The method I have adopted is as follows. Two days before the operation all patients are injected with 1 c.cm. of thyroxine. After three hours the temperature, pulse, and blood are examined. In the first group, which may be termed "thyroxine-sensitive," as a result of the injection, the pulse quickens and the temperature rises slightly; difference in the blood composition is noticeable by the variation of the quantity of the blood corpuscles—the number of the red corpuscles increases slightly, whilst the number of blood platelets decreases. After two or three injections of thyroxine these become two or three times more abnormal, and the time necessary for blood coagulation is longer. On a patient in this thyroxine-sensitive group any external influence, especially the operation itself, will produce the effects mentioned above, and for this reason I do not believe they can be affected by thrombosis. In patients in the second group, termed "thyroxine-resistant," as a result of the same injection it is impossible to detect any change in temperature, pulse, or blood composition. A slight variation in blood corpuscles is sometimes noticeable, but is of no importance. To these patients every operation will bring the danger of thrombosis.

The scientific explanation of the reaction to thyroxine is explained in different ways. Freund and Boshamer believe that the thyroxine acts on the vegetative nervous system, and at the same time directly affects the cardiovascular system, producing the clinical symptoms in the patient. In the thyroxine-resistant group of patients the effects of the injection are almost unnoticeable, because the systems mentioned above show very little reaction. In this group of patients the parasympathetic nerve system predominates, thus facilitating the formation of thrombosis. During the last three years I have experimented with more than two thousand patients admitted for operation. In every case thyroxine was administered and the patient afterwards examined. In this way patients were divided into two classes: thyroxine-sensitive and thyroxine-resistant.

PREVENTIVE TREATMENT

After the thyroxine effect had been discovered it was necessary to find some treatment to prevent thrombosis. During this experimental period all cases in which thrombosis occurred were examined and were found, without exception, to belong to the thyroxine-resistant group. This tended to confirm my conclusion that only patients of this group were liable to thrombosis. The treatment that was found to be successful consisted in the administration, hypodermically, of a combination of atropine and ephetonin in the proportion of 1/100 grain atropine to 1/4 grain ephetonin. Not less than three injections in all were given every other day, beginning from the fifth day after the operation. I am able to state that not one patient treated in his way suffered from thrombosis or embolism.

The scientific explanation of the influence of atropine and ephetonin is as follows. With the first injection a variation in the blood composition is noticeable. Six hours after the injection the number of blood platelets is less than 100,000 per c.mm., and the number of red cells is slightly increased. The decrease of blood platelets occurs after every injection until the third, when the same level is maintained. The time necessary for coagulation of blood is two or three times longer than usual. This is shown more clearly in the diagrams.

In small hospitals where there is no laboratory equipment and it is thus impossible to divide the patients into thyroxine-sensitive and thyroxine-resistant, I believe it is advisable to treat all patients as belonging to the latter division. The injection of atropine and ephetonin can never be harmful to the patient, and is even useful for

the prevention of post-anaesthetic lung trouble. In this way mistakes will be avoided and deaths from thrombosis will be reduced to the minimum.

CHART I.—Thyroxine-sensitive patients.

CHART 2.—Result of ephetonin-atropine treatment of "thyroxine-resistant" patient.

SUMMARY

The origin of thrombosis is not confined to one group of conditions. It is rather a special pathological state due to external influences such as constitutional predisposition, bacterial infection, or toxæmia. Not all patients are affected by the thrombosis, but by the use of thyroxine it is possible to divide them into two groups: those immune from, and those predisposed to, thrombosis. The symptoms which demonstrate the difference between the two groups are the pulse, temperature, and variations and change in the blood composition. The treatment is the hypodermic administration on alternate days, beginning on the fifth day, of a combination of ephetonin and atropine.

TORSION OF THE NORMAL FALLOPIAN TUBE

BY

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Torsion of abnormal uterine appendages is not an uncommon condition, but torsion of normal appendages is much less frequently encountered. Torsion may involve either the normal tube or the normal ovary, or both; but this brief review is confined to tubal torsion, of which the following is an example.

A girl, aged 14, was admitted to King George Hospital on April 5th, 1931, under the care of Mr. Harold Dodd. Pain in the right lower quadrant had begun four days previously. After twenty-four hours it had become central, and vomiting occurred. The pain persisted and remained central. The bowels had acted on the day of admission, and there were no urinary symptoms. The first menstrual period had begun on January 1st, 1931, and had lasted seven days; the second, on February 4th, lasted four days; and the third, on March 8th, lasted four days. (The fourth began on April 6th, twenty-four hours after the operation, and lasted two days.) Since the age of 2 she had suffered from attacks

of pain in the right iliac fossa, associated with headache. These attacks lasted three to four days, beginning and ending suddenly, and were usually associated with vomiting. Up to the age of 7 they occurred every one to two months, but subsequently they had become much less frequent.

On admission her temperature was 98.8° F. and her pulse 86. There was well-marked tenderness and rigidity in the right lower quadrant. Nothing abnormal was noted on rectal examination, and the urine was normal. A diagnosis of acute appendicitis was made, and the abdomen was opened by a gridiron incision. The appendix appeared to be mildly inflamed, and was removed. A mass was then detected in the right side of the pelvis, necessitating exposure through a right paramedian incision. This proved to be the right Fallopian tube, which had undergone two complete turns in an anti-clockwise direction (see Fig.) It was dark red and

Acute torsion of the right Fallopian tube.

much enlarged, due to swelling of its walls and haemorrhagic effusion into the wall and lumen. The mass was 5 cm. long and 3 cm. in diameter. It was removed, and the wounds were closed in layers. Recovery was uneventful, and the patient was discharged from hospital on the fourteenth day. The specimen was submitted for examination to Dr. Arthur Davies, who reported that there was no evidence of pregnancy, and that the haemorrhagic effusion was the result of torsion. In the two years which have elapsed since the operation there have been two attacks of pain somewhat similar to those which occurred in childhood.

In my opinion the interesting features of this case are:

1. The history of recurrent attacks of pain. This has been a feature of some of the recorded cases, but in this instance it seems probable that the repeated attacks were due to some other cause.

2. The definite relation to the pre-menstrual period.

3. The anti-clockwise direction of the twist, which is the reverse of that which is said to be usual on the right side. Kustner's law (quoted by Downer and Brines¹) says that, in torsion of uterine adnexae, twisting on the right side is clockwise and on the left side anti-clockwise.

The factors concerned in torsion of the Fallopian tube have been discussed from time to time in the gynaecological literature of the last twenty years, and a number of theories have been advanced to explain why a normal tube should suddenly undergo axial rotation to a pathological extent. It has been suggested that the tube is only apparently normal, and that it has been the seat of a mild inflammatory process, possibly associated with a slight hydrosalpinx. It is postulated that this may have resulted from vulvovaginitis in childhood, or that it may have occurred as a complication of one of the exanthemata.^{2,3} To reduce to a minimum the possibility of a low-grade infection of Neisserian or puerperal origin, this review is confined to torsion of the Fallopian tube in virgins. It would appear that in the recorded cases no very definite evidence of antecedent disease has been

found, and in the present case there were no adhesions and no suggestion of hydrosalpinx. I think, therefore, that it is probable that a tube indistinguishable from the normal can undergo torsion. Unusual mobility of a tube due to unusual length of the tube itself, the fimbria, or the mesosalpinx would predispose to unnatural twists; but in addition to a predisposing anatomical condition some further factor would be necessary to precipitate acute torsion.

Association in some way or another with menstruation occurs so frequently in the recorded cases that it would appear to have some aetiological relation. In this connexion two aspects of the menstrual function are of particular interest: first, it is conceivable that the passive congestion at this time may be one of the factors concerned; and secondly, it has been observed that during menstruation the tube occasionally contains menstrual blood. That this does sometimes happen has been established by Sampson,⁴ Bailey,⁵ and others; and it is generally believed that it is the result of regurgitation from the cavity of the uterus. Another view is that it arises by a process of tubal menstruation,⁶ but although this would appear to be embryologically possible it has never been proved to take place. The presence of blood in the tube may be concerned in the origin of torsion in one of two ways: it may be responsible for, or associated with, the initiation of abnormal contractions of the tube, or the simple factor of weight may be of importance. It is interesting that at least ten of the cases referred to have occurred after puberty, whereas torsion of the ovary or ovary plus tube before puberty is by no means unknown.

Another aetiological factor which has been commented upon is that of strain or sudden movement, but this does not appear to occur so frequently as association with menstruation. The most reasonable explanation of the way in which strain might be concerned in the origin of torsion is that contained in the haemodynamic theory of Payr, which is quoted by Gabe³ and Thorek.⁷ Payr showed, by injecting the vessels in the pedicle of the spleen, that torsion was imparted as a result of the spiral course of the vessels. It is conceivable that a sudden rise of pressure in tortuously disposed vessels may be a factor in torsion of the Fallopian tube, even though the anatomical arrangements are different from those obtaining in the pedicle of the spleen.

It would appear from these considerations that the important factors in the aetiology of torsion of the normal tube are anatomical predisposition, association with menstruation, and strain or sudden movement.

In the literature at my disposal I have found records of nine cases of torsion of the Fallopian tube in virgins. (1) Stark,⁸ 1911. Aged 46. Three previous attacks. Dermoid right broad ligament. Torsion left tube. (2) Davies,⁸ 1925. Aged 19. Clockwise torsion right tube. Long mesosalpinx. Association with menstruation. (3) Rogers,⁹ 1925. Aged 16. Torsion right tube, also left to less extent. Menstruating. (4) Darner,² 1926. Aged 13. Clockwise torsion right tube. Menstrual period began five days after operation. Onset of pain followed a kick. (5) Jefferson,¹⁰ 1926. Aged 32. Clockwise torsion right tube. Long mesosalpinx. Menstruated two days after operation. (6) Gillies,¹¹ 1926. Aged 23. Torsion left tube. Last period two weeks previously. (7) Corlette,¹² 1927. Aged 20. Clockwise twist of left tube occurring while confined to bed following fracture of tibia. Tube enormously distended. (8) Koster,¹³ 1929. Aged 16. Torsion of right tube. Menstruating. (9) Michon,¹⁴ 1930. Aged 26. Torsion of left tube. Koster¹³ gives references to five further cases reported by Auvray, Schweitzer, Hausen, Heil, and Gabe.

SUMMARY

1. A case of torsion of a normal Fallopian tube in a virgin is reported.

2. Nine other cases have been found in the literature, in addition to five quoted by Koster.

3. The aetiology in general is discussed, the salient points being preternatural mobility, association with menstruation, and strain or sudden movement.

My thanks are due to Mr. Harold Dodd for permission to publish this case.

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Memoranda

MEDICAL, SURGICAL, OBSTETRICAL

COMPLICATIONS FOLLOWING ANAESTHESIA WITH SODIUM EVIPAN

The case to be described is the only one in a series of 150 in which complications have followed the use of sodium evipan. It is of interest by reason of (1) the long latent period before the onset of symptoms, and (2) the toxic manifestations not being those commonly associated with poisoning by drugs of the barbituric group.

The patient, a man aged 23, was admitted to the hospital for the excision of a sebaceous cyst from the scrotum. There was some ptosis of the right eyelid, which had been present since birth, and atrophy of the right testicle, presumably due to mumps; otherwise he was apparently healthy and normal. Following my (E. L.) usual practice no preliminary medication was employed, anaesthesia being induced with evipan 10 c.cm. and supplemented with gas-oxygen anaesthesia. The cyst was removed, the course of the operation being uneventful. When the patient returned to consciousness about half an hour after the operation he was very restless; he was restrained, and there is no evidence that he injured himself in any way during that period. Eight hours later he was still restless, and was suffering some discomfort; 1/4 grain of morphine was injected and sleep ensued.

The following morning the patient appeared well, but complained of some headache; he was able to read, and took his food well. This persisted throughout that day, the following day, and until 9.30 on the evening of the third day, when he complained of sudden severe frontal headache, giddiness, spots before the eyes, and photophobia. The pulse rate dropped to 40; the temperature and respirations remained normal. He was seen by a colleague, who prescribed 1/50 grain of atropine and 10 minims of adrenaline. The pulse rate rose to 50, and the patient slept. On the morning of the fourth day he was somewhat drowsy, and lay back with neck and legs flexed. The temperature and rate of respiration were normal; the pulse rate was 36. The heart showed no signs of organic disease, and the blood pressure was 135/80. The pupils reacted to light and accommodation, but there was lateral nystagmus to the left. There was no evidence of paresis or anaesthesia in any part of the body. All tendon jerks were exaggerated, and the plantar response was flexor on both sides. The abdominal reflexes were brisk, and dermatographia was marked. Kernig's sign was negative, and the disks appeared normal. There were no sensory changes. The liver was not enlarged, and there was no jaundice. The urine contained no albumin, blood, sugar, or acetone bodies. Lumbar puncture was performed, and a clear fluid under slightly increased pressure was obtained. The manometer reading was 170 mm., 25 c.cm. of fluid being withdrawn. This fluid on examination revealed no excess of cells, no organisms, and

normal content of sugar, albumin, globulin, and chlorides. An electrocardiogram showed no abnormality. The nystagmus disappeared after the lumbar puncture.

On the fifth day the patient was still drowsy, and complained of occipital headache. The blood pressure was 140/85, and the pulse rate varied during the day between 40 and 50. Towards the end of the day the nystagmus reappeared, and the patient vomited twice. On the sixth day the patient's condition remained the same. The vomiting had become persistent, and he was unable to retain any food. Hypertonic glucose was given intravenously, and later in the day the blood pressure was found to have fallen to 120/70. The injection of hypertonic glucose was repeated on the seventh day, the vomiting ceased, and the headache became less severe. Eight days after the operation the pulse rate rose to 60, and the blood pressure fell still further to 115/60. There was no vomiting or headache, and the nystagmus had disappeared; the tendon reflexes remained brisk. From that day onwards the patient made an uninterrupted recovery, and was discharged on the sixteenth day after the operation. His pulse rate remained low, between 50 and 60, until two days before his discharge.

An x-ray examination of his skull showed no evidence of any abnormality or injury. The Wassermann reaction of his cerebro-spinal fluid was negative. It is difficult to assign a head injury as the cause of the symptoms; in these circumstances they can only be attributed to the sodium evipan.

We wish to thank Mr. Fedde Fedden, senior surgeon, St. George's Hospital, and Dr. Anthony Feiling, physician, St. George's Hospital, for their advice and help in the treatment of this case, and for their permission to publish these notes.

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FRACTURE OF FEMUR IN A WOMAN OF 80

This case seems to warrant recording because of the age of the patient at the time of the accident, and the satisfactory anatomical and functional state that has resulted after treatment.

In July, 1931, the patient, a woman of 80, fell heavily on a hard floor, and when I saw her, within two hours of the accident, it was obvious that there was a fracture of the right femur in the neighbourhood of the neck. She was moved carefully to bed in an adjoining room. An x-ray photograph was taken in her own bed the following morning by means of a Philips portable x-ray apparatus, and a paratrochanteric fracture of the right femur was demonstrated. A Thomas splint was applied to give temporary extension during transit, and the patient was removed with extreme care to the Butterfield Cottage Hospital, Bourne. On the following day a general anaesthetic was given, and the limb was fully abducted, extended—that is, protracted, while the foot was internally rotated—and a plaster-of-Paris spica applied. After four days had elapsed and on each day during her stay in hospital she was lifted from bed and placed in as upright a position as possible on a reclining chair. She was sent home ten days after admission to hospital.

After three weeks from the time of the accident the ankle was liberated and movements encouraged, and after a further three weeks the knee was liberated and again movements instituted. Three months from the date of the accident the patient was allowed to bear weight on the affected limb, and to walk as best she could, wearing the plaster case. In the week before Christmas the plaster cast was entirely removed, this being five months from the time of fracture. She was then able to move about almost unaided. There is now no disability, and the patient speaks of the right leg as being her "best leg."

It seems to me that several factors contributed towards the successful result: first, knowledge of the exact lesion, from the x-ray photograph taken in the patient's own bed without disturbance before removing her to hospital;