Today's Treatment

Diseases of the cardiovascular system

Cardiac failure

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Although the prognosis of congestive cardiac failure remains serious, with an outlook as gloomy as that for many malignancies, there is no doubt that modern treatment, particularly the more potent diuretic drugs, has greatly improved the situation for many patients, who are kept alive and relatively comfortable on the "diuretic life" in a way which would not have been possible 20 years ago.

Heart rate

The first question to consider in assessing a patient with congestive heart failure (using this term to include both pulmonary and systemic venous congestion) is: Is the heart rate appropriate? Either undue bradycardia or tachycardia may be a problem.

BRADYCARDIA

Congestive heart failure in complete heart block may be promptly relieved by pacing, particularly in the elderly patient with heart block due to isolated disease of the conducting system, as the working myocardium is generally normal and effective control of the heart rate will promptly relieve dyspnoea and oedema, restoring the patient to normal activities by control of the heart rate alone. Although Adams-Stokes attacks are the usual indication for pacing in complete heart block, congestive cardiac failure must not be overlooked as a complication of complete heart block which is potentially remediable with modern treatment.

TACHYCARDIA

Undue tachycardia is particularly seen as a factor producing congestive heart failure at the onset of atrial fibrillation. In a patient with a normal conducting system atrial fibrillation will produce a rapid and irregular ventricular rate. Patients with mitral stenosis, one of the prime causes of atrial fibrillation, are particularly sensitive to the effects of tachycardia, as the shortening of diastole further impairs left ventricular filling which is obstructed by the mitral stenosis. A similar effect may be seen if ventricular filling is impaired by myocardial disease. The duration of diastole is particularly affected by tachycardia as the time taken by each systole is reduced relatively little as the heart rate increases.

Pulmonary oedema at the onset of atrial fibrillation is characteristic of patients with severe mitral stenosis, and optimally mitral valvotomy should be carried out before such a critical complication can occur. Control of the ventricular rate in atrial fibrillation and other atrial tachyarrhythmias is the prime indication for digitalis treatment, but the onset of digitalis action is gradual even after intravenous administration and a prompt response may be obtained by using an intravenous beta-adrenergic-blocking drug, particularly if there is no serious myocardial disturbance which might be adversely affected. Practolol, up to 10 mg intravenously, has the advantage of familiarity, and a single intravenous dose is not associated with the risks of eye changes and other complications found with long-term oral treatment. Prompt control of the ventricular rate in this way may be followed by long-term control with digitalis treatment. In atrial flutter near toxic doses of digitalis are needed to secure the 4:1 ventricular response associated with a satisfactory ventricular rate. A frequent experience is that 4:1 flutter at rest changes promptly to 2:1 flutter with troublesome tachycardia on exercise. This effect is due to the improvement in atrioventricular conduction produced by sympathetic stimulation on exercise. Advantage can be taken of the synergistic effect of digitalis and beta-adrenergic-blocking drugs on atrioventricular conduction to combine a modest dose of digitalis with beta-adrenergic blockade to eliminate the effect of sympathetic stimulation on exercise. In this way satisfactory control of the heart rate may be obtained without producing toxic effects from either group of drugs. Similar principles may be applied to controlling the ventricular rate in the occasional patient with atrial fibrillation showing an undue tachycardia on exercise, but care must be taken that the adverse effect of beta-adrenergic blockade on myocardial function does not produce a deterioration in congestive heart failure in patients with myocardial disease. The improvement in congestive heart failure recently reported in patients with myocardial disease following beta-adrenergic blockade¹ may be related to control of undue tachycardia in this particular group of patients with myocardial disease after acute myocarditis, but the problems of this treatment are so great that it should not be embarked on without expert advice.

Conversely, in other cases tachycardia may be part of the compensatory mechanism. The shortened diastole of tachycardia helps reduce the haemodynamic disturbance in severe aortic incompetence, and tachycardia is a major feature of the gross sudden aortic incompetence seen with destruction or perforation of the aortic valve in infective endocarditis. Under these circumstances too rigorous control of the tachycardia may have an adverse effect on the cardiac output.

Digitalis treatment

Although digitalis treatment has long been regarded as mandatory in congestive heart failure, considerable doubt has been expressed recently about its long-term beneficial effect in congestive heart failure. Objective studies have failed to show sustained improvement² and it may be that the diseased myocardium has a limited response and that any improvement in contractility produced by digitalis treatment leads to a reduction in myocardial drive by other compensatory mechanisms such as sympathetic nerve stimulation or the

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Frank-Starling effect. The prime indication for digitalis treatment remains the control of the ventricular rate in atrial fibrillation and in this role it is effective and convenient.

Probably much digitalis toxicity has arisen from the inappropriate search for a beneficial effect of digitalis on myocardial contractility in patients with congestive heart failure in normal sinus rhythm. In atrial fibrillation the control of the ventricular rate provides a useful and convenient index of digitalis effect. The ventricular rate should be measured, as in the early stages of treatment the rapid and irregular ventricular rate may not be transmitted consistently to the peripheral pulse. With increasing atrioventricular block during digitalis treatment the ventricular rate becomes slower and less irregular and the difference between the ventricular and peripheral pulse rate is reduced. Nevertheless, ventricular ectopic beats produced as a toxic effect of digitalis treatment may not be transmitted to the peripheral pulse, and alternating ventricular ectopic beats may produce an apparent halving of the effective heart rate. The useful rule that a dose should be omitted if the heart rate falls below 60/min has saved many patients from digitalis poisoning. Digitalis overdosage is to be suspected if the heart rate becomes completely regular, a slow regular rate suggesting complete heart block with a junctional (atrioventricular nodal) escape rhythm, a more rapid regular rate the onset of atrial tachycardia with a regular, say 2:1, ventricular response. In patients with sinus rhythm the useful changes in heart rate found with digitalis treatment in atrial fibrillation are not evident; the tendency to slowing expected on pharmacological grounds may be obscured by other effects on the heart rate in patients with congestive heart failure and cannot be expected to give a guide to the control of digitalis treatment with subsequent risk of toxicity if digitalis treatment is pushed to obtain a probably illusory beneficial effect on contractility. The risk of toxicity from digitalis glycosides is so great that they should not be pushed in this way in patients with congestive heart failure in sinus rhythm.

DIGOXIN

The availability of a plasma radioimmunoassay for digoxin favours the use of digoxin rather than the other digitalis glycosides, for which little advantage can be shown. The recent problems of variation in biological availability with different preparations of digoxin tablet have been overcome and all now have a uniform standard of high availability. Digoxin is primarily handled by renal excretion and maintenance doses must be correspondingly reduced in the presence of renal failure. This policy seems preferable to using a different glycoside in patients with renal disease such as digitoxin, which is handled by hepatic excretion. Suggestions that particular glycosides are less likely to cause nausea and vomiting are unsupported by objective data and there is little evidence that any of the purified glycosides are preferable in this respect. Although nausea may not be the earliest sign of toxicity, in that arrhythmias may occur without nausea, it should be regarded as suggestive of excessive treatment. Antiemetic drugs should never be given to allow continuation of the dose of digitalis that produced nausea.

A loading dose of digoxin is necessary to start treatment but the onset of action is gradual even after absorption so there is little advantage from parental administration; if quick control of heart rate is needed it is better to use an alternative treatment such as a beta-adrenergic-blocking drug or electroversion (DC shock). Some of the most exciting examples of digitalis poisoning are seen when a loading dose of a digitalis glycoside is given to a patient who is receiving a maintenance dose of digitalis prescribed elsewhere. Careful inquiry must be made before prescribing, remembering that an unfamiliar preparation may be in use, particularly in patients treated abroad. If there is any doubt and treatment is not urgent a standard maintenance dose of digoxin will be effective in a few days. A loading dose of digoxin in an average sized man might be 1 mg followed by 0.5 mg twice daily for one day and a maintenance dose of 0.25 mg twice daily with further adjustment according to the response after a few days. Some patients seem to have problems of absorption even with tablets of known high availability and may require unexpectedly large doses. Patients with severe heart disease tend to need small doses, perhaps because of impaired renal function and may be maintained on as little as 62.5 µg daily. This small tablet is also useful in treating patients requiring smaller doses than usual; these include patients with renal disease and the elderly, probably because of reduced renal function, patients of small body build, and those with hypokalaemia. Although the potentiation of digitalis effect by hypokalaemia has been questioned,³ there is some

evidence⁴ that the effect is a real one, as expected on theoretical grounds although a definitive study is awaited.

HYPOKALAEMIA

Hypokalaemia is particularly troublesome in patients with congestive heart failure as it may complicate concurrent diuretic treatment. The observation of a patient developing digitalis toxicity without change in digitalis dose when a diuretic is added to the regimen may be due to hypokalaemia or to impaired renal function as a result of diuretic treatment. Every effort should be made to avoid hypokalaemia from diuretic treatment in patients who are also being treated with digitalis glycosides.

THYROID DISEASE

The variation in response to digitalis treatment in patients with thyroid disease is probably at least in part related to changes in renal function, renal impairment making the patient with myxoedema particularly sensitive to digoxin, and excessive clearance accounting for the lack of response in thyrotoxicosis. The finding of similar changes in sensitivity to other glycosides, such as digitoxin, which are not excreted in the kidney suggests that variations in tissue sensitivity with changes in thyroid function may also play a part.

DIGITALIS TOXICITY

Digitalis toxicity usually presents as a rhythm disturbance, although nausea and vomiting may precede or accompany the rhythm change. Coloured vision, though not infrequent, is rarely a cause of complaint. Intestinal disturbances with ulceration in the small gut occur, particularly in the elderly. Ectopic atrial or ventricular tachyarrhythmias or atrioventricular block may occur, and the combination of tachycardia and block as in atrial tachycardia with a 2:1 or an irregular ventricular response is particularly characteristic. Apparent failure to control atrial fibrillation with a persisting rapid irregular rhythm may be due to a toxic atrial tachycardia with an irregular ventricular response. Occasional ventricular ectopic beats may accompany apparently satisfactory control of atrial fibrillation, but are evidence that toxic levels are being approached, but regular alternating ventricular ectopic beats (bigeminy) should not be tolerated. Plasma digoxin levels taken at the steady state (such as four hours after an oral dose) are useful in controlling treatment but cannot give an absolute guide because of other factors such as the plasma potassium concentration and probably thyroid status which influence the effectiveness of digitalis treatment. A steady-state plasma digoxin between 1.3 and 2.6 nmol/l (1 and 2 ng/ml) is usually satisfactory. A concentration below 1.3 nmol/l generally indicates ineffective treatment, but some patients with atrial fibrillation have natural disease of the atrioventricular conduction system which may prevent a rapid ventricular rate even without digitalis treatment. Most patients with satisfactory control of atrial fibrillation have steady-state plasma digoxin concentrations between 1.3 and 2.6 nmol/l. Levels above 2.6 nmol/l indicate a risk of toxic effects. Patients being treated with other glycosides, however, may show some response to a digoxin radioimmunoassay, although this will not reflect the true effect of treatment. Radioimmunoassays for other glycosides are not generally available and there is some advantage in the complementary assay techniques based on the action of digitalis glycosides on the Na and K dependent ATPase of the red cell which is responsive to all the glycosides in a manner corresponding to their cardiac effect; this method is not as freely available as the digoxin radioimmunoassay.

In treating digitalis toxicity, stop the drug and correct any hypokalaemia. It may be worth while to withhold potassium-depleting diuretics until the condition is under control, when the whole regimen may be reconsidered to avoid any recurrence. Atrial tachyarrhythmias rarely need specific treatment but ventricular tachyarrhythmias are dangerous, as fatal ventricular fibrillation may follow. Multifocal ventricular ectopic beats are a serious warning sign. Electroversion (DC shock) must be avoided in the presence of excess digitalis, as the digitalis effect is potentiated after the shock. If such treatment is essential, as in cardiac arrest from ventricular fibrillation, the smallest possible energy of shock should be used. Beta-adrenergic-blocking drugs should not be used, as control of the tachyarrhythmia may be associated with potentiation of the digitalis effect on atrioventricular

conduction, leading to complete heart block with suppression of the usual escape pacemakers by the beta-adrenergic blockade. It is better to suppress the ventricular ectopic rhythms with a drug such as lignocaine, which has little effect on atrioventricular conduction, or phenytoin sodium (Epanutin), which has a positively beneficial effect, improving atrioventricular conduction. Lignocaine as used in cardiac infarction, an initial bolus of 100 mg followed by a continuous infusion at 1 to 2 mg/min, is generally satisfactory and should be the first choice as the most familiar treatment.

General measures

Bed rest is useful in allowing the best use of a limited cardiac output and avoiding the effects of exercise in diverting blood flow to skeletal muscles, myocardium, and skin (for heat loss) at the expense of the visceral and, most significantly, the renal blood flow. It is common experience that admission to hospital leads to improvement in congestive failure without change in drug treatment. Sunbathing is occasionally possible, even in Britain, and should be avoided as it produces an obligatory cutaneous vasodilatation which diverts blood from more important vascular beds. In tropical conditions air conditioning is an advantage. If there is gross oedema or prolonged bed rest is thought likely to be needed prophylactic anticoagulant treatment should be started, as venous thrombosis and pulmonary embolism are common complications of congestive heart failure which may interfere with an otherwise satisfactory response to treatment.

Diuretic treatment

The mainstay of current management of congestive heart failure, diuretic treatment, does not improve cardiac function but removes troublesome oedema, which relieves symptoms such as breathlessness and allows greater activity although there is good evidence that cardiac output falls further rather than improves with this treatment.⁵

Modern diuretics are generally so potent in removing excess sodium from the body that there is little need for a low-salt diet provided excessively salty foods such as kippers and bacon are avoided and additional salt is not added to the food. The excess of water in the body is generally secondary to sodium retention and limitation of water intake is not needed except in special circumstances (see below). Mercurial diuretics are now of historical interest only as their toxicity limited administration to twice a week. Carbonic anhydrase inhibitors had a brief vogue, but are relatively weak diuretics and tolerance develops quickly, so they are not important except as preliminaries to the development of the thiazide diuretics.

Thiazide diuretics are the mainstay of current treatment. Duration of action is the only real difference between the various analogues, and relative potency is of little importance as larger doses of the less potent agents may be given without toxic effects. Other moderately potent non-thiazide diuretics seem to offer little advantage. These drugs are often combined in tablet form with a modest dose of a potassium supplement, which can only be beneficial in helping to prevent potassium depletion secondary to the sodium diuresis, provided it does not obscure the fact that the dose of potassium provided may be grossly inadequate as a replacement treatment in an intensive diuretic regimen. As Slow-K is such a satisfactory potassium supplement I generally use the corresponding thiazide diuretic cyclopenthiiazide (0.25 mg) with potassium (Navidrex-K). In mild congestive heart failure 0.25 mg twice daily will generally prove effective in maintenance treatment. A bed-time dose may produce troublesome diuresis during the night and it is usual to give the second dose of the day after lunch, but there is little difference in effect if the whole dose is given in the morning. It has been suggested that treatment on alternate days will avoid potassium depletion by allowing the body to correct the potassium loss on the day off treatment, but the alternate-day treatment produces a return of congestion on the day off treatment, which may be inconvenient, and in all but the mildest cases daily treatment is needed for effective control.

Loop diuretics—The newer agents such as frusemide, ethacrynic acid, and bumetanide have a more potent effect on sodium transport in the renal tubules than the thiazides and as they are relatively free of toxic effects have allowed control of more severe degrees of congestive heart failure. My experience is mainly with frusemide, which is given in very large doses (1 g intravenously) without toxic effect in renal failure. Although the previously untreated patient may be sensitive to relatively small doses, the usual maintenance regimen is 40 mg twice a day by mouth. A single dose of 80 mg each morning seems

just as effective as divided doses. The response is relatively rapid and divided doses with the second dose on return from work may avoid nocturnal breathlessness without a noticeable diuresis during the night. In general the diuretic dose needed to eliminate oedema and reach dry weight in hospital is similar to the dose needed to maintain the position when the patient increases his activity on return home and the temptation to try to wean the patient off diuretics when free of oedema must be resisted.

Potassium depletion

The increase in sodium load reaching the distal renal tubules with diuretic treatment increases the activity of the sodium-potassium exchange system of the distal tubules which is under the control of aldosterone, and potassium and hydrogen ions are lost in exchange for retained sodium ions. Potassium depletion is a major feature of the patient with treated congestive heart failure held at dry weight by intensive diuretic treatment⁶ and the condition is accentuated by hyperaldosteronism.⁷ The potassium depletion may be partly evident as a fall in plasma potassium concentration, but the true depletion shown by measuring total body potassium is generally much greater than expected on the basis of plasma measurements. The plasma potassium concentration is important as it may be reflected in symptoms such as fatigue or lethargy and is associated with potentiation of a digitalis effect which may lead to toxicity if digitalis treatment is being given concurrently with diuretics, as is often the case. Fortunately the plasma potassium seems to form part of a labile potassium pool which is easily corrected by giving potassium supplements.^{8, 9} An increase in oral potassium intake can compensate for the renal potassium loss sufficiently to maintain a normal plasma potassium concentration. In Britain an attempt to increase potassium intake by dietary changes is generally unsatisfactory as unacceptable and too expensive, although instant coffee and fruit juice, but not squash, are good sources, and it is usual to give potassium supplements. In view of the associated alkalosis potassium should be given as the chloride rather than the bicarbonate, and local ulceration where the tablet dissolves has been overcome by using slow-release preparations such as Slow-K. Two tablets three times a day is generally satisfactory, but care must be taken that deteriorating renal function does not interfere with potassium excretion and lead to dangerous hyperkalaemia, so regular measurements of plasma electrolytes are a worthwhile precaution.

Aldosterone antagonists

An alternative policy is to block or reverse the distal tubular sodium-potassium exchange with drugs which oppose the action of aldosterone. Nicholls *et al*⁷ have shown that there is a modest increase in plasma aldosterone in untreated congestive heart failure, a fall to low levels during diuresis, probably as renin secretion is reduced by the high sodium load in the distal tubules, followed by a rise of plasma aldosterone to very high levels as the patient approaches dry weight. The use of aldosterone antagonists has the advantage over potassium supplements that more of the less labile cellular potassium pool^{8, 9} may be replaced, and loss of other ions such as hydrogen ions and more subtle and less well-studied factors such as loss of magnesium ions may also be reversed, although it seems unlikely that the depleted cellular potassium can be completely restored unless the underlying heart disease can be cured, as by surgical correction of severe valve disease. A good case can be made for using aldosterone antagonists in all stages of congestive heart failure because although plasma aldosterone is low during diuresis the exchange mechanism is particularly active then as a large sodium load is reaching the distal tubule.

Two main groups of aldosterone antagonists are available—the specific antagonists such as spironolactone, which is structurally analogous to aldosterone and acts as a competitive antagonist; and structurally dissimilar agents such as triamterene and amiloride, which produce opposite effects to those of aldosterone in the distal renal tubule without specific antagonism. With all potassium-retaining diuretics there is a danger of hyperkalaemia if renal function is impaired and potassium supplements should not generally be given in addition to these drugs. Spironolactone (Aldactone) has a moderately effective gradual diuretic action and has been suggested as a possible sole diuretic treatment for moderately severe congestive heart failure. It is more often used in doses of 25 to 50 mg two or three times a day to prevent potassium depletion during treatment with other diuretics.

Amiloride (Midamor), which has largely replaced the less effective triamterene (Dytac), is not a specific aldosterone antagonist, although this may be an advantage in the severe hyperaldosteronism of the dried-out patient being maintained at dry weight by continued diuretic treatment in which very large doses of spironolactone would be needed to overcome the aldosterone effect, and the contrary action of amiloride may still be effective in spite of the high plasma aldosterone levels. Used in combination with frusemide at a ratio of 5 mg of amiloride with 40 mg of frusemide, amiloride generally maintains a normal plasma potassium concentration and removes the need for potassium supplements.

Other complications of diuretic treatment

SODIUM DEPLETION

The sodium-retaining tendency in congestive heart failure is so potent that there is generally little risk of producing sodium depletion. A very dramatic diuresis, as produced by a conventional dose of frusemide or a moderate intravenous dose in a previously untreated patient, may produce a sharp reduction in circulating blood volume before oedema fluid can be mobilised to replace the loss. A similar shock-like state is well known after excessive venesection or the mechanical removal of ascites or the drainage of dependent oedema by acupuncture. These procedures are rarely needed nowadays as excess fluid can generally be removed gradually with diuretic treatment.

It may be injudicious to push diuretic treatment to restore venous pressures to normal in all circumstances as a raised venous pressure may be necessary for effective ventricular filling in some conditions. The large systolic wave in the jugular venous pulse from functional tricuspid incompetence can often be eliminated as the blood volume is reduced and the right ventricle becomes smaller, but a large venous "a" wave in the stiff right ventricle of severe pulmonary stenosis may play a vital part in right ventricular filling and should not be abolished. The similar situation in the left ventricle in severe aortic stenosis is less evident clinically but may account for some deterioration in left ventricle performance with intensive diuretic treatment. In constrictive pericarditis the high venous pressure is essential to fill the heart in the high-tension pericardial sac and should not be abolished. Treatment should stop short at the elimination of troublesome oedema.

The dried-out patient with congestive heart failure exchanges the inconvenience and dangers of systemic and pulmonary oedema for a persistent low cardiac output state producing fatigue with hyperaldosteronism and the severe metabolic disturbance of cardiac cachexia. Treatment of congestive heart failure is not in any way curative and can be regarded only as a palliative regimen which can keep the patient alive and may allow definitive surgical correction of the underlying disturbance if possible. Many units at any one time will have a few patients for whom cardiac transplantation seems the only feasible long-term answer.

WATER RETENTION

Not to be confused with sodium depletion, in which the plasma sodium remains normal but the extracellular blood volume is reduced, is hyponatraemia in congestive heart failure, which may occur in the dried-out state or be associated with persistent oedema. There is little evidence to support the suggestion that an excessive antidiuretic hormone effect is responsible. It seems more likely that a failure of free water clearance in the kidney results when the sodium load reaching the distal tubule is much reduced. There is a temptation to give sodium to improve the low plasma sodium concentration, but total body sodium is often increased in these patients as the extracellular fluid remains expanded. A response to administration of salt is reported at times, presumably as the sodium load to the distal tubules is transiently increased, but this treatment runs the risk of producing fatal pulmonary oedema. A better approach is to reduce water intake to daily losses plus 500 ml unless climatic conditions are extreme and continue conventional diuretic treatment. This regimen often provokes intense thirst, and it may be necessary to remove flower vases and other sources of temptation. An osmotic diuretic such as intravenous mannitol combined with intravenous frusemide to prevent any immediate increase in circulating plasma volume will often produce a water diuresis. In general hyponatraemia may be regarded as a terminal event, and if surgical treatment is considered feasible it may

be justified to remove the excess water by dialysis, although the risk of any operation in those patients is very great.

Pulmonary oedema

Left ventricular failure may present as dyspnoea on exertion or as paroxysmal nocturnal attacks, leading eventually to frank pulmonary oedema. Similar changes may occur in pulmonary congestion due to mechanical obstruction to left ventricular filling as in mitral stenosis. Radiological evidence of pulmonary congestion with distended upper-lobe veins, interstitial pulmonary oedema producing the horizontal Kerley B lines at the costophrenic angles, and perihilar oedema may precede other clinical evidence of pulmonary congestion. Wheeziness from bronchial mucosal oedema is often a major feature, leading to the description as "cardiac asthma," probably because the bronchial veins communicate with the pulmonary veins and become congested as the pulmonary venous pressure rises. Wheeziness may cause diagnostic confusion with obstructive airways disease, particularly if both airways obstruction and left ventricular disease coexist, as is often the case in elderly patients in Britain. As some treatments recommended for left ventricular failure may be dangerous in airways obstruction it is useful to have a therapeutic programme suitable for use if the diagnosis is in doubt.

Morphine is the first line of treatment for left ventricular failure if the diagnosis is certain. It relieves dyspnoea by reducing the sensitivity of the respiratory centre to reflex stimulation from the stiff congested lung, reduces associated anxiety, and probably helps to shift fluid from the pulmonary to the systemic venous system by dilating systemic veins.

Aminophylline—In airways obstruction morphine might be fatal by reducing the sensitivity of the respiratory centre and critically impairing ventilation; a useful alternative is aminophylline 250 mg intravenously or 500 mg intramuscularly, which has a dramatic effect in relieving airways obstruction as well as a potent action in increasing myocardial contractility. In less acute situations aminophylline suppositories are useful and two at night may prevent paroxysmal nocturnal dyspnoea in the early hours of the morning. The treatment produces rectal irritation after a few nights but it is useful while other measures are becoming effective.

Salbutamol—Sympathomimetic drugs help relieve airways obstruction, but isoprenaline may have an adverse effect on the diseased heart, producing undesirable tachycardia or ventricular ectopic rhythms, and it is preferable to use the more selective agent salbutamol (Ventolin), which has little cardiac effect, particularly if given by inhalation.

Oxygen treatment—The presence of central cyanosis in left ventricular failure indicates a possible improvement from increasing the oxygen content of the inspired air. The relatively minor interference with gas transfer across the alveolar capillary membrane in pulmonary oedema is overcome sufficiently by hyperventilation to prevent a rise in arterial carbon dioxide tension and the arterial hypoxia is chiefly due to ventilation-perfusion imbalance with perfusion of under-ventilated alveoli. In left ventricular failure there is no contraindication to producing as high a concentration of inspired oxygen as possible although in obstructive airways disease inspired oxygen is usually limited to avoid the complete removal of hypoxic ventilatory drive. In cases of doubt the use of a moderate increase in inspired oxygen is still of some beneficial effect. The Ventimask or similar system supplying a controlled mixture of inspired oxygen and ambient air is generally available for use in respiratory failure. Nasal catheters with a moderate flow of oxygen to avoid drying of the nasal mucosa similarly present a less controlled mixture of inspired oxygen and ambient air. It is in fact difficult to provide 100% oxygen unless the patient is being mechanically ventilated via an endotracheal tube and such a system produces a risk of pulmonary oxygen toxicity, one of the causes of "pump lung" after cardiopulmonary bypass. Other systems to supply 100% oxygen require a close-fitting mouth and nose piece with an oxygen reservoir to draw on during inspiration. In practice a moderate increase in inspired oxygen concentration is the best that can be achieved.

Pulmonary oedema is removed by diuretics as effectively as systemic oedema; this obvious treatment is sometimes overlooked as the oedema is not visible. In fact, removal of relatively small amounts of oedema from the lungs has a dramatic effect on symptoms.

Intravenous frusemide will produce a dramatic response, but in a previously untreated patient the dose is much smaller than the usual oral doses for maintenance treatment and 10 mg intravenously is usually adequate. It is said that the diuresis begins before the needle

can be withdrawn from the vein. In older men with prostatic problems caution is needed with diuretic treatment as acute retention may be provoked. A preliminary rectal examination, easily carried out in the sitting position, is worth while.

Digitalis treatment—In acute left ventricular failure there may be transient beneficial effects from digitalis treatment, and digoxin 1 mg by mouth in a patient of normal build with no undue sensitivity is useful for starting treatment. If the situation is urgent half the initial dose may be given intravenously although the slow onset of action of digitalis glycosides after absorption reduces the advantages of intravenous treatment. In pulmonary oedema secondary to the rapid ventricular rate associated with the onset of atrial fibrillation as may be seen in mitral stenosis, control of the tachycardia has first priority, and the rapid action of beta-adrenergic-blocking drugs may be preferred to the more gradual action of digitalis glycosides.

Mechanical measures—The presence of a pleural effusion may critically increase dyspnoea in the patient with left ventricular failure. Effusion may arise in isolated left ventricular failure by transudation from the congested lung or secondarily to pulmonary infarction from venous thrombosis and embolism. Aspiration of a large pleural effusion may produce striking relief of breathlessness.

Venesection is a time-honoured treatment which may be appropriate if the jugular venous pressure is raised and has the effect of transferring blood from the pulmonary to the systemic venous system. Removal of too much blood too quickly may critically reduce ventricular filling. Removal of one pint (half a litre) is usually satisfactory, conventional blood donor equipment may be used, and the blood is usually suitable for transfusion. A similar effect can be produced less traumatically by venous occlusion with blood-pressure cuffs on the limbs.

Intermittent positive-pressure ventilation—Some patients with pulmonary oedema remain resistant to all these treatments. Attempts to remove froth from the airways by inhaling agents reducing surface tension have relatively little value in the pulmonary oedema of congestive heart failure, and in the intractable patient the best solution is endotracheal intubation which facilitates cleaning the airways followed

by intermittent positive-pressure ventilation. The response is not due to forcing the oedema fluid back across the alveolar-capillary membrane but to the rise in intrathoracic pressure, which tends to keep blood in the systemic venous system and reduce the volume of blood in the pulmonary veins. In patients with mitral stenosis the induction of anaesthesia for mitral valvotomy may improve pulmonary oedema in a similar way.

Acute left ventricular failure carries serious prognostic implications. In hypertension it is a prime indication for effective control of blood pressure, and, although left ventricular failure can be controlled by antihypertensive drugs alone in these patients, this is a therapeutic tour de force which need not be repeated. Diagnostic confusion is sometimes produced by the transient rise of blood pressure which often accompanies the vasoconstrictor response to left ventricular failure in myocardial disease. In aortic stenosis one attack of left ventricular failure is an indication to consider aortic valve replacement as the natural history of aortic stenosis is such that further episodes of left ventricular failure are likely even with intensive treatment and each attack is progressively more difficult to treat. The tragedy of "death on the waiting list" is avoided only by prompt referral for operation after the first episode of left ventricular failure.

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Letter from . . . Paris

Medical writing in France

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There are two schools of medical writing in France: those who want all French research to be published in English; and those who, remembering that France is the cultural head of a great francophone community, believe that not to publish in French is a betrayal. Both groups are having difficulties and disappointments because neither realises the importance of the techniques of medical writing, as distinct from the language in which reports are written.

Before submitting papers to Anglo-Saxon journals, authors in the first group generally have the English revised by someone whose mother tongue is English. Yet to their surprise editors still send the papers back for rewriting. These authors have not appreciated that the structure of a paper is one thing, the

language in which it is dressed up is another, and that a paper may be written in faultless English yet be badly written, camouflaging the message. This was understood by the distinguished medical journalist R Veylon when he commented¹ on the failure of certain French journals whose sponsors had imagined they would penetrate Anglo-Saxon medical circles simply by transposing articles into the English language while keeping the original non-structure. Of this school, the "anglicists," one might say that although they are content to throw out the baby (the French language) they insist on keeping the bath water (amateur writing).

The second group, in contrast, would keep the baby, but they too refuse to part with the bath water. Modern scientific methods in medical writing are closely linked in their minds with writing in English. The patriotic Frenchman who rejects the one must also reject the other. The confusion of method with language has been well shown by Vargues.² After criticising Anglo-Saxon papers for their simple language and detailed precision, he, firstly, recommended that French research should be reported in the form of "scholarly dissertations, with thesis, antithesis, and synthesis," and, secondly, renewed the classical