Optimal value of filling pressure in the right side of the heart in acute right ventricular infarction

Sali Berisha, Adnan Kastrati, Artan Goda, Ylli Popa

Abstract

Haemodynamic monitoring was performed within the first 48 hours after the onset of symptoms in basal conditions, during volume loading, and during infusion of glyceryl trinitrate in 41 patients who fulfilled the diagnostic electrocardiographic and haemodynamic criteria of right ventricular infarction. In most patients an increase of mean right atrial pressure up to 10–14 mm Hg was followed by an increase in right ventricular stroke work index. But raising the mean right atrial pressure above 14 mm Hg was almost always accompanied by a reduction in right ventricular stroke work index. When the mean right atrial pressure was reduced by intravenous glyceryl trinitrate to <14 mm Hg the right ventricular stroke index increased. The same response was seen with cardiac and stroke index. The mean (SD) values of optimal right atrial and pulmonary capillary pressures were 11.7 (2.1) and 16.5 (2.7) mm Hg respectively.

Thus cardiac and stroke index increased and the right ventricle reached its maximum stroke work index when the filling pressure was 10–14 mm Hg. These values may be regarded as the optimal level of right ventricular filling pressure in patients with right ventricular infarction.

The ventricular filling pressure in patients with acute myocardial infarction has been the subject of a series of haemodynamic studies. The optimal value of left ventricular filling pressure during acute myocardial infarction has been known for many years. Studies on optimal filling pressure of the right ventricle in patients with right ventricular infarction are, however, few and not conclusive. In fact none of them true incidence of right ventricular infarction in the patients who were studied was not known. Others have studied only one subgroup of patients with right ventricular infarction and a cardiac index of <2.2 l/min/m², whereas the range of values in this syndrome is wide.

This study was designed to establish the optimal value of right ventricular filling pressure in patients with right ventricular infarction.

Patients and methods

Forty one (36 men and five women, mean (SD) age 56.3 (7.4) of the 106 patients admitted to the coronary care unit between February 1986 and March 1988 with a first acute inferior or posteroinferior myocardial infarction fulfilled the diagnostic electrocardiographic and haemodynamic criteria of right ventricular infarction and were the subject of this study. The diagnosis of inferior myocardial infarction was established by the presence of: (a) a positive clinical history; (b) a diagnostic increase in serum enzymes and (c) abnormal Q waves in leads II, III, and aVF.

The diagnosis of right ventricular infarction was established by the presence of the following electrocardiographic and haemodynamic findings: (a) ST segment elevation ≥1 mm in leads V3R–V4R or (b) QS or QR waves or both in the same leads; (c) mean right atrial pressure >10 mm Hg and greater than, equal to, or not more than 5 mm Hg lower than pulmonary capillary pressure (in basal conditions or after volume loading). We chose these criteria because they have a diagnostic specificity of 95–100% and a high sensitivity. We excluded from the study all the patients with a difference of >5 mm Hg between pulmonary capillary pressure and pulmonary artery diastolic pressure; those with previous myocardial infarction or other diseases such as valvar heart disease, pericarditis, pulmonary embolism, left to right shunt, chronic heart failure, and renal insufficiency; and all the patients with a pulmonary capillary pressure >20 mm Hg.

HAEMODYNAMIC VARIABLES

After informed consent was obtained from the patient a Swan–Ganz thermodilution catheter was placed in the pulmonary artery within the first 48 hours after the onset of symptoms. Right atrial, right ventricular, and pulmonary artery pressures were recorded with a Bentley Trantec Model 800 transducer on a CRG 1000 apparatus. Cardiac output was determined by thermodilution by an Edwards 9520A computer and the average value of three measurements showing a difference of <10% was calculated. Heart rate was derived from the electrocardiographic recordings. Arterial pressure was measured with a sphygmomanometer. The haemodynamic indices were calculated according to the following formulas: CI = CO/BSA (where CI is cardiac index in l/min/m², CO is cardiac output in l/min, and BSA is body surface area in m²); SI = CI/HR × 1000 (where SI is stroke index in ml/beat/m² and HR is heart rate in beats/min); RVSWI = SI × (MPAP – MRAP) ×
Optimal value between Figure stroke work right atrial pressure

The indicate pressures (R \textsuperscript{VSWI}) in (MRAP) ventricular is at highest.

 Relation ventricular of filling range (mean patients which pressure in the 0.0136 pulmonary artery pressure is 0.0136 is mean stroke work left ventricular is control. After resistance in MAP resistance). CO x x

pulmonary capillary pressure started volume that so was only pressure and cardiac output a action of may be increased instead of volume that did complicated by because ventricular function inferior increased by an increase of mean right atrial pressure up to 10–14 mm Hg was followed by an increase in right ventricular stroke work index. But if the mean right atrial pressure exceeded 14 mm Hg right ventricular stroke work index almost always fell. Subsequent reduction of a mean right atrial pressure to <14 mm Hg by intravenous glyceryl trinitrate was followed by an increase in right ventricular stroke work index.

VOLUME LOADING

After control measurements were made a rapid intravenous infusion of Dextran was started at a rate of 200 ml/5 min and the pulmonary capillary pressure was monitored so that it did not exceed 24 mm Hg. The total volume given to each patient varied from 200 to 1200 ml (mean 500 ml). Measurements of pressure and cardiac output were repeated only when there was a change of >3 mm Hg in right atrial pressure. The change between two consecutive values of right atrial pressure was >5 mm Hg in 75% of patients and it reached a mean of 6.28 mm Hg for the group.

We used intravenous glyceryl trinitrate instead of frusemid\textsuperscript{1} to lower the abnormally raised filling pressures. This was because the action of frusemid is not as easily controlled and may be deleterious in patients with inferior myocardial infarction complicated by shock and increased right atrial pressure\textsuperscript{2} and because ventricular function curves can be constructed by reducing ventricular filling pressure with glyceryl trinitrate.\textsuperscript{9} Ventricular function curves were plotted for each patient to relate ventricular filling pressure to stroke work index, stroke index, or cardiac index. We used mean right atrial pressure as the right ventricular filling pressure because it was more stable and more easily measured.\textsuperscript{10}

STATISTICAL ANALYSIS

We used a two tailed t test for paired observations to compare differences between means. p values of <0.05 were regarded as statistically significant. All values are expressed as mean (1 SD).

Results

Figure 1 shows the relation between right ventricular stroke work index and mean right atrial pressure in all patients. In most the increase of mean right atrial pressure up to 10–14 mm Hg was followed by an increase in right ventricular stroke work index. But if the mean right atrial pressure exceeded 14 mm Hg right ventricular stroke work index almost always fell. Subsequent reduction of a mean right atrial pressure to <14 mm Hg by intravenous glyceryl trinitrate was followed by an increase in right ventricular stroke work index.

The mean value of optimal right atrial pressure in the group (chosen in each patient as the value of mean right atrial pressure corresponding to the maximum right ventricular stroke work index) was 11.7 (2.1) mm Hg (fig 2). The mean value of optimal pulmonary capillary pressure (chosen in each patient as the value corresponding to the maximum left ventricular stroke work index) was 16.5 (2.7) mm Hg (fig 3). In 16 patients there was a descending left

0.0136 (where RVSWI is right ventricular stroke work index in g.m.m\textsuperscript{-2}, MPAP is mean pulmonary artery pressure in mm Hg, MRAP is mean right atrial pressure in mm Hg, and 0.0136 is a conversion factor); LVSVII = SI x (MAP – PCP) x 0.0136, where LVSVII is left ventricular stroke work index in g.m.m\textsuperscript{-2}, MAP is mean arterial pressure, and PCP is pulmonary capillary pressure in mm Hg); SVR = (MAP – MRAP)/CO x 80 (where SVR is systemic vascular resistance in dyn.s.cm\textsuperscript{-5} and 80 is a conversion factor); PVR = (MPAP – PCP)/CO x 80 (where PVR is pulmonary vascular resistance).

Figure 1 Relation between right ventricular stroke work index (RVSVI) and mean right atrial pressure (MRAP) in 41 patients with right ventricular infarction. The bars indicate the range of right atrial pressures at which right ventricular stroke work index is highest.
Figure 2  Optimal mean right atrial pressure (MRAP) in 41 patients with right ventricular infarction. Open circles, basal conditions; closed circles, after volume loading. Mean (1 SD) is also shown.

![Graph showing MRAP vs MRAP](image)

Figure 4  Right ventricular stroke work index (RVSWI) at different mean values of right atrial pressure (MRAP) for subgroups 1 to 5. See text for explanations.

![Graph showing RVSWI vs MRAP](image)

ventricular function curve and an ascending right ventricular one.

To show more clearly that 10–14 mm Hg is the optimal value of right atrial pressure, we selected pairs of points representing the relation between right ventricular stroke work index (and stroke index and cardiac index) and mean right atrial pressure that corresponded to two different values of right atrial pressure obtained consecutively by dextran or glyceryl trinitrate infusion. These pairs were divided into five subgroups.

Subgroup 1 included all those pairs in which the first point corresponded to a mean right atrial pressure of < 10 mm Hg and the second point to a mean right atrial pressure ≤ 14 mm Hg.

Subgroup 2 contained all those pairs in which the first point corresponded to a mean right atrial pressure < 10 mm Hg and the second point to a mean right atrial pressure > 14 mm Hg.

Subgroup 3 included all those pairs in which both points corresponded to a mean right atrial pressure from 10–14 mm Hg.

Subgroup 4 included all those pairs in which the first point corresponded to a mean right atrial pressure ≥ 10 mm Hg and the second point to a mean right atrial pressure > 14 mm Hg.

Subgroup 5 contained all those pairs in which the first point corresponded to a mean right atrial pressure > 14 mm Hg and the second point to a lower mean right atrial pressure obtained by infusion of glyceryl trinitrate.

In subgroup 1 the increase in mean right atrial pressure from 6·5 (2·1) to 11·5 (1·7) mm Hg (p < 0·001) was accompanied by...
a significant rise in right ventricular stroke work index from 4.9 (2.1) to 6.8 (2.7) g.m.m⁻² (p < 0.001, fig 4) and in stroke index from 40.0 (9.9) to 45.2 (10.4) ml/beat/m² (p < 0.001, fig 5).

In subgroup 2 the rise of mean right atrial pressure from 7.0 (1.8) to 16.0 (1.4) mm Hg (p = 0.009) was followed by an insignificant change of right ventricular stroke work index from 7.3 (3.1) to 6.5 (2.9) g.m.m⁻² (fig 4) and of stroke index from 37.9 (12.5) to 39.5 (9.8) ml/beat/m² (fig 5).

In subgroup 3 mean right atrial pressure increased from 10.3 (0.5) to 13.7 (0.5) mm Hg (p < 0.001), while neither ventricular stroke work index (5.0 (2.5) v 4.7 (2.3) g.m.m⁻², fig 4) nor stroke index (40.1 (14.5) v 38.3 (8.8) ml/beat/m², fig 5) changed significantly.

In subgroup 4 the increase in mean right atrial pressure from 11.4 (1.3) to 16.7 (1.4) mm Hg (p < 0.001) was accompanied by a significant reduction of right ventricular stroke work index from 5.0 (2.3) to 4.0 (2.5) g.m.m⁻² (p = 0.007, fig 4) and of stroke index from 38.4 (13.1) to 34.7 (13.5) ml/beat/m² (p = 0.002, fig 5).

In subgroup 5 the reduction in mean right atrial pressure from 17.8 (1.0) to 13.3 (1.9) mm Hg (p = 0.002) produced by intravenous glyceryl trinitrate was followed by a significant increase in right ventricular stroke work index from 3.8 (2.3) to 5.1 (2.1) g.m.m⁻² (p = 0.013, fig 4) and in stroke index from 39.1 (9.8) to 45.0 (8.3) ml/beat/m² (p < 0.001, fig 5).

The changes in cardiac index paralleled those for stroke index (fig 6).

Discussion

The filling pressure is a major determinant of ventricular performance in intact13 and diseased hearts.14,15 This pressure is of critical importance in evaluating cardiac performance in patients with acute myocardial infarction.14

Haemodynamic, clinical, and experimental studies13,16 showed that the increase in filling pressure of the infarcted left ventricle up to the optimal level of 14–18 mm Hg in some studies and of 20–25 mm Hg in others was followed by a parallel rise in cardiac output, stroke index, and left ventricular stroke work index. But data about the optimal filling pressure of the right ventricle in patients with right ventricular infarction are few and inconclusive.14

Some regarded as optimal mean right atrial pressures of 6–8 mm Hg, but in their patients the real incidence of right ventricular infarction was unknown. For others the optimal value was 15.6 mm Hg, but only in a subgroup of patients with cardiac index of <2.21/min/m². The range of haemodynamic function in patients with right ventricular infarction is much wider.

When we plotted ventricular function curves for right ventricular stroke work index (as a more specific indicator of the function of this ventricle) against mean right atrial pressure (fig 1) we found that an increase of mean right atrial pressure up to 10–14 mm Hg was almost always followed by a rise in right ventricular stroke work index, whereas increases in mean right atrial pressure to above these values resulted in a level or even descending line. When mean right atrial pressure returned to 10–14 mm Hg after treatment with glyceryl trinitrate right ventricular stroke work generally increased. The mean value of optimal right atrial pressure of the group, selected in each patient as the value of mean right atrial pressure corresponding to the highest right ventricular stroke work index, was found to be 11.7 (2.1) mm Hg (fig 2). This finding was supported by the analysis of pairs of points representing the relation between right ventricular stroke work index (and stroke index and cardiac index) and mean right atrial pressure that corresponded with the two different values of right atrial pressure produced by infusions of dextran or glyceryl trinitrate (figs 4–6). This analysis showed that the right ventricular stroke work index increased only in
subgroups 1 and 5. In subgroup 1, the rise of mean right atrial pressure from 6-9 (2-1) to 11-5 (1-7) mm Hg was followed by a considerable increase in right ventricular stroke work index and stroke index. In subgroup 5, too, right ventricular stroke work index and stroke index increased significantly when mean right atrial pressure was reduced from 17-8 (1-0) to 13-3 (1-9) mm Hg by infusion of glyceryl trinitrate.

The improvement in right ventricular performance at optimal filling pressure is a result of the stretching to their optimal length of the myocardial fibres that have survived necrosis; this, according to Starling's law, increases their contraction.

In subgroup 4, an increase in mean right atrial pressure from 11-4 (1-3) to 16-7 (1-4) mm Hg led to a significant decrease in the performance of the right ventricle. Others, too, have reported no improvement in right ventricular performance in patients with predominant right ventricular infarction when right atrial pressure is abnormally raised. The increase of right ventricular filling pressure above the optimal values reduces right ventricular performance because it augments the wall stress and the rigidity of the thin-walled right ventricle and affects left ventricular geometry and interventricular septal motion. For these changes that derive from ventricular interdependence the intact pericardium will also have constractive effects on the dilated heart.

We found that the optimal pulmonary capillary pressure was 16-8 (2-7) mm Hg (fig 3), nearly the same as that found in patients with left ventricular infarction. In 16 patients, however, the left ventricular function curves took the opposite course to those of the right ventricle. Others too have described patients in shock owing to right ventricular infarction who showed values of left ventricular filling pressure within optimal limits. These data clearly indicate that to maintain adequate cardiac performance in patients with biventricular infarction, both the left and right ventricular filling pressures need to be optimal.

Our study showed that the control of right ventricular filling pressure in patients with biventricular infarction enables the regulation to some degree of right ventricular performance according to Starling's law. Cardiac and stroke index increased and the right ventricle reached its maximum stroke work index when the filling pressure was 10-14 mm Hg. These values could, therefore, be considered as the optimal value of right ventricular filling pressure in patients with right ventricular infarction, though there were individual variations.

15 Rahimtoola SH. Hemodynamics in myocardial infarction. Am J Cardiol 1974;33:691.
Optimal value of filling pressure in the right side of the heart in acute right ventricular infarction.

S Berisha, A Kastrati, A Goda and Y Popa

*Br Heart J* 1990 63: 98-102
doi: 10.1136/hrt.63.2.98

Updated information and services can be found at:
http://heart.bmj.com/content/63/2/98

*These include:*

**Email alerting service**
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes