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ABDOMINAL DECOMPRESSION

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A Monograph

by

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INTRODUCTION

A SHORT EXPLANATORY foreword to this monograph appears imperative.

Abdominal decompression is the device offered for study and consideration to the reader of the following pages. If that reader accepts, for various cogent reasons, that abdominal decompression has certain effects upon the human organism—these being beneficial—and wishes to try its application, this is simple. Nothing is needed beyond the knowledge that the atmospheric pressure around the human trunk is to be reduced by one to three pounds a square inch of surface. The reader can then improvise some simple equipment and observe the effects of decompression upon him- or herself, upon another laboratory subject or on a patient who is pregnant, in labour, or suffering from menstrual pain or ordinary acute backache.

The present work is but a record of the observable phenomena and their variations: matters which can be ascertained independently by the reader. As there is a wide range of application of the method and much time is needed to put possibilities to the test, an experimenter will be saved two years of work if he or she sets out equipped with the knowledge at present available.

It goes, almost without saying, that explanations of the phenomena encountered have had to be considered in all their bearings as progress was made. In this particular research, most of the morphological events emerged unexpectedly and it is to be emphasized that the study has, in the main, been an empirical one. Upon emergence of phenomena attempts were then made to understand and explain the various manifestations, and experiments were devised to elucidate them.

In the pages which follow, all set down are the author's observations, thoughts, opinions and predictions. Some explanations will, no doubt, be modified—or increased in depth—with the increase of knowledge and resultant aggregations of factual material as time and research yield more. Yet it must not be thought that the refutation of some small part of the whole can destroy, or even vitiate, the validity of the main argument.

It would appear to be a temptation to many to think, because the effects of this elementary device are unusual, that they are also unlikely. Had its operation and mechanism been highly complex, by paradox, the average mind would tend to an immediate acceptance of what was seen or told—exactly as the average mind accepts without contention or even the element of scepticism, all that it is told about electronics, or rocketry as opposed to jet propulsion. It is axiomatic that nothing can be done with those who simply refuse to believe that an enormity of achievement can often be accomplished by an elementary minimum of device. At least one knows where one stands with such people. But there are others far worse—for example, those who are awakened and shaken from complacency by conviction, yet make objections and adverse criticisms or advise trials and experiments which are not primary, or, in some instances, practically possible.

To those who *are* interested in observing the effects of abdominal decompression (and their number grows quite amazingly as time goes on) the ensuing possibilities may be offered.

Facilitation and pain relief in the first stage of labour; reduction of the resistance of the birth canal to the descent of the presenting part in the second stage; extraction, by suction, of the placenta, leaving the uterus very firmly contracted; abolition of pain in primary dysmenorrhoea and relief from backache in both sexes and, finally, the improvement of foetal oxygenation in both labour and pregnancy. The last possibility is the most promising, although the most difficult to substantiate. It is necessary, therefore, to have some perspective of its probability.

In connection with the attempt to raise the physical and mental qualities of the new-born, by relieving foetal hypoxia, one has now to consider which things are certain, which are probable and which are speculative.

Things of which one can be sure: the excellent condition of the babies at birth, sometimes under the most adverse combination of circumstances; progress which is above average—often ranking with that of advanced infants; control of abdominal and intra-amniotic pressures, by decompression, and the absence of foetal bradycardia, even during contractions.

Probable things—those which have not, finally, been substantiated—the inefficiency of the placenta in respect of circulation and its effect on oxygen transmission. Similarly, the principle of syncytial sac low pressure states has been proved theoretically and experimental work to test this is being done elsewhere. Findings concerning the vascularity of the placenta and the significance of haemoglobins, the oxygen saturation of cord blood, and electroencephalograms have to be supported by more work.

Finally: that which is quite speculative—but an inference from reliable premisses—is the suggested effect on the brain. The possibilities, in order of achievement, would be the prevention of neuronal damage resulting from anoxia and leading to gross morbidity, the raising to betterment of inferior babies, improvement of the present average—physically and mentally—and, perhaps, the raising of the gifted child to a highly—or, occasionally, an exceptionally highly—gifted state.

It will be difficult to prove that the structure and function of the cerebral cortex can be improved—except empirically: by a follow-up of the infants who were subjected to the effects of decompression during their mothers' pregnancies. If, however, it can conclusively be shown that there is hypoxia in the third trimester of pregnancy and that this can be prevented to a significant degree, it would seem justifiable to presume that this will aid the development of arborization of the dendrites in the cerebral cortex, and perhaps, even the myelination of the axons. The potential of such a mechanism is so vital as to give high priority to trials on an extensive scale of daily decompression during the last ten weeks of pregnancy.

The reader is accordingly invited to study the evidence, so far accumulated. If the study has essentially been empirical it may, nevertheless, be claimed that the method of abdominal decompression is rational in its application.

PART ONE FUNDAMENTALS

GENERAL PROGRESS

HALLIDAY and the author spent nearly a decade studying uterine action (Halliday, Heyns *et al.* 1950-1958). Observations were made mainly on human parturients in whom the electrical activity of the uterus was studied intensively. Simultaneous records were made with a strain gauge placed on the abdominal wall. Thus we were driven to the conclusion that changes in abdominal contour produced the apparent gradient of activity seen in its descent from the upper abdomen or uterine fundus to the suprapubic area or cervix and lower segment. Doubt was thrown on the source of the electric potentials obtained (Halliday and Heyns) and consideration given to means whereby the action of the uterus upon the abdominal musculature could be simulated. Every possible device had the disadvantage of causing artifacts, particularly in the skin.

In May 1954, while endeavouring to make a cinematographic record of the abdominal outline, with the appropriate muscles in a passive state, a striking event occurred.

The subject was a primigravida in doubtful labour. After prolonged and anxious thought, arrangements were made to administer scoline (succinylcholine chloride) in the experiment. Because of distress caused by respiratory paralysis, and the need to inflate the lung artificially, it is necessary to employ a general anaesthetic. Thiopentone sodium (pentothal) was used. The inhibitory effects of such a narcotic on uterine contractions was expected to cause failure.

This was not so. Contractions occurred despite the influence of pentothal.

At the beginning of the experiment the subject, as has been stated, was in doubtful labour, but she appeared to be having good contractions at the end. Within 68 minutes, dilatation of the os was over 6 cm. To the best of one's knowledge she was in unequivocal labour for not more than six hours. This supreme eutocia was remarked only in passing.

The second patient, studied ten weeks later on 3 August 1954, was a 2-para, having painless contractions at the time. She was considered for the experiment and, with an os 3 cm. dilated, she appeared at best to be in spurious labour. The record ended 35 minutes after giving pentothal and she appeared to have had eleven contractions. Rectal examination showed full dilatation of the os and she was delivered six minutes later.

This experience with parturition, even if it was only a side effect, commanded attention; and the author resolved to pursue the matter to a conclusion. The next case, undertaken eight days later, had only three contractions during the experiment before labour ceased altogether. (A small series is mentioned later.)

Consideration was now given to the mechanics of the abdominal cavity and its walls. Curarizing and anaesthetic techniques were meanwhile abandoned. When

the effect of the abdominal musculature on the uterus in labour was better understood, a method was sought whereby the muscles could be influenced experimentally, and to discover what effect this might have on cervical dilatation.

The reasoning at this stage may be stated as follows.

Dick Read relaxed the general, and by chance also the abdominal, musculature by repeated suggestion.

When at first a safe and effective technique seemed to be unobtainable, one had almost in desperation to consider hypnotism. It was obvious that pronounced relaxation of the abdominal muscles of the flank was something outside the usual experience. The question also arose whether the flat muscles were under voluntary influence. It would accordingly require training during hypnotism to get the subject to understand what neuromuscular actions were required. Suggestion would have to be directed to the idea of a belly which is full from over-eating.

The undoubted power of anaesthetics made it imperative to explore their value for the purpose under consideration. Local analgesia might have to be extensive and reach the level of the 6th dorsal vertebra. In this connection, the rectus abdominis has attracted most of the attention. This is probably correct, for it is quick acting and is the voluntary anterior wall muscle *par excellence*. The flat muscles are concerned with changes in capacity and pressure inside the abdominal cavity.

Epidural analgesia was clearly the most desirable method for the purpose, but anaesthetists tend to be diffident about using this route. Intrathecal spinal analgesia often has such unpleasant sequelae that its experimental use was undesirable. All that remained was the curare-like relaxant. Therefore it was decided to use scoline. Light anaesthesia was required, because it is very distressing for the patient to be conscious of paralysis of the diaphragm.

The unexpected finding that uterine activity was not abolished by anaesthesia and the possibility that scoline exhibited an oxytocic effect were an important step forward. The significant result of these experiments had been a great acceleration in cervical dilatation.

It was concluded that enforced relaxation, or stretching of the abdominal musculature amounting almost to paresis, deserved attention. Dick Read's relaxation was uncertain and could be nullified in the timid by fear, anxiety and a loss of morale. Hypnotism might be precarious because few women could be expected to be sufficiently good subjects to respond to the requirements. The control of relaxation had to be certain in order to hold out a promise of any value, and the muscle stretch had to exceed what was normally found. While scoline might be satisfactory for experimental trials, a method had to be found that was less perilous and arduous.

The idea had been considered that the reduction of the atmospheric pressure outside the abdomen to about one-fifth of an atmosphere might relax the abdominal musculature and perhaps allow the wall to bulge forward.

While our belief in this possibility was firm, it was met with incredulity by others. It was pointed out, however, that the intestinal gas of the subject would expand because of a decrease in pressure, and would thus stretch the abdominal wall to some extent. There were the additional arguments that (*a*) descent of the

diaphragm would result from the atmospheric pressure of the inspired air opposing the low pressure of the abdominal cavity, and (*b*) that at one-fifth of an atmosphere half a ton of weight is taken off a large abdominal wall.

Experimental facilities were extended to us by the Assistant Surgeon-General (Air), Central Medical Establishment, South African Air Force. A decompression chamber, normally used for the flying personnel of Lyttelton, was set at our disposal.

The significant experiment was done with the Kifa type of cuirass, which is placed over the thorax in the treatment of poliomyelitis patients. Partial exhaustion of air was effected by means of the powerful pump available.

Adno and I fitted the cuirass over the abdomen and observed abdominal effects at pressures of 500 mm., 400 mm., and 300 mm. Hg. The atmospheric pressure at Lyttelton was 650 mm. Hg (altitude 4,700 feet). It was demonstrated, conclusively, that the abdominal wall projected forward on decompression. Also it was found that powerful retraction of the abdominal wall could not be maintained during decompression. One found it possible to speak without effort at a pressure of 500 mm. Hg, but at 400 mm. speech came in a whisper.

On our way home and for some hours afterwards both of us felt quite sore behind the xiphisternum, but this only reminded us of our success. It must be emphasized that, at this stage, we knew nothing of the mechanics of abdominal decompression and considered, only, the application of the discovery we had made.

It was judged that parturients would feel apprehension at pressures below 500 mm. Hg, but it was certain that at this two-thirds of a full atmosphere the muscle stretch would be sufficient for the purpose under consideration.

Between 29 April and 21 September 1955 another 11 cases were treated with scoline in early labour. About half the cases gave a poor response during anaesthesia, although some of these did well subsequently.

Meanwhile endeavours were being made to construct a small chamber to fit on to the anterior abdominal wall, to devise a means of sealing, and to discover of what capacity the roughing exhaustion pump would have to be. Delay was caused mainly by the bonding of the rubber seal to the metal and getting it to fit the abdomen, but the first parturient was treated with decompression in November 1955. There were two cases on 15 November, each one being so spectacular that the method gave promise of something unusual. Both were young primigravidae in the very earliest of labour. The first one was delivered in 3 hours after the start of decompression and the second in 2 hours 20 minutes.

Decompression was not, at this time, intended for second-stage use. However, the second stages were short and the most notable feature at the time was the relaxation of the patients, and their freshness. The short duration of this stage was dramatic.

Discussion

During the first stage of labour, when the uterus contracts, the anterior abdominal wall is pushed forward and stretched by the uterus. The energy responsible for this originates entirely in the uterus. It is necessary to visualize the relaxed uterus as hanging loosely and extending on either side of the lumbar vertebral

column. Its shape is such that it has one long axis and two quite short ones. Abdominal palpation puts this statement beyond all possibility of doubt. The growing uterus has taken the shape of the abdominal cavity, which possesses similar axes. As a result the uterus is like a balloon that has been compressed anteroposteriorly and is accordingly rather sausage-shaped. When this happens to a balloon with contents of constant volume it must stretch—and so does the uterus.

The abdominal cavity is entirely filled with its permanent contents and, if anything further is to be introduced into it, the only possibility of expansion is forward and to some extent laterally because of the stretching of the muscular wall. Space would be gained over the entire supero-inferior diameter for every unit of gain in the anteroposterior. Therefore an organ growing in such a cavity must, if compressible, assume an elongated shape with a shorter anteroposterior axis than if it had remained spherical.

It may be remarked that the spherical uterus seen in a pendulous abdomen would hold the necessary contents with less stretching than the sausage-shaped viscus. The pregnant uterus appears to be naturally round and, when there is sufficient tone or tension in its wall to overcome resisting forces (gravity and parietes), it will change its shape towards the spherical.

We may consider three effects of a uterine contraction in its early phases:

1. To the extent that it is isotonic, the wall will shorten, but without reducing the volume of the contents, which are incompressible.

2. As the uterus becomes rounder its available internal volume must increase. Instead of forming vacuums the myometrium shortens. (It seems obvious that, without shortening, the placenta would never separate and, if separation were to be effected manually or by haemorrhage, the parturient would quickly bleed to death —Heyns 1950.)

3. To become spherical, the flattened compressed uterus must gain in the length of its anteroposterior and possibly transverse axes. This requires the anterior abdominal wall to give. We know from simple inspection that it does so give, and that the upper abdominal wall moves well forward during a first-stage contraction. Descent of the diaphragm has the same effect, for it causes a reduction of the supero-inferior axis which of necessity leads to an increase in the short axes.

Therefore, if the abdominal wall yields freely, uterine contraction will effectively undo distortion by raising the viscus from its paravertebral flaccidity to a prevertebral erection of spherical shape. If the abdominal wall does not force the uterus back again, the so-called retraction will be highly efficient, for, after every contraction, the full shortening of the muscle will remain without any subsequent lengthening. The shortening normally occurs in the upper uterus partly because it is in the upper abdomen that change of shape can occur freely. The rearrangement of the myometrium lays the foundation, as it were, for dilatation of the cervix.

At rest, the upper segment is ellipsoid rather than spherical, with the long axis supero-inferior, the transverse axis sometimes next in size, and the anteroposterior certainly shortened by abdominal wall pressure. It is a matter of simple observation that the anteroposterior axis increases during a contraction, the transverse showing its lengthening by bulging of the loins. Expansion of the lower rib cage, as in

inspiration, would provide considerable space for both diameters in the uppermost fundal part of the uterus.

While shortening of the long axis must occur sooner or later during uterine activity, the length of this axis will depend on where its lower limit is considered to be. However, contraction results in an upper uterus which, if not spherical, would have an eccentricity nearer to 1 (unity) than that of the flaccid ellipsoid.

In this case the volume of the sphere would be greater than that of the ellipsoid but, as the uterine content remains of constant volume, shortening of the myometrial wall is possible. Proof of the potential change in volume may be stated as follows, but thinning of the wall can also be demonstrated by flattening a toy balloon containing water.

Suppose the original radius of the sphere was x.

Then volume of sphere $=\frac{4}{3}\pi x^3$

If the semi-axes of the ellipsoid are $(x-a)$, $(x-a)$, $(x+a)$ then volume of ellipsoid $=\frac{4}{3}\pi(x+a)(x-a)^2$.

Now we wish to show that the volume of the ellipsoid is less than the volume of the sphere. This will be so if:

$\frac{4}{3}\pi[x^3-(x+a)(x-a)^2]>0$

i.e. if $ax^2+a^2x-a^3>0$

Now the roots of this function are:

$x=-\frac{a}{2}(1+\sqrt{5})$ and $-\frac{a}{2}(1-\sqrt{5})$.

Since the function has the same sign as the coefficient of x^2 except when x lies between the roots, the inequality does not hold for:

$-\frac{a}{2}(1+\sqrt{5})<x<\frac{a}{2}(\sqrt{5}-1)$

Neglecting negative values of x we have that the inequality holds for:

$x>\frac{a}{2}(\sqrt{5}-1)$

i.e. $a<\dfrac{2x}{(\sqrt{5}-1)}$

Therefore for all $a<x$ the ellipsoid has a smaller volume than the sphere.

Accordingly the conclusion arrived at is—with change of shape of the uterus during contraction, isotonic contraction (real shortening) is possible. On the other hand, with no change of shape isometric contraction occurs with no resultant shortening.

The idea is elementary but fundamental in an explanation of how shortening and thickening of the myometrium of the upper segment take place. Any arc in the perimeter that changes shape may shorten but, if it is located in the fundus, it will either lengthen subsequently when the uterus becomes flattened, or overcome and stretch a length of lower segment proportional to the degree of shortening that persists. It is further possible that isometric contraction will raise intra-amniotic pressure without promoting dilatation and obliteration of the cervix; it certainly will not effect the requisite rearrangement of myometrium in upper and lower segments.

What is even more significant is that the sliding together of muscle elements in isotonic contraction with shortening may take place almost without a rise in intra-amniotic pressure, and in these circumstances is accompanied by less pain, possibly the backache moiety. Isometric contraction in which the stress fails to effect strain is accompanied by pronounced rise in tension of the uterine wall; but even pure isotonic shortening must cause some degree of tension having regard to the fact that a force operates in producing the shortening of muscle units.

It is depressing to think of the reverse of this picture, which is in fact the normal state of affairs.

In addition to what has been thus far considered, including the fact that the uterus dissipates much of its energy in overcoming a resistant abdominal wall, another result of tense abdominal musculature is the retention of the uterus in an unfavourable axis. Not only does the long axis of the foetus lie at a considerable angle to the axis of the brim, but the upper segment is at an angle to the lower so that the uterus is bent on itself or kinked at the sacral promontory. These faulty axes are present whether the foetal head is engaged or not. In the presence of a high tension myometrium the kink would tend to straighten out, thus bringing the fundus forward.

The first proposition is made. It is simply that the resistance to stretch of the muscular abdominal wall is a factor opposing cervical dilatation.

If this proposition is substantiated, there is an explanation for Dick Read's concept of relaxation. Whereas he said "tense mind—tense cervix", the truth lies in the fact that unresisting abdominal muscles allow the *corpus uteri* to come forward and to be in line with the resisting cervix.

Apprehension alone will cause tension of the abdominal muscles, force the viscus back on to and over the spine, and reduce uterine efficiency even to the point of inertia. The uterus itself will be producing the energy, but it will have to do so much more while working isometrically. If it is tending to become rounder even though prevented from changing its shape by the abdominal musculature, amniotic pressure will drop and some value be gained.

ABDOMINAL CAVITY

Decompression as applied to the abdomen in labour is not an empirical method. The two primary factors in the rationale are uterine and diaphragmatic action, and the former has now been considered. However, variations in the mechanism and technique for the second and third stages of labour require that the elementary structural and mechanical features of thorax, diaphragm, abdomen, pelvic girdle and pelvic floor be appreciated.

1. *Pressure*

The intra-abdominal pressure is at least atmospheric: the extra-abdominal weight of the atmosphere must of necessity compress the soft, flexible muscle walls so as to equalize the pressure inside and outside of these walls. It was found pro-

fitable to contemplate some of the more obvious effects of changes in abdominal shape and pressure, and it became clear that the subject was one of importance and offered fruitful applications both in health and disease.

2. *Mechanics*

A moment of reflection will suffice to bring to mind that the abdominal cavity is a single chamber (the pelvis being included) with the diaphragm as roof and with the pelvic floor below, the anterolateral walls being muscular and fascial. The vertebral column, pelvis and rigid thoracic cage provide for stable origins and insertions of the muscles. The abdominal contents fill it entirely and without leaving any dead spaces.

Within the viscera there may be gas, liquid and solid; but externally they are in close apposition and quite firmly pressed together. On the anterolateral abdominal wall alone there is a weight due to the pressure of the atmosphere of over half a ton. It is, therefore, not to be expected that a series of flat muscles of inconsiderable bulk could withstand such force sufficiently to allow intra-abdominal pressures below atmospheric. While it is possible, having regard to muscle power, it must, nevertheless, happen very rarely in nature that abdominal muscles are held contracted in a fixed position while the abdominal contents decrease and suffer a drop in pressures of perhaps a few mm. Hg.

It is different when the region of the uppermost abdomen that lies within the lower costal cage is taken, for negative pressures occur during respiration; and only—11 mm. Hg has been recorded in the stomach during decompression.

As the diaphragm is highly mobile and capable of reducing or increasing abdominal capacity by its descent or rise, it is necessary to appreciate its action very clearly.

It may be thought of as a piston moving up and down. If there really is an absolute movement, the abdominal contents will not only be forced downwards and their pressure increased, but room must be found for them unless there is some intestinal gas that can be compressed. The structure that resists least is the abdominal musculature, and simple inspection shows how the wall bulges outwards during inspiration and recedes during expiration.

The range of movement of the diaphragm is just over an inch in deep respiration, and about half an inch in quiet respiration. With the subject lying down the diaphragm is situated highest, but the dome descends in the erect posture, and it is even lower when sitting writing at a desk. The higher the diaphragm the greater its respiratory excursion.

The thorax and the abdomen are two adjacent compartments with the diaphragm as a mobile partition between. At its simplest we may regard the pressure in the thorax (strictly speaking in the bronchial system) as being presided over by the glottis. If the thoracic pressure is below atmospheric, opening of the rima will allow air into the lung. The normal thoracic pressure may, therefore, be considered to be atmospheric. When decompression lowers the abdominal pressure on the other side of the diaphragm sufficiently, the diaphragm will be forced downwards in an attempt to equalize the pressures in the two chambers.

The mechanism of increasing the capacity of the thorax is familiar to all, but it is necessary to point out that in abdominal breathing inspiration follows considerable increase in the girth of the lower costal cage which in itself causes an abdominal bulge. The movement is inevitable, because the flat muscles are fixed to the ribs. Thus the diaphragm reduces the supero-inferior axis of the abdomen, but there is an increase in the anteroposterior during forward bulging of the abdomen.

During decompression of abdomen and lower thorax by 50—150 mm. Hg below atmospheric pressure, the increase in girth is very great at the level of the transpyloric plane, slight at the umbilical level, and not appreciable at the transtubercular plane. The abdominal musculature being so intimately associated with respiration, it is possible that the neurones dominating the muscles of respiration also preside over those of the abdomen.

3. *Morphology and Phylogeny*

For the foregoing reasons the important question arises, of the extent to which there is voluntary control over the flat muscles of the abdomen.

One possibility is that the oblique and transverse muscles merely lengthen and shorten according to space requirements. In respect of this function it should be pointed out that the rectus abdominis differs greatly from these in its form and direction of fibres, and is strongly under voluntary influence. In most vertrebates the rectus abdominis extends from the shoulder region to the pelvis. As the sternum grows in length the rectus is restricted, and in mammals it is a purely abdominal muscle. It is in mammals that the diaphragm develops. Morphologically and because of its cervical nerve-supply, it is thought that the musculature of the diaphragm originated from a superior portion of the rectus, perhaps in the shoulder region, and migrated downwards at an early stage of pre-mammalian history in association with the heart and the lungs.

Another function of the flat muscle seems to be postural. Because *Homo sapiens* is erect he has different requirements from quadrupeds, when resisting gravity. These lateral hypaxial flat muscles go to constitute much of the body wall in fishes. Phylogenetically they are derived from the myotomes, but they arise in the embryo of at least some land vertebrates, from mesenchyme in the body wall lateral to the myotomes. Mesenchyme gives origin to all smooth and cardiac musculature.

In land life, as opposed to the aquatic in which there are forces opposing those of gravity, the function of supporting the trunk viscera is a new requirement. Then there develops in tetrapods a rib system of varying power and extent, but the continuity of the hypaxial musculature makes it conspicuously a unit. This develops into the subvertebral muscles dorsally and the rectus group ventrally, with the flank muscles following the curve of the trunk.

In tetrapods the flank muscles have little bulk and the bundles are scattered inside the periphery of the fascial covering of the belly. The muscle content suggests that it is included in the body wall for the purpose of contractility and because it affords greater stretching than fascia.

In erect Man, on the other hand, the muscle fibres themselves arise from bone, are of greater volume absolutely and relatively, and finally have a great iliac crest from which to arise. This part of the ilium in tetrapods is very small. In the erect posture, Man maintains his equilibrium with the help of the spinal muscles, but he must clearly make use of the abdominal musculature for versatility in poise and trunk agility. It is submitted that the flank muscles in Man are so powerful that their function may be mainly postural.

It would not be surprising if subsequent study of chronaxie values shows them to be relatively sluggish in response, and to a very small extent under voluntary control. Histological structure of the muscle and a clear demonstration of the nerve-supply in animals are required, but it cannot be expected that a great deal of light will be shed by these methods.

These myological considerations throw light on the problem of gaining paresis or marked relaxation of the abdominal wall. This was originally a requirement for the study of abdominal contours, but later was found to aid the efficiency of the uterus in labour.

A superficial assessment of the anatomy led to what may be an error, viz., that the infiltration with novocaine of only the rectus muscles was of small value. To provide local anaesthesia to the oblique and transverse muscles is an extensive undertaking reaching the mid-dorsal level. The recti can be directly infiltrated, but this would be undesirable for the entire abdominal wall. However, from what has been suggested, it now seems likely that in the abdominal wall the rectus is the striated muscle *par excellence*, and that its elimination must provide very nearly as great flaccidity of the parietes as is attainable.

Our theory is that the flank muscles merely conform to the crude forces acting upon them; nor must we lose sight of the great part played by diaphragmatic paresis in giving more space at laparotomy.

4. *Surgical Anatomy*

For didactic purposes it has been found necessary to cite a few examples of applied anatomy in the abdomen, as these assist in making the student familiar with the problems under discussion. It may accordingly be of value to begin with a consideration of what happens when a suprapubic incision is made into the peritoneal cavity of a patient in the Trendelenburg position.

Where the abdominal wall is fully relaxed gravitational force tends to pull the viscera towards the diaphragm. This does not cause a vacuum in the pelvic region because atmospheric pressure pushes in the paralysed abdominal wall and supports the inclined column of viscera. When the peritoneum is incised within a few inches of the pubes, air enters forcibly, sometimes whistling audibly, and then the viscera descend. The phenomenon is due to the elevated region, the bony pelvis being incompressible.

At the level of the umbilicus the internal pressure is higher than atmospheric, to the extent that there is tone or tension of the abdominal musculature. Here air cannot enter unless a low pressure space is made for it by pulling up the incised abdominal wall.

Nearer the pubes, however, atmospheric pressure supports mobile pelvic viscera, e.g. gut. The pelvic pressure is, therefore, less than the atmospheric by the weight of this column of its contents. Thus the outside air is under greater pressure than this region of the cavity, and it will flow in when the incision is made.

It would obviously be good practice to blow air into the peritoneal cavity through a needle locally before making the incision. Whatever the response to this suggestion, the belief must not persist that a relaxed wall in the Trendelenburg position may be incised without danger to the underlying gut.

The diaphragm, in successful anaesthesia, is high anatomically and provides more intra-abdominal space in the Trendelenburg position; but the unresisting flank muscles also bulge outwards to bear the mobile organs descending by gravity.

In the case of a tumour incarcerated in the pelvis the experience of letting in air behind, by prizing it up, is common. The explanation offered is that this phenomenon would occur where air enters the anterior part of the pelvis and makes pressure through the uterovesical pouch and, of course, in the direction of the pouch of Douglas. The small intestine descends by gravity, as happens with any body that is dropped. The pouch of Douglas is accordingly emptied. If the tumour which is being forced backwards by the air entry effectively seals the pouch of Douglas, it will be jammed with great force towards the cul-de-sac. Theoretically it may require a force of up to 200 pounds to dislodge such a tumour. Fortunately it is easy to wedge the fingers between the tumour and the sacral promontory and allow air to enter.

When William Hunter failed to dislodge the uterus in his case of incarcerated gravid uterus, vascular effects and oedema would have set in and must rapidly have increased the impaction. No doubt the injection of air into the cul-de-sac (from below if it were not possible from above) would have helped to deliver the uterus.

The board-like rigidity resulting from a perforated gastric ulcer must be due to the need to reduce peritoneal sliding which causes pain. A reflex would effect muscular spasm driving the diaphragm upwards into a fixed position, and only upper costal respiration would be possible.

The collection of gas in the intestine in subjects confined to bed for upwards of twelve hours is probably due to lowered abdominal pressure permitting some degree of dilatation of the gut. An adequately contracting gut reduces vaporization of the volatile contents and may force gas so formed into solution in the blood. Post-operative distension is probably due to a failure of this process or to the sucking in of air. The latter is usually undetectable at the time. It is likely to occur during anaesthesia.

It may be of value to pass a tube at the termination of the operation to let out all gastric air; then to apply firmly a many-tailed bandage to prevent laxity of the abdominal wall and consequent intestinal dilatation. If the patient shows distress because of excessive flatus, the binder will have to be removed. The possibility in this case is that air sucked in had advanced beyond the stomach. The higher the abdominal pressure, the more force would the gut have to exert in propelling its contents along. The flatus is more likely to advance distally if the abdomen is not compressed.

5. *Obstetrical Anatomy*

Trials of suction around the pelvic outlet proved successful.

The subject sat on a decompression chamber with a suitable rubber seal. The levatores ani forming the pelvic diaphragm or floor of the abdominal cavity may be regarded as the partition above which the pressure is abdominal and just higher than atmospheric, and below which there is the lower pressure induced by decompression. There is at present no doubt that there is a pressure head between the contents of the abdomen and the birth canal below the elvatores.

Thus second-stage expulsion of the foetus was accomplished without bearing down where decompression was applied during contractions, and actually shortened when accompanied by bearing down. Decompression appeared to facilitate descent of the head even where arrest had occurred above the brim.

Suction after separation of the placenta secured delivery in 20—30 seconds without any need to manipulate the abdomen. Hardness of the empty uterus was more pronounced than found in normal delivery. This is probably due to exsanguination of the uterus via the uterine veins which are subjected to the negative pressure inside the bony pelvis.

Postpartum haemorrhage due to atony has been treated satisfactorily by decompression and a firm uterus obtained.

The musculature *post partum* must be in a relatively flaccid condition, and its fortification by an efficient abdominal binder would prevent the uterus from becoming atonic and increased in size. Women are comforted by the binder *post partum*, and its use appears to be rational. Blood is apt to pool in the large veins, perhaps because the small intestine subsides into the relaxed loins. In cats it has been found that, after a fall of blood pressure from an induced haemorrhage, a rise to normal has followed injection of sufficient fluid to raise the abdominal pressure.

Discussion

The phrase 'plastic tonus' was applied by Sherrington to the state of certain muscles in decerebrate rigidity. Under normal conditions the inhibitory pathway from the cerebral cortex prevents this rigidity. The main reflex centre for decerebrate tonus is in the midbrain, and decerebration increases its excitability so much that slight peripheral stimuli maintain certain groups of muscles in a state of reflex tonic contraction. Thus the contracted limb extensors can be flexed to any degree and remain fixed in this position. It was this state that Sherrington called plastic tonus.

The expression is convenient for describing the behaviour of the muscular abdominal wall, even if it does not refer specifically to Sherrington's definition. Invariably in the living state there is tone in the abdominal musculature. Its behaviour is unusual in so far as it stretches and contracts to accommodate the ever varying abdominal contents. Two elementary properties may be recalled.

(1) Ingestion of substantial alimentary matter leads to stretching, but the muscles are soon in equilibrium or in relative rest. The feeling of fullness after feasting disappears as the fibres resume a state of normal tension at their new and

increased length. The same situation prevails with exaggerated respiratory activity.

(2) The rapidity of abdominal muscle activity can be similar to that of average skeletal muscle, a fact ascertained by palpating the contractions of the former during rapid trunk movements. With the slower stretch following diaphragmatic descent on ingestion, abdominal muscle function is reminiscent of the slow contractile rate of smooth muscle.

The question arises whether artificial stretching of the abdominal wall early in labour would allow of enhanced relaxation for several hours thereafter. The rapidity of action referred to may be due entirely to the rectus, which could counteract much of the elongation induced in the flat muscles.

Schafer in 1900 suggested that the abdominal wall movement of respiration was under the influence of the respiratory centre. Though difficult to prove, this seems to be sound. It accords with the possibility that the flat muscles are largely involuntary and postural.

MECHANISM OF OUTLET DECOMPRESSION

When decompression was first applied to the outlet, it was questionable how far into the soft tissues the partial vacuum would extend. It seemed certain that its effective limit would be the floor of the abdomen, which incorporates the levatores ani. Pressure in the abdominal cavity would be at least atmospheric, as under normal conditions.

Experiment gave the following results: an obturator fashioned from paraffin wax was inserted into the vagina and was expelled by decompression. If uncomfortably large, the obturator was not readily expelled. A Fergusson speculum placed in the vagina and held in place firmly by adhesive tape around the vulva was ejected with what seemed to be considerable force.

Decompression in the second stage was attempted next, and there was material evidence that a pressure head was at work. When a high foetal head in a small parturient (aged 15) descended quickly with suction and the child was born with unexpected ease, one was at a loss to explain the mechanism.

Finally, the application of decompression in the third stage was regularly followed by expulsion of the placenta. This and the unusual firmness of the postpartum uterus were explained tentatively by various mechanisms, but without confidence.

Matters were further complicated when it was observed that vaginal pressures remained positive during decompression of the outlet. It was only just inside the introitus that pressures corresponded with those in the chamber. Moreover, the wax obturators were not expelled when inserted well into the vagina, i.e. right up at the vault.

In the present attempt at explanation, the following morphological basis is employed.

The levator is inserted around the vagina very low, on average at the junction of the third and lowest quarters of the vagina. The levator is just as mobile as the

perineal skin covering the bony outlet. It is, in fact, virtually tacked down on to it at the central point of the perineum and along the anococcygeal raphe. In the angle between the two lies the ischiorectal fossa with its fascia lunata and compressible pad of fat.

In so far as the perineal surface moves downward about $2\frac{3}{4}$ inches (6.7 cm.) during the second stage of labour and the vulval cleft and its posterior extension can move upward from the midposition of erect posture for more than 2 inches (5 cm.), the mobility of the pelvic diaphragm is remarkable and must of necessity correspond with the range of movement of the integument. At rest, the pressure in the abdominal cavity is a few millimeters of mercury above the atmospheric: the soft walls are forced in so as to conform to the external pressure, but in addition the postural tone of the musculature makes a small pressure contribution.

Abnormal states like standing on one's head cause a negative pressure in the incompressible pelvis, but these do not apply here. When the trunk is erect, however, the pressure in the pelvis is about 35 mm. of mercury above the atmospheric level, owing to the column of mobile viscera supported by the pelvic floor.

Pressure above atmospheric is referred to as positive and that below as negative.

The first question to settle is whether the negative pressure can extend up along the vagina and perhaps into the uterine cavity. This seems unlikely, because most of the vagina lies above the levators in the abdominal cavity and is compressed by the surrounding positive pressure. The more negative the pressure inside the lumen, the more completely will it be obliterated.

This view would also explain why all vaginal pressure readings above the levator are positive in the presence of outlet decompression.

In view of the success of decompression in both second and third stages, one wondered whether a pregnant woman with a substantial presenting part in or over the pelvis would be different from the subjects used for the experiments. In considering the firm postpartum uterus, a conclusion was not unnaturally reached that the uterus was exsanguinated.

It is not clear on first thoughts how a negative pressure could develop around the uterine veins for the purpose of sucking out the intramural blood. An alternative mechanism would be the positive abdominal pressure on the outside of a uterus of which the cavity has been decompressed by some 2 pounds (0.9 Kg.) per square inch. The objection to this was the fact that the vaginal pressure readings during the experiment were never negative. The author, F. S. Crafford, pointed out that this should cause severe haemorrhage, and we have since demonstrated the correctness of this contention.

The only solution that can be offered is this. A normal presenting part, but also the third-stage uterus, is of such bulk that the pelvic brim is sufficiently blocked to prevent viscera passing from abdomen to pelvis minor. The smallest structure available for such transfer would be colonic contents, a loop of intestine or the omentum. Pressure changes in fluid contained in a vessel are transmitted freely so as to retain a uniform pressure, but in the peritoneal cavity the fluid elements are contained in relatively massive units, like loops of intestine, which can-

not pass through the correspondingly narrow gaps and slits remaining between the pelvic brim and a bulky uterus forced down upon it.

What is visualized, therefore, is that decompression, acting through the non-rigid pelvic floor, causes a drop in pelvic pressure, for the pelvic basin is now isolated from the abdomen proper. Whereas the abdomen has compressible walls, the pelvic walls are rigid. If by a progressive drop in pressure the levator is forced downward because of a difference in pressure between its superior and inferior surfaces, a transient increase in volume would result and cause a partial vacuum. The brim is obstructed by the uterus and its bulky contents, but no other viscera enter the true pelvis. (The intestine has to be compressed so tightly as to cause an insuperable obstruction.) Thus a low pressure region exists in the inferior part of the pelvis above the levator level.

It is accordingly clear how a foetal head above the brim, particularly in the presence of disproportion, maintains a negative pressure in the pelvis below it. Positive pressure in the abdomen above and negative pressure in the pelvis below the head cause it to be given momentum. At the same time, the aperture in the pelvic floor (levator gap) is forced open by decompression and offers less resistance, which in effect means that the levator is partly pushed out of the way.

These conditions affect the other situations similarly: thus, the placenta is expelled because there is a negative pressure in the region superior to the levators; the uterus is exsanguinated because there is high pressure around the upper segment and low pressure around the lower externally, and blood is squeezed above and finds its way along the paths of least resistance.

Conversely, the positive vaginal pressures occur in the non-pregnant because there is no means of blocking the pelvic brim, with the result that any variation in pressure on one compressible abdominal wall (levator) is met by an opposite change elsewhere in order to keep the volume constant and the pressure uniform.

Finally, in the case of the vaginal obturators, expulsion is possible only when a portion of the mass projects through the levator gap: when it lies entirely above the levator, there is no pressure gradient to drive it out. When its size is excessive, the force that can be derived from its relation to the levator gap may be insufficient to expel it.

Experimental Observations

1. Vaginal pressure readings in the third stage were made, a Statham head and galvanometer being used. At the termination of the second stage, decompression was started, the cylinder being applied to the patient lying in the dorsal position. The results were as follows: (*a*) Pressures were negative and followed the chamber pressure with a difference of about 60 mm. Hg. (*b*) With the descent of the placenta into the lower part of the vagina, the pressure rose to the atmospheric level. (*c*) Expulsion of the placenta was followed by a return to negative pressure.

2. Vaginal pressures were measured, as afore-mentioned and described, in the cases of women near term with the foetal head above the brim, engaging, and engaged in, the pelvic cavity. Pressures remained unchanged (i.e. 3 to 4 mm. Hg)

with decompression, except when the head obviously fitted tightly in the brim or lower part of the pelvis.

3. In the second stage of labour the effect of decompression was best seen when there was a tight fit of the foetal head in the pelvic brim. In a few cases of disproportion, descent—and even birth—were dramatic. When decompression failed to effect expulsion of a head that was in the pelvis, it was our impression that the head was too tightly impacted to be moved by the pressure gradient employed. It seems likely, however, that the vaginal pressures may not have been negative in this case. On the other hand, a small foetus has been sucked right into the decompression cylinder. In our opinion, crowning of the head is intimately connected with passage through the levator gap.

At this stage is seems that decompression should give some impetus in all cases.

4. It was a disappointment to find that outlet suction during the fourth stage caused rapid and severe haemorrhage from the placental site. It was noted that vaginal pressure was negative at this time. This corresponds with what we now call the F. S. Crafford mechanism, to which reference has been made. We still hope that closing off the lower part of the vagina by means of an obturator may prevent a drop in pressure during decompression for severe fourth-stage haemorrhage. What is presumably required is a negative pelvic pressure outside the uterus. A negative pressure inside must cause the Crafford phenomenon.

PAIN RELIEF IN LABOUR — A NEW REFLEX

Labour pain seems to be felt in the back, suprapubically, and generally all over the abdomen in front. Relief of pain during decompression was at first related to myometrial tension. In that section of the antenatal clinic where decompression is given during pregnancy many patients declared after treatment that their backs felt easier and less tired. In this atmosphere and following certain personal observations, relating to sudden cramp in the back musculature and pain of the lumbago type, one examined the possibility that the nerve conductor system of labour pain might lie within the proprioceptive nervous system.

Inspection and personal experience when subjected to abdominal decompression make it obvious that an exceptionally deep inspiration is caused. During such inspiration the ribs are drawn upwards and spread apart, an action that pulls upon the tendons and fleshy processes of the iliocostalis lumborum and longissimus dorsi columns. If the tension of the inferior fleshy mass of the sacrospinalis is high, or it is in spasm, or in a tetanic state of some kind, pull on the tendons and slips will also cause a stretch on the thick muscular mass from which they arise.

Therefore, if the conductor is proprioceptive, stretch of the muscle will break spasm. As decompression proceeds, a marked lordosis of the lower vertebral column develops. This would result in shortening of the sacrospinalis in the sense that it forms the bow-string of the vertebral bow that is now more curved. Such an expla-

nation would account for the empirical observation that decompression in labour relieves pain in the back.

With regard to the rectus, stretching of this muscle is a prominent feature during decompression, and this would explain the relief of the abdominal pain obtained. Decompression causes bulging of the abdomen to the extent that its girth increases by 2 to 3 inches, sometimes as much as 4 inches. The increase occurs at the umbilicus and just below, but it is seen most of all above—in the epigastric region.

To test our hypothesis several sets of electrical recording were done. As a first step electrical impulses were recorded over the sacrospinalis during labour contractions (Fig. 1). It was well known that even more marked electric potentials

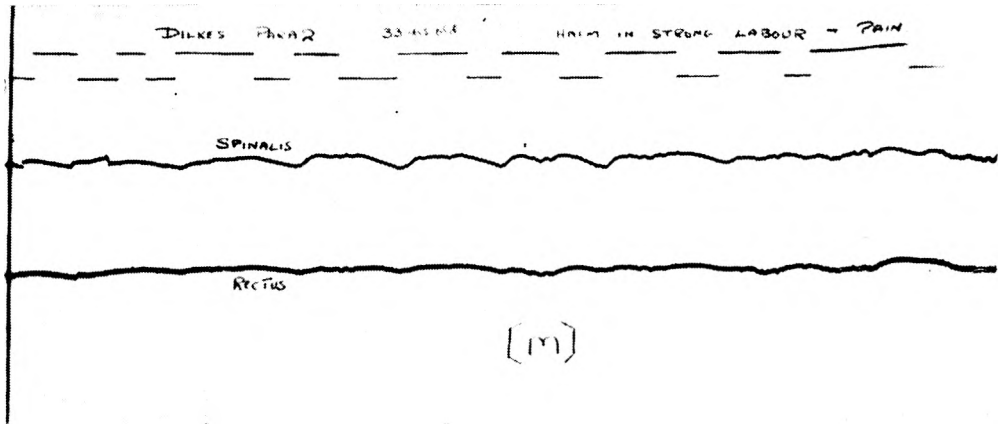


Figure 1
Electrical activity in sacrospinalis during labour

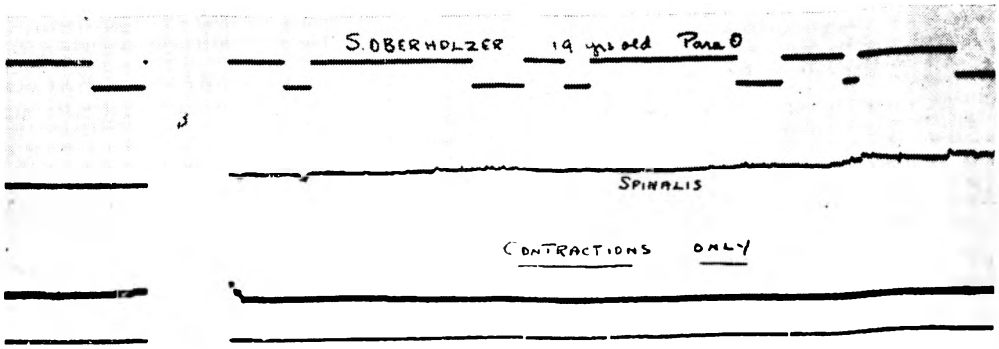


Figure 2a
Electrical activity in sacrospinalis during labour (without D)

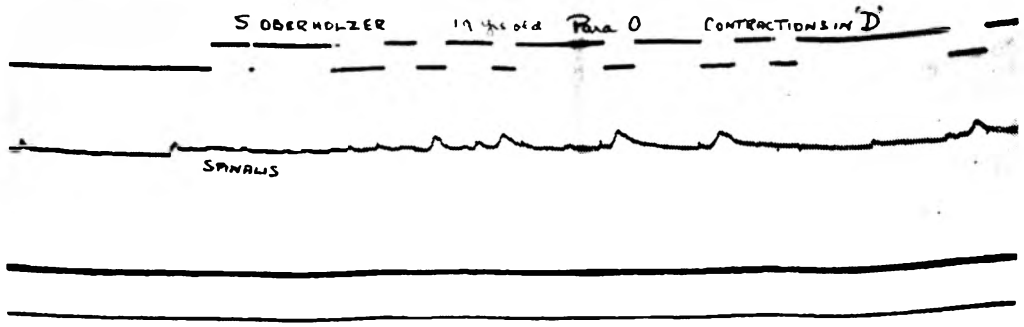


Figure 2b
Electrical activity in sacrospinalis in labour (with D)

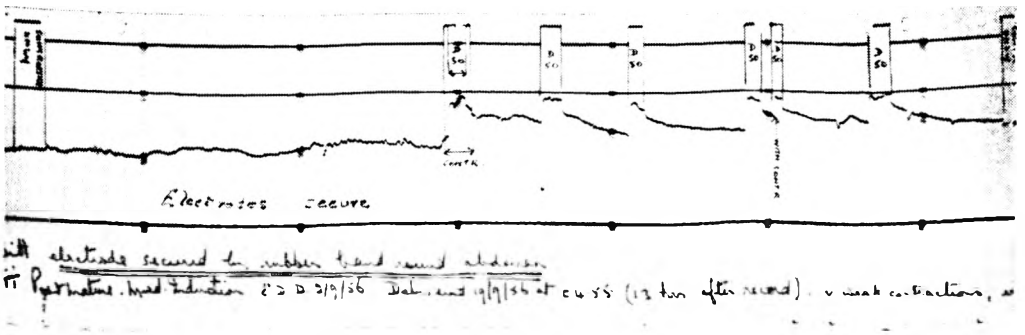


Figure 3
Electrical responses in labour: C→C+D→C

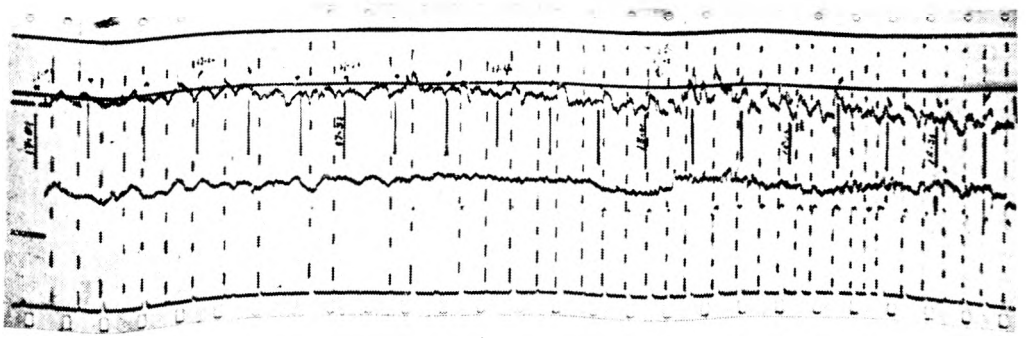


Figure 4
External electrical tocography in labour

occur in the rectus during uterine contraction (Fig. 4). At the same time electrodes placed over these anterior and posterior trunk muscles during abdominal decompression showed deflections of several hundred millivolts (Figs. 2b, 3, 18, 19).

The view was gaining ground that labour pain was not visceral but reflex. The receptor area would be the region of the vaginal vault, and the conductor would have an afferent path—probably somatic—with the cerebellum as head ganglion and spinal extensor and flexor muscles as effectors. The possibility was that few if any impulses reached the sensory cortex.

Experiments were accordingly devised to test the existence of the reflex.

Women at the postnatal clinic were examined according to routine, but electrodes were placed on sacrospinalis and rectus and a Foley catheter was introduced into the uterus. The aim was to stretch and pull upon the lower segment, cervical canal and vaginal vault.

Electrical impulses of the order of 10 to 50 millivolts resulted from these stimuli, but also when the vulva was touched and the introitus stretched, and most of all on bimanual examination (Fig. 5). The question that now arose was whether dysmenorrhoea could be relieved by abdominal decompression.

Cases of severe dysmenorrhoea were sought and found. The relief resulting from this treatment in primary dysmenorrhoea exceeded all expectation. Acute backache in both sexes was similarly relieved, often dramatically.

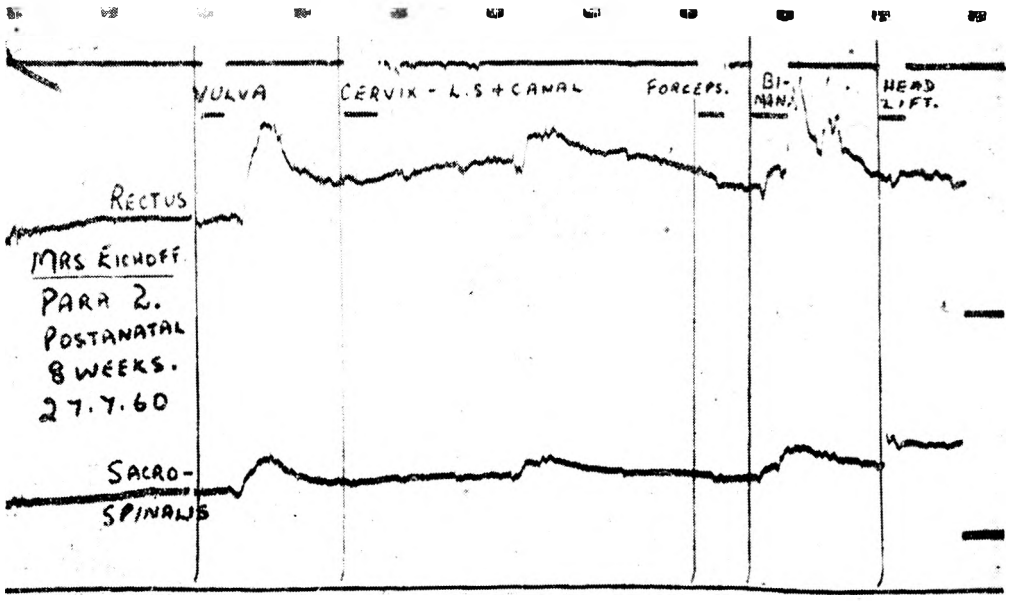


Figure 5

Electrical responses in rectus and sacrospinalis on stimulation of receptor area of new reflex

PART TWO CLINICAL

FIRST STAGE

INSTRUCTIONS pertaining to Decompression in the First Stage of Labour.

Technical

1. There are four main components. These are the chair, the disposable suit, the fibre-glass spacer, and the pump with its tubing and gauge.
   (*a*) The suit is laid open on the chair so as to allow its upper margin to reach between the base of the breasts and the armpits when the patient sits semi-erect in the chair.
   (*b*) The bucket-seat backing-plate is placed on the suit and the patient takes up her position on the seat. The cage is placed over the abdomen with its

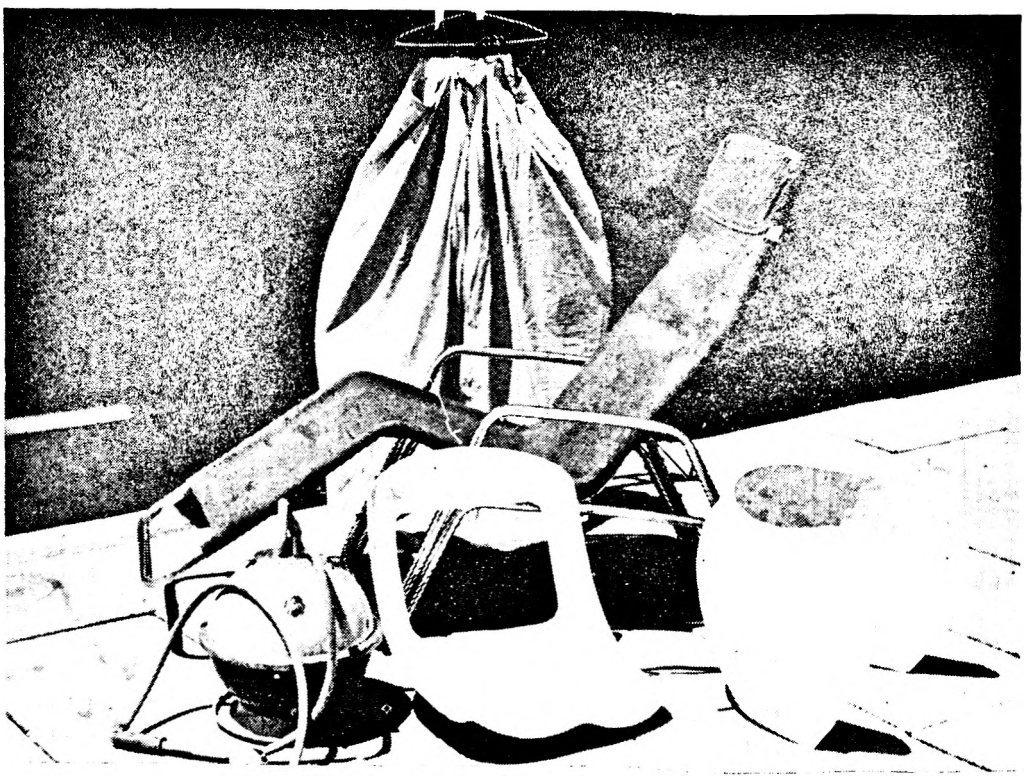


Figure 6
Stage I equipment for D

edges in the grooves on the bucket-seat, and with the metal pipe on the patient's right.

(*c*) The zip is closed carefully, using some degree of delicacy, and asking the patient to help steady it. If the fit over the cage is tight, the zip-containing edges can be pulled together very firmly when it will be found that the plastic material of the suit stretches readily.

(*d*) Sealing at the upper chest, above the base of the breasts, is essential. The fit need not be very tight, for sweating inside this non-porous material has proved to be the most effective factor in securing a seal. It is necessary in each case to insure that the back part of the upper opening of the suit is neither creased nor pulled down too far. Unless the parturient has a good layer of subcutaneous fat over the sternum, it helps if she presses the zip against the breast bone every time she starts suction.

2. Suits vary in upper chest girth at the sealing area in gradations of two inches. The plastic material is stretchable. A pleat can be put into it with the help of a piece of sticking tape. Above all, the presence of perspiration (which starts soon, owing to the air-tightness of the plastic) helps sealing more than anything else. This moist seal occurs below the uppermost edge and naturally requires a substantial area. During operation the suit may be pulled downwards, and attention should be given to the back to make sure that the suit is high enough to seal. In front, endeavours should be made to prevent a process of suit being sucked into the cage from above. The pressure in the process will be atmospheric, and it is essential that decompression must be operating around the lower rib cage and the epigastrium. In order to overcome the tendency to downward pull on the free edge of the suit in front and behind, a 'bib' 11 inches wide and 5 inches long has been added to the free edge behind with cords extending from each corner. The 'bib' reaches the lower part of the neck and the cords pass on either side of it to be tied loosely to the upper end of the zip.

3. If a backing-plate (bucket-seat) with a window cut out of it is used, lordosis during decompression is proportional to the area of the window. It has been proved, however, that some lordosis occurs with the backing-plate complete. Pain relief is partially dependent on lordosis, and operators may wish to experiment with these variations in the backing-plate.

   The torus on which the woman sits will fit into the vulval cleft. Its purpose is to keep the perineum, anus and anococcygeal raphe from being pushed downwards by the levatores ani. There is a pressure head from the upper surface of the levator ani downwards, because the abdominal cavity pressure does not fall below atmospheric, and the pressure around the vulva is negative.

   Without the torus there is downward pull on the vagina (and indirectly its vault) by the levator. It is possible that mobilization of the region of the vaginal vault is obtained most effectively by upward and downward pull, i.e. with and without the torus. Once labour is progressive the torus should be used, and perhaps it may be dispensed with towards the end of cervical dilatation.

Note. The above pressures at 50 mm. Hg below atmospheric are as much strain on the subject as 100 mm. when no backing-plate is used. With the torus the former 50 mm. is tolerated much better. However, we are not much in favour of pressure readings greater than these, although some women using only the cage have employed 130 mm. readings, and more, in an attempt to abolish all pain.

4. It will be appreciated that, when the tubing connects pump and gauge and suit, the circuit is arranged so as to be an open one. When, however, the subject closes the open T-piece with her thumb, the pump exhausts only air from the suit. As soon as the T-piece is open again, most of the suction will be from the outside air.

   Maintenance of the pump is necessary for efficient decompression. The main point in this respect is the ability to drop the pressure rapidly when a contraction starts. The tubing and intake of the pump should be cleared of clothing particles at regular intervals of about a month.

The Patient

It is desirable to explain the function of the mechanism to the patient and thereafter to seek a form of 'indoctrination' toward the end of pregnancy, so that she is fully instructed and given confidence. One session is usually enough, and a

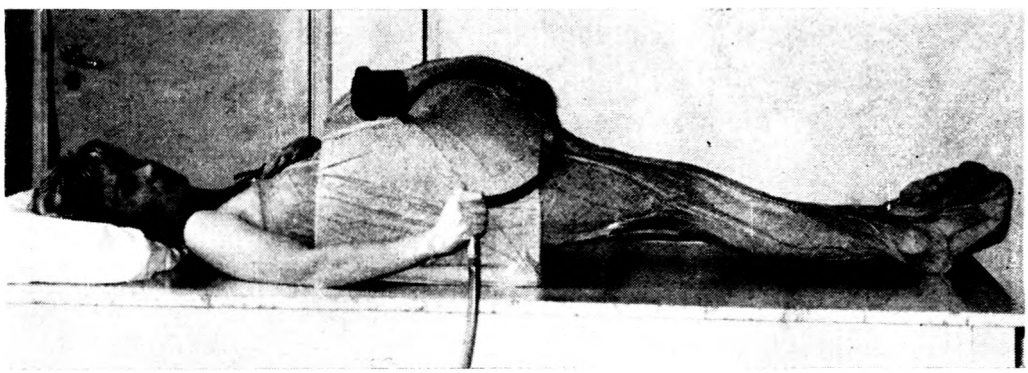


Figure 7
Alternative Stage I equipment for D

run of half an hour, with the patient experimenting, has been our practice. Some patients are apprehensive and may have to repeat their experience to gain confidence. This indoctrination readily becomes the decompression contribution to the ripening process that appears to be the basis of the onset of labour. If it is convenient, therefore, the two procedures may be combined by giving decompression on at least two occasions of an hour each, the last about the expected time of delivery.

The patient is shown how the apparatus works and how to read the pressure gauge. She operates the outlet valve herself. At pressure readings above 40 mm. Hg she will be conscious of interference with respiration. This is due to downward pull and fixation of the diaphragm during decompression. Upper costal



Figure 8
D equipment in use

breathing is readily carried out, except at readings beyond 100 mm. Hg. The average patient has no difficulty with respiratory adjustment, and she strives after pain relief in labour even if it means that she has to hold her breath for some seconds. During pregnancy she tends to co-operate because she has immediate relief of the back strain, ache, or pain that is characteristically present.

In labour, then, encourage the parturient to control the duration and strength of decompression.

Let her have an occasional contraction without decompression, in order to judge the degree of pain relief.

Check the foetal heart after an hour.

She may like to bring decompression back to 20 mm. Hg rather than zero at the end of the pain. The desirability of this practice is open to question, having regard to uterine efficiency.

She must try not to feel concern about contractions, but must rather take the contractions in her stride while providing decompression, and preferably retaining the continuity of her thoughts with a book.

TABLE I

DURATION OF LABOUR STAGES FROM START OF DECOMPRESSION
(100 PRIMIGRAVIDAE)

Time in Hours	Stage I (Number)	Stages I and II (Number)	Time in Minutes for the Groups Shown in Col. 2		Remarks
			Stage II (Average)	Stage II (Range)	
1–	7	4	27	5—47	
2–	15 (5)	9	34	20—75	
3–	9 (3)	15	29	5—60	
4–	19 (3)	11	31	10—75	
5–	14 (1)	22	25	10—60	
6–	8	7	43	10—95	
7–	0	3	48	20—67	3 occipitoposterior
8–	6	2			
9–10	6	5			
10+to 20	12	15	46	20—80	5 occipitoposterior
over 20	4	5	33	19—50	4 occipitoposterior
	100	98	35.0	5—95	

Results

Table 1 reflects the results obtained in the first systematic series of 100 primigravidae and (shown in brackets) 12 multiparae.

1. (*a*) In 78 cases decompression was started at 0 to 1-finger (2 cm.) dilatation.
   (*b*) In 22 cases decompression was started at 2-finger (4 cm.) dilatation.
   (*c*) In no cases was decompression started at more than 2-finger (4 cm.) dilatation.

2. Sometimes decompression was stopped with a rim of cervix present. This frequently took a long time to disappear but the time up to the unequivocal full dilatation was the one counted.
3. There were 2 Caesarean sections not shown under Stage II.

Note on the Four Stage I Failures (i.e. over 20 hours)

In 3 cases the pain relief was 70 per cent and in the fourth, 50 per cent. The first had decompression for 10 hours and in some respects was not a failure. Two had decompression for just over 7 hours, one having had contractions only during such treatment, the other having been in labour for 36 hours previously. The fourth was decompressed (for 5 hours) by request because inductions for postmaturity had failed.

TABLE II

RELIEF OF PAIN (100 PRIMIGRAVIDAE)

Percentage Relief Estimated by Patient	Primiparae	Duration of First Stage in 9 Individuals having less than 30% Relief	
		Hours	Minutes
100	2	1	0
90	3	1	10
80	5	2	40
70	31	2	50
60	11	3	35
50	25	4	0
40	9	6	0
30	5	8	50
		19	45
	91	9	

Table II shows the degree of pain relief in the 100 primigravidae of Table I. They were White women.

In 1959 I personally conducted a critical series of 56 primigravid parturients. The women were all given an hour session, sometimes more than one, to learn the use of decompression during pregnancy and were requested to come to hospital, but preferably to inform me, at the very earliest signs of the onset of labour. The intention was to take labour from its very beginning.

Five women had to be rejected: one failed to report, one had elective Caesarean section, for one there was no apparatus available at the time, one was a psychotic, and the last was an agitator in the antenatal ward and was refused decompression by me.

Labours under 5 hours were 19 or 37 per cent (control 13.5).

Labours under 7 hours were 24 or 47 per cent (control 24.5).

Labours under 8 hours were 27 or 53 per cent.

Labours under 10 hours were 32 or 63 per cent.

Failures were 37 per cent, the range of the duration of the first stage being 10 to 33½ hours, with some longer but difficult to estimate. In this series the objective was entirely to discover the extent to which decompression could facilitate labour. Even among the failures in this respect the pain relief justified the procedure and such remarks as "pain relief remarkable" were recorded in some cases of failure.

In order to determine, reliably, the duration of the first stage among our hospital primigravidae we observed 200 consecutive White parturients. The specific difficulty is to discover the time of irrevocable labour, disregarding spurious labour. De Snoo (1948) was perhaps nearest the truth when he counted the onset of labour from the earliest signs: it required 18 hours to 4 cm. and 24 hours to full dilatation. Three of us watched the labours. Difficulties were encountered, but we did our best as experienced and devoted obstetricians. The times were as follows.

The percentage of primigravidae having a first stage under 2 hours was 1, under 3 hours was 3, under 4 hours was 6.5, under 5 hours was 13.5, under 6 hours 19.5, under 7 hours 24.5.



Figure 9
Antenatal clinic patients having D

The percentage of primigravidae having a first stage over 24 hours was 8.0, and lasting 12 to 20 hours was 29.5. The average time was 13 hours 4 minutes.

The next step was to test the finding that decompression given on several occasions during the last 10 days of pregnancy effects changes in the birth canal leading to efficient dilatation of the os. A hundred women at the antenatal clinic were listed at random. Alternate women were to have decompression and the others would form the control group. Subjects are inevitably lost through going into labour before being contacted and for other reasons, and only 48 were finally used for the study. There were 20 treated women and 28 controls. A list of the total was given to each of three registrars who were not informed as to which were the controls. They had to conduct the 48 labours without using decompression and to ascertain the duration of the first stage, but also to assess the efficiency of the labour generally and on the basis of specified variables. There was an even sprinkling of first and subsequent labours.

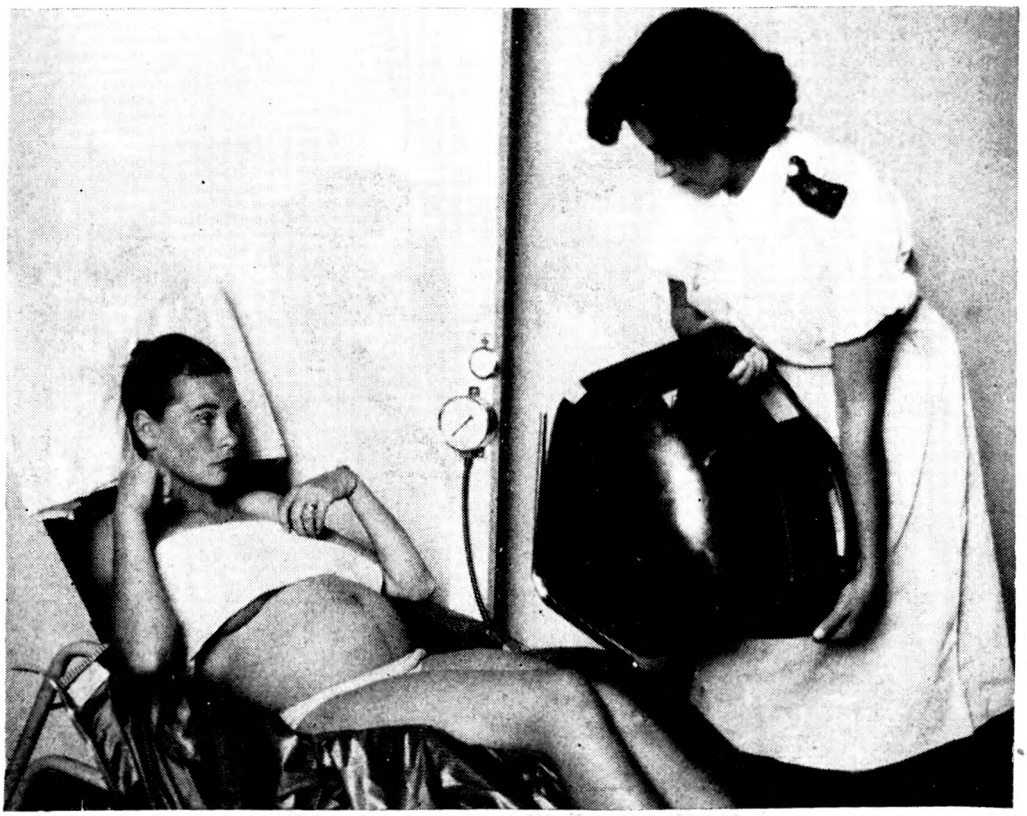


Figure 10

Placing spacer of D

The staff nurse subsequently had the D infant with a vocabulary of 200 words at 18 months

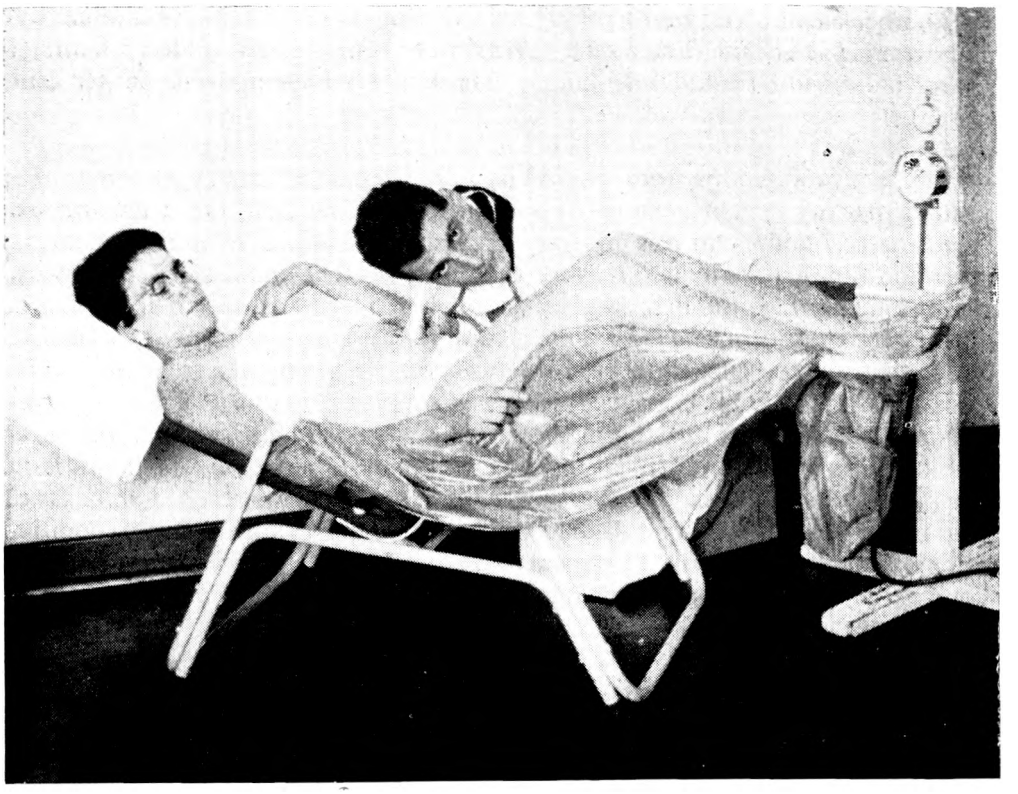


Figure 11
Listening to foetal heart during D

	With Decompression Stage I Time	Without Decompression Stage I Time
Mean for nulliparous patients ...	9 hrs 14 min.	14 hrs 15 min.
Mean for parous patients	6 hrs 15 min.	6 hrs 11 min.
Mean for Total	7 hrs 54 min.	11 hrs 5 min.

Of the 20 decompression women 2 had a first stage under 3 hours, 9 under 5 hours, 11 under 7 hours, and 16 under 10 hours. Thus 55 per cent were under 7 hours, but 9 were second or third labours.

The percentages for efficient labour were lower in the controls but the differences were not statistically significant. However, one felt that 270D was well worth prescribing. It is relevant to state here that 270D became "pregnancy D" because no cases of premature labour occurred. On this foundation it was first remarked early in 1960 that the decompression new-born were unusually fine babies.

Another small series of 18 primigravid decompression labours was done by a highly trained Assistant (Potgieter). There were 6 first stages under 5 hours, 10 under 7 hours, and 12 under 10 hours. The under-7 hour figure is 56 per cent.

Conclusion

The last four groups were studied by myself or under my supervision. It is admitted that my personal under-7 hour figure is 47 per cent, but 3 labours were only just over 7 hours and it is arbitrary to fix the time of onset within an hour, not to mention the quarter hour. It is for these reasons that one has stated elsewhere that in primigravidae the first stage of labour is reduced to under half the normal in just over 50 per cent of cases. This is significantly different from any unassisted average that may be alleged to prevail anywhere. If the figures from Bainbridge, Nixon and Smyth (1958) are taken, one is well supported in this and also in the presumption that 14 hours for the first stage is a reasonable estimate for the mean. Quite empirically, however, it is submitted that any time beyond 12 hours is not normal, even though it may be average. This conclusion was reached amongst the Bantu in the tropics when one studied child-birth under primitive tribal conditions and one was able repeatedly to remain with the parturient for virtually the entire labour. A progressive first stage judged in this way seems to be unduly prolonged when it exceeds 12 hours.

Clinical Notes and Case Histories

It would be ideal to postpone decompression until the patient is in irrevocable labour. Unfortunately, the women in spurious labour very frequently complain about their suffering, and there is no doubt that decompression gives relief. In effect, therefore, any women who are likely to be in labour and also those who are well advanced in the first stage should be regarded as candidates for decompression.

The patient is now shown what effect decompression of the abdomen and lower chest has on her breathing. From about 40 mm. Hg below atmospheric pressure the diaphragm becomes immobilized in its lowermost position. This is not very unpleasant, but it demands some mental adjustment. When the patient is having painful contractions, she appreciates from the outset that there is relief of pain. She is, therefore, an apt pupil. If she is in strong labour, she learns to control the decompression herself within 3 or 4 or 5 contractions. She is quite happy to remain in the chair for several hours. The experience of having a few contractions without decompression usually leads her to request decompression without cessation. The suit is easily opened and the cage removed for listening to the foetal heart. It may be emphasized that there is very little inclination for the parturient to leave the chair in order to walk about, lie on the bed, or even to wander off aimlessly to the toilet.

The patient starts decompression as soon as she becomes aware of the onset of a contraction. Decompression tends to increase the length of the contraction, and apparently also its strength. Nevertheless, the patient suffers very much less with its use. Varying technique has not been exploited to any great extent, and control of decompression has been left to the patient for the most part. However, the

level used normally is over 50 mm. Hg during contractions, but many patients are quite happy about dropping this to 100 mm. Hg, and sometimes to 125 and 150. Much depends on the relief of pain gained by the patients, but also upon the temperament and the robustness of the subject. There is no doubt that beyond 100 mm. Hg only upper costal breathing is possible, and a certain fortitude is required. In the laboratory some of us have withstood a pressure drop of 200, 250 and even 300 mm. Hg.

As the patient is delivered out of the suit in a routine fashion, ordinary clinical judgment is required to decide when the cervix is fully dilated. This is as straightforward as in a labour which is conducted on orthodox lines. When the os becomes fully dilated within 1, 2 or 3 hours, surprise on the part of the attendant is usually experienced. In no case has foetal distress been found to develop during decompression. Late rupture of the membranes seems to be characteristic, and it seemed probable to us that intra-amniotic pressure was reduced during decompression.

The seven cases that follow are from my personal series. They show the work involved and illustrate several significant points.

Mrs C. H. No. 3938 N. Para O. Age 24. E.D.D. 25.3.59. Height 5 ft. 5 ins. "Indoctrinated" 3.4.59. Keen. 50 mm. Hg. Suit 30. Notch 5. Weight 5 lb. 15 oz.

Stage I 9 hours 5 minutes
Stage II 35 minutes
Stage III 16 minutes

10.4.59

00.55 Decompression was started. Excellent patient. 50 mm. Hg. Contractions began to be more regular after her husband rang me 3 hours previously. He had phoned after 2 hours and then brought her in on my advice. She had appeared to start on the 8th, but had had only ½ hour contractions and had been sent home. She had some pains during the day and phoned several times. In the admission ward, contractions appeared to be coming every 3 minutes.

01.25 The last 3 contractions came in 3 minutes, 2 minutes and 4½ minutes. The next were after 4½ minutes and 4 minutes. The last contraction lasted 48 seconds.

01.38 Contraction lasted 40 seconds. Contraction 38 seconds. Complete relief. Contraction 48 seconds. Went to 75 mm. Hg. She was truly delighted with decompression. She said that she felt like a human being again. She said before that she was very happy to have had this experience. Contraction 43 seconds.

03.00 F.H.H. Patient had bowel action and fair show. Very pleased to be back at decompression.

04.30 to 04.45 Pains were stronger, 75 mm. D, and lasted over 1 minute.

05.45 P.R. 3 fingers.

06.10 Decompression resumed.
06.50 Probably fully dilated.
06.55 F.H.H.
07.35 Delivered.

Mrs F. F. No. 4472 N. Para O. Age 26. E.D.D. 10.4.59. Height 5 ft. 7 ins. "Indoctrinated" 2.4.59. Nervy. 50 mm. Hg. Suit 28. Notch 3. Weight 7 lbs. Occipitoposterior.

Stage I 5 hours 45 minutes
Stage II 55 minutes
Stage III 15 minutes

Note: This was the case of the spectacular misjudgment by Sister and me. The patient had returned after lunch and the cervix was fully dilated. She was the first person to make me realize how *Beta* can be overlapping *Gamma* and make labour sudden and short.

13.4.59

Had pain on and off for a few days. At 17.00 on 12.4.59 contractions seemed to start. Admitted 22.40. At 23.30 p.v. 1-finger, head near I.S. L.O.P. F.H.H. Contractions occurred at most every 5 minutes, lasting under 30 seconds. When I saw her, contractions were much less than this. Decompression started, in order to stimulate labour and ease pain.

A. 00.30 Decompression started. It completely removed all pain.
01.00 Contractions became longer. Decompression to 50 mm. Hg and just over.
02.10 Decompression stopped and contractions watched until 02.40. Contractions now occurred every 10 minutes. They were at 9—10 minutes and the best I saw was with a 6 minute interval. Patient clearly out of labour. Given chloral 30 gr. for a good sleep.
08.30 Patient looked very well groomed. Had a "wonderful" sleep, bath, etc. *Sent home.* (Said she had been having contractions every 20 minutes.)

B. *Later* This patient had been discharged by Sister and me with contempt. Rang soon afterwards to say she was having contractions. Sister impatient. Patient came in at lunch time fully dilated.

Comment This patient and her labour were the first and strongest factors to suggest 270D, or decompression in the last 10 days of pregnancy.

Mrs G. M. G. No. 1341 P. Para O. Age 19. E.D.D. 16.4.59. Height 5 ft. 7 ins. "Indoctrinated" 2.4.59. Very good. 100 mm. Hg.

Suit 34. Notch 5. Weight 7 lb. 4½ oz.
Stage I 3 hours 55 minutes
Stage II 10 minutes
Stage III 12 minutes
4.4.59
She came in prematurely. Heavy urinary infection with evidence of moderate pyelitis. This seemed to initiate contractions. The striking feature was a foetal heart rate varying 180, 188, 200. Dr Potgieter contacted me and was concerned about the foetal heart and thought that she would not be safe for decompression. I said I thought decompression would help and went down.

22.30 Decompression started. Stopped after 20 minutes to listen to heart—then 152. Decompression continued for an hour after which rate was 148. Rate remained 148 after that. (The uterus had been irritable on admission and until decompression was started. She had regular contractions which may not have been pains and were quite relieved by decompression.) Went gradually out of labour.
5.4.59

02.00 Decided to stop decompression as contractions were infrequent. We were quite satisfied as our main aim was to restore a normal heart rate. She was transferred to Ward 1 where the pyelitis was treated. Later discharged.
14.4.59
Woke up at 01.00 with pain. Admitted here, and I was informed at 03.00 that os was 3 fingers dilated. Did not go, because I did not catch the name.

03.10 Delivered.

Comment Quite remarkable, short labour ultimately. Baby very well. The favourable effect of decompression on the foetal tachycardia made a deep impression on all concerned.

Mrs F. J. No. 4109 N. Para O. Age 24. E.D.D. 12.4.59. Height 5 ft. 4½ ins. "Indoctrinated" 7.4.59. Good. 50 mm. Hg. Suit 28. Notch 5. Weight 6 lb. 8 oz.
Stage I 6 hours 15 minutes
Stage II 1 hour 25 minutes
Stage III 2 minutes
14.4.59
I was notified at 01.00 that she had entered hospital in painless labour with os 3 fingers. I did not go down as she needed no pain relief and was too advanced. She became aware of contractions at 22.00 (13.4.59).
Said to have painless contractions.

Contractions declined round about 03.00.
Delivered 05.50.
(She ultimately did not do as well as Mrs G.M.G. who took about half her time. Mrs G.M.G. had had the possible preparation and Mrs F.F. had this "wonderful" painless labour, as the midwives called it.)

Mrs H. A. R. No. 4147 N. Para O. Age 22. E.D.D. 28.3.59. Height 5 ft. 8½ ins. "Indoctrinated" 8.4.59. Fair. 35 mm. Hg. Suit 30. Notch 2. Weight 8 lb. 11 oz.
Stage I 3 hours 30 minutes maximum
Stage II 12 minutes
Stage III 4 minutes

11.30 Post mature. Medical induction 9.4.59 failed.
10.4.59
P.V. revealed very thin cervix, head well engaged and well applied. Os 1–1½ fingers. Membranes stripped and os easily stretched to 2 fingers. Pitocin drip started.

12.45 Membranes ruptured spontaneously. Appeared to be having contractions.

13.40 Decompression started.
Very poorly adjusted.

14.00 Pitocin drip removed.

15.00 Os fully dilated.

15.12 Baby born.

Contractions throughout were every 3 minutes. They became progressively longer and stronger lasting up to 65 seconds. This patient adjusted herself with difficulty and gave considerable trouble. I had to give her my individual attention or else I may have lost her for decompression; but she was agreeable with me.
Giving her an occasional contraction without decompression convinced everyone how much decompression helped to diminish her pain. When I still thought her to be not well adjusted I asked her whether she would have a contraction without decompression and she said "nothing doing". She had from 11.30 a maximum of 60 contractions, perhaps 50–60, i.e. 20 (2–3 p.m.), 20 (1–2 p.m.), less than 20 (11.30–1 p.m.).

Mrs S. T. No. 3789 N. Para O. Age 21. E.D.D. 13.4.59. Height 5 ft. 2 ins. "Indoctrinated" 1.4.59. Very good. 50 mm. Hg. Suit 30. Notch 3. Weight 8 lb. 3 oz.
Stage I 6 hours 40 minutes
Stage II 40 minutes
Stage III 25 minutes

18.4.59

Woke up with contractions at 02.30. Contractions became more frequent and stronger. Admitted 05.10. I was notified 06.00. P.r. os 1-finger. We decided I could wait a while before seeing her. (L.O.A. head high. Liquor amnii ++ F.H.H.)

She was clearly in very early labour at best. Decided on decompression.

09.00 Decompression started. Contractions did not come. But backache immediately relieved. First pain was totally abolished at 50 mm. Hg. Very short.

09.15 to 09.55 Nine contractions.

10.30 Said contractions were more frequent. Up to 70 mm. Hg suction. Contraction lasted 45 seconds; contraction after 4 minutes lasted 35 seconds; after 4 minutes lasted 25 seconds. No pain felt at all at 45 mm. Hg but used higher decompression. Contractions again after 4 minutes exactly. Contraction without decompression lasted 25 seconds. Contraction after 5 minutes. Contraction after 4 minutes lasted 50 seconds. Did stopping decompression delay the contraction from 4 to 5 minutes?

12.25 Out of decompression. Contractions had been declining, and were now absent.

20.05 Contractions every 5–6 minutes. This had been the case at best all day. She was a very good subject and would rather have got under way before resuming decompression.

19.4.59

at 19.15 p.v. 1+finger.

20.4.59

at 01.10 fully dilated.

Brilliant labour ultimately. In effect, unintentionally, 3½ hours of 270D (while in false labour) and the result was an easy, short first stage.

Mrs L. M. No. 4021 N. Para 2. Age 32. E.D.D. 3.4.59. Height 5 ft. 4 ins. [1952, rather long labour (8 lb. 8 oz.). 1954, few hours labour (7 lb. 8 oz.)] Weight 7 lb. 5 oz.

12.4.59

Membranes ruptured 04.00. Contractions felt 04.30. Came in later and had fair contractions.

13.00 P.r. 2½ fingers; then labour subsided; 15.00 p.v. 2½ fingers. F.H.H. R.O.P. level of I.S.

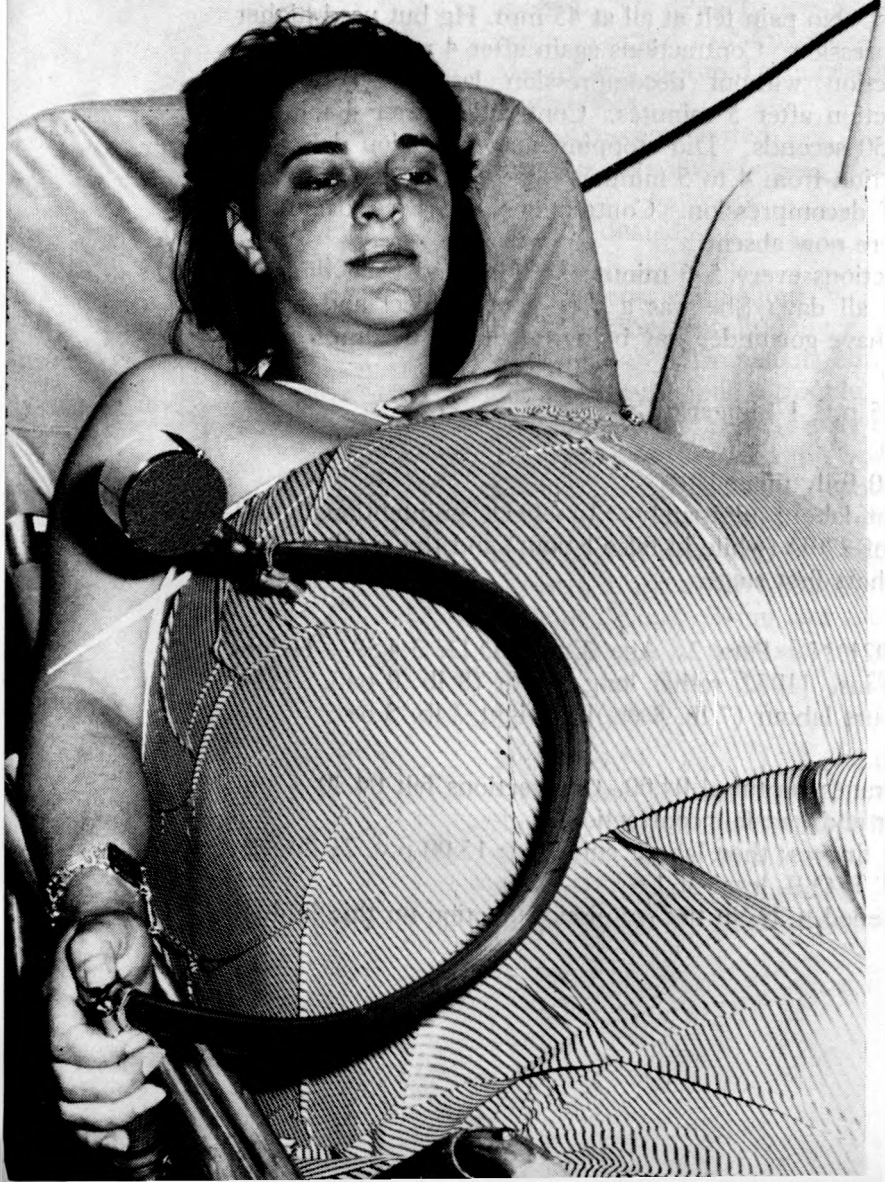
I happened to be in the Admission Section on this Sunday

Figure 12

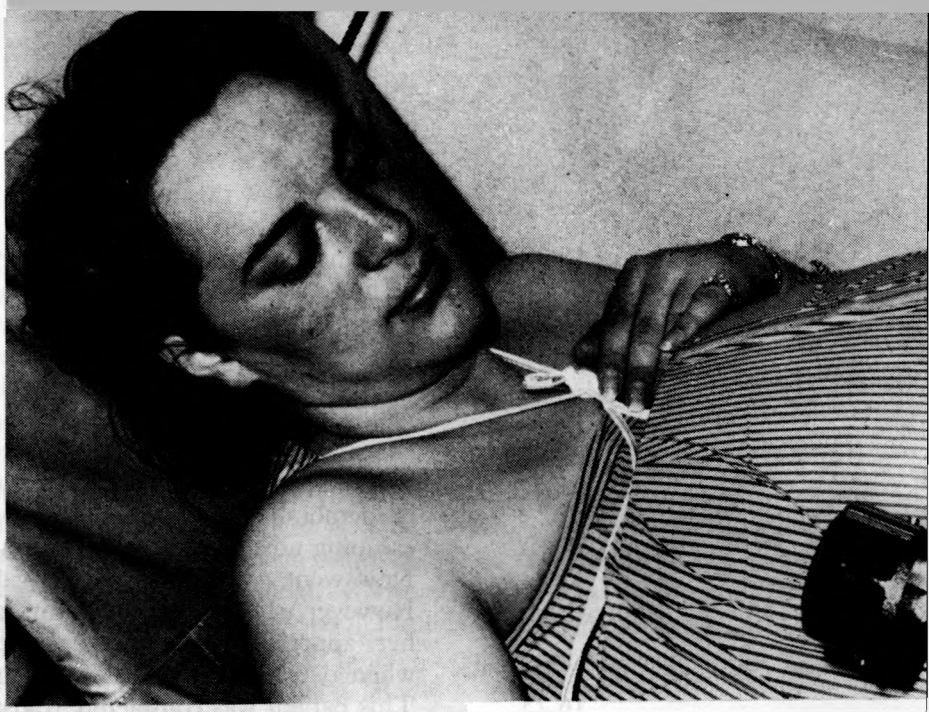
Sequences showing pain relief by D



1. **Mrs B. 22 year old primigravida during a contraction in strong labour.**



2. **First contraction in decompression (100 mm. Hg). Thumb on valve. Complete pain relief.**



3. Fourth contraction in decompression. Paturient now relaxed and confident. The male born of this labour walked unaided at the age of 7 months and 2 days.



4. Mrs P. 20 year old primigravida well advanced in labour. Second contraction in decompression. The smile breaking through the grease and sweat and tears is more striking in the original colour transparency.



5. Fifth contraction in decompression.

afternoon, saw this multipara who was not a patient of mine, and took pity on her.

15.25 Decompression with small pump. Very tentative suction at first, went to 25 mm. Hg happily, but was frightened beyond that! Noticed a difference immediately. She had been having marked inter-contraction backache and this was relieved at once by 25 mm. Hg.

15.40 According to herself, she was now having proper labour contractions. Working decompression better, seemed almost to be enjoying herself. Had been weeping and was thoroughly demoralized. Now she showed dignity, and air was escaping noisily and regularly from the control valve.

15.53 Now went gaily to 75 mm. Hg but still felt a little pain. However, she went a long way in 30 minutes, considering her apprehension about decompression and the labour, when decompression was first administered.

16.25 This patient was completely transformed and enjoyed much relief. She worked decompression very well—went to 75 mm. Hg and tended to keep it at 40 for a long time (3 minutes in all).

16.45 Apparently fully dilated. Removed from bag. F.H.H.—transferred to labour ward.

Decompression caused strong, afterwards regular but rather long drawn out, contractions and definite relief of pain.

17.25 Delivered normally. L.O.A.

Comment This parturient is included here to show the influence of decompression on a multipara who was treated at the same time as the primigravidae of the personal series, but had never heard of decompression.

Of the things I learned two are noteworthy. One is the observation that not infrequently dilatation of the os does not proceed beyond 2 cm., even after several hours of unequivocal labour, but that full dilatation may be reached within an hour from the 2 cm. stage. The second point is not a general one, but pertains to decompression. It is that '270D' has an effect on the birth canal like ripening, for labour is shorter and easier following it. It is very effective if given for a few hours in early doubtful or spurious labour. One's experience has been that at this stage contractions are stimulated to become stronger and to last longer, labour being effectively induced; or the patient goes out of labour for 6 to 72 hours but then has a dramatically quick, easy dilatation of the cervix. The second feature is mentioned because, whereas it caused great disappointment at first, it led directly to the much more fundamental observation on foetal response to decompression.

Labour Phases

In order to assess the first stage of labour, it is convenient to think of four phases.

ALPHA This phase lasts about two weeks. During this phase the so-called formation of the lower segment takes place.

BETA This phase lasts about two days. Further changes take place in the birth canal. These changes probably are centred around the vaginal vault and may be concerned with the obliteration of the angle existing between the long axes of the vagina and the uterus.

Comment: Little is known with certainty of the morphological rearrangement that occurs during the *alpha* and *beta* phases, but these are important in that they represent the ripening necessary for the onset, and irrevocable progress, of labour. There is no diagnostic means by which one can determine whether the necessary changes have occurred.

GAMMA This phase lasts for about eight hours (in first labours). At its termination the os should be 4 cm. (2 fingers) dilated. Contraction becomes stronger and more frequent and labour is progressive. Labour should not come to a standstill.

DELTA This phase lasts for about six hours (in first labours). At the end of this phase the os is fully dilated.

Comment: In normal circumstances one expects no delay after 4 cm. dilatation of the cervical os. In many cases, however, the delta phase seems to commence as soon as dilatation has exceeded 2 cm.

General Remarks

It is very difficult to predict whether the *gamma* phase has been reached, if all is judged by the quality of the contractions and by vaginal assessment of the state of the cervix and os. Medical personnel usually start taking charge late in the *gamma* or even early in the *delta* phase. However, a patient may be admitted to hospital in the *beta* phase and consequently be in spurious labour. She may appear to start in labour, and then go out of labour. She may then be discharged and be sent home or to an antenatal ward. In cases of postmaturity in whom for some indication (e.g. toxaemia) induction is carried out, one of two things may happen. (1) If labour cannot be started, it seems to be due to the fact that the patient still languishes in the *beta* or perhaps even the *alpha* phase. Preparation of the birth canal is not advanced to the necessary point of ripeness. (2) On the other hand, if labour is induced easily and in fact is short, *beta* has overlapped *gamma*; conditions for the latter are overripe, and a relatively minor stimulus is required to plunge the woman into labour.

A. It was found in pregnancy beyond 270 days that a few runs of decompression for an hour each—approximately with 40 mm. Hg decompression operating for alternate minutes—result in easy, short labours. Success here was greatest when this type of decompression was done near to the time of the onset of spontaneous labour. Thus, if the woman has been admitted in an early *beta* phase, decompression for a few hours may result in the cessation of labour. After a period varying from six hours to three days the patient comes into labour and characteristically delivers within three or four or five hours.

During 1961 patients were encouraged to drop the pressure by 70 or 80 mm. Hg in all types of pregnancy decompression. The need for this is still *sub judice*.

Note: If the parturient appears not to be in progressive labour, stop decompression after a few hours and discharge her from the labour ward if not unequivocally in labour. This plan suggests that decompression should not be withheld until a patient is clearly in the *gamma* phase.

B. The best progress may be expected with parturients in the *gamma* phase. Usually contractions occur at least every four minutes and last over 30 seconds.

C. Persistent occipitoposterior positions are associated with delay in the first stage of labour, although decompression seems to reduce the time in a pronounced manner.

Note: Decompression stimulates uterine action. The occipitoposterior head will descend further into the pelvis, but in some manner not understood dilatation of the os is complicated—in a few cases as late as when the head has reached the pelvic floor and where 9 cm. dilatation has been easily achieved.

D. The fearful patient of low morale who has weak and irregular contractions will prove a failure, except for the pain relief that decompression will afford for periodic sessions of a few hours.

Note: We do not at present believe that all of these are primarily timid women. Parturients of superlative physique and mental attributes can become consolidated in the commotion of the *beta* phase. Uterine activity now emerges from the subconscious plane, and there is progressive lowering of the pain threshold. It takes a long time to extricate the parturient from this disturbance.

SECOND-STAGE DECOMPRESSION

The question of the manner in which forces are transmitted to and along the foetus during its expulsion was first treated in Röderer's *Elementa* in 1753. One school of thought believes that propulsion results entirely from the rise in the intra-uterine pressure, and the other that so-called axial pressure along its spine drives the foetus out. The second group does not appear clearly to suggest that the force or pressure in the upper segment is greatest, nor is it explained how this can occur. The first group does not succeed in explaining why the foetus should advance rather than be gripped by a uterus which is powerfully embracing 95 per cent of its surface.

It is now submitted that at the initiation of a second-stage contraction the abdominal cavity is divided into three separate compartments which have virtually no intercommunication. As the uterus on erection rises from the spine to press firmly upon the upper abdominal wall, shortening of muscle fibres can occur only by change of shape to the more spherical. The uterus then effectively seals off the

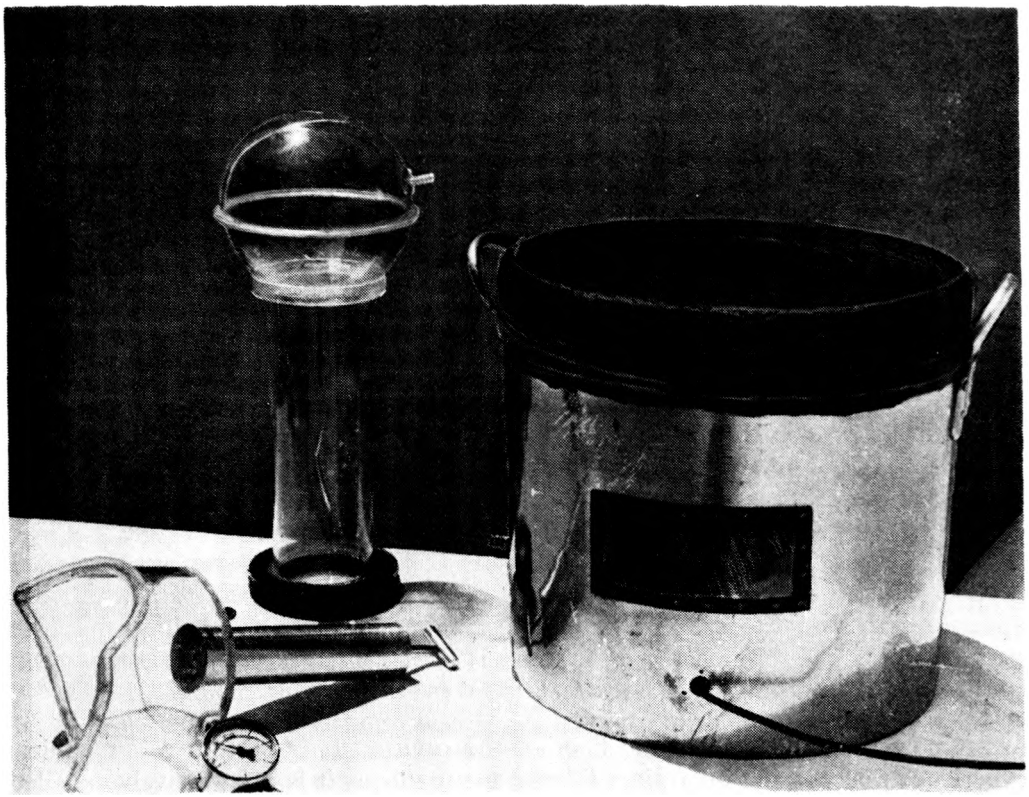


Figure 13
Stages II and III equipment for D

upper abdominal chamber, obliterating the lumen of the large gut and any small intestine that may coil in that region. The next compartment is of course that which is fully occupied by the uterus, and the remainder of the cavity is the third compartment.

When a contraction occurs the uterus pushes the abdominal wall a little forward; pain is felt and the wall is held tight. Then the secondary powers come into play with a moderate inspiration, closing of the glottis, and a bringing down of the diaphragm in strong effort. During the pressure against the closed glottis the abdominal musculature is powerfully contracted. When the upper chamber is isolated, it takes the full force of the descending diaphragm which presses down the available viscera upon the fundus of the uterus.

This pressure upon the top of the uterus can be powerful. The uterine wall itself is meanwhile rigid from contraction. Thus the suprafundal force moves uterus and contents downward. The os is fully open and the rigid foetus directs the presenting part towards the levator sling which offers a gap in the pelvic floor. As the foetus is by slow degrees pushed out of the abdominal cavity through the pelvic floor, the uterine wall shortens *pari passu* to embrace the smaller volume of

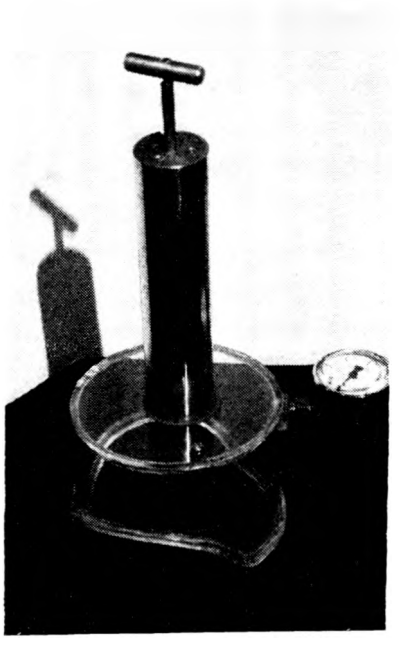


Figure 14
Gasyd for Stages II and III

foetus now remaining above the floor of the cavity. It is of course our clinical experience that the fundus in fact follows the foetus as it is progressively expelled from the abdomen. The uterus is not itself driven through the levator gap for the following reason.

It would take more than the distal empty part of the vagina to dilate a normal pelvic floor aperture. When the head is crowning, the direction of uterine pull on the lower vagina is still away from the aperture, forcing it inwards over the presenting part which itself is being pushed outwards in the opposite direction. *Post partum* the uterus must be of sufficient size to seal the lowermost abdominal cavity from the upper to allow a pressure gradient. If smaller, there would be no sealing and the uterus would be everywhere surrounded by uniform pressure. In view of the relative magnitudes of these structures, it becomes absurd to contemplate the descent through a normal postpartum aperture of a uterus large enough to act as seal and mobile enough to be a piston.

Therefore, we may conclude that the uterus cannot by itself expel the foetus and that axial pressure which is dependent on diaphragmatic descent and can occur only in the presence of uterine contraction, is responsible for propulsion through the levator gap.

Aid in the second stage was only a minor challenge at the time when we were fully engaged on decompression problems in the first stage. Anyone sitting on a lavatory seat will readily appreciate how a seal around the pelvic outlet, i.e. the perineum, could be obtained by adducting the thighs. Posteriorly the buttocks present

a substantial cushion of soft tissue and in front of this the thighs are even more satisfactory for the purpose. The matter was immediately put to the test by sitting on the circular rubber-bonded vent of a decompression chamber constructed for admitting the pregnant abdomen in the semi-prone position. The chamber was about 8 gallons in volume and was housed upon a tilting table. With the table in the horizontal position it was easy to sit with bare skin and legs adducted on the vent and to apply suction to the chamber. An effective seal was readily experienced, and pressure readings of 100 to 150 mm. Hg below the atmospheric were obtained. The rubber surface was plane, and a good seal was secured only when the bare skin was applied. Even thin silk panties impaired the seal.

A large aluminium pot was then purchased and a kidney-shaped piece, with axes measuring 9 by 7 inches (22.8 by 17.7 cm.), cut out of the lid. The lower surface was bonded with sponge rubber 1 inch thick so as to provide an air seal between the rim of the bucket and the lid. The whole of the upper surface was bonded with a flat layer of sponge rubber 1 inch (2.5 cm.) thick. It is on this plane surface of rubber that the parturient sits.

The chamber was mounted on a low wooden stand to raise it to a convenient height for comfortable sitting. A brass nipple for fitting rubber tubing was screwed into the side of the chamber, and a viewing window and a small internal electric lamp were added. The suction pump used was the Edwards RB4, which is a roughing pump with a capacity of 70 litres per minute.

The equipment thus consisted of a chamber with a loose lid, incorporating the requisite rubber for sealing purposes, a suction pump and rubber tubing.

Second Stage: Application

There is no doubt that decompression has some effect in the second stage of labour. While speed and violence in the passage of the foetal head through the lower birth canal is to be avoided, the more obvious effects of suction are as follows:

1. The difference in pressure provides a force that can replace the secondary powers. If the parturient bears down as well, descent of the head will take less time. She need not bear down, however, and will accordingly save energy.
2. The uterus does not have to work, because decompression can be employed even between contractions. Normally both primary (uterine) and secondary (bearing down) powers are required for expulsion of the foetus, the former to seal off the supra-uterine compartment of the abdomen and the latter to create the pressure head. Decompression can yield even more pressure gradient than is normally required and will complete the second stage without uterine action if desired. This may prevent 'exhaustion' (fatigue) in certain complicated cases, and so postpartum haemorrhage.
3. The resistance of the pelvic floor is reduced by a pressure of the order of 100 mm. Hg (2 pounds per square inch); the foetal head, therefore, is used less as a battering ram than in normal labour. Our calculations show that a force of under 8 pounds (3.6 Kg.) is required, instead of about 22 pounds (10 Kg.).

A structural effect produced by the pressure head is to open the pelvic floor aperture more widely and consequently to pull it lower. In this connection it is significant that in the normal expulsive stage the foetal trunk is used for the transmission of axial pressure. Thus, in so far as nothing but the foetus is used as a battering ram, its head takes the full force. On the other hand, with decompression the other pelvic viscera and their initial high pressure contribute as well toward the forcing down of the pelvic diaphragm. For this reason there is a modicum of work not required to be done in the birth canal itself.

Conclusion

One may say that decompression in the second stage is much like forceps extraction, the difference being in its favour and based on a reduction of resistance or friction to the foetal head: (*a*) less work has to be done in the birth canal itself, and (*b*) contractions not being necessary, placental circulation is not interfered with. Thus this foetal hazard is absent.

Note: Looking toward the ideal, one is struck by the fact that there is no lack of energy. Whereas the sources are (*a*) primary and secondary powers, and (*b*) the pressure potential of decompression, the first two do not even have to be called into service when suction is employed. The art here would be to drive the head through the pelvis in the shortest possible time, using the smallest force required for the purpose. The advantage that is being exploited, however, is the total omission of interference with the placental circulation. Our experience has shown that the increase of suction beyond 100 mm. Hg causes oedema of the vulva. If there is justification for using pressures beyond this, 150 mm. Hg should not be exceeded. Patience is required where the fit between head and pelvis is tight, but at the recommended pressures the work is halved if suction is applied during contractions. Patients have shown signs of appreciating this aid to the expulsive forces. In the presence of relative disproportion, unaided forces are most efficient for crowning.

THIRD-STAGE DECOMPRESSION

Expulsion of the placenta is effected by outlet suction precisely as in the second stage.

The policy for the third stage was to apply suction after separation of the placenta. No delay was involved except in complicated cases, when it was known that there was an adherent or unseparated placenta. There was no need to touch the abdomen, but as soon as the signs of placental separation were present, the parturient got up and sat on the bucket. Getting right out of bed at this stage of labour is objectionable, and a high wooden stand was improvised to allow the patient to sit at such a height that her feet rested on the bed instead of the floor.

A seal was readily obtained if the patient sat well back or erect, but it is possible to have some difficulty in effecting a partial vacuum in the chamber. It was found necessary to drop the pressure by 100 to 150 mm. Hg and to employ this level of

decompression in short bursts, each lasting about five seconds at peak pressure. Characteristically, a separated placenta dropped into the bucket in twenty to thirty seconds.

In recent clinical and other trials a perspex cylinder—as shown in the accompanying illustration—was used. It was perfectly mobile, in the sense that it could be applied to the perineum with the patient in the dorsal position and the thighs flexed and abducted. Thus the cylinder could be brought to the patient wherever she was. Experiments are also in progress with a cup and pump, the latter comprising a hand operated piston and cylinder. This modification is to serve remote areas where a power operated pump cannot be used.

Clinical Results

The most striking observation was, as has been stated, that expulsion of the placenta occurred in the majority of cases within a few seconds of the beginning of decompression. The impression gained was that the placenta had been forcibly impelled through the vagina. In each case the cord was clamped with artery forceps, which rested on the floor of the chamber, and in some cases the cord could be seen uncoiling out of the vagina as soon as decompression was applied and just prior to the expulsion of the placenta.

The second phenomenon observed was the postpartum hardness of the uterus. This was present in all but one complicated case. Palpated through the abdominal wall, the uterus could be encompassed in the hand and felt harder than anything previously observed *post partum*: it felt like a cricket ball, though a little larger, and gave the impression of being stonily hard.

Corresponding with the consistency of the uterus, there was a conspicuous lack of bleeding. (There was one exception to this, in which a patient, with other complications, lost forty ounces of blood one hour after delivery.) This itemized case apart, in practice the uterus was found invariably to be firmly contracted *post partum*, and there was no tendency to bleed. There is every indication that, *post partum*, ergometrine is unnecessary, although in the original series ergot was withheld in only 4 cases in which personal observation was possible.

With a view to publishing our observations, we have repeated the clinical work first done a few years ago. The early observations on both second and third stages have been confirmed.

In the most recent trials a vulval cup with hand operated pump (by us called Gasyd) has been tested critically. The simplicity of design and operation are reflected in the illustration. Better control of suction is possible with this device, and manipulation so easy that the operator can work even at arm's length and without any assistance. Birth of the placenta is accomplished with dramatic ease and the use of oxytocics has been found to be unnecessary.

The accompanying table contains the relevant details of each of the first 30 cases in which decompression was employed. All have been included in order that others may profit from our experience.

FINDINGS ON FIRST 30 CASES OF DELIVERY WITH DECOMPRESSION*

Case	Parity	Age	Time Before Use of Decompression Chamber, Min.	Time on Decompression Chamber, Min.	Total Duration of 3rd Stage, Min.	Blood Loss Before Use of Decompression Chamber, Oz.	Blood Loss During and After Use of Decompression Chamber, Oz.	Total Blood Loss, Oz.	Comment
1	4	30	4	3	12	—	12	—	Decompression had no effect—50 to 70 mm. Hg only
2	0	16	2	1	3	—	12	12	Oedema of labia minora
3	0	19	3	1	4	—	2	2	Admitted with retained twin
4	0	19	2	1½	6	—	6	6	Decompression 1½ min. only (bad air seal)
5	0	16	7	—	—	—	—	—	Placenta fell into chamber immediately, before decompression was started
6	0	35	120	—	—	—	—	—	Several minutes on chamber; no result constriction ring present which required deep anesthesia and amyl nitrite
7	1	18	6	½	6½	9	6	15	9 oz. loss before use of decompression chamber
8	0	20	10	½	10½	2	4	6	
9	0	22	10	2	12	2	8	10	
10	0	21	20	10	40	4	10	14	Placenta removed by Crede's manoeuvre
11	5	27	5	½	5½	14	2	16	14 oz. loss before use of decompression chamber (1 min.)
12	1	23	5	½	5½	4	3	7	Piece of membrane removed from introitus; ergot not used
13	2	32	2	½	2½	—	8	8	Ergot not used
14	6	33	3	5	8	4	35	39	History of previous postpartum haemorrhage
15	5	35	10	4	14	8	3	11	Ergot not used; history of postpartum haemorrhage
16	0	—	3	2	5	3	8	11	Ergot not used
17	0	—	5	2	7	2	4	6	
18	1	24	19	1	20	3	2	5	
19	0	18	3	1	4	3	10	13	
20	3	25	3	½	3½	1	2	3	
21	1	23	2	½	2½	—	16	16	N.B. 16 oz. in 2½ min.
22	0	21	10	1	11	7	2	9	
23	1	20	4	1	5	1	2	3	
24	6	33	(5 (4	10½)	20	—	2	2	Twin pregnancy; partial Brandt Andrew's
25	0	18	4	10	14	—	14	14	Obvious delay in separation of placenta
26	6	38	2	8	10	4	30	34	40 oz. lost 1 hour after delivery. Unequivocal diagnosis of concealed accidental haemorrhage had been made.
27	2	24	5	1	6	—	2	2	
28	3	27	5	1	6	—	2	2	Antepartum haemorrhage during pregnancy
29	0	22	3	1	4	1	4	5	
30	1	25	3	7	10	2	14	16	Marked vulval haematomas

*Cases 1, 5 and 6 should be omitted from the analysis. In Cases 2, 10, 14, 24, 26 and 30 there were complications after decompression. In the remaining 21 cases the treatment was successful. The patient in Case 3 was admitted with a retained twin; in Case 28 antepartum haemorrhage had occurred during pregnancy, and in Case 15 postpartum haemorrhage had occurred in previous labours.

SECOND AND THIRD STAGES

Summary and Conclusions

The equipment and method used in making suction around the pelvic outlet are presented. Suction was carried out in the second and third stages of labour. A table of all of the first 30 third-stage cases is included. An explanation of how the mechanism of decompression operates is offered. Experimental data correspond with the hypothesis, and in the author's opinion they substantiate the mechanism afore-mentioned.

The conclusions to be drawn from the investigations touch both basic and applied considerations. They are as follows:

1. Outlet decompression will deliver the placenta and the foetal head through the pelvis without the aid of bearing down or manual pressure. The placenta must not be adherent, and the foetal head must fit tightly in the brim or the cavity of the pelvis.
2. Suction is effective because the pelvis is separated from the upper part of the abdomen by the head or third-stage uterus effectively sealing the entrance to the pelvis.
3. The postpartum uterus is exceptionally firm and is apparently almost completely exsanguinated by decompression. It is not yet known with certainty whether the atonic postpartum uterus can be treated effectively by decompression.
4. Decompression in the second stage should be done apart from contractions, in order to avoid impairment of placental circulation.
5. Less work has to be done within the birth canal during expulsion, because with decompression other pelvic viscera also contribute to the forcing open of the pelvic diaphragm. The foetal head accordingly does not take the full force of the pressure gradient as is the case when expulsion results from longitudinal axial pressure. The force expelling the foetus naturally is of the order of 22 pounds (10 Kg.) but with decompression it is less than 8 pounds (3.6 Kg.).
6. The art that seems to be attainable in the second stage is to drive the foetal head through the pelvis in the shortest possible time, while using the minimum force required for the purpose.

RELIEF OF BACKACHE

In the last subsection of Part I entitled "Pain Relief in Labour—A New Reflex" (p. 15) it was indicated that the demonstration of the reflex led to therapeutic trials with decompression on women suffering from primary dysmenorrhoea. An explanation of the manner in which backache is relieved was given in this subsection.

Decompression was then tried for its effect on severe dysmenorrhoea. Relief of pain was obtained, but the duration of the former was often unexpectedly long. The following early case illustrates this point.

Miss A.P. aged 20, from the onset of menstruation, was incapacitated by dysmenorrhoea to the extent of being confined to bed for two or three days every month. She had consulted several doctors and had tried numerous forms of treatment without relief.

In January, 1961, she came for decompression. She had two half-hour sessions on the two days before bleeding began, followed by similar treatment on the first two days of the cycle. There was almost complete relief of pain. In February exactly half this amount of treatment, premenstrual, gave the same dramatic result. Since then she has had no further treatment whatsoever, and regards herself as cured.

Arising out of the experience with dysmenorrhoea and the backache of pregnant women, relief of pain in the back was sought by males who were incapacitated. The first applicants for decompression were young medical men, characteristically ones with athletic interests and of robust build. The patients were impressed by the results and their gratitude was touching! Soon, sufferers came from a radius of 200 miles and occupied much of our time.

Backache

Technique

Abdominal decompression was carried out, by decreasing pressure within the rigid spacer, by 100 mm. to 150 mm. Hg. The patient controls the valve for her- or himself, reaching the desired pressure in a few seconds. This pressure is maintained for about 10 seconds, the valve is released, and suction is re-applied within a minute. Treatment is continued for at least half an hour. In severe cases four daily treatments are given, sometimes more.

Results

A total of 34 patients, 20 men and 14 women, had decompression for backache. The results were as follows:

Excellent	24 per cent
Good	60 per cent
Satisfactory	12 per cent
Unsuccessful	3 per cent

The "excellent" group has not required treatment again and it is not yet possible to decide whether periodic decompression, say every six months, is advisable.

The "good" group had relief following decompression but came for 7 to 10 sessions, the results not being quite as dramatic as in the former group.

The "satisfactory" group had temporary relief but perseverance and repeated treatments were necessary.

One unsuccessful case had proved osteoarthritis of the spine and no improvement was expected. He had decompression for 2 sessions.

Sessions given to an individual averaged 5, with a range of 1 to 10. The decompression level was 100 mm. Hg or more, below atmospheric pressure.

Some of the patients had a history of chronic backache, but they usually presented with an acute exacerbation. Others had acute pain in the back which they had suffered previously, but repeatedly.

Decompression, in our antenatal cases, has given relief to many who, near their term, have been aware of fatigue or an ache in the spinal muscles.

DYSMENORRHOEA

The method of relief employed is similar to that described for simple backache. The patient, however, is sometimes not in robust health at this time, and has to proceed more gently. In a few cases a feeling of faintness enforced slow progress, particularly when the subject was unfamiliar with the effect of decompression on respiration.

Results

There were 93 patients in all. They were composed of 2 groups, 67 school girls in the second decade and 26 adults mainly in the third decade of life. The response was as follows:

	Girls	*Women*
Excellent	13–19 per cent	7–27 per cent
Good	33–49 per cent	15–58 per cent
Satisfactory	17–25 per cent	2–7 per cent
Unsuccessful	4–6 per cent	2–7 per cent
Total	67	26

	Combined Series (Girls and Women)	
Excellent	20	22 per cent
Good	48	52 per cent
Satisfactory	19	20 per cent
Unsuccessful	6	6 per cent
Total	93	

Two girls were discarded: one left school and no follow-up was possible, the other fainted at the start of treatment.

Seven school girls treated last year came for decompression when the second series was done over a period of months this year. Of these, 3 gave an excellent result, 2 good and 2 unsuccessful. The 2 women who were failures were nurses subjected to the earliest trials when treatment was entrusted to a ward sister. The above figures are given for the sake of simple guidance to the reader, but we feel bound to say that it is our experience that there are only two variables in the prognosis. Any patient with severe primary dysmenorrhoea who is incapacitated will give an excellent or good result: those with only malaise and discomfort will find relief, although this group will show an incidence of a few equivocal and poor responses. Of cases that could not be regarded as serious clinical problems the

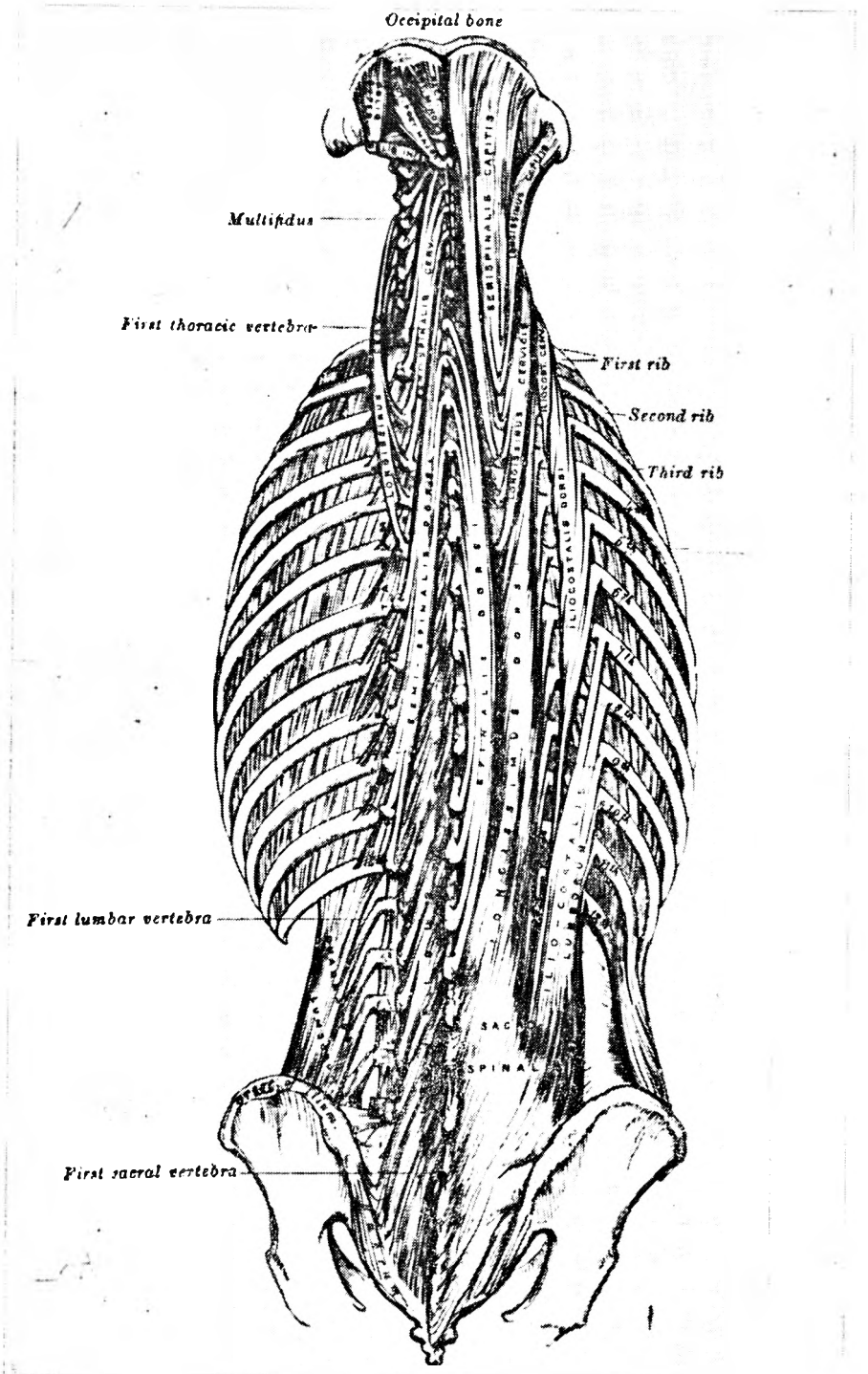


Figure 15
Sacrospinalis muscle

school girls presented a higher proportion than the women. Where patients are seriously inconvenienced by primary dysmenorrhoea in schools, factories and institutions, they can rely on substantial relief from abdominal decompression.

We are acquainted with the results of 20 cases of dysmenorrhoea treated by colleagues in private practice. There is also a success story in a series of pupil midwives. The latter have been diffident about consulting us, but made their own arrangements with a staff nurse to give them abdominal decompression in our clinic. In this spirit, Bantu midwives have also made liberal use of our apparatus installed at Western Native Township for applying decompression in pregnancy.

This spontaneous and unsolicited use of the device lies outside our control, but it testifies to the acceptance of the method by those within our orbit. Similar reports have reached us and gynaecologists in other countries where, it appears, midwives have actually monopolized apparatus provided for parturients.

Discussion

It is probable that the development of painful muscular tension depends on volleys of impulses from the receptor end-organs, released over a period to be measured in days. The suggestion is based on the observation that interruption of this tonic state is followed by relief lasting for days or even weeks. Furthermore, where treatment has been started on the day preceding menstruation, the results have been best. If the rate of discharge of the receptor is low it may take days for the development of sufficient tension to cause pain, but no pain may be present.

Measurement of the state of the muscle is not satisfactory by the electrical method, but it is likely that temperature readings with a thermocouple will give the necessary information. The tetanic spasm of simple backache is to be differentiated from that present in dysmenorrhoea, for it is related to an irritating local focus or to postural aberrations.

Our attitude to the patient with menstrual pain is to refer to the future only at the end of the first session. Even if she enjoys complete relief, she is advised to report for two more treatments on the ensuing successive days, and for three more periods. After this she may be allowed to go without treatment for a few months for the sake of assessment. One expects some patients to require therapy for 12 to 18 months, but thereafter to use decompression occasionally, when indicated.

Simple backache is more resistant to therapy and perseverance is often necessary. Follow-up treatment is likely to be required at intervals of six months. The immediate effects are pleasant and give a sense of suppleness of the trunk.

In so far as there may be criticism of the idea that spasm of the sacrospinalis occurs and in fact causes the pain under discussion, it can be stated with confidence that the objections are not supported by proof. We have made our own observations with electromyographic equipment and have studied the literature but at present are not dissuaded from our viewpoint.

Our own standpoint is that the following phenomena exist: pain in the back occurs during labour and dysmenorrhoea, there is a reflex from the uterus to the sacrospinalis as effector, and the stretch of the spine muscles by abdominal decompression abolishes the pain. It is for others to determine all the underlying processes.

One is only willing to suggest that the electrical method is probably not the best approach to an examination of spasm, tonus, tetanus, or increased tension in the postural muscles. Better understanding by the chemists of the Marsh (relaxing) factor may come in a decade or two, and an important facet will have been illuminated.

What is required now is some explanation of why pain is present in this tissue under intrinsic conditions that appear to be similar to the states giving rise to no pain. This contractile tissue is but one in which the mechanism of pain eludes the investigator.

MISCELLANEOUS

Cardiac Disease

In view of the fact that facilitation of labour and pain relief are obtained in the first stage, it was felt that parturients with cardiac disease should have decompression. A few cases have been treated as an experiment. Although no series can be offered, there is reason to expect that decompression will be a valuable aid, for patients with unfavourable conditions such as previous pneumonectomy showed no ill effects. There can be little doubt that second-stage decompression is strongly indicated in the occasional case of delay, and in the third stage the method should be routine.

The cases of cardiac disease that merit special attention in labour are not common. Types II+, III and IV are those likely to benefit from a reduction of the stress of labour. For those who insist upon the highest standards of accouchement, the aid to be derived from decompression in all three stages of labour complicated by cardiac disease is sure to make this addition to our armamentarium indispensable.

Pre-eclamptic Toxaemia

Because blood pressure often drops during pregnancy decompression, and placental function is believed to improve with this treatment, the possibility of using decompression to treat toxaemias has been considered since 1958. At present daily decompression for half an hour is being given to women admitted because of toxaemia. No doubt much more time than this should be devoted to severe and moderately severe cases which are the only ones likely to provide conclusive results. It will take two years to collect a useful sample and one hopes that other clinics will regard trials as worth their while. The promise thus far makes a search into this possible control of toxaemia a matter of considerable importance. It is significant on the prophylactic side that in 85 long term decompression women no pre-eclampsia has developed. Our clinic incidence is 7.6 per cent. Our first series of White short term decompression patients showed, on analysis, that there were only 2 cases of pre-eclampsia out of 300. Both were mild. I make this 11.5 times the standard error of the difference in our antenatal clinic.

Our resources have been organized for a therapeutic study with some rigidity. The improvement in placental circulation that appears to follow daily decompression is of sufficient moment to stimulate investigation. Furthermore, attention to this particular point will test the value of decompression fundamentally.

PART THREE TECHNOLOGICAL

LABORATORY AND ACADEMIC USEFULNESS

IN ESTIMATING the value of decompression as a research tool there is the danger of ascribing to it increased knowledge which has been derived mainly from greater care of the expectant and parturient woman. This section does not aim at a demonstration of the uses or value of decompression: perhaps it is valid to set out what its influence on obstetrics has been, even if, without it, modest attention to the phenomena of the first stage could have brought a rich harvest. The contribution—*sui generis*—opens new avenues in obstetrics and possibly may give an evolutionary new direction to research.

Uterine Potentials

This is the predominant item in this section, because the specific problem of

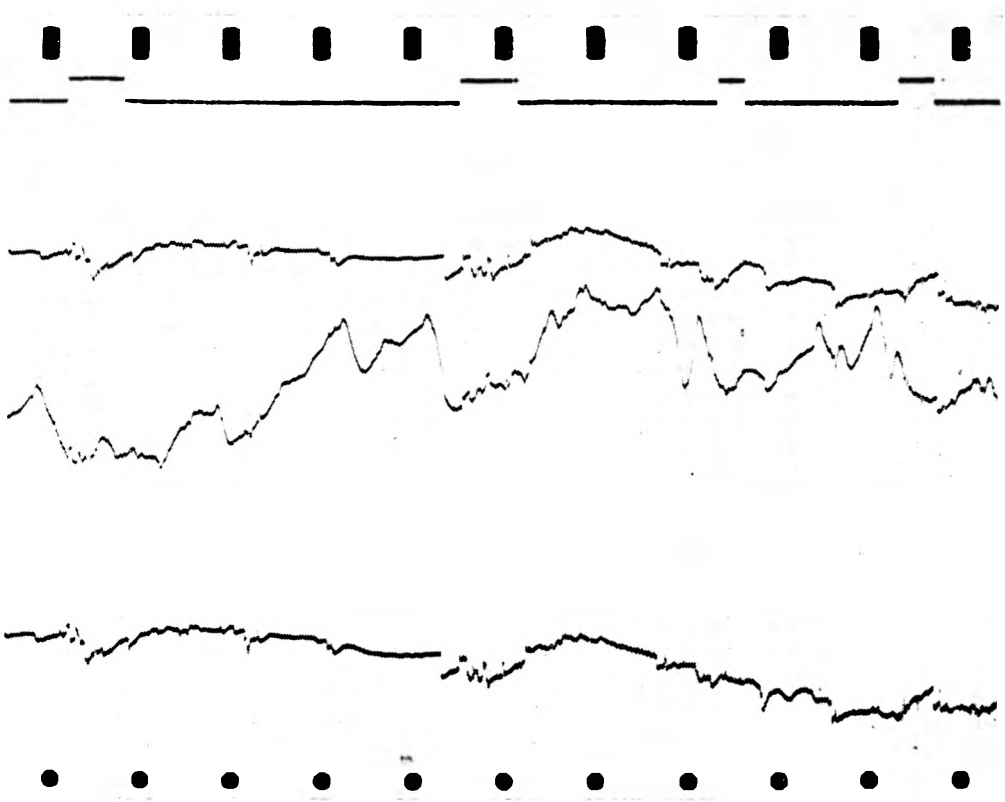


Figure 16
Comparison of rectus and myometrial electrical impulses in labour

electrical activity in the uterine and abdominal musculature was solved by means of decompression. This problem was referred to at the beginning of Part I, and it was a step forward, years later, when it became obvious that the abdominal bulge caused by decompression was substantially similar to what happened during a strong uterine

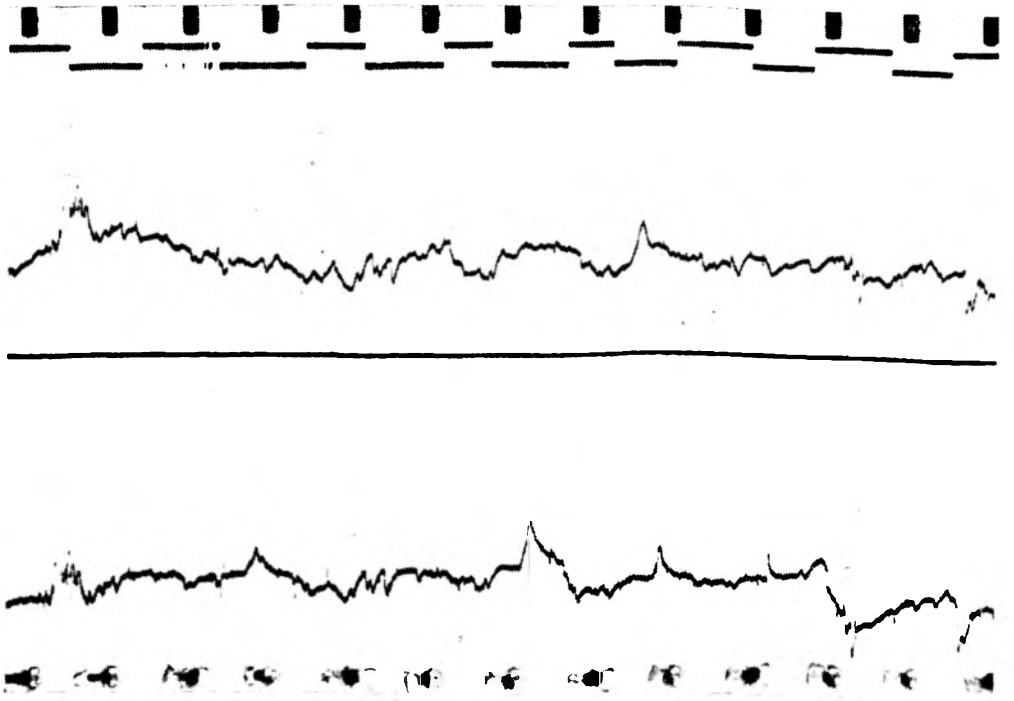


Figure 17
Intra-uterine potentials in labour

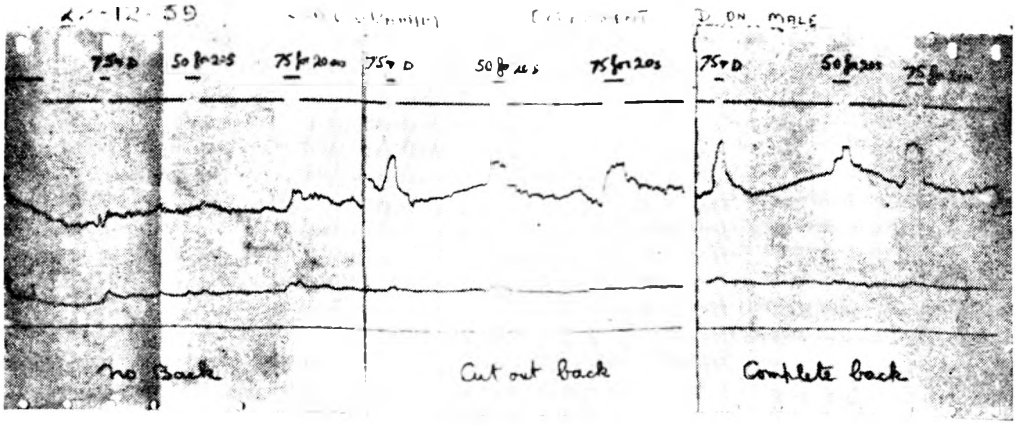


Figure 18
Differential electrical responses in the rectus using the plastic suit (male) (i) without a back-plate, (ii) with a back-plate containing a window and (iii) with a windowless back-plate. Abdominal decompression is partial in (i) but complete in (ii) and (iii).

contraction. Electric recordings obtained from abdominal wall electrodes showed big potentials (Fig. 19). In the early series partial decompression was applied with a rounded dome, but complete decompression was later done in the suit with and without a backing-plate which itself was used with and without a window (Fig. 19, 18).

It was accordingly found that the stretch of the recti during labour contractions gave a potential. Recordings were made during a contraction in the second part of which decompression was applied, and records during decompression with a superimposed contraction are also shown in Fig. 3. It seemed as though the electric potentials obtained from electrodes on the abdominal wall were mainly from rectus muscle stretch. The question remaining was whether the uterus made a contribution to this external potential. If the answer was in the affirmative, could the uterine moiety be interpreted usefully for, if it bore no relationship to the rectus tracing, mechanical tocography should replace electrical?

The main point, however, is that the primary effect of uterine action which is what tocography sets out to study—is a change of uterine shape from ellipsoid towards spherical. This can occur only by pushing away towards the front and sides the muscular abdominal wall overlying it. The latter is readily accessible to observation, and electrodes or strain gauges may be placed upon it. But what is it that one wishes to study?

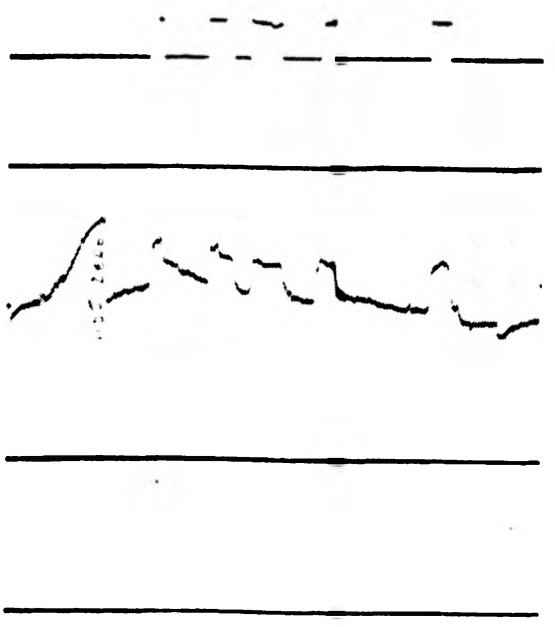


Figure 19
Partial D with a rounded dome

There is clearly some merit in studying all facets of uterine action or the entire range of the forces of labour. Thus electric responses from the myometrium are best obtained by using internal electrodes. Pressure reading also would be of the utmost value and would similarly be measured by internal mechanical tocography. The only activity that can be recorded from the surface of the abdominal wall is the stress and strain on its musculature resulting from uterine change of shape. Quite emphatically the electric response in the rectus or other muscles to this uterine thrust is all that the record can be relied upon to demonstrate.

No doubt the force of the uterine contraction can be assessed from the electric potentials measured in the stretched rectus, but the potentials are not so much a direct measure of uterine electrical activity as its mechanical action. To repeat, both the last variables are best measured internally.

The rectus muscle response is most favourable to labour when its resistance to the uterine thrust is minimal. It will accordingly stretch readily, and the contour of the abdomen anteriorly—but to some degree laterally—will change and become more curved. This will usually be most pronounced in the epigastrium but become progressively less in the direction of the pubes.

There is noquestion that a strain gauge is the instrument to use for the measurement of these changes, not an electrode. There is an electrical field produced by the parietes and no one would wish to record uterine electrical responses as they pass through this field. The strain gauge movements may be recorded through a galvanometer if desired, but this is a technological matter. The significant point is that external tocography should be mechanical and not electrical.

Pain Relief

Decompression produced pain relief. This was quite unexpected, because its earliest known effect of facilitation of labour was accompanied by longer and stronger contractions. Steps were taken to provide analgesia, for the increase in labour efficiency was now regarded as indispensable. In the circumstances the origin and mechanism of uterine pain became the subject of speculation.

It was Graham's antenatal decompression patients who first called attention to the sacrospinalis. They remarked upon the pleasant relief that decompression had produced in their tired backs. This led to the demonstration that electrical impulses were recordable over the sacrospinalis during labour pains. Our reflex was suggested. Stimulation of the receptor area around the lower pole of the uterus and vaginal vault—the polar receptor!—with somatic afferent conductors to cerebellum and thence to sacrospinalis and recti gave potentials of 10 to 50 mv. What is still obscure is the relationship between the presumably incompatible factors underlying painful and painless contraction of the sacrospinalis. At the outset certain phenomena have to be recognized. Pain in the back occurs during labour and dysmenorrhoea, there is a reflex from the uterus to the sacrospinalis (and rectus) as effector, and the stretch of the spinal muscles by abdominal decompression abolishes the pain.

In examining this problem one questions very seriously whether the electrical approach is the best for the study of spasm, tonus, tetanus or increased tension in

the postural muscles. Better understanding of the chemistry of the Marsh (relaxing) factor may come in a decade or two, and an important facet will have been illuminated. What is required, however, is some explanation of why pain is present in this tissue under intrinsic conditions that appear to be similar to the states giving rise to no pain. This contractile tissue is but one in which the mechanism of pain eludes the investigator. The question was referred to in Parts I and II and it was suggested that painful muscular tension might depend on volleys of impulses from the receptor end-organs, released over a period to be measured in days. Rate of discharge of the volleys is probably critical.

The Device of so-called '270 D'

The personal series of labours treated with decompression in 1959 was described in Part II. It provided unexpected failures. Treatment was repeatedly followed by a latent period of uterine inaction, after which progressive labour of very short duration brought the first stage to an end. There was also the occasional phenomenon of hours of clinically irrevocable labour that failed to progress beyond a 2 cm. dilatation of the os, a point from which the second stage could be reached within an hour. Some explanation was called for and will be dealt with below. The previous complication resulted inevitably in the use of '270 D'. This simple device —decompression during the last 10 days of gestation—was followed by longer spells of pregnancy decompression when the danger of premature induction of labour proved to be non-existent. It was easier to accept patients when precise timing was no longer a problem. This variation was in use by the end of 1959 and by the middle of 1960 Samson and Graham's antenatal patients were speaking more freely of their splendid decompression babies. It had been suggested (Heyns, 1958) that the painless contractions of pregnancy, but preferably those associated with decompression, insured improved circulation of blood and CO_2—O_2 exchange in the placenta and in consequence a proposal was made to provide daily decompression during the last trimester. During 1960 difficulties connected with a shortage of equipment were resolutely overcome and trials of prolonged decompression in pregnancy undertaken. Thus '270 D' developed into what is by far the most promising aspect of the entire project, that of the improvement of foetal oxygenation during pregnancy.

Morphology and Mechanics

When decompression replaced muscle relaxants and the duration of labour was often reduced, the idea of change of shape of the uterus was developed more carefully. It became clear that the intervillous space was subject to the same influences (Heyns, 1958) and a good deal later the promotion of this mechanical change by decompression was appreciated. There was delay in reaching a firm conclusion until intra-amniotic pressures were measured in 1960. Improved blood circulation and oxygenation in the placenta were thought to result and this would be the basis for the superiority of the new-born babies. Infant development could be expected to have gained in momentum, thus laying the foundation for subsequent improvement in physique and intelligence.

The restraining influence of decompression upon tense abdominal muscles allowed the contracting and anteriorly moving uterus to function more freely and efficiently. This provided a more satisfactory explanation than the Grantly Dick Read concept of relaxation, particularly in respect of the conclusion "tense mind, tense cervix". It was not the cervix that was tense during apprehension but the skeletal musculature, and thus the abdominal wall. This restricted the requisite change of the flattened uterus to a shape with an eccentricity nearer to 1 (unity). Resistant abdominal muscles cause the uterus to dissipate its energy which is essentially required for isotonic contractions culminating in a rearrangement of the myometrium into a thick upper and thin lower segment.

It cannot be denied that there was gross ignorance about the mechanics of the abdominal cavity, and this even in respect of all who operated in the abdomen. Eminent writers were guilty of the belief that the internal pressure was that of a vacuum. Anaesthetists thought that the Trendelenburg position allowed the intestine to drop away from the pelvis before incision. The reason for these *solecisms* no doubt arose from the rejection of the pressures measured in the cavity, values that might be questioned but not ignored. The simple device of decompressing the cavity not only provided specific applications but *sui generis* was the hard core of abdominal mechanics: it compelled one to give attention to the subject, to explain observations, but at the same time was an important part of the armamentarium that could produce the effects.

The picture that existed of the morphology and mechanics of the second stage of labour is illustrated by the fact that the expulsion of the foetus could not be explained after 200 years, and that the Malmstrom cup reached its present development after trials spread over 120 years. One wonders what delusions can have such disastrous effects but it must be remembered that the obstetric forceps was invented after millennia, and 200 years after such an elegant instrument as the violin.

Observations with suction upon the perineum or pelvic outlet have drawn attention to the forces acting upon both the birth canal and the foetal head. These factors are accepted as a matter of course, but such complacency must give way to the measures available for protection of the foetal brain.

The effect of decompression on the intervillous space indicates that this device may prove a useful tool in discovering something of the pathogenesis of the toxaemias of pregnancy. Even before the underlying mechanism is understood, its prevention and control may be possible by prescribing decompression during pregnancy. At the outset intensive treatment of a series of severe cases may provide valuable information.

The Onset of Labour

A working hypothesis that attempts to explain the variations in the onset of labour and its progress is required. It is precisely in this respect that the objection may be raised that considerations of this kind need not be traced to abdominal decompression, but the least that can be said by way of justification is that the hypothesis was proposed inside of the field of our experience with the device.

Less specifically, anything that might lead to greater care and attention during

the conduct of the first stage of labour must be recorded on the credit side; moreover, decompression is still concerned with an investigation of the finding that some parturients using it progress so well and others so badly. Calculation shows that theoretically 17 contractions should be sufficient to effect the rearrangement of the uterine muscle that is essential to the first stage. In fact, several cases have reached full dilatation of the os in under 30 contractions. The hypothesis referred to is the one illustrated in the diagram of the "1,000 C".

Uterine Contractions

We have designated the scheme shown in Fig. 20 "1,000 C". All uterine contractions, whether painless or painful, add up to a figure of the order of 1,000. The diagram shows contractions to become more frequent and stronger as pregnancy proceeds until, at term, they become 'labour pains'. At this time they penetrate the pain threshold which varies from patient to patient and it depends on this and the magnitude of the contraction as to when pain is first felt. To the patient and the accoucheur this heralds the onset of labour. The labour may be spurious if the pain threshold is low.

The inset figure shows the final phases of "1,000 C" in more detail. α, β, γ and δ phases are there, but parturition proper occurs at γ and δ. The pain threshold operates so as to cause painful contractions at the onset of labour but pain may first be felt later (see inset). It is improbable that the first stage ever draws to an end without pain except in those unique cases of congenital absence of pain sensibility.

The aspect of this matter that is elusive is why contractions should gain so much power at the end and why they are intermittent. It is possible that an active principle precipitates the onset of labour. Everything points to a slow process. Sensitization of the myometrium by one hormone (oestrogen) to another (oxytocin) is only part of the explanation, for there is substantial evidence that contractile protein is laid down in the myometrium under the influence of oestrogen towards the end of pregnancy. The ageing placenta no doubt initiates the release of oestrogen through its failure to produce progesterone. The actomyosin may be expected to cause a progressive increase in muscular contractility. When overcoming the stretch caused by continued growth of the foetus, the stimulation of muscle activity might be sufficiently great to amplify contractility even to the point of tetanus. Labour would then start, and it is difficult to say whether the phase would be α, β, γ or δ. It could be any of these, but is most likely to be β or γ.

Another consideration is as follows. Myometrial tension increases with shortening. Therefore the upper segment can work or contract more forcibly than throughout pregnancy. This means that contractions can occur more frequently. It is perhaps elementary to think that energy is needed for contractions, that energy has to be replenished, and that fatigue must play a part in providing periodicity. The apparent long duration of spasm in the adductors of molluscs, in the smooth muscle of the human uterus and in some postural muscles has misled us for too long. One must accept that a high tension muscle must obstruct its blood-supply: even in skeletal muscle there is intermittency in all natural actions; and in highly skilled movement a set of muscles alternates rapidly with its antagonist.

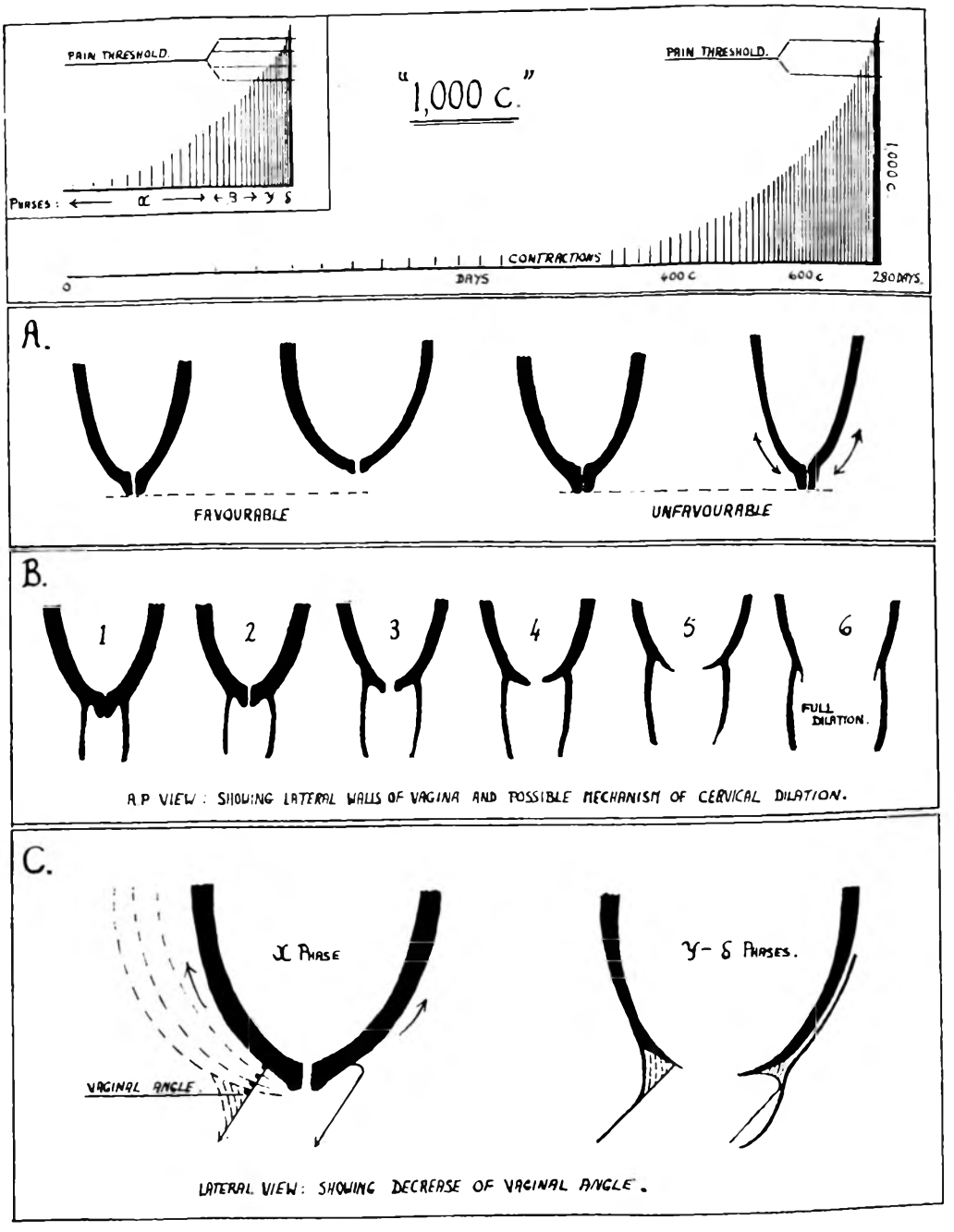


Figure 20

"1000 C" A lower uterine segment
B cervix and vagina
C vaginal angle

Labour Changes at the Utero-Vaginal Junction

The diagrams in Fig. 20 A represent the lower uterine segment. Assuming that the presenting part does not fit particularly well, the tangential forces resulting from muscular shortening can pull the half ellipse upwards into a more spherical shape. The result will be an increase in volume, but this can happen only when the vaginal vault is mobile as in a parous subject. Where the vault is immobile the upward pull will be resisted: if there is no movement the half ellipse will become even flatter with a consequent diminution in volume.

In B an anteroposterior view shows how lower segment, cervix and vagina increase in diameter with a flattening out of the lateral angles. The portio vaginalis of the cervix remains, but its substance is greatly thinned by the increase of its diameter to over 4 inches. It is pushed hard up against the vaginal wall.

C depicts a side view and the angle between the long axes of the vagina and the uterus. Deep manipulation during the second stage does not reveal an angle but at most the curve of the birth canal following the pelvic axis. The first diagram indicates certain preliminary changes in the cervix.

Until the lower segment is sufficiently 'formed' and progressively incorporates the upper cervix in such formation, and the cervix then opens up to reduce to less than 90 degrees the external angle between its wall and that of the vagina, the upper end of the vagina will not begin to open up in following its cervical insertion as the cervix becomes effaced. This is illustrated in the second diagram of C. If the external os has not dilated much, as happens so frequently in primigravidae, the tangential forces now in operation will be added to the internal pressure forces. Therefore, what was the α phase has now passed into the structural state of the γ and δ phases.

This may then be the spatial relationship to the chronological sequence of the α, β, γ and δ phases. It is only a first attempt at an examination of the changes that take place in the birth canal when the vagina is pulled up by the uterus in preparation of the birth canal for the second stage of labour. This presentation of the events occurring during parturition may be rejected. On the other hand, if it indicates a direction for investigation, it is to the credit of decompression that such a stimulus has been received.

APPARATUS

An account of our experience with equipment is given, not for historical reasons nor indeed for vainglory, but specifically to help others. It is doubtful whether in many cases the others wish to be guided. If this is the case, perhaps they can be dissuaded. At the risk of giving offence, it has to be stated for documentary completeness that in several countries modifications have been devised in the hope of gaining *lettres patent*, or for building something of the ego into the machine. This would be trivial if the satisfactory operation of the method were not impaired by such changes. On this point there is the paradox that in many quarters one's instructions and advice are ignored: failure and disappointment have been registered

and one has even been suspected of deliberate fraud. It seems to be a matter of the utmost difficulty to convince even one's own staff on points of technique that have been derived from a superior familiarity with the device.

What follows in this section is our experience with modifications, and it is recorded in the conviction that others may be saved two years of preliminary work when they set out with this knowledge.

The Evolution of Equipment and Methods

Starting from the premiss that a reduction of the pressure upon the abdomen will cause the anterior wall to bulge we might inquire a little more closely into the underlying factors.

Because of what was available, and because of one's preoccupation with the part of the abdomen that bulges when a woman is heavy with child, a cuirass respirator was the type of decompression chamber used in our first experiment. The test was successful, for the soft abdominal wall came forward and girth increased. Thus it was possible artificially to do some of the work normally performed by the uterus when it rises from the spine in its flaccid, flattened state and contracts firmly with increased tension in its wall to a round organ pushing the rectus and other muscles before it. The more resistant these muscles, the more energy does the uterus dissipate to overcome them.

There was, therefore, the promise that the abdominal muscles could be controlled. But even more, their constricting influence was eliminated so that the uterus was virtually set free—in effect brought out on to the table and capable of unrestricted action. So the first decompression chamber received only the enlarged abdomen and operated locally or partially. The abdomen bulged as expected and the recti were stretched.

This form of apparatus was followed by a light perspex dome with a sponge-rubber seal around the rim. The patient sat in a semi-reclining position in a *chaise longue* type of chair and the dome was applied to the abdomen.

It was subsequently appreciated that the flanks were pressed in by the atmosphere and that more of the abdomen should be subjected to decompression. For this purpose a casket with hinged back and front was constructed. It required sealing above and below, and sponge-rubber cushions were bonded to the aluminium. This device was rejected because it required too much exposure of the patient's body, and securing it caused undue disturbance in a sensitive parturient.

A radical change was indicated, and a decision in favour of enclosing the whole body below the arms was firmly made. A bag would be required. Experiment showed that it would be necessary to open and close such a bag, in order to get both patient and a rigid spacer into it. A zip 'lightning' fastener was indicated, and sealing was to be below the axillae. A voluminous bag was made of very thick nylon impregnated with polyvinyl chloride and an ordinary metal zip four feet in length ran down the middle of its front from the upper edge. For effective sealing this hemmed upper edge was controlled by lace up and other devices to let it out and pull it in.

Experimentation with spacers was extensive, inclusion of the lower extremities being usual. Although the aim was to construct something light and mobile and causing a minimum of distress while being adjusted, we made a species of tank respirator at this time to permit experimental observation. The tank had a viewing window and contained the entire body up to the axillae. Sealing was accomplished with flaps of nylon connected by zips. The patient was laid on a stretcher sliding into the tank on rails. It was found with the tank that a drop of pressure by 125 mm. Hg rapidly led to unconsciousness. This was explained as being due to a pooling of blood, mainly in the lower extremities, which had an effect similar to that of the Valsalva experiment. The heart thus beat on an empty ventricle and caused cerebral ischaemia.

It was now clear that the rigid spacer had to be confined to the abdomen, in order to leave the legs exposed to atmospheric pressure. A cage was made of $\frac{3}{8}$ of an inch steel rods welded to make a mesh. It covered the abdomen and had angled lower edges to fit into the sitting angle of the chair. Meanwhile the metal zip could not be made air-tight for pressures approaching 25 mm. Hg below atmospheric with vaseline, paraffin wax, tape or any substance at our disposal. Within a few months an airtight plastic zip became available which could be welded to the plastic material of which the suit was made.

The space left between the chest and the spacer above admitted a process of the suit when pressure differences were high. This impaired the seal. Unless sweating was free, the posterior edge of the suit descended and caused trouble. In order to dispense with the zip a large bag was made.

This voluminous suit was constructed by welding together two pieces of p.v.c. plastic material 50 inches above and tapering down to an 18 inch base below. The length was 66 inches. The top girth of nearly 9 feet made it possible for the spacer to be placed in the suit from above and for the patient to be manoeuvred into place as well.

Plastic straps were welded back and front on either side of the centre (neck position). These are tied over the shoulders once the patient is in position. The redundant side wings are folded over to reach the side of the chest underneath the armpits. A belt of the same material was tied tightly around the chest so as to enclose the folded side pieces. The seal obtained was superior to anything previously experienced.

At present, the posterior bib referred to in Part II is being used regularly. The improvement has been so pronounced that one is reluctant to make use of the older model for anything where pressures of more than 50 mm. Hg below atmospheric are indicated. For backache therapy at least 8/1000 inch nylon is needed.

The chair was exceptionally comfortable to sit in. The foetus was very nearly vertical as shown on X-ray films, the angles of the surface of the chair being 140 degrees below the knees, 115 degrees below the buttocks, and a short upright piece behind the head.

During decompression with this plastic bag and cage, atmospheric pressure forced the unprotected back forwards. It will be readily understood that the body above and below the cage had atmospheric pressure all around, and therefore these

parts were not pushed forward or in any other direction. On the other hand, the part of the trunk covered by the cage had reduced pressure in front and at the sides and was accordingly pushed forward, into the cage, by the higher atmospheric pressure operating against the back of the suit. As only this caged part of the body was so pushed, a marked lordosis developed; but, in addition, the chest was pushed so hard against the upper edge of the cage as to cause discomfort at high decompression.

Partly to counteract this disadvantage, but mainly to provide the complete decompression which the cage was obviously not affording, a backing-plate was tried. A bucket-seat mould was made of plaster of Paris using a pregnant girl as model. From this a fibre-glass seat was made which, although hard and rigid, was remarkably comfortable to sit in. This backing-plate, then, had a groove on either side to receive the edges of the cage as it existed before, except that *now* it was made of fibre-glass. Thus the spacer completely encircled the trunk.

The procedure was to lay the plastic bag or suit open on the chair, put the backing-plate in position, help the patient on to the seat, place the anterior portion of the spacer within the receiving grooves and zip the suit closed. The upper border of the suit reached to the top of the breasts, fitted well and provided the seal. The backing-plate could be used without a window or with a small or large window. The chest was now no longer pushed forward against the anterior edge of the spacer, but the whole of the abdomen was surrounded by reduced pressure during operations, so much so that, for the first time, measurement showed intra-abdominal pressures to fall below atmospheric.

It has to be stated that a further modification had been introduced by this time for, whereas the metal cage only reached to well below the ensiform cartilage and did not contain the breasts, an extension was made to include the lower thorax. Thus the breasts were within the spacer and the requisite expansion of the rib cage during decompression was not counteracted by atmospheric pressure. Inferiorly, the spacer extended so as usually to cover the vulva.

As the suit has to be airtight, it must be made of plastic material, the thinner the gauge the better. Gauge 6/1000 inch usually bursts at pressures 150 mm. Hg below atmospheric.

The above endeavours were directed to the control of the first stage of labour which is the great ordeal because of its long duration and intermittent flagellations. After the initial successes, some observers said, with what seemed to be a lack of perspective, that one should now attempt to add decompression in the second stage.

The body can seal off the huge part of a lavatory seat, because of the substantial size of the cushion of the buttocks, but mainly when the thighs are held together in adduction. This realization was immediately put to the test on a tilting table containing an 8-gallon chamber with rounded vent which was covered with plain surface rubber securing sealing of the pregnant abdomen. Provided the bare skin was applied to the vent, an effective seal was readily experienced and the pelvic outlet decompressed to any pressure that could be tolerated. Suitable apparatus was constructed and second and third stages of labour were subjected to decompression (Heyns *et al.*, 1960).

Whereas we started with a pump which evacuated 70 litres of air per minute, we now use commercial vacuum pumps which remove more than ten times this volume. Very rapid decompression is thus assured and a pump is capable of serving several chairs. Unfortunately these units do not singly give us the low pressures required and we are using pumps in series and experimenting with Venturi systems.

The original outlet decompression chamber was in effect a bucket on which the patient sat. It was raised to a convenient height by the use of a simple wooden stand. The lid could be detached, but made an air-tight seal with the bucket by rubber adjustments. The vent measured 9 by 7 inches and the rubber surrounding it above was of firm sponge-rubber 1 inch thick and bonded to the metal. This chamber required the parturient to sit and, therefore, to hold trunk and foetus vertical.

A variation in the form of a small perspex cylinder made it possible to make suction with the patient in the dorsal or lithotomy position. A pump driven by a motor exhausted these chambers.

The simple cup and hand pump referred to in Part II has given excellent results. Theoretically a cup a few millimetres in depth, or even much less, would give a partial vacuum around the outlet with minimal suction. What stultifies this principle is the downward bulge of the vulva on decompression. We started with a cup 6 inches in diameter and 3 inches in depth before its edges were shaped to the requirements of the outlet. This proved too shallow. A cup 4 inches in depth proved almost entirely satisfactory except that the placenta was sometimes held back. A 4½ inch cup served our requirements. A cylinder 9 inches in length and 1½ inches in diameter gave a reduction in pressure of 100 mm. Hg with two excursions when the seal was good. We have called this instrument Gasyd.

PART FOUR THE IMPROVEMENT OF FOETAL OXYGENATION

Preamble

OXYGENATION of the foetus has received much attention in recent years.

Intracranial damage during labour, but more specifically asphyxial effects during both pregnancy and labour, are incriminated more frequently than in the past. The interest in cerebral palsy is pronounced. Moreover, such matters as the effect of prematurity in lowering intelligence have led to an inquiry into aetiological factors on a wide front. Several authors have joined Barcroft in referring to "Mount Everest *in utero*", a phrase which reflects the belief that the foetus has to adjust itself to some degree of hypoxia. That is also why the haemoglobin at birth is so high.

While it is believed by many paediatricians, and others who deal with so-called retarded children, that anoxia lasting for periods measured in minutes can cause cerebral damage, there has been very little experimental work to substantiate this claim. What material is available is restricted to findings on animals.

Windle and his co-workers (1943, 1944) have reported illuminating studies on guinea-pigs which were totally asphyxiated *in utero* at Caesarean section. The animals presented the picture we know so well: pallid, atonic, apnoeic creatures with slow, weak pulse and relaxed sphincters. Structural changes were found in the brains of nearly all animals which had been asphyxiated for eight minutes or more. Chromatolysis of nerve cells was observed as early as 1½ hours after resuscitation. Extensive loss of neurones was present in 66 per cent of the animals which had survived for learning tests to be started eight weeks after asphyxiation.

The animals subjected to learning tests all looked outwardly normal and could not be distinguished from the control animals by inspection. Windle (1950) says, "Some of them could not solve the problem at all and others learned it more slowly and forgot it quickly. In no instance was an experimental animal superior to its control. Quite clearly, therefore, asphyxiation at birth produced changes in brain function as well as structure." He poses the question whether some human dullards, like his guinea-pigs, might not be products of asphyxia neonatorum rather than victims of social environment and defects of the germ plasm.

OUR PROGRESS

Whereas this research project began in 1954, the present method of applying abdominal decompression did not come into use until the end of 1957. In 1958 experimental work arising out of decompression was done and published. Certain conclusions were drawn, followed by the prediction that decompression during pregnancy should improve the quality of the new-born by ensuring adequate oxygenation at placental level.

In the light of this fact it may seem strange that attention to the baby was neglected in the first two years. Decompression in labour gave pain relief and facilitation and the mother was accordingly the focal point of interest. Extreme care was taken to ensure the safety of the foetus and at all costs to avoid placing it in peril, yet nothing was observed—except that the foetus fared particularly well under the new conditions of labour.

Thus it came as a complete surprise in 1960, when my collaborators spoke with increasing frequency about the splendid babies born to our decompression patients. This, however incredulous one was at first, became more conspicuous every day until there was a series of 300 such cases. Of these the majority had been looked at critically and it was believed that there was not a single inferior baby among them. At birth these babies cried lustily, were of good tone, strikingly pink colour and in the first days of life they showed an unusual vitality. In a special group an Apgar index was later applied.

What had preceded this harvest was the introduction of an experiment dictated by experience with a personal series. It was called 270D because daily half-hour sessions were given between days 270 and 280 of pregnancy. Its aim was to prepare the birth canal for labour, and to shorten the first stage.

When the procedure was found to be quite safe, the sessions often amounted to a dozen or more; but when the quality of the babies became manifest, 270D became more prolonged antenatal decompression. It is true that an occasional patient had been given decompression throughout the second half of pregnancy, and this was in accordance with our early belief that daily decompression for all of the last trimester would enhance foetal oxygenation. It meant of course that the patient would carry out decompression at home and that she had to have her own unit of equipment. This could be spared for only one patient at a time and accordingly this aspect of the project was left in abeyance.

Now it began to appear that even short spells of decompression improved the foetus, and excitement rose—not only among ourselves but in the mothers. These people were seen at subsequent postnatal clinics. On being questioned, their reserve would give way to pride and they would tell of how good their infants were—that they hardly ever cried, that they fed properly, and were 'advanced'.

Multiparae invariably remarked on the comparative superiority of the new baby, and a para-12 of superior status said that this was far and away her best baby.

The Argument

While a few sessions of decompression could hardly be expected to raise the quality of a baby by influencing developmental processes, protection from the anoxia of labour may be significant. These superior babies had had worthwhile decompression in labour in most cases, the exceptions being those who had had too short a first stage to receive treatment and those who for a multiplicity of excuses were denied treatment.

How then should the present position be viewed? Let us take stock.

Workers are concerned about asphyxia, but this represents the cruder aspects of anoxia. It now seems probable that some degree of hypoxia occurs in all labours,

having regard to the good condition of our treated babies. Nevertheless all that has been entertained by others is the problem of the prevention of such anoxia, even if only severe anoxia. No one has thought of raising the oxygen saturation of the foetal blood during pregnancy, i.e. of improving even our best of to-day rather than saving the worst from their well-known fate. This possibility was never considered, simply because there had been no way of accomplishing it. In abdominal decompression we had the device for the purpose.

Decompression during pregnancy promotes painless contractions and foetal activity in the form of movements. A wide experience indicates that these movements are not due to distress. The contractions are regarded as necessary for improved oxygenation, but the mechanism will be described later.

It is necessary at this stage to define an attitude, in order to show one's standing in this investigation which is one that can be based on a high degree of speculation or be respectably rational. To me it is a phenomenon that the intellectually highly gifted make a sporadic and usually random appearance.

Gifted parents often have mediocre children and it is remarkable how undistinguished the sons of the historically great have been. What happens to the germ plasm? Why is there no indication that under favourable conditions the *homo* intellect has made progress either in excellence or frequency? Why is it that a superior genetic integration, acquired by chance, is characteristically extinguished? How should this problem be approached? The starting point is this: if there is in existence such a phenomenon as outlined, where do we seek to understand it?

It is the function of Genetics to explain the phenomena of heredity. If it is an observed phenomenon that the offspring of the exceptionally highly gifted revert to the average intelligence, Genetics is begging the question when it submits that this in itself is a rule or law—irrespective of whether there are further suggestions about a fortuitous chromosomal arrangement. Genetics must seek an explanation in the full knowledge that intellectual superiority cannot be defined in morphological terms and that, therefore, totally unknown entities can hardly be related to specific genes.

Genetics is only one philosophical facet of Biology, and one should not slavishly follow it as though it were a deductive science. The orthogenetic impulse must be at its greatest in any primate brain, and de Vries must surely have claimed for variation in cerebral excellence a spectacular means towards progress.

The general conclusion must be that something counteracts the acquired potential of the zygote. In the zygote there is contained the very highest potential of a genetic legacy. From birth, through infancy, childhood and adolescence, environmental factors are so standardized that no more than slight variation of their influence will occur.

Foetal life *in utero*, on the other hand, is precarious, particularly having regard to the unreliability of the placenta. Nature's drive towards the highest primate brain is perhaps its principal focus, but corresponding placental development is quite inconspicuous. There are factors other than oxygen, that are of fundamental importance, for example foetal nutrition and metabolism, trace elements, the domination of umbilical cord vessels. One is familiar with their importance,

but oxygen tensions in the foetal blood appear to be the most vital factor. Thus the foetus may suffer nutritional deprivation for days but anoxia for only minutes.

It is accordingly proposed to examine the effect of a better oxygen supply during the last third of foetal life in the belief that genetic gains or potentials are depressed by poor placentation into a state where cortical morphogenesis is below normal. Where, however, uterine activity during pregnancy is sufficient (see appendix p. 79), it is believed that it is unimpaired oxygenation that secures a 2 to 3 per cent incidence of superior babies.

It is submitted that the oxygen trail is the one to follow, not only because oxygen is indispensable to the function of animal tissues but because of what is now known of intracranial consumption of the substance. Moreover, we have the device to influence oxygenation.

EVIDENCE FOR BELIEF

Placenta

The total foetal blood volume at term is about 350 ml., and the maternal blood serving it is probably less than 100 ml. This corresponds with the smaller pulmonary circulation in the adult in whom, however, the rapid flow varies with the impressive physical activity that prevails. The foetus is almost sedentary.

The so-called intervillous space, which is best thought of as the placental lake or syncytial sac, contains both the villi which are foetal and the circulating maternal blood. The foetal component is arborescent and its morphology is such as to suggest that it will grow indefinitely as long as there is adequate nutrition. It also appears likely that its requirements must be met by foetus rather than mother. Having regard to this, it is found histologically that the maternal blood spaces between the villi form only a fraction of the whole.

Microscopic studies are very crude because they provide only a two-dimensional picture, but in most preparations it is obvious that the maternal blood has drained out of the placenta after its birth. For this reason we injected different materials into cotyledons, distending them to bursting point. Microscopic examination then gave the greatest possible intervillous volume—representing the greatest possible quantity of maternal blood—and at best this volume was 50 per cent of the whole.

Many of the crypts and crevices between villi appear so isolated as to be almost inaccessible to maternal blood. On the other hand there are dead branches imprisoning erythrocytes which give the impression that some red cells may never escape from the placenta, that some may take a week to re-enter the mother's circulation, and generally that there could well be a rich reserve supply of effete reds to ensure the iron requirements of the developing foetus.

One is now in effect saying that the human placenta is partly histiotrophe. One does so didactically because of the unwisdom of imagining a primate placenta to be only haemotrophe. The morphological status of the system is so low that all biological devices are no doubt invoked for survival.

It is thought that there are just on 500 spiral arterioles entering the basal plate. Those who speak with the most authority think that this number is far too high. Even if these arterioles have a diameter of 150μ, which of course they have not, it has to be denied that they can transmit 1 ml. of maternal blood per minute. Thus one is forced to disagree that a volume of 600 ml. per minute circulates on the maternal side. We have tried to cut paraffin sections in various ways to make an arteriole count possible but without success. In some respects one estimates the number of arterioles to be of the order of 100, in others of 1,000.

Everything that was said at Princeton at the Foetal Oxygen Supply Symposium is eloquent of a sluggish circulation, and Assali's suspected low values are spoken of as if they indicate a rapid circulation. The arterioles transmit blood only when the physiological conditions permit. These are based on carbon dioxide and oxygen tensions, other metabolites and hormones; but physical considerations also exercise control. In a closed unyielding space the addition of a very small proportion of volume will raise the pressure enormously. The demonstration of this is striking. Driving a nail through the stopper of a full mineral-water bottle will burst it. The arteriolar pressure does not exceed 60 mm. Hg and could not be projected against the high pressure consequent upon additions to the I.V.S. volume. If high pressures cause venous outflow, it will be mainly peripheral blood that will flow thus because of the labyrinthine nature of the foetal placenta. The entire framework is such as to preclude rapid flow.

A point of interest is the finding of visible filaments that run right across the placental space from chorionic to basal plate. One wondered what these were because they were too white and firm to be blood-vessels. Microscopy showed them to be a core of homogenous material like umbilical cord, perhaps mesenchyme, carrying blood-vessels of up to 60μ diameter which were obviously arterioles. Further study showed that the villi seemed to bud off these columns.

One is led to suggest that growth activity of villi depends on increasing vascularity under foetal stimulation and control. It is inconceivable that villous growth is supported directly by maternal blood in parasitic fashion: it must not even compete with foetal needs, for then something sinister like hydatidiform mole or even chorionepithelioma would develop.

What is visualized is that villi serve the foetus which, according to its vitality, supplies more and bigger villous scaffolding containing vascular elements. It is readily admitted that the placenta might be expected to be highly vascular, but by comparison with active tissues and glands its blood-supply is very unimpressive. It is to be remembered that this is its appearance when one sees it after removal from the maternal organism when its function has ceased.

In studying the placenta of the long term decompression mothers, one was struck by two things. The more certain one was the poor vascularity of the post-mature placenta which gave the appearance of drying up and increasing infarction, and the other was the tendency towards hyperaemia and congestion. The latter vascular states were not by any means morbid but drove one to the conclusion that a placenta structurally deficient and under-weight might be the seat of a blood flow so rapid as to function perfectly. This possibility makes it easier to understand

why the foetus of the last two months of pregnancy fares reasonably.

The decline of the villi is seen very well in ordinary sections, for there is first deficient staining with unlaked erythrocytes, then haemolysis with extravascular homogeneous blood elements, and finally bloodless dead tissue. However, such decay is readily advanced by the growth of new and vascular villi.

Pressures

Theoretical prediction that abdominal decompression will lower intra-amniotic pressure during uterine contractions has been substantiated in the finding that decompression can drop the intra-amniotic pressure to zero and even negative.

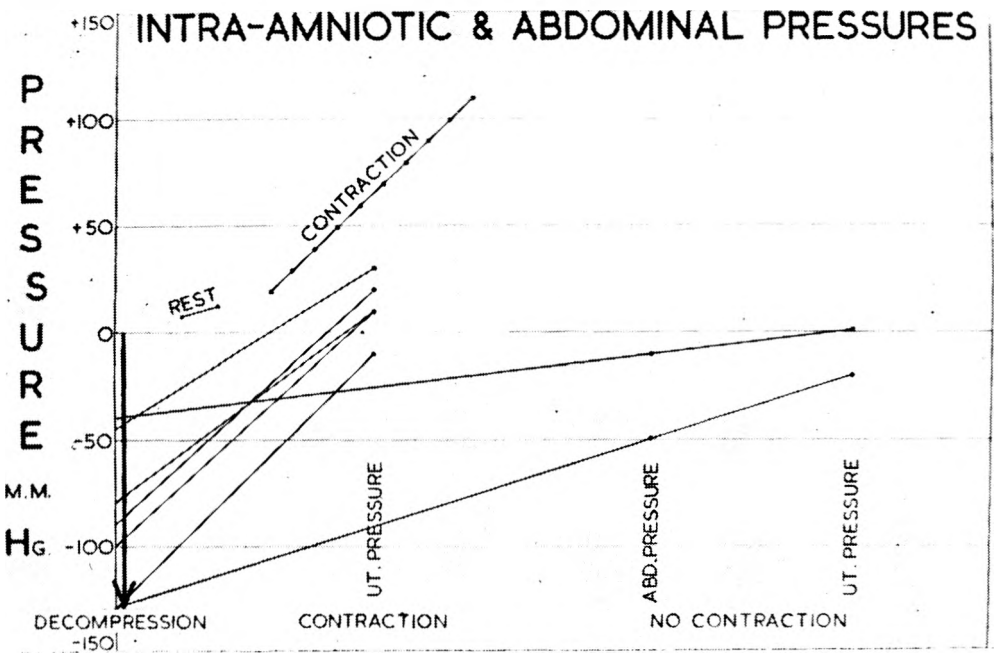


Figure 21

During an untreated contraction in labour the pressure rises from a resting 8 to 12 mm. Hg to 30, 40, 50, 60 and quite often 70 with a summit of over 100 mm. Caldeyro-Barcia and Alvarez (1950, 1952) have pointed out that progressive labour will occur only when pressures rise above 25 mm. Hg. Under 15 mm. there will be no progress and between 15 and 25 it will be very slow.

Williams (1952) showed that foetal bradycardia will develop if the amniotic pressure is raised above 80 mm. Hg for more than one minute in four. As the first stage ends, pressures get out of hand, and more so during the second stage, presumably because of bearing down and the consequent superimposition of positive intra-abdominal pressure.

As it is certain that foetal distress supervenes when pressures of 90 mm. Hg recur for one hour, this is a period fraught with danger for the foetus.

With the ranges of decompression used ordinarily in labour, pressures can always be kept below 30 mm. Hg, usually below 20 mm.; but with decompression being forced beyond 100 mm. Hg, zero and less. It is likely that any pressure below 20 is optimal and that nothing is gained by forcing zero or negative pressures. It is a fact, however, that negative pressures (that is below atmospheric) occur even during contraction, sometimes readily and at others with decompression pressures of between 100 and 150. It is clear then that decompression will give valuable protection to the foetus. (The mechanism will be explained later.)

So far from being brought to a standstill, labour is facilitated. Decompression permits unrestricted change of uterine shape from flattened to spherical, allowing for myometrial shortening. Thus isotonic contraction is possible—in its complete absence the cervix will not dilate. Without change of shape, isometric contraction occurs, causing great rise in myometrial tension and resultant increase in intra-amniotic pressure. With the isotonic, on the contrary, the muscle bundles slide over and between one another and the rise in tension is minimal. It is suggested that the higher pressures that are regarded as necessary for labour to proceed smoothly are caused by abdominal artifacts that vary from one woman to another and are in actual fact unfavourable to satisfactory progress in the first stage.

We have found that with the more complete decompression now in use abdominal pressure drops below zero (atmospheric) fairly early in decompression. Thus one can depend on getting minus 5 mm. Hg abdominal with decompression of 40 mm. Hg. The lowest pressures recorded have been minus 60. The intra-amniotic pressures have so far always been a few millimetres higher than the surrounding abdominal pressures and thus, accompanied by a lower myometrial tension isotonic contraction, it can be seen how the pressure is kept low.

The graph (Fig. 21) illustrates characteristic uterine and abdominal pressures for varying ranges of decompression.

Electro-encephalograms

Seventy-six infants had EEG. recordings. At the time the report was written there were 67 cases of which 22 were considered to be unsuitable for analysis. There were 23 controls. Nothing significant has emerged so far, but it seems unlikely that the first month of life will exhibit anything different from the usual EEG. pattern. Eleven infants were in the 1 to 10 month age group.

The report on this first series concludes as follows:

> No EEG. abnormalities were detected in any of the 45 babies tested. The findings thus revealed no EEG. differences between control infants and those born after maternal decompression. Possible effects of improved prenatal oxygenation on the cerebral maturation-rate were therefore not demonstrated.
>
> Research is continuing in the form of follow-up studies of individual cases over several months, because, although a cross-sectional analysis does not indicate either an advantageous or a deleterious EEG. effect

of maternal decompression, a longitudinal study might reveal individual EEG. changes with age that correspond to other observed differences.

Vascularity of Placenta

It has happened that the placenta born in decompression cases showed greater foetal vascularity than usual. There are several factors that influence this vascularity, but careful control material has been used for comparison. While there is no significant difference yet between the two, a strong trend is visible. This may guide us in the amount of decompression necessary for optimal effects.

Foetal Haemoglobin

It was reported at the April Interim Congress in Durban that the haemoglobin in decompression babies was significantly low. We have since done our own controls, not relying on the normal figures available to us. The haemoglobins of our own babies are lower than the controls with an average of 16.0 gm. per cent (range 11.9 to 18.8) against 16.7 per cent (range 15.2 to 20.3).

Oxygen Saturation of Foetal Blood

Cord blood taken as soon as the second stage ends shows in a small number of our special patients an average oxygenation of 59 per cent. This is high, but one does not expect to find a significant rise even if only for the reason that the normals in other clinics are rather mercurial. This estimation is at present in abeyance.

Inspection of Babies—their Behaviour

In a critical assessment of our theory and the interpretation of results one always returns to the high quality of our babies. This stands out like a beacon. There have been multiple pregnancies, maternal cardiac disease, occipitoposteriors, postmaturity, hard labours and other unfavourable conditions. None of these has caused observable impairment of the new-born, and we have been impressed to see babies emerge unscathed from situations in a manner quite foreign to our experience. All this is empirical, and so are the follow-up reports from the mothers.

In 53 last-trimester decompression babies the mean of the Apgar index was 9.6 points out of 10, with a range of 4 to 10. Forty-two babies scored 10 points!

We are engaged on the task of examining these babies periodically. An analysis appears at the end of this Part (p. 80).

Foetal Heart Response to Hypoxia

Having achieved as our best contribution the control of uterine pressure, it was necessary to discover the correlation between heart rate and intra-amniotic pressure. This was unproductive for two reasons: beats inaudible to auscultation are not necessarily absent electrographically: while they may change in quality, this cannot be measured quantitatively—and, of course, one cannot compare auscultation findings in one state with electrical in another. It is not easy to auscultate directly during decompression. The second reason depends on this. Elec-

tronic amplification indicates the heart beat even when weak and, therefore, it requires serious distress states to provide a bradycardia.

We have now succeeded in doing direct auscultation in labour with and without decompression. It has been demonstrated conclusively in cases of bradycardia during uterine contraction that no change of rate occurs with decompression. In addition we have repeatedly restored a foetal distress bradycardia to a normal 120 to 140 beats per minute (Heyns *et al.*, 1962). Bradycardia can be relied upon to occur throughout the second stage, but then abdominal decompression is not used and comparison is precluded. Finally, a great deal of our time and energy was devoted to these observations. Nor did we lack anything in apparatus.* The primary interest was in the prevention of anoxia to the brain, and the foetal heart was used only as our index of foetal oxygenation. If a means of assessing encephalic anoxia were available, one would ignore the foetal heart.

Mechanics of the Syncytial Sac

It was shown in 1958 that uterine contraction of the Braxton Hicks kind in pregnancy resulted in an increased volume of the syncytial sac. This would lead to lowered pressure in the sac with considerable vacuum in some of the crypts. It can be shown that oxygenation of the foetus and increased maternal blood flow through the intervillous space follow these changes. It was pointed out at the time that only one factor was unknown, namely the effect of higher pressures outside the sac collapsing it and so neutralizing the gain of decreased pressure.

This uncertainty disappeared when our uterine pressure readings were found to be of the order of minus 10 mm. Hg during decompression in pregnancy. From our experimental work it seemed necessary to develop a negative sac pressure around minus 40 mm. Hg. It seems probable that minus 20 mm. will be obtained with certainty. This in itself will give a pressure gradient from villous vessel to space of over plus 20 mm. Hg to minus 20 mm. This would be satisfactory.

Observations have made it clear that all of the syncytial sac is extruded at the birth of the placenta and that cotyledons are virtually independent units. This meant that each cotyledon formed an elliptical shape which, instead of having a myometrial wall on the outer surface as we thought before, had chorionic and basal plates for walls.

The problem of the pressure-flow system in the arterioles unfolded itself when the factors causing vasodilatation were considered. Hormones probably play a part, but the only impressive principle of this kind is that thought by Markee (1932) to be released by the foetus. Among the metabolites, carbon dioxide and oxygen cover the requirements sufficiently to explain the arteriolar circulation. Increased carbon dioxide and diminished oxygen both relax the vessels: conversely increased oxygen constricts. Physiologists even to-day cannot explain the mutual interaction of oxygen and carbon dioxide in anoxic anoxia, which must be the type that occurs in the foetus proper. Stagnant anoxia, according to our view, occurs in the intervillous space.

* Messrs Westdene Products loaned us their splendid Multi-channel Pressure and Sound Recorder

The mechanism suggested in the presence of decompression is that a uterine contraction occurs, the syncytial sac pressure becomes negative, foetal blood flows into the villi, carbon dioxide passes rapidly from foetal to maternal blood under a pressure head of 40 mm. Hg, maternal oxyhaemoglobin is reduced, oxygen diffuses to the villous blood, spiral arteriole vasodilatation and flow result from the temporary excess of carbon dioxide, the contraction subsides with a decrease in sac volume and consequent increase in pressure, and venous drainage of intervillous space blood restores the balance. Simultaneous with maternal blood outflow the rising pressure also forces blood from the villi back to the foetus. Thus a peripheral heart comes into play and the amount of oxygen released in the intervillous space is sufficient to simulate pulmonary respiration.

All these processes must be present with a Braxton Hicks contraction, but the reaction will be slow owing to the slight drop in the intervillous space pressure which will be counteracted by the comparatively high pressure outside the sac.

In the absence of uterine contraction the process will be slow, out of all proportion to foetal brain needs alone, quite apart from the rest of the tissues. Carbon dioxide will diffuse very slowly and be followed by oxygen exchange and, as often as the pH is low enough, the arterioles will transmit blood, raise intervillous space pressure and drive blood along foetal and maternal veins.

Central Nervous System—Intellect—Physique

The foetus exists almost in a state of suspended animation which might be relatively anaerobic. Yet proliferation of cells has to be at a high level. The question is whether this hypoxia is satisfactory or not. The foetus admittedly survives this state of depression, but at birth it cannot compare with the foal in spite of the inferior epitheliochorial placenta in *equus*. The *equus* brain has a highly convoluted cerebral cortex, but its lack of a Broca convolution may inhibit development *ab initio*. We are endeavouring to study the cerebral cortex in the foal at and near term. Once the vulnerability of the neuronal system to measurable anoxia is known in *homo*, we shall no longer be dealing with imponderables. An approximate estimate is that about 40 per cent of babies are in general inferior, about 60 per cent no more than mediocre, and 2 to 3 per cent superior. Gifted children—say with I.Q. 135 to 145—come from this 2 to 3 per cent: highly gifted (I.Q. upward of 150) and exceptionally highly gifted (I.Q. upward of 170) are so rare as not to require derivation. Why should the figure of 2 to 3 per cent be so low? It is very disappointing to all concerned. It is also the percentage of gifted children in highly civilized communities. It is to be emphasized that the modern child of superior intelligence is also outstanding in physique. The truth of this has been established beyond question by Terman and his followers. There are exceptions but, when one speaks of the best infants, there is the expectation that they will become gifted children. The question arises whether higher oxygen saturation of foetal blood is fundamental and, if so, whether the poor 40 per cent can be raised to a less pitiful level and the 2 to 3 per cent greatly increased.

Brain

We are bold enough to hope that decompression carried out daily from perhaps mid-pregnancy will thicken the neopallium. Where decompression has been given no earlier than the last fortnight of pregnancy and intensively during labour it is probable that the integrity of the brain has been preserved but that the increase, growth and arborization of the neurones have not been influenced. There is every inducement to improve the cerebral cortex structurally by ensuring a satisfactory oxygen supply.

Some morphological points may be of interest. The thickness of the adult cerebral cortex is not more than 4.0 mm. At 6 months of foetal age three layers can be made out and in the later months further differentiation occurs. Neurones occur in the grey matter and in ganglia and are numbered in billions—about twelve thousand million in the cortex. Having regard to the complexity of the dendrites in such cells as those of Purkinje and even Golgi, neuronal connections are seen to open up unlimited possibilities and indeed baffle description.

At birth, brain weight is one-quarter that of the adult—say 350 gm. At one year it is three-quarters its ultimate size, which latter is attained at 8 years. Growth is due partly to increase in the size of the neurones and of the cerebral blood-vessels, but principally to myelination. Continued growth after the first year is due to myelination.

In the cerebral cortex neurones are usually multipolar. There is only one axon, which is efferent; the others are dendrites and are all afferent or receptors. Nissl's granules extend into the dendrites but not into the axon, "and they disappear (i.e. chromatolysis) during fatigue or after section of the fibre" (Gray, 1923—1958).

A feature to bear in mind is the short vessels of the cortical arterial system, which are known to provide a poor circulation for the outer zone of the cortex. Physiologists entertain the idea that when one part of the brain is in action another part may be dormant. This of course is characteristic of all tissue activity—when one organ acts, another becomes less vascular: when an entire muscle like the biceps goes into action, not all its fibres shorten simultaneously. One's aim then is to improve oxygen transport to the brain. This seems likely to give the best chance for neuronal increase, but also for arborization and integration. In such cases the brain would require no circulatory preference, and the rest of the foetus can be adequately supplied with oxygen and nutriments and accordingly develop fully.

The contrary situation would be based on anoxia and greater blood flow through the brain in preference to other tissue. The question arises whether the result of this might be a richer vascular pattern with consequent increase in the short cortical vessels. This, however, may be counteracted by the greater need of mid- and hind-brain which are indispensable to foetal survival. In this case, the relative unimportance of the neopallium may result in circulatory preference for the more vital areas of the brain. This possibility also throws light on the occurrence of the physically superior man of poor intelligence who during the foetal period probably had perfect myelination in the spinal cord and brain stem but inferior myelination of the fibres of the large association areas (frontal, parietal and temporal). The

reverse, however, is indubitably more certain, namely the rule that boys and girls of great intellectual development are also physically superior.

DISCUSSION

In trying to define one's position and to crystallize such contribution as there may be, it is necessary to emphasize the conviction that we can prevent anoxia in labour with our device, and that probably an improvement in cerebral oxygenation during the last few weeks of pregnancy will result in a central nervous system that is unimpaired. In this manner the inferior 40 per cent will, for practical purposes, be obliterated. If one is in error about decompression, some other means must be found, because one does not think that the investigations on hypoxia are completely faulty; and the time has come to set aside complacency.

I have given my views on Genetics and think that we err when we suppose that the ament is in our midst because of chance arrangements in the germ plasm. This recalls the point about empiricism which at present is one's mainstay. While admitting the latter fact one is probably in the position, nevertheless, of being able to point an accusing finger at the attitude of all of us who have simply accepted cerebral quality as it is and have falsely believed that these things are inevitable. The truth will not be known until many cases have been treated and the sooner the problem is tackled on a wide front the sooner will a conclusion be reached. In the present state of one's knowledge the possibilities stand firm, and the harvest could be prodigious. Factors which are now unpredictable may stultify this expectation, but that would not dissuade me from my present purpose. There is much observation still to be made and several facets have hardly been smoothed or clarified at all, but one is firmly convinced that this does not weaken the case.

It would be wrong to imagine that this case is highly speculative or entirely hypothetical. Induction has in fact played a substantial part, for many of the phenomena appeared unexpectedly, and deduction was used once the inductive process had led to a conclusion. One has repeatedly submitted that the conjectural fraction of the argument is outweighed and indeed supported by the empirical certainty touching the babies. There is such high probability of raising foetal quality that it is imperative that the investigation be pursued to finality to obtain the true answer.

Calculations suggest that the foetus may get enough blood circulating but that the oxygen may be deficient. The situation in general may not be too bad, but it seems inevitable that there must be a period when the whole process is slowed down—sometimes dangerously.

At 28 to 30 weeks the placenta has reached full size.

From Assali's figures it seems as if the 2 to 3 pound foetus should get enough O_2.

Everyone agrees that placental growth does not take place in the last two months. Increase in foetal weight makes it inevitable that anoxia must occur unless there are very frequent Braxton Hicks contractions.

At term 30 ml. blood/100 gm./minute to the brain seem to be available.

Saturation must be 75 per cent at least and O_2 must be used to drop it to 25 per cent.

Values of 54 ml. blood and 3.3 ml. O_2 /100 gm./minute are needed by adult brains.

In adults a 10 per cent decrease in O_2 causes mental hebetude, a 20 per cent decrease has the effect of 3 to 4 cocktails, and as the decrease in O_2 approaches 40 per cent, coma ensues.

One envisages three degrees of anoxia:

(*a*) No damage is done to neurones, but their increase and development are retarded. This can be improved by decompression.

(*b*) The position is so bad that some neuronal damage is done which decompression could certainly have prevented.

(*c*) Superimposed on (*a*) or (*b*) there is a catastrophe of some sort—disease, cord compression, placental separation, premature spurious labour—when anoxia passes beyond the border-line of safety and neuronal loss (death) occurs. The result is the birth of children who are mentally deficient, cerebral palsies, idiots or perhaps only dullards.

The question is whether the routine use of decompression could prevent these set-backs under (*c*) and lower the incidence of 'retarded' children. I do not see why it should not. The exceptions will be the accidents that occur from 20 to 26 weeks having regard to the fact that they are very serious and severe and are based on pronounced renal, metabolic and circulatory defects.

According to our hypothesis both (*a*) and (*b*) above might be the basis of the primary agenesis invoked to explain congenital cerebral palsy. At least they throw some light on an otherwise unintelligible agenesia. Cases of atrophic sclerosis may show regional or generalized neuronal loss and are irreversible. Where there is a truly primary deficiency of neurones, progress in infancy is restrained because there is no neuronal proliferation after full gestation. Thus stunted growth or comparable arrest cannot be regarded as having a temporary effect, a handicap that can easily be made up in infancy. If a tree, an animal, or a baby is fragile early on, it tends to be hyposthenic later. Prematures certainly fail to catch up as a rule. But there are some dramatic exceptions.

How did Sir Isaac Newton manage to do so well mentally? Was he born before neuronal damage occurred? There is also Voltaire. It seems as if an individual can catch up after birth, particularly if birth is very premature. Why not? It is obvious that it is possible. It may be thought that there is no haste for the baby to develop in the first year of life and that matters could be left to take their course. But it seems to be the rule that gifted children have shown precocious development as babies. One is surprised that some are slow about dentition and other minor features, but the forebrain and even midbrain show more advanced function than in the average.

Perhaps the problem resolves itself into whether the following is true or not:

(*a*) Does the infant, born without decompression, have neuronal damage or not? If he has, the damage is done: he has suffered a certain loss of neurones which he cannot restore. He will never be the same again—in

fact, he is not of the same species as the one born with decompression.

(*b*) Newton is a classical example of what can be done before neuronal damage has occurred. He must have been an idiopathic premature having no morbid factor present except that of being contained in an irritable uterus. He will thus have had the advantage of many Braxton Hicks contractions and neuronal damage could be expected to have been absent. At 28 weeks babies are born for this reason or because of maternal disease, but never perfectly normally. There is accordingly no reference experience on the problem, but, hypothetically, very early termination of pregnancy by Caesarean section might produce some really good ones.

What is the ultimate vision? One does not know whether a barrier is set against further intellectual progress in *homo*. The evolutionist cannot think so, the Story of the Ages reveals unequivocal deterioration in man, and the anatomist will refer to the infinite possibilities of his cerebral cortex. The barrier has indeed been penetrated, notably by Goethe, J. S. Mill, Leibniz, but more unobtrusively by such as Hamilton (the Irish mathematician) and even this century by Betty Ford, Elizabeth (Erie), J.M. (Washburne), E.B. (Stedman) with I.Q. 214; and the six children, A to F, fully documented by Leta Hollingworth, all with I.Q. above 180.

The computed I.Q. for the exceptionally highly gifted of the past suggests a figure of around 200. The children here shown to have been tested this century rank over 180 and quantitatively seem to be in this highest class. That is open to question, however, for a property of the very top class is the ability to read some Greek at the age of 3 years; and this seems to have been out of the reach of the twentieth-century geniuses.

Heineken as a baby aged 10 months revealed something latent and a tutor was immediately engaged for him. At 12 months he had memorized the best stories in the Pentateuch and at 14 months stories from the Old and New Testaments. He died when he was 4 years and 4 months, but by this time many people in Europe had seen him. Audience was given him by King Frederick IV at which he displayed astonishing tact of speech and manner. Elizabeth Tuttle seems to be the most remarkable of those recorded as being the progenitor of several generations of greatly distinguished persons. The rarity of this expected genetic trend in man leads one to postulate for Elizabeth Tuttle a gene, in addition to the obvious ones, endowing the sort of uterine function in pregnancy that we have described as necessary but rare in erect man. She was a woman "of great beauty, tall and distinguished in stature . . . of strong will, extreme intellectual vigour, of mental grasp akin to rapacity . . . attracting many by her charm." From this line came Winston Churchill.

There is, then, the vision of eradicating cerebral palsy, reducing the incidence of inferior babies from 40 per cent to a few *per centum*, of raising the general standard, and of increasing the percentage and possibly even the excellence of babies of the first rank. There emerges a deeper meaning to *mens sana in corpore sano*—a healthy mind in a healthy body—for a sufficiently satisfactory foetal environment will promote superior development of body as much as of mind. One has said during the

past decade that the old orthodox Obstetrics had reached its peak about the end of the half-century and that for the mother pregnancy and labour were well under control. It was envisaged that advancement in favour of the baby would dominate the future scene in Obstetrics but the endeavours of many will be required to test the validity of the hypothesis presented here.

APPENDIX

Syncytial Sac (I.V.S.) Pressure Flow Dynamics

1. Contraction + Decompression

   When a contraction occurs in the presence of decompression, the following events take place in order, each one causing the next:

   (*a*) Uterine shape becomes less ellipsoid and more spherical.

   (*b*) Myometrium shortens.

   (*c*) The long axis of each cotyledon (I.V.S.c) is diminished by (*b*) above. Short axis must increase. Thus volume of I.V.S.c increases. Therefore total I.V.S. volume increases.

   (*d*) P.i.v.s. is decreased by (*c*) above. All pressures around I.V.S. are negative, say—10 mm. Hg. Therefore P.i.v.s. readily reaches—20 mm. Hg.

   (*e*) With villous vessels at a pressure of +20 mm. Hg there is a pressure gradient of 40 mm. Hg from foetal to maternal blood.

   (*f*) As a result of (*e*) above, CO_2 will cross villous membrane rapidly (Heyns, 1958). Incidentally there will be increased blood flow along the umbilical arteries.

   (*g*) CO_2 will reduce maternal $HHbO_2$ freeing O_2 which diffuses to the foetal blood. Excess $CO_2 \rightarrow$ pH drop $\rightarrow$ vasodilatation $\rightarrow$ filling of I.V.S. with blood $\rightarrow$ rise in P.i.v.s. $\rightarrow O_2$ transport to foetal blood.

2. Contraction subsides:

   Decrease in I.V.S. volume causing great rise in pressure which in turn causes venous outflow to both foetus and mother.

   Note: A peripheral heart action is shown to be present in 1 (*f*) and 2.

3. Braxton Hicks Contraction:

   All these processes are present with Braxton Hicks contractions in the absence of decompression. However, the reaction is slow, because I.V.S. is collapsed by positive surrounding pressures as soon as P.i.v.s. drops following on change in shape caused by the contraction. Thus P.i.v.s. may not drop below zero, and pressure gradient between foetal and maternal blood is below 20 mm. Hg.

4. No Contraction:

   In the absence of Braxton Hicks contraction:

   Diffusion of gases is very slow.

   CO_2 will diffuse very slowly, liberate O_2 and, as often as the pH is low enough, the arterioles will transmit blood, raise P.i.v.s. and drive blood along foetal and maternal veins.

THE ANALYSIS OF BABIES WHOSE MOTHERS HAD DECOMPRESSION DURING PREGNANCY

Eight months ago the first decompression baby was tested. Figures will be presented for 330 babies tested, but 80 of these were controls.

Material and Methods

The material was composed of:

(*a*) 60 control babies from our clinic at the Queen Victoria Hospital, 30 being taken at random and 30 as shown below;

(*b*) 20 Bantu control infants from the clinic at Moroka Township;

(*c*) 196 babies, the ANC Series, whose mothers had had up to 30 decompression (D) runs during pregnancy. These women attended our antenatal clinic at the Queen Victoria Hospital and were subsequently delivered in hospital;

(*d*) the Sanctuary Series of 38 babies whose mothers were unmarried and lived in a home during their pregnancy;

(*e*) a Bantu (South African Negro) group of 16 given daily decompression in Western Native Township.

Series (*b*) and (*e*) were non-European babies and the others White.

All infants of mothers who had had decompression during pregnancy were under 24 months of age, and all the available infants were used for testing. Of the White controls 30 were taken at random as they visited an antenatal or postnatal clinic with their mothers, and a sequence of 30 was tested at each month of age up to 30 months. The latter were ones born nearest in time to a decompression baby.

The ANC Series mothers were hospital material and as such a fair cross-section of what is seen under the circumstances. Nevertheless, they were volunteers to the antenatal decompression clinic and some of them were splendid women. The Sanctuary Series girls were taken as they entered their small institution without any selection whatsoever, and we know that one patient had an I.Q. of 55 and another of 60. The Bantu women had to be selected for their suitability of temperament and the demand that they had to attend their local clinic daily: their number of sessions was 57.6 per mother.

The unmarried mothers were used for long term decompression because there was an economy of apparatus in providing daily runs. Two sets of equipment were installed and were in use throughout the day. The average number of sessions per mother was 63. For daily decompression in the last trimester no woman can be expected to attend hospital, and we have endeavoured to lend equipment for domiciliary use to some special patients.

The tests were made on a Gesell pattern. The latter reflects, for each month of the age, the motor development, language, adaptive behaviour and personal-social behaviour. Gesell obtained averages in the United States by testing some 100 babies for each month. The available scheme accordingly saved us the onerous task of devising a pattern of our own. Our interpretation of Gesell values cannot lead to serious error, because we have the control babies for comparison. These

controls have scored a mean of 107 to Gesell's 100, but we do not believe that we have been at all lenient in our assessments.

Three of us, all obstetricians (Heyns *et al.*, 1962a), tested the babies. We have been objective and strict and are confident about the reliability of the findings. It is not difficult to appreciate comparative levels of development.

Ratings were obtained by dividing the developmental age by the chronological age. Motor and behaviour development were assessed together. This is unsatisfactory when the correspondence between physique and intelligence is poor. Developmental age was assessed on the proportion of tests correctly performed, but the figure arrived at was in units of months with a fraction of not less than half a month. Thus an infant aged 10 months who scored the equivalent of 9 months was rated −1 or with a Factor of 0.9. Similarly an equivalent of 10 months would be rated 0 or Factor (F) 1.0, scores at 11, 12 and 15 months representing +1 or 1.10, +2 or 1.20 and +5 or 1.50 respectively. For the sake of convenience the F figures were multiplied by 100.

Babies have been tested at all ages up to 24 months and on some the examination has been repeated. It has been easier to make observations from about 6 months onwards. Language as a means of communication is usually absent under the age of 20 months.

The Analysis

I. Table III contains the essential facts and can be used for calculations in

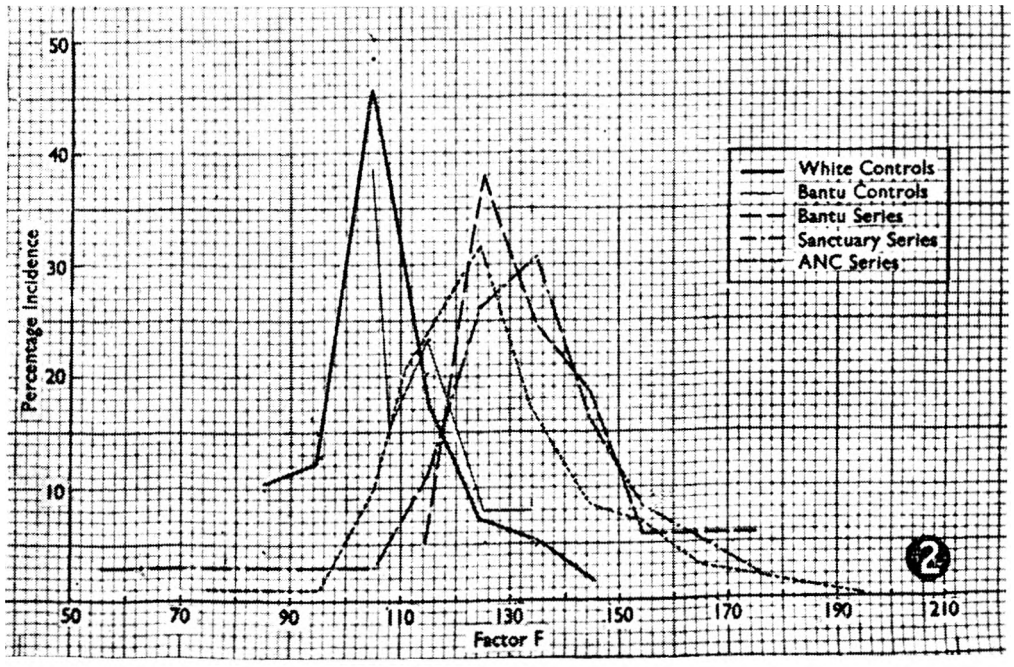


Figure 22

Baby assessment (Factor F) graphs

addition to those shown. One aspect is shown in Fig. 1 which gives a fair impression of the effect of decompression on the quality of the babies.

The graphs in Fig. 22 were drawn when the total figures were a little lower than the present ones. Thus the Bantu and White controls at present have curves that coincide with peaks at about the 50 per cent level, but are otherwise of the same shape. The control groups are clearly inferior in F scores. The significance of this is illustrated by the difference between the two very small Bantu samples, for it is 24 ± 4.8, which is 5 times its standard error. In the Whites the corresponding difference is more than 7 times the standard error.

The scatter diagram (Fig. 23) represents the distribution of 141, 16 and 36 of the three decompression groups of which the F values have been plotted against the decompression (D) runs. Inspection of the diagram reveals a measure of asso-

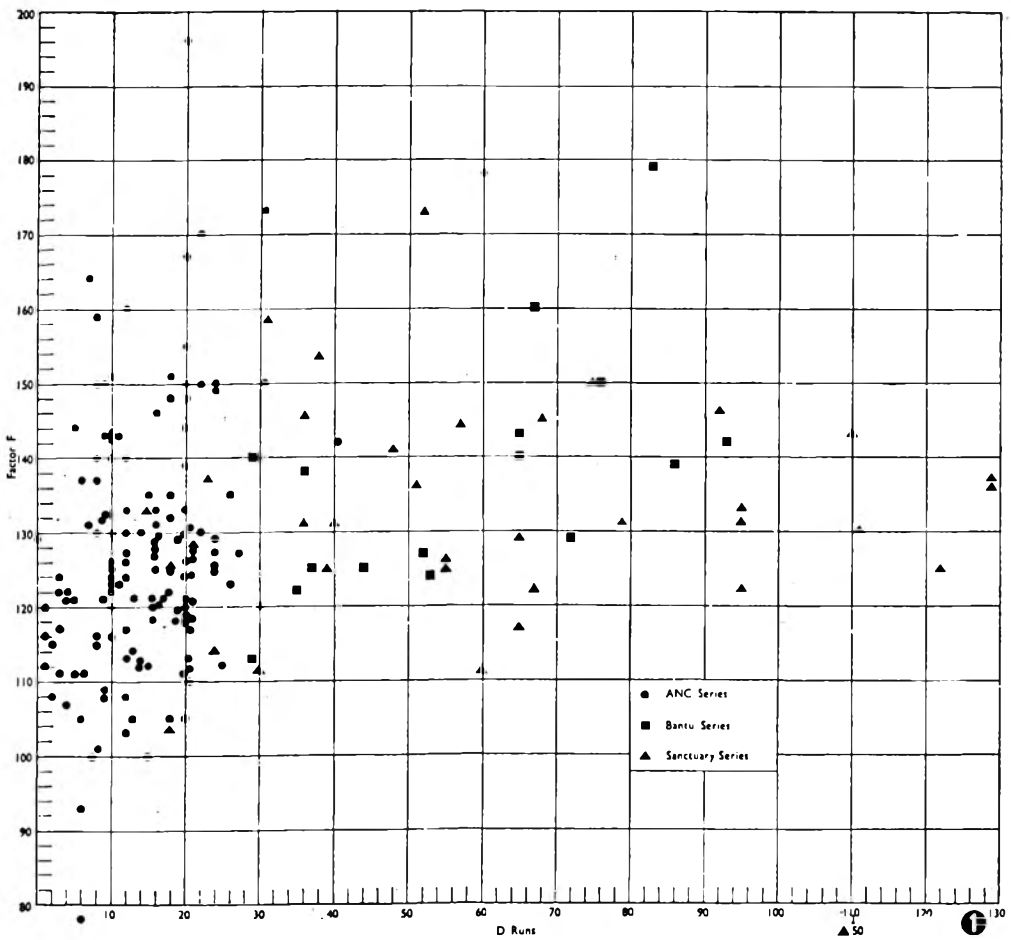


Figure 23
Baby scatter diagram

ciation between the two variables in its early portion, but nothing striking after 70 runs. Most of the examples hereabouts are Sanctuary babies who, for some reason, have not benefited from excessive runs although all tend to score about 120 F or better. The association may be tested by calculating the correlation coefficient. Table IV shows a fair measure of this for certain samples, the Bantu series and D runs up to about 30 being the most obvious. A coefficient of 0.63 for the Bantu is striking.

TABLE III

Number of infants (percentage in brackets) and, in the first column, their Factor F score

Assessment Factor F	White Controls	Bantu Controls	ANC Series	Sanctuary Series	Bantu Series
50—55				1 (2.6)	
75—80			1 (0.5)		
80—	2 (3.3)				
85—	4 (6.7)				
90—	3 (5.0)	1 (5.0)	1 (0.5)		
95—	4 (6.7)	2 (10.0)			
100—	17 (28.4)	6 (30.0)	7 (3.6)	1 (2.6)	
105—	12 (20.0)	4 (20.0)	15 (7.7)		
110—	4 (6.7)	4 (20.0)	22 (11.2)	3 (7.9)	1 (6)
115—	7 (11.7)		22 (11.2)	1 (2.6)	
120—	2 (3.3)	1 (5.0)	34 (17.4)	2 (5.3)	4 (25)
125—	1 (1.7)	1 (5.0)	23 (11.8)	7 (18.4)	2 (13)
130—	1 (1.7)		18 (9.2)	7 (18.4)	
135—	2 (3.3)	1 (5.0)	14 (7.1)	4 (10.5)	4 (25)
140—	1 (1.7)		13 (6.6)	4 (10.5)	2 (13)
145—			4 (2.0)	4 (10.5)	1 (6)
150—			11 (5.6)	2 (5.3)	
155—			2 (1.0)	1 (2.6)	1 (6)
160—			2 (1.0)		
165—			3 (1.5)		
170—			2 (1.0)	1 (2.6)	
175—180			1 (0.5)		1 (6)
195—200			1 (0.5)		
Totals	60 (100)	20 (100)	196 (100)	38 (100)	16 (100)
Mean F	106.8	107.2	127.8	130.8	134.1
S.D.	12.7	10.4	16.8	16.8	15.1
Average D runs ...	Nil	Nil	13.5	63	57.6

TABLE IV

Correlation Coefficients for Factor F and D runs

Maternal Group	r	Significance
ANC Series of 190	+ 0.36 ± 0.07	5.1 × standard error
16 Bantu	+ 0.63 ± 0.26	2.4 × standard error
D runs up to 60 (all groups)	+ 0.30 ± 0.07	4.3 × standard error
D runs up to 93 (all groups)	+ 0.30 ± 0.07	4.3 × standard error
D runs between 30 and 93	+ 0.13 ± 0.16	Absent.

Regression equations are possible and have been calculated. They are:

Up to about 30 runs F=0.85 D runs + 116.5

Up to 93 runs F=0.26 D runs + 123.5

II. Another way in which the relationship between decompression runs and the Assessment Factor—F may be tested is by counting, for a given range of runs, the number of scores up to 120 F and those above. Any statistical significance for the difference in percentage is estimated.

(1) Up to 10 runs and 120 F → 29 infants
Up to 10 runs above 120 F → 23 infants
per centum difference 6 ± 9. Not significant

(2) Up to 5 runs and 120 F → 12 infants
Up to 5 runs above 120 F → 7 infants
per centum difference 26 ± 15.7 Not significant
F above 120 is uncommon

(3) 6—10 runs up to 120 F → 17 infants
6—10 runs above 120 F → 26 infants
per centum difference 20 ± 10.5. Not significant
F above 120 is commoner than in (2)

(4) 8—12 runs up to 120 F → 16 infants
8—12 runs above 120 F → 31 infants
per centum difference 32 ± 9.8 = 3.3 × standard error

(5) 10—15 runs up to 120 F → 15 infants
10—15 runs above 120 F → 26 infants
per centum difference 26 ± 10.6 = 2.4 × standard error

(6) 10—20 runs up to 120 F → 33 infants
10—20 runs above 120 F → 62 infants
per centum difference 30 ± 7.1 = 4.2 × standard error

(7) 15—20 runs up to 120 F → 19 infants
15—20 runs above 120 F → 37 infants
per centum difference 32 ± 9 = 3.6 × standard error

(8) 20—30 runs up to 120 F → 15 infants
20—30 runs above 120 F → 34 infants
per centum difference $38 \pm 9.3 = 4.1 \times$ standard error

(9) 30—130 runs up to 120 F → 4 infants
30—130 runs above 120 F → 51 infants
per centum difference $86 \pm 4.9 = 17.5 \times$ standard error

From the above figures several conclusions appear to be valid. Five decompression runs seem to have little value but from 8 upwards significant gains occur. From (4) to (9) the significance is striking except that the numbers in (5) are rather small. The trend is for F values to improve as more runs up to 30 are given, and at present the exceptionally highly significant difference ($17.5 \times$ standard error) in (9) must be accepted as indicating a serious probability that F values below 120 might be preventable.

III. The danger to the foetus of parturition

(*a*) We ourselves have no doubt that labour has an adverse effect on the foetus, mainly because of anoxia. It has seemed to us that a prolonged and difficult labour might undo the benefit of pregnancy decompression. An inquiry was, therefore, made relative to the information at our disposal.

In the ANC Series labours were assessed as being bad, poor, poor to good, good and excellent. In 182 labours the respective numbers for these classes were 5, 14, 7, 60, 96. No decompression had been given in 2 poor, 13 good and 29 excellent labours.

TABLE V

Class (Labour)	Mean Factor F Ungrouped Series	Mean Factor F Grouped Series	Standard Deviation	Mean D runs	S.D.
Bad, Poor, Poor to Good	123.6	124.6 ± 4.1	20.3	14.6	8.6
Good	126.2	127.6 ± 2.1	16.0		
Excellent	128.7	129.1 ± 1.8	16.7	16.4	9.5

The F values in Table V have differences that in no case are more than the standard error. An equal lack of significance exists for the D runs given in the poor and the excellent classes of parturition, for the difference is less than the standard error.

Statistically, therefore, dystocias have not been proved to impair F values in the babies. It is to be emphasized, however, that the F difference between the 60 control and the 60 good labour babies is 21 ± 2.8 which is more than 7 times its standard error, and thus highly significant.

(*b*) Decompression during pregnancy, but also in labour, has had a good effect on parturition, as shown by the figures that follow:

Bad, poor and poor to good labours were 14.3% of the total.
Good and excellent labours were 85.7% of the total.
25% of labours had no decompression, but of these 95% had good or excellent labours,
75% of labours had decompression but of these 83% had good or excellent labours.

This paradox is to be explained by the fact that parturients with very short labours often had no decompression. It indicates that the patients were attended fairly conscientiously, although we are well aware that some did not have decompression when they should have had it.

Discussion

Three basic questions that arise are, whether the examiners were competent to assess the infants, whether the tests served their purpose and whether the comparative acceleration of development would be maintained.

We believe that we have mastered the art of testing the babies. Whether the tests applied to these babies discover what is fundamental, useful or indeed what is required, must be met by the admission that one knows of no other form of testing. Furthermore, the examiner looks beyond the surface to envisage the progress of nerve myelination in the infant. It is felt that one is on the right track. As a quarter of the babies are of outstanding quality, the investigation is of a special nature.

Subsequent examination will show whether high rates of development are maintained. It is our opinion that the *tempo* of development in the superior babies must remain greater than in the mediocre who will show small fluctuations but will in fact remain beneath the ceiling of mediocrity. The finding that infants in general or taken in groups revert to a uniform level when they enter childhood is derived from random samples in which the percentage of superior individuals, as stated, is 2–3. One is not aware of a follow-up of high quality infants such as are found in our own sample. Terman studied mentally superior children, but their quality in infancy could only be assessed in retrospect. The available information was invariably stated to be meagre.

The rate of development of decompression babies follows a curve. The available information points to a flattening of the curve at about 20 months of age. The relationship between Factor (F) and chronological age (A) is $F = A^n + c$ and, because n was found to be 0.97, the curve, so far as one can judge, is almost linear. The curve composed of means for every month does not pass through the origin and shows a mean advancement of half a month at birth. There can be little doubt that development should be calculated from a time several months before birth.

It is clear that experimental curves of repeated examinations of different babies must be constructed. These will show the trend and should provide reliable curves for any correction of tested F values that may be necessary. As testing has been in progress for only 8 months, one cannot at present even plot 20 month

values and those found under 10 months in individuals. Such considerations do not invalidate the present findings which reflect an accurate assessment of the overall quality of our babies.

The general conclusion is that the decompression babies on average are far superior to those not having decompression. Taken individually, the incidence of exceptionally advanced babies is very high. At the extreme of the range of quality there have been a boy who walked unaided at the age of 7 months and 2 days while a second was only a few weeks slower for the same achievement, a triplet female who scribbled with concentration at the age of 7 months, and a girl who had a vocabulary of 200 words at 18 months.

Decompression as used in the ANC Series may have no more effect than to improve the circulation of blood in the placenta. If so, the present advantages from a small amount of decompression are explained. On the other hand, more than one daily session may be required to cause a pronounced effect on neurones from improved oxygenation. The duration of Braxton Hicks contractions following a pregnancy decompression run has not been determined, although observations are in progress. Two apparent effects on the placenta may, however, be recalled here. One is that decompression babies are not born prematurely but tend to become postmature. The other is that pre-eclampsia is conspicuous by its absence, as judged from 385 cases. These observations point to a more active circulation in the placental villi and consequently less infarction and death.

In an empirical sense one is convinced that a bad labour, which is prolonged and represents increased work owing to hundreds of contractions, causes damage to the foetal central nervous system; but the analysis does not bear this out. The reason for this may lie in the fact that there were only 5 'bad' and 14 'poor' labours. Nor is it possible to decide how much better the babies concerned might have been with more satisfactory parturition. The problem will be examined by testing series of untreated children by grouping them according to the record of the labour in each case. In the present series there was undoubtedly some degree of devotion to the parturients comprising it, but much less than was expected.

PART FIVE SUMMARY AND CONCLUSIONS

SUMMARY OF PRESENT POSITION

ABDOMINAL DECOMPRESSION is simple in its conception, and is readily administered to the patient. It has required some application to devise standardized equipment, but it does not demand overmuch ingenuity to improvise apparatus for laboratory use. Some years ago a man in the heart of Africa prepared a successful device for his wife's confinement, having gained the idea from a press report on the subject.

The effects of the procedure on different structures have been considered, and several clinical, physiological, and investigational uses have emerged in consequence. Thus all three stages of labour may be served by it with advantage. The pain suffered in labour, in primary dysmenorrhoea and in simple backache can be relieved, in some cases completely. But the most valuable contribution of abdominal decompression is its influence on placental and foetal blood flow and oxygenation.

The pressure flow dynamics of the syncytial sac has been examined and on this, in the main, rests the hypothesis submitted. In this respect an extremely important side-effect is the reliable control of intra-amniotic pressures: even during labour contractions the pressure can be kept below 20 mm. Hg, but decompression by 130 to 150 mm. Hg will reduce the pressure to zero or below. Accompanying this, the abdominal pressure drops from +4 or 5 to −5 at 40 mm. Hg decompression, but −50 to −60 mm. Hg at 150 mm. Hg decompression.

Foetal distress during decompression has thus far not occurred in the first stage of labour. On the contrary, a number of cases of first-stage foetal distress have been treated by decompression, the worst having a bradycardia of 70 with irregularity and the passage of meconium. These have been restored to normal and have terminated with the birth of a baby in good condition. The foetal heart does not change in rate or force during first-stage contractions.

Daily decompression during the last trimester of pregnancy has not, in 85 women, been associated with any case of pre-eclampsia. There were 2 mild cases in 300 of a short term series. As stated, the condition of the new-born has been excellent, even where this could not have been expected owing to the abnormality of the labour (and sometimes of the pregnancy).

Decompression in pregnancy promotes Braxton Hicks contractions and a heightened activity of the foetus. The former are considered to increase, at placental level, carbon dioxide and oxygen exchange, and blood flow. As foetal hypoxia is believed to be the rule rather than the exception, particularly after the thirtieth week of gestation, any improvement in placental efficiency is a significant step forward. Furthermore, development of the central nervous system would be retarded under average normal conditions, and it is submitted that protection from hypoxia is possible during both labour and pregnancy.

The major experiment now in progress consists in conducting trials on White and Bantu women by giving daily decompression in the last trimester of pregnancy.

This should be done on a big scale, and workers elsewhere should do large series, for a decision one way or another should be possible within three years.

Meanwhile our own babies who have been subjected to decompression during pregnancy, for sessions varying from 3 to 123, but recently with a minimum of 12 to 20, now number some 500. The results of testing 250 decompression babies and 80 controls on a Gesell pattern of assessment show that our babies are far superior in physical and mental development to the controls with a difference that is highly significant statistically.

The present perspective of the hypothesis formulated to explain the role of decompression in childbirth may now be considered.

Braxton Hicks contractions are the primary need for increasing placental blood flow and oxygen transport. These contractions may be isotonic or isometric. It depends upon the give or resistance of the muscular abdominal wall which type predominates. Whereas the isotonic variety serves foetal needs efficiently, the reverse is the case with the isometric. We have studied this factor during labour, but foetal heart action as an index is not entirely satisfactory. In this respect the time factor seems to be important, a succession of isometric contractions being required to cause noticeable cardiac disturbance.

The value of decompression during pregnancy lies in this, that the soft abdominal wall is stretched artificially rather than by the growing uterus. Therefore subsequent pregnancy contractions are likely to be isotonic.

Isometric contraction causes uterine pressure to rise and, consequently, the entire foetus and the placenta function under higher pressures. With abdominal decompression the reverse occurs. As there is no air to displace in the foetal lung and diaphragmatic movement can be shown to be virtually absent whether the external pressure is high or low, thoracic and abdominal pressures are not equalized. Therefore the antenatal condition differs so much from the postnatal that thoracic pressure can be higher or lower than the rest of the body. When lower, the effect is for more blood to reach the right heart. Now unfortunately the stroke volume is opposed by greater resistance outside the thorax.

However, where a localized low pressure region develops, the position becomes significant; and this happens in the syncytial sac whenever an isotonic contraction occurs (Heyns, 1958). Abdominal decompression amplifies this. In addition it creates a range of regional pressures with foetal thorax highest and syncytial sac lowest, but with abdomen, extremities and head and neck intermediate.

In respect of pregnancy, placental blood flow and oxygenation are least efficient with an isometric contraction but become progressively better in the quiescent state of the uterus, with decompression alone, with isotonic contractions, with decompression accompanied by isotonic contraction—possibly in that order.

To the simplified scheme appearing in the Appendix of Part IV (p. 79), we may now add the observation that a cyclic sequence probably operates in the presence of an isotonic contraction.

Blood is sucked into the villi from the umbilical arteries and possibly vein, the foetal heart continuing to make its contribution in the same direction. Syncytial sac pressure begins to rise. During the first few heart beats CO_2 diffuses from foetal

to maternal blood. Whole blood releases H_2CO_3, HCO_3 and $HHbCO_2$ to a vacuum. As CO_2 crosses the membrane it is gaseous and, therefore, occupies some part of the I.V.S. volume. This movement slows down progressively as the reaction approaches equilibrium. I.V.S. pressure rises. O_2 is released from $HHbO_2$ by the CO_2 and is in gaseous form and there is no alteration in I.V.S. pressure. O_2 crosses the membrane into the villus. The syncytial sac pressure drops because O_2 combines with HHb, but it rises as a result of the CO_2 effect on the maternal spiral arterioles which pump blood to the I.V.S. The foetal heart continues to pump blood into the villi. Pressure is now so high that foetal blood is driven from the villi, and maternal blood through its veins. This foetal peripheral drive is inevitable.

The tendency to form a partial vacuum resulting from the isotonic contraction continues and the original negative pressure, which became positive until it was the same as that in the foetal abdomen, now becomes negative again. Blood is once more drawn into the villi—not into the I.V.S., because most of the CO_2 has been disposed of—and, if the suction is strong enough, blood may even flow back along the umbilical vein. And so the sequence of cycles continues every few seconds while the contraction lasts.

What effect decompression exerts when superimposed upon the contraction cannot be determined at present, for we have not succeeded in recording uterine pressure at the time.

The above attempt at explanation is made because of the striking improvement in quality of decompression infants. It is also of importance to try to arrive at the correct technique of administration in pregnancy, e.g. the degree of decompression and the duration and frequency of sessions. Reducing the resistance of the abdominal wall by stretching is undoubtedly fundamental, but insuring Braxton Hicks contractions that are isotonic is primary. Decompression probably excites Braxton Hicks contractions both because it effects change of abdominal cavity shape at the time and afterwards reduces abdominal wall resistance by stretching.

CONCLUSIONS

1. (*a*) Facilitation of labour occurs in the first stage. Over 50 per cent of primigravidae have a first stage lasting less than half the average time, i.e. under 7 hours. This is from the very earliest onset of labour. Thirty-seven per cent had a first stage of under 5 hours. The corresponding percentage figures for controls were 24.5 and 13.5, both highly significant.

   (*b*) Outlet decompression in the second stage reduces the total of work required for expulsion of the foetus, because the resistance of the levatores ani and the perineal muscles is reduced. The foetus is not used as a battering ram and the stresses acting on the head are minimal.

   (*c*) The placenta is delivered in 20 to 30 seconds after separation, without manual pressure. The postpartum uterus is exceptionally firm and appears to be exsanguinated by decompression. It is not yet known with certainty

whether the atonic postpartum uterus can be treated effectively by decompression.

In both (*b*) and (*c*) it is necessary for the uterus to lie over the pelvic inlet so as to seal the pelvic cavity from the rest of the abdomen.

2. (*a*) Pain relief in the first stage of labour was a prominent feature—77 per cent of parturients had 50 per cent or more relief, 41 per cent 70 per cent or more relief. Nine had under 30 per cent relief, but six of these had rather tumultuous labours lasting four hours, or less.
   (*b*) Pain at menstrual periods is brought under control and relief lasts, often for the rest of the period but in the majority of severe cases for several cycles. Secondary dysmenorrhoea does not appear to respond, but, in primary, a series of 93 gave a result that was excellent in 22 per cent, good in 52, satisfactory in 20 and unsuccessful in 6 per cent.
   (*c*) For acute backache decompression gave an excellent result in 24 per cent, good in 60, satisfactory in 12, and unsuccessful in 3 per cent. The average number of 30 minute sessions given was 5, with a range of 1 to 10. Decompression was 100 to 150 mm. Hg below atmospheric pressure.

Simple backache is more resistant to therapy than dysmenorrhoea, and perseverance is often necessary. Follow-up treatment is likely to be required at intervals of 6 months. The immediate effects are pleasant and give a sense of suppleness of the trunk.

In (*a*), (*b*) and (*c*) the effect of decompression upon the thorax is to produce an exceptionally deep inspiration which causes pull upon the tendinous slips of the sacrospinalis. It also stretches the rectus in proportion to the bulge of the abdomen. In acute backache the focus of irritation is local, but in the other states the rise in sacrospinalis tension is due to a reflex from uterine receptors.

Patients with backache have come to our clinic for treatment over a radius of 200 miles. Orthopaedic surgeons have shown an encouraging interest in the use of the method, several physiotherapists in private practice use decompression equipment daily, and nurses at home and abroad have made regular use of available apparatus for the treatment of their own dysmenorrhoea.

3. Experience with the new-born has forced the conclusion that decompression during the first stage of labour saves the foetus partly or wholly from hypoxia. The babies are pink and vigorous, in circumstances where depressed vitality has been the rule. This observation is supported by the fact that the foetal heart during first-stage contractions does not change in rate and force and, even more so, that bradycardias of under 100, sometimes accompanied by an irregular rhythm and the passage of meconium, have been restored to normal and in all of a small series delivery of a vigorous baby has followed some hours later.

In respect of pregnancy, on the other hand, we submit—and there are many who hold this view, although it is by no means clear on what they base it—that hypoxia is present in the last trimester and that it must be the rule in the last 8

weeks of pregnancy. Braxton Hicks contractions increase foetal blood flow and gaseous exchange at placental level, according to our hypothesis. In the absence of frequent Braxton Hicks contractions exchange must be very poor; but it can be shown that decompression during a contraction, in pregnancy as well as in labour, speeds up the reaction considerably. Quite apart from this, decompression during pregnancy promotes uterine action that appears in the form of mild Braxton Hicks contractions and heightened vitality of the foetus manifested by an appreciable increase in movements.

Theoretical treatment of the intervillous space is the foundation on which our hypothesis rests, but the predictions made three years ago have been justified by our findings in a harvest of 500 babies of which 250 have been tested to date.

Thus the question arises whether an element of neuronal damage or suppression of dendritic development is not a feature of the last trimester in *homo*. If there is a probability of 3 in 10 that our interpretation of the present information is correct, there will be full justification for carrying out trials on a wide front in an endeavour to improve foetal oxygenation and so benefit the central nervous system. Abdominal decompression is the only device with which one can even remotely hope to correct the *status quo*.

PART SIX GENERALIZATIONS AND PERSPECTIVES

THE FACTORS that, experimentally, have been shown to cause neuronal damage are two in number. Anoxia is first; but its effect is not instantaneous. The other is ischaemia which, whenever there is total interruption of the circulation, acts immediately. In the latter the immediate cause seems to be due to an accumulation of metabolites, an inadequate supply of glucose and increased acidity. An animal reacts perhaps twenty times as rapidly to total ischaemia as it does to suffocation. In the human foetus *in utero* it is impossible to estimate the relative importance of anoxia on the one hand and the group of factors resulting from an insufficient circulation on the other.

In asphyxiated animals, functional loss computed from the ability to learn has been studied by Becker and Donnell (1952) and Hurder (1952), and resultant structural damage demonstrated by Windle *et al.* (1944) and Ranck and Windle (1959). These researches have a precise bearing on the question of hypoxia during pregnancy in *homo*. The great volume of work carried out on experimental hypoxia and asphyxia is, however, not equally to the point.

Contemplation of the range of observations possible in human beings brings the realization that little can be done because of the impossibility of employing controls. Even the systematic accumulation of clinical and pathological information in known cases of asphyxia is at most fragmentary. Nevertheless, those paediatricians who are attracted to neurology and are attached to schools and similar institutions caring for the victims of cerebral palsy hold that anoxia lasting a few minutes will cause significant neurological damage. It is generally believed that, in the adult, deprivation of oxygen for 4 minutes causes irreversible changes in the neurones.

Birth trauma and asphyxia, and spastic children in particular, have received increasing attention in recent years. In the clinical field this development inevitably followed in the wake of the conquest of the problems of difficult midwifery, not only of the Herman type; but complications rectified by blood transfusion, the triumphs of premature infant care, and Caesarean section to save the foetus previously doomed, have brought the achievements of the accoucheur to fulfilment. This new midwifery seems to have found its inception in the London area in 1942. The perinatal salvage rate by comparison with pre-war days has been prodigious. Our own corrected still-birth rate at the Queen Victoria Hospital since 1950 has been just over 5 per 1,000 and the neonatal death rate well under 10.

It was in the nature of things that this advancement in favour of the baby would dominate the future scene in Obstetrics. One's own opinion was, that Obstetrics could be surrendered to personnel working under direction and that the old type of obstetrician should join forces with the paediatrician or disappear from the scene.

The view was that these partners, obstetrician and paediatrician, would direct themselves to the reproduction of improved qualities, as is standard practice with animal breeders. Nothing came of this. Perhaps there were too few children's specialists: at any rate they remained preoccupied with morbidity. This again departs from that which prevails in animal husbandry. The urban veterinarian, on the other hand, is possibly heading in the direction of the paediatrician!

There was an endeavour referred to as Child Health, but this was essentially didactic. No one seemed to dare to be a breeder of men. Perhaps this was traceable to the conscience evoked by the more violent side of Eugenics, as it is called. At most one formed the simple judgment that bonds should be forged between ourselves and our paediatric brethren. This new field was, therefore, completely outside our orbit: it was quite fortuitous that the decompression device fell into our hands; and the present endeavour is ironical indeed, for there could have been no more than a subliminal influence urging us in the direction we have taken.

It is important to realize that a tremendous stream of relevant research flows through the animal laboratories and the field of foetal physiology.

On the human side rich facilities have been created for the study, empirical and aetiological, of cerebral palsy. There is a resurgence, an awakening in fact, of interest in the reproductive product. So much basic work is being done and there is such collaboration between laboratory men and clinical research exponents that such symposia have been held as the one at Princeton on foetal oxygenation. The preoccupation with this subject is very easy to understand, *vis-à-vis* obstetrical progress.

What is below the surface, however, is a stirring of something exotic, perhaps rich and strange, which begins to look toward the people, the population and its biological welfare. When the quality of the persona as a biological unit is improved, the state must surely recognize this as its main asset. This is as it should be, for progress in anatomy and physiology is what it is because of the recognition of human needs.

The reproductive physiologist's interest is in the lack of oxygenation. This is anoxia, which kills rapidly. It comes by the way of asphyxia which in its duality of connotation represents circulatory as well as respiratory function in the placenta. The third and least abnormal degree of the syndrome is expressed by the term hypoxia. In so far as the interest is confined to conditions compatible with survival, hypoxia is the main theme.

The question to be dealt with is the frequency or otherwise of foetal hypoxia. Attention to this factor is only fortuitous and is restricted to the circumstance of labour. Its presence is recognized in certain morbid states during pregnancy, but virtually nothing is or can be done to alleviate it. There is no prophylactic approach, because its onset in pregnancy is unpredictable and there is in fact no means of prevention.

Thus it comes about that no serious consideration has been given to the possibility of the shadow that it casts irrespective of whether intervention has been solicited or not. There is, in any event, no means of influencing it. The possibility of its existence has had to be ignored. During parturition, on the contrary, certain

steps can be taken to alleviate it and immediate delivery, by section for example, will bring its action to an end.

Assali's figures show a poor blood circulation within the foetus and a poorer oxygen supply. My own arguments are in the direction of a poor placental circulation, of stagnant anoxia and consequently anoxic anoxia in the foetus. This applies in the last 10 weeks of pregnancy, but more acutely in the last 8 weeks. However, the lack of increase in bulk of the placenta does not necessarily mean a total circulatory decline, if the minute volume at this time is effectively increased.

If some degree of hypoxia occurs—and there is no ground for supposing this other than that just stated—we must examine the question of whether an infant can make up developmental loss after birth. The cerebral cortex is such a delicate structural organization, possibly more so than any other in the whole biological field, that impairment at any stage can hardly be regarded with complacency. Any damage or retardation will prevent the system from reaching far. Contemplation of the mental aberration and instability that is seen everywhere leaves little doubt of what has happened.

No informed person will deny Man's consciousness of his weakness, not so much physical as mental. How sublime to have infinite resources on which to draw! On this point the question of the suspected retardation of development arises. That is fundamental. Why is there not greater equality between individuals? Among healthy children the performance of other organs and systems is strikingly similar, but the one that varies characteristically is the seat of the intelligence. The facts would have been easier to understand if the system that presides over the rest was more stable, in fact uniform. Such is not the case. But is that not surprising?

The variability in cortical function may be due in part to genetic factors. The idea that it is based on a chance arrangement of genes seems to be far-fetched, and the stimulus of such a catalyst to neuronal growth and its linkages is something outside of one's experience. The geneticist's view is unnecessarily speculative (*see* Appendix 3, p. 104). Respiratory and metabolic requirements are more basic to the present consideration (*see* Appendix 1, p. 101). The high performance of the neurone may be glimpsed from the potential of the association areas in respect of memory, discrimination, reasoning—not to mention the production of speech of the question and answer kind (*see* Appendix 2, p. 103).

In so far as the genes play a part, why does an ontogenetic decline occur? There must be primary genes that have not been lost. Practically everyone can speak. *Homo* has shown no decline in this primary acquisition. Sometimes he speaks well! Is it a genetic matter that the highly gifted child has a great command of words, that he has this convenient faculty for the communication of his observations and judgments? What further is the basis for the normal vocabulary of five words at 17 months being hundreds sometimes, and thousands very rarely? Surely, it would be profoundly interesting to know whether this process of maturation could be influenced artificially.

There is now the possibility of making the oxygen supply to the foetal cortex optimal. Let us see how far this natural addition will take us.

The clinical indication of oxygen lack is a change in the foetal heart rate and the passage of meconium, but the seat of damage is no other than the brain. When an adult is suffocated, it is his brain cells that are endangered, although on the practical side one is concerned only for his life. However, we know what neuronal damage results neither in such cases nor in infants resuscitated after drowning. While it is inevitable that neuronal loss must occur, it is connived at. It never manifests itself noisily. Even in the victim of radical neurosurgery it is very striking that his brain functions extraordinarily well and that he does not give any impression of being sub-human.

It seems that a person whose behaviour is restrained and who keeps well within his intellectual limits—who perhaps even presents a facade of specific conduct to hide his shortcomings—will pass muster almost anywhere. If he has insight and some degree of judgment, a superficial observer will accept him as human, even reasonable. He is unlikely to be schizoid and may thus have an advantage over the majority of his associates.

One step further down the scale brings us to the dullard, another to the mentally deficient. Windle speculates with the idea that these latter may be like some of his asphyxiated guinea-pigs, animals that in fact showed a loss of neurones. These situations have been accepted for labour. What has not been considered, as suggested, is the degree of hypoxia that prevails during normal pregnancy itself.

Reproduction in *homo* on the basis of natural selection has been a dismal failure. When a high degree of cerebral development appears, it does not climb higher as would be expected generally: it reverts, as it is expressed, to the mean or mediocre. One is driven to suspect the environment of the zygote for being responsible for this decline, not the zygote itself. That is not to say that the genetic influence fails, for it is a commonplace that the highly gifted child is born almost exclusively to parents who are richly privileged and endowed. Even this selection does not raise the general standard appreciably, for so many mediocrities fill the ranks where the elect might have been expected to appear.

It may be said that Nature is likely so far to go into decay as to have defaulted seriously in the oxygen requirements of the neurones of the cerebral cortex. Among others, a young ophthalmologist taxed me with this improbability after a lecture. He returned after some minutes, looking very thoughtful, and said that there might be some truth in my contention, having regard to the numerous aberrations in the human optical system which he claimed was the most perfect in existence.

The excellent figure of only 26 per 1,000 live births dying in the United Kingdom in childhood contains the sting that the precise causes of these deaths are not reliably known (Davis and Tizard, 1961). These authors go on to say that most perinatal deaths are attributable to two causes, one being "environmentally determined asphyxia operating before or after birth". Mott in her review (1961) of the Symposium of Princeton published under the title 'Oxygen Supply to the Human Foetus' uses the phrase "the newborn infant who is usually grossly asphyxiated at birth". Clement Smith (1959), after considering the evidence in favour of foetal hypoxia as "physiological" and the vulnerability of certain parts of the central nervous system to the anoxia that the neonate can tolerate, sums up the

position thus:

> Certainly one may speculate as to whether such resistance to anoxia as probably occurs in the human foetus and infant may not be something of a mixed blessing . . . may allow the infant to exist as a damaged individual . . . Nature seems to have made provision for the survival of the species at a risk of incomplete development of some of its individual members.

The problem of how it happened, rather than why it happened, may be simpler, for there is a faint suggestion that this hypoxia may have been occurring in so many generations that the neurone has become conditioned to it and has acquired the present tolerance to it. Oxygen consumption varies at different levels of the rat's brain: it is high in the cerebellum and medulla but low in the cerebral cortex after birth, although these properties are reversed later in development. What is optimal is not known, but survival of the animal is no doubt more critical than maximal growth of the cortex.

Much of interest has been recorded by Terman and his followers; and the study of the gifted, highly gifted and exceptionally highly gifted—not only in individuals born in the first half of this century, but with close attention to geniuses of the past—has removed a great deal of prejudice and misunderstanding. The majority of responsible people even to-day are unsympathetic to the highly gifted child, will not admit his great value, and regard him as a liability. Such a child cannot be expected to benefit from what there is to offer in ordinary schools.

It has been an innovation to establish special schools for the gifted in the New World, and their value has been unlimited. What is a more serious deficiency is the scarcity of a teacher of the same mental stature as the student. If a crop of superior children can be produced, they will at the age of 15 be available to teach juniors. The juniors will be from 5 to 10 years younger. The atmosphere of the school will be different from what now prevails, and the gifted children will benefit from the clash of mind on mind and competitive exercises rather than explanation. Their school will operate as a repository and source of information.

An important feature of the gifted group is superior physique. It is the rule that, in stature and robustness, these young ones are far above average. They have appeared to lack ability for sport, because their companions have been several years older and have had pronounced physical advantages. The superior group, having highly developed intellectual interests, are easily discouraged from robust physical pursuits by the unequal struggle.

A second line of study in America is that by Gesell and his collaborators. Monumental work has been done in achieving average behaviour and performance at different ages and the rate of development of the infant and child is no longer a matter of conjecture. In dealing with the public, whence the human material comes, and with funds that may have the same origin, some care has been exercised in avoiding exaggerated preference for the gifted. The endeavour now contemplated deals with individuals still to be nurtured, not a *fait accompli* as with those already on one's hands.

The structural condition of the neurones was considered in Part Four. Very little is known about the extent of neuronal loss that can occur during development. What is even more basic is the inaccessibility of the neurone of the association areas to the observation of any kind of higher function. It will involve a great deal of work, but it is inescapable that a new vista is now open and that systematic histological studies of the cerebral cortex at all stages of development should be made. Clinical correlations will inevitably throw some light on neuronal integration, but the tissue will usually be obtained *post mortem*. Fortunately intercurrent death will not unsettle the stability of the neuronal pattern, for normal Histology is concerned with cells and tissues that are very nearly as perfect as those of the living state.

It will be difficult to determine accurately what the response of a neurone is to anoxia, but it may be possible to obtain indirect information in humans.

It is clear that there are many gaps in the information necessary to draw conclusions about the amount of oxygen that is optimal for neuronal development. Discussion on perinatal asphyxia always has a centripetal tendency towards those factors that are unknown. Contemplation of this unknown is overdone. One result is that the majority of authors say that there is a great deal of asphyxia, although valid evidence on the matter is lacking.

In contrast to this state of affairs, the application of decompression gives clear indications that unequivocal asphyxial states are improved. In the face of this, decompression cannot be ignored, and it would be a wrong approach to the present problem to discard it. Why indeed should it be discarded? It is another tool. It is a weapon. It is something that also sheds a little light where information is completely lacking. For that reason, then, it must be used *nolens volens*.

The constant presence of hypoxia in the last trimester and its possible adverse influence on neuronal development is a problem quite different from that of parturitional asphyxia. It has been accepted as a challenge because of the evidence that points to its existence, and because of our prediction (Heyns, 1958) and of what is found in those infants whose mothers have had decompression in pregnancy.

It is a problem more far-reaching and of considerably greater importance than the short term anoxia of labour: it lies within the sphere of Physiology rather than of Pathology and is amenable to control, and even improvement, on a much wider front.

The present position is, quite emphatically, that there is no evidence in *homo* to prove that, and perhaps no means of knowing whether, hypoxia really produces neuronal damage. Anoxia unquestionably does so, but it is quite definite that to-day we do not know anything with certainty about the hypoxic peril. There might possibly be such a mechanism, but it cannot be substantiated. On the basis of our experiments, however, there is a suggestion, certainly at least a suggestion, that hypoxia might impair the neuronal complex.

This would be extremely difficult to demonstrate or prove, but with decompression baby controls it is conceivable that autopsy material may become available and reflect fundamental differences.

If pursued, the present argument might reach the point at which one can say

that thought is imponderable and, in the same variety of sense, that at present hypoxia and its effect on the neurones is an imponderable.

Now, if that is the case, one could abandon the position and not trouble about it further; but we ourselves are going to deal with it for the simple reason that we get certain results. If in the process there is some infringement against the more rigid methods of science, let the device be exploited merely to see what can be produced in respect of the babies. Results thus far have been very encouraging and, if something substantial is produced without the philosophical support of science, what does it matter? There are serious limits to the well-worn methods of Science who is a servant—not the master. The proposed endeavour is no more than a reconnaissance, but the results might fortify man a little against his ignorance.

It is necessary to bear in mind that the brains of rabbits, guinea-pigs and other mammals—even the primates—are not so highly developed and not so necessary for high function as in human beings. Bringing animals into comparison with humans here is not profitable. The human cortex is a delicate, refined, elaborate and elegant system of dendritic development of the association areas never seen in animals. If neuronal impairment follows anoxia in animals, damage is much more likely to occur in human beings. It would be a matter of blind prophylaxis to avoid hypoxia. With prophylaxis the objective is never the eradication of something actually causing damage, but the erection of a screen through which the thing cannot penetrate. Let the approach accordingly be based on preventive measures!

If the state of the central nervous system at birth is superior, the starting time of mid-pregnancy rather than of full-term should be the reference point. The judgment might then be that it takes about five months of development for the neurones to reach the new-born point. The neonate that has 140, perhaps 170, to the 100 of the average, is progressing rapidly. This seems to be preferable to the view that the average child is a little behind but will catch up, or may do so. One does concede that the speed of development should be the same after birth, whereas oxygen lack made one slower than the other before it. It is important to recognize the structural implications.

If myelination can be shown, by means of motor tests, to be below average during the first weeks of life, there must be some qualitative fault in the central parent neurone. There must have been cytological impairment by some factor such as hypoxia.

The next step is the presumption that the dendrite system will be inadequate, and develop accordingly. It is difficult to picture it amounting to much later—functionally if not structurally. The third inference is not requisite to neurological significance, but it has a secure place in the probabilities. It is that the offending factor may indeed have killed some neurones and that there is in fact localized neuronatrophy. How far-reaching the effects will be is difficult to determine, although the potential dendritic loss is three-dimensional; and the first step considered above may involve the complete loss of a limited number of neurones rather than the loss of lustre in a single cell.

On this foundation there is the situation that the muscular action expected of the neonate may be imperfect in speed and strength. Thus it is difficult to imagine

the circumstances in which retarded neuromuscular function in the neonate can reflect anything but a poor prognosis. The delusion that backward children have caught up with the superior members of their age-group may be explained by the extreme difficulty that untrained parents and teachers have in assessing the child's intelligence. McGraw's testimony reveals that a comparison of infants continued from the early months onwards maintains the same relative order of merit later.

Our White decompression babies are derived from Provincial hospital material which comprises half the births in a metropolitan centre. The altitude is 6,000 feet above sea-level and the climate is exceptionally healthy, being sunny and for over half the year of very low humidity. The comparison with control babies from the same clinic leaves no doubt about the superiority of the decompression infants. In a few subjects decompression was not tolerated, neither during pregnancy nor in labour. When the labour time approached 10 hours, or pains were violent, the failure of decompression protection was obvious in the infants. They alone fell below average. It must be emphasized that these particular mothers were singularly poorly suited for reproduction, in a manner well known to obstetricians.

It is still a matter of conjecture as to which is the stage when the protection of decompression is the most effective. There is little doubt that the short labours, preferably those under two hours, are, in our experience, the most acceptable. It must be understood that the pregnancy decompression plays an important part in reducing labour time.

Whether there is a tendency to a chain reaction with the prenatal treatment is unknown. It will be recalled that Braxton Hicks contractions are provoked and foetal activity augmented. This continues for some time, of the order of hours, but it is difficult to observe, and we have not had an opportunity of studying it systematically. While these two factors are present, the rate of both oxygen transport and the circulation of foetal blood are raised. Although the following observation is purely empirical, a very small amount of physical exercise daily seems to raise the efficiency of the sedentary worker. The person able to translate such physical activity into recreation is at luxury level when 10 per cent of his time is devoted to sport and he is privileged when the figure is 5 per cent. Half an hour a day, although not optimal, would serve to keep him in good condition for mental work.

It is not known what katabolic advantages result from the induced increase in blood circulation, but there is the possibility that the foetus may benefit from this mechanism.

An important point to consider is whether an improved circulation through the foetal brain is not relatively more effective than the mere supply of oxygen. In *homo* oxygen can be withheld for two minutes, whereas the total interruption of the blood-supply to the brain causes unconsciousness in six seconds. Mental dullness or clouding of consciousness after awakening from sleep is abolished by a few deep breaths, and similar stimulation of the cerebral blood flow in the foetus may heighten its vitality. It may be objected that this is by no means in the nature of things that are best for the foetus. However, nutritional and respiratory processes

are not regarded as operating adequately in suppressed states like shock and suspended animation.

The views expressed about normal foetal oxygenation may prove to be incorrect. The promise contained in raising the oxygen supply is so profound, however, that a systematic investigation of the problem is indicated. Certain phenomena have been discovered and shown to have some degree of constant regularity. The manner of conducting the research and what is required is set out in the final chapter, and it is now possible to proceed with the third and final stage which will provide a conclusive answer.

Such is the nature of all investigation in the biological field of dynamic events, and the present research has not gone beyond the second stage.

APPENDIX I

Relevant Biochemistry

The development of the central nervous system has been depicted as being of the very simplest nature chemically. For the present research it is believed that no purpose will be served by taking into account the more precise knowledge about metabolism, the blood-brain barrier, tissue respiration and even genes. However, as we have built our hypothesis on a crude foundation and have considered only anoxia, ischaemia and the blood sugar level, it is advisable to pay attention to more delicate mechanisms which may harbour functions that are both protective and controlling.

Some seven years ago a symposium was held at Magdalen College, Oxford (Waelsch, 1955) to consider the biochemistry of the developing nervous system. From this and later writings one has formed the following impressions.

The energy requirements of the brain are obtained partly by glycolysis, but mainly by oxidation of glucose. Glycolysis provides only about 10 per cent of the potential energy of the combustion of glucose. Experiment indicates that glucose is the only substrate normally utilized *in vivo* but observations with tracer elements show acetate, glycine, methionine and even lipides to enter the brain, albeit slowly. The blood-brain barrier acts by reducing the rate of entry of metabolites.

Energy is needed by the brain for the performance of osmotic work, for the transport of ions in the neurones, for the synthesis of acetylcholine, and for other processes connected with the growth and activity of the cells. There is a metabolism of function as well as of growth. We have no means, however, of assessing the amount of work performed by the brain.

In not using fats and other metabolites, brain is unlike muscle and most other tissues. From embryo to adult its main fuel is glucose. It is characterized by a high over-all oxygen consumption. Metabolic activity is generally highest in phylogenetically newer regions, e.g. cerebral cortex and cerebellum, which represent the highest levels of functional activity in the brain.

Enzymic development is a function of which account must be taken. It tends to be proportional to cytological changes in the brain. Thus during a particular 'critical period' when the outgrowth of dendrites is most active the activity of

certain enzymes is very great. The highest oxygen uptake is in the (1) cerebellar cortex, (2) corpus striatum, (3) cerebral cortex, (4) thalamus, (5) globus pallidus, and (6) white matter of brain.

It was suggested that cyanide produces unconsciousness in a matter of seconds by preventing the oxidation of metabolites in the cells of the brain. This is unlikely to satisfy everyone, but it is mentioned to call attention to the delicacy of the chemical balance in the nervous system. The immediate effect of cyanide is no different from the dramatic suddenness of total ischaemia. Ammonia has also been shown to be very toxic and, as it is liberated during activity, its removal is important.

Whereas bile pigments, penicillin and many other substances readily penetrate other tissues, they do not cross the normal blood c.s.f. and blood-brain barrier. A pH difference of 0.2 is maintained across the barrier. For most substances that do enter there is an exaggerated delay. On the other hand, glucose appears to be actively transported, whereas oxygen and carbon dioxide do not appear to be in any way impeded in crossing the barrier. In morbid conditions the barrier may break down partially and permit a rapid entry of ions, organic acids and other substances. Abnormal function may be due to an abnormal barrier. About the growing brain there is unfortunately this reservation that "at the present time we are unable to assess the extent to which, during the developmental periods under investigation, vascularization changes and blood flow may affect the influx of substances from the blood into the brain" (Waelsch, 1955).

It has to be accepted that the mutual interaction of O_2 and CO_2 cannot be explained. CO_2 is utilized by animal tissues and may yet prove to regulate the rate at which physiological oxidations are carried on by the body (Ruch and Fulton, 1960). So also Lorente de No' has emphasized that CO_2 is essential to the normal behaviour of nervous tissue, because enzyme mechanisms of nerve use the stores of metabolic energy more readily in its presence.

Full-term babies seem not to suffer retrolental fibroplasia. That it has occurred in prematures seems to point to a degree of intra-uterine hypoxia *vis-à-vis* the high extraneous oxygen tension administered.

It is unusually difficult to discover how the foetal brain differs from the adult in metabolic respects. Glucose, O_2 and CO_2 are definitely the overriding metabolites. With the increased circulation probably present during abdominal decompression the supply of glucose is plentiful and oxygenation is increased. For the present purpose, therefore, these comparatively crude elements are all that it is worth considering when other mechanisms seem, according to the available information, to be less fundamental. The developing arterial system of the brain is quite unimpressive and any measure which extends it and the oxygen exchange may be accepted as exercising a primary influence.

APPENDIX 2

Early Development in the Cerebral Cortex

A great deal of thought and speculation concerning neuronal development was expressed in Part Four. This is of primary importance in the main theme of the investigation and is of the essence of what is believed about improved foetal oxygenation during the third trimester. The submission is that the placenta is inefficient and in a state of decay during at least the last 8 weeks of pregnancy. The objective has been to improve the circulation of blood through the brain and the supply of oxygen to its developing cortex.

It is generally believed that neurones have reached their maximum number before birth and that, with advancing age, there is a progressive loss of neurones accompanied by gliosis. But there is an important development in the form of the increase of dendrites which reflects growth of the cell systems and the cortical network that gives to the brain its unique complexity. Increase in brain weight is due to this growth in size of neurones and of the cerebral blood-vessels, but principally to myelination. Continued growth after the first year of life is due to myelination.

White fibres of the cerebral hemisphere do not all acquire their myelin sheaths at the same period. The process commences in the eighth month of foetal life. The afferent fibres become medullated first. Myelination of the great, efferent cerebrospinal pathway is not completed until the middle of the third month after birth.

It is generally believed that only medullated nerves can conduct impulses. Accordingly movements in the first two months of life are reflex and depend on the spinal cord and brain stem.

Last of all do the fibres of the large association areas (frontal, parietal and temporal) become myelinated. In these areas, the process may not be finally completed until the 18th year or even later. Complete mental development is not possible until these areas can be utilized.

Conel (1939, 1941) made a beginning with structural studies of the human cerebral cortex and published a volume on the cortex in the normal full term foetus and another for the age of one month. This study is of inestimable value and it is hoped that systematic information of this calibre will be available in time to reflect brain development *in utero* and in the first years of life. Conel had such a programme in mind and hoped to be able to correlate development in structure with the development in the behaviour of the growing child. His reproductions of neurones are an inspiration.

The changes found between birth and one month later are unmistakable. In the isocortex of both, the nerve cells are arranged in six horizontal layers, and each layer has the same types of cell in it everywhere. In the allocortex the six layers are not evident. The features on which Conel made a comparison between the two ages were:

> (1) width of the entire cortex, and of each horizontal layer; (2) number of nerve cells; (3) size of nerve cells; (4) condition of the chromophil

substance; (5) neurofibrils; (6) size and length of the processes of nerve cells; (7) pedunculated bulbs; (8) varicosities; (9) size and quantity of exogenous fibres; (10) state of myelination. A change occurs in each one of these criteria with advancing age (Conel, 1941).

Everywhere in the isocortex the processes of the extra large pyramidal cells in layer V show the greatest increase in calibre and length. Its width has also gained more than that of other layers. In the anterior central gyrus the dendrites and axons of these giant pyramidal cells are the largest and longest in the entire cortex. In the region of the hand the processes show the greatest increase in calibre and length; those in the region of the trunk, shoulder and arm come next.

In smaller measure than the above, but quite uniformly, there is evident a slight advance in the development of the nerve cells during this first month of life.

Afferent fibres radiating in the white matter towards the cortex do not, at this stage, always reach it. Of those that do enter, the majority end in layer VI, a few reach layer IV and an occasional one is seen in layer III. Within the cortex very few of these fibres are myelinated, nor does myelin extend beyond layer VI. It is thought to be improbable that these vertical fibres are capable of conducting impulses into the cortex.

The primary matter of interest in the present inquiry is the possible architecture in advanced babies of their cerebral cortex at birth and later. It is difficult to see how an active state of growth in a maximum number of cells can fail to surpass numbers reduced by loss. The latter in addition often exhibit impaired vitality.

APPENDIX 3

Heredity in the Decompression Babies

The question of the social or other stratum to which the mothers of the decompression babies belong has been asked by a variety of friendly critics. It is easy to understand the reason for this concern, but obstetricians cannot be expected to set so much store by control in this direction as do those who are unfamiliar with the obstetrical aspect of the general field of reproduction. The critics seemed to lay too much stress on the inquiry whether our pregnant women volunteered for decompression or had it imposed upon them. The precise origin of the mothers was shown in the analysis presented in Part Four. The ANC Series may have been derived from mothers who were on average superior to the mean of the total of our antenatal clinic patients, although we do not think so. We do not wish, however, to contradict this possibility.

One has been aware that some women are more liable to complications during gestation and parturition than others. Some are indeed very poorly suited to reproduction. Foetal development in these is likely to be impaired and one is not enthusiastic about the effect of pregnancy decompression on the quality of their babies. While the baby is expected to gain through use of the device, it is a matter of disappointment to the observer if unfavourable extraneous influences come into play.

Something that was unexpected happened in the Sanctuary Series and came to our notice recently. As these mothers were unmarried, many of them were not ideal material for our purpose. In addition, one has felt that the circumstances for the duration of their pregnancy were not to be compared with those prevailing in a happy home. Our belief was that optimal development might be impaired in these unfortunate cases.

One of the girls, herself of I.Q. 55, produced a baby which we assessed at F50; but another of I.Q. 60 had a child assessed by us at F129. This was surprising, but it called attention to itself when the infant of a special patient derived from the topmost intellectual bracket scored the same. It has become discouraging to choose parents in an endeavour now being made to obtain babies with scores upwards of F180.

It seems as if it would be more certain to offer decompression to parous women, preferably two-paras, in whom something of their aptitude for reproduction can be judged. It also seems as if our best babies have been born to ordinary women —pious women perhaps, and not uncultured—but not from the highest strata of opulence and professional distinction. One has much of the father's intellectual status in mind, but the hereditary potential of this might be lost if the mother is not well adapted for reproduction. This possibility may not be so rare as is commonly imagined.

Certain facts about the Sanctuary Series are revealing. It was possible to divide the mothers into five classes, viz. I. exceptionally intelligent, II. intelligent, III. normal, IV. slightly retarded and V. greatly retarded.

Class I had 5 mothers having babies with F range 117 to 146 and mean 131.

Class II had 3 mothers having babies with F range 122 to 154 and mean 136.

Class III had 22 mothers having babies with F range 111 to 159 and mean 133.

Class IV had 3 mothers having babies with F range 104 to 173 and mean 142.

Class V had 3 mothers having babies with F range 50 to 129 and mean 104.

Class Unknown were two mothers.

Classes IV and V had babies with F 104, 150, 173 and 50, 129, 133 respectively.

These phenomena will have to be studied more carefully. It seems as if something has been lost by not assessing the intelligence of groups of parents and their offspring for three generations. Much may still be learned. It is easy to accept the inheritance of structural features rather than functional complexes. Genes may be protective or activating factors, but are likely to operate simply by forging a chain of many links. The network of the association areas of the cerebral cortex, or exceptional intelligence, are unlikely to be influenced directly by genetic activation, but may result from a very complex chain effect. The observations referred to above may give some direction to a better understanding of the inheritance of intelligence. According to our findings the problem is not quite as simple as Sir Julian Huxley suggests when he states that we might produce a superior intelligentsia if we could breed intensively from the best university types.

PART SEVEN FUTURE RESEARCH

AT THE END of Part Six it was stated that the second stage of the present investigation had been completed. This conclusion was based on the view that an inquiry is initiated by the observation that a phenomenon or trend makes its appearance in compelling fashion. Its recurrence will be noticed in the field with which the observer is familiar whether it be related to the mathematical sciences or the biological. The second stage is concerned with determining the reality of the phenomenon, defining it and devising means to study its properties. From this restriction there can emerge the final stage of the inquiry which will be carried forward on a wide front. The project will prove to be of value if some underlying principle is discovered.

It is hoped that the present research which has been in progress for eight years has reached the final stage and that a contribution to knowledge might follow further endeavour.

Primarily nothing more has been gained than a simple mechanical device. But abdominal decompression is a useful tool. Its usefulness up to the present has been indicated, but its effect on certain states has merely opened new avenues for investigation.

The complex first stage of labour should be studied in order to give the accoucheur some morphological understanding of what is involved. The mechanics of the second stage has been suggested, but substantiation is needed. The precise effect of both these stages on foetal morphology is still a matter of conjecture but should be properly determined. Much may be done by assessing non-decompression infants with the sole purpose of correlating their quality with easy and difficult labours.

The physiological state of postural muscles like the sacrospinalis group during tension states giving rise to no pain on the one hand and those where pain is continuous or even periodic (as in labour pains) should be determined. Backache is a complaint so universal that a study would be justified if it did no more than to provide the therapy for relief.

Problems such as these are only by-products of abdominal decompression. It is the promise of improving foetal development by this means that deserves attention. The primary things to be discovered are the efficiency of the human placenta relative to foetal requirements, more specifically the pressure flow changes in the intervillous space in response to different conditions, and much more information about the rate of circulation within the foetus and through the umbilical cord, large superficial vascular sinuses and villi.

These are mere examples; but they are not nearly as critical as may appear to be the case.

Inevitably, as so often is the case, the study with the strongest appeal will be more empirical. In addition to demonstrating the degree of validity of the present

claims, it will throw light on some aspects of reproduction, not the least of which may be the hereditary.

It cannot now be decided whether the device has been fully exploited or whether the therapeutic applications described are at present incomplete. It would, however, be most profitable to examine the scope of the requirements for producing and testing decompression babies.

Pregnancy Decompression Commitments

1. A substantial organization is required to attend to those having decompression daily or thrice weekly and later in labour. After some time there will be babies who are to be observed, tested and photographed periodically. The mothers are best given professional antenatal care by a member of the team.
2. It would be well worth having an expert to instruct on the technical side for several months to begin with. A warning must be sounded for the benefit of individual workers. A number of workers have claimed that patients subjected to decompression have felt and shown benefit in certain specific directions. A few observers have come forward with the most unexpected failures and disappointments and always counter-claims. It is impossible to analyse their technical peculiarities, and their denials of the claims of the majority impede genuine progress. The advice to anyone embarking on a new technique, and decompression is no exception, is that he should adhere closely to the equipment and method advocated by the originator or other expert. Preceding this step there is his desire to experiment with the particular device. If the trials are unsuccessful, the experimenter may reject the method; but he would design modifications only after having satisfied himself of merit in the original claims. Any other course than this is unknown outside of the field of Medicine where, however, the autonomy coefficient is very high.
3. Domiciliary and hospital decompression must be provided during pregnancy. This implies substantial expenditure on sets of equipment. Decompression in labour may be given in the home in a small percentage of cases.
4. At an early stage it must be determined whether the semi-erect or the horizontal position is best during pregnancy D runs and an intensive study made of the duration and strength of Braxton Hicks contractions following a run. At this time observation of the foetus and its heart rate may be of value. The findings will indicate how many daily sessions are optimal and also their duration. It may prove difficult to get co-operation for intensive pregnancy decompression for any period longer than two months. Once the value of the method is established, co-operation will be had from all.
5. An assessment of the intelligence and reproductive aptitude of all couples must be made, and material from all levels should be exploited. The fact that we have experimented with such people as the Bantu should be a guide to others. We are meanwhile greatly impressed by their performance.
6. A studio for assessment of the babies is necessary. Records should include motion pictures of babies when comparisons or subsequent demonstrations to others are likely to be of value. EEG. records should be made at each baby visit.

7. Unmarried mothers are a splendid source of material. Adoption of their babies has led to loss because the foster parents have often been domiciled in almost inaccessible places. A small institution for these babies is imperative. The children can be adopted at the age of five years when their value for study will have diminished considerably.
8. In conclusion, certain things are inevitable. It takes two years to produce a harvest of babies of which one has a little knowledge. It is likely to require another two years before firm conclusions are possible. Meanwhile the priceless gift of knowledge of normal infants will have been gained, a splendid *esprit de corps* established at one's antenatal clinic and a more rational obstetrics practised. As the truth about the quality of the infants is unfolded opportunities will occur to make comparative and controlled studies of the cerebral cortex.

Mental and Somatic

Our standpoint has led us to investigate foetal development on specific lines, and the findings will indicate to what extent our hypothesis is valid.

At this stage the inquiry is based on the view that intelligence depends on brain structure, more particularly on the integrity of the cerebral cortex. The hypothesis is that there is a patchy distribution of normal neurones, this being due to impaired proliferation or actual loss by atrophy or gliosis. No decision has been taken on whether the patchiness reflects good tissue which is lacking in some gyri or substantial loss in some regions due to death from anoxia.

It is suggested that midbrain and hindbrain can be expected to be adequately nourished where cortical development is satisfactory and that, in consequence, both physique and intelligence should be superior. Under adverse circumstances a preference for at least the vital encephalic centres may lead to physique of high quality in the absence of a satisfactory degree of cortical morphogenesis. The problem of foetal anoxia comes into strong perspective when one contemplates the considerable percentage of persons who exhibit—in addition to mediocre intelligence—even physical inferiority.

One does not subscribe to the idea that Holism rather than structure is the soil in which Mind develops and functions; nor is it necessary to invoke Genes or any form of Evolution for our purpose. Nor again does the effect of Environment on development dominate the situation. Whether these abstract elements are present or not—and whatever metaphysical conclusions may be reached about them—they do not affect the simple objectives with which we are concerned. We stand firmly by the idea that the cerebral equipment of the new-born or the infant is the foundation upon which his intelligence develops. With poor equipment his ceiling is low but with good higher, whatever secondary influences may come into play.

What is thought of as Personality is to us the reflection of a neuronal organization which is balanced, and not patchy, in such measure that the several components of the cortex are in equilibrium and able to function with facility. The individual would at the lowest computation be gifted and his physique would match the co-ordination of the structure of his association areas, in so far as they reflect judgment,

discrimination and the critical faculty generally. Objections to intelligence tests do not affect the present endeavour, for we seek an even balance in the individual, with minimal structural deficiencies; in truth one who will be specifically sapient.

Structural perfection will be sought with decompression during pregnancy. Improved oxygenation of the brain in the last ten weeks of foetal life is no doubt of paramount importance. The promotion of the blood circulation is cruder, because its benefits are more nebulous, except for one obvious factor. More realistic even than the reproduction of chromatin and the consequent formation of genes is the other use of amino-acids for the production of the nucleoproteins present in the nuclei of our neurones. These with phosphorus and other necessary elements would be transported efficiently for the proliferation of neurones whenever the pressure flow system improves: in the absence of the latter the high saturation of metabolites in the villus would lower the concentration gradient of these substances. Facilitation of the supply of such metabolites to the foetus, even if sporadic, could be significant even if the periodic increase in oxygen transport is not.

Improvement of the structural *status quo* is, therefore, the primary objective which in turn is expected to raise the child to a true *mens sana in corpore sano* plane.

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