haemoglobin electrophoretic pattern, and improves clinically after the administration of methylene blue or ascorbic acid.

No treatment is indicated and the patient should be fully informed about the benign nature of the disorder. A Medic-Alert token may be useful to avoid unnecessary investigations and iatrogenic restrictions.

REFERENCES

The Action of Tetanus Toxin
A Case Report
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SUMMARY
A case of neonatal tetanus in an infant with myelo- meningocele is described, and the possible action of tetanus toxin is discussed.


Most of the research regarding the mode and site of action of tetanus toxin has been done on laboratory animals such as rats and cats. Infrequent reports on its action in human beings have been published. 1,2 We have made observations which substantiate the results of the animal experiments.

CASE REPORT
A 10-day-old infant was brought to Ga-Rankuwa Hospital, Pretoria, with a 1-day history of convulsions. He had been born at home with the grandmother assisting. The umbilical cord had been cut with a non-sterilized pair of scissors, ligated with a piece of dress material, and covered with a layer of petroleum jelly. The birth weight had not been recorded. The mother noted a red swelling low down on the infant's back, and since birth he had not been seen moving his lower limbs. He was breast-fed and sucked quite vigorously until the 9th day. When the convulsions started, he also started having difficulty sucking at the breast. The spasms involved his face and arms, but not his legs.

On examination he weighed 2.8 kg, his temperature was 35.9°C, the pulse rate was 180/min, and the respiratory rate was 36/min. The skull circumference was 37 cm (75th percentile). The trachea was central, but the entry of air was reduced in the right hemithorax, and no adventitious sounds could be heard. The umbilical stump appeared slightly septic. A type 1 myelomeningocele was present over the dorsum of the spine, with the upper border of the lesion at the level of L2. He had a total flaccid paralysis of both legs. There was one spontaneous spasm every 5 minutes, which was typical of tetanus with risus sardonicus, opisthotonos, and a rigid abdomen. Trismus and hypertonia were present between attacks. Spasms could also be started by stimulating the infant. Significantly, the upper limbs went into spasms, but not the lower limbs. Repeated tactile stimulation of the lower limbs did not result in spasms, but stimulation of the trunk and arms provoked it. The legs always remained flaccidly paralysed.

Although special investigations are not helpful in the diagnosis of tetanus, they do help to rule out other similar conditions. The results of investigations were as follows: the erythrocyte count was normal for age, and there was polymorphonuclear leucocytosis (27.6 × 10⁹/l). Blood chemical values were as follows: Na - 144 mmol/l, K - 4.6 mmol/l, Cl - 109 mmol/l, Mg - 0.83 mmol/l, and Ca - 2.32 mmol/l.
Aerobic and anaerobic blood cultures showed no growth. Cultures of umbilical swabs produced growths of β-haemolytic streptococci, not group A, and a Staphylococcus aureus, both sensitive to penicillin. Chest radiographs confirmed pneumonia of the right upper lobe. Lumbar puncture was not performed because of the myelomeningocele. An intravenous line was established and penicillin was given. Tetanus antitoxin was administered and the infant was sedated with diazepam and phenobarbital. Artificial ventilation was not considered. The spasms decreased in frequency over the following days, but on the 5th day after admission he died. We were not able to perform an autopsy.

DISCUSSION

Clostridium tetani produces two toxins,\(^4\) tetanolysin and tetanospasmin. Tetanolysin causes in vivo lysis of erythrocytes, but no adverse effect has been found in vitro. Tetanospasmin is a double-stranded protein\(^6\) which has the following five known effects on the nervous system:\(^7\)

1. It has a peripheral action on the motor end-plate, probably inhibiting the release of acetylcholine from the nerve terminals.\(^7\) Tetraplegia is observed in a small percentage of patients after severe tetanus.
2. It affects the spinal cord, and this action of the toxin is mostly responsible for the typical clinical features of tetanus. All parts of the nervous system are continually being bombarded by incoming sensory nerve impulses.\(^3\) The eventual effect on muscle would be to cause summation of contraction with complete fusion and sustained response, i.e. tetanus.\(^5\) This does not usually happen owing to the phenomenon of inhibition which enables the selection of those signals that are important, while blocking those that are not.\(^9\) Inhibition is possibly mediated chemically through the release of γ-aminobutyric acid by the inhibitory neurons. Tetanospasmin probably prevents the release of this substance.\(^8\) It is also possible that, additionally, the toxin may cause direct damage to the interneurons in those reflexes that have a polysynaptic pathway.\(^5\)
3. A similar effect to that seen in the spinal cord has been demonstrated in tests on the cerebral cortex of animals.\(^11\)
4. Kerr et al.\(^12\) described sympathetic nervous system manifestations. Hypertension, tachycardia, peripheral vasoconstriction and increased urinary excretion of catecholamines were some of the findings noted.

5. Cephalic tetanus is manifested by cranial nerve palsies and occurs as an early phase in some cases of tetanus. The 3rd, 4th, 7th, 9th, 10th and 12th cranial nerves may be involved — the 7th most often.\(^8\) In this case tetanospasmin blocks impulse conduction in the nerve itself and not at the motor end-plate as in the late phase of tetanus.\(^2\)

Abel and Chalian\(^13\) postulated a possible direct stimulatory effect of tetanus toxin on muscle or on motor end-plates. This work is still referred to in a well-known textbook\(^14\) on childhood neurology. No evidence for this action on striated muscle has been found in animals and it is not supported by the continuous flaccid paralysis of the legs in our patient, although the responsiveness of the muscles may possibly have been impaired by the congenital lack of innervation.

The existing knowledge of the muscular development of the patient with myelomeningocele seems scanty. Immature muscle fibres possess intrinsic contractility unrelated to neural influence as shown in cultures of embryonic muscle.\(^2\) This spontaneous in vivo action is associated with a low threshold of electrical excitability and is influenced by a variety of pharmacological agents. The in vivo situation is unknown and the study of muscular tissue from spina bifida paralytics should throw some light on this problem of embryological development.

No conclusions can be drawn as to motor end-plate involvement in this patient’s lower limb muscles, since the paralysis was present from birth and the effect of tetanospasmin at this site is to paralyse muscles.

REFERENCES