

Atrial Infarction of the Heart

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CLINICAL REPORTS of atrial infarction diagnosed during life are few. This may be due to two major factors. First, the clinical features of isolated atrial infarction are not known, for it is usually associated with ventricular infarction, which dominates the clinical picture. Secondly, the diagnosis of atrial infarction can be made only from the electrocardiogram by small, transient elevations¹ and reciprocal depressions of the P-Ta segment usually associated with changes in configuration of the P wave.

Langendorf,² in 1937, reported one case of atrial infarction found at autopsy that in retrospect could have been recognized ante mortem from electrocardiographic changes. Subsequently, the electrocardiographic manifestations of atrial infarction in the standard leads were described³⁻⁶ in cases in which necropsy evidence had prompted a retrospective reinterpretation of the electrocardiogram.

Hellerstein,⁷ in 1948, reported the first case with the correct ante mortem diagnosis of atrial infarction confirmed by necropsy, and other cases were subsequently reported.⁸⁻¹⁰ The number of cases described, however, is far below the anticipated percentage of 3 to 17 reported by others.^{3, 11, 12}

The purpose of this paper is to present the changes in the 12-lead electrocardiogram from six cases of atrial infarction associated with ventricular infarction, which were diagnosed clinically and confirmed by autopsy.

Case Reports

Case 1

A 54-year-old diabetic, hypertensive woman entered the hospital because of mental confusion and convulsive seizures. Four days later rapid atrial fibrillation developed that stopped shortly

after intravenous lanatoside C. The P waves were of the negative-positive type in lead I, predominately negative in V₁, and largely positive in V₃ (fig. 1a). Changing contour of the P wave in lead I suggested a wandering pacemaker. The P-Ta segment was depressed in leads III, aV_F, V₁, and V₂, and was elevated in leads I, aV_L, V₅, and V₆. Complete left bundle-branch block was present. A diagnosis of atrial infarction was made. The characteristic P-Ta changes in aV_L and V₅ almost completely disappeared during the next 3 days (fig. 1b). The patient died 3 weeks later in coma. The serum glutamic oxaloacetic transaminase levels were 62 and 36 units.

Necropsy revealed a 1-cm.² hemorrhagic area on the endocardial surface of the anterolateral wall of the right atrium, at the atrioventricular junction. Small hemorrhagic areas, 2 mm. in diameter, were seen on the endocardial surface of the left atrium. The left circumflex coronary artery near its origin was completely occluded.

In the atrial myocardium (fig. 2) were several areas of hemorrhagic necrosis surrounded by a mild neutrophilic and lymphocytic infiltration. There was moderate patchy fibrosis. The left ventricular myocardium showed necrosis.

Case 2

A 54-year-old white man entered the hospital complaining of chest pain with shortness of breath for 2 weeks. The electrocardiogram revealed incomplete left bundle-branch block (fig. 3), occasional premature atrial contractions, and a brief run of atrial tachycardia at 160 per minute. The P-Ta segment was depressed in leads II, III, aV_F, all the precordial leads, and elevated in aV_L. The P waves were of the positive-negative variety in V₁. The serum glutamic oxaloacetic transaminase levels were 58 and 56 units. Since there was no evidence of acute ventricular myocardial infarction, a diagnosis of isolated atrial infarction was made. The patient was digitalized for mild congestive heart failure. He died suddenly 2 days later.

Autopsy revealed severe generalized coronary arteriosclerosis with a recent thrombus occluding the left anterior descending coronary artery and an old occluding thrombus of the right coronary artery. An extensive infarction was seen in the ventricular septum from the anterior to the posterior wall of the left ventricle. The hemorrhagic area of the left ventricle extended into the left

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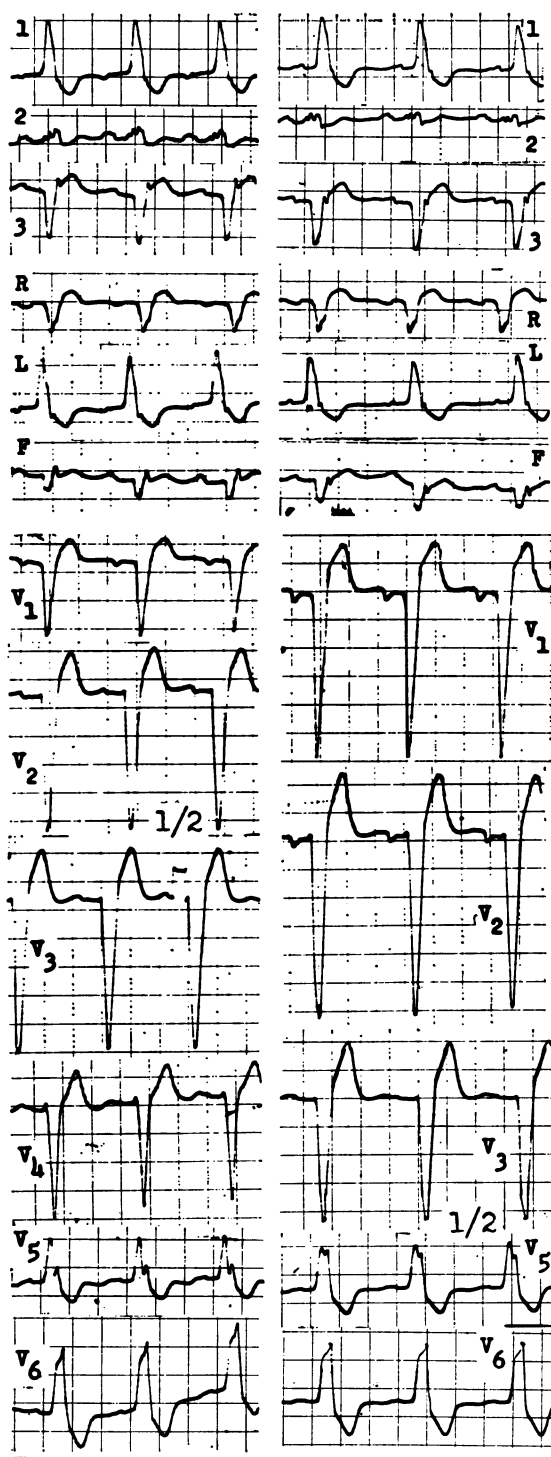


Figure 1

Left, case 1. Electrocardiogram 30 minutes after lanatoside C shows abnormal P and P-Ta waves and complete left bundle-branch block. Right, Case 1. Notched P waves are seen in lead I. P-Ta segments in aV_L and V_5 have almost returned to normal.

posterior atrial wall and appendage. Grossly the right atrium appeared normal.

Microscopically both atria showed perivascular lymphocytic infiltration, hemorrhage, necrosis, and hyalinization of the muscle fibers, typical of infarction (fig. 4).

Case 3

A 62-year-old white woman with severe ulcerative colitis, developed tachycardia with occasional premature beats, dyspnea, chest pain, and congestive heart failure. The next day the patient had more cardiac pain, transient multifocal atrial tachycardia of 200 per minute and hypotension. An electrocardiogram (fig. 5) at that time revealed a septal and inferior ventricular myocardial infarction. The P waves in lead II were wide and slurred and 1.25 mm. high in lead II. In V_1 and V_2 the P waves were diphasic and were followed by a depression of the P-Ta segment of 1.5 to 1.8 mm. The P waves were notched in V_3 and V_4 and low and notched in V_5 where the P-Ta segment was elevated 1.0 to 1.5 mm. The P wave in V_6 was wider and the P-Ta segment was slightly elevated. The deviations of the P-Ta segments in V_5 and V_1 and V_2 probably represented reciprocal changes of an atrial injury current. A definite diagnosis of left atrial infarction was made. The patient died 4 hours later in shock.

Neeropsy showed a 1 by 2 cm. area of hemorrhage in the left atrium and infarction of the interventricular septum. The coronary arteries showed only slight arteriosclerosis, with no occlusion. Microscopically congestion of the vessels and hemorrhages into interstitial tissue were compatible with an early stage of atrial infarction.

Case 4

A 52-year-old white man had repeated episodes of congestive heart failure for 4 years. An electrocardiogram (fig. 6) revealed incomplete left bundle-branch block and evidence of left ventricular enlargement. The P-R interval was 0.22 second. The P waves were notched in I, II, III, and aV_F with depression of the P-Ta segment. In aV_L the P-Ta segment was elevated 0.5 mm. The P waves in V_1 and V_2 exhibited a slight positive and wide negative wave with a total duration of 0.11 second. A diagnosis of atrial infarction was suggested.

Three weeks later chest pain and increased failure occurred. The serum glutamic oxaloacetic transaminase levels were 207 and 316 units, and the white blood count and temperature were elevated. An electrocardiogram showed changes in the P waves and P-Ta segments. The P waves in leads V_{1-3} were biphasic and 0.10 second wide; in leads I, II, and aV_F , the terminal portions of the P waves were depressed and notched. The P-Ta segments were depressed in leads I, II, III, aV_F , and V_6 and elevated in aV_R . Terminally the P-Ta seg-

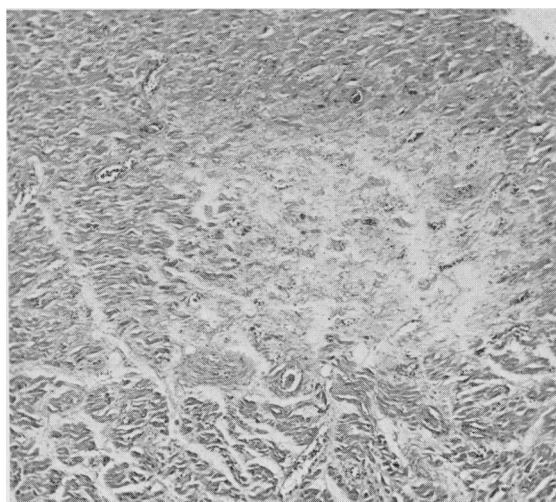


Figure 2

Case 1. Section of the atrium shows focal fibrosis and necrosis of the myocardium with polymorphonuclear infiltration.

ment became more depressed in lead I, and the terminal part of the P wave was more negative, being depressed 4 mm. in V_1 .

At autopsy the heart weighed 650 Gm. A 2 by 2 cm. clot was adherent to the posterior surface of the right atrium. Thrombi were also present on the posterior wall of the left ventricle. The coronary arteries showed extensive arteriosclerotic changes. Moderate interstitial fibrosis was present histologically. An area of acute inflammatory change with necrosis was seen under the mural atrial thrombus.

Case 5

A 79-year-old white man was admitted to the hospital because of chest pain of 2 weeks' duration associated with nausea and vomiting. He had basilar rales but no other signs of congestive heart failure. Leukocytosis was present initially, and the sedimentation rate became elevated. An initial electrocardiogram (fig. 7) showed subendocardial injury of the posterior surface of the left ventricle. The P-Ta segments were depressed 1 mm. in leads V_{1-3} and elevated from 1.0 to 1.5 mm. in leads V_5 and V_6 . The P-Ta segments in the limb leads were obscured by a changing T-P baseline. These changes in the P-Ta segments varied in subsequent tracings and a diagnosis of atrial infarction was made. The patient became cyanotic and hypotensive and died in 1 week. The serum glutamic oxaloacetic transaminase levels varied from 17 to 34 units.

At autopsy the heart weighed 300 Gm. The myocardium of the left and right ventricle was flabby and contained numerous petechiae and focal hemorrhages as did the posterior third of the interventricular septum. There were also hemor-

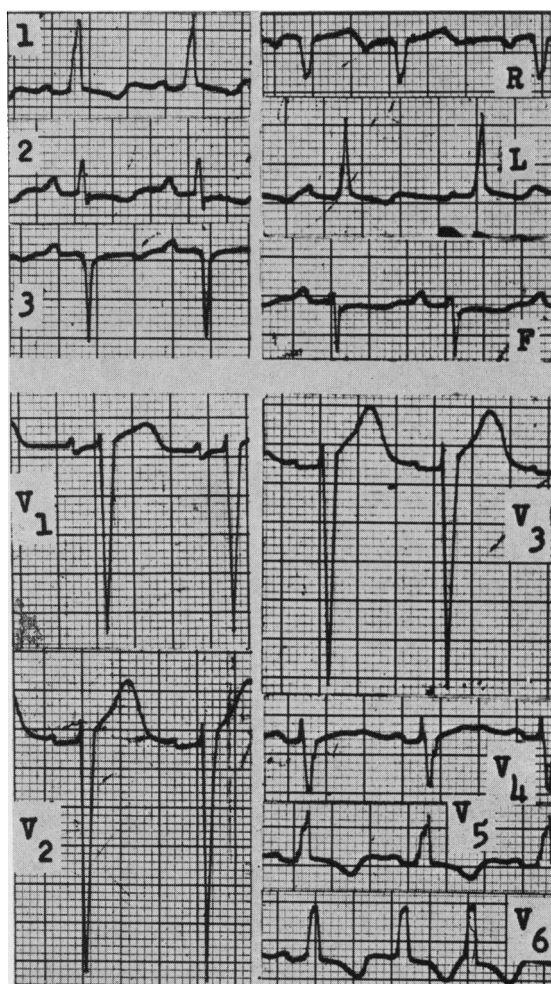


Figure 3

Case 2. Electrocardiogram shows incomplete left bundle-branch block and no evidence of acute ventricular infarction and P-Ta segment deviations. An atrial premature beat is seen in V_6 .

rhages in the posterior wall of both atria extending down to the atrioventricular junction. The right coronary artery was occluded 3 cm. from its origin by a thrombus and the left circumflex was occluded by a fresh thrombus. Histologic sections showed changes of acute myocardial infarction with necrosis and a large area of subepicardial atrial hemorrhage.

Case 6

A 72-year-old white man entered the hospital because of severe congestive failure and pain in the arms of 1 week's duration. Moderate congestive heart failure followed a myocardial infarction 3 years earlier. A leukocytosis of 23,000 per mm.³ and a serum glutamic oxaloacetic transaminase level of 26 units were present on admission. An electrocardiogram (fig. 8) revealed sinus tachy-

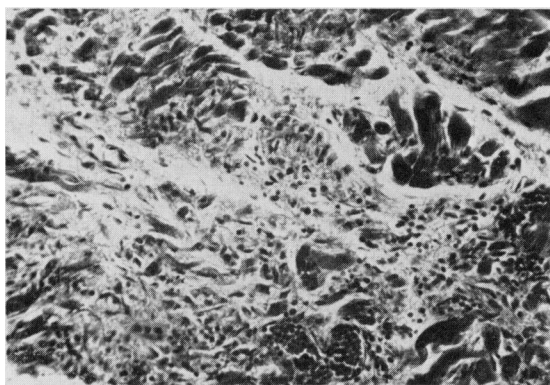


Figure 4

Case 2. Section of the atrium shows congestion of the small vessels with hemorrhage and necrosis and hyalinization of the muscle fibers.

cardia with a wandering pacemaker, premature atrial beats, and rare premature ventricular beats. The P wave was positive-negative in V_{1-3} . There was also evidence of apical myocardial infarction, age undetermined. Subsequently supraventricular tachycardia and electrical alternans appeared (fig. 8, lower). A diagnosis of atrial infarction was made. Finally ventricular tachycardia appeared and the patient died.

At autopsy the heart weighed 750 Gm.; it showed bilateral hypertrophy, an old scar at the apex, and a mural thrombus in the right atrium. The proximal portion of the left coronary artery was slightly narrowed by a calcium plaque. Histologic sections revealed the old apical infarction of the ventricle and showed an atrial infarction under the mural thrombus with subendocardial necrosis.

Results and Discussion

The clinical diagnosis of atrial infarction was made correctly ante mortem in six cases from the electrocardiogram. Both atria were involved in cases 1, 2, and 5, the right atrium alone in cases 4 and 6, and the left atrium in case 3. All cases had associated ventricular infarction. Changes of the P-Ta segment in the precordial leads were especially helpful in cases 1, 3, and 5. The diagnosis of atrial infarction is easily missed because the changes are less dramatic than those of the ventricular infarction usually accompanying it. Consequently relatively few cases have been reported in the literature, despite the reported incidence of 1 to 17 per cent among ventricular myocardial infarctions.^{3, 13, 14} The wide range may be due to the varying care and

criteria for its autopsy diagnosis. The atrium underlying a mural thrombus is probably rarely sectioned for histologic study, and in cases of ventricular infarction with grossly normal atria, histologic sections of the atria are seldom made.

Atrial infarctions produced experimentally in dogs and cats have been studied by Abramson¹⁵ and several other investigators,¹⁶⁻¹⁹ but the diagnostic electrocardiographic criteria have not been agreed upon. Elevations and depressions of the P-Ta segment have been of prime importance in the diagnosis of this condition. Analysis of the electrocardiograms of the patients presented shows that the P-Ta segment, when plotted vectorially, is directed toward the area of atrial infarction. This may be analogous to the direction of the current of injury (S-T segment) in ventricular infarction.²⁰ Therefore this segment might be called P-STa-Ta rather than the P-Ta segment. Because there is little atrial gradient,²¹ a direct comparison between ventricular repolarization and atrial repolarization may not be possible. For want of a better term, we use the P-Ta segment with the above understanding. The P-Ta segment usually extends through the QRS and S-T sections of the electrocardiogram, so that its complete examination is impossible unless complete heart block is present.

In our only case of isolated right atrial infarction (case 4, fig. 6) the P-Ta segment vector was directed to the right atrium and posteriorly. The P-Ta segment was depressed in leads I, II, III, aV_F , V_1 , V_2 , and occasionally V_3 . It became isoelectric in lead V_6 . It was elevated in leads aV_R and aV_L . In isolated left atrial infarction (case 3, fig. 5) the P-Ta segment was depressed in leads II, III, aV_F , V_1 , V_2 , and elevated in I, aV_R , aV_L , V_5 , and V_6 . In bilateral infarction the direction of the P-Ta segment vector may depend on which atrium is infarcted the more. In cases 1 (fig. 1) and 5 (fig. 7) (bilateral atrial infarction) the P-Ta segment vectors pointed to the left atrium and thus cannot be distinguished from isolated left atrial infarction.

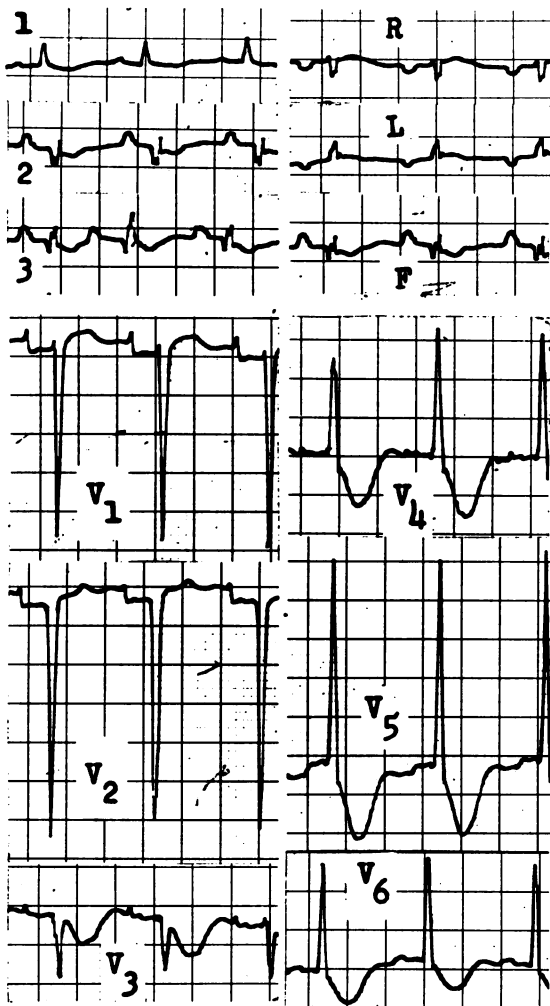


Figure 5

Case 3. Electrocardiogram shows P and P-Ta deviations and anteroseptal and diaphragmatic myocardial infarction.

In case 6 (fig. 7) there was no definite P-Ta segment vector that could be plotted. In this case, only supraventricular arrhythmias were present, which with the clinical picture of ventricular myocardial infarction led to the ante mortem diagnosis of atrial infarction as well. In this case the right atrium alone was involved where the sinoatrial node is located. It would be of interest to collect enough cases of left and right atrial infarction to determine whether these arrhythmias were more common in right atrial infarction.

Atrial arrhythmias are very common in

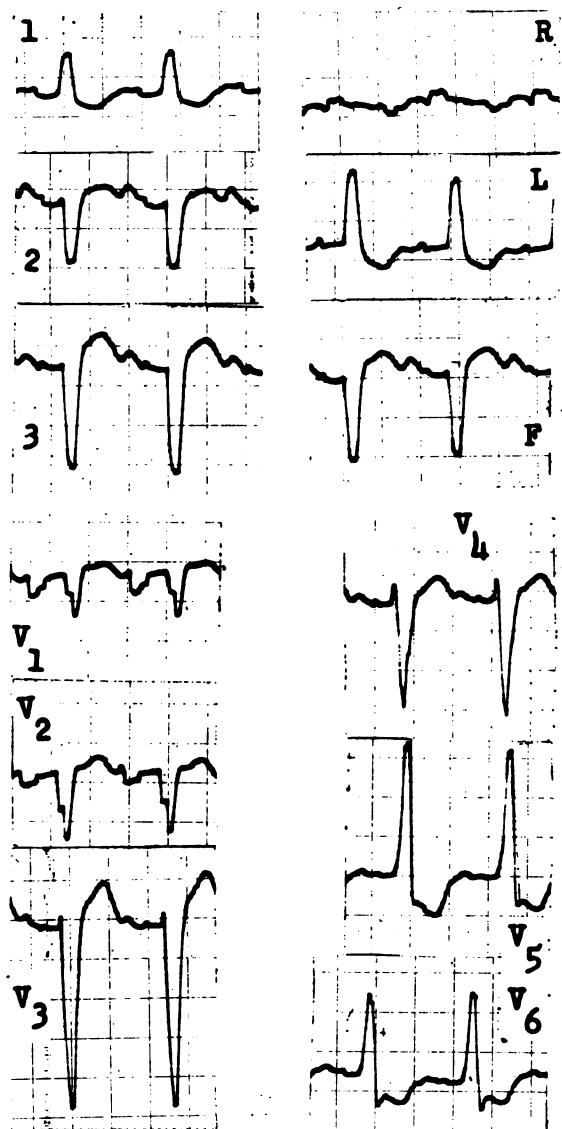


Figure 6

Case 4. Electrocardiogram shows depressed segments in leads I, II, III, and aV_F , positive-negative P waves in V_1 and V_2 , notched P wave in V_3 and V_4 , and some intraventricular block.

our cases of atrial infarction as well as those reported in the literature. The importance of a small infarction of the right atrium near the sinoatrial node has been stressed by Young and Koenig⁶ and could account for death by arrhythmia, despite its small size.

The beginning of the P waves may be fused with the T and U waves, especially in sinus tachycardia. The Q of the P has

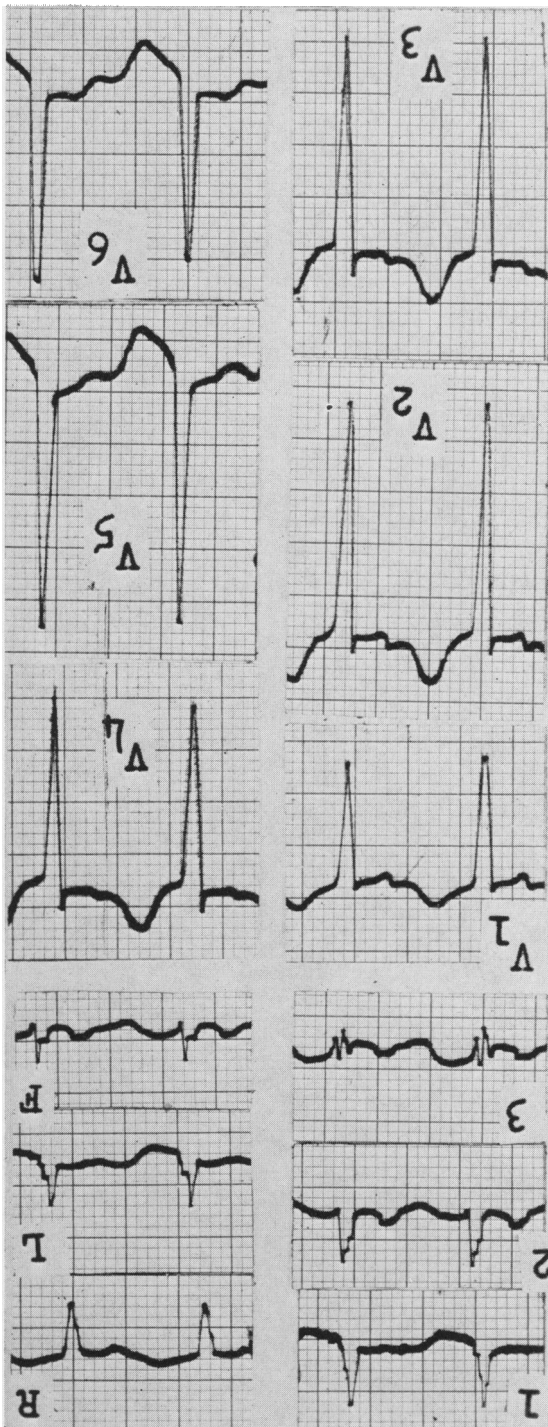


Figure 7

Case 5. There is a depression of the P-Ta segment in V_1 to V_3 , and elevation in V_5 and V_6 .

been described¹⁸ as in ventricular infarction, its vector may point away from the infarcted area. In case 3 in V_4 a negativity before the

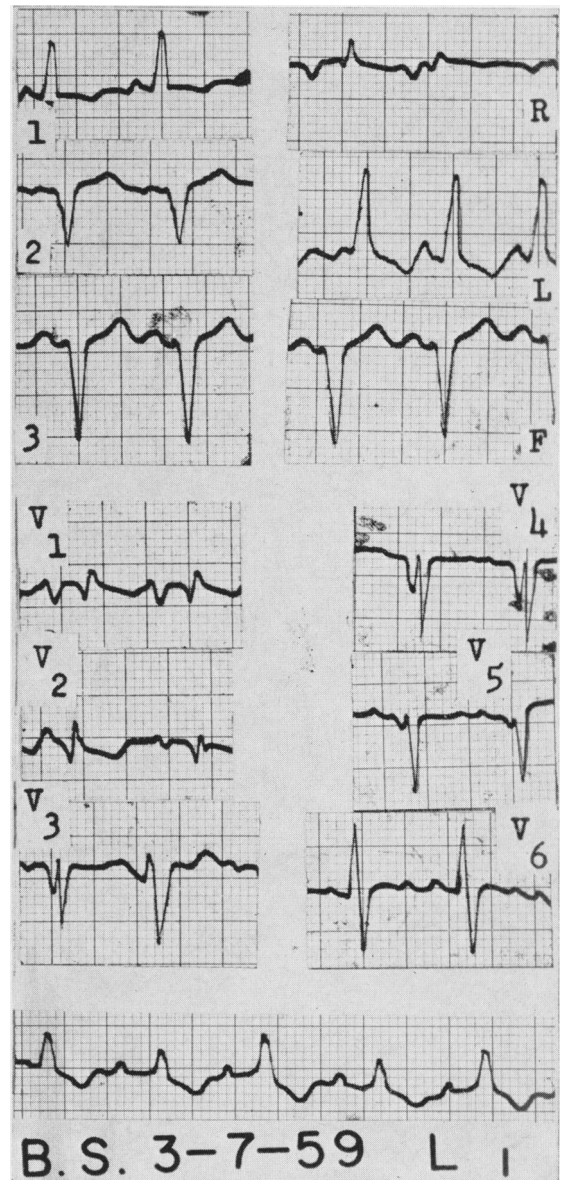


Figure 8

Case 6. Sinus tachycardia with wandering pacemaker and electrical alternans in a subsequent (lower) strip. Also present is an old apical myocardial infarction.

P might represent the atrial Q wave. It is at times difficult to determine where the P wave ends and the P-Ta segment begins, particularly in the right precordial chest leads and in tachycardia with short P-R interval. Often the end of the P wave merges with the P-Ta segment so that a clear separation is not possible. Some cases of atrial infarction reported in the literature^{4, 5, 9}

present P-Ta segment, elevations in leads II, III, and aV_F. These cases probably correspond to more inferior-posterior locations of the atrial infarction. Notching, widening, peaking, slurring, and positive-negative or negative-positive biphasic P waves are more difficult to evaluate in localization of atrial infarction.

The changes in the P-Ta segment in atrial infarction may last for only a few hours or a few days. In case 1, elevation of the P-Ta segment almost returned to the isoelectric line in 1 day. Complete electrocardiograms are advisable while the patients are having atrial premature beats, wandering pacemaker, or other forms of atrial arrhythmias. If atrial infarction is suspected, an electrocardiogram should also be taken immediately after sinus rhythm has resumed following supraventricular tachycardia or paroxysmal atrial fibrillation.

In our series, the left atrium was involved by infarction in all cases except cases 4 and 6. This is not in agreement with the reported incidence of the literature of more right-sided than left-sided lesions. Theoretically, one would expect to have a greater incidence of left atrial infarction because of the greater incidence of left ventricular infarction.

All six cases had sinus tachycardia during their illness. Atrial arrhythmias occurred in all but case 5. Among the atrial arrhythmias cases 2, 3, and 6 had atrial premature beats and runs of atrial tachycardia, case 1 had supraventricular tachycardia and paroxysmal atrial fibrillation, and cases 1, 3, 4, and 6 had a wandering pacemaker. Atrial flutter was not observed in our series. We consider that any form of atrial arrhythmia occurring in patients with ventricular myocardial infarction is highly suggestive of an accompanying atrial infarction as in case 3.

The diagnosis of atrial infarction is not only of academic interest but also of some clinical importance. In case 2, a diagnosis of acute ventricular myocardial infarction could not be made from the electrocardiographic changes but atrial infarction could.

We strongly believe that all patients with

electrocardiographic evidence of atrial myocardial infarction should be treated as if they had ventricular myocardial infarction because there may be ventricular involvement that does not show in the electrocardiogram.

The serum glutamic oxaloacetic transaminase levels were not helpful in the evaluation of isolated atrial infarction; in our cases elevations were always associated with ventricular infarction.

Since mural thrombi are so common in atrial infarction (2 of 6 cases) anticoagulant therapy seems indicated to decrease the probability of the formation of a mural atrial thrombus. Migration of atrial thrombi may be the cause of fatal cerebral or massive pulmonary infarction.

Summary

Six cases of atrial infarction associated with ventricular infarction are presented. All cases were diagnosed during life and were confirmed by autopsy.

A clinical diagnosis of atrial infarction should be suspected in patients with ventricular myocardial infarction having any form of atrial arrhythmia. Frequent electrocardiograms should be obtained, especially if sinus rhythm has just been re-established after episodes of supraventricular tachycardia or atrial fibrillation.

The *major* electrocardiographic criteria for the diagnosis of atrial infarction are as follows: elevation of the P-Ta segment of over 0.5 mm. in V₅ and V₆ with reciprocal depression of the same segment in V₁ and V₂; elevation of the P-Ta segment of over 0.5 mm. in lead I and its depression in leads II or III; depression of the P-Ta segment of more than 1.5 mm. in precordial leads and 1.2 mm. in leads I, II, and III in the presence of any form of atrial arrhythmia.

The *minor* electrocardiographic criteria in making the diagnosis of atrial infarction are as follows: abnormal P waves: M-shaped, W-shaped, irregular or notched; depression of the P-Ta segment of small amplitude without elevation of this segment in other leads cannot be regarded by itself as positive evidence of atrial infarction.

A diagnosis of atrial infarction can some-

times be made when the presence of ventricular myocardial infarction cannot be definitely established by electrocardiogram.

The treatment of atrial infarction is similar to that of ventricular infarction. Attention should be directed to the control of atrial arrhythmias and to the prevention of mural thrombi.

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