admitting the reality of the lesions described, it is difficult to follow the process of reasoning which convicts the degenerated nerve fibres of any share in the causation of the disease. If such a lesion were the true causative agent, or even one factor, by what magic do these cases recover so rapidly, so completely, and so permanently after the simple procedure of overstretching the constricting tissues?

Acute controversy has arisen as to the cause and pathology,^{12 13 15} in the course of which fact and fantasy have not always been clearly distinguished. Brown Kelly¹⁵ dissected two specimens and "demonstrated both muscular coats to be of uniform thickness and without any special aggregation of fibres at or near the cardia. . . . These findings seem to admit of only one interpretation—namely, spasmodic contraction." They certainly prove that hypertrophy of the muscle forms no essential feature of a case of cardiospasm, and effectually preclude any analogy with hypertrophic stenosis or Hirschsprung's disease, in both of which muscular hypertrophy forms a prominent and invariable feature. This analogy has gained a certain amount of credence by a parrot-like repetition, unsupported by facts. Pure theory is admissible if it helps to formulate treatment, but such a theory as this one neither satisfies reason nor directs practice.

There is a parallel to be found in the dysphagia due to spasm at the upper end of the gullet at the "post-cricoid" level.^{10 11 12} In the symptom-complex of "dysphagia with anaemia" described by Jones and Owen¹² pathological changes in the mucous membrane at the site of obstruction are stressed as a prominent part of the clinical picture. Again, Brown Kelly and Paterson¹⁰ give evidence that, in a goodly proportion of cases of spasm at the upper end of the gullet there have been observed fissures, erosions, bands or membranes, chronic glossitis, or early carcinoma. Sometimes no lesion of the canal is detected by the oesophagoscope,¹¹ but that does not negative their existence. Whether or no any lesion was found, all observers agree that the element of spasm was constant.

Wright⁹ states, "It is an undoubted fact that spasm at the entrance of the oesophagus occurs in association with superficial fissures and erosions." Although I can find no record in any *post-mortem* reports of cases of cardiospasm in which fissures and erosions were found at or near the cardiac orifice, it is possible that these would not be distinguishable through the oesophagoscope, and may readily escape detection at the necropsy unless definitely looked for. In Sir StClair Thomson's case¹⁶ of cardiospasm he remarks on the presence of a profuse growth of *Oidium albicans* on the wall of the dilated oesophagus. Certainly the demonstration of cracks and erosions would yield a more illuminating pathological picture than does the mere presence of degenerated nerve fibres in Auerbach's plexus. The latter might be expected as a natural sequence; their existence has been demonstrated by Rake⁸; that they are in any way the ultimate cause of cardiospasm is pure theory and extremely improbable.

Hurst¹³ writes of "the accompanying oesophagitis in the earlier stages, before the degeneration of Auerbach's plexus is complete." Surely when the nerve plexus becomes completely degenerated there is an end to all hope of cure or even improvement by bougies, mercury tube, or operation. There is an ingenuously about the statement that the mercury tube "closely approximates to what happens under natural conditions. . . . The weight of the mercury tube is about the same as that of the column of food in the dilated oesophagus."¹⁴ It might be inferred from this that the mercury tube is no more potent as a curative agent than the 8 in. column of food.

In the published cases there is a dearth of information regarding the habits of the patients in the years antecedent to the onset of the disease. Consideration of the case detailed above suggests a simple and, I believe, an adequate explanation of the origin of the disease. The repeated hurried swallowing of large masses of imperfectly masticated food, too large to pass at once through the cardiac orifice, so that they must remain for long periods in the lower oesophagus, causes a mechanical dilatation of that tube. The oesophageal wall, insulted by unduly prolonged

contact with disintegrating food, yields grudgingly; the irritation of the contiguous cardiac opening induces a reflex protective spasm.

Comments.

1. In the case reported there was very definite powerful spasm. Doubts as to the existence of spasm have been expressed, but the "pathology of the living" is more convincing than are theoretical speculations, or even experiments, on the cadaver.

2. The degeneration of the nerve fibres in Auerbach's plexus is interesting, but not germane to the question of cause. If the integrity of the plexus is essential to proper function the immediate and complete recovery after the various forms of stretching postulates an immediate and complete recovery of the nerve lesions, which is obviously impossible.

3. The disease is an acquired one, not comparable with congenital conditions, such as Hirschsprung's disease or congenital hypertrophic stenosis of the pylorus. Therein lies the hope of permanent cure.

4. The early age of onset (16½ years) in the case recorded is of interest.

5. The curious saltatory gymnastics adopted to obtain relief do not appear to have been previously recorded.

6. The bolting of food was clearly the cause in this case, and will probably be found to be the main factor responsible in all cases.

7. Plummer with the help of his bag only claims 81 per cent. of cures, and of these some relapse. Operation as advocated by Walton offers more certain, less tedious, and possibly more permanent cure.

8. Correction of faulty habits will go far to prevent relapse.

REFERENCES.

- ¹ *Lancet*, June 9th, 1928, p. 1174.
- ² Plummer: Cardiospasm, with report of 301 cases, *Med. Clinics of North America*, 1921, v, 355.
- ³ Hurst, A. F.: *Modern Technique in Treatment*, vol. iii, 1927, p. 317.
- ⁴ Walton, A. J.: The Surgical Treatment of Cardiospasm, *Brit. Journ. of Surgery*, vol. xii, No. 48, April, 1925, p. 701.
- ⁵ Kelly, A. Brown: Stenosis at Lower End of Oesophagus, *British Medical Journal*, October 19th, 1912, p. 1047.
- ⁶ Mutch: *Practitioner*, November, 1921, p. 342.
- ⁷ Hurst, A. F.: Case of Achalasia of the Cardia, *Proc. Roy. Soc. Med.*, January 22nd, 1915.
- ⁸ Rake, A. T., *Proc. Roy. Soc. Med.*, September, 1928, p. 1775.
- ⁹ Wright, A. J. M.: *Medical Annual*, 1923, p. 329.
- ¹⁰ Kelly, A. Brown: *Journ. of Laryngol.*, August, 1919, p. 288. Paterson, D. R.: *Ibid.*, p. 289.
- ¹¹ Mackenzie, Dan: *Ibid.*, 1918, p. 270.
- ¹² Jones, A. Mason, and Owen, Robert D.: *British Medical Journal*, February 18th, 1928, p. 256.
- ¹³ Hurst, A. F., *Ibid.*, April 21st, 1928, p. 690.
- ¹⁴ Hurst, A. F., *Ibid.*
- ¹⁵ Kelly, A. Brown: *Ibid.*, May 5th, 1928, p. 773.
- ¹⁶ Thomson, Sir StClair: *Journ. of Laryngol.*, April, 1919, p. 132.

THE PREVENTION OF COMPLETE RUPTURE OF THE PERINEUM.

BY

T. STRETHILL WRIGHT, M.A., M.D.Oxon.,
OKEHAMPTON, DEVON.

THE conduct of maternity cases by the general practitioner has recently been somewhat severely criticized by eminent gynaecologists; it is therefore with a certain amount of diffidence that I, one of the rank and file, venture to make this intrusion into the domain of obstetrics. My justification is that complete rupture of the perineum—the subject dealt with in this paper—is a disaster the importance of which can be scarcely exaggerated, and that I believe it to be preventable by the method of reinforcing the perineum presently to be described.

I do not propose to enter into the causes of severe perineal lacerations, nor need I stress the obvious dangers of impatient and violent intervention. There is, however, no doubt that from time to time cases will occur in which, in spite of every care, the perineum may be torn right into the rectum. In such cases after the tear has attained certain dimensions it is hopeless to expect the perineum to go on stretching—Nature has begun to take a short cut to secure the necessary room, and the line of least resistance will be followed until the orifice is big enough to let the head pass. Whether the necessary room will be secured

before the tear extends into the rectum is largely a matter of luck, unless the obstetrician can take active steps to limit the damage.

I suggest that, if he can strengthen the weak spot in the circle and can ensure that the perineum shall tear so far and no farther, he may make it possible for the extra room still required to be obtained by the resumption of the ordinary process of stretching. Acting on this idea I have for the past seven or eight years practised perineorrhaphy as a preventive rather than a reparative measure.

At the first sign of the perineum giving way I reinforce it by putting in a series of sutures of silkworm-gut—from three to five in number, according to the amount of room in each patient between the fourchette and the anal sphincter—starting at the level of the perineal tear and proceeding

from before backwards. The needle carrying each stitch is inserted about an inch to the left of the central line of the perineum, passes between the scalp of the foetus and the deep surface of the

FIG. 1.—Showing sutures inserted and tied in loops, which gradually become tighter as anus is approached.

FIG. 2.—Showing birth of head; sutures are now taut, preventing any further splitting of perineum.

FIG. 3.—Showing analogy of the loosened shoelace.

perineum, and is made to emerge again through the skin an inch to the right of the central line. By using a long needle and taking in a wide area on each side of the central line a good purchase for the sutures is secured.

Now comes the most important part—the tying of the stitches. The accoucheur has to decide in advance how far he is prepared to allow the perineum to split. The uppermost stitch is tied loosely enough to allow that amount of splitting, but not sufficiently loose to allow more; the next stitch must be a little less loose, and so on until the last one, just in front of the anus, is reached—this is tied firmly. We now have a sort of lattice-work of sutures which may be compared to the laces of a shoe after they have been loosened to allow the foot to be withdrawn.

As the head advances the stitches become taut; if they have been properly tied they should take the strain more or less simultaneously. If the top stitch is too tightly tied it will cut through the perineal tissue until the strain is taken by the sutures below. No single stitch would avail to hold in check a severe rupture of the perineum; it is the combination of a number of sutures all taking the strain together that saves the situation.

Several colleagues of mine have objected that the stitches must inevitably cut through. I can only urge sceptics to try this method for themselves. They will be surprised to find how well the stitches hold. So far I have not encountered a case in which there has been any serious cutting through.

In tying the sutures I do not use reef knots, for after the child is born the sutures have to be drawn tight to bring together the edges of the torn perineum. A double turn is sufficient* to fix the suture (already sticky from its passage through the perineal tissue) in the state of looseness temporarily desired, or it may be tied in a bow, which can be pulled apart later. Another alternative is to take a single turn and clamp the half-knot with pressure forceps.

Naturally the greatest care has to be taken to prevent too great and sudden a strain being put upon the now reinforced perineum. The use of midwifery forceps helps to control and steady the advance of the foetus's head. In occipito-anterior positions the forefinger should be inserted to steer the baby's eyes, nose, and mouth safely past the upper stitches. Taking this precaution I have never experienced any difficulty in preventing damage to the face.

* Since writing the above I have had a case in which the double turn was not sufficient to fix the suture as desired: the suture was subjected to a sudden and violent strain and began to slip, and a reef knot had to be made to save the perineum. I should therefore in future prefer the third method as the safest—that is, taking a single turn and clamping the half-knot with pressure forceps.

After the birth of the placenta it only takes a moment to draw the series of loops tight and to bring into exact apposition again the edges of the perineal tear. The sutures below the tear, having served their purpose, are snipped with scissors and pulled out.

It seems hardly necessary to warn the accoucheur that the stitches should be inserted *between* the pains, and that the needle must not be put in *too* deep; I happen to know of a case in which the doctor, trying this method for the first time, anchored the baby's scalp to the perineum.

As regards the value of these experiments, I must confess that I have not had enough cases to dogmatize. Rupture of the perineum is fortunately a relatively rare complication. The procedure described above is, at any rate, harmless, if proper precautions against sepsis are

taken, and the favourable results which I have experienced in every case in which I have employed it during a number of years encourage me to think that even in the worst cases it may, if rightly used,

prevent a tear from extending through the internal sphincter into the rectum. I think I may claim for it that it limits the damage and acts as a brake.

The description of this method is published in the hope that others will take the opportunity of testing its value and improving the technique. Hitherto I have only tried interrupted sutures; very probably the use of a single continuous suture (following more exactly the analogy of the loosened shoelace) would be simpler and equally effective.

THE RELATIONSHIP OF PLASMA CHOLESTEROL AND BASAL METABOLISM.

BY

J. ADDYMAN GARDNER, M.A.,

READER IN PHYSIOLOGICAL CHEMISTRY, UNIVERSITY OF LONDON;

AND

HUGH GAINSBOROUGH, M.D., M.R.C.P.,

PHYSICIAN TO ST. GEORGE'S HOSPITAL.

In a communication in 1922 Epstein and Lande¹ endeavoured to demonstrate the existence of an inverse relationship between the blood cholesterol and the basal metabolic rate. Such a correlation was suggested by the fact that in sub-acute and chronic parenchymatous nephritis the basal metabolism is subnormal, while the blood cholesterol is increased above normal. Epstein believes that this disease is really a metabolic disorder involving a hypothyroidism, and that the renal manifestations are only a part of the picture.

In support of this thesis Epstein and Lande have determined the blood cholesterol and basal metabolism in a number of cases of hyperthyroidism, in which they maintain that the same inverse relationship of these two factors also holds good. Their figures, however, hardly warrant the conclusions they have drawn. The colorimetric method they used for estimating the total cholesterol of whole blood, as has been repeatedly proved,^{2,3} is open to serious objection, but, perhaps, a more important criticism is the use of the total cholesterol of whole blood as a significant value. They ignore the fact that the total figure includes the cholesterol of the corpuscles, which is in the "free" form only, and which is remarkably constant in health and disease; and also the fact that the plasma cholesterol exists both in "free" and in combined form as ester. The ratio