RIGHT VENTRICULAR MYOCARDIAL INFARCTION

"L'infarctus du ventricule droit"

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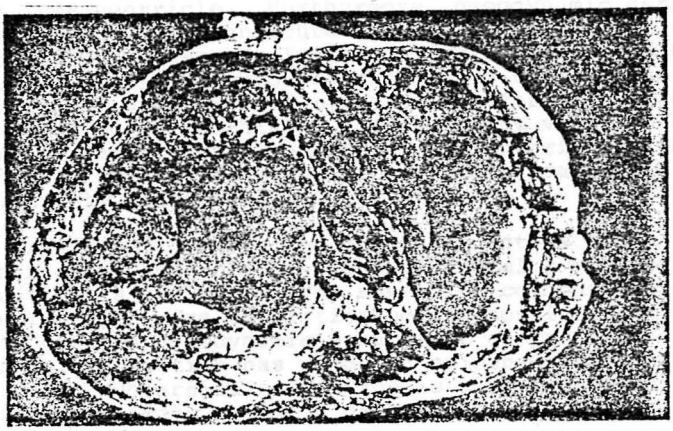
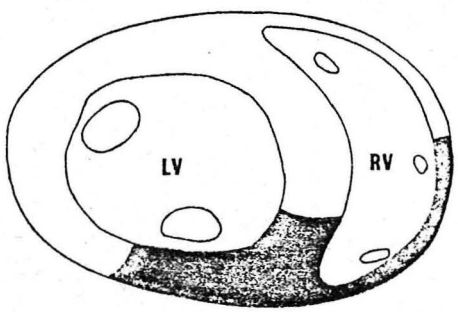


Right ventricular myocardial infarction associated with an inferior left ventricular infarction.



Introduction

Acute myocardial infarction predominantly involves the left ventricle. Furthermore, hemodynamic derangements produced by an acute myocardial infarction almost invariably present as left ventricular dysfunction. Until recently, diagnostic techniques utilized to confirm a clinical diagnosis of myocardial infarction were either nonspecific or non-diagnostic for right ventricular myocardial infarction. Thus it is not surprising that this clinical entity has remained a pathological curio until relatively recently.

While not common, and obviously not as common as left ventricular infarction, right ventricular myocardial infarction is not rare. The advent of coronary care units coupled with more sophisticated diagnostic techniques and more precise hemodynamic management of patients with myocardial infarction has produced evidence that right ventricular myocardial infarction is a well-defined clinical entity requiring specific management.

The following is a review of this clinical entity as it stands today.

Incidence

The initial reports of the incidence of involvement of the right ventricular myocardium in acute myocardial infarction were in cumulative autopsy studies. Table 1 lists the frequency with which right ventricular infarction was seen at post-mortem. The incidence ranges from 5 to 43 per cent, with a mean of 19 per cent. Erhardt[1] points out that in most of the early series, the right ventricle was either not examined or was not sliced correctly. Nevertheless, the series reported by Isner and Roberts[2] shows the incidence of right ventricular infarction to be no greater than some of the earlier studies.

Table 1. Incidence of right ventricular myocardial infarction in autopsy reports.

	Number	Per cent
Lisa and Ring (1932)[3]	22/160	14
Applebaum and Nicolson (1935)[4]	8/150	5
Bean (1939)[5]	95/287	33
Yater (1948)[6]		5
Wartman and Hellerstein (1948)[7]	22/60	14
Myers, et al (1949)[8]	19/161	12
Erhardt (1974)[1]	36/87	43
Isner and Roberts (1978)[2]	33/236	14
Ratliff and Hackel (1980)[9]	35/102	34
	Mean 19%	
	Range 5-43%	

The determination of the clinical incidence of right ventricular infarction is more difficult. The early literature is confined to case reports, usually of patients who were admitted in cardiogenic shock and subsequently died. Since the clinical syndrome associated with the condition was described by Cohn and his colleagues[10] and by Rotman, et al[11], the frequency with which right ventricular infarction occurs in a clinical setting has been addressed. Furthermore, several additional noninvasive techniques have been shown to be capable of diagnosing right ventricular myocardial infarction with reliability[12-16]. Table 2 lists the reports quoting the clinically diagnosed incidence of right ventricular infarction. Although one would expect it to be less, the mean incidence of 19 per cent is identical to the morphologically reported incidence. In this table I have listed the method by which the clinical diagnosis was made. The fact that this has an important bearing on the estimation of incidence is illustrated

Table 2. Incidence of right ventricular myocardial infarction in clinical reports.

	Number	Per cent	Method of Diagnosis
Cohn, et al (1974)[10]	6/74	8	Hemodynamic
Erhardt, et al (1976)[13]	25/92	27	ECG
Daubert, et al (1977)[14]	19/70	27	Hemodynamic
Wackers, et al (1978)[15]	24/64	38	PYP scan
Tobinick, et al (1978)[16]	7/43	16	PYP scan
Sharpe, et al (1978)[17]	6/26	23	PYP scan
Lorell, et al (1979)[18]	8/306	3	Hemodynamic
Coma-Canella, et al (1979)[19]	12/140	9	Hemodynamic
	Mean 19%		
	Range 3-38%		

by the series of Wackers and colleagues[15] who encountered right ventricular involvement much more often than other investigators. They note that only one patient in their series using technetium pyrophosphate scintigraphy as the method of identification had elevated central venous pressures. Erhardt, et al[13], on the other hand, using the electrocardiogram to identify right ventricular involvement, found 65 of their 92 patients to have evidence of right heart failure. Furthermore, the studies of Rigo, et al, Erhardt, et al, Wackers, et al, and Lorell, et al[12,13,15,18] include only patients with acute inferior wall left ventricular infarctions.

Thus, both the morphological and clinical estimates of incidence of right ventricular infarction are approximate and at best one can summarize the overall incidence as at least 19 per cent of all myocardial infarctions. Considering an incidence of one in five, it is surprising that this clinical entity has not received more attention.

Pathology

In 1952, Zaus and Kearns[20] stated that isolated right ventricular myocardial infarction was rare. They surveyed 11 autopsy sources reporting 1651 infarcted hearts and found only 29 isolated right ventricular infarcts, representing an incidence of 1.7 per cent. This concurred with Wartman and Hellerstein[7] who noted isolated right ventricular infarctions in

4 of 160 autopsied infarction patients. The three most recent pathological surveys listed in Table 1 noted no isolated right ventricular infarcts[1,2,9].

The remainder of the reported right ventricular infarctions are associated with left ventricular infarcts. In the recent pathological reports, while Erhardt[1] reported the majority (58 per cent) occurred with inferior left ventricular infarcts, Isner and Roberts[2] and Ratliff and Hackel[9] have reported that right ventricular infarction occurs exclusively in association with inferior left ventricular infarcts.

Several old wives' tales have surrounded the occurrence of right ventricular infarctions as described in the older literature. It was suggested by Wade[21] that right ventricular infarction was usually the direct result of an occlusion of the right coronary artery, with disease of the other major coronary vessels playing a minor role in the pathogenesis of the infarction. As the infero-posterior surface of both ventricles are supplied by the right coronary artery in 85 per cent of patients, this would seem to be a reasonable observation[22]. It was also pointed out by Wade[21] that when he compared 11 autopsied hearts with right ventricular infarction with 100 hearts with left ventricular infarcts, right ventricular hypertrophy was one and a half times more common and chronic pulmonary lesions (emphysema, COPD, and pulmonary fibrosis)were four times more common in the right ventricular infarction group.

The comments regarding both total right coronary arterial occlusion and the presence of chronic pulmonary disease with right ventricular hypertrophy in association with right ventricular infarction are interesting in the light of some experimental work by Ratliffe's group at Duke[23]. They developed a model for the experimental production of right ventricular infarction. They had reported that gradual occlusion of the proximal right coronary artery in the farm pig produces a myocardial infarction of the interventricular septum and the posterior wall of the left ventricle[24]. If, however, right ventricular hypertrophy is induced prior to right coronary arterial occlusion, right ventricular infarction is consistently produced[23]. A subsequent study by the same group showed that the experimental production of right ventricular infarction by induction of right ventricular hypertrophy impeded the development of collateral filling of the occluded right coronary artery[25].

These experimental observations would appear on the surface to be confirmed by the work on patients of Hamby, et al[26].

They reviewed the cardiac catheterization data of 465 patients and found that the collateral circulation appears to have a protective role against anterior myocardial infarction in a significant number of patients with complete occlusion of the anterior ascending branch of the left coronary artery. Conversely, patients with 100 per cent occlusion of the right coronary artery appeared to have no beneficial effect from collateral circulation. The frequency of an inferior wall infarction was identical with or without a collateral circulation (Figure 1). However, right ventricular angiograms were not performed in these patients and no comments regarding right ventricular infarctions were made.

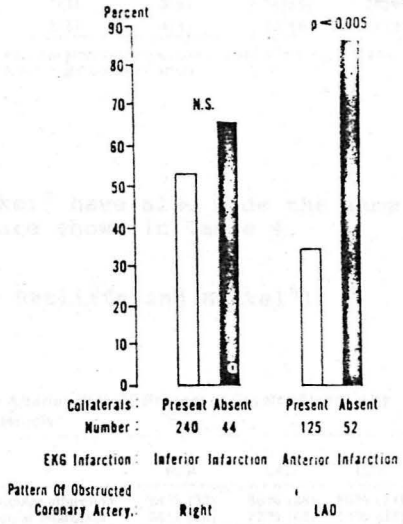


Figure 1. Patients with complete LAD occlusion and anterior infarctions compared to patients with complete RCA occlusions and inferior infarctions[26].

Isner and Roberts[2] have recently addressed the question of the association between complete right coronary arterial occlusion and right ventricular infarction. They compared inferior left ventricular infarctions with and without asso-

ciated right ventricular infarction (Table 3). No significant differences were found in the distributions of the coronary pathoanatomy and in particular no difference in the frequency of occlusion of the right coronary artery.

Table 3. (From Isner and Roberts[2])

Status of Right Ventricle and Coronary Arteries in Patients With Posterior Wall Left Ventricular Myocardial Infarct (MI) With and Without Associated Right Ventricular (RV) Infarct

LVMI	Cases (no.)	D (no. [%])	H (no. [%])	T (no. [%])	Patients With Histologic Study of Coronary Arteries (no. [%])			
					Total (no. [%])	With >75% Narrowing* of		
						RCA	LAD	LCx
With RV MI	33	12[36]	1[3]	3[9]	28[85]	26[93]	20[74]	20[74]
Without RV MI	106	10[9]	5[5]	4[4]	59[56]	50[85]	46[78]	45[76]

* More than 75 percent narrowing of cross-sectional area by plaque. D = dilatation; H = hypertrophy; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; RCA = right coronary artery.

Ratliff and Hackel[9] have also made the same comparison, and their findings are shown in Table 4.

Table 4. (From Ratliffe and Hackel[9])

Coronary Arteries With 75 Percent or Greater Stenosis by Atherosclerosis*

	Artery		
	RCA	LAD	LCx
Right ventricular infarction	91% (32)	80% (28)	60% (21)
Left ventricular infarction	66% (44)	72% (48)	51% (34)

* The number of cases is in parentheses. The differences are not statistically significant.
LAD = left anterior descending artery; LCx = left circumflex artery; RCA = right coronary artery.

Again, no significant differences in the distribution of the coronary pathoanatomy were found. They confirmed the conclusions of Isner and Roberts that the high incidence of right coronary arterial occlusion was apparent rather than real because comparisons were not made either

with the pathoanatomy associated with isolated left ventricular infarctions or with the incidence of occlusion of the other major coronary vessels in right ventricular infarcts.

The association between right ventricular infarction and right ventricular hypertrophy has also been looked into more recently. Erhardt[1] found no difference in directly measured right ventricular wall thickness between patients with right and with left ventricular infarctions. Isner and Roberts[2] found that right ventricular hypertrophy was more common in the group of patients without right ventricular infarcts, as shown in Table 3. Furthermore, none of the 236 patients had autopsy evidence of cor pulmonale. They did point out, as can be seen in Table 3, that dilatation of the right ventricular cavity was present in 12 (36 per cent) of 33 patients with, and in 10 (9 per cent) of 106 patients without, right ventricular infarctions ($p < 0.05$). The importance of this observation will be seen when we discuss the diagnostic features of patients with right ventricular infarctions.

The early anecdotal association of right ventricular mural thrombus with infarction has also been refuted recently[21,9].

Cauterization of the entire free wall is a freely applied experimental model used to study the hemodynamic function of the right ventricle and to simulate the effects of massive right ventricular infarction[27,28,29]. Investigators have shown that damage of this degree to the right ventricular myocardium did not increase pressures on the right side of the heart. They concluded that normal right ventricular function is not necessary for circulatory stability provided the pulmonary vascular bed is normal. Such conclusions appeared to be supported by the studies of Rodbard and Wagner[30], who demonstrated that pulmonary blood flow could be maintained when the right ventricle was excluded from the circulation.

More recent studies by Guiha, et al[31] showed that cauterization of the right ventricular wall does, in fact, produce right ventricular dysfunction. Their mean data are shown in Table 5. After cautery, the right ventricular end-diastolic pressure increased from 2.6 to 5 mm mercury. This increase was not as great, however, as the increase in left ventricular end-diastolic pressure after cauterization of the left ventricle, from 6.5 to 17 mm mercury.

Table 5. (From Guiha, et al[31])

Effect of Acute Blood Volume Expansion on Right and Left Ventricular End-Diastolic Pressures (mm Hg) in Open Chest Dogs (means ± standard error of the mean)

	RVEDP	LVEDP
Normal (6 dogs)		
Control	2.6 ± 1.0	6.5 ± 0.5
Volume load	10.5 ± 2.0	22.0 ± 4.3
RV damage (4 dogs)		
Control	5.0 ± 0.8	5.3 ± 1.2
Volume load	21.0 ± 2.3	17.0 ± 3.1
LV damage (2 dogs)		
Control	8.5 ± 0.5	17.0 ± 0.7
Volume load	15.0 ± 0.7	40.0 ± 2.8

LV = left ventricular; LVEDP = left ventricular end-diastolic pressure; RV = right ventricular; RVEDP = right ventricular end-diastolic pressure.

The effects of rapid volume expansion by 0.5 to one liter of saline before and after cauterization of each ventricle are shown in Figure 2. In particular, volume

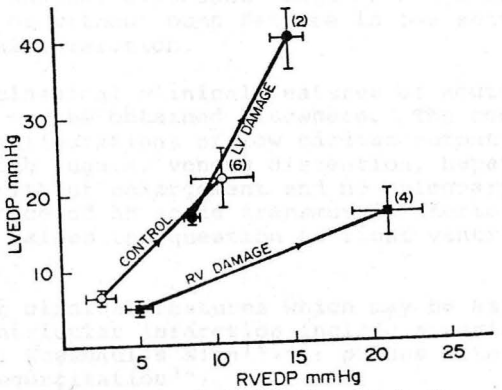


Figure 2. Right (RV) and left ventricular (LV) end-diastolic pressures compared pre and post volume expansion during control studies, after RV cautery and after LV cautery. (from Guiha, et al[31])

expansion of the damaged right ventricle demonstrates right ventricular dysfunction more clearly, the mean right ventricular end-diastolic pressure increasing from 5 to 21 mm mercury, with equalization of filling pressures of the left ventricle. Of interest is the more profound left ventricular dysfunction produced by extensive cauterization of the left ventricle with an increase in mean left ventricular end-diastolic pressure of 6.5 to 17 mm mercury. Volume expansion produced a further increase to 40 mm mercury. These findings allowed the investigators to conclude that although right ventricular damage produces disproportionate right ventricular dysfunction, isolated right ventricular damage should not in itself lead to irretrievable hemodynamic impairment. However, they felt that right accompanied by left ventricular damage should produce sufficient derangement that right ventricular dysfunction may be accompanied by a low cardiac output state. As will be seen later, this has important clinical as well as prognostic implications.

Clinical Features

It is only in the last decade that attention has been paid to the clinical diagnosis of right ventricular myocardial infarction. Prior to that, literature reports were confined to case reports accompanied by autopsy findings. Cohn, et al[10] drew attention to the clinical diagnoses as another important cause of congestive heart failure with or without pump failure in the setting of acute myocardial infarction.

The classical clinical features of acute myocardial infarction may be obtained elsewhere. The central and peripheral manifestations of low cardiac output syndrome associated with jugular venous distention, hepatic tenderness with or without enlargement and no pulmonary congestion in the presence of an acute transmural inferior myocardial infarction raises the question of right ventricular involvement[10,11].

Other clinical features which may be associated with right ventricular infarction include a pericardial friction rub[21,18]; Kussmaul's sign[18,32]; pulsus alternans[33]; and tricuspid regurgitation[34].

Diagnosis

Electrocardiography

Right ventricular myocardial infarction is invariably associated with a transmural inferior left ventricular myocardial infarction. Figure 3 shows the classical electrocardiographic pattern of Q waves in leads II, III, and aVF with associated ST segment elevation and T wave inversion.

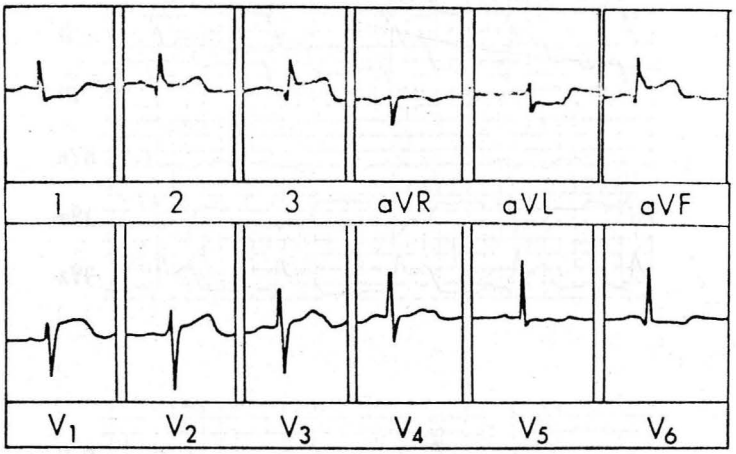


Figure 3. Acute inferior wall myocardial infarction (from Clark, et al[32])

Perhaps one of the reasons for the less than enthusiastic pursuits of the diagnosis of right ventricular infarction has been the inability of the electrocardiogram to specifically pinpoint infarction involving the right ventricular wall. Erhardt, et al[13] addressed this question in a series of patients admitted with inferior myocardial infarctions. In addition to the conventional 12 lead electrocardiogram, they included a recording of lead V_4R. Figure 4 gives an example of an inferior wall and right ventricular infarction, showing the ST elevation in lead V4R in addition to the classical changes in leads II, III, and aVF and reciprocal changes in leads I and aVL. Erhardt and his colleagues[13] noted the abnormal changes in lead V_4R in 25 of 92 patients with inferior

infarctions, and in 9 of 18 patients who subsequently died from their infarcts. It is interesting to note that this electrocardiogram also shows the changes classically associated with a true posterior infarction of R waves of 0.04 sec and upright T waves in lead V, with an altered R/S ratio in leads V_1 and V_2[35].

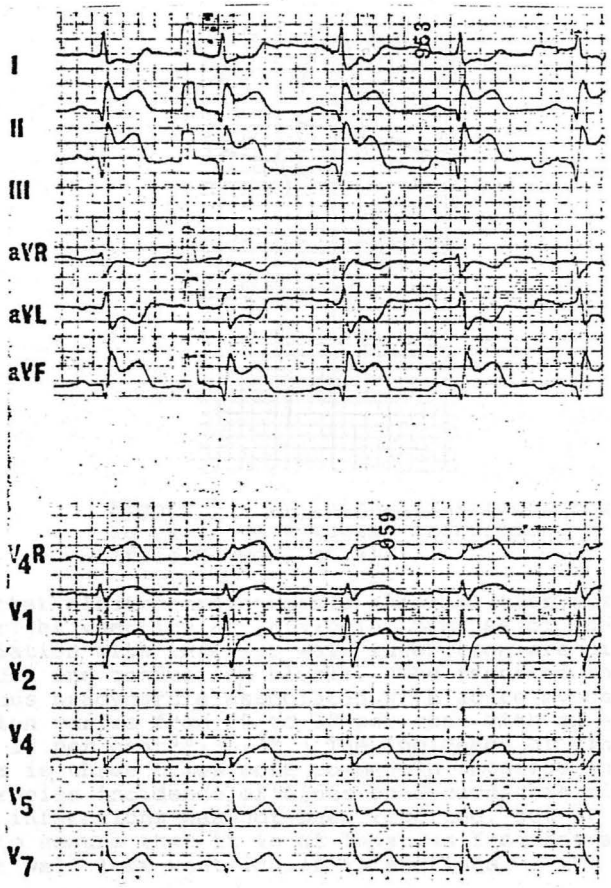


Figure 4. Acute right ventricular myocardial infarction showing the changes in the conventional leads and ST elevation in lead V_4R. (from Erhardt[55])

A recent observation has been the demonstration of a current of injury on a right ventricular intercavity recording following right ventricular infarction (Figure 5). This may be shown when the placement of a temporary pacemaker is necessary by attaching the distal pole of the pacemaker electrode to the chest lead of the electrocardiographic recorder.

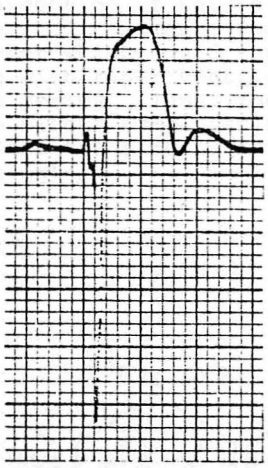


Figure 5. RV intracavity recording in right ventricular infarction. (from Marmor, et al[62])

Other electrocardiographic abnormalities associated with right ventricular infarction because of their close association with inferior wall infarction are sinus bradycardia and conduction blocks. The reported incidence of sinus bradycardia associated with acute myocardial infarction varies from 10 to 30 per cent with an average of about 15 per cent[36,37,38]. The incidence of sinus bradycardia is three times more common in inferior infarctions. The precise incidence of sinus bradycardia in right ventricular infarctions has not been reported, but it is reasonable to assume that it is at least as frequent as in inferior wall left ventricular infarctions.

The reported incidence of conduction blocks complicating acute myocardial infarction varies from 5 to 25 per cent[39,40,41]. The incidence of inferior wall infarc-

tions is approximately twice that of anterior infarctions. Again, the precise incidence of conduction abnormalities in right ventricular infarctions is unknown. However, it may be even higher than in inferior wall infarcts. Five of Cohn's six[10], one of Jensen's two[42], two of Sharpe's six[17], and four of Lorell's twelve patients[18] all had either second or third degree atrio-ventricular block. However, with the exception of Sharpe's series, identification of all these study patients was by direct, invasive hemodynamic assessment which would thus eliminate large numbers of patients with right ventricular infarctions from the respective series. Thus, the anticipated incidence of conduction blocks complicating right ventricular infarction is at least the same as inferior wall infarction and probably greater.

Myocardial Scintigraphy

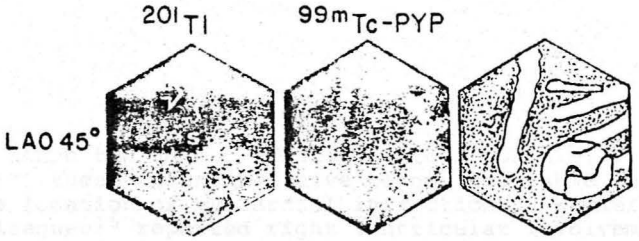


Figure 6. Thallium (Tl-201) and technetium stannous pyrophosphate (^{99m}Tc-PYP) scintigram accompanied by a line diagram of a patient with an inferior wall myocardial infarction with right ventricular involvement. (from Wackers, et al[15])

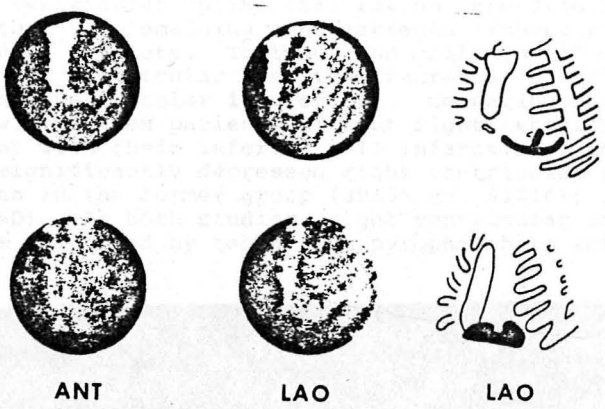


Figure 7. Technetium stannous pyrophosphate scintigrams of the patients with right ventricular myocardial infarctions. (from Sharpe, et al[17])

Since the advent of diagnostic myocardial scintigraphy [43,44], these techniques have become available for the precise location of myocardial infarctions. Wackers and his colleagues[15] reported right ventricular involvement identified by both technetium-99m stannous pyrophosphate uptake and by thallium-201 defects in twenty-four of seventy-eight consecutive patients studied with acute inferior left ventricular wall infarctions. Other studies[16,17] have used these techniques to diagnose right ventricular infarction. Although the sensitivity and specificity of both technetium pyrophosphate and thallium scanning are not known precisely with respect to the diagnosis of right ventricular myocardial infarction, there is no reason to suspect that they are any less sensitive or specific than in the diagnosis of left ventricular infarctions.

Sharpe, et al[17] have also looked at gated blood pool scintigraphy in patients with right ventricular infarctions. In their six of fifteen inferior infarction patients with right ventricular wall involvement, the right ventricular

to left ventricular uptake area ratios were significantly higher than the remaining nine patients without right ventricular infarcts. Tobinick and colleagues[16] calculated right ventricular ejection fractions in patients with right ventricular infarctions. Comparing seven patients with to ten patients without right ventricular involvement with their inferior wall infarctions, they showed significantly depressed right ventricular ejection fractions in the former group (39±5% vs. 51±10%, $p<0.001$, mean ± SD). In both studies, right ventricular infarction was diagnosed by technetium pyrophosphate scintigraphy.

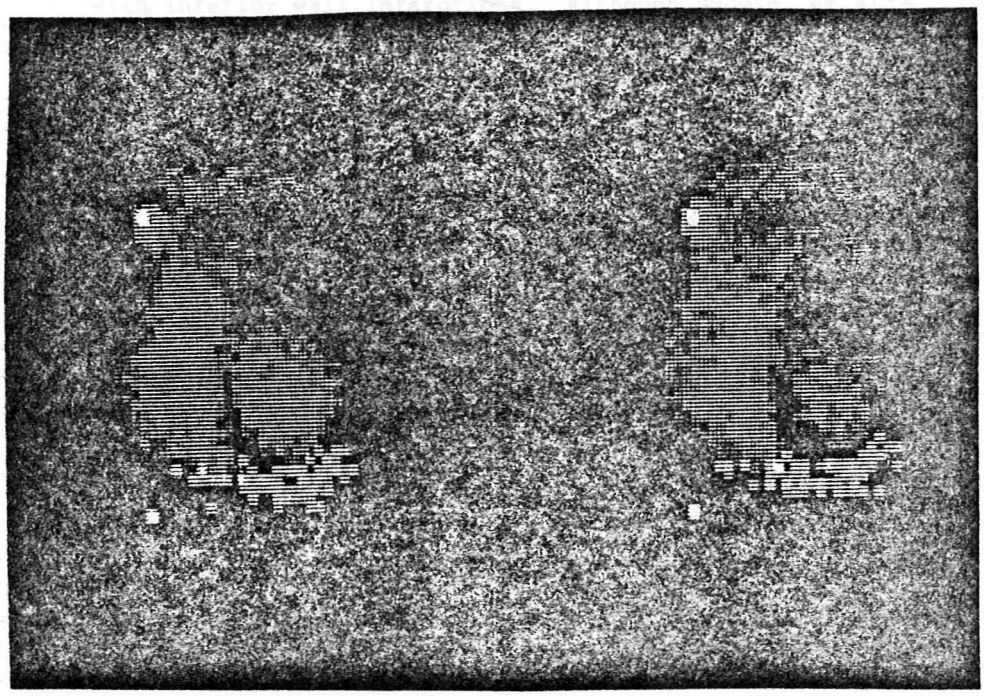


Figure 8. Simultaneous gated blood pool and technetium pyrophosphate scintigrams in diastole (left) and systole (right) of a patient with an inferior left and right ventricular infarctions. (from Corbett, et al[63])

Lorell's retrospective look[18] at twelve patients with right ventricular infarcts diagnosed by hemodynamic means revealed three patients who underwent multiple gated blood pool imaging, all three of whom had marked dilatation of the right ventricle with right ventricular apical hypokinesia.

Prior to this latter study, several groups had demonstrated the value of radionuclide ventriculography in the assessment of right ventricular performance following acute myocardial infarction. Rigo and colleagues[12] showed a higher incidence of right ventricular dysfunction in patients with inferior wall infarctions. Although Reduto, et al[45] showed a higher incidence of right ventricular dysfunction in patients with inferior left ventricular wall infarctions, they concluded that this only infrequently resulted in hemodynamic compromise. Furthermore, they did not distinguish those patients with right ventricular infarctions.

Echocardiography

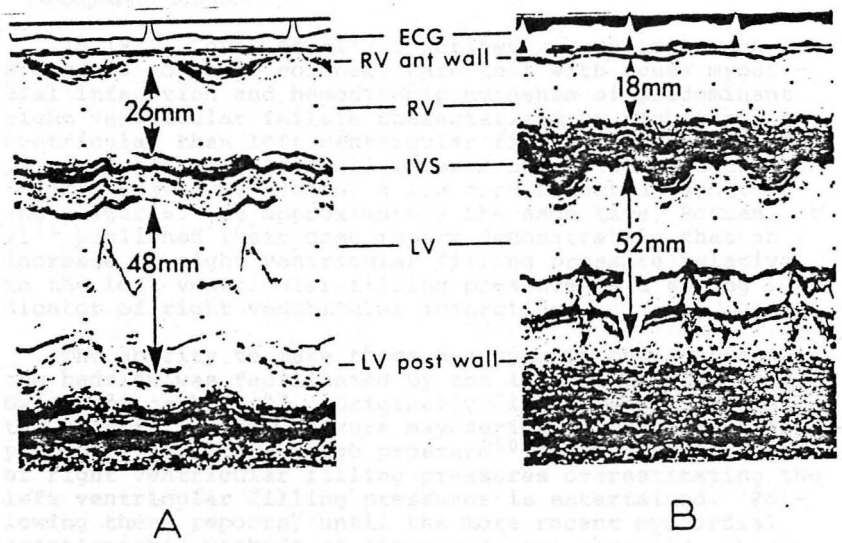


Figure 9. M-mode echocardiograms of a patient (A) with a right ventricular infarction, and a patient (B) with an inferior wall infarction. (from Sharpe, et al[17])

Both M-mode and two-dimensional echocardiography are known to be sensitive techniques for the demonstration of left ventricular dilatation and segmental left ventricular wall motion abnormalities after acute anterior and inferior myocardial infarctions[46,47]. Sharpe and colleagues[17] reported that following right ventricular myocardial infarction, in five of six patients the right ventricular dimension was increased, and compared to nine inferior infarction patients without right ventricular involvement, the mean right to left ventricular end-diastolic dimension ratio was significantly greater. These findings concurred with similar measurements obtained from dynamic scintigraphic studies on the same patients.

These echocardiographic observations have been subsequently confirmed in four of six patients in Lorell's study[18], and in case reports by Elkayam, et al[48], Clark, et al[32], Bansal, et al[33], and Raabe, et al[34].

Hemodynamic Studies

In 1974, Cohn, et al[10] described six patients who presented to their coronary care unit with acute myocardial infarction and hemodynamic evidence of predominant right ventricular failure characterized by higher right ventricular than left ventricular filling pressures. The patients had inferior wall infarctions, distended neck veins, clear lung fields, a low cardiac output syndrome and oliguria. At approximately the same time, Rotman, et al[11] published their case report demonstrating that an increase in right ventricular filling pressure relative to the left ventricular filling pressure is a strong indicator of right ventricular infarction.

The ability to make these hemodynamic observations at the bedside was facilitated by the introduction of the Swan-Ganz catheter[49]. Originally, it was reported that the central venous pressure may seriously underestimate pulmonary capillary wedge pressure[50]. Now, the paradox of right ventricular filling pressures overestimating the left ventricular filling pressures is entertained. Following these reports, until the more recent myocardial scintigraphic methods of diagnosis were reported, the diagnosis of right ventricular infarction was made by direct hemodynamic measurements.

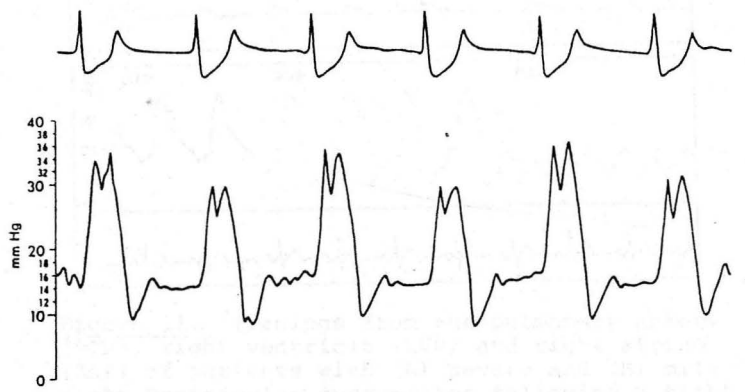


Figure 10. ECG (upper trace) and right ventricular pressure tracing of a patient with right ventricular infarction. (from Bansal, et al[33])

Figure 10 demonstrates the classical hemodynamic tracings in right ventricular infarction of sufficient severity to cause right ventricular hemodynamic compromise. The tracing shows elevation of the right atrial pressures with a mean pressure of 14 mm mercury, an elevated right ventricular end-diastolic pressure to the same level, and a right atrial mean pressure slightly greater than the mean pulmonary capillary wedge pressure. The right ventricular and pulmonary arterial systolic pressures are only minimally elevated. Furthermore, the right ventricular diastolic trace has an early diastolic dip followed by a plateau, the so-called square root sign characteristic of pericardial constriction[51]. I will return to this point when I discuss the differential diagnosis.

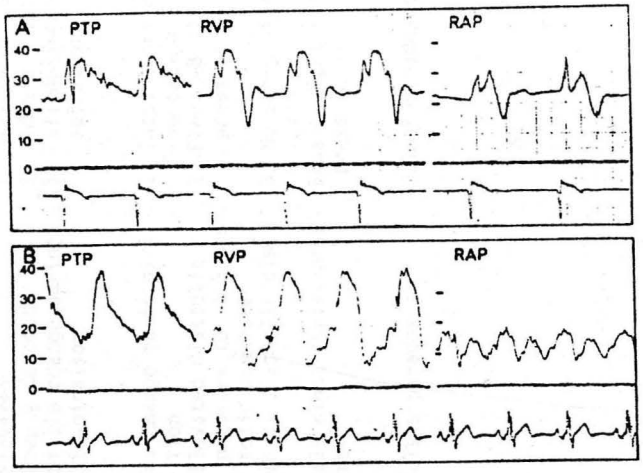


Figure 11. Tracings from the pulmonary artery (PTP), right ventricle (RVP) and right atrium (RAP) of patients with (A) severe and (B) mild right ventricular dysfunction following a right ventricular infarction. (from Coma-Canella and Lopez-Sendon[52])

Figure 11 permits a closer examination of the right atrial pressure tracing. This figure is taken from Coma-Canella's work[52] showing degrees of severity of right ventricular dysfunction following right ventricular infarction. The upper panel shows a severe and the lower panel a milder degree of dysfunction. The deep *y* descent which is also characteristic of pericardial constriction, is very obvious in the upper panel. These authors report that the deeper the *y* descent, the more severe the right ventricular dysfunction.

As the hemodynamic sequelae of pericardial disease may be masked by hypovolemia, so may those resulting from right ventricular infarction[53,34]. Raabe and Chester[34] describe a patient who was subsequently found at autopsy to have a large right ventricular infarction, in whom equalization of pressures through the right side of the heart and the development of an early diastolic dip in the right ventricular pressure tracing did not develop until after the administration of two litres of normal saline.

Table 6. Hemodynamics of right ventricular infarction, pericardial constriction and cardiac tamponade.

	Right ventricular infarction	Pericardial constriction	Cardiac tamponade
1. Right atrial (systemic venous) pressure	1. Elevated 2. Deep y descent 3. Little change during inspiration	1. Elevated 2. Deep y descent. 3. Little change during inspiration	1. Usually elevated 2. y descent absent 3. Fall during inspiration
2. Right ventricular pressure	1. Normal respiratory variation 2. Elevated diastolic pressure to >1/3 systolic pressure 3. Early diastolic dip	1. No change in inspiration 2. Elevated diastolic pressure to >1/3 systolic pressure 3. Early diastolic dip	1. Inspiratory augmentation 2. Elevated diastolic pressure 3. No early diastolic dip
3. Pulmonary capillary wedge pressure	Equal to or less than right atrial pressure	Equals right atrial pressure	Equals right atrial pressure
4. Arterial pressure	No pulsus paradoxus	± Pulsus paradoxus	Pulsus paradoxus

Differential Diagnosis

The clinical features and hemodynamic findings of right ventricular myocardial infarction closely resemble pericardial constriction. In the presence of a low cardiac output syndrome, the clinical features of elevated central venous pressure with or without Kussmaul's sign, a collapsing *y* descent of the jugular venous pulse, dyspnea with clear lung fields, and occasionally a pericardial friction rub are common to both conditions.

The hemodynamic findings in the two conditions are listed in Table 6 and compared to cardiac tamponade.

Awareness of the clinical diagnosis will reveal the typical electrocardiographic, enzymatic and scintigraphic changes of an acute myocardial infarction.

Treatment

The importance of diagnosing right ventricular myocardial infarction may lie in its management. Like cardiac tamponade, the use of diuretics in this setting may precipitate an impending, or worsen an already existing, low cardiac output state. In addition to the classical management of an acute myocardial infarction, the following forms of therapy have been advocated for right ventricular infarction, particularly those associated with a low output syndrome.

Volume expansion

Since Cohn, et al[10] reported the imbalance between right and left ventricular filling pressures, volume expansion has been advocated by several authors to improve or abolish the low cardiac output state[10,18,19,33,34,52,54-57]. Cohn and colleagues[10] pointed out that an increase in cardiac output by this therapeutic technique may only occur if the pressure gradient from the right to the left atrium could be increased, since without normal right ventricular contraction, flow from the right to the left side of the heart was accomplished passively across a low resistance pulmonary vascular bed. Unfortunately, if the left ventricle is also extensively damaged, volume expansion may be ineffective[55].

The volume of fluid administered during these studies varied greatly, but was essentially titrated against the left rather than the right ventricular filling pressure[19].

Inotropic Agents

Dopamine[18,19,33,57], isoproterenol[54], and dobutamine[32] have all been advocated as means of sustaining cardiac output in the low output syndrome complicating right ventricular infarction. All agents were used after volume expansion was unsuccessful implying marked left ventricular compromise in these patients, with variable results.

Unloading Agents

Since Cohn, et al[10] used nitroprusside successfully in a patient who had not responded to volume expansion, several authors have reported using unloading agents often in conjunction with volume expansion[13,34,52]. As Doctor Lipscomb has pointed out recently, therapy with unloading agents has been shown to improve left ventricular function and survival in patients with acute left ventricular infarctions[58,64]. Because these agents are known to have similar effects on both the pulmonary and systemic vascular beds, improvement in right as well as left ventricular function in the setting of right ventricular infarction would be expected[59,60].

Circulatory Support

Intra-aortic balloon pulsation has been advocated as a means of managing patients with right ventricular infarction who do not respond to volume expansion[18,55-57]. As with other advocated forms of therapy in this setting, the introduction and maintenance of circulatory support is dependent on the status of the left ventricle. The success of this intervention in cardiogenic shock is variable[61].

Prognosis

Because the initial clinical methods of diagnosing right ventricular infarction involved identifying the hemodynamic derangement found during Swan-Ganz catheterization for low cardiac output syndrome, it is understandable that the mortality rate in such patients was inordinately high, and certainly higher than the mortality associated with left ventricular myocardial infarction. The introduction of better diagnostic techniques for the diagnosis of right ventricular infarction, and in particular myocardial scintigraphic techniques, has corrected this imbalance. For example,

Sharpe, et al[17] and Tobinick, et al[16] reported no deaths in their clinical series of six and seven patients respectively. Wackers, et al[15] reported three deaths in twenty-four patients with right ventricular infarctions.

From all the pathological and clinical studies reviewed in this dissertation, it is clear that the conclusions of Guiha, et al[31] in their experimental studies that the outcome of a patient with right ventricular infarction is dependent upon the degree of left ventricular dysfunction due to the associated left ventricular infarct is correct. However, a comprehensive clinico-pathological review is still necessary to determine the precise prognosis of a patient with right ventricular infarction.

Summary

Perhaps the most important point concerning right ventricular myocardial infarction is to be alert for its occurrence. Approximately one-fifth of all infarctions and one-third of all inferior infarctions have some right ventricular involvement. All right ventricular infarcts are associated with inferior left ventricular infarctions.

When direct hemodynamic measurements are necessary in a patient with acute inferior infarction complicated by a low output state, the abnormal finding of higher right ventricular than left ventricular filling pressures should be associated with right ventricular infarction. Volume expansion is often beneficial in this setting, with the addition of an afterload reducing agent if necessary.

The prognosis following a right ventricular myocardial infarction would appear to be related to the degree of left ventricular dysfunction associated with the original infarction.

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