born baby is manipulated and strapped at weekly intervals for three weeks. If the shape is then satisfactory and overcorrection has been obtained, this method is continued and it is usually found that only a few weeks of further strapping is needed. But if at three weeks the shape or correction is unsatisfactory, operation is performed—and we feel it should not be delayed beyond the age of 4-6 weeks.

Through a medial incision the tendo achillis is elongated. The foot invertors and plantar flexors are then inspected and if any muscle is short and fibrous its tendon is elongated. In the few cases operated on before the age of six weeks we have so far not needed to elongate any structures other than the tendo achillis and tibialis posterior in order to obtain overcorrection without force; but in one case at 10 weeks the posterior capsule of the ankle also required division.

Following operation the foot is held overcorrected in plaster for six weeks, after which all splintage is discarded. The results so far have been extremely encouraging and the method has the obvious advantage that operation and splintage are over and done with long before the child is beginning to stand or walk.

With further experience we may well find that even in the early cases elongation of only the tendo achillis and tibialis posterior is not always sufficient. Attenborough (1966), for example, in his excellent account of the early treatment also implicates other structures. Although he does not advocate operation until the age of 6-9 weeks he has demonstrated contracture of the long toe

flexors and of the posterior capsule of the ankle joint in dissections of still-born infants. Denham (1966), another advocate of early operation, elongates the tendo achillis and divides the plantar fascia, on the assumption that contracture of these structures (which constitute a single morphological sheet interrupted by the os calcis) is the primary fault. Other writers (e.g. Irani and Sherman, 1963) have maintained that the primary anomaly is in the talus itself.

There may well be different varieties of resistant talipes equino-varus and, although the invertors and plantar flexors are undoubtedly of major importance, it would be unwise in the present state of our knowledge to assume that they alone are at fault. In any event there is no need for fixed pre-conceived notions of pathology—the dissection should proceed until overcorrection is obtained without force. The important point is to operate early, at a stage when simple soft-tissue procedures should prove sufficient and offer the best hope of restoring the foot to normal shape and suppleness.

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MUMPS

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Aetiology

The virus of mumps is readily isolated by inoculation of the yolk sac or amniotic cavity of the chick embryo but tissue culture, using monkey kidney, human amnion or HeLa carcinoma cell lines is more sensitive. Mumps virus varies in size from 90-135 m μ . It is composed of RNA and has an internal helical structure characteristic of the myxovirus group to which it belongs.

Mumps virus has been isolated from saliva, urine and cerebrospinal fluid for six days before the onset of symptoms and for thirteen days after. In children with sub-clinical infections, the virus has been isolated during the corresponding period

after exposure. Isolations have been made from the blood on the first day of illness. During the later stages of mumps, the virus is most readily cultured from urine.

Epidemiology

Mumps is endemic in most urban communities throughout the world. Epidemics appear at irregular intervals of 7-8 years and are often associated with overcrowding. The main incidence of clinical mumps occurs in the age groups 5-15 years. By adult life, 60% give a history of having had mumps. Serological tests, however, show a much greater incidence of mumps so it would

appear that 30-40% of infections produce a subclinical attack. Maternal antibodies can be demonstrated in cord blood. Although these antibodies disappear within 40-60 days, mumps is rare in babies under 9 months.

Mumps is only moderately infectious and fairly close contact is required for spread. Man is the sole reservoir of infection. The virus is spread by the inhalation of droplets of infected saliva or by oral contact with articles contaminated by saliva. The virus enters through the upper respiratory passages where it probably multiplies before being disseminated by the blood stream. Further multiplication takes place in the tissues and virus is excreted in the saliva and urine. There is no evidence that infected urine is important in the transmission of mumps. The subclinical case, excreting virus in saliva, is an essential link in the chain of infection and the large number of inapparent infections explains why the isolation of frank cases fails to curtail an epidemic. Second attacks of mumps have been confirmed but are rare.

Pathology

There is oedema of the salivary glands and surrounding tissues associated with focal degeneration of the acinus cells. Punctate haemorrhages are present and the interstitial tissue is infiltrated by mononuclear cells. During recovery, the debris is removed and normal structure is restored. In the testes, similar changes may be found. Little is known about the pathology of mumps encephalitis.

Clinical Course

Laboratory tests have shown how very limited is the concept of mumps as a disease primarily affecting salivary glands with complications involving other organs. It is now realised that mumps is a generalised virus infection with a variety of clinical themes. While there is a predilection for the salivary glands, inflammation of the testes, pancreas or central nervous system may be the sole manifestation. Occasionally, other structures may be involved. These include the ovary, prostate, breast, heart, joints, eye and ear. Since any combination of clinical signs may appear, it is proposed to discuss each organ separately.

Incubation Period

The incubation period usually lies between 18-21 days but may extend from 12-35 days.

Salivary Glands

Mumps commonly presents with parotitis. In 70% of cases, both parotid glands are involved. Swelling of the gland is frequently associated with oedema of the surrounding tissues and in some patients, particularly negroes, a jelly-like quivering is produced by tapping the side of the face. A helpful early sign is redness of the parotid papilla. Pain is very variable, and is often absent in

children. The parotitis reaches a peak within 48 hours, then gradually subsides, returning to normal within 10 days. In 10% of patients, the submaxillary glands are involved. Very rarely the sublingual glands are affected.

Other viruses may produce parotitis. Howlett, Somlo and Kalz (1957) isolated Coxsackie A virus from the throats of four patients with bilateral parotitis and herpangina but serum was not examined for antibodies.

Reproductive System

Testes—Orchitis is found in 25% of men and adolescent boys with mumps but is very rare before puberty. In 83% of cases, only one testis is involved. Although usually associated with parotitis, orchitis may be the sole manifestation of mumps. In unilateral orchitis, sperm counts sometimes show some impairment of fertility but sterility can only occur following severe bilateral orchitis and is a rare sequel. Orchitis caused by other viruses such as Coxsackie B and ECHO 9 can only be distinguished by laboratory investigations.

Epididymis—Epididymitis is usually associated with orchitis but may occur independently and is then readily mistaken for mild orchitis.

Prostate—Recurrence of fever or exacerbation of symptoms in an adult suffering from orchitis may be due to the onset of prostatitis. The condition is readily overlooked but on rectal examination the prostate will be found to be swollen and extremely tender. There is no urethral discharge.

Ovary—The ovary is involved in 5% of women with mumps. In severe oophoritis with high fever and large tender ovaries the diagnosis is obvious but mild attacks are easily missed. As a rule, there is no permanent disturbance of menstruation and no apparent effect on fertility. Mumps during pregnancy does not damage the foetus.

Breasts—Mastitis, unlike oophoritis and orchitis, may occur at any age and may affect both sexes. The illness is benign and recovery complete.

Pancreas—Pancreatitis occurs in 7% of cases and is usually associated with parotitis. Although the serum amylase is increased in pancreatitis, this test is not diagnostic for a moderate rise is commonly found in parotid mumps. As a rule, recovery is complete.

Nervous System

Meningo-encephalitis is a benign manifestation of mumps found in $\frac{1}{2}$ -10% of cases. It may be the sole feature or may develop during any stage of parotid mumps. When meningo-encephalitis is the only manifestation of mumps, it cannot be differentiated clinically from other forms of virus meningitis. The acute symptoms subside within a few days and recovery is usually complete.

Encephalo-myelitis — Encephalitis, developing suddenly during the second week of an attack of mumps, is a more sinister event similar to the post infectious encephalo-myelitis following other

virus diseases such as measles and rubella. This form of encephalo-myelitis may produce deep coma with evidence of focal damage to the brain and cord. The death rate is considerable and survivors may have permanent damage to the central nervous system.

Ear—Damage to the organ of Corti may appear during parotitis or encephalitis. In the majority of cases, deafness is permanent.

Heart

During the acute stage of mumps, abnormal electrocardiograms are found in 5-15% of cases. The cardiograms return to normal during convalescence.

Joints

Transient arthritis is found in 0.5% of cases.

Diagnosis

Isolation of Virus. During the first week of mumps, the virus can be isolated from saliva, cerebro-spinal fluid and urine but this laboratory investigation is not a routine procedure.

Serological Tests. The complement fixation test is the most reliable and most widely used of the serological tests for mumps. There are two components. The S antibody develops rapidly during the first fortnight, then gradually disappears and cannot be detected after six to twelve months. In contrast, the V antibody reaches a peak after one month, remains stationary for six months then slowly declines over the ensuing two years to a low but persistent level. In doubtful cases of mumps, a specimen of serum should be obtained as early as possible in the illness and a further specimen taken 2-3 weeks later. A raised S antibody level with a low V titre in the acute specimen followed by a fourfold rise in V antibody in the convalescent serum is strong evidence for an attack of mumps, with the qualification that antibodies may sometimes appear as a heterologous reaction to other infections.

Serum Amylase. The serum amylase level is raised in 70% of patients with mumps. The amylase is increased in the early stage, reaches a peak during the first week and returns to normal by the end of the second week. The test is not dependent on clinical involvement of the salivary glands or pancreas. It is a useful screening measure which may be conveniently performed on the acute specimen of serum. It should be supported later by complement fixation tests on paired sera.

The Blood.—The white cell count is very variable in mumps. It may be normal, may show a lymphocytosis or else a slight polymorph leucocytosis. The ESR is usually normal in parotid mumps but may rise when orchitis develops. Regular ESR estimations have been used to predict the onset of orchitis.

Treatment

Treatment for mumps is entirely symptomatic. Corticosteroids may be used for the severe forms of encephalomyelitis but the results are not spectacular. In orchitis, hydrocortisone intramuscularly 100 mg. six-hourly, or prednisone 10

mg./kg./day relieve symptoms but do not affect the course of the disease. The patient can be reassured that unilateral orchitis will not cause impotence or sterility.

Immunisation

There is no effective method of immunisation.

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