TETANUS

A REVIEW OF ONE HUNDRED AND THIRTY CASES SEEN AND TREATED IN THE SURGICAL PROFESSORIAL UNIT, GENERAL HOSPITAL, SINGAPORE

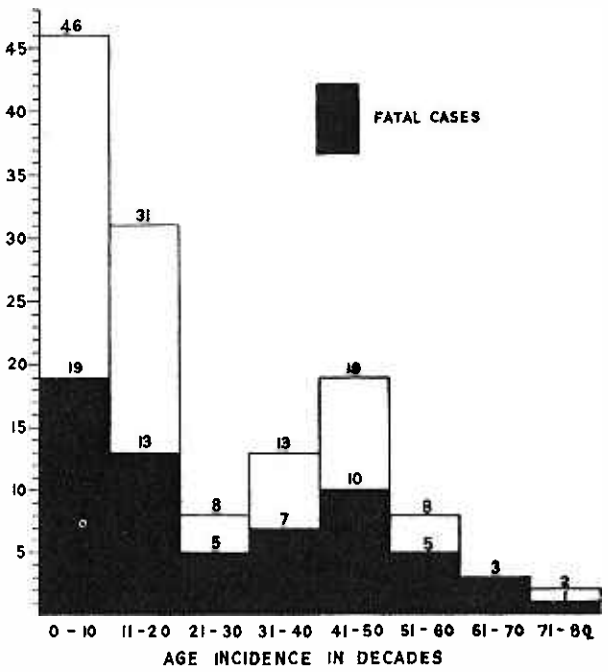
By G. S. Yeoh, M.A., M.B., B.CHIR., F.R.C.S. (ENG.), F.R.A.C.S., F.A.C.S.
(Department of Surgery. University of Malaya in Singapore).

Every medical man is familiar with the signs and symptoms of tetanus. Hippocrates described the symptomatology over two thousand years ago and since then many authorities have written on tetanus in the medical literature. The infective nature of tetanus remained unsuspected until shown by Carle and Rattone[1] in 1884. The spores of the bacilli were detected by Rosenbach[2] in 1887. Two years later Kitasato[3] cultured the bacillus and a year later in 1890, he and Behring[4] prepared the antitoxin. B. tetani is a gram-positive anaerobe and is a spore-bearing organism. Bacteriologically, it has been shown that there may be different strains of this bacillus as the exotoxin produced can be either haemolytic or neurotoxic. Tulloch[5] showed in 1918 that 5% of the stools of urban civilians contained B. tetani and the stools of farmers would show a higher proportion. The bacillus thrives in highly fertilised soils.

The wound most susceptible to an invasion of B. tetani is one that is deep and lacerated, resulting in the introduction of dirt and the production of necrotic tissues. An associated infection appears to increase the virulence of the bacilli. The mode of spread of the exotoxin is still in doubt as it has not been shown how and by what route or routes the exotoxin travels to the motor cells of the spinal cord. All authorities maintain that once the exotoxin reaches the motor cells, it becomes "fixed" to them and cannot be neutralised by the introduction of antitoxin. Although a deep and lacerated wound does provide an ideal nidus for the bacilli, some of the cases seen and treated in the Surgical Professorial Unit did not show any evidence of injury. This group made up seventeen of the one hundred and thirty cases seen in twelve and a half years. It is generally agreed that the mortality rate is inversely proportional to the incubation period, i.e., the shorter the incubation period, the higher the mortality. In our series, seventy-eight of the cases had an incubation period of ten days or less, with an average of 5.3 days. The mortality among these was forty-two cases, i.e., 53.8%. The over-all mortality of all the cases was sixty-three, i.e., 48.4%.

The average incubation period for the one hundred and thirteen cases with clinical evidence of injury was 6.9 days. The average stay in the wards for the fatalities was 1.6 days. The average stay in the hospital for the survivors was 19.5 days. Fig. One shows the age incidence of all cases and the number of deaths in each age group.

FIG ONE



The cases seen were from January, 1947 to June, 1959. There were ninety-one males to thirty-nine females, the youngest was a male infant aged eleven days with an infected umbilicus and the oldest was a female aged seventy-two. The sites of injury were found in most parts of the body and are shown in Table One.

In seventeen cases, there was no history of injury whatever, and this was confirmed clinically by not finding any trauma or focus of sepsis.

The commonest instrument for inflicting an injury in this series was the inevitable rusty nail as this accounted for thirty-two cases. Wood splinters were responsible for sixteen of the cases. Sharp instruments, e.g., knives,

Table One: Site of injury and the number of cases:

Feet	58 (69%in the soles)
Legs	27
Hands	12
Head	6
Back	1
Umbilicus	1
Multiple lacerations due to accidents	4
Second degree burns	1
Abortions	3

glass fragments and rusty pipes caused deep lacerated cuts in twenty-six cases. Thirty cases sustained injuries from falls, and of these, two suffered from fractures. One of them had a compound fracture of the right femur and came into the ward a week after the fall and died four hours after admission with a full blown attack of tetanus. The other was a Colles' fracture of the right wrist and made an uneventful recovery. The rest had only minor abrasions and grazes.

The treatment of tetanus cases in the Surgical Professorial Unit is not based on a concerted attack by a team of physicians, surgeons and anaesthetists. The patient is admitted into a general ward among other surgical cases and is, therefore, subjected to the usual extraneous noises and stimuli to be found in a busy ward. No provision is made to treat the patient in an isolated, quiet and darkened room. The patient is sedated with an intramuscular injection of paraldehyde, the dosage of which varies with the age of the patients and is between 2 and 6 ml. This sedative is given every six hours, if necessary. If there is an obvious wound, this is surgically treated in the operating room by wound toilet. Penicillin is given in adequate doses to control the associated secondary infection and to prevent any respiratory infection. Altemeir[6] has pointed out in 1946 that penicillin has no apparent effect on B. tetani. Antitoxin is given to the patient intravenously on arrival in the ward and here the dosage also varies with the age of the patients and the severity of the attack. Although it has been shown by Spaeth[7] and Cooke[8] in 1941 and 1943 respectively, that a satisfactory level could be maintained in the blood for five to six weeks or longer by a single intravenous injection of 30,000 to 100,000 units, our dosage is inclined to be too generous as up to 500,000 units have been given to very serious cases.

The nutrition of the patient is constantly borne in mind and feeds are given through an indwelling Ryle's tube. The dangers of aspiration-pneumonia have been indicated by Creech et al[9] in 1957 and they advocate doing a gastrostomy for the purpose of feeding and with the hope of preventing any aspiration into the lungs. Tracheostomy was performed in only two cases in our series, but this proved to be of no avail as they were admitted in extremis and died within a few hours of hospitalisation. Another consideration is the usual employment of an indwelling catheter to keep the urinary bladder empty as a distended bladder is too well-known as a form of stimulation to warrant further comment.

The treatment for the one hundred and thirty cases is divided into three phases or periods and it is of interest to compare the results of these three phases. During the first period, from January, 1947, to December, 1954, the standard treatment was based on the use of antitoxin, penicillin and paraldehyde. In the second and the third periods, the standard treatment of the first period was supplemented by other drugs. The second period was from January, 1955, to June, 1957, and to the standard treatment was added the following drugs: Largactil (Chlorpromazine Hydrochloride), Pethidine and Phenergan (Promethazine Hydrochloride). The purpose of these was to depress the reticular alerting system and thus to further sedate the patients. The dosage used varied with the age of the patients. The three drugs were added to a 5% dextrose solution and this was set up in the form of an intravenous drip. Each litre of dextrose solution might contain Largactil 25 mgm., Pethidine 25 mgm., and Phenergan 25 mgm., to Largactil 50 mgm., Pethidine 100 mgm. and Phenergan 50 mgm. The former amount was for patients under the age of fifteen and the latter for those above fifteen. The third period was from July, 1957, to June, 1959, and in this period, the supplementary treatment was the addition of a 6% solution of Polyvinylpyrrolidone in physiological saline ("Periston-N"). This solution is made up in 100 ml bottles and is given intravenously. The amount given varied with the severity of the attack and up to 300 ml have been given. According to the manufacturers of Periston-N, polyvinylpyrro-

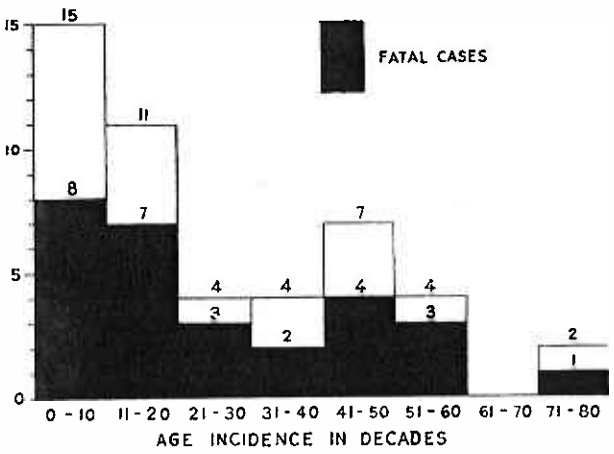
lidone has a mean molecular weight of 12,600 and has the property of adsorbing any toxin in the blood stream. In view of the small molecular weight, polyvinylpyrrolidone is easily filtered through the glomeruli and thus the adsorbed exotoxin is also got rid of. Table Two shows the results obtained in the three periods.

Table Two: The three periods, the number of cases and the mortality rate:

Period	Recovered	Died	Mortality Rate
January, 1947 to December, 1954	37	43	53.8%
January, 1955 to June, 1957	14	14	50%
July, 1957 to June, 1959	17	5	22.7%

In the first period, forty-seven cases were admitted with incubation periods of ten days or less and of these, twenty-eight died, i.e., 59.7%. Fig. Two shows the age incidence and mortality rate of these forty-seven cases.

FIG TWO



In the second phase, nineteen cases had incubation periods of ten days or less and eleven died, i.e., 57.9%. Fig. Three indicates the age incidence and the mortality in relation to each age group.

In the third period, twelve patients were seen with incubation periods of ten days or less and three died, i.e. 25%. Fig. Four shows the age incidence and mortality of these cases.

The remaining cases of the three periods with incubation periods of eleven days or more are shown in Table Three. Table Four shows the seventeen cases with no history of injury and the results obtained in the three phases.

FIG THREE

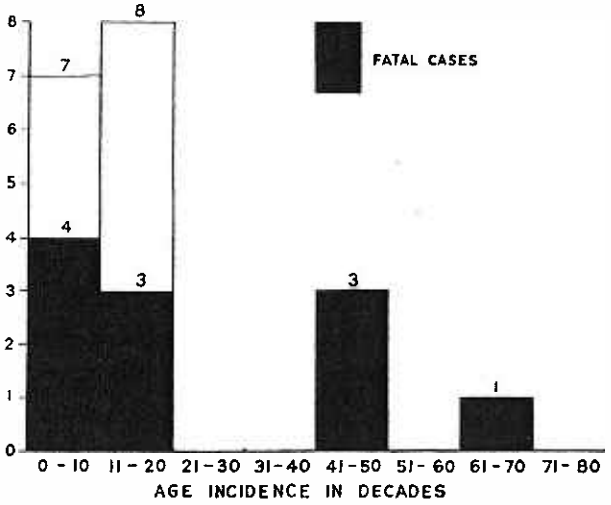


FIG FOUR

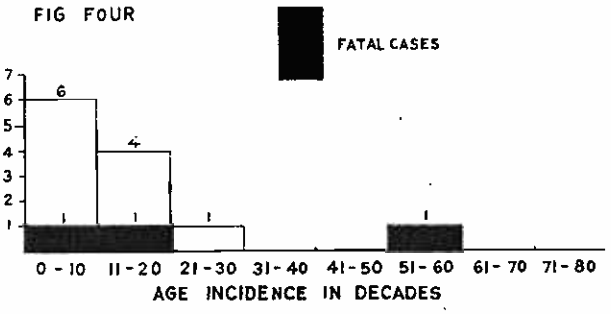


Table Three: Cases with incubation periods of eleven days or more:

Periods	Average incubation period	Number of cases	Mortality rate
First	19 days	22	6 (37.5%)
Second	19 days	7	6 (28.6%)
Third	20 days	6	0 (0%)

Table Four: Cases with no history of injury:

Periods	Number of cases	Mortality rate
First	11	6 (54.5%)
Second	2	1 (50%)
Third	4	2 (50%)

Of the five deaths in the third period of our treatment, three had an average incubation period of four days and they were dead within eight hours of admission. Of the remaining two, both had no history of injury and showed none on examination. The first was a male aged 55 and was the first patient to be put

on Periston-N. He was given only 100 ml. He died suddenly on the sixth day of admission without any appreciable improvement in the attack. The other was a female aged thirty-four admitted in an unconscious state and with generalised spasms. She received 200 ml of Periston-N, but she died within twenty-four hours of admission without recovering consciousness.

The results obtained in the third phase of our treatment have given us enough encouragement to put all tetanus cases on the regime instituted. The mortality rate in this group is the lowest in our experience. The purpose of this article is not to compare our results with those obtained by others as comparison with centres with better or worse facilities will not prove how effective our treatment can be. In this series, our results obtained in the three periods clearly indicated that under the same conditions, patients on the regime of a standard treatment supplemented by the addition of Periston-N stand a much better chance of recovering from tetanus. It is realized that the number of cases treated with Periston-N is still small and that a longer experience must be accumulated. The statistical significance between treatment of the cases in the Periston-N period and those in the second period is small ($X^2 = 5.4$, $P = 0.02$) but already approaches a valid difference.

SUMMARY

1. One hundred and thirty cases of tetanus were seen and treated in twelve and a half years. The over-all mortality rate was 48.2%.
2. Treatment was divided into three periods depending on the regime used.
3. The use of Periston-N in the third period indicates that under comparable conditions the mortality rate is substantially reduced.

REFERENCES

Carle and Rattone (1884)
Kitasato (1889)
Rosenbach (1887)
Tulloch (1918)
Von Behring and Kitasato (1890)
} As quoted by Ian Aird, 1949. A Companion in Surgical Studies. Pages 60-61. E. & S. Livingstone Ltd. (Edinburgh).

Altemeier, W.A. (1946) Penicillin in Tetanus. J.A.M.A., 130. 67.

Cooke, J.V. and Jones, F.G. (1943) The Duration of Passive Tetanus Immunity. J.A.M.A., 121, 1201.

Creech, O., Glover, A. and Ochsner, A. (1957) Tetanus. Am. Surgery. 146, 375.

Spaeth, R. (1941) Serum Therapy of Tetanus. Am. J. Dis. Child., 61, 1146.