the consequent increase in tachycardia, brought about by separation from familiar surroundings and perhaps frequent changes of attendants, all weigh heavily in favour of nursing the infant at home unless circumstances make this quite impossible. However exacting the task the mother should be persuaded to make the extra effort. It should be explained to her that in no disease is patient devotion to the interests of the child better rewarded in the end.

Intensive parenteral vitamin or liver therapy was not employed in this series. A high vitamin intake has been advised as a measure of protection against superimposed infection rather than from a predilection for any particular theory of causation. The main principles in management are: scrupulous attention to bodily hygiene, particularly when desquamation is in process; most careful segregation from any human source of nasopharyngeal or respiratory infection, and immense patience in feeding—all food should be boiled or cooked and served as cold as possible (sometimes the only way food or fluid will be taken at all), and food should not be forced into the mouth because of the danger of injury to the buccal mucous membrane and subsequent stomatitis.

The judicious use of sedatives and the local application of such soothing and cooling preparations or physiotherapeutic measures as seem to bring the greatest relief to the patient without causing maceration of the tissues must be a matter of individual experiment and ingenuity. There is no single royal road to success in easing the intense discomfort which is often the most harrowing feature of the disease.

Summary

A review of 65 cases of pink disease is presented. The progress in 62 was ascertained, 53 completely recovered, and nine were fatal.

The incidence, age of onset, and duration of the disease are given.

The symptoms are divided into two groups, and these are discussed.

Treatment and the prevention of secondary infections are dealt with, and the advantages of home nursing are emphasized.

PELLAGROUS ENCEPHALOPATHY

BY

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The following account deals with a deficiency syndrome which occurred among prisoners of war in Singapore. As the title indicates, the clinical manifestations suggested a brain lesion: the only pathological support for this contention was the naked-eye appearances of the brain and spinal cord at the necropsies on the fatal cases. No histological examination was possible. The syndrome occurred in the form of a comparatively small outbreak after large epidemics of vitamin B₁ deficiency resulting in neuritic and oedematous beriberi and Wernicke's encephalopathy, and later epidemics of B₁₂-complex deficiencies giving rise to angular stomatitis, glossitis, scrotal dermatitis, pellagra dermal lesions, retrobulbar neuritis, and the painful-feet syndrome. So far as I am aware, no such outbreak as this has been previously reported.

This neurological disorder was remarkable for the appearance of spastic paraplegia, and for the sake of description and recognition among the medical officers of the camp it was termed the "spastic syndrome." The initial illness varied in severity from a state of deep coma to some effects so slight as to be recognized only by the existence of extensor plantar responses. The residual symptoms similarly varied from a persistent spastic paraplegia so severe as to disable walking to a recovery so complete that no signs remained.

With the assistance of the ward medical officers who had charge of them I was able to keep a close watch on the first

group of cases to be admitted to hospital between August and November, 1942. Other cases arising later I saw at less frequent intervals, but the total number under close personal observation was 29. In January, 1944, I extracted from the hospital records data from 34 undoubted cases of this syndrome which were not included in my previous number. Under the prevailing circumstances it was impossible to see or arrange to see every case suspected of the condition, so that this report of 63 cases fails to embrace the total number figuring in the official camp statistics.

In this series there were 6 deaths. Four of these patients, after an insidious onset, deteriorated rapidly and died within 3, 8, 17, and 18 days of admission to hospital. Necropsies were performed by Capt. Lennox, R.A.M.C., and the naked-eye findings are included in the case reports. One patient died of pulmonary tuberculosis and no necropsy was carried out. The sixth died a year after onset with progressive mental deterioration; no changes were found in his brain comparable with those in the other cases.

The records I managed to keep with me and finally bring home in October, 1945, were of the briefest. As my notes on various diseases grew in number, so the records needed to be condensed still further to reduce their bulk, for there was the ever-present possibility of confiscation.

Fatal Cases

Case I

This patient, who was admitted on Aug. 31, 1942, had noticed for one week double vision, burning pains in the legs and left hand, general weakness, and difficulty in chewing his food. Mentally he was retarded, and was easily moved to laughter by unamusing remarks. There was general weakness of his limbs of moderate degree, most marked in the grip of the left hand. No changes in sensation were observed.

When I next saw him, eight days later, generalized rigidity of his limbs had developed and he was unable to sit up. His tendon reflexes were exaggerated and there was ill-sustained clonus at both ankles. The abdominal reflexes were weak, and the plantar reflex was extensor on the left side. He was given "marmite," 2 oz. (56.7 g.) daily, from then on.

Six days later he became incontinent of urine, and during the next few days he was mentally confused. By Sept. 16 his legs had become extended and his arms flexed rigidly across the chest. There was some weakness of the left facial muscles. His temperature rose to 101° F. (38.3° C.). The following day he became incontinent of stools consisting of blood and mucus, lapsed into unconsciousness, and died on Sept. 18.

At necropsy small translucent dots were to be seen scattered about the white matter of the frontal, parietal, and occipital regions of the cerebral hemispheres. These dots were 2-3 mm. in diameter and had the appearance of sago grains in milk. In the colon there was patchy erosion of the crests of the mucous membrane in the distal part of the sigmoid and rectum.

Case II

For a fortnight before his admission on Sept. 4, 1942, this patient had noticed dimness of vision, and for a week unsteadiness of gait and pain in the neck. On examination there was marked dulling of his mental faculties, with disorientation for time and place. Weakness and rigidity were present in all his limbs, with exaggerated tendon reflexes and extensor plantar responses. Other evidence of dietary deficiency was manifested by angular stomatitis, glossitis, and scrotal dermatitis. For three days he was given 1.7 ml. (1 ampoule) of "nicamide" (nicotinic acid diethylamide) intramuscularly and 1.7 ml. by mouth daily. For the next three days 3.4 ml. was given by mouth daily, and subsequently "marmite" 3 oz. (85 g.) daily.

Five days after admission he became incontinent of urine and faeces; he could not speak or swallow, but could understand simple requests and statements. Within the next two days he deteriorated rapidly; his legs became rigidly extended and his arms flexed across his chest. His face was expressionless and practically immobile. The blinking reflex was lost but some vision was present. He moaned continuously, so far as could be gathered because of pain in his limbs. This pitiful condition gave place to coma, in which he died on Sept. 21.

Changes in the cerebral hemispheres, identical with those in Case I, were found at necropsy.

Case III

This patient was admitted from another camp on March 22, 1943, in a stuporous condition, with a slip attached to his shirt bearing the words: "Fever. Choking feeling and failing vision." He could

be roused only to the extent of complaining of headache when asked his symptoms. His temperature was 104° F. (40° C.). There was an extensive stomatitis with angular fissures, pharyngitis, scrotal dermatitis, and keratitis of the right eye. The latter was occurring in epidemic form and was undoubtedly a deficiency manifestation. His heart sounds were of the tic-tac variety, and his blood pressure 120/90. Examination of his nervous system showed that the reaction of the pupils to light was sluggish; reaction to accommodation could not be tested owing to lack of co-operation. There were weakness of the left side of the face and generalized weakness of the limbs, which, so far as could be ascertained, was most marked in the left arm. The abdominal and cremasteric reflexes were absent, as was the right knee-jerk. The tendon reflexes of the arms and left patella were normal, but there were bilateral ankle clonus and extensor plantar responses. For treatment he was ordered "marmite," 3 oz. (85 g.) daily, in addition to milk and eggs.

The following day he became incontinent and passed seven semi-solid stools. A lumbar puncture was performed unsuccessfully by two competent medical officers. I could obtain spinal fluid only by aspiration. Examination of the cells and protein showed no deviation from the normal. On March 24 it was noticed that a keratitis had developed in the other cornea, and on the 25th he became deeply comatose and died.

At necropsy similar changes were found in the cerebral hemispheres to those in the two preceding cases. The colon showed extensive mild lesions of bacillary dysentery, with ulcers up to 8 mm. in diameter in the upper half of the large bowel.

CASE IV

This patient, who was admitted to hospital on March 16, 1943, presented what became recognized as the most typical triad—difficulty in walking, dimness of vision, and mental dullness. These symptoms had preceded his admission by two to three weeks. He appeared quite normal mentally, but stated that his memory was poor and his mental faculties slow and dulled. His cranial nerves were normal, and the only objective changes in his nervous system were the absence of abdominal reflexes on the right side and exaggeration of knee- and ankle-jerks. His diet was supplemented by "marmite," 3 oz. (85 g.) daily.

On the 22nd, six days after admission, he became quite rapidly confused. Within a few hours his limbs had taken up the position noticed in the previous cases—legs extended and arms flexed. With this there were associated exaggeration of all tendon reflexes, bilateral ankle clonus and extensor plantar responses, and loss of the abdominal and cremasteric reflexes. Two days later he developed double incontinence, and died, with a temperature rising above 105° F. (40.6° C.), eight days after admission. A lumbar puncture before death showed fluid under normal pressure and a normal protein and cell content.

At necropsy identical changes were found in the cerebral hemispheres to those in the three previous cases. In the colon there was recent necrosis of the crests of the mucous folds.

Comment

Perusal of these case histories shows (1) a fatal course terminating in from four to five weeks from the onset of the first symptom; (2) clinical evidence of interruption of conduction in the pyramidal system; (3) macroscopical evidence of a pathological process in the white matter of the cerebral hemispheres which would account for most of the physical signs and symptoms, including failure of vision. In three out of the four cases there was an ulcerative condition of the colon. Two showed other evidence of deficiency of part of the B₂ complex. None of them, to my knowledge, had recently suffered from dysentery, malaria, or other fever, and repeated blood films failed to reveal parasites. Other negative findings that call for comment are: (1) the absence of sensory loss in Cases I, II, and IV, from whom some co-operation was possible; (2) no naked-eye changes in the spinal cord; (3) an absence of meningeal reaction, as found by lumbar puncture on two of these cases.

The differentiation from the other neurological disorders—namely, Wernicke's encephalopathy and an acute encephalitis—which were occurring fairly often never gave rise to much difficulty, as the early symptoms and signs of this syndrome had become well recognized in the less acute cases which had preceded these four fatal ones. It is appreciated that difficulty might arise, especially in the case of acute encephalitis; and the history and nature of the early symptoms preceding by some days or weeks the more urgent manifestations of acute disorder of the nervous system are important to the diagnosis of the present syndrome. Wernicke's encephalopathy can usually be recognized by the symptoms of vomiting and

unsteadiness of the body or eyes, and the characteristic nystagmus and ocular palsies. The tendon reflexes are more frequently lost than increased. Only in encephalitis is any deviation from normal expected in the spinal fluid.

The association of this syndrome with other deficiency manifestations, and the argument for its recognition as such, will be adduced later.

Severe Cases with Ultimate Recovery

Case V

This man was admitted on Sept. 7, 1942. He had suffered from the very prevalent complaint of "pains in the feet" for six weeks. During the preceding three weeks he had developed general weakness, mental depression, anorexia, and failure of vision. On examination he was seen to be suffering from angular stomatitis and glossitis. He was mentally depressed and anxious, and apprehensive about himself. His face was without expression and almost immobile. His visual acuity was reduced to an inability to read newspaper print. There was general muscular weakness and rigidity, with exaggerated tendon reflexes, ankle clonus, and bilateral extensor plantar responses. The abdominal and cremasteric reflexes remained. No sensory loss, deep or superficial, was found. In addition to a general improvement of his diet he was given "hepatex," 4 ml. intramuscularly, and "marmite," 2 oz. (56.7 g.) daily.

Two days later he was unable to feed himself and became incontinent of urine. A week later he had developed dysphagia and dysarthria, and his limbs had taken up the characteristic position of flexion of the arms and extension of the legs. He moaned continually and became rather drowsy. His condition at this stage exactly resembled that of Case II during the last ten days of his illness. However, during the next week he began to improve, with a reduction in the rigidity and drowsiness, and in less than a month he was walking about unaided. Within a few days of the commencement of his recovery he developed an inversion of emotional expression and would cry when amused. Although his tendon reflexes remained abnormally brisk until June, 1943, his plantar responses had reverted to normal within a fortnight of his first sign of improvement. When last seen, in June, 1943, he was normal in all respects.

Case VI

This patient had noticed double vision and a tight feeling around the head for a fortnight before his admission on Aug. 3, 1942. He also described feelings of "pins-and-needles" and "shocks" all over his body. There was general slowing of his mental processes; he was easily amused, and was slow in his speech. He exhibited general muscular weakness and rigidity, with exaggeration of all tendon reflexes and knee and ankle clonus. The abdominal reflexes were absent, except in the right upper quadrant, as was the right cremasteric reflex. The right plantar response was equivocal, the left flexor. No sensory disturbance was discovered. "Marmite," 1½ oz. (42.5 g.), was added to an improved diet.

Four days after admission he had become quite euphoric. The muscular weakness and rigidity had increased, and he could not stand up. His physical and mental states deteriorated for the next twelve days, during which time he received intravenous vitamin B₂, 2 mg. daily. Incontinence of faeces developed. His spinal fluid was examined and found normal. On Aug. 20 there were signs of improvement in his general condition, but he was unable to move his ankle or foot joints, and his plantar responses had become extensor. From then onwards his mental condition improved and, except for occasional attacks of depression, returned to normal, but he was still bed-ridden in August, 1945, with a severe spastic paraplegia.

Case VII

This man, who was admitted on Aug. 12, 1942, had been weak and unsteady on his legs for two weeks, had become forgetful, and had developed a stutter. His condition at the onset was unique among the cases seen, and presented a picture usually the result of congenital cerebral injury or disease. At rest his face presented a vacant expression with open mouth, but this was distorted repeatedly by grimaces and rolling of his eyes and head, accompanied by athetotic movements of the limbs. His gait could only be described as fantastic—a combination of ataxia with the athetotic movements previously mentioned. He was disinterested in his food, and was quiet and reserved in his relations with other patients and friends. He was uncommunicative with everyone, and very little information could be got from him. Apart from increased activity of his tendon reflexes I could find no other abnormal signs.

Three days later there was no change in his condition, but I found that by taking a forceful line with him I could get him to walk normally. I erred in diagnosing his condition as hysteria and discharging him on the 19th. In mitigation I would like to point out that he was only the third case of this series to be admitted and that he presented a new clinical picture.

A month later he was readmitted with the history that he had been very quiet in his quarters until Sept. 12, when for some hours he became violently agitated, with much rolling of his head and eyes. He was then quiet again until the 18th, when he started screaming and throwing himself about on his bed. He was completely disorientated, and was found to have a temperature of 102° F. (38.9° C.). Later in the day, after his admission, he had an epileptiform seizure with twitching of the right side of the face and right arm, and incontinence. On recovery he behaved in a noisy, hysterical manner, but ceased on being reprimanded. For some hours afterwards he exhibited athetotic movements of his limbs and was incapable of speech. On lumbar puncture the fluid was found to be under normal pressure, with a normal cell and protein content.

When seen on Sept. 26 he had recovered some power of speech, using simple words slowly. His limbs were weak and rigid, with exaggerated tendon reflexes; ankle clonus and extensor plantar responses were present, but the abdominal and cremasteric reflexes were absent. During the next fortnight he became rather emotional and would have fits of crying for no apparent reason. By March 23, 1943, he was able to walk about, but with a fantastic heaving gait as if weights were attached to his feet. All the athetotic movements and rolling of his head and eyes had disappeared. When last seen in August, 1945, he had a spastic paraplegia which just allowed him to get about unaided.

Comment

The symptoms and progress of these three severe cases, while differing in some respects both from each other and from those in the previous group, accord well enough to be recognized as belonging to the same syndrome. Case V reached most nearly to the condition seen in the fatal cases just before death. All developed a spastic paraplegia.

General Description

The syndrome, as exemplified by the majority of cases, is ushered in by various combinations of the following symptoms: locomotor disability—taking the form of weakness, stiffness, or unsteadiness of the legs; failure of vision; diplopia; pains in the feet; pain in the back; paraesthesiae in the feet; cramps; changes in the mental sphere, such as depression or mental dulling. Nocturnal incontinence of urine occurred as an early symptom in three cases. Two had epileptiform seizures. Only one or two complained of cramps. Marked spasticity with or without extensor plantar responses was present in all cases. The following is an analysis of the commoner symptoms.

Symptoms	% of Cases
Difficulty in walking	53.9
Loss of visual acuity	46.0
Mental changes	41.2
Pains in feet (painful-feet syndrome)	38.0
Paraesthesiae in limbs	17.4
Pain in spine	6.3
Diplopia	5.0

In the early stages there may be no objective signs apart from unusually brisk reflexes and a rather hysterical behaviour. It was observed on several occasions that, on examination, men who had walked to the out-patient department complaining of pains in the feet revealed exaggerated knee- and ankle-jerks and extensor plantar responses. After a night's rest in hospital these signs would disappear. I assumed that in these cases there was only a minimal degree of the condition under discussion.

When this nervous affliction became well recognized medical officers began to report the occurrence of increased tendon reflexes, sometimes accompanied by clonus and extensor plantar responses, in ward patients suffering especially from dysentery and prolonged fevers. Both these groups of patients—those suffering from pains in the feet and those just mentioned—received special care, and I am not aware of any that failed to recover fully.

The difficulty in walking was due to a progressive spastic paraplegia with the development of the signs usually associated with it—viz., spasticity, weakness, exaggerated tendon reflexes, extensor plantar responses, and loss of abdominal and cremasteric reflexes. In just over 50 % the signs of pyramidal-tract involvement were confined to the lower half of the body; in about 28 % the upper limbs were included; and in the remainder there were signs of inclusion of the highest part of the system, with loss of facial expression, facial immobility, dysarthria, and dysphagia.

Under the conditions which prevailed it was not possible to test the visual acuity of all these patients; the loss was never severe and vision recovered rapidly, which was in striking contrast to the chronicity of the cases suffering from retrobulbar neuritis. In only two patients did I find any abnormality on ophthalmoscopic examination: in one the disks were very pink and the edges ill defined; he later developed further impairment of visual acuity and became established as a case of retrobulbar neuritis. The other had well-marked papillitis with exudate close to the disks; in this case the spastic syndrome was complicated by oedema of the legs and pellagra skin lesions, faucial diphtheria, S.T. malaria, and later a polyneuritis affecting all limbs and both sides of the face. He made a satisfactory recovery without visual defects.

The mental changes comprised general dulling, depression, loss of emotional control, and hysterical behaviour. One patient developed ideas of persecution which persisted until his death a year later, by which time there had been considerable physical and mental deterioration. Another developed grandiose ideas which lasted many months, and a third became confused and aggressive for a few weeks.

Pains in the feet were common, but this syndrome was widespread at the time, as were the other signs of deficiency disease which were found associated in the spastic syndrome in not more than one-third of the cases. The painful-feet syndrome has been well described by J. A. Page (1946). The paraesthesiae comprised numbness and tingling and were unaccompanied by objective signs. These symptoms tended to disappear rapidly after admission to hospital. The loss of bladder control arose early in some cases as nocturnal enuresis. Pain in the spine was usually located in the cervical or lumbar region, and occurred in severe cases with much rigidity and helplessness. The diplopia was usually of short duration and disappeared soon after admission. In some cases this symptom was intermittent.

Investigations

Laboratory facilities were limited, and the staff was continuously at work on blood films and stool examinations. Fractional test meals were performed on three cases, the results showing one to be achlorhydric and two normal. Lumbar punctures were done on six cases, and the cells and protein were normal in all.

A survey in respect of age, length of service in the East, previous illnesses, habits in regard to consumption of alcohol, and type of work as a P.O.W. failed to produce any facts of significance.

Course and Treatment

The onset and progress were fairly rapid in the majority of cases, and my records show that in 18, where information is available, an average period of 18 days' illness preceded admission to hospital, the extremes being 3 and 42 days. All non-fatal cases deteriorated for an average period of 21 days after admission. The shortest period from onset to recovery, when a paraplegia had developed, was 9 weeks.

Owing to the later division of the main Changi Hospital into two separate camps I am unable to give exact figures bearing on the late results of the disease in all cases. Of my original 29, 6 died (one from pulmonary tuberculosis), 9 recovered completely, and 14 were left with signs of paraplegia of varying extent, of which 2 were bedridden when last seen, in September, 1945.

The means available for the treatment of the earlier cases consisted in an addition to their diets of reconstituted milk (1-2 pints: 0.56-1.1 litre), "marmite" (1-3 oz.: 28.3-85 g.), "nicamide" (3.4 ml. daily) for a maximum of 11 days, parenteral vitamin B₁ up to a maximum of 16 mg. per case, liver extract up to a maximum of 12 ml., and red-palm oil (1-3 oz.: 28.3-85 g. daily). Rice polishings, pea-nuts, eggs, tinned meat, and fresh vegetables were given as available. It was not apparent that any addition affected the course of the disease.

Discussion

Evidence is presented that under certain conditions of nutritional deprivation a disorder characterized by signs of damage to the cortico-spinal innervation makes its appearance. When severe this condition is responsible for widespread disorder of cerebral function resulting in disturbance of memory, coma with quadriplegic rigidity characteristic of the decorticate animal, and in some cases convulsions. As recovery occurred spastic paraplegia was the common residual state. In minor affections less evidence of the initial cerebral illness may yet be followed by a crippling spastic paraplegia or a lesser degree of spastic weakness. Complete recovery may ensue after a severe initial onset.

Spastic paralysis has been reported in severe classical pellagra. Thus Heatley-Spencer and Biggam, in the *British Encyclopaedia of Medical Practice*, state: "Sensory changes are common in the lower limbs and consist of various paraesthesiae and numbness. Objective sensation usually remains intact and there is no alteration of deep sensibility. Romberg's sign is rare. Pain in the dorsal or lumbar regions is common: peripheral neuritic pain may be severe and is often a late symptom. Stiffness and some loss of power, with painful cramps in the lower limbs, are common. The reflexes are variable, the knee-jerks being in some cases exaggerated, with a spastic rigidity, and in others lost. Mental changes are important and begin with mental dullness, slow response, and loss of memory."

Manson-Bahr, in his *Tropical Diseases*, includes ptosis, diplopia, amblyopia, and rarely epileptiform convulsions among the signs and symptoms, and states that later the gait becomes uncertain and of the spastic paraplegic type. This author mentions a very acute form of pellagra, described under the title of "pellagra typhus," characterized by intense prostration, high temperature, muttering delirium, pronounced nervous tremors, generalized rigidity, and convulsions. The acute cases reported in this paper seem not dissimilar.

The additional point that this syndrome arose in the course of other manifestations of vitamin B deficiency which are recognized as part of the classical picture of pellagra lends support to the view that it is itself an isolated manifestation of that disease. The B complex comprises several factors, and the experience gained in our camp suggested that there was a different time factor for each before evidence of deprivation showed itself, the mucous membrane and dermal lesions of pellagra appearing first and the nervous lesions later. It is of interest that not more than one-third of the cases of pellagrous encephalopathy here reported showed other evidence of deficiency, but this lack of association was reported by Spillane and Scott (1945) in their account of deficiency manifestations among German prisoners of war in the Middle East.

Conclusion

The above findings are based on 63 cases of a deficiency syndrome, which has been given the name of pellagrous encephalopathy, occurring among prisoners of war in Singapore. There appears to be sufficient evidence to include it among the manifestations of vitamin B deficiency and as part of the pellagra symptom complex. Previous reports have been made in the literature of limb weakness, and spasticity with and without extensor plantar responses (Wilkinson, 1944; Spillane and Scott, 1945; Clarke and Sneddon, 1946; Pallister, 1940), in association with other deficiency manifestations, but a clear-cut syndrome as reported here appears to be unique.

My thanks are due to all those medical officers in the Changi Prisoner-of-War Hospital under whose care these patients were admitted; and to Dr. D. Denny-Brown, of the Department of Neurology, Boston City Hospital, for his help and encouragement in the writing of this article.

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A scientific meeting and the resumed annual general meeting (for members only) of the Nutrition Society will be held at the London School of Hygiene and Tropical Medicine, Keppel Street, W.C., on Saturday, Feb. 22, at 11 a.m. The speakers and subjects at the scientific meeting are as follows: G. Fraenkel, Requirements of Insects of Known and Unknown Members of the Vitamin B Complex; O. Kestner, Importance of Mixed Food for Nutrition; T. F. Macrae and G. A. Childs, Nutritive Value of Food Served in the R.A.F. during the War; J. Ganguly, S. K. Kon, and S. Y. Thompson, Form of Vitamin A in Cow's Milk; R. Braude, Stimulation of Growth and/or Fattening of Pigs by Synthetic Oestrogens; G. Dunlop, Nutritional Variation of Soils and Pastures on Scottish Hill Grazings; E. M. Hickmans, S. Bishop, and Y. Williams, Effect of High Protein Feeding on Marasmic Infants following Operation for Pyloric Stenosis; Z. A. Leitner, T. Moore, and I. M. Sharman, Vitamin A in Rheumatic Fever. Non-members are admitted to meetings of the society only by the introduction of a member. Meetings of the society are private and no unauthorized account of its proceedings may appear in the Press. Further details of the society can be obtained from the honorary secretary, Dr. Leslie J. Harris, Nutritional Laboratory, Milton Road, Cambridge.

Medical Memoranda

Case of Ectopia Testis with Seminomatous Change and Torsion

The following case is interesting in that it illustrates three pathological conditions: first, ectopia testis of an unusual type; secondly, a malignant tumour of that organ; and, finally, torsion. The liability of malignant change in an ectopic testis is a statistically supported, albeit disputed, fact. Gordon-Taylor (1938) states that 15 out of 50 testicular tumours occurred in undescended testes. Tumours of the testis, however, are not common, and Barrington (1939) remarks that out of 9,714 admissions to St. Peter's Hospital for diseases of the male genital system only 14 were testicular tumours. In our case an extreme example of undescended testicle had undergone both malignant change and torsion.

CASE HISTORY

A man aged 38, married but with no children, had recently been discharged from the Army with a clean bill of health. He had once during his service—in June, 1945—had an attack of abdominal pain, but after 24 hours' observation had completely recovered. In June, 1946, he called in his doctor one morning because of slight abdominal pain and having had several loose stools during the night. On examination there was a generalized abdominal tenderness with considerable rigidity, but temperature and pulse were normal. His doctor therefore decided to see him later in the day before coming to any conclusion. When seen during the afternoon he had increased pain, together with nausea, and there was marked tenderness in the right iliac fossa. There was also a rise of temperature to 101° F. (38.3° C.). He was then admitted to the Victoria Infirmary, Northwich. Here examination confirmed these findings, and a provisional diagnosis of acute appendicitis was made. It was, however, noted that the right testis was absent from the scrotum, and the inguinal canal was not patent to the exploring finger. Consequently a reservation was made that there was a possibility of some morbid change in an ectopic testis accounting for the symptoms.

The abdomen was opened by a pararectal incision in the right iliac fossa. When the peritoneum was opened a quantity of blood-stained fluid escaped, and on exploring the pelvis with the hand a rounded mass was found in the retrovesical pouch. This was delivered and seen to be a globular mass the size of a cricket ball, which was plum-coloured and had the appearance of a twisted ovarian cyst. It had a twisted pedicle 1½ in. (3.75 cm.) long which emerged from the parietes at about the site of the internal inguinal ring. It was clamped, ligated, and divided near its base and the mass removed. There were no adhesions, and no difficulty was encountered. There was no sign of pelvic or lumbar glands, and the liver was palpated and felt quite normal. The wound was closed without drainage and the patient returned to bed. He recovered uneventfully.

The tumour was incised, revealing that it was the missing testis. It was sent for section to Dr. W. H. Grace, who reported that it was a seminoma which had undergone torsion. The patient's urine was later also submitted to Dr. Grace, who determined that the Aschheim-Zondek reaction was negative, but as this was about three weeks after the removal it could only be an indication that there was no large metastasis as yet. After discussion it was decided not to recommend deep x-ray therapy at this stage, and on this point we invite criticism.

COMMENT

Perhaps the chief interest of this case lies in the abnormal anatomy of the testis and in the complete, or almost complete, absence of symptoms as demonstrated by a successful service in the Army. The testis had picked up a complete visceral peritoneum and had come to be a free intraperitoneal organ—a very unusual state of affairs. That it had undergone torsion was perhaps not surprising after, owing to the seminomatous change, it had attained the size and weight it had. Nevertheless it must be assumed that the symptoms which led to operation and ultimate diagnosis of the condition were due to torsion, as no doubt was his attack in June, 1945, although this must have corrected itself, and to this fact the patient may, we hope, owe his life.

We wish to thank Dr. W. H. Grace for his pathology report.

Hartford, Cheshire.

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