The cause of the murmur is not clear. Fishleder et al. suggested that it is produced by pulmonary insufficiency with backflow of blood to the right ventricle, resulting in a reduction of pressure at the pulmonary ostium. The latter may cause blood to flow from the aorta to the right ventricle, thereby producing a diastolic murmur. According to this concept, the murmur is made up of two components, the first caused by flow from the aorta to the pulmonary artery, and the second being the murmur of pulmonary incompetence.

ACKNOWLEDGMENT: The authors are indebted to Prof. J. Gafni, Department of Medicine, Dr. V. Deutsch, Roentgenology Department, and Dr. J. Bubis, Pathology Department, Tel-Hashomer Hospital, for their assistance.

REFERENCES


Reprint requests: Dr. Rosenthal, Heller Institute, Tel-Hashomer Government Hospital, Tel-Aviv, Israel

Reversed Reciprocating Paroxysmal Tachycardia*

M. C. Mehta, M.D. and S. V. Singh, M.D.

A case report of reversed reciprocating tachycardia sensitive to vagal stimulation which produced interesting patterns of coupling is described. The mechanism of reversed reciprocal rhythm is discussed. This arrhythmia and Wolff-Parkinson-White syndrome can be explained on the basis of anomalous pathway. In the former partial anterograde block through the A-V node is also present.

Katz and Pick have described a cardiac arrhythmia, a form of reciprocal rhythm, with the pacemaker of sinus or auricular origin. Under such a situation the auricular impulse travels to the A-V node and from there to both ventricles and atria producing a P-QRS'-p pattern. P' is the result of

*From the Department of Medicine and Cardiology, R.N.T. Medical College, Udaipur-Rajasthan, India.

DIS. CHEST, VOL. 56, NO. 4, OCTOBER 1969
retrograde conduction from A-V node to the auricle (different shape and size in comparison to first P wave). To this phenomenon they have given a name of reversed reciprocal rhythm.

In 1929, Wolferth and Wood\(^2\) were the first to describe this arrhythmia. Since then very few reports have appeared in the literature (Scherf and Cohen\(^3\)). This condition is extremely rare because of its basic prerequisites of an anomalous pathway, unidirectional block in the anomalous pathway due to its long refractory phase and a partial block in the A-V node. Normally the A-V junctional tissues have a long refractory phase and any impulse that returns from the ventricles finds it unresponsive. However, according to Scherf,\(^4\) retrograde conduction of an impulse from A-V node or ventricle is possible if a functional dissociation of the A-V node fibers is present. Some of its fibers remain undischarged by the initial forward activation wave. Retrograde conduction through the unused fibers then becomes possible. Codina-Atles and Pijoan de Beristian\(^5\) and Eldridge\(^6\) suggested an anomalous atrioventricular conduction pathway which permitted rapid retrograde conduction. They have explained the reversed reciprocal rhythm on the basis of presence of such a pathway. In contrast to the physiological properties of normal pathway which has slower rate of transmission and short refractory period, this anomalous pathway, although conducting impulses with extreme rapidity has a very long refractory period. The existence of these two paths of conduction explains production of this arrhythmia as follows:

An impulse arriving at the A-V node finding normal pathway nonrefractory travels down this path, but returns via anomalous pathway. This is probably due to a unidirectional block in the anomalous pathway as a result of its long refractory phase. This retrograde impulse will find the atria refractory because of their stimulation by anterograde sinus impulse. Therefore, it would be necessary for the retrograde and/or anterograde A-V block of such a duration so as to allow, recovery of the atria before arrival of the retrograde stimulus.

Reversed reciprocal rhythm is extremely rare. Few examples could be traced in the literature. In this report we describe one more case of this arrhythmia.

**CASE REPORT**

A 32-year-old Hindu man complained of attacks of palpitation. These attacks came on suddenly and had equally abrupt termination and lasted from a few seconds to a few hours. To start, these attacks were infrequent probably occurring at intervals of once a month for the first three years and ten months. However, for the last six months these attacks had become very frequent, occurring several times a day thereby making him conscious of the rapid beating of his heart. No symptoms or signs of hyperthyroidism were present, such as an increased appetite, loss of weight or intolerance to heat, goiter or tremors.

Examination revealed a well-nourished average built person of 130 pounds without any anxious look. There was no cyanosis or clubbing. His blood pressure was 120/80 mm. The pulse was 146 per minute. It did not change with respiration or exercise.

On occasions while the patient was being examined paroxysmal bouts of fast pulse and rapid cardiac action lasting for a few seconds were detected, and examination of the heart revealed no other abnormality. The balance of the physical examination was normal.

Investigation revealed no abnormality. Hemoglobin 13.8 gm per 100 ml. Total red blood cells 4.4 million per mm\(^3\). Total white cells 5,800 per mm\(^3\). Erythrocyte sedimentation rate 10 mm per hour (Westergren); urine and stools—NAD: serum cholesterol 200 mg per 100 ml. Heart was of normal size and shape on screening.

**THE ELECTROCARDIOGRAM**

Figure 1 illustrates an attack of tachycardia with auricular as well as ventricular rate of 150 per minute. During each cardiac cycle a tall spiked P wave and a QRS is present. This supraventricular tachycardia may be diagnosed as an auricular tachycardia with A-V block having a P-R interval of 0.32 seconds. It may also be a nodal tachycardia (retrograde conduction of nodal impulse) with R-P interval of 0.12 seconds. Carotid sinus pressure (marked by arrow) produced a sinus standstill of 1.2 seconds duration. On further analysis although the P wave seen at the restart of the tachycardia is of different type.

---

**FIGURE 1.** This figure shows a run of paroxysmal tachycardia as recorded in lead V\(_1\). Carotid sinus pressure terminated the attack (see text).

**FIGURE 2.** Electrocardiogram (lead V\(_1\)) showing termination of the paroxysmal tachycardia by vagal stimulation.
shape and size (P-R interval 0.22 seconds), yet the QRS complex is of the same configuration as compared to others. The balance of the P waves and the P-R intervals which follow are of the same contour and duration. This P wave (marked in Figure) thus represents sinus impulse which is conducted to ventricles with a partial A-V block. Return of this impulse to the auricle produces a P-QRS-P' pattern. This phenomenon is just the reverse of the common form of reciprocal rhythm and is therefore called reversed reciprocal rhythm. The cardiac rate was 150 per minute and the attack was of sudden onset and abrupt termination, and hence can be called reversed reciprocal paroxysmal tachycardia.

Figure 2 is one of those tracings obtained as a result of vagal stimulation irritation of fauces and eyeball pressure and Valsalva maneuver. It shows that the paroxysm has stopped; yet the beats showed the retrograde passage of sinus impulse to the atria. However, the anterograde conduction is now blocked.

This graph exhibits a sinus P wave to start with and a P-R interval of 0.24 seconds. Then a QRS complex and a retrograde P wave follows as the impulse returns from A-V node into the atria with an R-P interval of 0.14 seconds. This impulse travels anterograde to the ventricles describing an aberrant QRS complex. The P-R interval here is 3.2 seconds. Just after this QRS a retrograde P wave is noted. This returns anterograde (P-R interval of 0.28 second) to record another aberrant QRS complex. Similarly a further retrograde P wave is recorded after the third QRS complex following which the impulse returns to record fourth QRS complex which is of normal shape. This repetitive pattern can justifiably be called reversed reciprocating paroxysmal tachycardia. This patient was treated with digitalis, quinidine and finally, when these two drugs had little effect, with prostigmin. This latter drug was administered in doses of 15 mg four times a day, then 30 mg four times a day, then 45 mg three times daily and finally 60 mg three times daily.

In Figure 4(a) the electrocardiogram shows a continuous strip of lead V1 during digitalis administration. The tracing shows nodal rhythm with retrograde conduction due to atrial fusion beats and A-V dissociation. After prostigmin therapy [Fig 4(b)] the arrhythmia now consists of various reciprocal and nonreciprocal beats mostly grouped in pairs. The coupling is in the form of a Wenckebach phenomenon. The P-R interval varies from 0.18 seconds to 0.30 seconds. Those complexes which have P-R interval shorter do not show reciprocal return to atria while those which have partial A-V block exhibit a retrograde return. This impulse discharges the next sinus impulse ending the conducted stimulus.

DISCUSSION

Reciprocating rhythm was first of all produced experimentally by Mines. He ascribed this arrhythmia of alternating atrial and ventricular contractions to refractoriness of part of the A-V conducting system. The atrial stimulus first traveled through the nonrefractory pathway of the A-V node and stimulated the ventricles. By this time the refractory portion of A-V node had enough time to recover and transmitted the impulse in reverse direction, i.e., back to the atrium.

White (1915), Gallavardin and Gravier, Drury and Dock termed the nonrepetitive form of this arrhythmia reciprocal rhythm. This form, therefore, differed from the continuous reciprocating rhythm of Mines. In the reports of these authors the basic rhythm was a nodal rhythm. Occasionally a ventricular bigeminy was seen when a nodal P wave was "sandwiched" between two QRS complexes. White (1915) had hypothesized that such a pattern is produced when atria and ventricles were stimulated alternately with A-V nodal rhythm having slow retrograde conduction to the atria as compared to

Figure 4(a). Electrocardiogram (Lead II) shows an effect of digitalis intoxication. It illustrates A-V nodal rhythm with retrograde conduction and A-V dissociation to sinus rhythm (see text).
REVERSED RECIPROCATING PAROXYSMAL TACHYCARDIA

Figure 4(b). Electrocardiogram after prostigmin therapy (chest leads). It shows reciprocal and nonreciprocal beats in groups (see text).

anterograde conduction to ventricles. The stimulus which has reached the atria now finds the conduction tissue recovered and returns to the ventricles producing a ventricular contraction. Further, only 59 cases with reciprocal rhythm could be traced in the literature; (Decherd and Ruskin,\textsuperscript{11} Bix,\textsuperscript{12} Kistin and Bruce,\textsuperscript{13} Langendorf and co-workers.\textsuperscript{14}

Wolferth and Wood's\textsuperscript{2} patient had a basic rhythm of 2:1 heart-block. They were of the opinion that the coming of the retrograde P wave at about the same time the sinus P wave was anticipated, points to the fact that the retrograde P wave either blocks or discharges the sinus P wave. They emphasized their viewpoint on the basis of late occurrence of the retrograde P wave and believed that such a delay was necessary to allow recovery of the conduction tissues.

Naim\textsuperscript{15} in 1945 and Bix\textsuperscript{12} in 1951, independently described a case of paroxysmal tachycardia with reciprocating rhythm. When the rate was slow a sinus P wave preceded a QRS complex which was then followed by a negative P wave. This retrograde P wave was mostly blocked but whenever it returned it caused a ventricular contraction. This led to a paroxysmal tachycardia. While Codina Altes and Pijoan de Beristain\textsuperscript{9} on the basis of a clinical case of paroxysmal reversed reciprocating rhythm postulated that electrocardiographic phenomenon was due to dual pathway, Moe and his associates (1956) did so on experimental basis.

In 1949, Decherd and Ruskin\textsuperscript{11} and in 1957, Zakopoulos\textsuperscript{16} published a case of reversed reciprocal rhythm with double atrial stimulation.

Another form of reciprocal rhythm has already been referred to, that described by Katz and Pick.\textsuperscript{3}

In the present case the inverted P waves are of nodal origin as is obvious from the fact that vagal stimulation (Fig 2) produced nodal beats with retrograde conduction to the atria. This is evident from the shape of P wave and R-P intervals. They are the same as that of the reciprocal beats. That these are not due to auricular extrasystoles is further shown by the abnormal P waves coming only with those conducted beats which have a prolonged R-P interval—a phenomenon difficult to explain on the basis of their being auricular extrasystoles.

Physiologically the longer the cycle length, the longer the refractory period. The refractory period of bundle branches and A-V node will also depend on the duration of the preceding cycle length. In Figure 3, the second and third complexes are aberrant ventricular complexes which have resulted because of slight difference in refractory period of the bundle branches. The long R-R interval preceding first QRS complex allows recovery of one bundle branch at the time of anterograde return of the A-V node. The shorter R-R interval between first and second complexes permits a more complete recovery of both bundle branches at the time of the returning atrial impulse resulting more or less in a normal (third) QRS complex. The next QRS complex is completely normal because the R-R interval be-
between second and third complex is still shorter.

On the basis of assumption of anomalous pathway the sinus impulse is conducted slowly through A-V node to the ventricles. This impulse returns in a retrograde direction via the anomalous pathway to the auricle. Due to longer time taken for the impulse to travel through the A-V node the auricle now responds and a retrograde depolarization wave is inscribed. This impulse once again returns to the A-V node only to find it refractory and no more impulse conduction takes place. If the degree of block could be prolonged and the refractory period of the normal path could be shortened it would then be responsive to the returning stimulus establishing a reversed reciprocal rhythm.

In the mechanism of production of Wolff-Parkinson-White syndrome also, the theory of anomalous pathway was put forward by Holzmann and Scherf as well as Wolferth and Wood. The arrhythmia reported in this publication can also be explained on the basis of anomalous pathway. Recently Wolff has shown both patterns in the same tracing emphasizing such a mechanism of their production.

We agree with Schamroth that the difference between reversed reciprocal rhythm and Wolff-Parkinson-White pattern may be only of the presence of first degree A-V block in the former. Both the electrocardiographic phenomena can be explained on the basis of an anomalous pathway or extra bundle of Kent.

REFERENCES


Evaluation of Oxygen Therapy Devices by Arterial Oxygen Tensions

Morton Shulman, M.D., Gwen Schmidt, M.D., and Max S. Sadove, M.D., F.C.C.P.

Ten different oxygen administration devices were tested on volunteers in order to determine their relative efficiency in raising the arterial oxygen tension. The efficiency of any particular device was directly proportional to the oxygen flow into it. Those devices which utilized reservoir bags were the most effective. The nasal catheter and cannula techniques were also quite effective with the cannula appearing to be somewhat preferable because of its greater comfort, especially at high flow rates of oxygen. The importance of proper baffling in those mask type devices that did not utilize reservoir bags was demonstrated.

The relative effectiveness of oxygen administration devices has been studied previously. The most comprehensive study was that of Kory and others who compared eight different techniques.

*From the Department of Anesthesiology, University of Illinois Research and Educational Hospitals, Chicago, Illinois.

DIS. CHEST, VOL. 56, NO. 4, OCTOBER 1969