

The site of the bronchiectasis varies, and the condition may be due to a congenital abnormality of the bronchi.

Brain Abscess and Empyema.—Although brain abscess was a classical cause of death in bronchiectasis, it is now less commonly seen, presumably owing to better treatment of the disease. It is usually seen in a patient with well-established

moderate or severe bronchiectasis. Most cases of empyema, when they complicate bronchiectasis, do so in fairly well established moderate or severe cases.

(This article will conclude next week with a discussion on the treatment and prevention of bronchiectasis.)

ANY QUESTIONS?

We publish below a selection of questions and answers of general interest.

Coronary Disease and High Altitudes

Q.—Is it advisable for persons who have had a coronary thrombosis in the past to travel by air or to stay on holiday at a high altitude?

A.—Oxygen is readily taken up or released from haemoglobin for small changes in oxygen tension below about 60 mm. Hg (steep part of the oxygen dissociation curve) but is in more stable combination at higher oxygen tensions (flat part of the oxygen dissociation curve). This means that the saturation of arterial blood with oxygen falls relatively little until the ambient oxygen tension has dropped from the normal level of about 94 mm. Hg to below 60 mm. Hg.

The cabin pressures of most aeroplanes are maintained at or above 565 mm. Hg, which is equivalent to an altitude of 8,000 ft. (2,438 m.). At an atmospheric pressure of 565 mm. Hg the partial pressure of oxygen is therefore only 118 instead of the normal 149 mm. Hg prevailing at sea-level, and the oxygen tension of the alveolar air (fully saturated with water vapour) is only 70 instead of 100 mm. Hg. However, assuming a normal alveolar-arterial gradient for oxygen, the arterial blood will still be nearly 90% saturated at this level of oxygen tension. This altitude does not therefore lead to significant tissue hypoxia under resting conditions, although dyspnoea will be appreciated by healthy persons on hurrying until acclimatized.

At an altitude of 12,000 ft. (3,657 m.) with an atmospheric pressure reduced to only 430 mm. Hg the oxygen tension of alveolar air will be 51 mm. Hg. The arterial oxygen saturation will be down to about 82%, and rapid ascents even to this altitude have been associated with the development of pulmonary oedema, particularly when exercise has been attempted soon after arriving. At 15,000 ft. (4,572 m.) the arterial oxygen saturation will be only 65%, because at this range of altitude small decrements in atmospheric pressure give rise to considerable decrements in blood-oxygen saturation.

Altitudes of up to 5,000 ft. (1,524 m.) permit virtually normal arterial oxygen saturations. At 5,000 ft. the barometric pressure will be about 540 mm. Hg, the alveolar oxygen tension about 74 mm. Hg, and the arterial oxygen saturation about 94%.

The answer to the question, then, is that it is obviously inadvisable for patients with coronary artery disease to expose themselves

unnecessarily to reduced ambient oxygen tensions, because even a small reduction in the amount of oxygen available to an area of myocardium with a critically low blood flow can be dangerous. Specifically, air travel in pressurized planes where exertion is impossible probably imposes a negligible risk for most patients who have had a coronary thrombosis, but a man who develops anginal pain on mild exertion should be advised to take oxygen during the trip or, better, to go by sea.

A holiday at an altitude beyond about 3,000 ft. (914 m.) is a different matter. Hypoxia is more sustained and exertion will exacerbate its effects. Moreover, the situ-

ation is an avoidable one and so should be advised against.

"Binocular" Spectacles

Q.—Are spectacles with an adjustable focus (like "binoculars") made and in what cases are they of value?

A.—Spectacle "binoculars" are obtainable and give some magnification for distance (e.g., the Sportscope with a magnification of $\times 3$). The field of view is somewhat restricted, but they might be of value in helping the partially sighted person to enjoy a theatre or some sporting events.

Notes and Comments

Autotransfusion.—Drs. L. S. CHEN, J. M. CONNAN, J. S. HSU, and C. H. HUNG (Changhua Christian Hospital, Changhua, Formosa, China) write: In reply to a question on autotransfusion ("Any Questions?" 25 December 1965, p. 1533) your expert stated that this was rarely performed. The availability of blood and comparative rarity of acute ectopic gestation no doubt account for this. In this hospital it is used fairly frequently for ectopic gestation, and we find that it not only eliminates the dangers of incompatibility and hepatitis but it allows earlier operation even before donor blood is available, and a more economical return to a normal haemoglobin (100 ml. of blood costs about £2). In 16 cases a total of 14,600 ml. of autotransfusion was given, while the donor blood for these cases amounted to only 5,100 ml.

Last year of 28 cases of ectopic pregnancy 16 fresh cases had autotransfusion, and the largest amount of blood transfused was 1,600 ml. We use mechanical suction directly into the bottle of citrate, and filter the blood only through the drip chamber, thus eliminating the time-consuming process of first filtering through gauze. It is then possible to begin autotransfusion within 10 minutes of starting the operation. The amount of unusable clot is estimated and replaced by donor blood after autotransfusion.

We have also used autotransfusion in a clean case with post-operative haemorrhage into the peritoneal cavity. The surgeons here have used it for a child with ruptured liver with dramatic effect. Naturally, the greatest worry is contamination, especially in cases of trauma, and we have twice been exercised about the wisdom of using blood for an ectopic when a simple follicular cyst of considerable size has ruptured during operation. In neither of these cases was there any noticeable ill-effect.

Protection from Trichlorethylene.—Dr. A. PATON (Dudley Road Hospital, Birmingham 18)

writes: I have lately seen two patients in whom exposure to trichlorethylene was considered as a possible cause for their symptoms. Having therefore had to look up the literature on my own account, I would like to supplement the rather meagre description of the signs of poisoning given by your Expert ("Any Questions?" 19 February, p. 471). Three organs besides the brain seem particularly susceptible—namely, the liver, the heart, and the kidneys. Jaundice and hepatic failure due to acute hepatic necrosis and cardiac arrhythmias from myocardial toxicity have long been recognized. In fact sudden death during trichlorethylene anaesthesia has been attributed to ventricular fibrillation. Less well known is a renal syndrome which mimics acute nephritis (as in the two patients referred to above) and which may progress to anuria.¹ It is perhaps as well to remember that if the patient dies histological evidence of poisoning may be minimal, so that an occupational history may be the only evidence.

OUR EXPERT replies: The case to which Dr. Paton refers¹ relates to quite exceptional exposure. The man concerned worked for two hours at least in a badly ventilated room using trichlorethylene to remove excess asphalt from tiles which he was laying and also to clean his hands. Most chlorinated hydrocarbon solvents can affect the liver and kidneys, but trichlorethylene rarely does so.

REFERENCE

- ¹ Gutch, C. F., Tomhave, W. G., and Stevens, S. C., *Ann. intern. Med.*, 1965, 63, 128.

Correction

We regret that in the article by Dr. E. Posner on industrial chest disease (26 February, p. 525) the name of Dr. A. I. G. McLaughlin was misspelled.