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The Cardiodynamics of Tricuspid Insufficiency.*

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The purely physical effects of tricuspid insufficiency are often masked in patients by compensatory mechanisms as well as by the effects of concurrent cardiovascular disease.[1] While a few experimental observations of the dynamic changes produced by tricuspid regurgitation have been made,[2] little consideration has been given to the quantitative changes of pressure in the right auricle and large veins. Accordingly one phase of this study was undertaken to elucidate the immediate hemodynamic effects of uncomplicated lesions.

In order for the circulation to be maintained the output of the two ventricles must be equal. However, if a portion of the systolic ejection of the right ventricle is lost by regurgitation some mechanism must act promptly either to increase the tidal volume of the right ventricle or to decrease the systolic discharge of the left heart. The basic method by which the heart is able to maintain this equilibrium before extracardiac compensatory factors can come into action has not been fully worked out. Therefore, the second phase of this study was to determine the cardiac factors involved in effecting equivalent outputs of the two ventricles in the presence of an insufficient tricuspid valve.

Methods. Mongrel dogs were anesthetized with morphine and chloralose (ca 75 mg per kilo) or morphine and sodium barbital (ca 200 mg per kilo). The chest was opened by a sternal splitting procedure and the fifth or sixth rib removed. Mild but adequate artificial respiration was maintained which was interrupted when records were taken. In order to maintain an adequate venous return most animals received warm saline by slow intravenous drip into the femoral vein during the experimental period. The right auricle was cannulated through the external jugular vein and the pulmonary artery via a side branch. Anatomically the left pulmonary artery was found to be more accessible and was generally used. Right ventricular pressures were registered by means of a blunt 15 gauge needle introduced through the ventricular wall. Aortic pressure was recorded by a sound introduced through the carotid artery to the arch of the aorta. All cannulae were securely clamped to avoid vibrations.

Gregg type manometers were used in an optical system similar to that described by Alexander and Webb.[3] Valve lesions were produced by means of a trochar described by Wiggers.[4] This was thrust through the right ventricular wall and introduced through the tricuspid valves. By withdrawing the plunger an insufficiency could be produced, while replacing it would permit the valves to function normally by closing tightly about the instrument.

Following each record a short calibration record was made with a fixed standard pressure. The zero pressure in all experiments was the hydrostatic level of the animal board. A complete calibration was made at the end of each experiment.

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1 Bloomfield, R. A., Lauson, H. D., Cournand, A., Breed, E. S., and Richards, D. W., Jr., *J. Clin. Invest.*, 1946, **25**, 639.

2 Rosenbach, O., *Arch. f. exp. Path. u. Pharm.*, 1878, **9**, 1; Rhil, J., *Berl. klin. Wochensch.*, 1907, **44**, 825; MacCallum, W. C., *Johns Hopkins Hosp. Bull.*, 1911, **17**, 251; Wiggers, C. J., *Arch. Int. Med.*, 1915, **15**, 77.

3 Alexander, R. S., and Webb, E. A., *Am. J. Physiol.*, 1947, **150**, 272.

4 Wiggers, C. J., *Arch. Int. Med.*, 1915, **16**, 132.

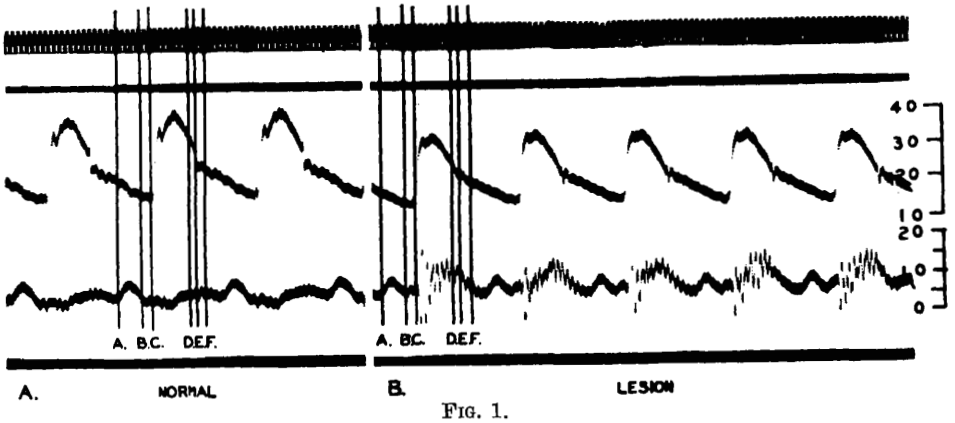

FIG. 1.

Record A shows the normal pulmonary (upper) and right auricular (lower) pressure curves. Record B shows the effects of tricuspid insufficiency. Time 0.02 second. Letters referred to in text.

Results and Interpretation. Records from 11 dogs were suitable for analysis. During tricuspid insufficiency the right auricular pressure rises at a steep slope during the systolic ejection phase of ventricular contraction (C-D, Fig 1), reaching a peak just before the second sound (E). A series of coarse vibrations are superimposed upon the curve during this phase. This is undoubtedly the murmur produced by the insufficiency. In all but one experiment of this study the auricular pressure began to fall during the phase of isometric relaxation (E-F). Wiggers and Feil[5] found in mitral insufficiency that the left auricular pressure continued to rise until the end of ventricular systole. The cause of this difference between the right and left heart remains speculative. A possible explanation is that the volume of blood that regurgitates through an insufficient mitral valve must be accommodated in a comparatively small vascular bed, while that which backs up through the tricuspid valve can be dissipated toward the periphery by way of the large cavae. Consequently, it seems possible for auricular pressure to fall even though a slight regurgitation takes place during this period. It is conceivable that the early fall in auricular pressure may be prevented if the venous pressure is high and the peripheral transmission of the regurgitated blood is impeded.

These studies confirm the observations of Wiggers and Feil[5] that regurgitation does not occur through an insufficient A-V valve during isometric contraction.‡

It has generally been accepted that tricuspid insufficiency *per se* causes a profound rise in right auricular and central venous pressures. That this is not necessarily the case is demonstrated in Fig. 1 and 2. Except during the systolic ejection and early diastolic phases of the cardiac cycle auricular pressure is not greatly elevated. The peak pressure reached during auricular systole is not changed as a result of the lesion. Apparently any elevation of mean venous pressure during uncomplicated lesions is due to the slight increase during ventricular ejection. The rise in venous pressure found in clinical cases perhaps is due to an increased blood volume or to mechanisms which restore arterial pressure and venous return to normal, or other concurrent lesions.

However, in all experiments the pressure in the auricle at the beginning of ventricular filling (F, Fig. 1) was always greater during the lesion than it was in the normal. Thus it is reasonable to assume that the rate of ventricular filling was increased.

The initial pressure in the right ventricle was measured by means of intraventricular

[5] Wiggers, C. J., and Feil, H., *Heart*, 1922, **9**, 149.

‡ Assuming the ventricle contracts isometrically when an insufficient valve exists.

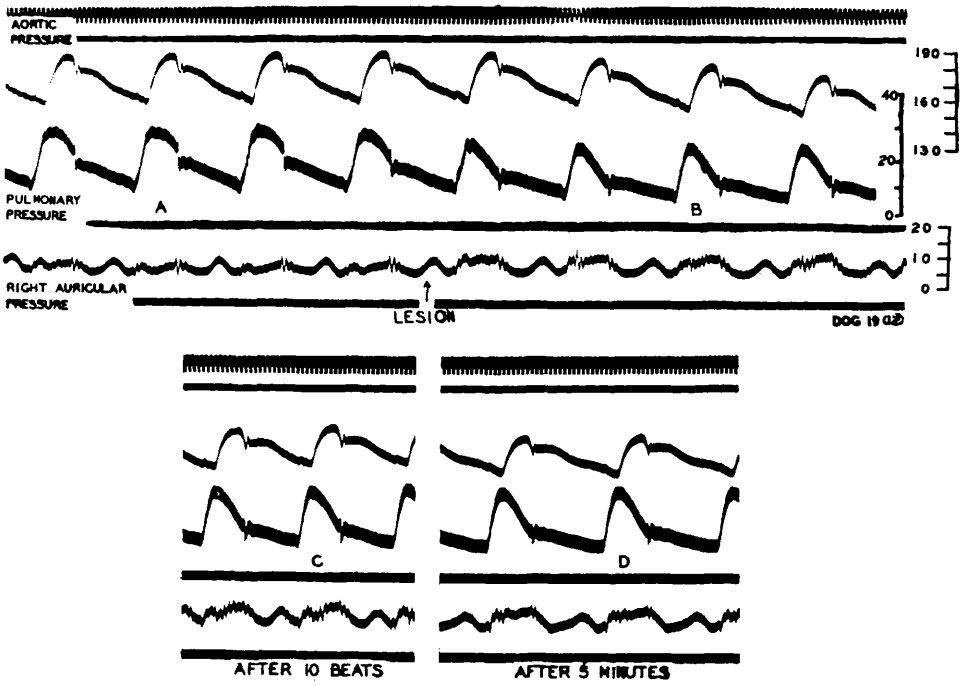

FIG. 2.

Sections of a record showing the immediate and subsequent effects of the lesion on aortic (upper), pulmonary (middle), and right auricular pressures (lower). Time 0.02 second.

pressure curves in 10 consecutive beats in 4 experiments both before and after the valvular lesion was produced. The initial tension increased in each case with an average rise of 2.3 mm of Hg, and a range between 1.7 and 2.4 mm. In spite of this increased initial tension with its accompanying dilation the amplitude of the ventricular pressure pulse was reduced due to the regurgitation.

Following tricuspid insufficiency the pulmonary pressure showed an immediate reduction in systolic, diastolic and pulse pressure. In seven experiments the pulse pressure fell an average of 2.7 mm of Hg with a range between 1 and 6 mm, while systolic pressure fell an average of 8.4 mm of Hg with a range of 5 to 10 mm.

The outstanding change in the form of the pulmonary pressure pulse is shown in Fig. 2. The smaller pulse pressure indicates a reduction in the effective systolic discharge of the right ventricle. The pulmonary pressure reaches its peak earlier and then falls rapidly to the incisura. This rapid drop from the peak to the incisura shows clearly that right ventricular ejection of blood into the pulmonary system is not sustained during the latter part of systole as is normally the case.

As a result of this smaller effective systolic discharge by the right ventricle less blood is delivered to the left and its systolic discharge must decrease. Consequently, as shown in Fig. 2, aortic pulse pressure is reduced and systolic pressure falls as a direct result of the lesion.

The change in the filling and discharge of the right and left ventricles can be shown by means of theoretical volume curves. These are presented in Fig. 3. It will be noted that due to the increased filling pressure the right ventricle has a larger diastolic volume than the normal. As the duration of systole is not appreciably changed by the production of the lesion, the ejection of the ventricle is much more vigorous. The part of the ejection curve A-B represents the early phase of rapid ejection. During this phase the major part of

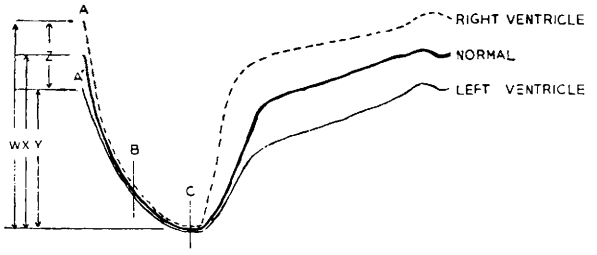

FIG. 3.
Theoretical ventricular volume curves showing the alterations in ejection and filling of right and left ventricles during tricuspid insufficiency. A and A^1, beginning of ventricular ejection; B, end of rapid ejection; C end of ejection. Further discussion in text.

the blood is ejected into the pulmonary system. The lower systolic pulmonary pressure and systolic collapse is explained by the dissipation of the systolic discharge back into the auricle. This regurgitated blood then causes more rapid and complete filling of the ventricle during diastole.

The left ventricle does not fill as completely as the right due to the lower pulmonary filling pressure; therefore, the diastolic volume A′ is smaller. This, of course, will mean a smaller stroke volume ejected into the aorta.

In the theoretical volume curves presented in Fig. 3 W represents the amount of blood pumped by the right ventricle; X equals the output of the normal ventricle, while Y shows the volume of blood ejected by the left ventricle into the aorta. As both ventricles must move forward the same amount of blood in order to maintain the circulation, this must also represent the effective output of the right ventricle. By difference, then Z will show the volume of blood that is regurgitated back through the insufficient tricuspid valve.

Data presented refers to the immediate dynamic effects resulting from the tricuspid insufficiency. Ordinarily, compensatory mechanism such as changes in peripheral resistance operate to restore the circulation to normal. Therefore, it is not surprising that in clinical tricuspid disease the lesion, *per se*, may have little noticeable effect upon the systemic circulation, and it is only after failure of these compensatory mechanisms that clinical signs and symptoms appear.

Summary. 1. The immediate cardiodynamic effects of uncomplicated tricuspid insufficiency were studied in dogs to elucidate the manner by which equilibrium between right and left ventricular output is maintained.

2. Pressure pulses were recorded from the right auricle, right ventricle, pulmonary artery and aorta by calibrated optical manometers.

3. As in mitral insufficiency, regurgitation occurs chiefly during ventricular ejection. However, except during late systole and early diastole, auricular pressure is not greatly elevated. The increased venous pressure found in clinical cases is apparently due to an increased blood volume or to the mechanisms associated with compensation or other cardiac lesions.

4. Comparison of pulmonary and aortic pressure pulses reveals that while the mechanism of left ventricular ejection is unchanged, that of the right is altered. The major portion of the stroke volume is ejected early in systole when regurgitation is less marked. The pulmonary pressure falls rapidly after reaching its peak, due to failing ventricular output later in systole when regurgitation becomes more pronounced. These changes are illustrated by theoretical volume curves of the two ventricles.