is in themselves to be relied upon as a "method" of feeding. The first does not deal with total quantities, the second does not treat of the quality or "balance" of the ingredients of the food, while the third does not take account of the relation of the weight to the age. It is often wiser, if the child's weight is not near the average for the age, to make the calculation of the requirements on a figure which represents the mean between the average weight for the age and the actual weight of the child in question. Each individual problem of feeding should be submitted to the first two tests taken in conjunction with one another, and not to one alone. Such a system of procedure ensures method in dealing with cases in practice and begets confidence and good judgment when confronted with difficult feeding cases which cannot be treated with any lasting success by haphazard methods.

I have not attempted to present outlying fields of thought on this subject, but have concentrated purely from a practical point of view on the common methods of feeding healthy or slightly ailing babies, deliberately leaving the discussion of some other types of feeding to be employed, in the case of feeble and ill babies, or in special circumstances, to a subsequent lecture.

THE HEART IN PULMONARY DISEASE.

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So frequently do pathological changes in the lung follow cardiovascular disease that the examination of a patient suspected of circulatory disorder is scarcely complete unless the respiratory system has also passed under review. Some of these changes, such

as pulmonary œdema or infarction depend upon exacerbations or complicating factors ; others, for example, hydrothorax and bronchitis, are contributory to the circulatory embarrassment and may demand special treatment. In general, the signs of secondary pulmonary damage are fairly obvious, though their exact interpretation may be difficult.

While the importance of respiratory signs in heart disease is generally accepted, it is not so widely appreciated how profoundly the cardiovascular system may be affected in certain pulmonary diseases. This is due in part to the fact that the state of the lungs affords an important clue to the clinical condition in cardiac disorders, while changes in the circulatory system secondary to diseases of the lungs occur late as a rule, if they occur at all. It is certain, therefore, that more subjects of lung disease than is generally realized die a cardiac death.

Embarrassment of the heart and circulation in pulmonary diseases arises from one of two main sources. First, an extrinsic burden may be laid upon the heart due to mechanical factors, such as obstruction in the lesser circulation, or to displacement or compression of the heart and mediastinum as often occurs in spontaneous and in open pneumothorax. Secondly, acute or chronic infections, such as pneumonia or tuberculosis, may cause intrinsic and serious impairment of the circulatory mechanisms. These infections may act by damaging the heart muscle, by attacking the pericardium or aorta, or by abolishing peripheral vasomotor tone, so causing circulatory failure.

This paper will therefore deal initially with the mechanical factors represented by chronic pulmonary disease and by disturbances brought about through alterations in the position of the mediastinum and through variations in intrapleural pressure. Pneumonia, as an ætiological factor in heart failure, will next be considered, and finally, the unusual involvement of the cardiovascular system in tuberculosis.

THE LESSER CIRCULATION IN CHRONIC PULMONARY DISEASE.

(1) *Right Heart Failure*.—Chronic bronchitis, emphysema, fibrosis of the lung and extensive pleural adhesions, are the common findings in patients who have died with congestive heart failure secondary to chronic pulmonary disease. Because the heart fails before an extrinsic barrier, it fails with normal rhythm, and it has been shown by Parkinson and Clark-Kennedy [1] that about one-fifth of patients dying from congestive failure with normal rhythm are subjects of chronic pulmonary disease. They found in all cases relative hypertrophy of the right auricle and right ventricle, while hypertrophy and atheroma of the pulmonary artery were also usually present. Microscopical examination showed no myocardial change apart from fatty degeneration.

Another less common factor in the production of right heart strain and pulmonary atheroma is syphilis. Ayerza was the first to observe the association of syphilis with chronic pulmonary disease, right heart failure and extreme cyanosis, and he gave his name to this syndrome. The historical aspects and clinical features of the disease have recently been summarized by Konstam [2]. As a result of the chronic infective process, arteritis of the smaller pulmonary branches supervenes, with ensuing obstruction to the pulmonary circulation from peri-bronchial and interstitial syphilitic fibrosis. The sequel to mechanical stress in the lesser circulation is atheroma of the pulmonary artery, and hypertrophy of the right heart with ultimate failure. These patients give a clinical picture essentially comparable to that of heart failure from chronic pulmonary disease apart from syphilis. In addition to the usual expressions of congestive heart failure, these two groups of patients have in common intense cyanosis, polycythemia, severe chronic headache, vertigo and lethargy.

(2) *Tricuspid Incompetence*. Attention has been directed by Lutembacher [3] to the

abrupt occurrence of tricuspid incompetence with rapid death from heart failure in patients suffering from chronic lung disease. Such lesions may or may not have a tubercular basis; however, if tubercle is present it is always of the fibrotic type with little or no accompanying cachexia. Lutembacher has given the name "terminal tricuspid syndrome" to this clinical entity, and claims that it may be recognized by the following: (i) the establishment of venous stasis, epigastric pulsation and cyanosis in the course of a disease previously pulmonary; (ii) the appearance of a systolic murmur maximal over the xiphoid process without other murmurs and in the presence of a rapid regular rhythm; (iii) increasing dyspnoea, oedema and ascites; (iv) the appearance of right heart gallop which may transiently disappear with rest; and (v) the failure of rest and digitalis to give other than temporary relief.

The X-ray in these patients shows "sabot" type of heart as in congenital pulmonary stenosis; the right auricle is distended and there is enlargement of the heart to the left owing to the hypertrophy of the right ventricle which extends over to the left, its extremity being represented by the lowermost part of the left border of the heart as seen on radioscopy.

(3) *Pulmonary Insufficiency*.—An uncommon sequel of increasing strain in the lesser circulation is pulmonary insufficiency. This may be secondary to atheroma of the pulmonary artery as in Ayerza's disease and is then exactly comparable to the atheromatous type of aortic incompetence. On the other hand it has been stated by Vaquez [4], that extensive long-standing lesions of the lungs may be the principal morbid change. As in the more frequent variety of pulmonary incompetence supervening in the course of mitral stenosis a diastolic murmur is heard along the left border of the sternum. It is distinguished from the murmur of aortic regurgitation by the absence of left ventricular hypertrophy, the normal pulse-pressure,

and the absence of marked disparity between the blood-pressure in the leg and in the arm. On screening, the state of affairs becomes at once apparent, for the brisk darting shadows at the hila demonstrate the collapsing nature of the pulsations in the pulmonary artery. In aortic incompetence such shadows are not seen, and the excessive pulsation of the aortic shadow is the outstanding feature.

The inefficacy of treatment in these patients with heart failure secondary to chronic obstructive lesions in the lungs has already been mentioned. Rest, digitalis, and the administration of oxygen often give relief when failure first appears, and venesection is frequently beneficial. Despite such improvement failure recurs, becoming less amenable to treatment, and the patient generally dies within a year of the appearance of œdema.

THE HEART IN PNEUMOTHORAX AND MEDIASTINAL DISPLACEMENT.

Theoretically, it is possible for the cardiovascular mechanism to be embarrassed by simple displacement of the heart as occurs in fibrosis at the base of one lung, by displacement to one side with supernormal intrapleural pressure in the other side as, for example, in some cases of spontaneous pneumothorax, and by the oscillations of a lax mediastinum that may be set up by the respiratory movements in open pneumothorax—mediastinal flutter. Actually, simple mediastinal displacement as occurs in fibrosis and pleural effusion is found to give little or no evidence of cardiac disability or insufficiency, and there seems to be great adaptability on the part of the large vessels, such as the venæ cavæ, to the traction and torsion that must arise. Further evidence of this immunity of the heart from distress or damage in these displacements where no excess of intrapleural pressure co-exists is contributed by the electrocardiographic studies of Perrin and Drouet [5], who showed that in artificial pneumothorax certain physiological modifications were found

in the electrocardiogram. These changes were referable to the axis of the heart becoming more vertical, and these workers concluded that no changes characteristic of myocardial defect occur in artificial pneumothorax or pleural effusion.

In spontaneous pneumothorax there exists a greatly different state of affairs. At its inception and until treatment is obtained there is in many cases not only displacement of the mediastinum but also increasing pressure in the air-filled pleura. This fact was demonstrated sixty years ago by Douglas Powell [6], who measured the intrapleural pressure in eight patients dead from pneumothorax, finding an average pressure of three inches of water. Both the lungs and the heart are thus subjected to pressure, and Powell further showed by animal experiment that although a lung collapsed by its own elasticity offers no appreciable additional resistance to the circulation, more complete collapse causes a very considerable resistance. The great relief frequently afforded by puncture of the chest in spontaneous pneumothorax is further proof of the cardio-respiratory embarrassment sometimes produced.

Appreciation of the dangers attendant upon open pneumothorax is of more recent date, and the study of this condition received fresh impetus from the large mass of material accruing from the war. Not only were cases of traumatic open pneumothorax available for study, but also the pneumothorax not infrequently produced in operations for empyema. The latter group has been exhaustively studied by Graham and Bell [7]. By whatever means the pleural cavity on one side is put into free communication with the exterior through the chest wall, the consequences are apt to be serious. The first and most obvious effect is dyspnoea from hindrance of the respiratory functions. With each inspiration the mediastinum is sucked over to the uninjured side, thus causing imperfect filling of the sound lung. To add to the embarrassment, part of the air

aspirated into the sound lung is drawn from the lung partly collapsed. These two factors co-operate to produce general anoxæmia.

Subsequent effects of open pneumothorax have been classified by Sauerbruch as: (i) heat loss; (ii) the danger of infection; and (iii) circulatory disturbances. The last have been contested by some observers on the grounds that a stabilized mediastinal displacement (e.g., in effusion) causes no cardiovascular distress. There is, however, the positive evidence produced by Sauerbruch that the femoral venous pressure is increased in open pneumothorax, the arterial pressure remaining unchanged. It seems likely that Lilienthal's [8] summary of the matter is correct when he says that the immediate symptoms must be due to respiratory effects, while the circulatory consequences must exert their influence later.

The treatment of circulatory disturbances secondary to open pneumothorax is that of the underlying condition. In the traumatic variety closure by some means of the aperture in the chest wall gives rapid relief. The dangers of mediastinal oscillations in operations for empyema are better avoided than treated. The discovery on exploratory puncture of a thin sero-purulent fluid in the course of a streptococcal pneumonia suggests that the pus is not yet encysted and that a pyothorax is actually present. If this fluid is repeatedly aspirated until frank pus appears and adhesions have formed, rib resection and drainage may be carried out without danger.

CARDIOVASCULAR DISTURBANCES IN PNEUMONIA.

In general, pneumonia disables the circulatory apparatus by virtue of its severe toxæmia. This process has been well summarized by Hay [9] who emphasizes the poisoning of the myocardium and the consequent reduction of its power. A lowering of the vasomotor tone is brought

about by the action of the toxins upon the medullary centre, while the venous return diminishes through progressive stagnation in the peripheral circulation. Owing to the gross lesions in the lungs, anoxæmia supervenes affecting among other structures the myocardium; the right heart is further embarrassed by the pulmonary consolidation and by the restriction of diaphragmatic movement through pain and abdominal distension.

The incidence of intrinsic myocardial damage, and the frequency of degenerative or inflammatory lesions in the heart-muscle have been studied by Stone [10] who analysed the results of microscopic examinations in seventy-one autopsies. He found a normal heart-muscle in only one-fifth of the cases of lobar pneumonia, and in two-fifths of the cases of broncho-pneumonia. The principal degenerations were cloudy swelling and fatty and hyaline changes. Actual myocarditis as expressed by leucocytic and round-celled infiltration was found in as many as 10 per cent. of cases.

The value of digitalis in the treatment of pneumonia has been investigated by many workers. Direct evidence on this point has been produced by Levy [11] who radiographed the heart in pneumonic patients, finding generally a dilatation. He showed further that the heart dilated less if the patient were digitalized. It has been suggested that this action of digitalis is due to some form of linkage with the muscle-fibres—thus precluding the usual poisonous effect of the toxins of pneumonia. Tincture of digitalis from 40 to 60 m daily, or its equivalent, is the dosage most profitably employed. The administration of adrenalin, cold or tepid sponging, and rectal salines are the measures of greatest value in combating peripheral failure.

TUBERCULOUS INVOLVEMENT OF THE HEART, PERICARDIUM AND AORTA.

The association between diseases of the heart and tuberculosis seems at first to be a

remote one. This infection may, however, attack the pericardium and aorta and may apparently cause cardiac insufficiency apart from the mechanical obstructive effect produced by chronic fibrotic pulmonary lesions, previously mentioned. The simplification of our notions concerning the ætiology of mitral disease has led many to discredit even the possible rôle of chronic nephritis; but it may be recalled that tuberculosis has not always been exempt from suspicion in this connection. Potain believed that mitral stenosis was attributable to tuberculosis. At the present time it is believed that these two diseases co-exist very uncommonly.

(1) *Cardiac Insufficiency*. — A syndrome has been described by Godel [12] in which a patient suffering from pulmonary tuberculosis becomes dyspnoic and experiences attacks of paroxysmal nocturnal dyspnoea with the expectoration of abundant blood-stained sputum. Other evidences of congestive failure appear and left ventricular extrasystoles are observed in the electrocardiogram. The occurrence of this syndrome is said to bear no relation to the extent of the pulmonary lesions, but to be associated with the evolution or recrudescence of the disease; it may precede or accompany hæmoptysis. For these attacks the intravenous injection of ouabain $\frac{1}{4}$ mg. daily has been advised.

Special reference has been made in this place to the observations of Godel relating to active pulmonary lesions since they are in direct contrast to the syndrome described by Lutembacher, in which tuberculosis if present was fibrotic and unassociated with cachexia—i.e., quiescent or extinct. The mode of production of these attacks of cardiac insufficiency and their relation to the "terminal tricuspid syndrome" are as yet problematical.

(2) *Tuberculous Pericarditis*. — Statistical evidence suggests that this lesion may occur at any age, that it is commonest in children, and is found at autopsy in 2 per cent. to

3 per cent. of patients with tuberculous lesions. It has, moreover, been shown that although the disease often originates from caseous tuberculous mediastinal glands, it may also represent an extension of infection from the pleura and lung; sometimes the pericardium is involved as part of a general tuberculosis of the serous membranes.

Clinically, the disease has been classified by Osler [13] into four groups: (a) latent tuberculous pericarditis in which the discovery is made accidentally at autopsy; (b) symptoms of cardiac insufficiency following dilatation and hypertrophy of the heart consequent upon chronic adhesive pericarditis; (c) acute tuberculosis, either general or with manifestations principally referable to the nervous system; (d) the syndrome of acute pericarditis, with præcordial pain, dyspnoea, œdema and progressive deterioration of the general condition.

(3) *Tuberculosis of the Aorta*. — This disease is very rare and is only included here for the sake of completeness. Adams [14] has collected thirty-five cases from the literature. Infection usually reaches the aorta from without by extension from caseous glands, tuberculous mediastinitis or pericarditis, or from vertebral disease extension to the intima may follow, and death from acute miliary tuberculosis. In some instances the intima of the aorta has been directly attacked by organisms in the blood-stream. The result of this is acute or chronic tuberculous endoarteritis, with subsequent generalization of the infection.

No treatment is of avail in these uncommon tuberculous invasions of the pericardium and aorta except when there are signs indicating acute pericarditis with effusion. The application of the ice-bag to the præcordium may limit the production of fluid; if despite this measure the effusion increases, paracentesis is advisable.

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POST-GRADUATE NEWS.

OWING to the Christmas vacation, it is not possible to hold many special courses in December. From December 1 to December 14 a course in Diseases of Infants with special reference to nutritional disorders and dietetics will be held at the Infants Hospital under the direction of Dr. Eric Pritchard. This course will occupy the afternoons only, and is of interest especially to those engaged in welfare centres. Some visits to other centres will be made, such as to the St. Margaret's Hospital, to the United Dairies Pasteurizing Plant at Willesden, &c. The fee for the course is £3 3.

From December 1 to December 12 an afternoon course in Dermatology will be given at the Hospital for Diseases of the Skin, Blackfriars. Instruction will take place in the Out-Patient Department every afternoon from 2.30 p.m., and special demonstrations of interesting cases will be given on December 2 and December 9. Fee, £1 1s.

We would remind readers that the first special course to be held in 1931 will be one in Cardiology, and as this course is extremely popular and the numbers strictly limited to twenty, early enrolment is desirable. The dates of the course are January 12 to January 24.

For the benefit of our new subscribers we would point out that the 1931 List of Special Courses is available.

M.R.C.P. LECTURE.

The last lecture in the M.R.C.P. Special Course will be given on Tuesday, December 2, at 8.30 p.m., at 10, Bedford Square, W.C.1, by Dr. A. Knyvett Gordon on "The Diagnosis of Diseases of the Blood-forming Organs," illustrated by epidiascope, followed by a laboratory demonstration. Fee, 10s. 6d., payable at lecture room.

LECTURES.

The following lectures will be delivered on Mondays at 4 p.m., at the Medical Society, 11, Chandos Street, Cavendish Square. There is no fee for attendance:—

- December 1.—Mr. W. H. Ogilvie, "Recent Advances in Surgery."
 December 8.—Dr. Philip Figdor, "Recent Advances in Osteo-arthritis."

DEMONSTRATIONS.

The following free clinical demonstrations in medicine and surgery will be given during December:—

- December 4, 2 p.m.—*King's College Hospital*, Denmark Hill. Dr. Terence East (Cardiology).
 December 4, 2 p.m.—*Western Ophthalmic Hospital*, Marylebone Road. Mr. T. C. Summers (O.P.).
 December 8, 2 p.m.—*Children's Clinic*, Cosway Street, Edgware Road. Dr. Bernard Myers, "Modern Methods of Infant Feeding."
 December 9, 2 p.m.—*Royal Northern Hospital*, Holloway Road. Mr. Kenneth Walker (Urological Cases).
 December 11, 2 p.m.—*Metropolitan Hospital (General)*, Kingsland Road, E. Dr. P. Hamill (O.P. Demonstration).

St. John's Hospital for Skin Diseases, Leicester Square, W.C. 2.

Chesterfield Lectures: Tuesdays and Thursdays at 5 p.m. Fee for hospital practice, including lectures, £1 1s. per month. Daily instruction given in O.P. Department. Lectures for December are as follows:—

- December 2.—Dr. A. C. Roxburgh, "Cutaneous Syphilis" (1).