MEDICAL TREATMENT

BY

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With a chapter on the Tropical Diseases by

Colonel W. R. M. DREW

C.B.E., F.R.C.P., D.T.M. & H.

In some standard textbooks of medicine emphasis is on aetiology and diagnosis and the sections on treatment do not always receive the attention they deserve. It is hoped, therefore, that this comprehensive volume dealing with this one aspect of medical practice will prove of value to general practitioners, house officers and senior students.

The problems of treatment are presented as seen through the eyes of a general physician and essential clinical features of the various diseases are given briefly. During the past two decades the tremendous developments in therapeutics have made it possible to offer curative treatment to sufferers from many diseases for which previously little could be done. The availability of successful therapy has been a criterion of inclusion, whether the disorder is common or a comparative rarity.

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With Chapters on the

TROPICAL DISEASES

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PREFACE

DURING the past two decades tremendous progress has been made in all aspects of medicine and nowhere has the advance been more rapid than in the field of treatment. The discovery of antibiotics, the advent of steroid therapy and the realisation of the importance of maintaining a correct fluid and electrolyte balance, are but a few of the many discoveries which have made it possible to-day to offer curative treatment to sufferers from many diseases for which previously little could be done. Nevertheless many forms of modern therapy are not without danger and it is more important than ever before that the doctor should be fully conversant with the possible hazards of the treatment he prescribes.

It is probably true, however, that in many of the standard textbooks of medicine, the sections on treatment have not received the increasing attention which they deserve and it seemed to me that there should be a place for a comparatively short, yet reasonably comprehensive, book which dealt primarily with this aspect of medical practice. It is in an endeavour to fulfil this need that the present volume has been written and I hope it may prove to be of value to general practitioners, house officers and senior students.

For the two chapters dealing with tropical medicine, I am greatly indebted to Colonel W. R. M. Drew, now serving as Consultant Physician to the British Middle East Land Forces. I was most fortunate in obtaining his services. The remainder of the book is my own, although I have incorporated much helpful advice given generously by my colleagues at Guy's Hospital.

I feel that there are certain advantages in a textbook of this type and size being written by a single author and I have endeavoured to present the problems of medical treatment as seen through the eyes of a general physician.

Before describing treatment, the essential clinical features of the various diseases are briefly enumerated and, in allotting space, I have been guided not only by the frequency with which the condition occurs, but also by the availability of therapy, so that a rare disease, which can be treated, has received more attention than a more common, but virtually untreatable, condition.

While a few references are given at the foot of the relevant pages, no attempt has been made to make these in any way comprehensive and if it is thought that undue emphasis has been given to papers by Guy's men, I can only make the excuse that, apart from the war years, the whole of my medical life has been spent at Guy's Hospital.

It is my great pleasure to thank my colleagues on the Hospital staff, Dr. Ian Mackenzie and Dr. David Stafford-Clark for their advice on the Neurological and Psychiatric sections respectively, Dr. A. S. Grimble for his help with the chapter on Venereal Disease and Mr. T. L. T. Lewis for his suggestions on the gynaecological aspects of Endocrinology.

Dr. P. R. Montgomery has given me extensive assistance in the preparation of the Dermatological chapter for which I am extremely grateful.

Much valuable criticism on the general medical part of the book has been given me by Dr. M. G. Thorne, now Consultant Physician to Torbay Hospital; by Dr. K. D. Allanby and Dr. J. R. Trounce, who have been my colleagues in the Department of Medicine; and by Dr. C. L. Joiner, senior medical registrar at Guy's Hospital. I owe them a special debt of gratitude.

I would also like to place on record my sincere thanks to Dr. P. H. Kendall, senior registrar in the Department of Physical Medicine at Guy's, for his help with the section on intra-articular injection; to Dr. Paul Polani, for his advice on the treatment of children; to Miss Newland, our chief Dietician, who has supervised the preparation of the sections and tables on diets; and to the Medical Illustration Department at Guy's Hospital for the preparation of Figure 1 on page 169.

I am also indebted to Dr. Margaret Waile, to Dr. Maurice Lessof, my present Registrar, and to all my house physicians over the past eighteen months for their great assistance in reading manuscript and proofs.

I wish to thank Mr. S. F. Izzard, Deputy Chief Pharmacist to Guy's Hospital, for the considerable time and trouble he has taken in checking the preparations and dosages throughout the book.

Mr. W. Hill has been responsible for the index and to him I owe most special thanks.

I would also like to acknowledge the great help I have received from Mr. J. Rivers and Mr. J. A. Rivers of J. and A. Churchill Ltd. No author could have had greater consideration.

Finally I should like to thank my secretary, Miss Sheila Bubb, for the many hours she has spent typing illegible manuscript, and my wife for her special encouragement on those not infrequent occasions when it seemed to me that this book would never come to an end.

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Diseases of the Heart

ACUTE CIRCULATORY COLLAPSE (SHOCK)

ACUTE circulatory collapse or shock occurs whenever there is a sudden substantial fall in cardiac output. In an attempt to ensure that the brain and heart continue to receive an adequate oxygen supply, the blood flow is deviated from less essential parts. Intense vasoconstriction therefore occurs in many tissues, notably the skin; later as a further compensatory mechanism much of the blood flow to the renal glomeruli may be short-circuited so that filtration is greatly reduced with resultant oliguria. A patient suffering from shock is pale with a cold clammy skin especially noticeable at the extremities. The pulse will be thready and usually rapid and the blood pressure low. Nausea is common and vomiting may occur. Eventually permanent changes may take place in the affected organs and the condition will become irreversible.

Causes. A sudden fall in cardiac output may be due to a central cause when associated venous engorgement may or may not be present. The commonest example of this is myocardial infarction. Alternatively, it may occur if there is an inadequate venous return of blood from the peripheral circulation, a state of affairs known as peripheral circulatory failure.

The causes of peripheral circulatory failure may be subdivided into two main groups. In one the essential factor is a reduction in the total blood volume such as occurs in haemorrhage, plasma loss or dehydration. The mechanism responsible for the second group is not fully understood. Probably changes in the capillaries occur in certain parts of the vascular bed, notably the splanchnic area, with resulting vasodilatation so that there is a pooling of large quantities of blood in these situations. The total blood volume is therefore unaltered, but the amount taking an effective part in maintaining the circulation is grossly reduced with the result that compensatory vasoconstriction in the less essential areas unaffected by the 'pooling' occurs, and a clinical picture of shock is produced. This 'pooling' mechanism is usually brought about by reflex action which may follow trauma or pain especially in the area of the solar plexus or testicle, or the perforation of a hollow viscus. In a transient form, it may occur after an unpleasant psychological experience and give rise to a fainting attack. Less commonly, the capillaries may be damaged by toxins as when states of shock occur during acute infective illnesses.

The more important causes of acute circulatory collapse are set out on the following page.

CAUSES OF ACUTE CIRCULATORY COLLAPSE

(*A*) **Central.** (Sometimes accompanied by increased venous pressure.)
1. Myocardial infarction.
2. Cardiac Tamponade. (Pressure from haemopericardium or pericardial effusion, preventing adequate filling of the heart.)
3. Massive pulmonary embolism. (Obstructing blood leaving the heart.)
4. Myocardial toxaemia in infections (e.g. diphtheria).

(*B*) **Peripheral. Diminution in Blood Volume.**
1. Haemorrhage.
2. Plasma loss from wounds or burns.
3. Dehydration and salt depletion.
   (*a*) Vomiting and diarrhoea (cholera, dysentery, etc.).
   (*b*) Polyuria. (Diabetes.)
   (*c*) Excessive sweating (heat exhaustion).
   (*d*) Addison's Disease.

(*C*) **Peripheral. 'Pooling' Mechanism.**
1. Reflex Mechanism.
   (*a*) Transient fainting.
   (*b*) Following trauma, especially to solar plexus area or testicle.
   (*c*) Following perforation of a hollow viscus.
   (*d*) Following severe pain.
2. Capillary damage from toxins. (Diphtheria, pneumonia.)
3. Sudden suprarenal failure. (Meningococcal septicaemia.)
4. Drugs.
   (*a*) Spinal anaesthetics.
   (*b*) Ganglion blocking agents. (Hexamethonium.)
5. Anaphylactic Shock. (Mechanism unknown.)

Frequently circulatory collapse may be due to a combination of several of these factors, for example peripheral circulatory failure may follow the pain of a myocardial infarction or pulmonary embolism, while shock following a major abdominal operation may be due to a combination of most of the peripheral causes shown above.

Treatment. Obviously this must be directed to the primary cause of the acute circulatory failure. The commoner medical conditions which may be responsible are enumerated in the foregoing table and are discussed elsewhere in this book. It is, however, now generally recognised that it is essential to restore the systolic blood pressure in all cases of shock to a minimum value of 90 mm. of mercury as soon as possible, and the various means by which this can be achieved are summarised below. Which methods are adopted will depend upon the primary cause of the shock.

SYMPTOMATIC TREATMENT OF SHOCK. *Warmth and rest.* Absolute rest is essential and patients must be kept warm. Care is necessary to ensure that they do not suffer burns from hot-water bottles, which must never be placed in contact with the unprotected skin. If pulmonary oedema is not present, the foot of the bed may be raised about eighteen inches.

Morphine. Morphine (10–20 mg.: gr. $\frac{1}{6}$–$\frac{1}{3}$) by injection is essential for the control of pain and may be of great value in restless and apprehensive patients. It should not be given to stuporose or comatose patients and is absolutely contraindicated in Addison's disease or acute suprarenal failure.

INTRAVENOUS BLOOD TRANSFUSION. It is first necessary to determine the patient's blood group and carry out emergency cross-matching.

The intravenous transfusion of from one to three or more pints of blood is indicated in all cases of shock due to haemorrhage and may be of great value in other types of acute circulatory failure. If the blood loss has been large, two pints may be run in as quickly as possible, and the transfusion may have to be continued until the source of haemorrhage has been controlled. Further administration must depend upon the clinical condition of the patient, the haemoglobin and the haematocrit level. Haemo-concentration may be so marked in the twenty-four hours following a severe haemorrhage that a haemoglobin estimation alone may be most misleading at this time, as a near normal level may occur in the presence of severe blood loss. If doubt exists as to the cause of shock, it is always wise to consider blood transfusion.

Intravenous transfusion is dangerous in the presence of either a raised venous pressure or pulmonary oedema, and if it is essential to give blood in these circumstances, the intra-arterial route is to be preferred. The neck veins and lung bases should be watched continuously, a rising venous pressure or the appearance of râles being indications for slowing down or stopping the transfusion.

INTRA-ARTERIAL BLOOD TRANSFUSION. This is indicated in very severe cases of shock, especially if they have failed to respond to blood transfusion via the intravenous route. The blood pressure may be restored by much smaller quantities of blood than are required intravenously, so that it may have a place in the treatment of shocked patients who exhibit a raised venous pressure. Fluids other than blood must never be given by this route as gangrene may follow if non-oxygen carrying solutions are used. In certain circumstances therefore intra-arterial transfusion is a valuable treatment, but it should only be employed by those familiar with its technique.

DEXTRAN AND PLASMA INFUSIONS. These are valuable substitutes for blood and may be given in emergencies while blood is being prepared and cross-matched. Dextran, a plasma substitute with a molecular weight equal to that of albumin, may be given intravenously as a six per cent solution in normal saline. It is preferable to plasma, which carries the risk of transmitting homologous serum jaundice. In shock complicating burns, plasma or dextran is preferable to blood, at any rate at first. As a rough guide, 75 ml. should be given in the first twenty-four hours for every per cent of body surface burned.

Dextran increases rouleau formation which adds to the difficulties of cross-matching. A sample of blood should therefore be withdrawn for this purpose before a dextran infusion is set up, in case blood transfusion is required subsequently.

SALINE AND GLUCOSE-SALINE INFUSIONS. These are indicated in cases of shock due to excessive water and electrolyte loss, as may occur after diarrhoea, vomiting or excessive sweating. The type of solution to be used and the quantities to be given will be fully discussed in Chapter 26. The danger of producing pulmonary oedema is greater than with infusions of blood, plasma or dextran.

SYMPATHOMIMETIC DRUGS. These raise the blood pressure by producing vasoconstriction throughout the peripheral circulation. Methedrine and noradrenaline are the most satisfactory, as these exert little or no

effect upon the pulse rate. These drugs act humorally and do not depend upon an intact nervous system, so that they are especially valuable in the treatment of hypotension due to hexamethonium and other ganglion blocking agents.

Methedrine (*Methylamphetamine hydrochloride*). This may be given by intramuscular or intravenous injection. The normal maximum dose is 30 mg., although it is wise to divide this dose into two equal amounts given at an interval of fifteen minutes if the intravenous route is used. The effect is usually transient, but repeated injections may be made at intervals of one or two hours. Overdosage may very occasionally cause cerebral excitation and convulsions. Methedrine is especially useful in correcting the hypotension following the therapeutic use of hexamethonium and other ganglion blocking agents.

Noradrenaline. This is the most efficient drug we have for controlling hypotension and may be used in most types of shock including cardiac infarction. It is of doubtful value in adrenal insufficiency. The most suitable preparation is Levophed, which is l-noradrenaline bitartrate. It is put up in ampoules, each ml. of solution containing the equivalent of 1 mg. of base. It must never be given undiluted. It is usual to add 4 ml. of Levophed to each litre of the intravenous solution to be given (saline, glucose, plasma, or blood) so that this contains 4 microgrammes per ml. Once diluted in this manner, the solution must be used within six hours. The speed of the infusion is regulated until the blood pressure is maintained at the desired level. This is achieved in most patients when about 2 ml. (15 to 27 drops) is being given per minute, but it may be necessary to exceed double this rate. If the patient's clinical condition makes it undesirable to administer fluid at this speed, a double strength solution (8 ml. of Levophed to each litre of fluid) may be used, when the effective rate at which the infusion must be given will be halved. The blood pressure must be charted every five minutes and the speed of the infusion adjusted. Overdosage may produce dangerous hypertension and the patient must never be left unattended. Levophed is not therefore suitable to add to intravenous solutions which are being given rapidly as replacement therapy after haemorrhage or in cases of severe dehydration. Its main use is in those hypotensive states which are not due to blood or fluid loss or which fail to show the expected response to intravenous replacement therapy. It may be continued as long as the acute hypotensive emergency exists.

OXYGEN. The administration of oxygen is sometimes of limited value in shocked patients, especially if there is any degree of pulmonary oedema. It should only be considered as a subsidiary method of treatment.

CORTISONE AND HYDROCORTISONE (p. 609). These substances are invaluable in the treatment of adrenal failure such as occurs in Addison's disease (p. 448), or after suprarenal haemorrhage. They have no place in the immediate treatment of other types of acute circulatory collapse, but some degree of adrenal exhaustion may complicate a period of shock lasting for more than forty-eight hours, especially in cases of burns, and in these circumstances from 100 to 150 mg. of cortisone may be given daily either orally in divided doses or by a single intramuscular

injection. The dose should be tailed off as the condition improves. Children seem especially liable to this complication.

INTRAVENOUS GLUCOSE. 20 G. of glucose in 50 ml. of saline may be given intravenously in cases where shock is suspected of being due to myocardial toxaemia (e.g. diphtheria).

NIKETHAMIDE. This drug has no direct action upon either the heart or blood vessels and is only of value in respiratory depression. This may occur in shocked patients after morphia. It is given in doses of 2–4 ml. either subcutaneously or intravenously as Inj. Nikethamid. B.P.

Summary. The various methods of combating shock have been enumerated. It is emphasised once again that the treatment of any individual case will depend upon the primary cause and an accurate diagnosis is essential if the most effective therapy is to be given.

CARDIAC FAILURE

We have seen that when sudden cardiac failure occurs, the clinical picture may be that of shock. More frequently, however, the condition produced is not that of acute circulatory collapse, but is dependent upon the high venous pressure in the systemic or pulmonary circulations.

Although the heart consists of but one muscle and therefore might be expected to fail as a single organ, it is often possible to detect on clinical examination that one or other of the ventricles is failing to perform its work efficiently. The recognition of right-sided and left-sided failure is a useful one, and the treatment of each type, although similar, varies in some respects and will be considered separately.

Right-Sided Failure

Clinical Types

Right-sided or congestive cardiac failure is recognised by the well-known triad of signs—distended neck veins, an enlarged tender liver and oedema of the dependent parts. The cardiac output is usually below normal and the patient will have cold extremities, with a clammy skin, while the pulse will be feeble and rapid. Sometimes, however, the unmistakable signs of congestive cardiac failure occur in the presence of a full, bounding pulse and a warm skin. This is called 'high-output failure' for in such patients the cardiac output may be considerably raised. It represents a partially successful attempt by the heart to compensate for the abnormal conditions present in the circulation in such diseases as anaemia, thyrotoxicosis, arterio-venous shunts or in chronic lung diseases where oxygenation of the blood is grossly impaired giving rise to the anoxic type of 'cor pulmonale'. If the cause of the high-output failure cannot be remedied, the heart muscle will gradually fail and the picture will change into that of the more usual low-output type with obvious evidence of an impaired peripheral circulation. It is therefore a mistake to attempt to divide cases of congestive cardiac failure into two watertight compartments, but the possibility that failure may represent a compensatory mechanism must always be borne in mind

when treatment is being considered. We shall discuss the special problems that arise in this connection shortly.

Therapeutic Considerations

Before deciding upon the treatment of a patient with right-sided failure, we must try to discover why the heart has failed. This may be due to an ischaemic myocardium, occurring either as the result of progressive atheroma or from the recent occlusion of a coronary artery; alternatively in emphysema, mitral stenosis or certain types of congenital heart disease the raised pulmonary pressure may finally prove too much for the right ventricular muscle. In these conditions, apart from the possibility of cardiac surgery, we have no means of remedying the fundamental cause of the failure and treatment must of necessity be symptomatic. On the other hand, failure may have been precipitated by some condition requiring urgent and vigorous therapy which may have complicated an already damaged heart. Examples which should always be borne in mind are (1) an uncontrolled cardiac arrhythmia such as auricular fibrillation or paroxysmal tachycardia, (2) an attack of rheumatic fever, (3) anaemia, (4) toxaemia from an infection such as pneumonia or diphtheria, (5) thyrotoxicosis and (6) bacterial endocarditis. Occasionally the failure may be due to such conditions as myxoedema, constrictive pericarditis or in tropical countries beri-beri. The detailed treatment of all these conditions is discussed under their appropriate sections. In patients who have compensated heart disease the onset of failure may be brought about by pregnancy or a change to a more active occupation.

If it is possible, therefore, to treat the primary condition which was responsible for the onset of failure, it is of paramount importance to instigate the necessary therapy without delay in addition to the general measures which will now be described. Unfortunately in many patients the primary cause of the failure is not amenable to treatment and symptomatic therapy is all that can be attempted. Nevertheless it is gratifying to observe the improvement that the measures to be described may effect.

Symptomatic Treatment. REST. As a general rule patients suffering from congestive cardiac failure should be confined strictly to bed. The amount of activity allowed will vary from case to case and must depend upon the amount that can be done without distress. In the acute stages a patient should be washed and fed, but when he can perform these acts himself without breathlessness or exhaustion there is no reason why he should not be allowed to do so. Visitors should be restricted to avoid fatigue, and it is important to remember how tiring a visit can be. The patient will be more comfortable propped up as long as dyspnoea is present at rest and every effort should be made to ensure that he does not slip down while asleep. In this connection a cardiac bed may prove of great value, or failing this it may be wise to let him sleep sitting up in an arm-chair. Sometimes in chronic cases where little further improvement can be expected, it is reasonable to allow limited activity about the home or ward as this may help morale, an all-important consideration in such patients.

BOWELS AND MICTURITION. Micturition should be carried out in bed with a bedpan or bottle. The use of a bedpan for defaecation is more open to question. Many people find great difficulty in getting their bowels open in this way and the ensuing struggle may prove far more exhausting than being helped onto a bedside commode. The insistence on the use of a bedpan is frequently an aggravating factor in causing constipation, which must be avoided at all costs in these patients. Unless there is severe dyspnoea at rest, the patient's own wishes should be taken into consideration and common sense decide the line to be followed. Regular doses of liquid paraffin together with a mild laxative such as infusion of senna pods (made by standing four to six pods in water for six hours) are an important part of treatment. Constipation, when it occurs, should be treated by a gentle enema. Any attempt at purgation is absolutely contraindicated.

DIET. The process of digestion calls for a sudden increase in the cardiac output and large meals should be avoided. Venous congestion in the gastric veins frequently causes nausea, anorexia and vomiting and these symptoms may be increased by digitalis which the patient is likely to be taking. The diet should be easily digestible and contain the minimum of calories. The total fluid intake must be limited to between thirty and forty ounces in the twenty-four hours. Initially milk alone may be ideal, but after a few days it will be necessary to increase the calorie intake to a little over 1,000 by the addition of such articles as lightly boiled eggs, boiled fish or white meat. Biscuits and thin toast with a little butter can be allowed. Vegetables should be given as a purée, and fruit juice provides a palatable source of Vitamin C. As progress continues, the diet is correspondingly made more liberal, but meals should always be small and frequent.

SALT RESTRICTION. Sodium retention is a feature of heart failure and some restriction in the intake of salt is essential. In most patients, however, the use of mercurial diuretics, which greatly increase sodium excretion, combined with a dietary regimen which reduces the daily intake of salt from the normal 10 grammes to something under half this amount, will prove effective and enable a satisfactory diuresis to occur. This can be achieved by forbidding the taking of added salt at table and avoiding the major salt-containing foods such as ham, bacon, sausages, smoked fish, pickles and tinned foods to which salt has already been added. All sauces and meat extracts such as Oxo, Bovril and also Marmite should be forbidden as well.

In cases with marked oedema or if a diuresis is not obtained on the above regimen, further measures are necessary. No salt or baking powder must be used in cooking, and the diet should be chosen from the following table. By this means the intake of sodium chloride can be reduced to a little over 1 gramme daily. In practice the diet of milk advocated above for the acute stages of heart failure has a very low salt content.

LOW SODIUM DIET

1. Use no salt or baking powder in cooking.
2. Avoid medicines containing sodium. The sodium content of sedative doses of sodium amytal is negligible.

3. Preserved, salted, smoked or tinned foods (except fruit) are prohibited.
4. Ration milk to 10 oz. daily. Additional 'low salt' milk (Edosol) may be used in cooking.
5. Use dripping, lard, olive oil or suet instead of butter or margarine (unless salt-free).

Type of Food	*Permitted*	*Forbidden*
CEREALS	Arrowroot: Barley: Cornflour: Flour: Macaroni: Oatmeal: Rice: Sago: Semolina: Tapioca: Shredded Wheat: Salt-free Bread.	Bread: Water Biscuits, Cream Crackers: Cornflakes: Grapenuts: Post-Toasties: Ryvita: Vita-Wheat.
DAIRY PRODUCE	Unsalted Butter or Magarine: Cream: Cream Cheese (home made): Eggs: Milk: Condensed Milk.	Salted Butter and Margarine: Cheese.
MEAT	Beef: Mutton: Pork: Hare: Rabbit: Sweetbreads: Fresh Tongue: Game: Brains.	Bacon: Silverside: Ham: Kidney: Sausage: Meat paste.
FISH	Bass: Herring: Red Mullet: Fresh Salmon: Plaice: Sole: Cod.	Crab: Lobster: Haddock: Shell-fish: Whiting: Fish-paste.
VEGETABLES	Root Artichokes: Asparagus: French Beans: Runner Beans: Brussels Sprouts: Lettuce: Leeks: Marrow: Mushrooms: Onions: Parsnips: Peas: Potatoes: Tomatoes.	Beetroot: Carrots: Celery: Radishes: Spinach: Watercress.
FRUITS	Nearly all fresh, stewed, preserved or tinned fruits: Fruit Juices: Unsalted Nuts.	Melon: Passion Fruit.
SWEETS	Sugar: Plain Chocolate: Honey: Ice Cream: Jam: Most sweets.	Blended and milk chocolate: Golden Syrup, Treacle (black): Toffee: Chutney.
CAKES AND PASTRIES	Doughnuts: Oatmeal Biscuits (made with lard and no salt): Shortbread: Sponge: Jelly: Pancakes: Yorkshire Pudding (no salt): Cereal puddings.	Currant and ginger cakes: Swiss roll: Anything made with baking-powder.
BEVERAGES	Coffee: Tea: Fruit Juices: Soft Drinks: Wine: Spirits.	Marmite: Oxo: Bovril: Bournvita: Cocoa: Horlicks: Ovaltine.

If facilities for the preparation of salt-free meals do not exist, further sodium restriction may be achieved by the administration of ion-exchange resins in the ammonium-potassium cycle which absorb sodium in the alimentary tract. Katonium in doses of 10–15 G. three times daily taken in dilute fruit juice with meals is a suitable preparation. Although more palatable than the earlier resins, it is somewhat irritating to the gastric mucosa and may sometimes increase the nausea of which these patients may complain.

Salt substitutes, for the most part containing potassium salts, are available but are not recommended.

Severe salt restriction will abolish thirst and no curtailment of fluid intake is necessary on such a regimen. Very occasionally the combination of this method of treatment with the use of mercurial diuretics may lead to a state of serious sodium depletion in the presence of oedema (predominant salt depletion, p. 594) and in these circumstances salt will be required therapeutically. Regular estimation of the serum sodium

should therefore be carried out if patients do not make the expected response to these methods of therapy.

OXYGEN. Oxygen therapy is indicated in the acute phase of congestive cardiac failure for patients who are cyanosed or dyspnoeic at rest. Although theoretically the administration of oxygen should hardly affect cyanosis due to a low cardiac output, in practice the result may be dramatic. This is probably due to its effect upon coexisting pulmonary oedema which is always present to some degree. The most efficient way to administer oxygen is by means of an oxygen tent, provided that this is well tolerated by the patient. Nothing is more undesirable than to attempt to keep a frightened, struggling patient in a tent, and it is wise to explain to patients that the tent is a means of making them more comfortable and that they need not remain in it unless they so wish. A B.L.B. mask provides a reasonably efficient alternative, but this must be fitted with a mouth-piece as dyspnoeic patients tend to mouth-breathe. Oxygen is usually given at the rate of about 6 litres per minute. The delivery of oxygen through a nasal catheter or by means of spectacles fitted with nasal tubes is of limited value and much less efficient than a tent or a B.L.B. mask. In the chronic type of right-sided failure oxygen is best avoided except as a means of tiding over acute episodes. Such a patient may too easily become dependent upon his oxygen tent, a state of affairs liable to have a disastrous effect upon his morale and from which it may be very difficult to wean him. When oxygen is being given, it is, of course, essential to ensure that no smoking occurs in the vicinity. In certain cases of cor pulmonale oxygen may be contra-indicated (p. 16).

SEDATION. Measures to ensure that these patients have adequate mental and physical rest are of the greatest importance. An attack of congestive cardiac failure, if acute, is a distressing and frightening experience and the doctor should always remember to spend a moment or so in reassuring the patient. It is wise to explain briefly the object of the treatment that is to be given, while painting an optimistic picture of the outcome. Sedative drugs are given with three objects in view:

(*a*) To relieve anxiety and so produce a more restful frame of mind. Here Sodium Amytal (60 mg. = gr. 1 t.d.s.) or phenobarbitone (15–30 mg. = gr. $\frac{1}{4}$–$\frac{1}{2}$ t.d.s.) are excellent, and should be given whenever anxiety is prominent. It is worth remembering that phenobarbitone may sometimes cause headache and depression, especially in older people.

(*b*) To ensure adequate sleep. For this purpose we have a wide range of hypnotics. The barbiturate group is generally satisfactory and Sodium Amytal (0·2–0·4 G. = gr. 3–6), Medinal (0·3–0·6 G. = gr. 5–10) or the shorter acting Seconal or Nembutal (0·1–0·2 G. = gr. $1\frac{1}{2}$–3) may be tried. Alternatively chloral hydrate given as Syrupus Chloralis B.P.C. (2–8 ml. = ℥ss 30–120) is a safe and effective preparation, although it is somewhat irritating to the gastric mucous membrane and should be avoided if nausea is prominent. If a quick and certain action is required, an intramuscular injection of paraldehyde (3–7 ml.) is excellent.

(*c*) To relieve pain or extreme distress.

The drugs described above allay mental anxiety and produce sleep but they have little effect upon pain in therapeutic doses. While this is not a typical feature of congestive cardiac failure, it may occasionally be severe in patients with acute liver congestion or if some complication such as pleurisy or pulmonary infarction is present. Furthermore, these drugs may have little effect upon the acute distress of severe dyspnoea. In such circumstances, the administration of a member of the opiate group is essential. A subcutaneous injection of morphine (10–20 mg. = gr. $\frac{1}{6}-\frac{1}{3}$) is most satisfactory. Sometimes this may cause vomiting, and very occasionally serious respiratory depression. If respiratory failure follows the administration of morphia it should be treated by injections of nalorphine (Lethidrone) or nikethamide (p. 585). Pethidine hydrochloride (100 mg. by injection) or Physeptone (10 mg. by injection) are two synthetic members of the opiate group which may be of considerable value. These drugs are rather less potent than morphia, but have comparatively little effect upon the respiratory centre and are therefore safer. They are also less likely to cause vomiting. As a general rule if severe pain or respiratory distress is present, morphia should never be withheld, for the beneficial effect to be expected far outweighs the small risk involved. If these symptoms are slight, however, pethidine or Physeptone will probably prove a safer and equally satisfactory substitute.

Alcohol taken in the form of whisky or brandy (1–2 oz.) has a valuable therapeutic action by allaying anxiety in patients who are used to it. Indeed in chronic alcoholics the administration of regular doses is essential during the acute stages of congestive failure, as sudden withdrawal on entering hospital may precipitate an attack of delirium tremens.

DIGITALIS. *Actions.* This is the most important of all the cardiac drugs, and its actions will be briefly summarised.

(1) It increases the strength of contraction of the heart muscle and it is to this that much of its beneficial effect is due.

(2) It reduces conduction in the A/V bundle and in large doses may cause complete heart block. Thus it reduces the number of impulses bombarding the ventricles in patients with auricular fibrillation.

(3) Its action on the carotid body causes vagal stimulation and thus slows the heart.

(4) It decreases the refractory period of cardiac muscle, and so increases its excitability. This may be dangerous, for large doses may cause ventricular fibrillation, especially if the ventricles are already damaged by disease.

In rapid auricular fibrillation its effect is dramatic, but in congestive failure where the cardiac output is low, the improvement following its administration may be almost as great. This is mainly due to its direct action upon the muscle, the reduction in heart rate playing a minor part. It is now believed that the reduction in the venous pressure seen in this type of case is the result of the improvement in the heart's action and is not a direct effect of the drug.

Digitalis is dangerous in the presence of paroxysmal ventricular tachycardia, as ventricular fibrillation may follow its use. It should also be used with caution after myocardial infarction.