

Circulation Research

JOURNAL OF THE AMERICAN HEART ASSOCIATION

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Circ. Res. 1964;14:387-391

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75214

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Direct Detection and Timing of Aortic Valve Closure

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With the technical assistance of Mr. Ivan Harvey

■ The aortic incisura and the second heart sound have been considered simultaneous with and caused by aortic valve closure for many years. While Wiggers¹ believed in a noiseless approximation of the semilunar valves and that after-vibrations of the *closed* valves and aortic blood column caused the second sound, the incisura was related to semilunar valve closure.² Thus, subsequent studies relating the second heart sound to the incisura associated the sound with closing of the valve.³⁻⁵ However, the possibility of an earlier closure of the valve has been suggested by recent cineangiographic studies,⁶ by ventricular-aortic pressure gradient reversal prior to the incisura,⁷ and by the presence of backflow in the aorta several milliseconds before the incisura.⁷ In order to clarify this, a means of directly detecting aortic valve closure seemed necessary. Additional requirements were that normal valve movements and vascular pressures and functions be maintained. Toward this end, an electrical contacting device and techniques for its insertion between the leaflets of the aortic valve in closed chest, anesthetized dogs were developed.

Methods

Two 28-gauge stainless steel wires, 80 cm

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Dr. MacCanon is the recipient of Research Career Program Award, HE-K3-13, 433, from the National Heart Institute, U. S. Public Health Service.

Dr. Arevalo is a Postgraduate Trainee with Grant H-5182 from the National Heart Institute, U. S. Public Health Service.

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Received for publication October 3, 1963.

long, were cemented together with epoxy leaving a 0.5 mm separation between them. A 3 cm contactor area was left bare just above the tip bead of epoxy. The general orientation is shown in figure 1. At the other end, the wires were left free for 20 cm providing external connections to the contactor. The device was inserted through a compression catheter adapter, luerlok fitting, and no. 10 cardiac catheter, so that the 3 cm contactor was exposed beyond the tip of the catheter. Additional epoxy was used to prevent shorting by the metal adaptors and to prevent leakage. All parts, except the contactor and lead tips, were sprayed with insulating varnish. Tested *in vitro*, the force required to close the contact was 9.5 grams.

The contacting device was used to time the relationship between aortic valve closure and the

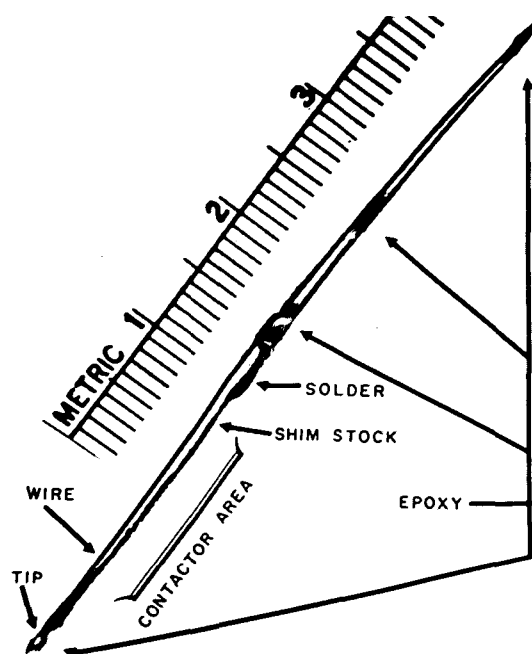


FIGURE 1

Photograph of tip of latest model of contactor device showing orientation of contacts and points of epoxy cement.

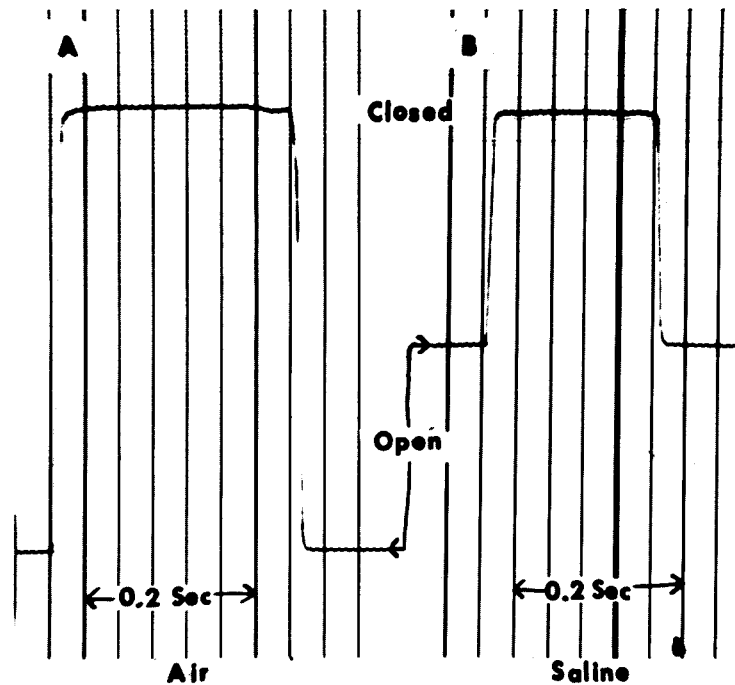


FIGURE 2

A. Tracing showing recorded deflections resulting from closing and opening the contactor in air and B, in a saline (0.9%) medium. Note sharpness of closure deflection, 10 and 7 msec response times, respectively, rounded shoulder of the trace near end of deflection, absence of any deflection prior to actual contact, slight shift in tracing A as the area of contact is changed slightly, and decreased response which results from immersion in a conductive medium.

aortic incisura in 11 adult mongrel dogs. The animals, in a postabsorptive state, were anesthetized intravenously with sodium pentobarbital (12.5 mg/kg) 30 minutes after premedication with morphine sulfate (10 mg/kg subcutaneously). Supplemental morphine (5 mg/kg) was given at two-hour intervals. Additional small doses of pentobarbital were given when required.

The contactor catheter was inserted into a common carotid artery and the animal heparinized (10 mg/kg). This catheter was guided into the left ventricle under fluoroscopic visualization so that its tip was just inside the aortic valve. A second no. 10 pressure catheter was passed through a femoral artery or the other common carotid artery and its tip positioned in the aortic root touching the aortic valve. The tip of the second catheter thus clearly marked the location of the valve. (When both common carotid arteries were used, disturbing carotid sinus reflexes were avoided by perfusing the peripheral common carotid arteries with blood from the femoral arteries.) The contactor was then inserted into the lumen of its catheter to its previously determined distance, and connected through a Wheatstone bridge to a Sanborn carrier preamplifier (350-1100 B). After balancing the bridge, the

catheter was carefully withdrawn until the contactor was located in the aortic valve. The pressure catheter was then withdrawn until its tip was approximately 1 cm from the valve. The contactor catheter was then carefully rotated until sharp and distinct deflections with stable intervening segments were observed on the monitor screen indicating that the contactor was properly oriented between valve leaflets.*

Aortic pressures were recorded using Statham P23D pressure transducers and carrier preamplifiers. Phonocardiograms were recorded from the third left interspace using a Sanborn dynamic microphone and heart sound preamplifier. High pass filtration at 200 cycles/sec (-24 db per octave) was used to clearly delineate the large high frequency vibrations commonly associated with valve closure. The tracings of pressure, heart sounds, and changes in electrical conductivity resulting from opening and closing of the contactor in the blood media were simultaneously recorded using a Sanborn photographic recorder

* A 0.1 × 3 cm piece of 5 mil thick shim stock substituted for one of the contacts in a later model greatly aided proper orientation (fig. 1).

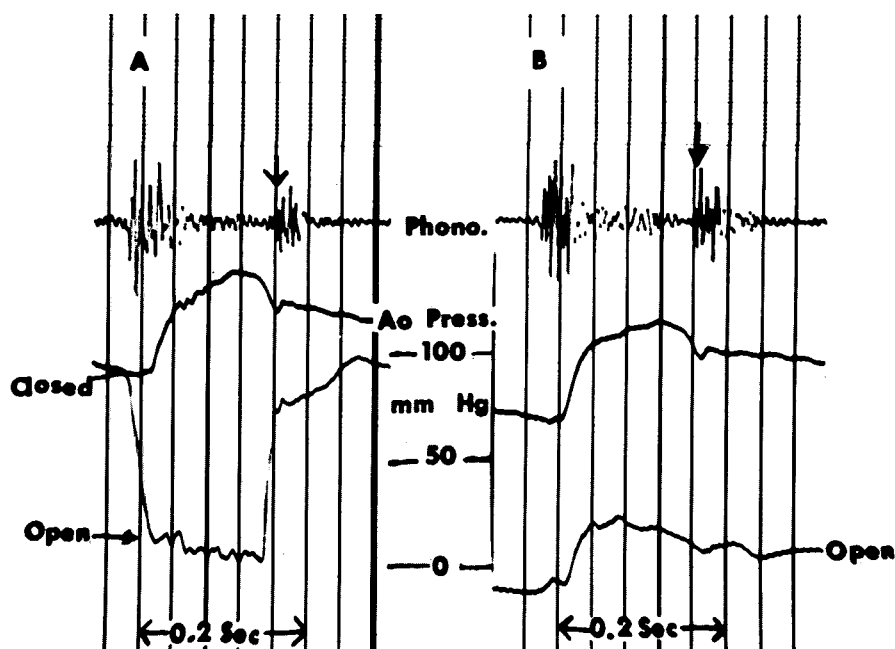


FIGURE 3

A. Sample tracing showing aortic pressure (Ao Press.) and phonocardiogram (Phono.) simultaneously recorded with conductivity changes resulting from opening and closing of the contacts (lower tracing). B. Sample of recording obtained a few minutes after A, after contactor device had been withdrawn from aortic valve into ascending aorta.

(550 M) at a paper speed of 200 mm/sec. The time difference from valve closure to the incisura was measured by two independent observers and compared for possible discrepancy. The rapid paper speed and sharpness of the deflections permitted a reading accuracy of 1 msec. The delay in pressure transmission of the catheter recording system was determined for each catheter and the presented data have been corrected for this delay.

Results

Typically, the changes in conductivity resulting from closure of the contacts produce a clear and distinct upward deflection. Examples of the contactor's response in vitro are shown in figure 2. The 10 and 7 msec response times and rounded shoulders of the trace near the end of each deflection indicate the presence of capacitance effects in the system. The instant of contact closure, according to electronic theory, is marked by the beginning of the sharp upward deflection. The absence of any change prior to the sharp upward deflection, plus the repeated observation that the elements of the contactor must

touch each other to produce the response, supports this view. In addition to attenuation of the signal in the conductive media of the blood it was anticipated that additional capacitance effects might alter the rise time in vivo, that vascular volume changes might add impedance factors, and that complicating electrical potentials might be generated by blood flowing through the field around the contactor elements. It was also expected that changes in the area of contact would cause some shifting in the recorded tracings.

That none of these factors obscured the typical sharp contactor response is evident in figure 3A. The beginning of the sharp upward deflection at the end of systole clearly marks the instant that the valve leaflets were approximated with sufficient force to close the electrical contact. On the sample tracing (fig. 3A) this occurred 15 msec before the recorded incisura. Subtracting 8 msec for the measured delay in pressure transmission shortens the interval to 7 msec. Withdrawal of the contactor from the valve abolished the

pattern seen in figure 3A and the tracing in figure 3B was obtained. While minor shifts due to impedance changes and flow effects are evident (fig. 3B), they differ markedly from the deflections which characterize closure of the contacts. Minor shifts in the tracing due to changes in contact area, impedance, or flow are also visible in figure 3A.

Simultaneous recordings of contactor signals and aortic pressures, in the 11 dogs, showed that the aortic valve was shut with sufficient force to close the contact an average of 7.8 msec (range 5 to 13 msec) before the incisura.

While the downward deflection in the conductivity trace at the beginning of systole seen in figure 3A might also seem useful to time the opening of the aortic valve, this was not usually the case. Ordinarily, this deflection had a rounded character making determination of its onset uncertain. Even in cycles where the start of descent was clear, there was no consistent time relation to events in the aortic pressure tracing. It seems probable that the sudden downward movement of the base of the heart at the beginning of systole,⁸ the impedance changes, and flow effects mentioned earlier complicated matters, rendering the contactor response unreliable at this time.

Discussion

The presented evidence that aortic valve closure occurs, at least 5 to 13 msec before the aortic pressure incisura, is an interesting phenomenon which has not been previously reported. This supports the concept that, when systolic ejection ceases, the valve closes as soon as ventricular pressure drops slightly below aortic pressure.⁹ Actually, measurements from recordings of carefully superimposed equisensitive aortic and ventricular pressures reveal that the pressure gradient from aorta to left ventricle 8 msec before the incisura is approximately 2 mm Hg.⁵ However, the demonstration of such a reversed pressure gradient during a large portion of late systole^{5, 7} makes efforts to relate such measurements to valve closure meaningless. On the

other hand, the calculated radius of curvature of 0.6 cm for the aortic valve cusps to obtain a circumferential tension of 9.5 g with a pressure gradient of 2 mm Hg does not seem unreasonable [$Tc (9.5 \text{ g}) = 2\pi r TM (2 \text{ mm Hg} \times 1.36)$].

The time interval between aortic and ventricular pressures at the level of the incisura (due to delay in pressure transmission to the aorta and "inertance" and compliance factors⁷) averages 9.8 msec (range 5 to 13 msec), nearly the same as the interval from contact closure to incisura. This implies that valve closure is more correctly related to blood movement. Therefore, it seems likely that the aortic valve closes the instant backflow commences, and that a large fraction of aortic backflow is due to aortic and valvular stretch and distention after the valve is closed.

The rapid cineangiographic films of Criley et al.⁶ showing that the start of the second heart sound coincided with a sharp descent and distention of the valve and aortic root several milliseconds after valve closure, plus confirmed association of the second sound with the incisura, supports this view.

In addition, Faber and Purvis⁴ recently concluded that the second heart sound originates and is transmitted as a transverse vibration of the aortic wall, which is inseparable from the high frequency components of the dicrotic notch (aortic incisura). Also, the transverse diameter tracings of Mallos,¹⁰ although limited in frequency response, followed internal arterial pressure changes very closely. Thus, Wiggers' belief that the second heart sound results from after-vibrations of the aortic blood column, walls, and valves, after a noiseless valve closing would seem to be substantiated. This view also seems compatible with Rushmer's hypothesis of vibration of the whole "cardiohemic system."¹¹

From these considerations, it seems probable that the aortic pressure incisura results from rebound of the aortic blood column and walls *after* aortic valve closure and that the second heart sound is *not* caused by collision of the valve leaflets but by the same

mechanical factors which are responsible for the incisura. It may also be hypothesized that the relatively higher frequency characteristics of the incisura and second heart sound result from elastic recoil of the aortic wall and valve in reaction to their distention by the rebounding aortic blood column.

Summary

A special electrical contacting device was developed and used to time the relationship between aortic valve closure and the aortic incisura in closed chest anesthetized dogs. High speed tracings showed that the aortic valve is closed at least 5 to 13 msec before the incisura. It is concluded that the aortic pressure incisura results from rebound of the aortic blood column and walls *after* valve closure and that the second heart sound is *not* caused by collision of the valve leaflets but by the same mechanical factors which are responsible for the incisura.

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