The average time taken to control these patients was seventeen days, and the limits seven to forty-six days respectively; figures which compare very favourably indeed with the average expectation of thirty-five days for mild chorea cases and the sixty-seven days for severe ones referred to in the earlier part of this paper. The drug diminished the discomforts of the patients at all stages of the treatment, and did not produce any of that mental depression which accompanies the use of chlorotone and certain other hypnotics. There was no undue drowsiness or digestive derangement. There was no relapse or secondary exhaustion when the drug was finally withdrawn.

Conclusions

Calcium aspirin (Coplans)* affords a method of treating chorea without occasioning exhaustion, discomfort, or risk to the patient. It is less toxic^{16 30} than acetyl-salicylic acid itself, and is free from the irritant action of the parent substance on the stomach and bladder. It is freely soluble, almost tasteless, and is rapidly absorbed. It has a triple action: (1) anti-rheumatic, (2) correction of calcium deficiency, (3) sedative to the brain.

The best results are secured when the patient is kept in bed during treatment and protected from undue emotional and mental stress.

REFERENCES

- ¹ Beidemann, W.: *Sitzungsber. der Wien. Akad. der Wissens.*, 1880, lxxxii, 3.
- ² Burnford, J.: *Lancet*, 1931, i, 351.
- ³ Cambell, A.: *British Medical Journal*, 1921, ii, 37.
- ⁴ Dennett, R. H., and Wetchler, S.: *Journ. Pediat.*, 1932, 203.
- ⁵ Gerstley, J. R., and Wile, S. A.: *Ibid.*, 1932, 458.
- ⁶ Heftter, E.: *Zeit. f. Kinderheilk.*, 1924, xxxviii, 403.
- ⁷ Hughes, H. M.: *Guy's Hospital Reports*, 1846, Series 2, iv; *ibid.*, 1855, Series 3, i.
- ⁸ Husler, J.: *Zeit. f. Kinderheilk.*, 1924, xxxviii, 408.
- ⁹ Jones, T. D., and Jacobs, J. L.: *Journ. Amer. Med. Assoc.*, 1932, xcix, 18.
- ¹⁰ Keller, R.: *Deut. med. Woch.*, 1928, lxiv, 1880.
- ¹¹ Loeb, J.: *Beit. z. Physiol.*, Fest. I. Adolf Fick, 1899.
- ¹² Lucas, K.: *Journ. Physiol.*, 1910, xl, 225.
- ¹³ Mackenzie, S.: *British Medical Journal*, 1887, i, 425.
- ¹⁴ Mandel, L.: *Ibid.*, 1933, ii, 78.
- ¹⁵ Mas de Ayala: *Ann. Fac. Med. Monte Video*, 1930, xxi, 73.
- ¹⁶ Mutch, N.: *Journ. Pharmacol. and Exper. Therap.*, 1934, li, 112.
- ¹⁷ Medical Research Council Special Report, Rheumatism, 1927, Series 114, p. 94.
- ¹⁸ Fletcher, H. Morley: *St. Bart's Hosp. Reports*, 1897, xxxii, 383.
- ¹⁹ Neale, A. V., and Eslemont, M. S.: *Arch. Dis. Child.*, 1928, xvi, 243.
- ²⁰ Neustaedter, M., Hala, W., and Tobstouchow, A.: *Journ. Amer. Med. Assoc.*, 1925, lxxv, 347.
- ²¹ Pilcher, J. M., and Gerstenberger, H. J.: *Amer. Journ. Dis. Child.*, 1930, xl, 1239.
- ²² Piltz, K.: *Arch. f. Kinderheilk.*, 1927, lxxxii, 810.
- ²³ Pritchard, H.: *British Medical Journal*, 1927, i, 794.
- ²⁴ Pye-Smith, P. H.: *Guy's Hospital Reports* 1874, Series 3, xix.
- ²⁵ Roeder, F.: *Therap. Monats.*, 1919, xxxiii, 54.
- ²⁶ Schmal, S.: *Deut. med. Woch.*, 1925, li, 1439.
- ²⁷ Sée: *De la Chorée et les Affections Nerveuses*, Paris, 1851.
- ²⁸ Shaw, B.: *Arch. Dis. Child.*, 1929, iv, 155.
- ²⁹ Sutton, L. P.: *Journ. Amer. Med. Assoc.*, 1931, xcvi, 299.
- ³⁰ Thompson, H. E., and Dragstedt, C. A.: *Journ. Amer. Pharm. Assoc.*, 1933, xxii, 1096.
- ³¹ Trawley, J. M., Pepper, M., and Wathe, L. E.: *Calif. and West. Med.*, 1932, xxxvi, 346.
- ³² Warner, E. C., Spurrell, W. R., and Carter-Braine, J. F.: *Guy's Hospital Reports*, 1929, lxxix, 473.
- ³³ Warner, E. C.: *Lancet*, 1930, i, 339.
- ³⁴ Weinfeld, G. F., and Cohen, R.: *Journ. Pediat.*, 1932, 210; *Journ. Amer. Med. Assoc.*, 1932, xcix, 33.
- ³⁵ Winter, J. E., and Richey, C. H.: *Journ. Pharmacol. and Exper. Therap.*, 1931, xlii, 2, 179.

* Calcium chloride is present in this stabilized preparation to an extent approximating to the formula

$$[C_6H_5(OCOCH_3)CO_2]_2CaH_2O + 1/3 CaCl_2$$

The Health and Cleanliness Council (5, Tavistock Square, W.C.1) has issued a revised edition of its pamphlet first issued in 1926, incorporating suggestions for a local health and cleanliness campaign. Supplies are available for distribution free of charge, and applications for copies should be addressed to the secretary, Miss Norah March, B.Sc., at the address given above. The advisory board of the council consists of Dr. George F. Buchan (chairman), Dr. Sophia Friel, Miss J. Halford, Professor H. R. Kenwood, and Dr. Eric Pritchard.

THROMBOSIS OF THE PENIS AND URETHRAL HAEMORRHAGE*

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Thrombosis may occur either in the veins of the penis or in the corpora cavernosa. The latter consist of spaces which fill with arterial blood when the penis is in a state of erection. This is only a part of the physiological process leading to enlargement of the organ. The glans is the terminal portion of the corpus spongiosum, and also distends with blood in the presence of sexual excitement.

The causes of a persisting enlargement of the penis are either nervous or vascular, and each can be recognized by the shape of the organ. Priapism is due to a nervous lesion, and the appearance of the penis is similar to that seen in the physiological state. On the other hand, thrombosis of the corpora cavernosa leads to the formation of a hard and tense swelling of the whole organ, *except the glans*. If the clotting is more extensive in one corpus than the other deviation occurs, and the penis is bent to the side which is more thrombosed. There are three diseases which may lead to the clotting of the blood in the cavernous spaces. They are malignant disease of the left kidney, lymphatic leukaemia, and arteriosclerosis. Whichever the cause, the condition is fortunately rare.

The explanation of the reason why thrombosis of the corpora cavernosa occurs in carcinoma of the left kidney is purely anatomical. The spermatic vein on this side of the body enters the renal vein. If the latter becomes blocked with growth at the point of entry of this tributary, stagnation of the blood occurs in the penis and left testicle, so that not only does pseudopriapism occur, but there is a temporary swelling of the latter due to the block in the pampiniform plexus. These veins can be felt as a hard mass in the inguinal canal. No original observations can be made on penile thrombosis due to lymphatic leukaemia, for the writer has never seen a case. However, most textbooks describe it as a complication of this blood disease, and it must be accepted as a fact unless proved to the contrary.

With regard to arteriosclerosis as a causal agent, two cases of thrombosis of the penis have been investigated in which the local lesion has been proved to be due to arterial disease. A study of the literature reveals that a considerable number have been described in which the cause is stated to be unknown. More accurate observation would have cleared up the mystery. In view of the fact that the nipple of a female breast consists of erectile tissue it is probable that thrombosis has occurred in this organ also in relation to arterial disease, but such a case has yet to be recorded.

Thrombosis: Case Records

The details of the two cases of thrombosis of the penis are as follows:

The first case to be carefully investigated was that of a man, aged 35, who was admitted to Whipps Cross Hospital with the whole of the penis, apart from the glans, swollen and hard. The enlargement extended back to the bulb. The history was that the swelling had been of gradual onset, a period of three days having elapsed before it reached its full size. During this time repeated coitus had failed to produce any change in the organ. On admission the patient was in considerable distress, and complained of difficulty in micturition. There was no pain. The medical officer,

* A communication read before the Dutch Urological Association at Amsterdam, April, 1934.

believing the condition to be due to a nervous lesion, tried the effect of spinal anaesthesia, with a negative result. Leukaemia was then excluded by an examination of the blood. The Wassermann reaction was negative. Marked thickening of all superficial arteries was noted. The systolic blood pressure was 145. The urine was normal. In three weeks the penis had resumed its natural size, and the patient was then discharged. It is now over twelve months since this patient was in hospital. Every effort has been made to trace him, but letters remain unanswered.

The second case is of a more recent date. The patient, aged 45, has an exactly similar history to that of the first case. Again the medical officer thought he was dealing with a nervous lesion, and injected stovaine into the spinal theca, with an unsatisfactory result. The patient was obviously suffering from arteriosclerosis, the radial arteries being hard and tortuous. The Wassermann reaction was negative, as was also the examination of the blood for leukaemia. The systolic blood pressure was 130. Urine normal. The patient discharged himself from hospital one week after admission, with no change in the size of the penis. This was at the beginning of July, 1932. Three months later, in answer to a letter, he presented himself for examination. The history was that the penis had become normal in size three weeks to a month after the onset of the swelling. He stated that he was enjoying good health apart from occasional headaches. His systolic blood pressure was 140, ten points higher than in July, 1932. Definite thickening could be felt along each corpus cavernosum. The patient stated that he was impotent. Sexual desire was strong, but there was no response in the penis, evidence that the nervous mechanism controlling coitus was intact, but the vascular system defective.

Local treatment in these two cases consisted of the application of evaporating lotions to the perineum, but it should be remembered that absorption of the clot occurs irrespective of either surgical or medical interference. Some writers advocate incisions into the corpora cavernosa, which shows their lack of understanding of the pathology of the lesion.

The other type of thrombosis of the penis occurs in acute infective processes of the urethra, the best example of which is gonorrhoea. In this condition it is the superficial veins which tend to clot, leading to oedema of the prepuce and subcutaneous tissues. It is really a case of phlebitis in an unusual situation. Resolution occurs slowly, but no permanent ill effects follow this complication. Dr. J. L. Morton reports to the writer that he has had under his care a man with thrombosis of the dorsal vein who has suffered for many years from syphilitic arteriosclerosis.

The study of urethral haemorrhage introduces problems of great pathological interest. Bleeding may occur either at the beginning or the end of micturition. On the other hand, it may be entirely independent of the passage of urine, and, lastly, it will take place during coitus.

Haemorrhage

There is no need to discuss in detail the causes of urethral haemorrhage in association with micturition, for they are well recognized. Any acute urethritis—gonococcal, tuberculous, or that due to the *Bacillus coli*—by the severity of the inflammation, will give rise to the dripping of blood, either before or after micturition. Also tumours of the urethra, such as papilloma and carcinoma (both rare diseases in this situation) cause haemorrhage. Again, a stone impacted in any part of the urethral tract may cause bleeding. One case of haemorrhage immediately after micturition is worthy of record on account of failure to discover the cause.

The patient was a well-built lad of 16½ years, who complained of a pain at the end of the penis after micturition, followed by a profuse flow of blood from the urethra. Quite two tablespoonfuls of blood were seen to escape immediately after micturition had ceased. The cause of the bleeding remains a mystery, in spite of all the known methods of

investigation, such as cystoscopy, urethrography, urethroscope, urine analysis, and examination of the blood. All proved negative to disease except the urethroscopy, which revealed what looked like a small ulcer on the ventral aspect of the mucous membrane of the bulbous urethra. Irrigation daily with a solution of one in 10,000 silver nitrate has failed to stop the bleeding, and one is forced to the conclusion that the condition is due to self-inflicted trauma.

The best-known examples of urethral haemorrhage independent of micturition are rupture of the tube, complete or incomplete, and damage to the mucous membrane due to instrumentation. It must not be forgotten that the passage of a bougie, catheter, or cystoscope, however gently handled, always causes bleeding. With the skilled urologist this is only detected by the microscope. Occasional spontaneous urethral haemorrhage will occur from the vessels which supply an adenomatous prostate. Oozing of blood may be continuous and of such severity as to cause clot retention. This complication is in no way caused by the act of micturition, but is probably initiated by straining at stool.

Lastly, we have to consider what may be termed urethral epistaxis in relation to coitus. It is only within the last few years that this phenomenon has been recognized as a manifestation of serious arterial disease. Previously it was labelled "malade imaginaire." Three cases have been carefully studied and kept under observation, one patient still being alive and the other two dead. Their notes are given in full in the presidential address to the Section of Urology, October, 1933, but it may be helpful to recapitulate some of the salient points in the clinical histories.

The first case was dismissed as an anxiety neurosis, and it was only when the patient, 35 years of age, brought his pyjamas stained with blood to the consulting room that it was realized a mistake in diagnosis had been made. It was then decided to examine carefully the vascular system. All the superficial arteries were clearly sclerotic and the systolic blood pressure raised. The urine showed evidence of changes in the kidneys, hyaline and granular casts being present. The loss of blood during coitus continued until the patient became seriously ill with chronic nephritis, from which he eventually died.

Thus the sequence of events in the medical history is that this young man suffered from arterial disease from an early age, that the strain of coitus on occasions caused rupture of a blood vessel of the urethral mucous membrane, and that the uraemia from which he died was the result of arteriosclerosis.

The second case was that of the editor of a well-known paper, a man aged 45, who had a severe haemorrhage into the prepuce during coitus. He also gave a history of urethral haemorrhage. It was already known that for some years he had suffered from arterial disease and hyperpiesis. A few months after being treated for the haematoma of the prepuce he died of cerebral haemorrhage.

The third patient is still alive, having been under medical observation for the past two years. He also has had urethral haemorrhage during coitus, due to arteriosclerosis and high blood pressure. Chronic nephritis is present, but, in spite of his disease and the fact that he is 64 years of age, he continues to work, and makes few complaints about his health.

Conclusion

These cases demonstrate very clearly how important it is that the urologist shall be just as much a student of medicine as he is of surgery. In fact, if he is satisfied to confine his interests to instrumental and operative technique, he will gradually disappear as an expert in diseases of the genito-urinary tract. The urologist of the future must be a man competent to diagnose accurately all constitutional diseases which first manifest themselves by purely urinary symptoms.