vascular congestion**. Case no. 55 (Haematoxylin and Eosin x 100). **VC** = vascular congestion. **NT** = neural tearing. **PNS** = perineural separation. **CTT** = connective tissue tearing.

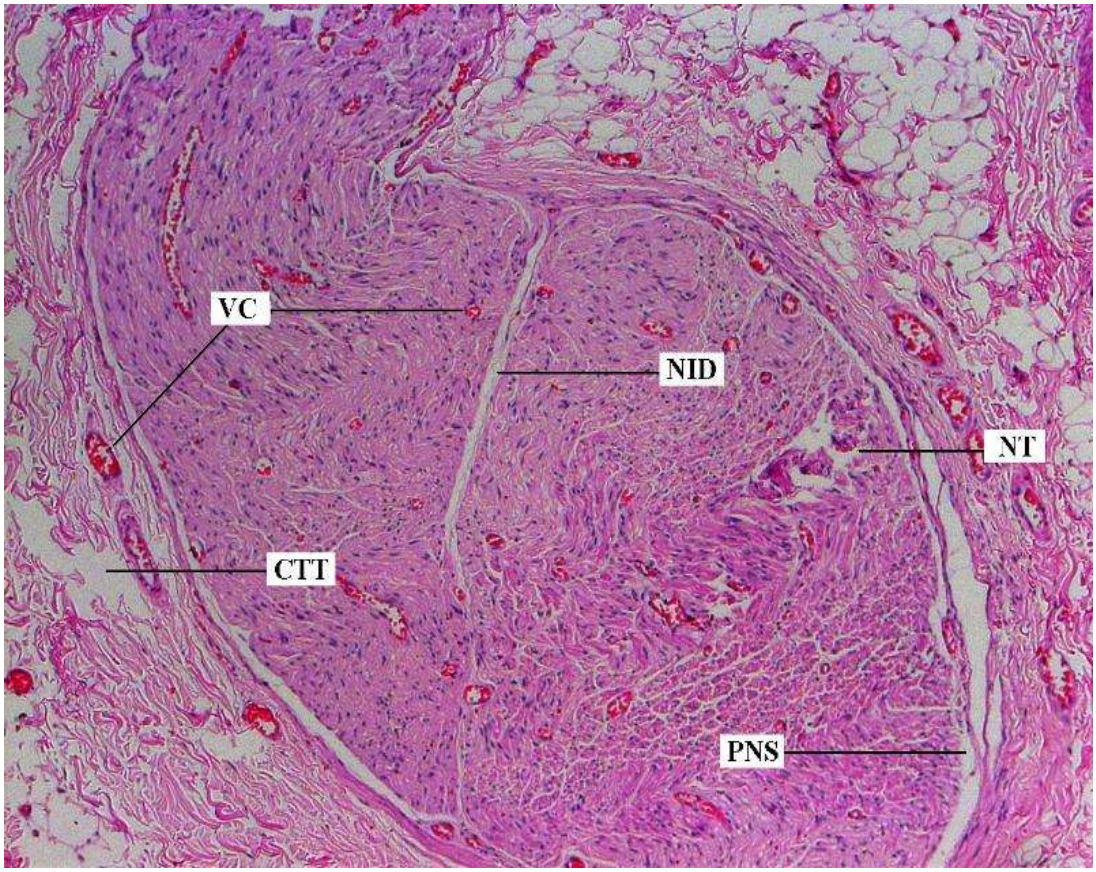


Fig. 7.65b. A high-power view of the **phrenic nerve** seen in Fig.7.65a showing **perineural separation** with bridging tissue strands, **internal dehiscence** with tissue strands along the area of separation, **neural tearing** and **vascular congestion**. The surrounding connective tissues show **disruption** and some degree of **vascular congestion**, better seen at the higher-power. Case no. 55 (Haematoxylin and Eosin x 100). **PNS** = perineural separation. **NID** = neural internal dehiscence. **NT** = neural tearing. **VC** = vascular congestion. **CTT** = connective tissue tearing.

The lower part of the nerve in the middle of the field beneath the area of **internal dehiscence** shows close apposition with a blood vessel above and a junctional vessel below. This area could therefore indicate a continuation of two components of the **phrenic nerve** bearing in mind that the **phrenic nerve** consists of

C3, 4 and 5 but mostly C4. The presence of close apposition between the two components, the absence of bridging strands of tissue and the presence of a vessel between the two components and a vascular supply at the peripheral junction lends support to the view that two components are indeed present. However, the presence of the area of separation and **internal dehiscence** above with tissue strands once again emphasizes the tendency of tissues to split at tissue interfaces in response to force application.

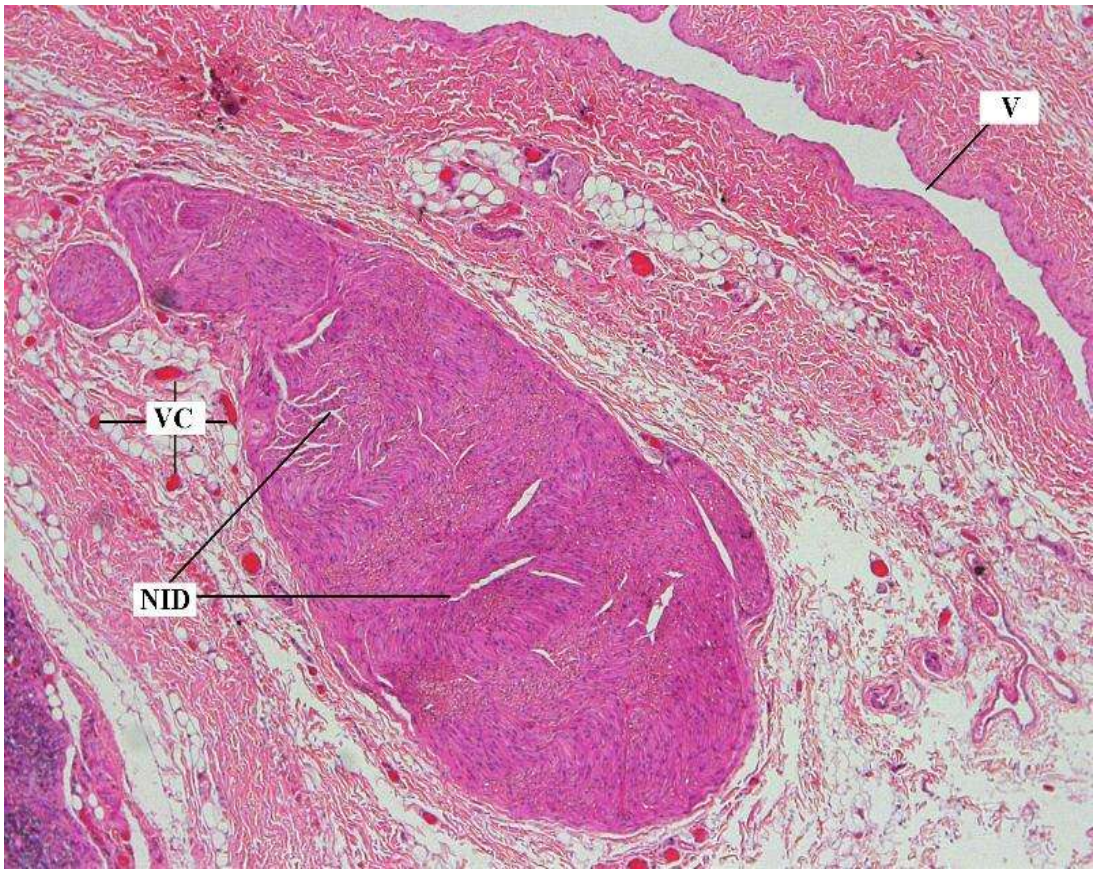


Fig. 7.66a. A view of the **phrenic nerve** showing **internal dehiscence** with **vascular congestion** in the surrounding tissues. Part of an empty adjacent **vein** is noted anteriorly on the right. Case no. 55 (Haematoxylin and Eosin x 50). **NID** = neural internal dehiscence. **VC** = vascular congestion. **V =** vein.

In contrast to the **phrenic nerve** seen in **Fig. 7.65b.**, here there is no real evidence of a second neural component within the large nerve although there are two smaller ones at the periphery, one on the right and the other to the left at the top. Continuation of the area of **internal dehiscence** indicated by the diagonal line of the annotation '**NID**' and to the right of the annotation '**VC**' is seen with space limitation inhibiting insertion of additional lines.

Nerves, as shown in the previous **Fig.7.65b.** and in **Fig. 7.66a.** above, may thus run together. Nerves may also branch and their blood vessels will branch with them. Hence, blood vessels will be found between nerves. On this basis, **dehiscence**, i.e. 'splits' within a nerve, with residual strands of tissue bridging, have been regarded as 'true splits' of a pathological nature and most likely due to the presence of the ligature, its compressive forces and the tensile and stretch forces of the weight of the body and the pull of gravity.

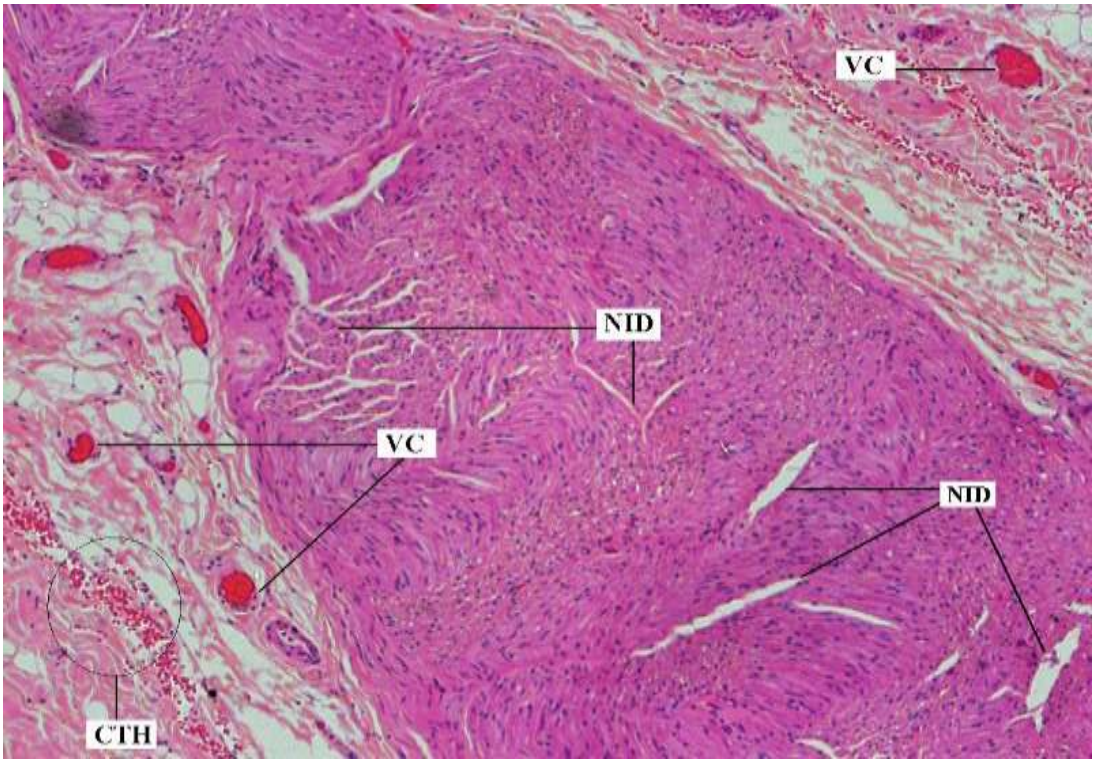


Fig. 7.66b. A high-power view of the **phrenic nerve** seen in Fig. 7.66a. showing marked **internal dehiscence**. The surrounding tissues show **vascular congestion** and **haemorrhagic infiltration**. Case no. 51 (Haematoxylin and Eosin x 100). **NID** = neural internal dehiscence. **VC** = vascular congestion. **CTH** = connective tissue haemorrhage.

Here, there is no clear evidence of nerve branching except possibly at the top above and to the left of the area of **internal dehiscence**.

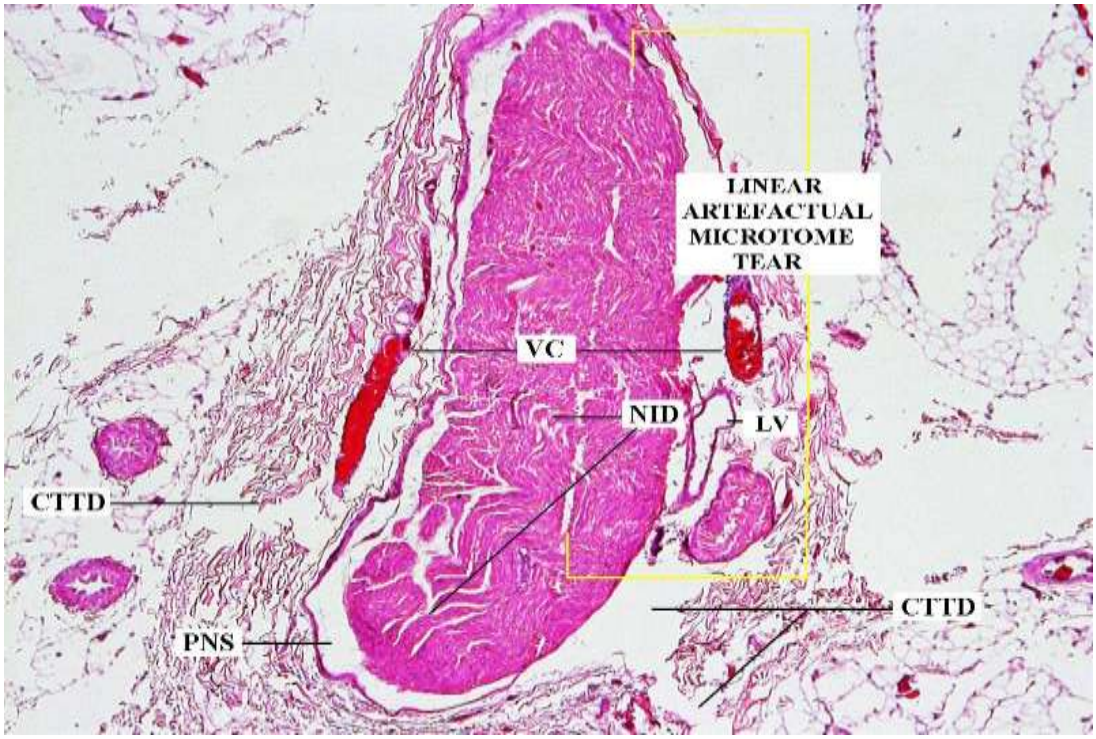


Fig. 7.67a. A view of the **phrenic nerve** showing **perineural separation** and **internal dehiscence**. **Vascular congestion** and **disruption** is noted in the surrounding tissues. A lymphatic vessel is seen lateral to the border of the nerve on the right. Case no. 54 (Haematoxylin and Eosin x 50). **PNS** = perineural separation. **NID** = neural internal dehiscence. **VC** = vascular congestion. **CTTD** = connective tissue tearing and disruption. **LV** = lymphatic vessel.

Comment: Nerves are traditionally ‘strong’ and are, therefore, unlikely to be damaged except with severe force. For example, the whole body may be lifted off the dissection table with a hook around one sciatic nerve.

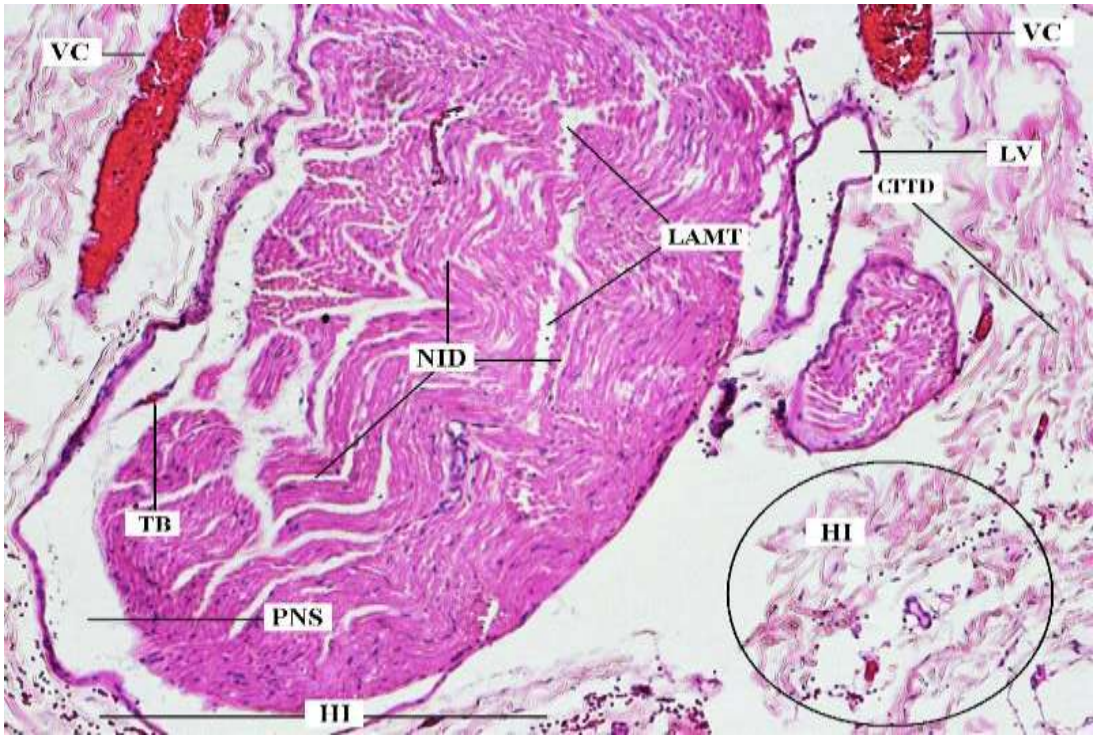


Fig. 7.67b. A high-power view of the **phrenic nerve** seen in Fig. 7.67a. **Perineural separation** and **internal dehiscence** are noted with **vascular congestion**, **tearing** and **haemorrhagic infiltration** of the surrounding connective tissues. Case no.54 (Haematoxylin and Eosin x 100). **PNS** = perineural separation. **NID =** neural internal dehiscence. **VC** = vascular congestion. **HI** = haemorrhagic infiltration. **CTTD** = connective tissue tearing and disruption. **LV** = lymphatic vessel containing lymphocytes. **TB** = tissue bridge. **LAMT** = linear artefactual microtome tear.

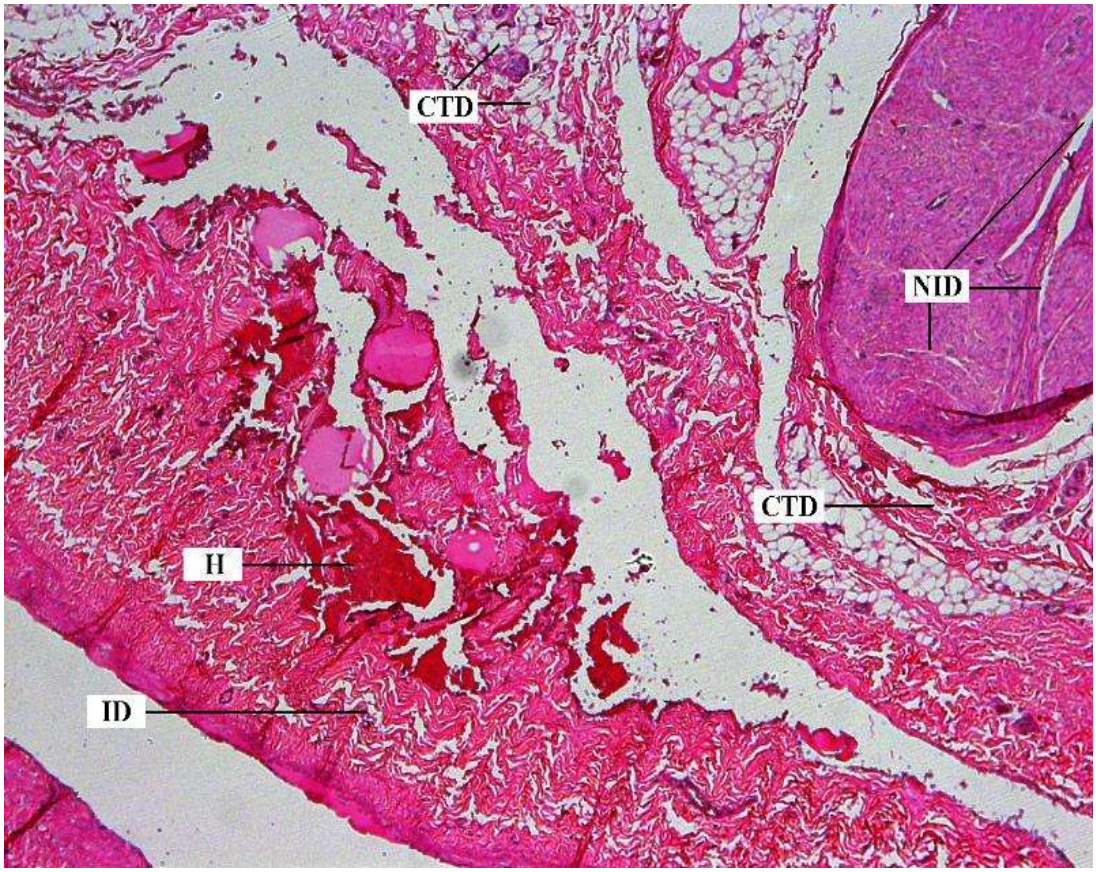


Fig. 7.68. A view of the **phrenic nerve** not having two components showing **internal dehiscence** and with part of an adjacent empty **vein** showing **internal dehiscence** with **haemorrhage** into the wall of the **vein**. The surrounding tissues show **disruption**. Case no. 53 (Haematoxylin and Eosin x 50). **NID** = neural internal dehiscence. **H** = haemorrhage. **CTD** = connective tissue disruption. **ID** = internal dehiscence of vein wall.

While, therefore, the findings in the **phrenic nerve** mirrored the **neural findings** seen in other **neural elements**, the most noteworthy finding emerging from examination of the **phrenic nerve and its surroundings** was the absence of **intraneural haemorrhage**. While **haemorrhage** into the surrounding connective tissues and overlying and underlying traumatised and disrupted skeletal muscles was

noted in abundance, the **phrenic nerve** itself appeared to be singularly unaffected. Transverse sectioning of the formalin fixed block had revealed a tough nerve 1 to 2 mm in diameter. Furthermore, could the cushioning effect of two muscle layers, the **sternocleidomastoid** muscle anteriorly and the **scalenus anterior** muscle posteriorly, have provided a buffering layer sufficient to prevent actual blood vessel rupture with **haemorrhage**? This finding highlights, perhaps, the relevance and importance of considering pathological findings not only from a mechanistic point of view but an anatomical one as well.

CONCLUSIONS

In essence, the findings emerging from the present study could be fundamentally classified into three broad categories, as follows:

1. **Vascular findings (section III)**;
2. **Additional Vascular Findings** (not previously reported in the literature)(**section IV**);
3. **Neural Findings (section V)**.

On a patho-anatomical basis, the vascular findings lend themselves to very specific differentiation and categorisation as one proceeds logically from the interior of the vessel wall, surrounding the lumen, to the outside and exterior of the vessel wall, i.e. the periadventitial tissues. Classifying the morbid anatomical features in this manner permitted further breakdown of the vascular findings into the **arterial elements**, namely, the **carotid arteries**, and those elements lying in the periadventitial tissues in close proximity to the **carotid arteries**, namely, the major **venous elements** and

the smaller vessels. The findings thus emerging from examination of these vascular elements could therefore be classified into three broad categories:

i) the pathological findings seen in the **arteries**, subdivided anatomically and pathologically into the three layers of the vessel wall, i.e. **intima**, **media,** and **adventitia**;

ii) the pathological findings seen in the **major veins**, i.e. the **internal jugular vein**, analogous to and identical with those seen in the arteries. These comprised **internal dehiscence** of and **haemorrhage** into the wall of the vein. These latter could be interpreted as being due to either the compressive forces of the encircling ligature or the tensile forces incident on the hanging process, i.e. stretch, and;

iii) the findings seen in the small **periadventitial vessels**, namely, **vascular congestion**. This latter finding could be attributed to a combination of the pathological damming of blood flow due to the compressive effect of the ligature and the physiological stagnation of blood flow consequent on a failing circulation.

As previously indicated, **adventitial haemorrhages** of the **thoracic** and **abdominal aortae** were found to be present to a greater or lesser degree in all the cases of suicidal hanging.

Concomitant with the study of the periadventitial tissues in proximity to the **carotid arteries** and **internal jugular veins**, there emerged the findings in the **neural elements**. These too could be classified into three major categories, all of

which, it is suggested, carry profound pathophysiological significance and consequences in the hanging process. These comprise:

1. damage to the **nerves** and **neural ganglia** related to the vessels;
2. damage to the **carotid bodies** and the **accessory glomi;**
3. damage to the **phrenic nerve** and its enclosing **muscular elements**, namely, the **sternocleidomastoid muscle** and the **scalenus anterior muscle**.

It is suggested that the **neural elements**, i.e. **nerves**, **neural ganglia**, **carotid bodies** and **accessory glomi**, and **muscular elements**, are of pivotal pathophysiological significance in the hanging process, not previously considered in this role. This lies in the twofold neural effect of **neural elements** on both brain and heart as a result of autonomic nervous system stimulation, sympathetic and parasympathetic, and the major function of the **phrenic nerve** in respiration.

The **phrenic nerve**, on the one hand, carries neural impulses to the diaphragm, the major muscle of respiration. The **sternocleidomastoid** and **scaleni muscles**, i.e. those enclosing the **phrenic nerve**, on the other hand, comprise accessory muscles of inspiration, expiration being an entirely passive process. Unremitting and continuously applied compression and stretching of the **phrenic nerve**, as in hanging, would induce a sustained physiological **action potential** in the **nerve**, resulting in the diaphragm becoming fixed in a state of contractual paralysis. As discussed in greater detail in the Discussion chapter which follows, sustained compression of these two muscles by a tightly applied constricting ligature, as well as stretching due to the pull of gravity by the weight of the body, produces both a **dynamic** and a **static stretch reflex** resulting in these muscles becoming fixed in a state of inspiratory paralysis, no further breathing being possible. This compounding

effect, combined with hypoxia as a result of carotid arterial damage and compression, as well as the hypoxic central nervous system effect of autonomic nervous system sympathetic stimulation, may, it is suggested, play a central role in unconsciousness and death causation in hanging. This will be further analysed and discussed in greater detail in the Discussion chapter which follows.

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CHAPTER 8

DISCUSSION

Introduction

As is evident from the foregoing chapters, there is a large amount of detailed information emerging as findings. Some of the information, as gleaned from the literature, is known, some is not known. Although in some chapters an attempt has been made to explain the findings in the form of 'miniature discussions', the present chapter is designed to pull together (and pool) all the information in an attempt to solve the problem as originally set out.

The discussion has been set out as follows:

A. **The vascular findings and their biomechanical causation.**

B. **The neural findings, their biomechanical causation and their pathophysiological implications.**

C. **The neuromuscular findings and their pathophysiological implications.**

D. **The biomechanical principles governing force application to biological tissues.**

E. **The principles of physics applicable to suicidal hanging.**

A. The Vascular Findings and their Biomechanical Causation

Detailed examination of the **vascular findings** shows a preponderance and predominance of lesions involving the **intima**, the **intima extending to or along the internal elastic lamina**, the **media** or muscular layer on its own rather than either **intimo-medial** or **adventitio-medial**, and the **adventitia**, the latter in the form of either an **adventitial tear** or an **adventitial haemorrhage**. In other words, while this study indicates that all three layers of the vessel wall may be involved by the trauma, the degree of trauma occurs in different proportions in various positions. In essence, all layers of the vessel wall are liable to damage.

Furthermore, while **intimo-medial** and **adventitio-medial tears** do occur, these combined **tears** occur with far lesser frequency than do the so-called 'pure' or discrete layer tears. This latter finding of a preponderance of discrete layer **tears** would be entirely in keeping with, and analogous to, **tears** occurring in other organ systems such as lung, liver, brain or meninges, where tissues tend to split in response to force application not only at tissue interfaces but also where different tissue densities are encountered. These findings therefore suggest either different vulnerabilities of the differing layers of the vessel wall to the forces applied, different types of forces acting on the various layers, i.e. stretch as opposed to compression, or differing compliances (the latter defined as the degree of yielding under applied force) of the different layers of the vessel wall, or a combination of the three.

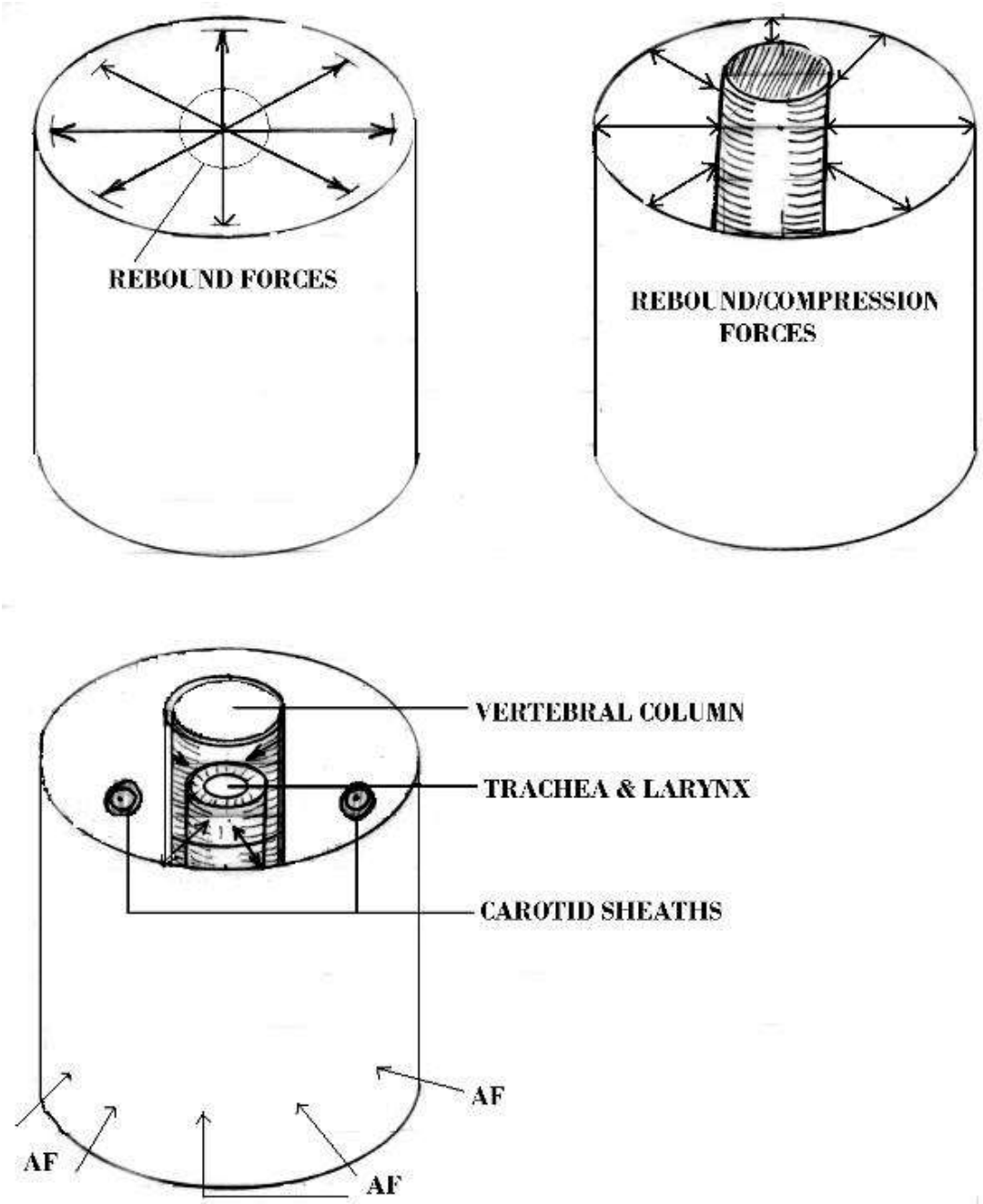
With regard to bilateral vessel involvement by the various types of lesion,while statistical inferences could not be drawn, pathological inferences could be made. Thus, only the **intima** and **adventitia** exhibited bilateral vessel involvement by the

same type of lesion. In other words, only the innermost and outermost layers of the vessel wall showed bilateral involvement. No involvement of the **media**, the middle or muscular layer of the vessel wall, showed bilateral involvement, either in the form of pure **medial tears**, **intimo-medial tears** or **adventitio-medial tears**. The question then arises: Why does this layer (the **media**) of the vessel wall appear to respond in an independent manner to the tensile and compressive forces applied? The **adventitia** and the **intima**, on the other hand, responded in a different manner, being equally affected by the circumferential externally applied constricting ligature. The mechanism of lesion production in the **media** thus appears to be different.

To reiterate, therefore, none of the subjects in the series showed bilateral involvement of the **media**, neither alone nor by **tears** extending either internally from the **adventitia** or outwards and externally from the **intima**. One inference that may be drawn is that the tougher **media**, consisting as it does of compacted, laminated layers of muscle fibres or 'plates' (this latter element enabling contraction of the **media**), of differing tissue density to that of either **intima** or **adventitia**, would be more resistant not only to an externally applied compressive force but also more resistant to subtle differences in force application. These differences could be attributable to the slight difference in angulation (+/- 5 degrees) of the **right common carotid artery** as opposed to the **left common carotid artery**. This is discussed in the Results chapter referring to the finding of a greater degree of atheromatous plaque involvement by the right-sided vessels. Alternatively, a slight degree of angulation of the head to one or other side, or subtle differences in tightness of application of the ligature may be present during the hanging process. These differences, insufficient in force and magnitude to affect the tough multiple laminated layers of the **media**, would nevertheless be sufficient to produce changes

in the more vulnerable and thinner **intima** and the more loosely disposed layers of the **adventitia**. Interestingly, the presence or otherwise of an atheromatous plaque producing intimal thickening appears to provide no protection to causation and production of an **intimal tear**, as seen in Figs. 7. 3a, 7.3b, 7.4a, 7.4b, 7.4c, 7.5, 7.9a and 7. 9b.

The position of the knot (when present) or sliding loop (Table II, p. 190), *vide infra*, showed an almost equal distribution between left and right sides with 19 on the left, 25 on the right, and with 6 cases showing an occipital position. Thus, it would appear to indicate that, regardless of the position of the knot, the **media**, in contradistinction to the more vulnerable **intima** and **adventitia**, remains impervious to or is more resistant to subtle differences in force application. These may consist not only of multiple shear and stretch forces (*vide infra*) but also the externally applied compressive force of the ligature. Resistance to the compressive force of the ligature may be provided by rebound forces from internal reinforcement of the circumferentially applied ligature by the incompressible fluid, i.e. tissue fluid contained within the surrounding extravascular interstitial tissues and blood, contained within a tubular structure which is, in turn, supported by the firm underlying bony and cartilaginous elements of the hyoid-larynx complex and vertebral column. An attempt to depict some of these forces diagrammatically is presented in Figure **8.1**.



With the technical assistance of Mr. J.Stevens

Fig. 8.1. A diagrammatic representation of the rebound forces produced by compression of cylindrical structures against the more rigid centrally situated hyoid-larynx complex.
AF = applied forces.

While the **intima** and **adventitia** appear to be more vulnerable and less resistant than the **media** to the tensile and compressive forces occurring in the hanging process, the mechanism of injury production may be both similar and yet different. If

a tubular structure is filled with fluid (which is incompressible) and an encircling compressive and constricting force is applied focally from the outside, the fluid contained within the tube will provide resistance to the force. However, as discussed in the section on 'pressure-flow relationships in collapsible tubes' (*vide infra*), a more important factor applicable to focal and localized application of pressure is that bending moments (the latter defined in terms of physics as a product of force and its distance from its line of action to a point) may occur at the point of pressure application. In other words, what this means in simple terms is that focal bending, kinking and deformation of the vessel wall occurs. These latter, it is suggested, may be responsible for the disruptive lesions noted on histopathological examination of the **arteries** and **veins**.

Compounding this mechanical factor will be, as indicated above, the anatomical factor of the resistance provided by the underlying rigid bony and cartilaginous elements of the laryngeal complex, trachea and vertebral column. It should be recalled that in 45 (90%) of the subjects (Table II, p. 190), the ligature lay over the upper third of the neck directly overlying these anatomical structures. These, by the nature of their anatomical location in relation to the level of the ligature, as well as their relative tissue density, provide resistance (counter-force) to the externally applied force by providing a firm underlying structure against which compressive force may not only be applied but also resisted by rebound.

A small explanation or clarification is, perhaps, appropriate at this point. Rebound or recoil is defined as a backward movement but with conservation of momentum. While, by strict definition, no loss of energy occurs on recoil this is not strictly true as energy goes into changing the state of the tissue by putting it into

compression. The latter produces damage, disrupting the targeted structure which is thus breaking and therefore absorbing energy.

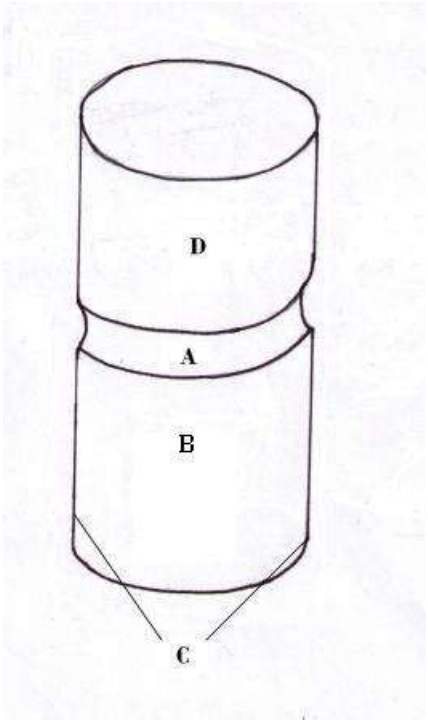
At this point, a simple example from mechanical and hydraulic engineering is, perhaps, appropriate. In a reducing volume of fluid, or when a constriction exists, the pressure exerted within the liquid increases. Thus, in an analogous manner, compressive forces applied by the externally constricting ligature impact against the different and multiple structures of the neck. These forces produce recoil backwards from the hyoid-larynx complex and vertebral column with pressure being directed and exerted on the neurovascular structures in an outwards direction. Part of this energy is then expended on bending and breaking the interposed and intervening neurovascular structures. An attempt has been made to depict these forces in Fig. 8.1.

With regard to other organ systems, the situation is somewhat analogous to that occurring in the context of frontal-impact motor vehicle collisions where the driver of the vehicle is propelled forwards striking the sternum against the steering wheel (in the absence of an airbag, as in older vehicles). Sternal impact then produces cardiac compression between the sternum anteriorly and the vertebral column posteriorly, resulting not only in the condition known as ‘commotio cordis’ with cardiac arrest but also in aortic rupture (although the latter has several mechanisms of causation).[1-5]

A further factor to be borne in mind, however, is that the contained column of blood within the **carotid arteries** would still be subject to the influence of cardiac contractile function with its 72 beats per minute, although this latter would slow down as a consequence of parasympathetic stimulation (*vide infra*, in discussion of the **neural findings**). Vagal inhibition then results with production of bradycardia and eventually in cardiac arrest. However, the physiological aortic pulse pressure

wave would still continue for several minutes although dwindling and diminishing in intensity and assuming that instantaneous cardiac arrest has not occurred. An aortic pressure wave travelling up through the **carotid arteries** at the rate of 3.2 to 4 metres per second could then be expected to impact 72 times a minute (although gradually lessening over the course of the next few minutes) against the arterial **intima** at the site of constrictive ligature application. The presence of a compressive and constrictive ligature would, it is suggested, similarly result in recoil of the contained column of blood at the site of constriction. Of course, the direction of such recoil would be back towards the heart, i.e. in a longitudinal direction although radial pressures do occur. In essence, the body of fluid contained with the vessel/vessels, i.e., the **carotid arteries**, is in equilibrium with pressure from the heart with longitudinal pressure being produced at the site of ligature constriction, i.e., at the interface with the fluid from the heart.

A further point which should be borne in mind is that hanging *per se* does not, unlike with gunshot or penetrating incised wounds, result in exsanguinating haemorrhage. Hence, a 'full' aorta is present, permitting propagation of the pressure wave. An attempt to depict the longitudinal tension and radial compressive forces produced is shown in Figure **8.2**.



With the technical assistance of Mr. J.Stevens

Fig. 8.2. A diagram showing the radial compressive and longitudinal tension forces acting on the vessel wall in hanging.
A = radial compression and longitudinal tension. B = longitudinal tension only. C = increased tension in vessel walls due to higher pressure loading from heart and longitudinal tension. D = no loading at all.

Thus, aortic pressure wave impaction with longitudinal tension and radial compression forces would tend to compound the stretch and shear forces taking place during the constrictive hanging process. The pathological consequence of such impaction would then be a stretching and stripping away of the delicate **endothelium** from its underlying **intima**. In other words, dissection would occur, analogous to and in the same manner as an aortic aneurysmal dissection occurring along the laminar planes of the **media**. This would account for the very high incidence of **endothelial**

elevation/avulsion noted in this study, i.e. 295 (98.3%) of the total number of 300 arteries examined and as seen in Figs. 7.4a, 7.4b, 7.4c. 7.7a, 7.14 and 7.25.

A further point which may be raised is that if **endothelial elevation/avulsion** is, at least, partially due to haemodynamics, it could, conversely, be argued that the presence of this finding indicates a continuing heart beat.

The question may arise as to whether direct pressure from the ligature could not also result in intimal stripping. While the wall of the artery is flexible and elastic, it is difficult to see, how the innermost and most shielded layer of the vessel wall from an externally compressive force can be affected in this manner. Put simply, the inner part of the vessel wall, i.e. the **endothelium**, under the ligature is protected while the 'stretched' part is at risk. Rather, as suggested above, the manner in which the ligature acts on the delicate **endothelium** is by 'strangulation', providing a localized constriction against which the aortic systemic pressure wave can impact. (The provocative use of the term 'strangulation' in the context of hanging is used with intent in order to emphasize the constricting effect of the ligature).

Notwithstanding the argument made immediately above, an attempt could be made to conceive whether direct ligature pressure could be conducive to intimal stripping. This would entail removal of an artery, placing a ligature around it, then filling and emptying it with fluid and then sectioning it serially to see the point of rebound damage. Unfortunately, the forensic pathology laboratory in Johannesburg is simply not equipped to conduct experiments entailing hydraulic engineering.

A further question which arises is how much pressure would be exerted within the **vertebral arteries**, which arise from the **subclavian artery**, a major branch of the **aorta**, when pressure is applied to one or more of the **carotid arteries** by the compressive ligature? In this regard, it should be borne in mind that the anastomosis

between **carotids** and **vertebrals** is mostly restricted to the **posterior communicating arteries** which are of relatively narrow calibre. Having removed the brain prior to performing a 'bloodless' dissection of the neck, rupture of these latter arteries as a result of blockage of the **carotid arteries** has not been noted in the present series of cases.

In ordinary circumstances, when the **carotids** are compressed, the volume and pressure below (i.e. on the heart side), would be increased. This would result in all anastomotic vessels swelling up to accommodate the loss of volume through the **carotids**. The **vertebrals**, being partly surrounded by bone, have a limited capacity to expand, i.e. limited 'expansibility'. The 'excess' blood, therefore, finds its way into surrounding anastomotic vessels. This would account for the **vascular congestion** noted in the periadventitial tissues. If these latter vessels rupture, **adventitial haemorrhage** ensues. And this occurs at the time of the hanging.

As indicated in the Material and Methods chapter, a number of cases of suicidal hanging not included in the present study were subjected to probe exploration of the **carotid arteries**. Prior to removal of the constricting ligature but after removal of the brain to provide a relatively 'bloodless' field, a probe was passed from below upwards into each **common carotid artery** on either side of the neck until the point of ligature application. In all cases, no further upwards passage of the probe was possible, attesting to the tightness of ligature application and completeness of vascular occlusion. The procedure was not carried out in the hanging cases included in the present study for fear that the probe itself might produce damage to the delicate **endothelium**. The inference that was thus drawn from the almost universal finding of **endothelial avulsion/elevation** in the study was that this finding was

genuine rather than artefactual, being due to the tightness of ligature application and its attendant forces.

The problem of fluid flow in deformable collapsible tubes transporting fluids within the body, i.e. arteries and veins and the analogous issue of wave propagation in such deformable tubes has been debated by biomechanical engineers for some time. These latter scientists have concluded that a so-called 'buckling phenomenon' occurs on wave propagation. [6,7] (While the word 'buckling' does not appear in the Oxford English Dictionary, to buckle means to give way or crumple up under longitudinal pressure). 'Buckling' of the vessel wall implies deformation, with consequent increased wall stress (defined in mechanical terms as the force per unit area exerted between contiguous bodies and which causes the 'buckling') at that site. It is suggested that 'buckling' would be augmented by the deformation of the vessel wall produced by the locally constricting effect of an externally applied compressive ligature and that this in turn would be one of the factors producing the arterial wall disruptions noted in the present study.

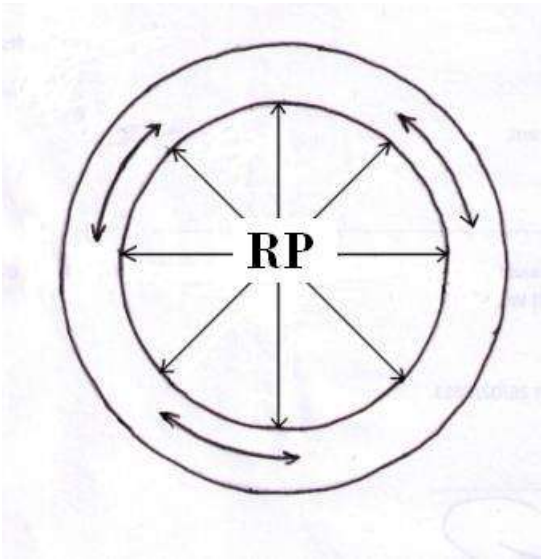
A further factor that should be considered in the context of the aortic pulse pressure wave is that synchronous with the heart beat the wave produces vessel dilatation which results in tissue stretch, similar in effect to that of a missile-induced hydrodynamic intravascular shock wave. [8-23] While similar in effect, the missile-induced shock wave differs in origin to the tensile forces occurring in hanging, although these latter are induced not only by the vessel stretch inherent to the hanging process but are compounded by the aortic pressure wave. It should be borne in mind that anatomically the course of the cervical **carotid arteries** is not only vertically upwards in the neck but that both the **internal carotid and external carotid arteries** are of narrower calibres than their parent **common carotid artery**.

These anatomical and structural characteristics could possibly contribute to upwards propagation of an intravascular pressure wave but, in addition, the anatomical narrowing in the calibre of the artery would tend to create a point of focally-directed internal pressure stress conducive to vessel stretch, if Young's modulus (*vide infra*) were different in the two types of tissue. The latter, in turn, could not only result in stripping of the **endothelium** but also manifest pathologically as the **fenestration of the media** seen in Figs. 7.2, 7.9a, 7.9b, 7.11 and 7.26.

'Pathological dissection' due to the trauma of hanging could further account for the histopathological finding of red blood cells tracking up not only between the delicate **endothelium** and its underlying **intima**, but also for the finding of red blood cells tracking along the **internal elastic lamina** as seen with **intimal tears** extending to and along this layer of the vessel wall in Figs. 7.3a, 7.3b, 7.4a, 7.4b, 7.4c, 7.5, 7.6, 7.15a, 7.15b, 7.10a and 7.10b in Chapter 7. In other words, one of the factors causing the damage seen in the innermost and most delicate layer of the vessel wall, the **endothelium** and its underlying **intima**, may be due to the repetitive aortic pressure wave impacting against an **intima** constricted and compressed by the externally applied but internally directed force of the ligature.

An additional interesting feature of **intimal tears extending to or along the internal elastic lamina** was the further longitudinal continuation of the **tear** along the **internal lamina** without breaching it. In other words, the **internal lamina** appears to have acted in these cases as a barrier to further radial disruption of the arterial wall with energy dispersion occurring in what appears to be the path of least resistance- the longitudinal plane. As indicated below, a similar tendency was observed along the laminar planes of the **media** with regard to **fenestrations** of this layer of the vessel wall.

From a mechanical viewpoint, tearing can only occur in areas of tensile stress (personal communication; Thomas Dessein, School of Mechanical, Industrial and Aeronautical Engineering, University of the Witwatersrand, Johannesburg). As shown in Figure **8.3**, radial pressure outwards causes circumferential so-called "hoop stress" which, in turn, causes longitudinal tears. Longitudinal tearing thus implies circumferential stress in the vessel walls due to a higher pressure in the vessel than the surrounding tissues.



With the technical assistance of Mr. J.Stevens

Fig. 8.3. A diagram showing the radial pressure producing circumferential "hoop stress" on the vessel wall . RP = radial pressure. Curved arrows = circumferential "hoop stress".

A further factor that should be borne in mind is that arteries have the conical geometry of a duct.[24] In other words, while a reduction in blood vessel diameter occurs as one moves further away from the heart, an increase in diameter takes place just before a bifurcation. Anatomically, this increase in vessel diameter is seen in the

case of the **carotid sinus**, where the diameter of the vessel wall increases just before the **common carotid artery** bifurcates into its respective **internal carotid artery** and **external carotid artery** branches. Studies of bifurcations at physiological flow velocities have shown that laminar flow is heavily disturbed at points of bifurcation and, furthermore, disturbances in the flow influence parameters at the walls of the vessels. [25-27] Upstream (i.e. away from the heart) of branches, vortices (defined in terms of physics as a portion of fluid whose particles have a rotatory motion, i.e. any 'whirling fluid' system) are <u>always</u> present at certain values of the flow rate. [25] These vortices then retard the flow of blood resulting in a zone of higher static pressure (defined in terms of engineering or physics as a body or force acting as a weight but not in motion) which stresses the arterial walls leading to wall damage. Thus, a close connection exists between possible wall damage and the mechanics of fluid flow and probably more so in disturbed mechanics of fluid flow.

However, an important, and additional, factor that should be considered in producing the vascular lesions noted is **vascular spasm**, either due to the effect of the constricting ligature with haemorrhage-induced vascular spasm or to reflex neural action.[28,29] It is suggested that vascular spasm might not only enhance but even produce disturbed mechanics of flow leading to pathological manifestations such as 'stripping off' of the delicate vascular **endothelium** from its underlying **intima**. Vascular spasm may further contribute to the decreased cerebral blood flow attendant on ligature application to the arteries of the neck and consequent haemorrhage-induced vasospasm. Could reflex vascular spasm thus be the cause of the early unconsciousness noted in the human experiments carried out in the 19th century, rather than pure vascular occlusion per se? Compounding these effects would be reflex vaso-vagal stimulation resulting in cardiac arrest with stoppage and

cessation of blood flow. This latter factor would, in part, account for the finding of empty **carotid arteries** and **internal jugular veins** noted as well as producing hypoxia and cerebral ischaemia.

A further point which comes to mind is that with incomplete obstruction of blood flow within the **carotid arteries** (although the tightness of ligature application would tend to negate this, complete obstruction to flow occurring) the period of consciousness would be prolonged and the consequent lapse into unconsciousness be delayed. This would then permit perhaps 'second thoughts' on the part of the victim with evidence of attempts to loosen the ligature being found in the form of fingernail abrasions around the site of application of the ligature. These, however, were not found, attesting once again to the tightness of ligature application with rapid unconsciousness then supervening.

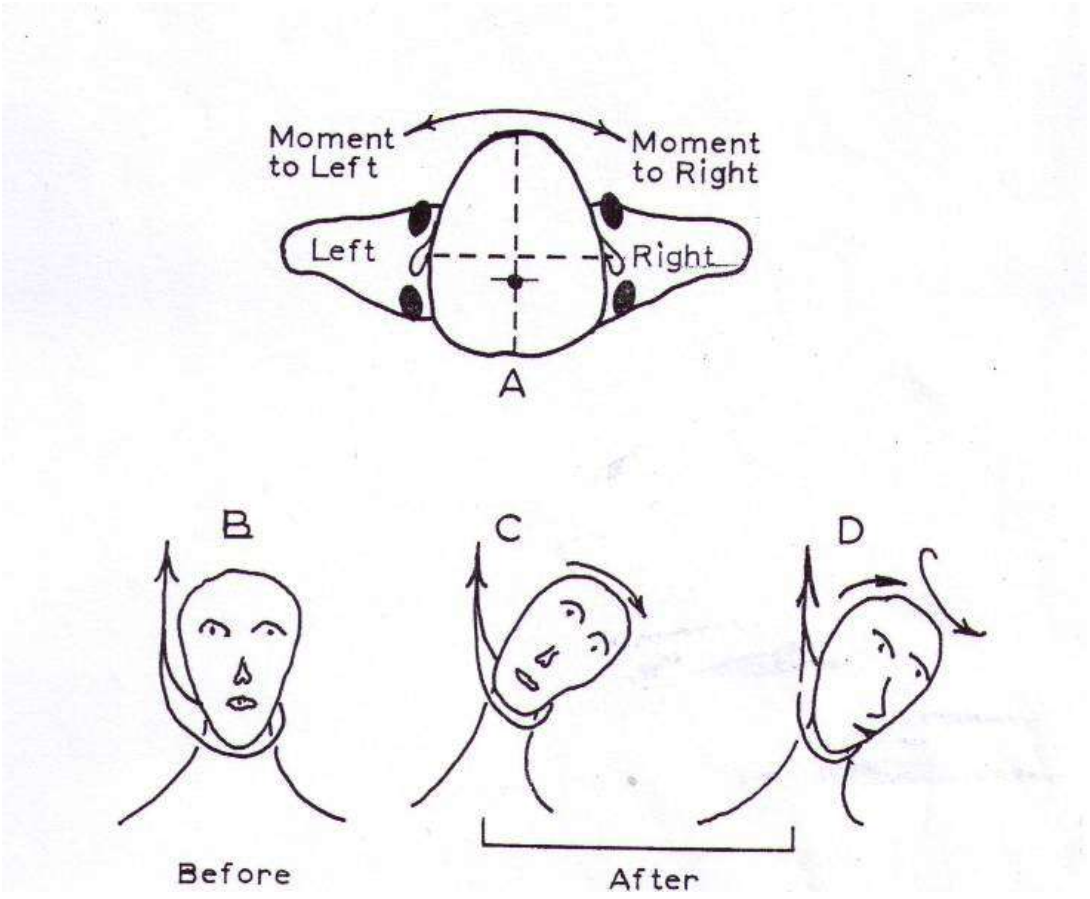
Reference to Table II (p.177) indicated that the level of circumferential ligature application lay over the upper third of the neck in 45 subjects (90%), i.e. as high as the ligature could possibly go in its application, that is, against the body of the mandible. (It should, however, be borne in mind that the initial application of the ligature around the neck by the victim may have been lower down but that either the weight of the body or a jump by the victim may have resulted in the ligature assuming as high a position as possible around the neck. The upper-third of neck level may, therefore, be a secondary phenomenon). In addition, the tightness of ligature application in many cases may indicate that either a large part of the weight of the body or its entire weight was applied to the ligature thereby enhancing constriction with all the pathological and physiological sequelae ensuing therefrom.

In those cases where the ligature had been removed at the scene by the paramedical personnel and not submitted with the body to the mortuary, the position

of a knot having been present was deduced by an irregular and wider area of abrasion along the circumference of the ligature impression. Conversely, the presence of a sliding loop when the ligature was not present was deduced by an area of absence of abrasion along the circumference of the ligature impression. An intriguing finding emerged with regard to the position of the knot or sliding loop. These were found to lie over the right side of the neck in 25 subjects (50%), i.e. half, over the left side of the neck in 19 subjects (38%), i.e. about two-fifths, and over the occiput in 6 subjects (12%), i.e. slightly more than one-tenth. In other words, the position of the fixed knot or sliding loop lay predominantly over the right side of the neck. In addition, a fixed knot had been present in 29 subjects (58%), i.e. about three-fifths and a sliding loop in 21 subjects (42%), i.e. about two-fifths. One would assume that the outermost layer of the vessel wall, i.e. the **adventitia** would bear the brunt of any **adventitial haemorrhage** due to vascular rupture in view of this being the layer of the vessel wall in closest apposition to the overlying ligature. The findings in the **right internal carotid artery**, for example, showed involvement by **adventitial haemorrhage** in 15 subjects (30%) while the left-sided artery showed involvement in 18 subjects (36%). Hence, the difference is only 3 cases out of 50 being nearly equal, thus confirming the inference of adventitial vulnerability to compressive and tensile forces. This, despite a sliding loop leaving a gap at the site of ligature application, no contact between the ligature and the underlying tissues having been made at this point. In addition, the five to six kilogram weight of the head falling to the side opposite, i.e. contralateral to the position of the loop would tend to further draw the tissues away from the loop. This would then produce an element of tensile stress due to vessel stretch on the open side of loop application, i.e. at the point of the gap between loop and neck, on the side contralateral to the position of the head. Thus, as

indicated, subtle and conflicting forces, some of which may be non-findable, come into play during the hanging process and may be different in individual cases.

What of additional factors that should be considered in contributing to possible vascular occlusion and vessel damage during the hanging process? These would include rotational forces producing neck rotation and consequent vessel 'twisting'. Considered anatomically, the **atlas**, or 1st cervical vertebra, is a ring supporting the skull. Phylogenetically, this vertebra has lost its body, the lost body forming the **dens** (odontoid process) of the **axis**, the 2nd cervical vertebra. The **dens** acts as a central pivot around (about) which the neck can rotate, the **atlas** thus rotating around its own lost body. A further anatomical factor contributing to and permitting neck rotation is the sturdy configuration of the **axis** and the point of attachment of its muscles. The bifid spine of the **axis** is massive because large deep muscles i.e., **Multifidus** and **Semispinalis Cervicis** extend upward to it but no further. This leaves the **atlas** free to rotate around the **dens** while these muscles are extending the **vertebral column**. Contributing further to the normal anatomical function of these latter muscles in extension of the **vertebral column** would be the additional extension produced by the weight of the body under the influence of gravity during the hanging process. The body itself might also rotate around the central pivot of the neck enhancing any vascular 'twisting' which might occur. An attempt to depict the rotational and 'twisting' forces which might occur is illustrated in the following diagrams (Figure **8.4**).



With the technical assistance of Mr. J.Stevens

Fig. 8.4. A diagrammatic representation of the rotational and twisting forces acting on the neck as it pivots around the central axis of the vertebral column.

With regard to the finding of **complete circumferential transverse rupture**, this was found to be present in only three subjects (6%). This appears to indicate that in these cases the force exerted was of sufficient magnitude to cause complete vessel transection. A further possible inference that could be drawn from this finding is that even if the victim jumped off a stool or a chair, thus producing a 'jerk' analogous to or resembling a judicial hanging, the 'drop', despite the weight of the body, was

insufficient to completely transect the artery. Of course, as may occur in hanging, ‘abduction’ or ‘twisting’ may further contribute to vessel disruption and tearing.

In none of the cases in the series was the so-called ‘hangman’s fracture’ found to be present on neck dissection. This fracture, seen in judicial hangings, consists of fracture of the pedicles of the axis (C-2) resulting in anterior dislocation of C-2 on C-3 with or without a fracture of the odontoid process. As an interesting point, in most cases of ‘hangman’s fracture’ the spinal cord may not sustain injury due to the anatomical feature of the considerable space surrounding the cord in this region. In other words, the anatomical space permits flexion or ‘twisting’ within the space surrounding the cord.

Mason, in his noted work “ The Pathology of Trauma”, has made the important point that in judicial hanging the weight of the body is used to provide a sudden distracting force upon the neck. This force then avulses one portion of the brainstem from another, or the brainstem from the spinal cord. However, Mason also makes the point that judicial hanging constitutes a major exception to the general rule, based on an hypoxic aetiology, that brainstem death is equivalent to whole brain death. Nevertheless, the point is made that the cerebral circulation is cut off very rapidly by the full-body suspension by ligature which inevitably follows the drop. The time to cerebral anoxic unconsciousness is, therefore, correspondingly short.

What conclusions could thus be drawn from the **vascular findings?** Certainly, varying degrees of vessel tearing and vascular haemorrhage could be expected during the hanging process due to the compressive and tensile forces taking place. However, what was unexpected was the sheer fragility and vulnerability of the outermost and innermost layers of the vessel wall to these forces. In the innermost layer of the vessel wall, the **intima**, these manifested histopathologically as **endothelial**

elevation, **endothelial avulsion** and **dehiscence along the internal elastic lamina**. In the outermost layer, the **adventitia**, these manifested as **adventitio-medial separation** and an anatomical **vascular plane of cleavage**. Even the layer most resistant to force application, the **media**, showed itself vulnerable to tensile stretch forces in the form of **fenestrations** (i.e. excessive 'separation' or 'gaps' between the layers of muscle. Moreover, the presence of **haemorrhage** within these 'splits' attested to the *intra vitam* nature of the lesion. However, a compressive force acting to produce **haemorrhage** as a consequence of vascular rupture cannot be excluded in view of the anatomical distribution of the **vasa vasorum** which are known to penetrate to the outer-thirds or middle-thirds of the **media**. In a sense therefore, and confirming the remark made above with regard to the rapid unconsciousness supervening in judicial hanging, this type of suicidal hanging with a 'drop', with its attendant severe stretch forces, may similarly produce the cerebral ischaemia and anoxia seen in judicial hanging, albeit with no fracture.

While the major arterial and venous vessels were noted to be devoid of blood, a very prominent feature was the **vascular congestion** of the small vessels noted in the adventitial and periadventitial tissues. This finding also comprised one of the features noted in and about the **nerves**, the **ganglia**, the **carotid body** and the **accessory glomi** (*vide infra*). What could account for this? Was this purely due to a failing circulation with decreased venous return or were other factors at play? It is known that postganglionic axons travel to local blood vessels with the parasympathetic fibres being vasodilatory. Could, therefore, the **vascular congestion** found in the periadventitial tissues and within and in relation to some of the **nerves**, **ganglia**, **carotid bodies** and **accessory glomi** be due to parasympathetic nervous system stimulation? This issue will be explored in greater detail under the **neural findings**.

What is the significance of the above findings in the light of various experimental studies dealing with the biomechanics of trauma and the response of organ systems to impact? While these studies primarily involve the relative tolerance of a number of organ systems other than arteries to trauma, [30,31] or the mechanism of traumatic rupture of the aorta, [32,33] a vessel with differing anatomical and haemodynamic characteristics than those of the relatively smaller calibre cervical **carotid arteries**, they nevertheless shed some light on the problem. However, a more detailed discussion of the biomechanical forces at play is undertaken in the section dealing with the engineering and biomechanical principles involved in producing damage to biological and in particular to arterial tissues.

Several mechanisms have been implicated in tissue damage following force application. The human body, from a mechanical viewpoint, is an extremely complex system which may be described as a heterogeneous viscoelastic mass within which run a number of channels filled with blood and other fluids such as lymph. When such a system is compressed or impacted, the immediately resulting disturbances are partly mechanical and partly thermal in nature. The most important changes are mechanical, and appear mainly at the site of impact or compression with displacement of parts of the system relative to one another. In the thoracic aorta, this mechanism has been invoked to explain rupture, [34] where the fixity of the ascending aorta, arch and descending aorta vary relative to one another, and that torsion and shearing stresses occur not only between these different segments but also between the aorta and other thoracic organs. It is assumed that at the instant of force application the relatively mobile descending aorta, with its contained column of blood, bends forward at its junction with the fixed aortic arch at the isthmus, generating shearing stress at this site with varying degrees of rupture. The factors

described for the aorta may be applicable to the **carotids** in view of the sudden application of an external force by a ligature. In other words, could hanging be construed as a form of acceleration/deceleration injury?

Bearing in mind the anatomical proximity, complexity and integration of the tissues in the neck as well as the 'take-off' of the **internal and external carotid arteries** at the point of bifurcation from their parent **common carotid artery** on either side of the neck, could similar shearing stresses conceivably explain the occurrence of **tears**, localised particularly to sites of vessel bifurcation, the point at which the tissue specimens were taken? Such stresses could, in part, explain the finding of avulsions and splits along the **internal elastic lamina** and the laminar planes of the **media**, but, in the context of hanging, would be compounded by the tensile and stretching forces to which the arteries were subjected.

The presence of a tightly applied constricting ligature to the neck and its arteries, compounded by the weight of the body and angulation of the neck as a consequence of the head falling to one or other side could reasonably be expected to produce tissue deformation. It is of **biomechanical** interest that tissue deformation and its shearing stresses leads not only to displacement of one part of the system relative to another, but also to the generation of a pressure pulse wave throughout the system. It has been experimentally demonstrated that within the vascular elastic tissues, whose principal physiologic function is to provide a hollow tube with considerable tensile strength, a so-called water-hammer effect may be caused by a pressure wave within the column of blood.[35] Furthermore, the magnitude of this water-hammer effect increases with the degree of velocity change. Thus, pressure on the **carotid arteries** may result in a pressure wave being transmitted along the column of blood in accordance with Pascal's law. [35] The **carotid arteries**, arising either directly from

the aortic arch or its brachiocephalic branch, are immediately in continuity for transmission of both the systemic head of pressure and this pressure pulse wave. Thus, these forces, acting in concert, are postulated to account for the 'stripping-off' of the delicate **endothelium** from its underlying **intima** as well as the tendency for **dehiscence along the internal elastic lamina**. In this regard, the effect of the systemic head of pressure would be magnified not only by impacting against a vessel constricted by an overlying ligature but would be compounded by the repetitive effect of this pressure wave occurring 72 times per minute, as alluded to previously. In this regard, it should be borne in mind that the heart continues to beat due to its inherent rythmicity. Furthermore, the **carotid arteries** lie distal to the heart but with 'take-off' from the **aortic arch** on the left and the **brachiocephalic artery** on the right, thus rendering the velocity of blood flow within the **carotid arteries** almost equal to that of the **aorta**. These anatomical and vascular flow factors thus place the **endothelium** and **intima** (and possibly other layers of the vessel wall) at risk at the site of vascular constriction due to the ligature.

A further interesting postulate arises in the context of the so-called water-hammer effect alluded to above. Could this mechanism be initiated in hanging not only on a pathophysiological basis but on an anatomical basis as well? While in the present study the particular circumstances of each hanging case were not known due to inadequacies in police investigation and history, it is reasonable to assume that, at least in some of the cases, a 'drop' from a chair or stool would have taken place. A sudden drop with its accompanying jerk would not only produce vessel stretch but would result in sudden arrest of the intra-abdominal organs against the **pelvic brim**.

Briefly, the **pelvic brim** forms the boundary line between the greater or major pelvis above and the lesser or minor pelvis below. Crossing the **pelvic brim** are

structures pertaining to the gastro-intestinal tract, the uro-genital tract and somatic structures, i.e. those pertaining to body wall. Of particular importance to this possible mechanism of injury in hanging are the structures pertaining to the gastro-intestinal tract, its accompanying arterial and venous vessels, and the somatic structures. The structures pertaining to the gastro-intestinal tract comprise coils of the ileum, the sigmoid colon, the mesocolon, the omentum (often loaded with fat), the inferior mesenteric artery and vein and the hypogastric plexus of nerves. The somatic structures of importance in the context of suicidal hanging comprise the **internal iliac artery and vein** and the sympathetic trunk. The **internal iliac artery** takes origin from the **common iliac artery** one-third of the way, i.e. 2 inches, along the line joining the **aortic bifurcation** to the midinguinal point. Of interest is that no muscles cross the **pelvic brim** and, therefore, fascia reaching the brim is attached to it. The component parts of the **pelvic brim** itself are, of course, bony, comprising the promontory of the sacrum, the anterior border of the ala of the sacrum, the iliopectineal line (which extends from the ala to the pubic tubercle), the pubic crest, and the upper end of the symphysis pubis.

Assuming that force diffusion occurs in <u>all directions</u> at the end of the ‘drop’ as the intra-abdominal organs come to an abrupt halt (with some oscillation), the **internal iliac artery and vein** would then be propelled backwards against the **pelvic brim**. It is suggested that sudden abrupt compression of the **internal iliac artery** against the pelvic brim would take place, in a manner analogous to the abdominal aortic compression which occurs in a pedestrian lying in the road after having been struck initially by a first vehicle and subsequently run over by a second oncoming vehicle. While sudden abrupt compression of the **internal iliac artery** against the **pelvic brim** is momentary, it could produce a pressure wave. The mechanism and

dynamics of pedestrian injury in persons run over by motor vehicles has been well-described in Mason's book "The Pathology of Trauma".[36]

In accordance with standard medico-legal autopsy techniques, all the body cavities were eviscerated and the individual organs dissected. During the course of the autopsies on the hanging victims in the present study, it was noted that in all cases **adventitial haemorrhages** of the **thoracic** and **abdominal aortae** were found to a greater or lesser degree. However, as indicated in the Results chapter, in none of the hanging cases examined was any damage found to the **hyoid-larynx complex**, its associated **cartilages**, or the **vertebral column**, although, as shown in **Figs. 7.55a.** to **7.68.**, and their accompanying legends and text, clear-cut damage to the surrounding **nerves**, **veins**, **connective tissue** and **lymph nodes** deep to the site of ligature application was noted. This is in keeping with suicidal hanging, where fractures of the **hyoid-larynx complex** are rarely encountered. This is in marked contrast to the situation found in judicial hangings, manual strangulation or accidental hangings. In these latter instances, excessive and violent force is applied to the neck, impinging and impacting upon the **hyoid-larynx complex** and **vertebral column**.

Schleyer attributed the **adventitial haemorrhages** of the thoracic **aorta** which he found in victims of throttling, strangulation, phenobarbitone overdosage, hepatic coma and gunshot wounds of the brain to an acute agonal rise of the intrathoracic blood pressure resulting in rupture of the **vasa vasorum**. [37] Could, however, the **adventitial haemorrhages** of the thoracic and abdominal **aorta** noted in the present study be due to the physical drop with impaction of the **internal iliac arteries** against the **pelvic brim** with production of a retrograde pressure wave be the cause of these **haemorrhages** ?

It is thus postulated that sudden **internal iliac artery** compression against the **pelvic brim** as a consequence of the anatomical factors described above, could similarly tend to induce a retrograde water-hammer effect within the **common iliac artery** and **abdominal aorta**, with propagation of a pressure wave both upwards and downwards. Impaction of this pressure wave against a **carotid artery** occluded and constricted by an externally applied ligature might then be a contributory factor to the **endothelial avulsion** and stripping observed in the present study. Of course, in the context of hanging, the force exerted would be vertical rather than horizontal as in a pedestrian run over by a vehicle but the effect would, nevertheless, be similar although of considerably lesser magnitude. Bearing in mind the delicacy of the **endothelium**, the magnitude of the pressure wave exerted might be sufficient to induce **endothelial avulsion** compounded by or contributed to by the systemic head of pressure impacting against a focal point of vessel constriction. Hence, both mechanical and pathophysiological consequences may ensue as a result of anatomical factors.

While direct vessel compression may account for the high incidence of **adventitial haemorrhages** found in the series, early historical observations documented in the German literature now assume greater importance in the light of modern well established laws of solid state physics. In 1893, Rindfleisch held the view that traumatic rupture is produced by a sudden stretching of the vessel. [38] This conforms to present day concepts of the Young's modulus or elasticity of arterial tissue [39] which has been described by Nickerson [40] and von Gierke. [31] These authors hold that while arterial tissues are endowed with considerable tensile strength, on rapid dynamic transverse stretching (i.e. 'stretchability'), as occurs in humans on application of force, the rate of stretching (rate being defined as a stated value of

numerical proportion prevailing between two elements) increases tremendously. The tissues become ‘stiffer’, more rigid, and fracture more readily.

What of other early investigations? While these were conducted on human aortic tissue, they nevertheless may have relevance to the processes occurring in those vessels arising either directly from the aorta, i.e. the **left common carotid artery**, or, on the right, from its brachiocephalic branch, namely, the **right common carotid artery**. These processes may have relevance not only in view of the origin and anatomical proximity of these latter vessels from the aorta or its brachiocephalic branch, but also in light of their being large muscular arteries, as is the aorta.

In 1918, Oppenheim [41] ligated the branches of human aortas and filled the vessels with water at a pressure of up to 3000 mm Hg. It was then noted that ruptures regularly occurred in the ascending part, an intriguing finding in view of the fact that both **common carotid arteries** and their respective **internal and external branches** constitute vessels which similarly ascend ‘vertically’, i.e. ‘vertical’ implying in the upright posture in hanging, (albeit with a **s**light degree of angulation on the part of the **right internal carotid artery**). While such pressures are unlikely ever to occur in the living, Oppenheim surmised that lower levels of increased intraluminal pressure may cause rupture *in vivo* because the resistance of the aorta to stress during life is lower than after death. Klotz and Simpson conducted similar experiments in 1932, and found that the postmortem aortas of young people resisted an internal pressure of about 1000 mm Hg without rupturing.[42] However, they nevertheless assumed that a sudden rise in blood pressure on application of force ruptured the vessel. As previously indicated, Schleyer attributed the **adventitial haemorrhages** of the thoracic aorta which he found in victims of throttling and strangulation to an acute agonal rise of blood pressure resulting in rupture of the **vasa vasorum**.[37] While

the **adventitial haemorrhages** of the **aorta** noted by Schleyer may indeed have been due to an acute agonal rise in blood pressure, the mechanism of blood pressure rise in hanging may lie in an abrupt backward movement of the **internal iliac arteries** against the **pelvic brim**, an anatomical mechanism and causation not previously considered.

As far back as 1847, M.G. Wertheim presented force-elongation data for arteries and veins, data particularly pertinent to these tissues in view of their both longitudinal and radial configuration. [43] Without going into the mathematical principles of his work, which is beyond the scope of the present study, Wertheim's data led him to conclude that soft tissues, including arteries and veins, do not obey Hooke's law. This latter law states that a linear relation exists between stress and a linearized measure of strain (strain in this context meaning a pull, stretching or tensile movement or an injury or change of structure resulting from such exertion or movement). Roy in 1880 came to a similar conclusion. [44] For example, Roy observed that arteries exhibit an anisotropic response, i.e. having physical properties exhibiting a directional response such as elasticity. This response manifests as an 'elastic after-action' which indicates the particular tissue as having a **biomechanical** viscoelastic character and which is time dependent. Roy additionally showed that the material properties of arteries differ with radial location within the wall, i.e. a local heterogeneity (defined as being composed of diverse elements or of diverse character) and along the vascular tree, i.e. a regional heterogeneity. In other words, arteries exhibit differing characteristics according to their histological type and according to their anatomical location along the numerous ramifactions of the vascular tree.

Thus, soft tissues are essentially non-uniform regardless of any overall homogeneity (defined as consisting of uniform parts). This non-uniformity, part of which depends on the radial location in the wall, for example at sites of bifurcation, explains, perhaps, the arterial disruptions and lesions occurring in this study. It should be recalled that the arterial sections in the present study were taken in the region of bifurcation of the **common carotid artery** into its respective **internal and external branches**.

As far back as 1926 Murray suggested that biological organization and adaptation are observed facts conforming to definite laws because, statistically at least, there is some sort of uniformity or determinism in their appearances. [45] Murray further suggested that the basic principle underlying the organization of biological systems was that the biological cost of operation of physiological systems tended to be at a minimum. Over the years, a number of investigators have used this concept of cost minimization of biological systems in an attempt to describe a variety of biomechanical observations of the vasculature. Thus, the bifurcation pattern, as observed with regard to the **common carotid artery** and its bifurcation into **internal and external carotids**, appears to follow from Murray's principl of biological organisation stated above. Of particular relevance to this study, however, is the tendency of a blood vessel to regulate its calibre in order to maintain wall shear stress at a particular value. [46] When this ability of a blood vessel to regulate its calibre is inhibited or obstructed by, for example, a constricting ligature, wall shear stress can no longer be maintained at a value optimal to the structural integrity of the arterial wall. Tearing must then occur due to imposition of non-physiological loads with their resultant dynamic instability. The manifestations of such instability would be dissection and over-stretching of the **media** with smooth muscle damage which

would account for the multiple **tears** of the vessel wall and the **fenestrations of the media** noted. It would further account for the atheromatous plaque disruption, plaque fracture, endothelial denudation, and delamination between the arterial layers encountered. All of these biomechanical manifestations of force imposition and shear stresses were noted in this study in the form of **endothelial elevation or avulsion, dehiscence along the internal elastic lamina, microtears of the intima, adventitio-medial separation** and the multiple types of tearing encountered. An excellent example of these mechanisms and manifestations under a controlled clinical environment occurs in the procedure of balloon angioplasty. In this procedure, a balloon-tipped catheter is inflated within a diseased artery for the purpose of weakening the plaque structure at the balloon interface with the arterial wall so that the normal distending systemic head of blood pressure can further expand the arterial lumen. Plaque fracture is, therefore, deliberately induced in order to enhance expansion of the artery lumen by the distending head of blood pressure. This does not occur against the constricting force of an encircling ligature. The systemic head of pressure impacting against a vessel compressed by an externally applied constricting force could, it is suggested, be a major contributory force to the various forms of arterial wall tearing noted in this study. A particularly expressive and florid example of vessel wall 'blow-out' is seen in Fig. 7. 22 in the previous chapter where **complete circumferential transverse rupture** in an outwards direction is demonstrated.

While sceptics may be tempted to conclude that the effect of tearing of the arterial wall is simply brought about by the sheer pressure of the sudden application of the ligature on the vessel wall, i.e. simply 'direct pressure damage', cognizance

must surely be taken of other factors that may have had an effect in producing the vessel wall damage observed in this study.

Pressure-flow Relationships in Collapsible Tubes

While some attention has been directed to the pathophysiological processes which take place in **arteries** on application of a constricting, compressive ligature to the neck, what of the **veins**? From a **biomechanical** viewpoint, veins are essentially thin-walled collapsible tubes transporting blood throughout the body. Hence, the pressure-flow relationships within these is of particular importance in understanding some of the pathophysiological processes taking place in the veins during the hanging process.

Thin-walled collapsible tubes such as **veins** are, biomechanically, 'structurally non-self supporting'.[47] This is in contrast to **arteries** where the **media** acts as their inherent self-supporting structure. It is in these venous vessels that the flow resistance may be controlled by sensitive changes in the transmural pressure which cause the tube to collapse or open very rapidly. The cross-section of the tube changes from a circular to an elliptical form (approximately) and then back to a circular section. Relatively large volume changes occur at an elliptical section of the vessel wall for small increments in the transmural pressure, whereas considerably larger changes in pressure influence the volume much less when the tube is near its fully distended circular cross-section. The reason for this lies in the distensible nature of vessel walls which must accommodate changes in blood pressure and blood flow. Caro and Saffman[48] have shown that the ellipticity (defined in engineering terms as having the characteristics of an ellipse, i.e. a regular transverse oval, traced by a

point moving in plane so that the sum of its distances from two other points is constant. Alternatively, a regular oval produced when a cone is cut by a plane making a smaller angle with the base than the side of the cone makes) becomes important for eccentricities (defined in geometrical terms as a circle not having its axis placed centrally) greater than 70 per cent.

Of relevance to the situation in hanging is that such collapsible tubes are typical of certain segments of the larger veins within the thorax [49] and those entering the thorax. [50] Thus, this applies particularly to the **internal jugular veins** which not only carry venous blood directly to the thorax but are constricted by the compressive ligature, enhancing the collapsing effect. Furthermore, in the case of the veins, two additional factors complicate the pressure-flow relationships:

i) the veins traverse sealed regions or chambers of different surrounding pressure, which include the thorax, and;

ii) ii) the pressures in these chambers change with the time variations of respiratory activities.

From the point of view of physics, the lateral dimensions of a tube decreases during tube collapse. The significance of this to the situation in hanging, where application of a compressive ligature to the neck is present, is that as a blood vessel collapses with decrease in vessel diameter, the shear forces acting at the site of constriction or deformation increase. The shear forces thus generated then carry implications with regard to disruption of vessel wall integrity.

In addition, the time-dependent transmural forces to which the vessel wall is subjected depend upon the arterial and venous pressures and, in the lung, alveolar pressures, as well as upon the transverse components of the longitudinal forces developed in the collapsing vessel. The summation of these forces results in localized

bending moments developing in the vessel wall. These, it is suggested, account at least in part, for the disruptive lesions of the venous wall noted in the present study, and as seen in Figs. 7.23, 7.61 and 7.68. In terms of physics, in the vicinity of the localized zone of collapse, i.e. implosion, the cross-sectional area and transmural pressure, under physiological conditions, fall off rapidly. Under the conditions occurring during the hanging process, rather than a fall off in transmural pressure, the converse obtains, an "unremitting external pressure" being applied to the vein wall.

As indicated in the section dealing with pressure-flow relationships in collapsible tubes (*vide supra*), the application of a ligature induces a 'bending' moment (moment being defined in terms of physics as a force producing 'rotation' or 'twisting', i.e. torque) in the vessel wall. In the case of a sliding loop, the 5 to 6 kilogram weight of the head falling to the side opposite, i.e. contralateral to the position of the loop, would tend to draw the tissues and their **carotid arteries** away from the loop. On the side of the loop, this would then tend to induce a compressive force on the medial, internal aspect, of the vessel wall and a tensile stretching force on the lateral, external aspect, of the vessel wall, i.e. 'bending' moments.

B. The Neural Findings, their Biomechanical Causation and their Pathophysiological Implications

These, as indicated, comprised **neural, ganglionic, carotid body** and **accessory glomal congestion, haemorrhage, internal dehiscence, tearing, adventitial separation** and **perineural separation**, i.e. findings which could be attributable to compressive, stretch and shearing forces. These, in large measure, reflected the **vascular findings**, albeit in different proportions, and particularly evident in the

notable paucity of **carotid body tearing** and **internal dehiscence** in comparison with these findings in the **vascular and neural elements**.

It is suggested that while the findings were remarkably similar in their pathological manifestations, the difference in proportional representation seems to lie in the different microanatomical structure of longitudinal elements such as **arteries** and **nerves** as opposed to rounded, discrete, multicentric and spherical structures such as the **carotid bodies** and their **accessory glomi**. Vascular elements, such as **arteries**, are, in addition, subjected to further forces applicable to distensible and deformable fluid-filled tubes under the influence of the systemic head of pressure. These forces have been alluded to above and will be further elaborated upon and discussed in the section dealing with the bioengineering and biomechanical principles pertaining to such structures (*vide infra*).

Beginning with the **carotid bodies** and their **accessory glomi**, what would account for the much lower incidence of traumatic disruptions noted as opposed to the **nerves** and **arteries**? The **nerves** and **arteries** are longitudinal structures with the conical (i.e. cone-shaped) geometry of a duct (defined as a channel or tube conveying liquids) rather than as spherical structures like the **carotid bodies** and their **accessory glomi**. It is suggested, therefore, that some additive congruity of forces may play a role in producing the disruptions noted.

Attention must perforce be directed to the configuration and structure of the **carotid body** itself. As indicated in Chapter 4 dealing with the anatomy of the neck, the **carotid body** is a multicentric, globular and spherical structure. In engineering, and, it is suggested, in biological terms a sphere is a stronger structure under conditions of general loading, i.e., bending or compression in any direction. A spherical structural configuration thus carries cogent and pertinent applications in the

fields of engineering, architecture and military design and technology. At the risk of deviating from what is essentially a study in forensic pathology rather than these disciplines, it is nevertheless felt that these latter may provide some answers to the question as to why curved structures such as the **carotid body** and its **accessory glomi** appear to be relatively resistant to the shear and stretch forces of hanging in contrast to the longitudinally configured **arteries** and **veins**.

It has been recognized since Roman times (and in all probability before) that the curved so-called Roman arch, employed in buildings and support of aqueducts, is the strongest and most stable structure, requiring no mortar (i.e. a mixture of lime, sand, and water) between the blocks to keep the individual elements in place. Each block provides structural integrity and support to its immediate neighbour. Further architectural examples are provided by the domes of St. Paul's cathedral in London and St. Peter's in the Vatican in Rome. In engineering, where numerous examples abound beyond the scope of this study, dam walls are constructed with a curved configuration at the point of greatest application of the water pressure. Similarly, in military design and technology, curved configurations are employed not to provide structural support but rather to aid in missile deflection. A classic example of the latter was the World War II Russian T-34 tank with its curved hull configuration, designed for this specific purpose.

A tragic example of failure to follow the above principles occurred in the design of the windows in the British Comet airplane disasters in the early 1950s. The windows of these aircraft were square rather than rounded resulting in force concentration and multiplication at the corners resulting in fracture at these points. It was subsequently realized that a rounded window configuration would tend to diffuse rather than concentrate forces. However, it is conceded that the actual point

and source of microfracture origin lay in the rivets which were employed in the window and door frames.

It has been recognized since the sixteenth century that mechanical factors play a key role in governing biological structure. Galileo Galilei (1564 – 1642) suggested that bones are hollow as this provides maximum strength with minimum weight, predating by centuries Murray's observation of 1926 that the biological cost of operation of physiological and, it seems, anatomical systems is at a minimum. [45] Half a century later, René Descartes (1596 – 1650) enunciated the philosophical idea that all material systems, including the human body, are simply machines governed by the same mechanical laws. These ideas resurfaced in the late nineteenth century with the study of soft tissues and bone by a number of workers. [39,43,44, 51]

In one of the seminal papers in biomechanics, Fung in 1967 suggested that a high degree of nonlinearity exists in the stress-strain relationship of biological tissues. [52] For ease of isolation, blood cells were among the first cells to attract detailed biomechanical analysis. [53] Blood cells may be modelled as spherical, deformable solid shells. In other words, they are vulnerable to compressive forces but, by the nature of their elasticity, would be less susceptible to tensile stretch forces. These characteristics would tend to impart a degree of dynamic stability to the structure. In relation to the **carotid body**, a different structure but with similar biomechanical characteristics and configuration, relative resistance to stretch forces would be encountered. This explains, perhaps, the very low incidence of **tearing, dehiscence** and **haemorrhage** encountered within the **carotid bodies** as opposed to the longitudinal **nerves** and **arteries**.

A further factor to be considered, however, is not only the curved spherical configuration of the **carotid body** with all that this implies in terms of force

deflection but the multicentric structure of the **body** itself and its **accessory glomi**. Each part of the **carotid body** and its **accessory glomi** is structurally (although not physiologically) independent. This feature would not only tend to enhance force diffusion and deflection but would also tend to maintain structural integrity. While, as indicated above, soft tissues are materially non-uniform, the material properties of a tissue result from the integrated manifestations of the homogenized properties of multiple constituents. Could, therefore, the presence of a surrounding spherical adventitial 'shell'resist the conformational changes noted in the **arteries** and **nerves**? A surrounding spherical 'shell' would tend to promote force deflection although concentrating tensile forces at this site despite the spherical 'shell' providing protection to the underlying constituents of the structure. Would this account for the very low incidence of **internal dehiscence** and **tearing** noted in the **carotid bodies** as well as producing the **adventitial separation** noted in the **accessory glomi**?

Action Potentials and their Physiological Implications

An action potential is an event in which the electrical membrane potential of a cell rapidly rises and falls, following a stereotyped trajectory, i.e. a formalized, constant and predictable path. [54, 55] Action potentials occur in several types of excitable cells, including neurons and muscle cells. In neurons, they play a central role in cell-to-cell-communication while in muscle cells an action potential is the first step in the chain of events leading to muscle contraction. It is in these two functions that the relevance of action potentials to the current study lies.

All cells in biological tissues are electrically polarized, i.e. they maintain a voltage difference across the cell's plasma membrane known as the membrane

potential. At the biophysical level, action potentials are generated by special types of voltage-gated ion channels embedded in the cell's plasma membrane. As the membrane potential is increased, Na^+ channels open, allowing entry of Na^+ (ions) into the cell. This is followed by opening of K^+ channels permitting the exit of K^+ (ions) from the cell. [56] Inward flow of Na^+ increases the concentration of positively-charged cations in the cell causing depolarization, where the potential of the cell is higher than the cell's resting potential. If the voltage increases past a critical threshold, typically 15 mV higher than the resting value of -70 mV, the Na^+ current dominates resulting in a runaway condition whereby the positive feedback from the Na^+ current activates even more Na^+ channels. Thus, the cell 'fires', producing an action potential.

Three points of significance should be noted:

1) currents produced by the opening of voltage-gated channels in the course of an action potential are typically significantly larger than the initial stimulating current;
2) the amplitude of an action potential is <u>independent</u> of the amount of current that produced it.
3) Action potentials follow an **all-or-none** principle, i.e. they either occur fully or not at all. Instead, it is the <u>frequency</u> of action potentials which is responsible for the intensity of a stimulus. This is in contrast to receptor potentials, whose amplitudes are dependent on the <u>intensity</u> of a stimulus. It is suggested that both these factors may play a role in the neural stimulation which occurs following application of a compressive ligature to the structures of the neck.

It is further suggested that two major neural effects may ensue following compressive pressure to the neck by a ligature:

1) cardiac inhibition as a consequence of parasympathetic stimulation, and;

2) central nervous system hypoxia and catecholamine surge as a consequence of vascular occlusion and sympathetic stimulation.

1. Cardiac Inhibition as a Consequence of Parasympathetic Stimulation due to the Application of a Tight Constricting Ligature to the Neck

Peripheral autonomic activity is integrated at higher levels in the brainstem, hypothalamus and cerebral cortex. These include various nuclei of the brainstem reticular formation, thalamus and hypothalamus, the limbic lobe and prefrontal neocortex. Ascending and descending pathways further interconnect these regions. It is now recognized that central control of the cardiovascular system is exerted by a longitudinally arranged series of parallel pathways involving specific regions of the neuraxis extending from the cerebral cortex to the spinal cord. [57]

While initiation of the cardiac cycle in vertebrates is myogenic, originating in specialized cardiac muscle cells known as cardiomyocytes, neural influences are of paramount importance in adapting the intrinsic cardiac rhythm to functional stimuli arising elsewhere in the body. In response to these stimuli, the autonomic nervous system exerts its effects on the cardiac nodal tissue and their prolongations, on coronary vessels and on the working atrial and ventricular myocardium. [58-60]

The autonomic innervation to the heart has both efferent (sympathetic and parasympathetic) and afferent components. Parasympathetic fibres reach the heart through vagal branches and the sympathetic fibres reach the heart from the branches

of the thoracic ($T_1 – T_5$) sympathetic trunk. Vagal preganglionic fibres proceed from origins within the brainstem, particularly within the medulla which includes the nucleus ambiguus, the reticular nuclei and the dorsal vagal nucleus. Preganglionic axons then leave in the cardiac branches of both the right and left vagus nerves to reach the cardiac plexus. Sympathetic preganglionic neurons, on the other hand, lie in the upper five or six segments of the intermediolateral column of the thoracic spinal cord from which postganglionic fibres proceed bilaterally to the heart from the cervical sympathetic ganglia.[61] It is suggested that because of their location in the cervical **ganglia**, these sympathetic fibres are in a position ideally situated to render them strategically vulnerable and subject to the forces exerted by a compressive ligature and the neural stimuli ensuing therefrom.

While sympathetic stimulation increases cardiac contractility, the force of cardiac contraction and the rate of contraction, parasympathetic stimulation carries opposing effects. These effects are mediated via a complex system of sarcolemmal receptors situated within the cardiomyocyte. Adrenergic or cholinergic stimulation initiates the activity of sarcolemmal and cytosolic messengers whereby an extracellular neural stimulus is converted to an intracellular physiological change, a process known as signal transduction. [57,62,63]

In the case of the parasympathetic system, the activity of the vagus nerve endings is specifically associated with a receptor known as the muscarinic (M_2) receptor with stimulation of this receptor resulting in decreased myocardial contractility. The interesting point, however, is that this latter effect of a decreased force and rate of cardiac contraction is best observed in the presence of beta-receptors. (These receptors respond to the sympathetic catecholamines, adrenaline and noradrenaline stimulation, when vagal effects counteract those of prior beta-

sympathetic stimulation.[61,64]). In other words, the inhibitory effects of vagal stimulation on myocardial contractility appear to override those of sympathetic stimulation, suggesting that 'braking' of beta-adrenergic stimulation is physiologically desirable.[61,62,65-68] This may explain why vagal effects predominate when both the sympathetic and parasympathetic nervous systems are activated following neural stimulation by a compressive ligature to the neck.

2. Central Nervous System Hypoxia and Catecholamine Surge as a Consequence of Vascular Occlusion and Sympathetic Stimulation

While it may be self-evident that vascular occlusion of the **carotid arteries** in the neck would produce ischaemia (decrease in blood flow) and hypoxia (decrease in oxygen level) of cortical tissue resulting in unconsciousness and death, it is less clear why sympathetic stimulation of **nerves** and **neural ganglia** in the neck should do so. However, experimental models of the effect of brain death have shown that brain death as a consequence of either injury or vascular compression can, in and of itself, lead to a catecholamine surge.[69-80] In other words, while neural stimulation of sympathetic **nerves** and **ganglia** in the neck would logically occur following application of a hanging ligature, the catecholamine surge which follows is secondary to the hypoxia which ensues on vascular occlusion of the **carotid arteries**. This is the reverse of the situation which pertains with activation of the parasympathetic system where the effects are primary. Furthermore, as indicated above, parasympathetic effects appear to override sympathetic effects as a protective mechanism in the cardiovascular system. Further evidence supporting the primacy of the parasympathetic system is that a mechanism for parasympathetic-sympathetic

interaction lies at the level of the sympathetic terminal neurons, where a presynaptic muscarinic M_2-receptor inhibits the release of norepinephrine (noradrenaline). [81] Where, however, brain damage is primary as, for example, following a head injury rather than secondary due to vascular occlusion, sympathetic overactivity then predominates, with ensuing cardiovascular sequelae. [82-88] Of clinical interest is that this surge in catecholamines following head injury has been exploited in predicting patient outcome in traumatic brain injury. [89] Hamill and co-workers, for example, reported that in 33 patients with traumatic brain injury, the levels of circulating catecholamines appeared to be excellent endogenous and readily quantifiable markers which reflected the extent of brain injury and predicted the likelihood of patient recovery. [90]

C. The Neuromuscular Findings and their Pathophysiological Implications

These refer to the findings within the **phrenic nerve** and its overlying and underlying muscles, the **sternocleidomastoid muscle** and the **scalenus anterior muscle**, respectively. The **neural findings** within the **phrenic nerve** mirrored the findings within the other **neural elements** with the sole exception of **intraneural haemorrhage** which was not encountered. This was attributed to a combination of the greater diameter and thickness of the **phrenic nerve** in comparison with the sympathetic and parasympathetic **nerves** as well as its anatomical position lying as it does between the **sternocleidomastoid muscle** and the **scalenus anterior muscle**. These latter muscles, it is suggested, then provide a buffering and cushioning effect

to the compressive forces exerted by the encircling ligature but not, however, to the tensile forces inherent to the hanging process.

While evidence of force application to both the **phrenic nerve** and its overlying and underlying muscles was, therefore, present, the question then arises as to what would be the pathophysiological consequences of force application to these neural and muscular elements. The **phrenic nerve** itself comprises the neural innervation to the diaphragm, the major muscle of respiration. In this regard, it should be borne in mind that respiration is dependent on its inspiratory component, expiration being an entirely passive process. Hence, any neural stimulus which is sustained and unremitting, as occurs with tight application of an encircling and compressive ligature, must, of necessity, result in the diaphragm becoming fixed in a state of inspiratory contractual paralysis. This effect would be compounded by the compressive pressure applied to the **sternocleidomastoid muscle** and the **scaleni muscles** which constitute parts of the accessory muscles of respiration. Force application to these accessory muscles with stimulation of the muscle receptors may similarly result in the thoracic cage around the lungs becoming fixed in a state of inspiratory paralysis. Paralysis of the respiratory muscles in turn reduces the **vital capacity**, which is the maximum amount of air that a person can expel from the lungs after first filling the lungs to their maximum extent and then expiring to the maximum extent. This amount is about 4600 ml, as described in standard texts of physiology. Paralysis of the respiratory muscles causes a great decrease in **vital capacity**, to as low as 500 to 1000 millilitres, an amount barely enough to maintain life. It is suggested that this reduction in **vital capacity**, when compounded with the cerebral hypoxia attendant on carotid artery constriction and occlusion, may be a further factor producing unconsciousness and death in the hanging process. Multiple

physiological mechanisms may thus be involved during hanging. To gain an understanding of these mechanisms, recourse must be had to the issues of lung compliance and the **dynamic** and **static stretch reflexes** initiated by the **muscle spindles**.

Pulmonary Compliance

As described in standard texts of physiology, the expansibility of the lungs and thorax is called compliance which is expressed as the volume increase in the lungs for each unit increase in alveolar pressure. The compliance of the normal lungs and thorax combined is 0.13 litre per centimeter of water pressure. In other words, every time the alveolar pressure is increased by 1 cm of water, the lungs expand 130 milliliters. However, the lungs alone, when removed from the chest, are almost twice as distensible as the lungs and thorax together. This is because the thoracic cage itself must also be stretched when the lungs are expanded *in situ*. Thus, the compliance of the normal lungs when removed from the thorax is about 0.22 litre per cm of water. This illustrates that the muscles of inspiration must expend energy not only to expand the lungs but also to expand the thoracic cage around the lungs. The important point with regard to this study is that any condition that decreases the expansibility of the thoracic cage such as chest wall deformities or muscle paralysis reduces the expansibility of the chest thereby reducing the total pulmonary compliance. This then introduces an element of hypoxia, contributing to and compounding the hypoxia consequent on the vascular constriction, occlusion and disruption of the **carotid arteries** incident to the hanging process.

In view of their relevance to one of the putative mechanisms of death in suicidal hanging, a brief review of the anatomy and physiology of the **muscle spindles** and the **stretch reflex** follows.

The Muscle Spindles

Introduction

Muscle spindles are sensory receptors found within the belly of a muscle, which primarily detect changes in the length of this muscle. They convey length information to the central nervous system via sensory neurons. The responses of muscle spindles to changes in length play an important role in regulating the contraction of muscles by activating motor neurons via the so-called **stretch reflex**.

Anatomy

Muscle spindles are found embedded in extrafusal muscle fibres ('fusus' is the Latin word for spindle). The spindles are composed of 3 – 12 intrafusal muscle fibres, of which there are three types:

· dynamic nuclear bag fibres (bag_1 fibres).

· static nuclear bag fibres (bag_2 fibres).

· nuclear chain fibres and the axons of sensory neurons.

The axons of gamma motor neurons also terminate in **muscle spindles**, synapsing at either/both ends of the intrafusal muscle fibres, regulating the sensitivity of the sensory afferents located in the central tendon of the **diaphragm**.[91] Fusimotor

neurons are classified as either static or dynamic according to the type of intrafusal motor fibres they innervate as well as their physiological effects on the responses of the sensory neurons innervating the **diaphragm**. In view of the role of the **diaphragm** as the major muscle of respiration, any interference in this regulatory pathway would therefore be be of major import in the pathophysiology of hanging.

While the static axons innervate the chain or so-called bag_2 fibres, the dynamic axons innervate the so-called bag_1 intrafusal muscle fibres. These latter increase stretch sensitivity by contracting the intrafusal fibres. The physiological function of the gamma motoneurons is therefore to modify the sensitivity of the **muscle spindle** sensory afferents to stretch, i.e., sensitivity modification. Thus, increased sensitivity would tend to enhance the effect of the stretch forces occurring during the hanging process upon the muscles.

The Stretch Reflex

The function of the **muscle spindle**, a complex organ, is manifested in the form of the muscle **stretch reflex**, that is, whenever a muscle is stretched, excitation of the spindles causes reflex contraction of the muscle. [92,93] This function carries considerable implications in the context of this study with regard to the effects of the **sternocleidomastoid muscle** and the **scalenus anterior muscle** on respiration folllowing application of a compressive ligature to the neck.

Hanging would result in the "stretching" of the **sternocleidomastoid** and **scalenus anterior muscles**. In the former muscle, the effect would be stimulation of the **phrenic nerves**, with fixation of the diaphagm in a state of inspiratory paralysis. In

the case of the **scalenus muscles**, stimulation results in the **intercostal muscles** similary becoming fixed in a state of inspiratory paralysis.

With regard to the effects on respiration, one neural centre and one physiological reflex limit inspiration. The **pneumotaxic centre**, located dorsally in the **nucleus parabrachialis** of the upper pons, transmits impulses continuously to the inspiratory area. The primary effect of these impulses is to control the 'switch-off' point of the inspiratory ramp, thus controlling the duration of the filling phase of the lung cycle. Hence, the function of the **pneumotaxic centre** is primarily to limit inspiration. The **Hering-Breuer reflex**, on the other hand, is primarily a protective mechanism for preventing overinflation of the lung. Located in the walls of the bronchi and bronchioles throughout the lungs are **stretch receptors** that transmit signals through the **vagus nerves** into the dorsal respiratory group of neurons when the lungs become overstretched. These signals affect inspiration in much the same way as signals from the **pneumotaxic centre** in that they limit the duration of inspiration. When the lungs become overly inflated, the **stretch receptors** activate the appropriate feedback response that 'switches-off' the inspiratory ramp limiting further inspiration. However, the crucial point to bear in mind is that these reflexes and mechanisms apply under physiological conditions. With application of a compressive ligature to the neck, normal physiology is disturbed and disrupted, pathological forces then pertaining and predominating, overcoming normal physiological responses.

The **stretch reflex** can be divided into two separate components, namely, the **dynamic stretch reflex** and the **static stretch reflex**. The **dynamic stretch reflex** is elicited by the potent dynamic signal transmitted from the primary endings of the **muscle spindles**. In other words, when a muscle is suddenly stretched, as occurs

with sudden initiation of the tensile forces in the hanging process, a strong signal is transmitted to the spinal cord. This causes an instantaneous, very strong reflex contraction of the same muscle from which the signal originated.

In addition to this function, the muscle spindle reflex acts as a so-called servo-assist mechanism. What is meant by this mechanism? When both the alpha and gamma motor neurons are stimulated simultaneously, as occurs with ligature application to the neck, the degree of stimulation of the muscle spindles will remain unchanged, neither increasing nor decreasing. However, when the muscle is suddenly contracted against a load, i.e. the imposed weight of the body in the hanging process, a mismatch occurs between the extrafusal muscle fibres and the intrafusal muscle fibres. This mismatch then stretches the receptor portions of the **muscle spindles** thereby eliciting a **stretch reflex** generating extra excitation of the extrafusal fibres. This then creates an accessory neuronal signal that increases the degree of stimulation of the extrafusal fibres. This biological principle has been applied in the design of engineering systems. For example, this principle, known as **bionics**, has been employed in the engineering design of the power steering in an automobile. Here, if the front wheels are resistant to following the movement of the steering wheel, a servo-assist device becomes activated which applies additional force to turn the wheels.

While the **dynamic stretch reflex** is over within a fraction of a second after the muscle has been stretched and stimulated to its new length, a weaker but more prolonged **static stretch reflex** continues thereafter. This **reflex** is elicited by the continuous static receptor signals transmitted by both the primary and secondary nerve endings. The significance of the **static stretch reflex** in the hanging process is that it continues to cause muscle contraction as long as the muscle is maintained at

excessive length, precisely the situation in hanging. The physiological consequences of this would then be the fixing of the thoracic cage and lungs in a state of inspiratory paralysis, due to both stretching and stimulation of the **phrenic nerve** as well as the accessory muscles of respiration. In essence, both the neck muscles, i.e. the **sternocleidomastoid muscle** and the **scalenus anterior muscle** and the diaphragm are affected; the neck muscles because of their location underneath the ligature and the diaphragm in view of its cervical innervation via the **phrenic nerve**.

D. The Biomechanical Principles governing Force Application to Biological Tissues

Definition:

Biomechanics is defined as the development and application of mechanics for the better understanding of physiology and pathophysiology. [46] By contrast, **mechanobiology** seeks, by observation, to extract the underlying general mechanisms while **bionics**, as indicated above, refers to the application of biological principles to the design of engineering systems.

In 2002, Van der Meulen and Huiskes formulated these concepts neatly and concisely by suggesting that 'form follows function [which] follows form'. [94] They suggested, therefore, that **biomechanics** focuses on whether or how function follows form, while **mechanobiology** focuses on whether or how function determines form. However, fundamental to both **biomechanics** and **mechanobiology** is histology, defined as the study of the fine structure of tissues. It is axiomatic in mechanics that the response of a material to applied loads depends upon its internal constitution. The

response of tissue constituents to abnormal loads and forces, as occurs during the hanging process, thus produces tissue alterations and disruptions which may then manifest as the lesions encountered in the present study. Figure **8.5b.** is a diagrammatic illustration of some of the vector forces occuring in the hanging process.

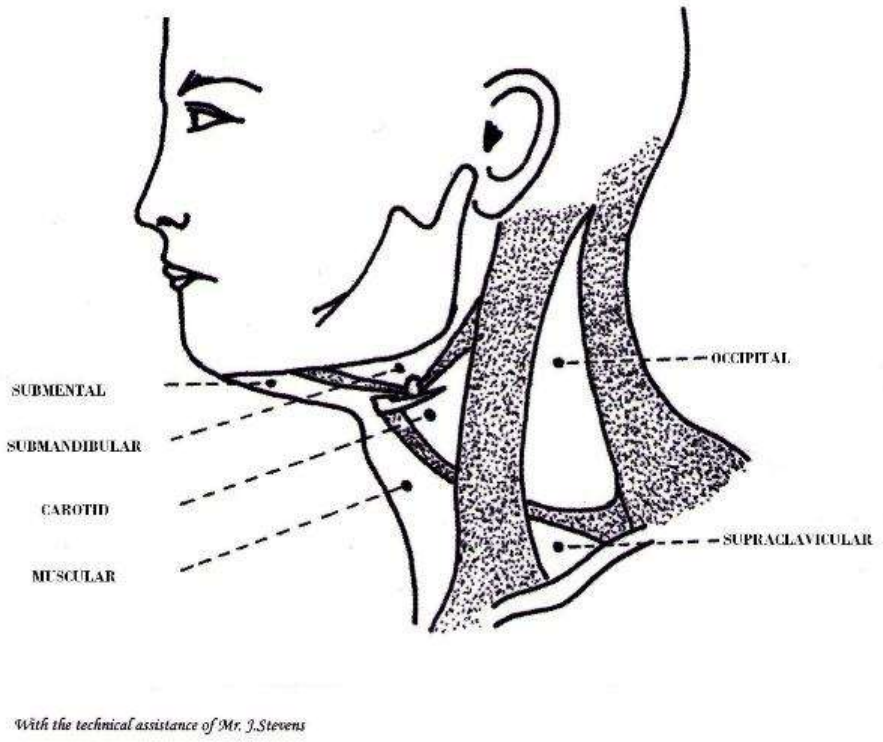
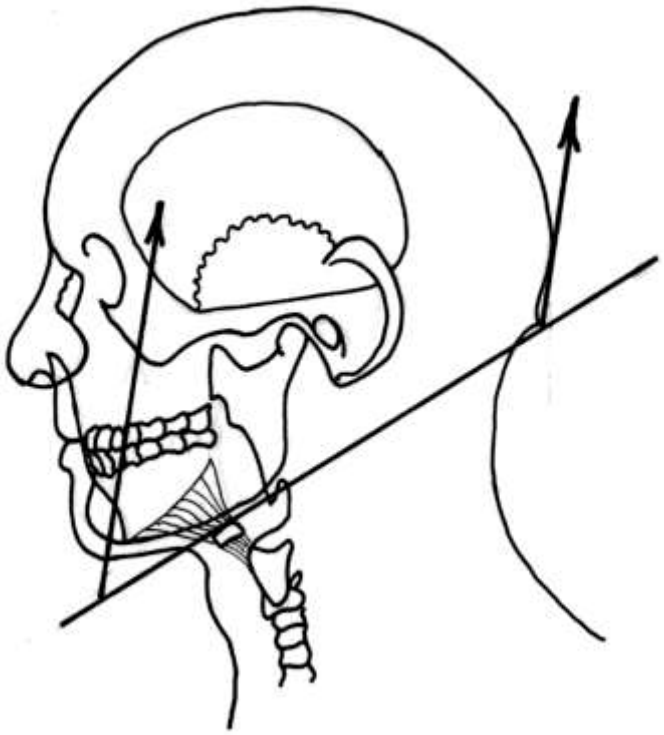


Fig. 8.5a A sketch showing the anatomical triangles of the neck.



With the technical assistance of Mr. J.Stevens

Fig. 8.5b. A diagram showing the direction of some of the force vectors acting during the hanging process.

The General Characteristic Behaviours of Biological Soft Tissues

"**As the force, so the extension**", a famous law enunciated by Robert Hooke (1635 – 1703), curator of experiments for The Royal Society, and an interesting observation in the light of the tensile forces occurring in the hanging process.

Biological soft tissues follow the basic postulates of mechanics such as stress (a mechanical force per unit area exerted between contiguous bodies), strain (a stretching or tensile movement/extension or change of structure resulting from such force), and elasticity (a spontaneous resumption of the normal shape of a body after contraction, distortion or dilatation).

It has long been known that biological soft tissue behave very differently from engineering materials such as wood, metals and concrete. While soft tissues may be best classified as mixture-composites that exhibit inelastic behaviours under particular conditions, their biological behaviour may best be modeled within six different theoretical frameworks in terms of elasticity rather than full mixture theory. Briefly, these theories are as folllows:

a. Finite Elasticity

It was a series of papers by Fung that highlighted the difficulty of applying the principle of infinitesmal elasticity to soft tissues which normally exhibit finite deformations. [52, 95-97]

Fung, in his seminal work, reported on data derived from tests conducted on the mesentery of the peritoneal cavity which showed a strongly nonlinear relationship between stress (a mechanical force perunit area exerted between contiguous bodies) and strain (a stretching or tensile movement/extension or change of structure resulting from such force), with extreme tissue compliance at the lower stretches. However, Fung further developed and refined a theory which suggested an exponential (defined as a function which increases with an increasing rate) stress-strain relationship. In other words, as stress, i.e., mechanical force increases, so does stretch. While this is of obvious relevance to the situation during the hanging process, it should be pointed out that stretch does not need to be exponential but could, in fact, be linear (personal communication; Thomas Dessein, School of Mechanical, Industrial and Aeronautical Engineering, University of the Witwatersrand, Johannesburg).

Of further relevance to the hanging process is that most biological soft tissues, in particular arteries, exhibit an anisotropic response (defined as having physical properties such as elasticity which depend on the direction in which a force is applied). In other words, arteries exhibit a viscoelastic character and which is time-dependent. The latter is discussed in detail below under the section dealing with viscoelasticity (section c) (*vide infra*).

b. Membrane Theory

Membranes are defined differently in biology and mechanics. In biology, a membrane is defined as a thin layer of tissue that covers a surface, lines a cavity or divides a space whereas in mechanics, the word membrane implies a thin structure which offers negligible resistance to bending. Most biological membranes can be modelled in terms of membrane theory, as biological membranes similarly offer negligible resistance to bending. These include, for example, cell membranes, the pericardium, the epicardium, the pleura, the intestinal mesentery, the epimysium which envelops muscle fibres, tendon sheaths, the meninges, the urinary bladder, the lens capsule and even the skin. Under pathological conditions, it includes vascular aneurysms.

Of particular interest to the present study, however, is that the theory of elastic deformation of membranes largely revolves around the general theory of plates and shells, i.e. flat, thin sheets and spherical outer casings. This has resulted in specialized approaches producing a specialized literature, appropriate for describing the behaviour of spherical membrane-bound structures such as red blood cells. [98] It has been suggested in the discussion of the **carotid body** and its **accessory glomi**

(*vide supra*) that the same response to force application may pertain to these latter structures, similarly spherical, curved bodies independent of one another and similarly surrounded by a protective 'membrane', an adventitia. As previously indicated, spheres constitute stronger structures under conditions of general loading such as bending or compression.

However, while membrane theory offers considerable simplification in comparison with theories of finite elasticity in attempting to explain the **biomechanical** behaviour of the **carotid body** and its **accessory glomi**, geometrical and material nonlinearities still pose major challenges in analysing dynamically loaded membranes.[99]

c. Viscoelasticity

In view of the fact that the human body consists of a high volume fraction of water, it is not surprising that biological soft tissues exhibit both solid-like and fluid-like behaviours. In other words, biological tissues exhibit characteristic behaviours of **viscoelasticity**. They creep, i.e. change in shape under conditions of stress and they exhibit hysteresis (defined as a lagging in effect when the cause varies in amount) under cyclic (defined as recurring in cycles) loading. However, some of these conditions do not appear to pertain when applied to the hanging process. Firstly, the high volume fraction of water in most biological soft tissues renders them nearly incompressible under physiologic conditions of loading which is clearly not the case during hanging when the weight of either part of or the whole of the body is exerted on the structures of the neck. Secondly, during hanging a force is suddenly applied upon the neck, particularly when a jerk has occurred. (This is the opposite of what

occurs under conditions of cyclic loading where the forces applied recur in cycles). Thirdly, rather than hysteresis, i.e. a lagging or retardation in effect which is supposed to occur in high water volume tissues, the actual physiological response of the tissues to neuromuscular stimulation is precisely the opposite, namely, an instantaneous initiation of **action potentials** and the **dynamic and static stretch reflexes** described above. Thus, because of the inherent nonlinear behaviour exhibited by most soft tissues, standard models of linear viscoelasticity are not, in general, applicable.

d. Mixture Theory

Biological soft tissues may be classified as mixture-composites as they consist of multiple solid constituents plus ample bound and unbound water. It is natural, therefore, to employ the concept of mixtures to describe certain behaviours of soft tissues. This applies particularly to significant changes of mass, energy between constituents or momentum. This latter is defined as the impetus gained by movement or the force with which a body moves (and which certainly occurs in hanging).

However, because of the complexity of biological tissues as well as the inherent geometrical complexities associated with most surrounding tissues (i.e. 'real boundary problems' in terms of physics), challenges are encountered in the direct application of the mixture theory to the forces applied during the hanging process. What this implies to hanging is that multiple forces occur simultaneously, or within seconds of one another, contributing to the difficulties arising in interpretation and analysis.

e. Growth and remodelling

It was long thought that the most important general characteristics of biological soft tissues are the often nonlinear and anisotropic (the latter defined as having physical properties such as elasticity [i.e., having no decrease in kinetic energy] which depend on direction), behaviours that they exhibit over a wide range of physiological and pathophysiological conditions. [100,101] However, an equally important characteristic is the ability of tissues to remodel, i.e., alter their configuration in response to stimuli and changes in their mechanical environment. A formidable body of literature has sprung up around this topic, predominantly involving haemodynamic stress-induced cardiac adaptive and maladaptive responses.[102-108] While this appears to be of no immediate relevance to the hanging process, where unconsciousness supervenes within seconds and death within minutes, a noteworthy point raised by several workers is that biological soft tissues are materially non uniform, regardless of their grossly apparent homogeneity.[109-127]

A pertinent example of this occurs in the context of motor vehicle accidents, where more solid organs such as the liver, having a high specific gravity, tend to shatter more readily than the lung which has a lower specific gravity due to the contained air. As noted in the Results chapter, statistical analysis of the weight of the body in relation to hanging indicated that weight plays no correlative role in the severity of arterial damage. Indeed, case no. 20 (Fig. 7.22), was one of only 3 cases in the study showing **complete circumferential transverse rupture**, despite having the lowest body weight of all the adults in the series, namely, 38 kg. Admittedly, however, and as previously indicated, this individual may have had the acquired immune deficiency syndrome. The severe weight loss accompanying the end stages

of this disease, with its decrease in body mass and subcutaneous tissues, would have rendered the arteries of the neck more vulnerable and exposed to the compressive forces exerted by a ligature and the systemic head of pressure acting from within the vessel. As an adjunct, could the reason for suicide be the knowledge of a terminal HIV+ state on the part of the person?

Non-uniformity of tissues may thus serve to explain in part why, for example, the different layers of the arterial wall exhibit different types and incidences of arterial disruption in response to the constricting force of a ligature. It may further serve to explain why a discrete, multicentric spherical structure such as the **carotid body** showed itself far more resistant to the forces occurring during the hanging process as compared to the **carotid arteries**, which are longitudinal fluid-filled structures. In biomechanical terms, arteries exhibit a cylindrical orthotropy (defined as having a straight or longitudinal directive response to an external stimulus), precisely because of their configuration.

In summary, biological soft tissues exhibit complex nonlinear, heterogeneous, anisotropic behaviours that differ from point to point, from time to time and from individual to individual. Despite the fact that tissue damage was, as noted in the present study, ubiquitous, the type and degree of damage noted, in particular with regard to the different layers of the arterial wall, was different, highlighting the nonlinear behaviour and response to injury of these biological tissues.

E. The Principles of Physics applicable to Suicidal Hanging

While biological soft tissues exhibit complex nonlinear behaviours which, as indicated, differ from point to point, from time to time and from individual to individual, they are, nevertheless, bound by the laws and principles of physics. It is, therefore, necessary to have recourse to some of these principles in an attempt to understand the mechanics of the hanging process, as follows:

Moment of Inertia

This refers to a concept of **rotational dynamics** analogous to the concept of **mass** in **translational dynamics**. The **moment of inertia** for a particular object is defined for a given point or axis of rotation as the sum of the mass of a particle multiplied by the square of the distance from the point or axis, or:
I (inertia) = $\mathbf{\Sigma mr^2}$, where **I** is the moment of inertia, **m** is the mass of a particle and **r** is its distance from the axis of rotation.

In Newton's second law of motion for a rotating object, one is concerned not with the net force on the object but rather with the net torque or moment of the force. Thus, the net torque (**L**) is proportional to the moment of inertia and the angular acceleration (**a**) i.e., net **L** = **Ia**. This is analogous to **F** (force) = **ma**. Similarly, the close relation of moment of inertia in rotation to mass can also be seen in calculating the kinetic energy of a rotating object. For each particle of the object, $\mathbf{KE = \frac{1}{2}\, mv^2}$, where **v** is the linear velocity of the particle. Linear velocity is related to angular velocity **w** by **v** = **rw** where **r** is the distance of the particle from the axis of rotation.

Momentum = measure of quantity of motion possessed by an object. It is equal to the product of the mass of the object (**m**) and its velocity **v** or **p** = **mv**. Because velocity is a vector quantity, momentum is also a vector, having both magnitude and direction. Angular momentum is defined as the product of a rotating object's angular velocity **w** and its moment of inertia **I**. When Newton's third law of motion is applied to an isolated system, it becomes the law of conservation of momentum, which states that the total momentum of a system is constant.

Linear and angular momentum are conserved separately. Newton's actual statement regarding law of motion was that force **F** equals the rate of change of momentum with respect to time or:

$$\mathbf{F} = \frac{\Delta \mathbf{p}}{\Delta \mathbf{t}} = \frac{\Delta(\mathbf{mv})}{\Delta \mathbf{t}}$$

Since mass is assumed to be constant in classical physics, this formula reduces to **F** = **ma**, where "a" is acceleration. In order to change the momentum of an object, a force must be applied to it. By rearranging the equation above, we have **Δp** = **FΔt**, so that the change in momentum depends not only on the force (**F**) but also on the time (**t)** during which it is applied. The quantity **FΔt** is called the impulse of the force **F**.

Let us draw an analogy from the mining industry (Figure **8.6a**). Assume that the hoist driver stops the skip or cage halfway between the ends (upper end and lower end) of the shaft. At that point, because the skip is poised within the shaft, it has potential, theoretical, and imaginary momental energy. The weight of the skip and its distance from the top and bottom of the shaft contribute to that energy despite the fact that it is not moving.

Let us say that at that point the rope breaks and the skip or cage plunges downward. Its weight (gravity) takes part in the plunge and as it goes downwards, it accelerates. The momental energy of the skip is:

E = **m x a** (mass x acceleration).

The faster it goes, the greater the accumulated energy, i.e., momentum.

Fig. 8.6a

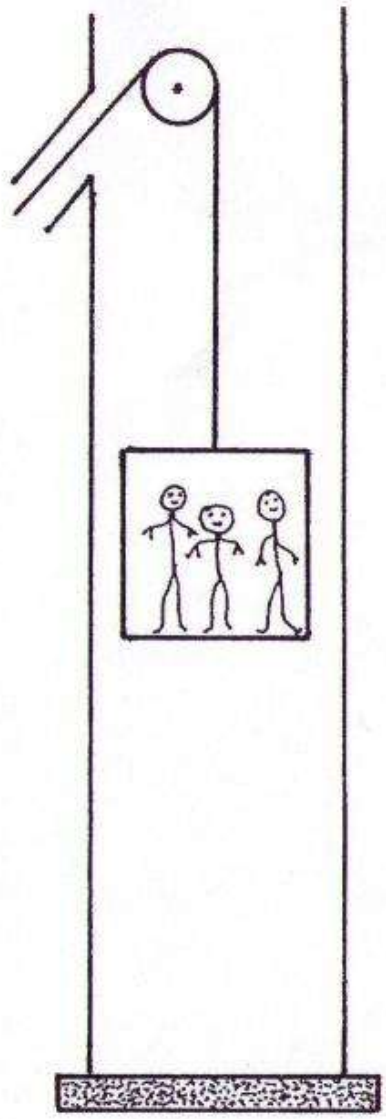
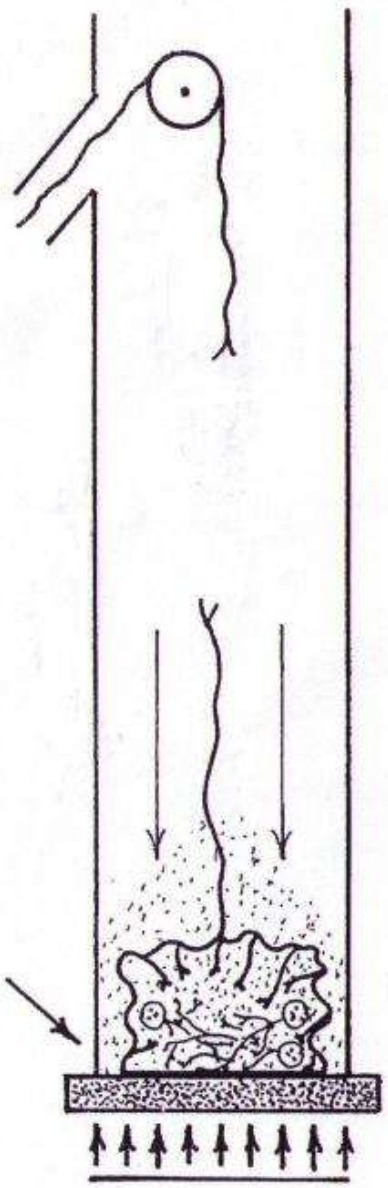


Fig. 8.6b



With the technical assistance of Mr. J.Stevens

Fig. 8.6a. A diagrammatic representation of a mining cage at a point midway between the upper and lower ends of the shaft. At this point the skip (cage) has potential momental energy.

Fig. 8.6b. The rope holding the skip has broken and the skip has plunged downwards impacting the base of the shaft and coming to an abrupt halt. The momental energy is released and the miners are killed (although cage plunge is retarded to a certain extent by various hydraulic systems which slow the cage's fall).

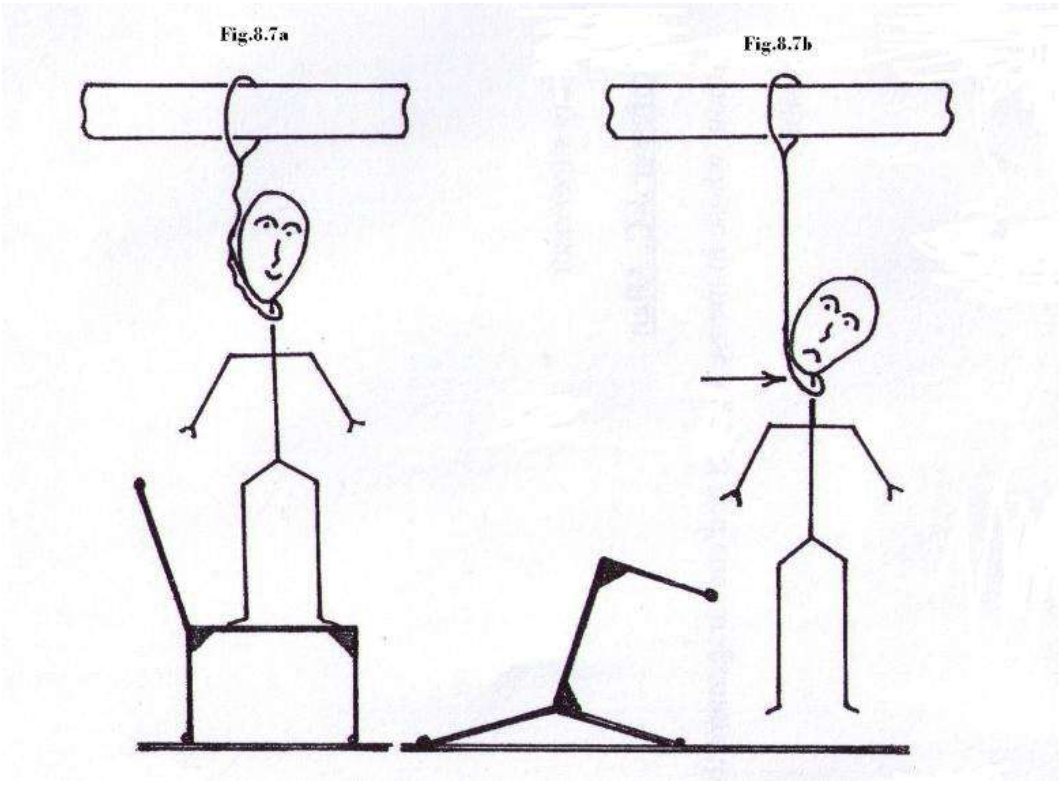
It should further be borne in mind that an electrical wire has much less "give" (elasticity) than, for example, a rope and therefore the impulse on the tissue is much greater because the body is brought to rest in a much shorter time.

Point of Impact

When the moving object (skip or cage) strikes and impacts the concrete base of the shaft, it comes to an abrupt halt. What then happens to the accumulated momental (moving) energy? This energy is released which then shatters and fragments the skip or cage. If the cage contains miners they are killed (Figure **8.6b**).

Let us therefore now look at Figure **8.7a**. The intended suicide is standing on a chair with a rope around the neck and fixed to a girder, beam or tree branch above. The rope in this position is slightly slack.

In Figure **8.7b**, the victim kicks the chair away and is left hanging by the neck. The point of impact comprises the upper third of the neck, the ligature in this case being constrained and restrained by the lower border of the mandible. The situation of the falling body is thus similar to that of the falling skip in that the damage occurs at the " point of impact".



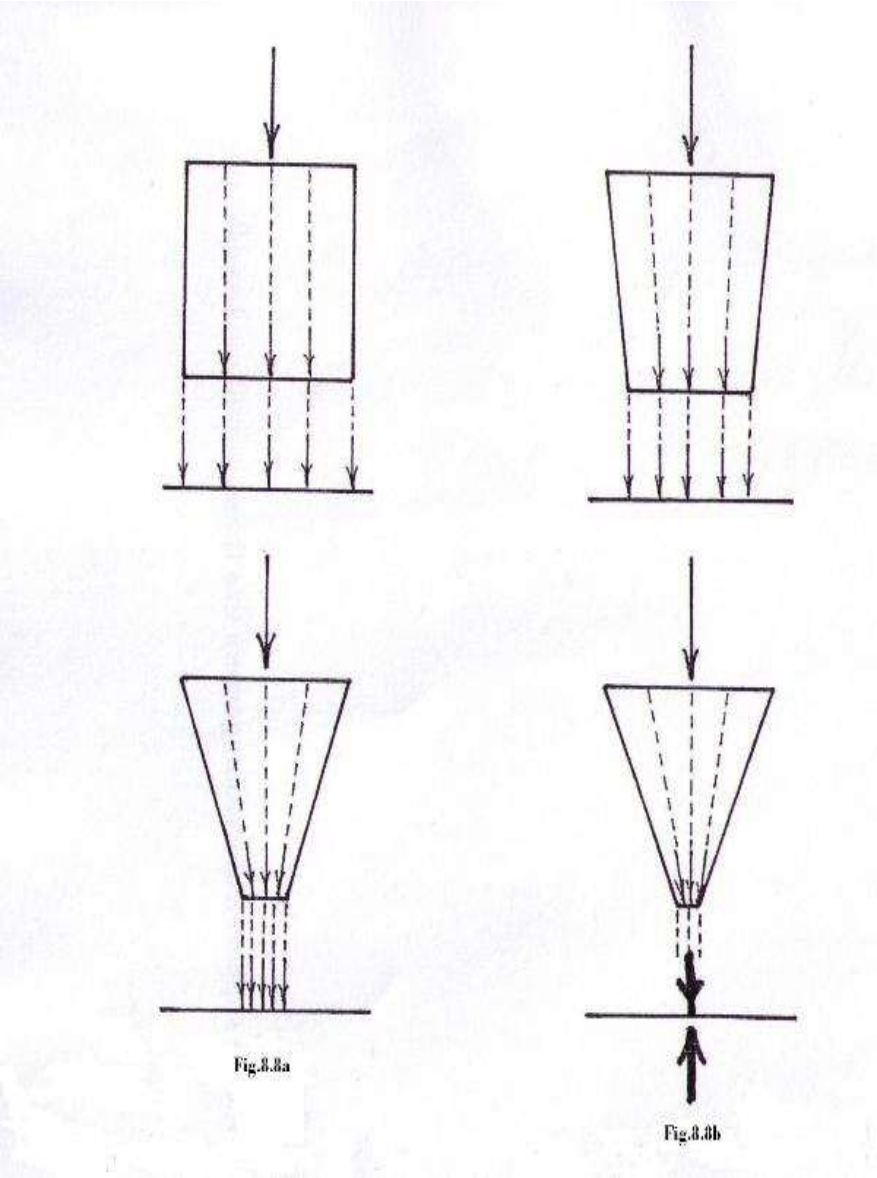
With the technical assistance of Mr. J.Stevens

Fig. 8.7a. A diagrammatic representation of the intended suicide standing on a chair. The rope is around the neck fixed to a beam above and slightly slack.

Fig. 8.7b. The chair has been kicked away and the victim is left hanging by the neck. The point of impact comprises the upper-third of the neck with the

ligature being restrained by the lower border of the mandible. The damage occurs at the point of impact.

However, a further analogy can be drawn. It is recognized that concentration and amplification of pressure at a single focal point occurs with a woman's stiletto heel, whereby a pressure of several tons per unit area ($1m^2$), sufficient to indent metal, is exerted at the focal point of pressure application i.e., at the contact point beneath the heel. A similar magnification and amplification of force may arise in the context of suicidal hanging, albeit in this case the pressure is applied transversely (rather than vertically as in the mine cage analogy) and distributed radially along the point of contact of the ligature with the underlying tissues of the neck (personal communication; Thomas Dessein, School of Mechanical, Industrial and Aeronautical Engineering, University of the Witwatersrand, Johannesburg).



With the technical assistance of Mr. J.Stevens

Fig. 8.8a. A diagrammatic representation of a falling mine cage with concentration of pressure at the point of impact.

Fig. 8.8b. A diagrammatic representation of the stiletto heel of a woman's shoe. Concentration, multiplication and amplification of pressure occurs at the focal point of contact with the underlying surface.

In the case of suicidal hanging, as opposed to judicial hanging, the distance dropped is relatively small, allowing little possibility for acceleration. However, with regard to application of force, the tissues of the neck may be classified as being either "hard" or "soft", the "hard" tissues being the **vertebral column** and the **hyoid-larynx complex**. The "soft" tissues, on the other hand, consist mainly of water which, for practical purposes, is non-compressible.

It therefore seems likely that the kinetic energy exerted by the ligature around the neck will be transmitted through skin and underlying subcutaneous tissue to the deeper soft tissues, i.e., the vessels, resulting in compression of these structures. (A similar situation occurs with regard to the mechanism of production of contusions, where blunt force is transmitted through the overlying epidermis to the blood vessels within the papillary dermis resulting in rupture of these vessels and extravasation of red blood cells into the surrounding interstitial tissues, producing the classical 'bruise').

While the blood vessels, being soft, are easily compressible, they contain fluid, i.e., blood, which is non-compressible. However, the contained blood is displaceable, resulting in "wall-to-wall" closure of the vessels. "Wall-to-wall" closure thus obliterates the lumen of the vessel while displacing the contained column of blood. The foregoing series of events therefore results in impedence of forward flow of blood within the **carotid arteries** producing cerebral ischaemia and anoxia for as long as the pressure from the surrounding ligature is present. In actual experiments (see 'The Early Research Findings', Chapter 4, pp. 90 to 109), the time required for the onset of unconsciousness is as little as 11 seconds. With the onset of unconsciousness, the victim is unable to save him-or-herself with death being the inevitable consequence.

After the victim is no longer standing upon an object such as a chair the body below the position of the ligature is now suspended by the ligature. This, in itself, would cause pain (described as excruciating by the test victim, as noted in Chapter 4) and attributed to the undue stretching to which the victim has been subjected. Pain would further be caused by the head (weighing five or six kilograms) being forced to the side as well as the neck being twisted, i.e., rotated in its vertical axis with torque being applied to the ligamentous, muscular, neural and vascular structures of the neck analogous in a sense to a "whiplash" effect.

Rapid onset of unconsciousness could, perhaps, be regarded as merciful as it would render the victim oblivious to the immediate effects of the suspension.

Mathematical Analysis to Estimate Minimum Peak Pressure

The following analysis estimates the minimum peak pressure that would be required to carry the weight of a person during hanging. The following assumptions are used to ensure a solution can be obtained:

- The analysis is done for the static case (no accelerations)
- The loop is perfectly round with a fixed diameter such that the rope does not apply any radial pressure above that required to support the weight of the person (thus the minimum pressure that would be required to support the weight of the person will be calculated).
- The loop is vertically orientated.

- The pressure increases linearly with angle (measured from the knot) from zero at the knot to the peak pressure opposite the knot. See Figure **8.9** below.

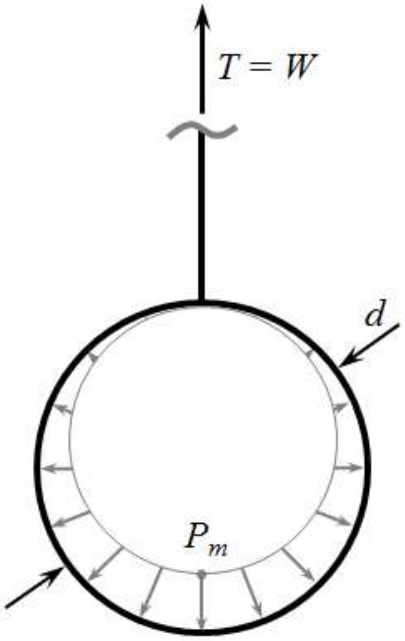


Fig.8.9. Pressure Distribution

The following standard units are used:

N = Newtons

Pa = Pascals

m = Metres

kg = Kilograms

s = Seconds

The table below defines the variables used:

Table: Variable definitions

T	=	Tension in the ligature above the loop	[N]
W	=	Weight of the person. (W = mg)	[N]
m	=	Mass of the person	[Kg]
g	=	Acceleration due to gravity	[m/s^2]
w	=	Width of ligature	[m]
d	=	Loop diameter	[m]
θ	=	Angle from rope as defined in figure below	[rad]
F_p	=	Perpendicular force due to pressure	[N]
p	=	θ dependant pressure/pressure distribution.	[Pa]
P_m	=	Peak pressure (at $\theta = 180^o$).	[Pa]
F_y	=	Total vertical force due to distributed pressure.	[N]

Figure **8.10** below shows the pressure distribution acting on an infinitesimal arc of the loop. This is used to develop the integral for the vertical force exerted by the pressure on the loop.

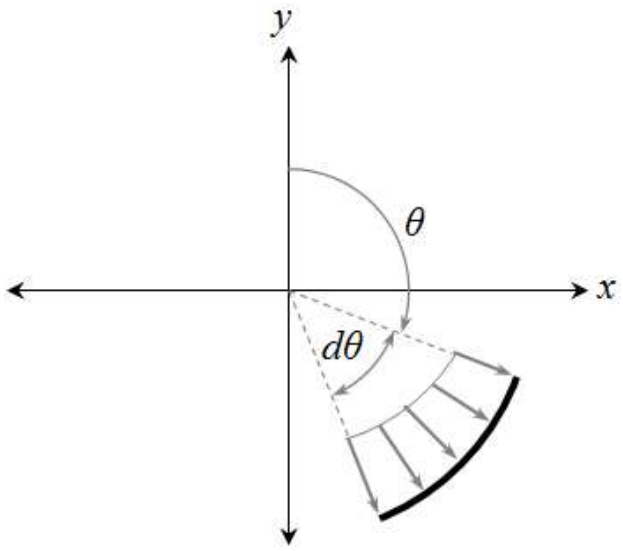


Fig.8.10. Pressure distribution acting on an infinitesimal arc of the loop.

The loading is symmetrical about the y-axis, such that the vertical force acting on the arc shown above (and its mirror image) is given by

$$dF_y = 2p(\theta)dA\cos\theta$$

Where the area of the arc is

$$dA = w\pi d\frac{d\theta}{2\pi}$$

The pressure distribution for $0° \leq \theta \leq 180°$ is given by

$$p(\theta) = P_m\frac{\theta}{\pi}$$

The total vertical force is therefore

$$F_y = \frac{wd}{\pi}\int_0^{\pi} Pm\theta cos\theta d\theta = -2\frac{wdP_m}{\pi}$$

Equating forces in the vertical direction:

$$T = W = mg = -2\frac{wdP_m}{\pi}$$

Solving for P_m:

$$P_m = \frac{\pi mg}{2wd}$$

For a thick ligature, let: m = 70 kg; w = 1 cm; d = 20 cm

$P_m = \frac{\pi 70 \cdot 9.81}{2 \cdot 0.01 \cdot 0.2}$ = 0,54 MPa

= 55 tonnes/m^2

For an electrical wire, let: m = 70 kg; w = 2 mm; d = 20 cm

$P_m = \frac{\pi 70 \cdot 9.81}{2 \cdot 0.002 \cdot 0.2}$ = 2.70 MPa

= 275 tonnes/m^2

These values represent the minimum peak pressure that could be expected. Further sliding of the knot to reduce the diameter of the loop could be expected to significantly increase this value.

These values highlight the magnitude of forces and pressures to which the victim is subjected during the hanging process.

Summary and Conclusions

In the above thesis, the principles of **dimensional analysis** i.e., the breaking down of a complex phenomenon into its component parts, have been applied.
However, in view of the complexity and proximity of structures to one another in the neck, consisting not only of the rigid hyoid-larynx complex and vertebral column but the integrated vascular and neural structures, it appears that not one single biological mechanism can be ascribed and attributed to the cause of death in suicidal hanging. Rather, it appears that unconscioness and death causation appears to be multifactorial. Both the sympathetic and parasympathetic arms of the autonomic nervous system are involved, often with antagonistic and therefore paradoxical effects. In addition, pressure to the **phrenic nerve**, not previously considered in playing a role in death causation in hanging, may, it is suggested, be a major contributory factor in death causation. This **nerve**, the innervation to the major muscle of respiration, i.e. the diaphragm, in a neural response to the compressive and tensile forces in hanging, fixes the diaphragm in a state of inspiratory paralysis. This latter effect would be further augmented by neural stimulation of the accessory muscles of respiration, the **sternocleidomastoid** and **scaleni muscles**, contributing to the thoracic cage becoming fixed in a state of inspratory paralysis. This latter effect, as described, is brought about by initiation of the **dynamic and static stretch reflexes** occurring in these muscles on application of a compressive or tensile stimulus. [128]

Compression of the **carotid arteries**, on the other hand, results, as has been shown, not only in major damage to these vessels and their accompanying veins, but, in addition, must produce a dramatic element of cerebral ischaemia. This would account for the rapid onset of unconsciousness, i.e. within 11 to 12 seconds. Moreover, the unremitting, unrelieved constrictive force of the ligature on these vessels, if perpetuated and left unchecked, would, it is suggested, lead inevitably to brain death in the ensuing 3 to 4 minutes. (This, of course, does not imply death of the individual as a whole, i.e. somatic death, as different organs die at different rates. Death, therefore, occurs by degrees).

Unconsciousness, thus, appears to be the critical factor for it is the state when the victim is unable to save himself or herself. Without unconsciousness survival may occur, but with it, death becomes inevitable. The question then arises – what is the cause of unconsciousness? In physiological terms, carotid artery occlusion induces rapid unconsciousness, i.e. within 11 seconds, resulting in ultimate death. In other words, the sudden and unremitting pressure of the ligature must inevitably result in death. On the other hand, the sudden application of a ligature with consequent vagal nerve compression may produce instantaneous cardiac arrest with cessation of blood flow to the brain and resultant loss of consciousness. This event would produce unconsciousness in less than the time period of 11 seconds of carotid artery occlusion (although the brain continues to survive for several minutes thereafter despite cessation of heart beat). If, however, unconsciousness is contributed to by phrenic nerve compression, it would not be instantaneous as shown by the fact that one can normally hold one's breath for several minutes (as underwater swimmers do) and unconsciousness does not supervene either instantaneously or within 11 seconds. In short, unconsciousness would not occur within 11 seconds in the case of compression

of the phrenic nerve unless a more critical factor supervenes. Thus, the rapidity of onset of unconconsciouness appears to be the critical factor in determining the progression to ultimate (and inevitable) death. Moreover, as has been pointed out in Materials and Methods, the **carotid arteries** in several tested cases would not allow the passage of a probe through the obstructed arteries beneath a tightly applied ligature. This obstruction would, therefore, appear to be the initiator of the deathly unconsciousness factor, although respiratory arrest would be compounded by neural and muscular factors.

While in the foregoing thesis the principles of **dimensional analysis** i.e., the breaking down of a complex phenomenon into its component parts have been applied, the principles of **integrated analysis** i.e., the combining and synthesis of separate parts into a whole have also been attempted. In essence, it appears that multiple factors, acting in concert, simultaneously or in rapid sequence to one another, all play a role in contributing to death causation in the hanging process.

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