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OTOGENIC BRAIN ABSCESS

By C. A. HUTCHINSON (Bath)

THE subject of Otogenic brain abscess is one of great interest to the otologist. Probably no other condition offers such difficult problems in diagnosis, particularly as regards differential diagnosis; and medical literature has been flooded with such a plethora of details and widely divergent views, that there is real danger of our not being "able to see the wood for the trees". Accordingly it is felt that there may be justification for a brief survey of the views held by various authorities and an attempt to marshall the salient points which they stress into some sort of co-ordinated order.

Classification

The first thing to recognize is that there are several different types of brain abscess; one type, however, to a certain extent merging into another, without any clear-cut distinction.

The two main varieties are :—

(1) Diffuse Encephalitis ("Septic softening").
(2) The localized abscess with a capsule.

There are, however, three varieties of this :—

(*a*) A pus-containing cavity with a ragged wall of softened brain tissue.

(*b*) A cavity with a thin capsule of young fibrous granulation tissue, surrounded by an area of soft, œdematous brain tissue.

(*c*) A cavity, perhaps small in relation to the size of the abscess, enclosed in a capsule of dense fibrous tissue, anything up to an inch in thickness, surrounded by more or less normal brain tissue.

Aetiology

Otogenic brain abscesses may be classified as "contact spread" and "metastatic". The vast majority belong to the first group, and the

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picture which they present is frequently obscured by other coexisting infective complications of middle-ear disease (Meningitis, Infection of the Choroid Plexus and Lateral Sinus Thrombophlebitis); whereas the comparatively rare metastatic abscesses are almost invariably accompanied by other evidence of generalized pyæmia. For the purposes of this article we shall confine our attentions to abscesses of the "contact spread" variety.

Ballance drew attention to the fact that there is usually a "stalk" to be found, representing the inward track of the infection, connecting the bone and the abscess[1]: while the depth at which the abscess lies is to some extent indicated by the facts that when it is sub-cortical, one usually finds bulging of the brain, with pallor, broadening and flattening of the exposed convolutions; whereas, when the abscess lies deeply, these characteristic appearances may be lacking.

As regards the precise route by which infection enters the brain substance: It has been remarked that the most intensive septic reaction is to be found just beneath the point at which the dura is penetrated; and Miles Atkinson[2] showed that, whereas the perivascular route is the one most commonly taken, the venous route is perhaps the one most likely to be followed in connection with lateral sinus thrombophlebitis.

The abscess makes its first appearance in the "avascular zone" of white matter left behind by the central and cortical vessels after passing through the cortex and up through the base of the brain: the earliest stage being a thrombosed capillary surrounded by a cuff of white blood cells. Then follows necrosis of a limited area of brain tissue, followed by pus formation[3]. Secondary abscesses may connect with the cavity of the primary abscess, or be found at a short distance from it without any visible connection. Such secondary abscesses are separated from the primary abscess by cerebral substance which, while it appears normal to the naked eye, is shown histologically to be actually necrosed brain substance[4]. It is probable that secondary abscesses are usually, and that multiple secondary abscesses are always, formed by diffusion along a vascular route. Very exceptionally, a secondary abscess is formed by the spread of suppuration from the pia mater into the brain substance—for instance, in the depths of a fissure[5].

Bacteriology

Piquet and Minne[6] as the result of examining abscess contents found that the infection is usually a mixed one, but that the streptococcus tends to produce a diffuse encephalitis. McFarlan[7], however, showed that the commonest organism present in the localized abscess is the Staphylococcus aureus (some 32 per cent. of cases, and, moreover, 27 per cent. in pure culture). His figures for streptococci were as follows:—

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Strep. pneumoniae	13 per cent.
Strep. pyogenes	8 per cent.
Strep. viridans	4 per cent.
Non-hæmolytic strep.	4 per cent.
Anaerobic strep.	20 per cent.
Unclassified strep.	6 per cent.

and he points out that it is obviously unjustifiable to group all these species of streptococci together. He found, moreover, that, contrary to the erroneous view previously held, encapsulation may occur in the presence of anaerobic streptococci; and he regards anaerobic culture as an essential part of the examination of pus from brain abscess, for anaerobes may be isolated from it; and it is probable that the anaerobic abscess may have originated in a focus of lowered O.R. potential produced by a vascular accident or by aerobic organisms, which later died out[8].

Symptomatology

The symptomatology of brain abscess is not yet clearly defined, and diagnosis is particularly difficult if the infection of the middle ear or mastoid is, or appears to be, insignificant—(when an erroneous diagnosis of cerebral thrombosis or encephalitis is apt to be made)—or if suggestive cerebral symptoms develop soon after a mastoidectomy.

Ballance[9] and Cairns[10] stress the need for careful, complete and repeated neurological examination, whenever intracranial complications are suspected: the latter, indeed, going so far as to say that this examination should take precedence over mastoidectomy. The tendency to a mistake in diagnosis is increased by the facts that symptoms are slight and may be transient, while progress is slow. At the same time, the very transiency of the signs is almost diagnostic[11]. What is certain, however, is the fact that focal symptoms are relatively late in appearing and it is not therefore wise to wait for them. The thing to go by is the general state of the patient; and should this make it seem likely that an abscess is present, it is advisable to explore without delay[12]. Sometimes the onset of an abscess is indicated by an easily distinguishable symptom, such as a Jacksonian fit; more often, however, the precise time of onset is uncertain. The following table, compiled by J. S. Fraser[13], gives a general idea of the most prominent symptoms and their relative frequency.

TEMPORO-SPHENOIDAL ABSCESS

Labyrinth functioning	80 per cent.
Marked Headache	90 per cent.
Vomiting	80 per cent.
Sub-normal or Normal Temperature ..	53 per cent.
Bradycardia	30 per cent.
Tachycardia	47 per cent.
Drowsiness to Coma	12 per cent.
Delirium alternating with Drowsiness ..	24 per cent.

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CEREBELLAR ABSCESS

Distorted balance, Nystagmus and Adiadokokinesis comprise the typical cerebellar syndrome. Vertigo is always, and constant occipital headache almost always, present from the outset[14].

Cairns[15], however, comments that the headache in brain abscess is frequently slight; and though it may be common at the outset, lethargy and drowsiness soon supervene and tend to obscure the picture.

Papillœdema is shown by some patients, but it may be almost or completely absent. Examination of the visual field is most important, but as the co-operation of the patient is needed it becomes impossible once he has become lethargic.

Suggestive transient signs are :—wrist drop lasting a day or two, a little opposite lower facial palsy, and transient ocular paresis.

Lastly, Lambert Rogers[16] has drawn attention to what he calls the " Posterior Fossa Compression Syndrome ", in which the patient collapses and respiration stops, while the heart continues to beat vigorously. This indicates the presence of a sudden subtentorial rise of pressure, due to either hæmorrhage, tumour or abscess; and demands, if it is not to become rapidly fatal, rapid posterior fossa decompression and the simultaneous administration of oxygen, either *viâ* an intra-tracheal catheter or an emergency tracheotomy or laryngotomy.

Differential Diagnosis

The diagnosis, and in particular, the differential diagnosis, may be extremely difficult. A glance at the symptomatology will show the most constant symptom to be headache; and indeed, one may take it that persistent headache following a mastoiditis or a mastoidectomy is a definite danger signal and demands that, at least, a very careful watch should be kept on the patient.

The enormous recent strides in chemotherapy have greatly improved the prognosis in middle-ear disease, but the universal adoption of these drugs has tended to obscure the diagnostic picture and lull us all too easily into a sense of false security. In a brief article by the author in the *Brit. med. J.* for August 2nd, 1941[17] he pointed out how the indiscriminate use of sulphonamides, particularly if in inadequate dosage and over an insufficient period of time, can mask a " Mastoiditis ": several months to some months later there may be signs of the development of some intracranial complication; but thanks to the sulphonamide already used the middle-ear signs may be so trivial at the moment, or forgotten in the past, that great difficulty in diagnosis occurs. Another cause for such difficulty is the pernicious " Mucosus Otitis ", which tends to show a greater incidence of complications than other middle-ear infections,

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and in which the initial clinical course may be so mild that the patient may not even have sought advice.

It should be noted that temporo-sphenoidal abscess may be closely simulated by purulent pachymeningitis following a mastoiditis; and a cerebellar abscess may sometimes be confused with a serous meningitis or arachnoiditis[18]; while both meningitis and cerebral or cerebellar abscess may coexist. A careful history and neurological examination, however, should enable a distinction to be made.

Differential diagnosis mainly lies between brain abscess (cerebral or cerebellar), otitic hydrocephalus and meningitis: other possible alternatives being intracranial tumour and hæmorrhage into the posterior fossa. What therefore are the principle differential points? In all these conditions there tend to be in varying degree, the classical signs of rising intracranial pressure, i.e. headache, vomiting and papillœdema; while Lambert Rogers[19] has drawn attention to the important posterior fossa syndrome, indicating a sudden sub-tentorial pressure rise acting on the more vulnerable respiratory centre: such pressure rise, however by its very suddenness suggesting hæmorrhage rather than the other alternatives.

Certain Diagnostic Inferences which may be drawn from Lesions found at a Previous Operation

(1) The finding of an extradural abscess during a previous operation, in a case which later presents vague cerebral symptoms, should awaken the suspicion either of a localized meningitis or brain abscess; as experience shows that associated intradural suppuration is present in a considerable proportion of cases of extradural abscess.

(2) The presence of osteomyelitis involving the squamous portion of the temporal is always suggestive of an underlying localized meningitis.

(3) Mastoiditis with labyrinthitis is a frequent forerunner of meningitis or cerebellar abscess.

(4) Mastoiditis with labyrinthitis and an associated sinus thrombophlebitis is especially apt to be followed by cerebellar abscess.

Lastly, in the case of mastoiditis the patient does not look sick until the terminal stage of blood-stream infection, although recording high temperatures with or without accented " swings ". He is apt to present an appearance of well-being—apparently too well in proportion to the temperature recorded. This favourable impression is on the whole a good prognostic sign, and as long as the pulse-rate is proportional to the temperature the prognosis remains favourable; but an ascending disproportionate pulse is often the earliest sign of complicating meningitis.

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C. A. Hutchinson

Valuable Diagnostic Points

		Meningitis	*Otitic Hydrocephalus*	*Brain Abscess*
1.	Optic Disc	Usually slight swelling.	Enormous swelling (Several Diopters).	Commonly slight swelling, but more usually only slight venous engorgement.
2.	Temperature	Continuous Hyperpyrexia.	Apyrexia.	In the majority of cases Subnormal or Normal.
3.	General Condition	Irregular attacks of Delirium.	Between attacks of Headache and vomiting, alert, lively and of good appetite.	Dull, constipated, no appetite.

Examination of the cerebrospinal fluid by lumbar puncture and the results of manometric readings are of the utmost value.

	Meningitis	*Otitic Hydrocephalus*	*Brain Abscess*
A.	Moderate to high C.S.F. pressure.	Very high C.S.F. pressure (neighbourhood of 300)	Less high C.S.F. pressure (150-250)
B.	Opalescent to Turbid.	Clear.	Frequently Turbid.
C.	Reduced Chlorides and Sugar in C.S.F.	Normal Composition of C.S.F.	Never normal composition of C.S.F.
D.	Excess Protein; White Cells raised; and frequently organisms.	No excess of Protein or Cells.	Always slight excess of Protein; usually slight excess of Cells (Mainly Lymphos. and a few Polys.; no organisms.

It is evident from the above that the diagnostic value of lumbar puncture cannot be over-estimated.

Two points of definite value for differential diagnosis are :—

(1) The presence of the Gradenigo Symptom Complex—consisting as it does of pain deep in the head, usually localized above the ear and/or deep within the orbit of the same side, accompanied by a low cerebrospinal fluid cell count and at times by paralysis of the external rectus of the homolateral side, coming on as a rule several days or weeks after a mastoiditis—indicates a meningitis secondary to a petrositis and *not* an abscess within the substance of the brain. It should be noted, however, that, occasionally, a brain abscess may co-exist.

(2) If at operation pus is found when a little cut is made in the dura, it is almost certain that there is *not* a brain abscess.

Course and Spread

A spreading abscess tends to make its way towards the ventricle; or rather perhaps, the ventricle filled with cerebrospinal fluid under pressure (due to toxic irritation of the choroid plexus and resultant over-secretion) tends to herniate into the softened brain; in which case the accident, not necessarily fatal, is indicated by the escape of cerebrospinal fluid from the wound[20].

Piquet and Boury[21] pointed out that the progress of an abscess towards the ventricle varies considerably. In some cases the abscess grows slowly

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and the ventricle becomes reduced to a mere slit, or perhaps occluded altogether by the formation of a homogeneous mass containing a few fibrils. In other more quickly growing abscesses, however, an irruption into the ventricle causes sudden death.

Dan McKenzie[22] pointed out that a brain abscess may rupture spontaneously and gradually discharge, either internally into the ventricle or externally into the meningeal spaces; and Cairns and Donald[23] stressed the fact that abscesses may leak into the lateral ventricles or subarachnoid space without producing fatal diffuse leptomeningitis.

In most cases of external rupture the pus travels by the route through which the infection entered the brain, the tissues there being devitalized and in some cases disintegrated. The relief afforded by leakage is rarely sufficient to neutralize the pressure effects of a brain abscess, or to keep its bulk from enlarging, or the infection of the brain from extending. Rupture into the ventricle, it should be noted, may occur in spite of the coexistence of a leak[24].

Lastly, the temporo-sphenoidal abscess is apt to grow to a larger size and to be quickly fatal after rupture, whereas the cerebellar abscess appears to be much more amenable to treatment.

Localization

Localization of the abscess is often very difficult indeed, as focal signs appear comparatively late; in fact the whole of one cerebellar lobe may be practically completely destroyed without there being either Arm Deviation or Past Pointing. A further complicating factor is the drowsiness of the patient, which comes on relatively early, making it still more difficult to elicit localizing signs. Accordingly, whereas McConnell[25] stated that he thought it essential to localize the abscess before anything is done, the general concensus of opinion nowadays seems to be that it is not wise to wait for localizing signs at all but to be prepared to explore the brain with much less evidence than that which has been insisted upon up to the present. It must be repeated that complete and repeated neurological examination, together with careful history-taking, are of the utmost value.

Terence Cawthorne[26] emphasizes the value of investigating cochlear function—for a generalized loss of cochlear function indicates that the lesion causing it does not lie within the brain, but must either involve the VIIIth nerve trunk or be located in the labyrinth; while, on the other hand, an alteration of vestibular function in the presence of a normal cochlear response always means a lesion within the brain tissue, usually in the posterior fossa and often in the brain stem.

Cerebellar abscesses are apt to give earlier indication of their presence[27] than do abscesses in the cerebrum itself, because of the ventricular distension which they tend to produce. Hypotonia and

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ataxia further suggesting such cerebellar localization, particularly in the presence of the typical cerebellar syndrome of distorted balance, nystagmus, and adiadokokinesis.

The value of nystagmus itself for localizing purposes is very slight. Vertical nystagmus is said to be always of central origin and is also seen in a brain stem lesion. Lastly, a brain stem lesion (e.g. a cerebello-pontine angle tumour) shows slow and coarse nystagmus on looking towards the side of the lesion but fine and rapid nystagmus on looking towards the sound side[28].

Visual aphasia is a very important localizing sign in left-sided temporal lobe abscesses; but does not help in the case of a lesion involving the right temporal lobe, except, according to some authors, in the case of a left-handed patient, when it *may* be present.

Radiological examination is of some use since the abscess very often contains gas, which is revealed by the radiogram[29].

When an abscess is suspected in one or other cerebral hemisphere[30] exploratory ventricular tapping is valuable, for the ventricle is always collapsed on the side of the abscess and is normal or slightly larger than normal on the opposite side. A ventriculogram is of still further assistance, in that it shows the presence of air on the sound side and its absence in the ventricle on the side involved. McConnell, indeed, advocates ventricular puncture in all cases as a preliminary to actual exploration of the brain; particularly in view of the facts that, while in the majority of cases the brain lesion lies on the side of the infected ear, in a small percentage of cases (metastatic spread) it may lie on the opposite side; and there is always difficulty in deciding which side the abscess lie when both ears happen to be infected.

In the present state of our knowledge, however, exploration of the brain is the only way in which a number of these abscesses can be definitely located.

Treatment

A. *The Principles.*—At present the essential treatment is surgical but the prognosis must always be extremely grave[31]. While the primary object is the effective drainage of the abscess, one must always bear in mind the fact that precocious or misdirected surgical measures may well result in fatal complications or, at least, involve risk of serious damage to brain function.

The surgeon, therefore, should start with a clear idea of what he wants to do and should handle the brain gently.

Neumann[32] advocates exploratory puncture as soon as the general state of the patient gives reason for suspecting that abscess is present. The risks entailed by undue delay (rupture into the ventricle; rapid onset of cerebral compression; development of cerebral œdema; and

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in the case of cerebellar abscesses in particular, sudden respiratory failure) being too great. The aim should be to operate in such a way as not only to save the patient's life, but also to prevent permanent crippling: at the same time, in such a dangerous condition it is a mistake to operate too timidly.

B. *Choice of Procedure.*—First try and decide which variety of abscess it is with which you are dealing, for measures clearly right for one variety may be absolutely wrong for another; and as Neumann points out, each abscess needs its own special method of operation and a uniform treatment can never be successful[33].

Cairns[34] states that it is doubtful whether abscesses in the acute stage should be operated on, but admits that occasionally this might have to be done. In his opinion the treatment of chronic abscesses (of moderate duration) should be to leave the wall of the abscess alone and drain the cavity. He emphasizes, however, that very chronic abscesses of long duration cannot be treated successfully by drainage, because the thick wall of hyaline fibrous tissue would never collapse completely or be entirely absorbed: while abscesses of such chronicity have a strong tendency to loculation, therefore enucleation should be carried out.

Guns[35] is of the opinion that the treatment of an acute cerebral abscess by aspiration is not ideal and carries a high mortality rate. He considers drainage of a cerebral abscess to be the procedure of choice, and holds that enucleation of the abscess should only be carried out in exceptional cases.

To summarize modern opinion therefore :—

For diffuse encephalitis (type 1) no surgical treatment is likely to be of any use except perhaps a simple decompression. When necrosis with pus formation has occurred (type 2) drainage may be undertaken with some hope of success. For type 3, embracing almost all otogenic cases, drainage is indicated. For type 4 (the very chronic thick-walled abscess), however, the proper treatment is not to drain but to enucleate the abscess and close the wound.

C. *Technique.*—The rules governing the opening of the abscess are (1) not to interfere with any protective adhesions of dura, arachnoid and pia, which may have already formed around the region through which the abscess is to be opened; and (2) to deliberately cause adhesions to form round it, should none already exist. If the "stalk" can be found, representing the inward track of the original infection and connecting bone and dura, it obviously affords the ideal route of approach: but if no "stalk" can be found the transarachnoid route must be adopted. In either case the bony opening need not be a large one, as the evacuation of pus will be followed by a fall in intracranial pressure; moreover, as Bozzi[36] points out, if a wide dural opening be made some encephalitis is set up and herniation of the brain is apt to follow.

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There is some difference of opinion as to the best technique to adopt for exploring for abscess. For instance, Sargent[37] advised against the use of trocar and cannula ; since these may pass by the capsule or, on the other hand, may fail to penetrate it but push it inwards and cause it to burst into the ventricle. He therefore favoured exploration with a small, flat seeker, which passes with the minimum of force through the softened brain tissue and permits easy recognition of the resistance afforded by an abscess capsule ; and if by any chance it penetrates into a thin-walled abscess, pus at once flows out along it. Cairns, on the other hand[38], reminds one that one must be prepared to explore over a wide field and therefore prefers exploration with a brain needle.

As regards the technique for opening and draining the abscess once it has been located and its depth ascertained, there is again some disagreement. The older school were in favour of tying off such cortical vessels as may be necessary and then incising the abscess cleanly with a sharp scalpel. Cairns, however[39], prefers to employ close drainage for relatively deep-seated abscesses as are most temporo-sphenoidal otogenic ones. When the abscess, however, is very near the surface he enlarges the bony and dural openings and incises the abscess as usual with a scalpel ; for close drainage he inserts a rubber catheter ; but sometimes the tip of this will not penetrate the abscess capsule. In this case, if the condition of the patient is not extremely urgent, it is best to leave the catheter tip in contact with the capsule for a few days ; then when the next dressing is done it will be found that some capsular erosion has been produced and it is possible to get in. If, however, the patient's urgent condition in the first place indicates immediate drainage, a firmer catheter or a stylet-strengthened one should be used.

The above procedures, however, only apply when exploration and drainage are done in the region of a " stalk ". When the transarachnoid route is adopted one should remember that with the evacuation of pus the resultant fall in pressure will be immediately followed by a collapse of the brain tissue from the surface, and the consequent opening up of the sub-arachnoid space : a two-stage procedure is therefore advisable. At the first stage the sub-arachnoid space is shut off by packing round the selected area with gauze strips (plain or moistened with 1 per cent. tincture of iodine) and leaving this *in situ* for 48 hours ; (if necessary aspirating some of the abscess contents at the time). The second stage of actually incising and draining the abscess can be then carried out with a far greater margin of safety.

The precise material for the drainage tube is of little importance. Rubber is rather too bulky for the size of the bore but it serves the purpose well enough. Aluminium or celluloid tubes have the advantage of larger bore ; while decalcified bone tubes have their advocates, including Macewen. The tube should be sutured to the edge of the dura or the

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skin. Lemaître and certain Danish clinics employ filiform drainage tubes: these work well at first but tend to become insufficient during the course of treatment.

The healing of an abscess must not be looked upon as a definite condition; there is not sufficient tissue material for the solid cicatrization of any great defect in brain substance, so that it is possible for one part of the resulting cavity to heal as a solid mass, while another part forms a cyst; and sooner or later, perhaps years after, this may become reinfected by an acute exacerbation, and there occurs what must be regarded as a relapse of the abscess[40].

When an abscess has been properly evacuated and drainage established the intracranial pressure falls, provided no complication, such as meningitis, encephalitis or a second abscess, exists. The appearance of a "Fungus Cerebri", however, means that the intracranial pressure has not been relieved. A "Fungus" must be clearly distinguished from a "Hernia Cerebri", for, whereas the latter means a protrusion of healthy brain, still covered by the pia, arachnoid and skin, with its circulation and functions unimpaired, the "Fungus" is a protruding mass of strangulated, necrosed and functionless tissue indicative of comparative failure to relieve intracranial pressure. If the brain protrudes persistently or increasingly, either the abscess is insufficiently drained or some complication (such as a second abscess or meningitis) may be present. It is unwise, however, to interfere locally too soon, as there is always some œdema, which tends to push the tube out, and this period can be tided over by withdrawing fluid from time to time by lumbar puncture[41].

After-Treatment

The after-treatment of acute or chronic abscesses is the same, whatever method of drainage be employed. Dressings should only be done at long intervals; it being borne in mind[42] that meddlesome interference after evacuation of the abscess (too frequent dressings, washing out with antiseptic, and packing the abscess cavity with gauze) all tend to produce the very thing it was desired to avoid. The first dressing should be left undisturbed for as long as possible, usually a week to ten days. It should be done in the operating theatre and by the surgeon who drained the abscess. The tube is not removed at dressing, but must be kept clean by very gentle syringing. It should be retained in place at least until all tendency of the brain to herniate has disappeared and then shortened very gradually before being finally discarded. The next important factor is prolonged rest in bed.

Complications during After-Treatment

Rise of temperature is almost invariable. The really important post-operative complication is post-operative œdema. If the tube had gone

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into the abscess cavity in the first place and œdema subsequently develops the right thing to do is to leave the patient alone and not interfere. Squints seem quite common after abscess but apparently are not of serious importance. Fits during the first week of convalescence may occur. Undoubtedly epilepsy may be a disappointing complication of an otherwise successfully treated brain abscess. It has probably according to Cairns nothing to do with the actual treatment[43] but is due to a pre-existent inherent tendency. Lastly, the partially subsiding abscess during convalescence should be most carefully watched for it is likely to erupt rapidly like a volcano.[44]

In conclusion, it should be stated that the question of chemotherapy in connection with the treatment of otogenic brain abscess has been purposely omitted since the whole question is still more or less in the experimental stage. It is certain, however, that chemotherapy will come to play an increasingly important part and that in the future we may confidently look forward to results which could never have been hoped for or dreamed of in the past.

BIBLIOGRAPHY

[1] BALLANCE, Sir CHARLES, quoted by Sargent when opening a Discussion in Section of Laryngology and Otology at Annual Meeting of B.M.A., Cardiff, 1928.

[2] MILES ATKINSON, E., speaking at a Discussion in Section of Otology, R.S.M., Feb. 7th, 1930 (*Brit. med. J.*, Feb. 15th, 1930).

[3] PIQUET, J. and BOURY, *Ann. Oto-laryng.*, Nov., 1936, 1,113.

[4] PIQUET, J. and MINNE, J., *Ann. Oto-laryng.*, Oct., 1936, 993.

[5] PIQUET, J. and BOURY, *ibid.*

[6] PIQUET, J. and MINNE, J., *ibid.*

[7] MCFARLAN, A. M., "The Bacteriology of Brain Abscess", *Brit. med. J.*, Nov. 20th, 1943, 643.

[8] FILDES, 1927, quoted by McFarlan, *ibid.*

[9] BALLANCE, Sir CHARLES, speaking at a Meeting of Section of Otology, R.S.M., March 2nd, 1934 (*Brit. med. J.*, March 10th, 1934).

[10] CAIRNS, H., speaking at a Meeting of Section of Otology, R.S.M., March 2nd, 1934 (*Brit. med. J.*, March 15th, 1934).

[11] MILES ATKINSON, E., speaking at a Meeting of Section of Otology, R.S.M., March 2nd, 1934 (*Brit. med. J.*, March 15th, 1934).

[12] Prof. NEUMANN, J., speaking at a Meeting of Section of Otology, R.S.M., Feb. 7th, 1930 (*Brit. med. J.*, Feb. 15th, 1930).

[13] FRASER, J. S., speaking at a Meeting of Section of Otology, R.S.M., Feb. 7th, 1930 (*Brit. med. J.*, Feb. 15th, 1930).

[14] Prof. NEUMANN, H., *ibid.*

[15] CAIRNS, H. and DONALD, C., *J. Laryng. and Otol.*, Feb., 1935, 73.

[16] LAMBERT ROGERS, *Brit. med. J.*, 1933.

[17] HUTCHINSON, C. A., "Chemotherapy in Acute Middle-Ear Disease, Masked Mastoiditis" (*Brit. med. J.*, Aug. 2nd, 1941).

[18] CAIRNS, H., speaking at a Meeting of Section of Otology, R.S.M., Feb. 7th, 1930 (*Brit. med. J.*, Feb. 15th, 1930).

[19] LAMBERT ROGERS, *ibid.*

https://doi.org/10.1017/S0022215100045291 Published online by Cambridge University Press

[20] SARGENT, Sir PERCY, opening Discussion in Section of Laryngology and Otology at Annual Meeting of B.M.A., Cardiff, 1928.
[21] PIQUET, J. and BOURY, *ibid.*
[22] MCKENZIE, D., *J. Laryng. and Otol.*, Dec., 1933, 797.
[23] CAIRNS, H. and DONALD, C., *ibid.*
[24] MCKENZIE, D., *ibid.*
[25] MCCONNELL, A. A., speaking at a meeting of Med. Section of Roy. Acad. Med. in Ireland on " Diagnosis of Brain Abscess ", Dec., 1934.
[26] CAWTHORNE, T., speaking at a Discussion on " The Otologist's part in the investigation of Brain Lesions " in Section of Otology, R.S.M., May 5th, 1939.
[27] SARGENT, Sir PERCY, *ibid.*
[28] CAWTHORNE, T., *ibid.*
[29] CAIRNS, H., speaking at a Meeting of Section of Otology, R.S.M., Feb. 7th, 1930 (*Brit. med. J.*, Feb. 15th, 1930).
[30] CAIRNS, H. and DONALD, C., *ibid.*
[31] SCOTT, SIDNEY, speaking at a Meeting of Section of Otology, R.S.M., March 2nd, 1934 (*Brit. med. J.*, March 10th, 1934).
[32] Prof. NEUMANN, H., *ibid.*
[33] Prof. NEUMANN, H., *ibid.*
[34] CAIRNS, H., speaking at a Meeting of Section of Otology, R.S.M., March 2nd, 1934 (*Brit. med. J.*, March 10th, 1934).
[35] GUNS, *Scalpel, Liége*, Dec. 26th, 1936, 537.
[36] BOZZI, E., *Ann. Oto-laryng.*, Feb., 1937, 98.
[37] SARGENT, Sir PERCY, *ibid.*
[38] CAIRNS, H., *ibid.*
[39] CAIRNS, H., *ibid.*
[40] Prof. NEUMANN, H., *ibid.*
[41] SARGENT, Sir PERCY, *ibid.*
[42] MILES ATKINSON, E., *ibid.*
[43] CAIRNS, H., *ibid.*
[44] CAIRNS, H., speaking at a Meeting of Section of Otology, R.S.M., Feb. 7th, 1930 (*Brit. med. J.*, Feb. 15th, 1930).

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