precaution to avoid provoking a possible reaction at a time when resistance is likely to be relatively low.

Summary

A case is reported of an 8-months-old normal child who, within 24 hours of a combined diphtheria–pertussis inoculation, became pyrexial, drowsy, and hypotonic. Convulsions and evidence of mental deterioration followed rapidly. At 3 years of age convulsions still persisted and mental retardation was gross (I.Q.=23).

Review is made of 107 cases of neurological complications of pertussis inoculation reported in the literature. Males predominated; reactions occurred in all age groups in which immunizations were done, irrespective of the dosage used, and followed first or subsequent inoculations. A wide variety of vaccines, single and combined, were implicated. Past personal and family histories indicated relatively few instances of neurological or allergic abnormalities. The early onset of neurological symptoms was characteristic, with changes of consciousness and convulsions as the most striking features. Pareses or paralyses were not infrequent, and eight children died within 48 hours of inoculation. Follow-up revealed a recovery rate of about 50%, a persisting morbidity rate (physical and/or mental) of about 30%, and a mortality rate of about 15%.

The question of aetiology is considered and contra-indications are discussed. The value of pertussis immunization is emphasized, as is the grave danger of further inoculations when a previous one has produced any suggestion of a neurological reaction.

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THE NEUROLOGICAL COMPLICATIONS OF MUMPS

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The neurological complications of mumps have been recognized since the eighteenth century, and a number of reports have appeared in the European and American literature describing various forms and the frequency with which they occur. Thus in 1911 Doptor reviewed 1,700 cases of mumps and estimated that 9.8% showed meningeal symptoms. Laurence and McGavin (1948) found 60 cases in an epidemic involving 208 New Zealand Service men; and Frankland (1941), in discussing an outbreak of 234 cases, put the incidence of meningitis as high as 30%. On the other hand, Paddock (1932) and Coe (1945) found neurological involvement in only 0.15–0.5% of their series, which were less selective and covered a wider age range.

It has been suggested that differences in frequency of meningitis in various epidemics are due to variations in resistance to infection, perhaps due to racial factors or to an altered virulence of the mumps virus leading to an increased affinity for nervous tissue. It is partly accounted for by the different criteria which have been used in the diagnosis of meningitis, some authors relying on symptoms, others on symptoms and physical signs, and some on the alterations in cerebrospinal fluid. To add to the confusion, it has been repeatedly confirmed, since Lavergne's observation in 1938, that cerebrospinal pleocytosis may occur in uncomplicated cases of mumps (Finkelstein, 1938) or even in contacts showing no signs of mumps (Frankland, 1941), and that meningeal symptoms may precede parotitis by as much as ten days (Bedingfield, 1927; Frankland, 1941).

These facts led Doptor and others to the view that the mumps virus is primarily neurotropic and that involvement of the parotid glands occurs as a secondary and not invariable complication. The existence of a separate neurotropic strain of the virus has not received much support (Macrae and Campbell, 1949), and Gordon (1927) was able to produce a fatal meningo-encephalitis in a monkey by intracerebral inoculation of material from throat-washings of uncomplicated cases. No correlation has been found between the occurrence of nervous complications and the severity of parotid disease or of epididymo-orchitis (Holden, Eagles, and Stevens, 1946).

Mumps meningitis is a remarkably benign condition usually appearing within a few days of the parotid enlargement and recovering without complications in three to four days; occasionally it may precede parotitis or occur alone. The cerebrospinal fluid contains a variable number of lymphocytes (10–1,000 per c.mm.) with slight increase in protein and normal values for chloride and glucose. Mumps virus has been isolated from the spinal fluid (Henle and McDougall, 1947).

The major neurological complications are much less common (approximately 1 in 6,000) and have been

reviewed by Miller, Stanton, and Gibbons (1956). They may occur at the time of parotitis or up to 23 days afterwards, the average latent period being 7.2 days. Encephalitis cases are less rare than the other varieties —myelitis, polyradiculitis, and polyneuritis. Mental symptoms are prominent in encephalitis, and clinical features include drowsiness, convulsions, headache, psychoses, ataxia, and hemiplegia. Neck stiffness, involuntary movements, increased reflexes, and extensor plantar responses may be found. Deep coma may occur, and is an unfavourable prognostic sign. The cerebrospinal fluid often shows a lymphocytosis, but may be normal. Death occurs in approximately 20% of cases, and 33% of those who recover may show major sequelae consisting of hemiplegia, ataxia, and involuntary movements.

Fatal cases show a patchy perivenular demyelination similar to that seen following other post-infective encephalitides (Donohue, Playfair, and Whitaker, 1955).

Cranial nerve palsies involving the optic, facial, trigeminal, or oculomotor nerves may occur (Laurence and McGavin, 1948 ; Macrae and Campbell, 1949), and the important complication of labyrinthitis may be permanent and unassociated with signs of neurological damage. Local weakness of the limbs with transient loss of tendon reflexes and subjective sensory disturbance has been described (Lightwood, 1946 ; Laurence and McGavin, 1948).

In the following report 18 cases of meningitis and one of encephalitis occurring in the course of a mumps epidemic are described.

Incidence

During February–October, 1956, there was an epidemic of mumps in Gurkha troops stationed at Sungei Patani, Malaya. A total of 287 cases occurred, and 84% of the patients came from " Recruit Company " and " Boys' Company," aged 14–17. A number of those infected had arrived from widely scattered areas in Nepal two months previously.

The infection reached a peak in June, when there were 58 cases.

In the great majority the disease ran a benign course over a few days, but in the latter part of the epidemic meningitis began to appear, reaching a maximum in July, when there were 12 cases, one in every five being affected.

Thirteen cases had salivary glandular enlargement and meningitis ; one had parotitis, orchitis, and meningitis ; one had orchitis and meningitis ; and three had meningitis alone.

TABLE I.—*Onset of Meningitis Related to Appearance of Parotitis*

	Days Before		Appearance of Parotitis	Days After									
	2	1	0	1	2	3	4	5	6	7	8	9	10
Cases of meningitis ..	1			1	1	2	4	1	1		2		1

The occurrence of these events is shown in Table I ; it will be seen that in one case the meningitis preceded the parotitis by two days.

In the three cases presenting with meningitis alone there was no opportunity of confirming the diagnosis by serological means as described by Kilham, Levens, and Enders (1949), but, in view of their similar benign course and the presence of an epidemic, it was assumed that they were variants of the same malady.

The incidence of all complications is set out in Table II, and it will be noted that, in contrast to meningitis, orchitis occurred in sporadic fashion evenly distributed throughout the epidemic.

TABLE II.—*Incidence of Complications*

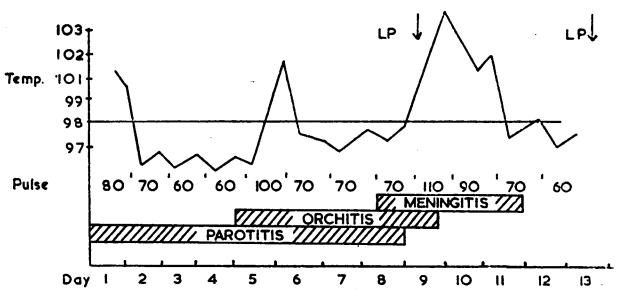
	Feb.	Mar.	Apr.	May	June	July	Aug.	Sept.	Total
Cases admitted	13	39	29	41	58	51	35	21	287
Uncomplicated	11	33	24	31	50	32	23	15	219
Orchitis ..	2	6	5	10	6	9	7	3	48
Meningitis ..					2	12	3	1	18
Encephalitis							1		1
Percentage incidence of meningitis					3·4	23·5	8·6	5·0	6·3

Clinical Features

Meningitis (18 cases)

The patients reached hospital on the first or second day following the appearance of parotitis. They were usually pyrexial, but in good general condition and reluctant to be confined to bed.

The pyrexia settled in 48 hours, but with the onset of meningitis it recurred (see Chart), and the cheerful swollen



Case 14. Boy aged 15. Temperature and pulse rate during illness.

faces were transformed overnight by expressions of extreme misery. The patients lay curled up in bed, complaining of severe headache and photophobia and refusing food and drink with such persistence that some had to be given fluids by tube. Vomiting occurred frequently and some had pain in the muscles of the legs.

On examination the most prominent finding was rigidity of the spine, which moved " in one piece " if an attempt was made to flex the neck. Kernig's sign was positive and there was slight muscle tenderness in the calves but no other objective neurological signs. Nystagmus, which had been reported in this condition, was not seen, and tendon reflexes were unaltered.

All the patients were irritable when disturbed and resented examination, but when induced to talk they seemed rational. They tended to spend a large part of the day asleep, but there was no nocturnal restlessness. The headache responded to aspirin in large doses, and appeared to be somewhat relieved by lumbar puncture.

The urine often contained ketone bodies but no albumin, and white-cell estimations showed values of 8,000–12,000 per c.mm., with a normal differential count. Recovery was rapid and complete in two to three days, and neurological examination four months later was negative. Two boys were thought by their instructors to be slower mentally after the illness, but apart from this equivocal finding there were no sequelae.

Cerebrospinal fluid was examined in all cases, and was usually repeated after 10 days. The results are summarized in Table III.

The cellular reaction was predominantly lymphocytic, although up to 60% of polymorphs was not unusual in the early stages. Estimations done later in the disease invariably

TABLE III

Case No.	Day of Disease	Fluid	Cells /c. mm.	Lymphocytes %	Polymorphs %	Protein mg./100 ml.	Chlorides mg./100 ml.	Sugar mg./100 ml.	Remarks
1	3	Turbid	230	60	40	48	750	57	Parotitis, meningitis
	9	,,	340	90	10	55	730	56	
	20	Clear	60	70	30	37			
2	5	,,	4	50	50	97	720	50	,,
	8	Turbid	140	35	65	95	690	52	
3	6	,,	940	50	50	240	690	55	,,
	16	Clear	2			37	760		
4	5	,,	6			49		55	,,
	12	,,	10	30	70	64	730		
5	3	Turbid	780	95	5	60	760		,,
6	7	,,	430	84	16	62	715	54	,,
		,,	60	80	20	47	740	45	
7	11	,,	930	80	20	95	670	69	,,
	17	Clear	Nil			49			
8	2	Turbid	460	32	68	94	760	45	Meningitis preceded parotitis
9	4	,,	130	38	62	25	730	41	Parotitis, meningitis
	16	Clear	1			17			
10	3	Turbid	1,260	70	30	110	740	68	,,
	14	Clear	3			47	670	46	
11	6	Turbid	86	70	30	80	720	82	,,
	14	Clear	9	88	12	58	740	64	
12	6	Turbid	340	90	10	60	750	36	,,
	13	Clear	80				710	58	
13	9	Turbid	370	90	10	70	730	65	,,
	20	Clear	30	90	10	25			
14	9	Turbid	480	90	10	61	710	34	Parotitis, meningitis, orchitis
	14	Clear	7	56	44	30	700	49	
15	5	Turbid	720	98	2	72	660	39	Orchitis, meningitis
16	3	Clear	80	28	72	100	720	50	Meningitis alone
17	4	Turbid	250	96	4	90	700	48	,,
		Clear	6			50			
18	2	Turbid	400	60	40	75	750		,,
	7	Clear	29	75	25	210	730	45	
19	11	,,	1			20			Parotitis, encephalitis

showed a preponderance of lymphocytes. Total counts in the acute stage varied between 200 and 800 cells per c.mm., falling rapidly in a few days, but in some cases being still significantly raised at the end of two weeks, when patients were asymptomatic and seemed fit for discharge. The protein was raised in proportion, with a usual reading at 60–100 mg. per 100 ml., and chloride and sugar estimations showed no significant abnormality. Six uncomplicated cases of mumps from the same epidemic and at the same time were subjected to lumbar puncture. Results were normal in every respect.

Encephalitis (1 case)

A previously healthy boy aged 14 with no past or family history of psychiatric disturbance was admitted with swelling of parotid and submandibular glands present for two days. He was slightly pyrexial on admission—temperature 99.4° F. (37.4° C.).

The swelling subsided over the first week, but on the ninth day he began to vomit and refused all nourishment. Over the course of 24 hours he became speechless and extremely drowsy, with episodes of mania when disturbed. In these he struggled violently with the attendants, grinding his teeth and biting anyone within reach, although never speaking a word. Headache was not prominent, but he appeared to have some abdominal pain. Temperature and pulse were normal. He was restless at night and slept fitfully during the day. He developed retention of urine, requiring catheterization.

Satisfactory examination of the nervous system was well-nigh impossible, but there was no gross disturbance of cranial nerves or limbs; there were no signs of meningeal irritation. The tendon reflexes were present and equal and the plantar responses were flexor. Lumbar puncture yielded normal cerebrospinal fluid under increased pressure. The urine contained no albumin or sugar and the urinary diastase was normal. Blood-sugar and blood-urea estimations gave no additional information.

After three days of mutism and grossly abnormal behaviour he began to improve; speech returned after a further 24 hours, and within a week he was completely recovered, having an amnesia extending over the encephalitis episode. Examination at this time and again three months later revealed no neurological or psychiatric abnormality and his subsequent mental state is reported as unchanged.

Comment.—There are many resemblances to previously reported cases, notably the acute onset nine days after the appearance of parotitis, and the predominance of mental over physical features. The absence of meningeal symptoms and of cerebrospinal fluid changes is well recognized. Unusual features are retention of urine unaccompanied by any signs of spinal-cord involvement, and the occurrence of mutism.

Discussion

The cases reported above conform to the description given by previous authors. They emphasize the important clinical distinction to be made between meningitis, a common and relatively trivial complication, and encephalitis, a serious condition with high mortality. The reason why the nervous system reacts in these two ways to infection with the mumps virus is not fully understood, although it has been the subject of much recent research.

The clinical syndrome of meningeal irritation, combined with the finding of cerebrospinal pleocytosis and the fact that the virus can be isolated from the spinal fluid, makes it likely that the changes of mumps meningitis are due to the direct action of the virus. In the present epidemic, an unusual one in that it occurred in an adolescent community only recently exposed to mumps, the overall incidence of meningitis (6.3%) is not unduly high, but it is significant that all the meningitic cases occurred in the course of a few weeks in the latter part of the epidemic. This suggests that the virus acquired an unusual degree of neurotropism during the epidemic, reaching a peak in July, when the incidence of meningitis was 23%. There was no increase in the frequency of orchitis or other visceral complication at this time.

If separate strains of virus with increased affinity for nervous tissue do arise in this way the incidence of meningitis in an epidemic of mumps is related more to the properties of the virus than to the state of immunity of the patients. This may help to explain the widely differing estimates of the frequency of meningitis as reported by various authors. It is, of course, also possible that the meningitic cases were due to a superadded infection with an unrelated virus, but this seems unlikely, and has received no support from serological studies (Kilham, Levens, and Enders, 1949).

The occurrence in the present series of two cases of meningitis without other features of mumps serves as a reminder that mumps should be included in the differential diagnosis of benign lymphocytic meningitis. The identity of the infection may be obvious during an epidemic, but isolated cases may give rise to difficulty, which is only solved by routine serological testing. In a recent series (Adair, Gauld, and Smadel, 1953) mumps was found to account for 12% of 854 cases of aseptic meningitis in America, and serological studies have been confirmed by the isolation of the virus from the spinal fluid (Kilham, Levens, and Enders, 1949).

Although most authorities agree in attributing meningitis to the direct action of the virus, there are conflicting views on the aetiology of encephalitis. This has been attributed to a further extension of the virus from the subarachnoid space into the neuraxis, possibly as a result of a high degree of neurotropism or a low resistance on the part of the patient

(Donohue, Playfair, and Whitaker, 1955). However, the latent period before the appearance of encephalitis and the occasional absence of meningeal symptoms and of cerebrospinal pleocytosis (as shown by the case reported here) do not support this view, and favour a primarily encephalitic process rather than a spread from the meninges.

The clinical resemblance between mumps encephalitis and the encephalitis which may follow measles, varicella, and rubella, and the fact that the histological appearances of perivascular demyelination are common to them all, suggest that they arise from the same pathological process (Miller, Stanton, and Gibbons, 1956). The nature of this process is unknown, but it may be a non-specific allergic reaction to virus protein (Miller and Evans, 1953), and similar lesions have been produced experimentally by the injection of brain material with adjuvants (Lumsden, 1949).

Summary

Previous reports on the neurological complications of mumps are reviewed. Lymphocytic meningitis is not uncommon, and recovery is full in a few days. Encephalitis is extremely rare, and has a mortality of 20%.

Eighteen cases of meningitis and one of encephalitis arising in an epidemic of 287 cases are described. The meningitic cases occurred only during the latter part of the epidemic.

The aetiology of the neurological sequelae is briefly discussed. Meningitis may well be due to the emergence of strains of viruses of increased neurotropism during an epidemic.

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Medical Memorandum

Advantages of "Polythene" Ring Pessaries

The advances achieved in surgical technique and, more particularly, in the art of anaesthesia in the past twenty years have made the surgical repair of genital prolapse the ideal method of treating this common disability. However, in spite of these advances there are some women whom it would be too great a risk to subject to an operative cure. We find this group to constitute less than 3% of the total number of patients with symptoms caused by a genital prolapse. A second group of patients complaining of genital prolapse, on whom a surgical repair is not immediately desirable, are the women of child-bearing years who have either been recently delivered or are about to embark on another pregnancy. A third group of patients refuse to undergo a surgical repair. These three groups can have their symptoms relieved by the fitting of a ring pessary into the vagina.

The ideal ring pessary should possess the following properties. (1) It should be made of a substance that is inert and produces no tissue reaction that will lead to a malignant change in the vaginal walls and no offensive vaginal discharge. (2) It should be sufficiently compressible to introduce through a small vaginal introitus, yet possess enough elasticity to regain its shape when introduced into the vagina, and it should not alter in shape or in its physical properties with prolonged use. (3) It should be technically easy to manufacture and cheap to produce.

The many varieties of material from which ring pessaries have been constructed can be divided into those of the preplastic era and those of the plastic era. The former group are the rubber watchspring pessary and the vulcanite pessary. The watchspring pessary is compressible, but it produces an offensive vaginal discharge. The vulcanite pessary has both the disadvantage of incompressibility and the property of producing leucorrhoea. Pessaries made from the many varieties of plastics can be constructed of compressible material or a plastic that is for practical purposes incompressible.

The flexibility of plastics such as P.V.C. depends on the presence of a plasticizer. The majority of plasticizers are toxic to tissues when in contact with them for any length of time and may induce a malignant tissue response. A plasticizer with a low vapour pressure, such as dioctylphthalate, does not produce a tissue reaction. It has, however, the disadvantage of escaping from the material with which it has been mixed, and brittleness and loss of flexibility follow.

Pure polythene, however, is chemically almost inert and has been buried in living human tissues for long periods of time without producing any toxic reaction. It is, furthermore, sufficiently flexible and compressible to use as a pessary and firm enough to support the vaginal walls and uterus in a genital prolapse. The material does not lose its flexibility whilst in the vagina. We have had experience of using the pessaries for three years in the vagina without any apparent physical change occurring in the material. The management of the cases treated with polythene pessaries does not vary from pessaries of other materials.

Patients are examined every three months, the vaginal walls are inspected for evidence of ulceration, and the pessary is washed and replaced.

Polythene is made in a number of different grades which, chemically speaking, differ only in their molecular weight. There is very little variation in the physical properties between different grades except at temperatures up to 100° or below 0° C. In particular the modulus of elasticity which determines the flexibility of the product is much the same for all grades at normal temperatures. The only advantage of using a grade of higher molecular weight is that it can be boiled without danger of deformation. From the manufacturer's point of view, however, the light molecular weights are easier to mould. For these reasons the choice of grade is not very important. A ¼ in. (0.6 cm.) diameter cross-section provides the necessary flexibility and at the same time is thick enough to support the vaginal walls yet will not cause ulceration.

Cost.—The polythene pessary is cheaper to manufacture than the rubber-ring pessary and its life is infinitely longer; while a rubber ring has a life of only six months at the most, a polythene pessary retains its shape, smooth surface, and flexibility for years.

We are indebted to Mr. D. Annat, M.P.S., chief pharmacist, Stoke Mandeville Hospital, for negotiating the original samples of plastic and polythene and organizing the supplies of the polythene pessaries from the manufacturers, Portland Plastics Ltd., Hythe, Kent.

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