THE FUNCTIONAL ANATOMY OF THE BASE OF THE HEART

J. C. VAN DER SPUY, *Division of Thoracic Surgery, Pretoria University and Pretoria General Hospital*

The works of the Lord are great, sought out by all them that have pleasure therein.

Psalm 111, 2.

The heart has both an anatomical and a functional base. The former is directed backwards and is formed by the atria and chiefly by the left atrium. The superior and inferior vena cava and the 4 pulmonary veins terminate in it (Fig. 1). The functional or clinical base on the other hand is formed by 4 valves, 2 atrioventricular and 2 ventriculo-arterial, and the related portions of the ventricles (Fig. 2).

When inspecting the functional base, it is striking that the base of the right ventricle accommodates the atrioventricular, or tricuspid, valve only, whereas the base of the left ventricle accommodates both the atrioventricular, or mitral, and the ventriculo-arterial, or aortic, valves. It also seems strange that the left atrioventricular valve is bicuspid and closely related to the aortic root while the right atrioventricular valve is tricuspid and only distantly related to the pulmonary root.

The construction of the semilunar cusps of the tricuspid ventriculo-arterial (pulmonary and aortic) valves as such does not play any special part in the function of the base of the heart as a whole. Their anatomical relationship to the corresponding atrioventricular valves, however, is of the greatest importance.

The one and only function of the left atrioventricular, or mitral, valve is to direct the unobstructed flow of the blood from the lungs to the heart. These are the only 2 organs in the body with functions so vital that complete cessation of the function of either for but a few minutes will inevitably lead to immediate death. It seems imperative, therefore, that a valve of such strategic importance should function persistently well even in the presence of the most adverse haemodynamic influences.

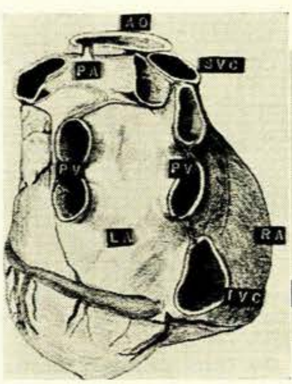

Fig. 1.

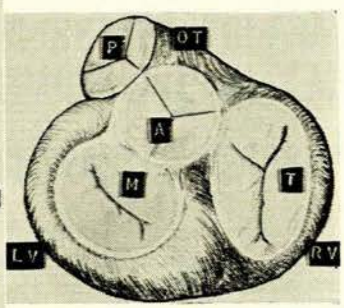

Fig. 2.

Fig. 1. The anatomical base of the heart. AO = aorta; PA = pulmonary artery; SVC = superior vena cava; IVC = inferior vena cava; RA = right atrium; LA = left atrium; PV = pulmonary veins.

Fig. 2. The functional base of the heart. P = pulmonary valve and root; A = aortic valve and root; T = tricuspid valve and ring; M = mitral valve and ring; LV = left ventricle; RV = right ventricle.

The right atrioventricular, or tricuspid, valve on the other hand guides the flow of blood from the systemic veins into the heart. It is a common clinical observation that severe engorgement of the systemic veins, even with marked oedema of the subcutaneous tissues, abdominal organs and peritoneal cavity, is tolerated relatively well, even over a period of many years, whereas oedema of the lungs is a most serious complication. For these reasons it must be of great significance that the mitral valve is a bicuspid unit intimately related to the aortic valve and that the right atrioventricular valve is tricuspid and anatomically only remotely related to the pulmonary valve.

Although like the mitral valve the tricuspid valve directs the flow of blood through an atrioventricular opening, its anatomical construction is such that, unlike the mitral valve, it appears to have a dual function.[1] Whereas competent closure under normal conditions is its usual function, incompetence under stress seems to be its more urgent function. Incompetence of the mitral valve on the other hand leads to serious pulmonary complications, particularly when the incompetence is of an acute nature.

The tricuspid valve is attached at its periphery to the entire base of the right ventricle via the tricuspid ring. Dilatation of the thin-walled right ventricle with consequent dilatation of this ring will therefore readily lead to valvular incompetence. Had the root of the pulmonary artery and the tricuspid ring both occupied the base of the right ventricle, then a dilated root of the pulmonary artery, as is found in pulmonary hypertension, would have encroached upon the tricuspid valve (Fig. 3). Then in instances of severe pulmonary hypertension the beneficial outcome of engorging the systemic venous system by transferring the hyperpressure in the pulmonary arterial system to the peripheral and portal venous systems through tricuspid incompetence, i.e. the development of 'congestive cardiac failure', would have been seriously interfered with.

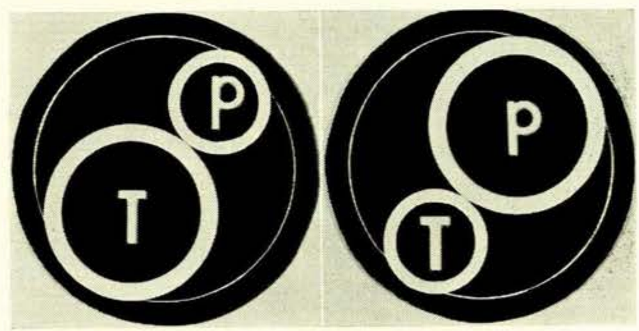

Fig. 3. The base of the right ventricle with the tricuspid ring (T) and the pulmonary root (P). Had the root of the pulmonary artery and the tricuspid ring both occupied the base of the right ventricle (*left*), then a dilated pulmonary artery, as in pulmonary hypertension, would encroach upon the tricuspid valve (*right*).

The outflow tract of the right ventricle is therefore merely an anatomical expression of the absolute necessity of having the pulmonary root some distance away from the tricuspid valve. In view of the above it seems wrong to replace a 'diseased' tricuspid valve with a non-anatomical prosthesis in patients with associated 'congestive cardiac failure'.

Tricuspid Valve

The tricuspid valve has 3 commissures but only two sets of papillary muscles (Fig. 4). The third commissure is attached directly to the interventricular septum. Both mitral cusps and commissures on the other hand are attached to the myocardium by papillary muscles and chordae tendineae. The chordae tendineae of the septal cusp of the tricuspid valve, which are attached to the entire undersurface of the cusp, are inserted directly into the muscular interventricular septum without the intervention of papillary muscles (Fig. 5). Dilatation of the right ventricle will therefore cause downward displacement of the entire septal cusp and so cause tricuspid-septal cusp incompetence. The chordae tendineae of the anterior and posterior leaflets of the tricuspid valve are attached only to the peripheral zones of the cusps and to the myocardium by papillary muscles. These two cusps can therefore billow towards the right atrium in advance of the peripheral zones — like the anterior mitral cusp — and therefore probably play a more important role than the septal cusp in valve closure.

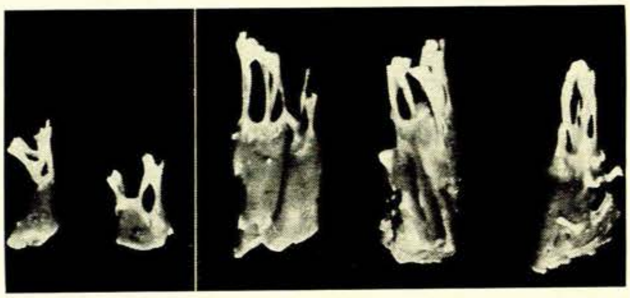

Fig. 4. Tricuspid (*left*) and mitral papillary muscles (*right*) removed from the same heart. Although the tricuspid valve has 3 leaflets and 3 commissures, it has only 2 sets of papillary muscles. The muscles are short and slender.

Fig. 5. The septal leaflet of the tricuspid valve. The chordae tendineae of the septal leaflet of the tricuspid valve, unlike those of the anterior and posterior tricuspid leaflets and unlike those of the mitral cusps, are attached directly to the myocardium without the intervention of papillary muscles.

The 2 sets of tricuspid papillary muscles, unlike those of the mitral valve, are short and slender, and when right ventricular dilatation occurs, their limited extensibility cannot nearly compensate sufficiently for the degree of ventricular dilatation to prevent the development of valvular insufficiency. The mitral papillary muscles are long and thick and will therefore respond more readily with the general left ventricular myocardium to the requirements of abnormal haemodynamic states (Fig. 4). Thus, in the case of aortic incompetence and left ventricular dilatation, the enlarged papillary muscles will help to prevent the development of mitral incompetence which may otherwise result from the tension produced on the chordae tendineae and mitral cusps by the dilated ventricular wall.

Mitral Valve

The mitral valve has 2 cusps. Only the posterior cusp is attached to the base of the left ventricle — the anterior cusp is attached to the root of the aorta — and dilatation of the base of the left ventricle will therefore not cause significant dilatation of the mitral ring. The mitral cusps are roughly triangular. Their apical margins correspond. Had the cusps been truly triangular and of equal size and had the mitral ring been annular, then the mitral valve would have been a regular cone with a large inlet and a minute outlet (Fig. 6a). Amputation of the apical zone of a cone increases the size of its outlet; and the shallower the cone the larger its outlet (Fig. 6b). To produce minimal outlet obstruction, a cone-shaped valve must therefore be of minimal vertical depth. This seems to be the reason why the posterior mitral cusp is short. In a shallow cone with a circular inlet complete valve closure would entail complete flattening of the entire valve, including the circular inlet, and therefore very considerable reduction in the diameter of the base of the left ventricle

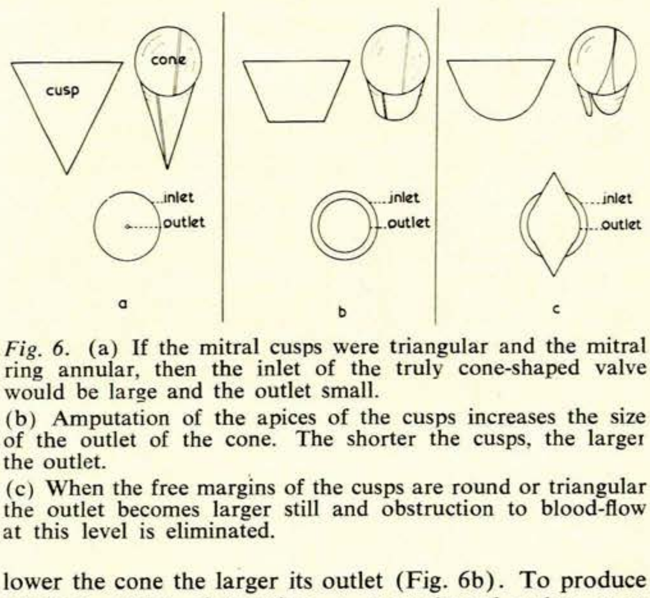

Fig. 6. (a) If the mitral cusps were triangular and the mitral ring annular, then the inlet of the truly cone-shaped valve would be large and the outlet small.

(b) Amputation of the apices of the cusps increases the size of the outlet of the cone. The shorter the cusps, the larger the outlet.

(c) When the free margins of the cusps are round or triangular the outlet becomes larger still and obstruction to blood-flow at this level is eliminated.

during the systole, as also very considerable backward and forward excursions of the cusps during systole and diastole.

A flattened or oval valve inlet, by approximating the cusp bases, would largely reduce the degree of cusp excursions so required to produce valve closure. A flattened or oval valve inlet will, however, produce some valve inlet obstruction. When the circular valve inlet is set obliquely[2] the cusps are also approximated but the circular shape of the valve inlet is maintained (Fig. 7). For the valve ring

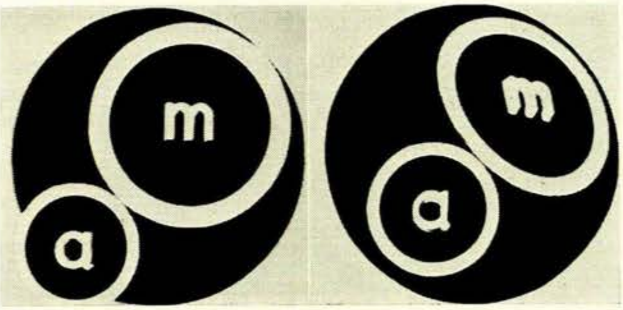

Fig. 7. Left. Base of left ventricle with mitral ring and aortic root set horizontally.
Right. Base of left ventricle with mitral ring set obliquely and aortic root horizontally (*see text*).

to be set obliquely, the cusps must be of unequal length, which is in fact so in the case of the mitral valve (Fig. 8). And, since the shorter posterior cusp is attached to the base of the left ventricle and the longer anterior cusp attached to the root of the aorta, the latter must lie at a more cranial level than the former.

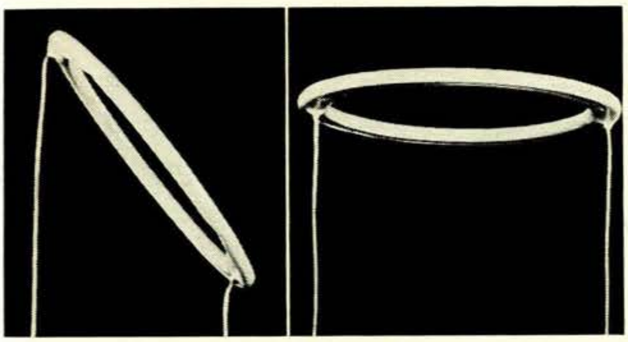

Fig. 8. Left. Oblique ring with cusps of unequal length. *Right.* Horizontal ring with cusps of equal length. Since the apical margins of the cusps correspond, the cusps must be of unequal length when the valve inlet is set obliquely.

Because the remaining half of the root of the aorta is attached to the interventricular septum, the base of the left ventricle must be oblique too. This obliquity is made possible largely through the presence of the membranous portion of the interventricular septum which extends between the root of the anterior portion of the aorta, above, and the muscular septum, below (Fig. 9). The oblique setting of the mitral ring not only has the important diastolic function of overcoming obstruction to flow at the inlet of a flattened valve cone, but perhaps an even more important systolic function. The greater length of the anterior cusp permits valve closure from the apex of the flattened cone as far as the base of the posterior cusp with minimal flattening of the true base of the oblique ring, and

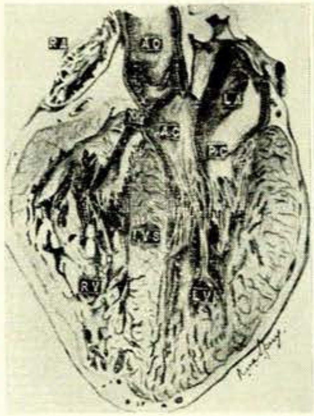

Fig. 9.

Fig. 10.

Fig. 9. Vertical section through the heart to include the base of the left ventricle (*see text*). AO = aorta; LA = left atrium; RA = right atrium; X = membranous interventricular septum; AC = anterior mitral cusp; PC = posterior mitral cusp; IVS = interventricular septum; LV = left ventricle; RV = right ventricle.

Fig. 10. Left ventricular outflow tract (cross-hatched). The function of the left ventricular outflow tract is to make provision for the long anterior mitral cusp and the oblique mitral ring, the two most important systolic and diastolic mechanisms of the mitral valve. OT = left ventricular outflow tract; M = mitral valve; T = tricuspid valve; X = membranous interventricular septum.

therefore of the base of the left ventricle, between the root of the aorta in front and the base of the ventricle behind.

The construction and functions of the more membranous left ventricular outflow tract are quite different from those of the thin-walled muscular outflow tract of the right ventricle (Fig. 10). Like the membranous interventricular septum it makes provision for the very important long anterior mitral cusp and the oblique mitral ring, the two most important systolic and diastolic mechanisms of the mitral valve.

In view of these functions of the membranous septum it is anatomically correct to employ a patch in all cases of ventricular septal defect where the membranous septum is absent, i.e. when the septal defect has muscular margins, or where it is insufficient to the extent that direct suturing of the defect will produce shortening of the membranous septum and so result in tension on the sutures and sutured tissues.

CONCLUSIONS

The anatomical construction of the tricuspid valve and its relationship to the pulmonary valve is such that it appears that the development of 'congestive cardiac failure' must commonly be attributed to a specific function of the tricuspid valve, rather than to myocardial failure, because:

1. Only the tricuspid valve occupies the base of the right ventricle and dilatation of this ventricle will therefore cause dilatation of the tricuspid ring ;

2. The pulmonary arterial root is some distance away from the tricuspid ring in order that dilatation of the pulmonary artery, in cases of pulmonary hypertension, will not interfere with dilatation of the tricuspid ring;
3. The chordae tendineae of the septal cusp of the tricuspid valve, unlike those of the anterior and posterior tricuspid leaflets and unlike those of the mitral cusps, are attached directly to the myocardium without the intervention of papillary muscles. Dilatation of the right ventricle will therefore cause downward displacement of the septal cusp and so produce valvular incompetence; and
4. The tricuspid valve, although it has 3 commissures, has but 2 sets of papillary muscles. These papillary muscles are very short and slender and, when right ventricular dilatation develops, their limited extensibility will not nearly compensate sufficiently for the ventricular dilatation to prevent the development of valvular insufficiency.

In the case of acute pulmonary capillary hypertension, before the development of pulmonary arterial hypertension, tricuspid incompetence can unfortunately not develop. The mechanical induction of a controllable degree of tricuspid incompetence should therefore be of great benefit in the treatment of severe acute capillary hypertension — pulmonary oedema — whether caused by myocardial or valvular disease.

The mitral valve guards probably the most important gateway in the body. This function it can perform persistently well, even under abnormal circumstances, because it is (*a*) a flat, (*b*) cone-shaped valve, with (*c*) a short posterior cusp, attached to the base of the left ventricle and (*d*) a long anterior cusp, not attached to the base of the left ventricle, with (*e*) an obliquely set mitral ring, (*f*) occupying with the aortic root the base of the left ventricle, and because (*g*) the chordae tendineae are attached to the myocardium by long, thick and extensile papillary muscles, which readily respond with the rest of the left ventricular myocardium to the demands of increased or decreased haemodynamic stress.

The right ventricular outflow tract owes its presence to the fact that the tricuspid ring and pulmonary arterial root must necessarily be some distance apart so that the development of triscuspid incompetence will not be interfered with in the case of pulmonary hypertension calling for 'congestive cardiac failure'.

The left ventricular outflow tract results from the most important diastolic and systolic mechanisms of the mitral valve unit, viz. the obliquely set mitral ring and the long anterior cusp.

SUMMARY

The functional anatomy of the base of the right ventricle, base of the left ventricle, right ventricular outflow tract, left ventricular outflow tract, tricuspid ring, mitral ring, membranous part of the ventricular septum, and also the function of the mitral valve and the functions of the tricuspid valve are discussed.

There is clear evidence to indicate that the construction of the tricuspid valve is such that it *should,* and of the mitral valve that it *should not,* become incompetent under adverse haemodynamic circumstances — in both instances to protect the lungs.

The mechanical induction of a controllable degree of tricuspid incompetence seems to be the specific treatment for severe acute pulmonary capillary hypertension — pulmonary oedema.

I wish to thank Mr. Theo Marais for the photographs and Miss Rianda van der Spuy for two of the drawings.

REFERENCES

1. Van der Spuy, J. C. (1964): S. Afr. Med. J., **38**, 771.
2. *Idem* (1958): Brit. Heart J., **20**, 471.