Atrial systole: its role in normal and diseased hearts

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HISTORICAL PERSPECTIVE

The importance of atrial systole in cardiac physiology was first recognized by William Harvey in 1628: "... and if at this time with its auricle alone beating you cut off the apex of the heart with a pair of scissors, you will see the blood flow out of the wound with each beat of the auricle. You will thus realise that the blood gets into the ventricles not through any pull exerted by the distended heart but through the driving force exerted by the beat of the auricles"[1].

It appears from this quotation, however, that Harvey believed ventricular filling was wholly active and caused by atrial systole. His observations were unsurpassed until the late 19th century when further evidence for the presystolic role of atrial contraction was derived from the intraventricular pressure curve of the horse. Chaveau & Marey in 1863 showed a "small but well defined wave before the sharp systolic rise" [2]. In the early 20th century more exact measurements of cardiac physiology were made and the diastolic filling of the ventricles was studied experimentally. Yandall Henderson [3] in 1906, using an isolated heart preparation and plethysmography to measure volume changes in the ventricle, showed that diastolic ventricular filling was mainly passive and found only a small increase in volume coincident with atrial systole. He concluded that "the contraction of the auricles increases the ventricular volume to the extent of a few drops at most". Henderson's results were later criticised by Gesell [2], who felt that the plethysmography equipment used by Henderson was insensitive to rapid changes in volume and that changes in the outer contours of the heart may not represent what was happening inside. This was borne out by the work of Hermann Straub [4], who in 1910, using isolated mammalian hearts and sensitive plethysmography repeated Henderson's early experiments. He showed a marked increase in ventricular volume after atrial contraction. Subsequently, Gesell performed a series of experiments [2,5,6] in isolated heart preparations in order to define the role of atrial systole and to study the factors which influenced it. Much of the knowledge we have about the physiology of atrial systole stems from these early experiments.

METHODOLOGY

Various techniques have been used to investigate the role of atrial systole. First, in isolated heart preparations, the timing of atrial and ventricular contraction was changed by either interference waves [6] or by surgically inducing complete atrioventricular block, thereby resulting in dissociation between atrial and ventricular contraction [7, 8]. Interference waves caused by Farradic stimulation of the atria interfere with the normal atrial activation sequence in much the same way as interference in sound waves. Once atrioventricular dissociation has been produced, the haemodynamic differences between synchronous atrioventricular contraction and asynchronous contraction can be found. Since the development of transvenous cardiac pacing, much research has been based on observations between ventricular only and synchronous atrial and ventricular pacing [9, 10].

The second way in which the value of atrial systole to overall cardiac function has been measured was by comparing haemodynamic parameters in sinus rhythm with those during atrial fibrillation [11–14]. Recently, Doppler ultrasound, combined with real time imaging, has provided a third, non-invasive, method of studying intracardiac flows in vivo [15, 16], including flow across the mitral valve. With this technique, continuous assessment of flow from left atrium to left ventricle can be performed.

ATRIAL PHYSIOLOGY

The early physiologists used isolated heart preparations with atrioventricular asynchrony to study ventricular filling and the role of atrial systole. Gesell showed that asynchronous atrial contraction caused a reduction in blood pressure of 55% [2], which he later showed to be due to a reduced cardiac output [5, 6]. There were two possible explanations for this observation. First, that when atrioventricular dissociation occurred, atrioventricular valve closure was incomplete and the reduction in
cardiac output was due to regurgitation of blood back into the atrium. Gesell argued against this because he observed only a small 'v' wave in the venous pressure trace during asynchronous contraction (later confirmed by Sarnoff in 1962 [17]), and in a later experiment [5] he showed that cardiac output varied with the type and strength of atrial contraction. Only recently have imaging techniques confirmed the absence of significant mitral regurgitation during atrioventricular synchrony [18]. Gesell showed also that when atrial contraction occurred against a closed atrioventricular valve a large 'a' wave was seen in the venous pressure trace [6]. Other studies have shown retrograde flow into the vena cava [19] and pulmonary veins [18] under these circumstances which further decreases atrial volume available for subsequent ventricular filling. The second explanation, therefore, for Gesell's original findings was that atrial systole does indeed augment cardiac output by its effect on ventricular filling. Repeated experiments in isolated mammalian heart preparations confirmed these views, although the magnitude of the observed effect varied [7, 8].

The factors influencing the relationship of atrial systole to cardiac output have also been studied using experimental models. Gesell and others [6, 7] clearly showed an increased effect of properly timed atrial systole with increased venous return and a diminished effect with vagal stimulation. It was shown that atrial muscle responded in much the same way as ventricular muscle to manipulations of the sympathetic nervous system via the carotid sinus [20] or stellate ganglion [21]. Indeed there are many similarities between ventricular and atrial haemodynamics. For example, ventricular function is influenced by after load, similarly, atrial function is influenced by ventricular end diastolic pressure. Thus Linden & Mitchell [22], in anaesthetized open-chested dogs with surgically induced complete heart block, showed that the relationship between left ventricular fibre length and cardiac output (by Starling) was influenced by the prevailing end diastolic pressure. At low left ventricular end-diastolic pressure (LVEDP), atrial systole caused greater changes in left ventricular fibre length compared with those at high LVEDP. Therefore atrial systole had a smaller effect on cardiac output when LVEDP was high. This was confirmed by Greenberg et al., [23], who showed that the atrial contribution to cardiac output during atrioventricular synchronous pacing in patients early after cardiac surgery was inversely related to the pulmonary wedge pressure (an indirect measure of left ventricular filling pressure). More recently, Doppler echocardiographic measurements of transmural flow showed that the ratio of passive to active transmural flow was inversely related to LVEDP measured simultaneously at the time of cardiac catheterization [15].

Another important factor in optimizing the effect of atrial systole on cardiac output is the timing of the atrial contraction relative to ventricular contraction. Gesell [2] first showed that timing was important, and in the isolated dog heart he found that atrial systole was best placed 0.0080–0.02 s before the onset of ventricular systole. Jochim [8], in the open-chested dog, found it was most effective at 0.08 s from the onset of ventricular systole. Benchimol et al. [24] showed that in man very long and very short PR intervals had little effect on cardiac output. Using atrioventricular synchronous dual-chamber pacing systems it has been further critically evaluated. The optimum atrioventricular interval appears to be between 50 and 170 ms but there is marked individual variation [10, 25, 26].

Although in the experimental animal model atrial systole appears to contribute between 20% and 50% to cardiac output, there has been much debate as to the value of its contribution in intact man. It appears that the ability of atrial contraction to augment cardiac output in man is dependent on the haemodynamic state of the ventricle. There have been few studies in normal man, but Benchimol et al. [27] showed that at rest atrial systole appeared to be of little importance in maintaining basal cardiac output. Recent echocardiographic studies of transmural flow also suggest that its contribution at rest is low [28, 29] but is age related, increasing from about 10% at age 20 to 46% at age 80 years [28]. This change may be related to decreased ventricular compliance with age. When the ventricle is damaged atrial systole appears to play a much larger supportive role.

**ATRIAL SYSTOLE IN VENTRICULAR DISEASE**

When the left ventricle is damaged, for example after myocardial infarction, it has been shown that atrial systole can support cardiac output, contributing up to 50% [30, 31], although as previously mentioned this is dependent on LVEDP [15, 22]. In some studies its contribution was not apparent at rest but became obvious on exercise as in atrial fibrillation where the absence of effective atrial contraction is associated with impaired effort tolerance [12–14]. When atrial fibrillation was first studied it was thought that this was due to the uncontrolled ventricular rate [11], but other studies have shown that when atrial fibrillation is converted to sinus rhythm with little change in heart rate there is still a measurable difference in cardiac output especially on exercise [12–14]. Moreover, successful drug therapy for atrial fibrillation has improved ventricular rates and symptoms of palpitation without improving exercise capacity [32]. These data would suggest that the role of atrial systole in cardiac physiology is more important during exercise. Recent studies with Doppler recordings of transmural flow in normal subjects have indeed shown that the contribution of atrial systole to ventricular filling is increased proportionately more than that of passive filling during mild exercise [29].

The value of correct atrioventricular synchrony to cardiac function has generated much interest recently because of the development of dual-chamber atrioventricular synchronous and single-chamber rate-responsive pacemakers. Most studies comparing various pacing modes have shown clear evidence in favour of rate responsiveness on exercise (versus fixed rate pacing [9, 10, 33, 34]) but little evidence in favour of atrioventricular synchronous systems or 'physiological pacing' over ventricular only rate-adaptive systems [35, 36]. The physio-
logical changes that occur on exercise are different in the three pacing modes. With fixed-rate pacing cardiac output rises during exercise by means of an increase in stroke volume. With single-chamber rate-responsive pacing, the heart rate is increased by the pacemaker in response to changes in activity, respiration or electrocardiographic parameters which themselves change with sympathetic drive, for example the QT interval. These rate-responsive pacing systems therefore produce an increase in cardiac output by increasing both the rate and also the stroke volume of the heart. In atrioventricular synchronous pacemakers the heart rate increases with exercise because increased atrial activity is sensed by the pacemaker and the ventricle is then paced after an appropriate time interval at the equivalent rate to the atrial activity. This utilizes the atrial contribution to cardiac output and the ventricular work of exercise is thereby minimized [37]. Theoretically therefore, atrioventricular synchronous pacing should be the most beneficial pacing mode for patients with impaired left ventricular function [38].

In summary, the factors which influence atrial physiology are similar to those which affect ventricular dynamics. The haemodynamic importance of correctly timed atrial systole in overall cardiac function is dependent on the functional reserve of the ventricle. When the ventricle is impaired, atrial systole can play an important supportive role in maintaining cardiac output. Non-invasive assessment using cardiac ultrasound may help in identifying those patients likely to benefit haemodynamically from atrioventricular synchronous pacing. It should also increase our understanding of the role of atrial systole in healthy and diseased hearts.

REFERENCES


