SYMPOSIUM
Cardiac Arrhythmias
(Part 2)

Hemodynamic Sequelae of Cardiac Arrhythmias

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SUMMARY
The hemodynamic consequences of cardiac arrhythmias depend on various factors, including the ventricular rate and the duration of the abnormal rate, the temporal relationship between atrial and ventricular activity, the sequence of ventricular activation, the functional state of the heart, the irregularity of the cycle length, associated drug therapy, the peripheral vascular vaso- motor system, disease in organ systems other than the heart, and the degree of anxiety caused by the disease processes. Sinus bradycardia, even with rates as low as 40 beats/min, may not be associated with significant hemodynamic consequences unless the stroke volume is limited by myocardial or valvular disease, as in acute myocardial infarction. Cardiac output usually, but not invariably, falls when atrial fibrillation replaces normal sinus rhythm, even at comparable ventricular rates, both at rest and during exercise. Similar observations have been made during the development of atrial flutter despite the persistence of effective mechanical atrial activity in at least some cases. Marked hemodynamic changes are frequent in the course of ventricular tachycardia with systemic arterial hypotension, a decrease in cardiac output, and evidence of cerebral, coronary, and renal vascular insufficiency. Cyclic variations in systemic and pulmonary arterial pressures are common during atrioventricular dissociation. Cardiac output is generally depressed during the severe bradycardia of acquired complete heart block with evidence of atrioventricular valvular insufficiency. Increase of the heart rate by ventricular pacing reverses all or some of these abnormalities. The changes in congenital complete heart block are considerably less severe because myocardial insufficiency is less frequently seen in congenital complete heart block.

THE PAST DECADE has witnessed a rebirth of interest in cardiac arrhythmias. This development has extended beyond considerations of the clinical and electrocardiographic differential diagnosis to evaluation of the hemodynamic and clinical sequelae of the arrhythmias.

A number of investigators1-5 have stressed the concept that the physiologic consequences of cardiac arrhythmias are caused by: (1) changes in ventricular rate and the duration of these changes; (2) the effect of atrial function including the temporal relationships between atrial and ventricular activity, atrioventricular valvular function, strength and effectiveness of atrial mechanical activity, and storage function of the atria; (3) the sequence of ventricular activation; (4) functional state of the heart; (5) the irregularity of the cycle length of the arrhythmia; (6) drug therapy; (7) preservation of vaso-motor control mechanisms; (8) The superimposed presence or absence of anxiety; and (9) disease in other organ systems.

Changes in Ventricular Rate
Modest increments in ventricular rate (185-230 beats/min) produced by atrial pacing result in only small and transient decreases in cardiac output in the dog.6,7 The changes in systemic arterial blood pressures and coronary blood flow are minimal. Further increments in ventricular rate following atrial pacing caused clear-cut decrements in these parameters.

A substantial body of data has accumulated in man as to the effect of variations in heart rate. Ross et al.8 reported "no striking alterations in the
cardiac index occurred” during the use of right atrial pacing alone to increase the ventricular rate: increases in heart rate from 80 to 121 beats/min altered cardiac index from 3.67 to 3.72 liters/min/m². Further increases in atrial pacing rate caused a small decrease in flow to 3.2 liters/min/m². Stein et al.9 reported similar results. On the other hand Samet et al.10 reported data on the effect of right atrial pacing in 33 normal subjects and 136 patients with cardiac or pulmonary disease. These latter data demonstrated that cardiac output increases in a statistically (but not physiologically) significant manner as the ventricular rate is increased modestly (to rates of 60–89 beats/min). Further rate increments produced no further output changes as the rate was increased to levels of 90–110 and 110–140 beats/min. Yoshida et al.11 came to similar conclusions. The therapeutic value of temporary right atrial pacing in management of the patient after open-heart surgery has been stressed.12,13 Augmentation of cardiac output is probably the primary beneficial hemodynamic effect of postoperative pacing. Increase in atrial and ventricular rate to 180 beats/min in man following right atrial pacing was followed by a fall in cardiac output in normal subjects; output fell at pacing rates above 140 in cardiac patients.14 Inadequate diastolic ventricular filling periods are the probable cause of these results. Data as to the effect of long-term increases in atrial and ventricular rate upon cardiovascular dynamics in man are not available.

An experimental study on the effects of increasing heart rate on the force-velocity relationships of the left ventricle has provided a basis for understanding the small increases in output observed by some investigators during atrial pacing. While mean aortic and left ventricular end-diastolic pressures were maintained constant, the heart rate was increased by atrial pacing. This shifted the isovolumic force-velocity curve up to the right with increase in maximum contractile element velocity and maximum isovolumic tension. These effects were evident despite shortening of the phases of systole.15 These changes probably provide the substrate for an increase in output.

**Effect of Atrial Function**

The basic function of the atrium is to aid in blood transport into the ventricle by atrial systole and to aid in closure of the atrioventricular valves thereby facilitating ventricular filling while left atrial mean pressure is maintained at a normal level.

**Temporal Relationships between Atrial and Ventricular Activity**

It has long been appreciated that significant changes in cardiac dynamics follow interruption of normal sequential atrioventricular electrical and mechanical activity both in the experimental animal and in man.1–3,5,6,16,17 The so-called “booster pump action of the atria” in increasing ventricular volume at end-diastole is 15–20% larger during atrial than during ventricular pacing.5 P-wave synchronous pacing (in complete heart block) elevates cardiac output by 10–15% more than does ventricular pacing at the same ventricular rates.18 These observations apply to ventricular rates of 60–100 beats/min as well as faster ventricular rates as high as 130–140 beats/min. The optimal P-R interval is 0.10–0.20 sec.19 As the heart rate increases, the P-R interval effect is increased.20 The atrial contribution may also be more significant in the presence of myocardial dysfunction.

**Atrioventricular Valvular Function**

A properly timed atrial systole results in essentially a presystolic reversed atrioventricular gradient,21 placing the atrioventricular valve leaflets in a position to close early with the onset of ventricular systole and thus prevent atrioventricular valvular regurgitation. Such regurgitation, in addition to the absence of the booster pump action of the atrium (atrial kick) may be responsible for the lower cardiac output during ventricular as compared to atrial pacing.

**Atrial Mechanical Activity**

Dissociation between the effects of the ventricular irregularity per se, the rapid ventricular rate, and the lack of effective atrial mechanical activity is not always achieved in studies of atrial fibrillation and flutter. Skinner et al.22 studied the effects of atrial fibrillation at constant venricular rates in the dog during ventricular pacing. Removal of the effective atrial contribution resulted in a rise of left atrial mean pressure despite a decrease in left ventricular and end-diastolic pressure, a fall in aortic pressure, and a decrease in output. A ventricular systolic rise in left atrial pressure was seen only during atrial fibrillation. These effects can also be observed in man.

The effect of loss of atrial mechanical activity in atrial fibrillation in man is somewhat difficult to analyze because of the multiplicity of factors involved in the studies in the literature. These include the effect of drugs and anesthesia plus the
problems mentioned above, i.e. the increase in ventricular rate and ventricular irregularity changes per se in addition to the loss of atrial mechanical activity. Morris et al.\textsuperscript{23} studied 11 patients before and after cardioversion under light thiobarbital anesthesia (duration 12–30 min). The average ventricular rate and cardiac index before and 2 hours after cardioversion were 78 beats/min and 4.41 liters/min/m\(^2\) before and 86 and 5.31 afterward, \(P<0.05\) for the output increase. Oxygen consumption was identical (316 ml/min/m\(^2\) control, 318 after cardioversion) in the two studies. Output rose more than 0.5 liters/min in seven of the 11 patients after conversion. Five of the 11 patients were also exercised before and after electrical shock. The control exercise data was oxygen consumption 1002 ml/min/m\(^2\), heart rate 110 beats/min, and output 8.36 liters/min; the postconversion figures were 988, 118, and 9.80, respectively. The average output changes were significant, \(P<0.01\). All five patients exhibited a significant output rise during exercise. Rodman et al. studied 19 patients (generally without anesthesia) before, immediately after, and 1, 2, and 3 hours after cardioversion from atrial fibrillation to sinus rhythm.\textsuperscript{24} Despite considerable individual variation, cardiac index rose from a control level of 2.36 to 2.40, 2.45, 2.56, and 2.65 liters/min/m\(^2\) at the four stated times after cardioversion. The average heart rate was 73 control and 70–74 beats/min after cardioversion. Full interpretation of the available data poses problems ranging from contradictory data reporting no change in output at rest following cardioversion without a fall in ventricular rate,\textsuperscript{25} to observations at rest showing output increment not immediately after conversion but only days later,\textsuperscript{26} to observations reporting more significant effects on cardioversion during exercise than at rest.\textsuperscript{27} Carlton and co-workers have stressed that atrial systole augments cardiac output and left ventricular \(dp/dt\) in patients without valvular heart disease but does not do so in the presence of mitral stenosis; study of 11 patients with mitral stenosis and atrial fibrillation during right ventricular pacing to induce artificial tachycardia led to the conclusion that the functional deterioration often seen in patients with mitral stenosis with atrial fibrillation is due to ventricular rate increase rather than loss of atrial systole.\textsuperscript{28} It has been noted that following successful cardioversion right atrial a waves may occur in the absence of left atrial a waves even in the presence of P waves on the electrocardiogram,\textsuperscript{29} i.e., right atrial function recovers more rapidly than left atrial function.

\textit{Storage Function of the Atrium}

The atria also serve as conduit and storage areas between the great veins and the ventricles.

\textit{Sequence of Ventricular Activation}

It has been stated that the sequence of ventricular activation and contraction has significant effects on cardiovascular dynamics. The fact that some cardiac pacing studies demonstrate differences in stroke output of up to 100% secondary to changing the site of ventricular stimulation has been interpreted as demonstrating the importance of altered ventricular sequence of contraction. However, studies in man have suggested that an abnormal pathway of ventricular depolarization is of little hemodynamic import.\textsuperscript{5} In analysis of the cause of the hemodynamic differences between atrial and ventricular pacing, two possible explanations were suggested—the absence of the atrial kick and abnormal depolarization during ventricular pacing. Observations were therefore made during atrial, ventricular, and sequential atrioventricular pacing to separate these two factors. The latter was done by applying paired pacing stimuli to the right atrium and right ventricle, at an interval less than the control P-R interval for a given subject. During sequential atrioventricular pacing the effect of the atrial kick is preserved in the face of abnormal ventricular depolarization. The results of studies in patients with normal hearts and with heart disease demonstrated that the cardiac outputs during sequential atrioventricular pacing were only slightly less than those during atrial pacing and that both were significantly greater than the outputs during ventricular pacing. Abnormal ventricular depolarization per se produced little effect on cardiac output.

The data suggest that asynchronous ventricular activation has relatively little hemodynamic effect, and are consistent with older observations that significant mechanical ventricular asynchrony is not a necessary consequence of the electrical ventricular asynchronism of bundle-branch block or ventricular premature beats.\textsuperscript{30} Braunwald and Morrow\textsuperscript{31} studied five patients with complete left bundle-branch block and 10 with complete right bundle-branch block. None of the former exhibited delay in onset of left ventricular contraction, and only four of the latter patients had as much as a
0.04-sec delay in onset of right ventricular contraction. Bourassa et al. have, however, presented data to the contrary in a patient with intermittent left bundle-branch block.22

**Functional State of the Heart**

It can readily be appreciated that any given cardiac arrhythmia may be better tolerated when the underlying myocardium is normal. Patients with congenital complete heart block are better able to tolerate severe bradycardia than patients with acquired complete heart block. In the former group the ability to compensate for bradycardia by increasing the stroke volume is greater than in patients with acquired block. Even minor arrhythmias may be hemodynamically significant in the face of severe myocardial disease.

**Irregularity of Cycle Length of the Arrhythmia**

The irregular pulse rate observed in many arrhythmias, especially atrial fibrillation, may per se have definite hemodynamic effects. Potentiation is a key mechanism for some of these effects. Potentiation is a mechanism in which an early ventricular depolarization augments the force of contraction of the succeeding contraction.33, 34 Greenfield et al.35 noted that peak flow, peak power, and stroke work correlated best with the two previous R-R intervals, not simply the preceding cycle length. A second mechanism whereby cardiac irregularities produce hemodynamic effects is via Starling's law of the heart. Braunwald et al.36 performed studies in 26 adult patients with atrial fibrillation at operation. Measurements of the length of a segment of the left ventricle by means of a mercury-filled resistance gauge were made together with arterial pressure and left ventricular end-diastolic pressure. Variations of end-diastolic segment length and pressure due to changing duration of diastole were measured. Changes in end-diastolic segment length correlated with peak systolic ventricular pressures. The longer the preceding cycle length in the atrial fibrillation, the greater is the length of the left ventricular segment.

A third mechanism is change in the refractory period of various cardiac tissues associated with sudden rate changes. As many as the 12 preceding cycles may affect the refractory period. An abrupt change from slow to rapid ventricular rates shortens the refractory period maximally in the first beat and progressively less in subsequent beats.37 Such changes in refractory period may be responsible for rate changes in arrhythmias.

**Drug Therapy**

Just as the state of the myocardium may influence the hemodynamic effects of cardiac arrhythmias, so may drugs alter the consequence of arrhythmias. These effects may result from direct drug effect on the myocardium or from effects on the peripheral vascular system. In addition, the drug involved may itself cause an arrhythmia.

**Preservation of Vasomotor Control Mechanisms**

The response to similar cardiac arrhythmias may be quite varied in different patients. At least part of these differences may be due to the status of the peripheral vascular system and its control mechanism. For example, Skinner et al.22 noted that bilateral vagotomy, with control of carotid sinus pressure, blunted the compensatory mechanisms which modified the hemodynamic consequences of acute induced atrial fibrillation in the dog. Drugs, circulating catecholamines, altered blood volume, redistribution of blood volume, venous tone, neurogenic reflexes, or intrinsic disease may thus alter the effects of cardiac arrhythmias.

**Superimposed Mental Status of the Patient, i.e. Level of Anxiety**

Cardiac arrhythmias may result in anxiety responses on the part of the patient. Outpouring of catecholamines may result38 with secondary effects on the cardiac rhythm as well as the peripheral vascular system.

**Disease in Other Organ Systems**

The hemodynamic effect of arrhythmias may also be conditioned by disease processes in other organs. Atherosclerosis of the bowel vessels may result in symptoms secondary to the arrhythmia which might be absent in patients without such vascular involvement. Mild pulmonary congestion in a patient with obstructive pulmonary emphysema may result in more profound symptomatology than in the presence of otherwise normal lungs. Anemia may also modify the hemodynamic result of cardiac arrhythmias.

**Effect of Arrhythmias on Regional Blood Flow**

Several lines of evidence indicate that the cerebral circulation may be compromised by cardiac arrhythmias. The electromagnetic flowmeter was employed to measure cerebral blood flow (carotid artery flow) during the control sinus rhythm, and various arrhythmias in the dog.39 The flow decrement averaged 7–12% during premature
beats, 14% during supraventricular tachycardia, 23% during atrial fibrillation, and 40–75% during ventricular tachycardia. The clinical counterpart of these experimental observations is well established. A 10-hour taped electrocardiogram was recorded in 39 adult patients with symptoms of cerebral ischemia. Ten of the 39 patients exhibited arrhythmias with bradycardia below 40 beats/min or heart rates above 150 beats/min. Morgagni-Stokes-Adams episodes in complete heart block have, of course, been well known for many years. Electrical pacing with correction of the bradycardia was associated with a significant increase in cerebral blood flow. Coronary artery flow may be reduced by atrial or ventricular premature systoles, atrial and ventricular tachycardia, and atrial fibrillation. The clinical counterparts of these experimental data are well known. Posttachycardia T-wave inversion is frequently seen even in young individuals with normal coronary circulations. Cardiac arrhythmias may also be associated with reduction in renal and mesenteric blood flow in the dog. Marked polyuria is often seen in patients during supraventricular tachycardia. Lower rhythm nephrosis may complicate arrhythmias.

**Specific Arrhythmias**

**Sinus Bradycardia**

Even ventricular rates below 40–45 beats/min are often not associated with severe hemodynamic consequences if the stroke volume is not limited by myocardial or valvular disease. In one group of 17 patients with only sinus bradycardia (Hildner FJ, Narula OS, Samet P: Unpublished data), the ventricular rate ranged from 37 to 59 beats/min (average 49). The average cardiac index was 1.93.

Patients with acute myocardial infarction and sinus bradycardia are often prone to increased vagal and vasovagal reactions with hypotensive episodes. Bradycardia, inability to increase stroke volume due to decreased myocardial reserve, and inadequate peripheral vasomotor controls all contribute to the problem. The bradycardia and hypotension may, in turn, predispose to serious ventricular arrhythmias and ventricular standstill especially in the presence of an acute myocardial infarction.

**Supraventricular Tachycardia**

The hemodynamic differences between nodal and atrial tachycardia relate primarily to the incidence of atrioventricular dissociation in the former but not the latter and to ventricular rate differences. The effects of atrial tachycardia depend on the myocardial state, duration of the rhythm, and heart rate. As the heart rate increases in atrial tachycardia, there is, at most, only a small change in cardiac output in the experimental laboratory, until rates of 160–230 beats/min are recorded, at least in normal hearts; at faster rates the output may then fall. The fall in output may occur at slower rates in the diseased heart. Several studies in man have revealed little change in cardiac output at the onset of atrial tachycardia. A fall in systemic arterial pressure may occur especially when in the upright position. Right atrial and pulmonary artery wedge pressures rose especially at the onset of tachycardia probably secondary to inadequate diastolic ventricular filling. Systemic pulsus alternans has been reported. The occasional development of heart failure or angina pectoris during supraventricular tachycardia is the clinical counterpart of the physiologic hemodynamic changes. Study of two patients with nodal tachycardia and the Wolff-Parkinson-White syndrome revealed evidence of tricuspid regurgitation with right atrial hypertension.

**Atrial Flutter**

Mechanical atrial activity may persist in atrial flutter unlike atrial fibrillation. Atrial contractions at rates of 300/min may be present. Cardiac output was decreased in nine patients with atrial flutter, five with heart failure (index 1.48–2.48), three with heart disease not in failure (index 1.67–2.10), and one without heart disease, cardiac index 2.57. In five patients studied in flutter and sinus rhythm, the index rose 40% on conversion to sinus rhythm as the ventricular rate also fell. Digitalization in four patients resulted in a significant rise in output with a significant decrease in heart rate and persistence of atrial flutter. A single patient was studied in sinus rhythm, atrial fibrillation, and atrial flutter during both rest and exercise, with comparable oxygen consumptions during the three rest and three exercise periods. The three resting ventricular rates were virtually identical. The cardiac indices were identical during sinus rhythm and atrial fibrillation but fell slightly in atrial flutter. During exercise, the cardiac index was greater during sinus rhythm than during the atrial arrhythmias.

**Atrial Fibrillation**

The development of the right atrial electrogram has demonstrated that differentiation between atrial
fibrillation and supraventricular tachycardia is not always possible on the basis of the surface electrocardiogram. Intracardiac recordings may be required for this purpose. Comments on the hemodynamic changes during atrial fibrillation were made above.

The hemodynamic data obtained in patients with atrial fibrillation during exercise deserve comment. Most studies have revealed significant hemodynamic improvement after conversion to sinus rhythm. Graettinger et al.\textsuperscript{25} studied 17 patients during exercise in both atrial fibrillation and normal sinus rhythm. Oxygen consumption was very similar during both rhythms, but the average cardiac index rose from 2.98 to 3.20 while the respective heart rates fell from 127 to 98 on conversion. Benchimol et al.\textsuperscript{50} studied eight patients during exercise in both rhythms and observed an 8% rise in cardiac output after conversion with a 30% rise in stroke volume since the heart rate fell 24%. Separation of the effects of rhythm change from heart rate change is difficult. Another observer reported that, in selected patients, exercise tolerance may not be affected by atrial fibrillation.\textsuperscript{51}

\textit{Ventricular Tachycardia}

The hemodynamic effects of spontaneous rapid ventricular tachycardia in the experimental animal include hypotension, a fall in cardiac output, and evidence of cerebral, coronary, and renal vascular insufficiency.\textsuperscript{5, 38} Pulmonary arterial and left and right atrial pressures may increase.\textsuperscript{58} The effect of ventricular tachycardia may also be observed in man by simulated ventricular tachycardia, i.e., ventricular pacing.\textsuperscript{1, 2, 5, 6, 15, 36} These studies clearly reveal that systemic hypotension, cyclic variations in systemic and right heart pressures, and decreases in cardiac output regularly occur even when the effect of rate, per se, is eliminated by comparing atrial and ventricular pacing at similar ventricular rates. The abnormal dynamics during ventricular pacing or ventricular tachycardia are probably due to both the rapid rates and the absence of sequential atrial-ventricular activity. Thus, the hemodynamic abnormalities seen in ventricular tachycardia are often more severe than those in supraventricular tachycardia even at the same overall ventricular rate. The importance of the ventricular rate per se in the hemodynamics of ventricular rhythms has been stressed by recognition of the relatively benign course of some ventricular rhythms (rates 60-90) in the course of acute myocardial infarction. The abnormal dynamics of ventricular tachycardia are accentuated during ventricular fibrillation when effective circulation ceases.

\textit{Brady-Tachyarrhythmia Syndrome (Sick Sinus Syndrome)}

The frequency of this syndrome in which slow and rapid ventricular rates alternate has recently been recognized.\textsuperscript{52} The hemodynamic effects vary with the different arrhythmias present in a given case.

\textit{Complete Heart Block}

Levinson et al.\textsuperscript{53} studied five patients in acquired heart block not in failure. Right heart pressures were elevated. Systemic and right heart pulse pressures are widened. The heart rates are 45-55 when the QRS complex is narrow, i.e. originates above the bifurcation of the His bundle. The rate is 30-45 when the ventricular focus is idioventricular in origin. The cardiac output was decreased but the stroke volume was elevated. Stark et al.\textsuperscript{54} made comparable observations in eight patients. Right ventricular end-diastolic pressure was generally normal. In the presence of heart failure, marked further decrease in cardiac output was observed. Samet\textsuperscript{55} reported low cardiac indices in 24 subjects not in failure. The abnormalities observed in these patients probably result from a slow rate, myocardial disease, plus abnormal P-QRS temporal relationships. The hemodynamic data in congenital heart block are somewhat different from those in acquired complete heart block. The effects of associated myocardial disease are probably absent in these patients. The heart rates are somewhat faster (40-80/min). The cardiac output is usually normal. Right heart pressures are generally close to normal. Right ventricular end-diastolic pressure is normal or minimally elevated.\textsuperscript{56}

Exercise studies in acquired complete heart block have revealed a further increase in the already large stroke volume and a rise in cardiac output if left ventricular function is relatively normal. If myocardial function is depressed, the stroke volume may be fixed during exercise.\textsuperscript{57} The heart rate is generally constant during exercise in acquired complete heart block. In congenital complete heart block, the ventricular rate and cardiac output usually rise during exercise, as myocardial function is commonly normal.\textsuperscript{58}

When the heart rate is increased by ventricular pacing in patients in complete heart block, the cardiac output generally rises.\textsuperscript{5, 55, 57, 59} A bell-shaped curve is often noted and the output may fall...
as the rate rises above 100 beats/min. P-wave synchronous pacing results in larger cardiac outputs than obtained during ventricular pacing at similar ventricular rates.18

Atrial, Nodal, and Ventricular Premature Beats

The effect of premature beats depends on several factors including the underlying heart disease, the degree of prematurity of the beat, the frequency and the site, i.e. atrial or ventricular, and the P-QRS temporal relationship.1 A very early premature beat may have little or no mechanical effect, with even little effect on the ventricular pressure curve. The electrical heart rate is then greater than the effective mechanical heart rate, as in the postextrasystolic potentiation phenomena.33, 34 If the premature beat is somewhat later in the cardiac cycle, a mechanical ventricular pressure curve may be generated, but the level of pressure achieved may not be sufficient to open the corresponding semilunar valve. Premature beats in mid-to-late diastole will usually have less abnormal hemodynamic effects than earlier beats. The left ventricular pulse pressure and systolic pressure peak and the systemic arterial pulse pressure increase after the compensatory pause of a ventricular premature beat except for patients with idiopathic hypertrophic subaortic stenosis. In these individuals the systemic arterial pulse pressure falls after a ventricular premature beat due to the increased contractility observed after the premature beat.30

The hemodynamic sequelae of premature beats also depend on the site of origin, i.e. whether or not normal atrioventricular sequential activity is maintained. The site of origin of ventricular premature beats is believed by some investigators to influence the hemodynamic effect of such beats.61 Other studies have, however, cast doubt on the importance of the sites of ventricular stimulation.62

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