Electrocardiographic Findings in 98 Consecutive Non-penetrating Chest Injuries*

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Non-penetrating bodily injury is a well established cause of heart disease; an increasing number of reports deals with traumatic heart injuries in which the role of trauma was well established either at necropsy or during surgical procedure. Clinically, a traumatic cardiac injury may be evident sometimes, or sometimes difficult or impossible to recognize.

Electrocardiography is usually considered a valuable diagnostic tool for detection of traumatic heart disease, and electrocardiographic abnormalities have been reported in high percentage in traumatized patients. However, in the majority of cases, such abnormalities are not specific and they have not been considered a conclusive diagnostic criterion of cardiac trauma.

Electrocardiographic studies of 98 cases of unselected patients with various degrees of closed chest injuries form the basis of the present report. Changes related to trauma will be discussed in detail.

METHOD

Ninety-eight consecutive patients were admitted to the emergency ward of the Arcispedale of S. M. Nuova from November, 1964 to November, 1965, immediately or within a few hours after having sustained various types of non-penetrating chest injuries. Seventy-seven had had an automobile or motorcycle incident, and 12 had hit their chest on the steering wheel. The remaining 21 patients fell from variable heights. The age of the patients varied from 13 to 83 with a mean of 48 years. Twenty-nine were women and sixty-nine were men. The stay in the ward varied from a few days to several months. Eight patients were again hospitalized one or two months after the incident. The clinical history was taken as soon as feasible after the event; it included the family history, normal activities of the patient, presence or absence of scarlet fever, diphtheria, rheumatic fever, syphilis, diabetes, arterial hypertension and coronary and/or myocardial disease. The patient was specifically asked if he had had any previous medical or laboratory (electrocardiogram, chest x-rays, etc.) check-up. The mechanism of the wounding force was investigated as accurately as possible.

A complete physical examination was done in every case. At least one chest x-ray picture and one electrocardiogram was taken of each patient. Additional electrocardiograms were done at frequent intervals in those patients showing abnormalities and in most of those who had normal electrocardiograms, but had been subjected to moderately severe or severe trauma (Table 1).

RESULTS

Severity of trauma: trauma was arbitrarily divided as light, moderately severe and severe either by an evaluation of the magnitude of the wounding force or of clinical findings (number of fractured ribs, presence of hemothorax, presence and severity of shock) or by both these criteria. Accordingly, 18 patients were considered to have sustained light trauma, 69 moderately severe and 11 severe.

Clinical manifestations: no patient developed symptoms compatible with the diagnosis of cardiac trauma. No sign of traumatic cardiac involvement could be found on clinical and radiologic examina-
tion. Pulmonary and pleural involvement were found in many patients with moderately severe trauma and in most of those who had severe trauma. Fever of low or moderate degree was absent in patients with light trauma, almost always present in the two other groups. It generally subsided within a week. Brachial pressure was at shock levels in the group of severely traumatized patients returning gradually to normal values within the first 12 hours from the incident.

Electrocardiographic abnormalities were found in 37 cases; they were considered to be preexistent in 18 cases either because they were demonstrated by a previous electrocardiogram or because they were in accord with the patient's history of heart disease and remained unchanged when repeatedly controlled after trauma. They included seven cases indicating myocardial alterations, six with left ventricular strain patterns, two with right bundle branch block, one with left bundle branch block, one with atrial fibrillation and one case with first and second degree A-V block.

In the remaining 19 cases, electrocardiographic changes could be attributed to trauma. They consisted in five cases of diffuse light depression of the ST segment and shallow inversion of the T waves; in four, there seemed to be a somewhat common pattern with increasing abnormalities (Fig. 1-4). In the fifth case, a different pattern was present (Fig. 5). In 14 cases, changes were limited to flattening or inversion of T wave in leads III, aVF and V1, or in flattening of T waves in one or two chest leads. There was a rough proportion between the degree of trauma and the percentage of electrocardiographic changes related to it (Table 1). Four of five cases with more marked changes (Fig. 2-5) were found in patients who sustained severe trauma. The duration of electrocardiographic changes ranged from five to 20 days.

**Discussion**

Electrocardiographic abnormalities following non-penetrating bodily trauma are reported in percentages varying from 17% up to 38% and 40% per cent of cases. In our series, they were present in 19 per cent.
The different figures may be related to the different degree of trauma sustained by the patient of each series, the timing of recording electrocardiograms and the type of electrocardiographic variations estimated worth to be reported by each author. Most of our patients sustained moderately severe or light trauma and the timing of recording first and second electrocardiogram could make detection of minor electrocardiographic changes difficult or impossible.

The impression one receives reading papers dealing with this subject is that any electrocardiographic variation encountered after trauma is considered equivalent to traumatic heart injury, though all the authors state that a great part of such variations are not specific. According to this point of view, the percentage of heart lesions thus calculated would appear even larger than that found at necropsy in series of fatalities for bodily injuries. Functional heart injury could be advocated to explain such discrepancy, but in our opinion, this is theoretic and a critical attitude towards this problem appears more scientifically sound.

![Figure 2](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21450/)

**Figure 2:** A 60-year-old active woman was struck by a car while crossing the street on May 31, 1965, but she walked by herself to the hospital the same day. Ribs were found to be fractured from the second to the sixth on the left. There was a fracture also of the 12th dorsal vertebra. Basilar left pleural effusion developed during her stay in the hospital.
Arrhythmias and A-V conduction defects of proved traumatic etiology,11 can be retained as important signs of myocardial damage, though they may be mediated through a nervous mechanism. Sinus tachycardia, exceeding 110 beats per minute, was found in ten cases of our series (Fig. 1, 3, 4), but many factors, such as shock, emotion, etc., besides heart injury, could be responsible for it.

Significant displacements of the ST segment, followed by pointed inversion of T wave, accomplished or not by changes of the QRS complex, usually indicate a myocardial contusion.11 For instance a coronary pattern was present in seven of 12 cases of traumatic perforation of interventricular septum,12 but was not encountered in our series. In one case, there was a shallow, transient inversion of the T wave in pos-

**Figure 3**: A 43-year-old man, suffering from chronic bronchitis, was struck by a car while riding his motorcycle on October 10, 1965 and was hospitalized in severe shock with subcutaneous emphysema and fractures of the fifth to the ninth left ribs. Left pleural basilar effusion appeared in the following days. Sinus tachycardia of 136 and 107 respectively is shown by the first and second records.

**Figure 4**: A 37-year-old woman was violently projected against the dashboard of her car. She was immediately hospitalized in severe shock on November 2, 1964. Ribs were fractured from the fourth to the sixth on the right. Bilateral pleural effusion and extensive linear atelectases were demonstrated by chest x-ray pictures. First record shows sinus tachycardia of 136 per minute and a pattern suggestive of right ventricular strain.
terolateral leads (Fig. 1), but this finding could only suggest a cardiac contusion.

In 18 cases, electrocardiographic changes related to trauma consisted in minor and major specific alterations of the ST-T (Fig. 2-5). Several factors need to be carefully evaluated before attributing changes of this type to myocardial injury. Increased heart rate per se could be partly responsible for abnormalities of the T wave in some of our cases (Fig. 1, 3, 4). Emotional strain, induced by trauma, could be advocated as another cause of ST-T abnormalities. Pulmonary contusions, an often neglected visceral injury, might determine pulmonary circulation derangements, either reflex or through hypoxia, the release of histamine and serotonin and therefore be responsible for patterns suggestive of right ventricular strain (Fig. 3,4). The role of respiratory dysfunction, which ensues in severe chest trauma, will have to be investigated through blood gas analysis and electrolyte determinations in future works on this subject. Transient nature of electrocardiographic changes observed in most of our cases favors the hypothesis that the above cited extracardiac factors are in operation and tends to discard that of myocardial injury.

It is the authors' opinion that previous considerations do not lessen at all the value

![Figure 5: A 35-year-old woman was thrown out of her car when it collided with a tree at high speed. She was hospitalized unconscious on November 24, 1964 with several lacerations of the head and trunk, fracture of right femur, but no rib fractures.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21450/ on 06/01/2017)
of electrocardiography in the detection of traumatic heart diseases. They simply stress once more the need for critical use of this technique.  

**Summary**

Ninety consecutive patients with non-penetrating chest injuries were observed for evidence of cardiac trauma. Eleven were arbitrarily considered to have sustained severe trauma, 18 light trauma and 69 moderately severe. No signs or symptoms of traumatic cardiac involvement were found on clinical and radiologic examinations. Electrocardiography revealed abnormalities related to trauma in 19 cases (19 per cent). In 15 cases changes were limited to flattening of inversion of T wave in leads III, aVF and V1 or in flattening of T waves in one or two chest leads. In five cases, which are reported in detail, there were diffuse light depression of the ST segment and shallow inversion of the T waves. Such changes have not been attributed to myocardial injury, since several extracardiac factors, such as tachycardia, emotion, pulmonary circulation derangements, pulmonary dysfunction, appeared to be more convincing factors in their production.

**Resumen**

Hemos observado 90 pacientes consecutivos con lesiones no penetrantes del tórax desde el punto de vista de posible traumatismo cardiaco. Once fueron arbitrariamente considerados como gravemente lesionados. Dieciocho con lesiones ligeras y 69 medianamente graves. Ninguno presentaba indicios clínicos o radiológicos de traumatismo cardiaco. El electrocardiograma reveló anomalidades relacionadas con lesiones traumáticas en 19 casos (19 por ciento). En 15 casos las alteraciones del ECG se limitaban a depresión de la onda T en las derivaciones IV, aVF y V6, depresión de la onda T en una o dos derivaciones torácicas. En 5 casos reportados en detalle hubo depresión difusa ligeramente del segmento ST, e inversión poco pronunciada de la onda T. Tales cambios no han sido atribuidos a lesión del miocardio, ya que diversos factores extracardíacos tales como taquicardia, emoción, trastornos de la circulación pulmonar y alteraciones funcionales pulmonares, aparecían como factores mas convincentes en su patogenia.

**Zusammenfassung**


**References**

Diseases


SERUM LACTIC DEHYDROGENASE ISOENZYMES IN MYXEDEMA HEART DISEASE

Serum lactic dehydrogenase isoenzyme studies have been made on 48 patients with hypothyroidism. In 17 (35 per cent), the isoenzyme distribution pattern resembled that of acute myocardial infarction with differential ascension of LDH-1 and LDH-3 isoenzyme activity. It is suggested that this evidence of myocardial damage represents a specific form of cardiomypathy resulting from chronic thyroid deficiency.

Re-establishment of a euthyroid status with hormone therapy was accompanied by gradual disappearance of the “heart” isoenzyme distribution, ECG improvement, and reduction in heart size. Serial LDH isoenzyme studies provided considerable help in the management of those patients with coronary artery disease and hypothyroidism in whom too rapid or excessive treatment may provoke myocardial infarction.


ANGIOGRAPHY OF PULMONARY EMBOLISM IN “MITRAL LUNG”

The value of pulmonary arteriography in the diagnosis of pulmonary embolism will be enhanced if sources of error are recognized. The vascular changes in mitral stenosis with pulmonary hypertension and other conditions of post-capillary hypertension which the author calls “mitral lung” are characterized by narrowing of the vessels to, and underperfusion of, the lower zones. Narrowed vessels, pruning, underperfusion and oligemia are otherwise secondary signs of embolism. In the mitral lung, they lose their diagnostic validity. One has to fall back upon the direct signs, the intraluminal filling defect, gross cut-off, or localized asymmetrical pruning. This limits the value of the method to some degree and with proper precaution leads to under-reading or “false negatives.” Pertinent precaution should be exercised in the diagnosis of pulmonary embolism in the mitral lung, both in the use of lung scanning with radioactive macro-aggregated albumin and in the plain roentgenogram.