

EFFECT OF COWDUNG SMOKE INHALATION ON E.C.G. OF RATS

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Summary : The present study was conducted on 20 adult albino rats divided into group I (n=10) and group II (n=10). Group I animals were exposed to cowdung smoke for 60 min twice a day for a period of 75 days whereas group II served as control. ECG of control rats was essentially similar to that of man. ST segment was absent, Q wave absent or rudimentary and the mean QRS axis $+ 45^\circ$. Rats exposed to cowdung smoke revealed a significant increase in PR interval and a mean QRS of $+ 117.5^\circ$. Our results indicate that inhalation of cowdung smoke leads to conduction defects and right axis deviation.

Key words : cowdung smoke

ECG

right axis deviation

INTRODUCTION

In rural India, respiratory disorders are common even though there is negligible atmospheric pollution by industries and automobiles. Moreover, rural non-smoker females also show a high incidence of bronchitis and chronic cor pulmonale (9, 12). This has been attributed to the exposure of the villagers to cowdung smoke while using dry cowdung as a fuel for cooking and warming their ill ventilated houses (9, 12). In spite of widespread exposure of rural population of India to cowdung smoke, the work on its pathophysiological effects has been negligible. In an earlier work (10) we have shown that rats exposed to cowdung smoke develop bronchitis, bronchiolitis, emphysema and atelectasis. In the present paper, we report the electrocardiographic changes in rats exposed to cowdung smoke.

MATERIALS AND METHODS

Healthy adult albino rats (200 ± 26 g) of either sex were randomly divided into group I (n=10) and group II (n=10). Group I animals were exposed to cowdung smoke

in an exposure chamber as described earlier (10). The smoke was produced by burning 350 g dry cowdung cake in smoking chamber. With the help of a respiratory pump, the smoke was delivered into the exposure chamber at the rate of 2.5 l/min. Simultaneously, atmospheric air was pumped at the rate of 2 l/min to avoid asphyxia. The animals were exposed to the smoke for 60 min twice a day for a period of 75 days. Group II (control) rats were kept in the exposure chamber in the same way as group I animals, but they were not exposed to the smoke.

At the end of the study period, ECG was recorded 30 min after anesthetizing the rats with ip injection of nembutal sodium (4 mg/100 g). The animals were fixed in supine position with the limbs gently extended. The forelimbs were kept at right angles to the body. Electrodes consisted of 26 gauge hypodermic needles placed subcutaneously for 1 cm. Tracings of leads I, II, III, aVR, aVL, aVF and precordial leads corresponding in position to conventional leads V₁, V₃ and V₆ were recorded on Grass Model 7 Polygraph at a paper speed of 100 mm/sec and standard of 20 mm/mV. Heart rate (HR) was determined from the RR interval and mean electrical axis for QRS from the hexaxial reference system. Amplitudes were calculated in mV and durations in msec. Corrected QT duration was calculated from the formula:

$$QTc = QT (\text{sec}) / \sqrt{RR (\text{sec})}$$

RESULTS AND DISCUSSION

The results are presented in Fig. 1 and Tables I and II.

Electrocardiograms were distinct in resolution and serial records taken from the same animal were readily reproduced. Lead III tracings were clear and not indefinitely defined as reported by Normann *et al.* (8). Earlier workers (1, 3, 5) have used ether anesthesia for recording rat ECG. We preferred nembutal because ether produces disorders of cardiac rate and rhythm as rat's upper respiratory tract is sensitive to ether (6). The amplitudes and durations recorded in lead II are given in Tables I and II.

ECG of control rats : Our control rats had a mean HR of 456.8/min which is close to our earlier observations (7). Other workers (1, 3) have reported a lower HR while recording ECG of rats under ether anesthesia. Ether produces bradycardia while nembutal increases the HR (6). The mean duration of P wave was 20.3 msec and this is in agreement with the observations of Beinfield and Lehr (2). PR interval of 36.1 msec is much lower than the findings of Fraser *et al.* (3), but close to the value of Kela *et al.* (5). The mean QRS and QT intervals were 23.3 msec and 60.4 msec respectively.

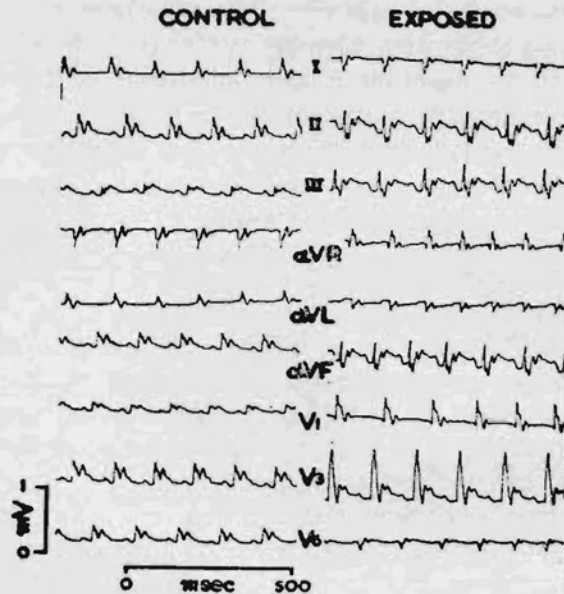


Fig. 1 : Electrocardiographic tracings of control and cowdung smoke exposed rats.
Paper speed : 100 mm/sec.

Although these values are different from those reported by Beinfield and Lehr (1), they are close to the observations of Fraser *et al.* (3). According to Normann *et al.* (8), accurate measurement of QRS duration is not possible, but our records did not present such a difficulty. Mean QTc of 0.165 is in agreement with an earlier report (3). The mean amplitude of P wave was 0.065 mV. This is in agreement with the observations of Kela *et al.* (5) but different from the values reported by Normann *et al.* (8), and Beinfield & Lehr (2). The amplitudes of QRS, R and S waves were 0.343 mV, 0.321 mV and 0.022 mV respectively and these values are comparable to those reported earlier (5). Since Q wave was absent or very small in standard limb leads (Fig. 1) it was not measured. T wave amplitude of 0.190 mV is in agreement with the findings of Beinfield and Lehr (1). Rat ECG is characterised by the absence of ST segment (2, 11). In the present study also, there was no ST segment in any of the leads (Fig. 1). According to Sambhi and White (11) the TP segment is frequently absent in those leads where the T wave is prominent even when the HR is exceptionally slow. But in our records, TP segment was well discernible even in presence of prominent T waves and high HR. The average QRS axis was $+45.0^\circ$. This is in close agreement with earlier reports (1, 3, 8) and shows that the anatomical orientation of rat heart is similar to that of man.

TABLE I : Electrocardiographic data (lead II) of control and cowdung smoke exposed rats.

	<i>P</i> (msec)	<i>PR</i> (msec)	<i>QRS</i> (msec)	<i>QT</i> (msec)	<i>QTc</i>	<i>HR</i> (beats/min)
Control (10)	20.3 ±1.63	36.1 2.84	23.3 5.77	60.4 4.83	0.165 0.010	456.8 40.1
Exposed (10)	23.0 ±3.4	40.7 5.73	21.5 5.54	61.7 3.94	0.174 0.011	483.0 49.6
P value*	<0.05	<0.05	NS	NS	NS	NS

Values are mean ± SD

Numbers in parentheses denote the number of animals.

*Groups were compared by students' 't' test. NS : Not significant.

TABLE II : Electrocardiographic data (lead II) of control and cowdung smoke exposed rats.

	<i>P</i> (mV)	<i>QRS</i> (mV)	<i>R</i> (mV)	<i>S</i> (mV)	<i>T</i> (mV)	<i>Axis</i> (degrees)
Control (10)	0.065 ±0.008	0.343 0.061	0.321 0.056	0.022 0.026	0.190 0.042	45.0 10.2
Exposed (10)	0.077 ±0.024	0.390 0.109	0.289 0.081	0.101 0.057	0.165 0.062	117.5 35.7
P values*	NS	NS	NS	<0.001	NS	<0.001

Values are mean ± SD

Numbers in parentheses denote the number of animals.

*Groups were compared by students' 't' test. NS : Not significant

ECG changes in exposed rats : In rats exposed to cowdung smoke, the amplitudes of P, QRS and T waves were not significantly different from the control rats. Mean QRS and QT intervals were very close to those of control animals. QTc and HR were insignificantly higher than the control values. There was statistically significant increase in PR interval suggesting an increase in atrio-ventricular conduction time. The amplitude of S wave in lead II was significantly more as compared to the control group. Fig. 1 shows predominant negativity in lead I and predominant positivity in aVR in exposed rat. Pre-cordial leads show predominant positivity (tall R) in V₁ with S wave persisting in V₆. Q wave which is absent or rudimentary in normal rat ECG (8, 11) was prominent in some of the exposed rats (Fig. 1, aVR) suggesting myocardial strain. Since there is no true

ST segment in rat ECG (2, 11), changes in this segment are not expected in our exposed rats.

These electrocardiographic features and a mean QRS axis of $\pm 117.5^\circ$ suggest that our rats exposed to cowdung smoke had developed chronic cor pulmonale. Heavy cigarette smoking is known to be associated with emphysema (4). In an earlier study (10), we have found that rats exposed to cowdung smoke develop bronchitis, bronchiolitis and emphysema as a result of inflammatory reaction to toxic constituents of smoke. A decrease in pulmonary surfactant activity resulting in an increase in the alveolar surface tension was also observed (10). This condition is known to result in collapse of the alveoli and pulmonary edema. Other workers have reported that females in Northern India have greater incidence of chronic bronchitis and chronic cor pulmonale even if they are non-smokers (9, 12). Since the females are directly exposed to cowdung smoke while using dry cowdung as a fuel for cooking, they are prone to develop pulmonary changes and chronic cor pulmonale. The pulmonary changes observed in our earlier study (10) and ECG changes observed in the present study corroborate with the clinical observations of Padmavati (9) and Wig *et al.* (12) that cowdung smoke could be one of the factors responsible for chronic bronchitis, emphysema and chronic cor pulmonale in the rural population of North India where dry cowdung cakes are commonly used for cooking purposes.

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