infection from inadequate sterilisation of the instrument, may follow fibreoptic bronchoscopy, but it has serious consequences only occasionally,[3 4] and usually responds promptly to treatment with antibiotics.

If the purpose of the examination is merely to inspect an intrabronchial lesion and take a biopsy specimen from it the risk of the procedure is very small indeed—so long as the patient's respiratory function is adequate, too large a dose of opiate is not used for sedation, and the operator is not foolish enough to occlude a tracheal stenosis with the tip of the bronchoscope. The fibreoptic bronchoscope (unlike the rigid bronchoscope, which is an open tube) acts as a space-occupying lesion within the main airway and impairs respiratory function both in patients with obstructive airways disease and in normal persons.[5 6] The partial pressure of oxygen in arterial blood (PaO_2) falls, but the changes are usually small and are reversed soon after the bronchoscope is withdrawn.[6] Nevertheless, a protracted examination by an inexperienced operator in a patient with severe airways obstruction—particularly if heavy sedation has been given—could well produce a critical reduction in PaO_2 to a level at which it might precipitate respiratory and cardiac arrest. Such events are known to be rare in specialised units where many examinations are performed and serious complications are fully recorded,[1 2 7] but they may be less uncommon elsewhere—and remain unreported—when the procedure is undertaken by "occasional" bronchoscopists. The rule should be that before any patient with exertional dyspnoea is submitted to fibreoptic bronchoscopy under local or general anaesthesia his respiratory function must be assessed by simple measurements of ventilatory capacity and arterial blood gas studies. Only then is it possible to weigh the risk of the procedure against the benefit the patient is likely to derive from it and to decide whether oxygen should be administered during the examination.

Two further serious hazards are pneumothorax (from penetration of the visceral pleura by biopsy forceps) and haemorrhage from a bronchial artery. Both are inherent in the technique of transbronchial lung biopsy for either localised or diffuse pulmonary lesions. In skilled hands the risks of this procedure are slight and are usually acceptable, because they are less than those of percutaneous needle biopsy and open thoracotomy. In one recent survey[2] the incidence of pneumothorax was reported to be 5%. This is a low figure, and the complication, if quickly recognised and treated, should not pose any major problems; but nevertheless to take biopsy specimens from both lungs at the same session in patients with diffuse pulmonary disease seems unwise. There is a strong case for performing every transbronchial lung biopsy under radioscopic control (which is, of course, essential in the case of localised lesions) because this must reduce the risk of penetrating the visceral pleura with biopsy forceps. As a further prudent precaution a chest radiograph should be taken before the patient leaves the *x*-ray department at the end of the procedure, so that pneumothorax can be detected without delay and an intercostal tube inserted if the pneumothorax is large or is causing any respiratory discomfort.

Haemorrhage of over 50 ml is uncommon during transbronchial lung biopsy[1 2 8] (and is indeed hardly mentioned in many reports), but the very nature of the procedure requires transection of the wall of a small bronchus. The neighbouring bronchial artery is unlikely to escape damage, so that some bleeding is inevitable, and, though it usually abates within a few minutes, few bronchoscopists will have been spared the uneasy experience of a total loss of vision when the distal lens of the bronchoscope is suddenly and completely obscured by blood which cannot be cleared by suction through the narrow instrument channel. If bleeding of this degree continues the patient may be in serious danger, and a rigid bronchoscope or an endotracheal tube should always be available so that large accumulations of blood can be removed quickly by suction through a wide-bore aspirating tube or catheter. Facilities for cardiac resuscitation should also be at hand.

Fibreoptic bronchoscopy has much to offer in the diagnosis of bronchial and pulmonary disease, particularly tumours in the segmental divisions of the upper lobe bronchi, peripherally situated lesions, and diffuse pulmonary abnormalities. Published reports suggest that it has a very low morbidity and mortality, but this may not always be the case if proper precautions are not strictly observed—as indeed they should be in every invasive investigative procedure.

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Typhoid fever

In Britain typhoid fever frequently produces the same reaction from the press as much more serious infections such as smallpox and Lassa fever. This is unjustified. The mortality rate from major smallpox and the viral haemorrhagic fevers may be as high as 30%, and there is no specific treatment available for these virus infections; in contrast, typhoid fever responds to several antibiotics, and, provided the diagnosis is made in time, no patient should die from this disease. Further, whereas smallpox is readily transmitted by the airborne route, direct person-to-person spread of typhoid rarely occurs.

Notifications of typhoid fever in Britain are rising annually, and every year there are one or two deaths. There were 199 notified cases of typhoid fever in 1977, and the number is likely to be considerably higher for 1978. In the past decade almost all the typhoid seen here originated in Asia, but in recent months the disease has again been seen in holiday-makers recently returned from Spain.

Early diagnosis is vital in typhoid fever; delay in starting treatment may result in complications such as perforation of, or haemorrhage from, the small bowel, which may be fatal. The diagnosis of typhoid may be delayed by the blind use of an antibiotic such as ampicillin, amoxycillin, or co-trimoxazole for treating an undiagnosed febrile illness. Recent reports from Britain have shown that delays of this kind may be as long as four weeks.[1 2]

The early symptoms of typhoid fever are non-specific, being related to the septicaemic phase of the illness. Sustained high fever is common, though swinging fever may occur; repeated rigors are less frequent than in other septicaemic illnesses. Headache is almost invariable at some stage. During the first week the spleen may be palpable, but apart from fever there may be no abnormal physical sign. That classic sign of typhoid fever, "rose spots," is frequently absent.[1–3] The rash is pink and may not be noticed on a pigmented skin. As the disease progresses further symptoms and signs develop; multiple

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systems are commonly affected, producing myocarditis, encephalitis, cough and pain in the chest, renal symptoms, and diarrhoea and vomiting.[1 2] In children there may be a less severe illness which may present with diarrhoea and vomiting, leading to a mistaken diagnosis of gastroenteritis.

Blood culture is the most important investigation. The Widal reaction may be helpful, but it can be unreliable, especially in the early stages of the illness. Leucopenia and anaemia are frequent, and there may be evidence of disseminated intravascular coagulation.[3] Although jaundice is unusual in typhoid, mild abnormalities in the results of liver function tests are common.[3]

Typhoid responds to chloramphenicol, amoxycillin, co-trimoxazole, and mecillinam; and studies suggest that, given sensitive organisms, there is little to choose among the various antibiotics.[4] Nevertheless, unlike other infections caused by Gram-negative bacilli, response to treatment is slow. The mean duration of fever after start of treatment is five days irrespective of the antibiotic used; while blood culture results may remain positive for up to 10 days after the start of treatment, a curious phenomenon for which there is no apparent explanation.

The best advice on enteric fever is contained in the *Memorandum on Typhoid and Paratyphoid Fevers:* prepared by the Standing Medical Advisory Committee for the Central Health Services Council: "Typhoid and paratyphoid fevers should be considered as a possible diagnosis in any patient who has unexplained pyrexia for three days or more; if the patient has recently been abroad it should be considered from the first day of illness."

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Manic states in affective disorders of childhood and adolescence

Whether or not children ever develop diagnosable and treatable affective disorders is still hotly disputed—despite a mass of publications over more than two decades[1 2] and an international conference.[3] A recent American paper[4] on the treatment of depressed children with learning disorders even carried a cautionary editorial paragraph on the dangers of diagnosing let alone treating this condition in children. Yet depressive illness in children accounted for 10% of the unselected-intake patients seen in a European urban child psychiatric service,[5 6] and the malignant effects of the untreated illness on the intellectual and general development of children is beginning to be realised and described.[7]

Clinical reality has forced the occasional recognition of manic states in childhood, and several reports of single cases or small groups have appeared from time to time. Such reports have become more common since the increasing acceptance of lithium treatment in adults.

The manic state, like the depressive, can be considered as an affective response to endogenous (physiological) or environmental stress.[8] This may be especially true in children. While manic behaviour is to be expected within the manic-depressive illness, a manic component may also complicate schizoaffective illnesses[9 10] and severe chronic anxiety states. Response to lithium treatment is occasionally used as a diagnostic pointer in all these groups, and several workers have described[8 11-13] some children who respond to lithium among apparently hyperactive anxious individuals difficult to identify within the total group of overactive children. Anthony and Scott[14] listed the characteristic features of manic illness in children as including the following: evidence of an abnormal psychiatric state close to the classical clinical description at some time of the illness; a family history suggesting a manic-depressive diathesis; and an early tendency to a manic-depressive type of reaction as manifested in a cyclothymic tendency with gradually increasing amplitude and length of the oscillations and in delirious manic or depressive outbursts occurring during pyrexial illness. The illness might be recurrent or periodic with at least two observed episodes; or diphasic with swings of pathological dimensions; or endogenous illness, with its phases showing minimal reference to environmental events. Often it would be severe enough to indicate the need for inpatient treatment, heavy sedation, or electroconvulsive treatment. Finally, they looked for an abnormal underlying personality of an extroverted type in the absence of schizophrenic or organic states—and emphasised that these should be current not retrospective assessments.

A more clinical approach may often be helpful, with particular attention being given, firstly, to affectivity—exuberant noisy hilarity, unrestrained playfulness, mischievousness, arrogance, aggressiveness; secondly, to stream of thought—ideas of grandeur, delusions of wealth or power, sarcasm, flight of ideas, illogicality, short attention span, and distractability; and, thirdly, to psychomotor activity—overactivity, noisiness, meddlesomeness, socially inappropriate behaviour, loquaciousness and emphatic speech, physical aggression and impulsiveness, and accident proneness. Typically, some vegetative disturbance is also found—failure to eat, insomnia, and abdominal pain—and, particularly in younger children, an admixture of depression which may suddenly supervene with suicidal intensity. Many manic-affective disorders may be misdiagnosed as behavioural disorders or personality problems.[15] The symptoms are intense but variable, and in children they merge all too easily into apparent boisterousness or attention seeking. Vegetative and intellectual problems should give the clue to the diagnosis.

Treatment with lithium carbonate is often effective in resolving manic illnesses in children and adolescents.[10 11 17 18] The problem is to recognise those patients who will respond among the anxious, the schizoaffective, and the manic manic-depressive patients—for a mixed manic-depressive picture is frequently seen in children.[16]

Youngerman and Canino[18] collected reports of 190 children and adolescents aged 3-19 treated with lithium carbonate. Full details were given of only 46—25 boys and 21 girls. Thirty of the 46 had responded. The important features among manic patients who responded to lithium seem to be a positive family history of affective illness,[16] especially if this had responded to lithium treatment; a strong affective component to the presenting illness, particularly with mood swings or fluctuations in learning ability or other periodic behaviour alterations; major mood disturbances with an irregular cyclic pattern (recurrent stupors, frequent outbursts or suicide attempts, or fluctuating psychotic illness[10]), and aggressive or hyperactive behaviour with a major affective component.

Lithium carbonate is certainly worth a trial in such patients

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