Clinical disorders of the heart beat : a handbook for practitioners and students / by Thomas Lewis.

CLINICAL DISORDERS OF THE HEART BEAT.

THOMAS LEWIS

FIFTH EDITION.

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CLINICAL DISORDERS

OF THE

HEART BEAT.

A HANDBOOK FOR PRACTITIONERS AND STUDENTS.

BY

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Fifth Edition.

LONDON:

SHAW & SONS, 7 & 8, FETTER LANE, E.C.4.

Printers and Publishers.

1920.

BY THE SAME AUTHOR.

"Clinical Electrocardiography."

"The Mechanism and Graphic Registration of the Heart Beat."

"Lectures on the Heart."

"The Soldier's Heart and the Effort Syndrome."

PREFACE.

DURING recent years our knowledge of the heart and of its affections has rapidly advanced. The newly acquired knowledge has been gathered largely by precise graphic methods. To the onlooker it often appears that this advance simply comprises a detailed analysis and accurate classification of cardiac irregularities. That is a quite inadequate review ; recent study has done much more for us than that ; it has thrown most of the major conceptions of heart disease into a melting pot, from which some have issued transformed, from which others will never emerge. The conceptions of heart failure, gradual or sudden, and of the rôle which valvular defects, and strain in the wider sense, play in its production, have undergone radical revision. The chief symptoms of heart disease, breathlessness, exhaustion, pain, syncope,

palpitation, and their relations to other phenomena of heart disease begin to be understood; so do a host of physical signs which formerly were quite obscure. The basis of prognosis and of treatment has been profoundly altered. Much of the cardiological work and teaching of the last century rested upon traditional doctrines which we now recognise to have been unsound. The methods employed were almost wholly subjective, and consequently open to very serious fallacies. Objective methods, and the graphic method in particular, have reformed our conceptions.

Familiarity with the heart's mechanism in health and disease is a first essential if these newer conceptions are to be appreciated. Judged by present-day standards, those who do not possess this familiarity are not competent to deal with cardiac patients. I make this statement after full consideration, speaking of cardiac patients generally, and without confining it to patients who exhibit irregular action of the heart. Confirmed in this view, I am asked again after eight

years—for it is now eight years since the first edition of this book was written—in what degree is an acquaintanceship with graphic method essential or expedient in the routine of busy practice? To this I reply, as I replied then, that the acquisition of the necessary manipulative skill and experience, which the taking and interpreting of graphic records involves, will often entail too great an expenditure of time and energy adequately to repay the practitioner or the patient he serves. A universal and detailed acquaintanceship with medical science as it exists to-day is no longer possible, but it behoves a practitioner to grasp new principles and to be conversant with new knowledge and its influence upon the care of patients afflicted with common maladies.

While the pursuit of graphic work by those who possess the special aptitude or a developing interest in the method is decidedly to be encouraged, yet it is clear that the universal adoption of graphic method is neither to be anticipated nor advocated.

And this conclusion is largely dictated by the belief that most of the every-day disturbances of the heart beat may now be identified by relatively simple means. If this is possible to a practitioner, then it is also possible for him to grasp the new general conceptions and to apply them in his daily work.

More than nine years ago, and at a time when I was actively engaged in studying cardiac irregularities by exact graphic method, these thoughts were in my mind; they led me to search for a series of relatively simple tests, based upon enquiry into the clinical histories of patients and upon bedside methods then generally in vogue, by which the common disorders of the heart beat might be recognised. For this purpose I made a large number of observations and incorporated in this handbook the new information so gathered with that gained from the contemporary writings of my co-workers. That its publication has been justified I judge from the repeated calls for new editions. I have again revised the text, and still

confine the graphic records almost to such as illustrate what may be seen and felt. There are few important disorders of the heart beat which the unaided and practised senses may not recognise. A single and portable piece of apparatus may be used in cases of doubt to supplement the observations so obtained. The Dudgeon sphygmograph is probably familiar to most medical men; it will readily trace a short curve from the radial pulse. Such a curve, especially if it be accompanied by a record of time, will usually complete an analysis of any common disorder of the heart beat.

The purpose of this handbook is not to discuss the evidence* upon which the diagnosis of cardiac disorders rests, but simply to recount such symptoms and signs as I have found to be serviceable in identifying these disorders, without recourse to complex graphic methods.

THOMAS LEWIS.

August, 1920.

* A full account of this evidence will be found in my other publications.

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CHAPTER I.

THE IDENTIFICATION OF DISORDERS OF THE HEART BEAT.

It seems desirable that I should open the chapters of this book by acquainting my readers with the general arrangement of the matter contained therein.

Since those who work amongst the sick usually become interested in a particular phenomenon by observing it rather than by reading of it, I begin the first chapter by describing in general terms the chief derangements of the rate and sequence of the pulse and heart beat as they are felt, seen, or heard by all practitioners. I take certain common and generally recognised physical signs as they are noted at the bedside and translate these signs into terms of mechanism, without attempting to describe the manner in which they are brought about and without suggesting their value in prognosis or treatment. I shall speak of seven forms of cardiac disorder, and they will be described under the following headings :—

1. *Sinus arrhythmia.*
2. *Heart-block.*
3. *Premature contractions or extrasystoles.*
4. *Simple paroxysmal tachycardia.*
5. *Auricular flutter.*
6. *Auricular fibrillation.*
7. *Alternation of the heart.*

There may be some to whom these terms are unfamiliar or to whom their meaning is still obscure. My immediate endeavour, therefore, is to offer them a preliminary idea of the meaning of these names, an idea which I hope may appeal to them from their past experience ; and I do so by citing clear examples of phenomena to which in future I must perforce refer in distinctive terms.

If we feel the pulse of a young adult, when the breathing is deep, or, better still, if we feel the beating of a dog's heart against the chest wall, a periodic irregularity of the pulsations is observed which accompanies the separate acts of breathing. I cite this disorder of the heart beat as a characteristic example of *sinus arrhythmia*, or one in which the whole heart is involved.

In many patients in whom the pulse and heart beats fall with perfect uniformity for long periods, an occasional and isolated disturbance is noticed. The *pulse* intermits ; it loses one of its serial beats at intervals. When the *heart* is examined it is found that at the moment of the disturbance a ventricular contraction appears before the rhythmic beat is due, and that this early beat is followed by a pause of unusual length. I cite this disturbance as a simple example of *premature contraction* or *extrasystole.*

If in a similar case, where the occasional pulse fails, a similar failure of ventricular action is discovered, so that on listening at the apex beat no abnormal sounds are heard, but the heart remains silent throughout the whole of the pause, the phenomena are evidences of another condition, namely *heart-block.* But lest I should create confusion at an early stage, I must add that heart-block manifests itself in many other ways, for example, by producing pulses of conspicuously slow rate.

Paroxysmal tachycardia is a term which is probably familiar to all, but I employ it in a restricted sense, and speak only of instances in which an absolutely abrupt acceleration of regular heart beat, which subsequently terminates in an equally abrupt manner, is repeated from time to time.

Sometimes, and especially in the elderly, we find regular and accelerated heart action at rates of 120 to 160 per minute. Such an acceleration, when notable for its constant rate under all sorts of conditions and for its tendency to persist without apparent cause, is generally the result of *auricular flutter.*

When a patient, who is known to have mitral stenosis and who requires treatment for cardiac failure, exhibits not only dropsy, venous engorgement and cyanosis, but a rapid and utterly disordered heart action in which there is no rhythmic sequence, he presents a characteristic picture of *auricular fibrillation.*

Finally, if in a case of renal disease or arterio-sclerosis the pulse tension is high, Cheyne-Stokes' breathing perhaps is present, and the pulse is regular in rhythm but varies in *force*, so that each alternate beat is strong and each alternate beat is relatively weak, an example of *alternation of the heart* is under observation.

I have deliberately chosen these examples because they are distinctive ; but the several forms of disturbance are not always so clearly differentiated. Were it so, my task would be simple. The examples are distinctive and consequently allow a preliminary idea of my terms to be entertained. It is into these terms that I shall in the first instance translate the commoner physical signs, and I do so with the object

of providing the student or practitioner with an immediate clue to the type of mechanism with which he is dealing. But as this preliminary description is inadequate, it is supplemented by a detailed account of each form of disorder in the remaining chapters of the book, in which an account of the pathology, prognosis and management will also be found.

Preliminary evidences.

Age. The first guide in identifying a disordered heart mechanism is the age of the patient in whom it occurs and a knowledge of the age incidence of the several disorders.

An irregularity of heart or pulse found before the tenth year is usually sinus arrhythmia. Heart-block may be present during the first decade, but it is rare ; premature contractions have been noted in quite young children, in most of whom enlargement of the heart has been present, or during the course of acute infections. Solitary examples of auricular fibrillation have been recorded at the ages of 5 and 13 ; it is very rare before the age of 17.

The relative frequence of the different disorders of the heart beat from adolescence to old age is in general hospital practice approximately as follows :—

Auricular fibrillation	40%
Premature contractions	35%
Alternation of the heart	10%
Paroxysmal tachycardia, sinus arrhythmia, heart-block and flutter	15%

Dealing with those in whom there is obvious cardiac failure, at least 60% of irregular hearts are irregular because the auricles are fibrillating.

Heart rate. Another valuable clue is the rate of the heart beat. *When the ventricle* beats regularly* and its rate is continually below 35 beats per minute, complete heart-block is probably present ; in similar circumstances a rate which lies constantly between 40 and 50 should arouse a suspicion of partial heart-block ; a persistent rate of 130 and over in a resting patient should always suggest a long continued paroxysm of tachycardia or auricular flutter.

If, on the other hand, *the ventricle beats irregularly* and its rate surpasses 120 per minute, fibrillation of the auricle is probably present, and as the rate is faster, so the probability that such is the disorder approaches certainty. Irregular hearts, beating at 140 and over, are scarcely ever affected in any other manner. Premature contractions very rarely accompany ventricular rates of 120 and over, sinus arrhythmias are almost confined to rates below 100 ; both these forms of irregularity become more frequent as the scale of rate is descended to the sixties and fifties. If an irregularity is observed, any influences, such as exercise, fever, or the administration of belladonna, which enhance the ventricular rate, tend to abolish all irregularities of rhythm, with the exception of that due to auricular fibrillation, and in this instance the disorder persists and is usually augmented.

Persistence of irregularity. Auricular fibrillation is usually persistent, and examination from hour to hour or from day to day reveals continuous irregularity. The other irregularities are transient, the pulse being perfectly regular from time to time ; shorter or longer periods of normal heart action intervene between periods of disturbance.

* When I refer to beats of the heart or of the ventricle, on this or future occasions, I do not refer to *pulse beats.* Ventricular irregularity and pulse irregularity are not always coincident ; the ventricular rate often surpasses the pulse rate.

Common types of disorder and their meaning.

Solitary pulse intermittences. An occasional pause of pronounced length, which interrupts an otherwise perfectly regular pause, is due to one of two causes,* namely, a premature contraction (common), or a dropped beat resulting from heart-block (rare). They are easily distinguished, for the premature beat is felt or heard at the apex, it gives rise to an early first, or early first and second sound. In block the heart is silent and motionless during the whole pause.

Coupled beats. If the *ventricular* beats are coupled and the couples are evenly spaced† they are the result of one of two disturbances, for either the alternate beats of the normal rhythm have been replaced by premature contractions—in which case the second beat of the couple is weak and may not reach the wrist—or else each third ventricular contraction has been lost and heart-block is present. If the *pulse* beats are coupled (*pulsus bigeminus*) a third possibility remains; the pulse pairing may be due to the occurrence of premature heart beats which replace each third rhythmic beat. If such is the case the premature beat will be appreciable at the apex, though it does not reach the wrist.

Triple beating. To recognise the cause we proceed along similar lines. Tripling at the apex is due to premature contractions which replace each third rhythmic beat, or to heart-block in which each fourth ventricular contraction is lost. Tripling at the pulse (*pulsus trigeminus*) may be due to a third cause, namely, premature beats replacing each fourth rhythmic beat, the early beats failing to reach the wrist.

* A long pause of a respiratory arrhythmia occurring during expiration may be mistaken for intermittence if the examination is cursory.

† Sometimes the pauses following the pairs are not of uniform length. The irregularity is then a complex one; it is due to auricular fibrillation, to which premature beats have been added. Patients who show it are usually under the influence of excessive doses of digitalis.

Halved pulse rate. When the ventricle beats at twice the pulse rate, the disorder is due to premature contractions in all but the rarest instances. Alternation has been known to occasion halving, the weak alternate beats failing to reach the wrist ; but this condition is of great rarety and, when it occurs, is very transient. The two are readily differentiated, for in the first instance the ventricular beats are coupled, while in the last they appear regularly.

When sudden and exact halving of pulse rate is noted and the ventricular rate is halved simultaneously, the disorder is the result of heart-block.

A grossly irregular pulse in which there is an intricate admixture of stronger pulsations with quick runs of almost imperceptible beats, and in which the lengths of intervening pauses are constantly varying, is due to auricular fibrillation.

A mild grade of irregularity which persists, which is not related to respiration even when the breathing is deepened and in which no definite sequence of events can be determined, is also due to auricular fibrillation in most cases. A similar irregularity which shows relation to respiration is a sinus arrhythmia.

Familiarity with the few rules which I have now given will enable the practitioner to identify most forms of disorderly heart action with which he meets. But when he is in doubt, or when he requires more explicit information, in regard to the arrangement of the beats, or in respect of the management of the case in which the disorder is discovered, he may refer to the more precise descriptions contained in the following chapters.

CHAPTER II.

SINUS IRREGULARITIES.

Definition.

Irregularities of the heart which are produced by interferences with the rhythmic impulses at the seat of their discharge.

The nature of sinus disturbances.

In a discussion of sinus irregularities, the nerve supply of the heart in relation to disturbances of rhythm occupies a prominent place. Let me state emphatically at the outset that we have nothing to do, first, with the functions of the intrinsic cardiac ganglia, nor, secondly, with the sympathetic nerve trunks; as we have little knowledge of the part they play in disease, so any hypothesis which ascribes a derangement of the heart to perversion of their functions is without practical significance. We have a limited but real knowledge of the vagus and its relation to pathology; my remarks upon the cardiac nerves are consequently confined to it.

The complete beat of the normal heart consists of a contraction of its chambers in orderly sequence. The wave of contraction has been proved to start in a small mass of tissue, the *sino-auricular node* (Fig. 1), which lies embedded near the upper and anterior end of the *sulcus terminalis*. The *sulcus terminalis* runs, as may be remembered, from the junction of the right auricular appendix and superior

vena cava towards the inferior vena cava (see Fig. 1). The tissue of the node, consisting of a network of small spindle-shaped muscle cells, richly supplied by the nerves of the heart which enter in this region, lies therefore at the mouth of the superior vena cava and is embedded in the wall of the right

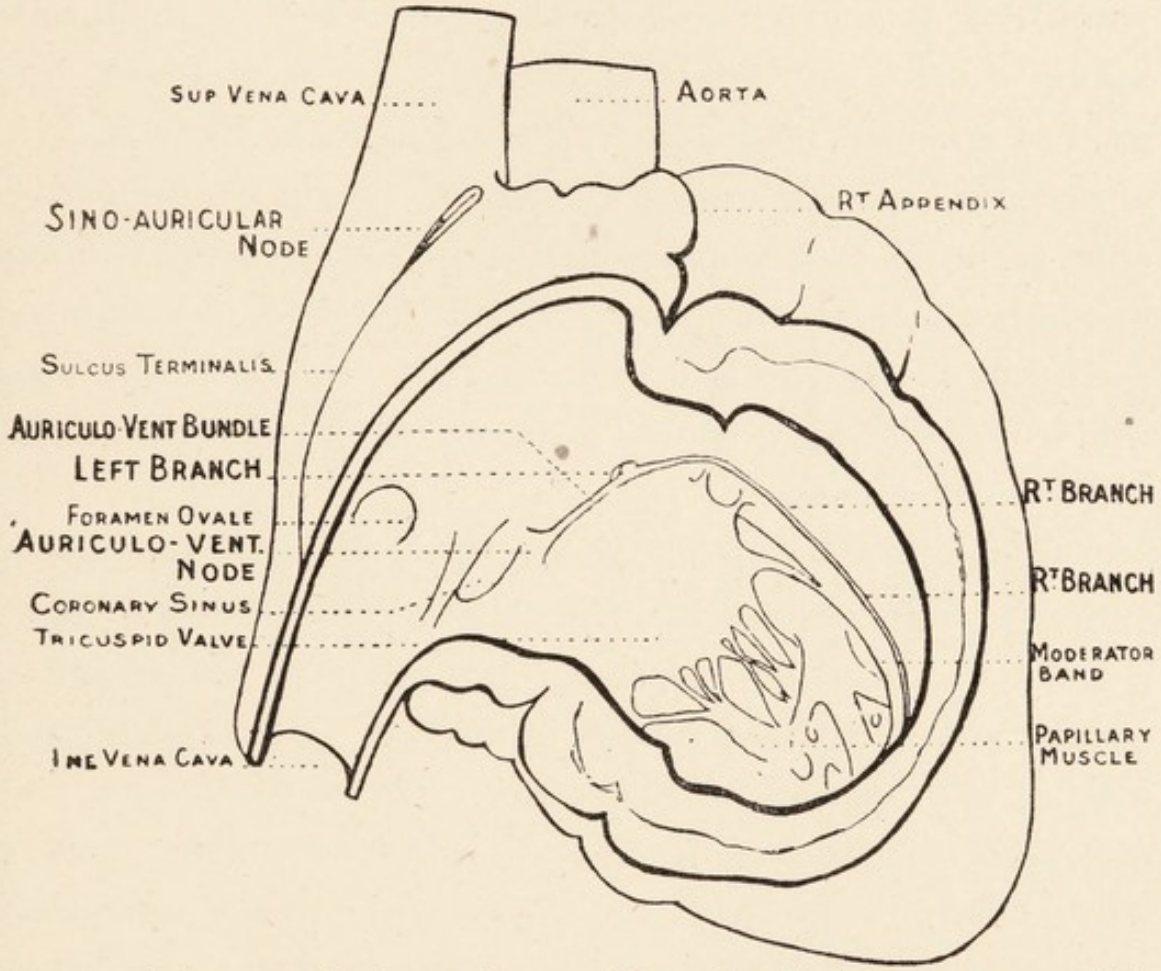


Fig. 1. A diagram of a human heart. The walls of inferior vena cava, right auricle and right ventricle, have been partially removed to expose the septa. The position of the sino-auricular node, in which the heart beat commences, is shown, as are also the position of the auriculo-ventricular node and the course of the auriculo-ventricular bundle and its branches. The last-named structures convey the contraction wave from auricle to ventricle.

auricle. The contraction which commences in its neighbourhood spreads through the walls of both auricles and is transmitted to the ventricles by a special band of tissue which will receive subsequent description. The orderly rhythm of the whole heart takes its origin in this node, to

which I have consequently applied the term heart's "pacemaker." In the normally acting adult heart the pacemaker sends forth waves of contraction at rates which *average* 72 per minute, and, the separate beats being evenly spaced, the systoles follow each other in a regular order or rhythm. The pacemaker is under the control of the vagi, or inhibitory nerves of the heart, and these normally restrain this stimulus producing centre, holding it, as it were, in leash. Destruction of the nerves, more especially that of the right side, or the administration of atropine, which paralyses the nerve endings in the heart, raises the rate at which the heart beats follow each other. In the human subject, the probable limit to which the rate may rise as a result of this denervation is 150-160 per minute.

In many subjects under special conditions, the vagus curbs the heart excessively, and this over-action of the nerve is apt to be relatively constant or rhythmic. There consequently results either a relatively uniform pulse slowing or a waxing and waning of heart rate. Let us deal with the persistent slowing first, for it is a subject with which we are only briefly concerned in these chapters. Pronounced slowing of the whole heart is comparatively rare; the lesser grades of slowing, many of which are of vagal origin, slowing to 50 or 60 beats per minute, are not uncommon and are especially prominent in athletes and in association with increased arterial pressure, pregnancy, jaundice, convalescence from the acute fevers and less frequently with other conditions. Pulse slowing of this degree has no great significance, and it is common to meet with people who enjoy perfect health and in whom during rest the pulse rate habitually lies between these limits.

Periodic or varying disturbances, which influence the rhythm of the heart at its source and produce a greater or lesser degree of arrhythmia, are more important, though only the commoner varieties of such irregularity need to be described.

In Fig. 2 a diagram of a characteristic sinus arrhythmia is given. I shall refer to similarly constructed diagrams in succeeding chapters. The figure is arranged so that each narrow black rectangle (*A*) represents a single co-ordinate beat of the auricle, and so that each broad black rectangle (*V*) represents a co-ordinate ventricular contraction. Where an

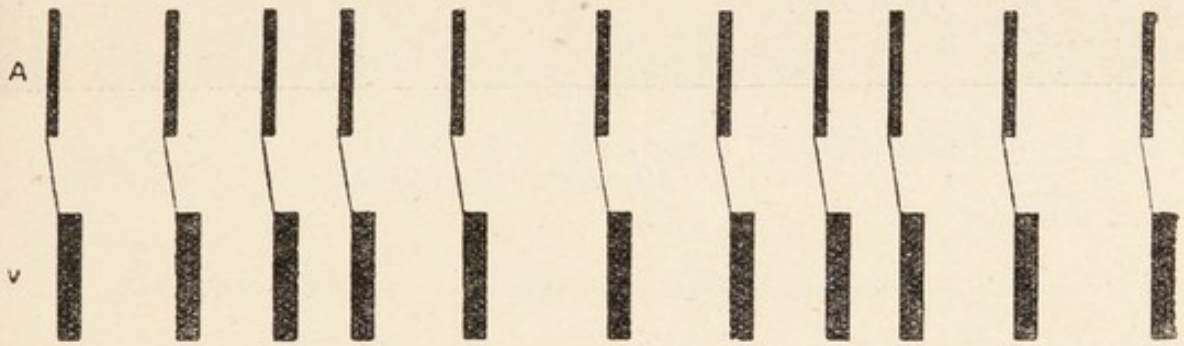


Fig. 2. A diagrammatic representation of the action of a heart affected by sinus arrhythmia. The contractions of auricle and ventricle are represented by the thin and broader black rectangles, *A* and *V* respectively. The auricle contracts at the beginning of each cycle, and sends its impulse, indicated by an oblique line, to the ventricle, which responds. The irregularity consists of a waxing and waning of rate, in which auricle and ventricle participate equally.

auricular contraction is followed by a ventricular response, an oblique line is drawn, joining the corresponding rectangles. The slope of the oblique line indicates the time interval between the contraction of auricle and ventricle. All such diagrams read from left to right. In the present example, a sinus irregularity, the whole heart is affected, so that each ventricular contraction is preceded by an auricular systole at the usual interval. The irregularity consists of a gradual waxing and waning of auricular rate which is repeated periodically, and which is followed exactly by the ventricle.

Respiratory irregularities.

It is well known that young adults manifest a very appreciable irregularity of the heart and pulse action when they breathe deeply (Fig. 3). The pulse quickens when the chest is inflated, and slows when the chest is emptied. But in young adults and in the aged also there is usually no respiratory variation of pulse rate, which the finger can

Fig. 3. A sphygmographic curve from a normal subject, breathing deeply. There is an increase of pulse rate during inspiration and a decrease during expiration.

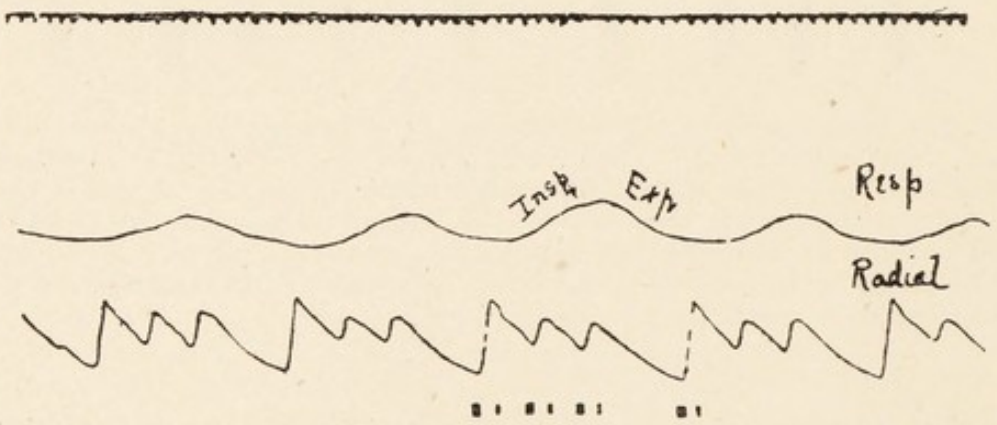


Fig. 6. A gross sinus arrhythmia of rare type ; a long pulse cycle accompanies each expiration. In this, as in all similar figures, the top line represents time in fifths of seconds.

discover, while *the breathing is natural.* On the contrary, a perceptible degree of natural respiratory irregularity of the pulse, characterised chiefly by one or more longer pauses during the expiratory period (Figs. 4 and 5), is not uncommon in young children, and sometimes it is sufficiently prominent to attract immediate attention. Irregularity of a very similar

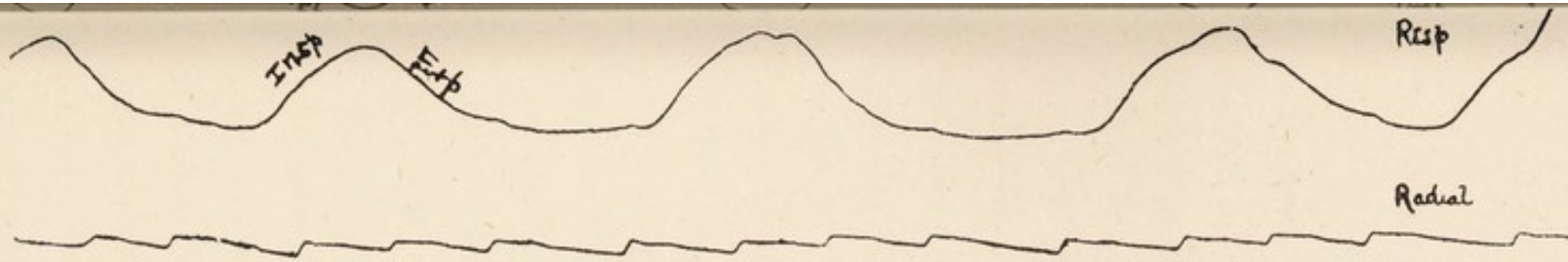


Fig. 4. A curve of pulse irregularity which is repeated with each complete cycle of normal respiration.

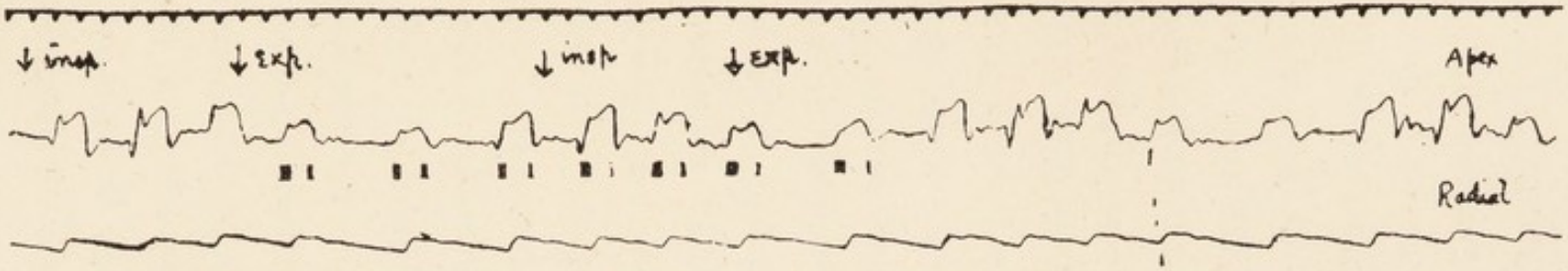


Fig. 5. Curves from the heart's apex beat and radial artery, showing a periodic increase and decrease of pulse rate with the respiratory cycles. The beginnings of inspiration and expiration are indicated by arrows. The heart sounds, which are represented diagrammatically, run parallel with the ventricular beats.

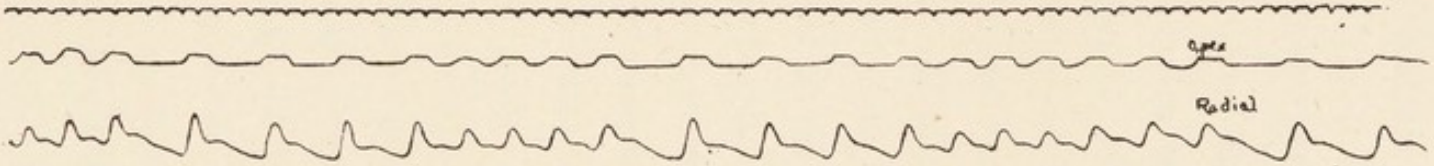


Fig. 7. A periodic irregularity of the heart of sinus origin, which shows no relation to respiration. The breathing was held while this curve was taken.

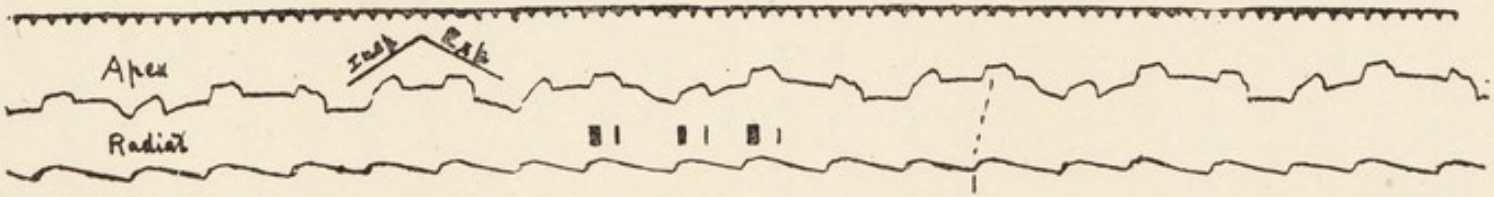


Fig. 8. Slight irregularity of the pulse in a child. The irregularity bore no constant relation to the acts of breathing while it was natural, but became purely respiratory when breathing was deepened. It is a sinus irregularity.

kind is found frequently at the age of puberty, and it is also seen on rare occasions in the adult (a striking though rare example of the last is shown in Fig. 6).

All these irregularities are of vagal origin.

Sinus irregularities which bear no relation to respiration.

While the vagal irregularities of heart rhythm generally show a respiratory relation, disturbances of similar origin occur where there is no association between the changes and the several acts of breathing. These disorders of the heart fall into three main categories. They are :—(1) Sudden and prolonged cessation of the whole heart beat usually associated with syncope ; it is a very rare condition and requires but a passing notice in this general survey. (2) Relatively abrupt and profound slowing of the whole heart (to 20 or 40 beats per minute), accompanied by an independent lowering of blood-pressure. This disturbance is often responsible for attacks of faintness or actual loss of consciousness. I have met with it frequently in soldiers invalided for the condition known as " irritable heart." It is, I believe, the commonest cause of fainting attacks in men and women. (3) Phasic variation of pulse rate, in which a retardation and subsequent gradual acceleration of the whole heart occurs ; the change is spread over ten, fifteen or more seconds, and may be repeated regularly or may occur from time to time ; it is associated with the administration of heavy doses of drugs of the digitalis group, but may be seen apart from them (Fig. 7) ; it is an uncommon type of irregularity. (4) An irregularity of the whole heart of mild degree, in which shorter and longer pauses are mingled indiscriminately. It is not infrequent, and is almost always combined with a general

reduction of pulse rate. It may be found in quite young and apparently healthy children (Fig. 8) and is also encountered in young adults in whom no other cardiac sign is apparent. It is specially frequent in patients who have rheumatic heart disease and who are under the influence of digitalis; it is accentuated when the heart slows after it has quickened in response to exercise.

These sinus irregularities, like those which are related to respiration, are all due to alterations of vagal tone.

The recognition of sinus irregularities.

Sinus irregularities are usually recognised with ease. It may be said that the great majority of pulse irregularities which occur before the end of the first decade are of this kind, and most of them are respiratory. When there is the definite relation to respiration, no further evidence is required; in most instances of sinus irregularity this relation is present, but if it is absent it becomes established if the breathing is deepened; a gradual waxing and waning of rate is always highly suggestive, if not conclusive. The radial beats and apex pulsations correspond; the heart sounds are simply modified according to the incidence of the ventricular contractions. The radial beats are full, and the apices of the several pulsations maintain an almost constant height in arterial curves (Figs. 4, 5 and 8).

Vagal irregularities are all abolished by any factor which notably increases the average pulse rate. Thus they disappear with exercise, with fever, or shortly after the administration of amyl nitrite or atropine. Atropine also cuts short or prevents standstill of the heart and the profound slowing described on the previous page.

The prognostic significance of sinus irregularities.

The commoner form of sinus irregularity (excluding those which may be responsible for syncope and the true phasic variation of pulse rate) are of little prognostic value. They are so frequent in patients who present no other sign of cardiac disturbance, either at the original examination or subsequently, that they are to be regarded either as slight exaggerations of a normal phenomenon (respiratory irregularity) or as evidences of a mild and insignificant instability of tonic inhibitory nerve action.* Unhappily, different forms of cardiac irregularity are still widely confused; irregular action of the heart in children is still regarded by many as a grave sign ; it is usually a phenomenon of health and when of sinus origin may be disregarded safely. The common sinus irregularities should not be allowed to influence the habits of those who exhibit them ; they neither suggest nor require special therapy.

* Occurring in children, this irregularity has obtained without reason an unenviable reputation, on account of its supposed relation to tuberculous meningitis.

CHAPTER III.

HEART-BLOCK.

Definition.

An abnormal heart mechanism, in which there is a delay in, or absence of, ventricular responses to the auricular impulses.

The nature of heart-block.

Under normal circumstances, the ventricle depends for its stimulus upon impulses which are sent down to it from the regularly contracting auricle. Each auricular systole transmits a stimulus to the ventricle, and this stimulus travels from auricle to ventricle along a narrow neuro-muscular path, the auriculo-ventricular bundle. This band of tissue starts in the right auricle near the coronary sinus and proceeds forwards and downwards to the membranous septum of the ventricle (Fig. 1), where it divides into two main branches on either side of the septum. The main branches subdivide and are connected to the ventricular musculature through the complex network of cells named after Purkinje. The sequence in which the chambers of the heart normally contract is diagrammatically illustrated by Fig. 9.

When from any cause the function of the tissues uniting auricle and ventricle is impaired, this sequential contraction is disturbed. The grades of disturbance which human hearts manifest are numerous.

There may be a simple prolongation of the interval which separates the beginnings of auricular and ventricular systole (the *As- Vs* interval, as it is termed). Such a conduction defect is illustrated by Fig. 10 ; the thin lines become more oblique in the diagram, and a gap is left between the end of auricular and the beginning of ventricular systole.

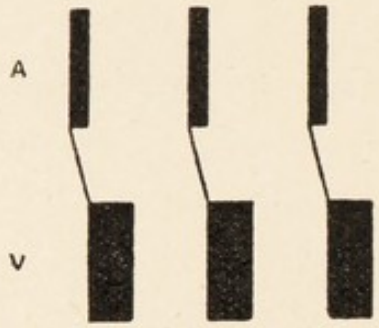


FIG. 9.

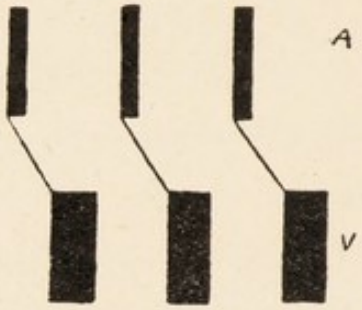


FIG. 10.

Fig. 9. A diagram representing the normal heart action. The auricle contracts first and transmits an impulse (represented by the oblique line) to the ventricle. The ventricle responds and commences to contract immediately at the cessation of auricular systole.

Fig. 10. A diagram illustrating the earliest stage of heart-block. An interval appears between the end of auricular and the commencement of ventricular systole. There is delay in the transmission of the impulse from auricle to ventricle (indicated by the increased obliquity of the line which joins the rectangles in the diagram).

Where the grade of heart-block is higher, the ventricle may fail to respond to occasional auricular impulses ; a condition commonly designated by the term " dropped beats." This form of heart-block is rarely a simple phenomenon ; it is almost always complicated by variations in the lengths of *As - Vs* intervals over the period of disturbance. The relation of chamber contractions may be studied in Fig. 11. A " dropped beat " or ventricular silence produces a pause of exceptional length and this pause breaks the natural rhythm of the ventricle. Where there is no associated variation in the *As- Vs* intervals, the long ventricular cycle

is necessarily equal to that of two regular cycles. But this is rarely the case ; the " dropped beat " is foreshadowed by a progressive increase of the preceding *As- Vs* intervals (Fig. 11, *1*, *2* and *3*). Moreover, the *As - Vs* interval which follows the silence is generally curtailed (Fig. 11, *4*). These changes in the conduction intervals shorten the long ventricular cycle and consequently diminish the grade of ventricular disturbance. The exact manner in which the changes happen requires closer study. Consider the first three *As- Vs* intervals of Fig. 11 ; as illustrated by the obliquity of the lines, the interval gradually widens, but it widens in a peculiar manner. The increase of the second interval

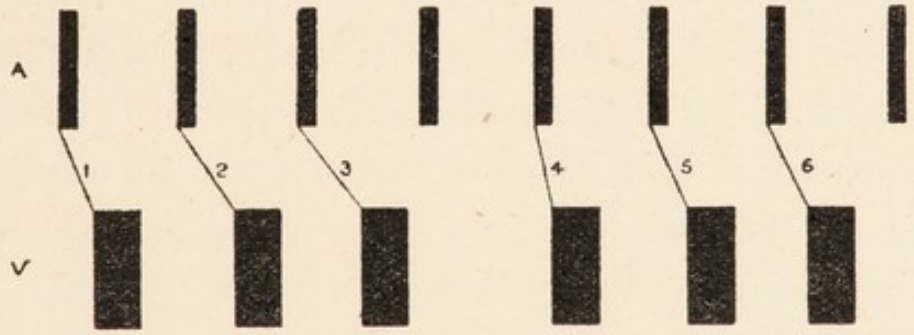


Fig. 11. The second stage of heart-block, to which the term " dropped beats " is applied. Up to the point where the chief disturbance occurs, the gaps between the auricular and corresponding ventricular contractions widen out. The impulses travel to the ventricle with increasing difficulty. The fourth auricular contraction stands isolated, it yields no response ; a ventricular contraction is "dropped." Following the ventricular pause, the *As- Vs* interval is short, for the tissues have rested, but it again widens as successive cycles follow.

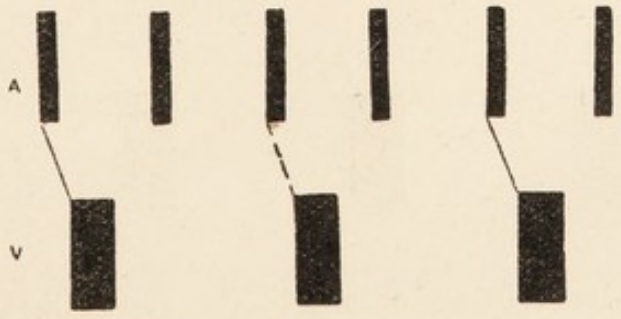


Fig. 12. A diagram of 2 : 1 heart-block, in which the ventricle responds to each second auricular beat.

over the first is greater than the increase of the third over the second. The result is a decrease of the interventricular period directly preceding the ventricular silence. The ventricle quickens to the point of the disturbance. The shortening of the *As- Vs* interval following the pause, and the subsequent prolongation of it, produces a similar quickening of the ventricle after the disturbance. The primary and secondary accelerations of ventricular rate, before and after the disturbance, help to identify the disorder in many arterial curves of heart-block.

As the grade of heart-block rises, and ventricular silences become more frequent, relatively simple ratios are established between the auricular and ventricular rates. When the ventricle beats at only half the rate of the auricle, because alternate impulses are ineffective, the condition is spoken of as 2 : 1 heart-block (Fig. 12). 3 : 1 and 4 : 1 ratios, in which each third or fourth auricular impulse alone yields a ventricular response, are sometimes encountered, but they are uncommon.

The disturbances which have been described are all included under the term " partial heart-block."

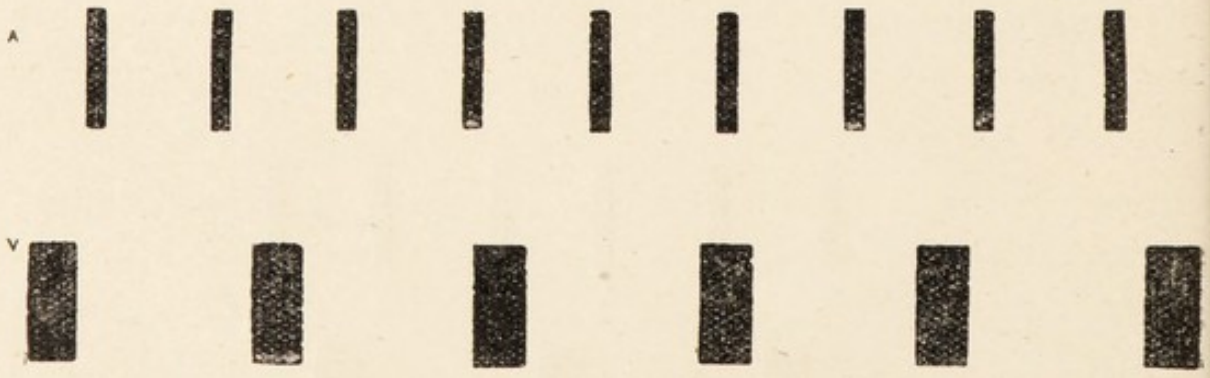


Fig. 13. A diagram of complete heart-block or dissociation. Both the auricle and the ventricle beat regularly, but at independent rates. The relative positions of auricular and ventricular contractions are very variable.

The highest grade of heart-block is reached when no impulses are transmitted to the ventricle. When this happens, the ventricle, having completely lost the controlling influence of the auricle, beats in response to a slow and regular series of impulses which it builds up intrinsically. In "complete heart-block" or "dissociation" two entirely separate rhythms are maintained; one starts in and controls the auricle, the other originates in and regulates the ventricle. The first has the usual rate, 72 per minute or thereabout; the last has an approximate rate of 30 to the minute. Though both are regular, the rhythms are mutually independent (Fig. 13) and the systoles of auricle and ventricle fall with varying time relations to each other.

Etiological and pathological associations.

Age and sex. Heart-block may occur at any age. It has been observed in the newborn child and at almost all ages into the eighties and nineties. Its age distribution is settled by the age incidence of the diseases which produce it. Thus, it is especially prevalent amongst those whose hearts have been severely damaged by rheumatic fever or chorea, so that a special class of case is grouped between the years 10 and 35. Senile affections account for another large group of patients who suffer from this cardiac disturbance; these patients are elderly. But the causation is so varied that no age is exempt.

Heart-block is much commoner in man than in woman.

Heredity. A single instance of the supposed occurrence of several cases of heart-block in the same family has been reported, but has not been substantiated. It is highly improbable that heredity plays any direct part in the

affection ; though a few cases of congenital heart-block are on record.

Relations to infective disease. Relatively, heart-block is not infrequent during the course of infective diseases, and of these rheumatic fever holds the first place ; the disturbance is usually temporary. The exact relation of rheumatic affections to acute and sub-acute inflammatory lesions of the heart is but imperfectly known, but there is a clear connection between them. Many instances of heart-block have been reported during the course of severe rheumatic fever and its complications, acute endocarditis and pericarditis. It is probable that the infection of the heart is never limited to its outer or inner layer ; the middle layer or myocardium is probably always involved. My own experience leads me to think that heart-block is almost a common accompaniment of acute or sub-acute rheumatism of the heart, for I have seen a number of patients in whom during the course of rheumatic fever involving the valves or pericardium, or both, dropped beats or partial heart-block in its several grades have been present. In other instances, temporary heart-block has appeared during short febrile attacks in patients who have been previously affected by rheumatic fever. It is certain that, being transient, it is often overlooked.

Other acute affections which should be mentioned are those of the more active pus organisms, and also those of diphtheria, influenza, typhoid, scarlet fever and pneumonia ; heart-block in these conditions is limited to infections of severity.

A large proportion of the reported cases of *chronic* heart-block and of those which have come under my own observation, have belonged to two groups ; the disorder has

followed single or repeated attacks of rheumatic fever, or has been the direct result of syphilis. Whether of rheumatic or syphilitic origin, whether acute or chronic, heart-block is generally but an expression of a widespread affection of the heart muscle in these patients, though the lesion may be confined to, or may fall most heavily upon, the tissues which establish functional connection between the auricle and ventricle. In a fourth of the cases in which the hearts have been secured after death the lesion has been gummatous. In a series of 42 cases which I collected in a few years, 6 gave a frank history of venereal infection and 14 a history of rheumatism.

The relation of heart-block to rheumatism in chronic heart affections is a peculiar one. The heart-block is often dormant or is detected only by exact instrumental methods ; it may be unmasked by the administration of drugs of the digitalis group, for the higher grades of heart-block are produced from the lesser by these poisons.

Relation to chronic degenerative processes of more obscure origin. Many cases of heart-block are in elderly people, and observation shows that the responsible damage is part and parcel of a widespread change, either in the heart alone, or in the heart and its vessels. A number of the lesions can undoubtedly be traced to syphilis or rheumatism, but the cause of a still larger number is obscure. Chronic inflammation, fibrosis, atrophy, calcification or fatty degeneration of the tissues, associated or unassociated with disease of the coronary arteries, are amongst the most frequent causes.

Heart-block as the result of digitalis administration. I have referred to the unmasking of dormant heart-block in

rheumatic heart-disease. When digitalis, or an allied drug such as strophanthus and squills, is given in toxic doses to young patients who have rheumatic hearts, it is not uncommon to observe the severer grades of partial heart-block as a sequel. In most, but not all, of the cases which react in this manner a slight defect in the conduction of impulses from auricle to ventricle was present before the drug was taken. The added effect may be due to the action of digitalis upon the vagus nerve, for sometimes it can be removed by atropine.

Heart-block may be induced in experiment by stimulating the vagus, and efforts have been made to establish a clinical group in which the heart-block is attributable to disturbed innervation. Up to the present time there is no very clear evidence that anything but a temporary disturbance of this character may be initiated purely by vagal impulses ; though, as I have stated, a pre-existing tendency may be exaggerated in this manner ; if the higher and enduring grades of heart-block are ever due to derangement of the vagi, this form is so rare that it scarcely needs consideration in practice.

Special morbid anatomy. It is in the main bundle, or in its auricular attachment, that the lesions responsible for heart-block are found. The kind of lesion has been spoken of already Gummata, chronic inflammatory processes and their accompaniments, fibrosis, atrophy, and calcification are most frequently seen. Examples of tumours (fibroma and endothelioma) affecting these special tissues have been recorded.

Ulceration invading the bundle (rarely), acute inflammation evidenced by deposition of leucocytes (commonly), or parenchymatous degeneration of the bundle are the lesions found in hearts damaged by acute or subacute infections.

The recognition of heart-block.

The disorders of the heart's mechanism caused by heart-block, in its several grades, are readily recognised by the exact graphic methods provided by the polygraph and galvanometer. The efficacy of these instruments and the certain analysis which they provide must be evident, for heart-block produces deranged sequence in the contractions of auricle and ventricle, and the polygraph and galvanometer supply separate and clear records of the systoles of upper and lower chamber. To compare the onsets of the several systoles, therefore, is relatively simple when these recording devices are employed.

But I speak to those to whom special methods are not available, and I hope to show that heart-block can be recognised in many of its grades by simpler means. It will be necessary to treat each grade of the disorder separately.

Often the earliest manifestations of heart-block consist in a **widening of the As-Vs interval** (see page 18); this defect can rarely be identified by ordinary clinical means; yet it may be responsible for two physical signs. It may not be known to everyone that auricular systole produces a distinct though muffled sound, and that while this sound is inaudible when the heart's mechanism is normal, it is frequently heard when the auricular and ventricular systoles are sufficiently separated. A slight widening of the *As-Vs* interval may lead to a reduplication of the first heart sound; a more pronounced widening may result in a double second sound, for the auricular systole will then fall in early diastole.

Another sign is confined to cases of mitral stenosis and is of similar origin; in these patients the systole of the auricle

causes or reinforces the diastolic rumble which characterises the valve lesion. Contraction of the auricle at an abnormal instant in diastole (for example, mid-diastole) is accompanied by a murmur and thrill at a corresponding time. When the pulse is regular, apical thrills or rough murmurs, confined to mid- or early diastole, are physical signs which should suggest, not alone stenosis, but also the beginning of heart-block.

Single dropped beats are not difficult to detect. Take the case of a pulse, which though it seems otherwise regular, is interrupted by an occasional pause of unusual length, while examination of the cardiac impulse reveals neither movement nor sound in the pause. If the pause is not associated regularly with the expiratory phase of respiration (see page 12) it can be attributed to a failure of the customary response of ventricle to auricle. The length of the pause in radial tracings may be exactly equivalent to that of two rhythmic beats. More frequently (as in the radial pulse tracing of Fig. 14) it is

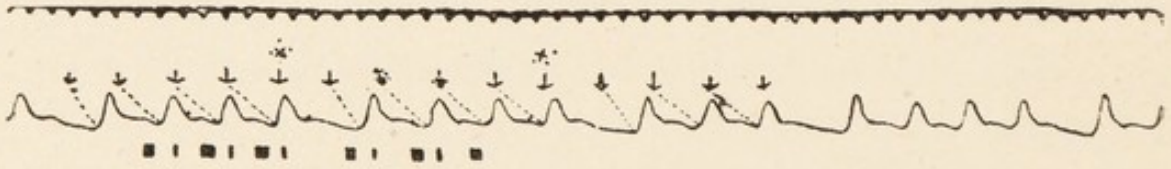


Fig. 14. A pulse curve showing "dropped beats." The arrows, which represent the positions of the regular auricular contractions, have been accurately determined in this and subsequent figures by means of polygraphic curves. There is, of course, a big delay between the auricular systole and the pulse beat. The heart sounds are shown diagrammatically. The arrangement of the pulse beats depends upon the lengths of the *As- Vs* intervals and upon the failure of response at the points marked by asterisks. Note the widening of the *As- Vs* intervals and accompanying increase of pulse rate before and after each dropped beat.

distinctly short of this, and is preceded and succeeded by slight pulse quickening. The nature of these changes has been considered (page 19), and the mechanism is indicated in

the present figure by arrows showing the points at which the regular auricular systoles fell. Responses to the auricular contractions marked by asterisks have failed.

With exercise* the pulse accelerates and becomes regular ; with rest the irregularity reappears and the first sign of its return is the occurrence of an unusually long *ventricular* pause.

When dropped beats are more frequent, the irregularity takes the form seen in Fig. 15. Here each third or fourth

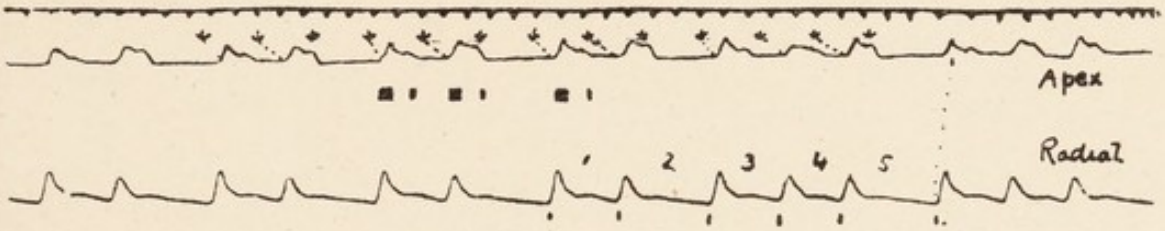


Fig. 15. Curves of heart's apex beat and pulse from a patient in whom ventricular responses failed frequently.

impulse miscarries, and the heart and pulse beats are grouped in twos and threes. This irregularity, like the last, may be recognised clinically by observing the action of the ventricle immediately at the end of exercise,* for it is abolished when the heart rate is raised and its resumption is signalled by a ventricular pause, as opposed to a premature beat, such as is heard in the corresponding extrasystolic irregularity in similar circumstances ; the same test applies to such irregularities as are seen in Figs. 16 and 17.

2 : 1 heart-block is to be suspected in any patient in whom the ventricle beats regularly and in whom the heart rate lies between 40 and 50 contractions to the minute. A sudden and exact halving of *ventricular* rate is always most suggestive of its onset. 2 : 1 heart-block is unstable, the ventricle quickening from time to time, and these changes

* Or a whiff of amyl nitrite.

in the rate of its response to auricle disclose the nature of the whole disturbance.

Exercise* abruptly doubles the ventricular rate; with subsequent rest the raised rate falls abruptly to one half.

In mitral stenosis partial heart-block is often characterised by peculiarities of the murmurs. They are often

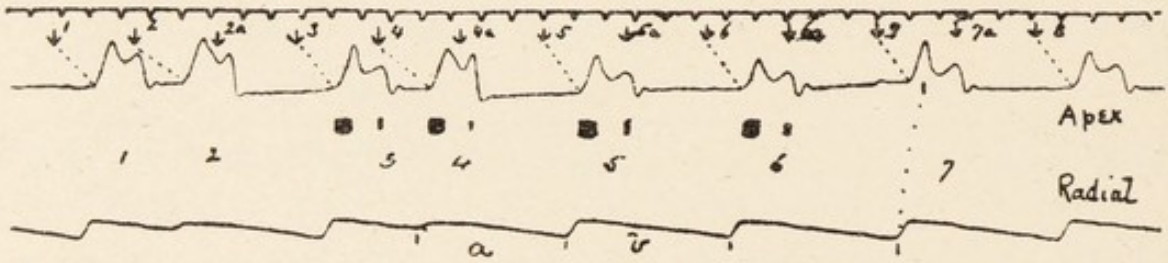


Fig. 16. Curves of heart's apex beat and pulse, taken during the transition from frequent "dropped beats" to 2 : 1 heart-block. The rate is reduced to exactly three-fourths the original at the change.

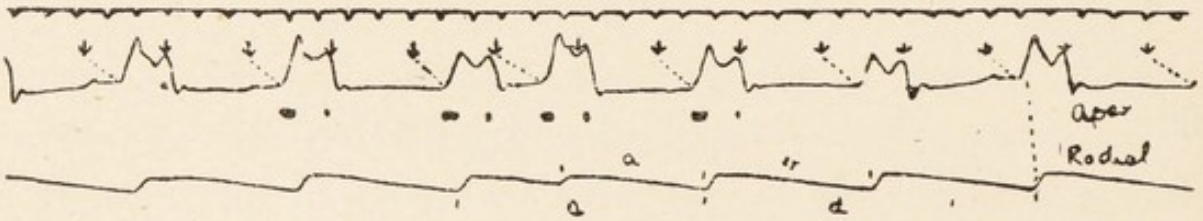


Fig. 17. Curves showing the interruption of a period of 2 : 1 heart-block by a single response of the ventricle to one of the series of alternate impulses which usually yield no ventricular contraction.

The transition between 2 : 1 heart-block and an arrangement previously studied, namely, the loss of each third response, is shown in Fig. 16. A bigeminal or coupled action of the ventricle passes over into a slow regular action. The features which proclaim heart-block in this curve are the increase in the length of cycle *b* as compared to *a*, and the exact reduction of rate to three-fourths. The lengths of the several pauses are understood by examining the positions of the auricular systoles which have been indicated by arrows drawn on the curve. Systoles *2a*, *4a*, *5a*, *6a* and *7a* do not affect the ventricle; and where the ventricle is silent a lengthy pause is found. The arterial cycle *a* is brief as compared with *b* because the auricular impulse *4* takes longer to reach the ventricle than does impulse *5*. Disturbance of a 2 : 1 period is shown in Fig. 17. An early contraction of the ventricle is followed by a pulse cycle *a* which is shorter than *b* and the succeeding cycles. The reason of this shortening has been explained in the description of the last figure. In Fig. 17 heart-block is also evidenced by the total duration (*c*) of the two short-cycles; it is equal to one and a half times that of the longer cycles (period *d*). In other words, *c* and *d* each correspond to three auricular cycles.

* Or a whiff of amyl nitrite.

extremely complex. Where 2 : 1 heart-block is present two thrills and two diastolic murmurs may accompany each ventricular cycle. The arrangement of the murmurs will be understood when it is remembered that the thrill and harsh murmur of mitral stenosis are produced largely by auricular

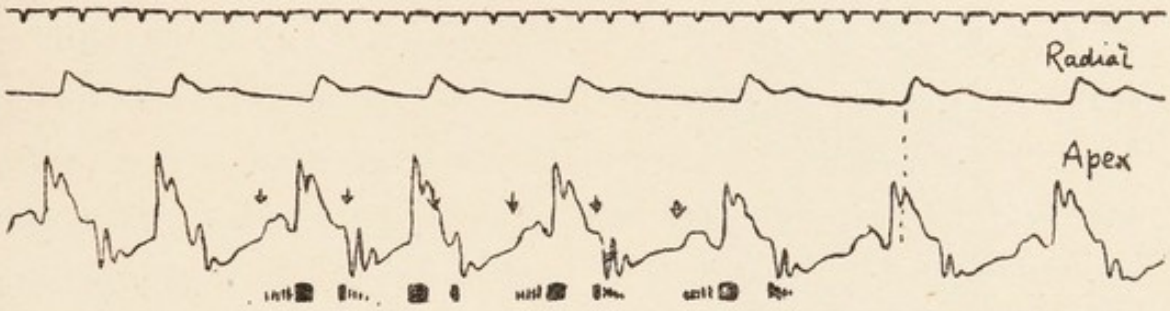


Fig. 18. Arterial and apex curves from a case of mitral stenosis, while there is a transition from dropped beats to 2 : 1 heart-block. Note the arrangement of the diastolic murmurs and their dependence upon those auricular contractions which force blood into the ventricle.

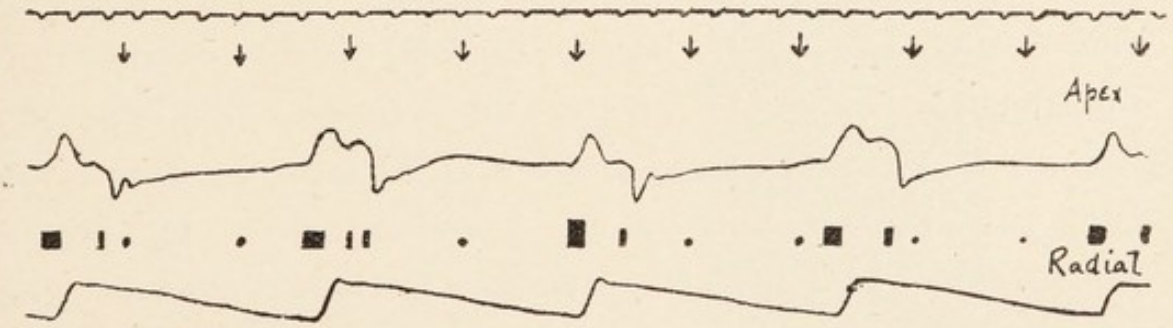


Fig. 19. Apex and radial curves in a case of complete heart-block. The heart sounds are modified by the auricular contractions, which are faintly audible. Where auricular and ventricular contractions begin together the first sound is exaggerated. The pure auricular sounds are shown as dots.

systole, and that in 2 : 1 block the auricle contracts twice as frequently as the ventricle. A more complex arrangement of murmurs is illustrated by Fig. 18. The ventricle beats at first in couples, and at such times the murmur occurs before the first and after the second sound of the first beat of a couple ; the second beat of the couple is accompanied by no murmur, for the single auricular contraction falling in its neighbourhood

coincides with that of the ventricle and forces no blood through the stenosed orifice. Over the last portion of the curve 2 : 1 heart-block is present, and each cycle is accompanied by presystolic and early diastolic murmurs.

In complete heart-block the action of the ventricle is phenomenally slow ; nearly all hearts which beat at rates of 35 and under are affected in this manner. The rhythm is generally quite regular. The rate is almost or quite unaffected by exercise, or by the administration of atropine or amyl nitrite. Each ventricular beat is accompanied by a first and second sound ; in addition, very faint muffled sounds are usually to be heard in the long diastoles. The latter are due to auricular systoles. A sign which is conclusive, and almost always present, is a changed character of the first and second heart sounds from beat to beat. When the auricular and ventricular contractions begin together, the first sound is intensified, and when they fall almost together the first or second sound may be reduplicated (Fig. 19). Definite and recurring change in the intensity of the heart sounds, which proves independent of respiration, forms, in cases of slow and regular heart action, a conclusive sign of complete heart-block. Evidences of the relatively rapid auricular contraction are generally seen in the neck ; small and regular pulsations (Fig. 20, *a* waves) appear in the jugular veins between the beats of the carotid (*c* waves). From time to time a prominent venous pulsation (Fig. 20, *a*/*c*) accompanies the intensified first heart sound, when auricular systole coincides with that of the ventricle and when as a consequence the blood cannot be ejected naturally from the auricle. A periodic waxing and waning of the venous pulsations, independent of respiration, is always highly suggestive

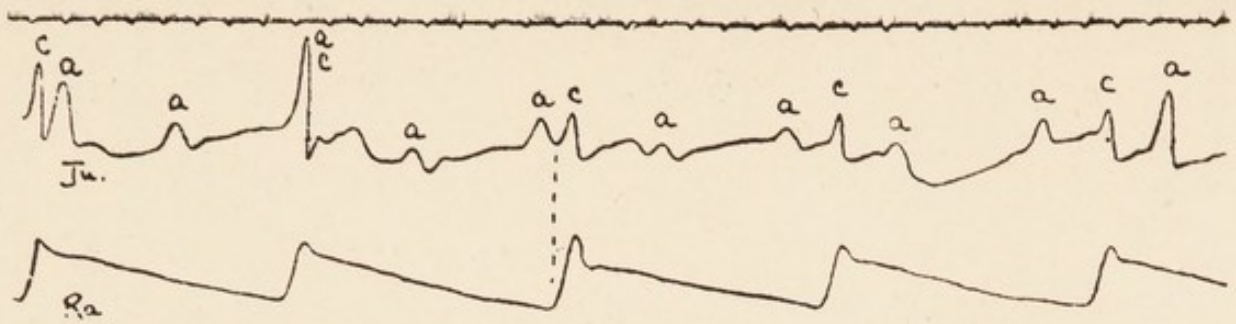


Fig. 20. Curves from the neck (*Ju.*) and radial artery in a case of complete heart-block. There are three pulsations in the neck during each radial cycle. Two of each group of three neck waves result from auricular contractions, *a*, while the third is the result of ventricular systole, *c*. When *a* and *c* fall together an exaggerated wave is produced and is visible as such in the neck. It is due to discharge of the auricular contents into the veins.

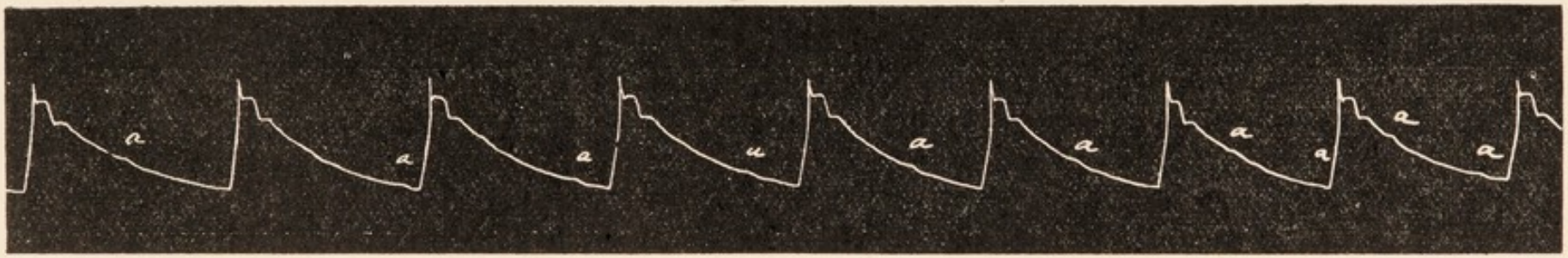


Fig. 21. A sphygmographic curve from the radial artery in a case of complete heart-block. As often happens, signs of auricular systoles (*a*) are visible in the curve and are easily recognised by their regularly changing position as they are traced from one radial cycle to the next. How these little waves are conveyed to the arterial system is not precisely known, but they are commonly conveyed to it.

of complete block. Traces of auricular pulsations upon the arterial curves are also evident in most of the patients from whom full pulse excursions can be obtained (Fig. 21). Where, as in the accompanying figure, the little waves on the downstrokes of the regular pulse beats show a gradual and orderly change of position, moving steadily away from the succeeding radial upstroke, the presence of complete heart-block is certain.

Effects on the circulation and the general symptomatology.

The symptoms suffered by the subjects of heart-block are divisible into two groups. On the one side are those which are especially associated with the condition itself, and on the other are those which result from co-existing disease in other parts of the heart. For disease is rarely limited to the bundle, and generally heart-block is but a local manifestation of a more widespread process. The effects of a lesion which transects the bundle differ from those of a similar lesion in another portion of the musculature in one chief respect ; the lesion so placed produces manifest disturbance ; there is no second strand which may fulfil the functions of that which is destroyed, whereas a defect in the general mass of muscle is hidden by the response of the remaining tissue. As in disease of the nervous system, where large masses of the tissue may be destroyed without gross outward signs of damage, but where a minute morbid focus in a given situation gives rise to obvious and profound disturbance, so it is with the heart. It should be emphasised that heart-block is usually an indication of a far graver condition than simple transection of the bundle ; it is a sign of diffuse invasion of the myocardium.

The symptoms produced by affections of the whole heart musculature do not lie within the scope of this book ; but it is important to state that the presence of heart-block demands an exhaustive study of the subject in whom it appears ; in all instances special attention should be directed to the fitness or otherwise of the heart as a whole. And this caution is not limited to heart-block ; it applies to all departures from the normal mechanism.

The special symptomatology of heart-block may be conveniently approached from two standpoints.

Heart-block of high grade is accompanied by a reduction of the rate of the ventricle, often to a half its former rate. What is the effect of this retardation upon the circulation, and what are the results of the lessened nervous control of rate which accompanies it ? It is certain that thereby a serious burden is imposed upon the efficiency of the heart as a pump ; but nothing is more remarkable than the accommodation of the cardiovascular system to conditions which diverge widely from the normal. Dissociation of auricles and ventricles, and the consequent establishment of a slow ventricular rhythm, is followed by some degree of ventricular hypertrophy. No doubt this increase in the mass of the ventricular muscle makes up in a measure for the loss of co-ordination and of the natural rates. During the long diastoles the blood is squeezed from arteries to veins and a low diastolic blood pressure results ; but the blood pours equally fast from veins to heart, whose chambers, receiving the extra load, expel it into the arteries. Fullness of pulse and high systolic pressure (170-200 mm. Hg) consequently characterise the arterial system when in persistent heart-block there is no lack of healthy cardiac tissue. In evidence

of the adaptability which the circulation as a whole shows to the new conditions, I may cite the case of a patient in whom, judging from the signs and symptoms, the damage to the muscle mass was but little. The patient, a man of 33 years, was known to have had a heart rate of 30 to 35, with occasional accelerations to 48, for 15 years. He was the subject of complete heart-block. There was a little hypertrophy of the heart, but no subjective symptoms. He led, when last seen, a very active business life, and passed in the street would have been judged a perfectly healthy person. There was no circulatory embarrassment, even after strenuous exertion ; he prided himself upon his sprinting power and had recently run in races. An instance of this kind offers a partial answer to the original questions ; the slow pulse of heart-block and the lost regulation of rate do not disable an otherwise healthy heart from performing its full work. In hearts more profoundly affected, the extra burden is less readily borne, but in these it is not easy to dissociate the effects of the new mechanism from those of disease of the remaining muscle.

In the second place, heart-block is responsible for a group of symptoms arising as a direct result of excessive slowing. Reduction of pulse rate beyond certain limits, or the check to the arterial flow for a certain time, is accompanied by grave disorders of nutrition, and the brain is an early and anxious plaintiff. The patient, who exhibits conspicuous pulse slowing in conjunction with fits, falls into the category of Adams-Stokes' syndrome. The higher grades of heart-block, whether of persistent heart-block in which ventricular responses are frequently missed (2 : 1, 3 : 1 ratios, etc.), or of complete dissociation, are frequently

accompanied by temporary periods of excessive pulse slowing or by cessation of the ventricular systole for prolonged intervals. The cause of change in the ventricular rate—the auricles continue to beat at the usual or at an enhanced rate—is not fully understood, and I do not propose to discuss it. The symptoms presented by the patient are intimately dependent upon the degree of heart slowing or upon the duration of isolated periods of asystole. When the pulse falls to 8 or 20 beats per minute, unconsciousness supervenes; suspension of the mental functions is also produced by a single period of asystole of from 3 to 7 seconds' duration. Patients who suffer from the higher grades of heart-block commonly relate a history of brief attacks of giddiness, fainting, temporary loss of consciousness and its dependent accidents. Seen in mild attacks, the subject of them is pulseless and momentarily pale. In severer seizures, where the pulse ceases for 15 seconds or more, there are additional phenomena. The blood is dammed back in the venous system, increasing pallor has cyanosis added to it, the breathing deepens and becomes stertorous; twitching of the face and upper limbs eventuates. The convulsive fit rarely spreads beyond the described area, but it may become more generalised. Urine is not passed, neither is the tongue bitten during the attacks. In most cases the condition is readily recognised by the silence of the ventricle and by the rapid undulations in the veins of the neck, signifying activity of the right auricle. Unexpected death is a by no means uncommon accident amongst the affected, but, considering individual attacks, it is rare. Death occurs after a period of status epilepticus in a number of the patients, and the status consists of repeated seizures of the forms described.

As a rule the patient has no warning of an impending syncopal or epileptic attack ; though on occasion he or his medical attendant may be informed of the approaching danger by a change in the heart's action, for example, by the occurrence of unusual ventricular slowing. Such sensations as the patient may experience at the commencement of long seizures are similar to those accompanying a brief cessation of the heart beat, and consequently do not properly constitute an aura.

The prognosis.

Heart-block in itself does not kill ; those who suffer or have suffered from it mostly die with the usual symptoms of general heart failure. Let me be clearly understood in this statement. Heart-block and the Adams-Stokes' syndrome are by no means synonymous terms ; the majority of patients who exhibit heart-block never have fits, for the higher grades of heart-block are not common. Lesser grades of heart-block are common in conjunction with rheumatic heart disease ; they produce no specific symptoms. Moreover, the syncope is not of necessity the cause of death even in chronic heart-block of high grade.

The prognosis in heart-block has to be dealt with from several points of view. In the first instance, let us consider the common variety, the milder grades of heart-block such as are associated with rheumatic heart disease (prolonged *As- Vs* intervals or " dropped beats "). Where such heart-block is *persistent*, there are usually a number of physical signs in addition to those dependent upon disturbed rhythm or sequence ; they are the signs of heart disease, muscular or valvular, in its several and universally recognised forms. Heart-block is often the least prominent phenomenon in

these patients, who often suffer from mitral stenosis. The only question that I raise is as to the manner in which heart-block affects the prognosis in these cases. It should be regarded as an evidence of myocardial damage, not necessarily limited to the bundle, but probably diffused throughout the heart. My experience of such cases is that they are serious; in fact, most of those I have seen are dead, though they have not died of heart-block. But *temporary* heart-block of mild grade is not uncommon during the febrile attacks to which rheumatic heart subjects are liable; it occurs also in pneumonia and typhoid fever, etc. The appearance of this abnormal mechanism is of importance, for it may be the only outstanding sign of myocardial damage. Whenever it complicates an acute infection it consequently dulls the prognosis; at the same time it should be understood that the normal mechanism is usually recovered. Occurring as an accompaniment of fever in a patient who has rheumatic heart disease, it should be regarded as an outward sign of an isolated injury which, if often repeated, eventually so weakens the muscle that life is no longer supported.

Where the higher grades of heart-block are present, the prognosis is based upon two chief considerations. The general evidence of the integrity and fitness of the muscle as a whole should be weighed first. The fits, especially their frequency and severity, are next taken into account; a number of the patients are free from them; others are in constant peril; and it is not easy, nay it is often impossible, to predict the ultimate effects of syncopal attacks or severer crises in a given case. Those patients especially who have progressive lesions, and those in whom partial is eventually

converted to complete and permanent dissociation, must pass through a time of particular danger; for, during the passage from one grade of block to the other, fits are very common and the period of passage may not be a short one. It is useful to remember also that those who have partial heart-block are more prone to fits than those in whom the obstruction is complete. Uncertain in both incidence and effect, the fits always dictate a cautious prognosis.

Regarded in its entirety, persistent heart-block of high grade is a grave condition. It is usually complicated, and then a few years generally close the scene. Nevertheless, some, and especially the younger patients, survive for many years, in comparative and absolute comfort. These are patients in whom the mass of heart muscle is comparatively healthy and in whom fits are rare or absent.

The treatment.

In all cases of heart-block a careful inquiry into the causation is to be undertaken, it if is not obvious. Syphilis as a possible cause in middle life is to be kept in mind especially.

Persistent heart-block of the milder forms requires no immediate treatment, but is an indication for repeated examination of the patients who show it. As such patients usually require treatment for the general condition of the heart, constant supervision is not difficult. Digitalis medication will frequently increase the grade of block. The increase of block should not deter digitalis administration, for it is not in itself detrimental; the drug or its allies may be given without restraint, and some seem to receive benefit from it.

When the *abrupt onset of partial heart-block* is observed, it is, as I have said, an index of active mischief. The patient should remain in bed and should be thoroughly searched for the provocative cause, which is attended to when found. The acute infections are suitably treated. Rheumatic patients are treated with salicylates, and scrupulous attention is paid to the hygiene of the mouth and throat. If, after the subsidence of remaining symptoms, the block remains and persists for several weeks, the patient is treated along the lines indicated in the previous paragraph. Heart-block in itself does not call for rest in bed or other interference, though a suspicion of an active or progressive lesion does.

Full exposure to open air has been advocated and deserves a thorough trial.

The *higher grades of heart-block* are usually chronic and stationary and the habits of the patient should be governed by his general fitness. Most patients of this class are up and about, and are able to undertake many of their ordinary duties ; yet it is only exceptionally that real bodily activity is either possible or permissible. Here again a suspicion that the lesion is active or progressive calls for rest and careful watching. A history or sign of syphilis constitutes an imperative demand for thorough and appropriate treatment, and in some cases success has attended the administration of mercurials and iodides or intravenous treatment.

All those who have *fits* should be warned of the danger which they run from accidents during these attacks if they do not appreciate it fully. Not a few have lost their lives by falling heavily or in traffic and suffering mortal injury. In many cases the fits occur in groups, and additional precautions are required until such attacks cease. Most patients have brief

warnings of the onset of unconsciousness, and, if advantage is taken of these, less risk is incurred.

A careful inquiry for causes predisposing to the fits may elicit a history of gastro-intestinal disturbance or over-exertion, upon which it is well to act.

For the fit when it is present I know of no remedy which is of avail to increase the pulse rate and restore the unconscious patient. Many drugs have been administered with this end in view, and the list includes oxygen, strychnine, strophanthine, digitalis and amyl nitrite. They appear to have no appreciable effect. Atropine is said to have abolished fits in isolated instances. As a rule it is contra-indicated. Adrenalin has been suggested ; I know not if it is of value.

CHAPTER IV.

PREMATURE CONTRACTIONS OR EXTRASYSTOLES.

Definition.

Contractions of the heart which disturb the rhythmic sequence by appearing early and in response to impulses newly formed in the musculature.

The nature of premature contractions.

A clear conception of disordered heart action can be attained only by those who are perfectly familiar with the normal beating. The orderly sequence of muscle movements, which constitute the normal heart cycle, is propagated, as I have already stated, from a single impulse born in the sino-auricular node. The contraction, starting from the mouth of the superior vena cava, travels rapidly through the auricle, reaches the auriculo-ventricular node and traverses this node and the bundle which is its continuation ; it is distributed in an orderly manner amongst the mass of ventricular fibres in which it ends. The normal rhythm of the heart consists of a regular sequence of such beats, so that auricle and ventricle contract in proper time relation to each other. Each stimulus elaborated at the sino-auricular node requires a certain time of preparation, and this time of preparation is very constant in given circumstances. It is relatively long, reaching nearly two-thirds of a second when

the heart is beating at a normal rate. Indeed it is the time of impulse preparation which controls the rate of a normally beating heart. A second characteristic of physiological impulses is regular repetition. Each impulse belongs to a regular or rhythmic series.

The premature contraction or extrasystole differs from the physiological beat in two fundamental respects. Firstly, the impulse which gives rise to it appears to require little or no time for preparation. It is to this quality that the pathological contraction owes its prematurity. Secondly, the pathological impulse is not necessarily one of a rhythmic series, and upon this character the usual isolation of the pathological contraction depends. Premature contractions originate abruptly and may spring from the auricle, from the ventricle, or from the tissues which unite these two contractile structures. For ordinary clinical purposes it suffices at the most, if we remember the two chief classes of premature beat, the auricular and the ventricular.

If, while the heart chambers are beating in a normal and sequential manner, a pathological impulse arises in the ventricle, the ventricular beat which it awakens disturbs this rhythm by anticipating the next rhythmic beat, whence comes the term "premature contraction." It disturbs the sequence of ventricular contractions in a definite manner. Excepting the premature impulse, the ventricle is dependent for its stimuli upon the impulses which descend to it from the auricle. Consequently, after the disturbance produced by a single premature beat, the ventricle rests until a rhythmic auricular impulse reaches it. If the accompanying diagram (Fig. 22) is studied, it will be seen that for the first three cycles the ventricle follows the auricle in contraction; a

premature beat (*p*) is then interposed and as a consequence the oncoming auricular impulse, represented by the broken line, arrives while the ventricle is already in a state of contraction. Being in contraction, the ventricle shows no response, its muscle is in the "refractory" state. The dominance of auricular impulses is reasserted during the succeeding cycle. Thus, the original sequence is restored by the fundamental heart rhythm which proceeds, unheedful of the disturbance. The ventricular contractions, subsequent to the disturbance,

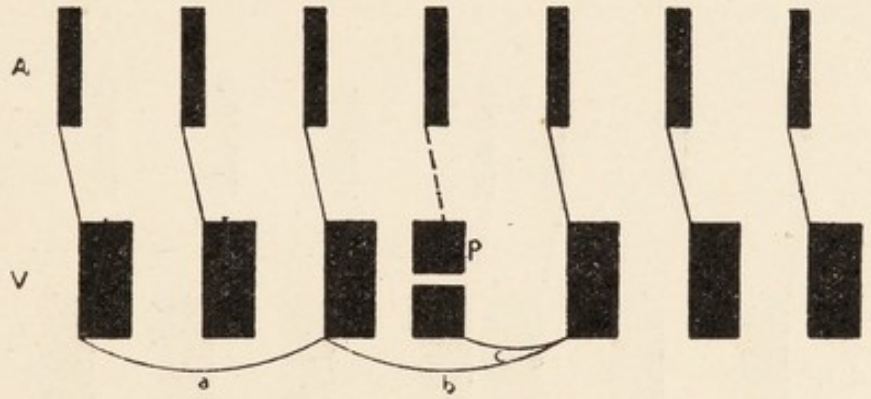


Fig. 22. The disturbance produced by a premature ventricular contraction (*p*) is represented diagrammatically. The auricle beats regularly throughout. The ventricle responds to six auricular impulses. The impulse of the central auricular systole is ineffectual, for it falls while the ventricle is in premature systole. The abnormal origin of the ventricular beat is indicated by the break in its centre. Note the equality in the lengths of periods *a* and *b*. *c* is the compensatory pause.

fall at points which may be accurately anticipated; the period of the disturbance (*b*) is exactly equivalent to the length of two complete cycles of the normal rhythm (*a*). The diastole which follows the premature ventricular beat is long; the ventricle is waiting. The length of this diastole (*c*) is such as to compensate for the brevity of the diastole which precedes it; consequently it has been termed the "compensatory pause."

When a premature impulse originates in the auricle the events are somewhat different. The premature con-

traction of the auricle, which it calls forth, is followed by a similar and parallel disturbance in the ventricle (Fig. 23), for the ventricle responds to each auricular contraction, normal or abnormal. In all but exceptional instances, too, there is a disturbance of the fundamental heart rhythm; the premature contraction (*p*) is followed by a long pause, but the whole period of the disturbance (*b*) is not equivalent, as in the case of the premature ventricular beat, to two full cycles of the normal rhythm (*a*). The original sequence is not restored.

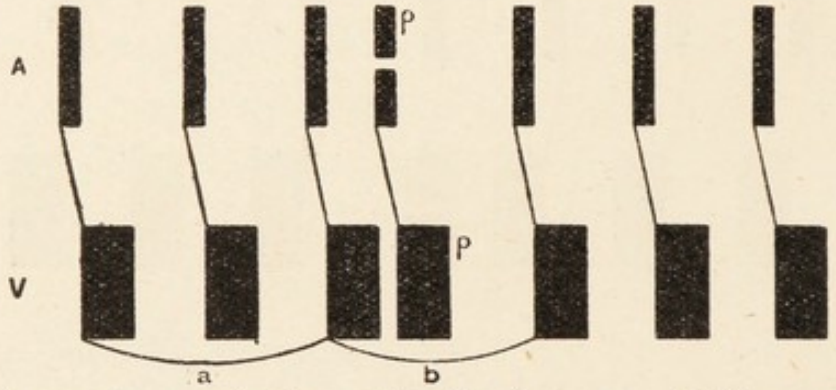


Fig. 23. A premature auricular contraction is represented diagrammatically. The auricular rhythm is disturbed by the abnormal auricular beat (*p*); the disturbance in the ventricular rhythm is parallel, for each auricular systole yields a ventricular response. The rhythm of the whole heart is dislocated, the period *a* is longer than the period *b*.

Etiological and pathological relations.

Age and sex. Premature beats have been recorded at all ages from a few weeks to old age. During the first decade they are rare. Their incidence in an age table is actually heaviest between 50 and 70 years; if the age distribution of the populace is considered in conjunction with this fact, it becomes evident that they are essentially connected with advancing years.

Premature contractions are twice as common in men as in women.

Associated conditions and provocative factors. It should be remembered that any statistics compiled to show the relations of premature contractions to associated conditions and infections suffer from a defect. Those cases which exhibit frequent and persistent premature beats preponderate in the tables ; for in these circumstances they are conspicuous, while if they are scarcer they often fail to attract attention. *It is probable that the majority of people who live to middle life or advanced years are affected in this manner at some time or other.* Amongst patients who attend out-patient departments or are admitted to the wards of general hospitals, frequent and persistent premature contractions are most common in those who exhibit definite symptoms and signs of cardiac disease. They are often found in association with aortic incompetence and mitral stenosis ; an even larger number of curves is collected from patients who present signs of degenerate heart muscle, as evidenced by enlargement and symptoms of muscle insufficiency in the absence of gross valve lesions. In yet another, and by far the largest group of patients, no evidence of impairment of the heart, leaving the irregularity out of consideration, can be discovered.

Extrasystoles are not uncommon accompaniments of infectious disease ; thus, in scarlet fever and diphtheria they may cause irregularity of the child's pulse, and local pus infections are not infrequent associations.

Of the factors which appear to be predominantly associated with them, gross lesions of the heart stand first. Otherwise an inquiry into the habits, history and state of the patient throws but an obscure light upon the causation. A history of rheumatic infection is certainly common ; it was present in one-third of the cases in the following series. In

Premature auricular contractions.

Cardiac group.		*Remainder.*	
Myocardial degeneration ..	12	Bronchitis and emphysema..	2
Mitral stenosis	5	Pulmonary tuberculosis ..	2
Aortic stenosis	2	Dyspepsia	1
		Lumbago	1
		Exophthalmic goitre.. ..	1
		Apparently healthy otherwise	4
	19		11

Premature ventricular contractions.

Cardiac group.		*Remainder.*	
Myocardial Degeneration ..	24	Tuberculosis (lungs & pleura)	5
Aortic disease	12	Bronchitis and emphysema ..	2
Mitral stenosis	13	Gallstones	1
Angina pectoris	7	Gangrene of toes (senile) ..	1
Bright's disease and granular kidney	5	Epilepsy	1
		Lipoma of neck	1
Arteriosclerosis	3	Fractured skull	1
Acute endocarditis	2	Abdominal tumour	1
Aneurism	1	Exophthalmic goitre.. ..	1
		Gastric ulcer	1
		Dyspepsia	1
		Apparently healthy otherwise	8
	67		24

young adults excessive tobacco smoking is recognised as an exciting cause of their appearance. Digitalis and its allies are not uncommonly responsible, when the patient is under the full influence of these drugs. There are also clinical associations between premature contractions, raised arterial pressure and digestive disturbances, but these are not fully understood.

Many things affect the frequency of premature contractions. Fatigue, subsequent to exertion, is provocative in all who are predisposed. The influence of heart rate is especially noteworthy. Hearts beating at 100 per minute and over are not often disturbed, and premature contractions are very rare when the heart rate exceeds 120. Fever usually rids the pulse of this form of irregularity, and so also does any cause which notably accelerates the pulse rate. Thus they are abolished during exercise and for a short period afterwards, but during the period of slow heart

action which often follows exercise, they are frequent in the predisposed. As we shall subsequently see, this knowledge may often be used advantageously to induce premature beats in patients predisposed to them. Suspension of respiration for a period compatible with comfort often suffices. The pathological beats are in evidence either in the apnœic stage or shortly after the resumption of respiration. Posture is often a potent factor. Patients who exhibit numerous premature contractions while standing may soon lose them in recumbency, and this happens despite a slight decrease of heart rate in the last position. In other patients, pressure upon the abdomen may abolish them.

Observations go to show that extrasystoles are not caused by nervous impulses playing upon a healthy organ; but it seems to be true that, if the heart manifests extrasystoles from time to time, nervous impulses, especially those of sympathetic origin, may exaggerate the tendency.

The recognition of premature contractions.

The work accomplished by premature beats is small, because the periods of rest that precede them are short. They may or may not *raise the aortic valves*. Accompanying the premature beat, a feeble pulsation or a prolonged pause is noted in the arterial pause; auscultation reveals early first and second sounds when the aortic valves are forced, but only an isolated and premature first sound if the ventricular pressure fails to top the arterial. The consequent grouping of sounds in threes and fours is comprehended when the nature and degree of the corresponding arrhythmia are discerned. The commonest arrangements of pulsations and sounds are described in the following paragraphs, and are illustrated by the accompanying diagram and tracings.

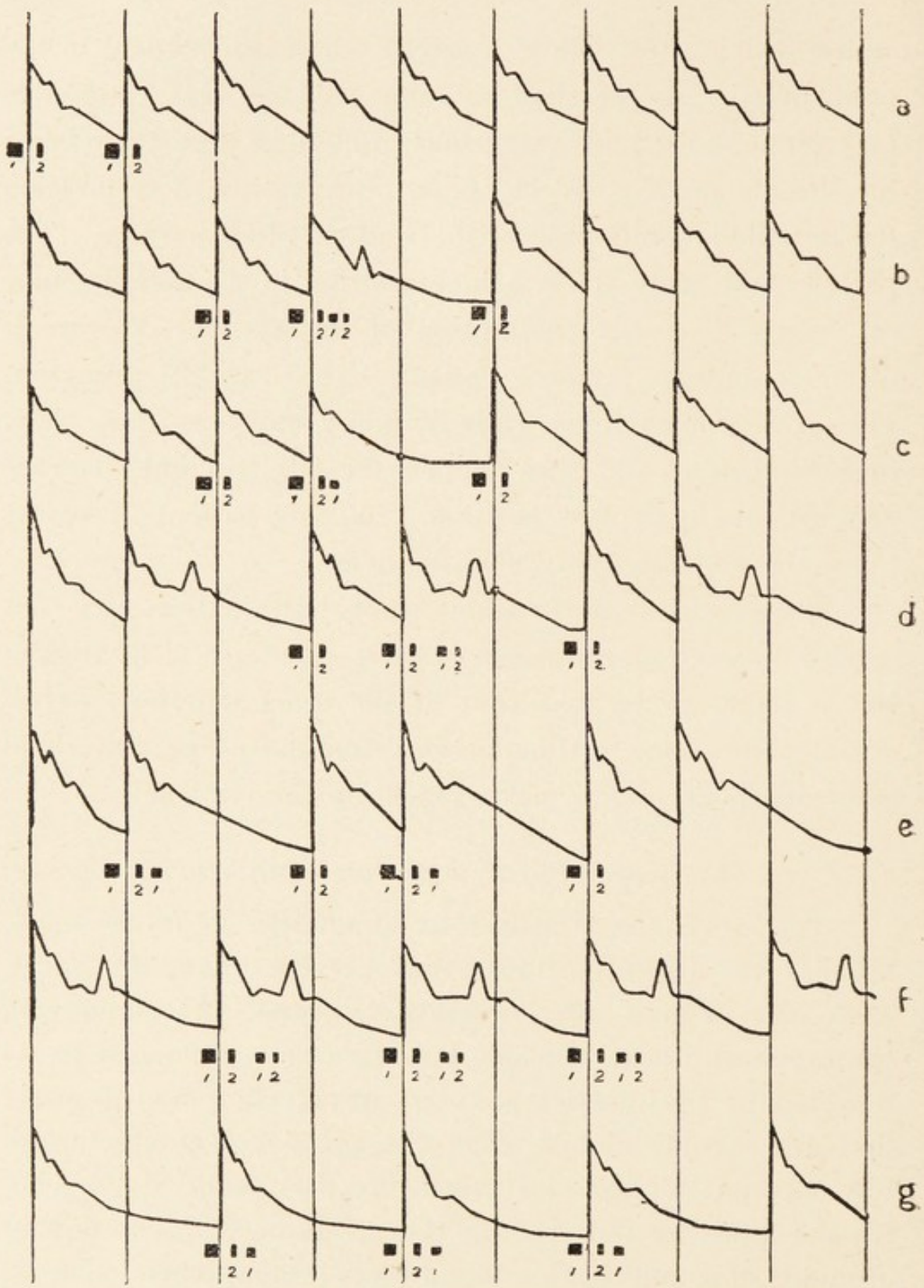


Fig. 24. A diagram showing common disturbances of the arterial pulse and heart sounds when premature ventricular contractions are present.

(*a*) Normal rhythm ; (*b*) Occasional premature beat, which affects arterial pressure ; (*c*) Occasional premature beat, which fails to affect arterial passage ; (*d*) Premature beat replacing each third normal beat and affecting arterial pressure ; (*e*) Premature beat replacing each third normal beat and failing to affect the arterial pressure ; (*f*) Premature beat replacing each second normal beat and affecting the arterial pressure ; (*g*) Premature beat replacing each second normal beat and failing to affect arterial pressure. The heart sounds occur in groups, and the groups are of four or three, according as the aortic valves are raised or remain at rest when the premature beat occurs.

In the succeeding paragraphs I have sub-grouped the signs according as the premature beat (α) raises or (β) fails to raise the aortic valves.

1. When a systole of a regularly beating ventricle is replaced by a premature beat, this abnormal contraction is accompanied by an early apex thrust and by (α) a weak arterial wave and two extra sounds, which, together with those of the preceding rhythmic beat, form a group of four (Figs. 24 *b* and 25) ; or by (β) an intermission in the arterial pulse and one extra sound, forming with the sounds of the preceding rhythmic beat a group of three (Fig. 24 *c*).

2. When each third beat of the regular ventricular rhythm is replaced by a premature beat, we find a grouping of the apex thrust in threes, of which the third beat in each group is premature. The arterial beats (α) are grouped in threes,* with groupings of the apical sounds, so that two normal heart sounds alternate with a group of *four* sounds (Fig. 24 *d*) ; or (β) are paired, with groupings of the apical sounds, so that two normal heart sounds alternate with a group of *three* sounds (Figs. 24 *e* and 30).

3. Premature beats which alternate with rhythmic beats give rise to pairing of the apical thrusts (Figs. 27, 28, 32), and to (α) pairing of arterial beats of which the second stroke is weak, and to groupings of heart sounds in fours (Fig. 24 *f* and 29) ; or to (β) halving of the rate of the arterial pulse, and heart sounds in groups of threes (Figs. 24 *g* and 28).

To decide whether the premature beat arises in auricle or ventricle is not usually possible without suitable recording instruments ; neither is the differentiation of any great clinical consequence.

* Premature beats may also be responsible for groups of three arterial beats when they replace each fourth rhythmic beat (Fig. 26).

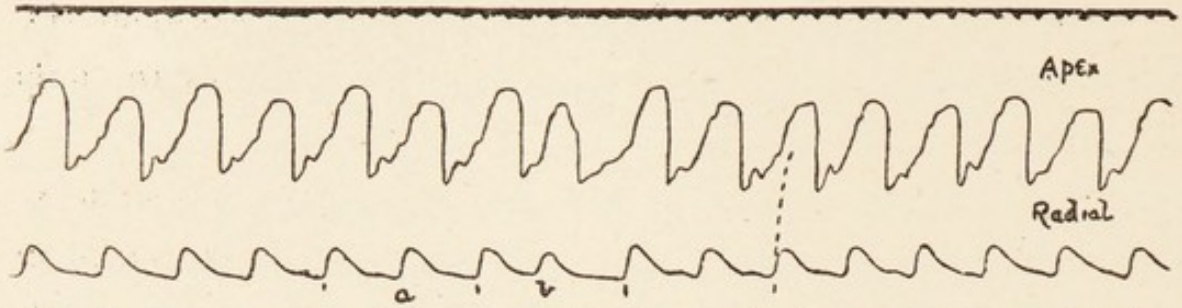


Fig. 25. Apex and radial curves, showing a single premature ventricular contraction. The intervals *a* and *b* are equal.

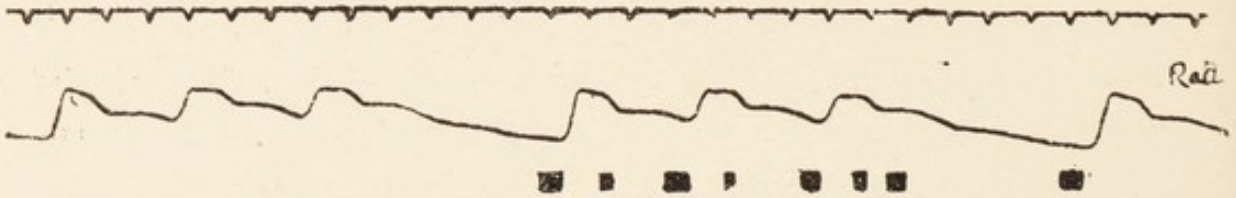
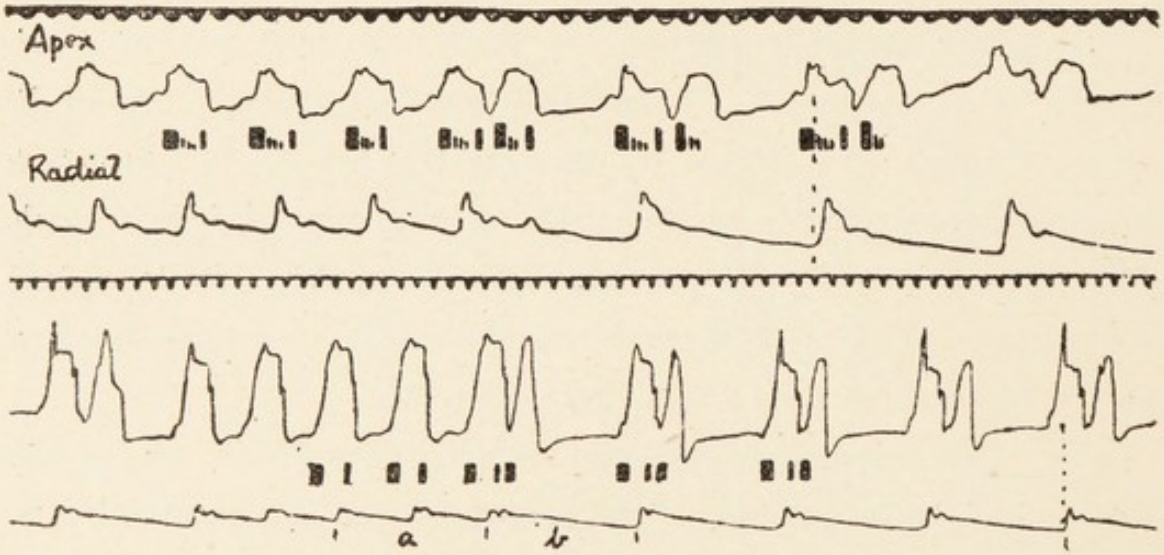


Fig. 26. Radial curve and heart sounds in a case in which premature ventricular contractions replace each fourth normal beat. The premature beats fail to affect the pulse.



Figs. 27 and 28. Apex and radial curves and heart sounds. The normal mechanism passes into one in which premature ventricular contractions replace alternate normal beats. Fig. 27 is from a case in which mitral regurgitation was present. In Fig. 28 the intervals *a* and *b* are equal.

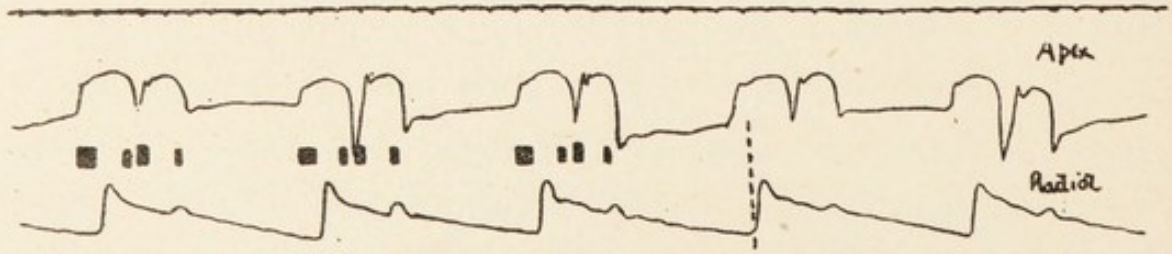


Fig. 29. Premature ventricular contractions replacing each second normal beat. The heart sounds are grouped in fours; the pulse is of the form termed *pulsus bigeminus*.

When an occasional premature beat occurs, the indications of its *ventricular* origin are as follows : (*a*) There is no disturbance of the fundamental heart rhythm. The presence of this phenomenon may sometimes be elicited, in feeling the radial pulse, by anticipating the points at which the rhythmic beats, following the disturbance, ought to fall to carry on the original rhythm ; but it is usually easier to detect disturbance of the rhythm than to exclude it by this method. A strip of radial curve by itself is usually sufficient to distinguish one from the other ; in the instance of the premature ventricular beat the full period of the disturbance is equal to two normal cycles (Fig. 25). In the instance of the premature auricular beat the full period is less (Fig. 31). (*b*) There is often a prominent jerk and swelling of the veins of the neck (Fig. 30) at the time of the premature ventricular beat. This is brought about in the following fashion. The ventricular beat, falling prematurely as it does, coincides with a rhythmic auricular contraction, so that the two heart chambers are in systole together (see Fig. 22). As a consequence of this simultaneous contraction, the auricle fails, for a single cycle, to empty itself into the ventricle, and pumps the blood back into the veins. (*c*) Synchronism of the premature ventricular beat and the rhythmic auricular contraction often exaggerates for a cycle the corresponding first sound.

When the premature beat follows pairs of normal beats or alternates with normal beats, signs *b* and *c* may be present, but *a* is usually valueless unless a transition from a period of disturbance to a period of normal rhythm is graphically recorded (as in Figs. 28 and 32). A comparison can then be instituted between the lengths of the disturbed and undisturbed heart cycles. Thus, in Fig. 28 the long cycles are exactly twice the length of the short ones ; *a* is equal to *b* ; premature contractions arising in the ventricle have created an exact halving of pulse rate. In Fig. 32 the pause following the premature contraction is not compensatory ; *a* is longer than *b* (see Fig. 23).

The effect of premature beats upon the auscultatory signs, when murmurs are present, are manifold ; yet most of

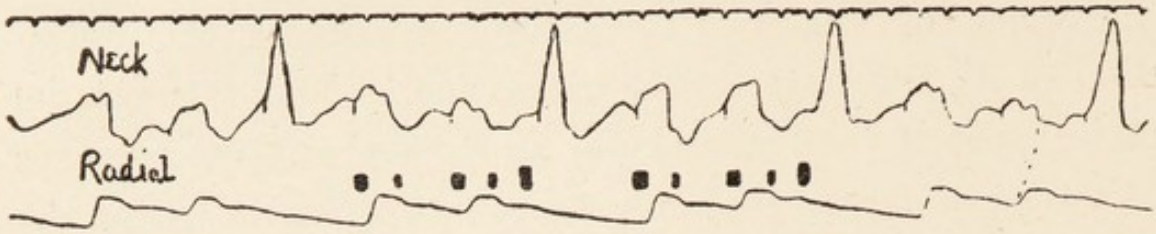


Fig. 30. Curves from the neck and radial artery. Premature ventricular contractions replace each third normal beat, but do not affect the pulse. An exaggerated first sound and a prominent wave, easily visible in the neck, accompanied each premature beat ; these phenomena result from simultaneous contraction of auricle and ventricle.

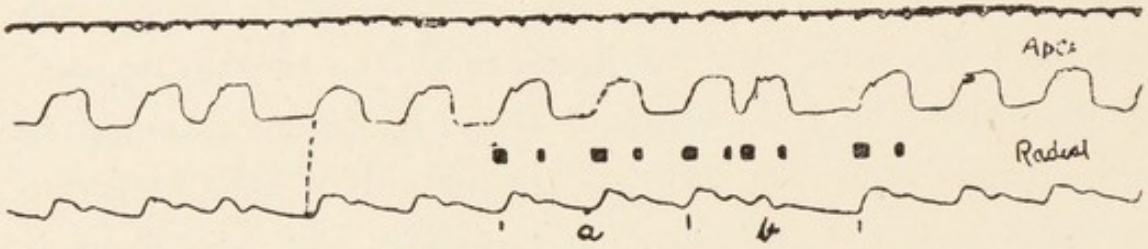


Fig. 31. Apex and radial curves showing occasional premature auricular contractions ; *a* is greater than *b*.

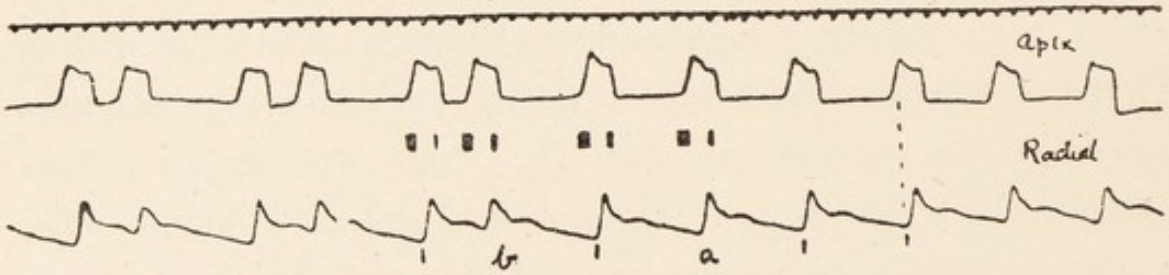


Fig. 32. A " bigeminy " or coupling of heart beats, resulting from premature *auricular* contractions, passing into the normal rhythm ; *a* is greater than *b*

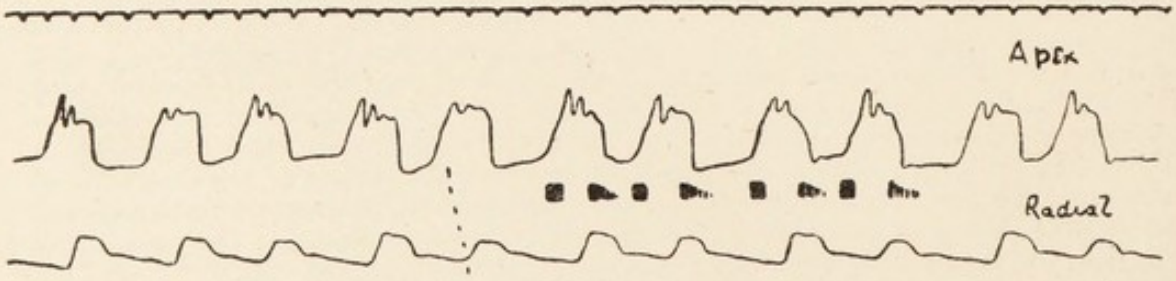


Fig. 33. A bigeminy, resulting from premature auricular contractions. The beats are paired in apical and radial curves. Aortic regurgitation was present.

them can be foretold if the general principles are grasped. A systolic mitral murmur will be found with the premature as well as with the rhythmic beat (Fig. 27), but it is usually short and may be absent. In aortic disease, a systolic or diastolic murmur is present at the base of the heart when the premature beat raises the aortic valves (Fig. 33). On the other hand, in mitral stenosis, a presystolic mitral murmur is usually absent whether the premature beat is auricular or ventricular, but in the former instance, it is often replaced by a presystolic sound. The absence of the presystolic murmur in the case of the auricular beat is attributable either to weakness of the premature contraction or to its coincidence with the preceding ventricular systole.

More complex heart sounds are heard when a premature beat raises the pulmonary but not the aortic valves, as sometimes happens; the second pulmonary sound occurs, but the second aortic sound is absent. This phenomenon has been erroneously ascribed to hemisystole, the presence of the second sound of the right heart and the absence of that of the left heart being taken as evidences of activity and quiescence of the respective ventricles.

The subjective phenomena which accompany premature contractions.

In a very large number of those affected, the disturbances of heart rhythm pass unnoticed. On the other hand, premature beats not uncommonly cause what patients term "palpitation." The symptom is more prominent in young subjects, especially those of female sex and those afflicted by nervous instability. When numerous, they sometimes occasion actual distress; by calling attention to the heart,

they often induce anxiety. The sensations experienced are exaggerated by depression of the general health, by fatigue and by emotion. They are often more noticed after the patient retires for the night, after excessive smoking, after a heavy meal, or after exertion.

As a general rule the premature contraction itself passes unperceived; the long pause which follows awakens a sense of uneasiness or oppression in the chest, or a feeling of void, while the succeeding contraction of the heart is accompanied by consciousness of shock to the chest wall and frequently by fullness or gripping in the throat. Patients in whom these sensations are vivid sometimes swallow, cough, or inspire as soon as they experience them. When a number of premature beats succeed each other at short intervals, and consciousness of them is marked, anxiety may be profound, and faintness, coldness of the extremities and even sweating may result.

The prognosis and treatment.

It should be clearly understood that, in speaking of the prognostic value of premature beats, I speak of these beats without reference to the conditions with which they are associated. That when frequent and persistent they often accompany grave affections of the heart will be evident from a study of the tables already given; but this fact does not affect the question discussed.* The associated lesions give prognostic indications of their own; our inquiries are as to whether a heart, which presents no other sign, can be regarded as healthy and as to whether, in the case of an unhealthy heart, the prospect has an added gloom.

* Some fail to grasp this point, and I would enforce it by a simple illustration. Scars on the tongue of an epileptic do not influence the prognosis of the disease.

It must be admitted that all such beats are evidence of a pathological condition and that the pathological process has its seat in the tissues of the heart. The presence of premature contractions is an indication of disturbance of cardiac nutrition, whether temporary or permanent, but it is an aspect that should not be allowed undue prominence. Very many people are temporarily affected by premature beats which do not reappear, while the heart manifests no sign of further damage, either at the time or afterwards. In such instances it is impossible to suppose that the disturbance of the cardiac function has been more than transient or that the nature of it has been serious. Observations and inquiry also teach that they may be present constantly and for long periods, and that those who manifest them may do so from an early to a good old age, such patients never showing any other sign or symptom of cardiac disability. It may be said, therefore, that in themselves premature beats cannot be regarded as evidences of serious involvement of the heart muscle, although such involvement is often found in conjunction with them.

The question can be regarded from another standpoint. The premature contraction, when present and frequent, inevitably increase the work of the heart, but the amount of the added burden is not easy to ascertain. It is probably not weighty, for where the muscle is evidently compromised and frequent premature contractions occur periodically, little change in the condition of the patient can be detected from time to time, and serious embarrassment of the circulation as a direct result of them is only suspected on rare occasions.

Because they frequently consort with relatively grave cardiac maladies, their detection demands a close scrutiny of

the heart from other points of view. When after such scrutiny, no further symptoms or signs attributable to the heart are detected, they may be neglected in the prognosis. It is also to be observed that if additional and significant symptoms or signs are found, the prognosis should be based on these, the extrasystoles again become negligible. In other words, they serve a purpose in diagnosis by directing attention to the heart, but are of little or no value in prognosis.

I may summarise in the statement that premature contractions have a relatively insignificant import, as compared to many forms of cardiac irregularity.

Their detection should not be allowed to cripple or hamper the patient who is the subject of them.

The first standpoint of treatment is already indicated. The presence of premature beats does not call for a limitation of bodily exercise ; it should not prejudice the vocation or pastime of the patient. Restrictions are necessitated only where other signs render them advisable, or where some particular act or occupation is definitely known to originate symptoms of a distressful kind. The anxiety to which the beats conduce in some subjects may be materially allayed by reassurance. No drugs are known which influence their prevalence ; digitalis as a direct measure is contra-indicated. The symptoms are usually masked or considerably modified by the bromides administered in doses of from fifteen to thirty grains or more a day ; and these drugs are especially useful in tiding a nervous or excitable patient over a period of disturbance.

In patients who complain of these beats and of this alone, the most suitable line of treatment is attention to general hygiene and to that of the gastro-intestinal tract in particular.

CHAPTER V.

SIMPLE PAROXYSMAL TACHYCARDIA.

Definition.

Paroxysmal tachycardia is a term which has been and still is applied to several distinct phenomena. It will be of material assistance if I restrict my description to the simple form and define it as a condition in which from time to time the normal mechanism is interrupted by a series of rapid and regular beats, varying in rate between 100 and 220 per minute,* the series starting and ending quite abruptly.

The nature of simple paroxysmal tachycardia.

It has been said that the normal pacemaker of the heart lies at the union of the superior cava and right auricle. The usual rate at which the rhythmic impulses proceed from this focus is about 72 per minute in the adult. If a new centre of impulse formation develops in any portion of the heart wall, and this centre forces responses at a rate surpassing that of the normal rhythm, then, while it is active, the new centre dominates the movements of the whole heart. Such are the paroxysms which we are about to study ; they consist of sudden accelerations of heart rate in response to new and pathological impulses. The paroxysms may be regarded

* In so defining it I have purposely excluded all accelerations of normal or sinus rhythm, for these are dependent upon altered innervation. I have also excluded two forms of tachycardia, which are related to that described in the present chapter ; one, which is regular, but in which the auricular rate exceeds 220 per minute (see Chapter VI) ; the other, which is irregular (see Chapter VII).

both clinically and pathologically as formed essentially of a regular series of extrasystoles. The new impulses are elaborated in a single focus, whence the regularity of the series, and this focus lies, usually or always, at a point which is removed from the pacemaker.* Fig. 34 opens with three normal heart beats, and the fourth auricular contraction (p) is premature. Up to this point the diagram is identical

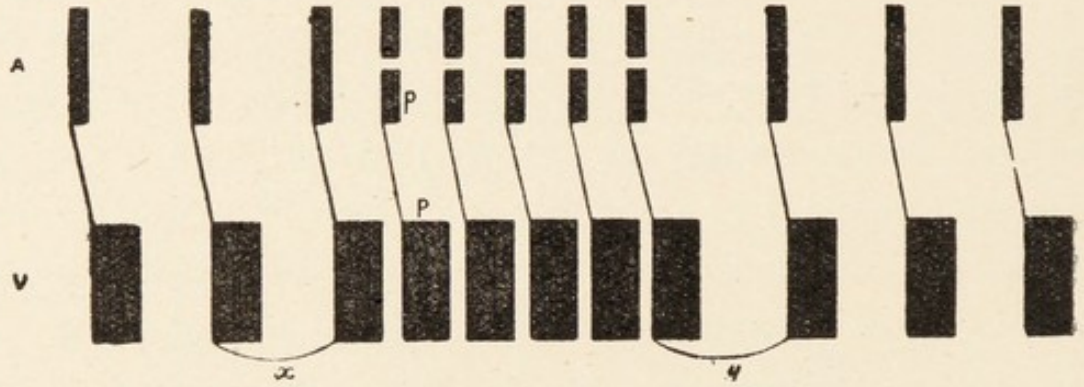


Fig. 34. A diagrammatic representation of a short paroxysm of tachycardia arising in the auricle. The abnormal auricular beats are broken in their centres. Each yields a ventricular response. The first abnormal beat occupies the same position in relation to preceding events as does that of Fig. 23. The short paroxysm ends in a cycle y. y is longer than x.

with that shown in Fig. 23† ; it differs from the earlier picture in the repetition of the abnormal contraction, five such beats following each other in rapid and regular succession. In each instance the ventricle responds. The paroxysm terminates, and its end is marked by a cycle (y) which is longer than the normal cycle (x)‡ ; the pause which follows a paroxysm derived from the auricle has generally the same length as that which in the same case succeeds an isolated premature contraction (see Fig. 23).

* A statement which is based upon electro-cardiography.

† In both diagrams I have broken the auricular rectangle, to emphasise the abnormal birthplace of the pathological contractions.

‡ The interval (x) has been deliberately chosen at the onset of the paroxysm, because the restored rhythm of the old pacemaker is often slow for a few cycles. For simplicity this retardation is not figured, but it will be referred to again at a later stage.

How important a clear conception of this disturbance is will be evident, for the nerve control of a new centre of impulse formation is not known from analogy ; as a matter of fact, the new rhythms show only limited subordination to vagal and sympathetic control.

The sites in which the new rhythms develop are numerous ; the abnormal focus is generally seated in the auricle, and the usual sequence of contraction is consequently maintained in the heart chambers ; but it may be ventricular, and the auricle then responds reversely to the ventricular beats. The chief features of the mechanism will be sufficiently impressed by a closer examination of the commoner auricular variety. The diagram exhibits a paroxysm of five beats ; this short series displays both onset and offset. Actually the attack may last a few seconds or a week or more ; whatever its length the mechanism is constant, but the symptoms vary with the duration.

The extreme ranges of rate in this the simple form of paroxysmal tachycardia are from 110 to 220 per minute ; during most paroxysms the heart contracts 160 to 200 times per minute.

Etiological and pathological relations.

Age and sex. Paroxysmal tachycardia may occur at any period from childhood to old age.

Instances have been recorded in quite young children ; but these are rare. The highest incidence is between the ages of 20 and 30.

The disorder is a good deal more frequent in men than in women.

Heredity has been blamed, but the evidence is insufficient to show that it has any direct influence.

Relations to infective disease. In quite half the cases no history of previous illness, other than perhaps children's ailments, can be traced. Rheumatic fever is the only infection which is at all common. Occasional instances appear to have followed immediately upon malaria, measles, pneumonia and scarlet fever; a few of the patients have been syphilitic.

Associated conditions. Most cases of paroxysmal tachycardia show no sign of valve lesion, and in a large number of the patients there is little or no evidence of dilatation during the intervals between the paroxysms.

Nevertheless, many of them exhibit a limited field of cardiac response and become breathless with slight exertion. Taking enlargement, in the absence of valve lesion, undue breathlessness upon exertion and the subsequent development of more serious signs of cardiac failure as evidences of degeneration of the mycardium, I have placed ten of the patients in a corresponding group in the accompanying table. The only valve lesion which figures prominently is mitral stenosis, being present in ten of the cases.

Paroxysmal tachycardia and associated conditions.

Mitral stenosis	10
Myocardial degeneration	10
Arterial disease (with and without angina)	4
Aneurism (thoracic)	1
Renal disease and cardiac enlargement	3
Early pulmonary tuberculosis	1
No other signs	16
	45

Factors promoting attacks. Exertion or emotional disturbance chiefly excite attacks in those predisposed to them, and the proportion of cases in which the history tells of paroxysms provoked in these ways is remarkably high.

Flatulence, other digestive disturbances, and especially the assumption of certain postures, are also provocative agents. The induction of a first attack by unaccustomed effort is often responsible for their hasty assignment to overstrain, but strain is never the complete story ; probably damaged or ill-nourished muscle is in all cases the underlying mischief.

Morbid anatomy. In the instances in which examination has been possible after death, the most prominent and frequent lesions have been in the walls of the heart. Fibrosis, pallor, friability, atrophy and interferences with the arterial supply are the chief naked eye changes recorded. In a few cases of tachycardia nerve lesions have been found, but their association with the specific condition with which we now deal is more than doubtful.

The recognition of simple paroxysmal tachycardia.

Whenever a patient is seen in whom excessively rapid heart action is a prominent sign, the nature of the acceleration should be determined ; the tachycardia may be a simple acceleration of the physiological rhythm or it may be a pathological rhythm ; in the first case it is not, in the second case it is primarily cardiac in origin.

A heart rate of 180 or more in an adult is usually the result of pathological impulse formation, and especially is this the case where a heart lesion is known to be present. A clinical sign of a pathological rhythm, which experience continues to impress me as most valuable, is the following : *the rate of the ventricular beating is preserved when the patient passes from the upright to the recumbent position* ; it is rarely altered by more than a few beats per minute even when a supine position is maintained for considerable periods of

time. A physical sign of equal diagnostic importance may be noted at the onset or ending of an attack, the increase and decrease in rate at these times is absolutely abrupt. In patients who are conscious of the rapid heart action, but in whom the beginning and ending cannot be observed, the sudden changes can usually be elicited by careful questioning.

When tachycardia is due to a rise in the rate of the physiological rhythm, as in exophthalmic goitre, effort syndrome, etc., the rate responds to posture, rest and other factors, and usually in an advanced degree ; for the rhythm is under some measure of nerve control. In palpitation, which owes its origin to quickening of the physiological rhythm, the rise of rate and the subsequent fall of rate is gradual and not abrupt.

In paroxysmal tachycardia other physical signs may be present, though their significance is not so great. A prominent and *palpable* pulsation in the veins at the root of the neck is often present ; it may be almost aneurysmal in force. The arterial pulse is frequently irregular in force, and at the first examination may give an erroneous impression of an irregularly beating ventricle. No observations are more unreliable than counts of pulse rates taken in the ordinary manner during the paroxysms ; they should always be checked at the apex beat, either by touch or hearing. The heart sounds are tic tac in quality, and murmurs which may have been noticed on previous occasions usually disappear while the heart rate is raised. To know this is of importance in mitral stenosis, in which such attacks are relatively common ; the presystolic murmur is abolished. When a rough presystolic murmur is lost by a patient who develops an accelerated and regular heart action, the dis-

appearance of the murmur is generally attributable to an abnormal rhythm. In patients who suffer periodically from tachycardia, the presence of occasional premature beats during the periods of quiesence is suggestive that the tachycardia is due to new rhythm production.

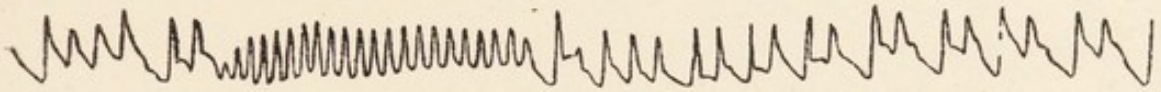


Fig. 35.

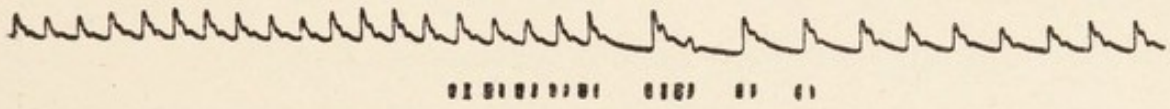


Fig. 36.

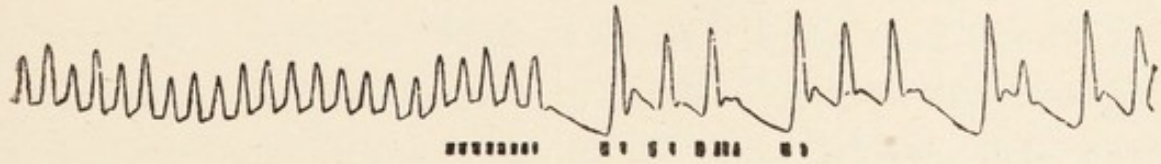


Fig. 37.

Figs. 35 to 37. Three radial curves, taken from separate cases of paroxysmal tachycardia. In Fig. 35 a short and complete paroxysm is shown. In Figs. 36 and 37 the terminations of longer paroxysms are seen. Note the abrupt beginning and ending of the paroxysms, the pauses in which they terminate, the irregularity of the slow periods and the regularity of the fast periods.

The curves are illustrated by Figs. 35-37. In Fig. 35 the beginning and ending of the period of tachycardia, due to abnormal impulse formation at a new auricular focus, is shown. The slow periods to left and right of it are irregular,

for premature contractions interrupt them. The terminations of two long paroxysms are shown in Figs. 36 and 37. The noteworthy features of such curves are several. The changes from the slow to the fast and from the fast to the slow ventricular rates are quite abrupt. Following each paroxysm is a relatively long pause, and this forms the first of a series of pauses in a period of retarded rate. The rate at the actual termination is usually slower than the average rate during the periods of quiesence; quickening, which is best seen in Fig. 36, occurs directly after the termination. The slow rhythm is interrupted by occasional premature contractions; usually these may be shown by special methods to arise from the same point in the heart as the paroxysm.

Symptomatology of paroxysmal tachycardia.

Broadly speaking, the less frequent the attacks the longer do they last. In a given patient the duration of attacks is fairly constant, so that the paroxysms are similar from time to time. Patients exhibiting paroxysms of a few seconds' duration are not uncommon; attacks which last for several hours are the most frequent; those of a fortnight's duration are rare; the attacks may be of any intermediate length. Paroxysms of accelerated heart action of the form considered in the present chapter, but of longer duration, are unknown (see succeeding chapter).

The symptoms accompanying paroxysms of tachycardia are variable both in their nature and in their degree. They are intimately dependent upon the duration of the attack, the heart's rate during it, and upon the capacity of the heart muscle. Amongst those in whom the attacks are brief, it is not uncommon to find that a patient is entirely

oblivious to the rapid heart action when it occurs, and this is more especially the case when the subject is elderly and of the phlegmatic type; or he may be conscious of transient attacks only when his attention is specifically drawn to them or to phenomena commonly associated with them. Paroxysms lasting half an hour or longer are almost invariably accompanied by symptoms, and these become aggravated as the attack proceeds.

The immediate onset is signalled by a sudden sense of discomfort in the region of the heart, amounting to slight or violent palpitation. A tremor or fluttering in the chest and a beating in the neck are common. General effects, such as lassitude, exhaustion, coldness and sweating are also amongst the early symptoms. Later, flatulence, salivation, nausea and vomiting are prominent. These alimentary symptoms are common within an hour or more of the onset, and, once established, usually persist so long as the heart rate is maintained. They hasten the exhaustion which is common and conspicuous in attacks of long duration. In many patients a number of symptoms which are directly referable to the heart are added. These may be divided into two groups. First, anginal symptoms, varying in intensity from slight precordial pain or a sense of compression with skin tenderness, to violent and continuous pain, radiating in the characteristic fashion over the chest, into the neck, into the left arm or both arms and into the abdomen. Wide areas of hyperalgesia, corresponding to the distribution of the lower cervical and upper thoracic nerve roots, are frequently present and persist after the attack has ceased; they are accompanied by tenderness of the tendons of the sterno-mastoids and of the bellies of the deltoid, pectoral and other

muscles. The patients complain of constriction of the chest, variously described as " a band of tightness," " a sensation of gripping " or " a difficulty in breathing." The second group of symptoms is a sequel to embarrassed emptying of the heart. In a number of patients, as the attack proceeds, the limits of cardiac dulness move steadily away from the middle line, and as pallor, which is often an early symptom, becomes more marked, cyanosis and general venous engorgement are added. The veins swell progressively ; the eyes seem sunken, dark areas appear below them and the patient becomes haggard and restless. The liver bulges downwards, its edge becomes palpable and may pass the umbilicus eventually. Tenderness is experienced when the organ is pressed upon, and pulsation is seen in it ; the abdominal muscles assume an increased rigidity ; aching pain develops in the epigastrium and right hypochondrium. In more exceptional cases, puffiness of the ankles and face develops after a long continued attack. A cough, accompanied by a frothy and sometimes blood-stained sputum, is not infrequent, and signs of engorgement of the lungs in the form of sibilant rhonchi and moist râles are found at the bases. Collapse of the patient is prominent in the latter stages. The attack may terminate in progressive failure, delirium, ascites, general anasarca and death. Unexpected death also ends the attack on occasion, but the great majority of the paroxysms cease at the abrupt resumption of the normal rhythm. The actual cessation of the attack is marked by symptoms of its own, a sharp stabbing pain in the chest, or one of more forcible thumps of the heart ; but as a rule the patient speaks only of instant relief. Nothing is more remarkable than the rapidity with which the natural circulatory conditions are restored when the abrupt fall of

pulse rate comes. The dilatation of the heart and the accompanying engorgement of the neck veins vanish, as it were, by magic. The liver recedes beneath the ribs, respiration becomes free, the pain is subdued and the remaining symptoms subside. Quantities of flatus and limpid urine are often passed after an attack.

A varying degree of exhaustion follows the severe attack, the cough may continue for a few hours or days, and skin and muscle tenderness commonly persists for a day or two.

Differential diagnosis.

The diagnosis of paroxysmal tachycardia, during an attack, rests upon careful attention to the history of the patient and to those physical signs and symptoms which have been enumerated already. As a rule, there is little difficulty. But a number of errors do occur, and the chief of these may be mentioned; they mostly depend upon the prominence of symptoms which are referred to other organs, and consequently upon a hurried examination of the organ at fault, or to neglect of the heart because it presents no murmurs.

The stasis of the lungs, with dulness and crepitations at the bases, has been attributed to pneumonia. It is an error which should not happen, for paroxysms at this stage are always accompanied by signs of venous congestion in other organs. When it has occurred, I believe it has been largely attributable to under estimation of the heart rate, and the mistake emphasises the rule that the heart rate should be taken from the apex beat and not from the wrist.

Anginal pain in the abdomen, or the pain of an engorged liver, and accompanied by abdominal rigidity, vomiting and

signs of collapse, has been mistaken for the symptom of a perforated gastric ulcer, and has led to a dangerous and needless laparotomy on more than one occasion to my knowledge; and this in patients in whom cardiac dilatation, engorgement of the veins and excessive heart acceleration, were overlooked in the absence of conpicuous cyanosis.

A large number of cases are grouped under the comprehensive term "heart strain," and this is applied especially to the patient in whom the first attack has been hastened by effort. In my judgment there is no sufficient evidence that a healthy heart is ever damaged by muscular exertion, however severe or prolonged that exertion may be. Patients who are brought forward to illustrate heart strain are unrecognised examples of paroxysmal tachycardia, examples of heart poisoning from foci of infection or examples of undetected structural heart disease, almost without exception. The capacity of the normal heart to withstand added strain is enormous. Is it to be supposed that the organ is so ill-protected that it is to be damaged by actions natural to man? Is it not curious that in the horse, the most heavily strained beast of which we have intimate knowledge, chronic heart affections are conspicuous by their rarity.

More than one instance of paroxysmal tachycardia has come to my notice, in which "acute cardiac dilatation" has sufficed as a diagnosis in a pregnant woman, suffering in reality from a rheumatic heart with mitral stenosis. Dilatation of the heart, let it be clearly understood, does not accelerate the pulse. I am aware that a contrary belief is commonly held, but it is an erroneous belief. When the heart is dilated and beating rapidly, these two phenomena are either due to a single cause, or more commonly, the dilatation is the response

of an injured muscle* to the burden of excessive rate. A rheumatic history is not uncommon in cases of paroxysmal tachycardia, and the characteristic murmurs of mitral stenosis, when this valve lesion is present, are usually masked during the attack. A history of rheumatic fever, or a slight systolic thrill and an accompanying apical murmur, may suggest a more correct interpretation of the case.

The chief difficulty arises, as these instances illustrate, when a patient is seen for the first time in an attack, and this is especially so when no clear history is obtainable. When a regular heart rate exceeds 160 per minute in an adult, the presence of a new rhythm, rather than acceleration of the normal rhythm, should always come first to mind ; it may be suspected even at lower rates. The reaction of the rate to posture is important. It is perfectly true that very high pulse rates are met with in exophthalmic goitre, in pulmonary tuberculosis, in alcoholism and other conditions, but the presence of absence of the diseases or intoxication in question may usually be ascertained and the conditions differentiated. Failing positive evidence from these sources, an examination of the heart rate in its response to posture is of service. In the aforesaid conditions a notable or conspicuous decrease of rate at or shortly after the assumption of the supine posture is the rule. Where we deal with a new rhythm, posture influences the rate inappreciably, if at all ; neither is it affected by repeated swallowing or the suspension of respiration, or by the inhalation of amyl nitrite. A persistent tachycardia of 140 or upwards, maintained under a variety of circumstances, is almost always due to a new and pathological heart rhythm.

* A normal heart responds to increase of rate by decreasing in size ; an abnormal heart responds either by an immediate increase or, as is more frequent, by a temporary decrease and subsequent increase in size.

Patients who are the subjects of relatively brief attacks occasionally seek advice during periods of quiescence on the score of attacks of faintness, palpitation, rapid heart action, etc.. The true nature of the condition may be suspected or proved by careful examination. The history of the sensations at the beginning and ending of the attack are then most valuable. The absence of symptoms or physical signs of cardiac involvement, and the absence of occasional or frequent premature beats, should suggest causes other than those which we are considering, though these are not to be finally excluded on such scores. In nervous, debilitated or infected subjects, an increased force or rate of the normal action or excessive consciousness of the beat are the most probable explanations. In cases of paroxysmal tachycardia a prolonged examination of the patient is sometimes rewarded by the discovery of brief attacks, for the patient so affected is often the subject of more attacks than those of which he is conscious. In all cases of doubt an effort should be made to see the patient during an attack.

The prognosis.

The prognosis contains one element of uncertainty. Death during paroxysms has occurred, but the overwhelming majority of the paroxysms are tolerated.

Speaking of individual attacks, several prognostic aspects need emphasis. The symptoms of the patient are largely governed by the reaction of the nervous system ; nervous subjects, especially women, awaken needless anxiety. The duration of the observed paroxysm and the length of previous seizures have to be considered. The outlook is more ominous when, after a continuation of several days, the heart shows

signs of progressive weakening, manifested by steady increase in its size and by the pulmonary and hepatic congestion which supervene. The strength of the pulse is not prophetic, it may be scarcely perceptible in repeated attacks. The gravest symptoms are those of increasing respiratory embarrassment, consequent upon œdema of the lungs, and the onset of delirium and general anasarca. Nevertheless, it usually happens that when embarrassment is profound the paroxysm ends, and the patient passes in a few minutes from a condition of acute distress, and seemingly the utmost gravity, to one of relative comfort and safety. Briefly, so far as the individual attack is concerned, it is the duty of the medical attendant to maintain a confident attitude ; however desperate the case may appear to the uninitiated, if its nature is certainly known, an abrupt cessation is to be predicted. And this prediction will rarely fail to be fulfilled ; although I have had exceptional opportunities of witnessing these attacks, a fatal termination has not come within my personal experience. The attack, except in the very rarest instances, does not last beyond 10 or 14 days ; consequently, if the nature of the attack is recognised, a confident prediction to this effect may be made.

The prognosis of the malady as a whole should be based upon two chief considerations ; first and most important, upon an estimate of the endurance of the cardiac muscle, and secondly upon the severity of the trials through which it has to pass.

The estimate of the first factor is formed from the signs and symptoms *between* the attacks and from the patient's reaction to effort. The prognosis in a case of paroxysmal tachycardia is the same as that in a similar

case which shows no attacks, but with the following reservations :—The attacks themselves indicate muscle damage, and the attacks are apt to place the life of the patient temporarily in jeopardy. The reaction of the heart to the attacks is also of importance. A healthy heart reacts to a pure increase of rate, amounting to a doubling of the normal rate, by *decreasing* in size, and the circulation may be maintained for long periods. A defective or diseased muscle reacts by dilating. The degree of dilatation and its rate of progress during the attack consequently gauge the degree of muscular involvement. In estimating the second factor a survey of the length and frequency of the attacks and the heart rate during such attacks is involved, as they are summed up by observation and the previous history ; but as the attacks may cease at any time never to return, the value of these considerations in completing the prognosis has its limitations.

The possibility of death in a seizure is an uncertain factor ; it necessitates caution in prognosis when the paroxysms are frequent and of long duration. Thus, although the factor of unexpected death may be neglected in the prognosis of individual attacks because its occurrence is remote, it may not be neglected entirely when the paroxysms are dealt with collectively.

The prognosis, where the heart between the attacks seems sound, and where the paroxysms are infrequent and of a few hours' duration, and the rate not very excessive, is very favourable ; with rare exceptions such paroxysms do not curtail life, and a prospect of long years may be spoken of to young subjects without hesitation. These patients always wish to know whether they will ever enjoy freedom. They

may be told that, although such freedom cannot be promised, the prospect of it is fair. The prognosis as a whole starts from this foundation, and as muscle or gross valve lesions are more in evidence, as the attacks are longer and more frequent, as the heart acceleration is greater, and as the patient is older, so the outlook is naturally less hopeful.

The treatment.

The treatment of paroxysmal tachycardia may be conveniently dealt with from two standpoints ; the management of the attacks themselves, and the care of the patient during the general course of the malady.

Are we aware of any remedy which will infallibly cut short a paroxysm of tachycardia ? The answer to this question is still in the negative. I have frequently seen attacks of several hours' duration terminate shortly after the administration of certain remedies or after certain interferences. The patients who are the subjects of them are often aware of and adopt certain curative measures. In some instances the assumption of a given posture, sitting and placing the head between the knees, for example, or lying supine, is a certain remedy. The induction of vomiting, the relief of flatulence, or the application of a tight abdominal binder may be immediately and constantly efficacious in given cases. I have seen the application of an icebag to the precordium, a remedy which always affords relief, speedily terminate attacks. Similarly, they have ceased shortly after the administration of a single intravenous injection of digitalin (1-100 gr.) or strophanthin (1-250 gr.). Firm pressure upon one or other vagus nerve as it lies in the carotid sheath has been successful. But much more often than not, such

remedies are without effect and the treatment finally adopted becomes palliative or symptomatic. Rest is enjoined, and attention is paid to the wishes of the patient in respect of posture. Most frequently these sufferers prefer to lie well supported with pillows; sometimes they prefer to stand. The dietary should be fluid, bland and restricted. Iced water or milk are well borne and are often beneficial.

Local applications, the icebag, a mustard leaf, cupping over a distended or pain-giving organ, be it the heart or the liver, often afford relief. Pain, if general, may be combated by more general remedies, such as chloral or morphia; but these drugs are not often needed. The induction of sleep in long-continued paroxysms is essential, and fortunately chloral and the opiates may be employed with safety. Serious engorgement of the heart and signs of progressive lung œdema or grave venous stasis are indications of venesection. The letting of 8 or 12 oz. of blood will be followed by improvement; fortunately the occasion for this does not often arise. Respiratory embarrassment is relieved and sleep induced by the administration of oxygen; this gas is best given through a tight-fitting mask which covers the whole face, so that high percentages are breathed.

The treatment of the malady as a whole is largely governed by the condition of the heart between the attacks. A searching inquiry may reveal exciting causes of paroxysms; often, sudden exertion or emotion is the chief provocative, so that to change or forbid the usual employment may be imperative. General care of the health, the cleanliness of the mouth and throat, the orderliness of the dietary and the remedying of dyspeptic troubles and constipation may ward off the crises. The continued wearing of a broad abdominal

belt, firmly applied before rising and discarded at bedtime, is sometimes accompanied by the happiest of results.

The paroxysms themselves do not contra-indicate the careful administration of general anæsthetics, should these be necessary.

CHAPTER VI.

AURICULAR FLUTTER.

Definition.

Auricular flutter is a condition in which, as has recently been shown, the contraction wave follows a circular and never-ending path in the auricle, the circuits being completed at a rate of from 240 to 350 per minute in different subjects.

The nature of auricular flutter.

When the heart beats normally, each beat starts in the sino-auricular node and the contraction thus begun radiates in all directions into the auricular muscle, more and more of which becomes involved as the wave spreads, until the whole is in contraction. The contraction wave spreads to the furthest lying points of muscle, and, finding no further path open to it, dies out; the auricle then remains quiescent until a new impulse is discharged and the events are repeated. In recent experiments, which have elucidated flutter, the beating of the auricle is ordered on a quite different and wholly abnormal plan. A single and continuous wave circulates in the auricle, usually around the combined mouths of the superior and inferior vena cava. Unlike the normal contraction wave, this wave is unidirectional; it pursues its own wake and passes back to and over the same path again and again. It gives rise to a very rapid and weak action of the auricle, the known limits of the rate being from 220 to 350 per minute; otherwise stated, each circuit is

completed in 1-220 to 1-350 of a minute ; the time taken for the completion of the circuit, which depends on the length of the circus and the rate of conduction, governs the auricular rate. As the wave is always circulating, the auricle, as a whole, never enjoys a true diastole; that is to say, it is never at rest as a whole. At each circuit the auricle sends an impulse towards the ventricle, which may or may not penetrate to it. One of the most notable features of flutter, or extreme acceleration of the auricle, is its almost invariable association with heart-block. Flutter, so far as we know, arises in the

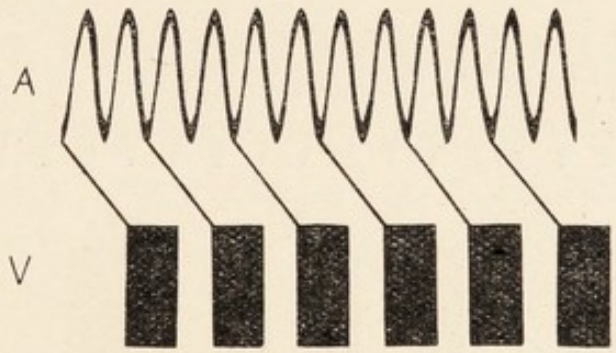


Fig. 38. A diagrammatic representation of auricular flutter. The contraction wave in the auricle is represented spirally, to indicate its continuation and constant re-entry of the same path. The auricular rate is extreme ; the ventricular rate is also rapid, though in this instance only half the auricular ; 2 : 1 block is present.

auricle only,* and the rate of the auricular contractions is so great that the ventricle can rarely keep the pace. The usual auricular rates are from 260 to 320 per minute. The usual ventricular rates are from 130 to 160, exactly half the auricular ; for 2 : 1 heart-block is generally present when the patient first comes under observation (Fig. 38). The auricles are driven in merciless fashion and the reins of control, the inhibitory nerves, are powerless. The auricle has veritably seized the bit with its teeth. The ventricle, shielded from

* Ventricular flutter is unknown clinically ; it is probably unknown because, continuing, it would kill the subject of it.

the whip by the auriculo-ventricular bundle, lags behind. 2 : 1 heart-block is the rule ; but any grade of block may be present. Thus it happens that while the auricle races at 300 per minute, the ventricle may beat at 150 (2 : 1 heart-block), at a normal rate of 75 (4 : 1 heart-block), or at 30 to 38 (complete dissociation). The speed of the auricle once set is wonderfully uniform ; it may vary but a few beats per minute over long periods of time ; its beating is always regular. The responses of the ventricle are often regular ; but they may also be irregular, when the impulses

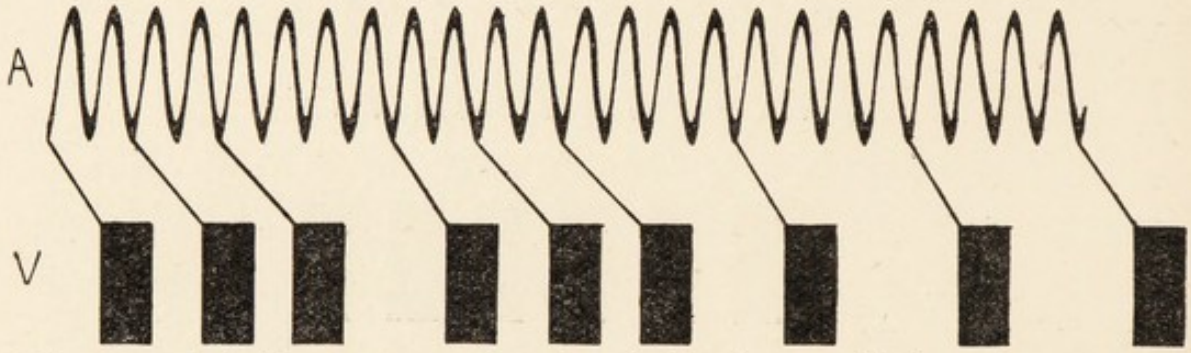


Fig. 39. A similar representation of auricular flutter with irregular response of the ventricle. The irregularity is of such a kind that the beats of the ventricle come in groups which are accurately repeated from time to time. Note the changes in the *As- Vs* interval, and compare with Fig. 11.

from the auricle are chosen at irregular intervals ; especially is this so during a transition from one uniform grade of heart-block to another. But even when the ventricle beats irregularly, as each of its responses is to an auricular contraction forming one of a perfectly regular series, the ventricular contractions lie in the curves at definite points (Fig. 39), which may be prejudged accurately if the grade of block is known. The point at which a ventricular beat is placed is governed by laws described under heart-block in Chapter III.

Attacks of flutter are sometimes of quite brief duration ; usually, and unlike simple paroxysms of tachycardia, they last for months or years.

Etiological and pathological relations.

Age and sex. Flutter is a comparatively rare condition, and though it may occur at any age it is essentially associated with advanced years. Once I have seen flutter in a child of nine months, and it has been reported in other children.

In my experience it is four times as common in males than in females.

Relations to infective disease, etc.. As a general rule, no previous infection can be traced. Rheumatic fever or syphilis has seemed responsible in some cases, and in others there has been an antecedent infection of the throat or urinary tract or a history of recurrent attacks of gout.

Associated conditions. Occurring as it does in elderly cases, flutter is often associated with arterial sclerosis; much increase of the heart's dulness is uncommon; as a rule there are no murmurs, but any of the valve lesions may be discovered. There are nearly always some signs of degeneration of the heart muscle, as witnessed to by the symptoms of the patient when the heart beats at normal rates.

Of a special *morbid anatomy* we have no knowledge.

The recognition of flutter.

In a number of patients the presence of auricular flutter may be recognised by ordinary clinical means; but in perhaps a larger number the diagnosis is only possible when a special method (the electrocardiographic particularly) is employed.

During the 2 : 1 *heart-block phase.* When patients who exhibit flutter are first seen, the rate of the ventricular action is usually half the auricular. The history often includes palpitation, whose onset was sudden months or years before

and was perhaps accompanied by fainting. A regular and persistent ventricular action of from 130-160 per minute in an elderly subject is a most suspicious circumstance, and its discovery should always be followed by a special examination for other signs of flutter. The patient may give a history of short paroxysms of many years' standing and may speak of this the final attack which he is unable to discard. If tachycardia persists for a month or more at one of the stated rates, and there is absolutely no change of rate with change of posture, rest or exercise, the condition is almost certainly flutter. A most suggestive incident is the constant repetition of the same high rate in the pulse chart, or the finding of exactly the same high heart rate at intervals of weeks or months.

From time to time, in certain individuals, and usually during periods of emotion or exertion, the ventricular rate springs momentarily to the full auricular rate ; the resulting disturbance is profound, and patients who retain consciousness subsequently give vivid accounts of the experience ; fainting is common in flutter patients.

Firm pressure upon the carotid sheath, on left or right side, sufficient to obliterate the vessel and stimulate the vagus nerve, often produces a conspicuous slowing of the pulse or a lapse of many beats (Fig. 40).* Digitalis, given in full doses, always slows the pulse and creates irregularity. The radial curves, when the pulse is fast, often exhibit alternation (see Chapter VIII).

Auricular flutter, yielding a 2 : 1 response of the ventricle, is the easiest form in which it is recognisable ; fortunately

* But it leaves the auricular rate unaltered, the vagus acts here by preventing the auricular impulses from reaching the ventricle (heart-block).

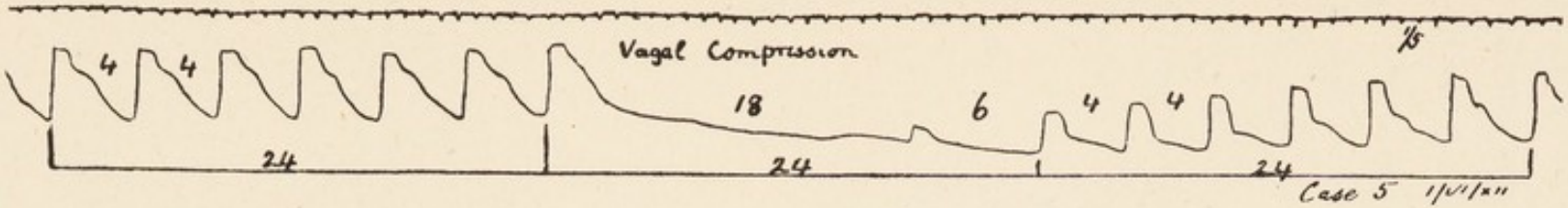


Fig. 40. An arterial curve in auricular flutter, showing the effect of pressure upon the right carotid sheath during a period of 4 : 1 heart-block. The ventricular responses after the long pauses occur at expected intervals. This is due to the beats being responses to auricular impulses and to the undisturbed and regular character of the auricular rhythm. The number of auricular contractions to each ventricular cycle is marked above the curve in this figure. The brackets include equivalent numbers of auricular contractions and equal stretches of the curve.

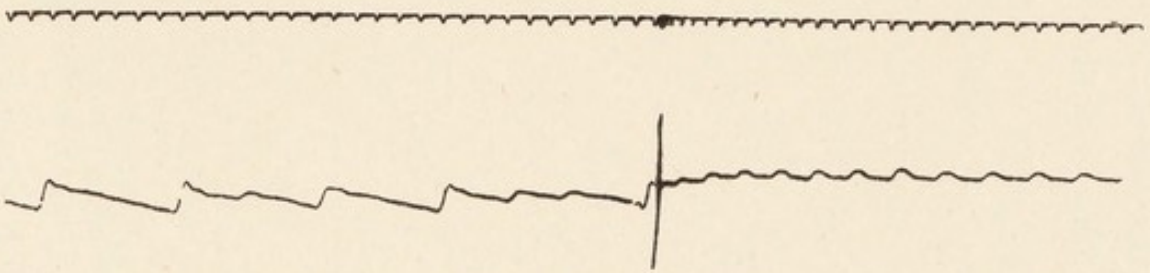


Fig. 41. A radial curve in auricular flutter. To the left of the ordinate is a period of irregular response ; to the right of the ordinate is the regular action produced by moderate exercise. This last period corresponds to 2 : 1 heart-block.

this is also the most important form in which to recognise it, for it then requires and responds well to treatment.

If flutter occurs, as it may do, in short paroxysms, it is apt to be confused with simple paroxysmal tachycardia, for the symptoms are similar and the signs are very similar. Often they may be differentiated clinically, nevertheless. Common ventricular rates in simple paroxysmal tachycardia are from 180 to 220 per minute. If the ventricle beats regularly at these rates, auricular flutter may be excluded, because auricular rates equalling or surpassing 360 (which is

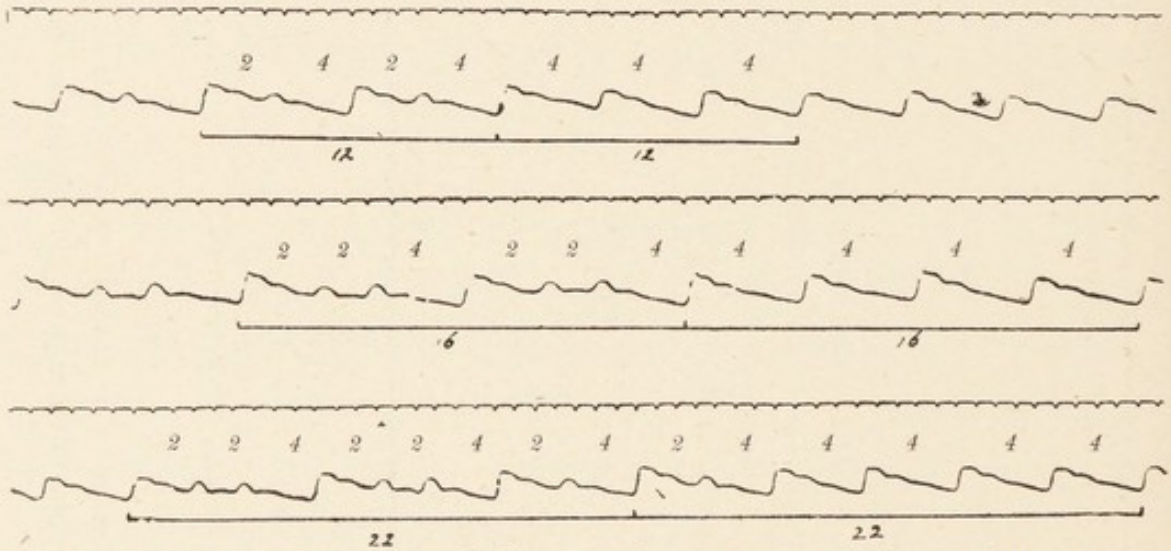


Fig. 42. A series of three radial curves from a case of flutter, showing the chief features of the irregularities produced by mixed responses. The curves have a superficial resemblance to premature contractions; but that the irregularity is not produced in this fashion is clearly indicated by the manner in which the curves "space." The bracketed portions of each curve are of equal duration, and they are of equal duration because they correspond to equal numbers of auricular cycles. The number of auricular cycles to each ventricular cycle is marked above each pulse beat.

the double of 180) are unknown. Actually the confusion is not of much consequence, since short attacks of flutter should be treated as are simple paroxysms unless the symptoms show extreme urgency.

As simple paroxysms of tachycardia do not last more than 14 days, and as flutter usually lasts months or years, they are not usually confused.

During the stage of irregular responses. If the responses of the ventricle are irregular, a little exercise, often no more than raising a limb off the bed, immediately accelerates the ventricular action and induces perfect regularity of the pulse (2 : 1 heart-block) (thereby differentiating the condition from auricular fibrillation), and this regular pulse action may then be tested in the manner described in the preceding paragraphs (Fig. 41).

The character of the radial curve gives an immediate clue to the condition (Fig. 42 and explanation).

When the responses of the ventricle are infrequent. It is under these circumstances that flutter is so difficult to recognise by ordinary clinical means. A patient may possess a fluttering auricle and the pulse may be within normal limits of rate and may be regular. Fortunately for diagnosticians, such cases are rare ; moreover, the failure to detect the flutter at such times is relatively of little consequence. In some patients the movements of the auricle are transmitted to the veins of the neck and may be identified as very rapid, minute and regular undulations (Fig. 42*b*).

The symptomatology of flutter.

The symptoms associated with auricular flutter need not long detain us. In patients in whom the acceleration occurs in short paroxysms the symptoms are identical with those of simple paroxysmal tachycardia ; they vary in intensity according to the heart rate and according to the resisting power of the ventricular muscle.

But in patients who experience longer periods of disturbed heart action, and these are more frequent, for flutter generally persists for months or years, the subjective sensations are

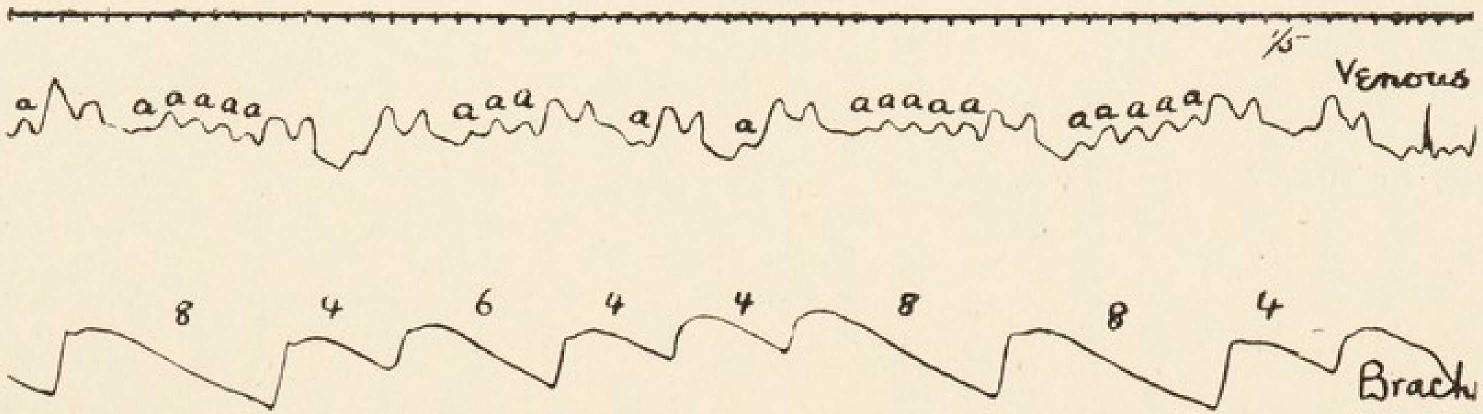


Fig. 42*b*. Venous and arterial curves in flutter associated with a high grade of partial heart-block. Showing the numerous and regular auricular waves (*a*) which appear in the long diastoles.

As in Fig. 40, the number of auricular contractions corresponding to a ventricular cycle is marked above the respective beat of the curve.

modified. Although the reason is not clear, the symptoms of flutter seem less profound than is to be expected from a study of many simple attacks of tachycardia. Thus, not infrequently, the action of the heart may be accelerated to 130 or 150 for periods of a year or more, and yet the symptoms

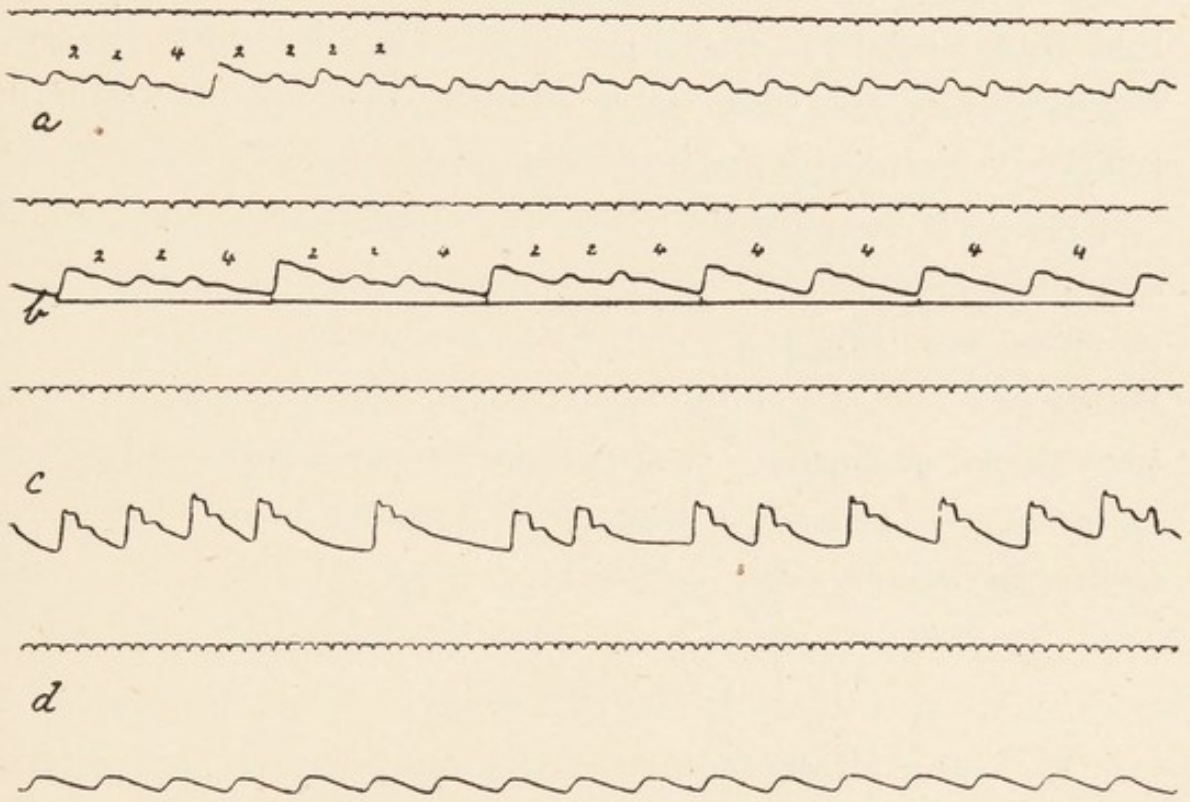


Fig. 43. A series of four curves, showing the effect of digitalis medication upon flutter. (*a*) The heart is beginning to respond to digitalis and occasional periods of 4 : 1 heart-block are seen, interrupting an otherwise regular 2 : 1 mechanism ; the rate of the auricles was 264 per minute, and that of the ventricles 132. (*b*) Further slowing a few days later. The beats of the ventricle now occur in groups, or the mechanism is reduced to a 4 : 1 grade of heart-block. (*c*) A few days later ; the ventricle becomes grossly irregular with the establishment of fibrillation. The digitalis having been withdrawn, the normal and regular mechanism is resumed (*d*) ; the rate of the auricle and ventricle being now 64 per minute.

consist of little more than a sense of exhaustion and fatigue after exertion. The occupations of such patients are limited, but signs of failure in the form of stasis and dropsy do not necessarily supervene. I have known a patient in whom the ventricle has beaten at 140 per minute night and day for seven years ; his condition was much the same at the

beginning and end of this period. Such cases illustrate the resistance of a tolerably healthy heart to prolonged and considerable strain in a most dramatic manner. Such tolerance is to be attributed perhaps to the reserve power of the ventricular muscle. Naturally there are cases of flutter in which signs of congestion are visible at an early stage; but in reviewing a series of cases, one cannot but be impressed by the infrequence of these signs of failure, as compared to their relatively high incidence in simple paroxysms of similar rate.

As I have already indicated, there is an additional symptom complex in flutter; it results when the ventricle assumes the full auricular rate; an acceleration of the ventricle to 300 per minute places the life of the subject in immediate jeopardy, the symptoms are profound and consciousness is usually lost; such attacks, being survived, are necessarily fleeting.

The prognosis.

As stated, I have known the condition to last uninterruptedly for seven years, the ventricle beating without cessation at 140 per minute. How much longer this high rate may be maintained in the presence of a tolerably efficient circulation cannot be stated.

We gather a general idea of the prospect upon the lines discussed in treating of the simple paroxysms of the last chapter. It may be based upon a general consideration of the strength of the heart muscle, and of the burden which this muscle has to carry. Important in this respect is the response to treatment, for most cases are amenable to specific measures, as we shall see in the succeeding paragraphs.

The treatment.

The treatment of long continued flutter of the auricles is often conspicuously successful. Even after the acceleration has lasted for many months, or even several years, the natural rhythm of the heart may at times be restored by suitable medication. The remedy is digitalis or an allied drug. My experience tells me that the ventricular rate can always be reduced by giving digitalis or strophanthus in full doses, and may be maintained at the reduced rate so long as treatment is continued.

Further, I have found that if, having obtained this reaction, the dosage can be increased, the flutter ceases and fibrillation (a condition described in the next chapter) takes its place ; if, now, the remedy is withdrawn, the fibrillation soon vanishes in most cases and the normal rhythm is immediately resumed. I have seen these changes in a number of patients and can speak confidently of the success of the remedy in them. Occasional intolerance to the drug, the onset of gastro-intestinal symptoms, appears to the sole limitation ; strophanthin may then be administered intravenously with equally happy and much more speedy results. It may be asked if the flutter ever returns when it has been abolished. In one of my cases it returned, but renewed treatment again restored the normal rhythm, and this persisted. The secret of the treatment seems to lie in throwing an obstruction in the path of the circulating wave; digitalis may conceivably do this by creating a region of block in the auricle. Flutter, once it comes, establishes and maintains itself; it constitutes a really vicious circle; the same tendency is found in fibrillation, a condition which we shall discuss

presently. Being checked, the cause of its persistence is removed.

When, in such patients as have signs of cardiac failure, flutter is removed and the normal rhythm, with normal heart rate, takes its place, the change in the general condition is remarkable and almost immediate. Engorgement and dropsy rapidly disappear; breathlessness and other discomforts are relieved; the customary occupations of life may be resumed.

CHAPTER VII.

AURICULAR FIBRILLATION.

Definition.

A condition in which the auricles fail to contract *en masse*, the muscle activity consisting only of fibrillary twitchings ; the normal and regular impulses transmitted to the ventricle are absent, while rapid and irregular impulses produced in the auricle replace them and produce gross irregularity of the ventricular action.

The nature of auricular fibrillation.

When we inspect the normally beating heart of an animal the systoles of both auricle and ventricle are readily discerned. The movement of the auricle is a sharp flick, most clearly perceptible in the length of the auricular appendix, for in this line the shortening is greatest. When the auricle is forced into fibrillation or delirium, the appearances are quite distinctive ; the muscular walls are maintained in a position of diastole ; systole, either complete or partial, is never accomplished ; the structure as a whole rests almost immobile ; but close observation of the muscle surface reveals its extreme and incessant activity, rapid and minute twitchings and undulatory movements are visible over the whole. Recent investigation points to the nature of the auricular disturbance. It is related to flutter, but instead of the single smooth circulating wave, which produces

a regularly beating auricle, the circulating wave or waves are sinuous, the path varies, and the waves seemingly collide or re-enter new channels where from instant to instant they find these free. This mechanism is not easy to represent in diagrammatic form, but I have attempted to depict multiple and variable circulating waves in Fig. 44. As in flutter, the auricle as a whole is never in a state of diastole; its tissues have suffered functional fragmentation. The precise manner in which this change comes is still unknown. When the auricle is fibrillating its normal, regular and co-ordinate

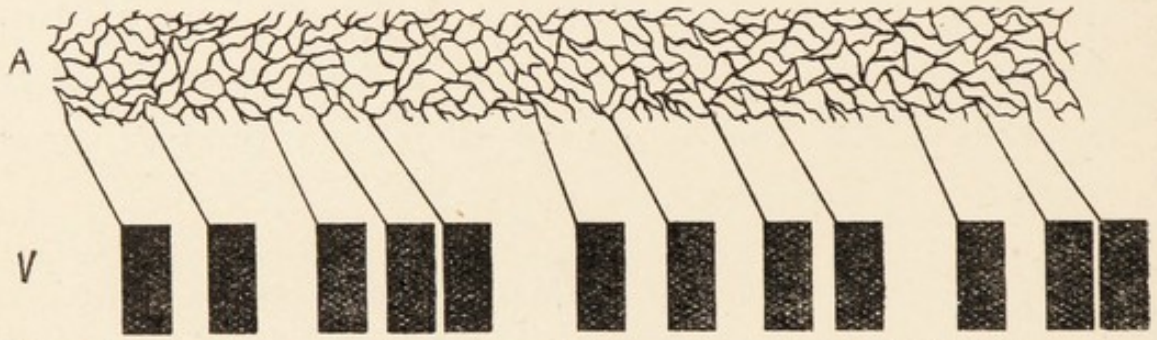


Fig. 44. A diagrammatic representation of auricular fibrillation. The fibres of the auricle do not contract co-ordinately. The contraction waves, which are multiple, follow varying and sinuous paths, colliding with each other or re-entering tracks through which they or their neighbours have previously passed. Occasional impulses leave the auricle at irregular intervals, and many of these proceed to the ventricle and stimulate it. Its action is therefore rapid and irregular.

contractions are in abeyance, and consequently the ventricle is robbed of the regular impulses which form its accustomed supply. These are replaced by numerous and haphazard impulses, escaping to the ventricle from the turmoil which prevails in the upper chamber; the change in the action of the ventricle is consequently profound. Its rate of beating rises considerably, and the contractions follow each other in a completely irregular fashion (Fig. 44)*.

* Ventricular fibrillation is incompatible with life. It is probably responsible for many instances of unexpected death; this end is more especially suspected in certain cases of auricular fibrillation and in death under chloroform anæsthesia.

Such are the events in experiment, and those of the clinical condition are identical, with one proviso ; since in the experimental heart the tissues controlling the conduction of impulses are healthy, the rate of the ventricular contractions is doubled or even trebled ; but in the human subject, the conducting tissues may be either intact or damaged ; consequently the ventricular rate varies widely in different patients, according as access to the ventricle is full or limited. While a free passage yields rates approaching 200 per minute, damage to the junctional tissues may reduce the rate to 40 or less : the usual rates lie between 90 and 140.

Etiological and pathological relations.

Age. While fibrillation has been observed from childhood to old age, it is extremely rare before the age of 17 years. In studying the age distribution, the cases are conveniently divided into rheumatic and non-rheumatic groups. This division clearly shows that, independent of rheumatism, the affection is related to advancing years ; the highest incidence is in the sixth and seventh decade. In the rheumatic group, the incidence is heaviest between the twentieth and thirtieth years ; it is almost as heavy in the fourth and fifth decade, but lightens as the years mount further.

Sex. Auricular fibrillation is much more common in men than in women, but the preponderance in males is chiefly in the non-rheumatic group. Where there is a rheumatic history the sexes bear the burden more equally. The relative frequency of fibrillation in rheumatic women is linked with the prevalence of mitral disease in this sex ; mitral stenosis and auricular fibrillation are bosom companions.

Relation to infections; associated conditions. Amongst a collection of 152 cases a rheumatic or choreic history was found 71 times; in four instances at least there was a history of one or other affection in the family. Amongst the remainder, mitral stenosis was present in 26, and pericardial adhesions or effusion in two cases. If these patients are collected to form a rheumatic group, the subdivision includes 101 cases, or 66 per cent. The prevalence of fibrillation amongst those who suffer from mitral constriction is especially noteworthy; 79 of the cases, or 52 per cent., had this valve lesion. The relation to mitral stenosis may be traced in another and equally emphatic manner. Of 106 cases of mitral stenosis collected in an out-patient department, 22, or approximately one-fifth, suffered from auricular fibrillation. The proportion amongst in-patients is much higher; it exceeds 50 per cent.

In the table I have classed a group as myocardial degeneration; it includes those in whom the heart irregularity was the outstanding feature, though many of the cases presented signs of cardiac failure in addition to the irregularity. Aortic disease, arterial disease and granular kidney are the chief lesions in other groups. Of all cases of cardiac failure admitted to a general hospital 60 to 70 per cent. manifest this disorder of the heart; it is difficult, therefore, to over-emphasise its importance.

Of etiological factors, rheumatism is predominant, as we have seen; a history of other infections, "influenza" amongst them, is given by many patients, but the influence of these infections is imperfectly understood.

An idea has been prevalent in the past that dilatation of the heart may itself lead to gross irregularity of the organ,

		*							
Rheumatic or choreic history	Mitral stenosis	**51**							71
	Myocardial degeneration		**11**						
	Pericardial adhesions						**1**		
	Aortic disease				**6**				
	Renal diseases			**2**					
Rheumatism or chorea in family	Mitral stenosis	**2**							4
	Myocardial degeneration		**2**						
No history of rheumatism or chorea	Mitral stenosis	**19**							70
	Arterial disease					7			
	Myocardial degeneration		17						
	Renal disease			11					
	Aortic disease				3				
	Aneurism							2	
	Emphysema and bronchitis							2	
	Subacute infective endocarditis							2	
	Pericardial adhesions and pericardial effusion						2		
	Tuberculous pleurisy							1	
	Syphilitic heart							1	
	Congenital heart							1	
	Chronic alcoholism							1	
	Pneumonia							1	
Rheumatism or chorea not noted	Mitral stenosis	**7**							7
		79	30	13	9	7	3	11	152

Mitral stenosis	.. ..
Myocardial degeneration	.. ..
Renal disease	.. ..
Aortic disease	.. ..
Arterial disease	.. ..
Pericardial adhesions and effusion	
Remainder	.. .. ..

such as is here described. This idea is fanciful. Dilatation does not produce irregularity, but irregularity, when associated with rapid ventricular action, may dilate an injured ventricle.

* The heavy figures mark the rheumatic group.

Morbid anatomy. That valve lesions are present in a number of the cases is obvious from the bedside examinations ; enlargement of the whole heart is common, and dilatation or hypertrophy of the auricles is more frequent than the valve lesions which might be held to account for them. The most constant structural alterations which are found are discovered by histological examination of the heart musculature. Usually it shows a more or less intense grade of subacute or chronic inflammatory change progressing to fibrosis, and the auricles are conspicuously affected. A diffuse fibrosis, accompanied by leucocytic infiltration and atrophy of the neighbouring muscle cells, is the most frequent change.

Such is the tale told by the microscope, but it does not justify us in holding that the inflammatory reaction is the cause of the disturbance. We examine the hearts of those who die, and most die with all the classical signs of heart failure. Many of the microscopic lesions are to be regarded as the result of infections producing heart failure rather than fibrillation. Similar lesions are found where fibrillation has never occurred, and hearts which have shown this disorder may not present the lesions described.

The recognition of auricular fibrillation.

Auricular fibrillation gives rise in a clinical case to two series of phenomena—the one dependent upon the virtual paralysis of the auricle ; the other dependent upon the disordered action of the ventricle.

It will be convenient to study the ventricular signs first. The irregularity varies in form according to the rate of the contractions. When the heart is beating rapidly at 100-160 per minute, the grade of disorder is

maximal. The pulse supplies indifferent news of the ventricular rate, many pulsations fail to reach it (such beats are marked with asterisks in Fig. 46). The pulse is a medley of beats of many sizes (Fig. 45), an intimate mingling of changing pauses; now the beats are almost uniform in strength and pacings; now feeble pulsations chase along rapidly; now the pulse is lost; now it returns with increased vigour. Feel the pulse in such cases and the nature of the disease is disclosed; the more turbulent the pulse, the more certain is the evidence. It is when the rate is slow that close attention and more experience are often asked, for with slower rate the disorder is less pronounced; all the heart beats now reach the wrist and the irregularity comprises minor variations in the length of diastole (as in Fig. 48); it may then escape notice, and a heedful examination, concentrating upon its presence or absence, alone brings it to discovery. Short pulse curves reveal the irregularity on all but rare occasions. The nature of the arrhythmia is such that the heart action is never quite regular, and seldom do two beats of a precisely similar character or length lie adjacent. No two whole strips of curve are alike; the cycles bear no simple length relation one to another. Proportion between the force of an arterial pulse and the length of rest preceding it is often lost (Figs. 45 and 46); a strong beat may succeed a brief diastole and a weak beat may succeed a long one. When the pulse is slow, only beat to beat measurement may disclose the irregularity.

The fully developed disorder of the ventricle is readily appreciated at the apex. The heart sounds are modified; they vary in intensity and the variation runs hand in hand with the strength of the beats. First and second sounds are present with each systole which yields arterial pulsation

Figs. 45 to 50. *Apex and radial curves from cases presenting auricular fibrillation.*

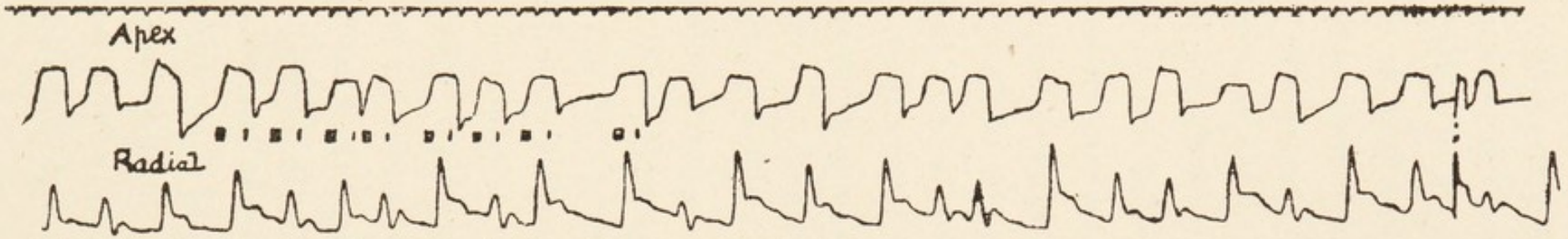


Fig. 45. The heart's action is grossly irregular, each ventricular beat reaches the wrist. The heart sounds are clear but irregularly placed.

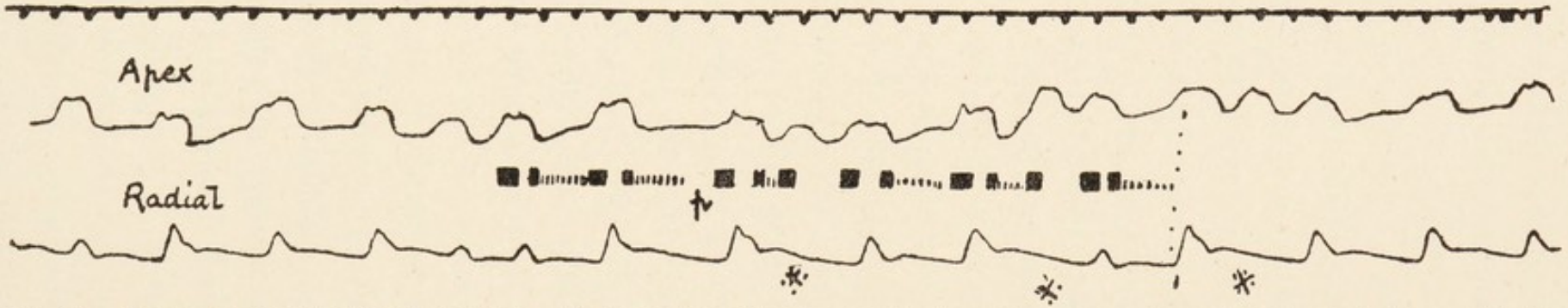


Fig. 46. From a case of mitral stenosis. The heart's action is grossly irregular, many beats fail to reach the wrist (*). A diastolic murmur fills the shorter pauses, but falls short of the first sound in the longer pauses (*p*). The murmur does not constantly extend to presystole.

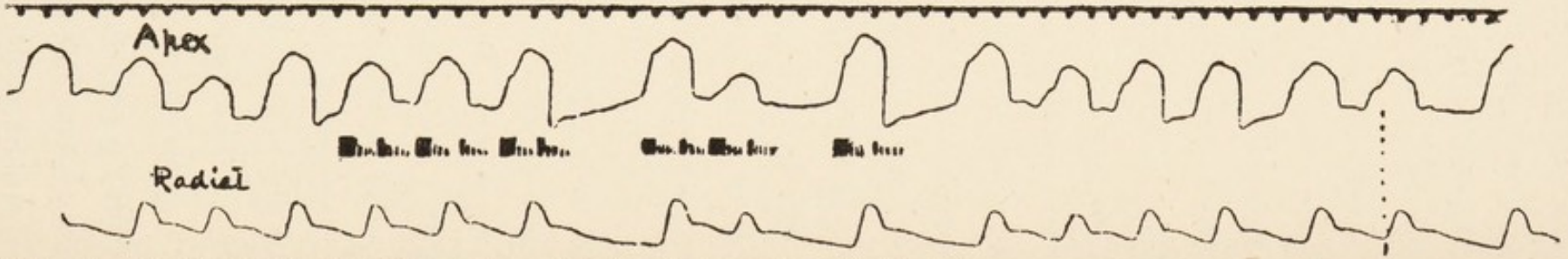


Fig. 47. Gross irregularity is present. Mitral systolic and aortic diastolic murmurs accompany each cycle.

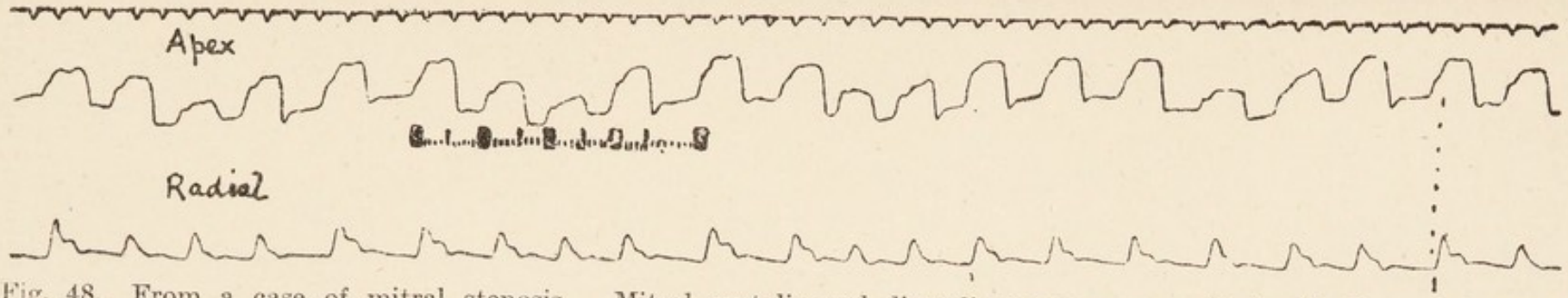


Fig. 48. From a case of mitral stenosis. Mitral systolic and diastolic murmurs completely fill the gaps between the heart sounds.

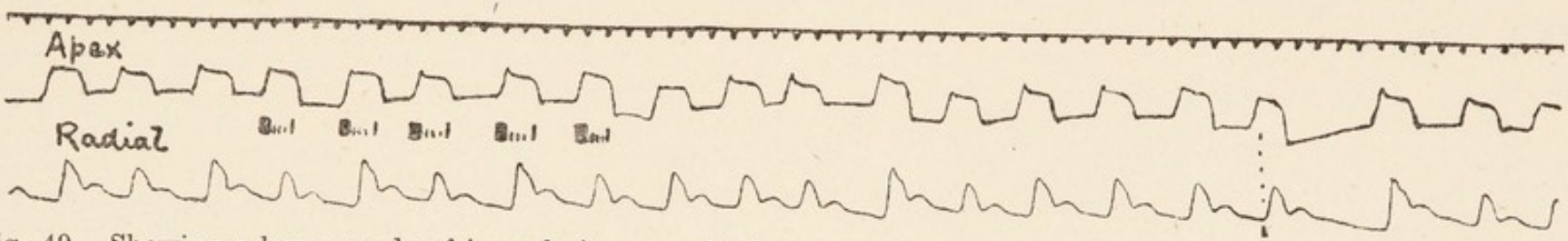


Fig. 49. Showing a lesser grade of irregularity; a mitral systolic murmur accompanies each ventricular contraction.

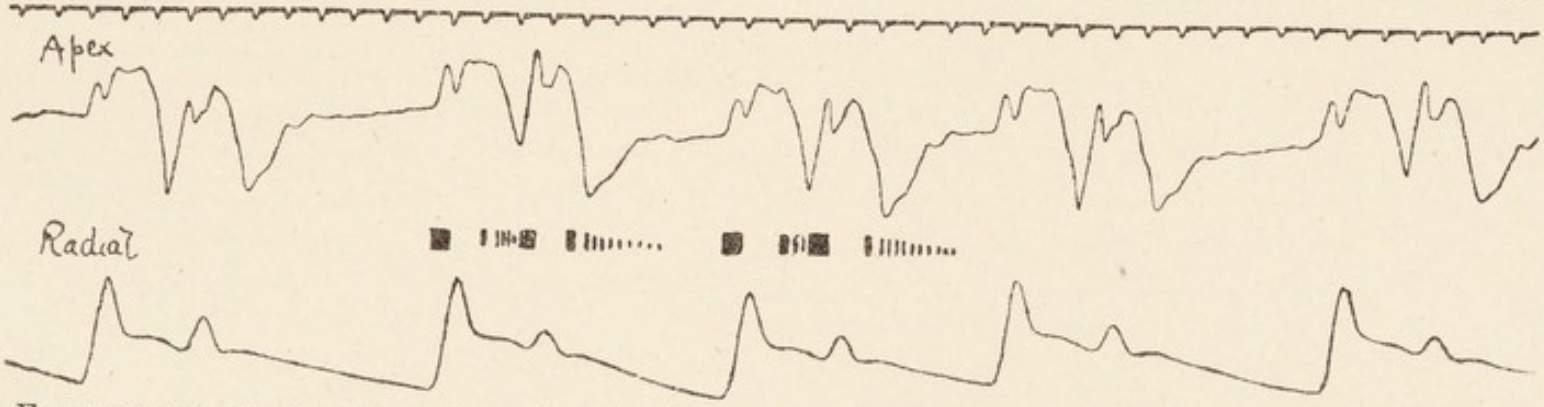


Fig. 50. From a case of mitral stenosis under the full influence of digitalis. "Digitalis coupling" is present. The presence of fibrillation is recognised by the variation in the lengths of the diastoles following the weaker pulse beats. The heart's action is slow. A diastolic murmur, commencing soon after but not immediately with the second sound, runs through early and mid-diastole.

(Fig. 45); a first sound stands isolated when the pulse beat is missing (Fig. 46). If a systolic mitral murmur is present, it accompanies each ventricular contraction (Fig. 49), except where the rate is fast, for then it is apt to vanish. Aortic murmurs obey the general rule, their presence or absence is controlled by the efficiency of the respective beats (Fig. 47).

The inactivity of the auricle is responsible for special alterations of the heart sounds in patients who have stenosis of the mitral orifice. It is customary to allude to disappearance of presystolic murmurs when the auricle fibrillates, but this statement is not exact. The change in the characters of the murmurs at the onset of fibrillation is oftentimes impressive, but it varies according to the heart rate and the degree of stenosis. If there are short presystolic murmurs while the heart beats regularly, these murmurs usually vanish when fibrillation begins; and more especially if the rate during fibrillation is rapid. If the presystolic murmur is long and rough, a murmur of similar character is preserved during fibrillation. But its time relations are altered. Attention should concentrate upon the position of the second sound at the apex. When the auricle is fibrillating, the diastolic murmur has a fixed time relation to this sound. If the heart is rapid, the murmur begins early in diastole and fills the whole gap to the first sound of the succeeding beat (Fig. 48); if the heart rate is less rapid, the murmur maintains its relation to the second sound but falls short of the succeeding first sound during the longest ventricular pauses (Fig. 46*p*); if the heart rate is slow, a long, though varying, interval separates the end of the murmur and the next first sound; the murmur is then confined to early diastole (Fig. 50), and often acquires a soft quality, making it difficult

to distinguish it from that of aortic regurgitation. The whole series of murmur arrangements may be observed in a single case which exhibits different heart rates from time to time. The reason of the changes will be clear when the pressures and mechanism are considered. The diastolic murmurs of mitral stenosis are dependent upon the rate of flow through the constricted orifice, and the rate of flow is controlled by the difference of pressure in auricle and ventricle at any given moment. Now, although the auricular pressure exceeds the ventricular during the whole of diastole, the excess is greatest at two phases, namely, when the auricle is in contraction and directly after the opening of the auriculo-ventricular valves. When the auricle contracts in normal fashion, mitral diastolic murmurs are in chief evidence at first in late and afterwards in early diastole; they are found in early diastole when the auricle is virtually paralysed, especially when, the heart rate being slow, stasis raises the ventricular pressure during the last phases of diastole.

Auricular fibrillation may be recognised clinically by the nature of the ventricular action, but its detection is aided, as we have seen, by other signs. It is possible to formulate a few general rules which serve as most useful guides in identifying it. *When the ventricle beats irregularly at a rate surpassing* 120 *per minute, the irregularity is almost always of this nature.* When an irregular ventricular action accompanies signs and symptoms of serious heart failure it is probably the result of auricular delirium, and the probability is increased if the heart rate is much accelerated. In patients in whom the heart is irregular, but in whom the heart rate is not much accelerated, and in whom signs of heart failure are absent or few, a test may be applied which is of

considerable value. Moderate exercise or the administration of amyl nitrite augment the ventricular rate, and this is so whether fibrillation is present or not; but there is a striking contrast in two given cases of irregularity, of which one is due to auricular fibrillation, while the other has a different cause (*i.e.*, extrasystoles, partial heart-block, etc.). In fibrillation the pulse becomes more irregular with its acceleration, while in the remainder the pulse steadies in rhythm. When premature beats are present, a sufficient acceleration of ventricular rate to abolish them temporarily may often be induced by several quick changes from the recumbent to the sitting posture; this is not so where fibrillation is concerned. Conversely, as the pulse shows subsequent to exercise, the irregularity of fibrillation decreases, while other forms of irregularity become more prominent. Fever similarly raises the ventricular rate, and during the febrile stage the disorder of fibrillation persists and is often augmented in degree. A study of the relation of irregularity to heart rate is the quickest and the most certain means by which auricular fibrillation can be recognised clinically. It does not matter how the rate is raised, providing it goes beyond 120 per minute, and the irregularity remains, the diagnosis is almost certain; and as the rate is higher so the diagnosis becomes more certain. Another clinical test which I have often employed with success is based on the absence of a respiratory control of rate. However deeply the patient breathes, quickening of the pulse fails to fall regularly in the respiratory phases, as it does in all other forms of heart irregularity, with the exception of those due to auricular flutter. Finally, the persistence of the irregularity, which is due to fibrillation, needs emphasis. In

most cases it is continuous from the time of observation until death. The other irregularities are present from time to time, so that there are intervals of regular ventricular action each hour or each day.

Auricular fibrillation is recognisable by means of these tests and without the aid of graphic methods in over 90 per cent. of the patients in whom it occurs.

The general symptomatology.

The symptoms complained of by patients in whom the auricles fibrillate are various, being dependent mainly upon the concomitant conditions. For the most part, they are the symptoms of degenerate and failing heart muscle, and these do not require reiteration at the present time. The symptoms which are now our special concern are those which appear to be the special effects of fibrillation itself. Patients who possess the persistent disorder often experience occasional fluttering in the chest and neck and may be conscious of irregular heart action. They are more prone to shortness of breath, exhaustion and other symptoms of over-taxation of the heart than are those with similar valve lesions and a like degree of cardiac dilatation* ; but it is not always easy to allot these superadded symptoms to precise causes ; they are in part the result of the graver myocardial condition which consorts with fibrillation ; they are in part due to the actual turbulence and embarrassment of the ventricular movements.† We know that the heart is taxed by its disorderly action, but it cannot be stated that any

* On the other hand, they seem peculiarly exempt from angina.

† The heart embarrassment is the result of ventricular irregularity and especially of rapid action ; the virtual paralysis of the auricle is probably without appreciable effect on the general circulation.

symptom, such as cyanosis, conspicuous dyspnœa, noticeable venous engorgement or dropsy, is the direct outcome of fibrillation alone ; for cardiac failure and these, its classical accompaniments are to be found where there is no fibrillation, and instances of fibrillation are common in which these symptoms have not developed. The symptoms, as has been stated, are provoked by the interplay of two factors, namely, the inherent muscle defect and the extra burden of disordered and rapid action ; while the signs of failure are proportioned to the degree of muscle damage, the whole of this symptomatic scale is raised by rapid heart action. In the healthy hearts of animals it is a general rule that fibrillation of the auricle produces a fall of arterial and a slight rise of venous pressure, but at the same time it is accompanied by a *decrease* in the heart's dimensions, a usual phenomenon when the rate is increased. The heart accommodates itself to the new conditions in a few moments ; the arterial pressure rises and the venous pressure falls, so that they almost recover their previous levels and the blood flow is maintained in a well-nigh perfect fashion. But if the heart has been damaged, the effect is both profound and lasting, and in place of *decrease* of heart volume, an *increase* may occur. So it is in patients. Patients may experience paroxysmal fibrillation at intervals of a month or perhaps a year ; many of them pass through their attacks with little or no sensibility of them ; neither can any sign, other than the disordered action, be discovered during their progress. Yet similar crises give rise in other patients to profound and serious disturbance, breathlessness, pain, cyanosis, and further indications of increasing dilatation of the heart. In these, the severest cases, the symptoms resemble those of long continued paroxysms of regular

tachycardia. Between the mild and most extreme reactions is the intermediate. The variation in the reaction is great, and, as I have said, is largely attributable to the grade of underlying muscle weakness. But there is another and equally important factor in the human subject; it is the grade of the ventricular disorder during the attack. Just as the muscle defect varies in its degree, so also does the burden imposed upon it; thus it is found that little reaction is shown in paroxysms of relatively slow ventricular action, while amongst those with grave disturbance the ventricular rate is usually rapid.

Influence upon thrombosis and infarction. If a series of diseased hearts is examined at autopsy, ante-mortem clots are found in much the heavier percentage in those auricles which during life exhibited fibrillation. The virtual paralysis of the auricles, and the consequent stagnation of blood in them, definitely predisposes to thrombosis in the appendices. Yet in mitral stenosis hæmoptysis, the result of lung infarction, appears to be no commoner where fibrillation exists than where the natural ryhthm is preserved. The explanation seems to be that although fibrillation aids the formation of clots, the passivity of the auricular walls safeguards these clots from detaching. In paroxysmal fibrillation I have observed a coincidence between embolism of lungs and brain and the resumption of the normal auricular contractions. Presumably, clots formed during the period of auricular fibrillation are detached if and when the heart resumes its natural mode of beating.

Remarks upon diagnosis.

The diagnosis, often suggested for cases which exhibit fibrillation of the auricles, is still that of the accompanying

valve lesion, though I am strongly of opinion that it is no longer warrantable. A diagnosis should include either the outstanding feature of the pathology, or it should be chosen that it may become associated with some specially beneficial form of treatment. In these patients a chronic affection of the myocardium is the essential lesion ; while the relations of the cardiac disorder to digitalis medication are so peculiar that the named disorder of the heart always brings this drug to mind.

But I wish to refer but briefly to this question of terminology under the present heading, having chosen it to emphasise a common and avoidable diagnostic blunder ; this comes from want of true appreciation of the mechanism in these cases. In discussing the signs associated with fibrillation, I have spoken of the modification of diastolic murmurs in mitral disease. A murmur, which originally occupies the full diastole of the shorter cycles, is replaced as the heart slows by an early diastolic murmur which is maximal in the region of the apex. It is the last murmur which so frequently misleads the physician and suggests to him an insufficiency of the aortic valves. It is said that in some cases of aortic regurgitation the characteristic bruit is *confined* to the apex, but this is certainly far less common than has been supposed,* and an erroneous conception of its frequence has arisen from inclusion of many of the cases to which I now refer. When mitral stenosis and auricular fibrillation are present in the same patient, *and the heart rate is slow*, an early diastolic murmur, most clearly audible at the apex but often audible around and above it, is

* Without actually denying its occurrence, I can but state that I have yet to see an example of it.

an expected sign. A diagnosis of aortic reflex is never justifiable when the heart is grossly irregular and slow, unless unequivocal signs of it are present apart from such a murmur. Uncomplicated aortic valvular disease and fibrillation of the auricles is a comparatively rare clinical picture. The combination, yielding a purely apical murmur, is so far an undescribed condition. Close attention to the character and accurate timing of the adventitious sound is often helpful. The early diastolic murmur of mitral stenosis is relatively low in pitch and it usually begins a little later than the second sound. The absence of a waterhammer pulse and of a murmur at the aortic cartilage should indicate the mitral valve as the probable seat of mischief.

The prognosis.

As in all other kinds of heart irregularity, the prognosis is largely governed by the remaining symptoms and signs, and in any individual case an estimate is formed, which includes consideration of the past history, the presence or absence of serious symptoms and their relation to exercise, the presence or absence of enlargement, of valve lesion, renal disease, etc.. Fibrillation is in many cases significant in that it is the chief or only reliable sign that the heart muscle has been affected. It is, as I have said, in itself an evidence of muscular damage. In most cases it heralds cardiac failure, temporary or terminal, so that few patients survive its onset for more than ten years. There are well authenticated instances in which it has persisted for a longer period, but they are few. A persistent ventricular rate of 120 or over is of serious omen, and according as the rate is maintained above this count, so the outlook becomes graver, providing

that the patient remains untreated. Rates of 140 and over are rarely maintained for many months, rates of 160 do not continue for many weeks. Such extra loads are not tolerated by a defective muscle. An extremely important consideration is therefore the reaction to treatment. As we shall see, most of these cases react to cardiac drugs; in many—probably in all where the original rate is much in excess of normal—the rate can be controlled, being reduced and maintained within limits which spare the heart excessive taxation of its strength. In dealing with a patient who has a given heart rate, the prognosis, in so far as it is affected by the fibrillation, does not depend so much upon the heart rate first observed as upon the heart rate which persists under treatment.

If two patients *showing equal signs of cardiac failure* differ in that the one presents a normal rhythm, while the other manifests a rapid ventricular action, the result of fibrillation, then the prognosis is more favourable in the latter; for in him the heart is burdened by a load which treatment will remove.

The treatment.

There is no other serious cardiac disorder which may be so speedily benefited as the well-managed case of auricular fibrillation. In no other cardiac affection can the medical attendant point with more thorough confidence to the effects of his remedies. As a direct result of active treatment the moribund may be restored, and many years may be added to their lives. Auricular fibrillation is the condition to which drugs of the digitalis group almost exclusively owe their well-founded reputation.

The guide to the physician is the rate of the heart beat, an index which rarely fails him. Auricular fibrillation is an absolute indication for the administration of a member of the digitalis group, whenever the heart rate exceeds 100 while the patient is at rest. In most of these patients the drug acts as a specific, impeding the passage of impulses from auricle to ventricle and thus reducing the ventricular rate. If the heart rate does not fall as a result of rest, and if it does not fall when digitalis or an allied drug has been properly administered, no other remedy is known which is of service in reducing the heart rate. In young people, and especially those who have been affected by rheumatism or chorea, it is certain that an absolute control of the rate can almost always be established and maintained. The treatment consequently consists of the administration of such doses as will keep the heart rate within reasonable limits. As more experience is gained of this treatment, the fewer become the failures; cases in which the heart rate fails to respond to full doses are now known to be very rare.

It does not necessarily follow that a patient who has fibrillation should lie up. But where the average heart rate exceeds 100 it is advisable, and the patient should remain in bed until his reaction to digitalis, or a similar drug, has been thoroughly investigated. Further treatment in bed is decided upon according to the general condition, and according to the tolerance and reaction to digitalis. In all where a high ventricular rate obtains, a reaction is speedily forced. These latter may be divided into three classes: the first, those in whom the reaction is a permanent one; these are patients in whom the rate remains slow though digitalis is omitted; they are comparatively rare: the second, those in whom the

reaction is permanent when small doses are subsequently administered : the third, those in whom relatively high dosage is persistently required to maintain control.

As a routine, the tincture or fresh infusion of digitalis is given, for it is a safe and most potent remedy. The tincture is given to adults in doses of from 10 to 20 minims three or four times a day (the infusion in 1 to 1½ drachm doses) ; if the reaction does not begin within four or five days, the dosage should be maintained or even increased until symptoms of nausea, diarrhœa, headache or pulse slowing appears. It usually happens that the desired fall of heart rate first comes just before or at the time when other signs of intoxication are manifested ; if these persist for several days the drug must be reduced or omitted, whether the rate has fallen or not. The dosage is also to be reduced if the heart rate falls, and the reduction is continued so long as the heart rate remains below 80. The quantity may be diminished to nothing in many cases ; often 5 or 7 minim doses eventually suffice. Usually the full reaction is obtained after six or eight drachms of the tincture, or an equivalent quantity of infusion, has been given. Whenever the average rate has reached 60 to 80 per minute, the drug is stopped, and it is given again only if the heart rate begins to accelerate once more. The appearance of coupled heart beats (Fig. 50) is always a sign of danger ; whenever they appear* the digitalis must be discarded. I have seen more than one case of unexpected death, attributable to continued dosing with digitalis after this stage has been reached ; it must be remembered always that digitalis is a poison, and that it has other actions than the simple reduction of heart rate.

* They do not usually appear until the rate has been reduced below the justifiable limits.

In the average, it may be said that a patient will usually use up or excrete some 15-20 minims of the tincture daily, and that it is not until such doses are exceeded that accumulation of the drug occurs in the body. To produce the full effects, and that is often necessary, an average quantity of 5 drachms has to be accumulated. This is accomplished by giving 7 drachms over a period of 7 days. With 5 to 7 minim doses there is usually no accumulation, but a previous accumulation may be maintained by these amounts.

In some instances where the patient has reacted, the drug can be stopped without the rate re-accelerating so long as he remains in bed. When he rises from bed a renewal of the small dose (5 to 7 minims) may be, and usually is, necessary. In other cases the result is less satisfactory and heavier dosage must continue. The administration of digitalis in small or moderate doses may be continued beneficially for years in many patients.

It sometimes happens that a patient is peculiarly intolerant to digitalis, and that, where a reaction is expected, a dosage of 15 to 20 minims of the tincture cannot be reached or maintained sufficiently long, without nausea or other discomforting symptoms supervening. The drug should be diluted and given directly after meals. Strophanthus (in its new B.P. strength) or squills may be tried, starting with doses of 10 minims or 20 minims of the respective tinctures. These drugs are pushed in the same manner, but though they are less apt to induce nausea or vomiting, and while diarrhœa is the chief disturbance produced by them, they are less reliable than digitalis. In some of these cases, too, recourse may be had to intravenous injections of strophanthin.

When a patient who has fibrillation is first seen, and the heart beats persistently at 170-200 per minute, the condition is urgent and heavy doses of digitalis (20 to 30 minims) should be employed. The intravenous injection of strophanthin is also valuable at such times. Two, or not more than three, doses of 1/250 of a grain, each in 40-60 minims of saline, are given at intervals of two hours. The reduction of rate begins almost at once, and heart rates of 90 or 80 are reached in from 6-12 hours. The remedy should be employed cautiously, and its adoption must be confined to the urgent case with very high ventricular rates, or to cases in which medication by the mouth has been hindered by the onset of gastro-intestinal symptoms.

The treatment of the case of auricular fibrillation in the patient who is up and about is guided mainly by the rate of the heart and the urgency of the patient's symptoms. The disorder is generally persistent, and most hospital patients eventually leave their beds and return to their former or somewhat lighter occupations; the latter are naturally advised. But even where the pulse rate is persistently low and symptoms are few, excessive exertion should be avoided; heavy manual labour, strenuous games and sports should form no further part in the daily life. If the pulse rate quickens readily, if drugs are constantly required to maintain a low ventricular rate, and especially if breathlessness or precordial uneasiness are easily induced, further restrictions are necessary. All patients of the female sex should be specially warned of the strain and danger of pregnancy.

Regular meals, consisting only of a sufficiency of solid and sustaining food, preferably dry, early hours, a placid existence, the avoidance of public buildings and all places

and seasons in which influenza and bronchitic troubles are contracted, and, lastly, scrupulous attention to the hygiene of the teeth and throat, are sound directions in this as in other serious heart maladies.

Belladonna, its ally hyoscyamus and their extractives should be avoided. Their customary action is to increase the rate of the ventricle in this condition.

In cases of urgency, or where the patient's life may be considerably prolonged by surgical operation, general anæsthetics may be employed. But where there is any hesitancy to perform an operation, apart from the cardiac condition, the presence of fibrillation should countermand it.

Paroxysmal fibrillation.

Most auricles which develop fibrillation maintain this mechanism to the end of the chapter; it is essentially a chronic and at last a terminal malady. But from time to time transient attacks are seen, and in some patients paroxysms of fibrillation lasting a few hours, days or weeks are noted. The affection, when it takes this form, is generally classed as paroxysmal tachycardia. In my discussion of paroxysmal tachycardia I have excluded it, desiring to deal, as I did, with the simpler mechanism alone.

The frequence of the paroxysmal affection may be gauged approximately by comparison. Of the 152 cases of auricular fibrillation included in the table on page 93, in only 16 was the disorder temporary and recurring. Paroxysms of regular tachycardia appear to be more common; while the 16 irregular tachycardias were being collected, simple and regular paroxysms were seen in 45 patients.

The symptoms of paroxysmal fibrillation have been spoken of already. They may be inconspicuous or profound. When the rate of ventricular response is rapid (160-200 per minute) the symptoms are those of simple tachycardias at similar rates, though they are on the whole more severe. The prognosis is reasoned in the manner stated for regular paroxysms ; the management and symptomatic treatment of the attacks are similar in the two. A few words are necessary upon digitalis medication. Drugs belonging to this group have been known, not infrequently, to excite fibrillation in those predisposed. In general they are therefore contra-indicated in paroxysms of short duration. When the paroxysm of fibrillation is prolonged and lasts for fourteen days, the fibrillation may be regarded as permanent and treated as is the persistent condition. In urgent paroxysms, where high ventricular rates prevail, digitalis medication may be adopted with advantage ; the dosage should be arranged for a speedy reaction. The drug affords quick relief, though by its use the duration of the attack may be prolonged.

Acute fibrillation.

Fibrillation of the auricles sometimes intervenes during the course of acute infections and continues for a few moments, hours or days. I have witnessed attacks of this kind in severe tonsilitis, acute cholecystitis, acute appendicitis, infective endocarditis and pericarditis, and others have been reported in scarlet fever and in pneumonia.

CHAPTER VIII.

ALTERNATION OF THE HEART.

Definition.

A condition in which the left ventricle, while beating with regular rhythm, expels larger and smaller quantities of blood at alternate contractions.

The mechanism in alternation of the heart.

Alternation in the size of pulse beats, so that each alternate beat is large and each alternate beat is small, is of obscure origin. The contractions of the ventricle are regular,

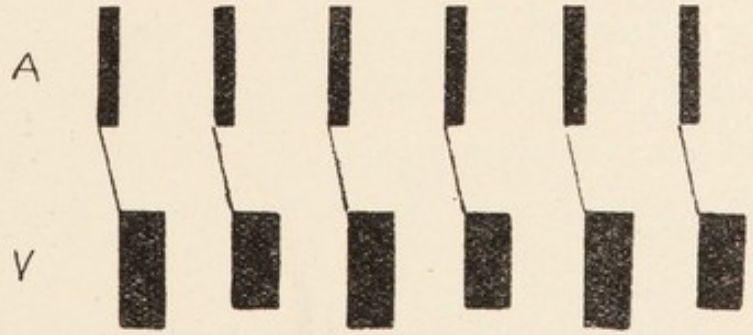


Fig. 51. A diagrammatic representation of alternation of the heart. The auricular and ventricular beats are placed regularly and in order, but alternate ventricular contractions are weak.

and each is preceded at a normal interval by a contraction of the auricle (Fig. 51). The disturbance is dependent upon an anomaly of the ventricular systoles, whereby at each alternate systole of the left ventricle a greater or lesser quantity of blood is thrown into the systemic arteries. In

the figure, I have represented this anomaly by varying the size of the ventricular rectangles. The hypothesis is that a different number of the ventricular fibres contract at alternate systoles.

Etiological and pathological relations.

Alternation of the pulse is seen in two classes of patient.

First, it occurs in those in whom the heart rate is unduly accelerated and more especially as an accompaniment of paroxysmal tachycardia. Associated with paroxysmal tachycardia, it has etiological and pathological relations in common with the last named disorder ; its prognostic significance is in these circumstances negligible, depending as it does chiefly, if not entirely, upon acceleration of the heart rate. It may be regarded almost as a physiological reaction to the increased frequency of the heart beat.

Secondly, it occurs when the heart rate lies within normal limits and at such times it is a sign of considerable import. Seen in elderly subjects and pre-eminently in the male sex, it consorts especially with angina pectoris, high arterial pressure, renal disease, and a fibrotic myocardium. It has been seen in pneumonia during the pre-critical stage, and also in patients under the influence of large doses of digitalis.

Alternation of the heart is encountered in experiment under similar circumstances, namely, when the heart rate is extremely rapid, or when the organ has been injured by the intravascular injection of poisons. Whenever it occurs, there is reason to believe either that a tolerably healthy heart muscle is carrying an excessive burden, or that a diseased or poisoned muscle is struggling to perform work of which it is barely capable.

In the remainder of this chapter I shall allude to pulse alternation as an accompaniment of heart rates which are not high. When the heart is disposed to alternate, the actual alternation is unmasked by anything which imposes a fresh and added strain upon that organ. Thus it is often made manifest by a slight acceleration of pulse rate; and in the earlier stages of its development, it is frequently exposed by single premature beat; in the last named circumstance it follows immediately upon the extrasystole, and continues for a varying number of heart cycles.

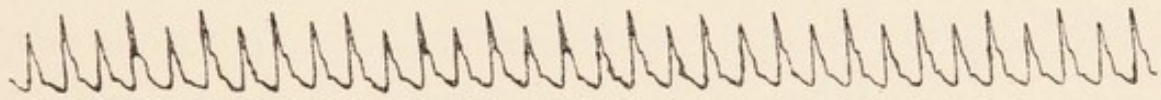


Fig. 52. Alternation of the pulse. Each alternate beat is strong and each alternate beat is weak.

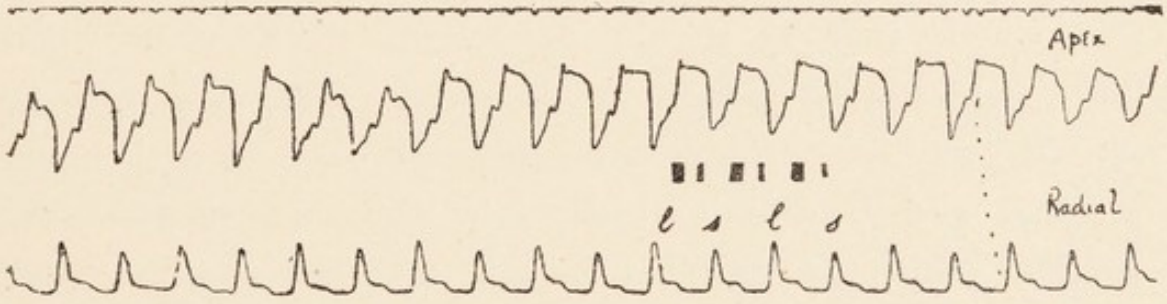


Fig. 53. Apex and radial curves and heart sounds in heart alternation. The curve is taken at a faster rate than the last and shows the slight variation in pulse pauses. As opposed to the picture when premature beats are present, the *stronger* beat is followed by the *longer* pause.

It is an affection of advancing years, its heaviest incidence being in the fifth, sixth and seventh decades; it is much commoner in men than in women. Dr. White, of Boston, who collected seventy-one cases in eight months, considers it is as frequent as fibrillation of the auricles; certainly it is a common disorder, generally passing unobserved.

The recognition of pulsus alternans.

It is unfortunate, but true, that most instances of *pulsus alternans* cannot be recognised other than by instrumental means. There are patients in whom it affects the pulse continuously, and in whom alteration in the force of alternate pulse beats is perceptible to the finger* ; but such cases are rare, and the feel of the pulse should not be trusted. Examination of the cardiac impulse gives little assistance, for the heart beats with regular rhythm and the differences in the force of ventricular systoles and the intensity of the heart sounds are inappreciable.

The systolic pressure of the large beats is several millimetres higher than that of the small beats ; by nicely regulating the pressure in the armlet of the sphygmomanometer, the small beats may be hindered from passing, and the pulse rate below the armlet is thus halved.

It is a sign of such importance and is so readily overlooked, that it should be sought deliberately when there is reason to suspect its presence. Thus it is wise to examine all cases of angina pectoris, all cases of high blood pressure and all elderly subjects in whom affection of the heart is suspected, or renal disease is known to exist, with a specific object, namely, to determine its presence or absence. It should be looked for, too, in all elderly people in whom premature beats are frequent. If such methods are adopted it will not often escape detection. It is so frequently confined to the few cycles which follow a premature beat that, in any class of the patients mentioned, it is important to obtain a curve which contains such a beat. This may often

* The separation from a dicrotic pulse is easy ; for where the latter is present the beating of the pulse is at twice the rate of the ventricle.

happen at the first examination. The patient should remain standing, for premature beats are more frequent in this posture, and if he has come some distance it is well that the examination should proceed at once, since premature beats are more conspicuous at such times. It should be remembered, too, that a held breath may evoke a premature beat, and the opportunity of detecting alternation in this manner should not be lost.

Single premature beats are usually followed by a pulsation of exceptional size, for the heart puts out more than its usual quantum of blood. It is the pulse which succeeds this tall beat which shows the first sign of weakening; it is less forcible than that which succeeds it. In Fig. 54 a regular pulse is interrupted by a single premature contraction (p);

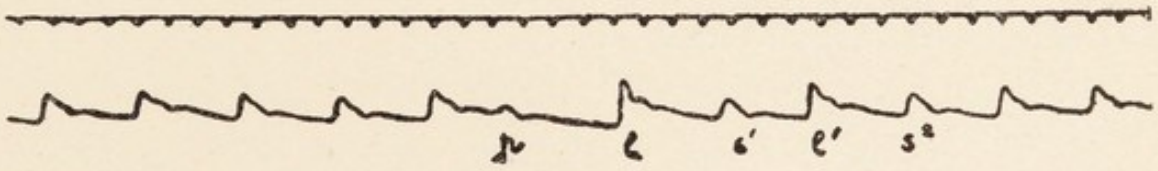


Fig. 54. Alternation of the pulse, appearing after, and as a result of, a single premature beat p. It lasts for four heart cycles.

it is followed by the usual pause, and this is succeeded by a tall pulsation (l); the next beat s^1 is small, it is followed by a taller beat l^1. The small beat s^1 is, as I have said, the earliest sign of the condition, and it may be the sole sign. In the actual figure s^2, the next alternate beat, is also low. Alternation has proceeded for four cycles before the normal pulse beats are restored. In Figs. 52 and 53 the condition is persistent throughout each curve; little and big beats are arranged alternately. Extreme degrees of alternation of the pulse are seldom encountered; but on very rare occasions

the little beats vanish entirely, and the pulse rate is halved for short periods.

The other irregularity with which *pulsus alternans* may be confused is a coupled pulse resulting from premature beats, but this only happens when the prematurity of the second beat of each couple is slight. An example is shown in Fig. 33 of an earlier chapter. There is sufficient contrast between them ; whereas the little beat in Fig. 33 is followed by the longer pause, if *pulsus alternans* is present the little beat is followed, if there is any variation in pauses, by the slightly shorter pause. In tracings written upon slowly travelling paper a difference in intervals is hardly perceptible (Fig. 52) ; but where the paper has moved faster, a measureable difference is often found ; it is well seen in Fig. 54, in which the pauses following l and l^1 are longer than those following s^1 and s^2.

The subjective sensations of patients presenting pulsus alternans.

Alternation of the heart is responsible for no symptoms ; the patient only complains of sensations which are referable to other causes. Thus, anginal pain is common. Breathlessness is even commoner ; it is often nocturnal, repeatedly awakening the subject of it after short periods of sleep and being accompanied by acute anxiety. Breathing of the Cheyne-Stokes type is rarely noted by those who manifest this respiratory abnormality, but periodic dyspnœa may be remarked by the friends, especially by those who sleep with the patients.

The prognosis.

Alternation of the pulse belongs to a small group of phenomena witnessed by those who attend the sick, which, treated as isolated signals, are in themselves emphatic and portentous. It ranks with *subsultus tendinum*, with *optic neuritis*, with the *risus sardonicus* and other ill-omened messengers. It is the faint cry of an anguished and fast failing muscle, which, when it comes, all should strain to hear, for it is not long repeated. A few months, a few years at most, and the end comes.

How grave is the condition of the patient whose heart produces this alternating pulse is often witnessed to by associated signs ; angina, nocturnal dyspnœa, Cheyne-Stokes breathing or high blood pressure are often encountered in the same subject. But here lies its special significance : each and all of these signs may fail, while alternation is there to tell the future.* Unexpected death is not uncommon.

I write of continued alternation of the pulse which alternates in force for many cycles. It is persistent while the heart yet lives. The prognostic value of the lesser grades of alternation is less certainly known ; but that their significance is grave, and that they are but too often the forerunners of the fully developed condition, should be understood. A favourable prognosis is always forbidden by the latter, and can be but rarely justified in the presence of the former. The only propitious circumstances are a history of exceptional and prolonged strain in the patient who shows the sign, strain which may be at once and permanently avoided, or the presence of acute intoxication which is vanishing.

* It is true, however, that alternation is never the sole sign of disease ; patients who exhibit it always manifest a poor tolerance of exercise.

The treatment.

The management of heart cases, in so far as it is affected by the sign alternation, may be stated in a few words, for it should be evident. Alternation speaks of over-taxation; it demands relief. In busy people it calls for prompt and drastic curtailment of work, be it mental or physical exertion. In the more sedentary, it is an indication for prolonging the hours of actual rest, both of body and of mind; the condition of such patients may undergo temporary relief by a long period of absolute quiescence. In each case sources of anxiety or emotion are to be avoided. The presence of alternation forbids the administration of general anæsthetics in major operations, unless the withholdment of the first immediately jeopardises life, or unless one or other is necessitated for the relief of intolerable pain.

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