TABLE V.—Toxic Symptoms

Case	Day of Treatment	Dosage Given (g.)	Symptoms	
			Duration	Nature
51	12th	224	3 days	Macular red rash on trunk; headache and tachycardia
34	9th	157.5	2 "	Slight fever; tachycardia; headache; nausea
23	8th	140	2 "	" " " "
29	8th	140	2 "	" " " "
46	8th	140	4 "	Maculo-papular rash; headache; nausea
48	7th	122.5	3 "	Generalized erythema; headache; slight fever
47	6th	105	3 "	" " " "
19	2 days after 4th	80.5	4 "	Maculo-papular red rash
6		4	2 "	Slight fever; headache; nausea

Conclusions**Acute Cases**

In all, 26 acute cases of bacillary dysentery were treated with sulphaguanidine. 19 cases were completely cured in from 14 to 21 days; 6 cases relieved, 1 of which became chronic; and 1 case hardly affected. The chronic case was subsequently cured by further treatment with sulphaguanidine.

Sulphaguanidine, like other sulphonamides, would appear to be bacteriostatic, the dysentery bacilli dying only slowly. Hence an efficient concentration of the drug must be maintained for some time after the active infective process has been inhibited. Such a stage is evidently reached when the stools are almost normal, and the failure to continue the sulphaguanidine makes a relapse likely. Cases 1, 2, 3, 4, and 5 were the first treated, and sulphaguanidine was stopped when the faecal stools had a consistency and showed no blood. The fact that these cases relapsed after a few days would appear to stress the importance of continuing treatment for 2 to 3 days after the stools are normal and while the final stages of healing are taking place. Further evidence in support of this is given by Cases 6, 23, 25, 26, and 51 of the chronic series, which received up to 80 g. in the acute phase but subsequently became chronic. Treatment had not been continued until the stools were normal.

The early acute infections showed a very quick response to treatment, and the longer the interval between the onset of the disease and the beginning of treatment, the longer was treatment necessary.

The more frequent and looser the stools the less the concentration of sulphaguanidine in the bowel (*Trop. Dis. Bull.*, 1942, 39, 34), and in the initial stages of treatment adjuvants such as tinct. opii, bismuth and chalk, and kaolin were used to lessen the frequency and increase the consistency. In the one case which failed to respond the stools remained fluid and frequent in spite of all treatment.

The feeling of well-being as toxæmia is overcome and the early fall in the frequency of the stools greatly reduced the exhaustion and dehydration of the patient. Fluid can be made up adequately by mouth, and the patient's early desire for food as his condition quickly improves allows his strength to be built up while the infective process is overcome.

Chronic Cases

A series of 51 cases of chronic bacillary dysentery were treated with sulphaguanidine. All showed an early and marked response to treatment. In the first 20 cases, as in the acute series, the nature of the stools governed the dosage given, and the average total dose was about 90 g. over 6 days. Only 11 (55%) cases were completely healed, without relapse. In the subsequent 31 cases a minimum dosage of 17.5 g. for 8 days was given, and the advent of normal stools was not taken as an indication for reducing or stopping treatment. The average total dose was 160 g. over 10 days. 24 (77%) cases showed complete healing, and 5 complete healing but for granular patches in the rectum; 2 responded but relapsed after treatment.

Stool examination and the general examination of the patient are inadequate as a means of gauging the state of the lesions in the bowel, and sigmoidoscopy is essential. It would seem that passage of normal stools and disappearance of symptoms in chronic cases indicate only that the active infective process has been inhibited and not that healing of the mucosa has taken place. If sulphaguanidine is not continued the infective

process may again become active, delaying healing for some weeks. A full daily dose of 17.5 g. for at least 8 days, and longer in cases showing other than moderate severity, would appear to afford adequate protection against this delay in healing. Nine cases of the chronic series showed mild toxic symptoms from the sulphaguanidine.

Summary

In all, 77 cases of bacillary dysentery were treated—26 acute, 51 chronic. Of the 26 acute cases 16 were Shiga infections, 6 Flexner, and 4 unidentified. Of the 51 chronic cases 20 were Shiga and 14 Flexner infections; in 17 the organism was undetermined.

Patients were put to bed, rested and warmed. The frequency and character of stools were noted, and specimens plated and examined microscopically. Diet was limited to fluid in the first 24 hours. A large fluid intake by mouth was encouraged in the early stages to replace fluid loss. No intravenous fluid was necessary.

Adjuvant treatment was given, cases of Shiga infection receiving 30,000 units of antiserum.

Acute cases received an initial dose of 7 g. of sulphaguanidine, followed by 3.5 g. at 4-hourly intervals. In very severe cases 3.5 g. was given 2 hours after the initial dose, followed in 2 hours by a further 3.5 g. In the main, sulphaguanidine was given until 2 days after the stools had become normal.

Chronic cases received 17.5 g. of sulphaguanidine daily, in 5 doses, for a minimum of 8 days, the length of treatment being increased in the severer cases. A preliminary period of treatment with colonic wash-outs while on a low-residue diet was carried out in 19 cases.

In chronic cases sulphaguanidine must be given until healing of the mucosa has taken place: to gauge this, sigmoidoscopy is essential.

Complete cure was obtained in 19 (73%) acute cases, in 11 (55%) chronic cases receiving 90 g. over 6 days, and in 24 (77%) chronic cases receiving an average dose of 160 g. over 10 days.

Toxic symptoms occurred in only 9 of the 51 chronic cases; there were none in the acute series.

Illustrative cases, showing the scheme of treatment, are recorded.

PROBABLE TETANUS DESPITE INOCULATION WITH TOXOID

BY

R. J. MCGILL, M.B.

Temp. Major, Indian Medical Service; O.C. Medical Division of an Indian General Hospital, M.E.F.

Preliminary reports upon battle casualties previously protected with tetanus toxoid seem to vindicate both its use and the administration of two doses at a six-weekly interval. In the B.E.F. in France and Flanders no tetanus occurred in the wounded who had been so inoculated (Perry, 1940), and the third dose after a six-months interval, which is now the standard method advocated by Boyd (1938) and in practice in the British and Indian Armies, should further raise the antitoxin level. The following is a probable case in a subject who had been protected by two doses at a six-weekly interval.

Case History

A sepoy aged 20 received 1 c.cm. of tetanus toxoid subcutaneously on April 25, 1941, and again on June 7 (as recorded in his pay-book). He was admitted to this hospital on Dec. 4 with a history of stiffness in the dorsal thoracic muscles and the muscles of the back of the neck for two days. Slight stiffness of the jaws and dysphagia appeared first on the day of admission. His only injuries were a wound on the left thenar eminence and one on the palmar surface of the left middle finger, each about 3/4 in. long, incised, superficial, and at the time of admission quite healed, with only the edges of the most superficial horny layer very slightly gaping. He had received them on Nov. 18 while serving on the Red Sea Littoral.

Moderate risus sardonicus and trismus were present, the jaws opening 1/2 in. There was marked stiffness of the back and neck. One could just pass the flat of the hand between the

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