Artificial ventilation and the heart

Intermittent alterations of stroke volume and asynchronism of output of the two ventricles are known to occur during both spontaneous breathing and artificial ventilation.1 The effect of intermittent positive-pressure ventilation (particularly when the inspiratory phase is prolonged) is to raise the mean intrathoracic pressure and to lower both the stroke volume and the cardiac output.2,3 This reduction of cardiac output has usually been attributed to reduced venous return, increased pulmonary vascular resistance or impedance, and reduced pulmonary blood volume.4 The finding that hypovolaemia increases the effect of artificial ventilation on the circulation has reinforced the view that the important factor is reduced filling of the right ventricle.5

Positive end-expiratory pressure6 is widely used to improve oxygenation in patients with acute lung damage. This technique of continuous positive-pressure ventilation raises the mean intrathoracic pressure substantially; and the cardiac output decreases as the pressure in the airways increases.7 During positive end-expiratory pressure ventilation a rise in the volume of the circulating blood raises the cardiac output,8 so that again reduced venous return has been thought to be the most important determinant of the effect of positive end-expiratory pressure on the circulation. On the other hand, the observation that dopamine is also effective9 suggests that myocardial depression may also be important in the circulatory depression induced by positive end-expiratory pressure. Furthermore, isolated heart muscle preparations are depressed by the addition of plasma from animals subjected to positive end-expiratory pressure.10 Promoting the suggestion that substances with a negative inotropic effect may be released as the lungs are stretched. Stretching of the lungs may modify the metabolism of prostaglandins,11 one of which is thought to possess negative inotropic properties.12 This hypothesis of release of negative inotropic substance into the circulation could explain the observation that positive end-expiratory pressure may greatly reduce the cardiac output even though the chest wall has been removed13,14 —when the lungs are stretched to their maximum but venous return is unimpeded. The same hypothesis could also explain why when the chest is open and the pulmonary pressure is raised positive end-expiratory pressure reduces cardiac output more than constriction of the pulmonary artery.15

Yet another possibility is that the cardiac performance might be depressed by circulatory reflexes elicited by stretching the lungs: but the reduction of the cardiac output induced by positive end-expiratory pressure persists in vagotomised animals treated with beta-adrenoreceptor antagonists16 whose circulatory reflexes have been substantially blunted if not abolished. Finally, the myocardial depression might be due to reduction of or abnormal distribution of coronary blood flow; but this possibility has been disproved by the persistence of the effects of positive end-expiratory pressure on cardiac output while coronary flow and its distribution remained normal.17

Some studies have found no evidence of myocardial depression8,18,19 during positive end-expiratory pressure, possibly because of inconsistencies in the evaluation of intravascular pressures. While positive end-expiratory pressure increases intravascular pressures relative to atmospheric pressure it may decrease transmural pressure if intrapleural, intraoesophageal, or intrapercardial pressures are taken into account. In some studies lower cardiac output was associated with reduced transmural pressures,20 while in others it was associated with raised transmural pressures.13 14 16 17 The latter condition has often been interpreted as evidence of cardiac depression, but an alternative explanation for lower stroke volume and higher transmural pressure is a decrease of diastolic ventricular compliance.

A factor receiving increasing attention is the part played by the pericardium. This structure has a low compliance and is an important determinant of the relations between the two ventricles.18,19 When the pericardium is intact the right and left ventricular filling pressures are tightly correlated and changes in right ventricular afterload modify the preload of both the right and the left ventricle. Right ventricular loading modifies the configuration of the left ventricle and the relations between diastolic pressure and volume20 by flattening the interventricular septum.21,22 Positive end-expiratory pressure inevitably causes right ventricular loading, and indeed in patients with acute lung damage positive end-expiratory pressure increases the radius of curvature of the septum.23 Positive end-expiratory pressure also decreases the cross-sectional area of the left ventricle both at end-diastole and end-systole in a dose-dependent fashion. At high levels of positive end-expiratory pressure the radius of curvature of the septum may become further increased—and what has been interpreted as left ventricular failure may, in fact, because of the interdependence of the two ventricles, represent failure to fill and not failure to eject.24

One more factor that needs to be taken into account is alteration of carbon dioxide tension, since hypocapnia is known to reduce the cardiac output—and the full complexity of the relation of intermittent positive-pressure ventilation and the heart is now apparent. All these factors may assume greater or less importance according to the condition of the patient.

Geographical variations in disease in Britain

D J P BARKER

In 1889 the British Medical Journal published a report on the geographical distribution of rickets, rheumatism, chorea, cancer, and urinary calculus in the British Isles. The report was based on questionnaires completed by more than 3000 doctors at the request of the BMA. Among the findings were that urinary calculus was especially common in Norfolk and that rickets was mainly a disease of industrial areas. The author of the paper, Dr Isambard Owen, offered no explanation of these and other findings, simply urging that the study of geographical distributions of disease in Britain should be continued.

This important report was one of the first large-scale attempts to document geographical variations in the frequency of common disorders, but general interest in this subject was by no means new. For example, doctors in Norfolk had long been aware that bladder stones were remarkably common in their county, with records going back to 1600. In 1874 a Norwich surgeon had written to other surgeons in the large hospitals throughout Britain and obtained from each the number of cases of stone treated in the previous five years as a proportion of all inpatients. Speculating on the causes of the geographical variation shown, he discounted climate in favour of associations with lack of milk, malt liquor, and hardness of drinking water.

A year after the BMJ report Palm published the findings of a survey of rickets. He had asked medical missionaries in many countries about “the prevalence or absence of rickets, the habits of the people, and their climatic and sanitary conditions.” Analysis of the replies led him to the brilliant discovery that lack of sunshine was the main aetiologic factor in rickets.

During the second half of the nineteenth century the annual reports of the Registrar General showed quite clearly that mortality varied from one part of Britain to another. A striking feature of this variation was consistently higher death rates in the north and west than in the south and east. As the report of the Local Government Board for 1915 and 1916 stated, a map of the coal measures in Britain would almost have served as a map of the chief areas in which child mortality was excessive.

Remarkably, the decline in mortality during this century has not eliminated the disparity between the north and west and the south and east. Figures for the largest county boroughs in England and Wales show that from 1911 to 1967 death rates among middle-aged people steadily declined, but the highest rates have remained just less than double the lowest. Mortality in towns such as Oldham and Salford has consistently been almost twice that in towns like Ipswich and Norwich. The magnitude of the current differences is shown by the variations in life expectancy. Based on regional death rates for 1974-5, the expectation of life at birth for men ranges from 67-9 years in the North-western region to 71-3 years in the East Anglian region; for women the figures are 74-3 and 76-9 years.