

# The medial papillary complex

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*The anatomy of the papillary muscle of the conus, also known as Lancisi's muscle, was studied in 100 normal hearts from pathological collections and in 8 embryonic and fetal hearts. Wide morphological variations were observed and because of this the name medial papillary complex is proposed. It is concluded that the value of this complex as an anatomical landmark in the right ventricle is a very restricted one. The development of the medial papillary complex is described.*

The pattern of the distribution of the papillary muscles and chordae tendineae in the right ventricle is variable. The present paper describes a study of the variability of what has been called the papillary muscle of the conus (Luschka, 1863), which has also been referred to as muscle of Luschka (Poirier, 1902; Barry and Patten, 1968), muscle of Lancisi (Tandler, 1913), or medial papillary muscle (Tandler, 1913; Bargmann, 1963). Sometimes this papillary muscle is said to possess tendinous cords attached to both the anterior and septal cusps of the tricuspid valve; sometimes insertions only on the anterior cusp are described. Part of this discrepancy may depend on the definition chosen. For, when the medial papillary muscle is defined as the most anterior one of a series of small papillary muscles, all the others being called accessory papillary muscles (Tandler, 1913), then it can be expected that the medial papillary muscle would give its chordae predominantly to the anterior valve cusp (Poirier, 1902). However, it has been suggested (Verduyn Lunel, 1964) that the muscle of Lancisi can be split up into two or more parts, and, thus defined, the muscle of Lancisi will give off chordae to both the septal and anterior cusps. According to Van Mierop (1974) the papillary muscle of the conus is absent in certain congenital malformations, and in such cases the uppermost accessory papillary muscle may mistakenly be interpreted as being the conal papillary muscle. These differences of identification are not without importance, because of the practice of relating to this papillary muscle the position of ventricular septal defects (Goor *et al.*, 1970; Moulaert, 1974) and of the conducting system (Tandler, 1913; Verduyn Lunel, 1964). This report describes the result of a study of the anatomy of this

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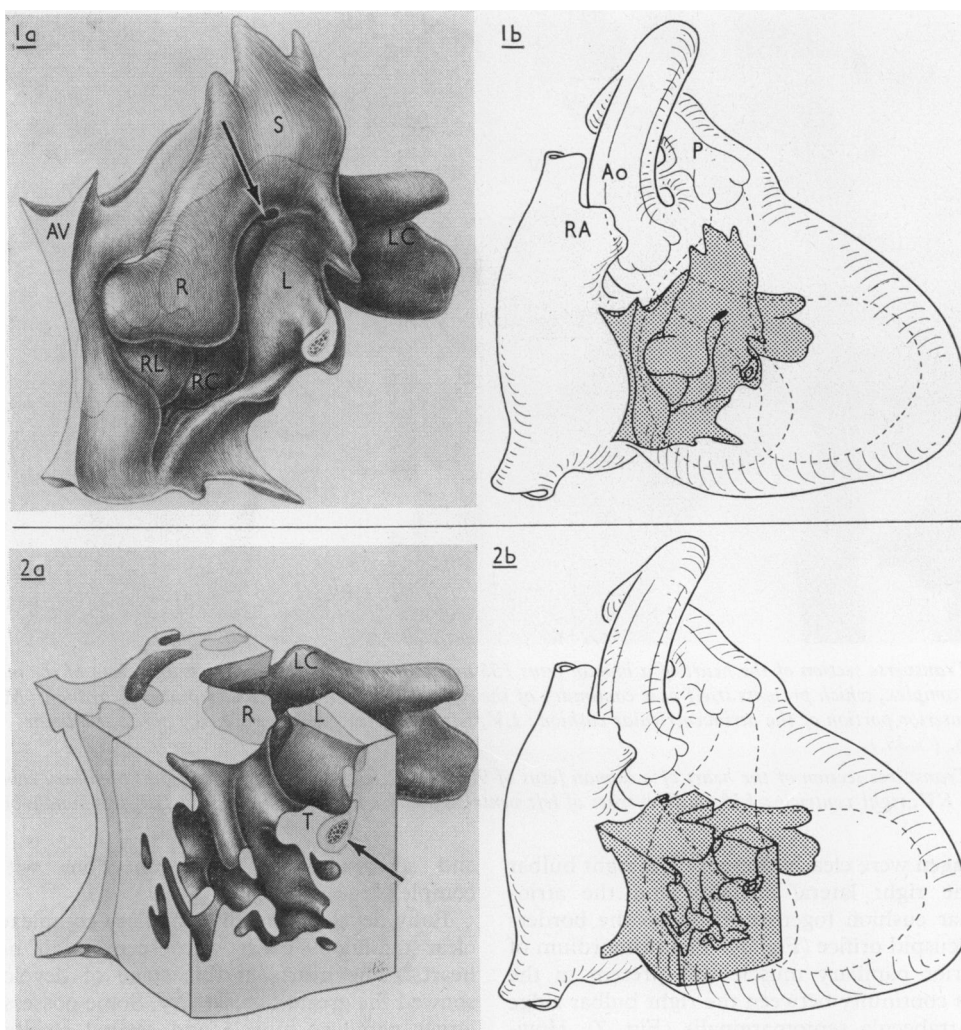
papillary muscle, from which it appears that the conal papillary muscle has a less constant anatomy than is suggested by its name or names, and that it is justifiable to speak of a *medial papillary complex* rather than of one medial papillary muscle.

## Material and methods

From the collections of both the Department of Pathology, Wilhelmina Gasthuis, Amsterdam (kindly made available by Dr. A. E. Becker) and the Department of Anatomy, State University Leiden, 100 normal hearts were available for study, of which 35 were obtained from neonates and 18 from subjects over 60 years of age. Sixteen specimens belonged to infants in their first year of life, and another 10 came from children in their first decade. Four hearts were available from the second decade, 8 hearts from the third decade, and 5 specimens from the fourth. The fifth and sixth decades were represented by two specimens each. In addition eight human embryonic and fetal hearts ranging from 14 to 90 mm crown-rump length were examined microscopically; the 10  $\mu$ m serial sections had been stained using routine histological methods.

## Definitions

For clarity some terms used in this paper will first be defined. The crista supraventricularis is the muscular structure intervening between the tricuspid and pulmonary orifices. The trabecula septomarginalis is that muscular elevation that descends from the crista supraventricularis along the right ventricular septum, and which, in the apical portion of the right ventricle, runs from the septum to the parietal ventricular wall; this latter part passes freely through the ventricular cavity



**Fig. 1** (a) Reconstruction of the valve-forming tissues surrounding the tricuspid orifice (human embryo of 17 mm CR-length). AV, right ventricular sulcus tissue; R, right bulbar ridge; RC, right portion of the atrioventricular cushion; RL, right lateral cushion; S, bulbar septum; L, left bulbar ridge; LC, left portion of the atrioventricular cushion; arrow: interventricular foramen. (b) Schematic representation of the entire heart, to show the position of the reconstructed part. Ao, aorta; P, pulmonary trunk; RA, right auricle.

**Fig. 2** (a) Part of the same reconstruction as shown in Fig. 1a, with addition of the myocardium. The trabecula septomarginalis (T) is clearly seen. Arrow, right bundle-branch; L, left bulbar ridge; LC, left portion of the atrioventricular cushion; R, right bulbar ridge. (b) Diagram, to show the position of the reconstructed part in relation to the rest of the heart.

and gives origin to the anterior papillary muscle. In the embryonic heart, the term bulbus is applied to that part which intervenes between the primitive ventricle and the future arterial orifices. The two endocardial swellings which play a role both in the septation of the bulbus and in the formation of the tricuspid valve are called left and right bulbar ridges, respectively (Wenink, 1971a).

## Results

In the material studied two extreme situations were recognised. One was found in relatively young embryonic hearts and the other in hearts from the older subjects.

In a 17 mm embryo the cusps of the tricuspid valve were, of course, not identifiable as such but

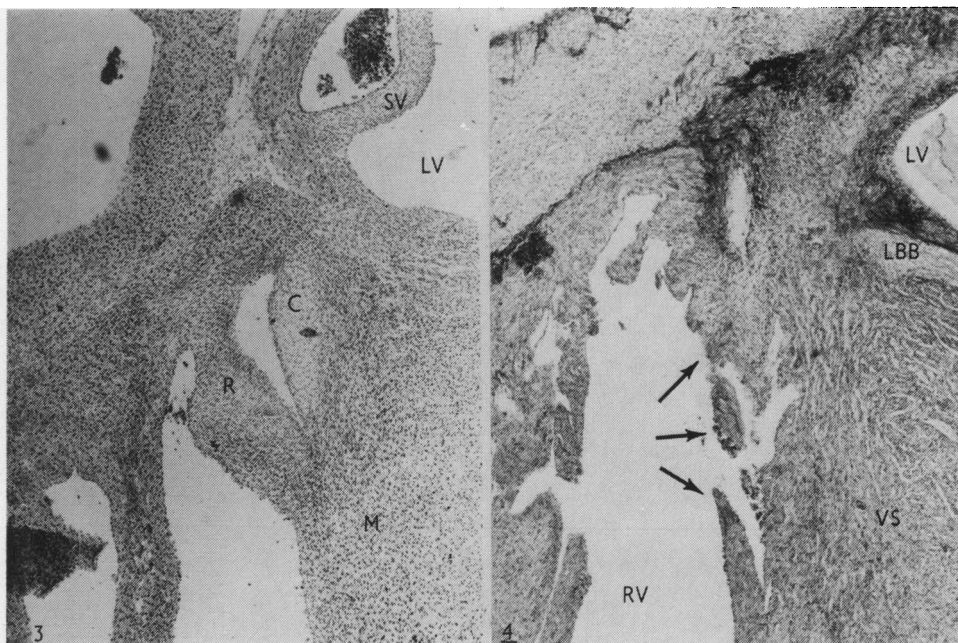


Fig. 3 Transverse section of the heart of a human fetus (55 mm CR length), showing the primordium of the medial papillary complex, which presents itself as a continuity of the right bulbar ridge (R) and septal myocardium (M). C, most anterior portion of the atrioventricular cushion; LV, left ventricle; SV, noncoronary aortic semilunar valve cusp. ( $\times 35$ .)

Fig. 4 Transverse section of the heart of a human fetus of 90 mm CR length, showing the medial papillary complex (arrows). RV, right ventricle; LV, outflow tract of left ventricle; VS, ventricular septum; LBB, left bundle-branch. ( $\times 35$ .)

their anlagen were clearly present. The right bulbar ridge, the right lateral cushion, and the atrioventricular cushion together made up the borders of the tricuspid orifice (Fig. 1). The primordium of the anterior papillary muscle was present in the form of a continuity between the right bulbar ridge and the trabecula septomarginalis (Fig. 2). However, in the corner between the right bulbar ridge and the atrioventricular cushion, that is at the future commissure of the anterior and septal leaflets of the tricuspid valve, no trace of any papillary muscle could be distinguished. The same was true for the entire septal wall of the right ventricle. Only in later stages of fetal development was the septal wall furnished with papillary muscles and in these stages the atrioventricular cushion had been partly loosened from the septum. At the site where the 17 mm embryo showed a massive right bulbar ridge, distinction could later be made between an anterior leaflet and a muscular crista supraventricularis. The youngest specimen with a recognisable medial papillary complex was a fetus of 55 mm crown-rump length (Fig. 3), but the complex consisted of less fibrous tissue than that in older fetal hearts (Fig. 4). Both the anterior

and septal leaflets had connections with this complex.

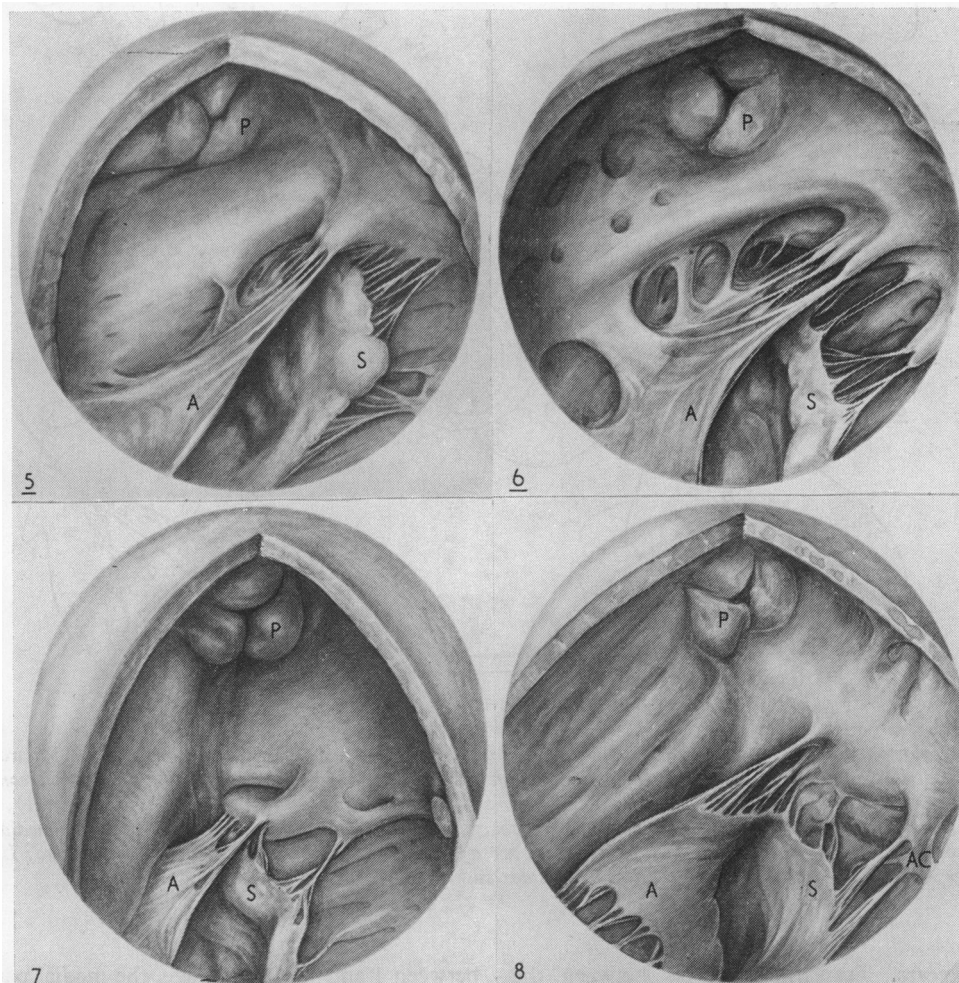
Fully developed medial papillary complexes with clear tendinous cords were seen in all neonatal hearts. The hearts at this stage of development showed the greatest variability. Some possessed one larger papillary muscle and several smaller ones with chordae attached to the anterior leaflet, while several other smaller muscles had their chordae attached to the septal leaflet (Fig. 5). In other cases there were two rows of several identical papillary muscles, each row belonging to one of the two tricuspid leaflets (Fig. 6). Yet other hearts had only one strong papillary muscle with both the valve leaflets tethered by its chordae (Fig. 7). In addition, the groove where the anterior leaflet and the crista supraventricularis met had a variable morphology. Sometimes it was smooth and straight but in other cases it was bridged by some papillary muscles with chordae connected to the anterior leaflet. Intermediate forms had only small trabeculations near the base of the anterior leaflet.

The other extreme situation, as distinct from that in the fetal hearts, was seen in the hearts of elderly individuals, which showed more uniform mor-

phology of the medial papillary complex. In the corner where the crista supraventricularis continues into the trabecula septomarginalis a convergence of chordae tendineae was found. Sometimes they ended together in one fibrous patch in the septum, recalling the *macula tendinea septi ventriculorum* as described by Holl (1912); in other cases (Fig. 8) they formed several strands which still had their own origin on the septal wall. These strands had

only slight resemblance to papillary muscles, for they consisted largely of fibrous tissue. The bundle of tendinous cords always inserted on both the anterior and septal cusps of the tricuspid valve. Real accessory papillary muscles were not encountered. Instead, many chordae tendineae inserted directly onto the relatively smooth septum.

A clear distinction could thus be made between neonatal hearts and specimens from subjects older



**Fig. 5** Neonatal medial papillary complex. *A*, anterior tricuspid leaflet; *S*, septal tricuspid leaflet; *P*, pulmonary orifice.

**Fig. 6** Neonatal medial papillary complex, consisting of two rows of papillary muscles. Only the row belonging to the anterior leaflet (*A*) is visible, hiding the septal row from the view. Note the trabeculations near the basal attachment of the anterior leaflet. *S*, septal leaflet, *P*, pulmonary orifice.

**Fig. 7** Neonatal medial papillary complex, consisting of only one papillary muscle, giving chordae to both the anterior (*A*) and septal (*S*) leaflets. *P*, pulmonary orifice.

**Fig. 8** The medial papillary complex of an elderly subject. *A*, anterior leaflet of the tricuspid valve; *AC*, accessory papillary muscles; *P*, pulmonary orifice; *S*, septal leaflet of the tricuspid valve.

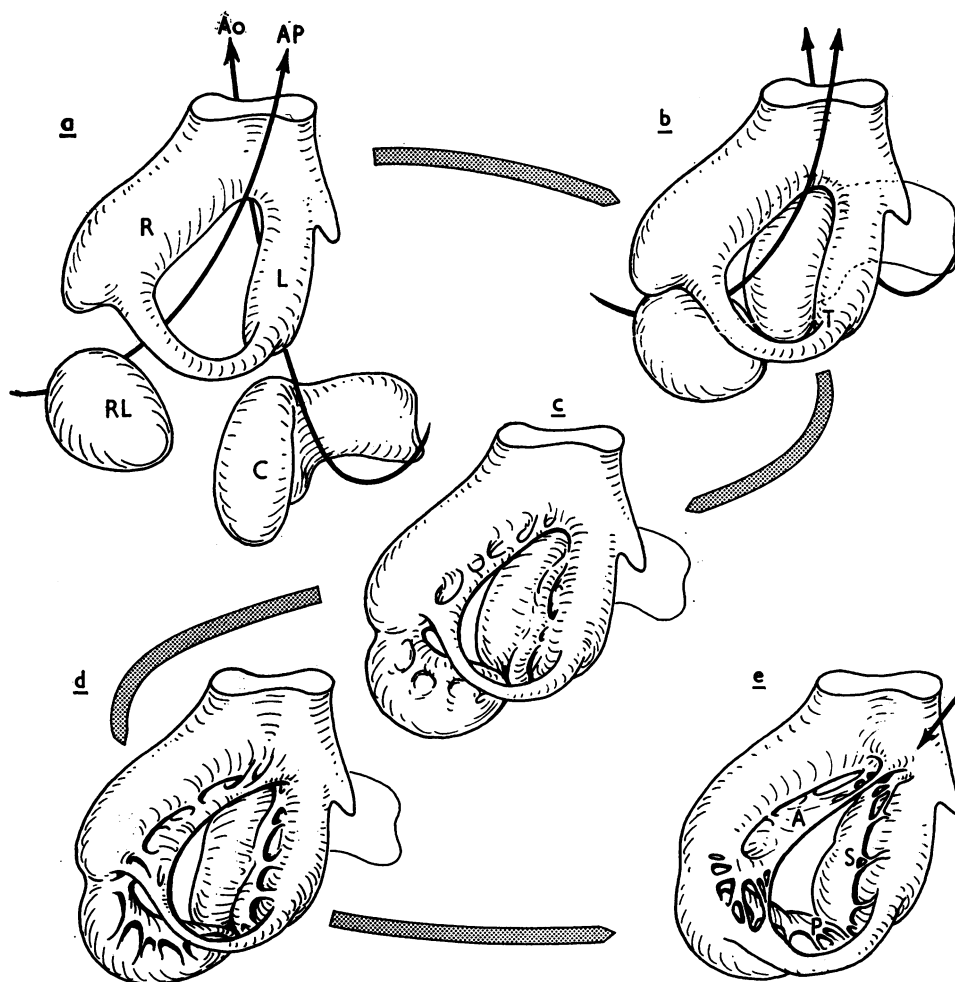


Fig. 9 Schematic representation of the development of the tricuspid valve and its tension apparatus. (a) Separated view of the valve-forming tissues, of which the normal relations are shown in b. The arrows follow the bloodstream in the right and left parts of the heart. (c) (d) The undermining process leads to the formation of anterior (A), septal (S), and posterior (P) leaflets. The trabecula septomarginalis (T) is present early (b), whereas the medial papillary complex (arrow) is only defined later (e). Ao, aorta; AP, pulmonary trunk; R, right bulbar ridge; L, left bulbar ridge; RL, right lateral cushion; C, atrioventricular cushion.

than 60 years. Transitional stages between the neonatal variability, as described, and the more constant and simpler morphology in the elderly, were seen in the intermediate age groups.

The infantile hearts largely showed the same pattern as that described in the neonatal ones. The proportional growth of the heart had even led to a still more striking medial papillary complex in some cases. The growth of the heart, however, appeared not to have proceeded proportionately throughout the older age groups. For, already in the group

between 1 and 10 years of age, the medial papillary complex was relatively smaller and less complicated than in the younger stages. The same was true for the accessory papillary muscles.

In many hearts from subjects in their third decade, it was already apparent that there was virtually no papillary muscle arising from the septum. At the site where the muscular medial papillary complex was assumed to have once existed, the morphology was as described for the oldest age group.

**Discussion**

There is a considerable lapse of time between the initial establishment of the definitive form of the heart and its final detailed morphology (Wenink, 1971a). After completion of the septational processes, development continues, and this is especially true for the atrioventricular valves (Van Mierop and Gessner, 1972; Van Gils, 1975). It can be asserted that cardiac development goes on long after birth and, taking into account processes of hypertrophy and atrophy, one comes to the conclusion that it ends only with the death of the individual. This is exemplified perfectly by the morphology of the medial papillary complex in elderly individuals (Fig. 8). An important factor in this continuing development is probably provided by the haemodynamics; it is known that, even in its early stages, haemodynamics play an important role in the morphogenesis of the heart (Pexieder, 1975). The bulbar ridges are particularly prone to haemodynamic influences and this could perhaps be explained by the plastic nature of these tissues. It seems plausible that haemodynamic influences operating relatively late in the development of the heart should effect more variations in detailed morphology than the primary morphogenetic processes do.

In Fig. 9, this difference is illustrated schematically. The undermining of the valve-forming tissues is directed towards the future commissure of the anterior and septal leaflets and produces one papillary complex belonging to both leaflets. The undermining of the right bulbar ridge can produce trabeculations or even chordae at the site where the crista supraventricularis and anterior leaflet meet (Fig. 7). These are still very reminiscent of, though not identical to, those below the septal leaflet which are called accessory papillary muscles (Tandler, 1913).

It is useful to reiterate that the atrioventricular cushion is always separated from the ventricular septum by the left bulbar ridge tissue and by tissue belonging to the atrioventricular sulcus. There is a continuity of the latter two tissues, which has been described elsewhere in detail (Wenink, 1971a). The fact that the atrioventricular cushion never touches the muscular ventricular septum is considered to be of great importance. Therefore, in Fig. 9 the embryonic situation has been slightly exaggerated. The role of the left bulbar ridge has been highlighted, whereas the muscular septum has been deliberately omitted. This does not mean that the septum is considered to play a minor role in development of the tension apparatus. But Fig. 9 does not show any myocardium, and all valve-

forming tissues are intimately related to myocardium. This holds for the atrioventricular cushion, even with the intervention of the left bulbar ridge.

The embryonic endocardial swellings should not be looked upon as the sole elevations on a smooth inner surface of the heart; they indicate the site of elevations in the myoepicardial mantle. The sharp boundaries shown for them in Fig. 9 are real as far as their luminal surfaces are concerned, but the boundaries are artificial with regard to their myocardial surfaces. Therefore, it is concluded that the medial papillary complex originates from left and right bulbar ridges, atrioventricular cushion, and myocardium (cf. Fig. 3 and 4). The exact role of the myocardium itself cannot easily be estimated, because of secondary muscularisation of the endocardial swellings (Wenink, 1971a). This histogenetic process continues into the period of undermining of the cushion tissue, during which the tension apparatus comes into existence.

Particular attention should be given to the continuous strand of endocardial swelling, which can be followed from the future medial papillary complex towards the anterior papillary muscle. It passes along the trabecula septomarginalis. Previously (Wenink, 1971a) it was stated that the left bulbar ridge continues for some distance along the trabecula septomarginalis. For a better understanding of cardiac embryology, the author wishes to extend this view. Like other transitional zones between the sequential compartments of the primitive heart, the bulboventricular transition is covered by more or less swollen endocardium. Thus, on the trabecula septomarginalis, which marks this bulboventricular transition from very early stages, there might be swollen endocardium connecting left and right bulbar ridges. In fact, the two ridges should be considered as part of one endocardial ring. This view closely corresponds with the concept of development of the conducting system (Wenink, 1976), in which four specialised myocardial rings have been described to be the anlagen of the adult conducting system. These rings are similarly interposed between the different heart chambers. They are covered by the endocardial swellings under discussion in the present paper.

The above considerations indicate that the inflow portion of the right ventricle is indeed what its name suggests. The region of the developing right ventricle, from which the tissues contribute to the tension apparatus of the tricuspid valve, is bounded by the trabecula septomarginalis.

With respect to the site of the medial papillary complex, the anatomy of the normal embryonic heart sufficiently explains the normal adult morpho-



logy. However tempting it may be to apply these observations to congenitally malformed hearts, one should be very careful in doing so, as structures which are continuous in the normal embryonic heart are not necessarily continuous in the case of a congenital malformation. This holds in particular for the very plastic tissue of the endocardial swellings. In the heart of a human embryo of 27 mm crown-rump length, a ventricular septal defect has been described as being caused by a defect in the left bulbar ridge (Wenink, 1971a, b). Such a defect can be interpreted as an interruption in the normal continuum of endocardial swellings as described above, and no difficulty arises in identification of these swellings. On the other hand, studies, by the present author, of a human embryo, in which the precise malformation has yet to be diagnosed, have revealed three endocardial swellings in the bulbus (unpublished observations). In such a malformation it is obviously difficult to make definite statements about the normal bulbar ridges being displaced, or about the presence of an extra bulbar swelling. At present, therefore, the interpretation of the transition from normal variations into clear-cut malformations cannot always be made reliably.

It can be concluded, from the wide anatomical variations of the medial papillary complex observed in the normal heart in this study, that one should be very careful in deciding about any abnormality of the complex in congenital malformations. Thus, it is not entirely clear on which criteria the diagnosis of its absence (Van Mierop, 1974) should be based. The early appearance in the embryo of the complex of the trabecula septomarginalis and the anterior papillary muscle makes this structure much less variable than the medial papillary complex. Consequently, it does not seem very logical to use the medial papillary complex as a landmark for the designation of the 'septal band', as has been suggested by Quero Jiménez and Pérez Martínez (1974). Further microscopical study of fetal hearts at more advanced stages of development is necessary to clarify the relations of the medial papillary complex to the right bundle-branch. A detailed description of these relations will be reported separately.

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