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Atrio-Ventricular Pressures and Their Relationships During Stellate Stimulation

Richard H. Ulmer
Loyola University Chicago

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**ATRIO-VENTRICULAR PRESSURES AND THEIR RELATIONSHIPS
DURING STELLATE STIMULATION**

by

Richard H. Ulmer

**A Thesis Submitted to the Faculty of the Graduate School
of Loyola University in Partial Fulfillment of
the Requirements for the Degree of
Master of Science**

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BIOGRAPHY

Richard H. Ulmer was born in Chicago, Illinois, November 11, 1936.

He was graduated from St. Ignatius High School, Chicago in June, 1953. In September of the same year, he entered Xavier University, Cincinnati, Ohio. Here he received his undergraduate training, and was graduated in June, 1957, with the Honors A.B. degree.

He enrolled in the Stritch School of Medicine of Loyola University, Chicago in September, 1957. During his sophomore year in Medical School he began work in the Graduate School of Loyola University.

At the present time, he has received an appointment to a rotating internship at the University of Chicago Clinics.

INTRODUCTION

The influence of the sympathetic cardiac nerves on heart rate and force of ventricular contraction has been investigated and described in great detail (1-6). It has been shown that sympathetic stimulation markedly increases both the rate and force of ventricular contraction, shortens the duration of systole, and decreases ventricular circumference. All these phenomena represent basic changes in myocardial contractility and result in significantly increased systolic ejection. Both right and left ventricles may respond simultaneously and in similar manner to stimulation of either right or left stellate ganglion, thus eliciting marked elevations in both pulmonary and aortic arterial pressures (6). Such augmented ventricular contractions with resultant increase in output must inevitably decrease the systolic residue to very low levels unless appropriate adjustments are made in the flow of blood from the atria and associated venous systems.

The contribution of atrial systole to ventricular filling was investigated by Y. Henderson in 1906 (7). Analysis of cardiometer tracings in the dog led him to conclude that the major portion of ventricular filling occurs early in diastole, with auricular systole contributing less than one cc. of blood. Gesell (8) critically reviewed this work and concluded that because of the insensitive thick rubber membrane employed, Henderson's cardiometers were unable to record the volume increment caused by atrial systole. By inducing atrial fibrillation, Gesell recorded a decline in arterial pressure

of 10-15 per cent of the control values. This was followed by a slow return of the arterial pressure to normal, as the venous pressure rose until a balance was reached. "This illustrates a very important function of the auricles, namely, an adequate filling of the ventricles with a comparatively low venous pressure — thus preventing a continued strain upon the venous system." (8). Straub, using a cardiometer with a more delicate membrane, observed ventricular filling during early diastole, the period of diastasis, and during auricular systole (9).

Wiggers and Katz determined that the amount of ventricular filling caused by atrial systole depends on the time that atrial systole comes in ventricular diastole, the completeness with which the ventricle has already filled prior to atrial systole, and on the vigor of atrial systole (10).

Little, et.al. (11-13) recorded pressures from both atria and also measured the distensibility characteristics of the atria. They found that 90 per cent of their animals exhibited a pressure gradient from the left to the right atrium during the major part of the cardiac cycle. They attributed this pressure differential to the fact that the right atrium is a more distensible chamber than the left, and also has an average volume twice that of the left atrium (7.0 vs. 3.4 cc.) (12).

Recent work by Sarnoff, et.al. has demonstrated important alterations in atrial hemodynamics during stimulation of the sympathetic nerves to the heart (14-19). During stellate stimulation, they observed atrial augmentation,

declining mean atrial pressures, and a constant mean pulmonary arterial pressure. There was also a lowering of left ventricular end-diastolic pressure. Similar changes were found after the administration of norepinephrine.

In a group of pilot experiments, we were able to confirm some of these findings. We did not observe the consistent decline in mean atrial pressures during stellate stimulation. A consistent increase in mean right ventricular pressure was also noted in our experiments, in contrast to the constant pulmonary arterial mean pressure recorded by Sarnoff, et.al. (18). Thus, it was determined to further investigate atrial hemodynamics and atrio-ventricular pressure relationships during stellate stimulation. This serves as the principal objective of this thesis.

MATERIALS AND METHODS

1. Animals and Operative Technique

Twenty-six mongrel dogs of both sexes were anesthetized with Nembutal (32.5 mg./kg.). Under positive pressure respiration, mid-sternal thoracotomies were done, unipolar electrodes positioned on both the left and right stellate ganglia, and all four chambers of the heart cannulated. In order to place the diaphragm of the transducer on the same horizontal plane as the tip of the atrial catheter, the first ribs were removed bilaterally. Each ventricle was cannulated with a light, freely movable teflon cannula which pierced the ventricular wall so that the cannula tip was fixed in position flush with the endocardium (20). Atrial pressures were recorded from polyethylene tubing which pierced the atrial wall with the tip suspended in the atrial chamber. Clotting was prevented by the use of heparinized saline in the cannulae. In twelve dogs, both vagus nerves were isolated in the neck and sectioned during the experiment. In some experiments, the force of atrial contraction was monitored by means of a Walton strain gauge arch (21) sutured to the atrium.

2. Recording Technique

All records of blood pressure from the four cardiac chambers were obtained by means of a Statham P23A transducer coupled to a 5P1 Grass polygraph preamplifier which in turn was coupled to a Grass model 5A driver amplifier. The driver amplifier was coupled to the Grass direct writing oscillograph.

The P23A pressure transducer has a nominal pressure range of zero to seventy-five centimeters of mercury and an approximate natural frequency of thirty-nine cycles per second. The 5P1 polygraph preamplifier is a chopper modulated and demodulated high gain, low noise, low frequency D.C. preamplifier which has a frequency response to forty cycles per second. Drift is less than three microvolts per hour, and is random. The input is designed to drive balance controls and to excite the strain gauge transducer for pressure. The model 5A amplifier is a push-pull, two stage direct coupled amplifier with a differential input. Its primary function is to amplify signals from the polygraph preamplifier sufficiently to drive the direct writing oscillograph. It also supplies voltages to operate the associated preamplifier. The maximum sensitivity of the driver amplifier combined with the pen writer oscillograph is greater than one hundred millivolts per centimeter. The entire system has a frequency response of forty cycles per second.

The frequency response of the recording system was checked by recording square-wave impulses from a Grass model S5 stimulator. It was found that the entire polygraph system was able to accurately record square-wave forms up to twenty-five cycles per second, and was overdamped beyond this frequency range.

The atrial or ventricular cavity plus the cannula, connecting tubing, and transducer cap form a closed fluid system. Any alteration in pressure of this fluid is transmitted directly to the diaphragm of the transducer.

The transducer is a strain gauge type in which a strain wire resistor is attached to the center of the diaphragm. This serves as the variable resistance in a bridge circuit. Changes in the diaphragm change the length of the wire gauge and thus alter its resistance. The electrical output of the transducer is then amplified to drive the pens of the oscillograph.

The pressure pulses recorded from the atria using this recording system were similar in form and in pressure levels to the pressures recorded by previous investigators of atrial hemodynamics (11, 13, 22-24).

The Walton strain gauge arch employed to record the force of atrial contraction consists of a strain wire resistor held in place on a bridge-shaped metal arch. As this arch is distorted by atrial contraction, the length of the resistor wire, and thus its resistance, is altered. The resistor wire serves as the variable resistance in a bridge circuit, the electrical output of which is fed into the polygraph and recorded. The record obtained is a qualitative record of atrial contractile force.

In some experiments, one standard limb lead of the electrocardiogram was recorded for purposes of timing the various components of the pressure waves.

A Grass model S5 stimulator was used to stimulate the stellate ganglia, thus providing isolated square-wave pulses having a duration of five msec., frequency of 10/second, and intensity of 3.0—4.0 volts.

3. Experimental Design

The animals were prepared as outlined above. Control records were made, and then the right stellate ganglion was stimulated. Recordings were

continued throughout the period of stimulation and recovery from stimulation at paper speeds of 2.5 mm/second. Following recovery, the same procedure was repeated using a paper speed of 25 mm/second to permit critical analysis of simultaneous events in the several cardiac chambers. Similar procedures were carried out during stimulation of the left stellate ganglion. This procedure was employed in 14 dogs.

In the remaining twelve dogs, the procedure as described above was followed by transection of both vagus nerves high in the neck, and the entire stimulation procedure was repeated. Pressures were recorded with the Grass amplifier " $\frac{1}{2}$ Amp. Hi Freq. Switch" set at 60 cps., and then switched rapidly to 0.5 cps. By this method, full scale pressure recordings could be made alternately with mean blood pressure recordings. Calibration and balance of the polygraph were routinely checked after each set of recordings.

RESULTS

Figure 1 illustrates the simultaneous pressure changes in all four chambers of the heart during electrical stimulation of the left stellate ganglion in an animal with intact vagi. Not only did intraventricular pressures increase remarkably, but there was definite elevation in amplitude of atrial pressure waves as well, with a somewhat greater response in the left atrium. Mean atrial pressures were alternately recorded. It is apparent that both left and right mean atrial pressures rose in this experiment, but this was not a constant response in all animals. In some, the mean pressures rose, in others they remained constant, and in still others they declined. In nearly all experiments the amplitude of atrial pressure waves increased, regardless of how the mean atrial pressures changed. In this experiment (fig. 1), heart rate increased from 160 to 180 per minute. All pressures returned to normal within 2 minutes after the stimulus was turned off.

Stimulation of the right stellate ganglion with vagi intact resulted in increased amplitude of the pressure waves in all four chambers of the heart (fig. 2). Acceleration in rate was generally more prominent, and right atrial pressures frequently showed relatively greater elevations. Left ventricular pressure increases, on the other hand, were less marked during right stellate stimulation. In figure 2, mean pressures were recorded alternately with pulsatile pressures. The influence of the right stellate in this animal is immediately apparent in comparing the degree of augmentation

Figure 1

Simultaneous pressure changes in the four heart chambers during electrical stimulation of the left stellate ganglion.

Figure 2

Simultaneous pressure changes in the heart during stimulation of the right stellate ganglion.

of the right ventricle with the left. Note that mean pressure increased 12 and 10 mm Hg in right and left respectively. These ventricular mean pressure responses were observed in nearly all dogs. In the right atrium, mean pressure remained essentially unchanged while rising 2 mm Hg in the left atrium.

Figure 3 illustrates an experiment in which only mean pressures were recorded during the stimulation period. The animal was bilaterally vagotomized. Mean right atrial pressure rose less than 1 mm Hg and right ventricular mean pressure rose 4 mm Hg. Mean left atrial pressure declined 1.7 mm Hg while mean left ventricular pressure rose 16 mm Hg. After the stimulus was turned off, full amplitude waves were recorded from the ventricles to show that augmentation had occurred. It is especially interesting that right atrial pressure started to rise promptly following application of the stimulus while a lag of 5 seconds occurred before left atrial pressure began to decline. This record thus illustrates a response in which mean atrial pressures progress in opposite directions.

Declining mean left atrial pressures during stellate stimulation was a more consistent, although still not invariable, characteristic of the vagotomized animals. The rate and amount of decline varied from animal to animal. Right atrial mean pressure alterations showed greater variability and were not consistently altered by vagotomy.

Recordings were made at higher speed to permit more precise timing and analysis of the atrial pressure waves (fig. 4). Control tracings showed

Figure 3

Mean pressure changes in the heart during stimulation of the left stellate in a bilaterally vagotomized dog. Ventricular pulsatile pressures were recorded after stimulation to show augmentation in the ventricles.

typical multiple waves related successively to atrial contraction (a-wave), a small c-wave concurrent with ventricular isometric contraction, and a slowly rising atrial filling (v) wave. Simultaneous points on the pressure traces reveal clearly that the a-wave precedes contraction in the ventricle, and this relationship was thoroughly confirmed by the ECG. Further examination of the a-wave revealed that the rate of increase in atrial pressure was accelerated during stellate stimulation resulting in a more peaked wave of greater amplitude and shorter duration. This was more prominent in the right atrial trace than in the left atrium, and culminated in a peak pressure in the a-wave of the right atrium somewhat in advance of that in the left atrium. It appeared that the right atrium contracted more rapidly than the left. An outstanding feature of the atrial response was a rapid and dramatic fall in the descending limb of the a-wave in both atria. This occurred during the period of isometric contraction in the ventricle and frequently marked the c-wave. Its termination represented the lowest atrial pressure during the cycle.

Mean left atrial pressures were generally higher than those in the right atrium and the amplitudes of the a-wave were greater. The c-wave was not prominent, and in spite of profound increases in left ventricular pressures, it generally was not markedly accentuated during stimulation. If the c-wave is indeed caused by bulging of the A-V valves, one should expect higher ventricular pressures to produce more prominent c-waves during augmentation. This did occur in a few experiments.

Figure 4

Fast pressure traces (25 mm/sec) recorded during stimulation of the right stellate. Segments of the original record have been reassembled with total elapsed time in seconds from start of stimulation shown at the top of the figure.

Finally, a strain gauge arch was carefully sutured to the atrium in vagotomized dogs, and atrial contractile force directly recorded during cardiac nerve stimulation simultaneously with atrial and ventricular pressures (fig. 5). A definite increase in force of contraction together with increased amplitude of the a-waves were superimposed on a progressively declining mean atrial pressure. These changes in atrial dynamics were accompanied by the familiar elevation in left ventricular pressure. Fast tracings revealed that the onset of augmented atrial force faithfully accompanies the first appearance of increased pressure of the a-wave, reached a plateau, and progressively declined in parallel fashion after stimulation ceased. Thus, it was conclusively shown that electrical stimulation of the stellate ganglia induced more powerful contraction in atrial muscle as well as in ventricular muscle.

Figure 5

Simultaneous recording of left atrial pressure and force of contraction together with ventricular pressure during stimulation of the right stellate ganglion.

DISCUSSION AND CONCLUSIONS

The elevation in ventricular pressures during stimulation of the cardiac sympathetic nerves confirmed the reports of other workers (2, 5). The changes in atrial pressures reported here and by Sarnoff, et.al. represent an important extension of knowledge of the cardiac responses to sympathetic excitation.

Little, et.al. (11-13) have very carefully set forth a detailed analysis of the individual components of atrial pressure tracings: "Pressure recorded at the beginning of atrial systole is in part a function of the elastic properties of the combined atrial-ventricular-venous system as well as the rate and amount of filling of the ventricle during diastasis. The pressure at the peak of atrial systole is influenced by the muscular contraction of the atrial myocardium and the distensibility of the ventricle. The pressure measured just before the beginning of ventricular systole is chiefly dependent on the volume-elastic characteristics of the common venous-atrial-ventricular cavity. Pressures recorded just before the second heart sound are a function of the distensibility of the atrial-venous system, the length of ventricular systole, and the rate of venous return." (13). Mean atrial pressure is clearly a composite of all these dynamic factors operating during a series of cardiac cycles.

The present experiments demonstrate significant alterations in atrial hemodynamics during electrical excitation of the sympathetic innervation of the heart. In accordance with Little's second point, a marked increase in

amplitude of the a-wave can only be explained by an increase in the velocity and force of contraction of the atrial myocardium. Direct measurement of the pressure gradient from atrium to ventricle at the peak of atrial contraction reveals a significant increase in filling pressure as a result of atrial augmentation. Table I includes data from a number of representative experiments in which the pressure gradient is illustrated. It is evident from this table that considerable variation exists in the magnitude of pressure change elicited by stellate stimulation, and that no important difference exists between the response of the right and left atria or in the resultant atrio-ventricular gradients. Linden and Mitchell demonstrated that atrial systole causes a substantial increase in ventricular diastolic pressure and on myocardial segment length when the ventricle is on the sensitive part of its pressure-length curve (16). Although we did not measure segment length in these experiments, we have confirmed the influence of atrial systole on right ventricular diastolic pressure.

Augmented ventricular contraction results in more complete systolic emptying (6) and markedly accelerated rate of ventricular relaxation during stellate stimulation (20) and during administration of epinephrine (25, 26). Thus there is less impedance to the inflow of blood during the early period of ventricular diastole. Ventricular filling is improved both early and late in diastole. The most logical mechanism translating increased impulse traffic in the sympathetic cardiac nerves into more powerful muscle contraction involves the release of norepinephrine (27, 28) with its direct action on excitability, conductivity, and contractibility.

TABLE I

Changes in Pressure Gradient (mm. Hg.) from Atria to Ventricles

During Stellate Stimulation

RA - RV			LA - LV		
Control	Stimulation	Change in Gradient	Control	Stimulation	Change in Gradient
3	10	7	2	7.5	5.5
2	6	4	3	4	1
5	5	0	5	7	2
4	7	3	2.5	5.5	3
2.5	4	1.5	2	4.5	2.5
5	7	2	1	6	5
3	5	2	4	8	4
2	3	1	3	3	0
2	3.5	1.5	3	2.5	-0.5
MEAN	3.2	5.6	2.4	2.8	5.3
					2.5

The variability of atrial mean pressures as recorded in these experiments differs significantly from the findings of Sarnoff's group in which mean atrial pressures fell markedly during stellate stimulation. It was thought that vagotomy might be the factor responsible for the difference between the two sets of results, but our series of vagotomized dogs did not show consistent decline in mean atrial pressures during either right or left stellate stimulation. In many cases, post-vagotomy pressure records were identical with pre-vagotomy records. In no instance did we observe the magnitude of decline in left atrial mean pressure shown by Sarnoff, et.al. who found little alteration in pulmonary artery pressure; we consistently observed an increase in right ventricular mean pressure. This increase more than offset any elevation in mean left atrial pressure and significantly increased the pressure gradient across the pulmonary vascular bed.

It might appear that increasing mean left ventricular pressure would offset any advantage which might accrue from increasing the pressure gradient from right ventricle to left atrium. This is not true, however, since the increase in left ventricular pressure occurs in the systolic portion of the cycle while the diastolic pressure remains constant or falls slightly. Since flow from the atria to the ventricles occurs during ventricular diastole, there is no increased resistance to flow into the ventricles. On the right side of the heart, the factors of increased ventricular distensibility and augmented atrial contraction would also enhance ventricular filling.

A possible explanation for the variable changes in mean left atrial pressures during stimulation of the stellate ganglia is the fact that in the

dog these ganglia send vasomotor fibers to the pulmonary vascular bed (29) as well as augmentor and accelerator fibers to the heart. The case for an active control of the pulmonary vascular bed by nervous stimuli rests upon the establishment of the fact that pulmonary vasomotor responses can be demonstrated when all passive regulatory mechanisms have been eliminated. In a carefully designed series of experiments, I. de Burgh Daly demonstrated that: "In experiments on atropinized perfused lungs, stimulation of the sympathetic pulmonary nerve path has raised the pulmonary vascular resistance sixty per cent or more in the absence of a functional bronchial vascular system and of a change in lung hindrance. . . It is therefore difficult to escape the conclusion that, in the dog, the upper thoracic sympathetic outflow contains constrictor fibers to some portion of the pulmonary vascular bed." (30). Daly also found evidence of vasodilator fibers in the thoracic sympathetic nerves, the cell bodies of which were located in the stellate ganglia. "Finally, mention should be made of one curious feature of the results, namely, the limitation of pure pulmonary vasodilator responses to stimulation of nerves on the left side of the body. . . The predominance of dilator responses to stimulation of the nerves on the left side is statistically significant. We have no grounds for the belief that this result has its origin in an experimental error, and the only reasonable explanation we have to offer is that it is due, during ontogenesis, to the development of an unequal distribution of pulmonary vaso-constrictor and -dilator fibers to each side of the body." (31). Thus, there is an anatomic basis for the

variation in left atrial mean pressure response to stellate ganglion stimulation. A consistent pulmonary vasoconstriction would be expected to elicit a fall in left atrial mean pressure. A dilatation, on the other hand, would contribute along with augmented atrial contraction to an increase in mean left atrial pressure.

The fact that the c-wave was not prominent in the left atrial pressure pulses and was generally not markedly accentuated in spite of profound increases in left ventricular pressures was an unexpected finding. One would expect this to be a prominent wave in the left atrial pulse because of the higher pressures achieved in isometric contraction of the left ventricle as compared to the right ventricle. Other investigators, recording left atrial pressure pulses during cardiac catheterization of human subject, have noted that the c-wave was barely perceptible or absent from their left atrial pressure pulses, but was consistently present in right atrial pressure pulses (23, 24). The explanation of this finding is not apparent, but the classic explanation that the c-wave is due to A-V valve bulging during ventricular isometric contraction does not seem entirely satisfactory.

Examination of the fast speed record (fig. 4) of the atrial pulses reveals that the duration of atrial systole was shortened while augmentation of the a-wave occurred. These atrial changes result in a more peaked wave of greater amplitude and shorter duration and reflect the same basic alterations in atrial dynamics reported previously in the ventricles (20).

They also confirm the changes induced in the cardiac cycle which are tabulated by Sarnoff, et.al. (18).

SUMMARY

- I. Pressures were recorded simultaneously from the four cardiac chambers in anesthetized open-chest dogs during electrical stimulation of the stellate ganglion before and after bilateral vagotomy.
- II. Augmentation of atrial contraction was shown to accompany that in the ventricles by increased atrial pressures as well as by the direct recording of atrial force. Fast speed records revealed that the atrial augmentation was primarily in the a-wave.
- III. In some experiments mean atrial pressures increased slightly, in others declined during stimulation. There was no consistent relationship between the change in left and right atrial pressures, although they showed simultaneous augmentation in amplitude of the a-wave.
- IV. Both right and left mean ventricular pressures consistently increased during stellate stimulation.
- V. Pulmonary vasomotor fibers which have their cell bodies in the stellate ganglia can exert a strong influence on left atrial mean pressure during stellate ganglion stimulation. These cell bodies are stimulated along with cardiac accelerator and augmentor cells. Activation of constrictor fibers would cause a fall in left atrial mean pressures; activation of pulmonary vasodilator fibers would cause the opposite effect.

- VI. The augmented a-wave resulted in a significant increase in the atrio-ventricular pressure gradient during late diastole, and thus contributed to increased ventricular filling.

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APPROVAL SHEET

The thesis submitted by Richard H. Ulmer has been read and approved by three members of the faculty of the Stritch School of Medicine of Loyola University.

The final copies have been examined by the director of the thesis and the signature which appears below verifies the fact that any necessary changes have been incorporated, and that the thesis is now given final approval with reference to content, form, and mechanical accuracy.

The thesis is therefore accepted in partial fulfillment of the requirements for the Degree of Master of Science.

May 29, 1961
Date

Walter C. Randall
Signature of Adviser