NEUROMYELITIS IN MUMPS

BY

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The occurrence of nervous complications in mumps points to the fact that the mumps virus possesses potential or even primarily—according to Dopter (1910)—neurotropic propensities. The experimental work of Gordon (1927), in which meningitis was produced in monkeys by intracerebral injection of a filtrate of saliva from mumps patients, lends support to this view.

Systematic lumbar puncture in cases of mumps by French physicians has shown that a meningeal reaction in the form of a lymphocytosis is almost constant. Definite meningeal symptoms are much less frequent, Dopter reporting 9.8% of cases with meningeal symptoms out of a total of 1,705. Typical meningitis is even more rare, Roux recording three examples among 274 cases of mumps. Meningo-encephalitis may also occur, and Howard (1919) reports three cases in which the encephalitis occurred without parotitis during a mumps epidemic.

The above complications seem to occur at the height of the attack of mumps or shortly before or after it. The rarest complication, and one which appears to have a more delayed onset, is neuritis, or neuromyelitis. This may be localized or widespread. The third, sixth, seventh, tenth, and twelfth cranial nerves have all been reported affected, the commonest being the seventh—possibly through mechanical causes rather than virus infection in some cases. Paralysis of the serratus magnus has been reported by Harris and Bethell (1938). Lightwood (1946) recorded a case of a flaccid paresis of the right lower limb occurring eight days after the onset of parotitis. While these cases are localized, a quadriplegia may develop, and five cases of this are reviewed by Collens and Rabinowitz (1928), their own case being of a man of 29 who developed a flaccid quadriplegia three weeks after the onset of epidemic parotitis, with complete bilateral facial paralysis, loss of the deep reflexes of the limbs and of superficial reflexes, and loss of position and vibration sense in the extremities. The cerebrospinal fluid was normal. Recovery was complete in four months. The four other cases were similar in the development of a quadriplegia at intervals varying from 7 to 21 days after the onset of parotitis. Position sense was lost in the case reported by Pitres and Marchand, and there was associated paralysis of both sixth nerves and left facial and right hypoglossal weakness in a case reported by Revilliod. Recovery was complete in all these cases, though Ford (1937) mentions fatalities due to paralysis of the diaphragm. The following provides another example of the quadriplegic type.

Case Report

A woman aged 37 developed bilateral parotid mumps on May 29, 1946, but ignored the attack so far as was possible, continuing with her housework. A week later she developed pains in the back between the shoulder-blades, which gradually became more severe until she was confined to bed. Two weeks after the onset of parotitis there was a gradually increasing weakness of arms, legs, and trunk until she was unable to sit or even move in bed. At the same time diplopia occurred, but there was no loss of sphincter control. The pain in the back continued all this time, but she had no headache. A week later, on June 19, the patient was admitted to Jersey General Hospital. On examination at this time she was apyrexial and mentally alert though very depressed. She was unable to move her position in bed; the tongue was dry and furred, and the parotid swellings had subsided. The fundi were normal; there was weakness of the third nerve bilaterally, paralysis of the left sixth nerve, complete paralysis of the right facial nerve, and weakness of the right accessory nerve. Cervical rigidity was marked. There was flaccid weakness of both arms with absent biceps jerks and weak triceps and supinator jerks. Position and vibration sense were impaired but other sensation was normal. Abdominal reflexes were absent. There was a paresis of both legs, greater in the left than in the right, with a positive Kernig sign, absent knee- and ankle-jerks, and absent plantar responses. As in the arms, position and vibration sense were impaired. Lumbar puncture was performed and a clear fluid, under 80 mm. pressure, was withdrawn. The Queckenstedt response was normal. The protein content was

20 mg. per 100 ml., there were 5 cells per c.mm., the W.R. and Kahn test were negative, and there was no change in the Lange test.

The patient was treated in the usual way for polyneuritis, including the injection of 50 mg. aneurine hydrochloride daily. The right facial nerve had fully recovered in seven days and the other affected cranial nerves in fourteen days. In five weeks she was able to walk, and in eight weeks returned home, with absent knee- and ankle-jerks as the only residual signs.

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CONSERVATIVE TREATMENT OF ACUTE PERFORATED PEPTIC ULCER*

BY

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The history of the treatment of acute perforated peptic ulcer shows a gradual trend towards conservatism. Four distinct phases in treatment during the last twenty-five years can be recognized.

Stage 1.—Closure of perforation. Thorough peritoneal toilet. Flushing and mopping out of all exudate and foreign bodies. Multiple drainage-tubes.

Stage 2.—Closure of perforation. Limited cleansing and mopping of peritoneum. Removal of obvious foreign bodies. Single supra-pubic drainage for twenty-four hours.

Stage 3.—Closure of perforation. Aspiration of fluid only if obscuring site of ulcer. No drainage.

Stage 4.—Conservative non-operative treatment.

It is a very short but adventurous step from Stage 3 to Stage 4. For many years I had deliberately made no effort to remove the beer and carrots from the peritoneum because the task seemed hopeless, yet not one case of subphrenic abscess resulted. It was pneumonia and cardio-renal failure which were responsible for the fatalities that did occur.

Three further observations seemed to point the way towards greater conservatism. First, all surgeons must have felt on many occasions that surgical intervention had done more harm than good. It was no uncommon experience to find the perforation already sealed off. Operation then merely breaks down the adhesions and attempts to repeat what Nature has already effected. Secondly, during the course of the last 400 gastrectomies for chronic peptic ulcer we have several times noted the presence of sheets of adhesions which indicated that a spontaneous closure of an acute perforation must have occurred in the past. Finally, I can remember more than one obstinate patient who, although threatened with the serious consequences of refusing operation, has nevertheless survived and remained well.

In spite of these reminders that spontaneous recovery can occur it was not until Bedford-Turner (1945) reported a series of six cases treated conservatively that I had the courage to follow his lead. For twelve months I treated every case of acute perforated ulcer conservatively. There was no selection of favourable cases, but unfortunately the series is small. Taken in conjunction with many similar series all over the country, it did provide encouraging evidence in support of a trial for non-operative treatment.

Technique

The technique of treatment was as Bedford-Turner described:

Intravenous morphine, repeated as often as necessary to give complete relief from pain. Stomach wash-out with large-bore tube if a recent meal has been taken. Intermittent suction half-hourly through a Ryle tube passed via the nose. No drinks allowed for

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