Cigarette Smoke

Its Effect on Pulmonary Function Measurements

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• **Inhaling cigarette smoke with each breath, with the subject at rest, by use of a smoking device that brought more smoke into the lungs than would be the case in ordinary smoking, produced consistent significant decreases in arterial blood oxygen saturation and in arterial pO_2 in most subjects who had severe or very severe pulmonary emphysema. In normal subjects and in those with a moderate degree of emphysema no significant changes in blood gas exchange resulted.**

No consistent significant changes in blood gas exchange were noted after the smoking of two cigarettes, either with the subject at rest or after a one-minute step-up exercise.

A decrease in oxygen uptake occurred when treadmill exercise was done after smoking two cigarettes, and the ventilation volume was also decreased, probably accounting for part of the oxygen decrease.

Pulmonary compliance measurements after smoking one cigarette were consistently and significantly decreased in most subjects—normal as well as those with pulmonary emphysema. The elastic work of breathing was increased in the majority of cases.

In two cases in which studies were done after the subjects stopped smoking, one for three months and one for two years, significant reductions in residual air were noted.

The results indicated that persons with severe or very severe emphysema would be better off to stop smoking.

THE RELATIONSHIP of chronic pulmonary disease, such as emphysema, and smoking is of particular interest in the Cardio-Respiratory Laboratory of the University of Southern California School of Medicine. A very high incidence of cigarette smoking has been noted commonly in the history of patients with severe emphysema when there was no apparent cause for the emphysema, such as a history of exposure to silica, asbestos, diatomaceous earth or other known irritating dusts, or a history of having had tuberculosis, asthma or some other chronic pulmonary disease.

Some physicians have arbitrarily ordered patients to stop smoking as part of the management of pulmonary emphysema. On the other hand, other physicians do not even advise patients with emphysema to stop smoking.

The Cardio-Respiratory Laboratory is frequently asked for an opinion as to whether or not a patient should stop smoking, and if so, what is the objective evidence. If pulmonary function measurements were to show definite and significant changes impairing lung function as the result of smoking, then it would be much easier to convince patients with emphysema that they should give up smoking.

Pulmonary emphysema may be the result of several factors and the evaluation of the importance of each one may be quite difficult. The present study was set up to explore the practical tests of pulmonary function which might be used to assess acute changes which could be induced by smoking one or two cigarettes, and thus permitting each subject to serve as his own control. The subjects studied were all smokers and the group included persons without disease and patients with chronic pulmonary disease of varying degrees of severity, especially pulmonary emphysema and pulmonary fibrosis. The subjects were advised to abstain from smoking from four to six hours before the tests of function were to be done, and in a few cases for as long as twenty-four hours. A longer time interval without smoking would be desirable; and it would also be helpful to obtain studies before and during the period of abstinence, and after resuming smoking. However, obtaining volunteers for such restrictions would be difficult. In two cases, follow-up studies were obtained after longer time intervals, as the patients decided to stop smoking; and as far as could be determined, no medication had been taken during this time which might be a factor in altering the pulmonary function measurements.

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METHODS

All the subjects—125 men and 16 women—were fasting and in an essentially basal state at the time of the studies. The ages ranged from 24 to 70 years. Each of the subjects had a complete pulmonary function evaluation including lung volume de-

terminations and blood gas exchange measurements while at rest and after exercise (except for three subjects who had heart disease and one normal subject).

Spirogram recordings on the 13.5 liter respirometer were made to determine the total vital capacity, the timed vital capacity for three seconds and the maximal breathing capacity, before and after administration of a bronchodilator drug.[12] Residual air was measured by the oxygen open circuit method and the helium closed circuit method.[13] The residual measurements were made with the subject supine in a fasting and resting state. The alveolar nitrogen sample was taken just above the mouthpiece after seven minutes of oxygen breathing. In a few cases, the nitrogen curve was obtained after a single deep breath of oxygen, using the nitrogen meter. The nitrogen wash-out was also obtained in a few of these subjects with the nitrogen meter with continuous recordings. All lung volume measurements were expressed at body temperature, pressure saturated (BTPS).

Arterial blood samples were obtained through an indwelling needle from the brachial artery, at rest and immediately after exercise. Oxygen content and capacity and carbon dioxide content determinations were made in duplicate by two trained technicians using different manometric Van Slyke analyzers and were repeated unless the results were close together. The arterial blood oxygen saturation (as per cent) was also determined on the Water's oximeter, double scale cuvette with the use of whole blood. The cuvette was calibrated daily against the Van Slyke analyzers, and in this laboratory it has been found very satisfactory, the results consistently staying within one per cent of the Van Slyke measurements when properly calibrated. The pH was measured on a Cambridge glass electrode. The arterial partial pressure oxygen (pO_2) and partial pressure carbon dioxide (pCO_2) were determined by the direct bubble tension method described by Riley.[15]

Oxygen uptake and carbon dioxide output were calculated from the percentage of oxygen and carbon dioxide in the expired air, as determined in a Scholander gas analyzer. The step-up exercise consisted of stepping up and down on a stool, 20 cm. high, 30 times in one minute; and the treadmill exercise was walking, level, at a rate of two miles per hour. The minute ventilation volume was measured during the basal rest period and during the one minute of step-up exercise, or during the sampling period on the treadmill after five minutes or more of walking. The minute ventilation and the oxygen uptake were expressed at standard temperature and pressure as liters per minute, or milliliters per minute per square meter of body surface area (ml./min./M^2, BSA). The alveolar pO_2 was calculated from the values obtained for arterial pO_2, the arterial pCO_2 and the respiratory quotient. The effective tidal air was calculated from the expired pCO_2 and the arterial pCO_2 using the average tidal volume. Hemoglobin was determined both by the oxygen capacity method and by converting all the hemoglobin to cyanmethemoglobin[8] and measuring light absorption at 540 mu. on the Beckman DU spectrophotometer.

Measurements of pulmonary compliance were determined by the continuous cycling method with modifications.[9] The recording device consisted of a 6-liter Benedict-Roth metabolism apparatus equipped with a helipot for volume changes. A blower was provided in the circuit to circulate the air, and all valves were removed to reduce breathing resistance. Oxygen was added to the system and the CO_2 was absorbed. Volume and pressure changes were recorded on a DuMont cathode ray oscilloscope through a compliance control apparatus employing a capacity transducer in a radio frequency circuit. The loop was interrupted by means of a blanking generator which allowed time measurements of 5, 10, 15 and 20 cycles per second intervals. In addition, the points at which there was no flow were determined by a flow-sensitive zero pressure device which produced blips on the oscilloscope at the end of expiration and inspiration. A balloon as described by Crane[7] was used to measure the intraesophageal pressure at its mid-position. The loop was then photographed with a polaroid Land camera. The patients were studied in the sitting position during spontaneous quiet breathing before and immediately after smoking one cigarette.

PROCEDURE AND SUBJECT MATERIAL

Studies were obtained on nine patients, all except two with severe pulmonary emphysema, in the supine position after smoking two cigarettes. The amount of smoke inhaled varied widely between different subjects during the smoking of two cigarettes; some inhaled deeply, others hardly at all.

It was felt that the acute effects of cigarette smoke on the lung could be intensified by the use of a smoking device so that with each breath some smoke would be inhaled in the lung with the respiratory gases. The subject was connected by a mouthpiece to a two-way directional valve which was connected on the intake side to a Douglas bag containing air on one side of a "Y" tube and on the other side containing a cigarette holder with a lighted cigarette. By the use of a three-way valve attached to the Douglas bag, the resistance could be varied so that a small amount of air with each breath was directed through the cigarette, with a resulting glow during

inspiration. The expired air was collected in a Tissot gasometer and the smoke was quite visible in the expired air from the subject.

In some subjects the inhaled smoke produced a sensation of burning, particularly on the posterior pharynx, and in a few it caused cough. Other subjects, most commonly heavy smokers with a history of smoking for many years, said that they were not aware that they were even getting smoke with the device. The use of the smoking device in the closed circuit insured that the smoke was distributed in the lung, so that the effect of smoke on the lung with respect to blood gas exchange could be studied. In all instances, the patients were allowed to smoke the cigarette of choice if they had their own. The rate of smoking was set so that usually it took about three to five minutes to completely smoke one cigarette, and in most instances at least 90 per cent of the cigarette was smoked. Fifty subjects were studied during the smoking of one cigarette, and these subjects were divided into three groups according to the severity of the pulmonary emphysema present. There were 23 cases in which the emphysema was insignificant, eight in which there was a moderate degree of emphysema and 19 in which it was severe or very severe (residual air greater than 45 per cent of total lung capacity).

Pulmonary compliance measurements were obtained on 41 subjects. In these subjects the residual per cent of total lung capacity had been determined (with the exception of three subjects who had cardiac disease and one who was normal). The vital capacity was determined in most of the subjects before and after smoking, utilizing the usual volume recording apparatus, but with the subjects in the sitting position. Compliance and the elastic work of breathing were the two specific measurements obtained at this study, and these data were correlated with the severity of the emphysema as measured by the residual per cent of total lung capacity, the ventilation factor, the resting arterial pO_2 and the vital capacity, before and after smoking.

A group of fifty subjects had blood gas exchange measurements made before and after smoking, using the one minute step-up exercise test. This group of fifty subjects was divided into three classes, depending on the severity of the emphysema. There were 19 with an insignificant degree of emphysema, 16 with a moderate degree of emphysema and 15 with a severe or very severe degree of pulmonary emphysema. The second exercise test was performed immediately after smoking two cigarettes and the measurements were compared to those of the first test. In 21 cases, treadmill exercise was used instead of step-up exercise, and the blood gas exchange measurements before and after smoking two cigarettes were compared.

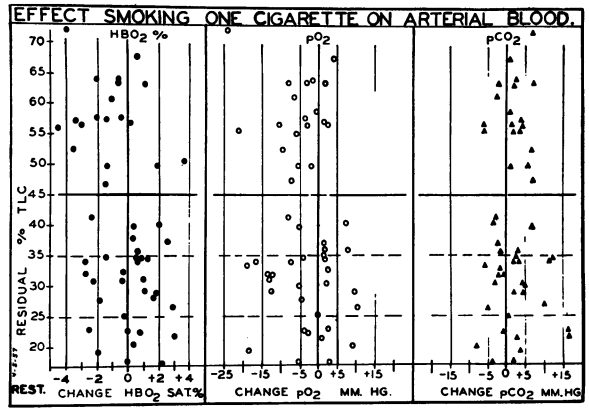

Chart 1.—Correlation of the residual per cent of total lung capacity with the change during the smoking of one cigarette with the smoking device, at rest, on: (1) Arterial blood oxygen saturation (HbO_2 saturation, per cent, (2) arterial pO_2, and (3) pCO_2 in mm. Hg., both by direct tension measurement. TLC (total lung capacity), HbO_2 per cent (arterial oxygen saturation, per cent), pO_2 (partial pressure oxygen), pCO_2 (partial pressure carbon dioxide). Measurements on 50 cases.

RESULTS

Blood gas studies in the supine position before and after smoking two cigarettes were obtained in nine subjects, seven of whom had severe emphysema. There was no significant average difference after smoking in the group, as shown by the arterial blood or the pulmonary ventilation measurements.* There was a slight increase in the pulse rate, but no significant change in the respiratory rate. In one subject at rest there was a significant decrease (—6.5 per cent) in the arterial blood oxygen saturation after smoking. This subject had a very severe degree of emphysema, and following the smoking the pulse rate, respiration rate and minute ventilation were significantly increased, but the arterial pO_2 was decreased from 73.5 to 56.1 mm. of mercury.

The smoking device with the closed circuit was next used in the supine position to insure that the smoke from the cigarette got into the lung. The data using the smoking device were compared in summary form by three groups based on the severity of the emphysema. There was no significant change in the arterial blood oxygen saturations before and after smoking either in the nonemphysema group or in the group with a moderate degree of emphysema. However, in the severe emphysema group there was an average decrease in the arterial blood oxygen saturation from 90.7 per cent at rest to 88.6 per cent after smoking. The changes in the arterial blood saturation before and after smoking have been plotted for each case on a basis of the severity of the

*More detailed data in tabular form will be published in the reprint of this paper.

emphysema as measured by the residual per cent of total lung capacity (Chart 1). It can be noted that none of the subjects with a residual per cent of total lung capacity below 45 per cent had significant change. In some the saturation was increased a little and in others it was decreased. However, in this group of 19 subjects with severe or very severe pulmonary emphysema there was definitely a decrease in the saturation after smoking, a decrease occurring in 14 and an increase in four, with no change in one. The standard deviation for the 19 subjects was ± 1.96 per cent with a standard error of 0.45 per cent. In a similar manner, the arterial pO_2, as determined by direct tension measurement, was correlated with the severity of the pulmonary emphysema (Chart 1), and a consistent decrease was present only in subjects with a residual ratio greater than 45 per cent of total lung capacity (decreased in all except four). The standard deviation of the arterial pO_2 was ± 7.6 mm. of mercury with a standard error of 1.7 mm. of mercury. When the arterial pCO_2 was correlated with the residual per cent of total lung capacity, it was noted that in the emphysema group in all except four cases there was an increase after smoking. The standard deviation of the arterial pCO_2 was ± 2.0 mm. of mercury with a standard error of 0.9 mm. There was no significant change in the arterial CO_2 content or pH after smoking with the smoking device. Also the alveolar-arterial pO_2 difference was not significantly changed after smoking. The mean alveolar pO_2 was calculated from the arterial pCO_2, the arterial pO_2 and the respiratory quotient. There was no significant change in the tidal volume or the effective tidal air with smoking, or in the oxygen uptake, the CO_2 output or the per cent of oxygen extracted from the inspired air breathed, using the smoking device.

The blood gas measurements with step-up exercises before and after smoking two cigarettes were studied in 50 cases. The changes, after smoking, in the arterial blood oxygen saturation, in the CO_2 content in volumes per cent and in the minute ventilation (in liters per minute per square meter of body surface area) are correlated with the residual per cent of total lung capacity in Chart 2. The subjects smoked two cigarettes while in the sitting position following the first step-up exercise. A few had reactions, possibly related to the fasting state. These reactions consisted of feeling dizzy or weak, and a few subjects had headache, sweating and tachycardia. If the subject had a reaction following the smoking, a rest period of a few minutes was given and then the second exercise was started. In practically all cases, the subjects felt better after the exercise. A few of the subjects complained that the cigarette did not taste good. In none of the three

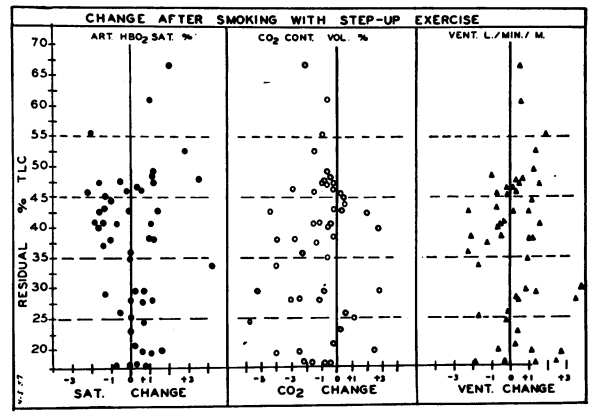

Chart 2.—Correlation of the residual per cent of total lung capacity with the change after smoking two cigarettes in the usual manner on the one minute step-up exercise measurements of: (1) Arterial blood oxygen saturation, (2) arterial CO_2 content and (3) the volume of pulmonary ventilation (liters per minute per square meter of body surface area). Measurements on 50 cases.

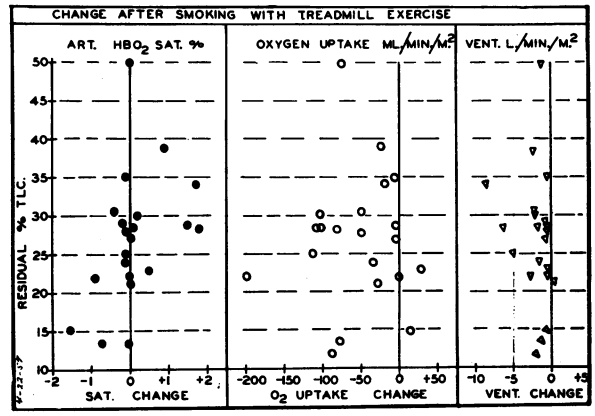

Chart 3.—Correlation of the residual per cent of total lung capacity with the change after smoking two cigarettes in the usual manner on treadmill exercise measurements of: (1) Arterial blood oxygen saturation, (2) oxygen uptake, and (3) the volume of pulmonary ventilation (liters per minute per square meter, body surface area). Measurements on 21 cases.

groups of subjects with varying degrees of emphysema was there significant change in the arterial blood oxygen saturation (Chart 2). Nor was there correlation between the change in arterial blood oxygen saturation after smoking and the residual per cent of total lung capacity. There was no significant change in the arterial CO_2 content and pH, before and after smoking with step-up exercise. The arterial pulse was consistently increased after smoking and the respiratory rate was increased slightly. A slight increase was noted in the average minute ventilation and oxygen uptake after smoking with step-up exercise. The data on individual subjects as to the CO_2 content change after smoking with step-up exercise revealed a tendency toward a decrease

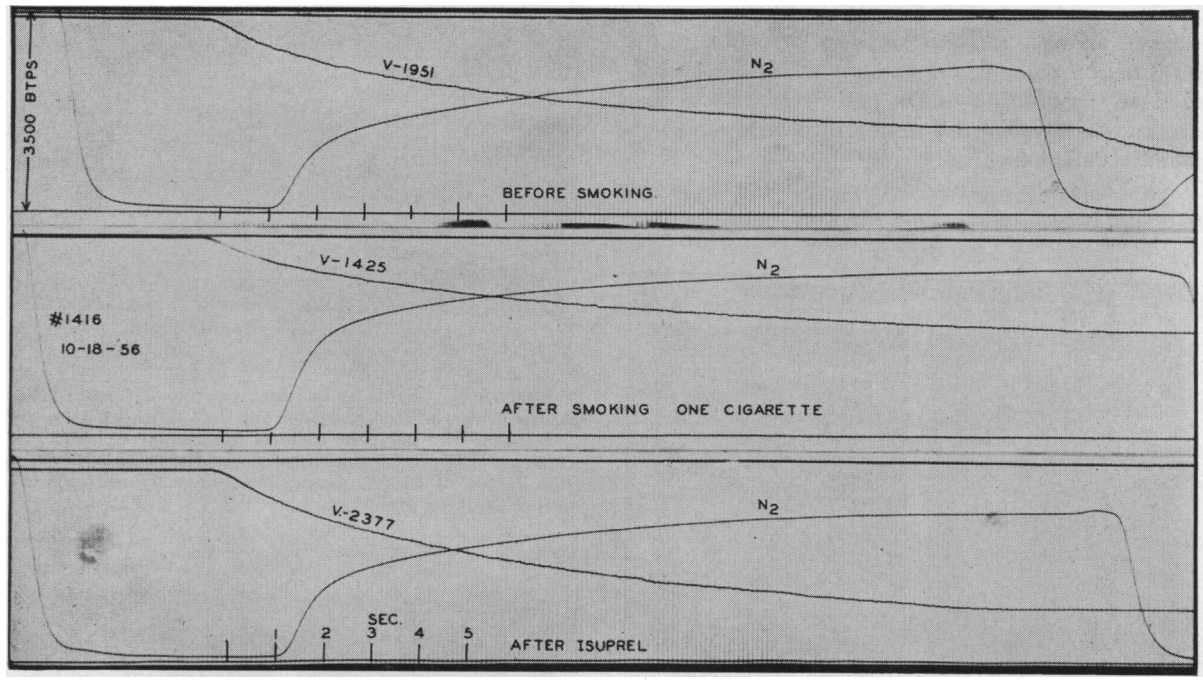

Chart 4.—The nitrogen curve (labeled N_2) and the corresponding volume (V) and flow during exhalation (total recorded as ml.) in a patient with severe emphysema, obtained by taking a single deep breath of oxygen and then blowing out as far as possible. The nitrogen was recorded from a nitrogen meter and the volume from the 13.5-liter Collins respirometer with a helipot to convert volume to electrical output. Impaired intrapulmonary mixing gives a prolonged sloping curve upward as shown in N_2 curve before smoking. After smoking the N_2 mixing curve was slightly improved (middle tracing) although the volume of the deep breath was reduced (vital capacity). After bronchodilator isuprel the vital capacity was significantly increased (V from 1425 to 2377 ml.) although the N_2 curve was not improved. Lung volume measurements are expressed at body temperature, pressure saturated (BTPS).

in CO_2. However, this was probably correlated with a slight increase noted in the minute ventilation (Chart 2). The average tidal volume was increased slightly after smoking.

Blood gas studies were obtained before and after smoking and treadmill exercise in 21 cases. The arterial blood oxygen saturation, the oxygen uptake in ml./min./M^2 BSA, and the ventilation as L./min./M^2 BSA are correlated with the residual per cent of total lung capacity for the 21 cases (Chart 3). There was no significant change in the arterial blood oxygen saturation after smoking. However, the oxygen uptake on exercise was decreased an average of from 565 to 516 ml./min./M^2 BSA after smoking. The standard deviation for the change in oxygen uptake on exercise was ± 53 ml. with a standard error of 11.5 ml. There was also an average ventilation change after smoking (Chart 3). The average minute ventilation was decreased from 13.05 to 11.62 L./min./M^2 BSA. The decrease in the oxygen uptake on exercise may be related in part to the decrease in the minute ventilation. However, no such decrease in minute ventilation was noted during step-up exercise after smoking two cigarettes. Direct tension measurements of arterial pO_2 and pCO_2 in the group of cases with treadmill exercises revealed no significant changes. The effective tidal air, computed on the basis of the expired pCO_2 and the arterial pCO_2, was not significantly changed in this group.

In several cases of severe emphysema, the single deep breath technique of oxygen with continuous recording of nitrogen on the nitrogen meter was employed and the volume of exhalation measured by electrical recording (Chart 4). No significant change in the air distribution as judged from the shape of the nitrogen curve was noted after smoking, even though the vital capacity usually showed some reduction. Although treatment with bronchodilator drugs after the smoking test increased the vital capacity significantly, the shape of the nitrogen curve was not improved.

In 41 subjects who smoked regularly the effect of one cigarette on pulmonary compliance was determined. The group consisted of eight normal persons and 33 patients with varying degrees of cardiorespiratory insufficiency as determined by complete pulmonary function studies. The pulmonary compliance was determined by a modification of the continuous cycling method, and timed pressure-volume loops were obtained over the range of quiet respiration. Pressure determinations were obtained by means of an esophageal balloon placed in the mid-esophagus. The resting compliance determina-

tion was obtained after the patient smoked a cigarette of his choice. There were no variations in the level of the balloon, the amount of air in the balloon or the position of the patient (variables that are known to affect the results in determining pulmonary compliance).

Vital capacity was also determined on most of the patients before and after smoking, utilizing the

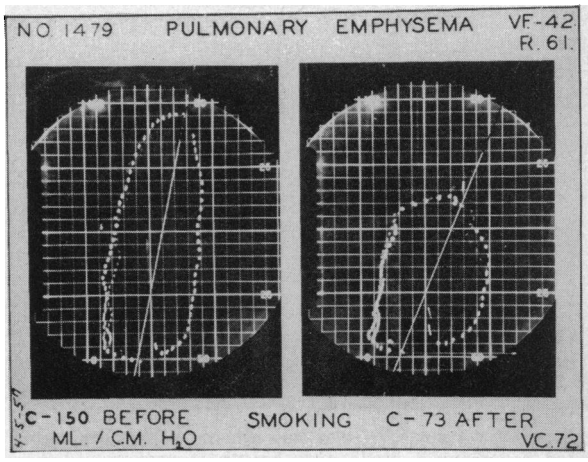

Chart 5(a).—Pulmonary compliance measurements before and after smoking one cigarette in the sitting position in a patient with severe emphysema (residual 61.0 per cent of total lung capacity (R), total vital capacity (VC) 72.0 per cent of normal, predicted and ventilation factor (VF) 42.0 per cent). Volume is recorded on the vertical axis and pressure on the horizontal axis. The interruption of the tracing permits timing, as the oscilloscope beam moves around counter clockwise, describing a complete loop for each respiratory cycle. The blip at the bottom is the no flow point at the end of expiration and the blip at the top the no flow point at the end of inspiration. The sloping line connects the points of no flow at the end of inspiration and expiration. Compliance (C) 150 ml./cm. H_2O before smoking and 73 ml/cm. H_2O after smoking.

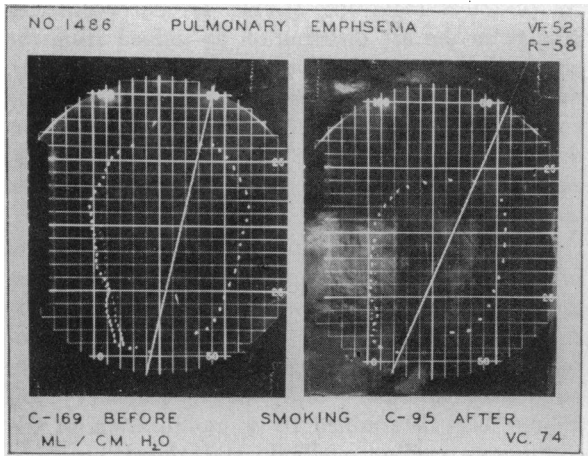

Chart 5(b).—Same as in Chart 5(a) except in a patient with a residual of 58.0 per cent of total lung capacity, total vital capacity 74.0 per cent of normal predicted and ventilation factor 52 per cent. Compliance 169 ml./cm. H_2O before smoking and 95 after.

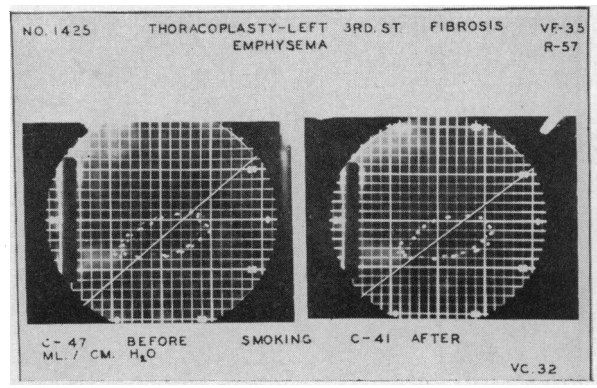

Chart 5(c).—Same as in Chart 5(a) except in a patient with fibrosis and emphysema and left thoracoplasty with a residual 57.0 per cent of total lung capacity, total vital capacity 32.0 per cent of normal predicted and a ventilation factor of 35 per cent. Compliance 47 ml./cm. H_2O before smoking and 41 after.

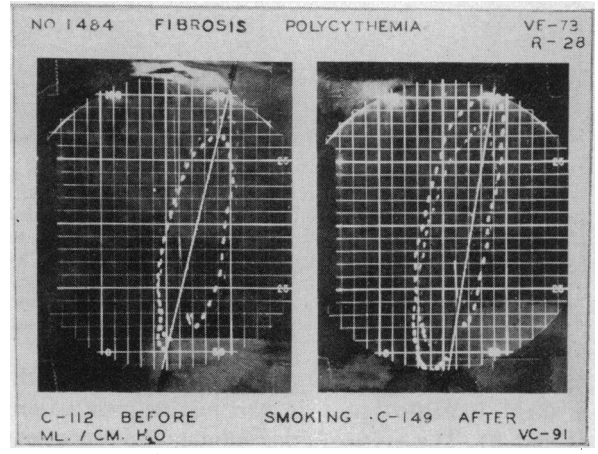

Chart 5(d).—Same as in Chart 5(a) except in a patient with fibrosis (x-ray) and polycythemia, with a residual of 28.0 per cent of total lung capacity, total vital capacity 91.0 per cent of normal predicted and ventilation factor 73.0 per cent. Compliance 112 ml./cm. H_2O before smoking and 149 after.

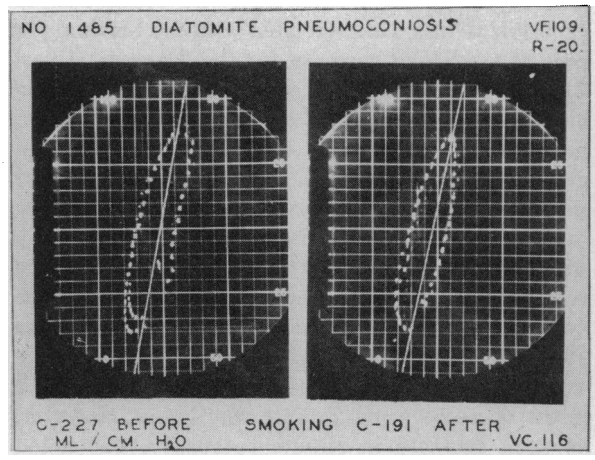

Chart 5(e).—Same as in Chart 5(a) except in a diatomite worker with a residual of 20.0 per cent of total lung capacity, total vital capacity 116.0 per cent of normal predicted and ventilation factor 109 per cent. Compliance 227 ml./cm. H_2O before smoking and 191 after.

TABLE 1.—*Effect of Cigarette Smoking on Pulmonary Compliance Measurements*

Case No.	Residual Per Cent of Total Lung Capacity	Ventilation Factor Per Cent	Resting Arterial pO_2 mm. Hg.	Sitting Vital Capacity		Compliance, ml./cm. H_2O		Elastic Work Breathing gm. M/liter of Ventilation	
				Per Cent Normal Predicted	Per Cent Change with Smoking	Observed	Change with Smoking	Observed Rest	Change with Smoking
1	61	42	75.6	73	— 5	1.0	— 73	45.00	+ 9.50
2	56	40	74.5	77	—16	83	— 22	57.50	+16.00
3	36	65	82.5	79	— 4	109	— 8	30.50	+21.50
4*	31	88	87.2	96	— 7	241	—131	17.50	+15.50
5	40	64	80.7	91	+ 1	169	— 15	27.50	+ 5.00
6	53	53	....	61	+ 8	168	— 26	32.00	+ 8.00
7	57	43	81.0	75	....	147	— 79	41.00	+13.50
8	35	64	92.1	41	—11	239	— 83	21.50	+ 5.50
9	23	100	97.5	92	—12	187	— 72	12.50	+ 7.50
10	48	55	87.3	60	....	48	+ 2	66.50	+12.50
11	43	69	81.8	95	....	96	— 11	43.00	0
12	67	61	85.6	60	....	179	....	18.00	— 5.00
13	62	32	....	54	....	153	— 50	15.50	+12.00
14	64	31	49.1	50	....	24	+ 3	12.00	— 1.50
15*	19	111	108.1	100	....	91	— 14	42.50	— 4.50
16	25	87	73.6	59	....	80	+ 24	38.50	—13.00
17	33	73	94.0	98	....	224	—109	16.50	+24.00
18	53	46	56.1	64	....	109	+ 56	22.00	— 3.50
19*	16	....	....	98	— 5	152	— 65	17.50	+19.50
20	57	44	63.0	30	—14	47	— 6	48.00	+ 5.00
21	....	....	....	95	— 2	143	— 57	28.50	+21.00
22*	18	114	90.2	116	— 1	297	— 28	8.00	— 2.00
23	59	31	66.4	62	—20	78	— 46	42.00	+10.50
24	48	55	91.3	83	+19	197	+ 19	27.50	— 5.00
25*	23	95	....	97	....	132	— 26	31.00	— 1.50
26	28	73	90.8	82	+ 4	115	+ 34	36.50	— 3.50
27*	20	109	102.0	110	— 3	239	— 49	23.50	+ 1.50
28	58	22	83.3	87	— 5	168	— 73	36.00	+18.00
29	29	71	....	67	+ 5	77	— 10	39.00	+ 1.50
30	....	....	....	63	+ 3	111	+ 25	45.00	—14.50
31	....	....	....	87	— 6	226	—108	23.50	0
32	32	72	....	102	....	267	— 56	21.50	— 5.50
33	66	29	59.9	66	— 8	77	— 15	34.50	— 7.00
34	31	86	88.5	80	+23	130	+ 44	18.00	+ 2.00
35	36	92	88.2	125	— 6	167	— 23	35.50	—10.50
36*	22	109	78.9	110	— 3	110	+ 21	38.00	— 7.50
37*	24	....	....	89	....	75	— 3	35.50	+ 9.00
38	....	....	....	75	— 5	111	— 6	50.50	0
39	44	66	....	86	—14	101	+ 1	86.50	— 7.50
40	68	32	75.6	29	—20	344	— 5	9.00	+ 2.00
41	24	89	84.6	92	+10	267	— 79	18.00	+23.50

*Normal subjects.

same volume recording apparatus with the patient in the sitting position. The elastic work of breathing was determined from the pressure-volume loop obtained at rest and again after smoking. Utilizing this method of determining pulmonary compliance in a group of normals, the mean pulmonary compliance was found to be 166 ml./cm. H_2O, with a range from 75 to 304 ml./cm. H_2O.

The results of the compliance studies shown in Table 1 and typical recordings are shown in Chart 5, a, b, c, d and e. There was a significant drop in pulmonary compliance in six of eight normal subjects in the study after smoking, with a mean decrease of 52 ml./cm. H_2O. In one subject no change was noted, while another had an increase of 21 ml./cm. H_2O. In the 33 patients with cardio-respiratory disease, 17 had a significant decrease in compliance after smoking. The maximum decrease was 109 ml./cm. H_2O with a mean of 57 ml./cm. H_2O. In ten patients no significant difference was noted between compliance while resting and while smoking. Six patients had a significant increase in compliance on smoking, with a maximum increase of 56 ml./cm. H_2O and a mean of 32 ml./cm. H_2O. There was no correlation of compliance change with the residual per cent of total lung capacity (Chart 6), as there were significant decreases in the group of normal subjects as well as in the emphysema group. On the whole, smoking tended to decrease compliance in the normal as well as in the emphysema group. The sitting vital capacity was decreased on an average 8.4 per cent in 20 cases after smoking one cigarette, and was increased 9.1 per cent in eight cases (Table 1).

The elastic work of breathing expressed, as gram meter per liter (gm. M/L) of ventilation, was de-

termined before and after smoking in the group of 41 subjects who were regular smokers. The eight normal subjects in the group had a mean value of 42.5 gm. M/L ventilation at rest. The 33 patients with cardio-respiratory impairment had a mean of 33.3 gm. M/L ventilation with a range from 9.0 gm. M/L ventilation to 86.5 gm. M/L ventilation. The values obtained for the elastic work of breathing by this method compare favorably with those reported elsewhere.[2,5,6,11,14]

After smoking, four of the normal subjects had an average increase of 15.0 gm. M/L ventilation in their elastic work of breathing while four had a decrease, the average for the group being 3.9 gm. M/L ventilation (Table 1 and Chart 7). In the group with cardio-respiratory impairment, 19 subjects had an increase, the average increase for the group being 11.5 gm. M/L ventilation. Eleven had a decrease, the average for the group being 6.95 gm. M/L ventilation. In three cases there was no change. The change in the elastic work of breathing after smoking was correlated with the residual per cent of total lung capacity (Chart 7). An increase in the elastic work of breathing after smoking was noted as often in the normal subjects as in those with severe emphysema. Similar changes were noted with regard to correlation with the ventilation factor. The elastic work of breathing is a static measurement, reflecting the elastic properties of the lung recorded at the instant of zero air-flow, and is therefore independent of time, but directly correlated to volume changes and rate of breathing. The elastic work of breathing was determined only at rest during normal tidal volume exchange.

No direct correlation was noted between the arterial pO_2 by direct tension measurements and changes in pulmonary compliance that were brought about by smoking. In 14 cases the arterial pO_2 after smoking showed a decrease; in four of them the compliance was increased, in two was unchanged and in eight was decreased (Table 1). In five subjects there was an increase in the arterial pO_2 after smoking; compliance was increased in two of them and decreased in three. In most of the subjects with a low compliance, there was a low arterial pO_2 also, although in some cases a low arterial pO_2 was associated with a normal compliance value.

There was a significant decrease in compliance after smoking in 56 per cent of the group, while 27 per cent showed no change and 17 per cent had an increase in compliance. Further studies of the effect of cigarette smoking on pulmonary compliance appear indicated in order to delineate the changes due to vascular effect, both in the greater and lesser circulation, and distinguish them from the direct effect on the respiratory mechanism.

In two cases follow-up studies of lung volume

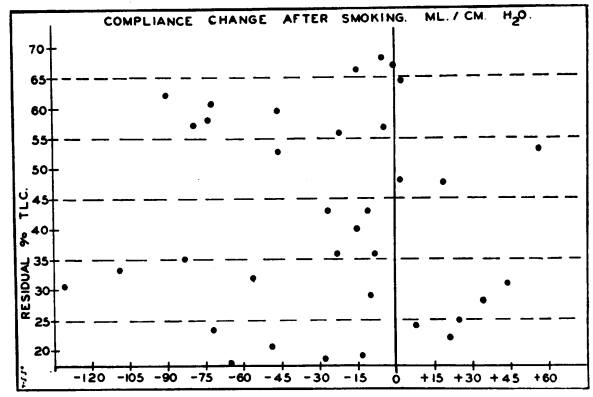

Chart 6.—Correlation of the residual per cent of total lung capacity with the change after smoking one cigarette in the usual manner on the pulmonary compliance measurement (milliliter volume change per centimeter H_2O pressure change). Pulmonary compliance change, ml./cm. H_2O, average mean —25, standard deviation ±39 and standard error 6.3.

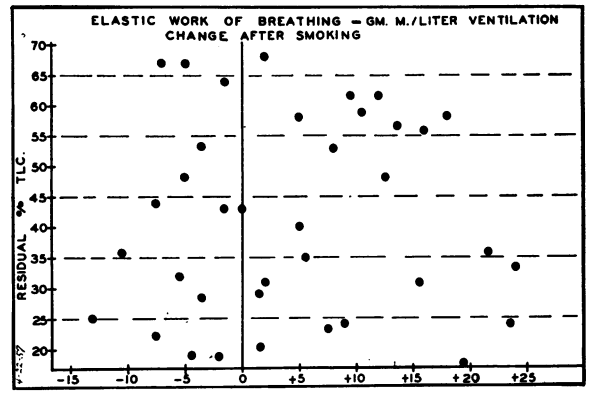

Chart 7.—Correlation of the residual per cent of total lung capacity with the change after smoking one cigarette in the usual manner on the elastic work of breathing (expressed as gram-meter per liter of ventilation).

were obtained after smoking of cigarettes had been stopped voluntarily by the subjects—for three months by one and two years by the other (Table 2). In both cases the residual air was decreased and the vital capacity increased after smoking was stopped. The timed vital capacity for three seconds and the maximal breathing capacity increased slightly in one case and decreased in the other. The subject in whom the decreases were shown complained of having had, for several weeks, a bad cold which had subsided very slowly. Bronchospasm was demonstrated in both cases before and after smoking was stopped (Table 2).

DISCUSSION

Loomis[10] carried out studies that led to a conclusion that there is a bronchoconstrictor factor other than nicotine in cigarette smoke. Bickerman and

TABLE 2.—*Lung Volume Changes After Cigarette Smoking Stopped*

Test	A Woman, Age 42 Years, Body Surface Area 1.67 Square Meters			A Man, Age 67 Years, Body Surface Area 1.90 Square Meters		
	Predicted	Observed June 25, 1953	Observed April 2, 1956*	Predicted	Observed Jan. 1, 1956	Observed March 12, 1957*
Total vital capacity, ml	3453	2540 (74%)	3320 (96%)	4165	2899 (70%)	3642 (87%)
Timed vital capacity, 3 sec. ml	3453	1650 (48%)	1929 (56%)	4165	1739 (42%)	1293 (31%)
Maximal breathing capacity, L./min.:						
Initial	104	26.2 (26%)	32.2 (31%)	121	35.0 (29%)	27.1 (23%)
After isuprel	104	48.0 (47%)	47.0 (46%)	121	51.0 (42%)	42.1 (35%)
Alveolar N_2, per cent	<1.5	6.89	5.05	<1.5	11.3	13.6
Residual air, ml	1190	2736 (227%)	2296 (193%)	1785	5263 (295%)	3382 (190%)
Total lung capacity, ml	4760	5276 (113%)	5616 (118%)	5960	8162 (137%)	7024 (118%)
Residual per cent of total lung capacity	25.0	51.9	40.9	30.0	64.5	48.1
Ventilation factor, per cent	100	41.0	49.0	100	39.0	39.0
Duration smoking stopped	.....	.....	2 years	.....	.....	3 months

*Study after smoking stopped.

Barach,[3] in a study of vital capacity and maximal breathing capacity in 122 subjects—91 with bronchial asthma and pulmonary emphysema and 21 normal persons—found no evidence of increased bronchospasm or impaired ventilatory function capacity in 108 of the subjects. There was a reduction of vital capacity and maximal breathing capacity in ten cases although unaccompanied by clinically perceptible increase in severity of asthma. An increase in vital capacity and maximal breathing capacity after smoking three cigarettes occurred in nine patients in whom coughing provoked by smoking resulted in expectoration of mucoid or mucopurulent sputum. A number of investigators have noted a decrease in vital capacity associated with long continued use of cigarettes.[4,16,18] Turley[17] noted no effect on vital capacity. Some of the observers who noted a decrease in vital capacity were of the opinion it may be associated with bronchospasm as a result of smoking, but the individual responses were quite varied. In some instances even an increase was noted. The decrease noted in pulmonary ventilation in some patients after smoking has been explained on a basis of the expectoration of mucopurulent secretions.[3] In some patients carbon monoxide may be a factor in stimulating pulmonary ventilation with smoking. However, Asmussen and Chiodi[1] carried out a study in which it was noted that the pulmonary ventilation was not stimulated by carbon monoxide poisoning as it was in hypoxemia due to breathing a low oxygen mixture.

The problem of allergic sensitivity to tobacco smoke has to be considered in studies like the present one. Some investigators regard allergic reaction to cigarettes as rather infrequent, although there can be little question that smoking or exposure to smoke from tobacco can aggravate asthma in some persons. The number of reported cases with detailed evidence of sensitivity to tobacco as a cause of respiratory manifestations is surprisingly small in view of the widely quoted statement that in one per cent of patients with asthma, tobacco is a factor.[19]

In a review of the literature very little information was found on changes in the blood associated with smoking, and not much more on gas exchange; what there was of the latter was principally on resting oxygen uptake. In the present study, when cigarette smoke was inhaled with the smoking device, there was a significant lowering in the resting arterial blood oxygen saturation in the majority of patients who had severe or very severe pulmonary emphysema. Direct tension measurements of arterial pO_2, using the bubble technique of Riley, also demonstrated a significant decrease in this group of subjects. However, the average pCO_2 was increased slightly but not to a statistically significant degree. There was no significant change in the ventilation volumes at rest when tests using the smoking device were carried out. Tests that were done in 50 cases before and immediately after smoking, with step-up exercise, showed no consistent significant changes in the blood gas exchange. Studies of 21 subjects before and after smoking, with treadmill exercise, did show a significant decrease in the exercise oxygen uptake after smoking. However, there was also a significant decrease in the minute ventilation, and this may have been the cause of the reduced oxygen uptake on exercise. One has to consider, however, that the reduced exercise oxygen uptake may possibly reflect some change in pulmonary vascular resistance, which could result from an increase in pulmonary blood volume or from bronchospasm.

When a very sensitive test such as pulmonary compliance was employed, significant changes in the pressure volume relationship were obtained in subjects who were essentially normal and also in those with various degrees of pulmonary insufficiency. When the elastic work of breathing was calculated, it was noted that smoking increased the work of

breathing in most subjects. There was no significant correlation of the change noted in compliance with smoking and the severity of the pulmonary emphysema or fibrosis.

In practically every case of severe emphysema which has been studied in this laboratory, when there was no other apparent cause for emphysema, there was a history of heavy cigarette smoking for many years. Significant changes in the arterial blood oxygen saturation were found only in the subjects with severe or very severe pulmonary emphysema when they inhaled the smoke with the smoking device, which delivered smoke in greater concentrations than was obtained by ordinary cigarette smoking. The experiment with the smoking apparatus was designed to try to intensify the effects of smoking as far as possible to see if blood gas exchange could be affected.

The data of this study in general indicated that patients with severe emphysema would be better off if they stopped smoking. No apparent difference was noted regarding the brand of cigarette smoked, or the presence of the filter tip. In normal persons the pulmonary compliance changes in some subjects were the only constant abnormality noted; this may be an undesirable change and may be a factor producing changes later on in some but not in all subjects, with respect to the development of a significant degree of emphysema.

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Social Security Footnotes

THE "SOUNDNESS" of Social Security depends on compulsion, high employment, and no wars.

—From the Department of Public Relations, American Medical Association