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REFERENCES

ACADEMY OF DENTURE PROSTHETICS (1960). Glossary of Prosthodontic Terms. J. Prosth. Dent., **10,** Supp.

CRADDOCK, F. W. (1956). Prosthetic Dentistry, 3rd Edition. Henry Kimpton, London.

HELLMAN, V. (1927). Changes in the Human Face Brought About by Development. Int. J. Orthodont, **13** : 475-515.

HICKEY, J. E., WILLIAMS, B. N., and WOELFEL, J. B., (1961). The Stability of the Mandibular Rest Position. J. Prosth. Dent., **11** : 566-572.

NAGLE, R. J., and SEARS, V. H. (1962). Denture Prosthetics. 2nd Edition. The C. V. Mosby Co., St. Louis.

SWENSON, M. C. (1959). Complete Dentures, 4th Edition. The C. V. Mosby Co., St. Louis.

TALLGREN, A. (1957). Changes in Adult Face Height. Acta Odont. Scand., **15,** Supp. 24.

THOMPSON, J. R. (1946). The Rest Position of the Mandible and its Significance to Dental Science. J. Amer. Dent. Ass., **33** : 151-180.

THOMPSON, J. R. (1954). Concepts Regarding Function of the Stomatognathic System. J. Amer. Dent. Ass., **48** : 626-637.

WILLIS, F. M. (1935). Features of the Face Involved in Full Denture Prosthesis. Dent. Cosmos., **77** : 851-854.

SYNCOPE — A POSSIBLE CONTRIBUTORY MECHANISM

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FAINTING is a phenomenon which is frequent enough, and usually harmless enough, to be regarded by most clinicians as a trivial occurrence. However if a faint is prolonged, or if a number of faints follow close upon one another, a condition very like haemorrhagic shock ensues (Cameron, 1958). Cardiac arrest, which is sometimes thought to occur as a result of excessive vagal tone during a difficult tracheal intubation, may be a more grave manifestation of a simple faint. Moon (1957) also observes that "primary shock" is a neurovascular reaction which cannot be distinguished from fainting.

The symptoms of syncope* are sweating, pallor, bradycardia, nausea and sometimes retching, often followed by unconsciousness, which is generally transient. Objectively, in the minutes preceding syncope the cardiac output decreases, but the heart rate and the total peripheral resistance increase sharply. The blood pressure shows a steady but gradual drop. With the onset of the faint there is a sudden precipitous drop in peripheral resistance, heart rate and blood pressure. Cardiac output levels off or rises slightly (Barcroft *et al.*, 1944). This latter finding indicates that the fall in blood pressure is mainly due to the decreased peripheral resistance and is not entirely cardiac in origin.

Syncope is peculiar to humans (Payling-Wright, 1958) and usually occurs in the tense person, who displays marked sympathetic nervous activity which may rapidly be superseded by activation of the parasympathetic division of the autonomic nervous system. The whole mechanism

*For the purpose of this paper "syncope" is used as a synonym for "vaso-vagal attack" — a specific type of faint.

of fainting formerly has been attributed to the decreased cardiac output resulting from increased vagal tone, and the associated decreased peripheral resistance which is due to loss of sympathetic tone. This theory implies that there is no actual loss of fluid volume or of electrolytes contributing to the faint and its after-effects.

It is the purpose of this paper to suggest that the sequence of events and subsequent recovery indicate that a change in fluid volume and probably a change in electrolyte content may occur during the faint.

NERVOUS AND PSYCHIC COMPONENTS OF THE MECHANISM OF FAINTING

Prior to the onset of a faint the effects of the adrenergic division of the autonomic nervous system predominate over those of the cholinergic division. Thus, for example, the skin is pale and the heart rate and respiratory rate are increased: the "fight or flight" response.

Persons who are subjected to stress — for example a soldier standing on the parade ground for a long period, someone anticipating bad news or an individual about to be subjected to some form of noxious treatment — are liable to faint when they finally realize that an unpleasant experience is inescapable, and they surrender mentally. When this occurs, sympathetic tone is suddenly decreased and is over-ridden by parasympathetic effects; or possibly parasympathetic activity is markedly increased with relatively little decrease in sympathetic tone. The fact that the skin remains pale during the faint supports the latter possibility. Barcroft and his associates (1944) stated that it seemed very likely that the vasodilatation of fainting is largely confined to muscle arterioles. They found that while adrenalin produced vasodilatation in muscle, it did not produce the other phenomena typical of fainting.

A small child who will not surrender and accept the fact that he is to be subjected to an unpleasant experience maintains his fight or flight response (his sympathetic tone), and therefore is not liable to faint. This is well borne out by clinical experience. In the same way, once a person under stress has decided to resist mental surrender — to "fight or flee" — a cholinergic response is less likely to supersede the adrenergic response than would otherwise be the case. Boyd (1947) has repeated, perhaps not quite seriously, that shock is more often encountered after a lost battle than after a victory. There may well be a modicum of truth in this generalization and it also seems singularly applicable to fainting.

A cholinergic response is evaluated clinically by depression of the heart rate, which is brought about by but a small part of the parasympathetic nervous system, the cardiac division of the vagus nerve. There is little evidence that the rest of the parasympathetic nervous system is over-active: it may merely have relative over-activity as a result of loss of sympathetic tone. However, over-activity in the gastro-intestinal branches of the vagus possibly may play an important rôle in the production of syncope.

THE GASTRO-INTESTINAL COMPONENT OF THE MECHANISM OF FAINTING

Recent work by Brooks *et al.*, (1963) has shown that the mortality in dogs is reduced after induced haemorrhagic shock if hypertonic saline is infused intravenously directly after the haemorrhage. This work has focused attention on the importance of electrolytes in haemorrhagic and, perhaps, in all forms of shock.

During syncope, incomplete compensation for the drop in blood pressure results in cerebral hypoxia and loss of consciousness. Consciousness is regained soon after vagal tone is decreased. Some patients who have fainted, however, state that they do not recover a feeling of well-being until perhaps 24 hours afterwards. The delay in complete recovery might have been assigned to mild hypoxic damage to the brain; but there are no residual signs of such damage. It is perhaps more likely to be due to alterations in the water and electrolyte content of the blood and of the extracellular and intracellular fluids.

The nausea and retching which frequently accompany fainting may be a result of transient hypoxia of the brain; or they may perhaps be due to a stimulus originating within the bowel.

The turnover of fluids and electrolytes in the gastro-intestinal tract is a complex process, but it has been estimated that in twenty-four hours the water secreted into

the bowel exceeds eight litres and contains about 60 grammes of sodium chloride. The chemical and nervous mechanisms regulating the secretions are so interrelated that the fraction resulting from each phase cannot readily be ascertained. Thus it is not possible to determine the total secretion owing to the nervous phase. However, the estimated normal of eight litres for the combined chemical and nervous phases is not the maximum possible secretion during twenty-four hours. The total probably could be considerably increased.

Since the total blood volume of an adult is four and a half to six litres, with an osmotic pressure equivalent to 0·9 gm. of sodium chloride per 100 ml., a very rapid loss of even as little as 500 ml. of isotonic fluid into the bowel would represent loss of a substantial proportion of the total blood volume and electrolyte content.

Thus if at the same time as the heart rate is reduced there is increased secretion of fluid into the bowel as a result of vagal activity, a considerable decrease in blood volume and electrolyte content could occur.

This loss would be made good temporarily by redistribution of fluid and electrolytes in the intravascular, extracellular and intracellular compartments. On reaching the large bowel, the bulk of the water and electrolytes which has been so "lost" is reabsorbed; a gradual redistribution of electrolytes must again take place. The minor residual electrolyte imbalance may account for the delayed return of a sense of well-being.

A serious objection to this hypothesis is the large fluid and electrolyte turnover which normally takes place in the bowel, as mentioned above. An additional loss into the bowel of even a litre of fluid and eight or nine grammes of sodium chloride is no very great excess over the eight litres and 60 grammes that are normally secreted into the bowel in 24 hours. However it is postulated that the secretion that takes place in fainting is sudden, and the ordinary physiological mechanism which is geared to compensate for the physiological fluid and electrolyte turnover does not adequately compensate for this additional sudden and widespread secretion into the bowel. It might even be that there is some mechanism, reflexly linked to the act of taking food, which compensates for the loss of digestive juices into the bowel. Of course digestive juices are not secreted simultaneously into all parts of the bowel, so there is no massive sudden fluid and electrolyte loss.

Some correlation seems to exist between the occurrence of fainting and the time at which the last meal was taken. Fainting is less likely if the person has had a good meal before being subjected to stress. For this reason it was formerly believed that hypoglycaemia plays an important part in producing syncope. However this is improbable, because under stress the secretions of the suprarenal gland and glycogen from the pancreas rapidly mobilize glucose from the liver, unless liver glycogen has been depleted. The reason why a person is less likely to faint after a meal is therefore difficult to explain; but it may be related to a high level of tonic activity in the gastro-intestinal division of the vagus nerve, associated with bowel activity. The hypothetical mechanism mentioned above, linked reflexly to taking of food might be sufficient to compensate for the fluid loss of the faint.

The physical presence of food in the stomach and upper small bowel will stimulate the chemical phase of secretion. This phase possibly may act as an inhibitor of the nervous phase of secretion and so counteract vagal activity.

It is therefore suggested that fainting is not entirely a nervous and vascular phenomenon as is implied in the often-used term "vaso-vagal attack", but that there is an associated fluid and electrolyte loss comparable with that which occurs in more severe types of shock.

OTHER TYPES OF SHOCK

In the final analysis the untoward effects of shock, no matter how it is produced, stem from disordered tissue metabolism, which may result from cellular anoxia secondary to prolonged hypotension, toxic agents or lowered temperature. It has been said of secondary shock that hypotension results from a disparity between the volume of the blood and the volume-capacity of the vascular system (Moon, 1957). Such a disparity could thus arise either from a loss of blood volume or from

an increase in the capacity of the vascular bed, or both.

Loss of blood volume appears to be one of the characteristic features of shock, no matter how it is caused. Thus in haemorrhage there is a frank loss of blood from the body: In crush injuries there is extravasation of blood into the tissues; in burns there is loss of plasma from the injured surface and into the surrounding oedematous tissue; in intestinal obstruction, diarrhoea and protracted vomiting there is loss of fluid into and from the bowel; and in the shock following surgical handling of the abdominal viscera or accompanying acute abdominal catastrophes, there is likewise fluid loss into the bowel. In each instance the fluid takes with it a greater or less amount of electrolytes. It therefore does not seem unreasonable to postulate that a similar fluid and electrolyte loss, though on a smaller scale, might partially account for a smaller scale type of shock, the common syncope. The most likely site of such fluid loss is the bowel.

An attempt was made to test the validity of this theory by using a peripheral parasympathetic depressant (atropine) to prevent the onset of fainting. However subjects with a history of syncope could not be made to faint either before or after taking atropine.

Lewis (1932) administered atropine to a number of subjects soon after they had fainted and found that while he could eliminate the bradycardia, the low blood pressure persisted. This finding does not contradict the theory suggested in this paper.

CONCLUSION

Although a number of arguments have been advanced to support the suggestion that, in common with other forms of shock, syncope may be partly caused by fluid and electrolyte loss, it must be emphasized that the authors have no experimental evidence for this theory. The main problem in obtaining experimental evidence is the difficulty of artificially reproducing the conditions under which any particular person would faint, without resorting to blood-letting or other methods which are entirely foreign to the usual sequence of events leading to fainting.

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REFERENCES

BARCROFT, H., EDHOLM, O. G., MCMICHAEL, J. and SHARPEY-SCHAFER, E. P. (1944). Lancet, **246,** 489.

BOYD, W. (1947). "Surgical Pathology".

BROOKS, D. K., WILLIAMS, W. G., MANLEY, R. W. and WHITEMAN, P. (1963). Lancet, **7,280,** 521.

CAMERON, G. R. (1958). "General Pathology", 2nd Ed. Edited by H. Florey. London, Lloyd-Luke.

LEWIS, T. (1932) Brit. Med. J., **i,** 873.

MOON, V. H. (1957). "Pathology", 3rd Ed. Edited by W. A. D. Anderson. St. Louis, The C. V. Mosby Co.

WRIGHT, G. Payling (1958). "An Introduction to Pathology". London, Longmans Green and Co.