POST-PARTUM HEART DISEASE

BY

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Post-partum heart disease has been described by several authors (Gouley et al., 1937; Hull et al., 1937, 1938; Lindeboom, 1950; Melvin, 1947) as a well-defined condition, exhibiting a uniform clinical picture, due to several possible etiological factors, many of which are often difficult to recognize. Notwithstanding these studies, the problem of heart failure occurring in the puerperal period in the absence of pre-existent cardiac disease is still far from being clear. The initial signs of decompensation usually appear during the last weeks of pregnancy and are often so slight as to be misinterpreted; they decrease immediately after delivery, often to reappear subsequently in a much more severe degree.

It is our opinion, based upon a series of 18 cases, most of which were followed throughout their entire clinical course, that this condition is not a specific or characteristic clinical syndrome, but is rather the result of multiple factors leading to cardiac failure. The presence of certain characteristics have enabled us to classify these cases into several groups.

Although the first cases of myocardial degeneration occurring during pregnancy and the puerperium were described in the last century (Porak, 1880; Virchow, 1870), and although a number were published in the earlier part of this century, emphasizing the importance of cardiac disease occurring in late pregnancy or in the puerperium (Blacker, 1907; Campbell, 1923), it was the work of Hull and his associates (1937, 1938) that suggested the existence of a well defined clinical entity and described its diagnostic features. These authors emphasized the favourable prognosis and the frequent occurrence of arterial hypertension, possibly related to a previous pregnancy or puerperium; they also found that embolism was common: two etiological factors mentioned were, pregnancy toxæmia and puerperal glomerulo-nephritis, both of which may cause a rapid elevation of the blood pressure. Another important contribution to this subject, published almost simultaneously, was the interesting paper by Gouley et al. (1937) describing 7 instances of advanced puerperal heart failure, with 4 deaths, mostly due to embolism from mural thrombi. The myocardial lesions differed considerably from the usual pathological findings in heart failure, showing great variation of distribution throughout the cardiac tissues. These authors thought that several factors were involved in such cases, and although the puerperal factors were mainly responsible, they emphasized the importance of pre-existing cardiac damage. Subsequently, a number of papers were published concerning the cardiac complications of pregnancy toxæmia (Benchimol et al., 1949; Dexter and Weiss, 1941; Reich, 1954; Szekely and Smith, 1947; Teel et al., 1937; Wallace et al., 1946), pointing out the clinical and pathological significance of these findings.

Some cases develop heart failure post partum (Benchimol et al., 1949; Teel et al., 1937), whereas others show nothing but electrocardiographic abnormalities suggesting myocardial damage, not necessarily related to the clinical syndrome (Benchimol et al., 1949; Dexter and Weiss, 1941; Wallace et al., 1946).

It has been implied (Szekely et al., 1947; Wallace et al., 1946), that these findings could explain
certain cases of post-partum heart failure of obscure aetiology, in the absence of obvious signs of toxæmia. The problem of acute glomerulo-nephritis was discussed in relation to this condition (Musser et al., 1938; Sodeman, 1940) and led to a classification of the post-partum circulatory syndromes into a nephritic and a non-nephritic group. The more recent publications on this subject refer either to isolated cases described as post-partum myocardosis (Woolford, 1952) or post-partal heart failure (Faerchtein et al., 1955) due to a non-specific myocardial degeneration, or to larger series of cases such as that of Melvin (1947), confirming the clinical syndrome described by Hull and his associates (1937; 1938).

It appears that the relationship of post-partum heart disease to arterial hypertension and to toxæmia of pregnancy cannot be precisely defined at the present time, and that the exact aetiology remains obscure in the majority of cases. More recently the existence of this condition has been seriously questioned by those who claim it is coincidental with the puerperal period (Bashour et al., 1954). We believe that the concept of post-partum heart disease is based upon a close relationship of the cardiac lesion to certain factors in late pregnancy and the puerperium, notwithstanding the variability of these factors and the difficulty of recognizing them in many cases.

We have attempted to classify post-partal heart disease in five groups according to the aetiological factors involved, as follows.

1. Cases that are undoubtedly related to toxæmia of pregnancy.
2. Cases that are probably related to toxæmia of pregnancy.
3. Cases that are due to non-specific myocarditis.
4. Cases with pre-existing hypertensive heart disease (due to essential hypertension, chronic pyelonephritis, glomerulo-nephritis, etc.).
5. Specific myocarditis, difficult to identify from the clinical standpoint, and usually requiring pathological data for a correct diagnosis.

The above classification represents an attempt to harmonize the divergent opinions that have raised much discussion among those who have studied this problem. We realize that it is far from satisfactory: at the present time, however, it seems to be useful from a practical standpoint. We have excluded all cases with previous heart disease excepting those due to hypertension, which constitute group 4. This was thought advisable, because arterial hypertension is one of the important predisposing factors in pregnancy toxæmia, which plays an important role in post-partum heart disease. On the other hand, it is often difficult to ascertain post partum, whether or not the elevated blood pressure is related to pre-existing hypertension. Thus, we prefer to consider all hypertensive cases that decompensate after delivery as post-partum heart disease, realizing however that we may be including some cases of chronic hypertensive heart disease, who develop cardiac failure coincidentally during the puerperal period.

PUERPERAL FACTORS THAT MAY CONTRIBUTE TO POST-PARTUM HEART FAILURE

In addition to the well-known hæmodynamic changes that occur during pregnancy and persist until shortly after delivery, other factors inherent in the puerperal period may also play an important role. It is known that the early puerperium is a critical period for the cardiac patient; there is a high incidence of heart failure and of cardiac deaths at this time as pointed out by Mackenzie (1921), and later confirmed by other authors (Hamilton et al., 1941; Hoffman et al., 1942). Recent studies show that the circulatory strain due to the hæmodynamic changes of pregnancy is maintained for several weeks after delivery, and although usually slight, the presence of hypervolemia, for instance, has been demonstrated in the second week of the puerperium.

On the other hand, labour itself may precipitate circulatory failure (Mendelson et al., 1942; Pardee et al., 1941; Sampson et al., 1945) which may occur after delivery (Gorenberg et al., 1941; Mendelson, 1944), possibly due to the sudden closure of a large arterio-venous shunt caused by the placental circulation (Burwell et al., 1937; 1938). It thus appears that the circulatory abnormalities of pregnancy do not disappear immediately after delivery, and that the maternal organism is sub-
jected to considerable stress as a result of the sudden emptying of the uterus, the elimination of the placenta, and the abrupt removal of the fetal circulation.

Brown et al. (1947), in a study of the circulatory abnormalities that appear during and after labour, observed that the changes in heart rate, blood pressure, vital capacity, and circulation time were not sufficiently conclusive to be attributed solely to pregnancy or to the puerperium. With reference to the venous pressure changes, several investigations have been made in pregnant patients (Burwell et al., 1937; Dellepiane, 1927; Luisi, 1938; McLennan, 1943), and revealed variable degrees of venous hypertension in the first 24 hours after delivery. This may be the result of several combined factors such as the effect of drugs, increase of venous return to the heart due to muscular effort, and increase of peripheral vascular resistance following closure of the circulatory shunt. These factors could conceivably precipitate heart failure in patients with a limited functional capacity. Hypervolemia with secondary hæmodilution observed during the first week after delivery (Albers, 1939; Brown et al., 1947; Crawford, 1940), probably represent contributory factors in heart failure that develops in patients with or without previous cardiac disease.

In addition to the influence of the above-mentioned haemodynamic changes, a number of other puerperal conditions may act as precipitating factors of post-partum heart failure, such as obstetrical hemorrhages, puerperal infections, thrombo-embolic phenomena, shock, and other minor conditions such as anæmia, hypoproteinaemia, and vitamin deficiencies. Under special circumstances some of these factors may be outstanding and may play the major role in cardiac decompensation; but this is rarely observed, and when it does occur is easily recognized as such. We believe therefore that they do not merit a special group in our classification. One of our cases was interesting in this respect, since the presence of severe nutritional megaloblastic anæmia associated with hypoproteinaemia were the main factors responsible for post-partum heart failure.

**Post-partum Heart Failure Due to Toxaemia of Pregnancy**

Eight patients of our series had pregnancy toxaemia and we believe that it was the main cause of cardiac involvement. In some cases, the clinical signs of heart disease appeared prior to delivery, but even so, there was aggravation either of the heart failure or of the electrocardiographic changes or of both, during the puerperal period. Although there are very few studies concerning the incidence of myocardial changes in toxaemia of pregnancy, it is apparent that cardiac damage occurs under these circumstances because advanced cases show orthopæna, cardiac asthma, and acute pulmonary œdema (Moore et al., 1927; Reid et al., 1939; Szekely et al., 1947; Teel et al., 1937).

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**Fig. 1.**—Teleradiograms of the heart of a 29-year-old primipara. The initial X-ray taken two days after delivery shows much enlargement with pulmonary congestion and bilateral pleural effusion. Twenty days later the heart shadow is considerably decreased and the lung fields are normal. Five years later the heart size is normal. In 1950 and 1951 she had two normal pregnancies.
FIG. 2.—From the same case as Fig. 1. Serial radiograms (leads 1, 2, 3, V4, 5, and 6), showing low voltage, primary T wave changes, and left ventricular enlargement. The T waves become progressively more inverted. Subsequently the tracing becomes normal. The period of the greatest electrocardiographic changes did not coincide with the most serious clinical phase.

Serial electrocardiographic (Benchimol et al., 1949; Szekely et al., 1947; Wallace et al., 1946), and post-mortem studies (Gouley et al., 1937; Hull et al., 1937; Teel et al., 1937) during pregnancy and the puerperal period in toxaemia reveal the importance of myocardial involvement, which occurs frequently and often results in advanced cardiac insufficiency. Among the eight patients of this group, there were two who showed that toxaemia of pregnancy may be responsible for severe puerperal heart failure with radiological and electrocardiographic abnormalities. Although we did not study these patients during the latter part of pregnancy; (they were in hospital in the puerperium), toxaemia of pregnancy was recognized by the presence of œdema and headache during the last weeks of pregnancy. The diagnosis was confirmed by the presence of severe angiospastic retinopathy and hypertension on admission, with the exception of one case with a normal blood pressure (100/80) throughout the second half of pregnancy. The fall of blood pressure to normal and the complete disappearance of the ophthalmoscopic abnormalities, confirmed the diagnosis of toxaemia, and excluded pre-existing arterial hypertension. In one of these patients the signs of heart failure were present in the last weeks of pregnancy and improved considerably following delivery, but reappeared a fortnight later in a more severe form. In the other case, however, the circulatory changes were already present on the sixth day of the puerperium and developed rapidly, culminating in severe dyspnoea and orthopnoea. The chest radiograms (Fig. 1) showed a great increase in heart size, with hilar and peripheral congestion in addition to bilateral pleural effusion. Subsequently, there was a rapid reduction in heart size and improvement in pulmonary congestion.

The electrocardiographic abnormalities (Fig. 2 and 3) were significant, particularly in this case (Fig. 2) in which there was a suggestion of anterior wall infarction in the chest leads, including high precordial levels,
as was previously described by Wallace et al. (1946). The usual electrocardiographic changes observed were flattened T waves, which occasionally became inverted in leads I, II, and aVL, and over the left precordium. The progressive changes in the serial tracings returned to normal after several weeks, although in a few cases the cardiographic abnormalities increased in the course of clinical improvement. Thus, in one case (Fig. 2) the T wave changes were still present on June 24, 1948, notwithstanding the patient's excellent cardiac condition at that time; this was also observed in another patient who showed maximal electrocardiographic abnormalities at the time of clinical improvement.

![Electrocardiogram of a 40-year-old negress. Following clinical improvement, the T waves become inverted. Four years later the tracing is almost normal.](image)

The course and prognosis of these cases were favourable in view of the excellent clinical response following a two to four week period, after which there was no further evidence of heart failure. One of these patients had two subsequent pregnancies without complication, and when she was last seen six years later, there were no cardiovascular abnormalities, from the clinical, cardiographic, or roentgenologic standpoints. In the differential diagnosis of post-partum heart disease, the following conditions should be considered: acute glomerulo-nephritis, myocardial infarction, and the
non-specific forms of myocarditis. The following points are important in differential diagnosis of glomerulonephritis and post-partum heart disease resulting from toxæmia.

Glomerulo-nephritis is extremely rare in the course of pregnancy (Tillman, 1951), and usually appears in the early stages, almost invariably before the 24th week; this is considered by many authors as a very important distinguishing feature from toxæmia of pregnancy. Other points are the absence of significant hematuria and nitrogen retention in toxæmia of pregnancy: the extremely unfavourable course of glomerulo-nephritis in the course of pregnancy: and the favourable prognosis observed in all our cases, none of which followed a chronic course, whereas this is the rule in cases of glomerulo-nephritis.

With respect to myocardial infarction, the electrocardiographic abnormalities were often suggestive of this condition; however, the diagnosis of coronary disease was ruled out in view of the patient's age, the absence of pain, the rapid and complete return to normal of the cardogram, as well as the absence of blood pressure changes and other characteristic signs of acute coronary occlusion. The distinguishing features from non-specific myocarditis are the following: absence of a prolonged infection, absence of muffled heart sounds or abnormal rhythms, and the presence of hypertension and other evidence of toxæmia of pregnancy.

Our remaining six cases of post-partum heart disease due to toxæmia of pregnancy did not completely conform with the clinical picture we have described. Notwithstanding the absence of several typical features such as heart failure after delivery, most cases showed other signs such as electrocardiographic changes, with associated fundoscopic changes, all of which, including arterial hypertension, disappeared completely in a relatively short period of time. The influence of other associated ætiological factors, such as hæmorrhage and obstetrical shock, nutritional deficiencies, pulmonary embolism, etc., as a cause of cardiac involvement, was demonstrated in two cases, both of which developed heart failure after delivery.

**Post-partum Heart Failure Possibly Related to Toxæmia of Pregnancy**

Toxæmia of pregnancy was considered the probable cause of cardiac disease in three cases that closely resembled the type of post-partum heart disease generally described in other

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*Fig. 4.—Radiograms of a 34-year-old negress who gave birth to a premature stillborn infant, and one month later developed congestive heart failure. The blood pressure varied from 170/120 to 120/90. After several pulmonary infarcts, death occurred seven months later. Radiograms show cardiac enlargement with right auricular dilatation; pulmonary infarction is seen in the second X-ray.*
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reports (Gouley et al., 1937; Hull et al., 1937, 1938; Melvin, 1947; Musser et al., 1938). Heart failure usually develops or is at least aggravated in the late puerperium (2 to 6 weeks after delivery). The clinical course is prolonged in most instances: embolic phenomena often occur, as well as a moderate and irregular elevation of the blood pressure. Response to routine treatment for heart failure is slow although eventually it becomes effective. The electrocardiographic changes

Fig. 5.—Heart from the negress referred to in Fig. 4. Above, the left ventricle with an organized thrombus adherent to the endocardium and with a local thinning of the heart at this point. The lower figure shows much of fibrosis in the interventricular septum.
are non-progressive and do not disappear rapidly. They are usually represented by T wave and S-T segment abnormalities, as well as signs of left ventricular hypertrophy. Cardiac enlargement is always present, and often great (Fig. 4). A unilateral elevation of the diaphragm associated with pleural effusion suggests the occurrence of pulmonary infarction. Repeated sub-pleural pulmonary infarcts may lead to extensive fibrothorax. There were two fatal cases. One came to necropsy and revealed an organized mural thrombus adherent to the apical wall of the left ventricle, in addition to extensive myocardial fibrosis (Fig. 5): the coronary arteries were normal and patent throughout, and the thoracic aorta showed mild atherosclerosis. In our opinion toxæmia of pregnancy probably played an important role in this group of patients, all of whom had suggestive symptoms in the last months of pregnancy, and two of whom had in addition arterial hypertension.

The fact that we have observed cases of toxæmia, in which the electrocardiographic and ophthalmoscopic changes become more severe after delivery, affords evidence as to the possible persistence of toxæmic factors after delivery (Hull et al., 1937; Szekely et al., 1947). We believe however, that the hypertensive toxæmias of pregnancy are not solely responsible for these cases of post-partum heart disease, although other ætiological factors are not readily recognized. The following factors were ruled out in these patients: pre-existent arterial hypertension, vitamin deficiencies, and non-specific myocarditis.

**Post-partum Heart Disease Due to Non-specific Myocarditis**

Two cases of our series exhibited fever, a rapid sedimentation rate, and leukocytosis, in the absence of any suggestive signs of toxæmia of pregnancy. In these patients with advanced congestive heart failure, progressive electrocardiographic abnormalities, and reversible radiological changes, the diagnosis of non-specific myocarditis of unknown ætiology was suggested, associated with other factors of minor clinical importance such as hemorrhage and nutritional deficiency. Cardiac enlargement, bilateral pleural effusion, and pulmonary congestion were present but reverted to normal after several weeks (Fig. 6). The electrocardiogram revealed primary T wave changes, which subsequently became normal in configuration (Fig. 7). In both cases the clinical course was favourable, and at the end of a few weeks no cardiovascular abnormalities could be demonstrated. In these two patients, thiamin deficiency was suggested among the possible causes of heart disease, in

![Image](http://heart.bmj.com/)

**Fig. 6.**—From a 33-year-old negress. Twelve days after delivery signs of left ventricular failure developed. The cardiac condition deteriorated and signs of congestive failure, without hypertension, developed and responded to digitalis. The initial roentgenogram shows enlargement of the heart, pulmonary congestion, and bilateral pleural effusion. Six weeks later there was considerable improvement in X-ray signs.
view of a history of chronic alcoholism, and the presence of polyneuritis in one case, and a nutritional deficiency in the other. However, this diagnosis was subsequently ruled out, at least as the sole causal factor, since heart failure did not occur during pregnancy, when the thiamine requirements are increased: there was no therapeutic effect of vitamin B1, but there was an excellent response to digitalis. We believe that this group of cases corresponds to non-specific myocarditis, frequently observed in the course of several types of infection, and often associated with thiamine deficiency. From the practical point of view, these two cases demonstrate the possibility of apparently unimportant infections causing myocardial lesions that lead to severe heart failure in the puerperal period, with obvious therapeutic and prophylactic implications.

**Post-partum Heart Failure in Patients with Hypertensive Heart Disease.**

In several reports on the clinical course of essential hypertension, glomerulo-nephritis, and pyelonephritis during pregnancy (Dexter et al., 1941; Dieckman et al., 1952; Fishberg, 1954; Page et al., 1938; Tillman, 1951) it has been suggested that these conditions are often responsible for cardiac
decompensation occurring after delivery. It is apparent that patients with hypertensive heart disease may develop heart failure during the puerperal period, due to haemodynamic changes that overload the heart and other unknown precipitating factors. Glomerulo-nephritis is a rare occurrence in these patients, since it usually leads to interruption of pregnancy or renal failure. However, when glomerulonephritis occurs in the early puerperium, it may be a causal factor of post-partum heart failure (Musser et al., 1938).

The importance of pyelonephritis is limited to chronic cases, since the acute forms are usually benign and rarely influence the course of pregnancy. Chronic cases, which frequently cause hypertensive disease during pregnancy, are probably responsible for the so-called "nephritis toxæmias" formerly attributed to chronic glomerulonephritis (Fishberg, 1954). It is important to know that arterial hypertension and renal insufficiency are usually late manifestations of this disease, and once it reaches this stage, it is practically irreversible although the urinary infection may be temporarily controlled. The disease may thus be prolonged for many years, with periods of remission and exacerbation. Cardiac failure may supervene, not only from long-standing arterial hypertension, but also from acute exacerbations of the renal condition, aggravating the hypertension.

The late stages of pregnancy undoubtedly predispose to stasis and infection of the urinary tract, tending to aggravate a chronic pyelonephritis which often culminates in heart failure. Although this may occur at the end of pregnancy, the full picture often appears only after delivery.

In our series of post-partum heart failure, there are two instances of chronic pyelonephritis with hypertension and heart failure occurring in the puerperal period. It is our impression that this renal condition is actually the most important cause of hypertensive heart disease in pregnancy, since glomerulo-nephritis usually leads to renal failure, and essential hypertension is well tolerated during pregnancy only in its milder forms. In the more severe cases, pregnancy seldom follows its normal course, since there is usually an aggravation of the pre-existent hypertension, and when heart failure occurs, it usually appears during pregnancy, as we have often observed (Benchimol and Carneiro, 1949). We have never seen cases of pre-existing hypertension, either of the essential type or due to glomerulonephritis, that have developed heart failure after delivery.

**Post-partum Heart Failure Due to Specific Myocarditis**

Specific types of myocarditis should be included in this classification as one of the possible causes of post-partum heart disease, since it is usually difficult to determine the exact nature of the cardiac condition before death, and also because the clinical picture often resembles the non-specific myocarditis related to the puerperal infections.

We have had the opportunity of studying two cases of specific myocarditis, leading to post-partum heart failure, both of which were confirmed at necropsy. One patient, correctly diagnosed as a chronic myocarditis due to Chagas' disease, decompensated during pregnancy but continued in heart failure until her death which occurred two months after delivery: this case was previously published (Benchimol et al., 1954), and was not included in our present observations. The cause of our second case was difficult to recognize, since the clinical picture, the electrocardiogram, and the radological aspects closely resembled those of the non-specific myocarditis due to puerperal infections. However, the nature of the myocardial lesion was determined at necropsy by the demonstration of a rare type of miliary tuberculous myocarditis (Fig. 8). It is obvious that once the specific nature of the myocarditis is established, it should not be included in the classification of post-partum heart disease, since under these circumstances, heart failure results primarily from the myocardial condition itself, and is not due to any factor related to the puerperium.

We believe therefore, that this group of cases should be included in our classification only when the precise etiology of the myocarditis is difficult to ascertain. We have described this group of patients, merely to emphasize certain practical aspects of the problem concerned with etiology and differential diagnosis.
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Fig. 8.—Myocardium of a 36-year-old negress, showing a central area of calcification and an intense infiltration of epithelioid and lymphocytic cells, characterizing tuberculous involvement of the myocardium.

SUMMARY AND CONCLUSIONS

Post-partum heart disease is not a well-defined condition with a uniform clinical picture. Evidence from the present series suggests that a number of aetiological factors may play a role, some of them probably related to pregnancy, whereas others are more directly concerned with the puerperium. The following classification of post-partal heart disease is proposed.

I. Cases undoubtedly due to toxæmia of pregnancy.
II. Cases probably related to toxæmia of pregnancy.
III. Cases related to non-specific myocarditis.
IV. Cases with pre-existing hypertensive heart disease (essential hypertension, chronic pyelonephritis, glomerulonephritis, etc.
V. Cases corresponding to specific myocarditis, difficult to identify, and often requiring pathological confirmation.

In addition to the influence of the hæmodynamic changes observed during pregnancy and the puerperium, several factors occurring after delivery may act as associated or precipitating causes of heart failure. Among these, the most important are anæmia aggravated by obstetrical hæmorrhage, puerperal infection, thrombo-embolic phenomena, shock, and other less important conditions.

Toxæmia of pregnancy was present in 8 cases of this series, and was believed to be the main cause of cardiac involvement. In some cases heart disease appeared before delivery, but it was always aggravated during the puerperium. The course and prognosis were most favourable in these cases, for after 2 to 4 weeks, all the manifestations of heart failure had disappeared as well as other evidence of cardiovascular disease. In two cases, other aetiological factors could be demonstrated.

Toxæmia of pregnancy was considered as the probable aetiological factor of cardiac involvement in 3 cases which resembled the type of post-partal heart disease previously described, where the initial manifestations occur late in the puerperium, the clinical course is prolonged and usually complicated by embolic phenomena, in addition to a moderate and irregular elevation of the blood pressure. The electrocardiographic changes are non-progressive, and do not tend to
disappear rapidly, and cardiac enlargement is always demonstrable radiologically. Two of these cases came to necropsy, and one of them showed myocardial fibrosis and mural thrombosis.

Two patients had advanced congestive heart failure occurring during the puerperium, and subsequently improved. A diagnosis of non-specific myocarditis of unknown etiology was made.

Hypertensive cardiovascular disease occasionally shows deterioration and heart failure, not only during pregnancy but also in the puerperal period, leading to post-partum heart disease complicated or precipitated by other additional aetiological factors. In this series, there were two cases of chronic pyelonephritis with hypertension and heart failure after parturition.

Specific myocarditis should be included among the determining factors of post-partum heart disease, in view of the fact that the cause is often difficult to recognize in life, and such cases closely resemble the non-specific myocarditis related to the puerperal infections. Pathological investigation may demonstrate the aetiology and the specific nature of the condition as in one case of tuberculous myocarditis, which was unrecognized clinically but was diagnosed at necropsy. It is obvious that once the specific nature of the myocardial condition is established, it should not be regarded as a puerperal cardiopathy.

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