Massive Infarction of the Right Ventricle and Atrium

Report of a Case

By Earl A. Zaus, M.D., and Walter M. Kearns, Jr., M.D.

This is a report of a case of massive infarction of the right ventricle and atrium, expanded to include similar cases found in the literature. The clinical manifestations are believed to confirm views expressed by others that peripheral congestive failure is not immediately related to failure of the right ventricle. The diagnostic value of the electrocardiogram in this lesion is examined.

In a well-reasoned paper in 1949 Starr1 presented clinical and experimental evidence which disputed the classic view that backward failure of the right ventricle was the sole factor responsible for congestive failure. He and his co-workers2 had found that complete destruction of the right ventricle in dogs did not produce a rise in venous pressure. This was confirmed by Bakos.3

Recently we observed the effects of destruction of the right ventricle in man due to infarction. A report of this case will follow. Included in this study are also 11 similar cases found in the literature. In necropsy material4 totaling 1651 infarcted hearts* there were only 29 isolated infarctions of the right ventricle. Only four among this group were documented well enough to be used for the purpose of this paper. From other sources5-11 an additional seven cases were found. The anatomic and clinical features of these cases are summarized in table 1.

Case Report

The patient, a 69 year old white male, was admitted for an attack of precordial pain which had occurred 10 days before admission. Three and one-half months previously he had consulted a physician for severe frontal headaches; a blood pressure of 176/104 was recorded. An electrocardiogram taken at that time is shown in figure 1A.

At home the precordial distress had subsided but he was extremely weak and showed cyanosis and dyspnea upon exertion, as he refused to stay in bed.

* There may be some duplication of cases in two of these reports6 and 7, as both originate from the Massachusetts General Hospital, 1914-1934 and 1926-1945, respectively.

At times he was rather stuporous. For one week after the onset he had been completely anuric. Slight swelling of the ankles had been present, clearing on bed rest.

Physical examination in the hospital revealed an acutely ill, stuporous, moderately dyspneic white male, weighing 220 pounds, 62 inches tall, with grade 2 cyanosis, temperature 98.0 F., pulse 56, regular and weak, and a blood pressure of 132/70. The exposed skin surfaces were cold and damp. The jugular veins were moderately distended while the superficial arm veins were in collapse. The sclerae were icteric and the breath ammoniacal. There was dullness, reduced fremitus, and absent breath sounds in the lower right chest posteriorly. Medium moist rales were present in the left base. The heart was enlarged to the left anterior axillary line, the tones distant, but no murmurs or friction rub were heard. There was marked tenderness in the right upper quadrant of the abdomen and a fluid wave was thought to be present. The liver was palpated three fingerbreadths below the right costal margin. There was slight sacral edema.

On the morning of the fourth hospital day, he suddenly became very dyspneic and expired.

Laboratory Findings

The urine showed a specific gravity of 1.010 to 1.014, no sugar, albumin or bile; but 20 to 25 red blood cells per high power field and a 2 plus urobilinogen were found on admission. Twenty-four hour urinary output ranged from 850 to 1200 cc. There were 23,000 white blood cells per cubic millimeter. The Kahn test was negative. Blood chemistry per 100 cc. showed 200.5 mg. glucose, 160 mg. non-protein nitrogen, 1.48 units of alkaline phosphatase, 156 mg. cholesterol, total protein of 5.9 Gm., 2.8 Gm. of albumin and 3.1 Gm. of globulin. The cephalin flocculation test was plus 2 at the end of 24 hours and plus 3 in 48 hours, and the icteric index was 27.

On the third hospital day, the nonprotein nitrogen was 131.5 mg. per 100 cc.

An electrocardiogram taken on the day of admis-
Table 1.—Anatomic and Clinical Findings in Reported Cases of Primary Infarction of the Right Ventricle

<table>
<thead>
<tr>
<th>Case and Source</th>
<th>Recent</th>
<th>Old</th>
<th>Description of Coronary Artery Pathology</th>
<th>Electrocardiogram</th>
<th>Clinical Picture</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>R</td>
<td>L</td>
<td>S</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. (5)</td>
<td>A</td>
<td>?</td>
<td>A</td>
<td>Thrombus in main right over-laying old clot; marked sclerosis of left.</td>
<td>QTc pattern</td>
</tr>
<tr>
<td>2. (4a)</td>
<td>A</td>
<td>P</td>
<td>A</td>
<td>Thrombus in main right 2 cm. from ostium extending well into infarcted area; marked arteriosclerosis; left normal.</td>
<td>QTc pattern</td>
</tr>
<tr>
<td>3. (6)</td>
<td>A</td>
<td>P</td>
<td>L</td>
<td>Thrombus in main right 2 cm. from ostium extending well into infarcted area; marked arteriosclerosis; left normal.</td>
<td>QTc pattern</td>
</tr>
<tr>
<td>4. (7)</td>
<td>A</td>
<td>P</td>
<td>L</td>
<td>Thrombus in main right 2 cm. from ostium extending well into infarcted area; marked arteriosclerosis; left normal.</td>
<td>QTc pattern</td>
</tr>
<tr>
<td>5. (4j)</td>
<td>A</td>
<td>P</td>
<td>L</td>
<td>Thrombus in main right 2 cm. from ostium extending well into infarcted area; marked arteriosclerosis; left normal.</td>
<td>QTc pattern</td>
</tr>
<tr>
<td>6. (4j)</td>
<td>A</td>
<td>P</td>
<td>L</td>
<td>Thrombus in main right 2 cm. from ostium extending well into infarcted area; marked arteriosclerosis; left normal.</td>
<td>QTc pattern</td>
</tr>
<tr>
<td>7. (4)</td>
<td>P</td>
<td>L</td>
<td>P</td>
<td>Complete occlusion of right 3 cm. from origin; also at right margin; narrowing of left anterior descending.</td>
<td>Low voltage; slurred QRS complexes; inverted T&lt;sub&gt;L&lt;/sub&gt;, L.</td>
</tr>
<tr>
<td>8. (9)</td>
<td>P</td>
<td>L</td>
<td>P</td>
<td>Recent clot in right; marked obliterative sclerosis of all arteries.</td>
<td>Not given.</td>
</tr>
<tr>
<td>9. (10)</td>
<td>P</td>
<td>L</td>
<td>P</td>
<td>Recent and old clots right; sclerosis of all arteries.</td>
<td>QTc pattern.</td>
</tr>
<tr>
<td>10. (11)</td>
<td>P</td>
<td>L</td>
<td>A</td>
<td>Congenital single artery arising from right, with anomalous large branch going left, with all branches.</td>
<td>QTc pattern; CF&lt;sub&gt;1&lt;/sub&gt; shows marked S-T segment depression.</td>
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<td>P</td>
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</tr>
</tbody>
</table>

Key: A = Anterior wall; P = Posterior wall; L = Lateral wall; At = Atrium; ? = Data indefinite; R = Right; L = Left; S = Septum.
Fig. 1A. Tracing taken about three months before terminal attack, suggestive of damage to the posterior wall of the left ventricle.

B. Taken on tenth day of attack: the heart is horizontal with moderate clockwise rotation; idioventricular rhythm with rate of 56 is present; corrected Q-T interval measures 0.447 seconds*; the limb leads show a QST pattern; the right-sided precordial leads show an Rs complex, plateau-type S-T segment, and T wave inversion; the left-sided leads show an RS complex, depression of the S-T segment, and diphasic T waves with major deflection downward; in aVL the S-T segment is depressed, while in aVF there is a QS complex, and depression of the S-T segment and the T wave.

The possible interpretations of these complexes are: (1) left ventricular anteroseptal infarct, (2) hypertrophy-strain pattern of the left ventricle, (3) right ventricular strain pattern with superimposed digitalis* effect, (5) subendocardial infarct of the anterior wall of the left ventricle plus right ventricular strain, (6) anterior pericarditis, (7) anteroposterior infarction of the right ventricle.

dition (figure 1B) was interpreted as being indicative of severe myocardial damage.

Postmortem Description

The heart weighed 500 Gm. There was moderate hypertrophy of both ventricles and moderate dilatation of the right ventricle. Almost the entire wall of the right ventricle was occupied by a recent infarct (fig. 2). In addition a small portion of the posterior wall of the left ventricle and interventricular septum was involved. The site of the infarct corresponded approximately to the location of the deep sinospiral muscle and a portion of the adjacent superficial sinospiral bundle. A large mural thrombus occupied the lateral and posterior walls of the right ventricle. There was a recent infarct and mural thrombus of the entire right auricular appendage and a small portion of the adjacent right atrium and the left auricular appendage. Fibrosis from an old infarct was present in the posterior wall and adjacent interventricular septum of the left ventricle. Microscopically, the recent infarcts were judged to be about two weeks old.

There was marked right coronary artery preponderance. All the coronary arteries showed moderate to marked arteriosclerosis, the smaller left coronary artery and its main branches demonstrating more advanced changes than the large right coronary artery. There was marked narrowing of the lumina of the left circumflex and left anterior

* No glycosides were used in therapy of this patient.
descending arteries. A recent thrombus totally occluded the main right coronary artery for a distance of 8 cm. starting from a point 0.5 cm. distal to the ostium. An old canalized thrombus was present just proximal to the right posterior descending artery and a recent thrombus was present here for a distance of 1.0 cm. Three large branches of the right coronary artery supplying the right ventricle were occluded in their proximal portions by extension of the thrombus in the parent vessel. There was a small patent accessory right coronary artery. The coronary sinus and anterior cardiac veins were patent. There was no gross or microscopic evidence of obstruction of the thebesian or arterioluminal groups of vessels.

The right and left lungs weighed 320 and 260* Gm. respectively. There were recent emboli measuring up to 2.0 cm. in length in the smaller branches of both pulmonary arteries. Both lower lobes contained recent infarcts measuring up to 3 cm. in maximum diameter. Histologically, the lungs showed passive hyperemia, and slight chronic pulmonary emphysema.

The liver weighed 1700 Gm. and showed passive hyperemia. The pancreas showed focal areas of necrosis with acute and chronic inflammation. The spleen weighed 250 Gm. and showed passive hyperemia. The kidneys showed marked arteriolar sclerosis and regenerating tubular epithelium consistent with a diagnosis of a lower nephron lesion.

**DICUSION**

**Pathologic anatomy.** Various anatomie14, 15-16 and physiologic† reasons may account for the rarity20-22 of isolated right ventricular infarction; either the right ventricle is favored with a better collateral circulation, or damage to it requires greater restriction in blood supply than is necessary to produce ischemic death in the left ventricle. The status of the coronary circulation in this and in cases 4, 5, 8, and 9 (table 1) suggest the importance of the latter factor.

Clinical findings. It is remarkable that this patient lived 14 days with such extensive damage to the right ventricle. In the reported cases (table 1) and the series of Wang and co-workers,41 death resulted within a few days. One is forced to speculate as to the source of the force which propelled enough blood through the lungs to the left ventricle to meet the needs of vital areas for so long. Bakos3 suggested that it may come from the deep sinus spiral muscle of the contiguous left ventricle.

The clinical picture in this and cases 2, 3, and 5 was that of low input-output failure of the left ventricle; that is, fall in blood pressure, collapse of peripheral veins, cold, ashen-gray skin, mental irritability, stupor, anuria, and azotemia. Peripheral congestive failure was not prominent, undoubtedly due to the fact that the factors1, 29-31 responsible for electrolyte and water retention were operative for too short a period. This supports the views of Starr.2

Electrocardiographic findings. The tracings in figure 1B and in the reported cases (table 1) show that there is nothing distinctive of right ventricular damage. Myers and his group24 found that the electrocardiogram failed to show any evidence of extension of left ventricular infarction into the right ventricle. In our case, the absence of progress tracings, and complication by the effects of an old posteroseptal infarct and the recent myocardial disturbance incident to the pulmonary emboli, may have been additional factors in reducing the diagnostic value of the electrocardiogram. Some suspicion of the possibility of right myocardial damage may be warranted, however, from the findings of (1) increase in the potential of the small right-sided R waves as the transitional zone was approached; (2) the reciprocal changes in the S-T segments on the right and left side of the precordium, as pointed out by Wolferth and his associates27 and Dressler and Roessler.28 A similar effect was reported in case 4 (table 1).
SUMMARY

1. Search of the literature confirms the clinical impression that massive right ventricular infarction is rare.
2. The pathogenesis of infarction of the right ventricle is briefly discussed.
3. The electrocardiographic diagnosis of right ventricular infarction is difficult because of resemblance of the changes to patterns found in several other cardiac situations. There are no absolute signs of right-sided lesions.
4. Most of the early clinical manifestations produced by damage to the right ventricle are due to low input-output failure of the left ventricle. Signs of peripheral congestive failure may appear but their severity is related to other factors in greater degree than to incompetence of the right ventricle per se. Usually death intervenes too soon to permit severe congestion to appear.

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