RADIOLOGICAL DIAGNOSIS OF RHEUMATIC PERICARDIAL EFFUSION

BY

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Twenty-one cases of rheumatic pericardial effusion have been studied in order to determine the precise nature of the radiological signs, including their frequency and the time of their appearance. The effusion recurred in two cases so that twenty-three episodes have been investigated in all. In fifteen the effusion developed after, and in the remainder before the patients were admitted. Seven of the patients died when effusion was still present. It was possible to study the radiological signs of effusions as they developed and of fourteen as they subsided. Proof of the diagnosis was obtained in twelve cases; at autopsy in seven and by cardiac catheterization in five: of the remaining nine, typical electrocardiographic changes occurred in five, pericardial fluid was aspirated from one, and convincing clinical signs of effusion were found in all. The course of the electrocardiographic and clinical signs followed the same pattern that was observed in the cases of pericardial effusion proven by autopsy or catheterization.

SIGNS OF DEVELOPING PERICARDIAL EFFUSION

Rate of increase in size of heart shadow. The rate of change in the size of the cardiac silhouette could be determined in 13 of the 15 cases. Generally, the cardiothoracic ratio increased rapidly to attain its maximum within a few days: it increased from a mean of 54 to 68 per cent during a period of eleven days, this being the average time for the effusions to reach maximum size (range, 4 to 25 days). The mean difference was 13.7 per cent (S.D. 6.5%). A significant change in cardiothoracic ratio was amongst the first major radiological signs of effusion in all cases.

Shape. A sudden change in shape was apparent in most cases: the normal sub-division of the silhouette became obliterated and the whole cardiac shadow assumed a more spherical appearance. The shape resembled that of a pear, carafe, or onion in different cases, depending on the size of the effusion. This change was amongst the early signs of effusion in ten and amongst the later signs in five.

Obliteration of the normal contours of the left border always occurred early: it resulted in the appearance of a straight left border extending upwards to include the lower segment of the aortic knuckle. With large effusions the left border developed a smooth convex bulge in its upper half, this change being of diagnostic value in four cases which already had straight left borders prior to the onset of their present attack of pericarditis. A change in shape of the left border was one of the first signs of effusion in fourteen cases.

A change in shape of the right border occurred fourteen times and was one of the first radiological signs of effusion ten times. The right cardio-diaphragmatic angle remained unchanged in eight, became more acute in four, and less acute in two cases at the same time as the shape of the right border changed.

Other signs. Disappearance of the shadow of the first part of the descending thoracic aorta was amongst the early signs of effusion seven times and amongst the later ones once. In the remaining seven the shadow had not been seen prior to the onset of pericarditis.
A mean increase of 42 per cent (S.D. 9·4%) of the initial width of the vascular pedicle, due to dilatation of the superior vena cava, occurred in twelve cases and was amongst the first signs of effusion in seven of them.

The outline of the posterior border of the heart shadow was always indistinct in the oblique views. In one this appearance preceded the onset of the present pericardial episode; in eight it occurred early in the development of the effusion, and in the remaining four later. In four cases the oblique views showed a hazy posterior outline at a time when the pericardial effusion was still small and before pleural effusions had developed (Fig. 1), so this haziness could not be attributed either to pleural effusion or to a massive pericardial effusion.

Details of the order of development of the individual signs are shown in Table I. Thus the radiological signs which were of most value in making a diagnosis of pericardial effusion were (a) a rapid increase in the transverse diameter of the heart shadow, and (b) straightening of the left border.

These were present in all cases and were generally the earliest signs. The following signs were of secondary value; they were not always present and often appeared only at a later stage: change in the general contour, changes in the right border, increase in width of the vascular pedicle, disappearance of the shadow of the first part of the descending aorta, and haziness of the posterior border of the heart shadow in the oblique views.

**DISCUSSION**

Our findings that straightening of the left border and a rapid increase in cardiothoracic ratio are the earliest radiological signs of pericardial effusion are in conformity with those of most previous authors (Holmes, 1924; Freedman, 1939; Roesler, 1943; Fenichel and Epstein, 1946). We did not observe any consistent change of the right cardio-diaphragmatic angle as described by Johnson and Palmer (1932) and Roesler (1943): they stressed the acuteness of the angle in effusion but in over half our cases it remained unchanged as the effusion developed.

An increase in width of the vascular pedicle occurred in 13 of our 15 cases, and was an early change in 7 of them. We have found only two authors who attribute significance to this sign, namely Smith (1935) and Friedberg (1950). Others (Freedman, 1939) have only noted shortening of the pedicle. However, widening in the supine position has been described by Schwedel (1946).

Increased density of the cardiac shadow with disappearance of the line of the descending aorta has been but rarely quoted in recent years, save by Oosthuizen (1943). The limited value of this sign is illustrated by the fact that seven of our cases showed it before the onset of their present pericardial effusion.
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Haziness of the posterior cardiac border in the oblique views occurred frequently in our series. This sign was described by Roesler with very large effusions, but was often seen by us when the effusion was still small.

**TABLE I**

| Signs of Developing Pericardial Effusion (15 Cases) |
|---------------------------------|-------------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Time of development             | Increase of cardio-thoracic ratio | Straightening left border | Change of whole contour | Bulging right border | Increased width of pedicle | Disappearance of first part of descending aorta | Hazy oblique |
| Early                           | 13                | 14              | 10              | 10              | 7               | 7               | 8             |
| Late                            | 0                 | 0               | 0               | 0               | 0               | 0               | 0             |
| Present before effusion         | 0                 | 0               | 0               | 0               | 0               | 0               | 0             |
| Unchanged                       | 0                 | 0               | 0               | 0               | 0               | 0               | 0             |

**TABLE II**

| Signs of Regression of Pericardial Effusion |
|---------------------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Time of regression                          | Decrease of cardio-thoracic ratio | Decrease of vascular pedicle | Hazy oblique | Decrease of right border | Restoration of normal contour | Changing left border | Reappearance of descending aorta |
| Early                                        | 13               | 12              | 6               | 5               | 6               | 2              | 2             |
| Late                                         | 0                | 1               | 4               | 7               | 8               | 9              | 5             |
| No return to normal                         | 0                | 0               | 1               | 0               | 0               | 3              | 7             |
| Never abnormal                               | 0                | 1               | 0               | 2               | 0               | 0              | 0             |
Thus the earliest sign of regression was a reduction in cardiothoracic ratio. This was followed by return to normal of the vascular pedicle, the posterior cardiac outline in the oblique views, and the right border. Straightening of the left border and absence of the shadow of the descending aorta tended to persist, and sometimes did not return to normal.

The Effect of Posture

The effect of posture on the pericardial contour was first noted by Williams in 1903. When there is an effusion, skiagrams taken with the patient recumbent have been reported to show characteristic changes in cardiac contour. Thus Holmes (1924) found the heart shadow to be rectangular in this position, and White (1944) found it to be globular. Lewis (1942) and Schwedel (1946) considered the horizontal position to be of value only for demonstrating a change in shape of the left border. There appears to be little agreement as to the diagnostic value of these postural effects on the cardiac contour. Widening and shortening of the pedicle in recumbency was described by Schwedel (1946), Levine (1945), and Arendt (1948). We have found similar changes in cases without effusion. The recumbent lateral view showed significant displacement of the heart shadow with effusions according to Roesler, but Arendt found this position of little value.

In 1943 Oosthuizen described the sign of divergent vascular shadows in cases of pericardial effusion skiagraphed in the recumbent position—shadows extending upwards and laterally on both sides of the superior mediastinum. We have found that this sign is best seen when the patients are tilted head downwards. We have, therefore, taken skiagrams (antero-posterior at three feet) with the patient tilted head downwards at 45° and compared them with films taken head upward at 45°.

Thirty cases have been investigated: eleven with pericardial effusion, two with possible pericardial effusion: twelve with large hearts from established valve lesions and five with cardiac enlargement and a previous history of pericarditis. The sign was positive in 8 of 11 cases with undoubted effusion (Fig. 2) but also in 4 of 17 with cardiac enlargement. The presence or absence of the sign could not be closely correlated with the height of the venous pressure: all the 11 with effusion had a raised venous pressure, but three did not show this sign, and two of the four cases of enlargement with divergent shadows had normal venous pressures. When positive in cases of effusion, the sign had always been preceded by other radiological evidence. Widening of the vascular pedicle occurred in 10 of the 11 with effusion when tilted head downwards. It also occurred in 8 of the 17 with cardiac enlargement.

It is concluded that these two effects of posture on the shape of the cardiac shadow are of limited value in distinguishing between pericardial effusion and cardiac dilatation.

Retrospective Diagnosis of Effusion

In the light of these observations, the skiagrams of all previous cases that had been diagnosed as having generalized cardiac enlargement from established valve lesions were reviewed, lest any of them should show similar evidence of pericardial effusion. Retrospective diagnoses of effusion were made in 6 of the 75 cases reviewed. The skiagrams on admission in these six all showed many of the characteristic features of effusion: a typical pericardial contour with a straight left border, altered shape of the right border, an increased vascular pedicle, and absence of the shadow of the descending aorta. Indistinct posterior borders were seen in those cases in which oblique views had been taken. Subsequent skiagrams in all six cases showed a decrease in size and a return to more normal contours. Two cases had previously been in other hospitals and X-rays obtained later from these hospitals showed undoubted effusions.

Five other cases had large heart shadows with contours somewhat suggestive of effusion, but none of them had a straight left border or widening of the vascular pedicle and all had clear posterior cardiac borders in the oblique views. They were regarded, therefore, as having cardiac enlargement from established valve lesions and not pericardial effusion. All five were observed over a period of one year or more and no changes were observed in heart size or shape. Two were catheterized and showed no evidence of effusion.
INCREASE IN HEART SIZE FROM CAUSES OTHER THAN EFFUSION

In the absence of pericarditis, the heart shadow increased in size during the period of observation in only 9 out of 215 consecutive cases with carditis. All of them had established valve lesions and active rheumatism, and one had signs of heart failure as well. In only one of the eight cases without failure was the rate of increase of size (5% in two weeks) in any way comparable to that which had been observed in effusion. In the case with failure, however, the rate of increase was more rapid, but could not be assessed accurately. In our experience, therefore, a rapid increase in heart size is due either to pericardial effusion or to congestive heart failure from active rheumatism and established valve lesions. We agree with Parkinson (1949) that it does not occur from dilatation alone in cases of early carditis. Keith and Brick (1942) reached conclusions similar to ours.

SPECIAL TECHNIQUES FOR THE DIAGNOSIS OF PERICARDIAL EFFUSION

Despite attention to points that have been emphasized in this paper, the differential diagnosis between pericardial effusion and cardiac enlargement is often difficult, particularly when the patient is first seen and before serial skiagrams are available. Various special techniques have been employed in view of this difficulty. The effect of position on the cardiac contour has already been discussed and is of little value. Fluoroscopy in our experience has failed to show any significant reduction of pulsation in the presence of an effusion. Pericardial paracentesis may, of course, be undertaken, but failure to obtain fluid does not exclude effusion owing to the frequency of early organization and loculation in some cases, and the mere demonstration of a few drops of fluid in

**Fig. 2.**—Tilt skiagrams taken before and after onset of effusion, showing development of divergent vascular shadows.
the pericardial sac does not necessarily indicate a significant effusion. Complete aspiration or partial removal of an effusion followed by air or lipodal replacement might provide more acceptable evidence, yet may well be thwarted by the loculations. As therapeutic aspiration is rarely necessary and the presence or early organization increases the hazards, diagnostic pericardial paracentesis has rarely been employed by us. Angiocardiography (Williams and Steinberg, 1949) would appear to be contra-indicated in acutely ill children.

Recently, cardiac catheterization has been employed in the diagnosis of pericardial effusion (Wood, 1950, 1951). We have used this technique in several of our cases and have found it to be of great value. Under visual control the catheter is passed into the right atrium and is then curled or looped so that the tip rests firmly against the lateral wall of the atrium. If there is then no opacity between the catheter tip and the lung field, no effusion can be present at this border. When a band of opacity separates the two, 7 to 10 ml. of diodone may be injected through the catheter, and skiagrams taken before and immediately after completing the injection: the dye then outlines the inner border of the lateral wall of the right atrium, and a pericardial effusion is seen as a shadow to the right of the catheter tip and the outlined atrial wall.

Fifteen cases of pericarditis have been investigated in this manner; two of them had undoubted large effusions, and were catheterized in order to confirm the value of the technique. Six of the remaining 13 had active rheumatism and cardiac enlargement, but it was not clear from routine skiagrams whether effusions were present or not. The catheter technique demonstrated a pericardial effusion as the main cause of the enlargement in all six (Fig. 3), and this was confirmed by

![Image](https://example.com/image.png)

**Fig. 3.—**Skiagrams showing increase of cardiothoracic ratio of uncertain ætiology. Cardiac catheterization with diodone injection, demonstrating pericardial effusion.
their subsequent clinical and radiological course. Two of these patients subsequently died, and in both there was evidence of pericarditis at autopsy. In five others, also with active rheumatism and cardiac enlargement, the presence of effusion was suspected on routine skiagrams but was thought to be unlikely. Catheterization demonstrated only small effusions in all of them, and subsequent serial skiagrams confirmed that true cardiac enlargement was in fact present. Two were catheterized after their effusions had apparently receded, and some residual effusion or pericardial thickening was demonstrated in both. Catheterization of these children proved to be perfectly safe and caused no distress provided adequate preliminary sedation had been employed.

We have also investigated two cases of mitral incompetence with aneurysmal dilatation of the left atrium. On fluoroscopy both showed systolic expansion of the atrium visible on the right and left borders of the heart in the postero-anterior view. Catheterization of these cases also showed a shadow to the right of the catheter tip. However, this shadow lies at a higher level than that of a pericardial effusion (Fig. 4) and may exhibit systolic expansion on fluoroscopy.

**Fig. 4.**—Contrast in catheter findings between (A) pericardial effusion, and (B) aneurysmal left atrium.

**SUMMARY AND CONCLUSION**

The radiological signs in 23 episodes of rheumatic pericardial effusion have been investigated. A sudden increase of cardiothoracic ratio and straightening of the left border were the most consistent and the earliest signs of developing effusion. Change of contour, bulging of the right border, widening of the vascular pedicle, disappearance of the shadow of the descending aorta,
and haziness of the posterior border in the oblique view were less consistent and usually later signs, but nevertheless useful ones. No constant change in the right cardio-diaphragmatic angle was observed.

A diminution in cardiothoracic ratio was the earliest radiological sign of resolution. Narrowing of the vascular pedicle, increased clarity of the posterior border in the oblique view, and return to normal of the right border followed later. Straightening of the left border and absence of the shadow of the descending aorta persisted longest, in some cases indefinitely.

The effect of posture on the shape of the heart shadow in pericardial effusion has been studied, particularly that of tilting head downwards to elicit the sign of divergent vascular shadows. This sign was found to be of limited value in the differentiation between effusion and cardiac enlargement.

Cases that had been diagnosed as having cardiac enlargement from established valve lesions were reviewed, and six of them were diagnosed in retrospect as having had effusions. Points that were of value in making this distinction are emphasized.

Cases with carditis have been reviewed to determine the incidence of rapidly progressive cardiac enlargement in the absence of pericarditis. It occurred in only 9 out of 215 cases; all of them had established valve lesions and active rheumatism, and one had failure. Only in the one with failure was the rate of increase comparable to that which had been observed in pericardial effusion. Acute dilatation does not, therefore, seem to occur in early rheumatic carditis.

Seventeen cases with and without effusion have been catheterized. The value of the technique in differentiating between pericardial effusion and cardiac enlargement is emphasized.

REFERENCES