False "Coronary Patterns" in the Infant Electrocardiogram

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Electrocardiographic patterns of myocardial infarction are rarely found in infancy and suggest, in the first place, an anomalous origin of the left coronary artery. Five infants with such electrocardiograms are reported. In all coronary heart disease could be ruled out, in 4 by autopsy and in 1 by the clinical course. Comparison of these 5 cases with previously reported cases with similar electrocardiograms and autopsy control revealed that coronary patterns in infancy occur in a true and false variety. Some distinguishing features are discussed.

ELECTROCARDIOGRAPHY in infancy is primarily of value in the clarification of abnormal rhythms or in the diagnosis of congenital heart disease by the early detection of hypertrophy or strain of atria and ventricles. Electrocardiographic alterations attributable to functional or organic myocardial derangement have received only scant attention because of the difficulties in distinguishing changes due to primary myocardial disease from normal changes taking place during the first months of life, and the rarity of alterations sufficiently characteristic to suggest specific clinical syndromes. Patterns typical of coronary disease became the subject of special reports, particularly when one of the several causes of occlusive coronary disease in infants and children could be ruled out on the basis of autopsy findings.

The fallacy of diagnosing coronary heart disease solely on the basis of "typical" electrocardiographic features is well known for the adult. It can be mimicked by a variety of etiologic factors—degenerative, inflammatory, parasitic, and neoplastic processes—as well as by the effects of drugs, physical agents, emotional states, and even body shape. Disregarding these possibilities, we erroneously diagnosed a congenital anomaly of the coronary blood supply in 2 infants in whom outstanding alterations of the electrocardiogram were the principal available source of information during an acute fulminating fatal disease. In 3 other cases with similar electrocardiographic changes coronary artery disease could be ruled out because of protracted clinical observation and a consideration of circumstances under which these changes developed. The electrocardiographic features of these 5 cases and a comparison of such "false" coronary patterns with true ones documented in the literature are the subject of this report.

MATERIAL

Case 1 was a 10-day-old Negro girl who developed cough and dyspnea a few hours before hospitalization. She was in acute respiratory distress with a respiratory rate of 80, a regular pulse of 160, and a temperature of 99.6 F. Except for wheezing over both lungs, the physical findings of the respiratory system were normal. The heart seemed enlarged, but no murmurs were heard. The liver was felt 3 to 4 fingerbreadths below the right costal margin. X-ray revealed cardiomegaly and signs of congestion in both lungs. The hemoglobin was 18.1 Gm. per cent, and the white blood cell count was 10,600, with a normal differential count. The electrocardiogram (fig. 1) showed sinus tachycardia of 140 with a P-R interval of 0.12 second. Prominent Q waves and convex elevated S-T segments merging with small inverted T waves were present in leads II, III, aV_R, V_5, and V_6. Reciprocal QRS and ST-T alterations were found in aV_R and in the right precordial leads. The electrocardiogram was interpreted to represent recent injury with necrosis of the posterolateral wall and apex, and on this basis an abnormal origin of a coronary artery from the pulmonary trunk was suspected. The infant died the next morning. At autopsy the heart was markedly enlarged.

*We are indebted to Dr. O. Saphir for the autopsy data in cases 1, 2, 4, and 5. Cases 1 and 2 were included in a study by Drs. Saphir and N. Cohn on myocarditis in infancy.7

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particularly to the right. The coronary arteries were normal in origin and distribution. There were a few petechiae on the septal surface of the left ventricle. The myocardium was pale and flabby and there were yellow grayish areas. Microscopic sections (fig. 1) revealed moderate separation of muscle fibers and bundles. In the left ventricle, approximately one third of the myocardial fibers were involved by necrotic alterations. A pleomorphic cellular exudate was scattered throughout the myocardium with increased affinity for the areas of necrosis, and with varying proportions of polymorphonuclear and mononuclear cells. Within the epicardium were aggregates of closely packed mononuclear cells, generally resembling lymphocytes. Inflammatory cells were present in small numbers in the subendocardial connective tissue and there was a small mural thrombus. No inclusion bodies were found. The pathologic diagnosis was acute myocarditis.

Case 2, a 9-month-old white boy had cough and nasal discharge 1 week prior to admission, and developed vomiting, shortness of breath, and progressive stupor before hospitalization. He was lethargic, pale, and apparently in shock. There was flaring of the alae nasi, and the throat was injected. The cardiac rate was 270 and regular. The liver was felt 2 fingerbreadths below the right costal margin. An electrocardiogram (fig. 2) showed a regular supraventricular tachycardia in which P waves could not be definitely identified. The contour of the ventricular complexes was very bizarre, with slurring of QRS in all leads, small Q waves in II and deep Q waves in III and aVF. The S-T segments were markedly elevated in II, III, aVF, and V4 to V6. Reciprocal S-T depressions were present in aVR, aVL, and in V4R to V2. T waves could not be separated from these S-T deviations. On this basis we suspected an anomalous origin of a coronary artery, causing necrosis of the posterolateral wall, with injury extending to the posterolateral and anterior walls.

The child died 2 hours after admission. Autopsy revealed a heart of normal size with normal origin and distribution of the coronary arteries. The myocardium was flabby and the posterior wall of the left ventricle appeared pale. Microscopically (fig. 2) there was diffuse interstitial infiltration by lymphocytes, mononuclear, and polymorphonuclear cells in varying proportions. A few polymorphonuclear cells were noted in the epicardium. The myocardium showed edema and focal areas of necrosis. Subepicardial areas were more involved than was the subendocardium. No inclusion bodies were found. The pathologic diagnosis was isolated myocarditis.

Case 3, a Negro girl was first admitted to the hospital at the age of 13 months because of severe respiratory distress. Her illness had begun 2 weeks before with symptoms of an upper respiratory infection. Two days prior to admission, anorexia, fever, and frequent vomiting were noted. Her past and family histories were noncontributory, her growth and development had been normal, and she had no previous illness apart from occa-

**Fig. 1.** Top. Case 1. Twelve-lead electrocardiogram showing a pattern of recent posterolateral wall infarction. Bottom. A section of left ventricle showing the dense cellular infiltrate within necrotic muscle fibers. The endocardium is at the upper right. (Hematoxylin and eosin. × 200.)
sional upper respiratory infections and atopic eczema. On admission, her temperature was 101.8°F, the pulse rate was 168, the respiratory rate was 63, and the blood pressure was 82 mm Hg. She had physical findings of right middle lobe pneumonia, and her liver was enlarged to 3 fingers below the costal edge. The heart showed moderate enlargement, a diastolic gallop, and an accentuated second heart sound in the pulmonic area; no murmurs or friction rubs were noted. At x-ray and fluoroscopy, all chambers of the heart appeared enlarged. The pulmonary vascularity was normal. The electrocardiogram (fig. 3, 8-23) showed as the outstanding abnormality an ST-T deviation of discordant type. In leads I, aV_L, and V_4 to V_6, the S-T was markedly elevated in contrast to equally prominent S-T depression in II, III, aV_P, and V_4R to V_2. Merging of these displaced S-T segments with T waves resulted in an almost “monophasic” appearance in some leads, pointing to severe acute injury of the anterolateral wall.

The patient was thought to be in congestive heart failure, caused either by myocarditis or, considering the electrocardiographic findings, by an anomalous origin of the left coronary artery from the pulmonary trunk. Studies of etiologic agents included all available viral and bacterial agglutination tests. All were negative, as were other laboratory studies except for acetonuria and a polymorphonuclear leukocytosis (17,100), a 2+ C-reactive protein, and a slightly elevated serum glutamic oxaloacetic acid transaminase on admission. No etiologic agent was established.

She was treated for pneumonitis with antibiotics and was rapidly digitalized, with prompt improvement. The diastolic gallop disappeared as did signs of congestive heart failure, but the heart size remained the same throughout the hospital stay. Serial electrocardiograms revealed a gradual regression of the effects of acute injury with an evolution of symmetrical T inversions (fig. 3, 10-29). Digitalis was discontinued after the pneumonitis had cleared and all evidence of infection had disappeared. A 6-week course with prednisone was instituted in an attempt to accelerate resolution of the myocardial disease, but no changes were observed. When the patient was discharged after 3 months of hospitalization, she was asymptomatic, active, and gaining weight. Cardiomegaly persisted and the electrocardiogram showed slow restitution to an almost normal configuration (fig. 3, 12-24).

At home she did well without medication. Two weeks after discharge, she was readmitted following ingestion of fuel oil. No ill effects were detected.

At 20 months of age she was hospitalized for the third time with symptoms of fever, cough, and anorexia. There was no evident edema, liver en-

**Fig. 2. Top. Case 2.** Twelve-lead electrocardiogram showing a supraventricular tachycardia and a pattern of recent posterolateral wall infarction. **Bottom.** A section of myocardium showing edema, cellular infiltration, myocytolysis, and early necrosis. (Hematoxylin and eosin. × 200.)
pronounced than previously (fig. 3, 2-22). The S-T elevations were now present in all 3 standard leads and aV_r, while reciprocal S-T depressions were absent in aV_L and over the right precordium, suggesting involvement by renewed severe injury of both anterior and posterior walls. X-ray showed normal lung fields and about the same degree of cardiomegaly as previously observed. The antistreptolysin O titer was elevated (333 units). There was a polymorphonuclear leukocytosis of 14,500 and an elevated transaminase of 56 units. Blood cultures, nose and throat cultures, bacterial and viral agglutinations, urinalysis, and serology revealed no abnormalities. She was treated with antibiotics and digitalis with prompt resolution of the infection, and was discharged after 8 weeks on a maintenance dose of digitoxin.

Again she did well at home until she developed a respiratory infection characterized by fever (105°F.), cough, and abnormal breathing. This occurred 2 weeks after “measles.” She was hospitalized for the fourth time and found to have right upper lobe pneumonia, which responded well to the usual methods of therapy. There was no change in the cardiovascular findings. The blood pressure was 90/45 mm. Hg. The heart was en-
The outstanding feature during the observation of the patient in the hospital was her relative well-being at the times that the gallop rhythm and the electrocardiographic abnormalities were most prominent. On the basis of clinical and electrocardiographic findings and the prompt response to digitalis therapy, it is believed that she developed acute myocarditis following a respiratory infection at 13 months of age with a recurrence at 20 months of age. The persistence of non-specific electrocardiographic abnormalities and of the cardiomegaly after several months, suggests chronicity of the myocardial process although the child continued to grow and develop normally and has so far remained without apparent symptoms.

Case 4, a Negro boy, was born at Michael Reese Hospital after 39 weeks gestation. He seemed normal at birth but was transferred to the pediatric service at 3 days of age because of persistent generalized cyanosis. His pulse rate was 112 per minute and respirations were normal. A grade II harsh systolic murmur was heard at the left sternal edge in the third and fourth intercostal space. A
second heart sound heard in the pulmonic area was of normal intensity and not audibly split. Femoral pulses were easily palpable. Fluoroscopy and x-ray revealed, in the posteroanterior view, a normal-sized heart with lifted apex and concavity in the pulmonary artery segment. The right atrium appeared enlarged and the pulmonary vascularity was decreased. The electrocardiogram (fig. 4, 8-24) showed sinus tachycardia of 158 with a relatively long P-R interval of 0.16 second. The P waves were large, narrow, and pointed in I, II, aV_{rb}, and in V_{1} to V_{R}. In other limb leads they were notched and diphasic. The QRS complexes were tiny in the limb leads with deviation of the QRS axis to the left in the frontal plane. The precordial leads and the T waves of all leads had a normal appearance. These findings were interpreted to represent atrial pathology and, considering the persistent cyanosis, to be compatible with tricuspid atresia.

At 4 weeks of age a severe attack of dyspnea and cyanosis occurred. From 4 to 6 weeks of age similar attacks became increasingly frequent and severe. He was digitalized at 6 weeks of age when he weighed 6 pounds 9 ounces. An unduly large dose (0.5 mg.) of digoxin was given intramuscularly within a 24-hour period. An electrocardiogram recorded the following day (fig. 4, 8-30) revealed the following changes in comparison with the first: the sinus rate was reduced to 136 and the P-R prolonged to 0.24 second. The QRS complexes were mainly inverted in leads I, aV_{L}, and aV_{P}, and predominantly upright in III and aV_{R} as well as in the right precordial leads. The S-T segment showed a conspicuous discordant displacement, being elevated in II, III, aV_{R}, and in the right precordial leads, and depressed in all other leads. T waves could not be separated from the S-T segment and the Q-T interval appeared abnormally short (0.20 second instead of 0.26 second ± 0.05, as expected at this rate. These changes suggesting acute injury involving the diaphragmatic and anterior walls were attributed to unusual digitalis effects, since they were transitory and disappeared in a few days.

An angiocardiogram was taken at 7 weeks of age. It showed that the left heart chambers filled immediately from the right atrium. A left aortic arch was seen to originate from the left ventricle, and a tiny pulmonary artery appeared to communicate with the right ventricle. The impression was of either tricuspid atresia with an atrial and ventricular septal defect or tricuspid stenosis. A surgical attempt to improve pulmonary blood flow by a Potts type of shunt operation was undertaken, but the child died from ventricular fibrillation before surgery was completed.

Autopsy revealed tricuspid stenosis, a thick-walled diminutive right ventricle, and atresia of the pulmonic valve. The foramen ovale and the ductus arteriosus were patent. The right atrium and the left ventricle were dilated and hypertrophic. The coronary arteries originated from a single ostium in the aorta and had a normal distribution. Histologically, the myocardium was slightly edematous and contained large amounts of fibrous tissue.

Case 5, a 10-week-old boy, born after 6½ months of gestation, was doing well until 5 days prior to admission when fast breathing, cyanosis, and slight cough were noted. Examination revealed a respiratory rate of 64 and a regular pulse rate of 140. The thoracic cage showed signs of retraction, and coarse rales were heard over both lungs. There were no murmurs and no signs of congestive heart failure. Chest x-rays showed a heart of normal size and signs of bilateral pneumonia. An electrocardiogram in which only the limb leads were obtained because of technical difficulties (fig. 5, 9-21) showed sinus tachycardia of 164 with a P-R interval of 0.12 second and no abnormalities in contour.

Signs of congestive heart failure developed a few days after admission. Digitalization was started parenterally (0.04 lanatoside C [Cedilanid] intravenously and 0.25 digitoxin intramuscularly) and continued orally in daily doses of 0.105 to 0.030 mg. An electrocardiogram (fig. 5, 10-29) showed an unchanged sinus rate and P-R interval. The precordial leads revealed prominent R waves with an upright T wave in the right precordial leads and predominant S waves in the left, suggesting right ventricular hypertrophy. In the limb leads were conspicuous, discordant S-T deviations—elevation in II, III, aV_{R}, and depression in I and aV_{L}; T waves were not clearly discernible. The Q-T interval appeared shortened but when measured in the precordial leads was 0.26 second, well within the normal range. These changes resembling effects of acute injury of the posterior (diaphragmatic) wall, were attributed to unusual digitalis effects, as in case 4. They disappeared in 2 subsequent electrocardiograms.

The patient died after 3 weeks from progressive respiratory insufficiency. Autopsy revealed acute interstitial pneumonia and hemorrhagic bronchopneumonia. There were multiple congenital abnormalities, including a partial anomalous connection of the pulmonary veins to the right atrium, atresia of the upper portion of the vena cava, and entrance of the hepatic vein into the right atrium. The foramen ovale was open. There was hypertrophy of the right ventricle, grossly and microscopically. The coronary arteries were normal in origin and distribution. Sections of the left ventricular myocardium revealed no significant changes.
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DISCUSSION
The 5 cases have in common electrocardiographic features of various injury patterns that have been shown11–13 to follow interference with the function of large areas of ventricular myocardium owing to sudden loss of blood supply or to action of chemical, thermal, or mechanical noxious agents. In all 5, discordant deviations of the S-T segments indicated the presence of injury currents.13 In cases 1 and 2, in addition, alterations were found in the direction of initial forces of ventricular depolarization, resulting in prominent Q waves in limb and precordial leads. In cases 1 and 3, inversion of T waves was recorded at the time of the S-T deviations or after the disappearance of the latter, indicating a change in the magnitude, direction, and duration of repolarizing forces in some of the affected myocardium. Unless the very unlikely assumption were made that electronegativity in the infant's heart differs from that in the adult human heart or in the experimental animal, the electrocardiographic features must be explained in the light of present knowledge concerning the genesis of injury effects in general and their manifestations in the scalar electrocardiogram with respect to the location of the exploring electrodes.14

In cases 1 and 2, there was an anatomic cause for the pronounced electrocardiographic abnormalities, consisting of a severe inflammatory lesion with diffuse interstitial-cell infiltration and focal necrosis of myocardial fibers, a combination considered by the pathologists to be characteristic of viral myocarditis.7 Electrocardiographically, the pattern closely resembled, in all aspects, a recent myocardial infarct of the posterior and posterolateral (diaphragmatic) walls of the left ventricle. That diffuse myocarditis in children and adults may imitate in the electrocardiogram a recent myocardial infarct has been shown previously.1,17 However, S-T deviations in particular are not a constant feature in this condition.18–23 In our cases the analogy with a pattern of acute infarct was particularly striking in view of the deep Q waves in the left precordial leads in one, and in leads III and aVr in the other. Since no confluent area of tissue necrosis was found anatomically, a severe functional alteration of a large portion of the myocardium must have been present, resulting in the failure of the latter to contribute electromotive forces to the initial stages of ventricular activation. The antemortem diagnosis of an abnormal blood supply to the myocardium, on the basis of a congenital anomaly, was disproved by the findings at autopsy of coronary arteries with normal origin and distribution.

On the basis of this experience, the possibility of an acute myocarditis was considered in the interpretation of the bizarre S-T deformations in the first record of case 3 (fig. 3, 8-23). This case bears a striking similarity in clinical and electrocardiographic aspects to a case reported recently2 and interpreted as myocardial infarction. Both children were older than 1 year and the onset of acute heart failure, cardiomegaly, and electrocardiographic abnormalities followed a respiratory infection. In both, clinical symptoms promptly disappeared after adequate digitalization, but enlargement of the heart persisted and serial electrocardiograms showed an evolution similar to a recent anterolateral wall infarct. Although the latter is the classical electrocardiographic manifestation of an anomalous origin of the left coronary artery, this diagnosis was discarded in our case, and that of an acute myocarditis was preferred, in view of the clinical course and the favorable outcome. Infants ill because of an anomalous origin of the left coronary artery very rarely live beyond 1 year of age and usually die in the third to fifth month,24 whereas recovery from acute myocarditis is not uncommon, especially in older infants.7 The assumption of an inflammatory etiology had further support in the recurrence after 4 months of a new episode resembling the first one in clinical and electrocardiographic manifestations. The latter, at this time, were even more conspicuous with involvement of additional leads (II, III, and aVr). Thus, during reactivation of the presumed inflammatory process, the resulting myocardial lesion appeared more extensive and diffuse, to include the anterolateral as
well as the diaphragmatic aspects of the heart, and became comparable to electrocardiograms recorded in the first 2 cases of proved myocarditis. This concordant type of S-T elevations also raised the question of a complicating acute pericarditis, but this possibility was thought to be excluded on clinical grounds, particularly by the presence of clear heart sounds and the absence of a friction rub and of venous distention. Theoretically, one would not expect that a widespread and scattered lesion of myocarditis would cause S-T deviations. While this is true in the majority of instances, currents of injury can be expected to become manifest in the electrocardiogram once a great number of anatomically and functionally compromised myocardial fibers are located close to the epicardium or extend transmurally.

In cases 4 and 5, the discordant S-T deviations resembling acute injury in the diaphragmatic aspects of the heart were attributed to unusual digitalis effects, since their development and disappearance coincided with administration and withdrawal of the drug. At autopsy no anatomic cause was detected to account for these transient electrocardiographic changes. A common coronary ostium in the aorta, present in case 4, does not interfere with normal blood supply to the myocardium. We are aware that electrocardiographic changes of the type illustrated in figures 4 and 5 are at variance with the usual effects of digitalis in adults as well as in children and infants. Ordinarily the S-T deviations develop in a direction opposite to, rather than parallel to, the main QRS deflection, in keeping with experimentally produced lesions in the subendocardium. However, in some of these experiments, S-T elevations of the type discussed were produced when the animals were exposed to toxic doses of digitalis.

Some of the other changes that developed in case 4, together with the S-T deviations, viz., the prolongation of the P-R interval and the foreshortening of the Q-T, are in keeping with known digitalis effects. More difficult to explain are the alterations in the direction of the QRS complexes that occurred in the limb and right precordial leads in the 2 records of figure 4. A change in the position of the heart appears unlikely, since the P-wave contour remained practically the same in all leads. A better explanation may be that, in this instance, the abnormal electromotive forces responsible for the S-T deviations were "injury currents of activity (demarcation potentials)" rather than "injury currents of rest." The latter are thought to produce S-T deviations primarily by shifting the T-P segment relative to the QRS inscription; the former come into action by virtue of incomplete depolarization of myocardial fibers and thus may result in significant QRS alterations in addition to deviations of the S-T segments.

Our error in diagnosing a congenital anomaly of the coronary circulation in cases 1 and 2 on the basis of electrocardiographic findings prompted us to review similar electrocardiograms reported previously in infants and young children. Considering only cases in whom the cause of abnormalities was established by autopsy, a total of 22 such cases was collected. Fourteen cases had an anomalous origin of the left coronary artery from the pulmonary trunk and in all, deep Q waves, S-T elevations, and inverted T waves in various combinations occurred resembling a lesion of the anterolateral wall. In 5 cases an acute myocarditis was established as the cause of the electrocardiographic changes; in 4 of these, the electrocardiographic abnormalities resembled an anterolateral, and in 1 a posterior wall infarct. The remaining 3 cases, all with a pattern of recent anterolateral wall infarction, had various etiologies: In one a primary malignant tumor of the heart was found, in another actual infarction of the left ventricle was due to thrombosis of the left coronary artery, and in the last, a case of complete transposition of the great vessels, necrotic changes in the left ventricle were attributed to acute subsidence of an intracardiac shunt partly compensating for abnormal hemodynamics. Thus, when the 4 autopsy cases of this report are added, the entire material can be divided into 2 almost equal groups consisting of 14 "true"
and 12 "false" coronary patterns. All cases of the former group displayed a pattern of anterolateral wall infarction; in the latter group about two thirds showed a more or less characteristic anterior, and one third a posterior or posterolateral pattern.

The distinction during lifetime between a "true" and a "false" coronary pattern occurring in infants may in the future have important clinical implications. Feasible surgical corrective methods have been developed for abnormally originating left coronary arteries, based on recent studies on the abnormal hemodynamics in this condition and the revision of older concepts of the causes of the associated myocardial ischemia. The electrocardiogram may prove of prime importance when, as in our cases 1 and 2, the observation time is too short to permit a correct diagnosis on clinical grounds and the need for a life-saving procedure is great. The present review would indicate that under such circumstances an anomalous origin of the left coronary artery from the pulmonary artery can be excluded when the electrocardiogram displays features of injury of the diaphragmatic (posterior or posterolateral) aspects of the heart. An anomalous origin of the right coronary artery, which theoretically could produce such changes, is extremely rare and does not give rise to clinically recognizable abnormalities. The finding, on the other hand, of an anterior or anterolateral wall injury pattern does not permit a distinction between a true and a false coronary pattern. Among the several causes of false coronary patterns, an acute myocarditis must be considered first.

Summary and Conclusions

Electrocardiograms are presented of 5 infants with characteristic changes of severe acute injury, imitating patterns of acute myocardial infarction. In 4 of the cases abnormality of the coronary blood supply could be ruled out by autopsy, in 1 it was thought to be excluded on clinical grounds. In 3 the changes were attributable to acute diffuse myocarditis, and in 2 to unusual digitalis effects.

A comparison with similar previously reported infant electrocardiograms, in which the etiologic factor could be ascertained by autopsy, suggests that coronary patterns in infancy occur in a "true" and in a "false" variety.

The true variety, caused by a congenital or acquired disorder of coronary blood supply to the myocardium, is associated with a pattern of anterior or anterolateral wall infarction. In the false variety either an anterior or posterior wall pattern may be found. The commonest cause of a false coronary pattern in an infant appears to be an acute diffuse myocarditis.

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Summario in Interlingua

Es presentate electrocardiogrammas de 5 infantes, exhibiente la alterationes characteristic de sever injurias acute, simile al configurationes de acute infarcimento myocardial. In 4 del casos, anormalitate del provision coronari de sanguine poteva esser eliminable como causa del alterationes mentionate, gratias al disponibilitate de datos necroptic. In 1, ille explication pareva esser eliminabile super le base de observationes clinic. In 3, le alterationes eseva attribuibile a diffuse myocarditis acute; in 2, a effectos inusual de digitalis.

Un comparation del presente constatazione con simile electrocardiogrammas infantil previamente reportate e permettente le identification de factor etiologic super le base de examines necroptic suggere que configurationes coronari occurre durante le infantia in le 2 varietates de "ver" e "false."

Le varietate ver, causaete per congenite o acquirite disorderes del provision coronari de sanguine al myocardio, es associate con un configuration de infarcimento de pariete anterior o antero-lateral. In le varietate false, le configuration pote esser illo de infarcimento del pariete anterior o illo de infarcimento del pariete posterior. Le causa le plus commun de un false configuration coronari in infantes pare esser le presentia de diffuse myocarditis acute.
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The test objects were dogs, anesthetized with morphine-nembutal. Quinidine intravenously regularly produce tachycardia in dogs. There was also an immediate hypotension of short duration. The tachycardia was more persistent. Twenty-five minutes after quinidine the cardiac output was not changed significantly. Work of the left ventricle did not change. Systemic arterial resistance was unaltered. However, there was a decrease in right ventricular work and total pulmonary resistance. Moreover, coronary blood flow and cardiac metabolism were markedly increased. Vascular resistance in the coronary bed was diminished. Myocardial efficiency was markedly decreased.

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