Calcification of the Pericardium in Apparently Healthy People
Electrocardiographic Abnormalities Found in Tracings from Apparently Healthy Persons with Calcification of the Pericardium

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Case histories are presented of five apparently healthy persons showing pericardial calcification. Calcium deposits form in the dense pericardial scarring which is responsible for changes in the electrocardiogram. Awareness by the cardiologist of the possibility of pericardial scarring makes him particularly alert to electrocardiograms showing normal QRS complexes with T wave changes, particularly if the T waves point in a direction opposite to the main QRS deflection and are associated with RS-T segment depression. Low QRS voltage may or may not be present. Suspicion will be increased in serial records when the abnormalities remain constant over a period of years.

Calcification of the pericardium is an uncommon condition of uncertain etiology. Calcium deposits do not form in the healthy pericardium but in the dense fibrosis and scarring which may follow pericardial inflammation. Tuberculosis, rheumatic fever, pyogenic infections, and trauma have been named as causative agents, but in many instances the exact nature of the primary lesion remains unknown. The deposition of calcium may continue for many years; long after the active inflammatory process has disappeared. Its presence constitutes strong evidence of diffuse scarring of the pericardium. Hence it is observed in patients presenting the clinical picture of chronic constrictive pericarditis. Harrison, Paul, and Evans reported it in 43, 55 and 70 per cent of their respective series. Pericardial scarring, with or without deposition of calcium salts, is known also to occur in persons showing no evidence of heart disease. Jones reported absence of cardiac symptoms in 9 out of 38 cases of calcified pericardium, while Armstrong at the London Hospital reported no symptoms in 31 of 114 cases showing a completely adherent pericardium at autopsy. That pericardial calcification may occur in apparently healthy individuals has been reported by various observers. It is important then to recognize that pericardial scarring may be present in persons who do or do not show clinical evidence of heart disease and who on radiological examination may or may not exhibit pericardial calcification.

The detection of pericardial scarring in asymptomatic persons rests upon the demonstration by x-ray examination of calcium deposits in the pericardium or the appearance of suggestive changes in the electrocardiogram. It is the purpose of this paper to present the case histories of five apparently healthy individuals who were found to have pericardial calcification and to discuss certain electrocardiographic findings which are thought to be of diagnostic importance.

Case Reports

Case I. On March 14, 1952 a business executive, age 52, underwent a routine medical examination. He was refused life insurance in 1920 supposedly because of a heart murmur and high blood pressure. In 1950 a gangrenous appendix was removed. There
were no other reported illnesses. His mother died of heart disease at age 52.

The patient's height was 5 feet 7 1/2 inches; weight, 203 pounds, and blood pressure, 150/86 to 144/80. General examination was otherwise normal. An electrocardiogram was recorded on March 24, 1952 and an x-ray film of the chest on April 7, 1952.

In the electrocardiogram (fig. 1) the RS-T segments in leads I and II are depressed and the T waves in lead I are inverted. In precordial leads V₅ and V₆ the RS-T segments are slightly depressed and the T waves are low or inverted. This record was considered to be abnormal but no clinical connotation was suggested. The heart was not enlarged by x-ray examination (fig. 1) and there was no visible calcification.

This man died from accidental causes on April 24, 1952. A postmortem examination carried out by Dr. D. W. Penner revealed a thickened calcified pericardium. Extracts from the autopsy report follow:

Gross findings. Both lungs are adherent to the parietal pleura by old firm adhesions and their roots are firmly adherent to the pericardial sac. The lungs themselves show no gross changes, other than marked congestion and some mucus accumulation in the bronchi.

The pericardial sac is thickened and firmly adherent to the visceral pericardium from which it cannot be separated. The heart (with pericardium) weighs 770 Gm. The valve rings measure: mitral 10 cm., aortic 6 cm., tricuspid 11 cm., and pulmonary 7 cm. The entire parietal pericardium is rather firmly attached to the visceral pericardium, although in areas it can be separated with blunt forceful dissection. The valves are all grossly normal. The wall of the right ventricle measures 5 mm. and the left up to a maximum of 18 mm. The coronary arteries appear relatively small. They are patent throughout and although there is minimal diffuse atheroma present there is no appreciable narrowing of the lumen at any site. Sections of the myocardium show no gross evidence of fibrosis. Located in the pericardium are four separate calcareous masses (fig. 2); the largest, measuring 3.5 by 2 by 1.3 cm. lies along the right lateral border of the right auricle. The inferior margin of it overlies the coronary artery which passes deep to it at a point 5 cm. from its origin. The vessel is not attached to the mass. Two similar, but smaller, calcareous masses overlie the course of the left coronary artery in the auricular-ventricular groove on the posterior surface of the heart, just at its left lateral border. These two masses adjacent to one another measure 1.5 cm. and 1 cm. and occur at a point 4 cm. from the origin of the left coronary artery. A fourth small calcareous mass, 3 mm. in diameter, is present at the apex. The gall bladder is normal in size. The lumen contains yellow bile, two mixed 1.2 cm. calculi, together with numerous tiny calculi and biliary mud. The bile ducts are not remarkable.

Microscopic Findings. Myocardium: Old fibrous
adhesive pericarditis is present with areas of calcification and ossification. There is almost no cellular infiltration present, i.e., no evidence of active inflammation. The myocardium shows patchy areas of minimal interstitial fibrosis (fig. 3). The liver shows early portal cirrhosis and some fatty changes.

Summary. Old adhesive pericarditis with calcification and ossification. Early portal cirrhosis.

Case 2. A 32 year old pilot was examined on Jan. 18, 1945 prior to his release from the Royal Canadian Air Force. A routine electrocardiogram at that time was found to be grossly abnormal. This tracing was identical with that of Sept. 20, 1945 which appears in figure 4. Here the RS-T segments are depressed and the T waves are inverted in leads I, II, III and CF4.

Between 1945 and 1954 this man was examined repeatedly because of the persistently abnormal electrocardiogram. At each examination he was found to be in good health and at no time had he symptoms of heart disease. There was no history of rheumatic or scarlet fever. The father, mother and family physician who looked after him from birth could not recall his having had any serious illness. He remained well in the Air Force. Since the war he has been continuously employed by a logging company as woods superintendent.

The family history was normal except that a younger brother had pleurisy during the war and was later treated for pulmonary tuberculosis. He had no contact with this brother. He was married in 1950 and has two children.

On examination he was 70⅔ inches tall and his weight increased from 149 pounds in 1942 to 175 pounds in 1954. Blood pressure was always within the range of normal (e.g., 122/74). The heart was normal in size and rhythm. A soft apical systolic murmur was heard once in 1945. There were no other noteworthy findings.

The consultant who examined him in 1945 summarized his opinion as follows: "I am unable to explain the unfavorable electrocardiograms. There are no symptoms and no physical signs to suggest heart disease, and there is no history of rheumatic fever, pericarditis, or trauma that could help explain the inverted T waves."

Serial electrocardiograms over the years have been identically abnormal, except for some increase in QRS voltage (fig. 4). Recently this man's heart was examined radiographically specifically for signs of calcification of the pericardium. The films taken on Jan. 12, 1954 are reproduced in figure 5. Extensive calcification of the pericardium is present at the apex of the heart and is seen to extend over its lower anterior surface.

The Department of Veterans Affairs kindly made available the 1942 and 1945 x-ray films taken on entering and leaving the Royal Canadian Air Force. In retrospect calcification can be seen in the 1945 film and with less certainty in the 1942 film.

Case 3. A 49 year old baker underwent a routine medical examination on Dec. 21, 1953. He stated that he was in good health. His father was still living at 85 and his mother died at the age 82. Apart from a history of pleurisy many years ago, there were no other known illnesses. He had received periodic medical examinations for the past three years. These examinations included photoradiographs of the chest. The height was 5 feet 5 inches, weight 191 pounds, blood pressure 122/88. Apart from mild varicosities of the right leg, examination was clear.

An electrocardiogram and a single x-ray film of the chest were taken on Feb. 1, 1954 (fig. 6). The electrocardiogram shows left axis deviation with depressed RS-T segments in leads I and II. The T waves are acutely inverted in lead I and low in lead II. The RS-T segments in V4 to V6 are depressed and the T waves in V2 to V6 are inverted. The x-ray film of the chest clearly shows calcification of the pericardium along the left border of the heart. The right costophrenic angle is obliterated by old adhesions.

Case 4. A 50 year old woman sought medical advice because of palpitation, nausea and a sense of heaviness in the abdomen. On June 7, 1954 during fluoroscopy in connection with an x-ray examination of the gastrointestinal tract, the radiologist observed calcification of the pericardium. She was first seen in January 1949 with hot flushes, mild mental depression and vague joint pains. Examination did not reveal any abnormality and her symptoms were believed due to menopause. Because her basal

Fig. 3. Case 1. Section showing densely fibrosed pericardium with calcium plaque. The relatively normal myocardium appears below.
metabolic rate was minus 28 per cent, desiccated thyroid gland was prescribed. In September 1950 she consulted her physician because of spells of weakness and perspiration. These responded to symptomatic treatment. Again in June 1953 she complained of palpitation intermittently for a period of two weeks. Nothing was found on examination and she improved with mild sedation. In March and April 1954 there were two further episodes of palpitation. These attacks lasted three hours, one following an emotional upset. The character of the attacks did not suggest paroxysmal tachycardia. There was no history of rheumatic or scarlet fever. On close questioning she admitted noticing dyspnea on exertion for the preceding year. She experienced no distress following her usual sedentary occupation of commercial artist.

The family history was not significant, with the possible exception of a maternal grandmother dying of tuberculosis and the coincidence of two brothers having stones removed from the bladder. The height was 5 feet 10 inches; weight 165 pounds; blood pressure 110/80. A soft pericardial rub, systolic in time was audible in the third intercostal space to the left of the sternum. Apart from some tenderness in the right lower quadrant of the abdomen, examination was normal. The venous pressure in the right antecubital vein was 120 mm. water.

The electrocardiogram (fig. 7) shows a broad P wave in lead II; the T waves are flat in lead II and inverted in lead III. The RS-T segment is depressed in V6 and the T wave here is flat. The x-ray films of the heart show in the anterior view calcification of the pericardium along the left border and at the apex. It can be seen to extend well up behind the heart in the lateral film (fig. 7).

**Case 5.** A 64 year old baker superintendent had an x-ray examination of the gastro-intestinal tract on May 31, 1954, and during fluoroscopy, the radiologist observed calcification in the region of the heart.
Recently he had experienced a "lump" high in the epigastrium for which he consulted his physician. This sensation was always present toward the end of the working day but not felt in the evening when working in his garden. He admitted during the past winter having shortness of breath on occasion but it did not interfere with his work which at times was quite heavy.

He was born in Belgium and came to Canada at the age of 39 years. Apart from influenza in 1918 he experienced no serious illnesses. The family history was negative.

The height was 5 feet 6 inches; the weight 141 pounds; the blood pressure 122/74. The veins of the forearms were prominent but there was no cervical engorgement. A soft cardiorespiratory murmur was heard at the apex. Examination was otherwise normal. Venous pressure measured in the right antecubital vein was 180 mm. H2O.

The electrocardiogram (fig. 8) is normal except for
There is a broad P wave in lead II of the electrocardiogram. The T waves are flat in lead II and inverted in lead III. The T waves in lead V₄, V₅, and V₆ tend to be low. Calcification of the pericardium is evident in the lateral film of the heart.

Apart from wandering of the pace-maker, the electrocardiogram is not remarkable. Diffuse pericardial calcification is present in the posteroanterior film of the heart.

Observations
Calcification of the pericardium in five apparently healthy persons was demonstrated by x-ray examination in four instances and by autopsy in one. In three of the five cases calcification was not identified in routine chest films on at least one occasion. In each of these cases the electrocardiogram was grossly abnormal.

In the one case which came to autopsy, the coronary arteries were not significantly narrowed and the myocardium was essentially normal except for superficial scarring.

In one case x-ray evidence indicated that the degree of calcification increased over a 12-year period.

The electrocardiograms varied widely, ranging from normal on the one hand to gross changes in form on the other. There were no major disturbances in rhythm and the P waves were normal in form with one exception. Auriculoventricular and intraventricular conduction were within normal limits. There were no changes in the form of the QRS complexes nor did any tracing exhibit low voltage, in fact voltage was of increased magnitude in one instance. The electrical position of the hearts varied from vertical to horizontal.
In three of the five records there was a definite depression of the RS-T segments. This occurred in lead II and in the left precordial leads in each instance but additional leads were involved in some records. Flat or inverted T waves in the limb leads and in a variable number of the precordial leads occurred in four of the five records. The RS-T segment depression and the T wave inversion occurred in the opposite direction to the main QRS deflection. Case 2 provided the only opportunity for a long term follow-up and here the observed abnormalities were constant.

DISCUSSION

Notching of the P waves and auricular fibrillation are reported to be common occurrences in chronic pericarditis but neither was observed in the present series. Possibly such changes occur only when there is pronounced scarring over the auricles involving specifically the region of the sinoauricular node. This study suggests that these changes may be uncommon in asymptomatic persons.

The abnormalities of the ventricular complex characteristic of pericarditis are restricted to the RS-T segments and the T waves—the phase of repolarization. The view is commonly held that these changes are the result of a disturbance in the subepicardial layer of the myocardium. During the acute inflammatory stage of pericarditis the RS-T segments are elevated and the T waves flat or inverted in one or more of the limb and precordial leads. With resolution, these changes tend to disappear. In chronic pericarditis the T waves are also flat or inverted but here the RS-T segments may be depressed. These changes occur in the opposite direction to the main QRS deflection. A residual adherent pericarditis with scarring of the outer surface of the myocardium may effectively delay the initiation of repolarization at this site, causing it to take place in a reverse direction, from the endocardium outward. If this is so it explains the observed changes and the fact that they tend to be irreversible.

RS-T segment depression has been recognized but not emphasized as part of the characteristic electrocardiographic picture of chronic pericarditis. Numerous examples of it are to be seen in the electrocardiograms of reported cases.

Not infrequently reference is made to the occurrence of low voltage of the QRS complexes in pericarditis. This reduction in voltage has been credited to the short circuiting effect of pericardial effusion or to the relatively poor conducting properties of thickened pericardial tissues and not to a decrease in the potentials of the underlying heart muscle which in many instances is singularly free from pathological change. In case 2 of the present series an increase in the QRS voltage occurred between 1945 and 1953. Here it is suggested that the heart, due to the contraction of adhesions between the pericardium and the anterior chest wall, was gradually tilted forward so that its anterior surface was brought progressively closer to the exploring electrode, resulting in voltages of increasing magnitude. The proximity of the anterior surface of the heart to the chest wall can be seen in the lateral film recorded Jan. 12, 1954 (fig. 5). It would appear that voltages of normal value as well as those which exceed these limits in either direction may be recorded in chronic pericarditis.

Subjects whose electrocardiograms show the RS-T segment and/or T wave changes which have been referred to should be subjected to a detailed x-ray search for calcification of the pericardium when these electrocardiographic alterations cannot be explained otherwise. It is to be understood, however, that failure to find calcification does not conclusively rule out the presence of pericardial scarring. The converse is equally true that a normal electrocardiogram may occur in the presence of recognizable pericardial calcification. Case 5 in the present series is an example and a similar case was reported by Forsee.

CONCLUSIONS

The wide use of the "routine electrocardiogram" will include the examination of persons with pericardial scarring who have no symptoms of heart disease. Awareness by the cardiologist of the possibility of chronic pericardial scarring in persons so examined will make him particularly alert to tracings
showing normal QRS complexes with T-wave changes, particularly if the T waves point in a direction opposite to the main QRS deflection and are associated with RS-T segment depression. Low QRS voltage may or may not be present. The degree of suspicion will be increased if in serial records the abnormalities remain constant over a period of years.

**SUMMARY IN INTERLINGUA**

Es presentate le casos de 5 apparentemente normal individuos con evidentia de calcification pericardial. Depositos de calcium se forma in le dense cicatrisation pericardial que es responsabile pro alterationes electrocardiographic. Le cardiologo qui es conscie del possibilitate de cicatrisation pericardial va esser specialmente alerte quando ille incontra electrocardiogrammas con normal complexos QRS e alterate undas T, particularmente si le undas T ha un direction oppone al principal deflexion QRS e es associate con un depression del segmentos RS-T. Un basse voltage QRS pote sed non debe esser presente. Le suspicion del cardiologo va augmentar se si registrationes serial revela que le anormalitates remane constante durante un periodo de plure annos.

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