CARDIAC ARRHYTHMIAS

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THE term "cardiac arrhythmia" indicates that an irregularity of heart-beat is present. Many states of disordered heart-beat, however, are associated with an entirely regular rhythm, for example paroxysmal atrial tachycardia. In the description that follows I propose to limit myself to cardiac arrhythmias presenting as an emergency requiring prompt recognition and immediate action. This will be preceded by a short account of the normal conduction pathway and the ætiology of cardiac arrhythmias.

Normal Conduction Pathway

The study of the conduction tissue of the heart began when Purkinje (1845) demonstrated the terminal part of the system in the heart of a sheep. Paradoxically, the pacemaker was the final structure to be identified (Keith and Flack, 1907). Lewis (1925) demonstrated that transmission between the sino-atrial and atrio-ventricular nodes occurred through the atrial muscle and not through specialised tissue. The function of the atrio-ventricular conduction system was established by Hering (1904) who damaged the bundle of His in dogs, and the effects of interruption of the branches of the bundle were demonstrated by Eppinger and Rothberger, (1910).

The normal impulse spreads from the sino-atrial node through the atrial muscle at about 1,000 mm. per second and produces the P wave of the electrocardiogram. Most of the P-R interval is taken up by the impulse spreading through the atrio-ventricular node, a structure with a long refractory period, incapable of rapid conduction (22 mm. per second). Once in the branches of the bundle of His and Purkinje fibres the passage of the impulse is rapid (4,000 mm. per second). Contraction occurs as the impulse enters the ventricular muscle and is followed by the resting state.

Ætiology of Cardiac Arrhythmias

Any abnormal state affecting the myocardium and any process interfering with the normal action of the valves of the heart may be responsible for an arrhythmia. In addition, many metabolic disturbances may present in this way, and in this connection the Na⁺ and K⁺ interchange across the myocardial cell

membrane, Ca⁺⁺ and Mg⁺⁺⁺ concentration are all of great importance.

Of great interest and clinical significance is the fact that many arrhythmias occur in hearts that are otherwise normal—sino-atrial block, premature beats, atrial tachycardia (35 per cent of these last attacks occur in normal hearts). Similarly, both atrial fibrillation and atrial flutter may occur in the absence of any recognisable heart disease.

Katz and Pick (1956) in a study based on 50,000 patients observed over 25 years, found that the most common arrhythmia was ectopic beats (14.5 per cent), then atrial fibrillation (11.7 per cent), heart block (4.1 per cent), atrio-ventricular dissociation (1.4 per cent) and atrial flutter (0.5 per cent).

Paroxysmal Tachycardia

This term was introduced by Bouveret (1889) and indicates rapid regular ectopic beats arising from one focus which may be atrial, nodal or ventricular. One special type is associated with the pre-excitation of the Wolff, Parkinson and White syndrome.

With all types the onset is usually abrupt and heart rates of between 110 and 250 per minute are usually found. Symptoms vary from none, to breathlessness, palpitations, dizziness, syncope, præcordial pain and anxiety. Polyuria (Wood, 1961) is frequently found during the attack. The effects of the attack depend on the functional state of the myocardium and its ability to increase cardiac output with the increase in rate. Normal hearts usually stand the period of tachycardia with no untoward effects but where the heart is abnormal cardiac failure may follow.

Attacks usually last for only a few hours but vary from seconds to weeks. The end is usually abrupt. Frequently the diagnosis is retrospective and depends on an accurate history. The exact nature of the arrhythmia can usually only be determined by an electrocardiogram during an actual attack.

Atrial Fibrillation

This may occur—

1. As a paroxysmal rhythm, or
2. As an established rhythm.

FIG. 1.—Atrial Fibrillation. ECG Lead 2.

It occurs most frequently in association with rheumatic heart disease (35 per cent), coronary artery disease (32 per cent) systemic hypertension (17 per cent), thyrotoxicosis (8 per cent), cor pulmonale (5 per cent). Congenital heart disease is an unusual cause of atrial fibrillation, of which atrial septal defect is the commonest, but this rhythm may also be found in other forms of septal defect especially if pulmonary hypertension is associated. Pericardial disease is a numerically small but functionally important precipitating cause of atrial fibrillation.

Atrial fibrillation may occur in the absence of any detectable organic heart disease and under these circumstances may precipitate heart failure if the ventricular rate is very rapid for a prolonged time.

Prinzmetal, Corday, Brill, Sellers, Oblath, Flieg and Kruger (1950) were able to show by high speed cinematography that in this arrhythmia two basic types of atrial contraction may be filmed. In the first minute irregular contractions at 10,000 to 40,000 per minute involve a small area of the atrial wall. In addition, larger wave-like contractions sweep across the atria at 400 to 600 per minute. The true mechanism underlying the arrhythmia is still under dispute but the most widely accepted theory suggests that it is initiated by a unifocal stimulus which is replaced by multiple foci from many portions of the atrial muscle. Irregular re-entries occur arising from anywhere within the atria. If this is so, a true mother circus movement does not exist.

Clinically the hallmark of the arrhythmia is the irregularity of ventricular rate. This has always been considered in the past as a chance occurrence. Recent studies, however, by Soderstrom (1958) suggest that in any one patient the nature of the ventricular response can be predicted.

The diagnosis depends on the recognition of a completely irregular cardiac rhythm. Because of the absence of organised atrial

contraction the jugular venous pulse shows no "a" wave and as in any arrhythmia careful inspection of the form of the venous wave will be rewarding. Carotid sinus compression may slow the ventricular rate but this is only temporary and in any case the irregularity is unaffected.

The rhythm should be differentiated from multiple premature beats arising from different foci, atrial flutter with a varying degree of atrio-ventricular block and any form of atrio-ventricular heart block with an irregular response. Any patient already receiving digitalis in whom a rapid irregular rhythm is found should have the possibility of paroxysmal atrial tachycardia with atrio-ventricular block seriously considered and the differentiation of this from atrial fibrillation will be considered later.

The effect of exercise (where the clinical state allows) should always be tried. In atrial fibrillation the rhythm becomes more irregular as the rate increases but with many other irregular rhythms this becomes less marked as the rate increases. This is specially useful for differentiating multiple premature beats which tend to disappear when heart rates exceed 120 per minute.

As with any arrhythmia an electrocardiogram will establish the diagnosis (Fig. 1).

Treatment

A. To control the ventricular rate:

Digitalis—This is the drug of choice and should always be used where the ventricular rate is raised.

If heart failure has resulted from a rapid ventricular rate and where no digitalis has formerly been given, digitalisation should be speedily performed. The approximate oral digitalising dose for an adult is 2 to 5 mg. of digoxin. 1.0 mg. may be given orally followed by 0.5 mg. every six hours until a desired effect has been achieved.

For extreme urgency 1.0 mg. digoxin may be given intravenously followed by a further

1.0 mg. in 3 hours. Thereafter 0.5 mg. may be given in three or six hours depending on the clinical state. This dose can be repeated at that time interval as needed but it must be stressed that intravenous dosage is not without risk and the instances demanding intravenous therapy are uncommon.

In patients with nausea or in whom oral therapy is not possible intramuscular digoxin should be used.

Where there is no urgency 0.5 mg. digoxin may be given orally and 0.25 mg. 8 hourly thereafter until the ventricular rate is slowed.

A maintenance dose will then be needed, usually 0.25 mg. 12 hourly but varying from 0.25 mg. on alternate days to 0.25 mg. 8 hourly in individual patients.

Occasionally digoxin, even in adequate dosage, fails to control the ventricular rate. This may occur in hypokalaemia or if thyrotoxicosis is present and specific treatment for these must always be given when needed. One of the presenting signs of digitalis overdosage is an uncontrolled ventricular rate and this should always be considered where the ventricular rate remains rapid or increases despite large doses of digoxin.

Occasionally patients respond better to one form of digitalis preparation than another. This is not a usual occurrence but equivalent doses of some of the commonly available preparations are:

Digoxin, 0.25 mg.

Powdered digitalis leaf, 60 mg.

Digitoxin, 0.075 mg.

Lanatoside C, 0.25 mg. (excretion more rapid than digoxin).

B. *To restore sinus rhythm.*

1. *Drug therapy.* Quinidine is the drug of choice. If quinidine is used alone it may allow a 1:1 ventricular response at a dangerously high rate. This can be prevented by preliminary digitalisation.

An adequate blood level (4.0 to 10.0 mg. quinidine per litre) is essential for successful version.

A satisfactory regime for oral quinidine is 0.3G two hourly for three doses on the first day, 0.6G two hourly for three doses on the second and 0.9G similarly on the third. Great individual response to quinidine is found and where facilities exist a blood examination to check the level obtained will be useful.

Hypersensitivity to quinidine is not uncommon, symptoms varying from skin manifestations to fever, nausea and collapse. Cardiac

standstill, rapid ventricular rates, ventricular ectopic beats or tachycardia may all occur and patients receiving quinidine in high dosage are better treated in hospital, preferably with constant oscilloscopic monitoring of the electrocardiogram. Facilities for immediate resuscitation should always be at hand.

A higher incidence of systemic embolisation may occur at the time of attempts at version of atrial fibrillation to sinus rhythm and anticoagulant therapy should be considered in every case. If used, a therapeutic level should have been present for at least 14 days before the attempt. Following successful mitral valvotomy or closure of an atrial septal defect and in cases of treated thyrotoxicosis anticoagulant therapy is probably not needed.

Attempts to restore sinus rhythm should always be made where there is no underlying heart disease responsible for atrial fibrillation but results may be disappointing (McDonald and Resnekov, 1964). Similarly, in successful post-operative mitral valve disease and treated thyrotoxicosis sinus rhythm should be aimed at. Likewise, atrial fibrillation precipitated by infection should be brought into sinus rhythm once the infection has responded to treatment.

Once atrial fibrillation develops in the natural history of a disease process, especially mitral valve disease, and where surgery is not contemplated attempts to restore sinus rhythm by any means are frequently unsuccessful and are not without risk, and should be avoided.

2. *Direct current shock*—this will be discussed in detail later in the paper.

Atrial Flutter

Like atrial fibrillation atrial flutter may occur in two forms, paroxysmal or established. It is far less common than atrial fibrillation; for every 15 cases of atrial fibrillation one of atrial flutter will be encountered. Nevertheless, it is not a rare cause of a sudden rapid arrhythmia that requires immediate treatment.

Atrial flutter is usually found in association with rheumatic heart disease, coronary artery disease, thyrotoxicosis and systemic hypertension and is less frequently encountered than atrial fibrillation in hearts that are otherwise normal. Atrial septal defect, especially post-operative, is the commonest associated congenital heart disease.

As in atrial fibrillation the arrhythmia depends on an irritable focus in the atrium which in an atrium in flutter beats at from 260 to 340 beats per minute. The rhythm is usually

FIG. 2.—Atrial Flutter. ECG Lead 2. An irregular ventricular response is shown.

FIG. 3.—Paroxysmal Atrial Tachycardia. ECG Lead V₁, P and T waves are superimposed.

regular, the ventricles responding to every second or third atrial impulse. By increasing vagal tone, as for example with carotid sinus compression, a greater degree of physiological block can be achieved. This is important diagnostically as carotid sinus compression causes a sudden temporary slowing of the heart rate which increases again as soon as the pressure is released.

Clinically the arrhythmia presents with a regular heart rate (but irregular ventricular response may be found) of between 120 and 220 per minute unaffected by exercise or respiration and with a varied intensity of first heart sound should the ventricular response be irregular (Harvey and Levine, 1945).

The clinical effects of the arrhythmia depend upon the ventricular rate. As long as this is less than 90/min. little fall-off in cardiac output occurs.

Atrial flutter has a very distinctive electrocardiographic appearance (Fig. 2). Atrial oscillations are seen, especially in standard leads 2 and 3 and in the præcordial leads V₁ and V₂. When atrial activity is in doubt, however, an œsophageal lead will show up the activity. In the usual type of atrial flutter inverted f (flutter) waves are present in standard leads 2, 3 and in aVF. The less common form presents with upright waves in these leads.

Usually there is a 2:1 or 3:1 ventricular response but occasionally atrial flutter occurs with a 1:1 conduction. Under these circumstances the very rapid ventricular rate constitutes a serious emergency.

Treatment:

1. Drug therapy.

(i) Digitalis—Adequate doses should be given. Oral digoxin is always to be preferred unless there is real clinical urgency. A satisfactory oral dose is digoxin 0.5 mg. 8-hourly which should be given to the point of toxicity or until atrial fibrillation, as shown on electrocardiogram, has occurred. Digoxin is then stopped and in at least one half of the cases sinus rhythm will follow.

(ii) Quinidine—this should never be used alone in cases of atrial flutter for it may allow the ventricles to keep pace with the atria and so result in a dangerous tachycardia.

In atrial flutter, therefore, its use should be reserved for those cases who have had their atrial flutter converted to atrial fibrillation but who have failed to come into sinus rhythm on withdrawing the digoxin. The dose to be used is as described under the treatment of atrial fibrillation and the same precautions and risks apply.

2. Direct current shock—see later.

In the small number of patients with atrial flutter who cannot be brought into atrial fibrillation as a preliminary to sinus rhythm a suitable maintenance dose of digoxin should be used to maintain a satisfactory ventricular rate. Alternatively direct current shock should be tried.

Paroxysmal atrial tachycardia

Here rapid regular beats occur from the same focus. The onset is sudden and the attack may last for a few seconds, hours or days before ending suddenly.

In a review of 350 cases of paroxysmal supra-ventricular tachycardia, Kissane, Brooks and Clark (1950) found that underlying heart disease was present as follows: none detected (34 per cent), rheumatic (34 per cent), arteriosclerotic (14 per cent), systemic hypertensive (3 per cent) and thyrotoxic (5 per cent). In the remaining 10 per cent, tachycardia was associated with a wide variety of other conditions.

The arrhythmia originates from a focus outside the sino-atrial node from which an impulse travels simultaneously in all directions and there is no evidence at present that it results from a circus movement.

In most cases the ventricular rate varies between 120 and 240 per minute although much faster rates have been recorded. In the normal heart an increase in cardiac output occurs with rates up to 170 to 180 per minute. Thereafter the stroke volume is reduced and cardiac output falls. In previously diseased hearts this fall-off occurs at slower heart rates so that congestive failure may be produced in prolonged tachycardia whilst with rapid heart rates, even shortlived, breathlessness, syncope and angina may all occur. Polyuria during the attack (Wood 1961) is an interesting association.

Paroxysmal atrial tachycardia may be suspected in the presence of a rapid regular heart rate or retrospectively following a characteristic history. The arrhythmia should be differentiated from nodal tachycardia, paroxysmal atrial tachycardia with atrio-ventricular block, atrial flutter, ventricular tachycardia (if bundle branch block is present in addition) and sinus tachycardia. Carotid sinus compression may have no effect or the heart rate may be temporarily halved or rarely, the arrhythmia may be cured.

The electrocardiogram (Fig. 3) shows P waves similar to those in atrial premature beats but these may be frequently superimposed on a preceding T wave. The rhythm is regular. The QRS complexes are normal in shape unless bundle branch block be present or a widening shown due to aberration.

Treatment

1. *During the attack*

(i) Cholinergic drugs and reflex vagal stimulation. These act by prolonging the refractory period of the sino-atrial node and atrio-ventricular node.

(a) Carotid sinus compression (one side at a time), ocular pressure (similarly) and the Valsalva manoeuvre may all be helpful.

(b) Digitalis—This is the drug of choice in paroxysmal atrial tachycardia. Full oral dosage as previously described is usually adequate and intravenous digitalisation should be reserved for the very rare case of genuine urgency.

(c) Prostigmine 0.5 to 1.0 mg. subcutaneously and acetylbetamethylcholine 2.5 to 60 mg. subcutaneously may terminate an attack. Untoward side effects include severe hypotension, urgent defaecation and micturition, and abdominal colic. Except for hypotension, these can be speedily ended by atropine sulphate intravenously which should be given only if essential. Cholinergic drugs should not be used in patients with a history of bronchial asthma.

(d) Sedation—heavy sedation may succeed where other measures fail.

(ii) Direct Current Shock—see later.

2. *Prevention of Attacks*

(a) Digoxin—in maintenance dosage, probably the drug of choice.

(b) Quinidine—0.3 to 1.0 G 8 hourly.

(c) Procaine amide—0.25 to 0.5 G 8 hourly.

(d) Sedation—regular mild sedation may help to prevent attacks.

In every patient with paroxysmal tachycardia particular attention must be paid to any underlying heart disease which is amenable to treatment and to the presence of heart failure or thyrotoxicosis. Even when all these have been excluded or dealt with satisfactorily there remains a small group with frequent attacks which respond poorly to the measures outlined. In these circumstances the outcome may be very serious unless the attack can be curtailed. Fortunately these instances are excessively rare.

Paroxysmal tachycardia with the Wolff-Parkinson-White Syndrome of pre-excitation.

Wolff, Parkinson and White (1930) described a condition which they called physiological heart block and which was characterised by a shortened P-R interval with widening of the QRS, (Fig. 4). The condition is probably due to accelerated conduction at the atrio-ventricular node (Prinzmetal and others 1950) and a characteristic delta wave is seen on the electrocardiogram for it is the first part of the vector of the QRS that is affected. An otherwise normal heart is present in over 70% and the abnormality is often intermittent.

Paroxysmal tachycardia may occur in 50% and the attacks are frequently related to effort.

Treatment is as for paroxysmal tachycardia unassociated with the Wolff-Parkinson-White

FIG. 4.—Wolff-Parkinson-White Syndrome. The P-R interval is short and the characteristic delta-wave abnormality of the QRS complex well shown. Positive deflections are shown over right ventricular precordial leads (Type A). This tracing is from a patient following a clinically-proven myocardial infarction and illustrates the difficulty of diagnosing a myocardial infarct in the presence of W.P.W. Syndrome. S-T segment and T wave changes may be part of the conduction abnormality and by themselves do not indicate myocardial disease.

syndrome but the tachycardia is often more resistant to therapy.

Paroxysmal atrial tachycardia with atrio-ventricular block

Although first described by Lewis (1909) and attributed to digitalis by MacKenzie (1911) this important arrhythmia received scant at-

tention until Lown and Levine (1955) emphasised again its relation to digitalis overdosage.

Clinically the arrhythmia is characterised by the development of a cardiac irregularity with ventricular ectopic beats in a patient receiving digitalis. Similarly an increase in heart rate, the development of a rapid regular

FIG. 5.—Paroxysmal Atrial Tachycardia with atrio-ventricular block. ECG Lead V₁. Atrial rate 215 per minute. Irregular ventricular response. Note the iso-electric line separating the P waves. Atrial fibrillation returned after discontinuing digoxin and diuretics and increasing the potassium intake.

FIG. 6.—Ventricular Tachycardia. ECG Oesophageal Lead. Clear evidence of dissociated atrial and ventricular activity is demonstrated.

rhythm when atrial fibrillation was present or an otherwise unexplained increase in heart failure in patients receiving digitalis should all lead the physician to suspect the development of the arrhythmia. It is particularly liable to occur in patients who have potassium depletion in the myocardial cell and in this connection mercurial and thiazide diuretics are potent potentiators of the arrhythmia. The arrhythmia may occur in an otherwise normal heart in the presence of digitalis overdosage and a lowered myocardial potassium as described by Oram, Resnekov and Davies (1960) and these authors also stress that as long as the arrhythmia is recognised and appropriate measures taken the prognosis is far better than assumed previously.

The electrocardiogram (Fig. 5) shows an atrial rate varying from 150 to 250 per minute with a varying degree of atrio-ventricular block (2:1, 3:1, 4:1 with occasional 1:1 response). Where digitalis is the cause, carotid sinus compression increases this block without affecting the atrial rate. The electrocardiogram shows P waves which are small, upright and peculiarly pointed and are usually separated by a short length of iso-electric line. Frequently other signs of digitalis over-

dosage are present clinically and confirmed on the electrocardiogram: for example, coupled ventricular ectopic beats and typical ST segment and T wave changes. Atrial flutter and atrial fibrillation may be recorded in a long strip of the electrocardiogram varying with the arrhythmia (Oram and others, 1960).

Treatment

A. *Of the acute attack.*

1. Where digitalis is the cause stop the drug forthwith.
2. Discontinue diuretic therapy and give additional potassium; 1G potassium chloride is equivalent to 13 m Eq. potassium and 26 m Eq. every four hours may be given. Where there is extreme urgency and assuming satisfactory renal function this amount may be given diluted intravenously.
3. Beta—adrenergic blockade. The efficiency of this group of drugs in stopping digitalis induced arrhythmias has been described recently. (Stock and Dale 1963).

B. *Prevention.*

Re-adjustment of digitalis and diuretic therapy with increased potassium supplements will usually prevent a recurrence.

FIG. 7.—Ventricular fibrillation. ECG Lead 2.

Ventricular Tachycardia

This is uncommon but is certainly found more frequently than previously thought. Although almost always associated with organic heart disease it infrequently does occur in the absence of any recognised disease.

The arrhythmia is rapid and the heart beat almost, but not quite regular. Cannon waves may be seen in the neck.

The electrocardiogram is usually characteristic but difficulty may arise in differentiating it from supraventricular tachycardia if the latter is associated with bundle branch block. An oesophageal lead may be very helpful, (Fig 6) for clear evidence of a dissociated atrial and ventricular activity may be obtained in this way.

Treatment

A. Drugs.

1. Quinidine—the drug of choice. Oral administration 0.3 to 0.6 G every three hours is to be preferred but where there is genuine need 0.6 G quinidine gluconate intramuscularly may be given every four hours.

In the very rare instances that intravenous administration is indicated 0.5 to 1.0 G of quinidine gluconate may be given diluted in 50 ml. 5% glucose over a period of 10-20 minutes under direct electrocardiographic control.

2. Procaine amide—0.5 to 1.0 G every two to four hours may be given orally, intramuscularly or intravenously. The intravenous administration is frequently followed by severe hypotension requiring urgent supportive measures.

B. Direct Current Shock—see later.

Despite a popular belief to the contrary congestive cardiac failure developing during ventricular tachycardia should be treated with digitalis unless there is good evidence that digitalis is the cause of the arrhythmia.

Ventricular Fibrillation

No effective ventricular systole is possible with this arrhythmia owing to the inco-ordinate type of ventricular contraction. Arterial pressure falls abruptly and death may occur within a few minutes. In the transient form, however, spontaneous recovery may occur. Frequently it is found as a terminal event in patients dying from heart disease. Irreversible cerebral damage may occur if the attack lasts longer than four minutes.

The electrocardiogram (Fig 7) is characterised by bizarre ventricular oscillations without any suggesting QRS complex or T wave.

Treatment

To be effective speed is essential.

1. Adequate oxygenation — mouth-to-mouth breathing with an adequate airway or an endotracheal tube with oxygen administered by bag inflation.
2. Closed chest massage—a firm support under the back of the patient is essential. In the accompanying record (Fig 8) a good brachial artery pressure pulse is maintained in a patient with ventricular fibrillation treated by external cardiac massage. A good pressure pulse does not of itself necessarily mean adequate flow, however.
3. Combating acidosis—intravenous sodium bicarbonate (1 ml. of 8.4 per cent $\text{NaHCO}_3 \equiv 1 \text{ mEq. HCO}_3^-$). Where large amounts are administered and up to 200 ml. may be needed to fully correct the acidosis the effects of a large infusion of Na^+ must also be remembered as renal function is often depressed at this stage.
4. Repeated doses of 1-5 ml. of 1/10,000 adrenaline intravenously.
5. Repeated doses of 5 ml. 1% calcium chloride intravenously.
6. Should normal sinus rhythm not develop within a short space of time external electrical

FIG. 8.—Ventricular fibrillation treated by external cardiac massage. Upper tracing—Brachial artery pressure pulse recorded with an indwelling cannula and pressure transducer. Lower tracing—Simultaneously recorded ECG Lead 2. Time markings in seconds.

NOTE.—With the onset of ventricular fibrillation pressure in the brachial artery falls to a mean level of 30 mm. Hg. External cardiac massage is followed by a brachial artery pressure of about 100 mm. Hg. within one second.

defibrillation with direct or alternating current should be attempted.

7. Once sinus rhythm has returned procaine amide 0.25-0.5G 6 hourly or slow infusion of 1G diluted in 540 ml. of 5 per cent dextrose may prevent further attacks.

8. Supportive measures for treating any cardiac failure.

It is doubtful whether there is still a place for internal cardiac massage unless cardiac tamponade is present or external electrical defibrillation fails despite the measures outlined above. If attempted it should be done only in an operating theatre or equivalent as a sterile procedure.

Stokes-Adams Attacks

In these attacks syncope results from ventricular standstill and constitutes a real emergency. Although they occur in about half the cases of complete atrio-ventricular dissociation they are especially common when partial heart block becomes complete. Loss of consciousness is abrupt and is not preceded

by any warning. Twitchings and convulsions follow unless the ventricles begin to beat quickly and recovery after 10 seconds is unusual. A vivid flush accompanies return to consciousness should the ventricles begin to beat. The atria continue to beat during the attack. Paroxysmal ventricular tachycardia or fibrillation may be associated and require urgent treatment.

Treatment

A. *Adequate airway and oxygenation.*

B. *Drugs:*

1. Adrenaline 1-5 ml. 1 in 10,000 solution intravenously.
2. Iso-prenaline 10 to 20 mg. sublingually every 3 to 6 hours to increase ventricular rate.
3. Long acting preparations of iso-prenaline, for example, "Saventrine" may be taken orally to a total of between 200 and 300 mg. per day.
4. Ephedrine 30 to 60 mg. three times a day has a time-honoured place in the management of these cases but it is

FIG. 9.—The Lown Cardioverter. An oscilloscope for monitoring the ECG Lead II and a tachometer with audible warning systems, are provided. In addition, provision is made for external or internal pacing should this be needed. Approximate dimensions are 22 x 28 x 12 in.

doubtful whether it is more effective than iso-prenaline.

5. Steroids—adrenal steroids accelerate atrio-ventricular conduction (Prinzmetal and Kennamer 1954) and prednisone up to 60 mg./day has been given, reducing slowly over one week. Results have been disappointing.

C. Artificial pacemaking.

This technique is described fully in another article in this issue—see R. W. Portal: Cardiac Arrest (p. 370).

Direct Current Shock

Although apparatus for both alternating and direct current shock was available at the end of the last century it is largely due to the work of Lown and his colleagues (1962 a, b) that this form of treatment is now accepted in the treatment of ventricular and supraventricular arrhythmias.

With the Lown Cardioverter* (Fig. 9) a capacitor of 16 microfarads is charged by variable DC transformer. The capacitor dis-

* American Optical Company Ltd.

FIG. 10.—Atrial Fibrillation treated with Direct-current Shock. Upper tracing—Pressure pulse in the abdominal aorta recorded from a catheter passed percutaneously into the femoral artery. Lower tracing—Simultaneously recorded ECG Lead 2. Time markings in seconds.

Atrial fibrillation is present at the beginning of the tracing. Following a discharge of 150 watt seconds phased to occur synchronously with the R wave, the ECG is lost (electrical interference) for 3.8 seconds. The pressure pulse in the aorta shows a short run of ectopic beats. With the return of the ECG sinus rhythm with a prolonged P-R interval is shown and is followed by nodal beats. Regular sinus rhythm then follows.

charges through an inductance coil and through the resistance of the body to give an under-damped discharge pattern over 2.5 m.seconds.

Electrodes are applied to the posterior and anterior surfaces of the chest when less energy is required to bring about version or both electrodes are applied anteriorly. Direct current shock may also be given by direct application of suitable electrodes to the surface of the heart. The energy required is from 25 to 400 watt seconds and to avoid the vulnerable phase of ventricular repolarisation is phased to occur synchronously with the R or S wave of the electrocardiogram. In this way the risk of ventricular fibrillation is avoided. The treatment is given under a light general anaesthetic.

Anti-coagulant therapy need be given only to those considered to be at high risk of embolisation.

The effect of the direct current shock is to depolarise the whole heart and to temporarily extinguish all electrical activity. This allows the sino-atrial node to resume as the dominant pacemaker.

In successful cases sinus rhythm may occur within one beat or be preceded by a short period of intervening rhythms (O'Brien, Resnekov and McDonald, 1964). (Fig. 10).

The treatment is remarkably successful even in cases previously resistant to version by quinidine or digitalis to toxic levels (McDonald, Resnekov and O'Brien, 1964). Complications are few.

The indications for direct current shock are essentially the same as for version by drugs. Atrial fibrillation, atrial tachycardia (paroxysmal or established), atrial flutter,

ventricular tachycardia and ventricular fibrillation (in the latter a synchronised shock is of course not used) may all respond to treatment by this means.

In a series of 75 consecutive cases of atrial arrhythmias associated with a wide variety of underlying heart disease in which successful version was achieved in 89 per cent. McDonald and Resnekov (1964) suggest that direct current shock is the treatment of choice in atrial arrhythmias of post-operative atrial septal defect, and post-operative mitral valve disease and that it may well become the treatment of choice in other forms of atrial and ventricular arrhythmias as well.

Direct current shock will not maintain sinus rhythm longer than version by drugs so that repeated attempts are not indicated. Furthermore it should be attempted only when persisting sinus rhythm can reasonably be expected.

Summary

The clinical and electrocardiographic features of cardiac arrhythmias which may present as emergencies are described.

The treatment of choice in each case is indicated and a plan of subsequent management outlined.

A brief description of the technique and indications for direct current shock in the treatment of atrial and ventricular arrhythmias is given.

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REFERENCES

- BOUVERET, L. (1889): Concerning Essential Paroxysmal Tachycardia, *Rev. Med. (Paris)*, **9**, 753.
- EPPINGER, H., and ROTHBERGER, J. (1910): Über die Folgen der Durchschneidung der Tawaraschen Schenkel des Reizleitungs-systems. *Schr. f. klin. Med.*, **70**, 1.
- HARVEY, W. P., and LEVINE, S. A. (1948): Changing intensity of the first sound in auricular flutter, an aid to diagnosis. *Amer. Heart J.*, **35**, 924.
- HERING, H. E. (1905): Nachweis, dass das His'sche Uebergangsbündel Vorhof und Kammer des Säugethiers herzens functionell verbindet. *Arch. ges. Physiol.*, **108**, 267.
- KATZ, L. N., and PICK, A. (1956): "Clinical Electrocardiography", Part I. The Arrhythmias. Philadelphia: Lea and Febiger.
- KEITH, A., and FLACK, M. W. (1907): The form and nature of the muscular connections between the primary divisions of the vertebrate heart. *J. Anat. Physiol.*, **41**, 172.
- KISSANE, R. W., BROOKS, R., and CLARK, T. E. (1950): Relation of supraventricular paroxysmal tachycardia to heart disease and the basal metabolic rate. *Circulation*, **1**, 950.
- LEWIS, T. (1909): Paroxysmal Tachycardia, *Heart*, **1**, 43.
- (1925): "Mechanism of Graphic Registration of the Heart-beat". 3rd ed. London: Shaw.
- LOWN, B., and LEVINE, H. D. (1958): "Atrial Arrhythmias, Digitalis and Potassium". New York: Landau and Berger.
- , AMARASINGHAM, R., NEUMAN, J. (1962a): New method for terminating cardiac arrhythmias: Use of synchronised capacitor discharge. *J. Amer. med. Ass.*, **182**, 548.
- , NEUMAN, J., AMARASINGHAM, R., BERKOVITZ, B. V. (1962b): Comparison of alternating current with direct current electroshock across the closed chest. *Amer. J. Cardiol.*, **10**, 223.
- MACKENZIE, J. (1911): Digitalis. *Heart*, **2**, 273.
- MCDONALD, L., and RESNEKOV, L. (1964): Atrial Defibrillation. Proceedings of the Symposium on Cardiac Resuscitation and Pacing. Glasgow, 1964.
- , RESNEKOV, L., and O'BRIEN, K. (1964): Direct current shock in the treatment of cardiac arrhythmias resistant to drugs. *Brit. med. J.* in the press.
- O'BRIEN, K., RESNEKOV, L., MCDONALD, L. (1964): Direct current countershock in the treatment of cardiac resistant arrhythmias. *Brit. Heart J.*, **26**, 137.
- ORAM, S., RESNEKOV, L., DAVIES, P. (1960): Digitalis as a cause of paroxysmal atrial tachycardia with atrioventricular block. *Lancet*, **ii**, 1402.
- PRINZMETAL, M., CORDAY, E., BRILL, I. C., SELLERS, A. L., OBLATH, R. W., FLIEG, W. A., and KRUGER, H. E. (1950): Mechanism of the auricular arrhythmias. *Circulation*, **1**, 241.
- , and KENNAMER, R. (1954): Emergency treatment of cardiac arrhythmias. *J. Amer. med. Ass.*, **154**, 1049.
- PURKINJE, J. E. (1845): Mikroskopische Neurologische Beobachtungen. *Arch. Anat. Physiol. wiss. Med.*, **281**.
- SODERSTROM, N. (1958): The diagnosis of partial atrio-ventricular block and Adams-Stokes syndrome in patients with auricular fibrillation. *Cardiologia (Basel)*, **33**, 397.
- STOCK, J. P. P., and DALE, N. (1963): Beta-adrenergic blockade in cardiac arrhythmias. *Brit. med. J.*, **1**, 1230.
- WOLFF, L., PARKINSON, J., WHITE, P. D. (1930): Bundle-branch block with short P-R interval in healthy young people prone to paroxysmal tachycardia. *Amer. Heart J.*, **5**, 685.
- WOOD, P. (1961): Polyuria in paroxysmal tachycardia. *Brit. Heart J.*, **23**, 457.