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# BRAIN ABSCESS

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BRAIN ABSCESS is not a very uncommon condition, and calls for close co-operation between physician and surgeon as regards diagnosis and prompt and accurate treatment, if recovery is to be expected. The sixteen cases recorded below have all been seen, during the major part of their clinical course at any rate, by one or both of us, all were proved to be brain abscesses, and six subsequently recovered. Almost all parts of the brain where abscess is likely to occur are represented. The cases are grouped according to their ætiology, the first two being metastatic from intrathoracic causes, the third traumatic, and the remainder from middle ear and frontal sinus infection.

### Metastatic Abscess from Intrathoracic Suppuration

CASE I.—Man, aged 41, admitted to a hospital ship on 3.1.41, complaining of pleuritic pain in the right chest since 1.1.41. A pleural effusion developed on that side, at first sterile but later becoming purulent. The empyema was drained posteriorly by Surgeon Lieut.-Commander W. G. Campbell, R.N.V.R., and later a second pocket in the axilla was drained. He was transferred to this hospital ship 15.2.41, when a large encysted collection of pus was found lying postero-laterally. This was drained by rib resection on 22.2.41 by Surgeon Lieut.-Commander F. J. S. Gowar, R.N.V.R., and closed drainage and irrigation with Dakin's solution were instituted. On 28.3.41 a small posterior pocket, which was becoming



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cut off from the drainage track, was drained by intercostal catheter. Irrigation was continued and all pockets cleaned up satisfactorily; the temperature became practically normal, and the general condition improved rapidly. He was allowed up for the first time on 30.3.41. On 1.4.41 he had a sudden attack of dyspnœa while up, and fell cyanosed to the ground. This was followed by a tonic spasm of short duration, after which he felt well except for a slight frontal headache. Two days later he had another fit, which was characterized by generalized clonic spasms, though there was no true loss of consciousness. After this, headaches continued intermittently, and were mainly frontal in character. Up to this time there had been no abnormal physical signs in the central nervous system. On 4.4.41, the patient stated that his headache was worse, and he felt drowsy. He also complained of slight weakness of the left arm and leg. Examination of the central nervous system at this stage showed a slightly hyperæmic right disc, a partial paralysis of the left facial nerve of the upper motor neurone type, and some loss of power and spasticity of the left limbs. The abdominal and cremasteric reflexes remained normal. Later, the left plantar response was extensor. There were no sensory changes. On 14.4.41, he became much worse; he was very drowsy and could only be made to answer with great difficulty, and he had conjugate deviation of the eyes to the left. Lieut.-Colonel F. A. R. Stammers, R.A.M.C., kindly saw the case at our invitation and operated that evening.

A large right osteoplastic flap was turned back, and an abscess was located in the right frontal lobe; this was drained. The patient did not recover from coma and died next day.

At post mortem by M.A.R.G. the right cerebral hemisphere was found to be considerably enlarged. A large abscess was found in the right frontal lobe, which was not encapsulated, and which had markedly compressed the anterior horn of the right lateral ventricle. By pushing the hemisphere across so as to block the left foramen of Monro, it had produced an internal hydrocephalus on the left side. The medulla showed mild signs of a pressure cone; indeed, death was probably due to a fall in intracranial pressure sufficiently rapid to give rise to œdema of the medulla. The chest condition was virtually healed. The pus from the brain abscess grew a short chained streptococcus and bacillus pyocyaneus.

Case II.—Man, aged 28. He was admitted to a military hospital on 29.7.41, with a history of cough for six weeks, malaise and dyspnœa, and he was pyrexial for about a week. Shortly after admission, he noticed a slight weakness in his right arm and leg, the weakness of his arm being first noticed when shaving. On 5.8.41 he was transferred to an army hospital carrier. At this stage there was weakness of the right limbs, and signs of a right pyramidal



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reflexes on the right side, and a right extensor plantar response. His chest showed numerous widely scattered rhonchi, suggestive of chronic bronchitis. The blood pressure was 95/70, the temperature 97, and the pulse 60. He complained of paræsthesia of the right forearm and leg. The next day he appeared rather cyanosed, and on 8.8.41 he complained of fairly severe headache, mainly occipital in character. He vomited that evening. He became very irritable, and some impairment of vision occurred, together with a right ankle clonus.

On the evening of 11.8.41 both of us were asked to see him. He had been given morphia gr. $\frac{1}{4}$ twice during the previous twenty-four hours. He was comatose, but could be roused to a state of irritability. Blood pressure was 140/95. There was a convergent deviation of the eyes to the left, with contraction of the right pupil and dilatation of the left pupil. There was marked left papillœdema and slight right papillœdema. A right facial paresis, especially in the lower part of the face, was present, but no other cranial nerve lesions. There was marked right spastic hemiplegia with knee and ankle clonus, bilateral extensor plantar responses, and absent right abdominal reflexes. It was not possible to test for sensory changes. Lumbar puncture gave clear fluid under some pressure (manometer not available), with 5 cells per c.mm., and normal protein content. The blood leucocyte count was 14,000. Divergent strabismus of the eyes occurred shortly afterwards. A diagnosis of cerebral abscess probably in the left fronto-parietal region was made, but it had been impossible to examine the sensory system or visual fields. He was transferred immediately to this hospital ship, and operation carried out by S.C.S. He was induced with chloroform changing over to intratracheal nitrous oxide and oxygen, and supplemented by novutox infiltration of the scalp. As the incision was begun the patient stopped breathing. There was a faint chance that spontaneous breathing would recommence after decompression, so a large fronto-parietal osteoplastic flap was turned down, while artificial respiration was maintained. The dura which was tense was incised, but exploration in front of the central fissure with the brain needle failed to locate the abscess. There was no sign of return of spontaneous respiration, so further exploration was abandoned, and the wound was closed. The patient died two hours later, on cessation of intratracheal oxygen and carbon dioxide.

Post-mortem examination by M.A.R.G. showed the left cerebral hemisphere to be larger than the right. An abscess was found in the left parieto-occipital region, just anterior to the calcarine fissure, and extending forward into the post Rolandic sensory area in its upper quarter. The abscess did not communicate with the ventricle or subarachnoid space. There was no gross degree of hydrocephalus



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chitis, the bronchi and bronchioles containing a fair quantity of purulent secretion. No lung abscess, bronchiectasis, or tubercle was seen.

### Traumatic Abscess

CASE III.—Man, aged 29. This exceptionally interesting case has already been fully recorded by Colver,[5] so that only a brief account will be given here.

At the age of 14 the patient was struck in the forehead by a detached flywheel, and rendered unconscious for two to three days, sustaining a compound fracture of his frontal bone and a broken nose. From this he made a good recovery, and remained well for fourteen years, until 1938, when he had an attack of meningitis following a head cold, and was in the Royal Naval Hospital, Hong Kong. A curious lance-shaped organism, probably belonging to the Streptococcus D group, was isolated from the cerebrospinal fluid. He was treated with sulphonamide and made a good recovery, except that he remained intolerant of noise and found concentration difficult. February 1939 saw the onset of uncinate fits. These consisted of attacks lasting from two to ten minutes, and occurred every three to ten days. During an attack the whole surface of the trunk would first go cold, as if spirit had been applied to it, then his whole body became enveloped in a peculiar smell, which he was unable to place, but it was not unpleasant and always the same. At the same time he felt himself " as a very little man, isolated from the world around ". In April 1939 he had a further attack of meningitis, similar to the first one, and was under the care of M.A.R.G. The same lance-shaped streptococcus was isolated from the cerebrospinal fluid, and with the aid of sulphapyridine he made an uneventful recovery as far as the meningitis was concerned, but the " smelling attacks " occurred more frequently and the headaches became much more severe. Then followed a curious period of invalidism, during which he appeared to be developing a hydrocephalus. It was chiefly manifested by a markedly raised cerebrospinal fluid pressure, together with the signs and symptoms of increased intracranial pressure, but without meningeal signs. The condition slowly cleared, the headaches became more localized behind the right eye and the uncinate fits went. Examination of the central nervous system at this stage revealed very little except a very slight weakness of the left side of the body, and on several occasions the left plantar response was queried. An X-ray of his skull showed an old fissure lying in the anterior fossa of the skull and involving the cribriform plate. No pneumatocele was demonstrable. On 13.12.39 he was acting in some amateur theatricals, and was about to walk on the stage, when suddenly he saw a



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kaleidoscopic view of coloured balls going round in circles in the upper left quadrant of his field of vision. He sat down, but shortly after his right hand began to move of its own accord; he lost consciousness and a generalized epileptiform fit followed.

He was readmitted to the Royal Naval Hospital, Chatham, and later transferred to the Royal Naval Auxiliary Hospital, Barrow Gurney, for further investigation. Headache became severe and constant, and was situated behind the right eye and in the occipital region. Electro-encephalography was then carried out on 29.12.39 and 15.2.40. The first showed an abnormal focus of discharge in the right temporo-parietal region; the second showed that this focus had extended considerably, and that the focus was low down in the right frontal lobe. On 16.2.40 ventriculography and craniotomy was carried out by Mr. Wylie McKissock at the Burden Institute. The cortex of the superior temporal convolution was found to be very abnormal, being soft, thinned, and of a greenish-yellow colour. The damaged cortex was excised, revealing a firm-walled tumour with a stalk extending forwards to the right frontal bone near the midline, just above the cribriform plate. The tumour was enucleated in one mass. The patient's subsequent course, following operation, was uneventful, and he was allowed up on the seventeenth day after operation. The tumour proved to be an abscess containing pale yellow sterile fluid. There was no evidence of tuberculous infection. Subsequent electro-encephalography showed the whole of the frontal, parietal and temporal regions to be free from abnormal slow discharges. The patient was subsequently invalided from the service, but in December 1940 he wrote to M.A.R.G. saying that he had remained fit since.

### Frontal Lobe Abscess following Frontal Sinusitis

CASE IV.—Boy, aged 14; admitted to St. Mary's Hospital, Islington, on 26.11.35 with a swelling of the left frontal region, which was incised. On 16.12.35 both frontal sinuses were opened by Mr. Archer Ryland, and osteomyelitis of the posterior wall of the left frontal sinus was present with a fistula into the left frontal lobe. On 13.1.36 the hernia cerebri was closed with a skin flap.

He was seen by S.C.S. in Mr. Ryland's absence on 1.9.36, four days after readmission to hospital. He had vomited two or three times, and had two generalized clonic convulsions. Mentally he appeared quite sensible, and the reflexes were normal. Pus was draining from the frontal scar. On 2.9.36 he had five convulsive seizures; the old frontal wound was reopened by S.C.S. that day, some necrotic bone was removed, and a small collection of pus in the lateral part of the left frontal sinus was evacuated. A finger went easily into the old abscess cavity in the left frontal lobe, but no pus was present. A rubber tube 8 mm. diameter was placed in



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the abscess cavity, with a flavine and paraffin dressing, and the skin flap left wide open.

4.9.36.—No fits had occurred since operation, there was no headache and he felt quite well.

7.9.36.—The tube was changed, and much pus was seen to be draining from the abscess cavity. He had no fits, but he had vomited on two occasions. The temperature tended to be sub-normal.

10.9.36.—The patient died relatively suddenly. At post mortem there was a large abscess in *both* frontal lobes, with a terminal meningitis.

CASE V.—Man, aged 47. An intranasal antral and frontal sinus drainage on the left side for chronic pansinusitis had been done elsewhere some ten days previously. He was admitted to the Central London Throat, Nose and Ear Hospital under Mr. Harold Kisch on 19.4.37 with œdema over the frontal bone. He was given three applications of deep X-ray therapy in the hope of localizing the osteomyelitis. On 6.5.37 a localized abscess on each side of the nasal bones was opened.

13.5.37.—Operation by Mr. Harold Kisch, an incision was made from one supraorbital margin to the other and the flap was taken upwards. Both frontal sinuses were opened, and pus was present in both. The anterior walls of both frontal sinuses were removed. There was no spreading osteomyelitis.

14.5.37.—Earlier in the day it was noticed that his manner was strange, and later that day he developed a right flaccid hemiplegia with a right facial paresis. The right plantar response was extensor and the left flexor. There was a right sided Jacksonian twitch. Lumbar puncture gave a turbid cerebrospinal fluid with a pressure of 80/90 mm. water.

Operation at ten that evening by S.C.S.; the flap was turned back and both frontal sinuses were full of pus. The posterior wall of the left frontal sinus was removed and a small extradural abscess was found with a sinus through the dura. The dura was opened and a large abscess in the left frontal lobe was opened and drained by a large rubber tube. The following day gross mental change persisted and the right hemiplegia remained the same. He died twenty-four hours after operation, and post-mortem examination was refused.

CASE VI.—Man, aged 29. He was admitted to the Central London Throat, Nose and Ear Hospital 23.4.37, with a history of acute left frontal sinus five months ago, which had been operated on four times prior to admission. There was X-ray evidence of osteomyelitis of the frontal bone with a fistula. Mr. Watkyn Thomas (S.C.S. assisting) operated on the day of admission. The anterior part of the frontal bone over an area of 8 × 8 cm. was removed and the dura found to be covered with granulations, suggesting an



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underlying frontal abscess, which, however, was not confirmed by any neurological signs. The wound was closed with a vaseline pack dressing.

17.5.37.—He had been drowsy and irritable since the day before but no localizing signs were found. The plantar responses were equivocal. Operation was carried out the same day by S.C.S. in Mr. Watkyn Thomas's absence. The flaps were turned back and the dura incised over the area previously suspected. Pus was found in the lower part of the left frontal lobe with the exploring needle. A large rubber tube was left in the abscess cavity, and a vaseline dressing applied.

The following day he felt much better and was able to read a newspaper. The improvement was, however, short-lived, the condition deteriorated, a fistula into the lateral ventricle developed with subsequent meningitis. He died ten days after the drainage of the brain abscess; there was no post-mortem.

CASE VII.—Girl, aged 15. She was admitted to St. Mary's Hospital, Islington, with an acute frontal sinus and proptosis of the right eye. 23.4.37. An external right frontal sinus operation was done by Mr. Archer Ryland. Much pus was found and a small area of the posterior wall of the frontal sinus was removed. As the patient had headache, vomiting, and right papillœdema on admission, the frontal lobe was explored by needle puncture through the area of dura exposed, but no abscess was found.

17.5.37.—She was seen by S.C.S. for the first time, in Mr. Ryland's absence. The vomiting and headache had persisted since the previous operation. For a week past she had had a daily lumbar puncture, the pressure being on an average 300 mm. That morning she had become quite comatose with a complete left flaccid hemiplegia. There was a persistent fistula into the right frontal sinus and a healing corneal ulcer at the outer limit in the right eye which had occurred during the time when the eye had been proptosed.

Operation at 1.00 p.m. 17.5.37 by S.C.S.; a flap from the right to the left supraorbital margin crossing the bridge of the nose and excising the fistula was turned back. Pus and granulations were present in the right frontal sinus. The frontal bone was widely removed above and including the posterior wall of the right frontal sinus, and a track leading through the dura from the sinus was exposed. A cruciate incision in the dura was made and the flaps turned back. A large abscess was found in the lower part of the right frontal lobe. A large rubber tube was left in the abscess cavity, and a vaseline dressing applied under the flap, which was sutured except for a gap at the right side of the nose for the drainage tube.

The coma and hemiplegia disappeared in two days. 11.6.37, both plantar responses flexor, abdominal reflexes present, general



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condition improving steadily, mentally bright, but very wasted. A cerebral hernia 5 cm. in diameter was present over the right frontal region, but it pulsated and had been slowly receding.

Subsequent convalescence was slow but steady, and the cerebral hernia receded, allowing the supraorbital skin to close over it.

17.10.41.—We are indebted to Mr. Archer Ryland, who has seen her from time to time since the war began, for the information that the drainage scar has been excised by Sir Harold Gillies with very successful cosmetic results, and that she remains well.

CASE VIII.—Man, aged 22. Admitted to University College Hospital under Mr. Watkyn Thomas 18.5.37, with a history of an acute cold eleven days previously, followed by pain in the left orbit four days later and proptosis the following day. Five days prior to admission there had been a remission of symptoms, but the day before admission he became worse, with delirium and severe left frontal headache. On admission his temperature was 103, and the pulse 86. Proptosis of the left eye with fluctuation of the upper lid was present. There was chemosis and limitation of ocular movements in all directions. Pus was present in the left nasal meatus.

19.5.37.—Left external frontal sinus operation was done by Mr. Watkyn Thomas. Much pus was present but no extension into the cranium was seen. Following operation the patient improved, and the proptosis and chemosis of the left eye subsided, but on 30.5.37 a fluctuant swelling appeared over the left frontal eminence. This varied somewhat from day to day, but on 10.6.37 a tender fluctuant swelling appeared to the lateral side of the left frontal area and pointed in the region of the upper eyelid. The blood leucocyte count was 15,000 with 79 per cent polymorphs.

11.6.37.—Operation by Mr. Watkyn Thomas, S.C.S. assisting. The scalp flap was turned up from the direction of the external frontal incision and extending into the left temporal fossa. The osteomyelitic bone was removed by bone forceps right back to the midline and into the left parietal bone ; the direction of spread was along the anterior temporal diploic vein as described by Bucy and Haverfield.[2] Two extradural collections of pus were found, the posterior one communicated with a superficial abscess in the cortex of the frontal lobe, similar to Gill Carey's case described by Symonds.[21] This was evacuated and dressed with paraffin gauze.

During the next three months several small sequestra were discharged, but the scalp healed up well, and he was discharged for observation in the out-patient department 21.10.37. On 6.6.1940 he was passed Grade 1 for the R.A.F. by a local medical board, but turned down by the R.A.F. Medical Board, owing to the skull defect. He was quite fit when seen in out-patients 10.10.1941. He rides a horse but finds that a hard cycling gives him a headache.



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### Temporo-Sphenoidal Abscess

CASE IX.—Man, aged 74. He had been under treatment at St. Mary's Hospital, Islington, from 16.3.35 until 24.4.35 with left chronic suppurative otitis media and cellulitis of the pinna, which had cleared up under conservative treatment.

He was readmitted 6.4.36 with a recurrence of the left chronic suppurative otitis media with pain over the left mastoid, and a history of fits and delirious attacks. 8.4.36 he became drowsy, and lumbar puncture showed clear fluid but increased pressure and protein content. On 12.4.36 he was seen in consultation by S.C.S., and had then a subnormal temperature and distended bladder with overflow retention. He was emaciated and semi-comatose, and there was a large foul polyp in the left external auditory meatus, a right facial weakness, right internal strabismus, doubtful abdominal reflexes, increased knee jerks and bilateral extensor plantar responses. The right pupil reacted to light, but the left pupil was dilated to 3 mm. and inactive, the discs were not seen.

Operation 12.4.36 by S.C.S.: a left radical mastoid was done, and pus found in the antrum. A large area of middle fossa dura was exposed, and at one point, where the dura was adherent to the bone, the dura was incised. Thick stinking pus was found in the left temporo-sphenoidal lobe at a depth of 3 cm. A 1 cm. diameter rubber tube was inserted into the cavity with a flavine and paraffin pack in the mastoid cavity. A plaster cap dressing was applied. He died two days after operation: there was no post mortem.

CASE X.—Man, aged 29. He was seen in consultation by S.C.S. on 21.5.37. Five days previously he had pain in the left ear and a chill, three days previously the discharge had ceased, but since then he had had severe headache. He had suffered from left otorrhœa at intervals for many years—an important factor in the history. Pharyngitis was present, and the left ear was full of purulent debris, behind which slight pulsation could be seen. There was no mastoid tenderness but the patient looked very ill with a temperature of 102·8, and seemed confused and slow in answering questions.

He was admitted to University College Hospital, London, and a left Schwartze mastoid operation was done the same night by S.C.S. The mastoid was sclerotic and there was thick green pus under pressure in the mastoid antrum. The dura of the middle fossa was exposed over a small area, but no abnormality was noticed.

23.5.37.—The headache persisted and cerebration was still slow. The temperature was 101 and the pulse had fallen to 60. An equivocal right extensor plantar response was present. A left temporo-sphenoidal abscess was suspected. The morning of 24.5.37



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a weakness of both arms and hand grip was present and the plantar responses were equivocal. Lumbar puncture gave clear cerebrospinal fluid under pressure of 220 mm. Later in the day right hemiplegic signs were manifest.

Operation at 6.30 that evening by S.C.S.: the mastoid was reopened and converted into a radical, the attic roof was completely removed. A bead of pus was seen oozing through the dura of the middle fossa at this point, from which a track led into the temporo-sphenoidal lobe. The abscess in the tempero-sphenoidal lobe contained stinking pus under pressure, and two small gum elastic drainage tubes were inserted into the cavity with a vaseline external dressing. Special care was taken in this case to avoid disturbing the surrounding adhesions.

He showed no improvement in his clinical condition and died two days after drainage of the abscess. Post mortem showed extensive marginal abscess in the left temporo-sphenoidal lobe which had been drained. There was no attempt at encapsulation. There was a terminal ascending meningitis along the fissure of Rolando.

Case XI.—Woman, aged 45. She was admitted to University College Hospital under S.C.S. 23.10.37 at 3.00 p.m. with a history of left otorrhœa for twelve years, which had ceased five days previously, pain behind the left ear for two days, and severe frontal headache for twenty-four hours with sweating and shivering. There had been no nausea or vomiting. Temperature 102·8, pulse 120, respiration 20, blood pressure 120/90. There was a perforation of the left ear drum with pulsating discharge, and œdema and bulging of the posterior meatal wall. There was no mastoid tenderness or swelling, no neck rigidity, and no Kernig's sign was present. The fundi were normal and the pupils normal in size and reaction. The reflexes were normal and no cranial nerve lesion was found. Weber's sign was to the right.

5.00 p.m. There was marked neck rigidity and equivocal Kernig's sign on the right side.

6.30 p.m. Lumbar puncture gave turbid cerebrospinal fluid under pressure of 300 mm. with a white cell count of 4,280, chloride 0·73, protein 0·055, globulin absent, sugar present, culture sterile.

7.15 p.m. Left radical mastoid operation by Mr. Lance Knox; there was free pus and cholesteatoma in the mastoid cavity. The dura covering the middle fossa was covered with black debris, the temporo-sphenoidal lobe was needled through this area, and pus found. Drainage was made by a gum elastic catheter, surrounded by gauze soaked in biniodide of mercury. A plaster head cap dressing was applied. The pus grew B. Proteus. (S.C.S. was not present at the operation, and is very grateful to Mr. Knox for these notes.)



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24.10.37.—10.00 a.m. The condition was quite good, temperature 101·8, pulse 100, blood pressure 120. She answered questions. Prontosil was begun, grammes 1·5 t.d.s. At 7.00 p.m. she was drowsy and could not recognize objects or people. At 8.00 p.m. the pulse had fallen from 100 to 80, and lumbar puncture gave a turbid fluid under pressure of 255 mm. At 11.00 p.m. she felt better, the headache was less and the pulse now 100.

25.10.37.—She vomited, but the headache was much better, pulse 106, blood pressure 110, temperature normal a.m. but 100 p.m. She was cyanosed and the blood showed methemoglobinæmia, so prontosil was stopped after only 8 grammes had been given.

1.11.37.—The first dressing was done in the theatre, and the tube withdrawn slightly. Lumbar puncture, pressure 210 mm., white cells 94, chlorides 75 per cent., protein 0·08 per cent., globulin not increased, sugar present, no organisms seen in the smear and culture.

5.11.37.—She could not find the words she wished to use, but she could describe the function of objects to which, however, she could not give a name. She felt very miserable.

9.11.37.—Dressing changed in the theatre and subsequently at weekly intervals from now on.

30.12.37.—She walked for the first time.

20.1.38.—Dr. Blake Pritchard reported " No evidence of recent increase of intracranial pressure, rapid irregular nystagmus to each side, slight weakness of right side of face and right hand grip. Tendon reflexes brisker throughout on the right side, abdominal reflexes brisk and equal, plantar reflexes both flexor. I think that these physical signs are consistent with some persistent damage in view of the earlier suppuration but do not necessarily indicate the presence of a persistent abscess or fresh suppuration."

She was discharged 25.1.38, and the last news from her was received in April 1941 saying that she had survived the air raids in London and was working in her stall.

Case XII.—Man, aged 26, with a history of admission to a Royal Naval Sick Quarters 18.8.41 with earache in the right ear for five days. On 20.8.41 a paracentesis had been done, as the lower part of the drum was bulging together with a swelling in Shrapnell's membrane. Pus was obtained. On 26.8.41 the leucocyte count was 14,000. On 1.9.41 a second paracentesis had been done, as the drainage was unsatisfactory. On 9.9.41 leucocyte count was 15,400, with 50 per cent. polymorphs. From then on the condition had begun to clear up. Sulphapyridine had been given intermittently. On 25.9.41 an X-ray had been taken of the right mastoid which showed cloudy and ill-defined cells. On 10.10.41 a sudden flare up with vomiting and vertigo had occurred, and for the first time the patient looked really ill. Temperature and pulse were normal.



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There was a nystagmus with slow movement to the right (depressed activity of right labyrinth). A right Schwartze mastoid operation had been done that day by Surgeon Commander C. B. Nicholson, R.N. The cells were extensive and necrotic, and on exposure of the lateral sinus and dura of the middle fossa, no gross abnormality was found. The pus from the mastoid grew streptococcus mucosus.

On 24.10.41 nothing abnormal had been noted apart from a slight slowness in his speech for the last three days until 6.00 p.m. when he became semi-comatose. The temperature and pulse were subnormal for the first time (Fig. 1). He said he had no headache or

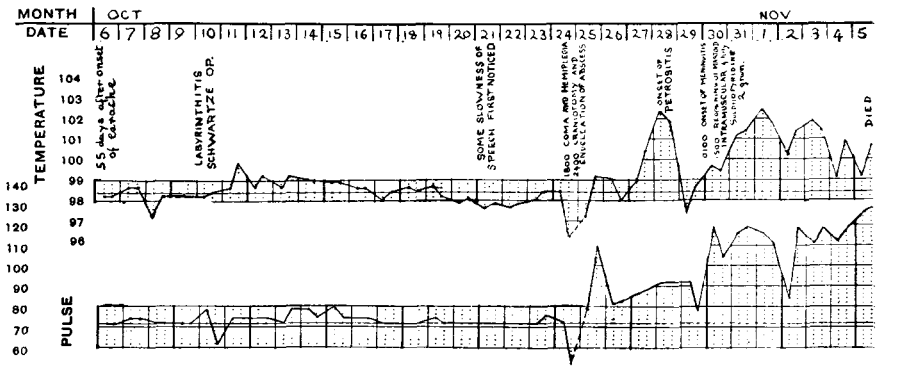



FIG. 1.

Case XII, the temperature chart during the last thirty-one days.

nausea, but could not be roused to answer any other question. The left hand grip was weak and he had a left facial paresis. There was no paralysis of the eye muscles and both pupils were equal in size and reacted normally; he did not appear to see objects to the left side on crude test, suggesting left homonymous hemianopia. The leucocyte count on the previous day had been 14,400.

10.00 p.m. He was seen in consultation by S. C. S. He shook his head when asked if he had headache, but no other response could be obtained. He lay curled up on the left side and exhibited eye rolling. The right pupil was now relatively contracted but reacted to light, and the right disc was congested. There was a marked left facial paresis of upper motor neurone type, weakness of the left arm and leg, absent left abdominal reflexes, left ankle clonus and a left extensor plantar response. The right external auditory meatus was dry, and the post-aural wound practically closed, with very little discharge. Regarding the history of slowness of speech, he was left-handed and his father was also left-handed. Lumbar puncture showed a cerebrospinal fluid under increased pressure, with 28 cells per c.mm. and sterile.

Midnight, operation by S.C.S., under intratracheal gas and



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oxygen by Surgeon Lieutenant-Commander C. V. Harries, R.N., and local infiltration of the scalp with novocaine and adrenalin. A right temporal osteoplastic flap was turned down, and a small cruciate incision was made in the dura, which was not unduly tense, about 2 to 3 c.mm. above the level of the external auditory meatus. The cortex began to herniate. The exploring needle located the abscess capsule at a depth of 2 c.mm. from the surface. The dural incision was slightly enlarged and the cortex removed over a small area down to the abscess capsule, which was now separated from the healthy cerebral tissue by ribbon gauze packing. When the dissection was well advanced, the capsule gave way at a friable point, but fortunately the whole area was protected by gauze and the pus was rapidly removed by suction. Subsequent dissection was now much easier and the whole abscess and capsule came out in one piece (Fig. 2). A tail extended forward to the anterior temporal pole, and the size of the abscess was approximately 7 × 5 × 4 cm., with a wall 2 mm. thick. A drainage opening was made in the lower margin of the osteoplastic flap at its hinge, and a roll of thin rubber sheet left in as a drain from the intracerebral cavity. The galea was closed with interrupted catgut, but a thin strip of leather sheet was left for drainage. The scalp was closed with interrupted silkworm gut.

At 9.00 a.m. on 25.10.41, five hours after the patient returned from the theatre, he was quite conscious, and was able to talk rationally, if slowly. Pulse was 110. The left extensor plantar response and ankle clonus was still present, but the power of the left arm and leg was greatly improved and the facial paresis was less obvious.

26.10.41.—Thirty-six hours after operation the scalp drain was removed and the intracerebral drain shortened slightly. He was seen at this stage by M.A.R.G. Mentally the patient was perfectly well orientated, and he had no difficulty in naming objects, but he was definitely slow in obeying commands. There was a slowness in comprehension though he understood quite well. The pupils and discs were normal. There was a moderate degree of nystagmus when looking to the left, the quick phase being to the left, that is, away from the side of the lesion. There was a residual partial left facial paresis of the upper motor neurone type. There was no sensory dysfunction or astereognosis, and no gross loss of power. The superficial and deep reflexes were normal. There was no gross degree of inco-ordination.

On 29.10.41 the temperature had been 103 on the previous night and he had had frontal headache ; the temperature now had fallen to normal and the post-aural wound which had previously been dry was now discharging pus. Leucocyte count was 18,000. The discharge of loculated pus from the mastoid seemed at the time



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sufficient explanation of the previous night's symptoms, but in reality this was the onset of petrositis. Unfortunately no cold caloric test was done but the nystagmus now present was not considered to be greater than that which had been present ever since the attack of labyrinthine vertigo on 10.10.41 for which the original operation had been done. The cranial wound was clean and dry.

On 30.10.41 at 1.00 a.m. he became restless and subsequently lapsed into coma. He was seen by S.C.S. at 2.00 p.m. when he had divergent squint, flexor plantar responses on both sides, absent abdominal reflexes, neck rigidity, and positive Kernig sign on both sides. Lumbar puncture gave an opalescent fluid under some increased pressure, which grew a fine growth of streptococcus mucosus similar to the cultures obtained at the original mastoid operation and from the encysted brain abscess. The intracerebral drain was removed and the cranial wound sealed off. The mastoid was reopened by Surgeon Commander C. B. Nicholson, R.N., and S.C.S. The dura of the middle and posterior fossæ was widely exposed by clearing away the bone in Trautmann's triangle and the posterior part of the petrous, where a small collection of pus was found. The mastoid cavity was filled with sulphapyridine powder before closure. Sulphapyridine was commenced intramuscularly at four hourly intervals, and intravenous gluco-saline given. The following day he was again conscious, the divergent squint had gone, but a paralysis of the right VIth cranial nerve was present. The temperature had fallen. He was obviously deaf in his right ear, but it was not possible to be quite certain whether this was complete. This improvement was not maintained, and in spite of a total dose of 40 grammes of sulphapyridine and a blood transfusion of 500 c.c. on 3.11.41, the patient died on 5.11.41, eleven days after operation.

A post-mortem was carried out by M.A.R.G. The osteoplastic flap was sealed off and adherent, and showed no signs of infection. There was a thick greenish-yellow exudate at the base of the brain, and extending a short way up the fissures, and it was greatest over the posterior surface of the right petrous bone. No hydrocephalus was apparent. The cavity from which the abscess had been enucleated was found to lie in the right temporo-sphenoidal lobe, and had largely closed. It showed no signs of infection. The petrous bone was opened exposing the labyrinth, which contained glairy pus. No evidence was found of further spread from the labyrinth into the petrous bone, nor of the original track into the temporo-sphenoidal lobe. No pathological changes were found elsewhere in the body. This case illustrates completely Watkyn Thomas's statement, "When labyrinthitis produces a cerebellar abscess, destruction of the labyrinth precedes abscess



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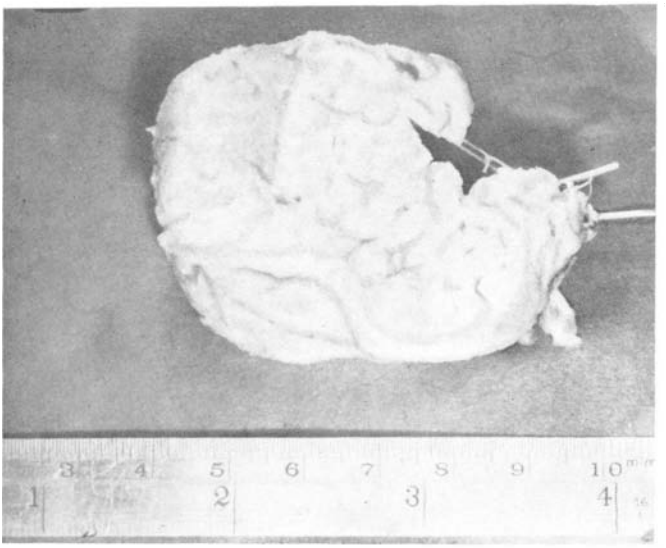


Fig. 2.

XII, the abscess after removal, somewhat shrunken by formal-saline. Photograph by Sick Berth Petty Officer R. B. A. Pay.

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FIG. 3.

Case XVI, antero-posterior radiograph by Dr. R. W. A. Salmond, showing drainage tube in position.

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FIG. 4.

: XVI, coronal radiograph by Dr. R. W. A. Salmond, showing metal drainage tu in position.

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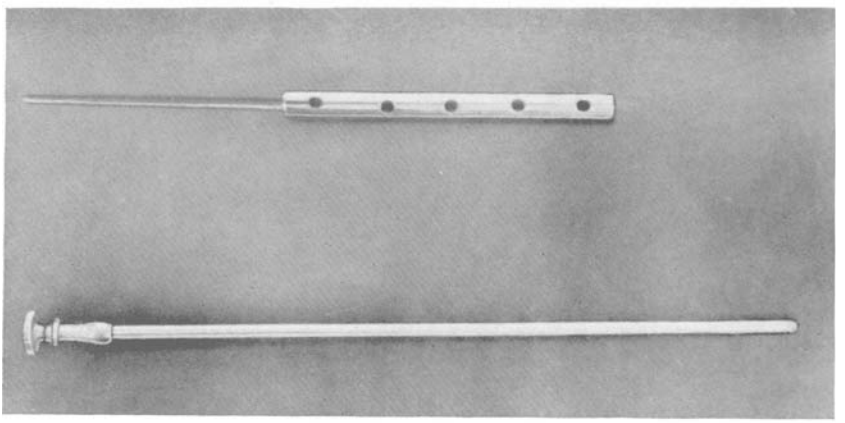


FIG. 6.

Brain-exploring cannula and blunt stillette made for S.C.S. by Willen Bros., and tl metal drainage tube used in Case XVI. The graduations are in centimeters. Phot graph by Sick Berth Petty Officer R. B. A. Pay.

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formation. In cases where labyrinth destruction and posterior fossa infection advance *pari passu*, meningitis, not abscess, is the result."[25]

### Cerebellar Abscess

CASE XIII.—Woman, aged 49. She was admitted to University College Hospital under Mr. Harold Kisch 19.2.33, when S.C.S. was his house surgeon, with a history of sore throat five days previously and left earache for three days. She had been seen outside by Mr. Kisch at the beginning of the attack when the affected left labyrinth was in the irritative hyperactive state, with the slow movement of the nystagmus away from the affected side, that is, towards the right. Vertigo was intense.

By the time she was admitted the nystagmus had changed direction and the slow movement was now to the left side, indicating depressed activity of the affected left labyrinth, and relative hyperactivity of the healthy right labyrinth. It is in this later-stage that acute labyrinthitis is usually seen, as the irritative stage is very transient. The left ear was discharging freely, and there was tenderness in the mastoid tip, but no œdema : temperature 98·2, pulse 88. A left Schwartze mastoid operation was done the same day by Mr. Kisch, and œdema fluid was found in the mastoid cells and much pus in the antrum. The pus grew hæmolytic streptococci. Ten c.c. of aolan were given intramuscularly, and repeated daily.

20.2.33.—The nystagmus was unchanged, and the left pupil was larger than the right, although it reacted normally ; temperature 103, pulse 100.

21.2.33.—Neck stiffness was present and two days later an erysipeloid rash appeared round the wound.

15.3.33.—Up to now the patient's condition had greatly improved and the temperature had been normal for two days, when suddenly the temperature went up to 99·6. A coarse nystagmus developed, this time with its slow movement towards the right, indicating a lesion of the left cerebellum, although, if the patient looked towards the right there was a tendency to change to the quick labyrinthine type of nystagmus previously noted. Vomiting began.

29.3.33.—Persistent vomiting and suboccipital headache with left sided ataxia were present. The mastoid was reopened that afternoon by Mr. Kisch : the lateral sinus was opened and plugged, and the dura of the posterior fossa exposed and opened through Trautmann's triangle anterior to the lateral sinus. Pus was found in the lower part of the cerebellum, and a superficial abscess was drained by two rubber drainage tubes : the pus grew hæmolytic streptococci.

For the first four days after operation the condition was good, but on 2.4.33 she became restless and delirious. On 3.4.33 the



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cerebrospinal fluid contained 14,400 cells, chiefly polymorphs, but was not under pressure. She died 4.4.33. Post mortem was refused.

CASE XIV.—Man, aged 55. He was admitted to Central London Throat, Nose and Ear Hospital under Mr. Archer Ryland on 27.7.36, with a history of a discharging left ear since 1918, and earache with pain in the left temporal region for five weeks. He had been unable to close his left eye properly for a week and had had vertigo, in which he had fallen backwards, objects having on occasions appeared to be turning to the left. On examination he had a small attic perforation of the left drum, which was thickened, and from which there was a seropurulent discharge. There was Rombergism backwards and to the left, and spontaneous nystagmus with a slow movement to the left, indicating depressed activity of the left labyrinth. There was an incomplete lower motor neurone paresis of the left side of the face. He was very deaf in the left ear with Weber's sign to the right.

28.7.36.—A left radical mastoid operation was done by Mr. Ryland. Pus oozed up from in front of the lateral sinus apparently from the posterior cranial fossa.

1.8.36.—"Definite cerebellar abscess," diagnosed by Dr. C. P. Symonds.

15.8.36.—A typical sub-normal temperature and slow pulse were present. He had not been so well since the previous night. The mastoid was reopened by S.C.S. The bone covering the lateral sinus and comprising Trautman's triangle was removed, and the posterior fossa exposed in front of the lateral sinus. The dura of the posterior fossa was incised at this point and pus found with the exploring needle in the cerebellum at a depth of 5 cm. Fifteen cubic centimeters of foul greenish pus were aspirated and a 7 mm. diameter rubber tube inserted with a flavine dressing and plaster bandage.

Subsequent dressings were done at three- to four-day intervals with change of tube each time, and his condition improved steadily until 1.9.36, when he took a change for the worst, and gave the appearance of a spreading encephalitis. He died 6.9.36; post mortem refused.

CASE XV.—Man, aged 38, admitted to the Archway Hospital 30.8.37. A left radical mastoid had been done at another hospital a month previously and he was transferred to the Archway Hospital complaining of headache, occasional vomiting and giddiness. On admission he had a sub-normal temperature, and a coarse nystagmus with slow movement to the right, characteristic of a left cerebellar lesion. There was ataxia of the left arm, but the optic discs and plantar responses were normal.

31.8.37.—Operation by S.C.S. The mastoid cavity was reopened



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and the granulations were cleared away. The bone covering the lateral sinus and middle fossa was removed until the dura of the posterior fossa was exposed in Trautman's triangle between the middle fossa above and the lateral sinus behind and below, until suddenly a bead of pus was seen oozing from a point where the cerebellar cortex was adherent to the dura. The track was opened up and a large stinking abscess found in the cerebellum. After the evacuation of the pus, there was a sudden gush of cerebrospinal fluid, and it was feared that the cisterna magna had been punctured, but no ill effects resulted. A rubber dam drain was laid into the cerebellar abscess cavity.

6.9.37.—Headache had gone, and the general condition was good. The ataxia of the left arm was less, but the nystagmus remained unchanged. Subsequent convalescence was uneventful, the rubber dam being shortened and withdrawn gradually until the cavity closed.

15.8.38.—A year after operation he was symptom free and at work, the radical cavity was epithelialized. There was a large post-aural depression, but it was covered by skin and there was no fistula into the radical cavity.

17.10.41.—We are indebted to Mr. Archer Ryland, who has seen him at intervals during the war, for the information that he remains well.

Case XVI.—Man, aged 26. He was was first seen by S.C.S. in his clinic at University College Hospital on 24.2.38, and he then had an old standing left chronic suppurative otitis media with a large polyp in the meatus. He was advised to have a radical mastoid, but admission was delayed for two months owing to his domestic difficulties.

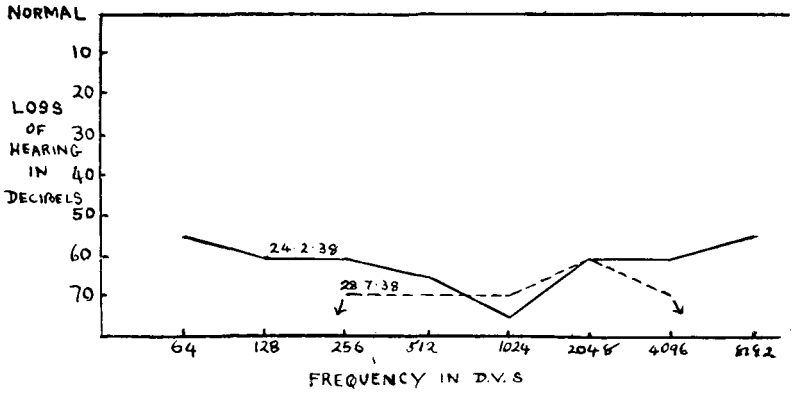



Fig. 5.

Case XVI, audiogram showing the hearing by air conduction before the original radical mastoid operation (continuous line), and after recovery from the cerebellar abscess (dotted line).



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29.4.38.—A left radical mastoid operation was done by S.C.S. Pus was found under pressure in the antrum, which was lined by granulations covering the dura mater. No ossicles were seen.

12.5.38.—The radical cavity was discharging freely, and he complained of morning headache, sometimes occipital, sometimes frontal. The temperature and pulse were normal. There was an intention tremor and dysdiadokokinesis of the left arm, and an atypical fine nystagmus, which changed its direction according to the side to which the patient was looking. No other neurological sign could be found.

19.5.38.—For the last two days the mental condition had deteriorated, and he was now drowsy and vomiting. There was marked left sided ataxia and atonia. The mastoid was reopened by S.C.S. The bone was removed over the posterior fossa in the area of Trautmann's triangle in front of the lateral sinus. A large sequestrum was removed from the posterior part of the petrous bone. It was obvious that the cerebellar abscess had arisen secondarily to a petrositis, so the dura was incised at this point. There was a gush of pus from a cerebellar abscess. A rubber drainage tube was left in, the wound left wide open and a plaster bandage dressing applied.

21.5.38.—He had no headache now, but the nystagmus had become typical with the slow component to the right and the quick component to the left.

27.5.38.—The dressing was done for the first time and the tube left undisturbed.

30.5.38.—There was a sudden onset of drowsiness, headache and vomiting. The left cerebellum was re-explored by S.C.S. and much pus found. The rubber tube was replaced by a chromium-plated copper tube 7 mm. diameter and 8 cm. long (Fig. 6), which was sutured to the scalp.

2.6.38.—The condition was again deteriorating. The metal drainage tube was changed for a rubber tube. Lumbar puncture showed a pressure of 100 mm. and the cell count was 32.

5.6.39.—As there was further deterioration in his condition the cerebellum was again explored by S.C.S. After much fruitless searching, a very deep loculus with much foul pus under pressure was found. At this point the anæsthetist noted an immediate improvement in respiration. The metal drainage tube was again inserted into the abscess cavity. It is shown in position in Figs. 3 and 4.

From now on improvement was uninterrupted and he was discharged 29.7.38. The radical cavity was not completely healed but the post-aural wound was soundly closed. He walked well but there was still a slight degree of nystagmus.

About six months afterwards he was examined by



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Dr. F. W. Kobrak ; it was interesting to see that if the index finger with rigidly outstretched arm was placed on a point in front of him, and he was asked to raise his outstretched arm and touch the point again at rapid intervals with his eyes closed, the arm approached the point in an arc of a circle instead of a straight line. This suggested that some permanent cerebellar damage had been compensated by an acquired cerebral reflex.

He was called up and passed by a medical board grade 1 in 1939, but subsequently released on representation by Mr. Watkyn Thomas in view of the continued necessity for attention at intervals to the radical mastoid cavity.

### Ætiology

For practical purposes the causes of brain abscess may be grouped under three headings, those due to trauma, blood borne infection, and direct extension. The last is by far the commonest cause, and accounts for all but three of our present series of cases, and accounted for 121 out of 194 cases in Evans's post-mortem series.[8]

Cases due to trauma and fracture of the skull are relatively uncommon and are usually associated with a meningitis, and are said to be especially liable to occur when foreign bodies such as fragments of missile or clothing are present. Case III is especially interesting in this respect, as fourteen years' interval supervened between the original injury and fracture of the skull and the subsequent removal of the abscess. In these cases it is possible that a pneumatocele may precede abscess formation.

Hæmatogenous abscesses fall into two distinct groups, those in which the primary focus is in the chest and those in which it is elsewhere. These two groups of cases are about equally frequent—in Evans's series[8] twenty-four were the result of extrathoracic pyæmia, and twenty-two resulted from thoracic lesions.

### Cases Secondary to Thoracic Lesions

A brain abscess may be the sole manifestation of pyæmia in these cases, and results from a septic embolus. A septic thrombus forming in the pulmonary veins finally comes to rest in one of the terminal branches of the cerebral arteries, where a localized encephalitis with subsequent suppuration and abscess formation follows. Chronic bronchiectasis is by far the commonest cause, being next in frequency to otitis, and may have been the cause in Case II, though no very obvious



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bronchial dilatation could be made out at post-mortem examination. Next in frequency is empyema, which was the cause in Case I. In this case it is worthy of note that by the time the brain abscess has become manifest, the empyema had virtually healed. Lung abscesses and neoplasms occasionally give rise to brain abscess in the same way. These abscesses are nearly always in the cerebrum, and occur about equally on the two sides, though curiously enough there is a tendency for the abscess to develop on the same side of the body as the original thoracic lesion. This latter feature, and indeed the curious tendency of thoracic suppurative conditions to metastasize in the brain, has never been very satisfactorily explained.

### Extra Thoracic Causes

These occur as part of a general pyæmia, the actual lesion in the brain often being unimportant, terminal, and often discovered accidentally at post mortem. Actually, in pyæmic states, the brain is comparatively infrequently the seat of septic infarction. Such abscesses tend to be small, are often multiple, and may arise anywhere within the brain. Infective endocarditis and other pyæmic states such as osteomyelitis are frequent causes, and several cases of amœbic abscess of the brain have been reported, though not infrequently the origin cannot be traced.

### Resulting from Contiguous Infection of Nasal or Aural Origin

The brain abscesses resulting from all forms of direct extension combined represent 62 per cent. of the total record of brain abscess post mortems reported by Evans.[8] In this group 9·1 per cent. resulted from nasal infection, and 83·2 per cent. from aural infection. In our series *thirteen out of sixteen* cases are due to nasal or aural infection, five being secondary to infection of frontal sinus, and eight secondary to aural infection.

**Incidence of Brain Abscess following Nasal Sinusitis at the Central London Throat, Nose and Ear Hospital during the Three Years 1935 to 1937 from the In-Patient Reports Compiled by S.C.S. when Registrar**

| | | | |
|---|---|---|---|
| Total in-patient admissions with acute sinusitis | .. | 35 | |
| Incidence of frontal lobe abscess : | | | |
| Died | .. .. .. | 5 | |
| Recovered | .. .. | Nil | |
| Total | .. .. | 5 | |
| Total of operations for chronic or sub-acute nasal sinus infection | .. .. .. | 1,123 | |
| Incidence of frontal lobe abscess : | | | |
| Died | .. .. .. | 2 | |
| Recovered | .. .. | Nil | |
| Total | .. .. | 2 | |



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# Brain Abscess

Even if allowance is made for the fact that only those cases of acute frontal or pansinusitis which develop serious symptoms are admitted to hospital, the high incidence of brain abscess following acute as opposed to chronic sinusitis is very striking. Moreover, in both cases in the above series which followed chronic infection, the brain abscess was secondary to osteomyelitis of the frontal bone, which in the first instance followed on acute infection.

In our experience all the brain abscesses following nasal sinus infection have been in the frontal lobe. We have not observed temporo-sphenoidal abscess secondary to infection of the sphenoidal sinus as described by Evans[8], nor have we observed brain abscess secondary to distant nasal infection such as furuncle of the nose, or abscess of the nasal septum.[14] These latter cases, we feel, belong more properly to the hæmatogenous metastatic group than to the contiguous group of brain abscesses.

The spread from the frontal sinus to the frontal lobe occurs either directly by local necrosis of the posterior wall of the frontal sinus or indirectly as a result of spread from the frontal sinus into the diploic space giving rise to osteomyelitis of the frontal bone.

The posterior wall of the frontal sinus is frequently as thin as paper, and an extradural abscess may or may not preclude the passage of infection across the subdural space. Abscess does not always occur in the frontal lobe of the same side,[2] and may occur simultaneously in both frontal lobes, as in Case IV. In view of the frequency in which the anatomical limits of one frontal sinus cross the mid-line, and the ease with which infection can pass through the paper like septum between the two frontal sinuses and infect the other frontal sinus, the appearance of abscess in the contra-lateral frontal lobe is not so surprising as at first appears.

Osteomyelitis of the frontal bone arises by spread of infection from the medial angle of the frontal sinus along the frontal diploic vein, or from the lateral angle of the sinus along the anterior temporal diploic vein, as in Case VIII. This type of spread is beautifully illustrated in the X-ray photographs of Bucy and Haverfield.[2] Extradural collections of pus form, and it is from these that the infection crosses the subdural space into the cerebral cortex.

It has been our experience that the incidence of spread



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directly through the posterior wall, or indirectly following osteomyelitis of the frontal bone is about equal, and this is borne out by the published data of other writers.

**Incidence of Brain Abscess following Aural Infection at the Central London Throat, Nose and Ear Hospital during the three Years 1935 to 1937 from the In-patient Reports Compiled by S.C.S. when Registrar**

| | | | |
|---|---|---|---|
| Total operations for chronic suppurative otitis media | | 460 | |
| Abscess in temporo-sphenoidal lobe: | | | |
| | Died | 2 | |
| | Recovered | Nil | |
| Cerebellar abscess: | | | |
| | Died | 2 | |
| | Recovered | 1 | |
| | Total | 5 | |
| Total operations for acute mastoiditis | | 533 | |
| Abscess in temporo-sphenoidal lobe: | | | |
| | Died | Nil | |
| | Recovered | Nil | |
| Cerebellar abscess: | | | |
| | Died | 1 | |
| | Recovered | 1 | |
| | Total | 2 | |

There was in addition one case of squamous carcinoma of the external auditory meatus and middle ear in a diabetic woman which spread into the temporo-sphenoidal lobe, causing extensive encephalitis and death from spread of infection into the ventricular system.

In contrast to brain abscess secondary to nasal infection, chronic disease of the middle ear shows a much greater incidence of brain abscess than the acute infection. Of the eight cases reported in this paper six resulted from chronic disease.

We have not found much difference in the rate of incidence of temporo-sphenoidal and cerebellar abscesses, but the records found in the literature suggest a slightly higher incidence of temporo-sphenoidal abscess.

Infection passes into the temporal lobe through the tegmen of the mastoid antrum and tympanic attic. Evans[8] found the average thickness of the tegmen to be 1·5 mm., but frequently it is much thinner. This is a point at which aural infection most easily enters the cranial cavities. The tegmen may necrose and an extradural abscess precede the intracerebral involvement, but frequently an adhesion between the tegmen and the dura, and between the dura and the surface of the cortex, is the only evidence of the route of entry. In chronic middle-ear disease a mastoid abscess cavity of which the dura forms one wall may be present for a long time before intracranial complications set in.

There are three routes by which infection enters the cerebellum from the middle ear: infection of the labyrinth



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and so to the posterior fossa, by way of the aqueduct of the vestibule and saccus endolymphaticus, the subarcuate fossa, or by necrosis of the labyrinth wall: spread of infection through the petrous bone which may contain cells: and, thirdly, the posterior fossa may be involved secondarily to a thrombosis of the lateral sinus. Case XIII is an example of spread by the labyrinthine route and Case XIV of spread from the perisinus region. Cases XV and XVI are both cases of petrositis, and in our opinion this is the commonest route of spread in the cerebellum.

### Prognosis and Prophylaxis

The more encapsulated an abscess becomes before giving rise to symptoms that demand intervention, the better is the prognosis. Globus and Horn[11] have shown how the capsule develops in brain abscesses. All their fifteen cases show advanced encapsulation, but it must be pointed out that none of these cases survived. Unfortunately rising intracranial tension frequently demands surgical interference before this ideal state of encapsulation has been reached.

Cairns's figures[3] show a survival rate of one-third, when those cases which were not deemed justifiable operation risks are included; and we do feel that any unselected series of brain abscess cases, which includes all types, will show a recovery rate greater than 30 to 40 per cent. Metastatic hæmatogenous abscesses have the greatest mortality—both our cases died. Horrax[14] had no recoveries from twelve cases in contrast to his excellent results with contiguous abscesses, and Cairns had no recoveries in ten cases of metastatic abscess following bronchiectasis.[3] Quite apart from the mortality of solitary brain abscess, pulmonary disease, pyæmic abscesses elsewhere, and often multiple brain abscesses increase the mortality very greatly. The prognosis of brain abscess from traumatic wounds appears very good, as shown by Cairns,[3] but the main factor here appears to be the high degree of encapsulation that occurs in the long interval between trauma and the need for surgical intervention.

Of the contiguous abscesses frontal lobe abscesses have the highest mortality. Sir St.Clair Thomson and Negus give the mortality as 80 per cent.[22] All seven cases at the Central London Throat, Nose and Ear Hospital in the period 1935 to 1937 died. Our own series shows a fairly high recovery rate,



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but the number of cases is too small to be of any significance. Of those due to middle-ear infection, temporo-sphenoidal abscess is usually considered to have a better prognosis than a cerebellar abscess, but without any precise evidence to substantiate it. Our own series is too small to throw any light on this point.

Prophylaxis consists essentially in early treatment of acute sinusitis, and operative treatment in all cases of chronic infection of the middle ear which do not respond to adequate conservative treatment. It is significant that no case of a brain abscess developing in a mastoid which has had a radical or modified radical operation at some previous date is known by us to have been recorded. Cases XV and XVI in this series had most certainly got their cerebellar infection before the actual mastoid had been done. Bucy and Haverfield[2] advocate very early external drainage in acute frontal sinusitis to avoid cerebral complications. We consider that their view is unnecessarily drastic. The majority of acute frontal sinus infections subside on conservative treatment, but we agree that if signs and symptoms do not show any remission within two or three days, or if proptosis and chemosis appear, external drainage should be done without undue delay. It is as yet too early to assess what part the general use of the sulphonamide drugs will play in diminishing the incidence of brain abscess, but recently Watkyn Thomas[26] stated, while discussing the question of intracranial complications in middle-ear disease, " that he was not convinced that these had diminished in frequency. That their mortality had diminished he admitted and this to an extraordinary degree ".

### Signs and Symptoms

The taking of a careful history and a thorough general examination of the patient are of paramount importance in the diagnosis and localization of brain abscess. Symptoms may precede signs by some weeks ; this is perhaps especially true in post-otitic cases where headache, drowsiness and vomiting may precede signs by a month or more. At the same time an exact symptomatology may be difficult to elicit. These patients are often drowsy and poor witnesses, and may be seen for the first time when they are already in coma. In Case II, for example, the patient was already in coma when seen by us, and a tentative diagnosis of cerebral tumour had been made :



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it was on the history of chest trouble and the present condition of his chest that a diagnosis of abscess was made.

It is customary to classify signs and symptoms under four headings, those due to local suppuration, those due to raised intracranial pressure, localizing signs, and finally the terminal extension of the infective process. Some symptoms—drowsiness, for example—may be due to a combination of factors, but for the purpose of classification the above is useful.

### Those Due to Local Suppuration

They vary in proportion to the acuteness of the infective process. They are, therefore, most marked in acute suppurative encephalitis and early abscess formation, and are often slight or entirely lacking in chronic abscesses. They tend to be more marked in contiguous than in metastatic abscesses, but they tend to be masked by suppuration elsewhere, which usually renders their assessment difficult. These signs and symptoms include pyrexia, rigors, and blood and cerebrospinal fluid changes. It is realized that certain other symptoms such as drowsiness, mental sluggishness and wasting may in part be due to the local suppurative process, but for various reasons are included under other headings.

The temperature is usually raised during the early stages when a diffuse encephalitis is present, but tends to be obscured by the original infection. Later when the abscess becomes encapsulated it tends to be normal, or sub-normal. A sub-normal temperature in a patient suspected of having a brain abscess is very suggestive. Case II had a sub-normal temperature when first seen by us, and the temperature chart of Case XII, inasmuch as it shows the temperature tending to become sub-normal four to five days prior to operation and finally dropping to 96 degrees twelve hours before operation, is suggestive.

Rigors may be expected in abscess formation in any situation, but frank rigors must be rare in brain abscess and their presence would be more indicative of systemic infection, or other complications such as lateral sinus thrombosis.

The blood picture usually shows some degree of secondary anæmia, and a moderate polymorphonuclear leucocytosis. This latter feature is more marked in the acute abscesses and may be lacking in the chronic ones. Again, it is often impossible to say how much is due to the original infection. Our



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cases for the most part had a leucocytosis of about 15,000 : Case II was 14,000 : Case VIII was 15,000 : and Case XII was 14,000.

The cerebrospinal fluid changes are important, and are mainly of use in differentiating brain abscess from other intracranial complications such as meningitis, and in assessing the degree of intracranial pressure. Information can also be gathered as to whether the abscess is leaking into the ventricles or subarachnoid space. The fluid is usually clear as long as the abscess remains localized. The pressure varies, and may be normal or raised. It is usual to find it somewhat raised. Case V gave a pressure of 80 to 90 mm. water, while Cases VII and XI both gave a pressure of over 300. There is usually some increase in cells, except in very chronic or deep-seated abscesses ; the cells are chiefly lymphocytes and are seldom more than a hundred per c.mm. Case II, although *in extremis*, and with a relatively superficial and only partially encapsulated abscess, had only 5 cells per c.mm. Case XI is interesting in that the cerebrospinal fluid was turbid, containing 4,280 cells per c.mm., whilst the chlorides and sugar remained normal : this was not an indication of meningitis and after operation the cells dropped to 94 and the patient finally recovered. The protein content of the cerebrospinal fluid is usually somewhat increased, whilst the chlorides and sugar content remain normal. A preponderance of polymorphonuclear cells together with a fall in chlorides and sugar indicates a leak into the cerebrospinal fluid. For diagnostic purposes not more than 1 c.c. of the fluid should be removed, and is especially true if there is a marked increase in pressure. Wasting may be a marked feature in some cases of the more chronic type. It was well marked in Case IX, but is seldom in itself an important diagnostic feature. Case XVI showed wasting during the period between the first exploration and the final re-exploration.

**Signs and Symptoms Due to Raised Intracranial Pressure**

These may be slight or absent, even when quite large abscesses are present. They are much more prominent in an acute and rapidly expanding abscess than in a slow-growing and encysted abscess. In the acute abscesses, moreover, the area of surrounding œdema tends to exaggerate symptoms by increasing the intracranial pressure.

Headache is perhaps the commonest symptom, except



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sometimes in young children who cannot complain. It varies much in intensity and is more marked in acute abscesses and may be absent altogether in chronic abscesses. It is more often paroxysmal than continuous in character, and is usually made worse by stooping and by exertion. The position varies, and frequently has some relation to the site of the abscess, often being occipital in cerebellar cases, temporal or bitemporal in temporo-sphenoidal cases, or frontal in frontal lobe abscesses. On the whole the position of the headache is only of real value when the abscess is in the frontal lobe, or in the occipital lobe and cerebellum. Elsewhere it may be misleading; Case XI, for example, had severe frontal headache with a left temporo-sphenoidal abscess. Cerebellar abscesses usually give rise to occipital headache, a useful point in abscess following middle-ear infection. Case II was interesting in this respect. The abscess was metastatic, the original focus was not, therefore, of any help in localization, and the patient was in coma when first seen, rendering examination more difficult. There was, however, the history of headache mainly occipital in character. Exploration was made rather too far anteriorly, and when reviewing the case afterwards we came to the conclusion that we had attached too little importance at the time to the occipital character of the headache. With the onset of hydrocephalus the headaches tend to be frontal or bitemporal. It cannot be too strongly stressed that the persistence of headache after adequate mastoid drainage demands careful investigation, and this is especially true if it is combined with vomiting.

Papillœdema occurs late and may be absent even in large and long standing abscesses. It is usually more marked on the side of the lesion, and is of value in this respect. In Case II, for example, papillœdema was much more marked on the side of the lesion, and in Case VII was only present on the side of the lesion.

Bradycardia is common, especially if the intracranial pressure rises quickly. On the whole it appears to be commoner in abscess than in tumour. The pulse rate slows in inverse proportion to the rise of intracranial pressure, and the blood pressure rises with the intracranial pressure and often serves as a useful ancillary guide. Respirations tend to become slow, Cheyne-Stokes breathing finally supervening, whilst respiratory and cardiac failure occur as terminal events. Drowsiness is a most useful sign. Occasionally in abscess, insomnia and some



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degree of restlessness is encountered. This is rare, but if insomnia does occur, it is a useful differentiating point from tumour, where insomnia is virtually unknown. Character change and a lack of vitality and vivacity, and disinclination to work may precede actual drowsiness in the more chronic cases. All our cases showed drowsiness or character changes to some extent. Case III, who was an exceptionally intelligent man, was closely questioned by M.A.R.G. as to what he felt or noticed peculiar about himself in the weeks prior to operation. He was aware that a change had come over him. He had formerly been a great reader, but now he felt little inclination to read, and then only light material. He was at times rather drowsy, and slept in the afternoon, which he never used to do before, but as rather violent paroxysmal headaches sometimes interfered with sleep, this was difficult to assess. He felt, at times, a sense of detachment from the world. He felt alone and looking at the world as a spectator, though he was well aware of what was going on. Serious brainwork, and especially prolonged concentration, was a great effort to him.

Vomiting is fairly frequent, especially if the pressure is rising rapidly. If combined with drowsiness or headache it is a valuable confirmatory symptom. Although usually it is a result of an increase of intracranial pressure, it can occur from the direct involvement of the cerebellum or the vestibular nerve.

### Local Signs

Contiguous abscesses occur near the site of the original infection and their localization is not as a rule so very difficult, but local signs are of extreme value in metastatic abscesses. On the whole, localizing signs are more marked in chronic than in acute abscesses, and there is often a long latent period before they become definite. In metastatic abscesses the original embolism may lead to transient weakness or localized convulsions weeks before other signs become obvious. This shows the need for keeping a constant check on the central nervous system in a suspected case. Oedema and hydrocephalus may lead to false localizing signs. The visual fields should be examined early. If a defect is found it is a most important localizing sign. Had we been able to examine the visual fields of Case II, it is reasonable to suppose that a defect would have been found which would have led to an accurate localization of



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the abscess. Metastatic abscesses can occur anywhere in the brain, and give much the same localizing signs as tumours in the same area. It is not proposed, therefore, to describe in detail every sign which might be produced by such an abscess, but to limit remarks to the commoner sites with special reference to our series of cases. The three common sites of abscess formation are the frontal lobe, the temporo-sphenoidal lobe and the cerebellum.

### Frontal Lobe Abscesses

Symptoms such as headache and apathy are earlier and more in evidence than signs, which are often absent altogether. Cases VI and VIII had no abnormal signs in the central nervous system, and Case IV presented no abnormal signs though fits were present. Fits are common, and may be the first definite sign of an intracranial lesion as in Case I. These may or may not be accompanied by complete loss of consciousness, and may be Jacksonian in type as in Case V. In Case I fits were the first indication that anything was amiss. The patient was up and about, apparently well, with an empyema wound which had virtually healed, when suddenly he had an attack of dyspnœa, became cyanosed, and went into a state of tonic spasm. Two days later he had another fit with clonic spasms and it was only after this that the other symptoms and signs appeared. A fit occurring in a patient who has nasal or aural infection, or who is suffering from chronic intrathoracic suppuration, is very suggestive of cerebral abscess. Fits occurred in three out of six of our cases of frontal lobe abscess, but they are apt to occur in any condition producing a raised intracranial pressure and as such may have little localizing significance.

Aphasia is a fairly common symptom, though mild degrees of aphasia, especially difficulty in naming objects, may be produced by a generalized increase of intracranial pressure, and can even occur in cerebellar lesions. It is not, therefore, always a very reliable localizing sign. In frontal lobe disease it may be of the verbal type, in which the patient names objects wrongly, is slow and hesitant in his speech, or has a tendency to stutter.

Some degree of hemiparesis occurred in three out of six of our cases of frontal lobe abscess, and it is due to involvement of the motor area by œdema or infection spreading from the frontal lobe. It usually manifests itself at first by a slight



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weakness of the contra lateral side of the body, progressing later to an actual flaccid paralysis. Unilateral anosmia and slight unilateral exophthalmus sometimes occur. We have not noted the presence of the grasp reflex in these cases.

Deviation of the eyes is worthy of mention. Case I developed conjugate deviation of the eyes to the opposite side to the lesion. The supranuclear fibres of the IIIrd and VIth cranial nerves originate in the second frontal convolution. Excitation of this area gives rise to conjugate deviation of the eyes to the opposite side.

**Temporo-sphenoidal Lobe Abscesses**

The localizing signs and symptoms vary somewhat with the position of the abscess. If the abscess lies far back, defects in the visual field and some degree of aphasia may be expected. If far forward, the uncinate gyrus may be involved with the production of uncinate fits. If high up, the motor area is likely to be implicated with the production of contra lateral muscle weakness and other hemiplegic signs.

The visual fields are of great importance and may be the only localizing sign. The lower fibres of the optic radiation are caught as they pass round the end of the descending horn of the ventricle, leading to an upper quadrantic homonymous hemianopia of the opposite side. This proved of value in Case XII, where rough assessment of the visual fields prior to operation suggested a contra lateral homonymous hemianopia, and would doubtless have proved of great value in Case II had we been able to carry it out.

Visual hallucinations are said to occur sometimes. They generally appear as actual objects, the figures being more formed than in the case of occipital lobe involvement. It is perhaps interesting to note that Case III who had a right temporo-parietal abscess had a visual aura before his one and only epileptiform convulsion.

Left temporal lobe abscess in a right-handed person frequently gives rise to some degree of aphasia. This is generally of the nominal type, in which a patient finds difficulty in finding his words, or names objects wrongly. The degree of aphasia varies, and may not be very marked, so that a prolonged test with numerous objects is necessary. More commonly a certain hesitancy is noticed in the speech. Case XII was slow and hesitant in his speech after operation, and it was reported



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to us that his speech had been somewhat slow for about three days before the onset of coma. This case is interesting, because a right temporo-sphenoidal abscess was present in a patient who was left-handed, and whose father was also left-handed. Case X had a marked slowness of speech when first seen, and it was difficult even to get a satisfactory history from him. Case XI had nominal aphasia after operation, which slowly cleared up. It is probable that this existed prior to operation. Aphasia of various types is more likely to occur if the abscess is situated posteriorly. It is likely to be quite absent in those situated far forward.

Unilateral weakness and hemiplegia and other signs of a pyramidal lesion are common and important. As might be expected when the representation of the body in the motor cortex is considered, the face of the opposite side is often the first area to be affected, and unilateral facial weakness may be the only sign. Paresis is usually more marked in the lower part of the face than in the upper part. The hand and arm are the next most frequently affected. Case XII showed this very well prior to operation, he had weakness of the opposite side of the face and a weak hand grip. Case XI developed both these signs, together with some degree of nominal aphasia, after operation. A further extension of the process may lead to a widespread hemiplegia with the usual signs of pyramidal pressure. This sequence of events is shown quite well in Case X: at first an equivocal right extensor plantar response only was apparent, whilst by the next evening a typical right hemiplegia was present. An equivocal left plantar response and, rather later, a slight weakness of the left side of the body were also the first physical signs to appear in Case III.

Jacksonian fits on the opposite side are sometimes seen when the temporo-sphenoidal abscess is superficial, as in the case described by Symonds.[21] Clonic convulsions begin in the face, followed by the arm and finally by the leg. A similar picture, however, may arise when the abscess leaks into the subarachnoid space and meningitis tracts up along the fissure of Rolando. Generalized epileptiform convulsions may occur in temporo-sphenoidal abscesses as with abscesses anywhere in the cerebrum. The type of aura, however, may have some localizing significance. Uncinate fits, when they occur, are valuable localizing symptoms. They are likely to occur in abscesses which are situated anteriorly in the temporal lobe,



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and which involve the uncinate gyrus. They are characterized by olfactory and gustatory aura followed by an abnormal state of consciousness, and perhaps certain motor components. They were very well shown in Case III, where a curious sense of smell was followed by a dreamy state, in which the patient described himself as feeling like a very little man detached from the world and regarding it as an onlooker.

The cranial nerves may be involved by pressure, especially the IIIrd and VIth nerves, and thus the pupils are affected. The pupil at first contracts, and later as paralysis sets in, dilates. In Case XII the pupil on the side of the lesion became relatively contracted in the two to three hours before craniotomy, but continued to react to light. In Case IX the pupil on the side of the lesion was dilated and fixed.

**Cerebellar Abscesses**

The signs and symptoms of an abscess of the cerebellum differ somewhat from abscess of the brain elsewhere. The diagnosis is frequently difficult if labyrinthitis is also present. The signs depend upon the involvement, usually by œdema, of the dentate nucleus. As has been pointed out already, headache in cerebellar abscess is sub-occipital in the early stage,[21] and may be associated with some degree of neck stiffness, as in Case XIII. Sub-occipital tenderness is sometimes present. Papillœdema is commoner in cerebellar than in cerebral abscesses, but is often absent.

The classical type of cerebellar nystagmus is coarse, and its slow component is to the opposite side, and as such was present in Case XV. It is, however, not always true to type. In Case XVI, for instance, the nystagmus was fine, and changed its direction according to the direction in which the patient was looking, but eventually in the post-operative period it became classical in type. Symonds[21] points out that nystagmus may be preceded in time by the deviation of the eyes away from the injured side when looking to the injured side; that is to say, the slow component only is present, without the correcting quick cerebral movement. It has, unfortunately, become customary to refer to nystagmus in terms of the direction of the quick component. Since this quick component is a cerebral correction, it is more logical and gives a more intelligible picture if the nystagmus is referred to in terms of its slow component, which is the true abnormal manifestation.



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If the cerebellar abscess develops following labyrinthitis the picture is often confused. The full course of events is shown in Case XIII. The first stage was that of irritation of the affected labyrinth, resulting in hyperactivity and a nystagmus in which the slow component was to the opposite side. As is almost invariably the case, this stage was only transient, and gave place to the stage of suppressed activity of the affected labyrinth with reversal of the direction of the nystagmus, that is, the slow component was now to the same side. Finally with the development of a cerebellar abscess, the direction of the nystagmus was again reversed, having now its slow component towards the opposite side. However, there was still a tendency for the earlier fine labyrinthine nystagmus to reappear if the patient looked away from the side of the lesion.

Nystagmus is accompanied by vertigo, Rombergism, and pointing errors. In labyrinthitis the vertigo is in the direction of the quick component of the nystagmus, while Rombergism and pointing errors are in the direction of the slow component. In labyrinthitis, past pointing shows a deviation of both hands in the direction of the slow component of the nystagmus, but in cerebellar abscess only the hand of the same side deviates, and that always outwards.[25]

Some degree of ipsilateral hypotonia is usual. It is generally easily palpable in the muscles themselves, and can be demonstrated by the diminished resistance to passive movement. This lack of muscular tone, together with inco-ordination, gives rise to hypermetria, or overshooting the mark if the patient tries to point to anything, and to dysdiadokokinesis. Dysdiadokokinesis is an early and constant sign. It was present in all our four cerebellar cases and in Case XVI dysdiadokokinesis and an intention tremor preceded other signs by four or five days.

Watkyn Thomas[25] has pointed out that these three signs of hypotonia, hypermetria, and dysdiadokokinesis indicate a cerebellar lesion whatever the state of the labyrinth may be. Moreover, in cerebellar abscess, motor signs such as ataxia and Rombergism are more in evidence than vertigo, in contrast to labyrinthine disease where vertigo is predominant. In Case XIII a marked labyrinthine vertigo dominated the picture in the earlier stages. In Case XIV the vertigo was due to irritation of the labyrinth by chronic infection in the middle



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ear, but in Case XV no labyrinthine factor was present and, therefore, in this case the vertigo was of cerebellar origin.

Cranial nerve lesions resulting from cerebellar abscess have been reported, but are rare. In Case XIII a dilated but normally reacting pupil on the side of the lesion was present at an early stage in the clinical course.

Cerebellar abscess, from its anatomical position, is prone to cause sudden respiratory embarrassment. In Case XVI, during the final re-exploration, the release of a loculus of pus under pressure caused the anæsthetist to comment on the immediate improvement of respiration.

### Terminal Signs

Increasing œdema causes a deepening coma and eventually paralysis of the respiratory centre. Alternatively the abscess or area of encephalitis spreads suddenly into the subarachnoid space or ruptures into the ventricular system, giving rise to the characteristic picture of meningitis.

### Special Methods of Investigation

Radiography by itself is of limited use in brain abscess, except in the traumatic variety. In Case III it was of use in showing the old fracture line in the base of the skull, and in cases where a radio-opaque foreign body is present, it is of actual localizing value. If the pineal body is calcified, the direction of the pineal shift is of use. The injection of radio-opaque substances into the abscess cavity is of use in indicating the size of the abscess and can be used to follow its migration to the surface if originally deeply situated.

### Ventriculography

Ventriculography is mostly of value in cases of metastatic abscess where there is no local focus or infection and a paucity of localizing signs. It is particularly useful when there is doubt as to which cerebral hemisphere contains the abscess.[3] The ventricle on the side of the abscess is collapsed, and the ventricle on the intact side is normal or slightly larger than normal. Before ventriculography is undertaken, preparation for craniotomy must be made. If a positive result is obtained in the ventriculogram, the abscess must be drained immediately, otherwise the consequences are the same as those which



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follow excessive removal of cerebrospinal fluid at lumbar puncture.

In our series Case III only had a preliminary ventriculogram, which was followed by craniotomy and enucleation of the abscess. Groff and Grant[12] make extensive use of ventriculography when localization cannot be made clinically, and four out of seven cases had a preliminary ventriculograph.

### Electro-encephalography

As a method of investigation of intracranial lesions the electro-encephalogram (E.E.G.) will probably come more and more into its own. At the present time the instrument is delicate, and does not lend itself to being moved from place to place, factors which greatly restrict its use. Details of its uses and interpretation of results may be seen in Walter's paper,[24] but briefly the instrument records changes of electrical potential in the brain, two electrodes being placed on the scalp and connected with a galvanometer. By this means it is possible to detect certain electrical rhythms in the brain. In a normal person the only discernible rhythm is the Alpha rhythm (the original Berger rhythm), which is best seen when the mind is at rest and the eyes shut. Walter showed that it is possible to locate an abnormally slow discharge from the neighbourhood of tumours which affect the cerebral cortex either by infiltration, expansion or interruption of fibre tracts: these he termed the Delta waves. Walter took over 200 consecutive E.E.G. readings of patients who were found subsequently to have a cerebral tumour, and in every case where Delta waves were seen a tumour was found near by, and this has been confirmed by other workers. As Walter himself remarks, "There is no doubt that in the majority of cases neoplasms of the cerebral hemispheres may be located as accurately by electro-encephalography as by any other means, and with less discomfort to the patient than when air replacement is resorted to." With raised intracranial pressure the Delta waves tend to be very widespread and of low potential. Cerebellar tumours are not so easily localized. They can, however, sometimes be detected and the side of the lesion determined.

In abscesses of the brain the E.E.G. is often of special use, for the primary septic focus may bring the patient to such a low state of health that neurological examination is difficult, air replacement is dangerous, and ventriculography liable to



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disseminate the infection. In these cases the E.E.G. has given particularly satisfactory results. The chief drawback is often that a patient is too ill to be removed to a suitable centre.

One case of ours was submitted to electro-encephalography and the results are most informative, as it was carried out twice before and once subsequently to operation. On the first occasion, December 29th, an abnormal focus of cortical discharge in the form of well-defined Delta waves of low potential was found, indicating a comparatively benign but destructive cortical lesion in the right temporo-parietal region and extending back therefrom. The second examination was carried out on February 15th, the day prior to operation. A marked change had occurred since the previous examination, the original focus now being low down in the right frontal lobe and the waves ten times as great in potential as formerly. There were also small diffuse Delta discharges, indicative of raised intracranial pressure. The large Delta discharge was probably due to a comparatively acute œdema of the cortex. The third examination was carried out on March 5th. This showed the whole of frontal, parietal and temporal areas to be quite free from abnormal slow discharges. There were, however, definite signs of Delta activity in the occipital region. This was thought to be due to interruption of the visual projection tracts caused either by the abscess or by the trauma involved in its total removal.

**Treatment**

For the purposes of treatment brain abscesses are conveniently divided into three groups, acute unlimited abscess, subacute localized abscess which though limited has not developed a thick walled capsule, and chronic or encysted abscess.

Case X is an example of an acute unlimited abscess. Cairns and Donald[3] have stated, " It is impossible to drain an area of acute purulent encephalitis, and it is probably unwise to disturb an abscess before it is localized." The difficulty arises, as is not infrequently the case, when the march of events is more rapid than the process of localization. The surgeon's conscience, and it is more often the aural than the cranial surgeon who meets the acute abscess, forces him to act when the condition is beyond the power of surgery to control.



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# Brain Abscess

As yet sulphanilamide and sulphapyridine have not been in use long enough to determine their ability to tide over an acute abscess into an abscess which is localized and amenable to surgery. Cases III, XI, and XII in this series received chemotherapy. In the one the drug was stopped owing to reactions, and in the other two it was in use for the treatment of intercurrent infection, and the time relation was too out of step with the treatment of the brain abscess to have had any real bearing on the issue.

Recently Gibb[10] using sulphapyridine and Hamby and his colleagues[13] using sulphanilamide have published successful cases in which these drugs played an important ancillary part. Hubert and Leroux[15] had a successful result with a girl of nine who had a temporo-sphenoidal abscess which was drained, and at the same time a course of neo-coccyl was given with a maximum daily dose of 2 grammes. She had remained well for a year at the time of publication. Frist and Rippy[9] have published a case in which the signs and symptoms of a brain abscess cleared up on full doses of sulphapyridine without operation. The difficulty is to prove that an abscess was actually present. Except in so far as it shows spontaneous recovery the clinical condition described by Symonds,[21] Leegaard,[17] and others under the name of otogenous encephalitis has the same clinical picture as brain abscess. Moreover in this connection Cairns and Donald[3] state, " From post-mortem studies there is no doubt that a cerebral abscess can subside spontaneously, and another abscess can subsequently form rapidly at the same site."

For the successful use of chemotherapy, the infecting organism must be identified, and the dosage adequate. Fortunately in many cases when the frontal sinus or mastoid has previously been opened a culture of the pus has already been done. A dose of three grammes by mouth, or if unconscious two grammes of the soluble drug intramuscularly, every four hours for the first twenty-four hours is necessary to give a satisfactory initial concentration. In this connection Whitby[27] points out, " An effective level is only maintained by regular four-hourly dosage and this means that the hours of night must not be omitted." We feel that future advances in the treatment of the acute abscesses depend on the development of chemotherapy.

Horrax[14] suggested that Dandy's[6] method of simple tapping



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may have a place as a means of lowering pressure. Dandy himself designed his method as a treatment for chronic abscess, but it has not found favour in the hands of others. In Dandy's original description the smallest workable opening in the skull and dura is made, and the needle left in until it ceases to drip. The abscess is neither aspirated nor irrigated.

Vincent[23] treats acute brain abscesses by a two-stage method. Firstly, following localization by ventriculography, a large osteoplastic flap is turned down and the abscess aspirated by needle puncture through the dura mater without incision. The osteoplastic flap is then replaced. Some three to four weeks later the flap is re-elevated, and the dura incised. The abscess, which is now encapsulated, is removed *en masse*, using electro-cautery and suction in the process of dissection. The osteoplastic flap is replaced and sutured without drainage. Vincent's operative technique in acute brain abscess appears so far to give the best results.

It is not out of place here to point out that when any case of brain abscess is being treated expectantly in the hope that further localization will take place, and this applies particularly to cerebellar abscesses, the ward staff must be warned of the danger of sudden respiratory failure. S.C.S. entered a ward one day when a colleague's case of suspected cerebellar abscess was under observation. The nurse mentioned that his respiration had been rather peculiar during the previous hour or two. Immediate inspection showed that he had stopped breathing completely, presumably a few moments before. It was quite easy to establish respiration again by alternate compression and relaxation with the hands on the lower part of the thorax, and to maintain it without moving the patient in bed, until he could be got into the theatre and an intra-tracheal tube passed.

Subacute or localized abscesses are mostly contiguous to frontal sinus or mastoid infection. The local condition has either not been opened, or only recently, and active drainage is, therefore, in progress; it is usually better to approach the brain abscess across the infected sinus or mastoid—that is, along the actual track of infection—and drain both abscess and focus simultaneously. However, there is no need to follow slavishly this suggestion and each case should be treated on its merits. Cairns[3] makes the important statement : " When intracranial complications are suspected neurological investigation should



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take precedence over mastoidectomy, since it will tell more about what is happening inside the dura than can be learned from looking at a small area of dura exposed in the mastoid region."

In the open drainage of brain abscess details may vary according to circumstances of the case, and the preferences of the operator, but the general conditions remain the same. These are, exposure of an adequate area of dura, minimum disturbance of the track and adhesions, a drainage tube of adequate size and rigidity to avoid blocking, avoidance of antiseptics, and a minimum of interference with the tube and dressings after operation.

An osteoplastic flap was used in Cases I and II. It is the method of choice for the exploration of metastatic abscesses and many cases of contiguous abscess. It gives a much wider field than a trephine opening, and the wide area of dura exposed allows insertion of the needle through the dura at more than one point. Failure to take advantage of this in Case II resulted in failure to locate the abscess, though in this particular case it would not have saved the patient. Having located the abscess, the dura is opened directly over the abscess and a corresponding hole made through the osteoplastic flap, so that the drainage tube passes vertically into the cavity from the surface after replacement of the osteoplastic flap. When exploring it is very important to keep the needle lumen patent, as if it becomes inadvertently blocked the abscess will be missed.

Smirnov[20] has designed an attachment arm to fit a 2 cc. "record" syringe, which is attached to the syringe barrel by a spring clasp and to the needle by a collar; affixed to this collar is a knife. The whole arm moves upwards and downwards on the barrel and needle of the syringe, carrying the knife along the surface of the needle. The needle with the syringe coupled to it, and the arm with attached knife slid up to the off position at its proximal end, is inserted through the dura. If pus is found the knife is slid down to the point of the needle by moving the arm attachment, thus cutting a path for a drainage tube. The syringe and its needle can be removed by springing the clasp band, by which the arm attachment is held to the barrel of the syringe, thus leaving the knife *in situ* to act as a guide for drainage tube or further exploration.

A useful exposure in the frontal area is given by continuation of the usual frontal sinus incision across the nasion and



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along the supraorbital ridge of the opposite side, combined with extension along the supraorbital ridge on the same side, and extending upwards at the fronto-temporal angle. This allows complete inspection of the frontal sinus, search for a fistula in the posterior wall—if necessary, obliteration of the whole posterior wall—and adequate exposure of the frontal bone for a separate trephine opening if required, after packing off the frontal sinus area. This incision was used in Cases V, VI, and VII.

If a temporo-sphenoidal abscess is to be drained in the course of a mastoid operation, it is necessary to do the radical operation and remove the whole of the tegmen tympani : it is at this point that the track of the abscess will be found if present.

Sydney Scott[19] in his historical survey remarks that both Balance and Preysing came to the same conclusion that cerebellar abscesses were more easily drained through the mastoid than through a separate trephine opening. While a radical mastoid is necessary to give adequate exposure for trans-mastoid drainage of a tempero-sphenoidal abscess, an extensive Schwartze operation will give an adequate exposure of the posterior fossa and cerebellar abscess, if the condition of the patient demands speed.

Whether the infection arises from extension of a perisinus abscess (Case XIV), or from the petrous bone secondary to petrositis (Cases XV and XVI) or secondary to labyrinthitis (Case XIII), the track leads into the posterior fossa by a precise route through an area of posterior fossa dura which lies between the lateral sinus behind and laterally, the petrous bone with the labyrinth in front, and the dura of the middle fossa above. If the bone covering this area of dura known as Trautmann's triangle, is removed, the route of infection into the cerebellum is laid bare. This area is well shown in a diagram by Watkyn Thomas,[25] Fig. 191. The large amount of bone that may be removed from the petrous in this area without damage to the labyrinth is shown in Fig. 4.

Although this route into the cerebellum gives the easiest access, the maintenance of satisfactory drainage is not such an easy problem. With this in view the exploring canula with round-ended stilette was made to the design of S.C.S. for use with the home-made 8 cm. metal drainage tube, shown with it in Fig. 6. It is 16 cm. long, the distal 8 cm. only is graduated, and the metal tube is held in position at the proximal end of



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the exploring canula. When pus is found, the drainage tube slides down over the canula into the abscess with the minimum disturbance of surrounding tissues. The tube is stitched to the skin edge by a stitch passing through one of its lateral holes. It was used with successful results in Case XVI (Figs. 3 and 4).

Whatever form of drainage tube is used, it is important to build up strips of gauze round the tube so that the covering dressings do not press on the drainage tube, thereby blocking or kinking it. Cairns[3] recommends covering the top of the tube with gutta percha to prevent the gauze dressings becoming adherent to it.

In most of this series of cases in which open operation has been carried out, vaseline dressings have been used. This is preferable to the use of the customary antiseptics, and is supported by the work of Dorothy Russell and Falconer[18] which demonstrated the destruction of cerebral tissue that accompanied the use of these antiseptics. They found that " Proflavine sulphate in 0·1 per cent. isotonic solution buffered to pH 6·2 is not substantially more injurious to the brain than normal saline." This is, therefore, to be preferred when the use of an antiseptic appears to be indicated. It is better to leave the dressing for a week and then to dress the case at approximately weekly intervals in the operating theatre. The tube is left in until pushed out by the gradual obliteration of the cavity. It keeps the dressings together better if plaster bandages are used to make a plaster headcap. The only disadvantage of the plaster cap is that patients sometimes complain of its weight.

### The Chronic or Encysted Abscess

These have a long history, a thick capsule, and give the best surgical results. Most traumatic, some metastatic and a minority of nasal and aural brain abscesses fall into this category. The local primary infection has been dealt with, or is at least quiescent and does not demand immediate attention ; therefore, the cerebral abscess should be explored by a clean cranial operation.

An osteoplastic flap is turned down, the brain tissue immediately over the abscess is removed, and the abscess is enucleated in the same way as a tumour is removed. The flap is then replaced, usually without drainage, but if contamination



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of the surrounding arm has occurred, a thin rubber sheet or gutta percha drain can be brought out through an opening made in the flap at the most convenient point for drainage. During the process of enucleation the field must be well protected with saline pads and strips of ribbon gauze between the abscess capsule and the brain tissue as dissection proceeds. If the abscess is large it is sometimes helpful to aspirate it, to reduce its bulk during the process of dissection.

Cairns[3] favours this procedure. Groff and Grant[12] state, " The complications of cerebral herniation, cerebral fungus, and an alarming, increasing, intracranial pressure are avoided. The constant wound care necessary when drainage methods are employed is eliminated." Enucleation was performed in three out of their series of six recoveries. Vincent[23] likewise uses this method. Horrax,[14] although his three cases in which this operation was carried out were successful, says it " must always be regarded as a potentially dangerous procedure," and prefers his method of marsupialization in which the abscess is opened after its walls have been anchored by sutures, and the abscess capsule is stitched to the galea round the margin of the opening through the cranium. Bucy and Haverfield,[2] after removing the overlying brain tissue by suction or electric cautery, pack the space between the cranium and the abscess with gauze, which is removed in two days' time. A week later the abscess is found to have migrated as a whole to the surface, and the surrounding subdural space to be shut off : the abscess is then opened and drained with large rubber tubes. The disadvantage of this method is the necessity of a week's delay before the abscess is opened. Kahn[16] prefers the two-stage method. His objection to total removal is that raw brain surface may be left in contact with the dura and prefers to marsupialize part of the abscess wall at the second operation. By the injection of colloidal thorium dioxide into the abscess cavity he was able to illustrate the migration of a deep abscess to the surface in the seventeen-day interval between the first- and second-stage operations. Vincent's pupils, David and Thiébaut,[7] record the successful removal *en masse* of an abscess in the right frontal lobe which weighed 100 grammes, and incidentally overcame the technical difficulties arising from adherence to the falx and cribriform plate.

Whatever method is used it is essential to remove the abscess capsule from the intracerebral cavity, for Cairns[3] says,



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" The wall, if left, persists in the brain as a foreign body which produces gliosis and is likely to result in further abscess formation later." In Case III Mr. Wylie McKissick enucleated the abscess with recovery of the patient. In Case XII the abscess was successfully dissected out as a whole, but unfortunately further spread of disease in the mastoid resulted in spread to the posterior fossa and killed the patient. It was at least encouraging to find aseptic healing of the cranial wound at autopsy eleven days after operation.

We consider that exposure of the abscess through an osteoplastic flap and its removal as a whole is the operation of choice for thick-walled encysted abscesses.

### Post-operative Complications

*Oedema* may be primary or secondary. In the first type the expanding lesion progresses in spite of drainage; it is particularly likely to occur after ill-timed interference in an acute abscess, but may occur also in a subacute abscess as in Case I of our series. Secondary œdema comes on a day or so after operation with reappearance of symptoms. It occurred typically in Case XI and, as is usual, passed off without any interference in twenty-four hours.

*Meningitis* arises either from the tract of entry into the abscess cavity from failure of the adhesions to shut off the area, or by spread of the infective process into the ventricles. Case X illustrates the spread of an ascending meningitis along the fissure of Rolando from an abscess of the temporo-sphenoidal lobe, and Case VI illustrates the spread of infection into the lateral ventricle from an abscess in the frontal lobe. It is worth recording here that Groff and Grant[12] twice opened the lateral ventricle accidentally, when stripping away the abscess wall, without ill effects.

*Rise of temperature* after operation is usual; it was seen characteristically in Case XI. None of our cases showed a rise of temperature prolonged as long as three weeks, as Cairns[3] has said may happen.

*Loculation of pus* is most likely to occur in a multilocular abscess, especially if encapsulation is not complete. Case XVI illustrates this complication and re-exploration was needed twice before the cerebellar abscess was completely drained and the patient began to recover.



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*Further spread of infection* occurs in acute abscesses whether drained or not (Case X), but may also occur in subacute abscesses. Case XIV showed a secondary fatal spread three weeks after what had first appeared to be satisfactory drainage of a cerebellar abscess.

*Multiple abscesses* may be present. This is frequently the case in metastatic hæmatogenous spread. In Case IV a second abscess was found post mortem in the contra-lateral frontal lobe.

*Focus of infection* persists and kills the patient. In Case XII we failed to control the infection in the petrous bone.

*Recurrence of the abscess* is due either to incomplete drainage or failure to remove the whole capsule. Ira Cohen[4] reports a case of cerebellar abscess which recurred and necessitated reopening three years later. Yet a second recurrence occurred six years after this reopening. The patient remained well at the time of publication six years after this third drainage.

*Change in character* appears to be rare, but Ira Cohen[4] demonstrated a case of a boy of 11½, who four and a half years before had had a temporal lobe abscess drained. He states, " As time has gone on the problem of management has increased and the character of the boy has changed, according to his parents, from what it was before the operation." S.C.S. can recollect one child who, following recovery from a temporo-sphenoidal abscess, developed fits of temper, according to the statement of the parents.

*Persistent speech defects* of minor degree have been recorded by Groff and Grant[12] and Ira Cohen.[4]

*Epilepsy*. Cairns[3] got this complication in four out of ten recoveries, but only in one case did it persist after discharge from hospital. Groff and Grant[12] have reported a case which had three major convulsions in the six months after operation, but none subsequently, and another with persistent sensory Jacksonian attacks in the contra-lateral arm. Ira Cohen[4] has reported a case which developed convulsive seizures five years after drainage, and another which developed Jacksonian fits in the contra-lateral arm and leg six years after. Both were improved following excision of scar tissue. Recently Asherson[1] has reported a case of temporo-sphenoidal abscess in which epilepsy began eighteen months after operation.

*Cranial nerve lesions*. One case described by Cairns[3] developed a squint, but this eventually cleared up. He



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considered that it was due to a patch of meningitis in the cisterna interpeduncularis. A case described by Sydney Scott[19] retained a trace of facial paresis. Case XI in our series had a slight weakness of the face, which took a long time to clear up. Just as the facial nerve lesion is so often the first focal sign in a temporo-sphenoidal abscess, so it is usually the last to disappear.

*Persistent defect of the visual fields.* One case described by Cairns and Donald[3] (Case IX), one case described by Sydney Scott[19] (Case VII), and a case recently reported by Asherson[1] had a permanent homonymous hemianopia. Fortunately it appears that this is a rare occurrence.

*Persistent traces of the original hemiplegia.* In a case of superficial abscess described by Symonds[21] weakness of the right arm and leg was still present six months after operation. In a case described by Cairns and Donald[3] (Case VIII) a slight defect of postural sensibility in the right foot and hand persisted. Two of the cases reported by Groff and Grant[12] had some persistent weakness of the contra-lateral arm. Case XI in our series had for some time a slight weakness of the right-hand grasp as well as of the right side of the face. It appears from the recorded cases in the literature that this complication occurs only infrequently.

In conclusion we should like to express our indebtedness to all those colleagues with whom we have worked in the handling of the cases we have reported, and to thank the Chief Medical Officer of the London County Council for his permission to include Cases IV, VII, IX, and XV.

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