The place of computed tomography and lumbar puncture in suspected bacterial meningitis

Although the incidence of acute bacterial meningitis has not changed significantly over the last 40 years, the death rate had declined considerably,1 presumably due to earlier diagnosis and advances in antibiotic treatment and paediatric intensive care. Between the years 1971 and 1986 the overall mortality from bacterial meningitis in children aged under 13 years in the North East region of Scotland was only 1-8% compared with 11-9% for the period 1946–61. In England and Wales between 1985 and 1987 the case fatality rate for infants with bacterial meningitis 1–12 months of age (a high risk group) was only 5-4%.2 As well as deaths in hospital, both these studies included cases of rapid death at home found to have meningitis at subsequent coroner’s postmortem examination. Similar low mortality rates have been reported from Sweden3 and the United States.4 The causes of death include septic shock, massive cerebral oedema, and brain coning. Agents such as corticosteroids and human monoclonal IgM antibody to endotoxin6 are currently being studied for their ability to prevent or reduce septic shock and will not be discussed further here. Might it be possible to reduce the mortality from cerebral oedema and brain coning?

Risk of coning

Raised intracranial pressure is the rule rather than the exception in children with acute bacterial meningitis. Minns et al studied 35 children and infants with pyogenic meningitis using a strain guage pressure transducer connected to the end of the spinal needle.7 Pressures were measured at the time of the initial diagnostic lumbar puncture in all the children and a number also had sequential measurements. Pressures were raised in 33 children with a median pressure of 15 mm Hg, range 4–70 mm Hg (mean upper normal limit of cerebrospinal fluid pressure was taken as 5–8 mm Hg for infants and 6-4 mm Hg for children). The initial pressures were usually higher than subsequent ones but pressures tended to vary throughout the illness. Cerebral perfusion pressures were calculated and in all but two children were acceptable (that is above 30 mm Hg). These data were thought to reflect a Cushing response (that is a reflex rise in systemic arterial pressure in response to raised intracranial pressure) acting to maintain cerebral blood flow. The response to intravenous mannitol was assessed in 15 patients with raised pressure. In some patients the pressure took as long as 25 minutes to return to normal, while in others the response was much more dramatic. The authors thought there were a number of possible explanations for raised cerebrospinal fluid pressure in pyogenic meningitis including occlusion of arachnoid granulations, generalised inflammatory brain oedema, basal occlusion of the subarachnoid spaces with the development of hydrocephalus, major seizures, and inappropriate antidiuretic hormone secretion. None of the children in this study died and there was no correlation between measured pressure and neurological outcome, although the median follow up was only 13 months.

The overriding concern with raised intracranial pressure is the formation of pressure cones, which are a common postmortem finding in acute bacterial meningitis6 and may be the direct cause of death in as many as 30% of such children. One cause of death is compression of the cerebellar tonsils through the foramen magnum or the medial temporal lobe structures through the tentorial opening. There is a tendency for tentorial herniation to precede foramen magnum herniation but either may occur alone. The early symptoms and signs of cerebral herniation are similar to those of uncomplicated meningitis but often have a rapid onset and progression. There is progressive diminution in the level of consciousness, decorticate or decerebrate posturing, loss of doll’s eye responses, pupillary abnormalities, tonic seizures, and respiratory abnormalities ranging through hyperventilation, bradynocea, and periodic breathing to respiratory arrest. Unilateral herniation of one temporal lobe through the tentorial opening typically causes ipsilateral pupillary dilation and a contralateral hemiparesis. Symptoms and signs of coning occur in about 5% of cases of acute bacterial meningitis10 and may recur particularly if prompt treatment is directed at controlling the underlying raised intracranial pressure with mannitol and passive hyperventilation.

Coning may occur in bacterial meningitis in the absence of lumbar puncture. Slack studied 88 of the 90 deaths from meningococcal infection in England and Wales in 1978 and reported coning in six,11 one of whom had not had a lumbar puncture but died after a convolution, an event known to increase intracranial pressure. However, the majority of meningitis patients who die as a result of a pressure cone do so within 24 hours of lumbar puncture. Some deteriorate immediately after the spinal fluid sampling but with many there is a delay of several hours suggesting that the amount of fluid removed may be less important than the continued leak of cerebrospinal fluid through the dural tear into the epidural space. Avoidance of lumbar puncture in children
with suspected meningitis in whom there is clinical evidence of raised intracranial pressure or early coning would be expected to reduce the death rate, provided the lack of diagnostic accuracy (for example, type of organism and its antibiotic sensitivities) is not too high a price to pay. The question is how best to manage such children without resort to lumbar puncture.

Other conditions
In addition there are a few children with suspected meningitis and evidence of raised intracranial pressure who have some other condition which mimics meningitis clinically and which must be diagnosed promptly for optimal treatment. Posterior fossa tumours, acute hydrocephalus, cerebral abscesses, subdural haematoma, bleeding from an arteriovenous malformation, meningocoecephalitis, herpes simplex encephalitis, Reye’s syndrome, and intracerebral haemorrhage may on occasions all present in a way that suggests acute bacterial meningitis with raised intracranial pressure. Lumbar puncture in many of these situations might be expected to carry a serious risk of coning. To illustrate this danger Richards and Towu-Aghantse described two patients who presented with impaired consciousness and neck stiffness and who were thought to have meningitis. Both had symptoms of pressure coning after lumbar puncture. Subsequently computed tomography showed a subdural haematoma in one and a cerebellar haematoma in the other. Both patients subsequently died from what were potentially curable conditions.

Computed tomography
The Vancouver prospective study of computed tomography in acute bacterial meningitis in childhood showed that the initial scan obtained within 72 hours after admission was normal in the majority of the 41 children studied. Abnormalities were noted in 14 children but were not specifically diagnostic of meningitis. Two children showed clear evidence of generalised cerebral oedema. Both had clinical evidence of raised intracranial pressure and subsequently died. Five children had focal low attenuation areas compatible with infarction and all had a haemiparesis on the opposite side. One child with pneumococcal meningitis showed obliteration of the basal cisterns consistent with the accumulation of pus. Eight children had evidence of subdural effusion but none of these were thought to be causing symptoms. Mild ventriculomegaly and widening of the subarachnoid spaces was a common finding in the second scan obtained 7–16 days after admission and was seen in 29 of the 36 children scanned at that time. None required shunting and the ventriculomegaly had reverted to normal in those children who had a third scan performed 5–23 months after admission. Contrast was not routinely given in this study. It is known that diffuse enhancement of the cerebral convexities and/or base of the brain may be seen after contrast if there is marked meningeal inflammation. Given the clinical context of suspected bacterial meningitis, such a finding on an enhanced computed tomogram would be virtually diagnostic. Unfortunately the finding is only seen in a small minority of children with acute bacterial meningitis at presentation. Thus it must be concluded that computed tomography rarely gives specific positive information on which to make a diagnosis of acute bacterial meningitis.

On the other hand computed tomography is very helpful in excluding those conditions that may mimic bacterial meningitis with raised intracranial pressure. Posterior fossa tumours, acute hydrocephalus, cerebral abscess, and intracranial bleeds are all readily diagnosed on computed tomograms. Scanning may be less diagnostically helpful in Reye’s syndrome, meningoencephalitis, and herpes simplex encephalitis where the findings may be non-specific or even normal.

Can computed tomography detect impending coning which is not apparent clinically? Unfortunately, the answer is probably not. Cerebral oedema may produce abnormalities on computed tomography including small lateral and third ventricles, generalised low attenuation of the white matter, and obliteration of the basal cisterns but there is considerable variation in ventricular and cisternal size which can make interpretation of a single scan unreliable in this respect.

Practical solutions
Avoidance of lumbar puncture in children with suspected acute bacterial meningitis and evidence of raised intracranial pressure should reduce deaths from coning but we must be sure that such a policy does not lead to delayed antibiotic treatment. Clinical features suggesting dangerously elevated intracranial pressure and incipient coning are given in the table. Papilloedema is an uncommon finding in acute bacterial meningitis even when there is markedly raised intracranial pressure as it takes 24–48 hours for the discs to swell but its presence would be an absolute contraindication to lumbar puncture. The absence of papilloedema in this situation does not mean that lumbar puncture would always be safe and the other clinical features of raised intracranial pressure need to be taken into account. Most children with bacterial meningitis are drowsy but increasing coma suggests raised intracranial pressure. Convulsive seizures are not uncommon in the early stages of bacterial meningitis and do not in themselves signify raised intracranial pressure, but any convulsion is associated with a transient increase of pressure and so lumbar puncture should not be done within 30 minutes after a short convulsive seizure or at all after a prolonged convulsion. Tonic seizures on the other hand may be a symptom of dangerously high intracranial pressure and lumbar puncture should not be carried out. Septic shock would also be a contraindication, not because of raised pressure, but because the stress of the procedure itself may aggravate the cardiovascular collapse.

For these children immediate lumbar puncture is not carried out (see figure) but blood and throat cultures are taken before prompt instigation of appropriate intravenous antibiotic treatment. Treatment for raised intracranial pressure may be required with intravenous mannitol, dexamethasone, and passive hyperventilation. Once the child’s condition has stabilised, arrangements should be made for computed tomography. The main purpose of this is to exclude those conditions which may mimic acute bacterial meningitis. In bacterial meningitis the computed tomography may show non-specific changes but is unlikely to give proof of the diagnosis. The results of the blood cultures will become available in 24–48 hours and can be expected to identify the organism in about 90% of cases of

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<thead>
<tr>
<th>Contraindications to lumbar puncture in the child with suspected acute bacterial meningitis (see text for discussion)</th>
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<tbody>
<tr>
<td>Papilloedema</td>
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<tr>
<td>Coma</td>
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<td>Hypertension</td>
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<td>Bradycardia</td>
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<tr>
<td>Bradypnoea or irregular respirations</td>
</tr>
<tr>
<td>Fixed dilated or unequal pupils</td>
</tr>
<tr>
<td>Absent doll’s eye movements</td>
</tr>
<tr>
<td>Recent or prolonged convulsive seizures</td>
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<td>Tonic seizures</td>
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<tr>
<td>Decerebrate or decorticate posture</td>
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<tr>
<td>Hemiparesis</td>
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<td>Septic shock</td>
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**Haemophilus influenzae** or pneumococcal meningitis but only about 50% of cases of meningococcal meningitis. 19 Failure to grow an organism means that the broad spectrum antibiotics must be continued for a full 10 days of treatment. After a day or two of treatment there may be the option of carrying out a delayed lumbar puncture if the contraindications are no longer present. This is rarely necessary but would allow cytological and chemical analysis of the cerebrospinal fluid with countercurrent immunoelectrophoresis for bacterial antigens if there is still serious doubt about the diagnosis. 20

Fortunately the majority of children presenting with suspected acute bacterial meningitis have no contraindications for lumbar puncture and this should be done promptly before starting intravenous antibiotic treatment. There is no place for routine computed tomography before or after lumbar puncture in the child with clinically uncomplicated acute bacterial meningitis.

**Flow diagram for management of the child with suspected acute bacterial meningitis. IV, intravenous; ICP, intracranial pressure.**

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