CARDIAC EMERGENCIES * (Part 1).**By CLIFFORD HOYLE, M.D., M.R.C.P.**

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Cardiac emergencies have a general interest and importance for there is no branch of practice in which they may not arise, and there are almost always peculiar difficulties facing those who have to deal with the emergency. Quite often, for instance, nothing is known about the medical history of the patient, and there is little time or opportunity to find this out during the exigencies of the attack. The distress and anxiety of both patient and onlookers often makes a coherent account of the emergency itself difficult, if not impossible to obtain. Physical examination can rarely be complete, and if we prefer that it should be, before taking steps in treatment, we are apt to be reminded that "the patient dies while the physician sleeps." Then again we are almost always deprived in these patients of the help of special investigations at the time, for they are either not available or too difficult to apply in a gravely ill subject. All these difficulties obstruct a rapid diagnosis, though actually it can be made in the great majority of cases.

To do this we need to know what are the essential facts upon which diagnosis depends in a cardiac emergency, or as Oliver Wendell Holmes remarks,

"If you must listen to his doubtful chest,
Catch the essentials, and ignore the rest . . .
So of your questions: don't in mercy, try
To pump your patient absolutely dry."

I should emphasize to begin with, the importance of concentrating upon the first, and generally the leading, symptom of the attack. All cardiac emergencies present clinically in one of four ways, either as an attack of syncope, or of palpitation, or of dyspnoea, or of pain. It is the predominance of one or other of these features during the early phases of the emergency that so often allows a rapid opinion to be formed about the probable cause. Details of the past history, and of other and minor features of the attack itself, can safely be left until later, for they more often confuse than clarify the issue; but if exception is to be made to this plan I should place most value upon enquiry into the patient's knowledge of past heart disease, or of similar attacks.

Knowing the central symptom of the attack we can proceed to the examination. The respirations, colour, state of consciousness will have been judged already; the rate and rhythm of the pulse, or of the heart sounds, are the next most important features. The rate and rhythm are of far more value than the actual volume of the pulse, for a rapid pulse is often a thready one, and is not necessarily of serious omen on that account. A "thin," "feeble," or so called "weak" pulse may imply no more than the change in volume that commonly occurs whenever the heart's rate rises above a certain level, for the shortening of diastole then leads to a reduced output that is reflected in this way. The rate and rhythm of the heart, the condition of the arterial walls, and a record of the blood pressure give the facts required from this part of the examination; and the presence or absence of cardiac enlargement then comes next in importance. Knowing the limitations of clinical methods we shall be satisfied to detect gross enlargement only, for there is

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no point in consuming time over a doubtful enlargement when using clinical methods that we now recognize to be fallacious for this purpose.

Cardiac murmurs are helpful only when they are characteristic of a particular valvular defect, such as the presystolic murmur or late diastolic murmur of mitral stenosis, the diastolic murmur of aortic incompetence, or the harsh systolic murmur of aortic stenosis, with its attendant thrill. Others are apt to mislead, and are often better ignored. Next in order comes the search for signs of congestive heart failure, the distended and pulsating veins in the neck, crepitations and râles in the lungs, especially at the bases, enlargement of the liver with overlying tenderness and rigidity, and perhaps œdema of the legs.

Such a history and examination should have provided ample evidence upon which to say whether we are dealing with a cardiac emergency or not, and in most cases will allow the kind of emergency present to be accurately recognized. I propose now to discuss each variety according to the simple plan suggested, and based upon the leading symptom.

Cardiac Syncope.

Cardiac syncope, producing unconsciousness within ten seconds, is a rare event. It has to be distinguished from other, and commoner, sources of unconsciousness unconnected directly with the heart.

The most important of these are epilepsy, cerebral hæmorrhage and thrombosis, auditory vertigo, a sudden and severe internal hæmorrhage, postural faintness, and vaso-vagal attacks. As a group these can be separated from true cardiac syncope by the fact that the pulse rhythm remains normal and the rate not markedly affected, except in a severe hæmorrhage. Clonic movements, tongue biting, and evacuation of the bladder are very exceptional in cardiac syncope, common in epilepsy. In the cerebral vascular accidents there is no pallor, and often stertor and paralyses, and in auditory vertigo a marked sense of rotation at the onset which is characteristic, and never occurs in cardiac syncope.

A severe hæmorrhage leads to striking pallor and to a rapid pulse rate which rises steadily almost from minute to minute. Postural faintness, the commonest cause of transient unconsciousness in patients over forty, is generally found in those of sedentary habit, and the attack comes on most often on rising to the erect position. A recent full meal and a stuffy room commonly predispose. The cause of these attacks is a poor vasomotor tone or lack of muscular tone in the abdominal wall, exposing the subject to faulty distribution of blood with changes of position. In the attacks there is pallor, a moderate rise in the pulse rate, and a moderate fall in blood pressure. Such attacks are quite harmless, and can often be prevented by a healthier life and an abdominal binder or belt. They are more commonly mistaken for the minor attacks of unconsciousness that occur with cerebral arterial disease than for a major cardiac emergency; for short of an actual cerebral thrombosis elderly patients may have attacks of transient unconsciousness from atheroma of the cerebral vessels. Such attacks occur generally along with hypertension, and the pulse remains normal in both rate and rhythm, there is neither pallor nor sweating, whilst there is often headache, loss of memory or paresis afterwards.

Another cause of unconsciousness, vaso-vagal attacks, first described by Gowers in his book "The Borderland of Epilepsy," account for the majority of faints in younger people. Poor health and an overcrowded room tend to bring

them on. As they can occur of course in those already subject to some form of heart disease such faints may be wrongly attributed to this. Emotion and pain, or a sudden surprise often bring on an attack, with rapid unconsciousness but no sense of rotation; often there is a peculiar sinking feeling in the abdomen, and also retching. The subject becomes pale especially about the face, less so in dependent parts, the heart rate falls to 50 or less, the blood pressure falls also, respirations become sighing and slow, and there is a profuse sweating. Such an attack presents the features of vagal stimulation combined with vaso dilatation, and is believed to be due to the reflex disturbance of some central mechanism. Vaso-vagal attacks nearly always occur when the subject is erect; this, and the profuse sweating, are often crucial in diagnosis, and separate them from postural faints.

Here we must leave those common conditions which are most likely to give rise to difficulty in dealing with cardiac emergencies that cause syncope. In the latter there is always disturbance of the heart's rate or rhythm which gives the clue to the cause of the attack. Ventricular arrest, ventricular fibrillation, and one of the abnormal rhythms with a very high ventricular rate are the three possible explanations of cardiac syncope and the only three. Leaving aside those cases with sudden death due to some gross accident to the heart such as a massive coronary occlusion, or rupture of the heart or of a diseased valve, and those examples of sudden ventricular arrest due to plugging of the aorta or main pulmonary artery with a large thrombus, we have to deal with few conditions which can actually lead to true cardiac syncope; ventricular arrest from heart block, and ventricular fibrillation in the anæsthetic catastrophies are the most common. Abnormal rhythms with a very high ventricular rate sufficient to induce syncope at the onset of an attack are rare, but this can happen with auricular flutter and with ventricular paroxysmal tachycardia. This group is distinguished from the two former by the presence of heart sounds at the rapid rate.

Taking those cases with ventricular arrest as the cause of the syncopal attack the majority are due to myocardial disease from coronary atheroma, the main auriculo-ventricular bundle or one of its branches having been affected. Thus it is in elderly individuals with other evidence of arterial disease that this type of attack occurs most often—the Adams-Stokes attack of heart block. Rarely it happens that an acute infection of some kind, more especially diphtheria, leads to the same kind of thing. In the attack the ventricular arrest can be partial, when the rate is usually below 20 a minute, or complete, in which case the attack is fatal unless the beat is restored within a minute or two. In both these groups of cases the fact that the auricles continue to beat at their normal rate can be told by watching the jugular pulsation in the neck; this combination of a normal auricular rate, so observed, with a very slow or absent ventricular beat as judged by pulse or heart sounds, is diagnostic of auriculo-ventricular block. Adams-Stokes attacks are thus recognized with ease except in those stout individuals in whom the neck veins are not easily seen. After the attack the heart rate will remain permanently slow if the block affects the main A-V bundle; if the rate returns to normal we are dealing with a block of one bundle only as a rule, and there is then quite commonly a double first sound to be heard at the cardiac impulse. A source of error in dealing with these Adams-Stokes attacks is to meet with vaso-vagal attacks in a subject with a simple bradycardia; the character of the faint with sweating and the pulse present at a rate over thirty during unconsciousness should decide between the two, but if the attack has not been seen an electrocardiogram will be the only means of deciding.

Another, but very rare, form of heart block may produce attacks of unconsciousness. This is the benign sino-auricular block, thought to be due to vagal action. In an attack the whole heart is arrested, just as lesser grades cause dropped beats. These may suggest the condition, as does the absence of any venous pulsation in the neck while the attack is on. An electrocardiogram taken in an attack settles the point.

Ventricular fibrillation, the other cause of fatal cardiac syncope, is so serious because there is no treatment known that appears to have any certain chance of restoring the beat. It accounts for some examples of sudden death in heart disease of various kinds, especially those with myocardial changes due to atheroma, but its major importance in practice is during anæsthesia. It is the condition which makes chloroform still the most dangerous of anæsthetics, a danger which is all too often met in perfectly healthy individuals. Modern anæsthetics such as evipan, have fortunately put behind us the day when such an individual lost his life over some quite minor operation for which chloroform had been given. The exact risk is difficult to compute but it is probably in the region of 1 in 3,500: though this sounds a small risk, much smaller than many surgical risks, our total inability to forecast when it is likely to come makes it a thing with which in regard to surgical risks we are often in the dark. In spite of all its advantages as a volatile anæsthetic I think that there is a very good case to be made out for the total abolition of CHCl_3 on the score of this cardiac risk. Once it occurs the chance of survival is minute; atropine is valueless, only serving to restore the heart's action in the far less dangerous vagal arrest, and the usual intra-cardiac adrenalin is only likely to make things worse by making any chance of a spontaneous restoration of the normal beat even more unlikely. Cardiac massage and artificial respiration are the only measures which offer a chance of success.

The third group of cases is that in which syncope occurs at the onset of some form of abnormal rhythm with tachycardia. Actually these cases will almost always have palpitation as the first symptom so that they are included with that group of cardiac emergencies. There are however one or two points to note here.

Auricular flutter is the most likely form of abnormal rhythm with a high rate to produce a syncopal attack, and it does so when the ventricles spontaneously assume the same rate as the auricles, i.e., a 1:1 flutter. Ordinarily in flutter the ventricles respond only to each second or third or fourth beat of the auricles because the auricular rate, 260 to 320, is so high that the ventricle is literally unable to keep pace. Occasionally however, the ventricles suddenly take up the full auricular rate; diastole of the ventricles is then so short that filling becomes grossly inadequate, the output drops quickly, and the subject loses consciousness. If the original auricular flutter was accompanied by a relatively slow ventricular rate, as can happen, i.e., 4:1 flutter, the pulse rate before and after the attack will be apparently normal in rate, and during the attack will rise suddenly to anything between 260 and 320 beats a minute. If the attack has not been seen we have the anomaly of a patient with sudden faints, none of the stigmata of an extra-cardiac cause, and a normal pulse rate with regular rhythm between attacks, and perhaps unexplained breathlessness. These cases are generally regarded as "neurotic" with postural or vaso-vagal faints, but a careful history of the attacks should exclude the latter. Only an electrocardiograph or polygraph tracing can reveal the true cause of the trouble, auricular flutter with a varying grade of ventricular response to the high auricular rate.

Turning now to prognosis and treatment in the syncopal cardiac emergencies, ventricular fibrillation has already been dealt with, and auricular flutter will be considered again later. In heart block the prognosis varies widely, but the majority of patients are dead within two years of the first seizure, and we have to remember that the first one may on occasion be fatal. In considering the individual attack, danger is often already past by the time the medical attendant arrives except in those patients who may have one attack after another with transient consciousness between. Here the danger is acute. Treatment is with one of two remedies, atropine or adrenalin. Atropine should always be tried if the attack is accompanied by a very slow ventricular rate, for some of these cases have the attack from an increase of vagal tone that can be abolished by this drug. One-sixtieth to one-thirtieth of a grain given intravenously, the vein then being massaged towards the heart, should be quickly successful in raising the ventricular rate and abolishing the unconsciousness if this is the correct explanation. When it fails, or when there is complete arrest of the ventricle, adrenalin should be given intravenously or into the heart in doses of 5 to 15 minims of the 1 : 1,000 solution. Repeated injections are sometimes required over a period of hours, and the drug has some effect, though not a marked one, in preventing future attacks if it is given regularly, say four hourly or three times a day. Ephedrine, gr. $\frac{1}{4}$ or $\frac{1}{2}$, is sometimes used in the same way. Barium chloride, which was fashionable a few years ago, has not stood the test of time. Both digitalis and strophanthin are contra-indicated, for they are likely to make matters worse by increasing the grade of block between auricles and ventricles, and they should never be given in the attack. Strychnine, camphor and other irritants, and alcohol are quite without any effect, as also is amyl nitrite.

Palpitation.

The second group of cases is those in which a cardiac emergency arises with palpitation as the leading feature of the attack. Palpitation, though so common as a symptom, is rarely severe enough or the subject ill enough for the question of an emergency to arise at all, unless we are actually dealing with one of the forms of disordered rhythm. A simple tachycardia occurring in an hysterical attack may look at first sight like a true cardiac emergency ; but here the functional stigmata, often a globus hystericus, that sensation of choking in the throat, the bizarre features of the attack with convulsions and noisy obstructionist behaviour, and the gradual slowing of the heart rate as the attack subsides are enough to distinguish this condition. Often also the rate of the heart will vary widely during the attack in these functional patients, whereas in a truly abnormal rhythm the rate is remarkably constant, and rest, excitement, and exertion have little or no effect upon it. A tachycardia of this kind should always be regarded with suspicion. Another and the most important point, in distinguishing an attack of palpitation due to some abnormal rhythm is that the onset and offset are here almost always dramatically sudden, a feature which the patient recognizes as a rule. It is at any rate partially on this account that they come to have the character of a cardiac emergency, certainly to the patient.

Such paroxysms may be due to one of three causes, true paroxysmal tachycardia, a paroxysm of auricular flutter, or one of auricular fibrillation. Although the clinical distinction between these is not always easy without electrocardiographic evidence, there are a few points that help and that often give a shrewd idea as to which variety is actually present. The rate of the heart, the response to pressure over the carotid sinus in the neck, and the clinical associations in regard to other evidence of heart disease are the most important points.

Paroxysmal Tachycardia is most often seen between 20 and 40 years of age, commonly occurs as the sole manifestation of heart disease, or else in association with mitral stenosis, generally shows a ventricular rate between 160 and 200 a minute, and is usually benign. The attack passes off in a variable time without leading to congestive cardiac failure unless there is serious underlying disease of the heart or the duration is exceptionally long. The pulse keeps at the high rate during the attack without variation from rest or exercise, and is absolutely regular. Any tachycardia at a rate over 140 with such characteristics is likely to be of this kind, and if it started suddenly and stops in the same way the diagnosis is most probable.

Although as a rule the attack passes without harm, and death in an attack is almost unknown, congestive cardiac failure can come on very rapidly in those who have an already damaged heart muscle. This is seen most often in cases with old standing rheumatic carditis, and it is important to recognize this sequence. Cardiac failure may then appear within an hour or two; the patient becomes breathless and cyanosed, the cardiac impulse diffuse, the veins in the neck distend, the liver enlarges with overlying tenderness and rigidity, and there is occasionally oedema of the lungs. Seen then, for the first time, the significance of the tachycardia and palpitation may become buried and obscured under this accumulating mass of signs unless the fact is elicited that the attack began with sudden palpitation and not with dyspnoea. This point distinguishes such cases from those with cardiac dyspnoea which we shall discuss shortly. There is one other point, a most important one, about these patients with this combination of a paroxysmal tachycardia that has led to an acute congestive heart failure. These are the patients commonly regarded as examples of "acute dilatation of the heart," or of "heart strain." In point of fact although X-rays may sometimes appear to show a little enlargement of the heart during an attack, it is always difficult to demonstrate, often does not occur at all, or the heart size is reduced, and any trivial enlargement is quite subordinate to the rapid rate which is the important feature. Even when enlargement can be shown during an attack it disappears within a few beats when the attack stops, the heart then rapidly returning to its former size, whether this was normal or not.

As attacks of paroxysmal tachycardia almost always cease spontaneously within a few days at the outside the prognosis is good in the immediate circumstances. The outlook for future attacks depends more than anything upon whether there is associated disease of the heart or not, and it is here that fuller investigation with an electrocardiogram and opinion upon the heart size gathered from X-ray screening is so valuable. Evidence of myocardial defects between attacks or of a large heart both imply a poor eventual prognosis, with cardiac failure of that acute type during attacks as a likely sequel before long. In others with no evidence of any form of heart disease except the fact of the attacks themselves the outlook is very good; though the attacks recur and are a nuisance they rarely lead to serious harm.

There is no remedy known to control paroxysmal tachycardia, except that quinidine may sometimes be successful, especially in those where the abnormal rhythm arises from a focus in the ventricles. It is always worth a trial, either in a dose of five grains by mouth or three grains given slowly and well diluted intravenously. It is better avoided if the heart is much enlarged, and in any event will often disappoint. Morphia, heroin, camphor and other irritants, are all unavailing in controlling the attacks. Digitalis and strophanthin have no effect either in stopping or preventing them, and as they increase the irritability of heart

muscle they may be dangerous by converting a rapid ventricular tachycardia into fibrillation with a sudden exitus. They should not be given in an attack unless there is clear evidence of congestive cardiac failure which is urgent and increasing; then one's hand may be forced, but a safer measure is to do a venesection and wait afterwards for the attack to subside spontaneously. Sometimes the attacks can be stopped by firm pressure over the carotid in the neck for a minute or so; one side and then the other should be tried and is successful in about 10 per cent. of cases with immediate relief.

In the prevention of future attacks drug treatment is again a disappointment. Quinidine, given in doses of six grains every few hours for five doses may be tried if attacks are quickly recurring; or three grains twice or thrice daily for longer periods sometimes reduces the number of attacks, but it often fails to have any effect. If cardiac failure has occurred in a previous bout digitalis should I think be given in small doses between, say a grain and a half of the powdered leaf daily, with the object of preventing failure again in later attacks. As a rule it has no effect upon their incidence.

Auricular Flutter. In auricular flutter the auricular rate is usually between 260 and 320 a minute, and this is so high that the ventricles rarely respond at more than half this rate—i.e. 130 to 160 a minute. A constant pulse rate, uninfluenced by rest, effort, or excitement, and between these limits should thus suggest flutter, and especially in an elderly subject, and if the condition is prolonged for some time. Paroxysmal tachycardia, with which it is most likely to be confused, rarely lasts for more than a few days at most; flutter tends to be more persistent and may go on for weeks or months. The exact distinction has to be made by an electrocardiograph, though the longer the attack the more likely it is to be due to flutter. As with paroxysmal tachycardia there is the risk of acute congestive heart failure in a prolonged bout or when there is already serious myocardial disease; in this event the same sequence of events occurs with increasing dyspnoea coming on some time after the attack began with sudden palpitation. Here, as the patient may have been ill for some days, the dullness at the lung bases with crepitations, may simulate pneumonia, or the tenderness and rigidity overlying an engorged liver suggest an abdominal emergency if the initial palpitation of the attack and the dyspnoea are not given their proper perspective and importance, the constant and high pulse rate taken into account, and the other signs of congestion recognized. In flutter the veins in the neck show pulsations synchronous with the auricles and if the difference between this and the ventricular rate is noticed auricular flutter is certain; another point is that in flutter pressure over the carotid sinus often slows the ventricular rate markedly for a short time. Again in flutter there is often to be observed a sudden and precise doubling or halving of the ventricular rate; this feature is almost characteristic of the condition and is due to a variable ventricular response to the very high auricular rate. We have already discussed those examples where the ventricular rate suddenly rises to that of the auricles, 1:1 flutter, with the production of a syncopal attack.

As in paroxysmal tachycardia the outlook in flutter depends very much upon the underlying condition of the heart, and as it occurs most often in those same conditions in which auricular fibrillation occurs the prognosis follows similar lines. The size of the heart, the presence of myocardial defects revealed by the electrocardiograph, and the presence of cardiac failure during and between the attacks, are the most important points. It is however more amenable to treatment than

paroxysmal tachycardia, and it is in general unwise to give any prognosis until the effects of treatment have been observed.

The essential treatment of auricular flutter is digitalis or one of its allies. Full doses will always control the ventricular rate; an initial dose of two drachms of the tincture will generally begin to do this in 8 to 10 hours, and if the slowing is not enough this dose should be repeated next day, and smaller amounts subsequently. Alternatively strophanthin can be given, 1/250th to 1/120th gr. intravenously in a drachm of saline; this can be repeated in two hours if required and once more if necessary in four hours. This is the immediate treatment and the method of choice in a gravely ill patient; its effect is remarkable. It is upon this type of patient with acute cardiac failure due to auricular flutter that strophanthin made its reputation as a life saver. Both strophanthin and digitalis act by producing a grade of heart block in the A-V bundle, thus slowing the ventricular rate by preventing it from being driven by the auricles at the fast speed of natural flutter. Up to this point the actual auricular flutter remains the same; all that we have done is to slow the ventricular rate so that instead of a 1:1 or 2:1 flutter there is a 3:1 or 4:1 type.

Two courses are open once the patient has recovered from his urgent symptoms. We can either continue with full doses of digitalis, about 30 to 60 minims of the tincture daily, giving enough to keep the ventricular rate at about 70 to 80 per minute, with the object that the auricular flutter will become converted to auricular fibrillation. This is recognized by the fact that the ventricular rhythm then becomes completely irregular, whereas previously it was of course regular. There are two advantages in doing this; in the first place if there is cardiac failure it is more easy to control when the auricle is fibrillating than when it is in flutter, and the second advantage is that in about half the cases normal rhythm is restored spontaneously if digitalis is now withdrawn after flutter has been converted into fibrillation. The second possible plan of treatment is to give quinidine in the hope of restoring normal rhythm; this is a complicated and less successful line of treatment only suitable for a limited proportion of patients on account of the dangers of the drug. Its chief value is in a case resistant to digitalis, but it needs the most careful control if risk is to be avoided.

In those patients in whom digitalis has failed to produce normal rhythm and who are left in auricular fibrillation, and in those unsuitable for quinidine or resistant to this drug, digitalis has to be continued indefinitely in doses sufficient to control the rate of the ventricles. Such treatment of course abolishes the attacks of palpitation and wards off cardiac failure, and may be quite successful for years.

Auricular Fibrillation. The onset of this condition is often so quiet that it has in no sense a claim to be regarded as a cardiac emergency. When it does appear in this fashion the ventricular rate is high and, though palpitation is the initial symptom as a rule, congestive signs follow quickly. The recognition of this abnormal rhythm is generally easy unless the ventricular rate is very high, when the characteristic complete irregularity is masked, and the rhythm may seem to be regular. It is wise to listen to the rhythm of the heart sounds and not to rely upon the pulse, but occasionally only an electrocardiogram can decide.

The prognosis in auricular fibrillation is guided by the same considerations as in flutter, and treatment in the emergency and later is on similar lines. The doses of digitalis and strophanthin are the same as those used in cases of flutter,

though personal fancy can be allowed some latitude in deciding. There is no one method and scheme of dosage with any claim to constant preference; the essential thing is to give enough, and neither more nor less, to control the ventricular rate. The urgency of the case decides whether strophanthin should be used; and if this is not needed we should aim to secure a full effect within a matter of a few days. Massive doses of digitalis always have the disadvantage that the amount required may be overshot, and it is better to start with amounts of twenty minims of the tincture thrice daily, and gradually reduce as the effect comes on. Those who need more than this may be considered at once as candidates for strophanthin or digoxin intravenously administered. But there is no scheme of choice and it is better to get used to one's own scheme and use it consistently than to change with each case. In that way one gets accustomed to the effect of a certain plan of campaign and variations to meet a particular case are easier to make with some degree of certainty.

As in auricular flutter so in auricular fibrillation quinidine is apt to be a disappointing drug though it has at times brilliant successes. It is only suitable for those cases with a heart of moderate size, who have had no embolic accidents nor cardiac failure, and who are young in years. Granted these points have been decided favourably it may be tried, for in 40 to 50 per cent. of such selected patients normal rhythm is restored by a full course. The trouble is that so many relapse later. If quinidine is used it should only be after the heart size has been judged by X-ray screening and after a careful preliminary period of observation. A trial dose of three grains should be given to exclude susceptibility and thereafter a good plan of dosage is to give five grains the first day increasing subsequently to 30 grains daily by increments of five grains a day. When a dose of 30 grains daily is reached this amount can be maintained for a further three to five days. If normal rhythm is not restored by then it is not likely that it will be and the drug should be stopped.

["Cardiac Emergencies" will be continued in our June Issue.]

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