perforation and mortality appears to be related to failure to understand the significance of symptoms in preterm neonates resulting in delayed diagnosis. The diagnosis was established on necropsy in 56% of reported cases. In others, diagnosis was established only on laparotomy, and in none of the reported cases the diagnosis was suspected clinically. This condition should be seriously considered in the differential diagnosis of necrotizing enterocolitis. Neonates with a lump or erythema in the right iliac fossa, signs of intestinal perforation or peritonitis during early neonatal period should be suspected to have appendicitis. An early diagnosis and timely surgical intervention can reduce mortality in this otherwise uniformally fatal condition.

REFERENCES

1. Diers F. Appendicitis. *In:* Baster Kinderspital Baste Diersentation. Lase 1908, 36: 63.
2. Hemlatha V, Spitz L. Neonatal appendicitis. Clin Pediatr 1979, 18: 621-622.
3. Martin LW, Glen PM. Prenatal appendiceal perforation. J Pediatr Surg 1986, 2: 73-74.
4. Martin LW, Persin EV. Neonatal perforation of appendix in association with Hirschsprung's disease. Ann Surg 1967, 166: 799-802.
5. Barlett RH, Eraklis AJ, Wilkinson RH. Appendicitis in infancy. Surg Gynec Obstet 1970, 130: 99-106.
6. Lassifer HA, Werner MH. Neonatal appendicitis. South Med J 1983, 73: 1172-1175.
7. Kottmeier PK. Appendicitis. *In:* Pediatric Surgery Eds Chicago, Welch KL, Randoloph JG, Ravitch MM. Yearbook Medical Publishers Inc. 1986, p 991.
8. Marcy SM, Klein JO. Focal bacterial infections. *In:* Infectious Diseases of Fetus and Newborns Eds Remington JS, Klein JO. Philadelphia, WB Saunders Co, 1983, pp 782-819.
9. Kliegman RM, Fanaroff AA. Neonatal necrotising enterocolitis: A nine year experience. I. Epidemiology and uncommon observations. Am J Dis Child 1981, 135: 603-607.
10. Snyder WH, Chaffin A. Appendicitis in first two years of life. Arch Surg 1952, 64: 549-560.
11. Hardman RP, Bowerman D. Appendicitis in newborn. Am J Dis Child 1963, 105: 99-101.
12. Parson JM, Miscall BG, McSherry CK. Appendicitis in newborn infant. Surgery 1970, 67: 841-843.
13. Kliegman RM, Fanaroff AA. Neonatal necrotizing enterocolitis: A nine year experience. II. Outcome assessment. Am J Dis Child 1981, 135: 608-611.

Local Tetanus Initially Mistaken as Compressive Thoracic Myelopathy: A Case Report with Electrophysiological Findings

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Tetanus is an acute and often fatal disease caused by an exotoxin in a wound and characterised by generalized increased

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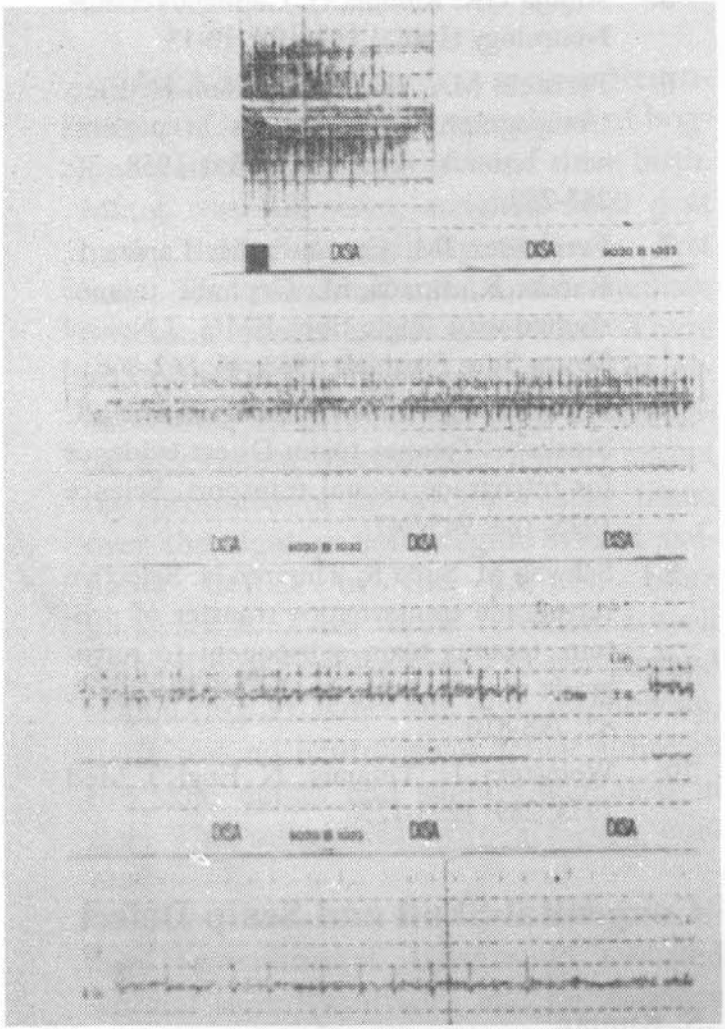

Fig. 1. Concentric needle EMG from calf muscles showing prolonged insertional activity with 'repetitive potentials'.

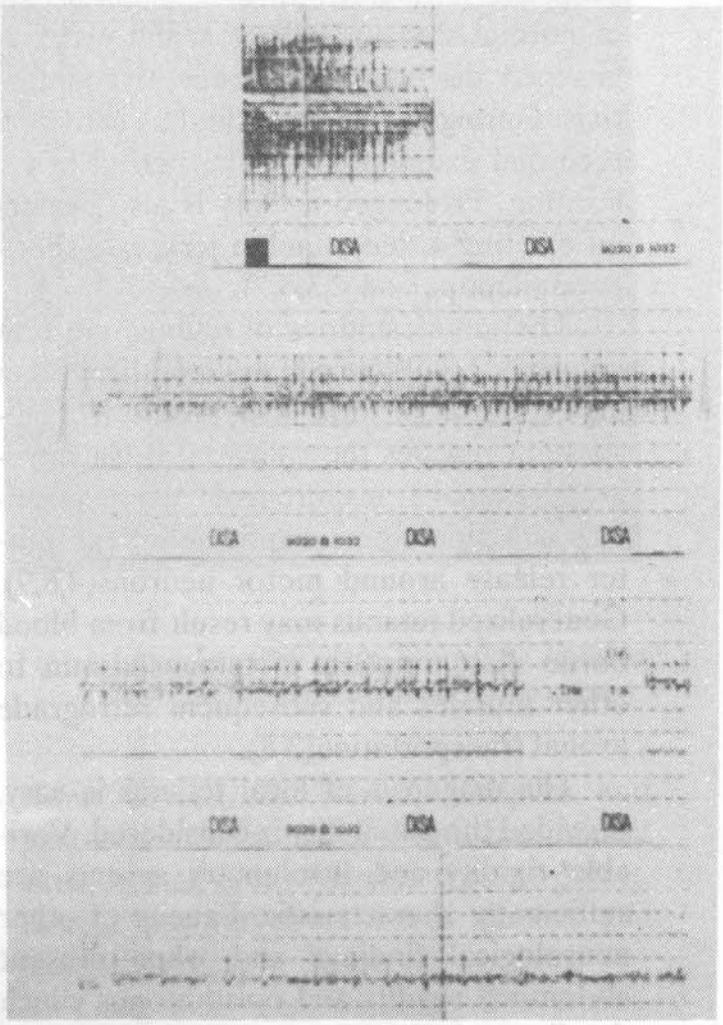

Fig. 2. Concentric needle EMG from quadriceps muscle showing prolonged activity with absence of the normal silent period after elicitation of knee jerk (arrow).

erature. Of these perhaps cephalic tetanus has been reported to be the commonest(2).

Local tetanus involving limbs is relatively uncommon. The clinical picture is typical and once seen it is difficult to confuse it with any other disorder. Twitching of muscles adjacent to the wound is usually the first manifestation(4). The affected limb exhibits marked rigidity which may vary from one examination to another, and often persists in sleep(3). Attempted voluntary movements induce involuntary spasms. The limb becomes extended at the hip and knee and plantar flexed at ankle. This so called "extensor sign" is also evident while eliciting the plantar response. Deep tendon reflexes may be brisk or normal. No upper motor or sensory signs are evident(2). Spasms gradually decrease with time and eventually recovery is complete. Rarely the disease may become generalized, usually with a fatal outcome(2-4).

The electromyographic findings though non specific are highly suggestive in an appropriate setting. Motor unit potentials are of normal size and duration and occur as grouped discharges, the repetitive potentials. During spams a continuous activity is recorded extending into the period of relaxation. Prolonged activity is also evident on eliciting a deep tendon jerk, *i.e.,* shortened silent period(2,3,6,7).

The manifestations of tetanus are now known to result from spinal and brainstem disinhibition of motor neurons. Tetanospasmin reaches the spinal cord via retrograde axonal transport from nerve terminals and blocks presynaptic neurotransmitter release around motor neurons(4,8,9). Generalized tetanus may result from blood borne dissemination of tetanospasmin to other muscles and subsequent retrograde axonal transportation(3,8).

The diagnosis of local tetanus is easy, provided the possibility is considered. Variable rigidity and involuntary spasms are sufficiently characteristic. Paucity of other neurological findings and when present abdominal rigidity and opisthotonus clinch the diagnosis(2,3,10).

REFERENCES

1. Beaty HN. Tetanus. *In:* Harrison's Principles of Internal Medicine, Vol I. Mc Graw Hill Book Company, New York, Eds Bdraunwald E, Issel Backer KJ, Petersdorf RG, *et al*. 1987, pp 558-560.
2. Struppler A, Struppler E, Adams RD. Local tetanus in man. Arch Neurol 1963, 8: 162-178.
3. Jain S, Ashik PP, Maheshwari MC. Local tetanus: A case report with electrophysiological studies. J Neurol 1982, 228: 289-293.
4. Griffin WJ, Roistachier K. Clinical conferences at the John Hopkins Hospital. Local tetanus. John Hopkins Med J 1978, 149: 84-88.
5. Ahjuja GK, Kamala G. Cephalic tetanus. Neurology (India) 1978, 20: 10-13.
6. Perlstein MA, Turner M, Elam H. Electromyographic observations in patients with tetanus. Arch Phy Med 1958, 39: 283-289.
7. Fernandez JM, Ferrandiz M, Larrea L, Ramio R, Boada M. Cephalic tetanus studied with single fiber EMG. J Neurol Neuro Surg Psychiatr 1983, 46: 862-866.
8. Price DL, Griffin JW, Young A, Peck K, Stocks A. Tetanus toxin: Direct evidence for retrograde axonal transport. Science 1975, 188: 945-947.
9. Schwab M, Suda K, Thoeren N. Selective retrograde transsynaptic transfer of protein, tetanus toxin subsequent to retrograde axonal transport. J Cell Biol 1979, 82: 798-810.
10. Weinstein L. Tetanus. N Engl J Med 1973, 289: 1293-1296.

Congenital Skull and Scalp Defect

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Congenital defects of bone and scalp are rare abnormalities. Scalp defects are more common than bony defects. Recently we came across a neonate with bone defect

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