Virchows Arch. A Path. Anat. and Histol. 362, 343—352 (1974)

Morphogenesis of Traumatic Ventricular Septum Ruptures of the Heart

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Received December 4, 1973

Summary. Following the attempt to arrange a clear description of the physical characteristics of blunt thoracic trauma, the morphological findings of a case of traumatic ventricular septum defect with interventricular and atrioventricular communications is presented in detail.

Accepting the principle that the occurance of a traumatic defect in the ventricular septum without a previous lesion of the myocardial texture is highly improbable, a embryologic based theory of the formal genesis of traumatic ventricular septum defects, in sense of a "Pathoklise", is reviewed.

The formal relationships between (1) "high-seated" traumatic ventricular septum defects (type I) and congenital heart anomalies following alteration of the vectorial bulbus turn and, (2) between the purely muscular, apical traumatic ventricular septum defects (type II) and connatal interventricular communications at "atypical location" are demonstrated.

Zusammenfassung. Nach dem Versuch einer übersichtsmäßigen Darstellung der physikalischen Gegebenheiten des „Stumpfen Thoraxtrauma" werden eingehend die morphologischen Befunde eines Falles von traumatischem Ventrikelseptumdefekt mit interventriculärer und atrioventriculärer Kommunikation dargestellt.

Ausgehend von der Ansicht, daß eine Entstehung traumatischer Defekte der Kammerscheidewand ohne Vorschädigung der betreffenden Strukturen kaum denkbar ist, wird eine embryologisch begründete Theorie zur formalen Genese traumatischer Kammerscheidewandrupturen im Sinne einer Pathoklise vorgelegt.

Dabei werden formalgenetisch-verwandtschaftliche Zusammenhänge zwischen „hochsitzenden" traumatischen Ventrikelseptumdefekten (Typ I) und Herzfehlbildungen infolge Alteration der vektoriellen Bulbusdrehung einerseits und zwischen rein muskulären, apexnahen traumatischen Kammerscheidewandrupturen (Typ II) und konnatalen interventriculären Kommunikationen in „ungewöhnlicher Position" andererseits aufgezeigt.

Congenital defects of the ventricular septum of the heart rank as the most prevalent within the complex of "Defects of the heart and the great vessels". Traumatic acquired septum defects are rare. An exact statistic as to their frequency is not available due to the nature of our accident statistics which do not document the full details of such lesions (Höpker, 1973).

The cadaver experiments by Barie in 1881 have shown that blunt thorax trauma is one of the most frequent causes of cardiac injury and especially ventricular septum ruptures. Trauma to the anterior middle of the thorax and left parasternal region between the third and sixth rib are most frequently associated with such injuries (Grosse-Brockhoff, 1960). However, a blow delivered to the back can also cause cardiac injury.

A direct correlation can seldom be drawn retrospectively between the actual physical trauma and the morphologic form and localization of the individual

cardiac lesion. However, we feel a few preliminary remarks on the physics involve are noteworthy:

Generally the intensity of the effective trauma is dependent upon the impulse (energy impact) of the force directed against the thorax. The energy impact $\overrightarrow{\Delta p}$ is the product of the energy F and the duration Δt of its application.

$$\overrightarrow{\Delta p} = \vec{F}\,\Delta t \text{ [Nsec]}$$

(Brenneke, Schuster, 1966).

The definition of the energy impact $\Delta p = F\,\Delta t$ is true only in the special case where the directed energy is constant. This prerequisite is seldom fulfilled. In general, the representation of the function $F(\Delta t)$ is a curve which rises to a peak and then returns to zero. The total impulse is then derived from the addition of the individual impulses:

$$\Delta p = \sum_{i=1}^{n} F_i\,\Delta t_i$$

and from the limiting value

$$\Delta p = \lim_{\Delta t_i \to 0} \sum_{i=1}^{n} F_i \Delta t_i = \int_{t_a}^{e} F\,(t)\,dt.$$

The energy impact applied to a body is thereby a time integral of the applied energy.

The injury incurred by the heart is primarily determined by the way the energy directed to the thorax is transferred to the myocardium. Aside from factors such as localization and surface area of the blow, the elasticity of the thoracic wall and organs which transmit the blow, play a deciding role.

The thoracic wall follows the principle of elastic deformation, especially in the young. Deformations are considered elastic when the affected body returns to its original shape by itself (Weizel, 1958). The elastic modulus ideally follows Hooke's law:

$$dl = \frac{Kl}{qE}.$$

During the elastic deformation the deforming energy exerts work on the body. The form-changing work is stored in the deformed body as E_{pot} which can be transformed into E_{kin} again, but no longer exert a force on the heart. This protective function of the thoracic wall for the heart and large vessels is only under certain circumstances grossly impaired, for instance by rib fractures, when the elastic protective continuity is interrupted allowing the possibility of a direct blow to the thoracic organs.

According to these physical preliminaries an impact to the thoracic wall can cause the following:

1. a deformation of the elastic thoracic wall skeleton;
2. a contusion of those areas of the heart which lie directly beneath the thoracic wall and
3. an acceleration of the heart in the direction of the energy application.

Depending upon the resulting energy lost in work necessary for the deformation, the following consequences for the heart are possible. Light trauma need not

affect the heart at all as a result of the energy lost. Stronger blows would lead to the clinical picture of cardiac commotion or contusion. Still stronger impacts can lead to ruptures especially at the attachment of the large vessels as a result of a slinging action.

An especially important factor determining the severity of the cardiac injury and development of internal cardiac damage by trauma to the thoracic wall is during which phase of the cardiac cycle the thoracic wall is deformed. Blows delivered directly following the ejection phase usually cause only external cardiac injury while those delivered to a filled ventricle are transmitted by the incompressible blood-fluid to internal structures (Grosse-Brockhoff, 1960) resulting in ruptures of the valves due to hydraulic pressures, rarely causing septum perforation.

Our Case

A 19 year-old student suffered a blunt trama to the thorax while working during his vacation. While unloading a truck a heavy box fell on the patient. Upon admission to the University Surgery Clinic in Heidelberg a loud systolic flow murmur over Erb region was heard, and neck vein distention was noted. A special cardiologic diagnosis was ordered and a defect in the septum ventriculorum found, which could only be a result of the trauma, shunting blood from the left ventricle to the right ventricle and from the left ventricle to the right atrium.

The defect was operatively closed with a patch under extracorporal circulation.

Postoperatively large amounts of hemolytic urine were produced followed by acute kidney failure. This was combated using daily hemodialysis. The clinical picture was complicated by an exaggerated bleeding tendency due to consumption coagulopathy and progessive respiratory failure.

The patient died 11 days after surgery under the clinical picture of acute cardiogenic shock.

Excerpts from the Autopsy Report
(SN 1015/73, Pathological Institute University of Heidelberg)

Body of a 182 tall, 69 kg heavy young male adult. No visable external injuries. A subcutaneous hematoma in the left inguinal region, 20 by 30 cm in size, superficial and only a few mm in depth.

The *heart* is enlarged (weight 430 g). A fresh fibrinous epi-pericarditis is present. In the area of the conus arteriosus pulmonalis is a 4 cm long, unsuspicious ventriculotomy scar in the flow direction. The opened heart presents a mild dilatation of all chambers. The venous entrances to the heart are normal, the atrioventricular ostia are typical. A membranous septum of the Jarisch type II is present. In the region directly beneath and dorsal to the crista supraventricularis is a 15 mm to 12 mm ventricular septum defect which is closed with a dacron patch. This defect lies in the area of the membraneous septum and, when not closed, allows a communication between the left ventricle and the right auricle as well as between the left and right ventricles. Observed from the left ventricle (see Fig. 1) the defect presents as subaortal between the right and posterior semilunar valves. It is placed in the middle of a half-moon shaped rip in the endocardium which includes the aortic leaf of the mitral valve in the area of Koch's point.

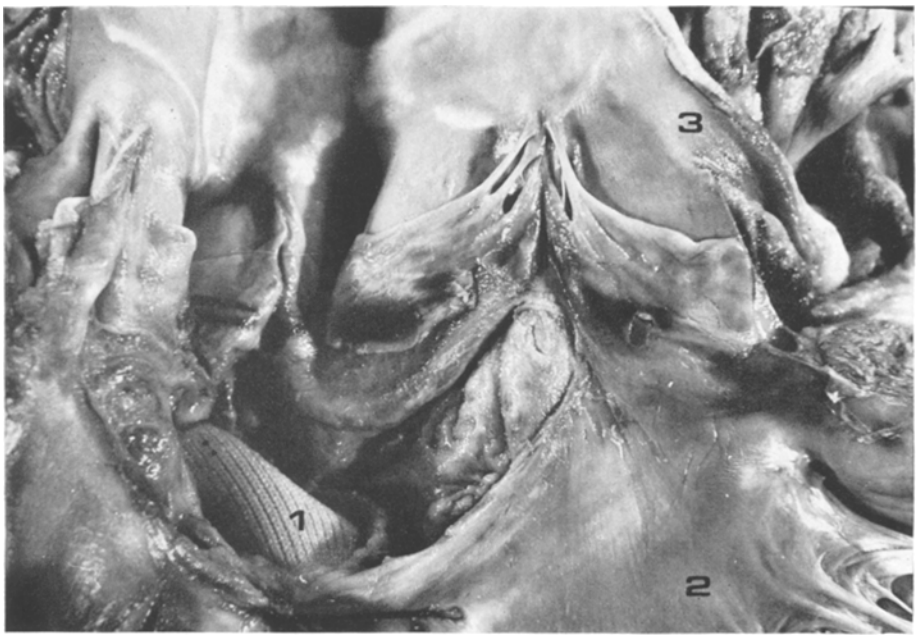

Fig. 1. Subaortal half-moon shaped tramatic rip in the endocardium with a central interventricular and atrioventricular communication, which is now closed with a dacron-patch. *1* Dacron-patch, *2* aortal leaf of the mitral valve, *3* origin of the left coronary artery

Inferior to the defect are diffuse areas of subendocardial hemorrhage in the anterior portion of the ventricular septum which however do not include the branching of the left AV-bundle-branches.

Inflammatory changes or congenital defects in the area of the lesion are macroscopically absent.

The outflow tracts of both ventricles are typical. Moderate lipoidotic deposits are found in the ascending aorta. The coronary arteries are normal. In the area of the left ventricular myocardium are miliary muscle fiber necroses, especially in the internal layer. Microscopic examination of the defect's borders excluded any injury to the central portion of the ventricular conduction elements (see Figs. 2, 3 and 4).

Further Pathologic-Anatomic Findigs

Diffuse hemorrhage into the mediastinum, the retropertoneal space and muscles of both thighs. Condition following long-term intubation: acute necrotizing tracheitis. Purulent bronchitis; shock lungs with beginning pneumonic process; purulent-fibrinous pleuritis bilaterally. Enlarged, fatty and edematous liver with subacute circulatory insufficiency. Spodogenic spleen tumor.

Acute tubulopathy of both kidneys with cortical ischemia and relative medullary hyperemia (so called "shock signs").

Traumatic fissure of the left shenoid bone with hemorrhage into the left inner ear. Moderately large subarachnoid hemorrhage without compression signs at corresponding locations. Brain edema with compression into the foramen magnum.

Minimal lipoidosis of the walls of the large arteries.

50 ml of coagulated blood in the posterior pharynx.

Condition after application of a Scribner shunt in the left arm.

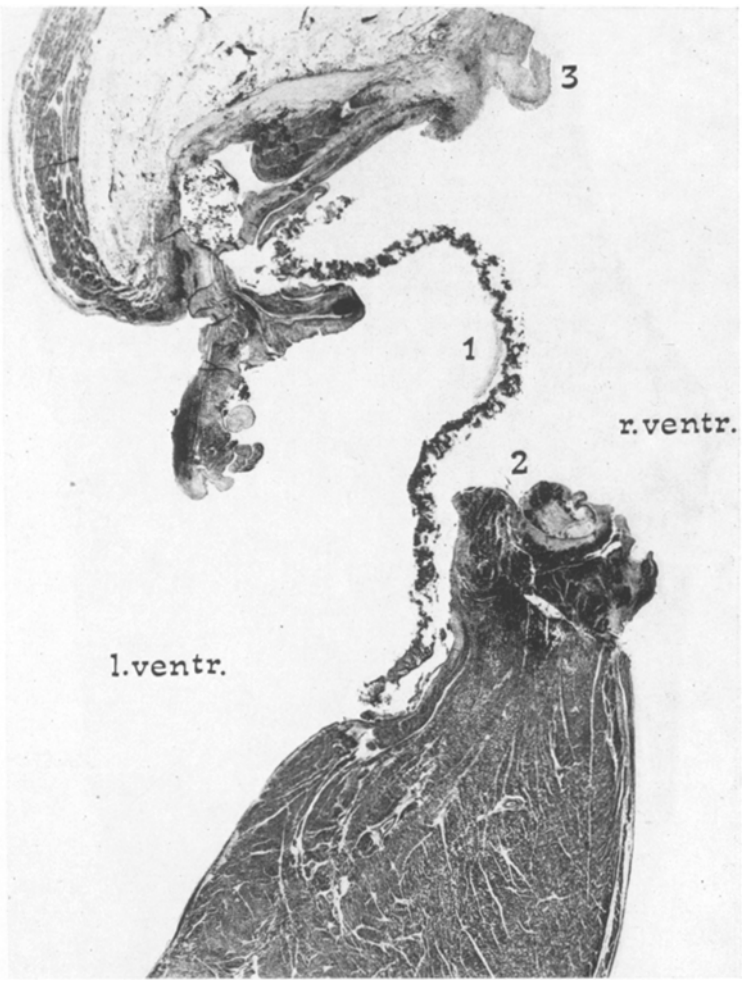

Fig. 2. Histologic section (magnifying glass) through the region of the defect in the area of the commissure between the right and the posterior semilunar valve of the aortic ostium (Topography see Fig. 1). *1* Dacron patch, *2* ventricular defect border, *3* right semilunar valve of the aortic ostium

Discussion

The diagnosis of a traumatic ventricular septum defect (VSD), once it has been considered in the differential diagnosis, is not very difficult. The possibilities of open-heart surgery allow adequate treatment in most cases. The question how an acquired, especially a traumatic VSD occurs has been experimentally and theoretically studied. The question *why* a traumatic VSD occurs had not until now stimulated much attention and should be discussed.

The characteristic type of accident which leads to a traumatic VSD is the "steering wheel injury" whereby the heart is compressed in the sagittal plane

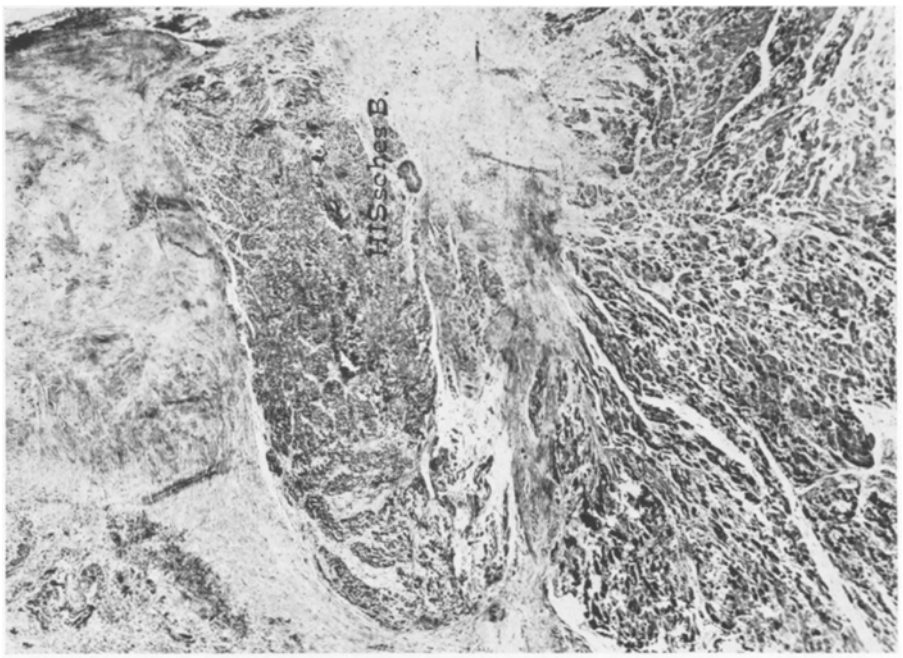

Fig. 4

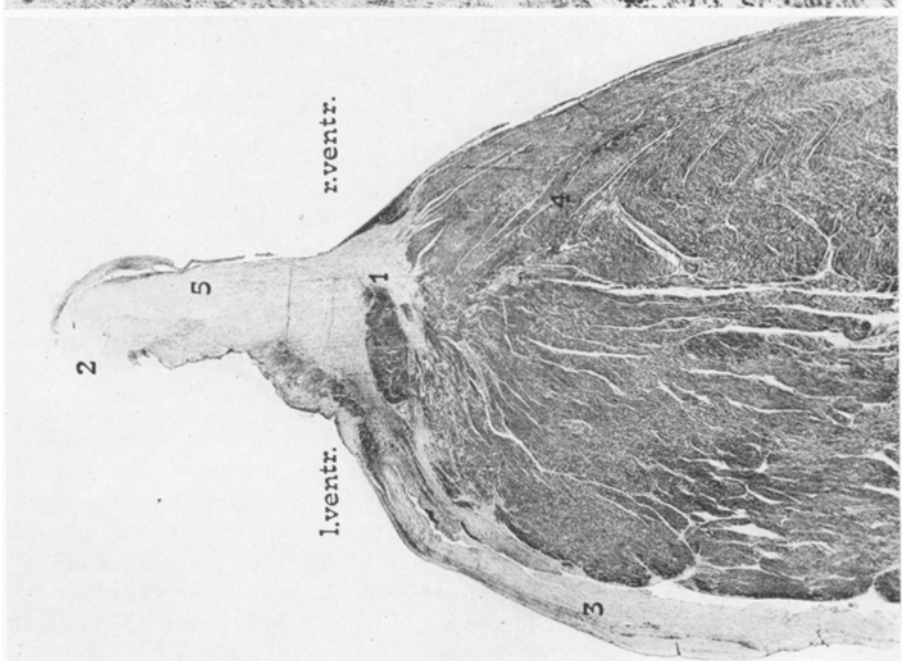

Fig. 3

and overexpanded in its diameter (Grosse-Brockhoff, 1960). It is quite obvious, however, that a definite discrepancy exists between the frequency of traumatic acquired ventricular septum ruptures and such thoracic injuries. It therefore appears legitimate to postulate that a previous lesion in the interventriculare septum texture, representing a weak location, must already exist, before a traumatic rupture can occur. The weak location can be circulatory or inflammatory in origin. However, in the special case of traumatic acquired VSD in previously cardiac healthy persons, we prefer to base the occurance on mechanically weakened locations as a result of impaired embryonic cardiac development.

Two types of traumatic defects can be differentiated depending upon their locations in the ventricular septum, each with its own formal genesis.

As type I we describe a high, subaortic defect related to the pars membranacea septi ventriculorum; depending upon the size of the septum membranaceum (Type classification according to Jarisch, 1911) it can lie interventricular, atrioventricular or both. The more apically placed, purely muscular defects, similar to the Morbus Roger lesion, we classify as type II.

According to that the case mentioned above would to classify as type I; comparable findings were reported by Meessen (1964) and Dunseth (1965).

The type II defect appears to be the more frequent lesion (Parmley, 1958; Feruglia, 1960; Cleland, 1961; Carter, 1967; Rotman, 1970).

Any attempt at an explanation of the formal genesis of traumatic ventricular septum defects must be accompanied by a typology of congenital interventricular communications. According to its localization an acquired VSD type I correspondents to a congenital defect in the posterior portion of the anterior ventricular septum (area 2–4; y, Y) (Classification of Rokitansky, Spitzer, Warden, Goerttler), which comprises 72% (Mayo clinic) of all ventricular septal defects.

The embryologic equivalents in the developing heart of these defects are the bulbar ridges A and B, the bulbar septum, portions of the muscular ventricular septum and the bulboauricular flange. These structures normally fuse to close the foramen interventriculare secundum and form the superior portion of the interventricular septum. This fusion necessitates a "movement of material", meaning a swiveling of the bulbus cordis out of the frontal plane into the parasagittal plane with simultaneous clockwise torsion around its long axis. This material movement has been described by Doerr in 1955 as "Vectorial bulbus turn". Primarily through this action are the provisions for a morphological coordination of related functional structures achieved. The actual contact surfaces between the corresponding portions of the ventricular septum anlage represent inhomogeneous zones wherein pathological processes can occur (Doerr, 1967). In other words: when structural defects or an incomplete vectorial bulbus turn induce develope-

Figs. 3 and 4. Histologic section (Magnification 1:25/1:40 both pictures stem from the same slide) through the region of the defect near the commissure between the posterior and the left semilunar valve of the aortic ostrium (topography see Fig. 1). The bundle of His is intact and sitting atop the muscular septum ventriculorum clearly distant from the defect border. *1* Bundle of His, *2* inferior defect border, *3* aortal mitral leaf, *4* muscular ventricular septum, *5* part of the membraneous septum

mental anomalies, these are manifested primarily at the fusion lines between membranous bulbar and muscular ventricular septum, at first in the form of more or less larger gaps in the ventricular septum. Anomalies of sufficient severity could cause a persistence of embryonic relationships prior to the 60th day of developement resulting in severe lesions of the "overriding vessel type" (Eisenmenger complex; Fallot's tetralogy; Taussig-Bing complex; transposition of the great arteries; double outlet right ventricle).

Following this same line of thought, it could be possible that a lesser degree of arrest of the embryonic development would still allow minimal contact and fusion of the corresponding septum components despite reduced tissue mass. The incorporation of the bulbus into the left ventricle would then not be "seamless", although the separation has fully occured, leaving a mechanically and structurally weakened area as expression of the disarrangement of the fibromuscular architecture, which would be then predisposed to perforation under conditions of abnormally increased pressure.

We prefer to classify this form of mild dextropositio aortae as a morphological pendant to the ring stenosis of the aortic conus, whose pathologic anatomy and formal genesis has been discribed detailled in 1959 by Doerr. The embryonic relationship of these two anomalies can be expressed as an "too much" or an "too little" of the vectorial bulbus turn. Should the aorta fail to reach its position (mild form of dextroposition) or overshoot it, grotesque confusions would result at the fusion line between the bulbus septum and the ventricular septum (Doerr, 1959), which are of fundamental importance for anomalies in the "région mitro-aortique" (Mall, 1912). The fact that the formal genesis of traumatic ventricular septum defects type I is coupled with the course of the vectorial bulbus turn leads us to the opinion that this anomaly belongs in the group of teratological cardiac defects which include ventricular septum defect, overriding vessels, the transpositions and dextropositions.

The type II of traumatic ventricular septum defects can be found in every part of the septum, especially in the area of the apex. They lie far from any fusion zones in the middle of the muscular ventricular septum. Experimentally, hypoplasia of the ventricular septum with persistence of the primitive spongiosa texture can be induced by trypan-blue (Wegener, 1961). These defects are the result of a hypoplasiogenic growth whereby the septal myocardium is retarded by a general cardiac enlargement and, in place of a gradual replacement of the spongiosa, an aneurysmatic dilation of the sinusoidal vessels perseists (Goerttler, 1968). These are then persistent spongiosa-spaces which develope during the formation of the interventricular septum from the ventricular metamer and remain at this arrested level of development. These interventricular spaces are usually small and probably often close spontaneuously, but secondary perforations are still possible in this weakened area (Goerttler, 1968).

These spontaeously closed, primary congenital ventricular septal defects in "atypical position" appear us to be an important factor for the predisposition to secondary traumatic defects. A histological documentation of this could certainly be achieved.

On the other hand, it cannot be conclusively excluded, that in an analogous way to type I, spongiosa gaps persist as retardation anomalies due to growth

arrest. These would be no defects of clinical significance, but these areas would be structurally weak in their tissue texture and not able to resist a sudden increase in pressure.

In general, any structure of the whole heart is endangered by the effect of a blunt thoracic trauma. We tried to demonstrate that there exists a disposition for circumscribed parts of the ventricular septum to be affected in a specific way on causes of embryogenic morphological facts and that this is linked up with a higher danger of injury as a consequence of a trauma to the thorax. The idea suggests itself to speak in this connection about a "Pathoklise" (pathoclisis) in the sense as defined by Vogt in 1925, similarly as Jansen explained it in 1962 for the sidediffering occurance of myocardosis in the right- and leftsided ventricular myocardium.

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