

When primary lung infection is the main aetiological factor the common causes are viral bronchitis or bronchiolitis, often complicating pertussis or measles, and bacterial pneumonia. Bronchiectasis frequently occurs in fibrocystic disease and in congenital or acquired hypogammaglobulinaemia, because these patients are especially prone to lung infections. Among primary lung lesions are congenital bronchiectasis; situs inversus and sometimes pulmonary sequestration (Kartagener's syndrome²), which tends to show an autosomal recessive mode of inheritance and to be associated with low serum IgA levels³; and bronchomalacia due to congenital absence of cartilage distal to the first division of the peripheral bronchi (Williams-Campbell syndrome⁴). More common are lung collapse and subsequent infection due to inhaled foreign bodies, infected congenital cysts, and inhalation pneumonia. In adults, bronchiectasis is most commonly a result of lung collapse, which is often due to bronchial carcinoma or tuberculous bronchial stenosis, and it may also occur in diseases complicated by bronchopulmonary aspergillosis.⁵

Whatever the underlying cause of the bronchiectasis the common infecting organisms are *Haemophilus influenzae* and *Streptococcus pneumoniae*, and infection with *Staphylococcus aureus* is an ever-present risk, especially in fibrocystic disease. The physical signs depend on the stage of the disease. There may be none, but in more advanced cases there is upper respiratory tract sepsis, purulent sputum (very rarely fetid), and finger clubbing, while signs of collapse and consolidation are frequent over the damaged and infected area of lung. Persistent medium or coarse post-tussive crepitations over the bronchiectatic area are a frequent and important physical sign. A plain chest radiograph may show areas of collapse and consolidation, cyst-like shadows, and bronchial dilatation, but for diagnosis and to demarcate the extent of the disease bilateral bronchography is essential. Complications of the disease include lung abscess and empyema and more rarely cerebral abscess or suppurative encephalitis.

The incidence of bronchiectasis is less than it was. Between 1952 and 1960 the hospital admission rate for children fell four fold.⁶ This reflects the control of infection by antibiotics and the reduced incidence of childhood respiratory infections attributable to immunisation against the specific fevers.

In treatment prevention is of first importance. Pulmonary infections should receive thorough treatment with appropriate antibiotics and with postural drainage to ensure that areas of collapsed lung expand. Upper respiratory sepsis must be sought out and treated, and in hypogammaglobulinaemia injections of concentrated gammaglobulin will be needed every four weeks or so. In established bronchiectasis the aim is to keep the airways clear of secretion by a properly learnt technique of postural drainage; this is combined with control of infection by broad-spectrum antibiotics such as tetracycline, oxytetracycline, ampicillin, or amoxycillin. Co-trimoxazole is an effective drug, but when given over a long period it may lead to folic acid deficiency. If *Staph aureus* is grown from the sputum ampicillin should be combined with cloxacillin, unless the organism is sensitive to benzylpenicillin. Antibiotics are probably best given only when the sputum is purulent, and in some patients vigorous medical treatment as outlined may lead to a reversal of the bronchiectatic disease.¹ As these young patients grow up they should be warned against the dangers of smoking. Surgical resection of the bronchiectatic area must be considered only when the patient has had an adequate course of medical treatment without any real improvement. Those most likely to benefit will have localised disease with prominent cough and sputum.

- ¹ Williams, H E, and Phelan, P D, *Respiratory Illness in Children*, p 198. Oxford, Blackwell, 1975.
- ² Kartagener, M, *Beiträge zur Klinik der Tuberkulose und spezifischen Tuberkulose-Forschung*, 1933, 83, 489.
- ³ Holmes, L B, Blennerhassett, J B, and Austen, K F, *American Journal of Medical Science*, 1968, 255, 13.
- ⁴ Williams, H, and Campbell, P, *Archives of Disease in Childhood*, 1960, 35, 182.
- ⁵ Crofton, J, and Douglas, A, *Respiratory Diseases*, p 429. Oxford, Blackwell, 1969.
- ⁶ Field, C E, *Archives of Disease in Childhood*, 1969, 44, 551.

Search for presymptomatic large bowel cancer

The quest to reduce cancer deaths, enshrined in the United States National Cancer Plan, is watched with a mixture of interest and scepticism in Western Europe. Apart from screening for cervical cancer the NHS has yet to commit its already stretched resources to a large-scale hunt for presymptomatic cancer. Nevertheless, it is worth considering what might be done to detect colon and rectal cancer—a common disease with a sufficiently optimistic outlook (compared, say, with lung cancer) to warrant the belief that finding early cases might be worth while. At present about 45% of the patients with large bowel cancer will die of the disease within five years of presentation: in the USA 50 000 persons will die of it this year.

The outcome of treatment of large bowel cancer is closely related to the stage of progression of the disease when first diagnosed. On the Dukes classification stage A is a tumour limited to the submucosa, B is with invasion through the muscularis without nodal metastasis, and C with spread to the nodes. The five-year survival of stage A lesions was 61-81%, stage B 39-64%, and stage C 27-28% in two major surveys of British and American experience of rectal carcinoma.^{1 2} The argument is that if cases were found earlier more would be in stage A and B and the results of treatment would be better.

In the USA, where the annual medical check-up is in vogue, the American Cancer Society's advertising campaign extols the virtues of annual proctoscopy for the over-40s. Such procedures are in practice confined to the middle and upper classes, so that they only touch the tip of the iceberg of the population at risk. The benefits are hard to judge objectively for the lack of suitable data for analysis. One trial begun in 1964 is worth attention.³ Two groups of subscribers to the Kaiser-Permanente health insurance plan aged 35-54 were studied. One group of 5156 were urged to attend for annual multiphasic health check ups, while a second control group of 5557 were left free to seek medical advice as they chose. After seven years there had been 40 deaths from cancer of all sites in the study group and 62 in the controls, but there had been only two deaths from colon cancer in the study group and ten in the controls. There was also a higher rate of detection of benign tumours of the colon in the study group. The trend in this small experiment suggests that the annual examination may have some merit so far as colorectal cancer is concerned, but the evidence is only tentative.

The carcinoembryonic antigen (CEA) was hoped to provide the basis of a screening test for bowel cancer to be used in the population at large. Nevertheless, extensive investigations in recent years have largely dismissed this idea. A study begun in 1969 in Busselton, Western Australia, illustrates the problems that would arise if the CEA test was used for mass screening.⁴ In an unselected population of 956 persons over the age of 60 a

total of 44 (4.5%) had a positive CEA test (≥ 5 ng/ml plasma) at the outset. During the next four years six of them died of CEA-associated cancers, two were found to have colonic diverticula, and 15 were found to be heavy smokers. By contrast 18 of the 912 CEA-negative individuals developed CEA-associated cancers during this period. Re-examination four years later of 21 of the apparently healthy persons who had been CEA-positive in 1969 showed that two had occult cancers, one of colon and one of lung. This small yield of cancers does not justify extensive clinical study of asymptomatic CEA-positive persons—which would have to be repeated—since half remain well for as long as four years and probably longer.

A simpler approach has been advocated by Lefall, who believes that annual testing of the faecal samples in the over 40s could reduce the toll of colorectal cancer.⁵ His idea is to issue the public with guaiac-impregnated paper slides, which they use for the test on three successive days after being on a high-residue meat-free diet. The slides are then returned to the doctor for development and reading. Lefall claims that this screening procedure yields a 5% positive rate and that 1% of these positives may be due to bowel cancer. His report did not state how the doctor decided whether to proceed to a formal examination of the bowel if the test was positive.

The simple occult blood test or the more complex annual medical check and proctoscopy would undoubtedly bring a crop of presymptomatic bowel cancers to the notice of the doctor and the patient. The crucial question is whether the lead time over the natural point of symptomatic presentation would result in a change in the mortality or only an extension of survival equal to the lead time.⁶ At present this question is unanswered; as it can be resolved only by formal testing, the size of the problem should not be underrated. Maybe this is the type of research that the regional cancer organisations might co-ordinate, by comparing survival in screened and unscreened populations. Large numbers would be required—the incidence per 100 000 at the age of 65–69 is 190 in men and 126 in women—so many general practitioners would need to be enlisted to help, and it is unlikely that there would be an answer in less than seven years. Such a programme might turn the tide of the disease; on the other hand such phrenetic activity with its extra demand for diagnostic facilities could hinder our ability to deliver health care for patients with overt disease—a nice point to debate. Maybe we should have been better off spending our time encouraging the patient with symptoms to seek advice and the general practitioner to keep the probability of bowel cancer well to the fore when dealing with the over-60s.

¹ Bussey, H J R, Dukes, C E, and Lockhart-Mummery, H E, in *Cancer of the Rectum*, ed C E Dukes, p 267. London, Livingstone, 1960.

² McSherry, C K, Cornell, G N, and Glenn, F, *Annals of Surgery*, 1969, 169, 502.

³ Dales, L G, et al, *Preventive Medicine*, 1973, 2, 221.

⁴ Stevens, D P, Mackay, I R, and Cullen, K J, *British Journal of Cancer*, 1975, 32, 147.

⁵ *Journal of the American Medical Association*, 1975, 234, 137.

⁶ Zelen, M, and Feinleib, M, *Biometrika*, 1969, 56, 601.

Nevertheless, while cardiac contusion is easily recognisable at necropsy from the obvious damage and bruising of the ventricles, it is a good deal more elusive clinically, both in its incidence and in its clinical definition.

Clinically, contusion is said to be present when, after blunt chest injury, there is electrocardiographic evidence of myocardial infarction or ischaemia which eventually disappears. Diagnosed by these criteria its incidence is increasing, owing to a general increase in such injuries and to heightened diagnostic awareness. Typically, there is chest pain of myocardial ischaemic type, but this may be difficult to elucidate since most patients have some radiological or clinical evidence of injury to the chest wall. The electrocardiographic changes are those of myocardial ischaemia and infarction,¹ usually ST-segment and T-wave changes. These may not develop until two days after the injury, which may cause the diagnosis to be delayed or missed. Serum enzyme levels are often raised, but this is of no diagnostic value as the rise may be due to injuries of liver, lung, or skeletal muscle. Other diagnostic procedures such as measurements of cardiac output² or technetium scans¹ are too complex for most accident units.

Cardiac contusion is not usually fatal in itself: death may be due primarily to other injuries sustained, though sometimes contusion occurs with no external evidence of damage to the chest wall whatsoever. The specific dangers are the development of pericardial tamponade and dysrhythmias, including atrial fibrillation, supraventricular and ventricular tachycardia, and complete heart block. These tend to occur in patients who are suffering from hypotension and hypoxia and who are therefore the most seriously injured.

The importance of diagnosing, or at least suspecting, cardiac contusion in an injured patient lies in preventing fluid overload and starting cardiac monitoring to identify the dangerous dysrhythmias when they occur. Giving large volumes of fluid, particularly blood, is usual in the management of major blunt trauma, and overload may be fatal. In one survey¹ four of the five patients with cardiac contusion who developed congestive cardiac failure did so during intravenous fluid replacement. If dysrhythmias are recognised quickly from the electrocardiogram and oscilloscope they can be managed effectively with, in the first instance, digitalis for supraventricular arrhythmias, lignocaine for ventricular tachycardia, and an intravenous pacemaker for heart block. Tamponade may be suspected from central venous and arterial monitoring and confirmed by injection of contrast medium into the right atrium, showing a wide shadow between it and the pericardium. It is treated by tapping with a needle or, better, a small catheter inserted under the xiphisternum by the Seldinger technique. Thoracotomy is rarely necessary for contusion alone.³

Management of cardiac contusion does not end with discharge of the patient from the ward. Complications may occur months or even years later,^{4 5} and these may include ventricular septal defects, left ventricular aneurysms, or coronary artery occlusion. Suspicion of cardiac contusion should, therefore, be enough to trigger a cardiac rather than a purely orthopaedic approach to blunt trauma. Its recognition may prevent a relatively benign condition leading to fatal complications.

Cardiac contusion

Contusion of the myocardium—recognisable damage to heart muscle short of rupture—is a fairly common concomitant of blunt injury to the chest, a type of injury that (until the oil crisis at least) has been steadily increasing around the world.

¹ Jones, J W, Hewitt, R L, and Drapanas, T, *Annals of Surgery*, 1975, 181, 567.

² Pomerantz, M, Delgado, F, and Eiseman, B, *Surgery*, 1971, 70, 865.

³ McDermott, F T, and Douglas, M C, *Australian and New Zealand Journal of Surgery*, 1967, 37, 147.

⁴ Doty, D B, et al, *Annals of Surgery*, 1974, 180, 452.

⁵ Stern, T, et al, *Journal of the American Medical Association*, 1974, 230, 1308