Diverticulum of the Pericardium
With Observations on Mode of Development

By Herbert C. Maier, M.D.

A diverticulum of the pericardium can often be confused with other lesions of the heart and great vessels as well as mediastinal cysts and other masses. Pericardial effusion, sometimes associated with heart lesions causing cardiac enlargement, is a frequent factor in producing diverticular formation. The mode of development of a pericardial diverticulum is better appreciated when certain features of the anatomic structure of the parietal pericardium are understood. Two cases of pericardial diverticulum that illustrate the etiologic, clinical, and roentgen features of the lesion are presented.

A diverticulum of the pericardium may develop secondary to cardiac disease and pericardial effusion. When discovered on roentgen examination, the appearance may simulate cardiac or aortic aneurysm, vascular anomalies, cardiac and pericardial tumors, and mediastinal cysts. Since a diverticulum of the pericardium is rare, there has thus far been little opportunity to follow roentgenographically the evolution of the lesion over a period of years. Two cases of diverticulum of the pericardium herewith reported have provided some information concerning the mechanisms of development. The opinion has been expressed by some observers that localized weakness of the pericardium with an additional factor of pericardial distention by fluid was the usual responsible mechanism. One of our cases in which clinical and radiographic data were available over a period of years demonstrated this sequence of events after an attack of idiopathic pericarditis. The other case indicated the close relationship between pericardial diverticulum and the more common pleuropericardial cysts, which are of congenital origin.1

The mechanism of development of an acquired pericardial diverticulum is more readily understood when certain points related to the histology of the pericardium are appreciated. The parietal pericardium is a fibroserous sac that consists of an outer fibrous layer and an inner serous layer. The pericardium merges with the sheaths of the great vessels with the single exception of the inferior vena cava, which pierces the pericardium from below. The outer fibrous layer is strong and inelastic due to interlacing collagenous fibers. The serous or inner layer of the parietal pericardium has a smooth and glistening lining that is covered by mesothelium. After splitting the outer fibrous sheath one can separate the inner serous layer from it and thus demonstrate grossly the 2 layers. After separation of these 2 layers over a small area it is then possible to evaginate the inner serous lining through the defect in the outer fibrous covering. Thus one can demonstrate the mechanism by which a herniation of the inner serous layer may occur through a cleft or weak place in the outer fibrous layer. Since the inner serous lining is far more distensible than the outer fibrous covering, a protrusion of the thin inner layer could gradually produce an enlarging diverticulum as a result of increased intrapericardial pressure such as is caused by cardiac enlargement and pericardial effusion. Study of the pericardium at autopsy demonstrates some variation in the ease with which the parietal pericardium can be split into its 2 component layers. In some cases a cleavage plane can be readily established between the outer fibrous and inner serous layer over a considerable distance because only delicate fibers join the 2 layers. In other instances, especially in the atrophic pericardium of the aged, demonstration of the 2 layers is much less satisfactory. In some areas of the parietal pericardium the interlacing collagenous fibers of the outer layer are obviously unevenly distributed. It is in the potentially weak spaces between groups of fibers, especially near

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the roots of the great vessels, that the thin serous layer may bulge through when the intra- pericardial pressure increases. The pericardial fluid can then distend the protruding serous layer so as to produce a gradually enlarging sac. Rarely a diverticulum may be produced by localized traction on a small area of pericardium.

It is stated in the literature that the wall of a pericardial diverticulum is similar to that of the pericardium but this does not appear to be entirely correct. Although on ordinary microscopic examination no striking difference in the appearance of the wall of a pericardial diverticulum and the normal parietal pericardium may be readily apparent, a macroscopic difference may be noted. The diverticulum does not possess the dense bands of collagen fibers that impart some degree of stiffness to the normal parietal pericardium. In contrast the diverticular sac is very flexible and flaccid. However, the wall of the diverticulum tends to be thicker than the serous layer of a normal pericardium. When a pericardial diverticulum is observed at operation, one notes the tendency of some pericardial fluid to flow back and forth between the pericardial sac and the diverticulum during phases of the cardiac cycle. This rhythmic distention of the herniating sac may well play a role in its gradual enlargement. The diverticulum readily varies in shape and size with changes in position of the patient. The sac usually hangs down from the site of communication with the pericardium and is often pear-shaped.

A critical review of the literature emphasizes the necessity of discarding from consideration those cases in which the diagnosis was not proved by autopsy or operation. There are some reports based only on radiologic findings that are not even suggestive of a pericardial diverticulum when considered in the light of recent surgical experience with mediastinal masses. In other reports no distinction is made between diverticulum, localized pericardial effusion with regional protrusion, or localized perforation of the pericardium from a pathologic process such as tuberculosis. Moreover, in some of the cases designated as diverticulum of the pericardium the sac with fluid did not communicate with the pericardial cavity but was merely attached to the pericardium. In recent years such pleuropericardial cysts have been recognized as relatively common lesions. This discussion is concerned only with cases in which there is localized outpouching of the pericardium so as to form a sac that protrudes from the remainder of the pericardium and communicates with the pericardial cavity. Only a relatively small number of cases in this category are available for study in the collected literature.

Cushing reviewed the literature on diverticulum of the pericardium in 1937. Some of the 40 cases he listed were unproved pathologically or may not be true diverticula. Various cardiac or other abnormalities that tended to distend the pericardial sac were recorded in a considerable percentage of the proved cases. The diverticulum presented most often on the right side. In most instances details concerning the exact site of the diverticulum were lacking but the sites seemed to vary considerably; in a few cases the origin was stated as being at the base near the origin of the great vessels. The size and shape of the diverticulum as well as the width of the neck of the sac varied considerably. None was very large. Most were incidental findings at autopsy in adults. Reports of pericardial diverticula in the newborn and in infancy seem to be lacking, if pleuropericardial cysts, which do not communicate with the pericardial cavity, are excluded.

The criteria of radiographic diagnosis of a pericardial diverticulum reported by Kienböck and Weiss on the basis of largely unproved cases seem unacceptable, since similar roentgen shadows may occur with pleuropericardial cysts, lymphatic cysts, and serous cysts. The density on the roentgenogram produced by a pericardial diverticulum is inseparable from that of the cardiac silhouette. The shape and location of the abnormal shadow may vary widely, and there may be a considerable change in the shape with changes in respiration and position. Some such alterations in relation to the cardiac silhouette may make it difficult to distinguish the diverticular outline from other mediastinal structures. The only absolute roentgen sign of a pericardial diverticulum...
other than with pneumopericardium is disappearance of, or conclusive evidence of diminution in the size of the cystic mass when the patient is so positioned, as in lateral recumbency, that the diverticulum lies above the pericardium. Then some of the fluid in the diverticulum may drain back into the pericardium. It is usually very difficult to be certain that the cystic lesion has changed its volume because a thin-walled cyst that is not tense can change its shape so markedly that it is impossible to tell whether the volume is altered, even when films are taken in several projections. Misinterpretation may also result from shifting of the cystic mass in relation to the cardiac silhouette, so that the abnormal density may be thought to have disappeared when it has merely shifted its position and become superimposed on the cardiac shadow. Even pericardial fat pads and lipomas can be confused with cystic lesions around the pericardium. Sometimes a diverticulum with a large free communication with the pericardium will not empty on change in position because the pear-shaped diverticulum with a long neck can shift the position of its lower bulbous portion so as to remain below the level of the opening into the pericardial sac. Also, if there is a very small communication between the diverticulum and the pericardial cavity, fluid will not readily drain from the diverticulum.

A pericardial diverticulum might be conclusively diagnosed in some cases by pneumopericardium but such a diagnostic procedure would appear to be undesirable.

Pericardial diverticula are often asymptomatic.\(^8\) Occasionally there is a complaint of vague discomfort in the thorax. In a few cases circulatory signs and symptoms such as dyspnea, dizziness, and slight cyanosis have been present that have disappeared postoperatively although the mechanism of their production was not apparent unless torsion of a long pedicle was responsible or unrecognized cardiac lesions were present. Physical examination and auscultation do not aid in establishing the diagnosis. Electrocardiograms are normal unless alterations are produced by cardiac or other pericardial disease.

**Case Reports**

**Case 1.** This 49-year-old man had a history of chest pain for 1 day in 1947 and at that time a definite pericardial friction rub had been heard by several observers. The chest roentgenogram had shown no abnormality or enlargement of the cardiac silhouette (fig. 1A) and the lung fields were clear. A diagnosis of idiopathic pericarditis was made by Dr. C. de la Chapelle. In 1952 another chest roentgenogram was taken, and at this time a small bulge was noted at the right cardiac border on the posteroanterior film. The cardiac silhouette also showed some enlargement as compared to the roentgenogram of 1947. The electrocardiogram was normal. Slight hypertension was present and the enlargement of the cardiac silhouette was thought to be related to the hypertension. A year later another chest film showed a definite increase in size of the abnormal shadow (fig. 1B and C) and operation was recommended. The patient remained free of pulmonary or chest symptoms. The mass was found to be definitely larger than it had been 15 months previously. At operation in July 1953 a large pear-shaped diverticulum of the pericardium having its origin in the region of the base of the aorta was found and resected. There was an increased amount of pericardial fluid present. The postoperative course was uneventful and the patient was discharged on the eighth postoperative day. A year after operation the patient was asymptomatic and feeling fine. Microscopic examination of the wall of the diverticulum revealed that it was composed of a small amount of fibrous connective tissue covered by a single layer of somewhat hyperplastic endothelial cells. There was a slight chronic inflammatory cellular infiltration as well as dilated thin-walled and congested blood vessels.

**Comment.** Preoperatively the lesion had been considered to be probably a thymic tumor because of its location. At that time it was erroneously assumed that a pericardial diverticulum would not be likely to make its appearance in a 49-year-old man who had no evidence of such a lesion on roentgenographic examination when he was 45 years old. At the time of operation it was noted that the pericardial fluid was increased in amount. The postoperative roentgenograms showed a definite decrease in the size of the cardiac silhouette, indicating that the increase had been due to an abnormal amount of pericardial fluid rather than to cardiac enlargement (fig. 1D). In retrospect it is thought that the increased pericardial fluid that followed the idiopathic pericarditis 6 years previously had resulted in the development of a pericardial diverticulum by gradual protrusion.
of the inner serous layer through a cleft in the fibrous bands of the outer layer.

Case 2. This 29-year-old man had occasional slight discomfort in the right anterior thoracic region. Roentgenograms of the chest showed a small mass of soft tissue or fluid density in the right supradiaphragmatic region adjacent to the cardiac border (fig. 2). The shape of the mass changed considerably with respiration; it was rounded on expiration and became elongated on inspiration. Barium studies of the alimentary tract revealed no evidence of a retrosternal diaphragmatic hernia.

At operation an irregular multilocular thin-walled cyst containing clear fluid was found attached to the anterolateral aspect of the pericardium. This was typical of a pleuropericardial cyst but a unique finding was a small pericardial diverticulum attached to the cyst by a fibrous strand. There was some excess fluid within the pericardium. The cyst and diverticulum were removed. The postoperative course was uneventful.

Comment. The lack of reports of cases of pericardial diverticulum in infancy and childhood and the frequent association of pericardial effusion or other factors that would distend the pericardial sac strongly suggest that most pericardial diverticula result from a gradual outpouching through a weak area in the pericardial wall. On the other hand, the close developmental association between pleuropericardial cysts, which are apparently usually of congenital origin, and abnormalities of pericar-
dial development seems evident. The pathologic findings in this case are of interest in this regard, since here a pleuropericardial cyst was intimately associated with the tip of a small diverticulum of the pericardium. Whether the development of this diverticulum was in part due to a traction effect of the cyst remains a moot question.

TREATMENT

It is questionable whether a pericardial diverticulum would usually require any therapy if a positive diagnosis could be established by radiologic and clinical criteria. At present it seems unjustified to assume that the radiologic features that have been described and discussed in the literature are sufficiently accurate to be considered diagnostic or reliable except in rare instances. Although alterations in the shape and contour of a pericardial diverticulum may be noted with changes in position or respiratory motion, similar variations may occur in pleuropericardial cysts and even other lesions. Therefore the management of the patient must be based on a realization of the limitations of radiographic differentiation of the large variety of mediastinal masses and vascular lesions that could simulate a pericardial diverticulum. Angiography will help to identify lesions of the heart and great vessels. When the radiographically detected mass is considered to be of nonvascular origin, thoracic exploration is advisable unless other conditions produce contraindications.

SUMMARY

The evidence concerning the mechanism of development of a diverticulum of the pericardium is discussed on the basis of 2 personally observed cases, some experimental autopsy observations, and an analysis of cases recorded in the literature. The development of a large diverticulum that was not roentgenographically demonstrable until some years after an attack of idiopathic pericarditis is recorded. Although congenital factors may play a predisposing role by providing a weak area in the parietal pericardium, an increase in intrapericardial pressure with pericardial effusion seems to be the important causative factor in many cases. A pericardial diverticulum usually results from the gradual stretching of a herniating portion of the inner serous layer of the parietal pericardium that bulges through the split or weakened outer fibrous layer of pericardium.

Evidence supporting the concept that pleuropericardial cysts and a pericardial diverticulum may have a common origin is indicated by a case in which both lesions were associated.
together. However, congenital diverticulum of the pericardium seems to be extremely rare.

SUMMARIO IN INTERLINGUA
Le mechanismo del disveloppamento de diverticulos pericardial es discutite super le base de 2 personalmente observate casos, un serie de necrotic observationes experimental, e un analyse de casos reportate in le litteratura. Es reportate le disveloppamento de un grande diverticulo que non eseva demonstrabile per medios roentgenographic usque a plure annos post un attacco de pericarditis idiopathic. Ben que il es possibile que factores congenite exerce un rolo predispositori per provider un area de debilitate in le pericardio parietal, un augmento del pression intrapericardial con effusion pericardial pare esser le causa immediate in un grande numero de casos. Le processo usual es que un diverticulo pericardial resulta ab le tension gradual del portion herniata del strato serose interior del pericardio parietal que protrude a transverso le findite o debile strato fibrose exterior del pericardio.

Le conception que cystes pleuropericardial e diverticulos pericardial ha possibilemente le mesme origine es supportate per un caso in que le duo lesiones eseva associate. Tamen, diverticulos congenite del pericardio pare esser extremely rar.

REFERENCES


Internal or common carotid artery ligation was performed in 63 patients because of intra cranial aneurysm (44) or arteriovenous communication (19). Preoperative evaluation included angiography to determine the status both of the carotid and the basilar systems. If these were satisfactory, the patient’s ability to withstand carotid ligation was then tested by high carotid compression for 10 minutes under electroencephalographic and electrocardiographic monitoring. Those individuals having an irritable carotid sinus were protected by the prior administration of 1.6 mg. of atropine. When carotid compression produced no untoward reaction, carotid ligation was followed by death in 2 patients both of whom were previously very ill, by transient hemiparesis in 4 and by no significant side effects in the remainder.

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