for the deleterious effects of such adversity, provided that it is compensated sufficiently by all other aspects of the environment—as well as the stimulus that used to be called tender, loving care. Perhaps malnutrition reduces human achievement only when it is added to other adversity. But, as we must confess to our collective shame, it usually is.

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Massive haemoptysis

All too often massive haemoptysis is a medical catastrophe: patients may die within a few minutes from asphyxiation before conservative or surgical measures can be carried out. Among the causes of massive haemoptysis are mycetoma, bronchial carcinoma, chronic cavitated pulmonary tuberculosis, lung abscess, and bronchiectasis; it has also been reported in necrotising arteritis. We should not forget, however, that pulmonary tuberculosis in its acute stages—with little or no obvious cavitation—may cause massive fatal pulmonary bleeding. A recent report described five deaths from haemoptysis due to acute pulmonary tuberculosis in a single hospital within six years.[1] All the patients were aged 45 or less, and, though all had been noted to have haemoptysis on admission, none had appeared to have such destructive disease that a massive haemoptysis seemed likely. Tuberculous cavities have a particularly rich bronchial blood supply, and when heavy bleeding occurs it comes from dilated bronchial arterioles[2]; such dilated bronchial vessels are also found in mycetoma and bronchiectasis.

How should the clinician attempt to predict the outcome in severe haemoptysis? The only reliable guide is that the faster the rate of blood loss the more likely is the patient to die, the mortality rate being about 75% if blood loss exceeds 600 ml in six hours.[3,4] Surgical intervention is not an easy solution, however, even when technically practicable: the mortality rate is between 15% and 20% after resection of bleeding lesions.[5–7] Surgical treatment is often precluded by the poor respiratory reserve often found in patients with mycetoma and widespread pulmonary tuberculosis or by inoperable or disseminated carcinoma. An alternative may be emergency induction of an artificial pneumothorax.[8]

The patient who develops more than minor haemoptysis should rest in bed—lying on the affected side if this is known. A surgeon should assess his condition early on. The pulse and blood pressure should be monitored as well as the amount of blood lost—in view of the prognostic importance of the rate of haemorrhage. Careful radiographic assessment, including tomography if necessary, is important. Six units of blood should be made available and the patient must clear the airways by gentle coughing; he should not be sedated.

Bronchoscopy is central to management.[3,9] Using the rigid instrument, the airways can be cleared of blood, some of it in the form of clots. There is a common misconception that when the patient is bleeding bronchoscopy is valueless for identifying the source precisely. In one series, however, it identified the site of bleeding in 65 out of 67 patients,[3] and in another in 18 out of 21 patients.[9] Occluding the bronchial artery by embolisation is a skill possessed by few clinicians, but many more are skilled in the use of the fibreoptic bronchoscope, so that the method of arresting bleeding described by Saw *et al*[4] is important. Combining flexible fibreoptic bronchoscopy with systematic lavage of the tracheobronchial tree they identified the source of bleeding and stopped it in all their 10 patients (six of whom had pulmonary tuberculosis). The bleeding was halted by balloon tamponade of the affected segment with a Fogarty catheter; the balloon was deflated after 24 hours and the catheter removed a few hours later.

Clearly, then, aggressive methods of diagnosis permit early surgical intervention should haemoptysis increase. Nevertheless, the selection of patients for intervention remains a daunting problem.

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Hypertension and oral contraceptives

Practically all women who take oestrogen-containing oral contraceptives have a rise in blood pressure. Severe or even malignant hypertension is rare,[1] but about 4% develop diastolic blood pressures of 90 mm Hg or more.[2] This prevalence is about twice that in women of the same age not using oral contraceptives. Furthermore, there is a significant and sustained rise in blood pressure even in women who do not develop hypertension. A major prospective study in Glasgow showed a mean increase of 9·2 mm Hg in systolic pressure and 5·0 mm Hg in diastolic pressure[3] among 83 women, though in none did the diastolic pressure exceed 90 mm Hg.

The long-term effects of this change in blood pressure are unknown. Many epidemiological studies of large populations have shown that the height of the systolic blood pressure is a potent predictor of subsequent cardiovascular disease.[4] Small rises which remain within the range conventionally regarded as normal or clinically insignificant might therefore be expected to be associated with increased risk—and, indeed, an excess of deaths from non-rheumatic heart disease and cerebrovascular disease is found particularly in older women taking oral contraceptives, though in such studies antecedent hypertension has not been documented.[5–7]

The results of attempts to understand the mechanism of the