# Generalised Left Ventricular Dysfunction after Traumatic Right Coronary Artery - Right **Atrial Fistula**

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## SUMMARY

A patient with traumatic right coronary artery to right atrial fistula, which was repaired by direct closure and aortocoronary saphenous vein bypass grafting, is described. Cardiac catheterisation and selective cine angiocardiography were performed pre- and postoperatively, and left ventricular (LV) function was studied in detail by invasive and non-invasive techniques. There was regional (diaphragmatic) LV asynergy but also generalised impairment of myocardial function, and these abnormalities persisted after operation.

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Traumatic coronary fistulae are rare. To our knowledge 11 patients have been reported, but detailed studies of left ventricular function have not been made.1 Unexplained abnormality of left ventricular end-diastolic pressure (LVEDP) has been reported: there was no information about left ventriculography in these patients and it is uncertain whether there was global or regional myocardial dysfunction.2,3

This report describes a patient who developed a right coronary artery to right atrial fistula after a penetrating stab wound of the chest. Surgical management was undertaken: the fistula was closed directly and an aortocoronary saphenous vein bypass graft inserted. Detailed haemodynamic and cine-angiographic studies were made preand postoperatively to assess the pathophysiological effect of the fistula on cardiac function and left ventricular performance.

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#### CASE REPORT

A 27-year-old Black male was admitted to hospital after receiving a stab wound in the right side of the chest. He developed cardiac tamponade, and urgent thoracotomy was undertaken: a clot was evacuated from the pericardial cavity and a laceration of the atrioventricular groove repaired. The immediate postoperative period was uneventful but signs of cardiac failure persisted.

On physical examination the patient was not distressed, the pulse was 88/min and regular; blood pressure was 130/90 mmHg. The jugular venous pressure was increased to 8 cm with A and V waves, the liver was 2 cm enlarged and there was mild bilateral ankle oedema. The cardiac impulse was normal. On auscultation a continuous murmur was present in the third left interspace radiating over the precordium and an atrial sound was heard at the apex. The respiratory system was normal.

Serial ECGs showed evolution of diaphragmatic infarction with apical ischaemia. The mean frontal Pwave axis was +80° (Fig. 1). Chest X-ray examination showed left ventricular (LV) enlargement and pulmonary venous distension.

The time intervals of the cardiac cycle in systole were measured at the bedside on three occasions: the ratio PEP/ LVET was persistently increased and this indicated impaired left ventricular function (Table I).4



Fig. 1. ECG 7 days after thoracotomy for cardiac tamponade, showing recent diaphragmatic infarction with anterolateral ischaemia.

### TABLE I. HAEMODYNAMIC AND ANGIOGRAPHIC DATA

	Pre-operatively	Postoperatively
Heart rate		
(beats/min)	88	75
PEP/LVET	0,54	0,59 (1 mo.
		0,46 (4 mo.
RA mean pressure		
(mmHg)	5	10
RV systolic early-dia	28	30
	stolic 2	4
(mmrig) end-diast	olic 6	12
PA mean pressure		
(mmHg)	17	20
LV systolic early-dias	102	120
	stolic 2	4
(mmrg) end-diast	olic 12	16
Peak LVdp/dt		
(mmHg/sec)	966	1 208
Cardiac index		
(L/min/m <sup>2</sup> )	2,3	2,9
Stroke index		
(ml/beat/m <sup>2</sup> )	25	31
LV end-diastolic volun	ne	
(ml/m <sup>2</sup> ) (angiographi	c) 133	133
LV end-systolic volum	e	
(ml/m <sup>2</sup> ) (angiograph	ic) 77	76
Ejection fraction (%)	42	43

Cardiac catheterisation was undertaken after intensive therapy with digitalis, diuretics and bedrest. Right heart pressures were normal and LV pressure 102/2 - 12 mmHg with a peak dp/dt of 966 mmHg/sec (Table I). Cardiac index (2,3 litres/min/m<sup>2</sup>) and stroke index (25 ml/beat/m<sup>2</sup>) were slightly reduced (direct Fick method). Blood sampling showed an increase in oxygen saturation of 4% at midright atrial level and this indicated a 17% left-to-right



Fig. 2a. Indicator dilution curves at pre-operative cardiac catheterisation. Indocyanine green 5 mg was injected into ascending aorta and then right coronary artery, sampling in main pulmonary artery large left-to-(MPA). A right shunt is shown on RCA injection. A small occurs from the shunt ascending aorta. AT arrival time.

Fig. 2b. Indicator dilution curves 1 month after repair of the fistula. A small left-to-right shunt is shown when injection is made into RCA and also ascending aorta. shunt. Indicator dilution curves were performed. Indocyanine green (5 mg) was injected into ascending aorta, right coronary artery and then left coronary artery, sampling at a constant rate of 38 ml/min successively in superior vena cava, inferior vena cava, right atrium, right ventricle and main pulmonary artery: this showed a shunt from right coronary artery to right atrium (Fig. 2a).<sup>5</sup>

Selective left ventriculography showed an enlarged left ventricle with akinesis of a large part of the diaphragmatic surface. The remainder of the LV contracted poorly and ejection fraction was reduced (42%) (Fig. 3a, Table I).



Selective coronary angiography was performed and showed a dominant right coronary artery (RCA) with an aneurysm and fistula into the right atrium (RA) in its proximal third. The distal segment was not visualised from right coronary injection but was opacified by cross-filling after injection into the left coronary artery (LCA), which was normal (Figs 4a and 4b).

tion fraction (EF) reduced.

 $24 \pm 8 \text{ ml/m}^2$ ; EF = 67 ±

70 ±

(Normal EDV =

8%.)

20 ml/min/m<sup>2</sup>; ESV

Operation was performed through a vertical sternotomy. Total cardiopulmonary bypass was established and the right atrium opened. Arterial blood squirted into the atrial cavity through a 0,5-cm vertical incision in the RCA. The defect was closed with 2 Tycron sutures cushioned with Teflon pledges. The distal RCA was explored and incised: good flow was present. A saphenous vein graft was inserted at the site of exploration and proximal anastomosis made with the ascending aorta, using interrupted sutures.

The postoperative period was uneventful. The time intervals of the cardiac cycle remained abnormal on 8 successive days after operation: the ratio PEP/LVET was 0,50 at 24 hours after the procedure and then 0,50; 0,53; 0,55; 0,45; 0,48; 0,44 and 0,50 on the succeeding days (normal value 0,35  $\pm$  0,04). One month after operation



Fig. 4a. Selective right coronary arteriogram (left anterior oblique projection) showing a small aneurysm of the RCA. The arrow shows contrast passing through the fistula into the right atrium. The distal RCA is not visualised.



Fig. 4b. Selective left coronary arteriogram (left anterior oblique projection). There is cross-filling of the distal right coronary artery.

it was increased to 0,59 and after a further 3 months an abnormal value of 0,46 was still present.<sup>6</sup>

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Elective cardiac catheterisation was repeated 1 month after operation. The haemodynamics were essentially unchanged: RA mean pressure had increased to 10 mmHg and LVEDP to 16 mmHg. The difference may have been a consequence of technical error or a change in the patient's blood volume after cessation of diuretic therapy (Table I). Cardiac and stroke index were normal. There was no step-up in oxygen saturation across the RA, but indicator dilution studies showed a small residual shunt from RCA to RA (Fig. 2b). The left ventriculogram was unchanged: there was diaphragmatic asynergy and generalised hypokinesis of the remaining myocardium. Ejection fraction was 43% (Fig. 3b).

Selective coronary angiography showed a small aneurysm of the RCA and a small fistula into the RA. This distal RCA filled progradely and there was retrograde filling of the vein graft (Fig. 5). There was no cross-filling of the distal RCA from LCA injection.

Four months after operation the pulse rate was 75/min and systemic venous congestion had disappeared. The continuous murmur was no longer present, but a fourth sound was still audible at the apex. The patient was well and returned to work.



Fig. 5. Selective right coronary arteriogram (left anterior oblique projection) 1 month after repair of the fistula. There is an aneurysm of the right coronary artery after its proximal third and a thin jet of contrast passes into the right atrium. The distal RCA fills progradely and there is retrograde filling of the aortocoronary saphenous vein bypass graft.

#### DISCUSSION

Coronary artery-right atrial fistula causes a volume load of all four cardiac chambers. At the same time there is an important change in myocardial blood flow. In our patient the left-to-right shunt was small (17%), and the major effect was due to decreased perfusion of the territory

supplied by the distal RCA: diaphragmatic infarction occurred. Over-all left ventricular function was poor and peak LVdp/dt and ejection fraction greatly decreased. particularly in the presence of regional ventricular asynergy when increased contraction of the viable myocardium is expected."

The pathophysiological mechanism of the depressed left ventricular function is uncertain. The syndrome of 'coronary steal' is a possible explanation.<sup>1,8</sup> Pre-operative selective coronary angiography showed cross-filling of the distal segment of the RCA without prograde filling of the vessel beyond the site of the fistula. Cross-filling was not demonstrated after operation.

Operation was performed and the myocardium revascularised in an attempt to improve function of viable myocardium, although diaphragmatic infarction had occurred at the time of injury.9 Myocardial function was not improved, although the left-to-right shunt was vir-

tually abolished. It is possible that irreversible, generalised myocardial damage occurred at the time of cardiac tamponade, during the period of 'coronary steal' or during cardiopulmonary bypass, although the last possibility is unlikely; the surgical unit performs 300 open-heart procedures per year with 10% mortality and low morbidity. It is also possible that ventricular function had been abnormal before injury in an asymptomatic patient.

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