suffering from a diminution in the amount of the internal secretion, whereas the rats had all ovarian tissue removed. If this were the explanation one would expect to find that rats suffering from a normal menopause would not all show adrenaline mydriasis, whereas all women suffering from a surgical menopause associated with total removal of the ovaries should show the reaction.

Dealing with the first point, an effort was made to obtain some rats of menopause age, and here considerable difficulty was experienced, and an interesting point arises which has an important bearing on claims which have been made in the rejuvenation of old animals. I have found that either old animals are pets, and are therefore not available for experimental work at any price, or, if they are not pets, for economic reasons are never allowed to grow old.

After considerable trouble, however, 6 rats, all guaranteed to be 2 years old or more, were obtained, but one of these has had a litter, 4 show oestrous cycles at regular intervals, and only one rat was found to have no oestrous cycles. It is interesting to find that this rat, which has a senile cataract in one eye, gives a constant mydriatic reaction.

With regard to the testing of women who have had all ovarian tissue removed, some difficulty has again been experienced owing to the reluctance with which most surgeons remove all ovarian tissue. Twelve women who have undergone Wertheim's radical operation for carcinoma of the cervix have been tested, however, and in only one instance was adrenaline mydriasis present. It is therefore obvious that some other explanation of the difference in the percentage of reaction between rats and women must be found. As I have shown elsewhere,⁶ adrenaline mydriasis in women invariably passes off when the symptoms of "flushing" cease, and that not all women who suffer from "flushing" at the menopause show adrenaline mydriasis. I have advanced a hypothesis in this *Journal*⁷ that the disturbance of the sympathetic nervous system, in particular the abnormal adrenaline effects, is but a temporary phenomenon consequent upon the withdrawal of part or whole of the internal secretion of the ovaries, and that the duration of the symptoms depends entirely upon the speed at which the compensatory activity of the general endocrine system occurs. It would seem, therefore, that this compensatory activity is much more rapid in women than in rats.

Finally, I would suggest that the experimental work which I have outlined in this paper justifies the following conclusions.

1. The adrenaline mydriasis phenomena associated with the menopause in women occur also in oophorectomized rats.
2. The adrenaline mydriasis reaction occurs with more regularity in rats than in women.
3. The oestrus-producing autacoid can cause rats to lose the adrenaline mydriasis effect if administered in adequate doses.
4. The autacoid is effective when administered by the oral route in doses corresponding to those necessary to produce oestrus.
5. Diminution in the amount of the oestrus-producing hormone is responsible for adrenaline mydriasis at the menopause, and, as this phenomenon is always associated with severe vasomotor phenomena in women, is also probably responsible for these symptoms of the menopause.
6. The oral administration of the oestrus-producing autacoid is rational treatment for the vasomotor symptoms of the menopause.
7. The dose will have to be repeated within seven days if a permanent effect is required.
8. Though administration of the autacoid at the menopause is rational treatment, the enormous doses required will prevent the maximum benefit from such treatment being attained until preparations of increased activity are available.

I wish to record my appreciation of the helpful criticism and advice which I have received from Professor Swale Vincent and from Mr. J. H. Thompson of this department.

REFERENCES.

- ¹ *British Medical Journal*, July 2nd, 1927, p. 14. ² *The Flushings of the Menopause*, Baillière, Tindall and Cox, London, 1927. ³ *Endocrinology*, 12 (1), 59-64, 1928. ⁴ *Ibid.*, 12 (2), 193-198, 1928. ⁵ and ⁶ *Ibid.*, 12 (1), 1928, p. 62. ⁷ *British Medical Journal*, July 30th, 1927, p. 192.

CARDIOSPASM.

BY

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THE use of the term "achalasia" implies a pathology analogous to that of Hirschsprung's disease and of congenital hypertrophic stenosis of the pylorus; both these diseases are probably congenital in their origin, both are accompanied by demonstrable hypertrophy affecting the muscular coat of the alimentary tube, whereas in cardiospasm the condition is certainly not congenital, and the existence of any hypertrophy of the muscular coat remains a subject of contention.

The term "cardiospasm" has an established place in the literature, and clinical observation bears out its descriptive accuracy; it would seem that the term "achalasia" is a premature attempt at generalization, facile and tempting, but quite unjustified by the present state of our knowledge.

The opinions expressed in recent literature by eminent authorities¹ show a lack of unanimity as to the cause of the disease, nor is there general agreement about the counsel of perfection in treatment.^{2 3 4 5}

The symptoms, slowly progressive, fall roughly into three stages: (1) Discomfort, not amounting to pain, in the epigastrium and behind the sternum. (2) Precipitate vomiting, coming at intervals, later occurring at every meal; pain, sometimes severe, usually precedes the vomiting and is relieved by it. (3) The vomiting becomes less precipitate. The oesophagus has become dilated so that it will hold a pint or more; the propulsive power of the oesophageal wall has weakened, and food enters the stomach mainly by hydrostatic pressure.

The disease usually occurs "in individuals of middle and late life" (Wright⁶). The youngest case in the series recorded by Walton⁴ developed symptoms at 21 years of age; the average age of onset was 46 years. In a case sent to me by Dr. Moore the age of onset was earlier—16½ years. The symptoms developed in the manner described above; there were, however, other features of an unusual character that are interesting.

The patient, an unmarried man, now aged 25, became an apprentice at the age of 16 in an engineering works one and a half miles distant from his home. The long hours of hard physical labour and his rapid growth during the period of adolescence demanded abundant food. Half an hour's rapid uphill walk, a similar period to return, left only a few minutes for a substantial meal at noon. In the evening, reaching home tired, at 6 o'clock, he had another hearty and hasty meal, hurrying to reach the technical college for evening classes at 7 o'clock. His teeth were very defective. In consequence all his meals were imperfectly masticated and were followed immediately by active exertion. The penalty of this constant bolting of food was the onset of symptoms of cardiospasm at the age of 16½ years. For several months the dilated oesophagus was emptied from time to time by vomiting, and relief from the pain followed. One day, after running rapidly up and down stairs, he discovered that the usual oppression in the chest and the sense of impending vomiting were suddenly relieved, and he felt something pass on into the stomach. He thereupon adopted the ingenious manoeuvre of springing up in the midst of a meal (when the symptoms invariably had their inception) and jumping with a curious side-to-side action, first on one leg and then on the other, until he felt a mass slide suddenly through an obstruction; he experienced an immediate freedom from pain, and he was able to resume his interrupted meal.

His general health began to suffer and he was obliged to abandon his employment at the works. He went abroad to Malay, and there, although now equipped with efficient dentures and no longer condemned to hurried meals, the symptoms persisted unabated. During the last few years they have, indeed, grown worse, and he has slowly but steadily lost weight; a stature of 6 ft. 1 in. consorted ill with a weight of 9 st. 8 lb. Sometimes pain and oppression were such prominent and urgent features that vomiting would alone serve to relieve his distress. He consulted various medical men and ingenious explanations were offered, but the cause was usually ascribed to "nerves," an attribution probably influenced by his curious saltatory movements.

Whilst on "long leave" he was referred to me, and I had several opportunities of seeing typical attacks during meals, ushered in by a growing expression of distress, and culminating in a sudden spring from his chair; the execution of this curious jumping dance, sometimes lasting a few minutes, sometimes as long as fifteen minutes before sudden relief came, was very striking, and suggested the existence of an oesophageal pouch or of cardiospasm.

A barium meal and x-ray examination by Dr. Godfrey Mitchell (April 16th, 1928) revealed clearly the typical appearances of an advanced case of cardiospasm. (See Fig. 1.) Since it was imperative that he should return to Malay in about three months' time, he wished to obtain a cure, as nearly certain and as permanent as was attainable.

The various possible courses were outlined to him; he rejected as impracticable the tedious and uncertain dilatation by the repeated passage of bougies, by Plummer's bag,² or by the mercury tube so confidently recommended by Hurst.³ Constrained by circumstance, he elected to submit to the operation so ably and convincingly advocated by Walton.⁴ With his preference I concurred the more readily in that his father, a medical man, counselled this course. Where time and the tolerance of the patient are limited it is well to remember that Mutch⁵ has recorded his having "seen patients who have passed the mercury tube 35 times a week without obtaining any permanent relief."

Accordingly, on April 22nd, 1928, under gas, oxygen, and ether, I opened the abdomen at Glenwood Nursing Home. He was very wasted, the chest long and narrow, the epigastric angle acute, the liver enlarged and low-lying (possibly from the frequent attacks of malaria that he had endured), so that access to the stomach was rather difficult. Contamination of the rest of the abdomen was prevented by large gauze packs. The stomach was then opened on the anterior aspect by an incision in its long axis, the free bleeding from the edges being arrested by "mosquito" forceps and ligatures of fine catgut. The initial difficulty encountered was the finding of the cardiac orifice, but by patience this was located, a small tightly closed dimple-like opening, resembling an anus spasmodically contracted by a fissura

enjoys an ordinary diet, and is steadily losing his emaciated appearance.

On July 2nd, 1928, Dr. Godfrey Mitchell again x-rayed the patient, and reported as follows.

"Food now passes very much more freely into the stomach, and even when thick paste was swallowed no large accumulation took place in the oesophagus. There is still some dilatation of the gullet, but considerably less than at the time of the previous examination."

The x-ray appearances are seen in Fig. 3. Although there is still some dilatation the tube of the gullet is seen to be flaccid; muscular tone is not yet regained, but there is no arrest of food. It is evident that any repetition of his former habit of swallowing large unmasticated masses of food, which could not at once pass onward into the stomach, would assuredly cause recurrence of symptoms. Herein there possibly lies the explanation of the relapses that occur in a certain proportion of successful cases. In like manner the recurrence of gastric ulcer and the formation of jejunal ulcer some months or years after a gastro-jejunosomy is more closely related to a reproduction of the same factors that caused the original lesion—oral sepsis, defective mastication, grossly unsuitable food, over-indulgence in alcohol and tobacco—than to any inherent

FIG. 1.—Antero-posterior view of dilated oesophagus, April 16th, 1928; before operation.

FIG. 2.—Oblique view of the same case, April 16th, 1928; before operation.

FIG. 3.—Radiogram after barium meal, July 2nd, 1928; after operation.

in ano. The little finger, then the forefinger, followed by the ring and middle fingers, were cautiously inserted, until, after about fifteen minutes, the thumb too, with the hand in the "obstetric position," gained admission.* Stretching was cautiously continued until the opening became lax; the fingers then passed easily along the 1 inch of intra-abdominal oesophagus to the diaphragm, where the aperture seemed slit-like and required less force to secure relaxation than did the true cardiac orifice.

Despite abstinence from food for the antecedent twenty-four hours, the anticipated gush of dark green fluid, flecked with fragments of undigested food, followed the dilatation; at least half a pint was mopped away and a similar quantity flowed later into the stomach whilst the gastric incision was being sutured; another 6 ounces were vomited seven hours later, after which none was returned.

The period of moderate shock, with intense pain in the left chest behind, lasted twenty-four hours, and would seem to be a sequel inseparable from any severe operation in a region so sensitive to trauma, howsoever gentle the manipulations employed. A bronchitis, with shallow distressed breathing, was not unexpected, and happily subsided at the end of three days. During these three days nothing was given by the mouth, abundant fluid (with sodium bicarbonate and glucose) being supplied by the rectum. From that time onward the swallowing of fluids and later of solid food was painless.

Healing and recovery were uneventful, the complete disappearance of all symptoms being remarkable. None the less gratifying has been the steady increase in weight, so that three months from the date of the operation he weighed 10 st. 9 lb.; he now weighs over 11 st.

The symptoms in this case etched clearly the clinical picture of cardiospasm. The ingenious and effective manœuvre of jumping in order to relieve the urgent sense of pain and oppression appears to be original.

The patient is now completely free from any symptoms,

defect in the operation, whose consequences would become apparent within a few weeks. It is always difficult to eradicate long-established habits whose evil tendency is not fully realized by the patient.

Hurst⁷ has denied the existence of spasm, and has assumed an exact parallel between cardiospasm and congenital pyloric stenosis. In the latter there is undoubtedly a hypertrophy of the circular muscle at the pylorus. One cannot but be impressed in doing Rammstedt's operation with the (relatively) enormous mass of muscle that needs division in an infant a few weeks old. Surely such a condition is either congenital or develops soon after birth, antecedent to and not caused by the spasm that gives rise to the symptoms of obstruction. It does not follow that the spasm has given rise to the hypertrophy. Nor does the absence of any hypertrophy of the circular fibres around the cardiac opening of the oesophagus negative the existence of a spasm seen and felt in a case which is clinically one of cardiospasm. After feeling with the finger the tightly closed cardiac orifice slowly, then more rapidly, yield to pressure, there can remain no doubt in the mind of the surgeon as to the presence of spasm.

A closer parallel is found in a fibrous stricture of the urethra on which has been superimposed a spasm that yields to persuasive measures; the absence of any demonstrable hypertrophy of the urethral sphincter does not invalidate a diagnosis of spasmodic stricture in the living subject.

Rake⁸ has carried a stage further: our knowledge of the changes found *post mortem* in Auerbach's plexus. Whilst

* This description applies to a small hand taking a 6½ glove.

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

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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