

CIGARETTE SMOKING AND PRESSURE-VOLUME CHARACTERISTICS OF THE LUNG

S. WALTER

Department of Physiology,
Christian Medical College,
Vellore - 632 002

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Abstract: This study was done to identify the immediate changes induced by cigarette smoking on the mechanical properties of the lung. Static compliance (C stat), static pressure-volume (Stat P-V) hysteresis, vital capacity (VC), frequency dependence of dynamic compliance (C dyn) and collateral ventilation (Coll V) of the lung were studied in six mongrel dogs. The smoking of one cigarette induced a fall in VC and static P-V hysteresis of the lungs. These changes indicate an increase in elastic recoil of the lung probably caused by inactivation of pulmonary surfactant. Frequency dependence of C dyn did not develop after smoking. Extensive collateral ventilation was seen in the lungs of all the experimental animals and in those of five other normal dogs, who had not been exposed to cigarette smoke. The significance of these findings are discussed.

Key words : cigarette smoking static P-V relations of lung collateral ventilation

INTRODUCTION

It is now well established that cigarette smoking even for only a few years, causes early changes in the peripheral airways of the lung (1-3). The mechanisms by which these changes are brought about are not definitely known. Factors such as inflammatory changes (4) and increased mucus secretion (5) play an important part. A probable mechanism which has not been much studied is related to the surface forces within the bronchoalveolar system. The alveoli and the bronchioles are lined by a surface film which contains surface active material. The alveolar Type II cells and the Clara cells are believed to be responsible for the secretion of this material (6,7). Inactivation of this surfactant, could increase surface forces and lead to narrowing of the peripheral airways and alterations in the pressure-volume characteristics of the lung. Information obtained from the immediate effects of cigarette smoking on the mechanical characteristics of the lungs may help in confirming this. The study reported here was therefore done to identify some of these effects in experimental animals.

METHODS

The studies were done on six mongrel dogs. The

following measurements were made.

Static compliance of the lung [C stat (1)].

Static pressure-volume [Static P-V] hysteresis of the lung

Vital capacity (VC)

Dynamic compliance of the lung [(C dyn (1)) at increasing breathing frequencies.

Collateral ventilation (Coll V) of the lungs.

The animals were anaesthetized with sodium pentobarbitone, using an intravenous dose of 30-40mg/kg. Generalized skeletal muscle paralysis was produced by intravenous succinyl choline, 3 mg per kg initially and repeated as necessary, during the entire experiment.

A tracheostomy was done and a tracheal cannula introduced. The animal was placed prone on a suitable stand, and a Y tube (A) connected to the tracheal cannula. One limb of the Y tube was used to monitor airway pressure. The other limb was connected through a silverman pneumotachograph and a second Y tube (B) to the inlet and outlet tubes of a Starling animal respirator. Artificial ventilation was maintained

with tidal volumes of approximately 25 ml/kg body weight. Pleural pressures were estimated from oesophageal pressures determined by the technique of Milic Emilie et al (8) using an oesophageal balloon and catheter. Pleural and airway pressures were recorded through a differential pressure manometer on one channel of a Grass model 7 polygraph. At zero airflow, airway pressure equals intrapulmonary pressure and therefore the difference between pleural and airway pressure equals transpulmonary pressure (Ptp).

Volume changes were obtained with the aid of a spirometer connected to the intake tube of the Starling respirator. Using a rotational transducer, these volume changes were converted to electrical signals and recorded on the polygraph. Respiratory airflows were measured with the aid of the pneumotachograph and recorded on the polygraph.

Calibration of the equipment was done before each experiment. Volume calibration was done with the aid of a one litre gas syringe. The differential pressure manometers were calibrated against a water manometer. Airflows were calibrated with the aid of a small vacuum cleaner and a dry gas meter. Fig. 1 shows a diagram of the experimental set up. The following procedure was used on each animal.

Measurement of VC, C stat (1) and Static P-V hysteresis: The lungs were fully inflated by manually closing off the outlet tube of the respirator until Ptp rose to about 30 cm H₂O. The outlet tube was then opened and lungs allowed to deflate. This was done

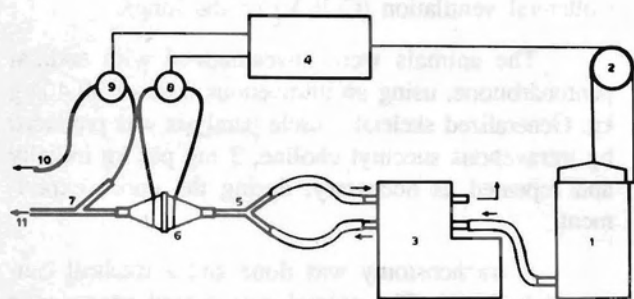
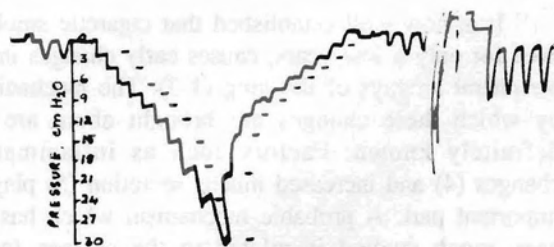


Fig. 1.: Diagram of the experimental set up.
 1. Spirometer
 2. Rotational transducer
 3. Respirator
 4. Polygraph
 5. Y tube B
 6. Pneumotachograph
 7. Y tube A
 8 & 9. Differential pressure manometers
 10. To oesophageal catheter
 11. To trachea

to ensure a uniform volume history. The pneumotachograph was disconnected from Y tube A, and a 100 ml syringe with a three way attachment was connected instead. Air was gently withdrawn until transpulmonary pressure fell to 0 cm H₂O. Next, air was introduced in steps of 50 ml until Ptp reached 30 cm H₂O. Air was then withdrawn in similar steps until Ptp reached 0 cm H₂O, the whole inflation and deflation taking 1 to 2 minutes. Static P-V curves were constructed from these values. VC was defined arbitrarily as the volume of air that could be withdrawn from the lung when Ptp fell from 30 to 0 cm H₂O (9).

Static P-V hysteresis was obtained from static P-V curves by plotting them on graph paper and measuring the area enclosed. C stat (1) was measured as the slope of the inflation static P-V curve over the tidal volume range used for the dynamic compliance measurements (10). Fig. 2 shows the pressure changes during the stepwise inflation and deflation of the lungs.

Measurement of C dyn (1): The lungs were again fully inflated and allowed to deflate to functional residual capacity, as variations in volume history can



Vol.	P _{insp.}	P _{exp.}
0.0	0.0	0.0
50	1.8	1.5
100	4.2	2.3
150	6.6	4.8
200	8.1	6.3
250	9.9	7.8
300	12.0	9.6
350	15.6	11.7
400	19.2	14.7
450	24.0	20.4
500	30.0	30.0

TEMP. 34°C
 BP = 740 mm Hg
 CF. 1.019
 VE = 500 ml, STPS
 = 509.5 ml, STPS

Fig. 2.: Static pressure-volume relations.
 BP. Barometric pressure
 CF. Correction factor for conversion of volume at ATPS to BTPS.
 Vol. Volume
 P_{insp.} Pressure during inflation
 P_{exp.} Pressure during deflation

affect C dyn (1) (11). The pneumotachograph was now reconnected to Y tube A. The spirometer was connected to the intake tube of the Starling respirator and inspired volume, airflow and Ptp recorded at respiratory frequencies of 15, 25, 35 and 45 per minute. Insp C dyn (1) was calculated as the ratio of the inspiratory volume change to pressure change taken at points of zero flow. At each frequency the mean value from five respirations was taken.

Smoking : The pneumotachograph was disconnected and replaced by a polythene tube, about 25 cm long connecting the two Y tubes. The differential pressure manometer was disconnected from Y tube A, and that limb of the Y tube closed off, to protect the manometer from the smoke. The spirometer was disconnected and a cigarette fixed to the intake tube of the respirator, and lighted. The animal was made to smoke one cigarette completely. No recordings were made during the smoking. Soon after smoking, all the above measurements were repeated within ten minutes.

Determination of Coll V̇ : Since the mechanical characteristics of the lung depend on the presence and extent of Coll V̇ between the lobules (12), this was studied using the method of Van Allen and Lindskog (13), in all the experimental animals after the measurements were completed and the lungs of 5 normal dogs not exposed to cigarette smoke. The lungs were excised, and one lobe of each lung carefully separated from the rest of the lung. The lobar bron-

chus was gently dissected free, down to its division and then cut. Generally at this level three daughter bronchial openings appeared. Into each a polythene tube was introduced and tied securely. One tube was connected to a glass syringe with a 3 way Luer lock attachment. The other two tubes were extended into a container of water (Fig. 3) Air was drawn from the

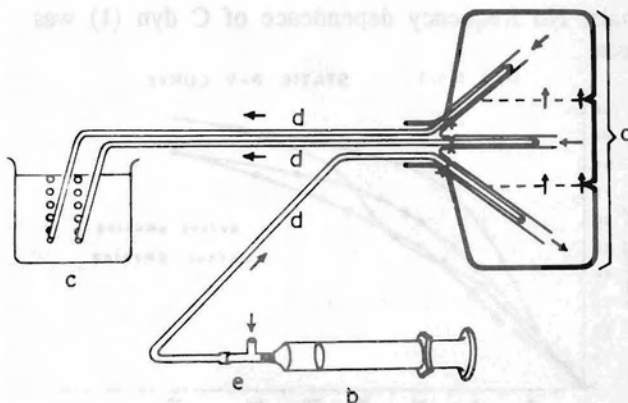


Fig. 3 : Experimental set up for demonstration of interlobular ventilation
 a. Lung lobe
 b. Syringe
 c. Container of water
 d. Polythene catheter
 e. Three way attachment
 → direction of airflow

atmosphere into the syringe and then very gently introduced into the lobule through the tube, and escape

TABLE I : Immediate effects of smoking on the static P-V characteristics of lungs.

No	Sex	Body weight kg	Lung weight gm	Procedure	C stat (1) ml/cmH ₂ O	VC,* ml	Static P-V* hysteresis mm ²
1	F	5.0	55.5	Before	25.5	583	1937
				After	24.3	510	1070
2	F	5.0	57.0	Before	39.2	453	1219
				After	39.2	448	543
3	M	11.5	101.0	Before	31.3	698	2708
				After	32.5	703	2313
4	M	15.5	131.0	Before	68.3	1275	4441
				After	68.3	1223	3123
5	F	10.0	78.5	Before	76.4	866	1457
				After	69.3	723	1268
6	F	2.5	27.0	Before	15.9	471	3360
				After	13.1	305	770

*P < 0.05

of bubbles into the water looked for. The syringe was now connected in turn to the other two tubes, and the procedure repeated.

RESULTS

Table I shows the results obtained and Fig. 4 shows the static P-V curves obtained from one animal. There was a significant decrease in VC and P-V hysteresis. There was a decrease in the C stat (1) in three animals. No frequency dependence of C dyn (1) was seen.

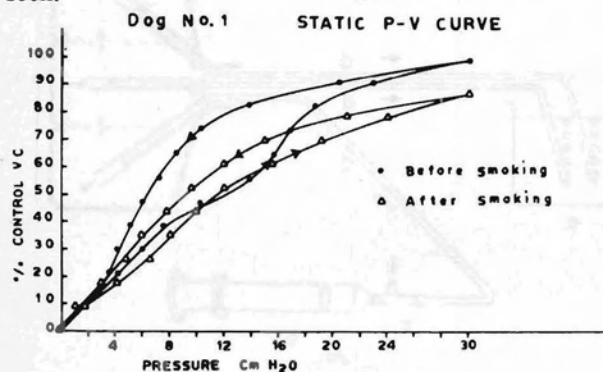


Fig. 4: Effects of smoking on static pressure-volume relations.

The lungs of all the animals tested showed extensive Coll \dot{V} , as evidenced by free escape of air from the other lobules when any one lobule was inflated.

DISCUSSION

A decrease in C stat (1) and VC could result from increased surface tension forces, increased bronchomotor tone in the small airways or haemodynamic changes in the pulmonary circulation, caused by cigarette smoking. The extent to which each of these factors contributes to these changes in this study is not clear. Haemodynamic changes in the pulmonary circulation are known to follow the smoking of a cigarette (14), but the nature and magnitude of these changes are such that they are unlikely to alter the mechanical properties of the lung (15).

Altered surface tension forces probably play a significant role in causing these changes. This view is supported by the decrease in P-V hysteresis that also occurred after smoking. Hysteresis is the "failure of a system to follow identical paths of response upon application and withdrawal of a forcing agent (16). In

normal lungs, most of the hysteresis is due to surface forces (17) and can be accounted for by the properties of the alveolar surface film. A decrease in static P-V hysteresis has been observed with a rise in temperature of the lung (18), following insufflation of kerosine into the lung (19), following prolonged exposure of the lungs to 70% oxygen (20), following rinsing of the lung with tween 80 (21) and in hyaline membrane disease (22). In all these cases, the decreased hysteresis was attributed to a decrease in pulmonary surfactant activity. The possibility of a raised temperature causing the decreased hysteresis in our experiments is unlikely, as the cigarette smoke tested at the airway opening was found to have reached room temperature. Volume history of the lung, which can influence C stat (i) and VC (ii), was kept constant in our experiments by full inflation of the lung before each measurement. The decreased surfactant activity may have been caused by inhibition of surfactant secreting cells or by inactivation of surfactant. The rapidity with which the effects occur indicate the later probability, although in chronic smoking a decrease in Clara cells has been reported (5). Our results support the findings of others, on the effect of cigarette smoke on lung extracts (23-25). This may not be a specific effect of cigarette smoke, but a nonspecific effect of particulate matter on molecular cohesive forces (25).

Increased bronchomotor tone leading to peripheral airway narrowing may also have contributed to the decrease in C stat (i) and VC. Although it has been said that only complete occlusion of these airways can cause these changes (9), it has been shown in smokers (26) and in nonsmokers (27) that bronchodilators cause a shift of the P-V curve to the left, indicating that an increase in bronchomotor tone of any magnitude may cause these changes. The lack of frequency dependence of C dyn (1) in our experimental animals does not prove the absence of peripheral airway narrowing. Frequency dependence of C dyn (1) has been considered to be a test sensitive to peripheral airway narrowing (28). However, if the lungs have extensive Coll \dot{V} , frequency dependence of C dyn (1) may not occur, even in the presence of peripheral airway narrowing. Hogg et al (29) using an electrical analogue have shown that when the time constant for Coll \dot{V} is less than 0.1 sec, frequency dependence of

C dyn (1) does not occur. As surfactant is present normally in the alveolar as well as in the small airway lining film, and since our results indicate inactivation of surfactant, it is very probable that inactivation occurs in both, the alveoli as well as in the small airways, contributing to increased lung recoil and to peripheral airway narrowing.

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