

THE PHYSICAL PROPERTIES OF NORMAL LUNGS REMOVED AFTER DEATH

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It has been known for over a century that the lungs retain some of their elastic properties after death. Carson (1820) was the first to investigate the elastic recoil of the lungs by measuring the force required to inflate them, and this type of experiment has been repeated by numerous investigators with confusing results. The only conclusion that can be drawn from the literature is that the elastic properties of normal lungs after death are unpredictable. In the experiments to be described the lungs have been inflated by negative pressure in a plethysmograph, and some of the factors responsible for changes in elastic behaviour after death have been analysed in an attempt to explain why previous investigations have not given consistent results.

Pulmonary elasticity has been defined as the ability of the lungs to recover their original size by elastic recoil when a force deforming them is removed. Until recently the lungs have been thought to obey Hooke's law, which states that the deformation of an elastic body is directly proportional to the force applied. This is a theoretical law to which no known material conforms. Recent work on the elastic properties of biological materials suggests that the lungs are more probably visco-elastic. A visco-elastic system is a combination of a viscous system in which the response to a force depends on the time the force is applied, and an elastic system in which the response depends on the magnitude of the force applied.

METHODS

The material studied consisted of human lungs removed between six and 72 hours after death, and animal lungs, in some cases removed immediately after death. Before removal a brass tube was inserted into the trachea at about the level of the cricoid cartilage and clamped in position with a "jubilee" clip. A water manometer was connected to the intratracheal tube and the pressure generated by elastic recoil measured on opening the pleural cavity. The lungs, with the intratracheal tube occluded to prevent the escape of air, were then

removed with the visceral pleura intact; the heart was cut off by severing the great vessels and the lungs set up in the plethysmograph without delay.

The plethysmograph consisted of an air-tight perspex box standing on a perspex tray with a mercury seal (Fig. 1). The lungs were suspended in the box with the intratracheal tube projecting through the top. The pressure in the box was reduced either by a vacuum pump or a simple hand pump until the pressure in the trachea was atmospheric. The intratracheal tube was then connected to the spirometer. Pressure changes in the plethysmograph were measured by a water manometer recording on a kymograph, or by a capacitance gauge. Although the water manometer showed lag and overswing, the error involved in its use was shown to be negligible under the conditions of the experiments. The volume of air passing in and out of the lungs was measured by a spirometer and recorded on the kymograph drum below the pressure.

Using the plethysmograph, it was possible to ventilate the lungs and measure their volume by the helium dilution method using the circuit described by Bates and Christie (1950). The intratracheal tube was connected to the lung volume circuit and the lungs ventilated until mixing was complete. Helium in air was used instead of helium in oxygen to eliminate the initial period of ventilation with oxygen.

RESULTS

From measurements of lung volume after inflation in the plethysmograph it was clear that the volume of air in the lungs when they were first set up in the plethysmograph was small. This finding suggested that air had escaped from the lungs between the time of death and removal of the lungs from the thorax. To confirm this suggestion a series of experiments was carried out.

In four cases the lungs were allowed to deflate completely *in situ* by opening the pleural cavity with the intratracheal tube connected to a bag. The volumes of air expelled are shown in Table I. The largest volume (650 ml.) was expelled in a case investigated six hours after death. In three other patients investigated between 33 and 65 hours after death the volume expelled was less than 200 ml.

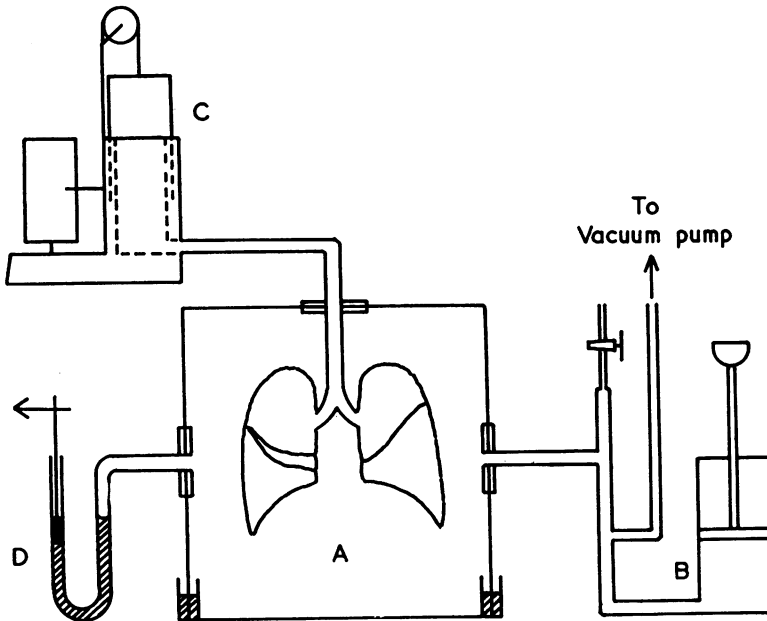


FIG. 1.—Diagram of apparatus. A=airtight plethysmograph; B=hand pump; C=recording spirometer; D=water manometer.

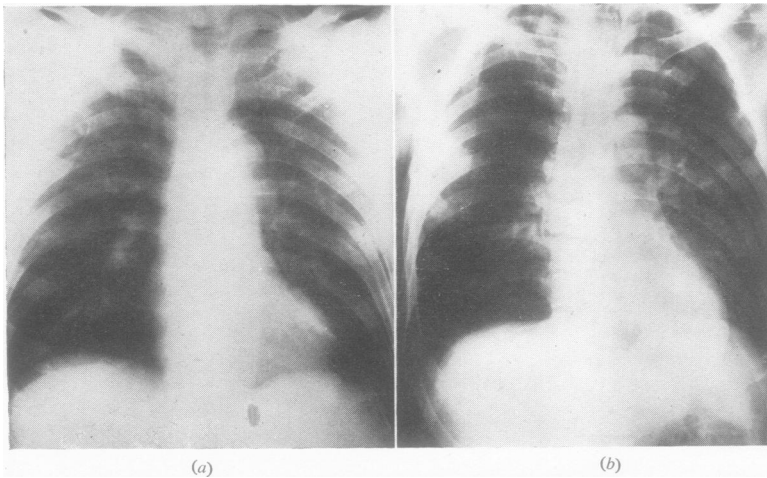


FIG. 2.—Radiographs of chest (a) ante-mortem and (b) post-mortem, showing ascent of diaphragm after death.

TABLE I
VOLUMES OF AIR EXPELLED FROM THE LUNGS BY ELASTIC RECOIL ON OPENING THE PLEURAL CAVITIES AT NECROPSY

Time After Death (hours)	Age (years)	Intra-tracheal Pressure (cm. H ₂ O)	Volume of Air Expelled (ml.)	Cause of Death
6	59	3.5	650	Perforated gastric ulcer
33	44	2.8	60	Cerebral haemorrhage
57	56	5.2	120	"
65	65	3.4	180	Cerebral tumour

The volume of air that would be expelled if the pleural cavities were opened during life is probably of the order of 1,500 ml., and the small volumes expelled in these post-mortem experiments, particularly those between 33 and 65 hours after death, suggested that the lungs had deflated in the period between death and necropsy.

The change in lung volume after death was further investigated by passing a cuffed intratracheal tube soon after death and collecting the expelled air in a bag. Six cases were investigated in this way, the intratracheal tube being inserted under direct vision between 20 minutes and two and a half hours after death. Results are shown in Table II. It can be seen that the sooner the collection of air was started after death the larger the volume collected. In the case in which the intratracheal tube was inserted 20 minutes after death 845 ml. was collected, 550 ml. out of this total being collected in the first two hours.

Reduction in lung volume after death was also investigated by comparing radiographs taken before and after death. Antero-posterior films were taken in two patients before and 24 hours after death. In each case the position of the patient—supine at the end of expiration—and the tube distance were the same. The difference

between ante-mortem and post-mortem radiographs is seen in Fig. 2; there is clearly ascent of the diaphragm after death. This was confirmed by planimetric measurement of the area of lung fields which showed a 12½% reduction after death.

These findings confirmed that a large amount of air was expelled from the lungs between death and necropsy, probably by ascent of the diaphragm. As a result, the lungs, when first set up in the

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TABLE II
VOLUMES OF AIR EXPELLED FROM THE LUNGS
BETWEEN DEATH AND NECROPSY

Time of In- sertion of Tube (minutes after death)	Age (years)	Volume of Air Collected (ml.)	Cause of Death
20	59	550 in 2 hours	Carcinoma of pancreas
		820 „ 4 „	
		845 „ 22 „	
60	55	535 „ 17 „	Coronary thrombosis
120	59	310 „ 4 „	
120	66	200 „ 2 „	Carcinomatosis
120	78	100 „ 16 „	Pulmonary embolism
150	38	150 „ 2 „	Acute leukaemia
		200 „ 3½ „	
		225 „ 19¼ „	

plethysmograph, were not inflated to the same volume as they had been in life.

PLETHYSMOGRAPH EXPERIMENTS. — The first expansion of the lungs in the plethysmograph was produced by reducing the pressure in steps of 2 cm. H₂O to about 10 cm. below the starting pressure, and a typical tracing is shown in Fig. 3. During inflation collapsed areas on the surface of the lungs were seen to expand one after another. The time needed for equilibrium to be reached at intermediate pressures was so long that a serious attempt to reach equilibrium was only made after the last step. The lungs were then allowed to recoil by increasing the pressure to its original level in similar steps. The volume change was

larger with each successive step, indicating that the lungs did not obey Hooke's law. Equilibrium was readily reached after each pressure step during recoil, but the lungs failed to recoil to their starting level.

In the experiment shown in Fig. 3 the post-mortem lung volume was initially 1,346 ml. After the first inflation the lungs recoiled to a volume of 2,206 ml., a value within the normal range of the functional residual air. A comparison between the post-mortem lung volume and the lung volume after inflation in 13 normal lungs is given in Table III, from which it can be seen that the first inflation of the lungs in the plethysmograph restored the lungs to a volume more comparable with that in life, presumably by opening up alveoli which had become airless after death.

The lung volume and presumably the number of alveoli opened up in this first inflation depended on how far the plethysmograph pressure was reduced. The arbitrary reduction to 10 cm. H₂O below the starting level was found to be satisfactory, for the alveoli on the surface of the upper lobes of the lungs were usually all expanded at this level.

The opening up of previously collapsed alveoli resulted in a complicated relation between pressure and volume which varied with each inflation as shown in Fig. 4. At A, when the pressure was -4.8 cm. H₂O, the lung volume was 770 ml. above the

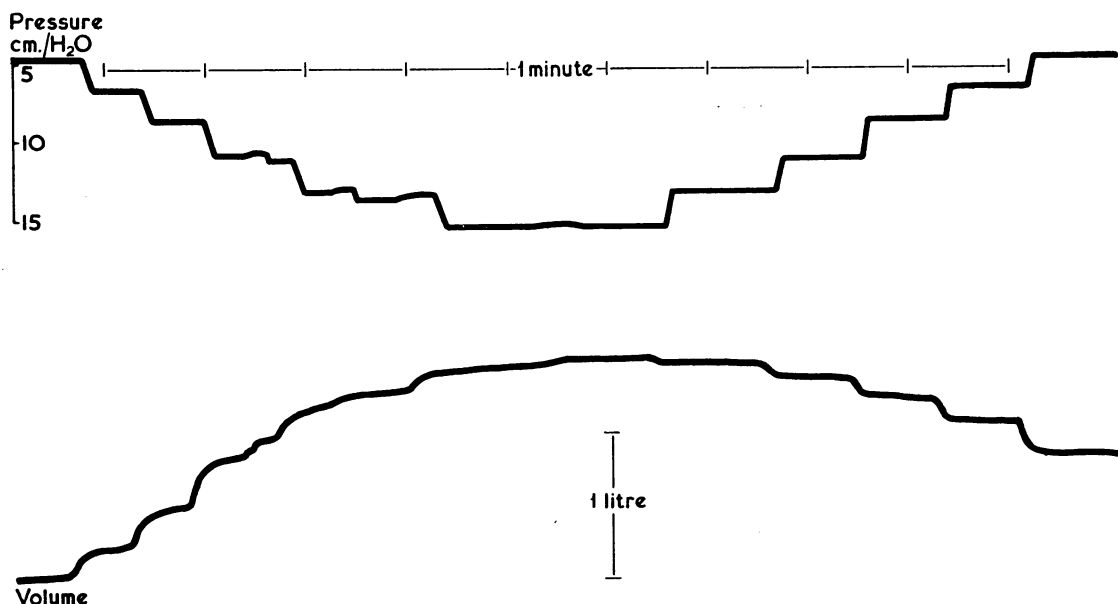


FIG. 3.—Tracing of first inflation of normal lungs in plethysmograph, showing failure to obey Hooke's law. Upper tracing represents plethysmograph pressure and lower tracing volume of air passing in and out of the lungs.

TABLE III
RESULTS IN EIGHTEEN NORMAL HUMAN AND FOUR DOGS' LUNGS INVESTIGATED AT DIFFERENT INTERVALS AFTER DEATH

Time after Death (hours)	Age (years)	Intratracheal Pressure (cm. H ₂ O)	Post-mortem Lung Volume (ml.)	Lung Volume after Inflation (ml.)	Diagnosis	Remarks
1	—	5			Normal dog	Unrefrigerated
1	—	5.9			" "	"
1	—	4.0			" "	"
12	78	4.2	820	3,960	Coronary thrombosis	
13	61	3.9	450	2,867	" "	
14	28	5.2			Fractured skull	
16	49	5.1	775	3,400	Coronary thrombosis	
21	55	3.4	410	2,589	" "	Bronchial obstruction due to inhaled vomit
21	68	2.8	800	2,560	Aortic stenosis	
21	64	4.7	380	3,160	Coronary thrombosis	
22	53	4.5	1,346	2,206	Carcinoma of cervix	
24	—	7.0		960	Normal dog	
24	70	4.5	1,110	3,906	Coronary thrombosis	
27	56	3.6	555	2,785	" "	
33	44	2.8	1,240	3,510	Cerebral haemorrhage	
48	50	6.2	1,560	2,610	Coronary thrombosis	Unrefrigerated
49	61	6.0	690	1,470	" "	"
49	50	—	—	3,324	" "	"
72	46	4.0			" "	"
72	64	3.7	1,535	2,835	" "	Lungs inflated with 400 ml. of air before removal
72	70	4.7			" "	"
72	60	3.0			" "	"

base line. After inflation B the pressure was increased to -4.8 cm. H₂O, but the volume level only returned to 850 ml. above the base line. After inflations C and D to -9.6 and -12.0 cm. H₂O the lung volumes at a pressure of -4.8 cm. H₂O were 1,020 and 1,800 ml. above the base line. In this experiment the lung volume at a pressure of -4.8 cm. H₂O depended on the negative pressure to which the lungs had previously been subjected. Each time the pressure was reduced below its previous lowest level new alveoli opened and as a result the lung volume at a pressure of -4.8 cm. H₂O became larger. When the

plethysmograph was opened to the atmosphere at the end of the experiment the lung volume returned to its original level. From this tracing it can be seen that the response of dead lungs to changes in pressure in the plethysmograph clearly depends on the previous history of inflation.

Another factor complicating the response of the lungs is the time taken to reach equilibrium following a change in pressure. In Fig. 3 two minutes were required before equilibrium was reached after inflation. Again in Fig. 4 one and half minutes were required for equilibrium to be reached at D, and on opening the plethysmograph

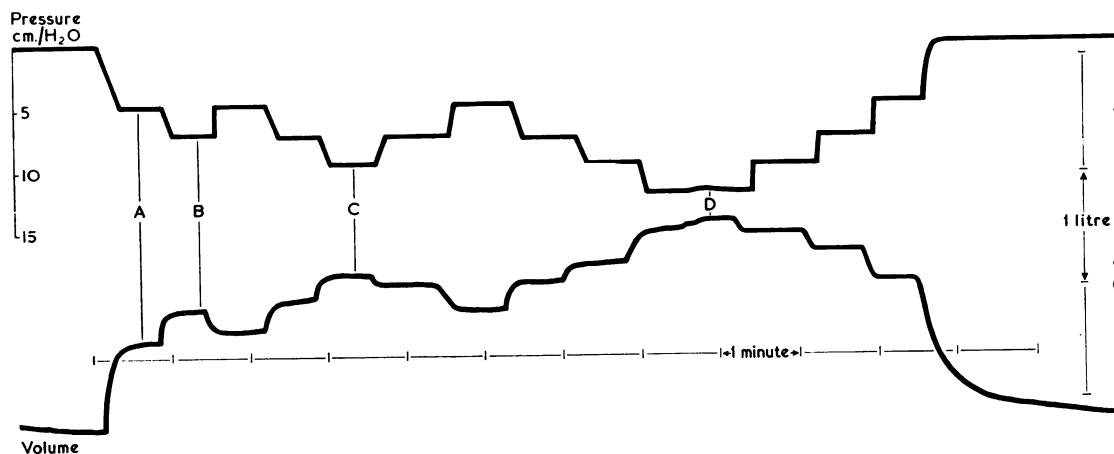


FIG. 4.—Tracing similar to Fig. 3, showing progressive increase in lung volume at plethysmograph pressure of -4.8 cm./H₂O, due to opening up of alveoli. After inflation A to -4.8 cm./H₂O lung volume is 770 ml. above base line; after inflation B to -7.1 cm./H₂O 850 ml. above base line; and after inflations C and D to -9.6 and -12.0 cm./H₂O 1,020 ml. and 1,800 ml. above the base line.

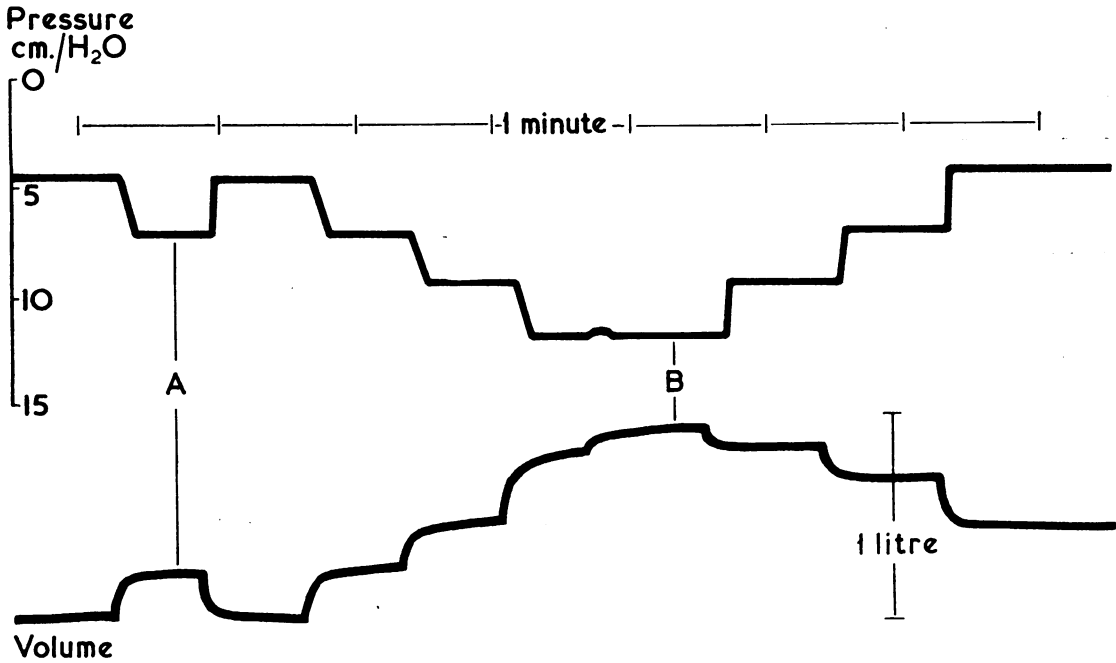


FIG. 5.—Tracing similar to Fig. 3, illustrating hysteresis after a large inflation. After smaller inflation A the lungs recoil completely. After the larger inflation B the lungs show hysteresis and fail to recoil completely.

to the atmosphere at the end of the experiment the lungs required time to reach equilibrium. The fact that time is required for equilibrium to be reached on inflation and recoil is evidence of the visco-elastic nature of the response of the lungs.

Variation in the number of alveoli open at different stages of inflation and the comparatively long time needed for equilibrium to be reached following a change of pressure were together responsible for the phenomenon of hysteresis, which was observed in all the lungs investigated. The term "hysteresis" has been used to describe the state of affairs in which the pressure-volume relationship on inflation differs from that on

deflation. It is best illustrated by an example (Fig. 5). In the first inflation, A, there was no hysteresis, the volume at a given pressure level being the same before and after inflation. In the larger second inflation, B, there was hysteresis, for the volume at intermediate pressure steps during recoil was greater than at similar pressures during inflation. Hysteresis was observed in all inflations in which the pressure was reduced sufficiently to open up alveoli and was more marked when time was not allowed for equilibrium to be reached after inflation.

From the preceding description of the complex nature of the response of the lungs to inflation, it

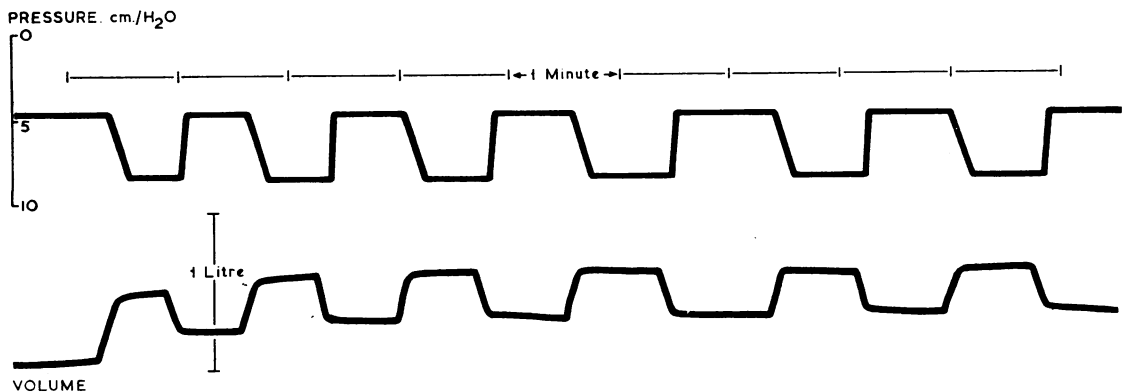


FIG. 6.—Tracing similar to Fig. 3, showing constant response to a constant pressure change repeated at intervals.

TABLE IV
CHANGES IN DISTENSIBILITY* OF ANIMAL LUNGS
AFTER DEATH

Animal	Time after Death	Distensibility
Dog	30 minutes	0.96
	35 "	1.37†
	3 hours	1.55
	7 "	2.0
	24 "	3.8
	30 "	4.8
Dog	30 minutes	1.55
	40 "	1.8‡
	7 hours	2.0
	26 "	3.6
Rabbit	10 minutes	62
	1½ hours	58
	3 "	75
	19 "	100
	23 "	118
Rabbit	10 minutes	80
	1½ hours	88
	3 "	98
	8½ "	100
	19 "	230

* Distensibility is expressed as force in cm./H₂O required to distend the lungs by 100 ml.

† After opening plethysmograph to atmospheric pressure.

‡ After increasing plethysmograph pressure to -1 cm./H₂O.

is clear that distensibility varies with the lung volume and previous history of inflation in a complex and almost unpredictable manner. It was also found to vary with the passage of time after death. The results of experiments showing a progressive increase after death in the force required to inflate the lungs are given in Table IV.

The only predictable features which were demonstrated in these experiments concerned the elastic recoil of the lungs. The positive pressure generated in the trachea on opening the thorax was measured in 17 human lungs and varied from 2.8 to 6.2 cm. H₂O with a mean of 4.3 cm. H₂O (Table I). The time elapsing between death and necropsy had no effect on this measure of elastic recoil. It was also found that, if a small pressure change such as occurs in ordinary breathing during life was repeated, the volume change with each successive breath became constant (Fig. 6). This predictable behaviour is further described in the next paper.

DISCUSSION

At least three factors are responsible for the failure of previous workers to obtain consistent results in studying the elastic properties of dead lungs. The first is failure to appreciate that considerable deflation of the lungs occurs in the first few hours after death. The second factor is failure to recognize that the lungs take a considerable time to reach a state of equilibrium following a change in pressure, and the third and perhaps most important factor is the difference between the behaviour

of the lungs when suspended in the plethysmograph and within the thorax. In the plethysmograph deflation may be associated with collapse of certain alveoli, which are then held by surface tension so that considerable force is needed to open them. In these circumstances the distensibility of the lungs depends largely on the proportion of alveoli which are patent, and this can neither be predicted nor measured. The elastic recoil of the lungs will also be profoundly affected by this factor, for if collapsed alveoli are opened up on expansion the lungs will not recoil to their previous volume when the force is removed. It is clear that in the conditions of these experiments, the behaviour of the lungs depends on the previous history of inflation or deflation.

The only circumstance in which a predictable response can be obtained is when exactly the same force is applied to the lungs on repeated occasions in a manner comparable to that which occurs in life. Under these circumstances a state of equilibrium is established in which the degree of inflation becomes constant. The significance of this constant response in analysing the elastic properties of the lungs after death will be discussed in a later communication.

SUMMARY

The properties of normal human and animal lungs removed up to 72 hours after death have been investigated on inflation with negative pressure in a plethysmograph. From measurements of lung volume after death it is clear that a considerable volume of air is expelled from the lungs mainly in the first two hours after death.

The distensibility of the lungs after death varies in a complex and unpredictable manner, depending on the number of alveoli open at any given time. The response of the lungs to a change in pressure is visco-elastic, several minutes being required on occasions for equilibrium to be reached. A predictable response to inflation is only observed when small pressure is applied to the lungs on repeated occasions. In these circumstances the volume of air entering the lungs with each inflation becomes constant.

The inability of previous workers to obtain consistent results in experiments with dead lungs is probably due to their failure to recognize these changes in lung volume occurring after death, the visco-elastic nature of the response of the lungs and the possibility of infinite variation in the number of alveoli open after different degrees of inflation.

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