Hereditary Occurrence of the Pre-Excitation (Wolff-Parkinson-White) Syndrome with Re-Entry Mechanism and Concealed Conduction

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The hereditary occurrence of the pre-excitation (Wolff-Parkinson-White) syndrome in 3 generations of 1 family is reported. The syndrome was observed in a grandfather, father, in a set of identical twin girls, and in the male of a second set of fraternal twins. Completed abortive circus movements with re-entry into the normal conduction pathway, as well as concealed forward conduction, in the pre-excitation syndrome are demonstrated. The importance of this observation for the understanding of the mechanism of the Wolff-Parkinson-White syndrome is discussed.

A national panel discussion on "Anomalous Atrioventricular Excitation" sponsored by the New York Academy of Sciences resulted in the advancement of 3 main concepts as an explanation for the pre-excitation syndrome.  

A mechanism was proposed by Sodi-Pallares, Calder, and associates that certain areas high in the interventricular septum are hypersensitive and, therefore, very easily stimulated and that the stimulus responsible for the excitation of these areas does not travel by any anatomicly recognizable pathway. These authors were able to produce experimentally by right heart catheterization ventricular complexes with "remarkable likeness" to those found in clinical examples of the pre-excitation syndrome.  

A second theory of Prinzmetal, Kennamer, and associates was that in the Wolff-Parkinson-White (W-P-W) syndrome the atrial impulse passes to the ventricles over the normal conduction system and not by way of anomalous connections, but that the normal delay in the atrioventricular (A-V) node is partially overcome, so that the impulse in part of the node passes through more rapidly than normal, a condition which they called "accelerated conduction." They also suggested that the A-V node and the intraventricular conduction system always "supply" the same portion of the ventricular myocardiunm and no others. In the experience of these authors, the W-P-W syndrome is more commonly acquired than congenital. They observed 20 patients in whom the W-P-W syndrome was thought to be acquired as a result of disease or as a functional disorder. The majority of these cases have been patients with myocardial infarction.  

The third concept implies the presence of 1 (or more) accessory conduction pathways bypassing the normal A-V conduction through the A-V node. This theory is supported by the anatomic demonstration of anomalous muscle bundles in human hearts carefully examined at autopsy. Recent histologic evidence suggests that the bundle of Kent, which lies subepicardially, and therefore outside the annulus fibrosus, is most likely not the accessory A-V bridge responsible for the pre-excitation syndrome. Other muscular A-V connections were described by different authors, which, on the basis of recent knowledge of the embryology of the heart, will replace the bundle of Kent in the hypothesis of an accessory muscular A-V pathway as the basis for the production of the pre-excitation (W-P-W) syndrome.

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The opinions or assertions contained herein are those of the author and are not to be construed as official or reflecting the views of the Navy Department or of the naval service at large.

Presented in part at the Third World Congress of Cardiology, September 1958, Brussels, Belgium.
Since the original description of the syndrome,\textsuperscript{14} voluminous monographs, excellent reviews, and many reports appeared advancing evidence in support of the different concepts. Hecht, moderator of the panel, stated in his summary and conclusion that "proof of the heredity of the syndrome would be in the observation of its occurrence in identical twins."\textsuperscript{1}

The purpose of this report is (a) to demonstrate the occurrence of the pre-excitation (W-P-W) syndrome in 3 generations of 1 family, (b) to describe the existence of the Wolff-Parkinson-White syndrome in a set of identical twins and in the male of a second set of fraternal twins in the same family, (c) to establish evidence for completed abortive circus movements in pre-excitation—forward conduction through normal A-V pathway—return through anomalous A-V conduction path—forward re-entry through normal path, and (d) to demonstrate concealed forward conduction in the pre-excitation syndrome.

**Report of Case**

A 16 month old white boy was admitted to the U.S. Naval Hospital, Portsmouth, Virginia, with acute meningitis.

On the next day, an electrocardiogram showed a supraventricular tachycardia at 300 per minute, and the patient was digitalized. He was quite resistant to digitalis therapy and received over the 32 hour period a total dose of 1.05 mg. of Cedilanid (levatoside C), which is more than double his theoretical digitalizing dose.

The paroxysmal supraventricular tachycardia converted only intermittently to sinus rhythm, at times following carotid sinus pressure. An electrocardiogram in such a period revealed evidence of a pre-excitation syndrome (W-P-W syndrome). After the W-P-W syndrome was recognized as the underlying cause of the paroxysmal supraventricular tachycardia, the patient received 100 mg.
of quinidine gluconate intramuscularly, and 2 hours later 200 mg. of quinidine sulfate orally. The supraventricular tachycardia was promptly controlled. The meningitis cleared with antibiotic therapy, and 5 days after admission he was afebrile, and the supraventricular tachycardia did not return. The digoxin was discontinued after 8 days and the quinidine after 3 weeks. The patient was discharged with the final diagnoses: meningitis, due to Bacillus subtilis pre-excitation syndrome (W-P-W syndrome); paroxysmal supraventricular tachycardia.

This patient was found to be of particular interest from several points of view, since he presented cardiac arrhythmias, which are readily interpreted by assuming an accessory A-V conduction bypass.

Figure 1 shows part of the electrocardiogram obtained 1 day after admission. Supraventricular paroxysmal tachycardia was diagnosed on the basis of the normal duration of QRS and the precise regularity of the rapid rhythm at the rate of 300 per minute. No P waves can be made out with certainty, although the upright peak before the QRS in V₂ probably represents a P wave. The possibility exists, but is remote, that this may be paroxysmal atrial flutter with 1:1 A-V conduction, because the rate is so rapid. Electrical alternans is especially well demonstrated during this rapid rhythm in lead V₅.

Figure 2, after digitalization with more than double the usual digitalizing dose, shows the pre-excitation syndrome diagnosed by the combination of a short P-R (0.06 second), a delta wave, and widened QRS to 0.10 second. In V₅ the W-P-W complexes change progressively in contour from left to right. The delta wave disappears and the small S wave becomes larger until the last complex in V₅ presents a diphasic R-S. In addition, the depressed S-T in the first few complexes becomes less depressed from left to right and the inverted T wave becomes less inverted and is finally upright in the last 3 complexes of V₅. The sinus rhythm shows a slight arrhythmia and varies between a rate of 120 and 130 per minute. In this tracing, we are dealing with intermittent A-V dissociation produced by nonparoxysmal A-V nodal tachycardia. The A-V node escapes readily as
soon as the sinus node slows down. This mechanism, which was repeated throughout the record, is of particular interest in this case of pre-excitation syndrome because of the coincidence of a sinus arrhythmia with A-V nodal tachycardia. The A-V nodal acceleration may find its cause in the infection or may be due to the high dose of digitalis.

Figure 3 represents part of long leads aV_{L} and aV_{P}. Evidence of the pre-excitation syndrome is
based in aVp, on the combination of a short P-R (0.08 second), a delta wave, and widened QRS of 0.08 second in beats 2 to 4. The sinus rate is 136 per minute. Compared with these pre-excitation beats, complexes of entirely different contour and direction are present (beats 6 to 14): no P wave precedes the QRS complexes in these beats; they are A-V nodal escape beats producing A-V dissociation. The P wave of the fifth beat is a trifle late, and close observation of the QRS reveals a different contour from the others as evidenced by the absent delta wave and the small downward deflection before the T wave. The slight slowing of the sinus impulse by 0.04 second was enough to permit A-V nodal escape, producing A-V dissociation and electric interference at the A-V junction. The same interpretation can be applied to the first beat of this strip. On examination of the A-V nodal beats, further interesting phenomena are observed. The T waves of the beats 6, 8, and 10 are not so deeply inverted as those of the 7, 9, and 11. In addition, the inverted T waves are followed by pauses producing pseudobigeminy. On detailed analysis of the S-T segment and T-wave contour of the seventh beat in comparison with the ninth beat, it will be seen that an upright P wave is superimposed on the ST of the seventh beat and an inverted P wave is superimposed on the T wave of the ninth beat. Furthermore, on exact measurement it will be seen that the pause following the ninth beat is 0.04 second longer than the pause following the seventh. The interpretation of these pauses is that after the seventh beat the impulse formation of the A-V node is retarded by the sinus impulse traversing deeper into the A-V junction producing forward concealed conduction. The pause following the ninth beat is produced by a different conduction pathway. The conduction of this A-V nodal beat spreads through the ventricle and in a retrograde fashion through an accessory A-V bridge into the atria, re-entering the normal A-V junction. The re-entry of the retrograde impulse is "concealed" in that it is not followed by another ventricular beat, but its effect on the A-V node is manifested by delay of the A-V nodal impulse formation. In support of this interpretation are (a) the inverted P wave superimposed on the T wave, indicating retrograde excitation of the atria, and (b) prolongation of the pause following an inverted P wave by 0.04 second as compared with the pause following an upright P wave. The difference of 0.04 second represents the re-entry time. The same abortive circus movement of conduction is seen in beats 11, 12, and 13. However, the second last cycle of the tracing is not prolonged, despite a retrograde P wave, indicating intermittent failure of re-entry to occur. In aVl, concealed forward conduction is clearly evident in beat 11, where an upright P wave is seen superimposed on the T wave of that beat and is followed by a pause indicative of retarded impulse formation of the A-V node. In summary, in this tracing the pre-excitation syndrome

Fig. 5. Intermittent A-V dissociation due to A-V nodal tachycardia and pre-excitation (W-P-W) syndrome. Two completed ventricular captures with aberrant ventricular conduction are seen in V2 (beat 10) and V3 (beat 8). From the ventricular capture the impulse is conducted in a retrograde fashion to the atrium and leads to concealed re-entry into the normal A-V pathway.
Figure 6. Pedigree demonstrating the pre-excitation (W-P-W) syndrome in 3 generations of the Hall family. The twin girls, 10 years of age, are identical twins.

As the basic mechanism plus A-V nodal tachycardia which produces A-V dissociation and electric interference at the A-V junction. In addition, there is concealed forward conduction and concealed re-entry of retrograde impulses through an accessory A-V bypass producing pseudobigeminy.

Figure 4 is assembled from selected parts of long limb leads of the same patient. This tracing is especially interesting for 3 reasons: fusion beats of unusual type in pre-excitation, concealed conduction in forward direction, and concealed retrograde conduction over an anomalous accessory A-V bypass following a ventricular capture. Again the main diagnosis is pre-excitation and intermittent A-V dissociation due to nonparoxysmal nodal tachycardia. In lead I, 2 basically different QRS complexes are seen. Beats 5 and 6 represent pre-excitation complexes. The first 3 beats as well as the last 2 beats represent A-V nodal beats with electric interference at the A-V junction as evidenced by the P wave superimposed on the T wave. Beats 4, 7, 8, and 9 vary in contour and to different degrees are intermediate between the 2 basic QRS complexes; these are fusion beats. The mechanism of these fusion beats can be understood by the assumption of an accessory A-V bypass. In pre-excitation, a single impulse originates in the sinoatrial node and splits in the atrium on its way to the ventricle to use the normal conduction path as well as an accessory A-V bridge, thus producing interference in the ventricles. In this tracing, a second ectopic focus in the A-V node has to be postulated, which promptly escapes as soon as the sinoatrial node slows. Here the fusion beats are the result of admixture of these 2 foci, the sinoatrial impulse plus the ectopic one.

In lead III, intermittent A-V dissociation as basic mechanism can again be recognized. An A-V nodal arrhythmia varying between the rate of 125 and 136 is present. On 2 occasions, after beats 1 and 4, a pause is seen, the cause of which can be interpreted as concealed re-entry of the retrograde impulse to the A-V node retarding its impulse formation as described in figure 3.

The bizarre premature beat in lead II (beat 7) represents a completed ventricular capture with aberrant spread in the ventricle and retrograde conduction through an accessory A-V bypass producing concealed re-entry of the retrograde impulse. Proof for this interpretation will be supported with figure 5, where in leads V2 and V3 the same phenomenon occurred.

In figure 5 we again see in the first 4 beats of V2 and V3 evidence of the pre-excitation syndrome diagnosed on the criteria outlined previously. In V2 the sinoatrial rhythm slows from the fifth to the seventh beat, whereupon the A-V node escapes, producing intermittent A-V dissociation with electric interference at the A-V junction. The explanation of the 2 premature complexes with detailed analysis of their mechanism is important for the understanding of the pre-excitation syndrome. Assuming the presence of an accessory A-V bypass, these 2 beats can be interpreted as follows:
In $V_2$ as in $V_3$ the basic mechanism is intermittent A-V dissociation. On examination of the A-V nodal beats in $V_2$ it can be seen that the P-R distance becomes progressively shorter from left to right until in beat 9, preceding the bizarre complex, the sinus P wave appears after the QRS. Evidence for this is seen by the different contour of the S-T segment as compared with the 2 previous A-V nodal complexes. The premature bizarre beat 10 is a completed ventricular capture with marked aberrant ventricular conduction. The same interpretation can be applied for the bizarre complex 8 in $V_3$. With this established, the second challenge in this record is the explanation for the delayed impulse formation of the A-V node. Assuming an accessory A-V bypass, it can be postulated that the conduction of the ventricular capture is spreading backwards through the accessory A-V bridge, activating the atrium in a retrograde fashion, reentering the normal A-V path, and retarding the impulse formation of the A-V nodal focus by concealed re-entry of the retrograde impulse. In support of this assumption is the fact that the duration of the R-R interval following the ventricular capture of 0.66 second is exactly the same R-R interval as in lead $aVF$ (fig. 3) where concealed re-entry of a retrograde impulse was established. Further, the negative deflection following the aberrantly conducted ventricular capture is a superimposed inverted P wave. Finally, the R-R interval is 0.04 second longer, representing the re-entry time, as compared with the R-R interval where forward concealed conduction was present. This tracing is unique and represents convincing evidence of the property of retrograde conductivity postulated for an accessory A-V bridge as an explanation for the mechanism of supraventricular paroxysmal tachycardia in the pre-excitation syndrome.

This represents the only demonstration in the literature for a completed abortive circus movement with re-entry into the normal A-V conduction pathway in the pre-excitation syndrome.

The possibility that the pre-excitation syndrome was a hereditary anomaly was suspected more and more in recent years since observations increased that the syndrome was found in 2 brothers and parent and child. Wolff observed 5 cases in a single family.

Curiosity led me to the examination of the entire family after the diagnosis of the W-P-W syndrome was established in the baby twin. The pre-excitation syndrome was observed in 2 identical female twins, sisters of the patient, and it was traced back into the third generation of the same family. Figure 6 demonstrates the pre-excitation syndrome in a grandfather, father, identical set of female twins, and in the male of a second set of fraternal twins. Documentation of the individual electrocardiograms is furnished in figures 9 to 13.

The 35 year old father of the twins was totally unaware of the presence of the W-P-W syndrome. During his career in the U.S. Navy he passed several physical examinations, but an electrocardiogram was never taken. There was no history of paroxysmal supraventricular tachycardia. The history was similarly negative in the 10 year old identical twin girls. However, 1 of the twin girls...
presented a systolic murmur of grade II, which was best audible along the left sternal border in the second and third intercostal space, with a slightly accented pulmonic sound. The chest x-ray was negative. Her electrocardiogram differed from the one of her twin sister, as can be seen in the illustrative appendix, but both are type A according to the classification of Rosenbaum and co-workers. The murmur was classified as insignificant for the dynamics of the heart.

The grandfather of the twins, 57 years of age, suffered shortness of breath and occasional palpitation of short duration for 5 to 7 years. He was not known to have pre-excitation syndrome. Pulmonary function studies revealed marked pulmonary emphysema.

Since the monozygotic identity of the 10 year old female twins is of prime importance for the hereditary occurrence of the pre-excitation, further support for this fact was established by examination of their blood groups. Twins with dissimilar blood groups or of different sexes are obviously dizygotic. Of all twin pairs, 65 per cent are found to be like-sexed and 35 per cent unlike-sexed. If 35 per cent are unlike-sexed and dizygotic, the same percentage will be expected to be like-sexed and dizygotic. The remaining 30 per cent will be like-sexed and monozygotic pairs. Figure 7 demonstrates the blood groups of the like-sexed twins and the ones of their parents. As can be seen, the blood groups of the twins are the same and, furthermore, are like the blood groups of their father, who also presented the W-P-W syndrome. The blood groups differ from the ones of their mother in the MN, S, and Fy (a-) groups. It can therefore be concluded that the like-sexed twins are, with a high degree of certainty, really identical monozygotic twins.

**DISCUSSION**

A discussion of the observations described above in relation to the concept of the mecha-
nism of the pre-excitation (W-P-W) syndrome is of interest in relation to the advances made in embryology. In support of an accessory A-V pathway is not only the anatomic demonstration of muscular A-V connections, but experiments in embryology which established further evidence that these muscular A-V connections are actually able to conduct the sinoatrial impulse to the ventricle. Elegant and very instructive experiments performed by Patten,\textsuperscript{26} as shown in figure 8, present convincing evidence of the conductive capacity of embryonic cardiac muscle. The sinoatrial impulse to the ventricle is still conducted in spite of cutting away all the tissue around the atrioventricular constriction except for a narrow connecting strand, which then serves functionally as a sort of artificial bundle of His. Patten found that it does not make any difference whether this connecting strand is left at or near the place where the bundle of His will later develop or whether, as in figure 8, the strand is left on the opposite wall of the heart at the farthest possible distance from the normal site of the bundle of His. The utilization of a strand of muscle from a part of the heart that never becomes involved in the formation of the bundle of His avoids any questions that might arise as to the possible functional differentiation of a conduction bundle, before it is histologically differentiated. Thus, it seems only natural that certain re-
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tained tracts of it come to serve as the path of impulse conduction and, on rare occasions, of impulse formation in the adult heart.

There seems to be general agreement on several points. 1. The W-P-W complex is the result of a ventricular fusion beat, the QRS complex is the resultant of 2 different conducted stimuli in the ventricle.\(^1\), \(^2\), \(^3\), \(^6\), \(^8\), \(^9\) 2. A limited area of 1 ventricle contracts prematurely; the remaining ventricular myocardi-um is still contracted.\(^1\), \(^2\), \(^3\), \(^8\) 3. In tracings designated as type A (positive delta wave in all precordial leads), the premature contraction occurs in the left ventricle and in type B (negative delta wave in right precordial leads) the pre-excitation occurs in the right ventricle.\(^1\), \(^2\), \(^3\)

There are considerable divergent explanations, however, for the production of the "fusion beat" in anomalous atrioventricular excitation.\(^1\), \(^2\), \(^3\), \(^8\), \(^9\), \(^10\), \(^11\) In assuming an accessory muscular A-V bridge, the physiologic properties of conductivity and rhythmicity of such muscle fibers are essential prerequisites. That the property of rhythmicity can "exist, although rarely, in the accessory A-V connection was convincingly proved by Pick and Katz.\(^23\)

The property of conductivity of such fibers both in a forward and in a retrograde direction is, however, the primary factor responsible for the usual manifestations of the syndrome. The mechanism for the forward conduction in the W-P-W syndrome was already described. Thus, as the final link in the chain of evidence for an accessory A-V conduction path it remained necessary to demonstrate conduction in a retrograde direction from the ventricle back to the atrium via the anomalous path. Proof of such evidence is given in figures 3, 4, and 5 of this report. The demonstration of a completed circuit movement of conduction in a human heart is of particular importance for the understanding of the mechanism of supraventricular tachycardia in the presence of pre-excitation.

As demonstrated in figures 4 and 5, impulses conducted in a normal fashion through the A-V junction to the ventricles may return back toward the atria over the accessory A-V path and continuation of such a re-entry mechanism may initiate and perpetuate rapid heart action. Further evidence for the correctness of this view is the demonstration that in cases in which the onset of supraventricu-lar tachycardia was recorded, the last beat preceding the tachycardia was not of the pre-excitation type.\(^20\), \(^23\)

The recent report of Pick and Fisch\(^31\) on 3 cases of the W-P-W syndrome in the presence of bundle-branch block is another important contribution to the existence of an accessory muscular A-V bypass. They reported left bundle-branch block and the pre-excitation syndrome in 2 cases, and right bundle-branch block and the W-P-W syndrome in 1. Thus, it is possible to explain in a rational way and on the basis of physiologically acceptable principles, the production of the ventricular "fusion beat" as well as all varieties of arrhythmias in the pre-excitation syndrome, by assuming the existence of
an accessory muscular A-V bridge. In addition, with the first demonstration of the hereditary nature of the W-P-W syndrome over 3 generations and in a set of identical twins, together with the cardiac arrhythmias reported, strong evidence is established for the correctness of the hypothesis of an accessory A-V connection as the most appropriate one to account for all of the known aspects of the pre-excitation syndrome.

**SUMMARY**

The possibility that the pre-excitation syndrome can be a hereditary anomaly, as suspected in recent years, is further supported by this observation of its occurrence in 3 generations of 1 family. Convincing evidence for this is the existence of the Wolff-Parkinson-White syndrome in a set of identical twins and a set of fraternal twins in the same family. The monozygotic identity of the twins was proved by their blood groups.

The concept of the mechanism of supraventricular tachycardia in pre-excitation syndrome by impulses returning back to the atria over an accessory atrioventricular path, and continuation of such a re-entry mechanism perpetuating the rapid heart action, was strongly supported by the demonstration of completed circus movements of conduction in a human heart. The operation of a completed retrograde re-entry mechanism—forward conduction through a normal atrioventricular pathway, return through an anomalous atrioventricular conduction path, retrograde excitation of the atria, forward re-entry into the normal atrioventricular path—was demonstrated for the first time.

Atrioventricular dissociation in the Wolff-Parkinson-White syndrome with effect of concealed forward and concealed retrograde conduction upon impulse formation of the atrioventricular node is demonstrated, and the importance of this observation for the understanding of the mechanism of the pre-excitation (Wolff-Parkinson-White) syndrome is discussed.

**ACKNOWLEDGMENT**

The author gratefully acknowledges Dr. A. Pick for his comments and constructive criticism during the preparation of this paper. He is indebted to Mrs. Phyllis Bailey, Dennis O. Brown, HM2, the members of the Cardiopulmonary Function Laboratory, and in particular to C. C. Ward, HMC, and H. Petras, HM2, from the Photographic Laboratory of the Naval Hospital, Portsmouth, Va., for their valuable technical assistance.

**Summario in Interlingua**

Le possibilitate que le syndrome de pre-excitation pote occurrer como anomalia hereditari—como on lo ha suspicite in recente annos—es supportate additionalmente per le hie-reportate observation de su occurrence in 3 generationes del mesme familia. Un forte corroboratio es le presentia del syndrome de Wolff-Parkinson-White in un par de geminos identic e un par de geminos fraterne in le mesme familia. Le identitate monozygotic del geminos esseva demonstrate por lor gruppos de sanguine.

Le conception que le mechanismo del tachycardia supraventricular in le syndrome de pre-excitation depende del returno de impulsos al atrios per un via atrio-ventricular accessori e que le continuation de iste mechanismo de re-entra percutua le rapide action del corde, iste conception esseva fortemente supportate per le demonstration de complete circos de conduction in un corde humana. Esseva effectuate le prime demonstration del curso del mechanismo de un complete re-entra retrograde, i.e. le conduction in avante per un normal via atrio-ventricular, returno per un via anormal de conduction atrio-ventricular, excitation retrograde del atrios, e re-entra in avante in le normal via atrio-ventricular.

Dissociation atrio-ventricular in syndrome de Wolff-Parkinson-White, con le effecto del celate conduction in avante e del celate conduction retrograde super le formation del impulso del nodo atrio-ventricular, es demonstrate. Le importantia de iste observation pro le comprension del mechanismo del syndrome de pre-excitation (syndrome de Wolff-Parkinson-White) es discutite.

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Diagnosis.—One must be a professional Ulysses in craft and wisdom not sometimes to err in estimating the nature of an attack of severe heart pain. There is no group of cases so calculated to keep one in a condition of wholesome humility. When you jostle against a hale, vigorous specimen of humanity, who claps you on the back and says, "The deuce take you doctors! I have scarcely yet got over my fright," you would like to forget that five years before you had almost signed his death warrant in a very positive diagnosis of angina pectoris vera. On the other hand, Mr. X. has left you with the full assurance that his cardiac pains are due to overwork or tobacco, and you have comforted his wife and lifted a weight of sorrow from both by your most favorable prognosis. With what sort of appetite can you eat your breakfast when, a week later, you read in the morning paper the announcement of his sudden death in the railway station? Or take another aspect—poor Mrs. Doe has gone softly all these years in the bitterness of her soul since you took that grave view of her vaso-motor or hysterical angina!—William Osler, M.D. Lectures on Angina Pectoris and Allied States, 1897.
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Circulation. 1959;19:28-40
doi: 10.1161/01.CIR.19.1.28

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/19/1/28

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