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Electrographic changes due to blunt trauma to the thorax

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E.C.G. CHANGES DUE TO BLUNT TRAUMA TO THE THORAX

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E.C.G. CHANGES DUE TO BLUNT TRAUMA TO THE THORAX

There are various types of E.C.G. changes
connected with blunt trauma to the thorax such as
the arrhythmias, RST components, T waves, and low
voltage. (1,2,3)

Before exploring these in greater detail the
following is a discussion of some basic principals
of electrocariography. (4)

A volume conductor is a medium which permits
the conduction of electricity in three dimensions,
and the human body can be classed as such. Current
may be caused to flow if two electrodes are inserted
in a saline bath and if the ends are connected to the
poles of a battery. For all practicle purposes the
human body may be classed as a large saline bath.
We can then consider the patient as a volume conductor
and the electric impulse originating in the heart as a
source of potential differences. With the use of a
galvanometer the magnitude and direction of the cur-
rent produced may be measured. If some method of
making a permanent record from the galvanometer's
readings is employed we will have an electrocardio-
graph.

If a galvanometer, the electrocardiograph, is

attached to the right and left arms of a patient, the differences in potential between these points may be measured and recorded as lead I of the electrocardiogram. By arbitrary construction of the electroc rdiograph, the current is conducted intentionally through the galvanometer so that whenever the right arm is relatively negative and the left arm is relatively positive, there is recorded an upward or positive deflection on the completed electrocardiogram.

If the electrodes are attached to the right arm above the wrist and to the left leg just above the ankle this will record lead II. Current is conducted through the galvanometer in such a manner as to produce an upward or positive deflection in the finished electrocardiogram when ever the right arm is relatively negative and the left leg relatively positive.

When the electrodes are attached to the left arm and left leg this records lead III. The current is conducted through the galvanometer in such a manner as to produce an upward or positive deflection in the finished electrocardiogram whenever the left arm is relatively negative and the left leg relatively positive.

in restitution of the positive and negative charges to their respective positions along the surface of the membrane. Process of repolarization begins at those points where depolarization first began. The wave recorded here n repolarization will be in the opposite direction of that of depolarization. Since repolarization is slower than depolarization, the wave of the former does not fall and rise as abruptly as that of the latter.

Cardiac muscle acts in a manner analogous to the membrane and cell described previously. As a depolarization impulse travels through the myocardium, a positive charge precedes and a negative charge follows the wave of depolarization.

If the heart were a completely enclosed sphere of muscle and the electric forces acted equally in all directions, no potential difference would be detected by the ordinary electrodes employed clinically and no electrocardiogram or potential difference would be recorded. The ventricles however are not a completely enclosed sphere of muscle. They form a more or less irregularly shaped shell with the open region at the base in the area of the atrioventricular valves. It is because of this open region that electric forces are detectable by the ordinary

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The interventricular septum with its more or
less equal and opposite forces normally exerts its
influences on the electrodes to a negligible extent,
depending in a large part upon the position of the
electrodes in relation to the heart.

The electric axis is continually changing in
direction and magnitude as depolarization of the
cardiac musculature progresses. It is not a single
static vector force as the discussion immediately
preceding might suggest. The direction of the axis
depends upon many factors, such as the health of the
muscle and the relative thickness and positions of
the ventricular walls. If the electric axis is
plotted at any given instant during the depolariza-
tion process, there is derived a vector force which
is termed the mean instantaneous electric axis.

The average direction and magnitude of all the
mean instantaneous axes produced during depolariza-
tion of the ventricles constitute the mean electric
axis of the QRS complex or the mean electric axis
during depolarization of the auricles constitute
the mean electric axis of the P wave. The same def-
initions hold for the repolarization processes.

The following is the mechanism of production of

the electrocardiographic pattern seen in myocardial
infarction which is similar to that seen in hearts
injured by blunt trauma to the thorax.

The normal resting heart is fully polarized
and there is no difference of potential existing
between the electrodes of the right and left arms.
When an impulse is delivered to the subendocardial
surface by the Purknji system a wave of depolariza-
tion migrates out perpendicular to the epicardium.
After certain physicochemical processes have occured,
the recovery process or wave of repolarization moves
perpendicular to the endocardium beginning at the
epicardium.

The repolarization force in the left ventricle
is directed away from the right toward the left arm
where as the smaller force results in a vector force
directed toward the left arm making the left arm
electrode positive and the right arm electrode neg-
ative. Therefore during the period of repolarization
the galvanometer swings to the positive direction or
upward in lead I and a positve repolarization or T
wave is inscribed. When the heart has been complete-
ly repolarized and the resting state has been acheiv-
ed no differences in potential will exist and the gal-
vanometer will swing back to zero or isopotential

line completing the downstroke of the T wave.

The following is an explanation of how an infarct effects an electrocardiogram.

There are three different zones in the region of the myocardial infarct, the dead zone, the injury zone, and the zone of ischemia.

The dead zone is the central zone which is composed of dead cardiac muscle. The zone is free from any physiologic or active electrocardiographic phenomena and therefore may be considered a physiologic hole or cavity in the myocardium.

The zone of injury is a shell of cardiac muscle of variable thickness that surrounds the dead zone. The muscle in this zone is injured to a variable degree. Some portions are progressing to recovery and others are regressing toward death. This zone is responsible for effects of currents of injury in the E.C.G.

The zone of ischemia is a shell of cardiac muscle of variable thickness surrounding the zone of injury. The muscle in this zone is injured slightly, that is, to a lesser degree than the muscle of the aforementioned two zones. This zone accounts in particular for change in the processes of repolarization reflected in the E.C.G. as T wave changes.

The following is an example of the E.C.G. changes occuring because of an infarct in the right ventricle.

If an impulse is delivered by the Purkinji system to the subendocardrial layer of muscle of the right and left ventricles, a wave of depolarization is started in both ventricles and progresses towards the epicardium. The septum is omitted from the discussion because its role is of little significance except in special situations.

The resulting electric force produced in the wall of the right ventricle exceeds the magnitude of that produced in the noninfarcted subendocardial shell of muscle in the left ventricle. Therefore, the vector addition of the forces yields a force which is directed toward the right arm electrode. The right arm then is relatively positive and the left arm is relatively negative. When such a polarity exists for lead I, the galvanometer is deflected in the negative direction. As the process of depolarization progresses the right ventricle becomes completely depolarized. This makes the left arm electrode relatively positive terminally in the depolarization process with respect to the right arm electrode and, therefore the galvanometer is deflected positively.

Because much of the right ventricle has been destroyed by the infarct a relatively small R wave is produced as the terminal deflection of the QRS. The larger the infarct the smaller the R wave will be.

It is then evident that the dead zone or infarcted area itself is indirectly responsible for the Q deflection and the small R wave or the QRS changes encountered in myocardial infarction.

The elevation of the S-T segment is due indirectly to the current from the control circuit of the electrocardiograph. It is thus evident that a shift of the S-T segment is due to a current of injury or is due to the zone of injury.

After a short time, while the physicochemical processes occur , the excited state begins to return to the resting state. The process of repolarization begins at the epicardial surface of the right ventricle and migrates towards the endocardium. An electric force is created that is directed toward the right arm electrode. In the case of the infarcted region of the right ventricle, the area of ischemia retards the physicochemical process concerned with recovery. Repolarization begins at the subendocardial surface where the muscle is more normal and migrates toward the epicardial surface. The electric

force thus produced is directed toward the right arm to be relatively positive and the left arm relatively negative. With such polarity of the arm potentials in lead I, the galvanometer is directed downw rd. Thus it is evident that the zone of ischemia is responsible for the T wave changes observed in infarction.

Arrhythmias caused by infarcts or by trauma to the heart are caused by a disruption of the conducting mechanism producing blocks and other rhythmic abnormalities because of muscle death.

The arrhythmias are the most common changes connected with blunt trauma to the thorax. The most common of the arrhythmias are premature contractions. Auricular fibrillation, auricular flutter and auricular paroxysmal tachycardia are the next most common type of cardiac arrhythmias caused by blunt trauma. Some other arrhythmias which are less frequent are sino-auricul r block, idioventricular rhythm, ventriuclar paroxysmal tachycardia and ventricular fibrillation. The last three arrhythmias are frequent causes of death in fatal cases. (5,6,7)

The RST component changes are similar to those changes seen in myocar ial infarction and pericarditis, the RS-T segment often consists of rounding,

elevation, and upward concavity in one or more leads as seen in pericarditis but there may also be coving as seen in myocardial infarction. (7,8,9)

The QRS complex change may consist of variations in height of various components, the development of slurring and notching and the disappearance of some of the waves of the complex. (10-11)

T wave changes are similar to those changes seen in myocardial infarction and pericardial inflammation. There may be characteristic T-1 or T-11 changes with the corresponding changes in the unipolar chest leads or there may be flattening and negativity of the T waves. (7)

Unipolar leads are also useful in locating the position of the injury and identifying the position of the injury or traumatic pericarditis. These findings are not reciprocal and when pericardium only is involved do not show the characteristic QRS changes that are seen in myocardial infarction. (6)

All the above findings may be effected by a generalized low voltage and alteration of the E.C.G. does not occur until 24-48 hours after injury.(12-13)

Cardiac damage resulting from chest trauma has been known to occur ever since postmortem examinations became part of medical investigation. (10) Until

recently it was beleived that most cases of cardiac damage were fatal, and that they occurred only in severe, penetrating chest injuries. In the past three decades however and particularly with the advent of electrocardiography, numerous clinical and experimental studies have demonstrated that many cases of severe cardiac damage including rupture of the heart result from non-penetrating chest injuries, although the thoracic cage remains intact without as much as a fractured rib. There is no doubt that many cases of myocardial contusion or damage to the heart in non-penetrating chest trauma are missed and not diagnosed. Since physical signs of cardiac contusion or of myocardial damage are often absent if the pericardium is not involved, the condition is not recognized unless repeated E.C.G. tracings are made. The symptoms of pain, particularly on respiration, are usually ascribed to muscular contusion, or to traumatic pleurisy but more definite symptoms of an anginal syndrome or of myocardial insufficiency may develop or become apparent later, after recovery from the initial chest trauma had taken place.

The portion to follow is a series of examples taken from the literature to illustrate the above mentioned changes.

Example number one published by Cary, Jurst and Arentzen. (14)

A 21 year old white male received a crushing injury to the chest in an automobile accident. The patient was treated for shock then transferred to the U.S. Naval Hospital, Bethesda, Maryland for more definitive treatment.

E.C.G. changes compatable with muscle death, injury and ischemia, and a conduction disturbence suggestive of right bundle branch block were all observed. Finally the blood pressure could not be maintained even with intra-arterial blood transfusions, and he died suddenly 52½ hours after admission. At autopsy there was an interventricular septal rupture one centimeter below the membranous portion and extended 5.5 centimeters in length.

Example number two published by Sigler. (15)

A 67 year old white male fell on the ground and sustained a fracture of the left humerus, the left third, fourth and fifth ribs, and contusions of the chest. The patient complained of pain in the injur-

♦

ed areas but no symptoms referable to the heart. E.C.G. two days later shows left bundle branch block. Four days later the E.C.G. still showed left bundle branch block but some changes are noted in the con-

figuration of the complexes especially in lead II.
Eleven days after the accident another E.C.G. shows
further changes in the configuration of the ventricu-
lar complexes and left bundle branch block is still
present. The bundle branch block probably existed
before the accident, but the changes in the appear-
ance of the complexes from time to time assume
sig-nificance in indicating acute alterations in
inter-ventricular conduction.

Example number three published by Sigler. (15)
A 46 year old physician collided head-on with another
car and was thrown against the steering wheel. He
sustained fractures of the second to the sixth ribs at
the costochondral junction with dis-placement. An
E.C.G. obtained one and one-half years before the
accident shows a tendency toward left axis deviation.
One obtained six days after the accident shows a
definitely lower voltage T wave in all leads. Another
tracing obtained about seven weeks after the accident
is the same as the one before the accident. Later he
went back to his practice.

The following are some examples of E.C.G. changes
because of trauma to the thorax which happened here in
Nebraska.

Example number one by personal communication

with Dr. Taylor. (16)

Miss M. C., a 58 year old white female was struck by an automobile on 9-6-56 and received superficial cuts and bruises to the head and neck but also receive bruises on the anterior chest wall and also complained of mild precordial pain. Chest x-ray was not remarkable and was negative for frac-tured ribs. E.C.G. taken on 9-8-56 showed an invert-ed T wave in lead AVL but was otherwise with in norm-al limits. A repeat E.C.G. on 9-13-56 showed that the T wave in lead AVL had reverted to normal and the rest of the E.C.G. had no changed.

Example number two by personal communication with Dr. Taylor. (16)

Mr. H.M., a 65 year old white male who fell down a 10 to 12 foot deep man hole nd struck his chest against a ladder on the way down breaking the fifth, sixth and seventh ribs on the right. The acci-dent occured on 4-19-57 and an E.C.G. taken that day showed an inverted T wave in leads II and III and slurring of the s-t segment in lead AVL. Repeat E.C.G. on 4-24-57 showe a reversion back to a tracing with in normal limits.

Example number three by personal communication with Dr. Taylor. (16)

Mr. A.⅃., a 38 year old white male was involved in

an automobile accident on 5-4-50. An E.C.G. on

5-5-50 showed an occassional premature from an abber-ant

focus. Repeated E.C.G. on 5-8-50 showed a normal rhythm,

a wandering pacemaker and auricular flutter alternating

1:1, 2:1, and 3:1. The auricular rate varied between

200-300 per minute and ventricular rate of 100-150. The

tracing on 5-24-50 showed right ven-tricular strain nd

digitalis effect. On 6-3-50 there was no change. By

6-18-50 the tracing was back to with in normal limits.

The following is an experimental study of

non-penetrating wounds of the heart by Kissane, Fidler,

and Koons. (17) The subjects used were male dogs 17 to

25 kilograms in weight. E.C.G.s were taken before and

after the experiment was preformed. the heart was

exposed under aseptic precautions and then the

pericardium was incised. At this time the myocardium was

bruised and usually this was done by striking the

myocardium with a metal dilator weighting 40 grams.

Repeated strokes with the dilator were applied to a

localized area over either the right or the left ven-

tricles. The E.C.G.s in these experiments showed a

variety of alter tions from the normal. These devia-

tions from the normal E.C.G. in general had disappear-

ed after about a month but some of these alterations persisted for alonger period of time. It is interesting to note that the E.C.G. obtained in the experiments in which blood was injected into the interventricular septum was some what similar to the E.C.G. obtained in the experiments in which the myocardium was bruised. This together with the fact that most of the E.C.G. ch·nges disappeared after a few weeks would indicate that these alterations were largely due to hemorrhage.

Since serious myocardial injury is possible in an injury to the thorax without fracture of ribs or sternum it is important to know something of pathologic anatomic and histologic character of the cardiac lesions since other objective evidence of injury may not be present.

Moritz and Atkins' study (18) of myocardial lesions produced experimentally in dogs by cardiac contusion stated in about 44% of the cases secondary exudative changes in the lesions gave them a character that might be designated as traumatic myocarditis. This consists of capillary hemorrhage, infiltration with leucocytes, edema, resolution and finally scar tissue formation.

The experimental method of producing cardiac

of trauma in direct continuity with it, the characteristic lesion was a profound disorganization of tissue with diffuse hemorrhage. Muscle cells were fragmented and displaced, and the orderly streaming of fibers was completely interrupted. In some instances, the local injury was less diffuse and was represented by single or multiple lacerations extending in various directions and to varying directions and depths in o the myocardium.

Injuries not in direct continuity with the site of contusion consisted of foci of ruptured muscle fibers. Some times these ruptures included several and sometimes many adjacent muscle fibers. Both large and small lacerations were occupied by hematomas. With in 24 hours the lesions became infiltrated with poly-morphonuclear leukocytes and at the end of three days the leukocyte infiltration was diffuse and in places quite dense. The tissue was edematous and in addition to the hemorrhage and leukocyt oses there was obvious necrosis of muscle fibers with loss of nuclear integrity, loss of crossstria-tions and swelling and granularity of cytoplasm. Although there was diffuse extravasation of erythro-cytes, the hemorrhage appeared less pronounced than in dogs that were examined within 24 hours after

18

trauma.

Hearts examined between one and two months after contusion revealed advance organization of the lesions. Collapse of the damaged tissue had occurred and where the damage had been superficial there were depressed scars in the epicardium. On incision these scars were irregularly shaped and sharply defined and were mottled red and golden brown. Remote noncommunicating sites of injury did not as a rule lead to epicardial or endocardial deformity because of their deep position in the myocardium.

In some pericardial changes one month or longer after contusion there were pericardial adhesions in eleven of the twenty one dogs.

Endocardial changes seen in this series of dogs with cardiac contusions bore striking resemblances to the gross and microscopic changes seen in myocardial infarction in man.

Possibilities of a myocardial injury once it occurs are that the symptoms disappear hours or days after the accident and the patient may remain well. Symptoms may persist for years and may be accentuated with exercise. The heart may fail hours of days after the accident or the contusion may soften and rupture may take place. (most commonly after two

weeks but may occur any time during the first month)(5)

Since this is the case, theraputic measures may
be taken to guard against myocardial rupture .nd
failure such as absolute bed rest, use of morphine
and other sedatives as necessary, heart saved as much
as possible and all exertion avoided, use of mild
laxatives if necessary to avoid straining at stool,
digitalis may be indicated for the tachycardia or
auricular fibrillation, surgical intervention if and
when rupture occurs, and if there is evidence of a
pericardial effusion tapping the pericardial cavity
will improve circulation. Also sutures placed at site
of hemorrhage and if myocardium is extensively bruis-
ed or softened, grafts of pericardium should be plac-
ed on the area of contusion and securely sutured to
myocardium.

Careful rest for weeks or months (a minimum of
three to four weeks) should be prescribed in order
to assure as sound a healing of the myocardial in-
farct or contusion as possible, with a very gradual
and careful convalescence; by wise treatment at the
start, life may doubtlessly be prolonged for many
years in some cases. (19) At times shortly after
coronary thrombosis when the patient is feeling well
and therefore possibly too active, sudden death from

cardiac rupture or other cause may occur. Sutton
and Davis (20) m de the interesting observation that
in dogs, rest for six days after the production of
cardiac infarction permitted the formation of a small
well-contracted scar without thinning of the wall of
the ventricle, while exercise within three days of the
infarction produced aneurysmal bulging of the ven-
tricular wall with a thin scar. The absolute need
of complete rest for two weeks after a large acute
myocardial infarction in man has been clearly dem-
onstrated by the finding of rupture of the heart
during the first twelve days in 73% of psychopathic

patients, in contrast to only 9.5% of patients in the
wards of a general hospital.

In summary and conclusion this study shows that
blunt trauma to the chest can cause E.C.G. changes
which are similar to those found in coronary throm-
bosis. Because the lesions of coronary thrombosis and
blunt cardiac trauma are rather similar patholog-
ically, a case of cardiac contusion should be treat-
ed much the same as a coronary thrombosis to decrease
the possibility of rupture of the heart due to ane-
urysmal bulging of the ventricular wall at the site of
trauma.

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