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CASE REPORTS

Right to Left Shunt, With Severe Hypoxemia, at the Atrial Level in a Patient With Hemodynamically Important Right Ventricular Infarction

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This report describes a patient with a massive right ventricular infarction, complicated by severe hypoxemia. Contrast echocardiography demonstrated a right to left shunt through a previously asymptomatic atrial septal defect. This phenomenon should be considered as a possible cause of hypoxemia in the presence of right ventricular infarction.

Arterial hypoxemia in acute myocardial infarction may be due to left ventricular failure with pulmonary congestion, pulmonary embolism or chronic lung disease. Hemodynamically important right ventricular infarction is characterized by right-sided heart failure, presenting the clinical picture of "venous hypertension with clear lung fields" (1). Hypoxemia is not a typical symptom. We recently observed severe hypoxemia due to right to left shunting through an ostium secundum atrial septal defect in a patient with acute right ventricular infarction.

Case Report

A 61 year old woman without previously known heart disease was admitted to our coronary care unit 3 hours after the onset of acute constrictive, retrosternal pain that radiated to the left shoulder and was accompanied by nausea and profuse diaphoresis. On admission she looked ill. Her mental state was normal. The skin was warm and dry, without cyanosis. Blood pressure was 150/80 mm Hg. The pulse rate was 50/min. Slight jugular venous distension was noted, but no ankle edema was present. The heart was not enlarged, and had soft heart sounds; there were no gallop sounds or murmurs. The lungs were clear. Liver and spleen were not palpable.

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The electrocardiogram (Fig. 1) showed sinus rhythm with 2:1 atrioventricular block and acute inferior wall infarction, with ST segment elevation in the right precordial leads, suggestive of right ventricular infarction. A chest roentgenogram showed no cardiomegaly or signs of pulmonary congestion.

Hemodynamics and treatment. A Swan-Ganz thermodilution catheter was positioned in a pulmonary artery through a Cordis introducer placed in the right subclavian vein. Increased right atrial pressure with normal pulmonary capillary wedge pressure constituted proof of hemodynamically important right ventricular infarction (Table 1). Thermodilution cardiac output was low (3.0 liters/min) with low mixed venous oxygen saturation and arterial hypoxemia. No atropine or isoprenaline was given; instead, VVI pacing was started and the patient was treated with ouabain (0.875 mg) because of right-sided heart failure, heparin (15,000 U/24 h), prophylactic lidocaine (3 mg/min), intravenous isosorbide dinitrate (10 mg/h), plasma expanders and nasal oxygen (6 liters/min). Hemodynamic variables remained unchanged. Urine production was adequate and no sign of left ventricular failure developed. However, despite increasing oxygen supply, arterial oxygen saturation did not improve. Blood sugar was high and the patient received repeated injections of insulin. A decrease in hemoglobin concentration and benzidine-positive stools suggested gastric bleeding and cimetidine was administered intravenously (50 mg/h).

Right to left shunting. Because of confusion and worsening arterial hypoxemia the patient was intubated and ventilated with 100% oxygen, without improvement of arterial oxygen saturation. Right to left shunting was suspected.

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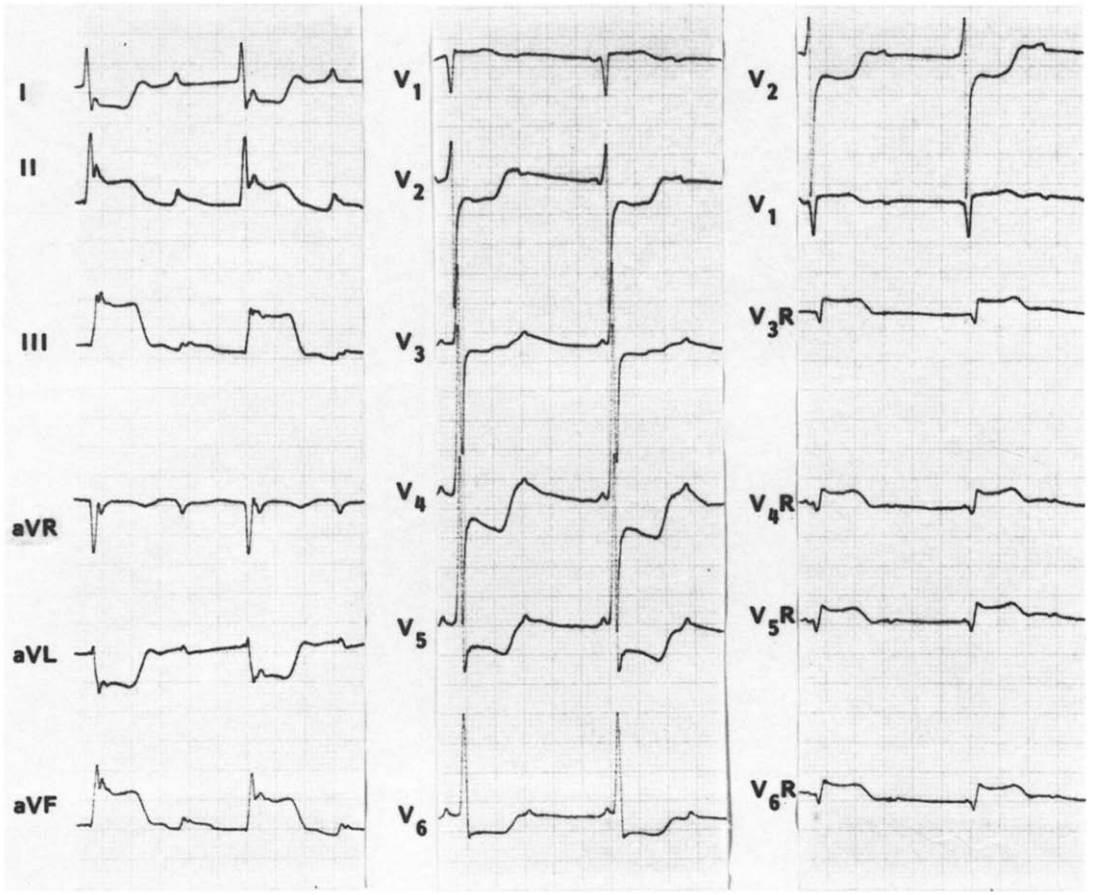

Figure 1. Admission electrocardiogram showing the 12 standard leads and right precordial leads (V_3R–V_6R), showing 2:1 atrioventricular block and acute diaphragmatic infarction with evidence of right ventricular infarction in the right precordial leads (2).

Contrast echocardiography (Fig. 2), by rapid injection of a 10 ml bolus of glucose 5% in water through the side arm of the Cordis introducer into the right subclavian vein, showed massive passage of ultrasonic contrast medium through an atrial septal defect. The balloon of a Swan-Ganz catheter was introduced through the atrial septal defect in an attempt to close the shunt. However, the patient remained comatose, the pupils became dilated and nonreactive to light. The patient was declared neurologically dead 70 hours after the first symptoms of myocardial infarction.

Autopsy. The dominant right coronary artery was occluded 2 cm beyond its origin, with an acute infarction of

Table 1. Time Course of Relevant Hemodynamic Measurements

Time After Infarction (h)	PCWP (mm Hg)	RAP (mm Hg)	MVO_2 Sat (%)	Art Po_2 (mm Hg)	Art O_2 Sat (%)	Oxygen Therapy (liters/min)
4	14	15	55	48	77	0
16	10	15	63	57	79	2
28	13	16	40	50	81	6
52	11	13	47	47	77	6

Art O_2 sat = arterial oxygen saturation; Art Po_2 = arterial partial pressure of oxygen; MVO_2 Sat = mixed venous oxygen saturation, PCWP = pulmonary capillary wedge pressure; RAP = right atrial pressure.

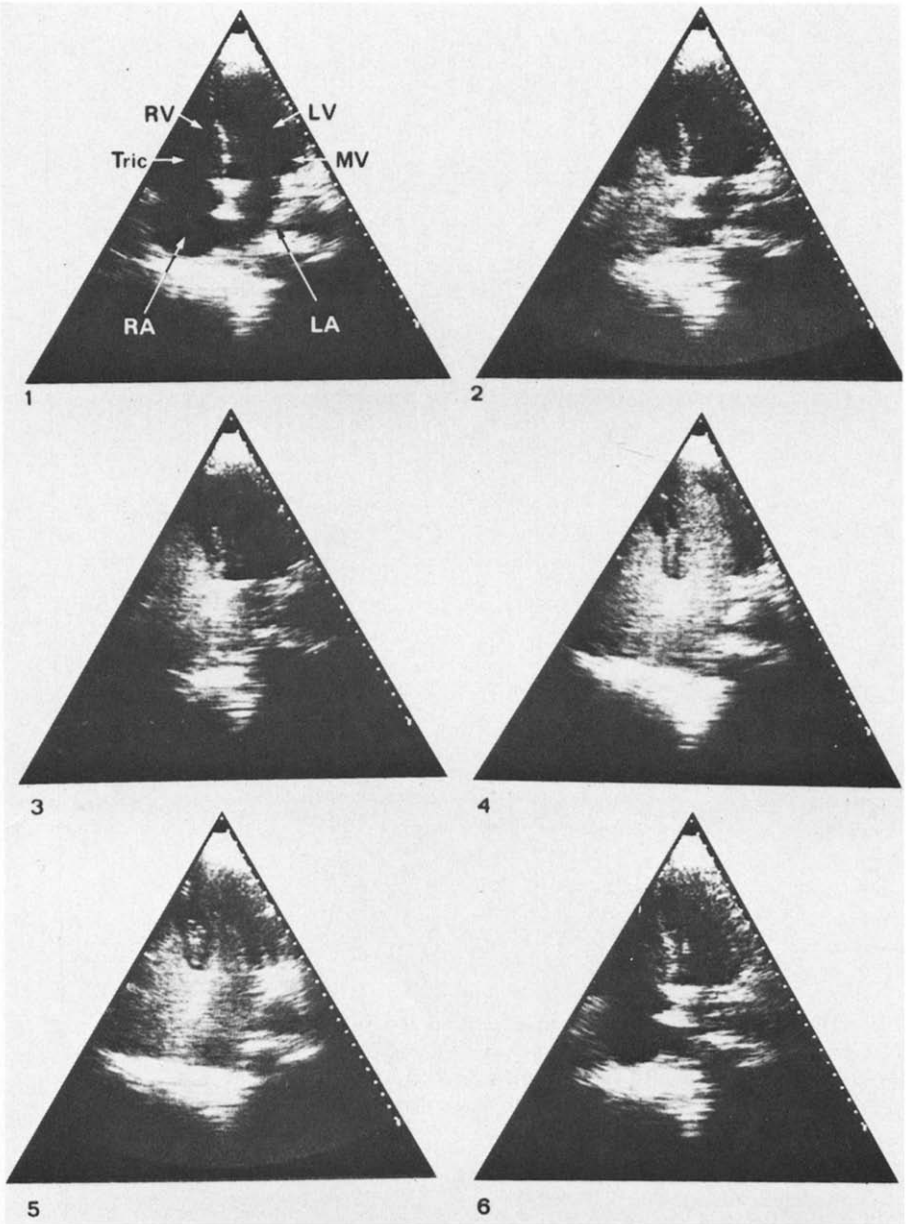

Figure 2. Frame by frame analysis of a continuous recording of an apical four chamber view of the heart during contrast two-dimensional echocardiography. From **top left**: **1**) before contrast injection followed by appearance of ultrasonic contrast medium in the right atrium (RA) **2**), then in the left atrium (LA) **3**), followed by filling of the right ventricle (RV) and the left atrium (LA) **4**), then contrast medium in both ventricles **5**), and finally emptying of the heart **6**), five frames later. LV = left ventricle; MV = mitral valve; Tric = tricuspid valve.

the entire right ventricle, extending into the inferior wall of the left ventricle. An ostium secundum atrial septal defect was found (Fig. 3). Liver and spleen were severely congested. The lungs showed no embolism or pneumonia. A gastric ulcer was observed. The brain was edematous.

This patient was admitted with acute right ventricular infarction extending into the left ventricle and complicated by right to left shunting at the atrial level. This type of shunting has been attributed to pulmonary stenosis, pulmonary hypertension associated with pulmonary embolism, positive end-expiratory pressure ventilation and severe chronic obstructive pulmonary disease (3). It has also been observed after right pneumonectomy (4), even in the absence of pulmonary or right atrial hypertension (5).

Right to left atrial shunting in acute myocardial infarction. Autopsy findings in 236 patients with acute myocardial infarction (6) revealed that 14% of patients with left ventricular infarction showed involvement of the right ventricle (24% of all diaphragmatic infarcts and none of the anterior infarcts). Clinically the syndrome of predominant right ventricular infarction may be suspected when right atrial pressure equals or exceeds the simultaneously measured pulmonary capillary wedge pressure, especially in the presence of inferior infarction (7). The difference between right and left atrial pressures introduces the possibility of right to left shunting at the atrial level whenever there is a coexisting atrial septal defect. This defect is not an absolute prerequisite, because a patent foramen ovale also constitutes

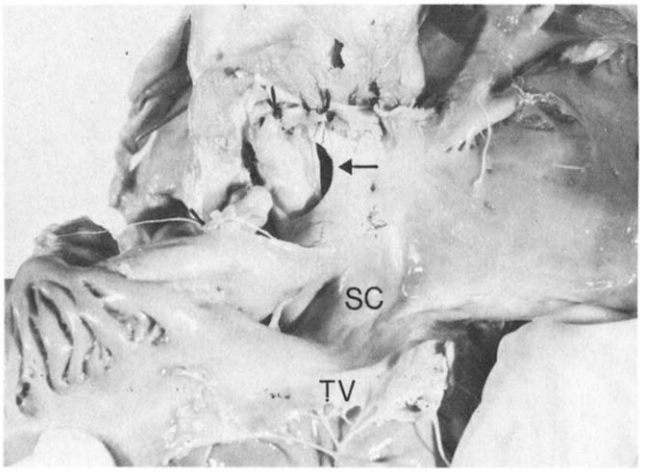

Figure 3. The opened right atrium at autopsy showing the atrial septal defect (**arrow**); note how the septum secundum seems too "short" to close the ostium secundum. SC = coronary sinus; TV = tricuspid valve.

a potential site for right to left shunting, with a corresponding degree of hypoxemia. Patency or incomplete seal of the foramen ovale occurs in 29 to 35% of adults (8). Its presence usually remains undetected, unless a paradoxical embolism occurs.

Diagnosis. In patients with an acute diaphragmatic infarction and arterial hypoxemia, right to left shunting at the atrial level must be suspected, particularly when pressure recordings suggest a hemodynamically important right ventricular infarction. Contrast echocardiography at the bedside will rapidly establish the diagnosis.

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