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The Flow of Blood Supplying the Cardiac Atria.

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Experimental evidence suggests that disturbance of the blood supply of the cardiac atria may lead to auricular fibrillation,[1,2] or to other auricular arrhythmias.[3,4] Aside from anatomical studies, little attention has been directed to the flow of blood to the atrial structures and to factors which may impair that blood supply. These experiments were undertaken in order to study the inflow of blood into one of the atrial arteries, and to note the effect of increased intraatrial tension upon the inflow.

Method. Dogs weighing about 15 kg were anesthetized with veterinary nembutal. In each experiment a Starling heart-lung preparation was set up. The use of the heart-lung preparation permitted closer observation and control of many features of cardiac performance. The largest, and most accessible atrial artery to perfuse was the *left anterior auricular artery*,[5] easily identified as a branch of the circumflex coronary artery at the base of the left auricular appendage. This artery was dissected free; the left circumflex artery was also dissected from its origin for a distance of about 2 cm. A flexible rubber tube, with small glass cannula, was led from the aortic cannula and was inserted into the left circumflex about 2 cm from its origin so as to perfuse the vessel distally. Such perfusion of the circumflex served to maintain myocardial function. The circumflex was ligated at its origin, and a second tube (of metal) was led from the aortic cannula, through the flow-meter, and attached to the proximal segment of the circumflex vessel (as shown in Fig. 1). Arteries arising from this segment, other than the auricular, were securely ligated.

Inflow of blood into the auricular artery was measured, phasically and quantitatively, by the differential pressure orifice-meter devised by Gregg and Green.[6] Calibration of the instrument and interpretation of the flow curves have been described by them, and these were carefully followed in the experiments. The flow curves were synchronized with the cardiac cycle by simultaneous recording either with an electrocardiograph, or with aortic pressure curves using a Wiggers manometer, on the same bromide paper.

Tension within the left auricle was measured by a simple manometer connected into one of the pulmonary veins close to the auricular wall. Estimates of cardiac output were made by collecting outflowing blood in a graduate or siphon-recorder. Twelve experiments were performed on 12 heart-lung preparations showing good function.

Results. For observing auricular arterial inflow under normal conditions, the output of the heart-lung was about 600 cc per minute (commensurate with the capacity of the heart). The blood pressure was adjusted to 100 mm Hg. Tension within the left atrium varied from 3-6 cm of water in different hearts. The venous reservoir was 12 cm above the right auricle.

The normal pattern of inflow into the auricular artery is shown in Fig. 2. The curve indicates that forward flows occur during ventricular systole and diastole. Abrupt interruptions of the stream occur at the onset of ventricular systole; early in ventricular diastole, there is momentary cessation of inflow followed by a short period of backflow.

[1] Resnik, W. H., *J. Clin. Invest.*, 1925, **2**, 125.

[2] Smith, J. R., and Wilson, K. S., *Am. Heart J.*, 1944, **27**, 176.

[3] Cushing, E. H., Feil, H. S., Stanton, E. J., and Wartman, W. B., *Brit. Heart J.*, 1942, **4**, 17.

[4] de Boer, S., *Ergebn. d. Physiol.*, 1923, **21**, 1.

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[6] Gregg, D. E., and Green, H. D., *Am. J. Physiol.*, 1940, **130**, 144.

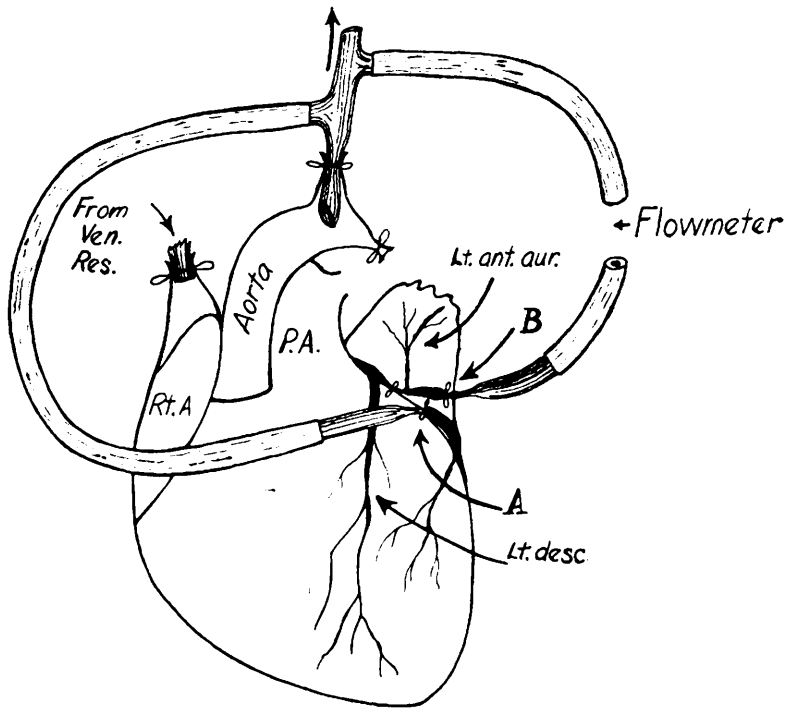

FIG. 1.

Diagram of the arrangement of cannulæ for the perfusion of the left anterior auricular artery. The left circumflex coronary artery is perfused distally (A) to maintain myocardial function. A second system, including the flowmeter, is used to perfuse a proximal segment of the left circumflex (B) of which the auricular artery is a branch.

The peak of rapid inflow in ventricular systole is slightly out of phase with the peak of aortic blood pressure owing, possibly, to delay in the stream flowing through the artificial circuit and flow-meter.

Quantitative analysis of the curves show that in most instances auricular arterial inflow during ventricular systole and diastole are nearly equal, systolic inflow being greater or lesser than that of diastole by a few hundredths cubic centimeter. In these hearts, total inflow for each cardiac cycle varied from 0.08 cc to 0.581 cc. A casual examination of the tracings suggests that the arterial stream during ventricular systole is of greater quantity than in diastole—a discrepancy which is more apparent than real. Rate of inflow in ventricular systole is greater for a short time, producing this form of curve. Tracings of blood inflow into the auricular artery resemble, in general, the metered curves of flow into the principal coronary arteries.[6]

Effect of Raising Left Intra-auricular Tension Upon Atrial Arterial Inflow: Elevation of intra-auricular tension was produced by: 1. induction of cardiac dilatation by raising cardiac output above optimum levels (4 experiments); 2. by inducing anoxia of the preparation (3 experiments), and 3. by producing cardiac dilatation by the administration of 20% chloral hydrate[7] (2 experiments). Cardiac dilatation by any of these methods readily caused a progressive rise of left intra-auricular pressure to 20-25 cm of water. During the period of myocardial dilatation, the blood pressure was maintained at 100 mm Hg, and the cardiac output showed only minor degrees of variation. Heart failure was not permitted to become more severe, for when

[7] Fahr, G., and Buehler, M. S., *Am. Heart J.*, 1943, **25**, 211.

TABLE I.
Examples of Quantity of Inflow into the Left Anterior Auricular Aftery in 2 Normally Beating Heart-Lung Preparations.

	Cardiac output, cc per min	Blood pressure	Inflow systole, cc	Inflow diastole, cc	Total inflow, cc per cycle
Exp. 7	660	100	0.084	0.072	0.156
" 8	640	100	0.099	0.141	0.240

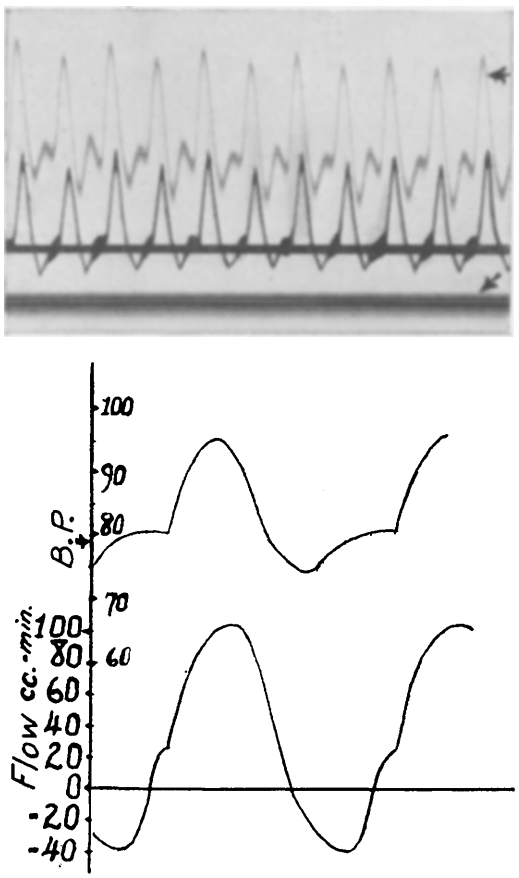

FIG. 2.

Upper: Segment of a curve showing aortic blood pressure tracing (marked by arrow), and the curve of blood inflow into the left anterior auricular artery in a heart-lung preparation. The flow curve has been inked to facilitate reproduction. Black line through the flow curve is line of zero flow; base line for blood pressure is marked by arrow. Lower: Blood pressure and inflow curves redrawn to linear ordinate scales. Analysis of flow curve shows inflow during ventricular systole of 0.249 cc; inflow during ventricular diastole of 0.332 cc. Inflow during cardiac cycle 0.581 cc. Cardiac output was 625 cc per minute.

marked failure occurred blood pressure and output could not be controlled, and the preparation became useless.

TABLE II.
Experiment 10, Normally Beating Heart-Lung Preparation, Blood Pressure Constant at 100 mm Hg. Quantitative Changes in Inflow into Left Auricular Artery During Rise of Left Intra-auricular Tension from Anoxemia.

Left intra-aur. tension, cm water	Cardiac output, cc per min	Aur. art. inflow per cycle, cc
3.3	580	0.140
8.8	640	0.123
24.0	600	0.096
8.8	610	0.136

With blood pressure and cardiac output remaining essentially constant, left auricular arterial inflow decreased as pressure within the auricle rose from myocardial dilatation. Decrease in the quantity flow became apparent as pressure within the auricle rose 8-10 cm of water; diminution of inflow became more marked as auricular tensions increased to 15-20 cm of water (Table II). In general, a rise of intra-auricular tension to 15-20 cm of water, or more, reduced arterial inflow approximately 30-40%. As the heart gradually recovered, the inflow again increased as intra-auricular pressure declined. Analysis of the curves further indicated that the inflow in ventricular systole and diastole was usually uniformly decreased as distension of the auricle occurred. Exceptionally, the inflow during ventricular diastole was greatly curtailed, so that forward flow into the auricular artery took place almost entirely during contraction of the ventricles.

Discussion. These experiments utilized a system conveying blood directly from the aortic cannula of the heart-lung preparation to the left anterior auricular artery. Therefore, the phasic changes observed in the inflow curves depended upon aortic pressure and upon alteration of factors affecting the auricular vascular bed. They were not due to changes of flow in the left circumflex artery.

The pattern of the inflow curves, in the normally beating preparation, suggests that the systolic rise of aortic pressure produces a rapid forward progression of blood in the auricular artery. During ventricular diastole, the flow is less rapid than in systole but is somewhat more sustained. Forward flow is momentarily interrupted at the onset of ventricular systole and diastole, coincident with rapid changes in aortic pressure. Generally there is a sharp backflow from the auricular artery during the rapid fall of aortic blood pressure, early in ventricular diastole.

The diminution of inflow into the auricular artery accompanying elevation of left intraauricular pressure appears to result from changes in the resistance in the auricular vascular bed, since cardiac output and blood pressure were maintained during the period of mild heart failure. It has been suggested that stretching of the cardiac walls may attenuate the intramural vessels, diminishing their capacity.[8] Spalteholz[9] and Unger[10] have shown that the greater part of the venous blood from the left auricle passes by vein to the coronary sinus, but that numerous Thebesian veins exist to account for the escape of a portion of venous blood directly into the auricular cavity. Therefore, a rise of intra-auricular tension may diminish venous outflow from the Thebesian vessels. More probably the distension of the atrial wall exerts pressure upon all of the intramural vessels preventing the free passage of blood through them.

The results of these experiments warrant the suggestion that distension of the auricles, (*e.g.*, resulting from heart failure or valvular disease) may curtail the blood supply to the auricles, favoring the development of auricular fibrillation or other auricular arrhythmias.[2]

Summary. The flow of blood into the left anterior auricular artery was measured phasically and quantitatively in the heart-lung preparation. The curves indicate that forward flow into the auricular artery occurs during ventricular systole and diastole, with abrupt, momentary interruptions of inflow at the onset of ventricular systole and diastole. Elevation of tension within the left auricle diminishes auricular arterial inflow; inflow again increases as pressure within the auricle is restored to normal levels. It is suggested that the interference with auricular blood supply, due to increased intra-auricular tension (as in heart failure) may enhance the establishment of aberrant auricular mechanisms.

[8] Vannotti, A., and Blunschy, A., *Z. f. d. ges. exp. Med.*, 1939, **105**, 447.

[9] Spalteholz, W., *Anat. Anz.*, 1934, **79**, 212.

[10] Unger, K., *Z. f. Anat. u. Entwickl.*, 1937-38, **108**, 356.

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Pyroninophilic Structures of Liver Cells in Carbon Tetrachloride Poisoning.

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The present report is concerned with changes in the pyroninophilic structures of the liver cell, as an early effect of carbon tetrachloride poisoning.

In the hepatic cells of various mammals and lower animals a peculiar type of granulation has repeatedly been observed and described.[1] These structures are characteristic in that they stain an intensive red with methyl green-pyronin (Pappenheimer's meth-

[1] Krause, R., *Arch. f. mikr. Anat.*, 1893, **42**, 53; Koiransky, E., *Anat. Anz.*, 1904, **25**, 435; Berg, W., *Anat. Anz.*, 1912, **42**, 251; *Arch. f. mikr. Anat.*, 1920, **94**, 518; *Pflüger's Arch. f. d. ges. Physiol.*, 1926, **214**, 243; *Z. f. mikr.-anat. Forsch*, 1927, **12**, 1; *Z. f. mikr.-anat. Forsch.*, 1934, **36**, 87.