

## THE RELEVANCE OF A. T. S. AND BETAMETHASONE IN THE CONSERVATIVE TREATMENT OF TETANUS\*

Experience of a Rural Hospital on the Nepal Bihar Border

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It is always a privilege to share clinical experiences with medical colleagues, and I count it a greater privilege today to be able to address so many who are in all essentials one's professional neighbours. Our neighbourhood admittedly includes the greatest possible topographical variations, from the world's highest mountains to one of its greatest and flattest plain. We encompass a dozen or more language groups and dialects. Yet we have in common a community which possesses a very low or non-existent standard of hygiene, is ignorant and superstitious of disease processes, and is bound by a great variety of dietary inhibitions. These circumstances in themselves increase the mortality and morbidity of the diseases we face, and in this task we also share a common state of impecunity. We are constantly short of supply and facility to do the things which we know can and ought to be done, and are frequently frustrated by the wish "if only we had such and such".

Nevertheless, we are here not to make excuses for ourselves but to improve the situation. It is for us to so work as to constantly maintain before the public a brighter and yet brighter image of sound health, so that it will demand and will be prepared to learn and work for, the eradication of disease. In achieving this we must not only aim to keep our methods of treatment within the financial reach of the community but also a little ahead, like a golden carrot, drawing it on to a healthier life. We do not, however, succeed if we embark on high-powered therapy which is impractical in our situation. In many instances we know a lot more than we can do, and so we should. But we do our best medicine when it is generally acceptable, applicable and steadily progressive.

These remarks are nowhere more relevant than to the treatment of tetanus in our area. It is a disease born of ignorance and uncleanness. The mortality is frightful, and the treatment costly in drugs and nursing care. Thus in our approach to this disease, bearing in mind the shortage of rupees and trained personnel, we in the Duncan Hospital have always sought to find the optimum which allows of the best being done for the greatest number of patients. Our treatment is essentially conservative; we have only ever done three

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tracheostomies among the thousands of tetanus cases we have treated. We aim to maintain a degree of sedation which allows of conscious co-operation in feeding and expectoration, but with the greatest relief from spasms. We mainly use Sparine (promethazine) and Luminal or paraldehyde, with regular antibiotics. We consider isolation to be unnecessary. Tetanus patients are treated in the general wards; they are segregated into the quietest part of the hospital to facilitate nursing, but not from any fear whatsoever of cross infection. Where feeding is not possible, nutrition is maintained by Ryle's tube or parenteral means. With this usual basic treatment, we add two controversial agents—anti-tetanus serum, and betamethasone. We need to consider the use of A.T.S. because many would have us believe it is unnecessary. We need to consider the use of betamethasone as it will enable us to salvage many who would otherwise die.

### Mortality Grouping.

Before we go further it is necessary to define our criteria of high and low mortality. We are all well aware that in tetanus any evaluation of success in treatment must always be measured against the severest cases, for many cases of established tetanus will survive with minimal nursing care, though I doubt if many survive in the villages on their own. We adopt a very simple but at the same time exacting definition of high and low mortality groups. By this we assess cases according to the probability of their dying. The high mortality group includes only those cases whose chances of survival are very poor (less than 20%) despite skilled and specific tetanus treatment. The low mortality group includes the rest, even though many of them are extremely severely affected and many die. In the high mortality group we eliminate any who might have survived anyway with normal nursing care and sedation. Our definition of a high mortality case therefore is a patient

- 1) who reaches hospital within 48 hours of the onset of symptoms, termed Admission Delay, and
- 2) either in whom generalised spasms have developed within 24 hours of the onset of symptoms, termed Onset Period,  
or who has arrived in hospital within 24 hours of the first symptom but in whom generalised spasms have been masked or delayed by sedation. If in such a case generalised spasms or total muscular rigidity develop within 48 hours of the first symptom, the case is included in the high mortality group.

Cases developing generalised spasms or total rigidity more than 48 hours after the onset of symptoms, despite 24 hours or more of sedation, are regarded as low mortality for statistical purposes.

The significance of admission delay in our circumstances is related to two things. Firstly, to the rapidity with which tetanus kills. In 1961 (Sanders 1964) of 99 cases brought to the Duncan Hospital within 24 hours of the onset of symptoms, 66% ultimately died, but 33% died within 48 hours of the onset of symptoms, despite hospital treatment. Some of these died within 12 hours of the onset of symptoms. Thus any patient seen 48 hours or more after

the onset of symptoms had its potential mortality at least halved. Secondly, the more quickly symptoms develop at home, the more quickly the patient is brought to hospital, which fact is closely related to the onset period. We have found that an admission delay of 48 hours corresponds to an onset period of 24 hours: of cases with an onset period of 24 hours or less, 85% had arrived within 48 hours, and of those arriving within 48 hours 91% had an onset period of 24 hours or less. Cases with an admission delay of more than 48 hours are never regarded for statistical purposes as high mortality whatever the stated onset period may be, and no case with an onset period of more than 24 hours is regarded as high mortality, unless as already stated the case was admitted within 24 hours and generalised spasms or stiffness were delayed by sedation. We do not find the incubation period of much value in assessing potential high mortality, except in neonates. In one third of our cases it is impossible to ascertain when the bacillus gained entry, and even then only 63.5% of cases with an incubation period of 0-8 days have an onset period of within 24 hours. Where the history given is uncertain the admission delay is the fact most reliably obtained, unless the patient develops generalised spasms after admission. We find the above guide to assessing inherent high mortality simple, reliable, and quickly made. Of course many patients outside this high mortality group develop the severest forms of tetanus and are treated accordingly, but are not regarded as high mortality cases for statistical purposes. Over the years as hope for this condition became more known, more people have been brought early. In the years 1950-56 the proportion of high mortality cases was 13.5%; in 1956-61 it was 35%; in the present no-betamethasone group of 1965-66 it was 48%, while in the with-betamethasone group of 1966-67 it was 55%. So if you get a name for tetanus treatment, your mortality percentage is liable to go up and not down.

#### Anti-Tetanus Serum.

There was a time when it was thought that A. T. S. was only of value if given in very high doses of several hundred thousand units. The cost of this alone was prohibitive, and we were not persuaded that the high dosage of A.T.S. was of any value. Thus in 1957 (Sanders 1965) we at the Duncan Hospital embarked on a series of clinical trials covering five years, in which we treated succeeding random groups of patients with 10,000 units of A. T. S. given intramuscularly or intravenously, and given on one day only or as repeated doses for up to 4 or 5 days. Whereas we did not have enough in the multiple intramuscular doses group for adequate comparison, what we did find, however, was that repeated intravenous injections of A. T. S. gave statistically better results than single intravenous dose and better than single intramuscular injection. Since May 1966 we have reduced the amount of A.T.S. still further. Our standard dose is now 1500 units A. T. S. given daily up to four days, and intravenously where possible. Reducing the amount of A.T.S. has made no difference to the mortality.

The question naturally arises, therefore, as to whether A. T. S. is of any value at all in established tetanus. Some have ventured to conduct series in which A. T. S. has been withheld, and results from these trials vary considerably. The trial by H. Vaishnava et al (1966) showed no difference in mortality whether A.T.S. was given or not. But in that trial

no account was taken of high and low mortality, besides which it is very doubtful if the stated incubation period alone is an adequate standard for assessing the severity of tetanus except in neonates. The trial by P.S. Singh et al (1966) from Delhi indicated a lower mortality among cases not given any A.T.S. This trial, however, appears to make no allowance for inherent seasonal variations in mortality, which would be likely in Delhi. Also, they used high doses of A.T.S., which Patel et al (1963) suggest may well be harmful. Patel et al however, in the more constant climate of Bombay, have shown a statistically significant rise in mortality when A.T.S. is not given. Similarly a trial in Nigeria (Brown et al 1960) resulted in a statistically significant increase in mortality when A.T.S. was not given. There is in fact, very good reason for not even contemplating the routine denial of therapeutic A.T.S. The reason is quite simply that prophylactic A.T.S. is known to prevent tetanus by the neutralisation of tetanus toxin as it is produced. Thus in the established disease it is reasonable to assume that there may be some free toxin which can and should be neutralised and so reduce the degree of ultimate intoxication. It is not surprising either that very small amounts of prophylactic A.T.S. should suffice to neutralise any heavy infection. 171 units A.T.S. is capable of neutralising at least 1,000 M.L.D. in a 60 Kg. man, or if you like, 1 unit of A.T.S. can neutralise the M.L.D. for six men.

We need to be aware of the influence of Britain and America which is increasingly in favour of forbidding the prophylactic use of A. T. S. (Sharrard 1965). Many reasons for this prejudice are valid as the situation in these countries is very different from ours. These differences are, briefly, 1) the populations of the U. K. and most of North America are to a great extent actively immunised. 2) uterine and auricular contamination is negligible. 3) trauma is promptly and expertly handled, with also penicillin coverage. 4) the risk of serum sensitivity is enormous in those highly inoculated communities, resulting in more casualties from tetanus. On the other hand we, in our localities virtually do not get serum sensitivity. Added to the risk of serum reactions is the knowledge that once sensitised to the serum, subsequent injections of A. T. S. give virtually no protection, as the carrier serum is destroyed within 24 hours. Thus in Britain and N. America it is common to find cases of established tetanus in people who were given prophylactic A. T. S. This is also true in cases where there appears to have been no previous serum inoculation. Hence, because A. T. S. has apparently "failed" there to prevent tetanus in some instances, many would discard its use completely, not only prophylactically but therapeutically too. But it needs to be realised that all prophylactic measures, whether toxoid, expert surgical debridement of wounds, or penicillin all have apparently failed to prevent tetanus in some cases. Added to which, it is pertinent to point out, that contracting tetanus itself is no guarantee against further infection. We have had four patients who contracted tetanus twice; one a boy of six, who had first come with neonatal tetanus, actually died through the second infection. The reason for this is that the amount of tetanus toxin which produces the established disease is so small, and is often insufficient to stimulate antibody response.

Up until recently, it has been acknowledged that the value of prophylactic A. T. S. was conclusively demonstrated in the early months of the First World war, when the average incidence of tetanus fell from 81000 to 21000 wounded. Recently A. T. S. sceptics (Stafford 1955; Cox et al 1963) have suggested that the dramatic drop in incidence of tetanus

during that period was not due to A. T. S. but to better surgical technique. But the Official history of the War makes it clear that prodigious though the efforts were to establish top-grade advance casualty clearing stations, the incidence of tetanus had dropped before adequate early routine surgical treatment could be established. Undoubtedly, good surgery obviated the use of much A. T. S., but the fact remains that A. T. S. prevented tetanus developing from many inadequately treated wounds. Prophylactic A. T. S. also eliminated cases of tetanus occurring from trench foot. It is important to hear again the evidence of those days, and to note the conclusion reached. I quote (Official History of the War) "While giving full weight to the importance of efficient surgery in ameliorating the severity and diminishing the mortality of tetanus, more especially when excision of the wounds is practised as in the latter part of the war, neither this nor any other factor can be so closely associated with a diminution of incidence, severity and mortality and with a prolongation of the incubation period, as can the administration of anti-toxic serum". I would add that this was the opinion among others, of such neuro-physiologists as Sherrington and Sharpey Schafer. It also needs to be emphasised that this successful prophylaxis was accomplished by the use of only 500 units A. T. S. at a time.

Today, of course, it would be difficult to emulate the circumstances of the First World War. But without doing a trial, circumstantial evidence (Lucas et al., 1965; Ellis 1965) weighs very heavily in support of A.T.S. We have had only one case develop tetanus following the giving of A.T.S., a severe 70% burns developed tetanus five days after having A.T. S. Some 5,000 patients at risk have been given prophylactic A.T.S., in an area where the village incidence of neonatal tetanus is 10% or more, and tetanus cases excluding neonates comprise 10% of all admissions to our hospital.

Regarding the use of A. T. S. in established tetanus, therefore, we have no reason to doubt that it can and does neutralise free tetanus toxin, in the body. Considering the very high potency of even the minutest amounts of the toxin, neutralising the free toxin before it is fixed makes the difference between life and death for many (but not all) of our patients. The question therefore is, not how much A. T. S. we should give, but how little, perhaps only homeopathic amounts, but never none.

Before we leave the subject of A. T. S. perhaps some attempt should be made to summarise the application of tetanus prophylaxis in our situation. First, too great emphasis cannot be made on washing, and the application of clean material to wounds and vagina. Secondly, we are justified in adopting a discriminative use of prophylactic A.T.S. A.T.S. may be withheld in early, superficial, clean wounds, and where the individual has previously had A.T.S. In dirty deep wounds, whether A. T. S. has previously been given or not A.T.S. should again be given with high doses of penicillin, and also toxoid—not forgetting to clean the wound as much as possible. Thirdly, a full course of toxoid (preferably alum precipitated) gives one the protection against absorbed tetanus toxin. For optimal protection I would think that in our area, after initial full active immunisation, subsequent toxoid need only be given in the event of injury, contamination, or pregnancy.

However, the prospect of having to inoculate the whole of our enormous population is of course just fantasy. But the following scheme might perhaps be practicable in localised communities, and possibly for the whole of Nepal. It lies in the inoculation of woman. We reckon that adult women alone account for 22% of all cases of tetanus. Tetanus neonatorum accounts for a further 34%. We know that active prophylaxis in the mother prevents neonatal tetanus, thus the inoculation of adult women would immediately reduce the incidence of tetanus by 56%. Furthermore, I find no evidence that ante-partum immunity is not still active in childhood. A practical scheme therefore would be to inoculate initially all females between 10 and 40 years of age (about one third only of the population). As girls between the ages of 10 and 15 years account for 3% of tetanus, we could thus achieve a reduction of 59% in the incidence of tetanus within nine months. Five years later, with the inoculation of children only (both boys and girls) between the ages of 5 and 15, (about 20-25% of the population) the incidence of tetanus would be reduced by a further 30%. Subsequently, routine inoculations need only be conducted every five years, and that only necessitating the giving of a single injection to all children between the ages of 5-10 years, about 10-15% of the population. The rest of the population could be reasonably protected by the regular giving of a booster tetanus toxoid in the presence of contaminated wounds, infection, and pregnancies. Thus, within six years, we could reduce our present incidence of tetanus by 89%, with the prospect of continuing annual decreases. It would be up to local authorities to decide whether the cost in manpower and money to achieve this is greater than the losses incurred through developed tetanus. But I would commend this suggested scheme to you for consideration.

#### Betamethasone

Cortico-steroids have been used for many years in the treatment of tetanus, but with greatly varying results (Lawrence et al 1963). What help they gave was considered to be only supportive in nature, helping a flagging suprarenal to withstand long term, generalised exhaustion. Because of its unproven value in tetanus we did not include it in an already costly therapeutic regime at the Duncan Hospital. When, however, it became apparent that patients died of tetanus toxicity, quite apart from spasms, we gave batamethasone as we did for other toxic conditions such as typhoid. In a pilot trial in 1966 the overall mortality was reduced from 61% (11 out of 18) to 18.5% (5 out of 27) (Sanders 1966.) This result stimulated full year's trial, through 1966-67, of 192 consecutive cases treated with betamethasone. This treated group was compared with 170 consecutive cases from the previous twelve months 1965-66, when no betamethasone was given. Neither of these groups include neonates for whom we are obliged to provide only out-patient treatment.

The overall mortality in the no-betamethasone group was 61% (103 out of 170), as compared with 37% (70 out of 192) in the with-betamethasone group. Among high mortality cases the results were 80% (66 out of 82) and 50% (53 out of 100) respectively. This difference is highly significant; using  $X^2$  formula, the probability of this being a chance result is considerably less than 1/1000.

The two groups had the same basic routine treatment, with two minor exceptions. 1) As has been mentioned, we reduced the standard dose of A. T. S. from 10,000 units to 1,500,

this occurred half way through the no-betamethasone series, but it made no difference to mortality. 2) We found in using betamethasone that it was dangerously easy to over sedate a case if morphia was also being used, thus in the with-betamethasone group, morphine was not used, as it had been in many of the non-betamethasone group. We have shown in previously reported trials that the use of morphia in no way embarrasses a tetanus patient, and previously we had our best results with using morphine in small doses, together with chlorpromazine, or promazine and Luminal. One justifiable objection to this trial of betamethasone is that the control series was not conducted concurrently with cases allocated at random. We were for administrative reasons not able to do this, besides which, following the success of the pilot trial, it would have been regarded as unethical by staff and patients to have withheld betamethasone in any case. Nevertheless obtaining these most significant results from among only cases of potentially very high mortality, and covering all seasons, I believe is a more convincing basis of evaluation than comparing random groups in which the potential mortality may vary considerably. It is futile to assess the efficacy of treatment among cases who are going to get better whatever you do. I would suggest, too, that the with-betamethasone group was probably the more severe, as in many instances betamethasone delayed the onset of spasms to such a degree that cases which, but for betamethasone would have been regarded as high mortality, were put in the low mortality group of the with-betamethasone series.

In view of the cost of betamethasone, we further varied the oral dosage of the drug to ascertain how much we could save on injectable betamethasone. These assessments were made among high mortality cases only during the winter months of November to February. The results were surprising. Doubling the doses of oral betamethasone resulted in rather more injectable being needed and not less. For example, adults given a daily oral dose of 8 mg. for the first ten days of treatment, required a daily average of 14.3 mg. injected betamethasone as well. Reducing the oral dose to 3 mg. a day resulted in an addition of only 13.7 mg. by injection. Similarly with children and infants, 6.9 mg. by mouth per day required an average of 16.3 mg. only by injection, while an average of 2.6 mg. by mouth per day required an average of 13.3 mg. only by injection. Since we have discontinued oral betamethasone, I suspect (but have not actually analysed) that we have needed even less by injection. Incidentally too the mortality was significantly lower among the low oral dose cases, 44% (7 out of 16) as compared with 57% (21 out of 37)  $\chi^2=7.583$ ,  $10 > P > .001$ . The reason why oral betamethasone apparently increases the demand for injectable betamethasone is perhaps related to the possibility of decreased gastrointestinal function due to local betamethasone. This would result in diminished absorption of oral sedatives, resulting in more having to be given parenterally.

We looked for complications attributable to betamethasone, and found very little. Betamethasone potentiates minor oral, nasal and subcutaneous haemorrhage, and this is soon rectified by reducing the amount of betamethasone. Significantly, not even oral betamethasone increases the incidence of gastric petechial haemorrhage which occur with severe brain stem tetanus intoxication. The giving of high doses of betamethasone resulted in slight

subcutaneous oedema, especially of the head, as we keep the foot of the bed on high blocks. This oedema is easily enough rectified by the giving of oral potassium chloride. The incidence of bronchopneumonia, the commonest complication of tetanus, was probably higher among cases treated with betamethasone, but of course patients lived longer to get pneumonia and even then survived. Betamethasone is extremely well tolerated. 50 mg. or more a day by injection will not harm the patient, although early on some cases were probably over-sedated, until we realised that the use of betamethasone reduced the requirements of other sedatives. We found that intravenous betamethasone gave the best results and was the most economical. However, from an analysis of all adult high mortality cases it is apparent that the giving of more than 24 mg. per day intravenously does not materially increase the chances of survival. In other words, if a case is so bad that 8 mg. of betamethasone given 8-hourly intravenously (together with other sedatives) does not control spasms and toxicity, then increasing the amount of betamethasone will not help. With children it was impossible to ascertain an optimal level of betamethasone as sizes varied and injections could not always be given intravenously.

Why betamethasone should be so effective in tetanus is a question to which we would all like to know the answer. I am afraid we haven't time to say more here other than to suggest it is related to intraneural anti-histamine action (Richter, 1957 Kwiatkowski 1943); to direct anti-toxic action (Finel et al 1960) to countering the cholinergic action of tetanus toxin (Fal et al 1963) and by preserving (Fal, 1963) and supplementing (Lawrence et al 1963) the suprarenal cortex. Whatever the rationale of its action may be, the important thing for us is that it works. Not only does betamethasone reduce mortality from tetanus, but it considerably facilitates the management of the patients. Necessary nursing procedures are less disturbing, the patient is more consciously co-operative in breathing and expectoration, he is more relaxed and is able to take food by mouth earlier. Furthermore, perhaps the greatest benefit of betamethasone comes from being able to give large doses intravenously in order to get a new case quickly under control. Patients can be lost by giving too little and too late, but no harm will come from giving a patient too much too early.

Having said all this, we do not regard the challenge of tetanus as closed. Far too many die despite the full use of betamethasone. We pray God's help and blessing on continued endeavours to find the specific cure to this most terrifying disease.

#### Acknowledgements

We wish to emphasise that the work reported here is the result of most congenial teamwork with Dr. T.N. Strong as Medical Superintendent, Nurses and Office clerks, orderlies and doctors. Through care of patient and care of chart, communication and supervision, all are important in the management of tetanus.

I would like to publicly express our appreciation of the help given to us by Glaxo Laboratories (India) Ltd. for considerable free quantities of 'Betnasol' brand of betamethasone, and to Pfizer (India) Ltd. for a free allocation of 'Betacortril' brand of betamethasone,



and to Mr. R. H. Sanders for his financial help; without all this assistance our work would not have been possible. Then lastly, the nursing care, because it is probably the most important single criterion in the treatment of tetanus. I wish to pay a sincere tribute to the consistent conscientious care given by the nursing staff of the Duncan Hospital to the very many tetanus patients. Some diseases involve heavy nursing work and some constant hour-by-hour care, but few are frightening. Tetanus demands continual hard, quick work, constant vigilance, and enduring courage to support these whose very disease strikes fear into the heart of patient and attendant alike. I believe it is to such persistent, vigilant and courageous nursing that most patients owe their survival.

## APPENDIX

### THE DUNCAN HOSPITAL TETANUS TREATMENT SCHEDULE

#### HISTORY

The most important information to obtain is—

- (1) When did the first symptoms develop,
- (2) How soon after the onset of the first symptoms did generalised spasms develop.

Initial treatment is largely based on this information. Any case presenting within 24 hours of the onset of symptoms whether spasms are present or not, is to be regarded as very serious, until shown to be otherwise. Also regard as potentially severe cases of deep causal injuries, e. g. compound fractures, uterine infection, etc. Patients having generalised contractions at the time of admission are obviously in a serious condition.

#### TREATMENT

**Sedation.** All severe cases (as defined above) should be quickly sedated, before further transportation or nursing procedures are commenced. When well sedated a Ryle's tube may be passed to facilitate feeding, hydration and oral administration of drugs. Try to keep the patient at a level of sedation in which he can breathe and expectorate adequately but at the same time remain oblivious to outside stimuli.

**Betamethasone.** For all severe cases 2 ml. of betamethasone should be given intravenously on arrival. For all children 2 ml. intramuscularly is better. For subsequent treatment at least 2 ml. intravenously every 12 hours, and 2 ml. p. r. n., should be prescribed. For the worst cases 2 ml. 8 hours or 6 hours may be ordered, with 2 ml. p. r. n. For children, 2 ml. or 1 ml. may be ordered every 6, 8, or 12 hours as required. It is better to give too much than not enough. In the severest cases 2 ml. intravenously and 8 hourly may be given to even 7 year olds, for three to four days if necessary.

For the not so severe case, an initial 2 ml. intravenously facilitates the passage of the Ryles tube and guards against unexpected spasms. Otherwise 2 ml. intramuscularly at any time in the event of sudden severe spasms.

**Promazine (Sparine)** 50 mg. of promazine intravenously to serious cases, or intramuscularly to others should be given on arrival. Subsequently promazine may be given by Ryles tube 25 mg. to 75 mg., 6 or 8 hourly. Where generalised spasms have occurred, regular intramuscular promazine 50 mg. 4, 6 or 8 hourly (25 mg. in children) greatly facilitates the control of spasms. When promazine is being given by injection, none should be given by mouth.

**Barbiturates.** With chlorothiazides such as promazine, it is good to combine another sedative, and barbiturates are good drugs to use. Phenobarbitone 30-60 mg 6, 8 or 12 hourly by mouth (or Ryle's tube), and injectable phenobarbitone sodium 100-200 mg. given as required, help to control spasms and maintain hypnosis.

**Paraldehyde** and other injectable sedatives may be given at times.

**Pethidine** 50-100 mg. may be given intravenously initially, together with betamethasone and promazine in order to effect immediate control of the patient. It may occasionally be used to alleviate painful rigidity when the patient is recovering.

**Promethazine (Phenergan)** 50-mg. Intravenously is a good drug to use in emergency to control spasms, but its continuous use in effective doses, especially with promazine and betamethasone is probably unwise.

**Potassium Chloride** 30-45 ml. daily should be given to offset potassium loss resulting from very high and prolonged betamethasone therapy.

**Vitamin B. Complex** two to three tablets daily should be given routinely to prevent minor degrees of dementia which sometimes develops after a long period of tetanus intoxication and treatment.

**Antibiotics.** From the first 400,000 to 800,000 units procaine penicillin should be given daily. In the presence of uncontrolled bronchopneumonia or other infections, penicillin should be increased or other antibiotics introduced. Remember that betamethasone masks the presence of all but the severest degrees of hyperpyrexia. Antibiotic cover should be continued until the patient is mobile, as a safeguard against hypostatic pneumonia.

**Roundworms.** Some patients especially children, die of intra-tracheal roundworm asphyxiation. We find the prophylactic and therapeutic treatment of roundworms in tetanus is dangerous and not to be advised.

**Anti-tetanus serum.** 1500 units A.T.S. should be given intravenously on admission, and repeated daily for 4-5 days in severe cases.

#### NURSING PROCEDURES

It helps to put the foot of the bed up on 10-15" blocks from the very first, to prevent aspiration-pneumonia, and to help in draining the chest. The patient should be turned from side to side regularly. Constant vigilance is necessary to remove bronchial and nasal secretions. Damage to the tongue must be prevented. In severe cases a rubber airway kept permanently between the teeth and over the tongue is a good procedure and

is well tolerated. With frequent asphyxiating spasms, the Ryle's tube is best removed for a day or two until the crisis is over. Where fluids cannot be given by mouth subcutaneous or intravenous infusions must be given.

When spasms are at their worst the otherwise necessary back rubbings and bed baths should be omitted, to keep the patient as quiet as possible. Similarly, relatives who are noisy or who persist in handling the patient should be out of the ward.

Bladder retention and incontinence is a common complication which may necessitate an indwelling catheter. But indwelling catheters irritate, and may precipitate spasms. Similarly, the too energetic treatment of inevitable constipation may do more harm than good. Early suppositories, or enemata after the worst of the disease is over, is usually all that is required to relieve rectal discomfort.

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