Anatomic Evidence for Spontaneous Closure of Ventricular Septal Defect

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The probability that postnatal spontaneous closure of ventricular septal defect occurs with some frequency has been evident to clinicians for a number of years. This postulate is based upon the presence of characteristic murmurs in childhood which disappear with the growth and development of the patient.1-7 Convincing evidence of spontaneous functional closure of ventricular septal defects is found in several recent reports of serial cardiac catheterization in children.8-13

Hoffman and Rudolph (personal communication) studied by serial cardiac catheterization 40 infants with ventricular septal defect. They observed that while the size of the defect did not appear to change in 11, it either closed or became smaller in 29 cases. In 10 of these 29 cases, there was evidence of closure of the ventricular septal defect.

Ventricular septal defects constitute about 20% of all isolated congenital cardiac malformations in children but only 7% of the congenital defects in adults. This difference cannot be explained because of early mortality in those with ventricular septal defect.14, 15

In spite of these observations which suggest the phenomenon of spontaneous closure of ventricular septal defect, description of closed ventricular septal defects is limited to a few isolated reports.

The purpose of this communication is to demonstrate the findings in seven cases which are considered to represent spontaneous closure of isolated ventricular septal defect.

Observations

During a 4-year period, 1,605 necropsies on patients of all ages were reviewed for evidence of spontaneous closure of a ventricular septal defect. Seven adults with spontaneous closure were observed (about one per 225 necropsies). The ages at death of the seven subjects ranged from 39 to 90 years. No histories suggesting a ventricular septal defect had been elicited in any of the cases, the malformation of the ventricular septum presenting as an incidental finding at necropsy. Two anatomic types of spontaneous closure of ventricular septal defect were encountered.

The first type, seen in two cases, was observed as a defect in the ventricular septum which was closed by an adherent tricuspid valve overlying the right ventricular aspect of the defect (figs. 1 and 2). In one of these cases examination of the left ventricle revealed a depression in the muscular part of the basal portion of the ventricular septum (fig. 1). In the second case, there was a subaortic depression in the ventricular septum (fig. 2). In each of these cases, probing of the depression revealed a barrier which prevented communication of the left ventricle with the right ventricle. From the right ventricular aspect, the septal leaflet of the tricuspid valve and its associated chordae were found to be grossly scarred and the chordae were interadherent. The conjoined substance of the leaflet and chordae were adherent to the right ventricular side of the ventricular septum and represented the barrier between the two ventricles. No jet lesions were ob-
observed in the right ventricle opposite the site of the altered and adherent tricuspid valve. In each case, histological examination revealed scarring of the septal leaflet of the tricuspid valve and adhesion of the free aspect of this leaflet to the endocardium of the right ventricle inferior to the ventricular septal defect.

The second type of lesion was observed in the remaining five cases (figs. 3 to 5). In each of these cases, a peculiarity of the left ventricular aspect of the ventricular septum was noted. The central part of the ventricular septum was uncommonly trabeculated. An endocardial-lined depression was present between two columns of muscle, which ran obliquely with respect to the long axis of the ventricular septum. Probing of this depression revealed a barrier at the right ventricular side of the ventricular septum. Sections made through the depression revealed a funnel-shaped defect of the muscle in the ventricular septum. The apex of the funnel was closed by a plug of dense fibrous tissue which was the barrier encountered while probing. No inflammatory exudate was evident in relation to the lesion and the surrounding myocardium was normal histologically. No jet lesions were observed in the right ventricle.

**Comment**

It has been shown that in seven cases observed pathologically a ventricular septal defect had become closed spontaneously by the interposition of fibrous tissue. In the first two cases, the evidence for postnatal spontaneous closure of a ventricular septal defect seems incontrovertible. A defect in the ventricular septum was closed by adhesion of the septal leaflet of the tricuspid valve to the right ventricular side of the tissues around the defect. The findings suggest that healed bacterial endocarditis of the tricuspid valve had been present and that in the process of healing the assumed bacterial endocarditis, closure of the ventricular septal defect occurred. Certain reported instances of spontaneously closed ventricular septal defects suggest such a sequence of events.

The remaining five cases (figs. 3 to 5) were similar in that a defect in the midportion of

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the ventricular septum was closed by a plug of dense fibrous tissue.

Spontaneous closure in a muscular ventricular septal defect has been previously suggested by one of the authors (J.E.E. and associates). Recently Roberts and co-workers demonstrated closure of the ventricular septal defect by fibrous tissue in a case of tricuspid atresia. In this case, a jet lesion opposite the plug of occluding fibrous tissue provided circumstantial evidence of a previously functional ventricular septal defect and crystallized the concept of spontaneous closure. Seven cases of fibrous closure of defects and three with "fistula-like tracts" in the muscular ventricular septum were recently reported by Bloomfield in a series of 20 cases which were considered to be examples of probable spontaneous closure of ventricular septal defect.

In our five cases, in which a fibrous plug closed a defect of the muscular septum, one might, for discussion, reject the hypothesis that the lesion represented spontaneous postnatal closure of a muscular ventricular septal defect. An alternate explanation for the lesion might be entertained. The lesion could be considered to represent: (1) antenatal closure of a ventricular septal defect, or (2) spontaneous closure of an acquired ventricular septal defect such as might result from trauma or myocardial infarction.

The fact that no evidence was present of damage of the myocardium in relation to the lesions with which we are concerned denied that an acquired ventricular septal defect had been present. Had a defect been acquired and then closed, it seems likely that the underlying cause of such a defect would leave evidence in the form of scarring in the sur-

**Figure 2**

Case 2. (a) Viewed from the left ventricle (L. V.). There is a small defect (point of arrow) in subaortic region. (b) Low-power photomicrograph of a sagittal section through the ventricular septum shows the left ventricular opening (L. V.) of a ventricular septal defect which is closed by the adhesion of the tricuspid valve (T. V.) to the thickened endocardium at the lower aspect of the ventricular septal defect. V. S., ventricular septum; R. V., right ventricle.
Figure 3

Case 3. (a) In the central portion of the left ventricular aspect of the ventricular septum is a depression between two bundles of muscle; the depression represents the left ventricular opening of an interventricular communication. (b) Close-up view of the left ventricular aspect of the ventricular septal defect (between arrows). Characteristically, obliquely oriented bundles of muscle outline the position of the left ventricular side of the opening. (c) Cross section through the ventricular portion of the heart exposing within the ventricular septum (V. S.), the left ventricular opening (arrow) of a tract between the muscles which leads toward the right ventricle (R. V.). (d) Low-power photomicrograph in same plane as c shows the tract between the left ventricle (L. V.) and right ventricle (R. V.) closed by the interposition of a collagenous fibrous plug.
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Figure 4

(a, b, and c) Case 4. (a) Right ventricular aspect of ventricular septum reveals a highly trabeculated ventricular septum. Although suggestive of the presence of a muscular ventricular septal defect, it is within the usual range for the degree of right ventricular trabeculation. (b) Interior of left ventricle (L. V.). Between two obliquely oriented muscle bundles within the ventricular septum is a cleft (between arrows) representing the left ventricular aspect of an interventricular communication, now closed. (c) Cross section through the ventricular septum (V. S.). From the cavity of the left ventricle (L. V.), an opening (arrow) leads into a fibrous mass which is within the ventricular septum. The fibrous tissue represents the factor closing a pre-existing ventricular septal defect of the muscular part of the ventricular septum. R. V., right ventricle. (d, e, and f) Case 5. (d) The left ventricular opening of an interventricular communication (point of arrows) is circular in outline. (e) A sagittal section has been made through the center of the ventricular septum showing within it the continuation of the channel seen in e. (f) A cross section of the right ventricular side (R. V.) of the ventricular septum (V. S.). The right ventricular side of the channel which began in the left ventricle is closed by the interposition of a collagenous plug.

rounding myocardium. No history of trauma was elicited in any case nor were there other lesions which could have been interpreted as being of traumatic origin. In no case was there a history compatible with the temporary presence of an interventricular communication. This negative factor seems an important unit of evidence against an acquired communication since acquired interventricular communications are usually associated with profound hemodynamic consequences. Lack of convincing evidence for closure of an acquired communication and the regularity in the nature of the lesions favor a congenital basis for the lesions.
CLOSURE OF VENTRICULAR SEPTAL DEFECT

Figure 5

(a and b) Case 6. (a) Left ventricular opening (between arrows) of the interventricular communication is represented by a slit-like depression between two obliquely oriented bundles of muscle of the ventricular septum. (b) Cross section through the ventricular septum (V. S.) showing the opening (arrow) on the left ventricular (L. V.) aspect of the ventricular septum. Toward the right ventricle the tract between the two ventricles is closed by fibrous tissue. (c and d) Case 7. (c) At the junction of the lower two thirds and upper one third of the ventricular septum is a slit-like opening (between arrows) representing the left ventricular side of an interventricular communication. (d) Low-power photomicrograph of cross section through the opening observed in the left ventricle and the ventricular septum (V. S.) It is apparent that at this level the communication between the left ventricle (L. V.) and right ventricle (R. V.) has been closed by the interposition of fibrous tissue. The latter is partly dense collagenous tissue and contains some elastic fibers while the remainder is loosely collagenous.
The remaining question is whether the fibrous tissue which separates the two ventricular cavities was formed before or after birth. The absence of a history suggesting cardiac disease in early life and the absence of jet lesions in the right ventricle opposite the lesion in question suggest that the defect in the muscular septum was closed many years before the death of the subjects concerned. From the size of the fibrous plug, one would be led to the view that the interventricular communication was comparatively large at the time of closure, and hence may have existed as a defect at the time of birth and for an unknown period thereafter. It is perhaps possible that these cases represent the pathologic counterparts of clinical examples of spontaneous closure of certain ventricular septal defects in infancy or childhood.

Summary

Pathological evidence for spontaneous closure of ventricular septal defect in seven adults has been presented. These cases were observed among 1,605 necropsies (an incidence of about 1 per 225 necropsies).

In two of the cases, the ventricular septal defect was closed by an adherent tricuspid valve, possibly the result of bacterial endocarditis.

In the remaining five cases a muscular ventricular septal defect was closed by a fibrous membrane.

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100 Years Ago—Cardiac Catheterization

(a) Recording apparatus; (b) pressure curves from right atrium, right ventricle, and apex beat of a horse.—E. J. Marey: Du mouvement dans les fonctions de la vie. Paris, Germer Bailliere, 1868, pp. 141 and 143.
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