High-Rate Cardiac Pacing Increases Blood Pressure and Decreases Right Atrial Pressure in Patients with Hemodynamically Significant Acute Right Ventricular Myocardial Infarction and Bradyarrhythmia

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Summary

Background: In an 84-year-old patient with acute right ventricular myocardial infarction (RVI), complete heart block, and low cardiac output, a significant increase in blood pressure (BP) and decrease in right atrial pressure (RAP) were accidentally observed during the performance of high-rate ventricular pacing.

Methods: Based on that observation, the acute effects of high-rate cardiac pacing (VVI or AAI) on BP and RAP were studied in 15 consecutive patients (67.4 ± 7.7 years), with hemodynamically significant RVI. Ten had advanced heart block and five had sinus bradycardia. Cardiac pacing with simultaneous recording of BP and RAP at intrinsic rhythm and at heart rates of 70, 90, 110, 130 beats/min was performed.

Results: Systolic BP (SBP) increased significantly from 94.6 ± 15 mmHg during intrinsic rhythm to 101.9 ± 13.8 mmHg– 127 ± 12.2 mmHg at heart rates 70–130 beats/min ($p < 0.0001$). Diastolic BP (DBP) also increased from 48.2 ± 8.7 to 53.9 ± 3.7 – 69.1 ± 3 mmHg at heart rates 70–130 beats/min, ($p < 0.014$ – 0.0001). Mean RAP decreased from 14.5 ± 5 to 14.1 ± 5 mmHg– 11.1 ± 4.1 mmHg at heart rates 70–130 beats/min ($p = 0.16$ – 0.0001). Significant elevation of SBP ($p < 0.007$), DBP ($p < 0.0075$), and decrease of RAP ($p < 0.038$) were also detected by comparing the usual pacing rate at 70 beats/min with pacing rates at 90–130 beats/min.

Conclusions: These findings, if demonstrated over a prolonged period during the acute state of RVI, may influence the

management of patients with RVI to include high-rate cardiac pacing, probably in the range of 80–110 beats/min.

Key words: right ventricular infarction, cardiac pacing, cardiogenic shock, noncompliant right ventricle

Introduction

Acute right ventricular infarction (RVI) is a condition frequently associated with acute inferior-posterior myocardial infarction. The clinical, hemodynamic, and electrocardiographic (ECG) signs of that condition are of considerable interest, since RVI frequently presents with low cardiac output state and even gross cardiogenic shock.^{1–6}

Inferior acute myocardial infarction (AMI) is frequently accompanied by bradyarrhythmia and usually needs temporary heart pacing.^{1,7,8}

The management of hemodynamically significant RVI requires, apart from standard therapeutic measures, temporary heart pacing in the case of bradyarrhythmia and detailed hemodynamic assessment by right heart catheterization in order to preload the left ventricle sufficiently (by massive fluids administration).^{1,2–4,7}

During atrial synchronous ventricular pacing (VDD) using transesophageal atrial wave sensing in an 84-year-old patient with acute inferior AMI associated with RVI, complete heart block (CHB), and cardiogenic shock,⁹ we happened to observe that BP, which was increased by the restoration of atrioventricular sequential contraction, remained high when the pacing mode changed from VDD to VVI at the sinus rate (115 beats/min) (Fig. 1). This was also observed when the basic rhythm changed to atrial fibrillation while the patient was still in CHB.

These unexpected observations led us to study prospectively the acute effects on BP and RAP of high-rate cardiac pacing in patients with hemodynamically significant RVI and advanced heart block (greater than Mobitz II) or sinus bradycardia.

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FIG. 1 Right arterial pressure (RAP) and electrocardiographic (ECG) lead I in intrinsic rhythm are shown for Patient No. 1 (recordings made with the central station recorder). Note the prominent descending y wave of the RAP waveform. (B) and (C) Tracings of ECG lead I with blood pressure (BP) is illustrated. Note the BP elevation during VDD pacing (B) and the virtually same BP elevation during VVI pacing from VVI at 60 to 115 beats/min (C). An increase in BP waveform width is also apparent (arrowheads). The arrows indicate the time of VDD (by transesophageal atrial wave sensing) and VVI pacing. The instability of BP tracing during VDD pacing is due to unstable transesophageal atrial wave sensing. Paper speed at 25 and 1 mm/s. CHB = complete heart block, SR = intrinsic rhythm.

Methods

Fifteen consecutive patients (11 men, 4 women, aged 67.4 ± 7.7 years), with clinical, ECG, and hemodynamic characteristics of significant RVI and bradyarrhythmia were studied. Ten patients had advanced heart block and five had sinus bradycardia.

The diagnosis of RVI was confirmed by using the following criteria:

1. Presence of inferior-posterior AMI
2. Mean RAP ≥ 2 mmHg from mean pulmonary capillary wedge pressure (PCWP)^{1,5,7,8}
3. Pattern of poor compliant right ventricle, that is, RAP waveform with prominent descending y wave^{1,2,5,10} (Figs. 1, 2).

4. ECG signs of RVI in right precordial leads, namely, ST elevation in V_3R-V_5R and QS or QR pattern^{1,11-14}

All patients received standard treatment, including thrombolysis if indicated.

Massive fluids, if indicated, were given after the completion of this study (itself of short duration), in order not to interfere with the hemodynamic profiles of the patients.

The study protocol included:

1. Continuous invasive radial artery blood pressure recording
2. Subclavian vein transcutaneous catheterization, using standard technique and insertion of a Swan-Ganz catheter, to confirm the hemodynamic profile of RVI by measuring RAP and PCWP

FIG. 2 Top to bottom: Electrocardiogram, blood pressure (BP), and right atrial pressure (RAP) registrations of Patient No. 11 with right ventricular myocardial infarction (RVI) and sinus bradycardia. The increase in BP and the decrease in RAP are apparent. Note the gradual elevation of BP as the AAI pacing rate increases from 56 to 110 beats/min (arrowheads). The poor compliance pattern of RVI is indicated by a prominent descending y wave of the RAP waveform. An increase in BP waveform width is also apparent. Paper speed at 25 and 1 mm/sec.

3. Insertion of a temporary bipolar pacing electrode from the same sheath in the right ventricular apex in patients with advanced heart block and in the high right atrium in patients with RVI and sinus bradycardia
4. Continuous recording of RAP and BP in a three-channel thermal paper recorder (Hellige SMV 107); recordings were made during pacing (either VVI or AAI) at paper speeds of 1 mm/s and 25 mm/s and included
 - (a) recordings of BP and RAP for 10 min in intrinsic heart rhythm or at a pacing heart rate of 50 beats/min in the case of pacemaker dependence
 - (b) continuous recordings for 10 min of RAP and BP at pacing rates of 70, 90, 110, and 130 beats/min alternately with the intrinsic rhythm. If BP was not increased during the pacing rates of 110 and 130 beats/min, pacing at these rates was reduced to 2–3 min.

The characteristics of the patients studied are presented in Table I.

All patients or their relatives gave informed consent. No patient with atrioventricular block had ventriculoatrial conduction. No adverse reactions were observed in any of the patients.

One patient (Patient No. 10), a 76-year-old woman, died due to irreversible brain damage as a consequence of ventricular fibrillation and prolonged cardiovascular resuscitation in another hospital. All remaining patients were discharged alive and remain alive at 6-month follow-up.

Statistical Analysis

The results of SBP, DBP, and mean RAP were compared using the Student's two-tailed *t*-test for paired data between intrinsic heart rhythm and paced heart rates above (70–130 beats/min).

TABLE I Basic characteristics of the patients studied

Patient No.	Sex	Age	Intr-rhyt	BP S/D	MRAP	PCWP
1	M	82	52	60/20	22	9
2	F	67	46	75/46	26	12
3	M	72	62	106/51	10	8
4	M	64	39	115/54	18	13
5	M	59	42	109/48	10	9
6	M	73	48	105/50	13	7
7	F	66	40	98/49	12	11
8	M	58	56	103/52	14	12
9	M	54	52	77/42	16	6
10	F	76	46	80/50	21	12
11	M	71	58	100/46	13	8
12	M	74	63	96/58	12	7
13	F	69	66	94/52	10	8
14	M	58	51	99/55	9	9
15	M	68	56	102/50	15	12

Abbreviations: BP S/D = blood pressure systolic/ diastolic, F = female, M = male, Intr-rhyt = intrinsic rhythm, MRAP = mean right atrial pressure, PCWP = pulmonary capillary wedge pressure.

Using the same parameters, every pacing rate was also compared with all the pacing rates above it. A *p* value of <0.05 was considered statistically significant.

Results

The results are presented in Figure 3 and Table II.

Systolic BP increased significantly from 94.6 ± 15 mmHg during intrinsic rhythm to 101.9 ± 13.8 , 117 ± 10.3 , $126.6 \pm$

FIG. 3 Results in bar charts (mmHg $\pm$ SD) of systolic blood pressure (dark columns), diastolic blood pressure (light columns), and right atrial pressure (open columns). In. rhyt. = intrinsic rhythm.

11, and 127 ± 12.2 mmHg at heart rates of 70, 90, 110, and 130 beats/min, respectively ($p < 0.0001$).

Diastolic BP also increased from 48.2 ± 8.7 mmHg to 53.9 ± 3.7 , 59.3 ± 5.6 , 66.8 ± 3.6 , and 69.1 ± 3 mmHg at heart rates of 70, 90, 110, 130 beats/min, respectively ($p = 0.014-0.0001$).

Mean RAP decreased from 14.5 ± 5 mmHg to 14.1 ± 5 , 12 ± 4.7 , 11.1 ± 4.1 , and 11.6 ± 4.1 mmHg at heart rates of 70, 90, 110, and 130 beats/min, respectively ($p = 0.16-0.0001$).

By comparing SBP at the usual pacing rate (70 beats/min) and that at higher pacing rates, a significant elevation of SBP was detected: from 101.9 ± 13.8 mmHg in intrinsic rhythm to 117.7 ± 10.3 mmHg at a heart rate of 90 beats/min, 126.6 ± 11 mmHg at 110 beats/min and 127.1 ± 12.2 mmHg at 130 beats/min heart ($p < 0.0007$).

By comparing DBP at the usual pacing rate (70 beats/min) with that at pacing heart rates of 90–130 beats/min, a statistically significant elevation was detected: from 53.9 ± 3.7 mmHg at 70 beats/min to 59.3 ± 5.6 , 66.8 ± 3.6 , and 69.1 ± 3 mmHg at pacing rates of 90–130 beats/min, respectively ($p < 0.0009$).

Mean RAP at the usual pacing heart rate (70 beats/min) was decreased in comparison with pacing rates at 90–130 beats/min: from 14.1 ± 5 mmHg to 12 ± 4.7 , 11.1 ± 4.1 , and 11.6 ± 4 mmHg at pacing rates of 90–130 beats/min ($p < 0.0002$).

Finally, by comparing SBP, DBP, and RAP at a pacing rate of 110 beats/min with that at 130 beats/min, only DBP increased significantly: from 126.6 ± 11 to 127.1 ± 12.2 mmHg ($p = 0.8$), 66.8 ± 3.6 to 69.1 ± 3 mmHg ($p = 0.0084$), and 11.1 ± 4.1 mmHg to 11.6 ± 4 mmHg ($p = 0.16$), respectively.

TABLE II Cumulative results of blood pressure (S/D) and mean right atrial pressure

Patient No.	BP/IR			BP/70			BP/90			BP/110			BP/130		
	S	D	MRAP/IR	S	D	MRAP/70	S	D	MRAP/90	S	D	MRAP/110	S	D	MRAP/130
1	60	20	22	78	53	21	110	55	19	130	70	18	135	76	18
2	75	46	26	85	56	24	118	66	21	145	68	17	138	71	18
3	106	51	18	107	53	18	110	49	15	120	69	14	125	72	14
4	115	54	16	122	60	16	136	65	13	142	72	12	135	69	13
5	109	48	10	121	54	9	128	56	8	136	60	6	136	69	6
6	105	50	10	119	55	9	128	68	7	140	70	6	138	71	6
7	98	49	12	106	53	10	124	54	9	128	64	9	152	68	8
8	103	52	14	105	54	16	113	58	11	121	65	12	129	65	10
9	77	42	13	84	46	13	134	62	9	128	69	10	119	71	12
10	80	50	21	85	53	21	115	61	20	120	65	18	125	68	18
11	103	48	11	104	48	10	117	54	8	131	64	8	122	65	9
12	96	58	12	102	61	11	110	68	9	116	71	8	115	71	10
13	94	52	10	96	54	9	99	58	8	110	67	7	108	68	8
14	99	55	9	107	54	9	109	57	8	110	61	8	108	65	10
15	102	50	15	108	55	14	114	59	13	122	67	12	122	67	12
Σ	94.6	48.2	14.5	101.9	53.9	14.1	117.7	59.3	12	126.6	66.8	11.1	127.1	69.1	11.6
SD	± 15	± 8.7	± 5.1	± 18.8	± 5.6	± 5	± 10.3	± 5.6	± 4.7	± 11	± 3.6	± 4.1	± 12.2	± 3	± 4
p				0.0001	0.014	0.16	0.0001	0.0003	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001

Abbreviations: BP/IR = blood pressure/intrinsic rhythm, MRAP/IR = mean right atrial pressure/intrinsic rhythm, D = diastolic blood pressure, S = systolic blood pressure, Σ = the mean values $\pm$ standard deviation (SD), P = comparison of the intrinsic heart rhythm with the higher pacing rates.

Discussion

Acute right ventricular myocardial infarction accompanies inferior-posterior AMI in a proportion of 40–70%.^{1, 3, 13} Inferior AMI is frequently associated with sinus bradycardia or atrioventricular block and is much more common in RVI patients (48 vs. 13%).^{1, 8} The preservation of atrioventricular sequential contraction in permanently paced patients is a well-known factor in improving heart performance and survival.¹⁵ In the particular condition of RVI, the restoration of atrioventricular contraction and the contribution of atrial contraction to the ischemic and noncompliant right ventricle has been proven beneficial.^{1, 7, 16}

High-rate VVI pacing in normal subjects has shown decrease in SBP, increase in DBP, decrease in stroke volume, and increase in catecholamine release during high-rate pacing.^{17–19} These observations probably explain the elevation of DBP—but not the elevation of SBP—in our patients as an effect of increased catecholamine release during high-rate cardiac pacing.

The results in the patients we studied are difficult to interpret; however, one can postulate:

In a patient with RVI, the systolic strength of the right ventricle is acutely reduced, the right ventricular cavity is dilated, and the end-diastolic volume and pressure are increased.^{2, 5, 8} In addition, the expansion of the overfilled right ventricle is impeded by the pericardium.²⁰ The prominent descending y wave of the right atrial pressure waveform (Figs. 1, 2) reflects the noncompliant hemodynamic pattern of the right ventricle, which is rapidly overfilled with a small volume of blood. Normally, an increase in heart rate reduces ventricular filling time. However, this is probably not the case in hemodynamically significant RVI, since the ventricular filling time lasts for a short period during early diastole. Thus, tachycardia in patients with hemodynamically significant RVI would not reduce right ventricular preload and stroke volume.

The decrease in RAP during high-rate pacing indicates that the right ventricle poses less impedance to ventricular filling, which may increase right ventricular preload and stroke volume. This is supported by the increase in BP waveform width during high-rate cardiac pacing (Figs. 1, 2).

In the case of severe RVI, the right ventricle behaves more or less as a passive conductor of blood from the right atrium to the pulmonary artery, as is the case in Uhl anomaly.^{2, 7} Thus, in the case of RVI and sinus bradycardia, the increase in pacing rate increases the volume of blood that the right atrium pumps directly to the pulmonary artery. This obviously increases the cardiac output.

The increase in mean BP and the decrease in mean RAP increases the perfusion pressure (ΔP) of the coronary circulation. By considering the equation of blood flow (F) in the coronary circulation: $F = \Delta P/R$, where R is the resistance, the coronary blood flow is expected to increase in proportion to the increase in ΔP .²¹ That may increase the oxygen delivery to the ischemic right ventricular myocardium.

High heart rate is expected to increase the myocardial O_2 demand, which may increase the probability of myocardial damage in patients with RVI. However, this is probably not the

case in patients with predominantly RVI, because the intracardiac pressure in the right ventricle cavity is much lower than in the left ventricle and the oxygen supply actually continues during the whole cardiac cycle (not only during diastole).²²

The established treatment of volume overload in hemodynamically significant RVI is not without problems, since the elevated RAP pressure would increase further, which could lead to systemic edema. Dell' Italia *et al.* reported that volume overload is not as beneficial as the administration of dobutamine in patients with RVI.²³ The latter could be partly explained by increased heart rate on dobutamine infusion.

Our findings differ from those of Love *et al.*⁷ The difference can probably be found in the hemodynamic profile. Of the patients they studied, 4 of 7 had a mean PCWP > 12 mmHg, versus 1 of 15 patients in our series. This probably reflects differences of left ventricular involvement. Considering the results of the present study, one realizes that it could be harmful to increase the heart rate for a prolonged period of time in a patient with significant RVI.

As the acute beneficial effect was the same between 110 and 130 beats/min, a more conservative heart rate of 80–110 beats/min would seem to be indicated. However, this needs further investigation.

Limitations

The major limitation of the present study is that we did not perform detailed hemodynamic studies to delineate the pathophysiology of our findings. Further investigations are therefore needed to assess the hemodynamic effects of high-rate cardiac pacing in patients with predominant RVI and bradyarrhythmia. In particular, stroke volume, cardiac output, pulmonary artery pressure, and PCWP should be performed. Also, echocardiographic studies would be of interest to assess the influence of high-rate pacing on right chamber volumes. Furthermore, prolonged observations are needed in patients during the acute stage of RVI and probably in a control group of patients with inferior myocardial infarction and bradyarrhythmia but without right ventricular involvement.

Conclusion

The results of an acute increase in heart rate in patients with predominant right ventricular myocardial infarction, low cardiac output, and bradycardia, if demonstrated over a prolonged period of time, could be beneficial in the management of these patients by indicating the usefulness of high-rate cardiac pacing (VVI or AAI) in the range of 80–110 beats/min.

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