—empyema, lung abscesses, and even myocardial abscess.[2-4] Nevertheless, generalised septicaemia is rare.

The cause of pyomyositis remains a mystery. Trauma, various parasitic and viral infections, and nutritional deficiencies have all been suggested as antecedents. None of these possibilities has stood up to close scrutiny, and there is still no plausible unitary theory of aetiology. Two features that such a theory would have to explain are the pronounced male dominance in reported cases and the frequent concurrent eosinophilia.

There is no specific method of making the diagnosis before suppuration. In the early stages management is based on a presumptive diagnosis made from the clinical features. In view of the almost universal role of *Staph aureus*, the optimum treatment is a β-lactamase-resistant penicillin in large doses.[5] If suppuration has occurred and pus is obtained on aspiration, then drainage must be assured by an adequate surgical incision. Pus should always be sent to the laboratory for culture and determination of antibiotic sensitivities of the organism. Because of the occasional occurrence of pyogenic lesions in the chest a chest radiograph is essential.

Though pyomyositis may destroy a large volume of muscle, functional recovery is usually good. Furthermore, cosmetic defects are unusual: the cavity left by the destroyed muscle apparently soon becomes filled in, presumably because adjacent muscle hypertrophies to occupy the space.

[1] Chiedozi, L C, *American Journal of Surgery*, 1979, **137**, 255.
[2] Sayers, E G, *Transactions of the Royal Society of Tropical Medicine and Hygiene*, 1930, **23**, 385.
[3] Taylor, J F, *et al*, *African Journal of Medical Science*, 1973, **4**, 409.
[4] Taylor, J F, Templeton, A C, and Henderson, B H, *East African Medical Journal*, 1970, **47**, 493.
[5] Buttner, D W, and Westhoff, H, *East African Medical Journal*, 1973, **50**, 74.

Six-month-old persistent vomiters

Nearly all infants bring some milk up after feeds, often with a belch, but some do it more than others. Factors which may persuade a mother to seek medical advice include her faith in her doctor, belief in the magic of medicine, normal anxiety, and her threshold of tolerance for the constant soiling of the baby's clothes and the ever-present sour smell. Her doctor has to decide whether the vomiting is within normal limits. He will be guided by the baby's weight and weight gain: there is less likely to be organic disease if the weight is normal in relation to the birth weight, the duration of gestation, and the build of the parents. If the weight gain is defective, the family doctor should be able to suspect or rule out the possibility of underfeeding and child abuse from his knowledge of the family.

More than anything else diagnosis will depend on the history. The vomiting may be due to giving the baby solid foods (such as raw apple, biscuits, or rusks) before he can chew[1] —average age 6 to 7 months, but later in a backward child. The baby may be ruminating—and this is diagnosed by the history and by observation. The baby, usually aged between 4 and 9 months, arches his back, contracts his abdominal muscles, and by rhythmical movements of the tongue, jaw, and pharynx brings up milk; some escapes from the mouth; with the remainder he may seem to gargle, the milk disappearing and reappearing in his throat, with an apparent feeling of satisfaction. He may not perform the act when asleep, when watched, or when he is distracted or interested in something. He may hasten the regurgitation by inserting his fingers into the back of the throat. The child may present as "failure to thrive," and rarely there is serious loss of weight; only when a careful history is taken will the diagnosis be made.

In 1907 Brockbank,[2] reviewing old stories of ruminators with horns growing from the forehead, suggested that psychological stress was a factor: many others subsequently ascribed rumination to emotional deprivation,[3-7] and there have been reports of favourable response to family psychotherapy[8 9] and aversive conditioning with an electric stimulation belt.[10] Fleisher[9] described an 8-month-old infant who before the diagnosis of rumination had been subjected to an *x*-ray examination of the skull, encephalogram, three barium meals, oesophageal manometry, endoscopy, treatment with propantheline, thickened feeds, soya milk, postural and other restraints, and threatened pyloroplasty or gastrojejunostomy—all causing a long, painful, costly delay in management. Treatment directed to reduction of emotional deprivation slowly but successfully cured the child.

Nevertheless, many paediatricians doubt whether all cases of rumination can be explained by psychological factors. The editor of the *1959-60 Year Book of Pediatrics*[11] commented, "We are not satisfied that the condition is due to emotional instability on the part of the mother. What do we know about the gastrointestinal tracts and the central nervous system of these patients?" At least some, probably many, ruminating infants have oesophageal reflux. Herbst *et al*[12], after describing three examples of ruminators with hiatus hernia, wrote that "although rumination has sometimes been considered a manifestation of psychiatric illness, the clinical features, anatomic and functional abnormalities, and therapeutic regimens advocated in the literature indicate that oesophageal disease, especially hiatus hernia, is the basic cause of the syndrome." Certainly the association is common: presumably the reflux makes it easier for the child to regurgitate and then, perhaps, emotional deprivation may somehow encourage rumination.

Hiatus hernia is often difficult to diagnose with certainty, but what is more important is the presence of reflux. Nevertheless, neither reflux nor emotional deprivation may be found in some ruminators, and there may be other factors. The rumination syndrome has been found, for example, in biotin-responsive propionic acidaemia.[13]

Persistent vomiting may be due to reflux or hiatus hernia without rumination. Symptoms may begin late (at several years of age) as a result of the development of oesophagitis and ulceration. The prime symptom which should alert the physician to the possibility of reflux with ulceration—and a symptom which should always be sought by direct questioning—is blood in the vomit. Occasionally reflux presents as a persistent cough, owing to inhalation of regurgitated material, causing patchy pulmonary consolidation.[14] Only on careful questioning will the persistent vomiting become apparent. Hiatus hernia occurs more often in children with cerebral palsy[15] and was found in 15 out of 20 persistent vomiters among 136 severely mentally defective children.[16] Hiatus hernia is sometimes found in association with abnormal head and neck movements, notably opisthotonos (Sandifer's syndrome).[17] When there is clear evidence of oesophagitis due to reflux, surgery may be indicated if the symptoms cannot be controlled by medical treatment, if the child is failing to thrive, or if there is a stricture.

Persistent vomiting should also suggest the possibility of

oesophageal or duodenal stenosis, especially if the child has Down's syndrome or some congenital anomaly of the alimentary tract. A small tracheo-oesophageal fistula may pass undiagnosed for several months; when a stomach tube is passed, with the proximal end under water, and the tube is slowly withdrawn up the oesophagus, bubbles may reveal the presence of the fistula. Sometimes coeliac disease presents as vomiting. Other remote possibilities are allergy to cows' milk or increased intracranial pressure. Finally a child may be brought to the doctor with vomiting when the history indicates that he vomits only when he has a spasm of coughing, and the cause is whooping cough.

The good clinician makes his diagnosis by a careful, detailed history, by observation and examination of the child, and then by sitting down and thinking—before deciding whether any investigations are needed. His decisions are based on clinical judgment, and he should be prepared to make a mistake.

[1] Illingworth, R S, and Lister, J, *Journal of Pediatrics*, 1964, **65,** 839.
[2] Brockbank, E M, *British Medical Journal*,1907, **1,** 421.
[3] Cameron, H C, *British Medical Journal*, 1925, **1,** 872.
[4] Richmond, J B, and Eddy, E J, *American Journal of Diseases of Children*, 1957, **94,** 412.
[5] Richmond, J B, Eddy, E J, and Green, M, *Pediatrics*, 1958, **22,** 49.
[6] Fullerton, D T, *Archives of General Psychiatry*, 1963, **9,** 593.
[7] Menking, M, *et al*, *New England Journal of Medicine*, 1969, **280,** 802.
[8] Stein, M L, Rausen, A R, and Blau, A, *Journal of the American Medical Association*, 1959, **171,** 2309.
[9] Fleisher, D R, *American Journal of Diseases of Children*, 1979, **133,** 266.
[10] Luckey, R E, Watson, C M, and Musick, J K, *American Journal of Mental Deficiency*, 1968, **73,** 139.
[11] *1959-1960 Year Book of Pediatrics*, p 421. Chicago, Year Book Medical Publishers Inc, 1961.
[12] Herbst, J, Friedland, G W, and Zboralske, F F, *Journal of Pediatrics*, 1971, **78,** 261.
[13] Hillman, R E, Keating, J P, and Williams, J C, *Journal of Pediatrics*, 1978, **92,** 439.
[14] Christie, D L, O'Grady, L R, and Mack, D V, *Journal of Pediatrics*, 1978, **93,** 23.
[15] Abrahams, P, and Burkitt, B F E, *Australian Paediatric Journal*, 1970, **6,** 41.
[16] Sondheimer, J M, and Morris, B A, *Journal of Pediatrics*, 1979, **94,** 710.
[17] Kinsbourne, M, *Lancet*, 1964, **1,** 1058.

Cubital tunnel syndrome

In contrast to the many articles published on the carpal tunnel syndrome, little information is available about the cubital tunnel syndrome—ulnar nerve compression at the elbow. Panas[1] elegantly described four cases in 1878; one patient had his elbow explored and subsequently died of wound infection. After this surgical interest lagged, apart from occasional case reports, until Feindal and Stratford[2] described compression of the nerve by a fibrous aponeurosis stretching between the humeral and ulnar heads of flexor carpi ulnaris. Like Osborne[3] and Clark[4] they favoured simple decompression rather than the more elaborate process of transposing the nerve. Diagnosis of earlier, milder cases more amenable to treatment became possible with the advent of electrodiagnosis.[5]

The factors that may play a part in causing the condition include decubitus pressure during anaesthesia, occupational pressures, sleep, bed rest, and unpadded elbow crutches. Chronic deformity of the elbow joint may cause recurrent dislocation of the nerve from the sulcus, or the nerve may be damaged by acute trauma. Ulnar nerves impaired by diabetes, alcoholism, leprosy, or hereditary neuropathy are more susceptible to entrapment pressure. Ischaemic mononeuropathy may occur at the elbow as a result of subacute vasculitis of the vasa nervorum in rheumatoid arthritis and polyarteritis nodosa. Yet despite this wide range of predisposing causes none can be identified in about one-third of cases.

Most patients present with both motor and sensory symptoms. When the fibres compressed are mainly motor appreciable weakness may develop before the patient seeks help—whereas the predominantly sensory symptoms in median nerve compression usually prompt earlier referral. Typically, there is slight clawing of the fourth and fifth fingers, impaired sensation over the fifth finger and the ulnar side of the fourth, and weakness and wasting of hypothenar and interosseous muscles. Because of weakness of adductor pollicis adduction of the thumb can be achieved only through flexor pollicis longus, causing flexion of the thumb tip (Froment's sign). There may be weakness of ulnar deviation of the wrist (flexor carpi ulnaris) and flexion of the tips of the fourth and fifth fingers (flexor digitorum profundus), which confirms that the lesion is at or above the elbow.

Payan[6 7] has studied the electrodiagnosis of ulnar nerve compression and its response to transposition. The segment of the nerve that traverses the cubital tunnel is subjected to standard tests of motor and sensory conduction and compared with other segments of the same nerve and of the other ulnar nerve. The most common finding is slowing of motor and sensory conduction across the sulcus. Prolonged latency and increased temporal dispersion of the response evoked in flexor carpi ulnaris is a useful sign in severe cases, where fibres to the hand may have ceased to conduct. The sensory volleys recorded from the fifth finger will be appreciably reduced compared with those from the second finger. Differential diagnosis needs to distinguish intravertebral compression of the eighth cervical nerve, which produces weakness and denervation of the thumb extensor and abductor muscles. Since the root lesion is proximal to the sensory ganglion the ulnar sensory action potentials are normal. Compression of the first thoracic root in the thoracic outlet usually produces signs of denervation of the thenar muscles and the motor conduction studies usually give normal results.

Several points about management are worth emphasising. When the nerve damage is due to closed trauma there may be a predominant neurapraxia from local demyelination of the conducting fibres; spontaneous recovery may be expected in several months. More severe damage causes degeneration (axonotmesis) of fibres, attenuation of conduction, and signs of denervation in the muscles supplied. Since reinnervation proceeds at an average rate of 1 mm a day it will take at least a year to reach the hand. Serial electrodiagnostic studies will help to show whether recovery is occurring and, if not, whether the nerve needs decompression. Finally, all concerned need to remember that no recovery can be expected in the hand until over a year after transposition for chronic compression.

[1] Panas, P, *Archives Générales de Médecine*, 1878, **2,** 5.
[2] Feindal, W, and Stratford, J, *Canadian Journal of Surgery*, 1958, **1,** 287.
[3] Osborne, G V, *Postgraduate Medical Journal*, 1959, **35,** 392.
[4] Clark, C B, *Journal of the American Medical Association*, 1979, **241,** 801.
[5] Simpson, J A, *Journal of Neurology, Neurosurgery, and Psychiatry*, 1956, **19,** 275.
[6] Payan, J, *Journal of Neurology, Neurosurgery, and Psychiatry*, 1969, **32,** 208.
[7] Payan, J, *Journal of Neurology, Neurosurgery, and Psychiatry*, 1970, **33,** 137.