PULMONARY OEDEMA

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Pulmonary oedema is a well-recognized symptom complex which may occur in either chronic or acute form and be precipitated by a variety of different causes. The factors which give rise to oedema in other parts of the body also operate in the lungs, but it must be appreciated with a difference in as much as the alveoli and capillaries of the lungs are directly exposed to the atmosphere and the whole of the circulation of the blood must pass through the pulmonary circuit.

The general factors which influence the production of tissue oedema may be stated as follows :—

- (1) Normally there is a higher hydrostatic pressure at the arterial end of the capillary bed than at the venous end, as a result there is a tendency for fluid transudation into the tissue spaces at the arterial end, and absorption at the venous end, owing to the greater osmotic attractive force in the blood vessels. In consequence any form of increase in venous pressure will tend to cause accumulation of tissue fluid.

As Weiss points out in his admirable review, the hydrostatic pressure in the pulmonary capillaries is low in relation to the osmotic pressure of the blood with the result that there is normally a force of absorption rather than transudation. He quotes the experiment of Colin in 1873, who administered twenty-one litres of water intratracheally to the horse in the course of three-and-a-half hours without ill effects.

- (2) The presence of serum protein in adequate concentration exerts sufficient osmotic pressure to retain and draw

fluid into the vascular system. • Any serious depletion of protein will therefore favour the formation of oedema.

- (3) Bacterial infections and toxins apart from their local effect may give rise to widespread capillary damage with increase in their permeability.
- (4) Chemical poisons and irritants by causing damage to the capillary endothelium will give rise to oedema.
- (5) Nervous reflexes which have been the subject of much experimental work but are not yet clearly understood may play an important part in the production of oedema, particularly of the lungs. An excellent and comprehensive review of the experimental work is given by Luisada.

The intravenous administration of large amounts of fluids to animals results only irregularly in the production of pulmonary oedema, but this is made much more certain after cutting the vagi (Farber). Brown Séquard demonstrated that stimulation of the stellate ganglion can give rise to pulmonary oedema, and many workers have confirmed the fact that the intravenous administration of epinephrine gives rise to pulmonary oedema which can be controlled or abolished by sedatives and narcotics. The intratracheal administration of adrenalin can also produce oedema.

Luisada and Sarnoff have recently produced pulmonary oedema in dogs by rapid intracarotid infusion. They have shown that denervation of the carotid sinus prevents this oedema, and further that it is possible to prevent this type of oedema by hypnotics. They stress the importance of specific recep-

tors of the cardiovascular apparatus and their stimulation which leads to an increase in the permeability of lung capillaries. Luisada feels that up to the present, too much attention has been focused on left ventricular failure in the production of pulmonary oedema.

There seems little doubt that it can occur in the absence of disease of the heart and kidneys. Its neurogenic origin has been described in hypoglycaemic coma by Parkes-Weber and Blum, and in cerebral trauma by Schlesinger amongst others. From the foregoing discussion it will be realized that the interplay of various complex factors, local, vascular and neurogenic, take part in the production of pulmonary oedema.

The conditions in the course of which pulmonary oedema may be found are :—

- (a) Failure in the circulation due to heart disease, primarily of left ventricular origin associated with hypertension, aortic valvular disease, and following coronary thrombosis. It sometimes occurs in mitral stenosis of high degree, but is not usually associated with right ventricular failure unless the whole heart fails. In fact, the lung oedema of left ventricular origin may improve with the onset of rightsided failure.
- (b) It may occur in the course of acute or chronic nephritis and the toxæmia of pregnancy. In acute nephritis its onset is determined by a number of disturbances, chiefly generalized capillary damage and the appearance of congestive failure with elevation of venous and capillary pressure. In chronic nephritis the oedema is usually precipitated by left ventricular failure, lowered protein or cerebral vascular accident.
- (c) In the course of acute infections such as influenza and pneumonia.
- (d) Following infarcts in the pulmonary fields.
- (e) Following trauma of the chest wall and pleura and as a consequence of blast injuries of the lung ; also it may follow too rapid removal of fluid from pleural effusions.
- (f) As a result of exposure to noxious gases and dusts in the course of in-

dustry or warfare, and in the course of anaesthesia and sensitivity to lipidol in bronchography.

- (g) In peripheral circulatory failure in secondary shock and as the result of overadministration of intravenous fluids.
- (h) In diseases which favour the retention of fluid in the tissues such as nephrosis, beri-beri and in famine oedema.
- (i) Oedema of central nervous origin due to disturbance of the respiratory and vasomotor centres, following trauma, haemorrhage, and the local vascular changes and oedema produced in the course of operations for the removal of tumours particularly of the posterior fossa.
- (j) It occurs occasionally in the course of epileptic convulsions and in encephalitis.
- (k) As a complication of angio-neurotic oedema.

Pathological findings

At autopsy the lungs may be pale or dark, depending on the amount of congestion, and will be heavier than normal with pitting oedema. On section with a knife frothy fluid escapes.

Clinical manifestations

Pulmonary oedema may arise suddenly and dramatically in acute form, or may be superimposed on a previously chronically congested lung. In chronic cardiac or renal disease it occurs often in the night with alarming symptoms of dyspnoea, orthopnea, suffocation and anxiety, and with the production of large quantities of bloodstained and frothy mucous from the mouth and nose. On examination the heart sounds are obscured and coarse, the bubbling râles will be heard all over the chest. Hydrothorax may also be present.

All efforts should be directed to preventing the onset of an acute attack, and therefore the recognition of chronic congestion and oedema is important, as in cardiac disease it can be controlled by appropriate treatment. Chronic oedema may be recognized by dyspnoea, irritative cough, and on physical examination by the presence of râles starting at the bases and then later extending upwards. It must be

emphasized, however, that oedema may exist without râles necessarily being heard in the lungs. It can be recognized earlier by radio-scopy when increased hilar and perihilar densities can be seen, fanshaped in distribution and spreading to the bases. In more advanced cases of congestion and oedema the radiological appearances tend to be confusing, with mottling, usually basal but sometimes more generalized and resembling bronchopneumonia or miliary tuberculosis. Early oedema may also be recognized by spirometry when a diminished vital capacity is present.

The common type of left ventricular failure is often characterized by attacks of paroxysmal dyspnoea, unassociated with exertion or excitement, and with the patient in the recumbent position in bed. In these so-called attacks of cardiac asthma râles and rhonci need not necessarily be present. The attacks are precipitated in the recumbent position because the lungs are then more engorged than when the body is in the erect posture. In the upright position there is also more room for ventilation and the diaphragm occupies a lower position. It is also possible that in the recumbent position there is increased venous pressure on the respiratory centre. Cardiac asthma may be distinguished from ordinary asthma by the history, evidence of cardiac enlargement, and in its more acute phases by the fact that in bronchial asthma the sputum is coughed up after and not during an attack.

The treatment of pulmonary oedema is dependent on the operative causes. By far the largest proportion of cases met with in practice are associated with cardiac disease. If seen in the acute phases an immediate injection of morphia gr. $\frac{1}{4}$ is essential. The addition of atropine gr. 1/100 has often been combined, but in view of the fact that the oedema fluid is a transudate rather than an exudate, its efficacy is doubtful. Should signs of venous congestion be present a venesection of at least one half pint is often helpful, and in the more severe cases pure oxygen if available should be administered. An intravenous injection of 0.25 gm. of cardophylin also often relieves the dyspnoea by causing relaxation of bronchi in any spasm which may be present. For the subsequent treatment rest in bed is necessary with a course of ammonium chloride and intra-

venous mercurial diuretics such as neptal to lessen the oedema. Digitalis in the form of powdered leaf is most valuable if there is any evidence of congestion, and should be given irrespective of the presence or absence of irregular cardiac rhythm. As has been previously mentioned the prevention of an acute attack is far better than its subsequent alleviation; therefore any patient who gives evidence of lung congestion by increasing dyspnoea, orthopnea, the presence of râles at the lung bases and attacks of paroxysmal nocturnal dyspnoea should be put to bed for some days and given mercurial diuretics and digitalis. In the course of acute or subacute nephritis the restriction of fluid and sodium will help to diminish the chances of oedema. Saline purgation is also helpful in eliminating fluid. Penicillin should be given for any (usually streptococcal) infection still active. Should acute oedema occur, venesection with the administration of morphine and oxygen is indicated. During the course of chronic nephritis the onset of acute pulmonary oedema is often terminal and again venesection, morphia and oxygen are treatments of choice. In all forms of nephritis mercurial diuretics are contraindicated. In nephrosis a high protein diet is rational and should oedema occur intravenous protein in the form of plasma can be given, provided no evidence of heart failure is present. The oedema of wet beri-beri is believed to be largely the result of right heart failure, but lung oedema may occur also with left heart failure. The patient should be treated with vitamin B, by injection, and also by mouth. If there is evidence or deficiency in plasma protein this may be administered after the heart failure has been dealt with. Replacement of serum protein is of course essential in the treatment of famine oedema.

Fortunately the last war produced no cases of gas casualties, but in the 1914 war they were all too frequently seen as a result of exposure to chlorine, phosgene, chloropicrin, and the fumes of mustard gas. The patients were often unaware of their exposure, particularly to phosgene, and the onset of pulmonary oedema was delayed. One of the pointers to such exposure was the victim's reluctance to smoke a cigarette. In peacetime practice oedema following gassing is likely to be met in industry by

exposure to the fumes of nitric acid, nitrous dioxide, hydrogen sulphide, and as was seen following the Coconut Grove fire in Boston, inhalation of fumes.

In all these cases absolute rest with morphine and the administration of oxygen with humidification under pressure if possible is indicated. If shock is present intravenous plasma or serum may be given, but not more than 2,000 c.c. Oedema may occur during the administration of anaesthetics such as ether and N_2O . When this happens the table may be tilted to help drainage from the lungs, and pharyngeal or intratracheal intubation carried out with pure oxygen given under pressure. Atropine gr. 1/100 must also be given intravenously. In cases due to cerebral oedema intravenous hypertonic solutions such as 50 per cent. sucrose are of benefit together with pharyngeal and intratracheal intubation with suction when necessary. The oedema following rapid paracentesis is now uncommon, presumably due to improved technique.

In past pandemics of influenza rapidly occurring oedema was often seen, but with the present attacks it is uncommon. It should be treated when it occurs with humidified pure oxygen.

In conclusion it is hoped that the foregoing summary will have shown the variety of conditions in which pulmonary oedema may arise. The exact vagal and sympathetic distribution to the lungs is not yet quite clear, and the importance of the interplay of vascular and central nervous reflexes remains yet to be elucidated. It is possible that the stretching caused by capillary engorgement, initiates reflexes giving rise to increase of permeability.

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PENICILLIN IN GENERAL PRACTICE

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Introductory

This lecture purports to be no more than an elementary summary of what is generally known about penicillin at the present moment, and it is as well to state at the outset that a good deal of that knowledge is still of a tentative nature. In treatment particularly, the establishment of a remedy and its method of use depends not upon the reported cure of a few, but of a statistically significant number of

cases—a criterion which has not yet been fulfilled in the case of many diseases now coming under penicillin treatment.

You will find, therefore, that you are in some sense thrust into the position of experimenting upon your patients, that cut and dried procedures are not yet wholly the guide but are going to be partly the result of your efforts. Ordinary streptococcal tonsillitis may serve as an instance; does it always, as some say, or