Alternation of the Heart

By David Littmann, M.D.

ALTERATION of the heart is an uncommon disorder of function that can involve tissues from the sinus node to the ventricular myocardium (table 1). It may be clinically obvious as in alternation of the blood pressure and pulse or conjectural as in alternating atrioventricular conduction block. Other forms are identified only by electrocardiogram and constitute a group of electrical alternations. With a single important exception due to shifting cardiac position, all the alternating mechanisms are on a cellular level resulting from incomplete recovery or persistent refractoriness. Whatever the underlying cause, it follows that recovery in some part of the heart is not wholly attained within one cycle length. Fiber response becomes delayed in fractions or multiples of the established interval and bears an arithmetic relationship to it as 3:2, 2:1, and the like. Alternation ensues when the ratio is 2:1. Block of this degree occurring at normal heart rates implies a pathologic or abnormally prolonged refractory period. At extreme rates, particularly in ectopic tachycardias, alternation can result from normal differences in the recovery rate of individual fibers.

Mechanisms, Significance, Illustrations

Alternating Sinoatrial Block

The sinoatrial node has a histologic appearance resembling the atrioventricular node and, like it, can exhibit block.1 Normally, sinoatrial activity is not separately apparent but is inferred from the atrial manifestations that follow. Minor delay in conduction through it (1° block) is therefore not evident. Sinoatrial block with 2:1 response is similarly undetectable, if it is persistent, though it may be suspected in inappropriately slow heart rates. When it is intermittent, however, the true sinus rate is revealed during intervals of normal conduction (fig. 1A). Two-to-one sinoatrial block results in a failure of atrial activity at the anticipated time in alternate beats, with a slowing of the atrial rate by one half. Three-to-one sinoatrial block causes the loss of every third beat and an electrocardiographic pattern that resembles bigeminy resulting from coupled atrial ectopic beats (fig. 1B). However, the P waves are identical in all contractions, and the long intervals are approximately or exactly equal in length to two short ones. The Wenckebach mechanism, which frequently occurs in this situation, causes increasing sinoatrial intervals in the conducted beats and a P-P time longer than the interval between sinus stimuli. This asynchronism is corrected during and at the expense of the nonconducted beat.1 It results in a long interval which may be considerably less than two short ones.

Sinoatrial block occurs, probably, for the same reasons responsible for atrioventricular block; they are occasionally seen together. Both may be seen with sinus arrhythmia, possibly because of vagotonia. Sinoatrial block is unusual in the absence of cardiac disease or toxicity; it sometimes follows digitalis excess and may be suspected in the extreme bradycardia that occurs with inferior myocardial infarction. Alternating (2:1) sinoatrial block does not produce symptoms other than an awareness, when it occurs, of a shifting heart rate. Greater degrees of block can, however, cause faintness and even syncope.2 Except

From the Veterans Administration Hospital, West Roxbury, Massachusetts.
when due to organic disease of the node, sinoatrial block can be abolished by atropine and exaggerated by carotid sinus pressure.

**Alternating Atrioventricular Block**

Alternating failure of conduction through the atrioventricular node, more familiarly known as 2:1 atrioventricular block, occurs at normal heart rates as the result of abnormal refractoriness. It is generally due to inflammatory or ischemic disease of the myocardium or to drugs. It probably does not occur as a normal variant and is unusual even with severe digitoxicity. With rapid atrial dysrhythmias some degree of atrioventricular conduction failure is frequent and is the rule in special circumstances as with atrial flutter and fibrillation. It may respond to vagolytic agents at normal heart rates by a change in response as to 3:2 or by a reduction to first-degree atrioventricular block.

Retrograde spread from an atrioventricular nodal pacemaker is subject to the same influences that affect normal forward conduction. Alternating or 2:1 retrograde block permits atrial activity and abnormal P waves (falling between QRS and T waves) in alternate beats (fig. 2). This is a singular phenomenon which, however, has no significance beyond that implied by the underlying atrioventricular nodal rhythm. It is seen in digitoxicity.

**Alternating Bundle-Branch Block**

Alternating or 2:1 bundle-branch block is an exceptional mechanism which may, however, occur at some time in most patients who later develop permanent bundle-branch block. As with delays elsewhere in the heart, it is the result of incomplete fiber recovery within the diastolic interval. It occurs at critical heart rates and can go on to complete block at higher frequencies. Alternate beats may show lesser degrees of block or completely normal conduction (fig. 3A). It is of interest that this type of alternation is seen almost exclusively with disease of the left branch and rarely, if at all, in "normal" right bundle-branch block. It has no additional clinical significance and may be considered a variant of rate-controlled bundle-branch block.

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**Table 1**

<table>
<thead>
<tr>
<th>Classification of Cardiac Alternations</th>
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<tbody>
<tr>
<td><strong>I. Due to abnormal refractoriness</strong></td>
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<tr>
<td>A. Of the conducting system</td>
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<tr>
<td>1. At normal heart rates</td>
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<tr>
<td>a. Sinoatrial node, 2:1 S-A block</td>
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<td>b. Atrioventricular node, 2:1 A-V block, 2:1 V-A block</td>
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<tr>
<td>c. Bundle branch, alternating B. B. B.</td>
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<tr>
<td>d. Peri-infarction block, alternating P.I.B.</td>
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<tr>
<td>e. Ectopic conductor, alternating ventricular pre-excitation</td>
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<tr>
<td>f. Purkinje system, &quot;pure&quot; electrical alternation</td>
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<td>2. With ectopic tachycardias</td>
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<tr>
<td>a. Supraventricular</td>
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<tr>
<td>b. Ventricular</td>
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<tr>
<td><strong>B. Of the myocardium</strong></td>
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<tr>
<td>a. Atrial, P-wave alternation</td>
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<tr>
<td>b. Ventricular, Pulsus alternans</td>
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<tr>
<td>Left ventricular</td>
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<tr>
<td>Right ventricular</td>
</tr>
<tr>
<td><strong>II. Due to alternating shift of cardiac position, total electrical alternation</strong></td>
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<tr>
<td>A. Pericardial disease with effusion</td>
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<tr>
<td>a. Neoplastic</td>
</tr>
<tr>
<td>b. Tuberculous</td>
</tr>
<tr>
<td>c. Uremic</td>
</tr>
<tr>
<td>d. Other</td>
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Peri-infarction block is a form of left intraventricular block frequently seen with myocardial infarction. It is believed to result from ischemic injury to one of the main subdivisions of the left branch. Block of the anterolateral division is the more common, particularly with anterior injuries, whereas inferior segment blocks are more likely with diaphragmatic infarctions. The QRS time need not be greatly prolonged but sometimes approaches the duration of classical bundle-branch block. However, since the injury spares the proximal segments of the conducting system, the septum is normally innervated, the left cavity is initially negative and abnormal Q waves can appear. This is despite a QRS configuration that may otherwise resemble ordinary left bundle-branch block. In addition, because of the abnormal order of accession, the electrical axis rotates to an unusual degree, horizontally or less often ver-
tically. As might be anticipated, 2:1 conduction through either left branch division may precede permanent block and appears, rarely to be sure, as alternating peri-infarction block (fig. 3B). It is not believed to have any significance beyond that of permanent peri-infarction block. It does not occur as a normal variant.

**Alternating Pre-excitation**

Another interesting variant of 2:1 bundle-branch block is seen with alternating activity of the ectopic conductor in anomalous ventricular pre-excitation (fig. 3C). In this circumstance the abnormal complexes occur with premature ventricular depolarization through an accessory conductor. These alternate with normal beats when the anomalous conductor fails to function.¹ There is no way, in any one record, to distinguish this form of alternation from coupling with ventricular ectopic beats when these come so late in the cycle that they follow the P waves and appear between them and the anticipated normal ventricular complexes. The resultant beats are fused ventricular premature beats exactly similar to those seen with ectopic conduction. Alternating activity of the anomalous conductor probably occurs fairly often in patients with the Wolff-Parkinson-White syndrome and has been seen prior to complete and possibly permanent loss of function of the accessory conductor. It has no other known significance.

**Alternating Arborization Activity**

Alternating or 2:1 block of the conducting

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**Figure 1**

A. Inconstant 2:1 sinoatrial block in a 63-year-old patient with arteriosclerotic heart disease. The longer P-P intervals are approximately twice as long as the shorter. There is sinus arrhythmia, however, which disturbs an exact relationship. In V₁, for example, each of the first two long intervals is almost exactly as long as the sum of the two preceding short ones, though the latter are of unequal length. The third long interval is considerably shorter than the first two: 1.54 second, 1.52 second, and 1.42 second. There is also atrioventricular block with somewhat shorter P-R duration after intervals of sinoatrial block than in its absence. B. An example of 3:2 sinoatrial block simulating coupling with atrial ectopic beats; 2:1 block is briefly present in lead II. The P waves are all alike but the P-R intervals are longer in the more rapid beats. The long intervals are somewhat less in duration than the sum of two short ones. This is due to the Wenckebach effect on the sinoatrial interval. From a 64-year-old man without known heart disease.
system below the level of the bundle branches and their main divisions produces what, by some definitions, is designated "true electrical alternation." This term is applied where the origin of the cardiac stimulus, the intervals between beats, and the QRS duration remain constant. Stated otherwise, only the amplitude, form, or direction of the ventricular complex exhibits alternating differences. Measurable variations in the QRS are always accompanied by secondary T changes, though these may not always be apparent on conventional recordings. Differences limited to the ST segments and T waves without change in the QRS have been obtained in animal preparations but have not, prior to this paper, been reported in human subjects.

The electrocardiogram illustrated in figure 4B was obtained from a man of 32, admitted for treatment of prostatitis. He was not very ill when the tracing was made; he had not yet been begun on therapy and his temperature was normal. There was no evidence of heart disease. The alternation was gone when the patient was restudied 2 years later. There is a well-marked T alternation with barely measurable differences in the QRS complexes.

When it appears at normal heart rates this type of alternation is almost always evidence of important myocardial disease. Under rather special but wholly obscure circumstances, myocardial disease can result in, among other things, a pathologically prolonged refractory phase involving segments of the Purkinje system. This alters the pattern of depolarization which is inscribed, however, with no measurable delay. Depolarization of the incompletely innervated myocardium occurs by spread from neighboring fibers in the same manner and at the same time taken by the normally distal myocardium of the outer layers of the heart. When this type of arborization block occurs in a ratio of 2:1 it produces pure electrical alternation. It is a subtle variant of 2:1 bundle branch and of peri-infarction block, and has a comparable significance. Superficially, it appears less ominous, since none of the beats is so grossly deformed as in the other conditions (fig. 4A). With one exception, however, I have not seen electrical alternation at ordinary rates in records that would otherwise have been considered normal. It may be permanent, transient, or repetitive. It can disappear, despite other evidence of progressive cardiac deterioration.

At very rapid frequencies, inherent but normal differences in recovery rates between individual fibers of the conducting system may become manifest and produce electrical alternation. For example, in adults at atrial rates in the vicinity of 240 per minute a blocked atrioventricular response is anticipated, as in atrial flutter. In some individuals with rapid rates, 2:1 block of the lower segments of the conducting mechanism develops...
Figure 3

A. Alternating left bundle-branch block. The second and fourth beats exhibit the pattern of left bundle-branch block; the first and third have normal conduction. From a 68-year-old man with hypertension and coronary heart disease. Not long after this record was obtained left bundle-branch block became established and normal conduction was never again observed. B. Alternating peri-infarction block. The first, second, and fourth beats in each lead have a duration of 0.16 second and the pattern of anterolateral peri-infarction block. The alternate beats are of normal duration and indicate an anterolateral infarction. Note the alternating axis shift from +60° to −30° and the complete change of direction in V₅. From a 60-year-old man with ischemic heart disease. C. Alternating anomalous ventricular pre-excitation; 2:1 block of an ectopic conductor except for the last two complexes in V₅, where it operates for two successive beats. The characteristic pattern of W-P-W conduction (type B) in a patient with a history of paroxysmal rapid heart action.

instead and produces electrical alternation of the QRS complexes (fig. 5A). The first reported case of electrical alternation by Lewis¹⁴ was of this type. Despite the logic of this concept, however, it is doubtful if electrical alternation during atrial or supraventricular tachycardia is truly benign and wholly innocent. It is an unusual manifestation in the great majority of paroxysmal atrial tachycardias, even at rates approaching 300 per minute. It is seen rather more often in patients with some underlying myocardial disease (fig. 5B) and especially during attacks of rapid heart action in patients with the Wolff-Parkinson-White syndrome (fig. 5C). It is conceded, however, that hearts driven by tachycardia to or beyond the limits of normal function may exhibit alternation which is less significant than at normal rates.

**Ventricular Tachycardia with Alternation**

Electrical alternation during ventricular tachycardia is not unusual and can exhibit complexes written in diametrically opposite directions. In this form it is designated bidirectional ventricular tachycardia¹⁷ (fig. 6A). It is never seen as an innocent finding, but occurs with extreme or terminal myocardial disease, usually with digitoxicity.¹⁸,¹⁹ Despite the fact that alternating impulses appear to originate in opposite ventricles, this is not believed to be the case. It is possible, in some instances at the beginning and ending of periods of alternation, to see the development or waning of differences between beats (fig. 6B). Electrical alternation in ventricular tachycardia is an invariably significant finding. However, it is perhaps very slightly less ominous than otherwise uncomplicated ventricular tachycardia, since it suggests digitoxicity as an underlying complicating factor. When this is the case, it sometimes can be abolished by appropriate therapy.
Disorders of the Myocardium

Atrial

Isolated alternation of the atria has been observed and reported just twice. However, since P waves are neither large nor angular, minor variations could easily escape notice. One published example occurred in a patient with amyloid disease of the heart. The other was briefly present in an individual with thyrocardiac disease soon after reversion to normal rhythm from atrial fibrillation. It was believed to have resulted from prior medication with propyl thioracil and quinidine. In both instances alternation would have to be attributed to disease of the atrial myocardium. The extraordinary rarity of this manifestation prevents any conclusions regarding its importance.

Ventricular

Alternation of the pulse and blood pressure is a form of mechanical alternation that can be clinically detected. It is relatively common in advanced myocardial disease from many causes, especially in ischemic and hypertensive heart disease and in aortic stenosis (fig. 7A). It may also occur as an unimportant concomitant of paroxysmal tachycardia. It is readily recognized by careful auscultation of the systolic level while obtaining the blood pressure. In this manner, slight variations of 2 to 5 mm. may be seen that escape notice by palpation of the pulse. Actually, pulse variations are difficult to appreciate when they are less than 20 to 30 mm. of mercury. It may involve either ventricle separately. Right ventricular alternation is encountered generally in pulmonary hypertension and other disorders of the right ventricle. It is detected only during cardiac catheterization (fig. 7B). The cause for mechanical alternation is probably quite similar to that responsible for electrical alternans. Abnormal refractoriness of portions of the myocardium leads in some hearts to 2:1 myo-
cardiac block or failure to respond. Unlike electrical alternation, however, this results in a variation of stroke output and generated pressure. It follows that noncontraction of a significant bulk of myocardium should be accompanied by an altered electrical pattern. Some observers, in fact, believe that the underlying mechanism is fundamentally the same in the two conditions and that one cannot be present without the other.\textsuperscript{7-11} It has even been suggested that inadequacies of recording methods prevent their more frequent recognition. These opinions notwithstanding, modern, quite accurate recording devices fail, in the great majority of cases, to reveal coincident electrical and mechanical alternans.\textsuperscript{12} It appears more likely that normal or customary depolarization of all the myocardium takes place with every beat, while incomplete or otherwise feeble contraction of some of the fibers (hypostole) obtains in half of them. Despite their many similarities, pulsus alternans and electrical alternation are practically never found simultaneously. An exceptional case was the moribund patient reported by Groedel and Miller.\textsuperscript{8} Despite his agonal state, the two forms of alternation occurred at the same time only during the apneic periods of Cheyne-Stokes respiration. Probably much of the myocardium had already sustained near-lethal injury and was unable, during periods of added hypoxia, either to depolarize or contract with alternate beats. Another patient who exhibited both

\textit{Alternation of QRS is best seen in} V_4 \textit{but T alternation is somewhat more prominent in lead II. The rate is only 155 per minute. B. Electrical alternation during paroxysmal atrial tachycardia. From a 33-year-old man with cardiac enlargement of unknown etiology. QRS alternation is well seen but no differences in T waves can be identified. C. Electrical alternation in atrial tachycardia. Variation in the height of the ventricular complex and of the R:S ratio without change in intervals. Marked secondary T-wave alternation. The rate is only 120 per minute. This was obtained from a 30-year-old concert singer with a history of paroxysmal rapid heart action who exhibited anomalous ventricular excitation at other times. Tachycardia would occur when he “reached” for an unusually high note.}

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types never had them together. Still another with simultaneous mechanical and electrical alternation suffered from a disorder of the pericardium without, as far as is known, any myocardial disease.

Mechanical alternation (pulsus alternans) is never a benign finding except at very rapid heart rates. Like electrical alternans it can be permanent, intermittent, or very transient. Characteristically, it is often initiated by a premature beat which is followed after a compensatory pause by an unusually large contraction. This exceptional effort appears to exhaust the myocardium and is followed in turn by a smaller beat, than another large one and so on for varying numbers of pairs (fig. 7A and B). In a somewhat analogous fashion, a slight effort such as getting up from the seated position can initiate alternation in a susceptible patient when it has not been present at rest. It is seen in severe aortic stenosis and may clear after successful surgical therapy. Similarly, it may disappear after improvement of hypertension. However, it can vanish even with worsening of the underlying heart disease.

**Alternation of Position**

Attention has been directed recently to a form of mechanical alternation unrelated to disorders of the myocardium or conducting system. In certain important diseases of the pericardium, shifting position of the heart during every other beat results in alternation of all the components of the electrocardiogram (fig. 8) and sometimes alternation of the pulse as well. Normally, rotation of the heart around its long axis takes place with each beat. This results largely from contraction of the spiral musculature of the heart. Systolic unwinding of the great vessels, which comes with the rise in pressure, may also contribute. The diastolic position is restored by muscular relaxation, ventricular filling, diminishing tension, and the gentle pressure of the lungs and other mediastinal structures. In the presence of an effusion of critical volume the normal mediastinal restraints are diminished or lost. This, it is postulated, permits greater freedom of rotation during systole and incomplete restoration in diastole. It allows, therefore, the development of a rotational pendular movement, a type of cardiac nystagmus. It
so happens that the natural frequency of this oscillation at times approximates one half of an average rapid heart rate (such as might be encountered in severe pericarditis) and results in alternating exaggeration of and interference with the normal cyclic rotation.

This, in turn, permits the heart to present a regularly varying anatomic aspect to any given electrode.\textsuperscript{21} Since rotation around the long axis appears greatest in the horizontal plane of the chest, the widest variation in the electrocardiogram is seen in the precordial leads. A lesser degree of electrical alternation is projected to the limb leads. It involves all components, P, QRS, T, and probably U waves. Though alternation of the P and U waves is anticipated in each such instance, the differences from beat to beat are often so slight as to escape notice. When the ratio of rotational oscillation to heart rate is other than 1:2, a variety of positional rhythmic variations can ensue, resembling those seen normally with vigorous respiratory movements.\textsuperscript{21}

\textit{Figure 7}

\textbf{A.} \textit{Pulsus alternans.} Alternation of pressures simultaneously obtained in aorta and left ventricle during catheterization in a patient with aortic stenosis. Note absence of alternation before and its appearance after the ventricular ectopic beat and its compensatory pause. It is much greater in the ventricular tracing than the aortic and is purely systolic. There is no electrical alternation. \textbf{B.} \textit{Mechanical alternation of the right ventricle.} From a patient with idiopathic pulmonary hypertension obtained during cardiac catheterization. Alternation present in slight degree in the first part of the record becomes exaggerated after the ventricular ectopic beat with compensatory pause. There is no electrical alternation.
Rather extensive positional changes must take place to account for the striking electrical alternation that is sometimes seen. Cases have been observed in which alternating beats are almost completely bidirectional (fig. 8). This degree of rotational swing probably accounts for the extraordinary findings I saw (but unfortunately did not record) in a patient with tuberculous pericarditis. This patient exhibited, when first seen, an alternating pulse and an alternating friction rub. The electrocardiogram made at the same time showed wide, total, electrical alternation (fig. 8B). Variations in the friction rub would be anticipated with large differences in cardiac rotation. But this was also, it would appear, so great in degree as to limit cardiac output and blood pressure in alternate beats. To be sure, myocarditis of unknown extent could have been present at the same time to account for the pulsus alternans. However, all of the alternating phenomena cleared simultaneously.

Curiously, total electrical alternation has been described only in serious types of pericardial disease, neoplastic, tuberculous and, in one case described elsewhere in this report, uremic pericarditis. Though it has not been reported, there seems to be no good reason why it might not occur in benign idiopathic pericarditis with effusion. Here, however, the relatively normal parietal pericardium still transmits the restraining influence of the lungs in contrast to the thicker, more rigid pericardium of neoplastic or tuberculous disease, which does not. Additionally, some degree of tamponade must coexist, since removal of relatively small amounts of effusion serves to abolish the alternation. Although the atrial rhythm is not specified in the definition of electrical alternation, it is commonly assumed to be constant and of sinus origin. Two rather interesting exceptions have been noted and are presented here for, it is believed, the first time.

**Alternation with Lower Nodal Rhythm**

A 35-year-old patient with glomerulonephritis and renal failure was being considered for a kidney transplant from a twin brother. While under observation he developed uremic pericarditis and coincidentally atrioventricular nodal rhythm with retrograde atrial depolarization. The ventricular complexes exhibited well-marked alternation and the P waves, which appeared between the QRS and T

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*Figure 8*

A. Alternation in pericardial disease, neoplastic invasion of the pericardium. From a 60-year-old man with cancer of the lung, spread to the pericardium with hemorrhagic effusion and tamponade. Lead V1. Extensive alternation of the QRS complexes, less so of the T waves, and slight but definite alternation of the P waves; the tall complexes are preceded by the larger P waves. Note that the tall beats have Q waves while the small ones have none. B. Tuberculous pericarditis. Total electrical alternation with bidirectional complexes in V1. Rather slight alternation of the P waves is best seen in V5. A 25-year-old man very ill with acute tuberculous pericarditis, effusion, and tamponade. There was coincident pulsus alternans and an alternating friction rub.
waves, also alternated (fig. 9A). The alternation lasted but a short time although the nodal rhythm and uremic pericarditis persisted for some weeks thereafter. He made a striking recovery following successful transplant of his brother’s kidney.

**Alternation with Atrial Fibrillation**

A 62-year-old patient with primary cancer of the lung developed pericardial spread with hemorrhagic effusion and tamponade. The electrocardiogram made while he had regular sinus rhythm showed total electrical alternation of the shifting position variety. The rhythm changed, not long before death, to atrial fibrillation with a rapid and fairly regular ventricular response. This, too, showed unmistakable electrical alternation (fig. 9B).

**Summary**

Alternation of the heart occurs whenever any of its tissues, contractile or conductive, fail to function during alternate beats. This results in a diversity of 2:1 conduction blocks with electrical alternation and myocardial blocks that produce alternation of the pulse. In a general way, the more distal blocks, involving bundle branches, arborization, and myocardium are the more ominous, implying serious underlying heart disease. They can occur normally, however, at rapid heart rates.

Another type of alternation involving atrial as well as ventricular waves is sometimes seen in serious pericardial disease with effusion. It is believed to be due to an unusual rotary oscillation of the heart released from its normal inhibitory mediastinal restraints by the surrounding effusion. This is an anatomic rather than a cellular form of alternation and can exhibit other mechanical disorders such as alternating friction sounds.

**References**

3. **First, S. R., Bayley, R. H., and Bedford, D.**

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ALTERNATION OF THE HEART


Concerning the Effects of Electricity on Muscular Motion

I thought, therefore, that I should be doing something worth while, if I reported a brief and accurate account of my discoveries and findings in the order and relation in which partly chance and fortune presented and partly diligence and industry revealed them to me; not so much lest more be attributed to me than to fortune, or more to fortune than to me, but that either I might have on a torch to those who had wished to enter this same pathway of experiment, or might satisfy the honest desire of scholars who are wont to be interested in things which contain some novelty either in origin itself or in principle.—LUIGI GALVANI. Commentary on the Effect of Electricity on Muscular Motion. Translated by ROBERT MONTRAVILLE GREEN, M.D. Cambridge, Massachusetts, Elizabeth Licht, Publisher, 1953, p. 23.
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DAVID LITTMANN

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