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THE EFFECTS OF EXPERIMENTAL ACUTE PETROL VAPOUR INTOXICATION (ETHYLINE 78) ON THE CIRCULATORY SYSTEM IN RABBITS

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Studies were performed on the effects of an experimental acute motor-car petrol (ethyl petrol 78) vapour intoxication on the circulatory system in rabbits. Some rabbits were kept for two hours in a specially constructed inhalation chamber in which they inhaled the air containing petrol vapour of an average concentration of 310 mg/l. The recorded electrocardiogram showed a slowed-down heart rate, prolonged QT interval, disturbed repolarization period of the ventricles, and disturbed intraventricular conductivity.

The statistically significant decrease in the sodium, potassium and magnesium levels found in the homogenates of the heart proves that the disturbances of the repolarization period of ventricles are due to electrolytic disturbances. Coexistant muscle lesion however cannot be excluded. A decrease in the histochemical activity of alkaline phosphatase in the heart muscle suggests the possibility of disturbances in the active transport. An increase in the ATP-ase reaction in the vessel endothelia of the heart muscle was also observed.

A statistically significant decrease in $\alpha_1 + \alpha_2$ globulins in the blood serum and an increase in the acid phosphatase caused most probably by its liberation from the tissue were also found as was a decrease in the activity of acid phosphatase in the heart muscle homogenates.

Modern civilization and its further development depends to a great extent on mineral oil products. Among these motor-car petrol plays a very important part. It has been widely used as engine fuel and organic solvent and has becomc quite indispensable in everyday life. The toxicity of various petrols is conditioned by the percentage content of paraf-

fin hydrocarbons (mainly pentane, hexane, heptane, octane and nonane) and contamination with sulphur and nitrogen compounds. The toxicity grows parallelly with the amount of unsaturated and aromatic hydrocarbons (5, 9, 26). Certain kinds of petrol contain ethyl fluid (motocar petrol: 0.1–0.15%) whose main component – tetraaethyl lead – acts as an antiknock agent (10, 14, 18).

The present development of chemical industry and a growing use of motor vehicles make the problem of petrol toxicity an up-to-date topic of utmost importance.

In acute cases of petrol vapour intoxication cyanosis and pulse irregularity were found (3). Sudden death record may be due to a paralysis of the respiratory centre or to a noxious effect on the circulatory system (16). Hydrocarbons present in petrol make the heart muscle more sensitive to the epinephrine circulating in the blood producing in ventricular fibrillation (8). Electrocardiographic changes were observed in subacute (27) and acute (1) intoxications. As chronic effects of small concentrations appeared hypotension, bradycardia and disturbances of the liver (6, 15, 25). In chronic petrol vapour intoxication of rats (22) the following symptoms were found: thickening of the arterial walls, loss of the normal pattern of the muscular coat, hyperplasia of the internal membrane, multiplication of the endothelia, parenchymatous degeneration of the heart muscle. The harmful effect of petrol vapour on the circulatory system is quite evident, but the mechanism of its action has not yet been explained. For this very reason this study has been carried out.

MATERIAL AND METHOD

Experiments were performed on 35 one-year old rabbits of mixed strain and both sexes, weighing 2.500–3.150 g. All rats were fed the same standard diet 3 months prior to the experiment and during the experiment. Twenty rabbits were poisoned with petrol vapour (ethyl petrol 78) while the remaining 15 served as a control group for biochemical and histochemical examinations.

The animals subjected to intoxication were kept in a chamber (Fig. 1) in which they breathed the air containing petrol vapour of an average concentration of 310 mg/l for two hours. This petrol vapour concentration was maintained constant by means of a glass beaker placed into the chamber, 12 cm in diameter, containing 500 ml of petrol. The petrol was kept at 38° C by warm water circulating through an immersed metal coil connected to the ultrathermostat with a rubber tube. The air was blown over the surface of the evaporating petrol by means of an electric blower of 600 l/h capacity. To ensure a constant flow and a uniform distribution of the mixture the chamber had an outlet. The concentration of the vapour was measured every 10 minutes by reading the decrements

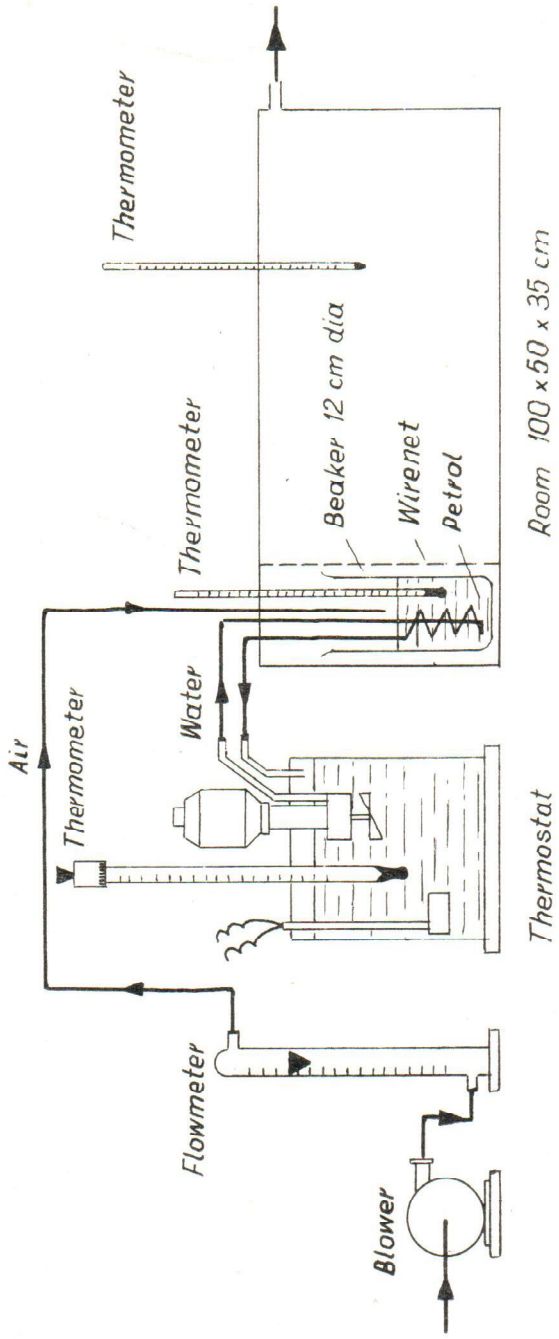


Fig. 1. *Exposure chamber to petrol vapour*

of petrol volume and transforming them into weight units which – knowing the capacity of the chamber and the flow-rate – could be further expressed in concentration units (mg/l) (Fig. 2).

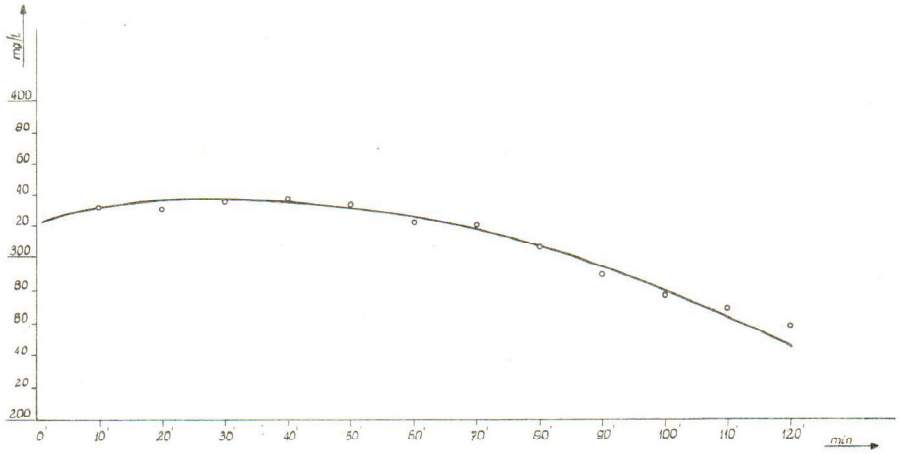


Fig. 2. *Concentration of motor-car petrol vapour in the exposure chamber*

The concentration of tetraethyl lead was determined by the method used by *Klichowski* (11). The petrol vapour-air mixture leaving the chamber was filtered through the glass wool moistened with nitric acid. Through the chemical reaction tetraethyl lead was transformed into lead nitrate $Pb(NO_3)_2$. After washing the filter with hot water the liquid was evaporated and the dry remainder was dissolved in a small volume of water. In the obtained $Pb(NO_3)_2$ solution the lead content was determined by means of the dithizone method (4).

1. Three days prior to intoxication all rabbits were subjected to electrocardiographic examination under evipan anaesthesia using needle electrodes. The animals were lying on their right side and were earthed through the skin of the right leg. On the average 25 mg of evipan per kg of body weight was injected into the marginal auricular vein (acc. to 12).

Three-channel Hellige Multiscriptor 9400 T electrocardiograph and bipolar extremity leads I, II, III were used in the experiment. The examinations had to be carried out at the paper velocity of 100 mm/sec because of high heart rate of rabbits. Taking into account the low voltage of the deflections the amplification 15 mm = 1 mV mark was used.

Immediately after intoxication, electrocardiographic examinations were repeated in a similar way except that no evipan narcosis was needed. The blood for biochemical examinations in the control and intoxicated group was taken from the central auricular artery and the rabbits

were decapitated. Heart tissue specimens were taken at dissection and homogenized in the routine way.

2. In the blood of both groups of animals the following parameters were determined:

a) the total protein level by means of the biuret test and each particular protein fraction by means of paper electrophoresis using veronal buffer with pH = 8.6. The separation lasted for 7 hours at 300 V. The strips were stained with bromphenol blue solution, cut into separate fractions, lixiviated in 8.4 per cent NaOH and the values estimated in a spectronic – 20 spectrophotometer. Since some proteinograms gave unsatisfactory separation α_1 and α_2 fractions were estimated jointly.

b) the lipoprotein fractions by means of paper electrophoresis, at 225 V, veronal buffer pH = 8.4, separation time 9 hours. The strips were stained with the alcohol solution of Sudan black according to *Swahn* (21) and read on a Pulfrich photometer. The percentage ratio of lipoproteins was calculated.

c) the total serum cholesterol according to *Turner* and *Ealls* (23).

d) the activity of alkaline and acid phosphatase in the blood serum according to *Bodansky* (2). The amount of inorganic phosphorus liberated enzymatically from the substrate was determined in MME units (Milimoleinheiten MME). The enzymatically liberated phosphorus was determined according to *Fiske* and *Subbarow* (7).

e) the activity of glutamic-oxalacetic transaminase (SGOT) and glutamic – pyruvic transaminase (SGPT) in the blood serum according to *Reitman* and *Frankel* (19).

f) sodium and potassium levels in the blood serum with the use of Zeiss III flame photometer at the air pressure of 0.4 atm. and acetylene pressure of 36 mm water. Calcium and magnesium levels were determined according to *Kovacs* and *Tarnoky* (13).

3. In heart muscle homogenates the following determinations were made:

a) the activity of acid and alkaline phosphatase as in 2 d.

b) the levels of sodium, potassium, calcium and magnesium as in 2 f. Phosphatase activity was expressed per mg of complete homogenate protein nitrogen of the respective tissue and electrolyte levels per mg protein nitrogen of the supernatant fluid.

4. The specimens for histochemical examination were always taken from the same region of the heart apex. The dissection material was fixed in cooled fixing fluid (acetone and Baker's fluid), cut on the microtome into 6 μ or 15 μ sections, and placed on albuminated slides for histochemical examination. The part of the unfixed dissection material was cut in a cryostat into 15 μ sections succinic acid dehydrogenase reaction. Control incubation was used for all reactions. Each section was stained with hematoxylin and eosine. The following enzymes were determined histochemically:

1) succinic acid dehydrogenase according to *Pears* (17);

2) ATP-ase and pentanucleotidase according to *Wachstein* and *Meisel* (24);

3) acid and alkaline phosphatases according to *Gomori* (17).

EXAMINATION RESULTS

All exposed rabbits had periods of restlessness, disturbances of equilibrium, convulsions, and fell into deep narcotic sleep after about 35 min.

I. *Electrocardiographic examinations*

The initial electrocardiographic record taken prior to intoxication showed no abnormalities. The electrocardiograms of all affected rabbits (Table 1) showed the slowing-down (on the average by 96 min) and disturbances of the ventricular repolarization period such as: flattening of T in ten (Fig. 3), inversion of T in seven (Fig. 4), biphasic T in 3 and ST depression in 10 rabbits (Fig. 5). The depression of the ST section ranged from 1 to 6 mV. The QT interval was prolonged in respect to normal values based on formula by *Bazett*, *Hegglin* and *Holzman* ($QT = K\sqrt{R} - R \pm 0.0.1$ at $K = 0.01$ at $K = 0.304$) in 16 animals. The prolongation of the = QRS complex above 0.03 sec. was observed in 7 rabbits.

II. *Results of biochemical examinations*

Table 2 shows the values of total serum protein and its fractions in animals exposed to petrol vapour. A small decrease in the level of total blood serum protein was due to a decrease in the albumin level. The level of globulins was similar in both groups of rabbits. In certain globulin fractions of poisoned rabbits a statistically significant decrease of $\alpha_1 + \alpha_2$ fractions and a nonsignificant increase of B fraction were observed. No distinct change was observed in β globulin fraction. Within lipoprotein fraction there was a statistically nonsignificant decrease of high density α fraction (Table 3) and a nonsignificant increase of β and γ fractions. The level of cholesterol in affected rabbits did not differ distinctly from that of the control group (Table 4). Both the SGOT and SGPT activities showed a slight increase which however was not statistically significant (Table 5). The poisoning with petrol vapour brought about a statistically significant increase of acid phosphatase activity and a nonsignificant increase of alkaline phosphatase activity in the blood serum (Table 6).

The levels of magnesium and calcium in the blood serum were similar in both groups. A decreased sodium level and increased potassium level in the blood serum were observed in intoxicated rabbits. These differences, however, were not statistically significant (Table 7). A statistical-

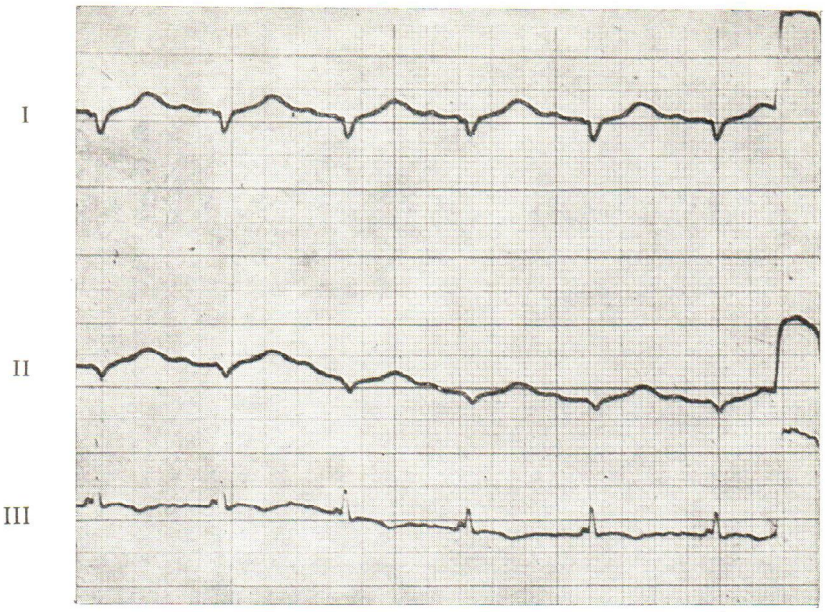
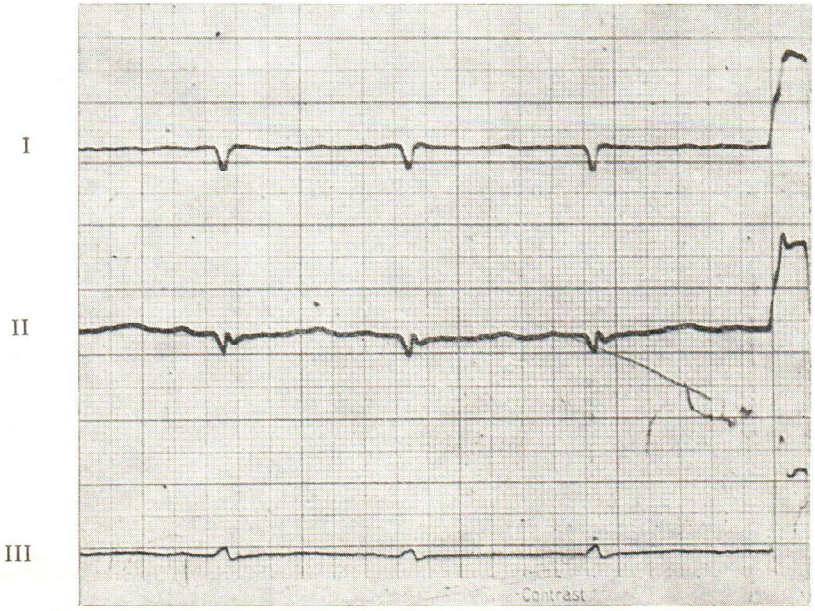


Fig. 3. *Curve A: electrocardiogram taken before intoxication. Heart rate 316/min.*



Curve B: electrocardiogram taken 2 hours after intoxication by petrol vapour. Heart rate 207/min., QS elongation, flat T wave in the leads I and II

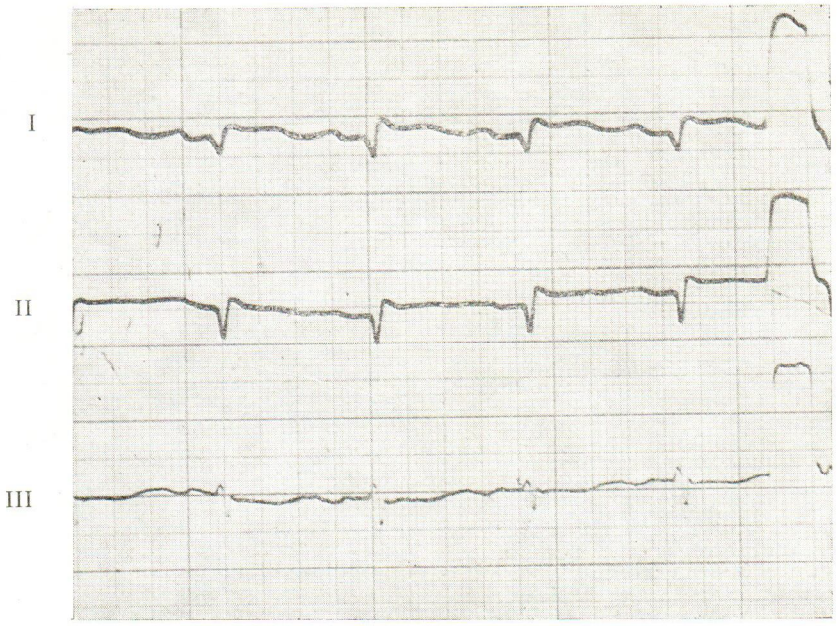
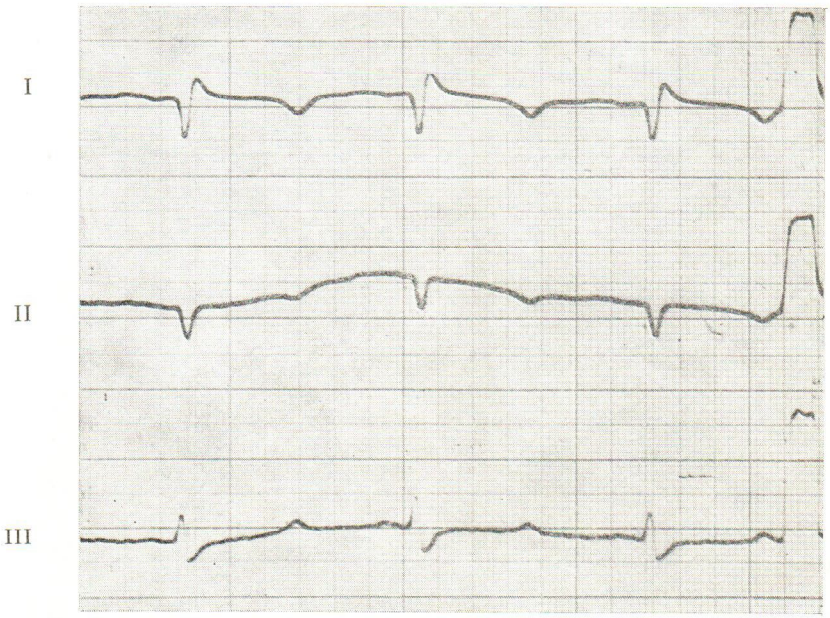


Fig. 4. *Curve A: electrocardiogram taken before the experiment. Heart rate 300/min*



Curve B: electrocardiogram after intoxication by petrol vapour. Heart rate 176/min. QS and QRS elongation, negative T wave in the leads I and II

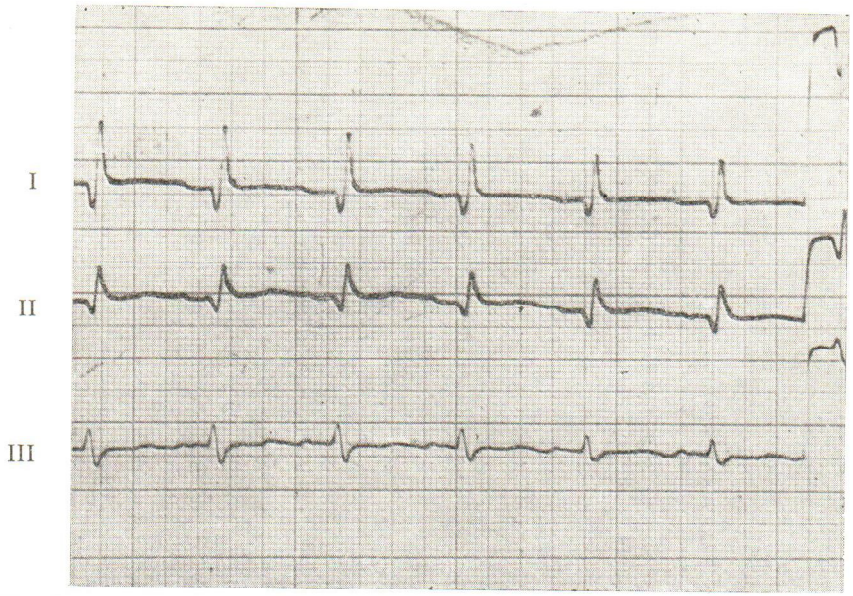
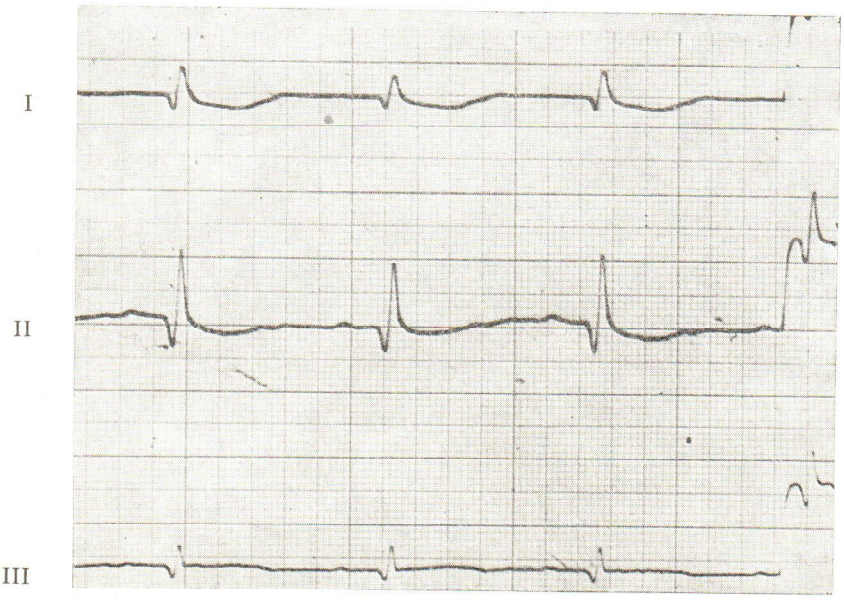


Fig. 5. *Curve A: electrocardiogram taken before intoxication. Heart rate 316/min.*



Curve B: electrocardiogram taken after intoxication. Heart rate 187/min. Sinking ST interval, in the leads I and II, flat wave in the lead III

Table 1

Features of electrocardiographic curve in examined rabbits

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Decreased heart rate	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
P wave																				
P-Q interval																				
QT interval		+	+	+	+	+		+		+	+	+		+	+	+	+	+	+	+
QRS Complex		+				+			+			+		+		+		+		
ST segment		+		+		+			+			+	+	+		+		+		
T wave: flat	+		+	+			+	+		+			+			+		+		+
negative		+			+	+					+			+			+		+	
biphasic									+			+			+					
Pathological angle of electric axis																				

Table 2

Statistically significant changes in serum protein levels in healthy and poisoned rabbits

Type of protein (g %)	Mean physiological values, 15 control rabbits	Mean values after 2 hours of poisoning, 20 rabbits	Difference between arithmetic means	*t*-test	Statistical significance α
Total protein	6,4 ± 0,77	6,1 ± 0,63	0,3	1,2	nonsignificant
Albumins	4,05 ± 0,94	3,71 ± 0,64	0,34	1,1	nonsignificant
Globulins	2,35 ± 0,37	2,39 ± 0,62	0,04	0,25	nonsignificant
α_1 and α_2 globulins	0,73 ± 0,20	0,66 ± 0,22	0,07	2,9	$< 0{,}01$
β-globulins	0,66 ± 0,16	0,75 ± 0,21	0,09	1,29	nonsignificant
γ-globulins	0,96 ± 0,25	0,96 ± 0,42	0,02	0,16	nonsignificant

Table 3

Serum lipoprotein fractions in healthy and poisoned rabbits

	Mean physiological values, 15 control rabbits	Mean values after 2 hours of poisoning, 20 rabbits	Difference between arithmetic means	*t*-test	Statistical significance α
Lipoprotein α	32,4 ± 10	26,8 ± 7,9	5,6	1,64	nonsignificant
Lipoprotein β	35,5 ± 9,3	36,2 ± 10,6	0,7	0,18	nonsignificant
Lipoprotein γ	32,2 ± 7,2	34,4 ± 8,6	2,2	0,76	nonsignificant

Table 4

Total cholesterol levels in the serum of healthy and poisoned rabbits

Rabbits	Number of rabbits	Arithmetic mean M mg %	Standard deviation	Difference between starting mean and mean in the experiment $m_1 - m_2$	*t*-test	Statistical significance α
Controls	15	107,4	35,1	7,8	0,23	nonsignificant
Poisoned	20	99,6	45,6			

Table 5

SGOT and SGPT activity in the sera of healthy and poisoned rabbits

	SGOT		SGPT	
	Control rabbits	Poisoned	Control rabbits	Poisoned
Number of rabbits	15	20	15	20
Arithmetic mean	29,4	34,5	44,0	52,1
Standard deviation	15,2	23,4	20,7	25,1
Difference between starting mean and mean in the experiment $m_1 - m_2$	5,1		8,1	
t-test	0,25		0,41	
Statistical significance α	nonsignificant		nonsignificant	

Table 6

Acid and alkaline phosphatase activity in the sera of healthy and poisoned rabbits (expressed as MME)

	Acid phosphatase		Alkaline phosphatase	
	Control rabbits	Poisoned	Control rabbits	Poisoned
Number of rabbits	15	20	15	20
Arithmetic mean	1,1	1,5	2,0	2,2
Standard deviation	0,37	0,30	0,58	0,77
Difference between starting mean and mean in the experiment $m_1 - m_2$	0,4		0,2	
t-test	3,3		0,8	
Statistical significance α	$< 0{,}01$		nonsignificant	

Table 7

Electrolyte levels in the sera of healthy and poisoned rabbits

	Na mEqu/l		K mEqu/l		Mg mEqu/l		Ca mEqu/l	
	Control rabbits	Poisoned	Control rabbits	Poisoned	Control rabbits	Poisoned	Control rabbits	Poisoned
Number of rabbits	15	20	15	20	15	20	15	20
Arithmetic mean m	160,7	156,2	4,59	4,86	3,1	3,0	6,4	6,3
Standard deviation	10,0	11,3	0,27	1,3	0,014	0,014	0,74	0,74
Difference between starting mean and mean in the experiment $m_1 - m_2$	4,5		0,27		0,1		0,1	
t-test	0,19		0,30		0,71		0,33	
Statistical significance α	nonsignificant		nonsignificant		nonsignificant		nonsignificant	

Table 8

Acid and alkaline phosphatase activities in heart muscle homogenates calculated per milligramme of tissue nitrogen of healthy and poisoned rabbits

	Acid phosphatase		Alkaline phosphatase	
	Control rabbits	Poisoned	Control rabbits	Poisoned
Number of rabbits	15	20	15	20
Arithmetic mean	0,38	0,19	0,42	0.28
Standard deviation	0,14	0,09	0,26	0,18
Difference between starting mean and mean in the experiment $m_1 - m_2$	0,19		0,14	
t-test	4,7		1,8	
Statistical significance α	$< 0{,}01$		$< 0{,}1$	

Table 9

Electrolyte levels in heart muscle homogenates calculated per milligramme of tissue nitrogen of healthy and poisoned rabbits

	Na mg		K mg		Mg mg		Ca mg	
	Control rabbits	Poisoned	Control rabbits	Poisoned	Control rabbits	Poisoned	Control rabbits	Poisoned
Number of rabbits	15	20	15	20	15	20	15	20
Arithmetic mean	0,086	0,043	0,166	0,079	0,0129	0,0049	0,098	0,093
Standard deviation	0,035	0,048	0,040	0,052	0,0039	0,0022	0,0049	0,0027
Difference between starting mean and mean in the experiment $m_1 - m_2$	0,043		0,087		0,008		0,0005	
t-test	3,1		5,4		7,2		0,35	
Statistical significance α	$< 0,01$		$< 0,01$		$< 0,01$		nonsignificant	

ly significant decrease of acid phosphatase activity and a statistically nonsignificant decrease of alkaline phosphatase were observed in the homogenised tissue (Table 8).

The intoxication also caused a decrease in the level of sodium, potassium and magnesium in the homogenates of the heart. These differences were statistically significant. Calcium levels were similar in both groups (Table 9).

III. *Results of histochemical examinations*

Heart

Succinic acid dehydrogenase. The reactions were strong, particularly in the myocardium. Much weaker reactions were observed in the epicardium. No distinct changes in the intensity of the reaction were observed in the intoxicated rabbits in comparison with the control group.

5-nucleoidase. A weak reaction was observed only in some endothelial cells of blood vessels. No distinct difference in the reaction intensity was found in the intoxicated rabbits.

ATP-ase. It was present in the vessel endothelia of the heart muscle within the inlays, cell membranes and in sarcolemma (Fig. 6). As far as the intoxicated rabbits are concerned, the intensity of the reaction increased only in the vessel endothelia. No change was found in other structures (Fig. 7).

Alkaline phosphatase. Positive reaction was found in the cell membrane and blood vessel endothelia (Fig. 8).

As far as the intoxicated rabbits are concerned, only a slight decrease in the reaction intensity was found (Fig. 9).

Acid phosphatase. The reactions in the heart muscle were negative in both groups.

DISCUSSION

The slowing-down of the heart rate shown in the electrocardiograms of intoxicated rabbits occurred as a consequence of vegetative disturbances. Disturbed intracellular conduction observed in 7 cases was due to the noxious effect of petrol vapour upon the conductive system of the heart. The observed changes in the repolarization period of ventricles could be explained as a result of electrolytic disturbances because a decrease in the level of Na^+ K^+ and Mg^{++} was found in the homogenates of the heart. The changes of the ST–T segment in potassium deficiency are generally known. *Seta* et al. (20) found that in dogs with the deficiency of both K^+ and Mg^{++} in the diet diphasic deep negative T wave occurs. The negative T observed in my experiments only in 7 and diphasic T in 3 poisoned rabbits might be accounted for by a lower degree of electrolytic disturbances.

In an experimental acute petrol vapour intoxication in guinea-pigs *Di Blasi* et al. (1) found rhythm disturbances besides the changes of ST-T segment. The absence of rhythm disturbances in my experiments

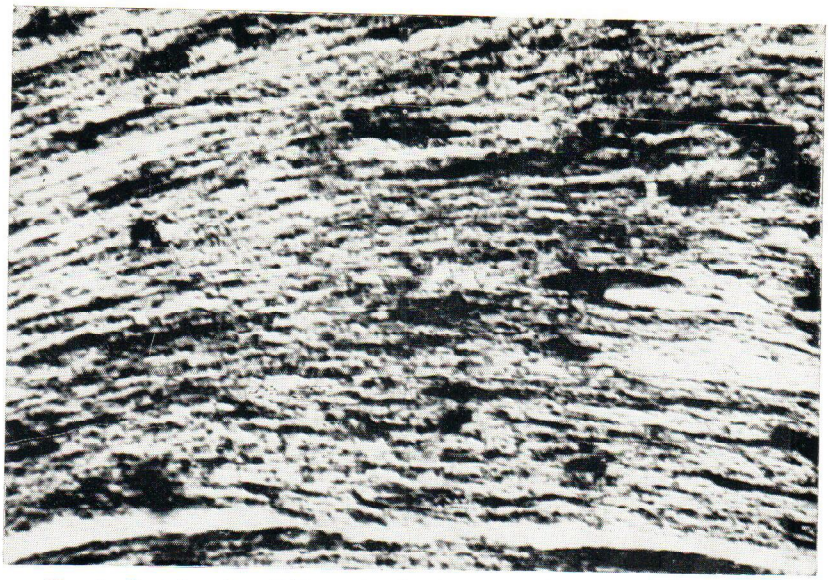


Fig. 6. *Reaction for ATP-ase in the heart muscle of control animals. Incubation 30 min. Magn. about 300 ×*

Fig. 7. *Increased ATP-ase reaction in vessel endothelia in poisoned animals. Incubation 30 min. Magn. about 300 ×*

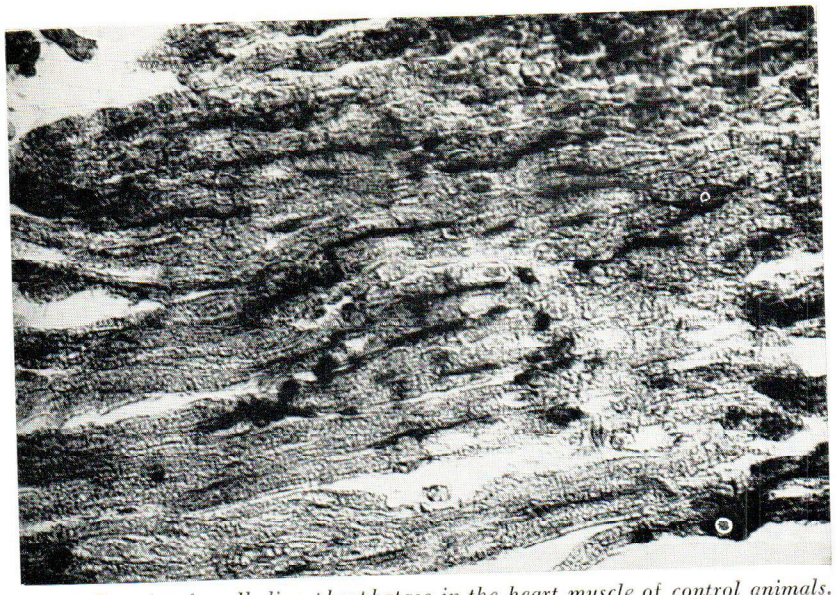


Fig. 8 *Reaction for alkaline phosphatase in the heart muscle of control animals. Incubation 120 min. Magn. about 300 ×*

Fig. 9. *Slight decrease in the reaction intensity for alkaline phosphatase in poisoned animals. Incubation 120 min. Magn. about 300 ×*

might be due to the species differences. *Di Blasi* provided no details of the technique of their experiment. The observed rhythm disturbances might have been due to the effects of CO_2 inhaled by animals in lack of continuous flow of air in the chamber. Partial improvement of ECG in II and III leads was obtained owing to the administration of 0.15 ml of 0.3% potassium chloride solution per kg of body weight into the marginal auricular vein. The absence of the total recovery of ST–T section suggests muscle changes or effects of multielectrolytic disturbances. The decrease of alkaline phosphatase reaction in the cell membrane and heart vessel endothelia might suggest the possibility of disturbances in the transport of substrates from the blood to the sarcoplasm. The ATP-ase increase in the vessel endothelia is probably the result of increased transport of Na^+ ions from the tissue into the blood circulation.

Decreased acid phosphatase level in homogenates of the heart and possibly in other organs is most probably the result of its liberation into the circulation. This may also be the cause of its increase in the blood serum. The observed statistically significant decrease in $\alpha_1 + \alpha_2$ fractions of blood serum globulin might be explained by the disturbances in the tissue metabolism, though a direct effect of petrol vapour on serum proteins cannot be excluded. The absence of statistically significant changes in the remaining fractions of the proteinogram, lipidogram as well as in the level of electrolytes, cholesterol and in the activity of SGOT and SGPT in the blood serum may be due to a very short period of exposure.

Analysis of the petrol vapour used in the experiment showed rather low concentrations of tetraethyl lead (up to 0.22 γ Pb/l of air). This is compatible with the data on the low volatility of tetraethyl lead (10) and shows that in cases of acute petrol intoxication the volatile hydrocarbons are the toxic agents. The results discussed above suggest that in experimental acute petrol vapour intoxication the activity of some enzymes in the heart muscle is changed and electrolytic disturbances occur which result in the changes of electrocardiogram.

It seems possible that the treatment of electrolytic disturbances brought about by acute petrol vapour intoxication may prevent circulatory complications.

CONCLUSIONS

1. The acute petrol vapour intoxication in rabbits brings about the following changes in the electrocardiogram: slowing-down of the heart rate, prolongation of the QT interval, disturbances of the repolarization period of ventricles and disturbances of intraventricular conductivity.
2. The observed changes of the electrocardiogram are caused by electrolytic (decreased level of sodium, potassium and magnesium in the homogenates of the heart) and vegetative disturbances and by direct effects of petrol vapour on the heart conduction system.

3. An acute petrol vapour intoxication causes a decrease in the level of $\alpha_1 + \alpha_2$ globulin in the blood serum and an increase in the activity of acid phosphatase.

4. The histochemical methods showed a change in the activity of acid phosphatase and ATP-ase in the heart muscle.

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Sažetak

DJELOVANJE EKSPERIMENTALNOG AKUTNOG OTROVANJA BENZINOM (ETHYLINE 78) NA CIRKULATORNI SISTEM KUNIĆA

Elektrokardiografsko ispitivanje kunića akutno otrovanih parama benzina pokazalo je ove promjene: usporenje srčane akcije, produženi P-Q interval, poremećenje vremena repolarizacije ventrikula i poremećenje intraventrikularne provodljivosti. U homogeniziranom tkivu srca nađeno je statistički značajno smanjenje natrija, kalija i magnezija. Akutno otrovanje parama benzina uzrokuje sniženje nivoa alfa 1 i alfa 2 globulina u serumu i povećava aktivnost kisele fosfataze. Histokemijske metode pokazale su promjene u aktivnosti kisele fosfataze i ATP-aze u srčanom mišiću.

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