full carafe of burgundy. Further haemorrhage may cause a break-through of the hyaloid membrane, to produce a vitreous haemorrhage.

To avoid this serious complication, a slow rate of injection and limitation of the amount injected are obvious precautions. The development of a headache, due perhaps to the rise of intracranial pressure during the procedure, gives warning that the speed of injection needs slowing, but this guide is not available when a general anaesthetic is used. Further tests are necessary to determine how long the raised C.S.F. pressure lasts, but if this is not long, gentle pressure on the eyes through

the closed lids at the time of the injection would raise the intraocular pressure and help to support the distended retinal vessels.

Summary

Two cases of retinal haemorrhage apparently caused by epidural injection of saline are recorded, and it is concluded that they were produced by a sudden rise in cerebrospinal pressure causing acute retinal venous congestion.

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MUMPS ARTHRITIS

BY

RONALD LASS, M.R.C.P.

Consultant Physician

AND

EDMUND SHEPHARD, F.R.C.S.

Consultant Orthopaedic Surgeon

West Kent General Hospital, Maidstone, Kent

Though the occurrence of arthritis in epidemic parotitis is referred to in textbooks of joint disease (Hollander, 1960), paediatrics (Abt, 1925; Griffith and Mitchell, 1941), and infectious disease (Wesselhoft, 1951; Stimson and Hodes, 1956), the condition seems to deserve wider recognition.

Maisondieu (1924) credited Rilliet with first recording this condition in 1850, added 6 personal cases, and reviewed 12 others. Apart from one other case mentioned by Wesselhoft (1951), who alone quotes Maisondieu's paper, we have found reports of only eight additional cases (Wilson, 1926; Magida, 1951; Applebaum *et al.*, 1952; Senise *et al.*, 1957), bringing the total to 28 cases, of which four were in females.

Maisondieu's six cases of arthritis occurred among 1,334 cases of mumps, giving an incidence of 0.44%.

This paper presents three further cases of arthritis complicating mumps.

Case Reports

Case 1.—A boy aged 5 years suffered severe transient pain in the right ankle 12 days after the onset of parotitis. Two days later the left ankle became painful and he began to limp. This ankle was swollen, warm, and tender over the posterior and lateral aspect. Movement was limited by severe pain. The temperature was 100.6° F. (38.1° C.). Radiographs of both ankles were normal. The E.S.R. was 50 mm. in one hour (Westergren). The white-cell count was 8,100/c.mm., with normal differential count. The antistreptolysin O titre was normal. The titre to mumps virus was 1/128 to V and 1/64 to S antigen. Within three days all pain and swelling had subsided.

Case 2.—A boy aged 5 years complained of severe pain in the left thigh, made worse by coughing, two weeks after the onset of parotitis. There was tenderness in the left groin with severe muscle spasm on attempted movement of the left hip. The temperature was 100.4° F. (38° C.), and fever continued for seven days. A diagnosis of arthritis of the left hip was made and penicillin was given. Salicylates were administered, but there was no immediate relief of pain or resolution of fever. The total white-cell count on the second day after onset of hip pain was 18,000/c.mm., and on the sixth day 7,000/c.mm., with normal distribution. On the third day abdominal pain and vomiting suggested the possibility of appendicitis, but these symptoms subsided without operation. The abdominal symptoms may have been due to mumps pancreatitis, especially as tenderness

persisted in the left hypochondrium for a few more days. The patient was discharged on the fourteenth day free from pain and with full hip movement.

Case 3.—A man aged 43 suffered bilateral parotitis at the same time as one of his sons and two weeks after another son had been affected. Two weeks after the onset of parotitis he developed fever, nausea, and headache and complained of pain and swelling of the left ankle, which confined him to bed. Two days later the left ankle showed slight residual swelling and tenderness. The patient then developed a severe left orchitis. The white-cell count was 14,000/c.mm. (polymorphonuclear leucocytes 89%). The E.S.R. was 34 mm. in one hour (Westergren). The mumps titre was less than 1/4 to V and less than 1/16 to S antigen. Two weeks later it had risen to diagnostic level, being 1/64 to V antigen. The joint recovered fully.

Discussion

In the reported cases arthritis showed a male predominance of seven to one. Other complications of mumps also show a predilection for the male, notably mumps paralysis (Lennette *et al.*, 1960), in which all 11 patients were boys. The age incidence was 11 to 47 years. Onset was from 8 days before to 29 days after the parotitis, and the duration was between two days and two months. One or many joints were involved and recovery was complete.

When performed blood sedimentation rates were found to be increased and complement-fixation or viral-agglutination titres were diagnostic. Usually a polymorphonuclear leucocytosis was found.

Mumps virus has been recovered from the saliva and urine (Utz *et al.*, 1957), thyroid (Eylan *et al.*, 1957), and cerebrospinal fluid (Lavergne *et al.*, 1937), and was found in the blood by Woolstein (1918). We can find no reference to isolation from the joint. Nevertheless, the arthritis may be due to viral infection of the joint, for it occurs commonly at the same time as other complications in which the virus has been recovered from the affected organ or system. It is our intention, when next we meet this complication, to attempt to grow virus from the joint.

Summary and Conclusions

Three cases of arthritis complicating mumps are reported, and the literature is reviewed.

If mumps were considered in the differential diagnosis of arthritis in the young, especially in males, we suspect that more cases would be recognized.

Numerous cases of infection with mumps virus have been recorded without parotitis; among these are pancreatitis (Friedjung, 1928), orchitis (Connolly, 1953), and meningitis (Van Rooyen and Rhodes, 1948), the diagnosis being made on the basis of a significant rise in agglutination titres. We can find no reference to arthritis sine parotitis in the literature, but suggest that estimation of viral titres and attempts to grow virus from an affected joint may perhaps lead to recognition of this condition.

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Preliminary Communications

Achalasia of the Oesophagus in Children

Achalasia of the oesophagus is seen and well recognized in adults but is rare in children. There may be failure to diagnose it in some children, for the symptoms can be confusing. A case history is presented of achalasia in a baby operated on at the age of 6 months. I had not previously seen this condition in patients under 17 years. Clagett (1961) stated that during 1936-59, 32 patients aged 15 years and younger with achalasia were seen at the Mayo Clinic; the youngest was under 1 year, and definite symptoms were present in 10 patients before they had reached 1 year of age. Swenson and Oeconomopoulos (1961) reported six cases in children, none of them being under 1 year of age.

CASE RECORD

A male infant of Maltese nationality was admitted to Westminster Children's Hospital on April 26, 1960, with the following history. He was born on September 14, 1959, after eight hours' labour, and was the first child of a father aged 25 and mother aged 23. The parents suffered no illnesses during the pregnancy. For vomiting of pregnancy

after the first month the mother received 16 tablets of ancotoxin. Birth weight was 8 lb. 10 oz. (3.9 kg.). On the day after birth he was inadvertently given two teaspoonfuls of hot water, the mother being unaware of its temperature. At his baptism on September 27 he swallowed well coarse grains of local salt and was breast-fed up to 19 days. On October 3 the mother developed mastitis and the child started feeds of Cow & Gate half-cream milk. After taking the hot water when 1 day old, the child would not feed from the breast, and the hospital doctor declared, "He does not know how to swallow," but this phase passed after two days and no importance was attached to it. On swallowing tepid water subsequently the child would gulp down the liquid with gurgling noises.

During the second week in December he first choked while asleep in his pram a short time after a milk feed. He was thriving normally but started to regurgitate part of his feed after a few minutes. Milk of magnesia and tincture of belladonna were given. On December 24 he started regurgitating yellowish-white fluid. On December 28 he was examined by a throat consultant who found no abnormality down to the pharynx and advised precaution against milk and phlegm ingestion into the bronchi. On January 3, 1960, the infant developed fever, and bronchopneumonia with dehydration was diagnosed and antibiotic given. He was admitted to hospital. Deglutition became worse, and bottle-feeding continued until January 12, when a "polythene" tube was passed, but a radiological examination showed this to be in the lower end of the oesophagus. On January 21 the tube was passed into the stomach.

Before bronchopneumonia developed "nestargel" was given on the supposition that the cardia was too relaxed, but this aggravated the dysphagia. Atropine methonitrate ("eumydrin") and all other medical treatment failed to give relief. The tentative diagnosis of achalasia was made on x-ray examination with propyl iodine ("dionosil").

On examination the child was happy and looked well; weight 16 lb. 15½ oz. (7.8 kg.). The temperature and the pulse and respiration rates were normal. No abnormality was detected in the heart, lungs, or abdomen.

Investigations.—Hb, 115%; W.B.C., 16,800/c.mm. Urine normal. Blood chemistry normal. Blood group O, Rh positive. Stool cultures negative. Radiological examination of the oesophagus (Fig. 1) with a barium swallow showed that the oesophagus was widely dilated throughout its extent and its lower end tapered to a point at the junction with the cardia, forming a short smooth stricture which relaxed at intervals allowing barium to enter the stomach. Some of the barium spilled over from the oesophagus into the bronchi, which are outlined in the x-ray film. The appearances are characteristic of achalasia of the oesophagus. The child was transferred to the country hospital to prepare him for operative treatment. He was readmitted on March 11, 1960.

Operation.—On March 17, 1960, Heller's operation was performed through a left supra-umbilical paramedian incision. The abdominal segment of the oesophagus was identified, and the peritoneum was incised over it so that it could be mobilized into the oesophageal hiatus of the diaphragm. The vagus nerves were carefully safeguarded from injury. An incision 4 cm. long was made through the hypertrophied muscle of the lower end of the oesophagus and extended through the seromuscular layers of the stomach as far as the fundus. All the muscle fibres were carefully divided throughout this incision until the mucosa of the oesophagus and stomach bulged through the divided muscle-coats. Fig. 2 shows the oesophagus after the operation.

Post-operative Course.—The child made an uninterrupted recovery from the operation. On the day after operation he took all his feeds by mouth without any difficulty or regurgitation. There was no further regurgitation or vomiting and he was discharged from hospital on April 14. His weight was then 17 lb. 11 oz. (8 kg.).

Subsequent Course.—The child returned to Malta on April 15 and migrated with his parents to Australia in