DIFFUSE SUBDURAL SUPPURATION

By PHILIP H. WOOD (Newcastle)

DIFFUSE suppuration within the subdural space is probably the least known of the intracranial infections that may complicate sinusitis or otitis media.

Pus spreading within the preformed space between dura and arachnoid has been described under a variety of names including Cortical abscess (Blegvad and Mygind), Meningitis circumscripta (Lemoyez), Purulent internal pachymeningitis (Blegvad), Intra-pia-arachnoid abscess (Eagleton), Phlegmonous meningitis (Witzel), Subdural empyema or abscess (Kubik and Adams). Subdural empyema seems the best of the terms and is the most generally accepted.

Receiving scant recognition in rhinological literature, subdural empyema is generally regarded as a rare and fatal condition, clinically indistinguishable from other forms of intracranial suppuration. This paper, based upon a study of eleven personally treated cases and of the literature, purports to show that this condition is in fact far from unusual, that it can usually be diagnosed with confidence and that it can be treated with success.

Historical Note

Subdural empyema was originally described as a complication of head injury by Guthrie (1847). Practically all subsequent cases have arisen as a result of middle-ear or sinus infections, and of these the first was published by Ceci and Onetti (1889). MacEwen (1893) reported four cases in his famous monograph *Pyogenic infections of the Brain and Spinal Cord* but gave scant clinical data.

Blegvad (1910) collected forty-four cases and he recognized that infection spread from the ear either by direct extension or by thrombophlebitis. The clinical syndrome was first formulated by Hecquet (1934). He described the essential features as acute or chronic otitis with evidence of systemic involvement as a background on which signs of meningeal irritation, increased intracranial pressure and focal cerebral involvement develop rapidly.

There is no good explanation of the fact that whereas practically all cases cited up to 1934 have followed aural infection, the majority since that date have followed sinusitis. A review by Kubik and Adams

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(1943) led to wider recognition of the clinical picture, but as yet treatment was unsatisfactory and the condition almost uniformly fatal.

The introduction of penicillin changed the entire course of this infection and the therapeutic principles of systemic and local penicillin combined with prompt surgical drainage were discussed by Schiller, Cairns and Russell (1948). These successes have led to new problems, for the lease of life granted by penicillin may result in chronic smouldering infection.

Case Reports

During the past five years eleven cases of subdural empyema have been personally treated. Cases I to VII came under my care whilst serving in N.W. Europe as surgical specialist R.A.M.C. The remainder were admitted to the Liverpool Regional Neuro-Surgical Unit, whose director, Mr. A. Sutcliffe Kerr, kindly allowed me, as first assistant, to treat them. All cranial explorations were performed under local anæsthesia unless otherwise stated.

Case I

A man of 36 years complained of severe frontal headaches shortly after external frontal sinus operation. A few weeks later he became feverish and vomited, within three days left facial twitching developed and he sank into semicoma.

On admission next day the patient, whose temperature was 101·8° F. and pulse 90, was confused and could do no more than give name and address. There was a little fullness over right frontal sinus and some right ptosis. Examination revealed left hemiparesis with weakness of the left lower face and twitching mouth, slight neck rigidity, positive Kernig and left plantar extensor response.

A provisional diagnosis of leaking right frontal lobe abscess was made. A right frontal burr-hole disclosed thick greenish pus in the subdural space; this was drained, but no intracerebral abscess was found. Penicillin was given systemically, subdurally and intrathecally. The pus grew hæmolytic streptococci, *Strep. mucosa* and diphtheroids. After operation there was a temporary improvement but death in coma occurred on the third day.

Post mortem showed a subdural collection of pus extending over right hemisphere and along falx cerebri but no brain abscess. Localized osteomyelitis had caused sequestration of the posterior wall of right frontal sinus but the dura was intact.

Comment

This case showed the tendency for pus to pool alongside the falx and

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the inadequacy of a single burr-hole to drain an extensive subdural empyema. In retrospect the error of diagnosing a leaking frontal lobe abscess was avoidable. The tempo of events was too fast, and the focal signs too widespread for an ordinary brain abscess. The orderly pattern contrasted with the sudden fulminating onset of a bursting " silent " abscess.

CASE II

A man of 32 years had undergone several operations for chronic sinusitis. Two days after external frontal sinus operation, severe frontal headache developed followed by fever and increasing stupor. Jacksonian fits then started in the left leg. Next day the patient was semicomatose and his eyes deviated to the right. Temperature was 102° F. and pulse 80. Examination showed neck rigidity and positive Kernig, together with a left sided flaccid hemiparesis, partial in the arm but complete in the leg.

A right subdural empyema was drained by burr-holes in posterior parietal and fronto-temporal regions. The medial face of the right hemisphere was explored through a para-sagittal burr-hole sunk ½ inch from midline in coronal suture. Swelling of the cerebral cortex hindered drainage of pus, but this was immediately relieved by spinal drainage although access to the falx remained poor. Penicillin was injected daily into the subdural space *viâ* indwelling soft rubber catheter besides being given systemically and intrathecally. The subdural pus grew non-hæmolytic streptococci.

The patient's general condition and focal signs rapidly improved. Limb movements were completely recovered in two weeks. Subsequently he returned to full duty, symptom free.

COMMENT

Chemotherapy at time of sinus operation did not avert spread of infection to the subdural space. The major concentration of pus alongside falx was localized clinically by the fact that hemiparesis was more profound in the leg than the arm. The value and limitations of spinal drainage in evacuation of subdural pus was demonstrated.

CASE III

A youth of 18 years developed an acute upper respiratory infection after swimming. Increasing lethargy, irritability and feverishness accompanied by severe frontal headache preceded swelling in the right frontal region. When seen four days later he was toxic and ill, drowsy and unco-operative with temperature 103° F. and pulse 100. Œdema over the right frontal bone accompanied a localized tenderness well beyond the sinus. Pus could be seen in the right middle meatus.

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Examination disclosed slight neck rigidity, lower left facial weakness and left hemiparesis with inability to look towards the left. Cerebrospinal fluid pressure was 270 mm., it contained 46 cells per c.mm.

Frontal sinusitis and osteomyelitis with extradural and subdural abscess was diagnosed. A right frontal burr-hole was sunk through infected calvarium and an extradural abscess was evacuated. Following deliberate incision of the dura, pus welled up from the subdural space. It was drained here and through a right temporal burr-hole. Penicillin was given systemically, intrathecally and *viâ* subdural catheter. Both *Staph. aureus* (coagulase positive) and hæmolytic streptococci were grown from extradural pus but only the latter from subdural pus.

Dramatic improvement followed operation and focal signs cleared up within ten days. Incisions healed by first intention. Sinus and bone infection cleared up without further operation.

COMMENT

The acute severe illness and focal neurological signs allowed diagnosis of subdural spread in the presence of extradural abscess and justified deliberate opening of dura through a grossly infected field. Minimal bone surgery with adequate penicillin sufficed to deal with frontal osteomyelitis and achieved excellent cosmetic result. The primary sinus infection subsided and required no direct attention.

CASE IV

Multisinusitis followed a bilateral Caldwell-Luc operation in a man of 50 years with chronic sinusitis and bronchiectasis. Three weeks later he suddenly became confused and lost ability to speak whilst his temperature rose to 104° F. Marked global dysphasia and complete motor aphasia accompanied right lower facial weakness and left hemiplegia. Neck rigidity and positive Kernig were also evident.

A left subdural empyema was drained *viâ* frontal, temporal and parietal burr-holes. The right subdural space was explored with negative result. Penicillin was given as in previous cases, the infecting organism being *Strept. viridans*.

Focal signs other than aphasia cleared rapidly, but recovery of speech was slower. The use of single words was followed by ill-constructed sentences which tailed off unintelligibly. Within five weeks he could hesitatingly recount simple newspaper passages and in two months speech was nearly normal. Thereafter he made complete recovery.

COMMENT

The interesting feature is the occurrence of hemiplegia on the same side as the lesion. This type of false localization is well-known with

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temporo-parietal tumours. The brain-stem is forced against the sharp edge of the opposite tentorium and this pressure interrupts contralateral pyramidal impulses at mid-brain level.

CASE V

A man of 21 years had been ill three weeks with fever, nasal discharge and sore throat when admitted to the medical division. Shortly afterwards he vomited and rising temperature to 102·8° F. accompanied drowsiness and meningeal irritation. Meningitis was diagnosed and penicillin given. The cerebrospinal fluid was cloudy and under pressure. It contained 300 cells per c.mm. but no organisms and sugar content was normal.

Two days later left-sided Jacksonian fits occurred and right abducens palsy was noted. Slight left hemiparesis appeared and increased as stupor deepened to semicoma. When I saw him for the first time next day a diagnosis of right subdural empyema was easy.

Parietal, temporal and frontal burr-holes drained pus from the right subdural space, the left side being clear. Routine penicillin given.

Focal signs cleared within three weeks and recovery seemed complete. Four months after discharge he complained of infrequent but severe headaches and had had two " black-outs " that probably were epileptic fits. Luminal and epanutin given. When last seen three months later he complained of occasional headaches but no further fits. Examination was negative.

COMMENT

Although the initial clinical state simulated meningitis, the cerebrospinal fluid findings were typical of meningeal reaction due to adjacent suppuration, subdural or intracerebral. The occurrence of hemiparesis is unusual in acute meningitis and such focal signs frequently call for ventricular studies to rule out loculated pus.

Intermittent headaches and occasional epileptic fits are not surprising sequelae, but in retrospect, late ventriculography was desirable to eliminate the possibility of a quiescent intracerebral abscess.

CASE VI

A man of 27 years had acute maxillary sinusitis followed by headaches and pain about the right eye. Two weeks later chills and high fever procured his admission to a Belgian Field Hospital. Next day neck rigidity and nominal dysphasia was noted, then increased drowsiness. Some hours later he could be roused to express himself volubly but unintelligibly in true " jargon dysphasia ". Penicillin therapy was commenced. When I first saw him two days later his temperature was

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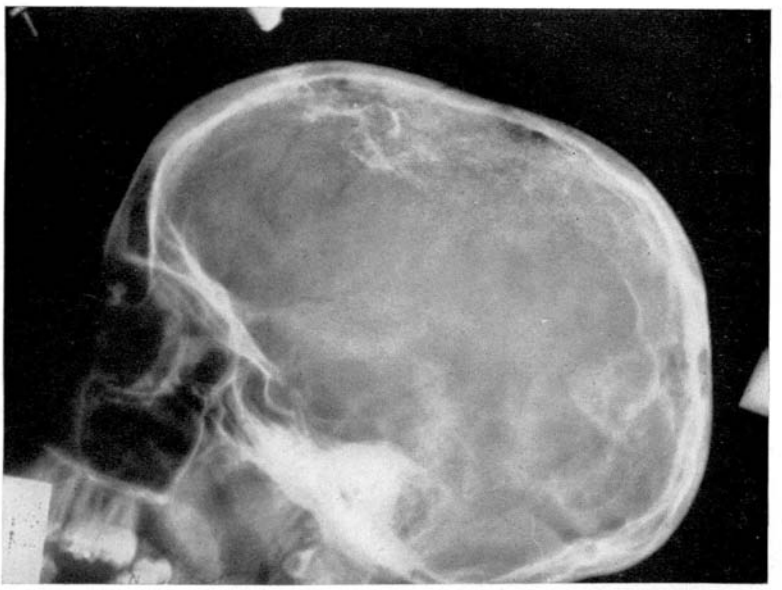


FIG. 1.

A subdural empyema outlined by thorotrast. The wide spread of pus over the cerebral hemisphere and along the falx cerebri is clearly shown.

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103° F. and pulse 105; his mental state varying between stupor and restless irritability. Examination revealed neck rigidity and Kernig's sign, bilateral ptosis and deviation of the eyes to the left. The left pupil was widely dilated but not fixed, whilst the edges of the left optic disc was blurred. The right limbs showed marked paresis, increased tendon jerks and an extensor plantar response.

A left subdural empyema was diagnosed and the patient transferred to a British Military Hospital. During this short journey generalized epileptic fits commenced and status epilepticus supervened.

At operation under pentothal anæsthesia and oxygen, a left subdural empyema was drained *viâ* frontal, temporal and parietal burr-holes. A penicillin tube was left *in situ* and systemic penicillin continued. Clinical improvement was immediate with cessation of convulsions and restoration of ocular movement. Power soon returned in the right arm and improved in the leg.

Over the next four weeks the patient's condition fluctuated, then right hemiplegia and hemianæsthesia suddenly reappeared.

Another burr-hole was made 1 inch to left of mid-line and the subdural space appearing normal the brain was needled towards the falx, 15 cc. pus being obtained. After two days without improvement a second para-sagittal burr-hole was sunk posterior to the first, a further 10 cc. pus being aspirated from alongside the falx.

Transient improvement preceded deterioration and death.

No post-mortem was obtained.

Comment

The initial operation should have included exploration of the medial face of the left hemisphere. It is significant that this case with loculation of pus alongside the falx was already late when first seen.

Case VII

A three-year-old girl developed left otorrhœa due to acute otitis and three days later a rigor with fever of 105·4° F. On admission next day, this child was semicomatose with deathly pallor and profusely sweating skin. Examination showed slight opisthotonos with marked neck rigidity and positive Kernig. Both pupils were widely dilated and reacted poorly to light whilst the right corneal reflex was absent. Right hemiparesis was associated with Brudzinski's sign in the right leg. Lumbar puncture gave turbid cerebrospinal fluid at 310 mm. pressure, the fluid containing hæmolytic streptococci and 1,510 cells per c.mm. Systemic and intrathecal penicillin therapy commenced.

Twenty-four hours later right hemiplegia was severe and disproportionate to the neck rigidity. Subdural suppuration seemed likely and

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called for exploration. Bilateral parietal burr-holes showed diffuse pus in left subdural space and a left fronto-temporal burr-hole was used to assist drainage. Penicillin therapy was now reinforced by subdural administration.

Complete recovery resulted, the acute aural infection resolving without further treatment.

COMMENT

Acute otitis media led to subdural empyema and streptococcal meningitis. Because cortical paralysis was out of all proportion to general signs of meningeal irritation, exploration was undertaken.

The primary focus resolved during penicillin treatment and did not require definite surgery.

CASE VIII

A man of 44 years complained of frontal headaches for three weeks, then his speech suddenly became unintelligible and entirely lost within a few hours. He was admitted to hospital next day. Examination showed percussion tenderness over the left frontal sinus, complete global aphasia, weakness and frequent twitching of the right lower face, right homonymous hemianopia and paresis of conjugate deviation to the right. Early papillœdema was associated with neck rigidity and positive Kernig. Right sided motor and sensory loss was more marked in the arm than the leg, right tendon jerks all brisk and the plantar response extensor.

A left subdural empyema was drained *viâ* frontal, temporal and parietal burr-holes. Routine penicillin was given systemically, intrathecally and subdurally.

Subsequently there was partial recovery of power in the right arm but Jacksonian fits in arm and leg continued. On the third day his condition suddenly deteriorated and he died within the next twenty-four hours.

COMMENT

Unfortunately no post mortem was obtained, nor the infecting organism identified. The complete cortical paralysis of the left cerebral hemisphere was due to diffuse suppuration within the subdural space.

CASE IX

A girl, 14 years old, had frontal headaches for a week before drowsiness and weakness of the right leg developed. On admission two days later she was toxic, drowsy and ill with temperature 103° F. and pulse 120. Right frontal sinusitis was associated with signs of meningeal irritation, high papillœdema, left homonymous hemianopia and loss of conjugate

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deviation to the left. The left face and arm were weak, the latter showing impaired co-ordination and joint-sense despite normal tone. Voluntary power was completely lost in the right leg and both plantar responses were extensor.

Frontal, parasagittal and parietal burr-holes were made. An extradural abscess in right frontal region and a subdural empyema that lay chiefly alongside the right side of the falx were drained. The pus grew *Strept. viridans*. Penicillin was given systemically and subdurally. The right frontal sinus was drained by a fine tube inserted through its orbital plate.

After operation there was immediate improvement and focal signs gradually cleared up. This patient was discharged after six weeks, a slight weakness of the left leg being the only residual symptom. When reviewed four months later she was a normal adolescent, the sole abnormal sign was a slight irregularity of disc margins.

COMMENT

A case of extradural and subdural abscess secondary to right frontal sinusitis. Simple surgical drainage and penicillin resulted in complete recovery. That high papillœdema subsided without secondary atrophy or visual impairment confirms the opinion that it is unnecessary rapidly to resort to subtemporal decompression to save vision.

CASE X

A female of 29 years whose chronic left otorrhœa culminated in the sudden onset of severe occipital headache and vomiting. Examination revealed a left attic perforation with cholesteatoma, typical meningeal signs but no focal defects. Lumbar puncture gave turbid cerebrospinal fluid under pressure. The fluid was sterile and had normal sugar content, but contained 2,500 polymorphs per c.mm.

Sulphatriad and penicillin therapy was commenced. Radical mastoidectomy was performed next day, extensive cholesteatoma and lateral sinus thrombophlebitis being found.

Over the next three days the patient's condition deteriorated as focal signs appeared. These were global dysphasia (complete nominal aphasia and difficulty in comprehension); right homonymous hemianopia; early bilateral papillœdema; difficulty in conjugate deviation to the right; right facial paresis, hemiparesis and hemianæsthesia. The patient was then transferred (3.2.50) to the Liverpool Regional Neuro-Surgical Unit.

A left temporal burr-hole was made but the subdural space appeared normal and the temporal lobe was needled in various directions without result. This negative exploration suggested extensive cortical thrombophlebitis and with definite clinical improvement next day, ventriculo-

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graphy was postponed. Posterior parietal burr-holes were made on 8.2.50 as no further improvement had occurred. A left subdural empyema was now found, drained and a penicillin tube inserted ; previous systemic chemotherapy being continued.

The patient continued to alternate between noisy delirium and semi-coma. On re-exploration (13.2.50) only localized collection of subdural pus over the tentorium was found and 10 cc. pus obtained. This grew *Staph. aureus*, insensitive to penicillin, hence streptomycin therapy was substituted. With continued failure to improve, ventriculography was performed on 18.2.50, but no significant abnormality was demonstrated. A fresh burr-hole was sunk 2 inches behind the left external meatus and the dura opened immediately above the lateral sinus. Œdematous cortex bulged through, so the lateral ventricle was tapped to reduce pressure. After raising the occipital lobe another loculus of pus over the tentorium was evacuated.

Restless delirium and focal signs persisted until leptomeningitis supervened on 5.3.50, when further deterioration rendered the patient completely inaccessible. On 22.3.50 the cerebrospinal fluid contained *Strept. fæcalis* that was completely resistant to penicillin and streptomycin but inhibited by aureomycin. Previous antibiotics were discontinued and aureomycin given by mouth (500 mgr. 6-hourly). Flickering pyrexia quickly settled and the cerebrospinal fluid became sterile. Despite general improvement, jargon dysphasia, hemianopia and hemiparesis persisted. On 24.4.50 recurrent pyrexia and signs of meningeal irritation initiated further deterioration and death occurred a week later.

Post mortem revealed a clean radical mastoid cavity and a thrombosed lateral sinus. The subdural space between tentorium and left occipital lobe was filled with irregular masses of pyogenic granulation tissue containing small pockets of pus ; these extended into adjacent cerebral sulci leading to patchy erosion of the cortex.

Comment

A case of subdural empyema complicating an acute exacerbation of chronic otitis with lateral sinus thrombophlebitis. Delay in diagnosis and drainage was associated with loculation of pus and granuloma formation in the subdural space.

Case XI

A female of 22 years with recurrent nasal polypi was treated by bilateral intranasal ethmoidectomy. During the next few days she complained of severe headaches and then became delirious as the temperature rose sharply, right ptosis and left hemiparesis appeared. Her condition deteriorated rapidly and on the fifth day she was admitted to the Liverpool Regional Neuro-Surgical unit.

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On examination the patient was semicomatose, wasted and toxic. Cerebrospinal fluid was leaking from the left nostril. Constant twitching of the left face and hand gave way to strong clonic contractions of the entire left side. Marked neck rigidity, Kernig's sign, right ptosis and left hemiplegia were present.

The subdural space was explored forthwith. A right sided empyema was drained in frontal, temporal and parietal burr-holes and a penicillin tube left *in situ*. The left side was clear. Penicillin therapy initiated whilst *Staph. aureus* and pneumococci were cultured from the pus. Recovery was dramatic and limb movements were regained within a week. The cerebrospinal rhinorrhœa persisted, however, and pus was aspirated daily from the right subdural space until the tube was removed on the 28th day. During this period penicillin prevented the onset of leptomeningitis and shortly thereafter intracranial repair of the dural fistula was undertaken. After bifrontal craniotomy and extradural approach, the floor of the anterior fossa was exposed back to the optic chiasma. An irregular fracture of the left cribiform plate was thus revealed, and opposite this a dural tear was defined and covered with a free graft of fascia lata.

Uneventful recovery without sequelae resulted.

COMMENT

The infecting organisms are unusual, but gained direct access *viâ* operative tear of the dura. This necessitated intracranial repair to avert recurrence.

Anatomy

Although Logan Turner and Reynolds (1931) portray the dura as being in intimate contact with the arachnoid, the reality of the subdural space is easily demonstrated. Careful incision of the dura displays the arachnoid as a glistening, transparent film enclosing the brain and cerebrospinal fluid. The opposing surfaces of these two membranes are lined by a single layer of epithelial cells and separated by a small amount of serous fluid.

This subdural space is co-extensive with the dura and thus extends a short distance along the cranial nerves emerging from the skull. Along the vertex it is crossed by superior cerebral veins entering the sagittal sinus. Within this space, pus can spread widely over the brain, extending over the upper border of the cerebral hemisphere to gain the opposite side after passing under the free edge of the falx.

Ætiology

Subdural empyema may follow infection of the ear or paranasal sinuses; metastatic spread from a distant focus, or direct implantation. In the present series, six followed frontal sinusitis; two followed antral

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infection ; two arose from otitis media and one case was caused by a dural tear during intranasal ethmoidectomy. Study of other case reports confirms that the primary focus is most commonly in the paranasal sinuses and much less frequently in the middle ear. The high incidence of frontal sinus infection has also been noted by Kubik and Adams (1943), Ray and Parsons (1948). It is readily explained by the arrangement of the perforating veins of the frontal sinus and the proximity of the dura.

Both direct implantation and metastatic spread of infection are rare, isolated cases being reported by Courville and Cutler (1936), Clifton and Werner (1938), Kubik and Adams (1943).

There is fairly general agreement that the infective focus is usually chronic, and acute exacerbation generally preceding intracranial spread.

The factors leading to a subdural empyema rather than brain abscess or leptomeningitis are not understood, but the nature of the infecting organism may be important. In eight of the nine bacteriologically verified cases the organism was a streptococcus. Non-hæmolytic forms were more frequent than the β-hæmolytic type. Case reports seldom describe bacteriological findings, but similar results were recorded by Schiller, Cairns and Russell (1948). This preponderance of streptococci contrasts with the equal incidence of streptococcal and staphylococcal osteomyelitis and with the relative rarity of streptococcal meningitis without focal intracranial infection.

These facts suggest that it is the degree of invasiveness of the infecting organism that leads to subdural empyema rather than to brain abscess or leptomeningitis. Thus streptococcal osteomyelitis tends to spread diffusely through skull and dura, but staphylococcal osteomyelitis localizes. Thus the bacteriological findings in Case III are particularly interesting : an extradural abscess due to mixed staphylococcal and streptococcal infection gave rise to a subdural empyema from which only hæmolytic streptococci could be cultured.

Pathology

Infection generally reaches the subdural space through the wall of the frontal sinus, ethmoidal roof, or tegmen tympani following localized osteitis and necrosis, with or without the development of extradural abscess. Less frequently, infection extends by thrombophlebitis of veins leading from the mucosa of paranasal sinuses or tympanic cavity to large dural sinuses (Courville and Blomquist, 1941).

Thick yellow pus in the subdural space spreads freely over the hemispheral convexity, thence it may pass beneath the falx to the opposite side as in cases reported by Geraghty (1936), Schultz (1946), and Anderson (1947). Untreated, the patient dies before the infection can be walled off, but in the longer period of survival ensured by

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penicillin the pus thickens, fibrinous adhesions form and finally a loculated abscess may be produced. A tendency for the pus to pool alongside the falx was noted in Cases I and II. Since the gravely ill patient lies recumbent and still the exudate may gravitate towards the junction of falx and tentorium. This would explain the sites of loculation noted in Cases VI, X, and those recorded by Schiller, Cairns and Russell (1948).

Histological studies (Geraghty, 1936) show that the usual inflammatory proliferation of blood vessels and macrophages occurs in the inner layer of dura and extends into the fibrin and pus within the subdural space. Thus a shaggy friable membrane develops adjacent to the dura and tends to encyst the empyema. As fibroblasts follow and lay down collagen fibres, this membrane is slowly transformed into a layer of fibrous granulation tissue. Concurrently, this process excites a non-purulent cellular infiltration of the leptomeninges and thrombophlebitis spreading in underlying cerebral veins may give rise to discrete cortical abscesses. These events, culminating in a terminal meningitis, formed the common lot of pre-penicillin cases.

The widespread use of penicillin has profoundly altered the natural progress of this condition. The rapidity and completeness of recovery shows that cerebral thrombophlebitis is quickly arrested and that cortical destruction is now exceptional. Longer survival has introduced a new clinical entity, for now the patient may live whilst infection smoulders on in a chronic granuloma. As in the case reported by List (1950) apparent recovery may be followed by recurrence of headaches and focal signs due to presence of irregular masses of pyogenic granulation tissue in the subdural space.

Thrombophlebitis of cortical veins may lead to the development of a brain abscess, the first signs of which may occur only weeks after complete symptomatic recovery (Schiller, Cairns and Russell, 1948).

Clinical Features

Reference to the cases reported here or in the literature will show that the clinical picture is remarkably uniform. Usually a patient with sinusitis or mastoid disease becomes restless, feverish and complains of headache. This initial stage of intracranial extension is easily missed, the signs being attributed to the more evident local condition, or to osteomyelitis arising from it. Suspicion should be aroused as the headache, at first localized about the site of primary infection, becomes increasingly severe and more generalized. Rapidly the temperature rises to 102° or above, and the patient's restless confusion gives way to apathy and drowsiness. This is the usual state a few days after onset of intracranial extension and examination at this stage reveals some neck rigidity and perhaps a positive Kernig's sign.

Focal neurological signs develop with startling abruptness whilst

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drowsiness deepens to coma. Weakness of an arm or leg appears, frequently accompanied by local Jacksonian fits; within twenty-four hours there is an almost complete flaccid hemiplegia. The eyes are frequently noted to deviate away from the paralysed side, or examination reveals loss of conjugate deviation towards it. Should the dominant hemisphere be involved, the earliest focal sign is often a difficulty of expression rapidly becoming complete inability to speak. Comprehension of speech is retained longer but next day this, too, may be lost. In these circumstances it is not surprising that the examiner may fail to note the sensory loss in paralysed limbs, or the homonymous hemianopia which is so often complete. These may be deduced, however, from the patient's indifference to painful stimuli applied to the affected limbs or lack of response to menacing movements from the appropriate side.

The completeness of this cortical paralysis is difficult to understand for neither blood in the subdural, nor pus in the subarachnoid space will produce this picture. Indeed it is unusual to see a completely paralysed limb in purulent leptomeningitis.

This clinical picture may be more subdued when the primary infection has been treated by systemic penicillin. Evidence of cranial osteomyelitis may be absent, paralysis may be confined to one limb. Nevertheless, signs of cortical paralysis, without obvious leptomeningitis, developing suddenly after gross infection in paranasal sinuses or temporal bone demands exploration of the subdural space.

Jacksonian fits tend to become continuous and then merge into generalized epileptiform convulsions. Less frequent signs are papill-œdema, ptosis, dilated pupils and cranial nerve palsies. Papillœdema is seldom marked, but slight blurring of disc margins and fullness of retinal veins are equally important. Although bilateral ptosis occurred in but one case, its interest derives from the fact that I have found this sign in head injury to be almost pathognomonic of subdural fluid collection. (These observations may be published elsewhere.)

The pupils may be grossly unequal, but a dilated pupil, unless fixed, is not a reliable indication of the side of the lesion. Facial nerve palsy of upper motor neurone type usually accompanies the hemiplegia. Abducens palsy has no particular significance, being frequent enough in any compressive lesion above the tentorium. It is probably due to the nerve becoming kinked over the sharp edge of petrous bone as the brain-stem is pushed downward. Oculomotor palsy, although unusual, is important in that it points to the formation of a tentorial pressure cone, and this calls imperatively for surgical relief.

Clinical Localization

All subdural fluid collections, whether blood, hygroma fluid or pus, tend to spread widely and often bilaterally. The tendency of pus to pool

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alongside the falx has been noted. This is liable to lead to late loculation and the possibility of its clinical recognition deserves emphasis.

With pus over the convexity, Jacksonian fits affect the face and arm and paralysis tends to be more complete in the arm than in the leg. When pus lies chiefly alongside the falx, the initial fits affect the lower limb, paralysis is earlier and more complete in the leg and homonymous hemianopia is prominent. The empyema generally occurs on the side opposite the paralysed limbs, but one must beware the false localization exemplified in Case IV.

Diagnosis

The first essential is the recognition of intracranial extension. Once focal signs occur only cortical thrombophlebitis and brain abscess need be seriously considered.

Lumbar puncture may help but the diagnosis can generally be made without this. Pressure may vary from normal to over 500 mm. water. The cerebrospinal fluid is clear or faintly turbid and though pleocytosis may amount to 500 cells per c.mm., the sugar content will be normal. Culture will be sterile until the arachnoid is penetrated, but in no case can diagnosis wait upon such laboratory report.

It must be emphasized that the diagnosis is confirmed only by exploratory burr-holes; treatment demands these be made immediately a subdural emypema is even suspected. Better unnecessary exploration in the face of meningitis than delay in a subdural empyema.

From Meningitis

Meningitis generally presents a less dramatic picture and focal signs are unusual. Whilst meningitis associated with cortical vein thrombosis may confuse, cerebrospinal fluid findings will help. A moderate pleocytosis, normal sugar and the absence of organisms suggest subdural empyema. Case V exemplifies this differential but both lesions may co-exist as in Case VII.

From Extradural Abscess

Extradural abscess is a much milder disease. Although the patient generally complains of severe localized headache, neck rigidity is unusual and focal signs rare. In the presence of stupor, meningeal irritation and gross focal signs, extradural pus should not be accepted as the only complication for subdural empyema may co-exist. This possibility justifies opening the dura even through an infected field (Case III), a procedure which does not appear to have been reported before.

From Cortical Thrombophlebitis

This can be most difficult for in the course of otitic infection the sudden onset of Jacksonian epilepsy and hemiplegia may be due to

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cortical thrombophlebitis (Courville and Nielsen, 1935 ; Symonds, 1937). Venous thrombi may be infected and clinical signs of meningeal irritation accompany a pleocytosis in the cerebrospinal fluid. Further difficulties may arise when this lesion is associated with meningitis or brain abscess. Helpful points in distinguishing subdural empyema are a history of chronic sinusitis, signs of preceding osteomyelitis and the occurrence of high fever. It is in otitic cases that confusion is more likely to arise, and it was this that led to grave delay in establishing drainage in Case X.

Irrespective of diagnostic difficulty the surgeon's duty is clear for the possibility of subdural empyema can be reputed only by negative exploration. Case X provided the salutary lesson that a single burr-hole, well placed though it be, is not an adequate exploration !

From Brain Abscess

Brain abscess generally has a more insidious onset and more protracted course. Headache is often less, the temperature is seldom high and the pulse may be notably slow. Focal signs are most unlikely to be so widespread as in subdural empyema. Occasionally brain abscess presents a more fulminating course and differential diagnosis is then more difficult. Chiefly from lack of acquaintance with subdural empyema at that time, the writer's pre-operative diagnosis was erroneous in Case I. In any such case, however, an exploratory burr-hole is required and this will show the real state of affairs.

Treatment

Pre-penicillin, practically all recorded cases of subdural empyema died. Death generally occurred within a few days of the onset of focal signs and survival beyond two weeks was rare. The outlook has now changed, but the possibility of effective therapy is too recent for agreement to have been reached as to the means.

Faced with the intracranial extension of infection, otologists have naturally sought to eliminate the primary focus. Several authorities (notably Fischer and Wolfson, 1943) have advocated this in subdural empyema. My contention, and it is amply supported by clinical results, is that once infection has entered the subdural space, the primary focus becomes of secondary importance and can wait. Conversely, search of the literature fails to reveal a single case where the elimination of the primary focus has stayed the fulminating course of subdural infection. Inevitably one must conclude that in these circumstances, operations on sinuses, frontal osteomyelitis or temporal bone are, at best, a dangerous waste of time. This is not to suggest that the primary focus is unimportant, in due course it must be treated on its own merits.

Theoretically it might be possible to treat subdural empyema by systemic penicillin alone. The infecting organisms have almost invariably

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proved remarkably sensitive to this drug which passes freely from the blood to subdural space (Cairns, 1949). In the thorax, however, we have learned that a sterile empyema is not a cured empyema, hence it is less surprising that antibiotics alone seem never to have produced a cure.

Briefly stated the objects of therapy must be :

(1) To control infection.
(2) To drain pus.

The first is obvious, but the urgency of surgical drainage must be emphasized for the liquid pus rapidly becomes a plastic exudate virtually impossible to remove. Chronic loculation occurred in Cases VI and X ; both received prompt and adequate dosage of penicillin but considerable delay attended surgical drainage. Study of cases with chronic loculation reported by Schiller *et al.* (1948), List (1950), reveals a similar pattern of events. Thus it appears that the prime cause of chronic loculation is inadequate drainage—either too little, or too late !

The Drainage of Pus

Various methods of drainage have been employed. Some writers (Fischer and Wolfson, 1943 ; Brunner, 1946) advocate the extensive removal of large portions of bone and wide cruciate incision of the dura. No surgeon with experience of the massive removal of infected calvarium, however, could wish to undertake this in a patient desperately ill with subdural empyema. Fortunately it is now accepted that the wide removal of infected bone is no longer required in the early stages of osteomyelitis for with penicillin most cases subside without obvious sequestration. Furthermore wide incision of the dura invites herniation of the brain and thus the subdural space becomes sealed off by pressure at the edges of the incision.

The tendency for pus to pool alongside the falx led Keith (1949) to propose resection of a parasagittal strip of bone, but this would increase the magnitude of operation and has the further disadvantage of uncovering a long segment of the superior sagittal sinus. Nor is there any probability of better drainage than can be attained with multiple burr-holes.

In all subdural lesions it is an essential principle that both sides must be explored, irrespective of the results of operation on the suspected side. Schultz (1946) records an instructive case of right frontal sinusitis followed by signs of intracranial extension and left hemiparesis. A subdural empyema was drained on the right side and considerable clinical improvement ensued. Three days later swift deterioration occurred and the patient's life was saved only by drainage of the opposite side.

In the cases presented here the subdural pus has been drained *viâ* multiple burr-holes sited in accordance with clinical localization. The most generally useful sites have been :

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(1) Posterior parietal, 8 cm. above the external occipital protuberance and 3 cm. from the mid-line. These bilateral burr-holes are ideally placed for ventriculography if required.

(2) Just with frontal hair-line, 4 cm. from mid-line.

(3) Low in the temporal fossa.

Technique

Local anæsthesia is employed, with 100 mgr. Pethidine as premedication. The completely shaven head is suitably positioned, incisions are marked and local anæsthetic injected. A meticulous aseptic technique is essential. Whilst the scalp is compressed to avoid blood-loss, an incision little over 1 inch long is made straight down to bone. Pericranium is stripped back and the wound held open by a small self-retaining retractor. The skull is opened by a perforator down to the inner table, then by a Hudson's brace and burr. Considerable force is required and to prevent the burr plunging on into the brain the brace must be held under rigid control, pressure being relaxed immediately the increased bite on the burr indicates that the inner table is breached. Calvarial bleeding is controlled by Horsley's wax.

The dura, which may be whiter than usual, is opened by cruciate incision and the pus that wells up is sucked away. When the flow is interrupted by brain bulging into the gap, an Adson's separator slid gently under the dura is used to depress the cortex. Saline washed from one burr-hole to another aids in clearing the subdural space. Pools of pus alongside the falx are similarly cleared by soft rubber catheters passed along the falx from a burr-hole sunk $\frac{1}{2}$ inch from mid-line just behind the coronal suture. Whilst experience suggests that this is always a wise precaution, those unused to intracranial manipulations may be advised to omit this step and remain at safe distance from the major cerebral veins draining into the sagittal sinus.

Spinal drainage induces the brain to slacken and recede from the dura, thus it is often helpfully employed during operation. Nevertheless this manœuvre does not improve access to the medial surface of the cerebral hemisphere for the cerebral veins draining into sagittal sinus anchor the brain against the falx.

Finally one or more catheters are left partly within the subdural space and the scalp incisions are closed in two layers.

The Use of Penicillin

Because the infecting organisms occur not only in the subdural space, but also in bone, cerebrospinal fluid and venous thrombi, penicillin is best given by several routes simultaneously.

Subdural administration gives a high local concentration of the drug

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than can be achieved systemically. Further, mechanical irrigation is necessary to avoid blocking the catheters. List (1950) advocates 50,000 units local penicillin thrice daily, relying on anti-convulsant therapy to reduce the likelihood of epileptiform convulsions. Unwilling to accept this calculated risk the writer has preferred to instil 10 cc. penicillin (500 units per cc.) into the subdural space twice daily.

Systemic penicillin has been given throughout treatment, 100,000 units every three hours at onset continued in the form of " Distaquaine ". Cairns and Schiller (1948) claim that this should be continued for eight weeks to obviate the liability of bone infection to relapse if antibiotics are discontinued too early. Such opinion commands respect, but seldom within my experience has penicillin been so long continued and relapse has not proved a troublesome feature.

To ensure adequate concentration in the cerebrospinal fluid, 10,000 units penicillin are given intrathecally on each of the first few days.

General Treatment

General supportive care is essential for these patients may be semi-comatose for several days. Feeding can be maintained by an indwelling tube and fluid balance is corrected as required. Any anæmia or hypoproteinæmia needs correction and it is important to ensure that protein intake at least equals the requirements of basal metabolism.

Pulmonary secretions are apt to be excessive and may require aspiration of the tracheo-bronchial tree. Undue restlessness is often due to a distended bladder and an indwelling catheter may facilitate nursing care.

Epileptiform convulsions or Jacksonian fits are frequent and Sodium phenobarbitone gr. 3 is at first given intramuscularly as required. Thereafter it is continued orally in reduced dosage and maintenance doses are continued for six months.

Results

Early recognition, prompt drainage and adequate antibiotic therapy have greatly changed the picture of almost uniform fatality presented by Courville and Blomquist (1941), Kubik and Adams (1943), Ray and Parsons (1943), Kahn (1946).

This series of 11 cases with 7 recoveries parallels the 10 cases with 7 recoveries recorded by Schiller, Cairns and Russell (1948). These authors had 23 pre-penicillin cases and all died.

In favourable circumstances marked improvement is evident within a day or two of commencing treatment. Paralysed limbs quickly recover but the return of lost speech is slower. The patient may remain mute for two weeks and the halting recovery of speech is likely to last another

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four weeks or more. All manifestations of cortical paralysis tend to recover completely and subsequent intellectual function is generally unimpaired.

After apparently complete recovery only two sequelæ remain to be feared: epilepsy or brain abscess.

Epilepsy is unlikely if anti-convulsant therapy has been used early and maintained for six months. Cairns and Schiller (1948) describe three cases with late development of brain abscess and advocate ventriculography to exclude a symptomless space-occupying lesion before the patient is discharged.

Some cases fail to improve (Case X), others relapse with reappearance of drowsiness or focal signs (Case VI). These events indicate the chronic loculation of pus. In late cases the subdural space adjoining the falx or tentorium is occupied by irregular masses of pyogenic granulation tissue containing numerous small pockets of pus with tongue-like extensions into the cerebral sulci. Even now the case is not hopeless, for such foci may on occasions be localized by ventriculography and treated by block resection *viâ* a suitable osteoplastic flap (List, 1950).

Here is a problem taxing the utmost resource of the neuro-surgeon and evidence has been adduced to show that it can be avoided by early diagnosis and prompt drainage. Initially the treatment of frontal sinusitis and other causal infections belong to the rhinologist; it is his responsibility to recognize its complications, and he may with advantage add to his armamentarium these neurosurgical methods.

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Summary

1. Eleven cases of subdural empyema with seven recoveries are presented.

2. The characteristic clinical features are described and the diagnosis is fully discussed.

3. Treatment is reviewed and prompt drainage *viâ* multiple burr-holes together with penicillin therapy is advocated. Such treatment has profoundly altered the former uniform fatality of this disease.

4. A new entity of chronic subdural granuloma is discussed, it is attributed to delayed or inadequate drainage.

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