The trial group of patients (5 male, 10 female) were selected only because of their severity, and were in fact consecutive admissions. They happen also to give a reasonable selection of the various disorders associated and often attributable to hypertension. Cases 1, 10, 11, and 12 (male), and 2, 3, 8, and 13 (female) were probably cases of malignant hypertension. Cases 2, 9, and 13 (female), and 11 (male) had cerebral thromboses, Cases 3, 4, 5, 6, 7, 8, 9, 10, 11, and 13 had recurrent attacks of left ventricular failure. Case 11 had angina of effort for at least a year, Case 6 had severe angina of effort and decubitus angina, and Cases 1, 2, 8, 9, 10, 11, 12, 13, and 14 had severe incapacitating occipital headaches often accompanied by vomiting and made worse by reclining. Gross vascular arteriosclerosis in peripheral vessels was easily demonstrable in Cases 2, 4, 5, 6, 7, 8, 9, 11, 13, and 15. The ages of the patients were 23, 28, 32, 38, 49, 51, 52, 54, 55 (two patients), 56, 57 (two patients), 58, and 59 years, and all except the 23-year-old woman and the 32-year-old man were known to have had severe hypertension for more than two years, and in some cases (Nos. 5, 6, 7, 8, 9, and 13) for more than seven years. Every patient in this series was fully investigated for phaeochromocytoma (phenolamine and piperoxan in every case) and had full radiological investigation, including intravenous pyelography and often aortograms—for example, Case 3.

In no case were any of the hypertensive signs and symptoms made worse by treatment. In every case in which a change occurred it was for the better. In every case attacks of left ventricular failure stopped when treatment began, breathlessness was relieved, and angina of effort ceased to occur. When electrocardiographic changes occurred they were towards normal, and heart size was reduced. It was believed that the papilloedema in Cases 1, 2, 8, 10, 11, 12, and 13 was subsiding or has subsided. Only one patient (Case 10) in this group had grossly impaired renal function. There was no evidence to suggest an increased liability to thrombosis in arteriosclerotic vessels; in fact, the evidence in these patients and many others was to the contrary. Those headaches which were hypertensive in origin were immediately ameliorated, but they recurred when treatment was inadequate to maintain the lowered blood pressure.

In conclusion, there can be no doubt, from our experience with this group of patients, that the ability to maintain a sustained hypotension with the drug 356c54 and, better still, with 139c55 resulted in a rapid relief of symptoms and a reversion to normal of many of the changes known to be associated with persistent severe hypertension.

Summary

This paper is a report on the use during the past year and a half of two of a new group of ganglion-blocking agents (compounds 356c54 and 139c55) in the treatment of 15 severe cases of hypertension. These compounds were given subcutaneously, and the results so far have been most satisfactory. The advantages of the compounds, particularly 139c55, in the treatment of hypertension lie in the more gradual onset of activity, the greater duration of action, which makes treatment possible with a single or at most two daily injections, the slowing effect on the heart rate, and the relative lack of action on the small intestine.

I am indebted to a number of lay, technical, and medical helpers, too numerous to mention. I am grateful to my scientific collaborators, Dr. D. W. Adamson, Mr. J. W. Billingham, and Mr. A. F. Green, without whose close chemical and pharmacological association this work would never have been accomplished. I am indebted to Dr. F. Prescott, from whose original suggestion this entire investigation arose, and to the Wellcome Foundation for supplies of ganglion-blocking agents and for help in many other ways. I wish to acknowledge the careful and ungrudging work of Mr. H. Williams, Mr. A. Richardson, and the other staff of the Oldchurch Hospital pharmacy; and Mr. D. Stephenson, of the Wellcome Chemical Works. I am grateful to Dr. J. Stewart and the technical staff of the radiological department of

Oldchurch Hospital for undertaking a multitude of x-ray investigations, and to the sisters and doctors of my medical wards for the thousands of blood-pressure readings necessitated by this work; also to Sisters Turner and Tredway and my registrar, Dr. A. J. Gray. The biochemist, Dr. W. S. M. Grieve, and the laboratory technical staff have, as always, rendered yeoman service. Finally, I am also grateful to my secretary, Mrs. E. D. Price, for keeping my numerous records.

REFERENCE

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INFECTED SUBDURAL EFFUSIONS

BY

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The striking improvement in the results of treatment of purulent meningitis in recent years has led to the increasing recognition of the relative frequency with which subdural effusions complicate convalescence in such cases in childhood. Partly responsible also is the present awareness in paediatric circles of subdural collections of fluid of various kinds in infancy.

The condition has long been recognized and cases have been described; thus Schwartz (1916), in a paper on the aetiology of pachymeningitis haemorrhagica interna in infants, describes a fatal case of meningitis with bilateral subdural effusions. The pathological findings were described by Spitz, Pollak, and Angrist (1945). Only two cases were encountered in a series of post-mortem examinations of 95 consecutive deaths from meningitis in a total of almost 5,000 necropsies. One was an elderly man, the other a child of 3 years. Spitz *et al.* concluded that "subdural suppuration is a rare complication of purulent meningitis, although a higher incidence will undoubtedly be revealed as a result of more complete necropsy studies."

The contrast in recent American experience is expressed by McKay *et al.* (1953), who state that 60% of all infants under 1 year of age suffering from meningitis due to *H. influenzae*, pneumococci, and meningococci develop subdural effusions. Likewise Smith *et al.* (1951), in a period of 11 months, found 20 cases of subdural effusion in 43 infants under 2 years admitted with proved meningitis. The role of *H. influenzae* (Pfeiffer's bacillus) as the causative organism in meningitis associated with subdural effusion is impressive, appearing in at least 50% of the American cases, the pneumococcus and meningococcus accounting for the remainder. *H. influenzae* was the fourth commonest cause of meningitis; but in infants it was the most frequent, with the highest incidence in the second six months of life. It is possible, however, that subdural effusions may be peculiar to the age of the patients rather than to the causative organisms.

The case-fatality rate of *H. influenzae* meningitis was reported by Mutch (1941) as being between 90 and 100%. The very significant change in mortality which has occurred was recorded by Bloor, Grant, and Tabris (1950). Their 44 cases showed a mortality of 22.7%; but when divided into cases above or below 9 months of age the mortality was 8% and 42% respectively. These authors make no mention of the presence of sub-

dural effusions either in the fatal cases or in those surviving. However, about 50% of the survivors were considered to be retarded and 20% had severe neurological defects, including hydrocephalus, hemiparesis, bilateral pyramidal signs, and deafness. It appears probable, in view of the relatively high incidence of subdural effusions recorded in other series, that some of these defective children are suffering from the sequelae of subdural effusions as well as the primary cerebral damage attributable to the meningitis.

Of the five cases of infected subdural effusions presented here, four were associated with purulent meningitis and one was an unusual sequel to bilateral subdural haematomas treated four years previously, in which no organism was isolated. The organisms responsible for the meningitis were *H. influenzae* in two cases and meningococci in one case; no organism was grown in the fourth. All the cases were seen at hospitals in or near Chicago.

Case 1

A 12-months-old girl was admitted to hospital because of vomiting and a left hemiparesis. Seven weeks before admission she had a convulsion associated with a high fever. Subsequently she vomited frequently, and had respiratory difficulty with continued fever. At this time the parents noted that there was weakness of the left arm and leg and that the child was extremely irritable. She was admitted to another hospital and given sulphonamide, penicillin, chlortetracycline, and oxytetracycline, which had little effect on the fever, although the child vomited less and her general condition appeared to improve. She was discharged from hospital, but after two weeks was readmitted. Her temperature continued to be elevated and stiffness of the neck was noted. She was given streptomycin and serum. *H. influenzae* was cultured from the cerebrospinal fluid following lumbar puncture. During the week before admission vomiting had again increased considerably and the child developed a left hemiparesis. She had had no previous serious illness and had developed normally.

On admission to the Children's Memorial Hospital, Chicago, her temperature was 102° F. (38.9° C.), pulse 132, and respirations 26. She was irritable and appeared ill, but was fairly well nourished. The anterior fontanelle was bulging and the veins of the scalp were engorged. The fundi were normal, the pupils were equal in size and reacted normally to light. (The pharynx was not injected and the ears were normal. The heart, chest, and abdomen were normal.) The right arm and leg moved normally, the left leg moved not at all and the left arm only slightly. The deep tendon reflexes were increased on the left side, but both plantar responses were flexor.

The subdural spaces were tapped at the lateral angles of the anterior fontanelle (Fig. 1). No fluid was found on the right side, and on the left there were a few drops of clear cerebrospinal fluid only. Examination of the blood revealed a moderate anaemia with a white-cell count of 23,000, predominantly polymorphonuclear leucocytes. Streptomycin was given, but there was no real change in the child's con-

FIG. 1.—Case 1. Showing difficulty of needling this effusion from the anterior fontanelle.

dition. Accordingly on the sixth day in hospital bilateral posterior parietal burr-holes were made under general anaesthesia. The left subdural space was normal, but on the right side 30 ml. of pus was evacuated from a subdural empyema. A further burr-hole was then made in the right parietal region, but the dura mater and cortex were adherent and congested and nothing further was done. The subdural empyema was irrigated with warm saline and penicillin by a catheter through the posterior burr-hole and the wounds were closed. The child made a satisfactory recovery. Culture of the pus obtained from the subdural space resulted in no growth.

The child's subsequent progress was satisfactory. Her temperature was normal on the third post-operative day and remained so with continued clinical improvement. The hemiparesis improved slightly during her stay in hospital and she was discharged on the ninth post-operative day. She has since been followed up as an out-patient, and there has been a return of function of the left upper and lower extremities and she is developing normally. This child has been under observation for more than one year.

Case 2

A 5-weeks-old female infant developed fever and irritability 10 days before admission. On the fifth and third days before admission generalized convulsions occurred. At this time she was admitted to another hospital and received an unspecified amount of penicillin. Lumbar puncture there revealed a purulent meningitis, but no organism was isolated. She was immediately transferred to the Contagious Disease Hospital, where she was treated with intravenous oxytetracycline, 50 mg. every six hours. The temperature of 102° F. (38.9° C.) returned to normal in 24 hours.

The baby was transferred to the Children's Memorial Hospital because of diarrhoea and abdominal distension, which followed recommencement of oral feeding. The abdominal condition rapidly improved, but on the day following admission her temperature was again 102° F. (38.9° C.) and lumbar puncture yielded a cloudy fluid containing 110 lymphocytes per c.mm., protein 92 mg. per 100 ml., and glucose 34 mg. per 100 ml. The white-cell count on admission was 26,000 per c.mm. Antibiotic therapy was continued and the temperature was again normal on the fifth day after admission.

On admission the anterior fontanelle was said to be flat, but on the third day it was described as "full," and three days later as "bulging but not tense." Bilateral subdural taps were then performed and 6 ml. of light yellow fluid was obtained from the left side and 4 ml. of a darker-coloured fluid from the right. The fluid from the right side contained protein, 2.8 g. per 100 ml., that from the left 0.9 g.

Subdural taps were repeated on four occasions during the next 10 days with decreasing amounts of fluid at each tap from the right side until no further fluid was obtained. The left side continued to yield 6 ml. on each occasion, containing protein about 0.6 g. per 100 ml.

The child was making good progress, was feeding well, and had a normal temperature. The white-cell count was now 10,000 per c.mm. She was accordingly discharged home and returned on two occasions subsequently, at intervals of one week, when the left subdural space was tapped. There was 2 ml. of yellow fluid at the first tap (protein 0.67 g. per 100 ml.; 52 W.B.C.). No fluid was obtained on the second occasion.

She subsequently made good progress. There were no neurological defects, and at 18 months of age she appeared to be normal. At no time were organisms seen on smear or grown by culture from either the C.S.F. or subdural fluid.

Case 3

The patient was a baby girl of 5 months. Her antenatal development and birth were normal. Post-natal development so far was normal and she was said to have been quite well until three weeks before admission to hospital, when she had an upper respiratory infection which progressed to

bronchopneumonia. She had a high fever during this time, ranging from 101 to 106° F. (38.9 to 41.1° C.), and had several generalized convulsive attacks. At the hospital where she was treated for this illness a heart murmur was detected and she was transferred to the Children's Memorial Hospital for further investigations. She was very lethargic and irritable when examined. Occasional generalized convulsions occurred and at all times there appeared to be increased muscle tone in the limbs. There was also a cardiac abnormality which was diagnosed as an intraventricular septal defect. Lumbar puncture on the day of admission yielded an abnormal fluid containing 92 mg. of protein, 41 mg. of glucose, and 686 mg. of chloride per 100 ml. *H. influenzae* was cultured from the spinal fluid.

The child was treated with oxytetracycline and penicillin, but made little progress over the next few days; the drowsiness and spasticity continued. The anterior fontanelle was full but not tense, and one week after admission bilateral punctures of the subdural spaces produced 25 ml. of orange-coloured cloudy fluid from the right side and only a few drops from the left. During the next 12 days about 20 ml. of orange-coloured fluid was removed on six occasions from the right subdural space. The left space gave no significant amount of fluid. On the two succeeding taps the fluid was much reduced and finally disappeared. During this time the child's clinical picture rapidly improved and she had no more convulsions. She has subsequently progressed normally.

Case 4

A 6-months-old boy developed an upper respiratory infection 48 hours before admission to another hospital and was subsequently seen in consultation. Early on the day of admission the child vomited and had generalized convulsions which lasted for about half an hour. When admitted to hospital he remained unconscious for a few hours with a high fever and was treated by cooling, rectal aspirin, sedation, and 400,000 units of penicillin. The following day lumbar puncture yielded cloudy fluid with protein of 115 mg. per 100 ml., sugar 8 mg. per 100 ml., chloride 675 mg. per 100 ml., white-cell count 3,700 per c.mm., predominantly polymorphs. Smear showed Gram-negative diplococci. The white-cell count at that time was 22,000 per c.mm. Cerebrospinal fluid: meningococci were cultured which proved to be sensitive to oxytetracycline, chloramphenicol, penicillin, and chlortetracycline. He was treated with 50,000 units of aqueous penicillin and 50 mg. of chlortetracycline every

two hours, and with adrenal cortical extract 0.5 ml. every eight hours. Feeding was extremely difficult and the child was maintained on parenteral fluids. There was no real improvement in his condition over the next few days; his temperature remained elevated and he was lethargic and irritable.

One week after admission, because of the child's continuing lack of progress, bilateral subdural taps were done through the anterior fontanelle, and 5 ml. of blood-stained fluid was obtained on the left side with no significant amount on the right. The next day the subdural taps were repeated with similar results, but there had still been no improvement. Accordingly, on the same day left temporal and parietal burr-holes were made. Upon opening the dura there was a large amount of xanthochromic fluid under considerable pressure and the brain had been depressed from the dura. A catheter was run in both directions and the areas were irrigated gently with saline. There was no significant improvement after this procedure. The child's course was steadily downhill and he died a week later.

At post-mortem examination large loculated bilateral purulent subdural effusions were found.

Case 5

This child was admitted to hospital at the age of 4½ years for lethargy, vomiting, and unsteadiness for the previous five days. The pregnancy and birth had been normal, but when he was 6 weeks old the mother fell while carrying him and he received a bruise on the head but otherwise seemed unaffected. About a month later the parents noticed that the head was larger than normal and he was brought to hospital. The anterior fontanelle was large and under increased tension. Daily bilateral aspirations of the subdural spaces on three days yielded 40 ml. of blood-stained fluid on each occasion. There appeared to be no improvement and he was accordingly operated on. The subdural spaces were exposed by bilateral parietal crown-trephine openings, and the operative note states that bilateral subdural haematomas were evacuated and the cortex was found to be depressed to a distance of 2.5 cm. by huge haematomas. The child made a good recovery and subsequently developed fairly well, although he was always somewhat retarded. He sat up at 8 months and could stand holding on to objects at 16 months. When seen at the age of 2½ years he had been walking for six months and could speak about 16 words and was able to feed himself.

FIG. 2.—Case 5. One year before present illness, showing previous operative defects and normal suture lines.

FIG. 3.—Case 5. Ventriculogram showing depression of frontal horns and suture diastasis.

Examinations at yearly intervals revealed delayed progress. He was admitted to the Children's Memorial Hospital at the age of 4½. According to the parents the child had been well until five days before admission, when he started to vomit and became unsteady, and for the past two days was said to have vomited everything he had eaten. They stated that the child had had an upper respiratory infection about a month previously and had not seemed normal since.

He was found to be lethargic and irritable, but fairly co-operative. He could move all his limbs, could sit and stand up, but was unsteady in walking. He was very pale and looked as if he had lost weight. The head appeared to be larger than normal and x-ray films of the skull showed diastasis of the sutures (Fig. 3). The fundi were normal and the pupils reacted normally to light. There were no definite localizing neurological signs. A right ptosis and slight divergent strabismus of the right eye were present. All reflexes were very brisk but equal; the plantar responses were equivocal. He had a pyrexia of 100 to 101° F. (37.8 to 38.3° C.). Haemoglobin was 7.6 g.; red cells, 3,600,000; white cells, 23,800 per c.mm., predominantly polymorphs. There was some generalized lymphadenopathy, most marked in the posterior cervical region on each side. No other abnormal signs were found. The chest was normal; the Mantoux reaction was negative; liver and spleen were not palpable.

Posterior parietal burr-holes were made on the day after admission and the ventricles were encountered at a depth of 5 cm. There was some increase in pressure, the fluid was clear, and subsequent examination showed no abnormal constituents. The dura mater was noted to be very thick, but the underlying cortex seemed normal. Subsequently he improved a little in that he was more alert and the vomiting ceased. During the next three weeks his temperature remained normal and he was more alert and was eating fairly well; however, his white cells remained between 20,000 and 30,000. Examination of an enlarged lymph-node and of a marrow smear revealed only reaction to infection.

His condition then began to deteriorate, with further vomiting and some increased temperature. Accordingly ventriculography was performed by puncture through the burr-holes previously made. The left ventricle yielded 20 ml. of clear C.S.F. under pressure which was replaced by air. The right ventricle was not encountered, but on withdrawing from the right side thick pus was obtained. The ventriculogram showed a shift of the ventricular system from right to left which seemed to involve the whole hemisphere. On the same day a large right parietal flap was turned; the dura mater was found to be very thick and was incised high on the hemisphere. A large quantity of pus, amounting to about 160 ml., under considerable pressure, was immediately obtained. The empyema extended from the frontal pole to the posterior parietal region and was about 1 in. (2.5 cm.) deep, having a thick capsule. At the frontal pole there was a small defect in the falx, about 5 mm. in diameter, through which pus was running from the left frontal region; both spaces were irrigated and drained, and the bone flap was not replaced.

For the next week the child's condition was fairly good and

a further burr-hole was then made in the left frontal region, which yielded about 10 ml. of pus. The medial limb of the right craniotomy incision was reopened and a small quantity of pus obtained, but it was found that the brain had considerably re-expanded, almost obliterating the subdural space. During this period he received streptomycin and oxytetracycline. Subsequently he made very good progress and was discharged home wearing a protective head-dress. At this time the scalp flap was depressed (Fig. 4) and pulsated. Presumably this child had small bilateral subdural effusions remaining from the subdural haematomas in infancy. Probably these became infected during the upper respiratory infection which occurred a month before his present illness. No organism was cultured from the pus in this case, although specimens of pus were taken on three occasions.

Discussion

Apart from the atypical case (No. 5) with previous subdural haematomas, the patients all showed one or more of the indications for subdural taps during the course of meningitis. There were persistent fever, convulsions (Case 4), vomiting, or gross neurological defect (Case 1). In particular, abnormal tension of the anterior fontanelle or, at a later age, suture diastasis is considered an absolute indication for investigation of the subdural space. The opinion has been expressed, and sometimes by those with long experience in the treatment of contagious diseases, that subdural effusions complicating meningitis may be ignored, as they are spontaneously absorbed. There may be truth in this in some cases, but it appears certain that the earlier such effusions are removed the less likely will a membrane form and that neurological damage and the necessity for more extensive treatment will be obviated. If the effusion is contained in a definite sac with well-formed membranes it is necessary to remove these by craniotomy.

The amount of fluid obtained at daily tapping of the effusion should rapidly decrease, and at the same time there should be steady improvement in the clinical condition. It is inadvisable to persist in this simpler type of treatment for more than a few days. When progress is unsatisfactory in these two respects, or when a definite membrane has been seen in the burr-hole or is suspected by reason of the chronicity of the illness, craniotomy should be undertaken. During the few days prior to this the child's condition can be expected to improve with tapping of the effusion, with antibiotic therapy, and by hydration. A moderate-sized parietal craniotomy suffices to remove the greater part of the superficial and deep walls of the membranous sac. The brain usually expands to obliterate the subdural space sufficiently, although occasionally it may be necessary to sacrifice the bone flap, temporarily at least, allowing the dura and scalp to fall in to achieve this. After operation the child should be nursed flat and should be well hydrated. Antibiotics are continued for a number of weeks until all signs of infection have passed.

In the three cases treated early by tapping, the period of treatment was short and the results were satisfactory. In Case 1, in which there was delay, the patient developed focal convulsions and a severe hemiplegia which only slowly improved. This case also illustrates the point that the effusion may be so situated as to escape detection by subdural puncture as ordinarily performed through the anterior fontanelle. It was only when posterior parietal burr-holes were made that the large subdural empyema was encountered. This occupied the posterior parietal and occipital regions, and, although an effusion had been suspected from the time she came under our care, it was clear why needling at the anterior fontanelle had been ineffective (Fig. 1).

The presence of the membranes of the original subdural haematomas in Case 5 added greatly to the difficulties of diagnosis and treatment. It was thought that these membranes had been removed at the original operation at another hospital when the child was 11 weeks old. This was evidently not the case, and it seems likely that the lack

FIG. 4.—Case 5. Patient 10 days after craniotomy. Note extreme depression of scalp flap.

of neurological signs, apart from those of increased intracranial pressure, and the absence of convulsive attacks, may have been due to the protection of the cerebral cortex by the thick membrane. Although attempts to isolate organisms from three specimens of pus from the bilateral subdural collections were made, nothing was grown; his clinical condition was one of moderate toxicity, with a persistent low fever and high white-cell count. Vomiting and lethargy were the striking abnormal signs throughout the course of his illness. At operation the tough membrane seemed too firmly attached to the underlying cortex to justify its removal, but, by removal of the bone flap and by some expansion of the cortex, the subdural space became quickly obliterated. This child gave no evidence of having had meningitis, although he was said to have had an upper respiratory infection a short time before admission. The advisability of removing well-formed membranes around subdural haematomas in infancy would appear to be re-emphasized by this experience.

The condition of Case 4 was very poor from the time of admission to hospital early in the illness. However, in view of the convulsions and lack of improvement under treatment, subdural taps should have been performed before the elapse of a week. In addition, bilateral burr-holes should have been made despite, on fontanelle punctures, the appearance of subdural fluid on the left side only.

Summary

Five cases of infected subdural effusion seen in young children in America are reported. Four were secondary to acute meningitis, the other the sequel to a traumatic subdural haematoma.

In infants, recovery from meningitis, otherwise adequately treated, may not occur because of the unsuspected development of subdural effusions. Their early recognition and treatment will also diminish the incidence of serious post-meningitic neurological defects.

It is our impression that more intensive antibiotic therapy than is often employed should be instituted in children with meningitis and that careful observation of the neurological state be made as often as possible so that subdural effusions may be recognized early.

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By the end of this year the mileage of water mains in the country will be half as great again as it was when the war ended. Every year mains of a total length sufficient to reach from London to New York are being laid. These facts were given recently by Mr. ENOCH POWELL, Parliamentary Secretary of the Ministry of Housing and Local Government. "The number of households in the country without a piped water supply," he added, "has been halved in the last five years, and schemes before the Minister at the moment will go far towards halving the remainder. Even in the present period of financial stress rural water schemes are being approved at the rate of 6½ million pounds' worth a year, and nearly all attract Government grants under the Act of 1944."

SUBDURAL HAEMATOMATA AND EFFUSIONS IN INFANCY*

BY

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In discussing subdural effusions in infancy I can add nothing except my own limited experience to what has been said so well and so fully by Ingraham and Matson (1944, 1954) in the United States and by Guthkelch (1953) in England. There are few reports on the subject by British paediatricians, a fact which leads to the assumption that subdural effusions do not occur in Britain so often as on the other side of the Atlantic. This may seem to reflect credit on the conduct of our obstetrics in so far as birth injuries can be held responsible for such effusions; but first let us be sure that we are not sometimes missing the diagnosis. It is because of this possibility that I take leave to remind you of the features by which these intracerebral collections of fluid can be identified.

Aetiology and Pathology

Subdural haematoma resulting from birth trauma is due to the rupture of small vessels passing from the pia through the arachnoid and subdural spaces on their way to enter the venous sinuses, notably the sagittal sinus. It is likely that the decisive factor producing this rupture is unusual stress on the foetal head, leading to abnormal head-moulding and overriding of the cranial bones. However, one has seen extensive moulding and overriding without this damage, so that other influences such as hypoproteinaemia or immaturity of the endothelial tissues may be at work as well.

A remarkable thing is the extent of these effusions as seen at operation. Most often bilateral, and usually larger on one side than the other, the clot may cover the frontal lobe and extend down the temporal lobe to the floor of the middle fossa, and backwards a shorter distance over the parietal lobe. This does not necessarily correspond with the area covered by the original haemorrhage, which may be quite small. Ingraham and Matson (1949) concluded from the available experimental evidence that the blood becomes surrounded by a fibrinous wall which organizes and acts as a semi-permeable membrane, so that surrounding fluid passes into the sac so formed. As the blood disintegrates the oncotic pressure of the fluid rises and more fluid is drawn in. The increase in bulk of the encysted fluid leads to the rupture of the small capillaries in the wall of the sac, still more blood gets into the sac, and the cycle is repeated. Gitlin (1955) does not agree, however, that high oncotic pressure is the decisive mechanism leading to swelling of the sac, but presents data from which he postulates that the increase in the accumulation of fluid is an effusion through irritated and damaged capillary walls, and that repeated haemorrhages into the sac do not, in fact, occur.

From the practical point of view symptoms and signs arise when the growth of the cyst causes a crucial increase in intracranial tension, which happens rather suddenly between the third and eighth months of life, though it may be sooner or later. It is not clear why most cases do not reveal themselves in the early neonatal period. Some infants undoubtedly suffer small "silent" effusions after apparently uneventful deliveries, while others exhibit mild and transient symptoms, doubtless often put down to birth injury from cerebral anoxia and haemorrhage. In still others the signs are overt and respond completely to immediate treatment, as the following history relates.

*A lecture delivered at Nicosia on March 23, 1956, to the Cyprus Branch of the British Medical Association.