shoulders and the bed. The abdominal muscles were rigid, but moved with respiration. Spontaneous paroxysms of opisthotonos were occurring about every half-minute; they lasted about 5 seconds, and were accompanied by clenching of the jaws and spasm of the thoracic and abdominal muscles. The limbs were unaffected. These paroxysms had first appeared on the night of Dec. 3. He was quite rational, but his face bore an appearance of intense anxiety. The only findings of note in the C.N.S. were loss of plantar reflexes (a usual finding in the sepy in health and disease) and a complete absence of abdominal reflexes. The pupils were moderately small, and reacted normally to light.

The patient had received 15,000 units of A.T.S. (therapeutic) in his unit on the previous day, and 15 c.cm. intravenously and 5 c.cm. subcutaneously with adrenaline and calcium lactate in a reception station earlier on the day of admission. (There he had also undergone lumbar puncture, the fluid revealing no abnormality of pressure or constituent.) In this hospital he was given 40,000 units (therapeutic) intramuscularly b.d., and was kept stuporous with 4 drachms of paraldehyde by mouth, the paraldehyde being repeated in 2-drachm doses when necessary, day and night.

On the morning of Dec. 5 his temperature was 99.4°, pulse 90, and respirations 20, and his condition was unchanged. On the 6th the spasms were of 5 seconds' duration as before, but occurred only every 2 minutes, and their severity was much less marked. The neck rigidity and trismus were unchanged, and the morning temperature was 100°. Treatment was repeated. Early on the morning of the 7th he suddenly died. The temperature had just been taken—99°.

Necropsy.—The examination was made by Capt. A. E. Wilkinson, R.A.M.C., about 6 hours after death. There were two small completely healed wounds on the left hand; rigor was noticeably more pronounced in this hand than in the right, but was absent in both arms. The right ventricle and auricle were engorged with fluid blood, while the left heart was comparatively empty. Numerous petechial haemorrhages were present in the epicardium over both auricles and ventricles. The posterior cusp of the pulmonary valve was ruptured at its free border. The remaining valves were normal, the coronaries were patent, and the cardiac muscle appeared excellent. The lungs were pale and emphysematous, with no subpleural haemorrhages; the liver was very dark and congested; the kidneys were congested; and the brain was normal. There was a pronounced smell of paraldehyde, most noticeable in the lungs, liver, and brain. The remaining viscera appeared to be normal. The tissue from the healed wounds was not cultured for *Cl. tetani*.

Differential Diagnosis.—The diagnosis was not confirmed bacteriologically, hence the doubt expressed in the caption of this article. The other possibilities were rabies and strychnine poisoning. As regards rabies, points of similarity were the spasms and rising temperature, but the respiratory rate was only slightly increased and there was no manifestation of air hunger. The larynx was involved in the spasms, but between them speech was normal. Similarly, fluids were taken freely by mouth between the spasms. There was no hydrophobia or hypersalivation. The trismus, risus sardonicus, and constant neck and back rigidity were distinguishing features, and the incubation period of 14 days agrees with a diagnosis of tetanus arising from such a wound in the hand. Strychnine poisoning is excluded by the duration of illness (5 days) and the persisting trismus and neck and back rigidity. Poisoning by brucine has been suggested, but since this drug is similar in action to strychnine but is only slightly toxic (Lynch and Pryce, 1939), it also is excluded. Furthermore, I have been unable to find any evidence of the use of poison on arrow tips or otherwise by the natives of this district. There was no history of an arrow wound.

Comment

No other case of tetanus has been reported in this area during the eight months of British occupation. The country is dry, sandy, hot, and arid, and tetanus is rare in such soils (Boyd, 1939). However, the Italians left the site of occupation in a filthy condition, and there had been much recent contamination of the soil by horse, mule, and donkey manure. No A.T.S. had been given at the time of the injuries, the patient considering them too trivial to report. At the post-mortem examination the pronounced rigidity of the left hand was a noticeable feature. The atmospheric temperature was similar to that of a fine English spring day, and could not have appreciably accelerated the onset of rigor mortis. There was no factor in the environment which might have allowed one part to cool more quickly than another.

The man was not seen by me just before he died, so the severity of the spasms then occurring cannot be stated accurately. The nursing sepy attending him reported that they seemed less severe than on the previous day, and that he had his back to the patient at the moment of death. The rupture of the pulmonary valve cusp was mute evidence of the immediate cause of death. It is extremely rare for a pulmonary valve cusp to rupture as a result of violent physical exertion, and there was no congenital defect in the cusp to weaken it. Death must have occurred in diastole. One can imagine that the severe respiratory effort with closed glottis forced blood out of the alveolar walls, and that the tension within the pulmonary artery was further increased by the general intrathoracic pressure's acting on its outer surface until a cusp ruptured. The emphysema in one so young is confirmatory evidence of the high pressure in the thorax at the time of death. Despite the improvement after his admission to hospital and until the moment of death, the ultimately fatal issue agrees with Cole's gloomy prognosis (Cole, 1941) in cases in which spasm begins within 48 hours of the onset of trismus.

It is admitted that tetanus usually arises from unhealed wounds or disturbed scars, but it does occur where no obvious wound can be found (the so-called "idiopathic" cases). The mere fact that the wounds in the left hand were healed does not invalidate the diagnosis.

If this was undoubtedly a case of tetanus the two protective inoculations had not even sensitized the reticulo-endothelial system, and the stimulus of toxin from the wound produced no rise of antitoxin to "the highly protective level," which has been suggested as 0.01 international unit per c.cm. (*British Medical Journal*, 1940, 2, 910). It would be similar to a persistently Schick-fast reactor. Marvell and Parish (1940) report that 26 out of 27 subjects reacted satisfactorily to the third dose of tetanus toxoid, and that the remaining titre rose from 0.05 to 0.05–0.1 unit. This, in conjunction with Perry's report, is an indication of the rarity of the case recorded above.

The occurrence of such a case vindicates the present practice of giving antitoxin to all wounded, irrespective of previous inoculation with toxoid.

I am greatly indebted to Col. J. S. K. Boyd for his criticism of this paper and personal communications upon it. I wish to thank Capt. A. E. Wilkinson, R.A.M.C., for performing and reporting upon the necropsy; Capt. G. J. H. Maud, I.M.S., Lieut.-Col. E. Oastler, R.A.M.C., and Major H. Fidler, R.A.M.C., for their criticism and advice; and the D.M.S., Middle East Forces, for his permission to publish.

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Medical Memoranda

A Case of Acute Aseptic Meningitis

Acute aseptic meningitis was first described as a separate disease by Wallgren (1925). Since then numerous cases have been reported, though the cause remains obscure. It is distinct aetiologically from the virus form of lymphocytic choriomeningitis. It is often referred to as benign aseptic meningitis. The term "benign" is accurate as regards prognosis, for all cases recover; but if applied to symptoms it may be misleading, as in the present case, in which the illness was so severe that I did not at first think it could possibly be what I had always known as benign aseptic meningitis.

CASE HISTORY

I saw the patient, a girl aged 5, at her home on June 17, 1942. Two days before, she had returned from school feeling sick and complaining of sore throat: three days previously she had been perfectly fit. She had had a convulsion on the morning of the 17th. She had moderate neck rigidity and a doubtful positive Kernig's sign. I admitted her as a probable case of meningococcal meningitis. On admission, lumbar puncture yielded a clear fluid under greatly increased pressure. The pupils were widely dilated and fixed: no reaction to light. Knee- and ankle-jerks were absent, as were all arm-jerks; plantars were extensor. The child resented all examination. Shortly after the lumbar puncture she had twitchings of the right side of face and body, which later spread to the left

and became violent. These violent choreiform movements continued all afternoon and most of the night, and the child became desperately ill. On the assumption that this was a case of poliomyelitis, though a very unusual one, she was given 20 c.cm. of human serum intramuscularly. The following morning the movements were less pronounced, but the child was semi-comatose, with marked head retraction; her limbs were all held flexed, and it was difficult to extend them. An obvious left facial paralysis and slight left foot-drop were present, and there was some difficulty in swallowing, but the palate moved freely. She was given 20 c.cm. of poliomyelitis horse serum intravenously and 20 c.cm. intramuscularly. The facial paralysis had largely disappeared within a few hours, and by June 19 was scarcely appreciable. The foot-drop had also almost gone and there was no difficulty in swallowing. The child remained semi-conscious, with marked head retraction and fixed flexion of all limbs. The most likely diagnosis at this stage seemed to be tuberculous meningitis. On the 20th, however, there was a distinct improvement in the child's condition, and by June 22 she appeared almost normal. She was discharged on July 8, and has been seen several times since, quite fit. The following investigations were carried out:

Lumbar Puncture	Appearance of Fluid	No. and Type of Cells	Protein (mg./100 c.cm.)	Chlorides (mg./100 c.cm.)	Culture
17/6/42	Clear	380 { Lymphocytes, 55% Polymorphs, 40% Large hyalines, 5%	40	640*	Sterile
18/6/42	"	320 { Lymphocytes, 96%	20	720	"
20/6/42	"	410 { Lymphocytes, 94%	20	700	"
2/7/42	"	90 { Lymphocytes, 80% Polymorphs, 20%	20	730	"

* This figure was reported as "not guaranteed" the following day: it obviously supported a diagnosis of tuberculous meningitis.

Radiographs showed no skull abnormality and the lungs were clear. Animal inoculation revealed no signs of virus infection from inoculation with patient's serum or cerebrospinal fluid (June 20). Complement-fixation and neutralization tests were both negative on the patient's serum on August 17.

COMMENT

Points of interest are: (1) the extreme severity of the symptoms, the term "benign" being rather misleading; (2) the absence of a preceding febrile illness (in the virus form, a febrile illness of one or two weeks' duration is frequently, though not always, recorded); and (3) the very transient paralyses. The last two points are well discussed by Baird and Rivers (1938). Diagnosis from tuberculous meningitis could be made only as signs of recovery became apparent. The wild choreiform movements followed by fixed flexion of the limbs were unlike poliomyelitis, though the absence of tendon-jerks was in its favour on the first day; failure to develop any flaccidity or marked paralysis in a case in which the central nervous system was so acutely involved, and a high cell count a fortnight after the onset, suffice to exclude it.

I am greatly indebted to Dr. MacCallum of the Wellcome Research Foundation, who carried out the animal inoculation and serological tests; to Dr. May of the Emergency Pathological Service, who reported on the cerebrospinal fluid; to Dr. Sleight Johnson, who saw the case in consultation; and to the medical officer of health, Southend-on-Sea, for permission to publish.

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Thrombophlebitis of the Cavernous Sinus: Recovery

The following case of cavernous sinus thrombosis, which recovered after a combined treatment with blood transfusions and sulphathiazole, may be considered worthy of record.

CASE HISTORY

On May 25, 1942, I was consulted by a vigorous, healthy married woman, aged 56, about a nasal furuncle situated inside the left alar cartilage. It was a typical staphylococcal lesion, not of large size, and there was the usual oedematous swelling of the adjacent cheek, with intense pain and slight pyrexia. I treated her from the outset with 1 tablet (0.5 g.) of sulphathiazole (M & B 760) four-hourly, and advised glycerin packs for the interior of the nose. Next day I was sent for and found a good deal more swelling of the cheek, with oedema of the lower eyelid and temperature 101°. Pus was being discharged from the furuncle. She also complained of severe supra-orbital headache, but it was not till the following day that I suspected the possibility of cavernous sinus thrombosis. On May 28 there was no longer any doubt: the left eye was proptosed, both lids being greatly swollen, and chemosis was present; the right eye was also involved, but not to the same extent. Frontal headache was very severe, and oedema extended over the supra-orbital region, more pronounced on the left side. Detailed examination of

the eyes was difficult, but the patient volunteered the information that she "saw double on looking to the left," and there appeared to be definite paresis of the left external rectus—from involvement of the sixth nerve, which runs along the inner wall of the cavernous sinus. There was some rigidity of the neck muscles, and temperature had risen to 103°. The patient was seen with me by Mr. C. Gill-Carey and removed to hospital.

A blood count showed R.B.C. 4,740,000 and W.B.C. 10,200 per c.mm. A blood culture was also taken that night (from which, after five days' incubation, a *Staphylococcus aureus* was grown of the same type as that found in the nasal lesion). Immediate treatment given was a transfusion with 400 c.cm. of whole stored blood (freshly taken that day), and intramuscular injection of sulphathiazole was started, alternating with oral administration of the drug—4 g. of each within the first 24 hours. The local condition was rather worse next day, proptosis and chemosis being more noticeable; but the temperature fell gradually to normal within two days, and the general condition was obviously better. The same dosage of sulphathiazole was continued for three days, when it was reduced to 6 g., but next day the temperature began to rise, and 8 g. was again given. On June 2 there was a sharp rise of temperature to 102°, so a second transfusion of stored blood was given, once more with good effect, the temperature falling to normal next day and remaining normal for four days. There was a great improvement in the patient's condition, general and local, except for the appearance on June 6 of a papular rash on the neck, arms, legs, and abdomen. As she was complaining of a good deal of nausea the sulphathiazole was reduced to 3 g. daily on June 6 and 7.

On the 7th, however, she felt very ill, with sudden rise of temperature to 102°. Again 400 c.cm. of whole blood was given, and sulphathiazole 6 g. daily. The dramatic improvement, however, was not repeated this time, so on the 9th 400 c.cm. of blood from a donor was administered. A blood count taken at the time showed W.B.C. 7,800 per c.mm., with no sign of agranulocytosis, so the dosage of sulphathiazole was stepped up to 8 g. daily. For the next four days the patient remained very ill, the rash extending into large confluent areas, and headache being severe; but the local condition of the eyes showed marked improvement. On June 13, as the rash was so distressing, and the patient had by then had 95 g. of sulphathiazole, it was decided to discontinue it. Next day the temperature remained normal for the first time for a week; the rash gradually faded and the swollen eyelids returned to normal fairly rapidly. Headache, though still severe at times, became spasmodic in character, and convalescence began.

DISCUSSION

Thrombophlebitis of the cavernous sinus is a grave complication of suppurative lesions of the face; the mortality has been notoriously high, and all the textbooks which I consulted record that it is "almost invariably fatal." In the *Lancet* for May 2, 1931, W. G. Scott-Brown, in a comprehensive review of six fatal cases collected from the records of St. Bartholomew's Hospital, alludes to it as "an invariably fatal complication of minor facial sepsis, unless the sinus can be drained." He goes on to describe the methods of approach to the sinus: (1) the Gasserian-ganglion route; (2) the orbital route; (3) the nasal route, through the sphenoidal air sinus. But each of these represents a surgical manœuvre fraught with the gravest peril of injury to important structures and of spreading the disease to the meninges. Bischer reported in 1893 a successful case of drainage through the petrous portion of the temporal bone (Scott-Brown), but his success has apparently never been repeated.

With regard to surgical treatment of the local lesion, it is generally recognized that free incision is dangerous and apt to lead to rapid dissemination of the infection. It is therefore gratifying to feel that methods of treatment exist to-day which give a good chance of recovery. I cannot say whether the repeated blood transfusions or the sulphathiazole therapy played the greater part in curing the condition, but I feel sure that the combination of the two was the important thing. A study of the temperature chart did make it appear that the fresh antibodies provided by transfusion caused dramatic improvement on the first two occasions, and, though the temperature persisted after the third and fourth transfusions, I am inclined to think that the patient had by then had as much sulphathiazole as she could deal with. The rash was very severe, and it seems likely that the drug itself may have been responsible for keeping the temperature up at this stage, the sudden cessation of fever on discontinuing it pointing strongly in that direction.

The case has, to my mind, proved conclusively the great value of sulphathiazole in the treatment of serious staphylococcal infections, and bears out the claim of the makers that its toxicity is only half that of sulphapyridine. Before the appearance of the toxic rash my patient had had about 60 g. of the drug, and she was able to take in all 95 g. in 18 days, without serious detriment.

I am indebted to Dr. Frank Knott, pathologist to Guy's Hospital, and to Dr. J. Verdon, R.M.O. at Surbiton Hospital, for their invaluable help in treating this interesting case.

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