Electrocardiogram In Coma Due To Intoxication

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Summary:

We analyzed electrocardiogram changes of 20 patients treated because of intoxication coma at the Department of Internal Medicine. Particular attention was paid to the corrected Q-T interval. The etiology of intoxication coma was the use of anxiolytics, ethanol, pesticides, antidepressants, anti-epileptics, sedatives, hypnotics, trichlorethylene and anti-diabetic chlorpropamide. According to Reed's classification of the depth of coma, nine patients were in degree O-I, seven in degree II-III, and four in degree IV coma. The duration of coma ranged from one hour to six days. Four patients had died, while in 16 the course of the illness was satisfctory. Sinus tachycardia was registered in 11 patients, atrial fibrillation in two, and in seven patients the rhythm was regular. Fifteen of the 20 patients (75%) had had the corrected Q-T interval prolonged with values of up to 0.664 seconds on admission, while in two patients the corrected Q-T interval became prolonged during hospitalization. Malignant dysrhythmia was registered in one patient. The relative risk of a prolonged corrected Q-T in patients with intoxication coma measured 87. The relative risk of malignant ventricular dysrhythmia in a group of coma patients amounted to 30.

Key words:

Intoxication, coma, corrected Q-T interval, malignant ventricular arrhythmia.

Introduction:

Changes in the circulatory system such as alterations in blood pressure, heart rate and heart rhythm may occur in a coma[1-3].

The Q-T interval, a standard electrocardiographic measurement, marks the electrical systole of the ventricle and the refractivity duration of the myocardium. With a prolonged Q-T interval the recovery of the conduction system varies. Consequently, an impulse may come across unequally repolarized myocardium and cause "re-entry" ventricular tachycardia and polymorphous ventricular tachycardia ("torsade de pointes") the result of which can be ventricular fibrillation[4-6]. Ventricular dysrhythmia may develop due to an imbalanced influence from the right and left cervicothoracal ganglia, i.e., reduced activity of the right or increased activity of the left stellate ganglion. However, the above dysrhythmias may appear without the presence of a change in the repolarized process[7].

The aim of our study was to analyze the electrocardiogram changes in acutely intoxicated pa-

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tients, particularly a prolonged Q-T interval, and the relative risk for ventricular dysrhythmias.

Patients and Methods:

A group of 20 consecutive patients admitted to the Department of Internal Medicine because of intoxication coma were studied. The following data were analyzed: age, sex, etiology of coma, time from intoxication to hospital admission, drug or poison concentration responsible for comatose state, degree of coma, electrocardiogram (constantly monitored), serum electrolyte values, blood count and acid base. The degree of the depth of coma was classified according to Reed's classification[8] in the following categories: degree 0 (unconscious but can answer questions and carry out simple suggestions, somnolent), degree I (responsive to painful stimulus), degree II (unreactive to painful stimulus with intact reflexes), degree III (most reflexes are absent but cardiorespiratory function maintained), and degree IV coma (deep coma, all reflexes absent, cardiorespiratory insufficiency, arterial hypotension, pulmonary oedema and shock may be present).

Corrected Q-T interval recorded in the II-lead ECG corrected to the heart frequency was determined according to the following equation[9,10]:

$$\text{corrected Q-T s} = \frac{\text{measured Q-T s}}{\sqrt{\text{R-R interval s}}}$$

The relative risk for a prolonged Q-T interval was also calculated[11]:

$$RR = \frac{a \times d}{b \times c}$$

The value is considered significant if it is higher than one. In the equation a signifies a prolonged corrected Q-T interval in patients with intoxication coma, b is the normal value of the corrected Q-T in patients with intoxication coma, c is a prolonged corrected Q-T interval in the control group, while d is the normal value of the corrected Q-T interval in the control group. Control group was selected at random from the register of our outpatient department, consisted 30 persons aged 32.4±24.1 year of both sexes—one have had prolonged corrected Q-T interval (3%).

Results:

A total of 20 patients with intoxication coma were studied. There were more males than females (12:8) and they were frequently younger than 30 years (26.7±9.8). The patients were between 16 and 80 years. Four of these 20 patients were in degree 0, five in degree I, two in degree II, five in degree III, while four patients were in the most serious comatose state—degree IV.

The majority of patients had been intoxicated by taking anxiolytics, ethanol and pesticides. Anxiolytics were taken mainly by women and alcohol and pesticides by men. Sedatives and hypnotics, anti-depressants and antiepileptics were all recorded twice as a cause of intoxication and both trichlorethylene and chlorpropamide once.

Table 1 shows the changes in the electrocardiogram on admission. Sinus tachycardia was present in 11 patients, sinus bradycardia was once registered, and atrial fibrillation was permanently present in two patients. The flat T wave was registered in three, inverted T wave up to 1 mm in one, S-T segment depression of descending type in two and polymorphous ventricular premature beats of approximately 25 per cent frequency in one patient (intoxication with ethanol and trichlor ethylene) that had been preceded the previous day by ventricular paroxysmal tachycardia with a prolonged corrected Q-T interval. A prolonged corrected Q-T interval was present in 5 women and 10 men.

Nine of 13 patients with metabolic acidosis on admission had a prolonged corrected Q-T interval. Four of five patients with hypoxia and a prolonged corrected Q-T interval. No correlation was found between serum potassium values and the corrected Q-T interval. In one patient, autopsy results revealed bleeding esophageal varies as cause of death, hepatic cirrhosis, aspiration of hemorrhagic content and 0.7 per cent ethanol. In two patients massive bronchopneumonic infiltrates were present as a complication of coma, and in the third fe-

male patient tuberculosis pneumonia was present. The patient intoxicated with ethanol and trichlorethylene had a fatal bradycardia as well as respiratory arrest and sudden death was not the result of ventricular dysrhythmia.

Table 1.

Changes in the electrocardiogram in intoxication coma

	ECG finding			
Sex	Sinus rhythm	Sinus tachycardia	Atrial fibrillation	Prolonged corrected Q-T interval
Males	5	6	1	10
Females	2	5	1	5
Total N = 20	7	11	2	15

A relative risk in patients with intoxication coma who had a prolonged corrected Q-T interval was 87. Among 15 patients with intoxication coma, the relative risk of malignant ventricular dysrhythmia amounted to 30.

Discussion:

A prolonged corrected Q-T interval in ECG was found in three-quarters of the 20 patients with intoxication coma. In six patients who had taken phenothiazine, tricyclic antidepressants or organophosphates, the corrected Q-T interval was prolonged. It is well known that this parameter may be prolonged with these drugs. However, no significant difference was found in these patients in relation to the group with a prolonged corrected Q-T interval intoxicated with other substances. In the same way, no correlation between a prolonged corrected Q-T interval and serum potassium concentration, metabolic acidosis, partial oxygen pressure which does not indicate metabolic phenomenon as the reason for the above ECG changes was found. According to some authors[8] the adrenergic mechanism has an important role in the occurrence of ventricular paroxysmal tachycardia in coma. Early changes possibly occur because of sympathetic stimulation (rarely, parasympathetic) due to an increase in the intracranial pressure, and later probably due to the secretion of catecholamines[12,15].

In 15 patients with a prolonged corrected Q-T interval, this parameter measured as much as 0.664 sec. In one patients together with the corrected Q-T interval of 0.491 sec. ventricular paroxysmal tachycardia was casued by coma and was treated successfully.

Seventyfive per cent of the patients with intoxication coma had a prolonged corrected Q-T interval in our study with a high relative risk of 87.

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