Clinical review

Fortnightly review

Postnatal depression

Peter J Cooper, Lynne Murray

Winnicott Research Unit, Department of Psychology, University of Reading, Whiteknights, Reading RG6 6AL

Peter J Cooper, *professor*

Lynne Murray, *professor*

Correspondence to: Professor Cooper P.J.Cooper@reading.ac.uk

BMJ 1998;316:1884–6

There has been considerable recent clinical and research interest in postpartum depression. This has been largely provoked by the accumulating evidence that postnatal depression is associated with disturbances in child cognitive and emotional development.[1] This evidence, which is reviewed below, has renewed concern about the epidemiology of postnatal depression, its aetiology, methods of prediction and detection, and the most appropriate form of management.

Summary points

- Postnatal depression is associated with disturbances in the mother-infant relationship, which in turn have an adverse impact on the course of child cognitive and emotional development
- Postnatal depression affects 10% of women in the weeks immediately post partum
- There is little evidence for a biological aetiology; antenatal personal and social factors are more relevant
- Postnatal depression is commonly missed by primary care teams despite the fact that simple reliable detection procedures have been developed
- The treatment of choice in most cases of postnatal depression is counselling, which can be effectively delivered by health visitors
- There is a need to develop preventive intervention strategies

Methods

This article is based on a review of the recent research concerned with the impact of postnatal depression on child development, and the epidemiology, prediction, detection and management of the disorder. Authoritative recent reviews are cited as well as the most impressive research papers. To supplement our immediate knowledge of the literature we performed literature searches with Medline and PsychLit (1980-97) using the relevant key words ("postnatal/postpartum depression" in conjunction with "infant/child development/outcome, epidemiology, aetiology, prediction, detection and treatment").

Impact on parenting and child outcome

There have been several recent prospective studies of samples of women with postnatal depression and their children.[1] They indicate a definite association between the maternal mood disorder and impaired infant cognitive development. Thus, in Cambridge a community sample of children of mothers who had had postnatal depression were found to perform significantly less well on cognitive tasks at 18 months than did children of well mothers, especially the boys.[2 3] Two London studies of more socioeconomically disadvantaged populations have found that this effect still obtained when the children were 4-5 years old.[4 5] Poor emotional adjustment has been shown to be similarly associated with postnatal depression. Thus, most studies that have systematically examined infant attachment in the context of postnatal depression have found a raised rate of insecure attachments.[2 6 7]

There is evidence that these emotional problems persist. A follow up of the Cambridge cohort found that the 5 year old children of mothers who had had postnatal depression were significantly more likely than controls to be rated by their teachers as behaviourally disturbed.[8] One major conclusion from these studies is that the mechanism mediating the association between postnatal depression and adverse child developmental outcome is the impaired pattern of communication occurring between the mother and her infant.[1]

Impact on child development

- Cognitive development in the context of postnatal depression is adversely affected, especially among male children and socioeconomically disadvantaged groups
- The children of postnatally depressed mothers tend to have insecure attachments at 18 months, and the boys show a high level of frank behavioural disturbance at 5 years
- The adverse child outcome in the context of postnatal depression is related to disturbances in the mother-infant interactions

Epidemiology and course

Epidemiological studies of puerperal samples have consistently shown that the prevalence of non-

psychotic major depressive disorder in the early weeks after delivery is about 10%.[9] Although this rate does not represent an elevation over the non-postpartum base rate,[10–13] the inception rate for depression does seem to be raised in the first three months postpartum compared with the following nine months.[10 13 14] The duration of postnatal depression is similar to that of depressions arising at other times—that is, episodes typically remit spontaneously within two to six months.[9 10] Some residual depressive symptoms are common up to a year after delivery.[9 10]

Epidemiology, aetiology, prediction, and detection of postnatal depression

- Postnatal depression affects about 10% of women in the early weeks post partum, with episodes typically lasting two to six months
- There is little evidence for a biological basis
- Previous depression is a risk factor, especially when paired with obstetric complications
- The main risk factors are ones indicative of social adversity
- The only large scale study of the predictive value of antenatal factors produced an index of some use; its performance could be improved by including assessment of postpartum blues and infant temperament
- Detection, while generally poor, presents no difficulty

Aetiology

There is little evidence to support a biological basis to postpartum depression.[9] Despite extensive research into steroid hormones in women during the puerperium, no firm evidence has emerged linking these hormones to the development of postnatal depression.[15] It has been suggested that in a small subgroup of those experiencing postnatal depression there might be a thyroid dysfunction.[16] Although this hypothesis merits attention if substantiated, it remains possible that the thyroid dysfunction could be secondary to immunological changes brought about by stress.

The presence of maternity blues in the period immediately post partum has been found to be related to the subsequent development of postpartum depression, but no hormonal basis to this association has been identified.[9 15] Obstetric factors are important in a vulnerable subgroup of women: among those with a history of depressive disorder, complications during delivery are associated with a raised rate of postnatal depression.[17 18]

The consistent finding of the epidemiological studies carried out to date is that the major factors of aetiological importance are largely of a psychosocial nature.[9] So, the occurrence of stressful life events in general and unemployment in particular, the presence of marital conflict, and the absence of personal support from spouse, family, and friends have all consistently been found to raise the risk of depression post partum.

A psychiatric history is also commonly reported to be a risk factor for postnatal depression, especially a history of depressive disorder. This latter association has recently been clarified in a five year follow up of a cohort of primiparous women who had had a postpartum depression as a recurrence of previous non-postpartum mood disorder and a cohort for whom the postpartum depression was their first experience of affective disturbance.[19] The first group were found to be at greater risk for subsequent non-postpartum depression but not to be at risk for depression after a subsequent delivery. Conversely, the second group were found to be at greater risk for subsequent postpartum depression but not for subsequent non-postpartum depression. This suggests that for a subgroup of those with postpartum depression the puerperium carries specific risk, for either biological reasons or psychological ones surrounding the demands of infant care.

Prediction

Although several studies have reported on antenatal factors associated with postnatal depression, all but one have been based on samples that were too small to derive a reliable predictive index. The single large scale predictive study to be conducted revealed that the most reliable predictors of postpartum depression (such factors as the absence of social support and a history of depression) each approximately double the odds over the base rate risk.[20] The predictive index derived from this study of several thousand women is of some use: at a cut off score with a sensitivity of 75% the specificity is 52%, and at a cut off score with a specificity of 75% the sensitivity is 44%. It is unlikely that there could be much improvement on the positive predictive value of this instrument using only antenatal factors.

Prediction of postpartum depression could be improved if account were taken of certain postpartum factors. Thus, in a recent study of the impact of neonatal factors on the course of maternal mood it was found that, over and above the predictive contribution of antenatal factors, both a high score for "maternity blues" and certain neonatal factors (irritability and poor motor control) were significantly related to the onset of postnatal depression.[21] Since both the blues and the neonatal factors contribute predictively over and above the predictive antenatal variables, the positive predictive value of the collective critical antenatal factors could be augmented by taking account of both these postpartum variables.

Detection

Postpartum depression is often missed by primary care teams.[22 23] Its detection does not, however, present any special problem. The clinical features of the disorder are not distinctive,[9–11] and its assessment is straightforward. Indeed, a simple brief self report measure, the Edinburgh postnatal depression scale (EPDS) has been developed as a screening device.[24] It has sound psychometric properties. A large community study has revealed a specificity of 92.5% and a sensitivity of 88%.[25] The questionnaire is easy to administer, simple to interpret, and could readily be incorporated within the routine services provided to all postpartum women. Sensitive clinical inquiry in high scorers would be sufficient to confirm the presence of depression.

Treatment

Drug treatment

There has been little systematic research on the drug treatment of postnatal depression. Although progesterone treatment has been advocated,[26] there has been no systematic evaluation of its clinical usefulness. The

Treating postnatal depression

- There is no systematic evidence to support the use of progesterone
- One study has shown a benefit of oestrogen in severe and chronic cases
- An antidepressant (fluoxetine) has been shown to be helpful in elevating maternal mood
- Counselling has been shown to be of significant benefit in improving maternal mood and aspects of infant outcome
- Counselling can be effectively delivered by trained health visitors

efficacy of oestrogen treatment has, however, recently been evaluated in a placebo controlled trial.[27] In a sample with severe and chronic postpartum depression, mood improved in both groups but significantly more so among those receiving oestrogen than among those receiving placebo. The appropriateness of this form of treatment in more typical samples of postnatally depressed women remains to be evaluated.

There has been only one controlled trial of an antidepressant drug.[28] In a factorial design involving the use of fluoxetine and counselling, both the drug and the psychological treatment showed a significant antidepressant effect. However, there was no additive effect of the two treatments, and the drug treatment was not superior to the psychological treatment. It is notable that less than half of those invited to take part in the study agreed to do so, mainly because of "reluctance to take the medication."

Psychological treatment

There have been three controlled trials of psychological treatment of postpartum depression. Holden et al found that women visited by health visitors trained in non-directive counselling, an average of nine visits over 13 weeks, showed substantially greater improvement in maternal mood than did the control group receiving routine primary care.[29] Similarly, a significant benefit in terms of remission from depression has been found for six weekly counselling visits by child health clinic nurses in Sweden.[30] Finally, a recent controlled evaluation of three brief, home based, psychological forms of intervention (including a session of non-directive counselling) found that they improved maternal mood.[31]

Treatment and the mother-infant relationship

Few studies have examined the impact of treating postnatal depression on the quality of the mother-infant relationship and child development. One controlled trial of psychological treatment found that the intervention was associated with significant improvement in maternal reports of infant problems, both immediately after treatment (four to five months post partum) and at 18 months post partum.[31] In addition, early remission from depression, itself significantly associated with treatment, was related to a reduced rate of insecure infant attachment at 18 months. Similar benefits have been reported in a study of health visitors' practice.[23] When training was provided to all the health visitors working in one NHS sector a cohort study was conducted, with assessments made of the health visitors' clientele both before and after the training. Treatment significantly improved both maternal mood and the quality of the mother-infant relationship.

It seems that the adverse child outcome arising in the context of postnatal depression is driven by disturbances in the mother-child relationship, which begin in the early postpartum weeks (or days). This highlights the importance of early detection and treatment by primary care teams. It also suggests that preventive interventions might prove particularly profitable.

1 Murray L, Cooper PJ. Effects of postnatal depression on infant development. *Arch Dis Child* 1997;77:99-101.
2 Murray L. The impact of postnatal depression on child development. *J Child Psychol Psychiatry* 1992;33:543-61.
3 Murray L, Fiori-Cowley A, Hooper R, Cooper PJ. The impact of postnatal depression and associated adversity on early mother-infant interactions and later infant outcome. *Child Dev* 1996;67:2512-26.
4 Cogill S, Caplan H, Alexandra H, Robson K, Kumar R. Impact of postnatal depression on cognitive development of young children. *BMJ* 1986;292:1165-7.
5 Sharp D, Hay D, Pawlby S, Schmucher G, Allen H, Kumar R. The impact of postnatal depression on boys' intellectual development. *J Child Psychol Psychiatry* 1995;36:1315-37.
6 Lyons-Ruth K, Zoll D, Connell D, Grunebaum HU. The depressed mother and her one year old infant: environment, interaction, attachment and infant development. In: Tronick EZ, Field T, eds. *Maternal depression and infant disturbance. New directions for child development.* San Francisco: Jossey-Bass, 1986:34.
7 Teti DM, Gelfand CM, Messinger DS, Isabella R. Maternal depression and the quality of early attachment: an examination of infants, pre-schoolers, and their mothers. *Dev Psychol* 1995;31:364-76.
8 Sinclair D, Murray L. Teacher reports of adjustment to school of postnatally depressed and well mothers' children. *Br J Psychiatry* 1998;172: 58-63.
9 O'Hara MW. The nature of postpartum depressive disorders. In: Murray L, Cooper PJ, eds. *Postpartum depression and child development*. New York: Guilford, 1997:3-31.
10 Cooper PJ, Campbell EA, Day A, Kennerley H, Bond A. Non-psychotic psychiatric disorder after childbirth: a prospective study of prevalence, incidence, course and nature. *Br J Psychiatry* 1988;152:799-806.
11 O'Hara MW, Zekoski EM, Phillips LH, Wright EJ. A controlled prospective study of postpartum mood disorders: comparison of childbearing and non-childbearing women. *J Abnorm Psychol* 1990;99: 3-15.
12 Troutman BR, Cutrona CE. Nonpsychotic postpartum depression among adolescent mothers. *J Abnorm Psychol* 1990;99:69-78.
13 Cox JL, Murray D, Chapman G. A controlled study of the onset, duration and prevalence of postnatal depression. *Br J Psychiatry* 1993;163:27-31.
14 Cooper PJ, Murray L, Stein A. Postnatal depression. In: Seva J, ed. *The European handbook of psychiatry and mental health*. Zaragos: Anthropos, 1991:1255-62.
15 Harris B. A hormonal component to postnatal depression. *Br J Psychiatry* 1993;163:403-5.
16 Harris B. Biological and hormonal aspects of postpartum depressed mood. *Br J Psychiatry* 1994;164:288-92.
17 Murray L, Cartwright W. The role of obstetric factors in postpartum depression. *J Reprod Infant Psychol* 1993;11:215-9.
18 O'Hara MW, Schlechte JA, Lewis DA, Varner MW. A controlled prospective study of postpartum mood disorders: psychological, environmental, and hormonal variables. *J Abnorm Psychol* 1991;100:63-73.
19 Cooper PJ, Murray L. The course and recurrence of postnatal depression. *Br J Psychiatry* 1995;166:191-5.
20 Cooper PJ, Murray L, Hooper R, West A. The development and validation of a predictive index for postpartum depression. *Psychol Med* 1996; 26:627-34.
21 Murray L, Stanley C, Hooper R, King F, Fiori-Cowley A. The role of infant factors in postnatal depression and mother-infant interactions. *Dev Med Child Neurol* 1996;38:109-19.
22 Briscoe ME, Williams P. Emotional problems in the clients of health visitors. *Health Visitor* 1985;58:197-8.
23 Seeley S, Murray L, Cooper PJ. Postnatal depression: the outcome for mothers and babies of health visitor intervention. *Health Visitor* 1996;69:135-8.
24 Cox JL, Holden JM, Sagovsky R. Detection of postnatal depression: development of the 10-item Edinburgh postnatal depression scale. *Br J Psychiatry* 1987;150:782-6.
25 Murray L, Carothers AD. The validation of the Edinburgh postnatal depression scale on a community sample. *Br J Psychiatry* 1990;157: 288-90.
26 Dalton K. Progesterone prophylaxis used successfully in postnatal depression. *Practitioner* 1985;229:507-8.
27 Gregoire AJP, Kumar R, Everitt B, Henderson AF, Studd JWW. Transdermal oestrogen for treatment of severe postnatal depression. *Lancet* 1996;347:930-3.
28 Appleby L, Warner R, Whitton A, Faragher B. A controlled study of fluoxetine and cognitive-behavioural counselling in the treatment of postnatal depression. *BMJ* 1997;314:932-6.
29 Holden JM, Sagovsky R, Cox JL. Counselling in a general practice setting: controlled study of health visitor intervention in treatment of postnatal depression. *BMJ* 1989;298:223-6.
30 Wickberg B, Hwang CP. Counselling of postnatal depression: a controlled study on a population based Swedish sample. *J Affect Disord* 1996;39: 209-16.
31 Cooper PJ, Murray L. The impact of psychological treatments of postpartum depression on maternal mood and infant development. In: Murray L, Cooper PJ, eds. *Postpartum depression and child development*. New York: Guilford, 1997:201-20.

Lesson of the week

Acute obstructive hydrocephalus complicating bacterial meningitis in childhood

Helen Mactier, Paul Galea, Robert McWilliam

Two children presented with signs of raised intracranial pressure and sepsis. Computed tomography showed relatively large ventricles, suggesting obstructive hydrocephalus. Raised intracranial pressure was confirmed at ventriculostomy, but cerebrospinal fluid was sterile. *Streptococcus pneumoniae* infection was subsequently documented in both patients, and findings at postmortem examination in one confirmed that he had acute purulent basal meningitis.

Case reports

Case 1

A boy presented to a district general hospital on the eve of his first birthday. He had collapsed at home, and his parents reported that for three days he had had fever, anorexia, vomiting, and increasing drowsiness. On arrival at hospital he was poorly perfused, could barely be roused, and his anterior fontanelle was bulging. The boy's blood pressure was 117/66 mm Hg and his occipitofrontal circumference was 50.6 cm (>90th centile). Initial investigations showed haemoglobin concentration 81 g/l, white cell count 23.9×10^9/l (neutrophils 18.9×10^9/l), platelets 226×10^9/l, serum urea and electrolytes normal, and C reactive protein 137 mg/l. He improved after resuscitation with plasma; then, because of recurrent episodes of bradycardia, he was intubated and ventilated. Intravenous ceftazidime and penicillin were given. The boy had been previously well, was fully immunised, and had been making appropriate neurodevelopmental progress. His large head had been noted previously but was attributed to a familial tendency and was not investigated.

The patient was transferred to the regional paediatric intensive care unit, where computed tomograms showed dilatation of the lateral and third ventricles with some reduction of the extra axial spaces (fig 1). There was no crowding of the foramen magnum, effacement of the basal cisterns, periventricular oedema, or loss of grey-white differentiation. Taking into account the history of a large head, it was concluded that this child had longstanding ventricular dilatation. Six hours later the boy's condition deteriorated—he had fixed dilatation of the left pupil, loss of amplitude on the electroencephalogram, and increasing blood pressure. Intravenous mannitol (0.5 g/kg) was given before an intraventricular catheter was inserted. The initial cerebrospinal fluid pressure was 52 mm Hg, and analysis of the ventricular cerebrospinal fluid showed white cell count 3/mm^3, red cell count 1500/mm^3, protein 0.34 g/l, glucose 2.9 mmol/l, and latex tests for *Neisseria meningitidis*, *Haemophilus influenzae*, *S pneumoniae*, and group B streptococcus all negative. Direct culture of the cerebrospinal fluid had negative results; enriched culture produced only coagulase negative staphylococcal species.

The boy's intracranial pressure continued to fluctuate at or above mean arterial pressure, and next morning repeat computed tomograms showed widespread cerebral necrosis (fig 2). Intensive care support was withdrawn. A lumbar puncture was performed after he had died. The results were white cell count 45/mm^3, red cell count 3/mm^3, protein 4.05 g/l, and glucose 2.2 mmol/l, in keeping with obstruction to flow of cerebrospinal fluid. Latex agglutination tests on the blood culture taken on admission to hospital and on lumbar cerebrospinal fluid were positive for *S pneumoniae*. Postmortem examination confirmed that he had acute purulent meningitis with exudate around the base of the brain; culture of cerebrospinal fluid grew *S pneumoniae* serotype 19. There was no congenital cerebral abnormality.

Case 2

An 11½ year old boy presented to this hospital with a five day history of worsening fever, headache, anorexia, vomiting, and drowsiness. There was nothing noteworthy in his medical history and he had not been given any medication. On admission to hospital, he was poorly perfused, with fever and noticeable nuchal rigidity. The boy's Glasgow coma score was 9 and deteriorating, his pupils were small and reactive, and his fundi were normal. The results of initial investigations included haemoglobin 140 g/l, white cell count 29.8×10^9/l (neutrophils 28.6×10^9/l), platelets 246×10^9/l, serum urea, electrolytes and ammonia normal, C reactive protein 63 mg/l. The boy was resuscitated with intravenous plasma and given intravenous

Bacterial meningitis may present as acute obstructive hydrocephalus, and cannot be excluded by sterile ventricular cerebrospinal fluid

Department of Child Health, Royal Hospital for Sick Children, Glasgow G3 8SJ
Helen Mactier, *senior registrar.*
Paul Galea, *consultant*
Fraser of Allander Unit, Royal Hospital for Sick Children
Robert McWilliam, *consultant*
Correspondence to: Dr Mactier

BMJ 1998;316:1887–9

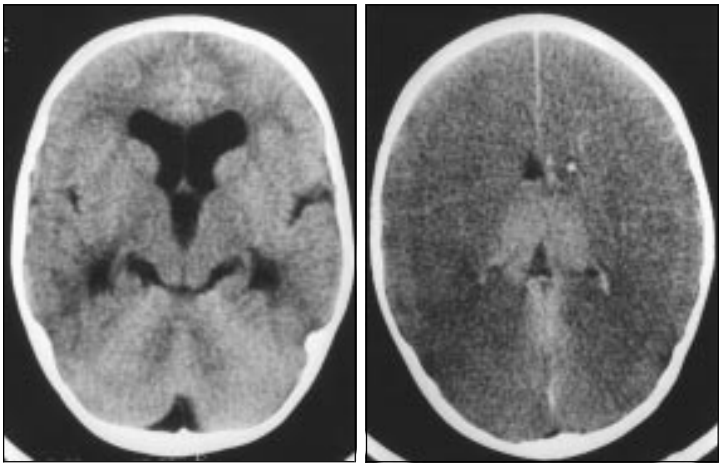

Fig 1 (left) Computed tomogram of patient in case 1 showing dilatation of the lateral and third ventricles with reduction of the extra axial spaces; grey-white differentiation is well preserved
Fig 2 (right) Computed tomogram of patient in case 1 showing widespread cerebral necrosis

Obstructive hydrocephalus in bacterial meningitis

Presentation

- Decreased level of consciousness
- Low grade or fluctuating fever
- Age appropriate signs of meningism

Management

- Avoid lumbar puncture
- Consider measures to reduce intracranial pressure (intravenous mannitol 0.25 g/kg, repeated up to a total dose of 1.5 g/kg if necessary)
- Urgent brain imaging
- Ventriculostomy and drainage

cefotaxime, benzylpenicillin, and mannitol before emergency computed tomography. Scans showed prominent temporal horns with effacement of the basal cisterns and some diminution of grey-white differentiation, in keeping with early brain swelling and developing obstructive hydrocephalus. The contrast study showed no focal lesion but ill defined enhancement around the posterior cerebral arteries, suggesting basal meningitis (fig 3). An intraventricular catheter was inserted soon afterwards, and the initial cerebrospinal fluid pressure was >30 mm Hg. Cerebrospinal fluid analyses showed white cell count 180/mm^3, red cell count >5000/mm^3, protein 1.2 g/l, glucose 5.5 mmol/l, and negative results on latex tests for *N meningitidis*, *H influenzae*, *S pneumoniae*, and group B streptococcus. In addition, direct and enriched cultures had negative results. Blood culture taken on admission to hospital subsequently grew *S pneumoniae* serotype 14, although repeat latex agglutination of cerebrospinal fluid for this particular serotype was negative. Chest *x* ray showed left lobar pneumonia.

Treatment with antibiotics and dexamethasone was continued, and by day 5 of his stay in hospital repeat computed tomograms were normal. The boy was successfully extubated next day, and discharged home on day 20 to make a full recovery.

Discussion

Acute bacterial meningitis remains a relatively common and potentially fatal condition in childhood, and *S pneumoniae* is found to be the infecting organism in 10-20% of cases.[1] As the box shows, presentation is generally with fever and signs of cerebral dysfunction. Classic nuchal rigidity may be absent in younger children, but most will have a peripheral leucocytosis.[2] Cerebrospinal fluid is formed by the choroid plexus in the lateral ventricles, from where it flows via the third and fourth ventricles to the subarachnoid space. It is reabsorbed subsequently by the arachnoid villi in the intracranial venous sinuses.[3] The flow of cerebrospinal fluid may be blocked at the third or fourth ventricles (obstructive hydrocephalus) or at the arachnoid villi (communicating hydrocephalus). In bacterial meningitis, neutrophil migration into the subarachnoid space follows bacterial invasion. The resultant purulent exudate tends to collect in the Rolandic and Sylvanian sulci over the cerebral hemispheres and in the basal cisterns, where the subarachnoid space is deepest[4] and where, presumably, cerebrospinal fluid flow is most sluggish. The exudate interferes with absorption of cerebrospinal fluid by the arachnoid villi and may also cause obstructive hydrocephalus by obstructing the foramina of Luschka and Magendie. Typically, the obstruction occurs towards the end of the second week of the illness, when neutrophils begin to degenerate and fibroblasts proliferate in the exudate. In case 1, however, findings at postmortem examination were of an acute inflammatory exudate blocking the exit foramina, entirely consistent with the three day history of illness.

It was surprising that in both patients, culture of ventricular cerebrospinal fluid had negative results. Bacterial invasion into the cerebrospinal fluid from the nasopharynx in pneumococcal meningitis occurs via the choroid plexus and cerebral microvasculature,[5] and in the primate model the highest concentration of organisms early in the course of *H influenzae* meningitis is in the lateral ventricles.[6] Previous exposure to antibiotics does not explain failure to culture the infecting organism from ventricular cerebrospinal fluid in these two patients. Indeed, in case 1, *S pneumoniae* was grown from cerebrospinal fluid sampled after death and despite more than 48 hours of appropriate intravenous antibiotic treatment. A latex agglutination test performed with prior knowledge of the phage type of the pneumococcus (as in case 2) detects fragments of the infecting organism at a concentration of 5 ng/l (500 bacteria/ml) (LE Smart, personal communication). The absence of white blood cells in both ventricular and lumbar cerebrospinal fluid from the patient in case 1 is consistent with infection localised to the posterior fossa and obstructing the flow of cerebrospinal fluid. Presumably the route of infection was not via the choroid plexus. The acute inflammatory exudate in pneumococcal infection is particularly tenacious. Only one major textbook of paediatrics acknowledges that ventricular cerebrospinal fluid can

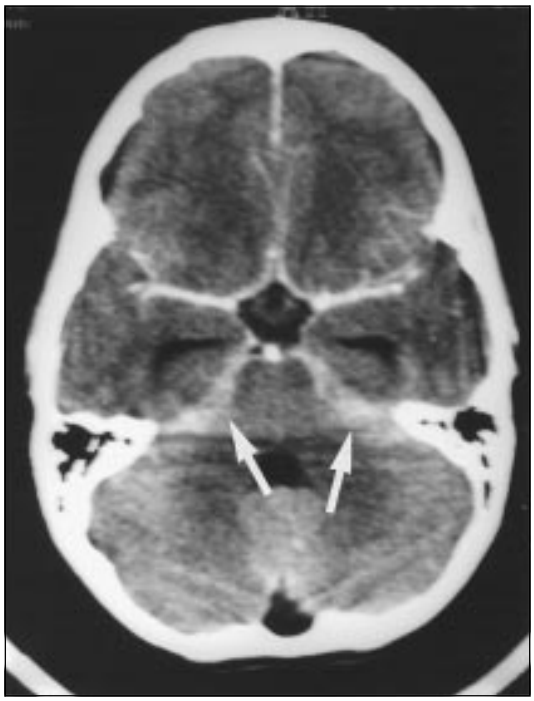

Fig 3 Contrast study of patient in case 2 showing ill defined enhancement around the posterior cerebral arteries (arrows) suggestive of basal meningitis

be sterile at the same time as lumbar cerebrospinal fluid is purulent.[7] We believe that this fact is not generally recognised.

Cerebral herniation is well recognised as a complication of pneumococcal meningitis.[8 9] However, diagnosing critically high intracranial pressure is difficult—particularly after seizures and in sedated ventilated patients—and a high index of suspicion is necessary.[9] Early use of osmotic agents, with or without ventricular drainage, may reduce the risks of long term morbidity or death, but mortality and morbidity are still appreciable.[8–10] Computed tomography is not an accurate measure of intracranial pressure[11] but is appropriate to exclude mechanical causes of raised intracranial pressure before lumbar puncture in children with suspected meningitis and a reduced level of consciousness.[12] Ventriculomegaly is relatively common in acute bacterial meningitis in childhood and may be progressive in the absence of raised intraventricular pressure, presumably as a result of cerebral atrophy.[13] Thus, computed tomograms must be interpreted in conjunction with the clinical status of the patient, and computed tomography may need to be repeated, particularly if there is no response to treatment. With hindsight, the patient in case 1 had simple familial macrocephaly—appreciation of this fact should have prompted earlier ventriculostomy.

Ventriculostomy is a safe and relatively simple procedure.[12 14] It enables cerebral perfusion pressure (mean arterial pressure minus intracranial pressure) to be calculated and intracranial pressure reduced by removal of cerebrospinal fluid. Unlike cerebral oedema, which is common in meningitis,[15] hydrocephalus can be treated by appropriate drainage of the cerebrospinal fluid, and therefore needs to be identified.

Acute obstructive hydrocephalus is thought to be an uncommon presenting feature of bacterial meningitis, usually occuring in younger children who have had previous treatment with antibiotics.[8 13] We could find no record of the incidence of this complication in published reports, nor any other case report, but since preparing this manuscript we have seen two other children with obstructive hydrocephalus as a presenting feature of bacterial meningitis. All patients with suspected meningitis and decreased level of consciousness should have urgent brain imaging to exclude obstructive hydrocephalus before lumbar puncture.

Funding: None.
Conflict of interest: None.

1 Roos KL, Tunkel AR, Scheld WM. Acute bacterial meningitis in children and adults. In: Scheld WM, Whitley RJ, Durack DT, eds. *Infections of the central nervous system.* Philadelphia: Lippincott-Raven, 1997:335-401.
2 Geiseler PJ, Nelson KE. Bacterial meningitis without clinical signs of meningeal irritation. *S Med J* 1982;75:448-50.
3 Walton JN, ed. *Brain's diseases of the nervous system. 8th ed.* New York: Oxford University Press, 1977:121-2.
4 Adams RD, Kubik CS, Bonner FJ. The clinical and pathological aspects of B influenzal meningitis. *Arch Pediatr* 1948;65:408-41.
5 Saez-Llorens X, Ramilo O, Mustafa MM, Mertsola J, McCracken GH. Molecular pathophysiology of bacterial meningitis: Current concepts and therapeutic implications. *J Pediatr* 1990;116:671-84.
6 Smith AL, Daum RS, Scheifele D, Syriopolou V, Averill DR, Roberts MC, et al. Pathogenesis of Haemophilus influenzae meningitis. In: Sell SH, Wright PF, eds. *Haemophilus influenzae: epidemiology, immunology, and prevention of disease.* New York: Elsevier, 1982:89-109.
7 Campbell AGM. Infections. In Campbell AGM, McIntosh N, eds. *Forfar and Arneil's textbook of paediatrics.* Edinburgh: Churchill Livingstone, 1992:1345.
8 Stephenson JBP. Timing of lumbar puncture in severe childhood meningitis. *BMJ* 1985;291:1123.
9 Horwitz SJ, Boxerbaum B, O'Bell J. Cerebral herniation in bacterial meningitis in childhood. *Ann Neurol* 1980;7:524-8.
10 Goitein KJ, Tamir I. Cerebral perfusion pressure in central nervous system infections of infancy and childhood. *J Pediatr* 1983;103:40-3.
11 Hyderman RS, Robb SA, Kendall BE, Levin M. Does computerised tomography have a role in the evaluation of complicated bacterial meningitis in childhood? *Dev Med Child Neurol* 1992;34:870-5.
12 McWilliam R, Stephenson JBP. Bedside intracranial pressure monitoring. *Lancet* 1985;ii:341.
13 Snyder RD. Ventriculomegaly in childhood bacterial meningitis. *Neuropediatrics* 1984;15:136-8.
14 Polpe IK, Muhlbauer MS, Sanford RA, Kirk E. Results and complications of intracranial pressure monitoring in 303 children. *Pediatr Neurosurg* 1995;23:64-7.
15 Odio CM, Fainezicht I, Paris M, Nassar M, Baltodano A, Rogers J, et al. The beneficial effects of early dexamethasone administration in infants and children with bacterial meningitis. *N Engl J Med* 1991;324:1525-31.

(Accepted 3 December 1997)

A memorable patient

It pays to be specific

When working as a general practitioner on a housing estate in south east London in the 1960s I went to a lecture on psychiatry. It was mainly about how depression presents in many different ways, mimicking other conditions. Often a patient arrives at the psychiatric clinic having been through a battery of investigations, the psychiatrist being used as a last resort.

My receptionist remarked one day that her next door neighbours, an elderly couple, were to get a flat on the estate. I was warned that their previous doctor would be pleased to get rid of them.

"Him and his back," she said, "*x* rays by the score. Doctor says he's got nothing more than we'll all get when we get to his age, but he's such a misery he drives his poor wife round the bend."

The old man was obviously depressed. It all fitted in nicely. I started him on a small dose of antidepressant, working it up gradually, warning him that he might feel a bit giddy at first. He was very cooperative. After a few months there was a great improvement in his mood; his wife was happier too.

Unfortunately, picking up his tablets from the chemist one day he said, "Some more of my wonderful pain killers. The pain in my back has almost gone."

The chemist promptly enlightened him. "No, they are not for your back, those tablets are for your nerves," he said.

Both husband and wife left my list. They were angry with my receptionist too. I never heard how their new doctor managed them.

I did not make that mistake again. I spent hours explaining to people as best I could how conditions commonly referred to as "nerves" are not to be ashamed of and should respond to treatment. It is hard for the average patient to see depression as an illness in its own right, with mood changes as simply one of the symptoms.

"What's he got to be depressed about?" they say.

I often think we ought to change the name. For example, we no longer talk about mental defect.

Jean Troughton, *retired general practitioner, Bexley*

We welcome articles up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake,* or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk. Permission is needed from the patient or a relative if an identifiable patient is referred to. We also welcome contributions for "Endpieces," consisting of quotations of up to 80 words (but most are considerably shorter) from any source, ancient or modern, which have appealed to the reader.