injected subcutaneously. The lung signs remained unaltered. Later in the day the pulse became more forcible. Flatulence was now a marked and troublesome feature, but was relieved by enemata. The urine contained bile and chlorides, but no albumin or sugar. Temperature had now fallen to 97°.

On March 5th the pulse was considerably improved, and had become full and regular. Flatulence was still marked. An interesting feature from now until his death was the excessive amount of urine passed by the patient, as much as 104 oz. being passed in the twenty-four hours on two or three occasions. There was little change in the physical signs in the lungs. Temperature was 97.6°, respirations 26, pulse 98.

On March 6th nausea became evident. Flatulence was still pronounced, and hiccup occurred in the morning. The mouth was by now exceedingly raw and painful, and, for the first time, the stools appeared putty-coloured. The chest signs seemed less marked. Gardenal was given at night.

On March 7th, after the patient had passed a delirious night, the jaundice seemed to be a little less pronounced. The abdomen was very distended, and enemata only gave temporary relief. Hiccup was not so obvious as on the previous day. The stools were soft and had the appearance of "silver fish." Examination of the chest now revealed an increase of sounds at the right base. Temperature was 98.4°, pulse 106, and respirations 28.

By March 8th the patient was very distressed, flatulence causing considerable discomfort. A faint, slightly raised, morbilliform eruption was now just discernible through the jaundice all over the body. The heart sounds were feeble, but with a regular pulse and apex beat. The abdomen had become a little more tender over the gall-bladder area, but no intra-abdominal viscus was palpable. Stools were still loose, with the "silver fish" appearance. Urine passed late at night was cloudy, and microscopical examination disclosed red blood corpuscles and epithelial debris. Temperature was 99.8°, and respirations 40. The temperature rose to 101° just before midnight. Gardenal was taken at night.

On March 9th, the last day of the illness, after a very delirious night, the patient appeared more rational. Respirations increased to 50. At about 5 p.m. respirations had further increased to 56 and the pulse became progressively feebler. At 7 p.m. the patient passed about two ounces of brown faeces streaked with blood. At 9.15 p.m. a large, soft, "silver fish" stool was passed. Urine was now loaded with red blood corpuscles and epithelial debris. At 11 p.m. the patient was in a state of collapse, and the pulse was barely perceptible. Respirations increased to 60. Cardiazol 1 c.cm. was again taken. Oxygen was administered throughout the day, but with no beneficial effect. Just before death, which occurred at 11.30 p.m., the patient passed a putty-coloured stool and about two ounces of bright red blood per rectum.

Necropsy

Post mortem, intense jaundice was the outstanding feature. All the internal organs examined were markedly bile-stained. There was no evidence of new growth and no pus to be found anywhere. The liver was slightly enlarged, but the gall-bladder was normal in size and contained inspissated bile. The kidneys were both enlarged. Nothing abnormal was found in the stomach, beyond bile-staining. Both lung bases were consolidated, but no pus was discovered. The pleurae covering the lung bases were loosely adherent to the diaphragm. There was no evidence of any endocardial lesion in the heart or of myocardial degeneration visible to the naked eye.

Histological and other Examinations

The wall of the gall-bladder was congested, and section revealed acute inflammation of the mucous membrane. Both kidneys showed cloudy swelling. There was round-celled infiltration about the portal canals, otherwise the liver was normal. The spleen was markedly congested.

A throat swab taken on February 26th was negative for diphtheria. Blood Wassermann reaction was negative. Culture from blood taken on the seventh day of the illness remained sterile after forty-eight hours' incubation. Culture from throat swab taken on the seventh day of the illness yielded a pneumococcus. White cell count

taken on the sixth day of the illness showed 11,700 per c.mm.—polymorphs, 86 per cent.; lymphocytes, 7 per cent.; hyaline, 3 per cent.; myelocytes, 4 per cent.; and abnormal leucocytes.

Discussion

The aetiology of this case presented great difficulties throughout, and, unfortunately, it will never be discovered whether or no it was a case of leptospiral infection. This man, who spent his days among chickens and the usual mud and filth associated with them, may quite easily have picked up such an infection. He also, strangely enough, had a sore place on his heel about a week before the onset of his illness, but not much importance was attached to this finding at the time or afterwards. A haemolytic streptococcal infection was a possible explanation, but the intense degree of jaundice and the sterile blood culture would hardly fit in with such a diagnosis. The sudden onset of the jaundice rather suggested the sudden flaring-up of a malignant condition in the liver as the result of an acute inflammatory condition inflicting an already damaged organ; post-mortem examination completely negated this theory. A liver abscess was considered as a possibility until a white cell count was done; this disclosed a leucocytosis, which was not even equal to the degree usually associated with pneumonia.

The feature which struck one above anything else was the intense bronze jaundice; within twenty-four hours of its onset it was of a greater severity than anything I have seen before. I can only conclude by saying that the patient was clearly being poisoned by some extremely toxic process, and that his resistance, as shown by the steady fall in the temperature, was not strong enough to combat it.

CEPHALIC TETANUS OCCURRING IN CIVIL PRACTICE

BY

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The infrequency of cephalic tetanus—even amongst war injuries—prompts me to record the following case, together with a few notes on the subject.

The patient, a man aged 71, was seen in the casualty department of the Metropolitan Hospital on February 4th, 1935. He gave a history of having fallen down and cut his right forehead while defaecating in an outside garden lavatory. A laceration was present over the right frontal region, and was 1 inch long and about a 1/4 inch deep. It was treated with a flavine and paraffin dressing. On February 6th the patient was seen in the out-patient department and appeared well. The next day he was again seen in the out-patient department, and still appeared well. On February 9th, five days after the accident, weakness of the right side of the face was noted; it was only slight, the patient being able to whistle. On February 11th, seven days after the injury, a bilateral facial weakness was noted, and the patient was admitted to hospital.

Closer examination during the evening of the same day showed, in addition to the bilateral facial weakness, a degree of stiffness in the muscles of the face: the eyes could not be closed, but if the lids were held down and the patient told to open his eyes, the upper lids moved up weakly. The tongue, when protruded, deviated to the right. The next day (eight days after the injury) a degree of trismus was noted. The jaws were not tightly clenched, but the mouth could not be opened forcibly, and the patient took up an attitude whereby he held his lips apart with his fingers. Nasal breathing was absent, there being a long-standing history of nasal obstruction, so that there was some degree of dyspnoea. The abdominal and the arm and leg reflexes were normal. The wound was suppurating. During the same evening the

trismus increased and the dyspnoea became greater. Attempts were made to introduce a Hewitt's airway between the gums, but, although the patient realized what was intended and tried to co-operate, we failed, because each time the tube was inserted between the lips and came into contact with the edentulous gums the spasms of the jaw muscles increased and the mouth closed. A Mason's gag failed to overcome the muscular spasms. Accordingly, a tracheotomy was performed under local anaesthesia, and afforded considerable relief to the patient, although it took several minutes before he realized there was no further need to hold his lips apart with his fingers. On February 13th, nine days after the injury, his condition was easier, there being no dyspnoea. Slight neck rigidity was noted. The next day the patient sank into coma and rapidly died.

Post-mortem examination, performed by Dr. H. C. Lucey, revealed no macroscopic abnormality in the body, apart from the local condition. The brain appeared normal. The wound on the forehead was suppurating—but to no great degree—and on examination was found to contain several small pieces of black, soft organic material. The wound was excised and cultured anaerobically on blood agar in a M'Intosh and Fildes jar. A mixed culture grew, which contained the typical spore-bearing tetanus bacilli.

The diagnosis of cephalic tetanus was made on February 12th, eight days after the injury, when slight trismus was first noted. Anti-tetanic serum in daily doses of 50,000 units was given intramuscularly. Heroin was also administered repeatedly, but all to no avail.

General Commentary

OTHER INSTANCES

Cases of cephalic tetanus, apart from the general form described in the literature, are rare. It is very interesting, however, to note that the first experimental observations on the pathology of tetanus were made on a case of cephalic tetanus. This work was done by two Italians, Rattone and Carle,¹ in 1884. They had a patient who developed cephalic tetanus after scratching an acne pustule in the region of his chin. A few hours after his death the matter from the diseased area, injected into rabbits, produced the disease which Carle and Rattone regarded as tetanus. During the great war, among the many cases of tetanus occurring in the military hospitals of North London² only two cases of cephalic tetanus are described, both with only slight head injuries. In one, following a small head wound, unilateral trismus and weakness of the right frontalis muscle occurred; in the other there was a right facial palsy and right unilateral trismus. In both cases evidence of spread of the toxin was present. In the former spasms of the neck and shoulder muscles, and in the latter general opisthotonos, occurred.

SOURCE OF INFECTION

The organism usually occurs in soil and dust which has become contaminated with the faeces of horses and cattle. It also occurs in human faeces. This was first demonstrated by Pizzini³ in 1898. Further work has shown that the occurrence of the organism varies in different localities. Tulloch⁴ in 1919 found the organism in the faeces of five civilian patients out of thirty-one examined, whereas Fildes⁵ in 1925 found the organism in only two out of 200 cases examined. The mode of infection in our case is doubtful, but it is interesting to note that the patient volunteered the statement that he "did not bother to wipe himself properly." Whether or not he infected himself by carrying the organism on his hand is pure surmise. It is probable that when he did cut his head after falling he put his fingers on to the wound to feel what had happened. Of what significance this may be I do not know, but even if it is only mere coincidence it is an interesting theoretical possibility of a method of infection. The alternative, of course, is that the lavatory floor was the source of infection.

SYMPTOMATOLOGY

Cephalic tetanus may present itself in more than one way. Following a head wound, dysphagia and dyspnoea may develop; extrinsic or intrinsic ocular palsies may occur; the hypoglossal nerve may be affected; facial palsy may occur with spasm of the paralysed muscles. A wound on one side of the face may be followed by a unilateral facial palsy or even a unilateral trismus. The particular physical sign which first shows is due to the spread of the toxin along the individual motor nerve. Probably the more distant symptoms are explained by a local diffusion of the toxin either through the synapses in the cord or bulb, or through the cerebro-spinal fluid. Toxin has been recovered from the cerebro-spinal fluid in experimental animals, but this has not been substantiated. The toxin is fixed more in the grey than in the white matter. The rapid spread from the seventh nerve nucleus to the twelfth and the fifth, in our case, suggests a local direct spread through the synapses rather than through the cerebro-spinal fluid. Rapid death, probably due to involvement of the vital centres in the bulb, prevented the general manifestations of tetanus. If the spread was through the cerebro-spinal fluid it is more likely that generalized tetanus would have occurred before death.

In war wounds, experience led to the early recognition of signs and symptoms of tetanus, but in civil practice, when the disease is not anticipated, early premonitory symptoms are likely to be overlooked. Among the premonitory symptoms may be mentioned sleeplessness, dreams, delirium, headaches, giddiness, yawning, an anxious facial expression, and difficulty in micturition due to sphincter spasms. In our case the diagnosis was not made till trismus was present, and by this time, of course, the disease was well established and the toxin had united with the nerve cells. It is probable that if cases of head tetanus were not rapidly fatal the local disease would become general and the condition and diagnosis obvious.

By the time the disease has manifested itself clinically the toxin (a) has combined with and damaged the nerve cells, (b) is circulating in the blood and lymph stream, and (c) is still being elaborated in the wound. The circulating toxin may be neutralized by antitoxin, and the elaboration of the toxin in the wound may be obviated by local excision and the use of hydrogen peroxide. Whether antitoxin given intrathecally can be expected to act on the damaged nerve cells is conjecture, but, following Sherrington's⁶ work on experimental animals, daily doses of about 20,000 units should be given into the subarachnoid space.

PROGNOSIS

The prognosis in cases of tetanus is interesting. It seems to depend on the length of the incubation period. If this is less than ten days the outlook is bad. In civil cases in Danish hospitals,⁷ 95 per cent. of patients died when the incubation period was under ten days, whereas in England only 35 per cent. died when the incubation period was over ten days.⁸ The injection of serum as a prophylactic measure lengthens the incubation period and so improves the prognosis. The outlook in cases of local tetanus is in the main good, but if the disease affects the head the proximity to the higher centres means a short incubation period and diminishes the chances of recovery. In our case the signs first showed on the fifth day, so that, whatever the treatment, the probability was that the patient would have died, and we were not surprised when he died.

In a busy hospital in the metropolis one sees many lacerations of varying degree, but although one is taught to give anti-tetanic serum in all doubtful cases the lesson is soon forgotten. Perhaps this is because the disease is

so seldom seen, and further because cases which would appear to have the propensities for tetanus seldom behave as we would expect. Nevertheless, antitoxin should be given as a prophylactic in all doubtful cases, and it is quite possible that if our case had been so treated a fatality would have been avoided.

My thanks are due to Mr. Acton Davis, under whose care the patient was admitted, for permission to publish this case, and to Dr. Lucey for the post-mortem and pathological findings.

REFERENCES

- ¹ Carle and Rattone: *Giorn. Acc. Med. Torino*, 1884, 3 s., xxxii, 174.
- ² *Medical History of the War*, i, 162.
- ³ Pizzini: *Zentralbl. f. Bakt.*, 1898, Abt. 1, xxiv, 890.
- ⁴ Tulloch: *Journ. of Hyg.*, 1919, xviii, 103.
- ⁵ Fildes: *Brit. Journ. Exper. Path.*, 1925, vi, 62.
- ⁶ Sherrington: *Lancet*, 1917, ii, 964.
- ⁷ Permin: *Mitt. Grenzgeb. Med. Chir.*, 1914, xxvii, 1.
- ⁸ Choyce: *System of Surgery*, i, 858, 863.

Clinical Memoranda

Defective Ossification

The photograph is that of a stunted Indian, with multiple, badly united fractures, the worst malformations being at both ankles: the right foot is completely upturned inwards, lying in front of his loincloth, and the left foot rests on a ledge of his cart.

The case is one of osteogenesis imperfecta, also known as fragilitas ossium. It was formerly called "foetal rickets," a term covering various conditions, which included achondroplasia and cretinism. There are two kinds—pre-natal and post-natal, osteogenesis imperfecta congenita and tarda. In the former the skull is defectively ossified: it is a mosaic of thin broken bones, and there are fractures of the limb bones, which are short and thick. In the post-natal cases the long bones are chiefly affected. Fractures occur more frequently during infancy than in after life, and on slight provocation, without violence or pain, because of the small amount of con-

tusion in the soft tissues. Patients with this kind of deformity are able to lead a fairly normal life, though their bodies are stunted.

Osteogenesis imperfecta can be hereditary. A curiously consistent feature is its association with grey-blue eyes. It is because of lack of fibrous tissue and of lime salts in the connective tissue that the sclerotics become transparent and transmit the colour of the choroidal pigment, resulting in blue eyes. There is, moreover, a lack of fibrosity throughout the general framework of the body. In the bones the lamellae arranged in circular plates round the Haversian canals are lightly bound together by the diminished amount of fibrous tissue, and this, combined with loss of phosphate and carbonate of lime, renders the bones brittle and without spring—bricks without straw. There is no evidence of syphilitic or thyroid gland involvement. Some observers, mostly German, have thought that the posterior lobe of the pituitary gland has an influence on the metabolism of lime salts, and that some

calcification takes place directly in the capsules of cartilage cells—metaplasia instead of neoplasia. Metaplasia is the direct transformation of cartilage into bone, and is probably compensatory and not primary in character. The osteoblasts, it is generally agreed, are abnormal, both in structure and in function. Endochondral bone formation being especially restricted, this results in the shortening of stature. This shortening is most marked in intra-uterine cases: in these the fractures are often thought to be due to the bones being warped round by the umbilical cord. But if the warp were strong enough to compress the limbs sufficiently to cause fractures it would mean compression of the cord to the extent of stopping foetal life.

Cases have been reported: of a woman 26 years old, who had thirty-nine fractures; of a young man of 16, who had forty-five fractures; and many others of similar figures. In these days of compensation for accidents it is conceivable that this disease may not be without medico-legal significance. The pathological chemistry may be summed up as progressive loss of calcium and phosphorus. Osteogenesis imperfecta was known to Ambroise Paré, who, in his monumental Latin work, cited instances of it and gave woodcut illustrations.

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Rupture of Bronchiectatic Abscess: An Unusual Case

The following case of rupture of a bronchiectatic abscess seems to be of sufficient rarity to be worth recording.

The patient, a male aged 36, was admitted to Preston Hall Sanatorium suffering from extensive bilateral pulmonary tuberculosis, with involvement of the spine, the sternum, and the right knee-joint. He had suffered from tuberculosis for fifteen years, and had been bed-ridden for the last three years. His condition was very poor, the breath had a putrid, typically bronchiectatic odour, and he brought up 8 to 10 oz. daily of foul, purulent sputum. There was well-marked clubbing of the fingers and toes. Towards the end of February he developed severe abdominal pain with persistent vomiting of a foul, greenish-looking material. There was definite tenderness over the epigastrium, but no rigidity. Vomiting was, in fact, the main feature of the abdominal condition, and the patient rapidly succumbed.

In this sanatorium routine post-mortem lipiodol bronchograms are taken in each case, and in this patient the presence of gross bronchiectasis was confirmed, but, in addition, it was noted that there appeared to be a quantity of lipiodol in the abdominal cavity. A necropsy was thereupon performed, and a large pulmonary abscess was found at the base of the right lower lobe, having an uneven, necrotic wall, which had perforated through a narrow pin-hole orifice in the diaphragm. A quantity of free pus was found in the abdominal cavity, but no point of entry into any part of the bowel could be traced.

The occurrence of a subphrenic abscess following an empyema, and, similarly, the occurrence of empyema following the rupture of a subphrenic abscess through the diaphragm, are events well recognized in medical and surgical practice. The actual rupture of a bronchiectatic abscess through the diaphragm appears, however, to be something of a rarity, and a preliminary search through the literature on the subject has failed to reveal any case at all on record.

We are indebted to Dr. J. B. McDougall, the medical director of the sanatorium, for permission to publish this case.

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