sclerotomal pain in the head and neck. They also point out that the spinal tract of the fifth cranial nerve descends to the second cervical level or below. This may be important, for with its spinal connexions a possible pathway exists for reference of pain to the orbit.

It has also been pointed out to us that the small muscles of the suboccipital triangle, which take origin from the first cervical transverse process, and which are so important in balancing the head on the neck, are supplied by the posterior division of the first spinal nerve (which does not supply any structures near the surface of the body). The sternomastoid and trapezius, like the small muscles of the suboccipital triangle, also receive some exclusively deep innervation through the bulbar fibres of the accessory nerve.

These anomalies of innervation probably have some bearing on the unexpected localization of pain from these structures. We have, however, no certain knowledge of the anatomical basis of the reference of orbital and frontal pain from the basiocciput. Kellgren (1949) says: "It is probable that the false localization of deep pain results from a central misinterpretation of its source." He also warns us that "we should avoid constructing those fascinating diagrams of unorthodox pain pathways and pain mechanism whose intellectual neatness and completeness belie the facts and discourage further inquiry."

The question has been raised, "Is this migraine?" but it might be more helpful to inquire whether some examples of migraine originate in a deep lesion in the neck.

We are not yet prepared to say what is the nature of the lesion we have been studying, and have avoided debatable terms such as "fibrositis" and "rheumatism." We can describe it as a tender bony point (the first C.T.P. tip) often associated with tender areas in muscle, but not usually with any limitation of movement. X-ray pictures, so far as they have been done, show no abnormality. We feel that it is an important fact that the tenderness disappears with the relief of the headache.

With regard to treatment, it is difficult to say how much of the improvement was directly related to the exercises. In some cases suggestion may have played a part. In others the fact that someone had time to discuss the symptoms and examine the child thoroughly seemed to give both mother and child confidence and relief from anxiety regarding a serious underlying cause. Tension has been mentioned as a contributory factor in causation, and relief of this, either by removing anxiety or by doing the exercises, or both, has probably been helpful.

Conclusion

This work shows that headache and eye pain related to tenderness of the first C.T.P. tip are not uncommon in schoolchildren and may occur as early as 5 years of age. It is often thought by parents to be connected with the eyes and leads to the unnecessary wearing of spectacles in some children. It may persist for years, and gives rise to the excessive taking of aspirin.

In a few children it cleared up spontaneously, while in the majority it responded rapidly to simple explanation and relaxation exercises. It is notable that most cases had tender areas in the neck muscles which, together with the tenderness of the first C.T.P. tip, disappeared after this simple treatment.

The region of the basiocciput seems to have received little investigation. Palpation of it, and of the adjoining neck muscles and cervical spine, does not usually form part of a routine examination. We think the region merits systematic examination so that we may know more of its contribution to and connexion with the genesis of headache in childhood.

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THREE RECENT CASES OF ENCEPHALITIS LETHARGICA

BY

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Encephalitis lethargica was first described by von Economo in 1917, although in retrospect it seems probable that similar cases had occurred before the outbreak in Vienna. During the next few years the disease was pandemic, and in 1929 von Economo wrote: "To-day there is hardly a medical man anywhere who has not seen a number of cases of this disease." However, it has now become much less frequent, and many modern authorities doubt whether the acute form still occurs. In view of this, three recent cases thought to be examples of the acute disease are reported here. The first is of particular interest because signs of post-encephalitic Parkinsonism, associated with characteristic behaviour disturbances, developed three years after apparently complete recovery from the acute encephalitis. The second and third cases occurred within a few days of each other in the same area of Northern Germany, and the acute illness in each case merged into a chronic post-encephalitic state with Parkinsonism.

Case 1

A police cadet aged 16 was of exemplary character. He had never been vaccinated. For several days before admission to hospital on April 22, 1951, he had a cold. On the day of admission he complained of frontal headache, and at 3 p.m. was found unconscious on his bed, with saliva pouring from his mouth. His tongue was not bitten and he had not been incontinent. By 4 p.m. he had regained consciousness, but was confused and talked nonsense. At 6 p.m. he lost consciousness again and was admitted to the Radcliffe Infirmary.

On examination he responded only to painful stimulation but did not speak. His temperature was 100° F. (37.8° C.), pulse 68, respirations 24, and blood pressure 110/70. There was a mucopurulent nasal discharge and slight conjunctival injection. Hiccups occurred intermittently and myoclonic twitching was observed around the mouth and in all limbs. Kernig's sign was positive, but there was no neck rigidity.

The pupils were contracted and did not react to light, and there was lateral deviation of the right eye. All limbs were flaccid, tendon reflexes being normal, but both plantar responses were extensor.

Cerebrospinal fluid (C.S.F.), obtained by lumbar puncture, was under a pressure of 240 mm. It contained 1 lymphocyte per c.mm. and 45 mg. of protein per 100 ml. Lange colloidal gold curve and Wassermann reaction (W.R.) were negative. X-ray films of the skull were normal. An electroencephalogram (E.E.G.) on April 23 showed a generalized disturbance with no localized or focal abnormalities. The white blood count (W.B.C.) was 9,000 per c.mm. and blood urea 20 mg. per 100 ml.

Progress

On the day after admission his fever subsided. During the next three weeks his level of consciousness fluctuated; sometimes he obeyed simple commands, whilst at other times he would respond only to painful stimuli. There were almost continuous involuntary movements which included chewing and swallowing, yawning, writhing of his limbs, and rubbing his nose to such an extent that it became obviously sore. He was occasionally incontinent of urine. The pupils became dilated, the right more so than the left, and both reacted briskly but transitorily to light. There were defective conjugate movements of the eyes in vertical directions and slight left facial weakness. Plantar responses remained extensor.

On May 1 the C.S.F. pressure was 70 mm., and other C.S.F. findings were as on admission. The E.E.G., recorded again on May 1 and 23, showed a gradual return of normal activity.

Thereafter he recovered slowly and was discharged home on June 21. By then he was out of bed most of the day, but was apt to have spells of crying and was almost completely mute. By July, however, he was speaking normally, and in the autumn he returned to work. For the next 18 months he complained of undue sleepiness during the day-time, and was treated for this with dexamphetamine sulphate. In other respects he appeared to have recovered completely, and worked full time as an apprentice toolmaker.

Subsequently, however, he committed a series of crimes. These were mainly violent and unpremeditated, and there was little attempt to avoid detection. The legal proceedings which followed brought him under medical supervision again in February, 1955. He then described episodes lasting 15–20 minutes during which his eyes involuntarily turned upwards and to the right in a manner strongly suggesting oculogyric crises. There was occasional titubation of his head, his facial expression was stiff, and the glabellar tapping test (Wartenberg, 1945; Garland, 1953) was positive. There was slight cogwheel rigidity of the upper limbs, all tendon reflexes were normal, and the plantar responses were now flexor.

Case 2

This patient, a corporal in the Regular Army aged 28, was stationed in Northern Germany. He had last been vaccinated in August, 1950. On January 31, 1954, he felt dizzy and almost collapsed. He complained of pins-and-needles down the left side of the face and body and had some weakness and incoordination of his left arm. His speech became slurred and his gait unsteady. A little later he complained of frontal and occipital headache and was admitted to a British military hospital. During the day his condition deteriorated rapidly. His temperature rose to 101° F. (38.3° C.), he became increasingly drowsy, and he developed paresis of conjugate eye movements in all directions. There was right-sided ptosis and the right pupil was larger than the left. He became anarthric and unable to swallow. By the evening he had a complete right spastic hemiplegia, with slight hemiparesis and ataxia on the left. Both plantar responses were extensor.

C.S.F. obtained by lumbar puncture was under normal pressure and contained 6 lymphocytes per c.mm. and 45 mg.

of protein per 100 ml. The left ventricle was punctured through a parietal burr-hole and no abnormality was revealed.

On the following day he was evacuated by air to the Military Hospital, Wheatley. On admission there his temperature was 100° F. (37.8° C.), pulse 60, respirations 28, and blood pressure 140/75. The striking feature was his level of consciousness: he appeared comatose, but it was remarkable how easily he could be roused to become alert and co-operative, although within a matter of seconds he would relapse into unconsciousness and have to be roused again. External ophthalmoplegia and ptosis were now bilateral and almost complete. The pupils were equal, but a few hours later the right pupil again became larger than the left, and they both reacted very sluggishly to light. He could not open his mouth, but there were very frequent involuntary champing movements of his jaw and lips. Hiccups occurred occasionally. There was bilateral facial weakness, more pronounced on the right than on the left. The palate and tongue were also paralysed. Excessive salivation combined with the inability to swallow or to cough resulted in a severe aspiration pneumonia which required intensive treatment.

During the first week he had albuminuria and considerable glycosuria. Blood sugar was 104 mg. per 100 ml. and blood urea 20 mg. per 100 ml.; W.B.C. was 16,500 per c.mm. (74% polymorphs). Blood culture was sterile. The blood W.R. was negative. Skull x-ray films were normal. Further specimens of lumbar C.S.F. had a slightly raised protein content, but were otherwise normal. Lange and W.R. were negative. The blood/C.S.F. bromide ratio (Taylor, Smith, and Hunter, 1954) was normal.

Progress

A coarse rhythmical tremor of his head and left arm began 12 days after the onset of the illness and persisted throughout. The somnolence lasted for approximately one month and then gradually lessened, and at this time he developed extreme pathological laughter and crying.

After two months the pupils, though slightly irregular, had become normal in size and reactions, and the ptosis had completely recovered. External ocular movements, however, remained extremely limited, the axes of his eyes were not parallel, and he had diplopia. Conjugate downward movement returned first, but adduction of both eyes remained paralysed and separate lateral deviation was accompanied by coarse nystagmus. Swallowing also gradually returned and he made attempts to talk. His speech improved very slowly, being grossly dysarthric at first, and remained "lalling" and monotonous. There was still excessive salivation. The right spastic hemiplegia showed very little recovery, and on the left side the weakness, ataxia, and tremor persisted. The Parkinsonian picture was recognizable at this time (May, 1954)—facies was mask-like with divergent strabismus. By November the hemiplegia and ataxia were still severely disabling, but he could feed himself with his left hand and walk a few steps with assistance. The pathological laughter was less marked, and his speech and conjugate ocular movements had improved slightly.

By July, 1955, his condition seemed stationary in spite of continual treatment with benzhexol hydrochloride ("artane") and physiotherapy. He could feed and dress himself, and walk with assistance, but he still laughed frequently and inappropriately and his speech remained slurred. His facies was still mask-like. Nystagmus persisted in horizontal and vertical directions. General incoordination with some athetotic movements, right spastic hemiplegia, and bilateral extensor plantar responses remained.

Virus Studies.—Specimens of blood, faeces, and C.S.F. were collected on February 25, and of blood and faeces again on March 12—that is, 25 and 40 days after the onset of symptoms—and were examined by Dr. A. D. Macrae at the Central Public Health Laboratory, Colindale. Neutralization tests showed unchanged antibody levels in the two samples of serum when these were tested against the three

types of poliomyelitis virus. No neutralizing antibody against a strain of encephalo-myocarditis virus was found. The leptospiral agglutination test was negative. Complement-fixation tests against mumps and lymphocytic choriomeningitis viruses were negative. Attempts at virus isolation made with the C.S.F. and faeces by inoculation (1) into newborn mice, subcutaneously; (2) into 4-6-weeks-old mice, intracerebrally and intraperitoneally; (3) into culture of monkey kidney tissue; and (4) on the chorioallantoic membrane of fertile eggs (C.S.F. only) were negative.

Case 3

A private in the R.A.M.C., aged 19, was stationed 30 miles from the previous patient in Northern Germany. He had last been vaccinated in January, 1953. On February 8, 1954, he developed generalized incoordination, slurred speech, and diplopia. On the following day he was admitted to a British military hospital. On admission he was alert, but dysarthric and incoordinate in all limbs, with defective conjugate eye movements and nystagmus in all directions. Salivation was excessive.

On February 10 the C.S.F. was clear, under normal pressure, with 8 lymphocytes per c.mm. and 40 mg. of protein per 100 ml. He gradually developed left-sided pyramidal signs, and between February 15 and 18 he became very drowsy, with almost complete anarthria and ophthalmoplegia. All limbs were ataxic, the deep reflexes were increased and both plantar responses were extensor, but there was no marked loss of power.

On February 20 he was evacuated by air to the Military Hospital, Wheatley. On admission there his temperature was 99.4° F. (37.4° C.), pulse 95, respirations normal, and blood pressure 130/90. His somnolence had the same unusual characteristics shown by the previous patient. In addition to the neurological abnormalities just described he had bilateral facial weakness and slight neck stiffness.

During the next five days he ran a low-grade pyrexia with a tachycardia up to 140 a minute. He became increasingly drowsy, so that at times he was quite unrousable. His pupils varied in size, were unequal, irregular, and did not react to light. The same champing movements of jaw and lips occurred as in Cases 1 and 2, and there were frequent myoclonic twitches and athetoid movements of both arms.

For several days there was albuminuria. Blood urea was 20 mg. per 100 ml. and the W.B.C. 9,100 per c.mm. (80% polymorphs). Blood culture was sterile. Blood W.R. was negative. Skull x-ray films were normal. On February 22 the C.S.F. was under a pressure of 220 mm. It had now become very xanthochromic and contained 1,400 red cells per c.mm. There were 220 white cells per c.mm. (40% polymorphs, 60% lymphocytes) and 100 mg. of protein per 100 ml. On February 25 the C.S.F. was less xanthochromic, and contained 30 red cells per c.mm., 50 white cells per c.mm. (2% polymorphs, 98% lymphocytes), and 50 mg. of protein per 100 ml. On the 27th the pressure and constituents of the C.S.F. in both lateral ventricles were normal. The C.S.F. Lange and W.R. were negative, and the blood/C.S.F. bromide ratio was normal.

Progress

For three weeks his condition was critical. He had irregular, at times Cheynes-Stokes respiration, and he developed an almost complete right hemiplegia. However, during the next month, April, his general condition improved remarkably and his hemiplegia largely recovered but gave place to a coarse tremor of his right upper and lower limbs. External ocular movements improved, but conjugate eye movements and adduction of each eye separately remained limited. There was also nystagmus, ataxia of all limbs, a mask-like facies with divergent strabismus, and the same lalling dysarthria as in Case 2.

Until this time he had remained placid and lacking in emotional expression, but then he developed extreme pathological laughter. Intellectual functions remained unaffected.

During the next year his Parkinsonian appearance remained striking, his head and trunk were held flexed, there was mild titubation of the head, and a coarse rhythmical tremor of the right upper limb. The diplopia cleared, the pathological laughter decreased, and he could walk with a stick. However, he remained severely dysarthric, and had considerable ataxia of trunk and all limbs.

Virus Studies.—Specimens of blood, faeces, and C.S.F. were collected on February 25, of ventricular C.S.F. on February 27, and of blood and faeces again on March 12—that is, 17, 19, and 32 days after the onset of symptoms. These were examined by Dr. A. D. Macrae at the Central Public Health Laboratory, Colindale. Similar tests to those described for Case 2 were carried out, again with negative results.

Discussion

Cases of acute encephalitis of undetermined and probably varied aetiology continue to occur with relative frequency. Some of these cases have been remarkably reminiscent of encephalitis lethargica: Leigh (1946) reported two cases, and thought that certainly one (if not both) might be regarded as a classical example of this variety. Bickerstaff and Cloake (1951), Barrett *et al.* (1952), Brewis (1954), and others have also reported cases in which encephalitis lethargica was thought to be at least a strong possibility.

There is no specific laboratory test for encephalitis lethargica, and, although many studies have been carried out, none of the claims to have isolated a causative organism have been substantiated (Ministry of Health Report, 1922; Macrae, 1954, personal communication). Thus the aetiology remains unknown, and, as in the past, the diagnosis still has to be made on the clinical picture.

The classical features of the disease consist of somnolence and ocular palsies in the acute stage, combined with the development of motor disorders and Parkinsonism. The characteristic motor disorders in this condition are of extrapyramidal type, including involuntary choreiform, athetoid, and myoclonic movements, tremors, abnormal postures, and rigidity. Hemiplegia and cerebellar disturbances were also occasionally recorded, while oculogyric crises are regarded as pathognomonic of previous encephalitis lethargica. Similarly, after the pandemic of the 1920's, behaviour and emotional disturbances often drew attention to a post-encephalitic state, particularly in children (Ford, 1946), though sometimes not until many years after the initial illness. Antisocial offences, petty pilfering, and violent crimes were often committed impulsively without thought of gain or concealment. Although personality changes and deterioration of standards might be gross, intellect was often preserved. Case 1 exemplifies these points extremely well.

In encephalitis lethargica the C.S.F. was frequently clear with normal constituents. Xanthochromia and other abnormalities were, however, occasionally found (von Economo, 1931; Kinnier Wilson, 1940). It is also evident from reports gathered by Hall (1924) and Neal (1942) that definite meningeal haemorrhage had sometimes occurred in the course of the disease, and that fairly large haemorrhages had been found in some fatal cases. Buzzard and Greenfield (1919) also described venous thrombosis and haemorrhage with hemiplegia in some cases.

From the clinical picture and subsequent course of the disease we have no doubt that these three cases should be regarded as examples of von Economo's encephalitis lethargica. In Case 3 a diagnosis of brain-stem angioma was for a time seriously considered in view of the xanthochromic C.S.F. However, the overall picture (Teilmann, 1953; Logue and Monckton, 1954) and the absence of any of the radiological features described by Hoare (1953) rendered it unlikely, and it was finally rejected, as were other brain-stem neoplasms, in view of the subsequent clinical course, now followed for over 18 months in each case. Similarly, the clinical picture and course in none of the three cases suggest even an unusual form of a primary demyelinating

process such as disseminated sclerosis. Nor was there any evidence to support a diagnosis of toxic or nutritional encephalopathy.

That the condition was polioencephalitis, which can at times be extremely difficult to differentiate from encephalitis lethargica (Brewis and Neubauer, 1948) and may present with almost any combination of brain-stem and cranial nerve signs (McAlpine, 1947), is unlikely in view of the absence of evidence of involvement of anterior horn cells and the negative virus and serum antibody studies in Cases 2 and 3. The prolonged initial illness, the C.S.F. findings, and the nature of the sequelae are also much against this diagnosis. Furthermore, the three cases reported here occurred at a time when poliomyelitis was not locally epidemic, and there were no other cases of encephalitis of any type notified in the military or civilian population of Northern Germany at this time.

An illness similar to encephalitis lethargica is only very rarely produced by the many known types of virus encephalitis, and ophthalmoplegia and Parkinsonism are conspicuous by their absence, except in von Economo's disease (Schlesinger, 1952). Moreover, many of the other types are summer infections, whereas the seasonal incidence of encephalitis lethargica is strikingly different, the majority of cases occurring in the first three or four months of the year (Ministry of Health Report, 1922; von Economo, 1931), as did the cases reported here. Virus studies in Cases 2 and 3 were negative for three neurotropic viruses, in addition to poliomyelitis. Further work is required to determine the aetiology of clinical encephalitis lethargica and to show whether it can be produced by more than one organism. At the present time, while the diagnosis remains a clinical one, it must be applied to these and similar cases.

Summary

Three cases which occurred recently have been diagnosed as encephalitis lethargica. They add further evidence that this disease still occurs and that post-encephalitic Parkinsonism may be due to recent infection.

The diagnosis is discussed. If the characteristic clinical picture and sequelae of this disease are observed, there can be no justification for rejecting the diagnosis on the doctrinaire ground that encephalitis lethargica is a disease of the past. The diagnosis must be based on purely clinical criteria.

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CONGENITAL ABNORMALITIES OF THE UTERUS AND PREGNANCY

BY

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Pregnancy associated with congenital uterine malformation is not uncommon. However, it has been given little attention in the standard textbooks of obstetrics. Isolated cases have been reported, but few papers on the subject have been published. In the maternity department of Farnborough Hospital, Kent, I had the opportunity of observing six cases. Three further cases were observed in the Obstetric Department of St. James' Hospital, London. This paper is the outcome of interest aroused by these cases. The cases are reported briefly, and some aspects of the association of congenital uterine malformations and pregnancy are discussed.

The earliest reference to the subject is a case reported by Mauriceau in 1675. Isolated case reports followed this, and Kussmaul published a paper on the condition in 1859. Miller (1922) summarized the literature up to that time and Smith (1931) reported 35 cases. In the last decade papers have been presented by Jarcho (1946), Fenton and Singh (1952), and Baker *et al.* (1953). In the British literature, Way (1945) and Hunter (1950) published papers describing the condition.

Incidence.—In the cases reported from Farnborough Hospital, the six latest pregnancies occurred among 5,000 deliveries, giving an incidence of 0.12%. The three latest pregnancies in the cases seen in St. James' Hospital were among 3,500 deliveries, giving an incidence of 0.08%. From the literature it would appear that the incidence of the association of this abnormality with pregnancy varies greatly. Smith (1931) gives an incidence of 0.003%, Way (1945) reports an incidence of 0.014%, while Baker *et al.* (1953) give 0.03%. It would seem that 1 in 1,000 would be a reasonably correct figure.

Development.—The Müllerian ducts appear around six weeks as two longitudinal folds on the dorsal aspect of the coelomic cavity. They are situated on the outer aspect of the intermediate cell mass, lateral to the genital ridge and Wolffian system. The ducts turn inwards, cross internal to the Wolffian ducts, and lie side by side in the midline. The lower portions fuse at about 14 weeks and they push into the urogenital sinus to form the Fallopian tubes and the uterovaginal canal. Anomalies arise because of: (1) failure of fusion (types A, B, and C); (2) persistence or partial persistence of the septum between them (types D, E, and F); (3) failure of one side to develop (type G).

Classification.—The classification I have followed is shown in Fig. 1. It is adapted from Jarcho (1946). The classification is a simple one and embraces all the common types. As is shown later, the proper classification of a congenital abnormality is of great importance where pregnancy is concerned.

Other Congenital Abnormalities.—Where abnormality of the uterus exists it is probable that other abnormalities will be found, chiefly involving the structures developed from the intermediate cell mass. Thus there is often associated congenital abnormality of the urinary tract, and sometimes congenital abnormalities in other parts of the body. Associated urinary tract anomaly is so