THE EFFECTS OF SULFUR DIOXIDE UPON THE MECHANICAL PROPERTIES OF THE LUNGS IN DOGS

by

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Introduction

The harmful effects upon the workers of sulfur dioxide have been the important problems in many industries.¹⁾ The first report on this effect was one of Bérard et al.²⁾ in 1821 and since then, the studies on the effects due to industrial exposure of sulfur dioxide have been successively undertaken by many authors. On the other hand, with the great industrialization of the past several decades the contamination of the atmosphere by sulfur dioxide became almost inevitable and reached to the extent never before realized. Such an increasing concentration of sulfur dioxide in the atmosphere would produce an unfavorable effect upon man's health.³⁾

Experimental studies concerning the effect of exposure to sulfur dioxide have been attempted by many authors, but little has been studied about the details of its effect upon the pulmonary function until recently. The change of the tracheobronchial reflexes by sulfur dioxide was indicated by Widdicombe^{4,5)}. Amdur et al. studied the effects upon the pulmonary function, especially pulmonary resistance and compliance of guinea pigs and man⁶⁻⁹⁾. Balchum et al. studied pulmonary resistance and compliance with concurrent radioactive sulfur absorption, distribution and retention in dogs breathing $S^{35}O_2$ ¹⁰⁻¹³⁾. Toyama studied the effect upon airway resistance in man, using the airway

interruption technic.¹⁴⁾

According to the results of Amdur and Mead⁶⁾, quantitatively measurable changes in both the elastic properties of the lungs and the flow resistance of the lung and airway may be produced before any external sign of respiratory distress appear. From this aspect, these both factors may be useful measures determining the toxicologic effects of sulfur dioxide. On the other hand, extra load would be imposed to the function of many organs when the change in the mechanical properties of the lungs were produced by the exposure to sulfur dioxide. But there are some gaps yet to be filled about these problems.

From these considerations stated above, the measuring for the successive changes of ventilatory mechanics, including ventilatory work, and concurrently the change of arterial oxygen saturation during and after the inhalation of sulfur dioxide of a known concentration by dogs via tracheostomy was attempted by us.

Methods:

Preparation of gas mixtures:

Applying the technic by Balchum et al.,¹²⁾ various concentrations of sulfur dioxide in air were prepared in thick-walled vinyl bag 200 L. in volume. Sulfur dioxide was generated with reaction between copper and sulphuric acid in flask and then transferred into a 50 mL. glass tube attached leveling bulb apparatus containing mercury. A measured volume of sulfur dioxide, according to the concentration previously determined, was introduced into air entering the bag, thus making

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the mixture of sulfur dioxide in air to be as complete as possible. The air was measured into the bag by means of wet gas-meter connected to pump. The concentration of sulfur dioxide was measured by p-rosanilin-formaldehyde method of West and Gaeke¹⁵⁾ or fuchsin-formaldehyde method. The change of concentration of sulfur dioxide-air mixture in this bag was found to be within 10% in the periods of 2 or 3 hours.

Methods of measurement:

Rates of air flow in and out of the respiratory system were measured with pneumotachograph of Lilly type connected to a differential strain-gauge manometer.

Intrapleural pressure had been preliminarily measured with the method of intrapleural puncture. But it had been frequently occurred that the puncturing needle was clogged with blood coagulum so that the recording was disturbed. On the other hand, intrapleural pressure change can be evaluated indirectly from endoesophageal pressure measurements^{16,17)} although the absolute value of the endoesophageal pressure differs from the intrapleural pressure and especially the former tends to be higher the later in the supine position. Also according to the record of the dog in the supine position by the present authors (Fig. 1.) the change of both

Fig.1 Simultaneous recording of intrathoracic and endoesophageal pressure in dog.

pressures runned parallel, Thus, as a measure of transpulmonary pressure the pressure difference between the proximal end of tracheal cannula inserted into the trachea and a point within the lower third of the esophagus was recorded. Endoesophageal pressure was transmitted to one side of a differential strain-gauge manometer by means of an air-filled polyethylene tube 90cm. in length, 1.0mm. in internal diameter. The distal end of the tube was sealed with a thin-walled latex balloon 10cm. in length, 2.3 cm. in perimeter. A number of spirally arranged holes were made in the

tube wall covered by the balloon. The other side of the manometer was connected to the side-tube attached to the tracheal cannula by polyethylene tubing for the transmission of the pressure at the proximal end of the tracheal cannula. The volume of air in the balloon and tube during recording was 1.0ml.

Rates of air flow and pressures were recorded simultaneously via a direct-current amplifier on a direct-writing recorder. Volume changes were measured by planimetry of the areas under the curves of rate of air

flow. The pneumotachometer was calibrated using a method of water-replacement and pressure gauges were calibrated using a water manometer after each study.

By relating the change of both the transpulmonary pressures and volumes on each 0.1 second during respiratory cycle from the beginning of inspiration to the end of expiration, ventilatory pressure-volume loop was obtained, the area of which represented the mechanical work done on the lung in order to overcome viscous resistance. The elastic-like property of the lung was expressed as effective pulmonary compliance (Liter/cm.H₂O) by Mead et al.¹⁸⁾ and Otis et al.¹⁹⁾ The work required to overcome the elastic recoil force of the lung was estimated by the method of McIlroy et al.²⁰⁾ and Comroe et al.²¹⁾ Total work done on the lung was calculated by adding the elastic work, inspiratory viscous work and additional expiratory viscous work, following the method of Cherniack.²²⁾ Each work was expressed as the values per liter of ventilation (gram meter) by dividing the work per respiration by the liter volume of breath. Viscous

resistance was calculated separately on inspiration and expiration following the method of Frank et al.²³⁾ In some cases, air flow-resistive pressure diagram was obtained. Because variation occurred due to changes in endoesophageal pressure accompanying cardiac contraction, average values for compliance, resistance and ventilatory work were calculated from at least 6 measurements. Arterial oxygen saturation was estimated by manometric method of van Slyke-Neil.

Experimental Procedure:

The 11 dogs were exposed to concentration of sulfur dioxide in air ranging from 50 to 350 p.p.m. and the 2 dogs were studied as control dogs, breathing air alone for about 2 hours. Measurements were all performed in the supine position. Dogs were anesthetized with subcutaneous morphine hydrochloride followed with intraperitoneal urethane, thus allowing the dogs to breathe spontaneously with as possible as constant respiration throughout the experiment.

Schema of experimental procedures was indicated in Fig. 2; Tracheal cannula was

Fig. 2 Schema of SO₂-exposure experiment.

inserted into the trachea opened by means of tracheostomy and connected via 3-way stop-cock to low resistant respiratory valve. Pneumotachograph was connected to other

one arm of 3-way stop-cock. At the recording of rate of air flow, 3-way stop-cock was turned toward pneumotachograph for about 1 minutes, through which dog breathed air.

Fig.3 Successive change of ventilatory pressure-volume loop and air flow-resistive pressure diagram in SO_2 -exposed dogs: No.15. (a), No.14. (b). C., G. and N. represent control period, exposure period and 30 min. after the end of the exposure, respectively, and the number by G represents the time(min.) after the start of the exposure.

Esophageal balloon was passed to the lower third of the esophagus by oral route. Blood-sampling needle was held in femoral artery. That the balloon had been situated at right position was confirmed with autopsy after the finish of experiment.

A control period of about 30 minutes on air alone was followed by 40 to 60 minutes exposure period during which dog inhaled sulfur dioxide-air mixture from the bag which was connected to respiratory valve. Following the exposure period, the dog was allowed again to breath air for about 30 minutes. Throughout the experiment recording of the rates of air flow and transpulmonary pressures were made at about 10 minutes intervals, and serial arterial blood samples were drawn.

Results:

Ventilatory pressure-volume loop and air flow-resistive pressure diagram obtained in two cases were given in Fig. 3 a, b. In No. 15., having breathed 50 p.p.m. sulfur dioxide for 40 minutes, the marked increase in the area of ventilatory pressure-volume loop, namely, viscous work, resulting from the marked increase in viscous resistance, was noted. These values for viscous resistance and work were decreased toward the control-level, when the dog was again breathing air after the end of exposure. On the other hand, effective pulmonary compliance was almost unchanged throughout the experiment. In No. 14., having breathed 100 p.p.m. sulfur dioxide for 60 minutes, resistance and work remained elevated or continued further increasing when the dog was breathing air again after the end of exposure, although the change of these values during the exposure period followed the similar pattern observed in No. 15. . In air flow-resistive pressure diagram of these 2 dogs the similar pattern of changes was noted, viscous resis-

tance being increased by the exposure to sulfur dioxide.

The results of measuring for tidal volume, respiratory rate, arterial oxygen saturation and ventilatory mechanics in dogs at the end of exposure and 30 minutes after the end of exposure were listed in Table 1..

In control dogs, the values for ventilatory mechanics and arterial oxygen saturation were almost constant throughout the observation period, while the slight variation in tidal volume and respiratory rate were noted.

In exposed dogs, the change of tidal volume and respiratory rate was found to be the same order of the change observed in control dogs. Effective pulmonary compliance was almost unchanged, the percentage change ranging from -4.7% to $+9.6\%$ at the end of exposure, while the similar finding was noted in the compliance 30 minutes after the end of exposure, thus the percentage change ranging from -1.9% to $+12.4\%$. The increase in viscous resistance and viscous work was so marked that the striking contrast presented to the results obtained in the control dogs, while the increase in total work was less remarkable than in viscous work. There was no significant relationship between the change of these mechanical values and the concentration of sulfur dioxide exposed. The average percentage increase of inspiratory and expiratory resistance at the end of exposure were 346% and 268%, respectively, although individual values scattered considerably widely. Average percentage increase by 228% and 235% were noted for inspiratory and expiratory resistance, respectively, 30 minutes after the end of exposure, although the further increases beyond the values during the exposure period were found in some cases. Average percentage increase of viscous work at the end of exposure was 296%,

Table I. Result of SO₂-Exposure.

Control Dog												
No.	Wt. kg		Exp. Time	TV	RR	SaO ₂	EC	IR	ER	VW/LV	TW/LV	
No.2	8.0		0'	136.0	11.0	89.3	0.060	0.28	0.34	1.04	19.88	
		Room air	30'	148.0	11.0							
			60'	176.0	11.0	91.5	0.064	0.32	0.30	1.06	18.80	
			130'	128.0	12.0	93.7	0.065	0.28	0.32	1.00	19.70	
			170'	184.0	13.0	89.5						
No.17	7.3		0'	144.6	12.7	99.3	0.029	0.67	0.70	4.43	27.10	
		Room air	30'	161.9	12.9		0.030	0.41	0.56	3.97	28.35	
			60'	151.8	13.3	100.0	0.030	0.18	0.49	2.79	25.95	
			70'	181.5	13.7							
			90'	167.3	14.0	99.9	0.027	0.42	0.60	2.17	27.60	
Exposed Dog												
No.	Wt. kg	Exp. Time	SO ₂ ppm	Control	TV	RR	SaO ₂ %	EC	IR	ER	VW/LV	TW/LV
No.1	8.0	52'	50	Control	107.0	32.5	94.7					
				End of exp.	119.0	26.0	84.6					
No.15	18.0	40'	50	Control	298.3	11.3	101.0	0.068	0.10	0.64	0.99	19.85
				End of exp.	309.0	11.6	101.0	0.072	1.45	1.79	8.90	22.25
				30' aft. end.	264.7	11.9	102.7	0.072	0.86	0.64	5.05	21.95
No.16	7.0	54'	60	Control	177.5	11.5	99.1	0.042	0.83	0.93	3.38	24.15
				End of exp.	194.1	11.8	98.9	0.040	1.44	1.45	7.00	26.55
				30' aft. end.	195.5	12.3	99.8	0.045	-0.14	0.36	1.18	19.10
No.13	5.0	55'	100	Control	148.4	15.6	98.7	0.059	0.13	0.19	0.81	13.20
				End of exp.	183.9	16.3	98.7	0.061	0.97	2.00	4.76	13.40
				30' aft. end.	188.5	16.7	99.1	0.058	0.56	0.87	2.68	17.30
No.14	7.0	60'	100	Control	134.4	10.2	100.0	0.031	0.54	0.46	1.04	21.75
				End of exp.	185.4	12.0	100.0	0.034	1.06	1.02	4.50	28.30
				30' aft. end.	164.0	12.1	98.3	0.034	1.10	1.86	6.10	25.75
No.11	6.8	50'	150	Control	215.0	14.0	96.0					
				End of exp.	229.0	14.8	97.2					
				30' aft. end.	187.0	15.5	96.7					
No.9	7.0	40'	170	Control	157.7	15.9		0.040	0.41	0.29	2.00	20.25
				End of exp.	151.0	17.4		0.043	1.18	1.76	3.59	21.20
				30' aft. end.	156.0	16.3		0.043	-0.86	2.69	2.39	15.00
No.11	11.5	40'	270	Control	250.8	18.6	100.5	0.108	0.52	1.17	5.05	13.10
				End of exp.	262.5	17.3	101.9	0.108	1.39	1.43	10.55	16.75
				30' aft. end.	247.6	19.6	101.4	0.111	0.76	2.24	19.00	13.50
No.7	15.0	35'	350	Control	104.5	16.1	89.0	0.034	4.69	3.04	8.00	20.50
				End of exp.	121.7	14.7		0.034	6.77	6.21	13.60	25.10
				30' aft. end.	153.5	13.9	89.6	0.034	5.56	5.56	14.50	23.10
No.5	7.5	60'	350	Control	108.0	14.0	99.3					
				End of exp.	212.0	10.0	96.1					
				30' aft. end.	157.0	11.5						
No.12	3.0	60'	350	Control	198.3	9.2	89.6	0.030	0.79	0.99	2.22	32.75
				End of exp.	213.4	12.8	91.0	0.030	2.45	1.92	10.80	35.40
				30' aft. end.	202.8	11.5	93.5	0.030	1.65	0.81	5.30	33.95

※ These values were omitted for statistical analysis.

TV: Tidal Volume (ml.)

RR: Respiratory Rate

SaO₂: Arterial Oxygen Saturation (%)

EC: Effective pulmonary Compliance (L/cmH₂O)

IR: Inspiratory Resistance (cmH₂O/L./Sec.)

ER: Expiratory Resistance (cmH₂O/L./Sec.)

VW/LV: Viscous Work per Liter of Ventilation (g.m.)

TW/LV: Total Work per Liter of Ventilation (g.m.)

while total work was increased by 15%. Either further increasing or decreasing of ventilatory work compared the values during the exposure period were noted 30 minutes after the end of exposure. Average percentage increase by 235% and 8% were

found for viscous and total work, respectively. The change of ratio of expiratory time to inspiratory time, calculated from the curve of air flow, by the exposure to sulfur dioxide, was presented in Fig. 4. . In control dog this ratio was almost con-

Fig. 4 Successive change in ratio of expiratory time to inspiratory time of control dog (No. 17) and SO_2 -exposed dogs. Vertical lines on the graph of exposed dogs indicate the time of the start and the end of the exposure to SO_2 .

stant for 2 hours, while in exposed dogs this ratio rose considerably during the exposure period.

Arterial oxygen saturation was almost unchanged throughout the experiment in all dogs except one (No. 1.), arterial oxygen saturation of which was decreased by 10% at the end of exposure.

Discussion :

According to the recommendation by the Committee on Threshold Limit of the

American Conference of Governmental Industrial Hygienist in April, 1958,²⁴⁾ threshold value for sulfur dioxide was 5 p.p.m. . On the other hand, Henderson and Haggard²⁵⁾ indicated 50-100 p.p.m. as the maximum allowable for men for exposure up to 1 hour and 400~500 p.p.m. as dangerous for even short exposure. Kisskalt²⁶⁾ indicated to be unable to breath 300 p.p.m. sulfur dioxide any length of time. Referring the concentration reported by them, we have chosen

the concentration of sulfur dioxide used in the experiment.

The elastic-like property of the lung was expressed as effective pulmonary compliance, according to Mead et al.¹⁸⁾ and Otis et al.¹⁹⁾ According to the result by the present authors, effective pulmonary compliance was regarded almost unchanged at the end of exposure to sulfur dioxide, considering the technical errors accompanied in measurements of such a small change of transpulmonary pressure or tidal volume as in dogs. From this finding no evidence suggesting the alternation of elastic-like property of the lungs of the exposure to sulfur dioxide was obtained.

Viscous resistance was calculated following the method of Frank et al.²³⁾. In assessing the values obtained for viscous resistance, however, consideration must be given to the fact that the compliance is a component of the calculation. As Cherniack²²⁾ and Shiraishi²⁷⁾ indicated, the line representative of the compliance will not be a straight line joining end expiration and end inspiration, but will be a curved line proportional to the rate of air flow. Hence, it is necessary to pay more attention to the interpretation of the results for viscous resistance. In fact, there were some cases which showed negative inspiratory resistance. Despite these considerations, there were the marked increase of both inspiratory and expiratory resistance of dogs at the end of exposure to sulfur dioxide. The marked increase in viscous resistance was also indicated by air flow resistive pressure diagram. The ratio of expiratory time to inspiratory time was increased during the exposure period, this finding being consistent with the increase of the resistance to breathing. The prolongation of expiratory time in the patients with the obstructive impairment of breathing have been in general accepted, while

Cain and Otis²⁸⁾ showed the increase of this ratio by the addition of external resistance to the mouth of man.

According to the results of Amdur and Mead by means of bodyplethysmograph and intrapleural puncture technic, the exposure to sulfur dioxide ranging from 26 p.p.m. to 350 p.p.m. for 1 hour resulted the moderate decrease in compliance and the marked increase in resistance of normal guinea pigs and tracheostomized guinea pigs. Balchum et al.¹⁰⁻¹³⁾ indicated the slight decrease in the compliance of the lungs and thorax and the marked increase in nonelastic resistance to breathing occurred in dogs breathing sulfur dioxide-air mixture under control respiration. The reason why these discrepancy between the results as to the compliance by Amdur and Mead, Balchum et al. and the present authors have occurred is not clear. A probable cause is the difference of experimental animals. The lungs in guinea pigs have the highest proportion of smooth muscles among the lungs in all mammals. And the response of smooth muscles of bronchioles to histamine, foreign protein and anaphylaxis differ distinctly due to species of animals, for example, the marked contraction of bronchioles of guinea pigs was induced when the high concentration of histamine was injected intravenously, while the degree of the response in dogs was much lower.²⁹⁾ Furthermore, guinea pigs are considered most sensitive among the experimental animals to irritant gases. It may be possible that more remarkable contraction of bronchioles and the accompanying greater response of pulmonary vessels in guinea pigs than in dogs are produced by the exposure to sulfur dioxide so that effective pulmonary compliance in guinea pigs are more affected by the exposure to sulfur dioxide. On the other hand, Amdur³⁾ pointed out the probable

reduction of the amount of gas reaching the lung in the case of a soluble gas. However, because of no data in hand, conclusion as to this effect on compliance was not established.

The conclusion we can draw from this data is that sulfur dioxide has an action to increase the viscous resistance in the lungs. Amdur and Mead⁶⁾ and Balchum et al.¹¹⁾ considered this action of sulfur dioxide either the stimulation of nerve endings or a direct action upon the bronchial muscles. The data for the action of sulfur dioxide upon the bronchi by Widdicombe^{4,5)} indicated that nerve fibers in the cat bronchi were stimulated by sulfur dioxide. According to the results by the present authors, the resistance in some cases remained elevated or continued further increasing 30 minutes after the end of exposure. This finding would suggest that the actual change of the tissue in the airway was also involved, either a edema of airway wand or increased mucous secretion with consequent narrowing of airway, although such a factor as reflex contraction of bronchi by the exposure could not be neglected, because it was probable that reflex activity was depressed under the condition by anesthesia.

A marked increase in work was thus required in order to overcome the viscous resistance. Although elastic work was not indicated in table, elastic work was almost unaltered because of almost unchanged compliance. Total work was increased as much as the increase of viscous work. That the percentage increase of total work was less marked was due to the smaller proportion of viscous work to total work, the average value being 15.7% during the control period. Amdur⁸⁾ indicated likewise the increase in ventilatory work of guinea pigs produced by low concentration of sulfur dioxide. Thus the increase of viscous resistance to

breathing imposed an added load of ventilatory work on animal, although it is necessary to be further investigated what degree of stress could be produced by the change in ventilatory work of this order of magnitude.

There was no evident relationship between the change of resistance or work and concentration of sulfur dioxide exposed, the reason of which was not clear. Toyama³⁰⁾ indicated that no relationship between the change of airway resistance in man and the concentration of sulfur dioxide inhaled was found when sulfur dioxide was mixed with the larger particles of H_2O_2 mist, which might act mainly on upper airway.

On the other hand, no significant reduction of arterial oxygen saturation was not noted in all dogs except one breathing sulfur dioxide under the condition performed by the present authors, this finding suggesting that significant alveolar hypoventilation, nonuniformity of ventilation-perfusion relationship or impairment of diffusing capacity would not be yet produced.

The quantitative aspects of our data obtained in anesthetized dogs can by no means be extrapolated to man. The effect upon the mechanical properties of the lungs in man by the exposure to sulfur dioxide, however, can be understood at least qualitatively from working with dogs. We think careful control of lung function on employees may be needed in industries occurring sulfur dioxide. On the other hand, as Amdur stated,⁸⁾ the increase of respiratory work might be kept in mind for future consideration from the point of view of a stress superimposed upon existing respiratory impairment. Oxygen cost of breathing in the patients with chronic pulmonary emphysema is higher than in normal subjects.³¹⁻³³⁾ Even slight but prolonged increasing in ventilatory work, which is insignificant in

normal subjects, would probably overtax such a patient. According to the data by Motley et al,³⁴⁾ the severity of emphysema was exaggerated by Los Angeles smog. Furthermore it has been indicated by the mortality statistics of London³⁵⁾ that those who have had pre-existing respiratory or cardiac impairment would be most sensitive to acute air pollution incidents.

Summary:

For the purpose of clarifying the effect of sulfur dioxide upon the pulmonary function, ventilatory mechanics by means of endoesophageal pressure technic and arterial oxygen saturation were studied in anesthetized dogs breathing sulfur dioxide-air mixture via a tracheostomy. The results obtained were as follows;

- 1) No significant change in effective pulmonary compliance by the exposure was noted.
- 2) Viscous resistance and viscous work were markedly increased by the exposure to sulfur dioxide, while the increase in total work was less marked. During post-exposure period, these values for resistance and work were either decreased toward the control level or remained elevated.
- 3) Arterial oxygen saturation was almost unchanged throughout the exposure period and post-exposure period.

These data indicated that sulfur dioxide had an action to increase the ventilatory viscous work.

Significance of extra load on pulmonary function imposed by the increase of the viscous work was discussed by the present authors.

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References:

- 1) Greenwald, I.: Arch. Indust. Hyg. Occup. Med. 10;455, 1954
- 2) Bérard, S. et al.: quoted from 1)
- 3) Pemberton, J.: in "Air Pollution", Butterworths Scientific Publication, London, 1957
- 4) Widdicombe, J. G.: J. Physiol. 123;55, 1954
- 5) Widdicombe, J. G.: J. Physiol. 123;71, 1954
- 6) Amdur, M. O. and Mead, J.: Proceedings of the 3rd National Air Pollution Symposium. pp.150, 1955
- 7) Amdur, M. O.: Amer. Indust. Hyg. Assoc. Quart. 18;149, 1957
- 8) Amdur, M. O.: International J. Air. Pollut. 1;170, 1959
- 9) Amdur, M. O. et al.: Lancet. 2;758, 1953
- 10) Balchum, O. J. et al.: Fed. Proc. 18;6, 1959
- 11) Balchum, O. J. et al.: Amer. J. Physiol. 197;1317, 1959
- 12) Balchum, O. J. et al.: J. Appl. Physiol. 15;62, 1960
- 13) Balchum, O. J. et al.: Arch. Indust. Health. 21;564, 1960
- 14) Toyama, T.: Japanese J. Indust. Health. 4;86, 1962 (In Japanese)
- 15) West, P. W. and Gaeke, G. C.: Anal. Chem. 28;1816, 1956
- 16) Mead, J. et al.: J. Appl. Physiol. 7; 491, 1954-55
- 17) Mead, J. and Gaensler, E. A.: J. Appl. Physiol. 14;81, 1959
- 18) Mead, J. and Whittenberger, J. L.: J. Appl. Physiol. 5;779, 1953
- 19) Otis, A. B. et al.: J. Appl. Physiol. 8; 427, 1956
- 20) McIlroy, B. B. et al.: Clin. Sc. 13;127, 1954

- 21) Comroe, J. H. Jr., et al.: The Lung, Clinical Physiology and Pulmonary Function Tests, Chicago, Year Book Publishers., 1955
- 22) Cherniack, R. M.: J. Clin. Invest. 35; 394, 1956
- 23) Frank, N.R. et al.: J. Clin. Invest. 36; 1680, 1957
- 24) Committee on Threshold Limits, American Conference of Governmental Industrial Hygienists: Arch. Indust. Health. 18;178, 1958
- 25) quoted from 6)
- 26) quoted from 1)
- 27) Shiraishi, T.: Tokyo J. Med. Sci. 67; 709, 1959 (In Japanese)
- 28) Cain, C. C. and Otis, A. B.: J. Aviat. Med. 20;149, 1949
- 29) Field, M.E. and Drinker, C. K.: Am. J. Physiol. 93;138, 1930
- 30) Toyama, T.: Clinics Res. Organ. 16; 621, 1961 (In Japanese)
- 31) Cherniack, R. M.: J. Clin Invest. 38; 494, 1959
- 32) Campbell, E. J. M. et al.: J. Appl. Physiol. 11;303, 1957
- 33) McGregor, M. and Becklake, M. R.: J. Clin. Invest. 40;971, 1961
- 34) Motley, H. L. et al.: J.A.M.A. 171;1469, 1959
- 35) Mortality and Morbidity during the London Fog of December, 1952.: Reports on Public Health and Medical Subjects. No. 95, 1954, London: Her Majesty's Stationary office.

SO₂ の犬の換気力学に及ぼす影響について

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SO₂ 吸入の肺機能に対する影響を明らかにする目的をもって, 11頭の麻酔犬に 50~350ppm の SO₂ を 40~60分吸入せしめ, 食道内圧法による換気力学諸値及び動脈血 O₂飽和度の変化を追求した。

(1)有効肺圧縮率は吸入によりほとんど不変であった。

(2)粘性抵抗及び粘性仕事の増加は顕著であった。しかし吸入終了後30分では, 吸入前値に恢復傾向を示すものと然らざるものとが存在した。なお全力的な仕事は, 粘

性仕事分だけ増加した。

(3)動脈血 O₂ 飽和度は吸入によりほとんど不変であった。

即ち SO₂ は, 呼吸粘性抵抗を上昇し, それにより呼吸粘性仕事を増加せしめる作用を持つ。この呼吸仕事の増加が長期持続する場合いかなる負担を人にあたえるかは不明であるが, 少なくとも心肺疾患患者には, 有意の負担を与える可能性があると思われる。

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