PULMONARY ŒDEMA

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ŒDEMA of the lungs was defined by Lænnec (1829) as . . . "the infiltration of serum into the substance of this organ, in such degree as evidently to diminish its permeability to the air, in respiration." Lænnec also observed that pulmonary Œdema was most commonly caused by disease of the heart, and could manifest itself as "suffocative orthopnoea". James Hope (1832) noted that obstruction of the left heart would lead to pulmonary congestion associated with severe dyspnoea, and wrote that . . . "asthma has been too much regarded as independent of the heart. Long treatises have even been written upon it without ever mentioning disease of this organ as one of its causes. It is, therefore, necessary to dwell a little on this subject, not only for showing the magnitude of this error, but of making the reader acquainted with all the habitudes and aspects of a complaint, which is perhaps the most distressing in the whole catalogue of human maladies".

Causes of Pulmonary Œdema

Pulmonary Œdema may be due to multiple causes, and extensive aetiological classifications have been given by Cameron (1948), Luisada and Cardi (1956) and Visscher, Haddy and Stephens (1956). The more common causes are listed in Table 1.

Aetiological Considerations

In the ultimate analysis pulmonary Œdema must be due to one or more of the following factors; a rise in pulmonary capillary pressure, a fall in intravascular osmotic pressure, an increase in capillary permeability, or a fall in tissue pressure.

Left Ventricular Failure:

Failure of the left ventricle is the most common cause of pulmonary Œdema, and may be acute or chronic. Left ventricular failure leads to damming back of blood in the lungs,

TABLE I. THE CAUSES OF PULMONARY ŒDEMA	
A.	<i>Heart Disease:</i>
	1. Hypertension.
	2. Aortic and Mitral Valve disease.
	3. Myocardial disease.
	4. Pulmonary embolism.
B.	<i>Central Nervous System:</i>
	1. Trauma.
	2. Hæmorrhage and Thrombosis.
	3. Encephalitis.
C.	<i>Respiratory System:</i>
	1. Pneumonia—(especially influenzal).
	2. Drowning and asphyxia.
	3. Inhalation of irritant gases.
	4. Chest trauma. (Traumatic wet lung).
D.	<i>Allergy:</i>
	1. Angioneurotic Œdema.
	2. Serum sickness.
E.	<i>Miscellaneous Causes:</i>
	1. Distension of œsophagus, stomach, gall bladder.
	2. Excessive transfusion.
	3. Overhydration in acute renal failure.
	4. Manipulation of stellate ganglion.
	5. Overdosage with morphia, heroin, methyl salicylate.
	6. High altitude pulmonary Œdema.

and the resulting pulmonary congestion renders the lungs turgid and less distensible, and the resultant difficulty in expanding and retracting the stiffened lung is responsible for the primary symptom of dyspnoea. If the pulmonary capillary pressure rises still further and exceeds the osmotic pressure, then true pulmonary Œdema occurs. Congestion of the bronchial mucosa and secondary bronchospasm may lead to airway obstruction and thus further contribute to the sensation of dyspnoea.

The classical relationship between cardiac dyspnoea and posture (orthopnoea) is probably due to increased right ventricular output consequent on increased right atrial filling in the horizontal position (McMichael, 1937); and it has been estimated that the lungs contain an extra 500 ml. of blood in the recumbent posture (McMichael, 1939). The decrease in vital

FIG. 1.—The vicious circle operating in left ventricular failure whereby increasing pulmonary oedema progressively impairs myocardial function, thereby leading to further degrees of pulmonary oedema.

This chain reaction is potentially self perpetuating and unless broken at some point may prove fatal.

capacity due to upward displacement of the diaphragm is a further factor leading to increased pulmonary congestion in the horizontal position.

Paroxysmal nocturnal dyspnoea (cardiac asthma, acute left ventricular failure) usually occurs in patients with chronically congested lungs, and is due to the sudden trapping of an extra few hundred millilitres of blood in the already congested pulmonary circulation. These attacks usually occur at night and various explanations have been offered to account for this phenomenon. The failing heart is more able to meet demands during the night when the requirements are reduced, and thus balance is temporarily restored. This allows the resorption of postural oedema which has collected in the dependent parts of the body during the day, leading to a nocturnal increase in blood volume, and the chronically raised pulmonary venous pressure tends to redistribute this oedema fluid in the lungs. The assumption of the horizontal position during sleep also tends to increase pulmonary congestion by increasing right ventricular filling pressure and decreasing vital capacity. Compensatory mechanisms are delayed because of sleep, and thus the patient does not wake and sit up until pulmonary congestion is well advanced. During sleep less fluid is lost by evaporation from the lung, thus potentiating the oedematous state. Recent experimental studies in rabbits in whom cardiac failure has been induced by artificial A-V shunts (Uhley, Leeds,

Sampson and Friedman 1962) have demonstrated very large increases in lymph flow from the lung, and it has therefore been suggested that reduced lung motion at night decreases pulmonary lymph flow and hence predisposes to pulmonary oedema.

Once the attack has started a vicious circle develops (Fig. 1), whereby increasing degrees of pulmonary oedema further reduce myocardial function. There are several mechanisms by means of which pulmonary oedema further embarrasses the left ventricle. Thus the severe dyspnoea leads to intense anxiety, and the resultant tachycardia and diminished coronary filling time place severe demands on the failing ventricle. The intense inspiratory efforts tend to suck more blood into the pulmonary circulation thus tending to further oedema formation. Pulmonary oedema may produce a severe ventilation defect due to obstruction of the airways by frothy fluid, and the resulting anoxia and metabolic acidosis further impairs myocardial function leading to further degrees of failure and progressively severe pulmonary oedema. This chain mechanism is potentially self perpetuating and unless broken at some point the sequence of increasing pulmonary oedema and diminishing myocardial function may prove fatal.

Mitral Valve Obstruction:

Obstruction at the mitral valve is usually due to a tight mitral stenosis, but on rare occasions ball valve thrombi and atrial myxomata may be present. Severe obstruction at the mitral

valve leads to pulmonary congestion with a raised pulmonary capillary pressure and subsequent pulmonary oedema, which can be acute or chronic.

Acute pulmonary oedema in mitral stenosis, unlike that in left ventricular failure, usually occurs during exercise. The resulting tachycardia decreases the diastolic filling period, thus leading to a hold-up of blood in the left atrium. Tachycardia associated with acute infections and the onset of an abnormal rhythm such as auricular fibrillation may similarly precipitate an attack of acute pulmonary oedema. It is also well known that a chest infection may act as a precipitating factor by increasing capillary permeability, and the increased blood volume associated with pregnancy may also precipitate an episode of pulmonary oedema.

Other Causes:

Irritant gases such as phosgene produce lung oedema by a direct action on the pulmonary capillaries, and increased capillary permeability is responsible for pulmonary oedema associated with serum sickness and acute angioneurotic oedema. Acute renal failure produces oedema by excessive fluid intake in the face of anuria or severe oliguria, with resultant circulatory overload. Excessive transfusion may have similar effects in elderly subjects with poor renal and cardiac reserves.

Pulmonary oedema may occur in association with intracranial disease of many kinds, and it is of historical interest that asthma was linked with the central nervous system long before the relationship to heart failure was suspected. Thomas Willis (1679) divided asthma into two groups, one associated with bronchial obstruction, and the other he called convulsive asthma, in which the lungs were normal and the cause of the disorder lay in the nerves controlling respiration. Thus Willis stated that he had . . . "clearly unfolded by anatomical observations that the cause of a convulsive asthma sometimes consists in the hinder part of the head near the origin of the nerves." Morgagni (1769) in the famous *De Sedibus et Causis Morborum* (The Seats and Causes of Disease) describes the relationship between head injury and subsequent pulmonary oedema. It is now suggested that any acute cerebral disturbance may lead to massive stimulation of the sympathetic system with a resultant shift of blood from areas of high constrictor potential in the systemic circulation to the pulmonary circulation which is of low con-

strictor potential (Sarnoff, 1952). The resultant overloading of the pulmonary circulation predisposes to pulmonary oedema. The peripheral shut down in the systemic circulation impairs left ventricular output and thus strains the left ventricle, and there is evidence that neurogenic pulmonary oedema usually occurs in association with hypertension and coronary artery disease which act as predisposing factors (Paine, Smith & Howard, 1952). The extreme bradycardia which sometimes occurs in severe intracranial damage may also predispose to pulmonary oedema (Campbell, Haddy and Visscher, 1949).

Acute pulmonary oedema can occur in heroin poisoning, particularly in "main-line" addicts. (Silber and Clerkin, 1959). The pathogenesis is not clear, but hypoxia due to respiratory depression may lead to increased capillary permeability (Drinker, 1945). Morphia is also known to increase intracranial pressure (Keats and Mithofer, 1955) and may stimulate the vasomotor centres (Schmidt and Livingstone, 1933) and thus the oedema might be partly neurogenic in origin. Pulmonary oedema in salt water drowning is due to an imbalance of hydrostatic and osmotic forces in the pulmonary capillary bed set up by the ingestion of salt water into the alveoli; hypoxia, damage to the capillaries and neurogenic factors probably also contribute (Lancet 1962). Pulmonary oedema may accompany lung infections, particularly influenzal pneumonia and is probably due to the local release of vaso-dilator substances as part of the inflammatory reaction. The mechanism of high altitude pulmonary oedema is uncertain (Hultgren, Spickard and Lopez, 1962).

Clinical Picture

Cardiac Asthma. (Acute left ventricular failure).

The clinical picture of acute pulmonary oedema is similar whatever the underlying cause. In routine clinical practice however acute pulmonary oedema is usually due to left ventricular failure.

The clinical picture was well described in 1832 by James Hope (Fig. 2) who in discussing asthma noted that cardiac asthma comprised . . . "by far the greatest number of the most severe and fatal cases of the disease." Hence the term cardiac asthma derives historically from the fact that hitherto severe paroxysmal dyspnoea arising from the heart had not been differentiated from other forms of asthma.

Hope's description of cardiac asthma is s-

FIG 2.—James Hope (1801-1841). (Courtesy of the Wellcome Historical Medical Museum). Born in Stockport, Cheshire, and became a Physician to St. George's Hospital. Attributed his success to the advice given him by his father: 1. Never keep a patient ill longer than you can help. 2. Never take a fee to which you do not feel yourself entitled. 3. Always pray for your patients.

detailed and well written that it could hardly be bettered today . . . "The fit is, on the whole, more apt to supervene during the evening or early part of the night. From a slumber fraught with the horrors of a hideous dream he starts up with a wild exclamation . . . sitting with the trunk bent forwards and the elbows or forearms resting on the drawn-up knees. The latter position he assumes when attacked by a paroxysm of dyspnoea, sometimes however extending the arms against the bed on either side, to afford a firmer fulcrum for the muscles of respiration. With eyes widely expanded and starting, eyebrows raised, nostrils dilated, and the head thrown back at every respiration, ghastly and haggard he casts around a hurried, distracted look of horror, of anguish, and of supplication; now imploring, in plaintive moans, quick, broken accents and stifled voice, the assistance already lavished in vain, now upbraiding the impotency of medicine; and now, in an agony of despair, dropping his head on

his chest and muttering a fervent invocation for death to put a period to his sufferings." Hope also notes that the respiration might be . . . "wheezing and performed with violent efforts of all the muscles of respiration both ordinary and auxiliary. The inspirations are high and accomplished with apparently little descent of the diaphragm, and the expirations are short and imperfect. The surface is chilly, the extremities are cold, and the face is pale and sometimes, livid." He goes on to say that often . . . "There is violent cough and in suffocative paroxysms, accompanied, at first, with scanty expectoration of viscid mucus, but ending in a copious and free discharge of thin, transparent, frothy fluid, occasionally intermixed with blood. This evacuation by disgorging the pulmonary capillaries, affords great relief to the cough and dyspnoea."

Apart from noting that the patient will often go to the window in an attempt to get fresh air, and that he is usually soaked in perspiration, there is little to add to Hope's clinical description. There is a tachycardia and there may be a pulsus alternans. The blood pressure may be either raised or lowered. Occasionally an arrhythmia is present, and a gallop rhythm is usually audible. Pulmonary signs depend on the site of the oedema; thus if it is pericapillary little abnormal will be heard, but if it is intra-alveolar then crepitations and fine rales will be audible throughout the lung fields. Oedema of the bronchial walls together with excessive bronchial secretions may lead to secondary bronchospasm. In some patients bronchospasm may appear to dominate the clinical picture, and this is said to occur mainly in those with pre-existing bronchitis or asthma.

Investigations are rarely carried out in acute pulmonary oedema. The chest X-ray will show fleecy mottling spreading from the hila (Figs. 3 and 4). The electrocardiogram may show an arrhythmia or evidence of myocardial damage, and catheter studies have demonstrated a markedly raised pulmonary artery pressure.

Subacute Pulmonary Oedema

The dividing line between pulmonary congestion (pulmonary venous hypertension) and subacute pulmonary oedema is not clear, and it is likely that dyspnoea in chronic heart failure is partly due to pulmonary oedema.

Several factors tend to prevent the development of oedema in the chronically congested lung. Recent studies on artificially induced failure in animals (using A-V shunts) have

FIG. 3.—(a) Pulmonary œdema associated with acute nephritis.
(b) Resolution of œdema 10 days later.

demonstrated greatly increased lymph drainage from the lung (Uhley and others, 1962). It has therefore been suggested that chronic excessive ultrafiltration leads to an increased run-off capacity of the lymphatics, with resultant protection from overt œdema. That this mechanism is operative in man is well demon-

FIG. 4.—(a) Acute pulmonary œdema caused by left ventricular failure and coronary artery disease in a patient with chronic bronchitis.
(b) Resolution of œdema 48 hours later. Note presence of fibrosis and emphysema together with an old tuberculous lesion at the right apex.

FIG. 5.—Long standing pulmonary venous hypertension with basal septal lines and a fibrosed interlobar fissure in a patient with mitral stenosis (By courtesy of Dr. C. M. Ogilvie).

strated by the engorged basal pulmonary lymphatics or septal lines, so commonly seen in mitral stenosis (Fig. 5). Further protective mechanisms such as thickening of the capillary walls and pulmonary hypertension, probably contribute to the long survival in mitral stenosis.

The clinical picture requires no elaboration. There is dyspnoea which is at first exertional, but which gradually progresses until present at rest. The dyspnoea due to pulmonary congestion is related to posture (orthopnoea). The sudden development of "bronchitis" or a nocturnal cough in the elderly should always raise the suspicion of impending left ventricular failure. Bronchitic symptoms such as a chronic cough associated with a little sputum and wheezing, may occur in subacute pulmonary oedema, and are particularly common in patients with mitral stenosis.

Physical examination may reveal evidence of the underlying cause, such as aortic or mitral valve disease, and there may be a history of anginal pain, and appropriate electrocardiographic evidence of underlying coronary artery disease. Basal crepitations or small basal effusions may be detected; occasionally rales

and bronchospasm dominate the picture. The heart is often enlarged, pulsus alternans may be noted, and a triple rhythm is often heard. The pulmonary circulation time is increased, and there is a reduction in all fractions of the lung volume except the residual air. Catheter studies will show a raised end-diastolic pressure in the left ventricle, and a raised left atrial pressure with a steep Y descent. A large "a" wave may be seen on the apex cardiogram (Coulshed and Epstein, 1963).

Much recent work has shown that subacute pulmonary oedema and pulmonary venous hypertension may first be demonstrated radiologically. The radiological signs have been well documented and include enlarged hilar shadows with fuzzy outlines, enlargement of the upper lobe pulmonary veins, small bilateral pleural effusions and some perihilar shadowing (Simon, 1961). Subacute pulmonary oedema is common in mitral stenosis, and engorged basal pulmonary lymphatics (Kerley's lines) are well seen, but also occur to a lesser extent in other forms of pulmonary oedema. Haemosiderosis may be seen in long standing cases and occasionally intrapulmonary ossific nodules may develop (Galloway, Epstein and Coulshed, 1961).

Treatment of Acute Pulmonary Oedema

1. *Posture*: Placing the patient in a sitting position will reduce intrapulmonary blood volume by increasing the vital capacity and by lowering right ventricular filling pressure. The conscious patient will naturally assume the most comfortable position, but the importance of posture should not be forgotten in the confused or semi-conscious patient. This simple procedure will often abort nocturnal episodes of cardiac asthma without recourse to other methods.

2. *Morphia*: is undoubtedly the most valuable drug in the treatment of acute pulmonary oedema, and a $\frac{1}{4}$ to $\frac{1}{2}$ grain (15-20 mg.) should be given intramuscularly or a $\frac{1}{2}$ grain (10 mg.) intravenously. Its life saving action rests on its ability to break the vicious circle (Fig. 1) which operates in acute pulmonary oedema. The intense dyspnoea produces great distress and anxiety, leading to a rapid tachycardia which further increases the work of the already failing left ventricle, thus leading to further decompensation and pulmonary oedema, and hence to even greater dyspnoea. As the pulmonary oedema increases oxygen exchange is impaired and severe hypoxia may develop which

further reduces myocardial efficiency, and may lead to loss of vascular tone and the development of profound circulatory collapse. The principle site of action of morphia is on the higher cortical centres, lowering the level of consciousness and thus relieving the intense anxiety which perpetuates this vicious circle. The direct depressant action of morphia on the respiratory centre is probably of secondary importance.

3. *Aminophylline*: This drug is commonly used in this country, and is given intravenously in a dose of 0.25-0.5 gram. Its principle action is the relief of airway obstruction due to expiratory bronchospasm. It also lowers venous pressure, acts as a respiratory stimulant, and may be a myocardial stimulant. The value of aminophylline in acute pulmonary oedema has however been questioned (Luisada and Cardi, 1956), and it should never be used in preference to morphia. In certain cases it can be difficult to distinguish between cardiac and bronchial asthma, especially in subjects with a history of chronic bronchitis or asthma; and in these circumstances morphia could be dangerous and a trial of aminophylline is justified.

4. *Digitalis*: It is well known that digitalis strengthens the myocardial muscle in the heart-lung preparation, and recent studies using strain gauges applied to the exposed human heart during extra-corporeal by-pass procedures have fully confirmed this finding (Braunwald, Bloodwell, Goldberg and Morrow, 1961). Intravenous use of the short acting glycosides such as strophanthin (1 mg. of ouabain) is usually recommended, but it is doubtful whether they have any advantage over intravenously administered digoxin (1 mg.). Care should be taken if digoxin has been given previously, and smaller doses used. Digoxin is absolutely contraindicated in acute pulmonary oedema due to mitral stenosis, as it will increase right ventricular output and further increase the pulmonary oedema. It has furthermore been suggested that digoxin may always have a greater effect on a healthy ventricle, and it may therefore increase right ventricular output to a greater extent than that of the failing left ventricle, thus exaggerating the degree of pulmonary oedema. Thus some authorities would not recommend intravenous digoxin in the treatment of acute pulmonary oedema due to left ventricular failure.

5. *Venesection*: If other methods fail venesection may be life saving in otherwise intract-

able left ventricular failure. It acts by rapidly reducing intrapulmonary blood volume. About one pint of blood should be taken from an arm vein, but in exceptional circumstances intra-arterial puncture of the femoral artery can be employed. Medical venesection using tourniquets applied to the limbs will have the same effect.

6. *Oxygen*: Severe pulmonary oedema may produce arterial hypoxia due primarily to airway obstruction caused by fluid. The resulting anoxia further reduces myocardial efficiency and the administration of oxygen is of value.

7. *Atropine*: This drug has been recommended to reduce bronchial secretions, but it is of doubtful value in pulmonary oedema. It has been used in neurogenic pulmonary oedema associated with extreme bradycardia of vagal origin.

8. *The Management of Abnormal Rhythms*: In cases which do not respond to treatment fairly rapidly it is always worthwhile doing an electrocardiogram, as an abnormal rhythm such as ventricular tachycardia or uncontrolled auricular fibrillation may be detected. The abnormal rhythm may have precipitated the attack of pulmonary oedema, or it may have started during the attack. Reversion to sinus rhythm by suitable methods, such as intravenous procaine amide (under ECG control) in ventricular tachycardia, will often terminate the attack of pulmonary oedema. The sudden onset of a rapid auricular fibrillation often precipitates acute pulmonary oedema in patients with mitral stenosis, and the use of digitalis in this situation is justified. Complete heart block may precipitate pulmonary oedema following a myocardial infarct, and the use of steroids may restore sinus rhythm, possibly by reducing oedema and hence hypoxia in the region of the A-V node (Printzmetal and Kemmamer, 1954). In these circumstances the sodium-retaining action of steroids should be counteracted by the concomitant use of a diuretic.

9. *Intermittent Positive Pressure Breathing* (I.P.P.B./I-O₂). This technique utilising oxygen and inspiratory pressures up to 45 cm. of water, has been used by Miller and Sproule (1959) in over 350 cases, and they describe their results as usually remarkable and often dramatic. In the apparatus described, 100% oxygen was used and the inspiratory pressure could be readily adjusted. This technique should be used only when simple measures have failed.

It is now well recognised that the anoxic disturbance in acute pulmonary oedema can be profound, and arterial oxygen saturations

as low as 30% have been recorded. The anoxia is not due to a diffusion defect, but is a distribution defect, and is largely due to the frothy fluid which obstructs the airways. Physiological studies have shown that I.P.P.B./I-O₂ rapidly corrects the arterial oxygen saturation, and this in turn will improve cardiac function and increase vascular tone.

It has previously been pointed out that the dyspnoea in acute pulmonary oedema is primarily due to the increased work of breathing resulting from the increased airway resistance due to frothy oedema fluid, and the diminished compliance. The latter factor is due to a combination of interstitial congestion, increased pulmonary vascular pressure, and increased pulmonary blood volume.

I.P.P.B./I-O₂ may be of value in several ways. I.P.P.B./I-O₂ significantly decreases the work of respiration and so relieves the sensation of dyspnoea. The improved ventilation relieves hypoxia and also increases lymphatic flow, thus helping to reduce the oedema. The positive pressure during inspiration increases the intra-alveolar pressure, and so decreases the trans-capillary pressure gradient, and hence the tendency towards oedema formation. The relief of dyspnoea will tend to reduce the increased depth of respiration during extreme voluntary effort, and will therefore lower the cardiac in-flow load and hence reduce right ventricular output.

Acute pulmonary oedema can never exist in a "steady state", and must therefore always be progressive, increasing pulmonary oedema throwing an extra load on the heart and leading to further ventricular decompensation. I.P.P.B./I-O₂ breaks this vicious circle of progressive decompensation at many points, and may therefore be life-saving.

10. *Antifoaming Agents*: Oedema fluid in the alveoli and smaller bronchi is not of great significance until the surface tension of the fluid reaches a critical point and foaming occurs. This leads to an enormous increase in volume and rapidly gives rise to a ventilation defect and hypoxia, which reduces myocardial efficiency and produces further transudation and foaming. This vicious circle can be broken by the use of antifoaming agents (Luisada and Cardi, 1956). Ethyl alcohol is an efficient antifoaming agent, and oxygen bubbling through a 95% alcohol solution is administered via a nasal catheter, and can also be given by I.P.P.B./I-O₂. The flow rate is gradually increased to 10 litres a minute. In order to avoid excessive absorp-

tion of alcohol, inhalation should be discontinued for 15 minutes per hour.

11. *The Artificial Kidney*: Dialysis offers a rational method of rapidly removing excess fluid from the body, and thus reducing pulmonary congestion. Pulmonary oedema due to circulatory overload is most commonly seen in patients with anuria when the fluid intake has not been satisfactorily regulated, but can also occur in elderly subjects who have been over-transfused. Ultrafiltration with the artificial kidney can be achieved by raising the pressure in the venous circuit, and lowering the dialysing fluid pressure by limiting its flow. The successful use of this technique in pulmonary oedema associated with acute renal failure has recently been described (Logan, Williams, Edwards and Watson, 1964). It should of course be emphasized that rigid restriction of fluid intake (to about 400 ml. daily) will usually prevent, or control, pulmonary oedema associated with anuria or severe oliguria. Exchange transfusion can also be carried out during dialysis by the replacement of whole blood with packed cells. This procedure effectively reduces plasma volume and there is no risk of inducing hyperkalaemia during dialysis.

12. *Diuretics*: There is no time for diuretics to act in acute pulmonary oedema, but they are of great value in the management of sub-acute pulmonary oedema. With the plethora of diuretics now available, resistant oedema is not commonly seen in medical wards. The commonest cause of failure to respond to diuretic therapy is hypokalaemia, and severe hypokalaemia may be present with a normal serum potassium level. The diagnosis of potassium depletion is best made by a high index of clinical suspicion of the circumstances, such as prolonged diuretic therapy, in which potassium loss may have arisen. The clinical picture of anorexia, apathy, mental confusion, unusual muscle weakness and abdominal distension, together with oliguria and a moderately raised blood urea should suggest hypokalaemia; and the electrocardiographic picture of S.T. depression, T wave inversion, and "U" waves in the left praecordial leads should support the diagnosis. Hypokalaemia is best corrected with oral potassium. Diuretic resistance also occurs with low chloride levels, and ammonium chloride 8 grammes daily in divided doses for a few days is worth trying. Sodium restriction is a valuable adjunct to diuretic therapy, and severe restriction using a rice diet for a few days will often improve resistant oedema as-

sociated with pulmonary congestion and otherwise intractable dyspnoea. In terminal cases which have proved resistant to all diuretics great relief from dyspnoea may be afforded by simple mechanical measures such as the use of a cardiac bed, pleural aspiration and acupuncture.

13. *The Underlying Cause:* It is rarely possible to treat the underlying cause during an attack of pulmonary oedema, and one has to rely on general measures to reduce intrapulmonary blood volume.

The reversion of abnormal rhythms is an exception to this rule, and occasionally in resistant pulmonary oedema due to hypertension it is justifiable to lower the blood pressure; subcutaneous ansolysin in small doses is a powerful hypotensive agent, but must be used with caution. Intractable pulmonary oedema occasionally occurs in mitral stenosis, especially during pregnancy, and emergency mitral valvotomy has been used in these circumstances, but carries a high mortality. Infection is known to increase capillary permeability and may precipitate acute pulmonary oedema, and in this situation antibiotic therapy is indicated. In acute allergy where the pulmonary oedema is due to increased capillary permeability, cortisone and antihistamines are valuable.

Preventive Measures

The most common cause of acute pulmonary oedema is left ventricular failure, and it is now generally accepted that this is usually due to the sudden trapping of an extra few hundred millilitres of blood in a previously congested pulmonary circulation. In other words acute pulmonary oedema is usually a complication of subacute pulmonary oedema and rarely arises in a previously normal lung. The vital importance of this point is not always appreciated, but it does suggest that the early recognition and treatment of subacute pulmonary oedema should prevent many cases of cardiac asthma.

Diuretic therapy supplemented by salt restriction is of great value in controlling subacute pulmonary oedema, and maintenance therapy with digoxin will improve myocardial function. Patients with mild heart failure should avoid excessive exercise, and the amount of rest required should be estimated for each individual patient. With modern diuretic therapy the tendency has been to allow more activity, and it should be remembered that the present trend in cardiology is to avoid prolonged immobility because of the twin dangers of pulmonary

emboli, and introspection leading to mental depression.

It should be remembered that rest includes mental rest, and reassurance and nocturnal sedation are therefore of great importance. Fear of the night is very common in patients with orthopnoea, and the provision of a good night's sleep can lead to considerable clinical improvement. If the simpler barbiturates do not prove satisfactory, then more powerful sedatives of the morphia group are justified.

Attention to the general health is important as the correction of associated conditions will often greatly improve myocardial function. Thus anaemia, obesity and minor grades of thyrotoxicosis should be looked for, and corrected if necessary. Infections increase capillary permeability in patients with mitral stenosis. It is therefore mandatory to treat infections promptly and vigorously, and there is a case for prophylactic influenza vaccination in patients with mitral stenosis. Women with valvular disease should be advised as to their ability to undertake a pregnancy. If pregnancy is thought to incur a significant risk, then operative intervention or some form of birth control should be considered.

Treatment of the underlying cause is occasionally possible in heart disease, and would include the use of hypotensive agents, and the correction of valve lesions by valvotomy and valve replacement.

Summary

1. The various causes of pulmonary oedema are reviewed.
2. It is stressed that acute pulmonary oedema cannot exist in a "steady state", but is usually progressive by means of a vicious circle mechanism.
3. Morphia is the most valuable drug in the treatment of acute pulmonary oedema, but numerous other measures are currently available.
4. Acute pulmonary oedema in routine clinical practice is usually due to left ventricular failure. It is emphasized that acute attacks of pulmonary oedema (cardiac asthma) usually occur in a previously congested lung, and hence the recognition and treatment of subacute pulmonary oedema may prevent their occurrence.

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