THE MECHANICAL PROPERTIES OF THE LUNGS IN PATIENTS WITH PERIODIC BREATHING

BY

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Cheyne (1818) first described periodic breathing in "a case of apoplexy in which the fleshy part of the heart was converted into fat". In describing the pattern of breathing he wrote: "his breathing was irregular; it would entirely cease for a quarter of a minute, then it would become perceptible, though very low, then by degrees it became heaving and quick, and then it would gradually cease again. This revolution in the state of his breathing occupied about a minute, during which there were about thirty acts of respiration." A similar case was described 36 years later by Stokes (1854) and since then the condition of Cheyne-Stokes breathing has been the subject of numerous papers. Although there has been a tendency to give the name of Cheyne-Stokes breathing to all forms of periodic breathing, it will be seen from the original description that it is more accurate to reserve the term for cases in which there is a definite period of apnoea and the length of the cycle is of the order of one minute.

As a result of the work of Pryor (1951), Gilmore and Kopelman (1954) and Guyton *et al.* (1955), it has been shown that changes in the activity of the respiratory centre and prolongation of the circulation time are important factors in the production of Cheyne-Stokes breathing. Periodic breathing occurs in patients with cerebral or cardiovascular disease and it has been suggested in the past that it is possible to distinguish a central and a peripheral type of periodic breathing. It is generally recognized that in patients with cerebral lesions, the cycle is shorter and apnoea is more likely to occur. Christie and Hayward (1943) suggested that the type of periodic breathing found in patients with left ventricular failure was due to waxing and waning pulmonary congestion. They measured the intra-pleural pressure and tidal volume in anaesthetized cats and rabbits and found that although the intra-pleural pressure swing remained constant the tidal volume underwent cyclic changes. Their findings could only be interpreted as indicating cyclical changes in the mechanical properties of the lung.

This paper reports the results of study of the mechanical properties of the lungs in patients with periodic breathing in which an attempt has been made to assess the part played by waxing and waning pulmonary congestion in the production of periodic breathing in man.

METHODS

The mechanical properties of the lungs were measured from simultaneous records of intra-oesophageal pressure and air flow. The patients, who were all sufficiently ill to make it difficult to obtain their co-operation, were studied sitting or semi-recumbent in bed. Intra-oesophageal pressure was measured either with a water filled tube (Dornhorst and Leathart, 1952) or a long air filled balloon (Mead *et al.*, 1955) connected to an electromanometer. The tidal volume was measured by several different methods, none of which was entirely satisfactory. In most of the patients a spirometer or pneumotachograph was used and in the others a combined thoracic and abdominal pneumograph (Dornhorst and Leathart, 1952) gave a record that could be calibrated in terms of volume. The principal difficulty in the use of a spirometer or pneumotachograph is that the

disturbance produced by breathing through a mouthpiece is often sufficient to abolish periodic breathing. The use of a pneumograph avoids this difficulty but is less accurate and difficult to calibrate.

The compliance of the lungs and the inspiratory resistance were measured, whenever possible, during the cycles of periodic breathing. In half the cases, however, the breathing became regular when the patient breathed through a mouthpiece. In these patients a record of intra-oesophageal pressure alone was obtained during periodic breathing and the mechanical properties of the lungs measured from the tracing taken when the breathing was regular. The length of the cycle of periodic breathing, the length of the period of apnoea, if present, and the respiratory rate were measured in each case. In one patient the oxygen saturation was measured with an ear oximeter.

RESULTS

The results in ten patients with periodic breathing are shown in Table I. All were men and all had evidence of cardiovascular disease. Four had classical Cheyne-Stokes breathing with a period

TABLE I
RESULTS IN TEN PATIENTS WITH PERIODIC BREATHING

Name	Age	Length of complete cycle in seconds	Number of breaths per cycle	Length of apnoea in seconds	Maximum respiratory rate during cycle/min.	Minimum respiratory rate during cycle/min.	Lung compliance l./cm.H ₂ O	Inspiratory resistance cm.H ₂ O/l./sec.	Simultaneous tracings	Diagnosis and remarks
A.L.	49	48	13	10	20	—	0.13	2.3	—	Coronary thrombosis Heart failure
W.S.	58	40	21	—	33	28	0.13	1.9	+	Cerebral hæmorrhage Coronary thrombosis Dissecting aneurysm (P.M.)
A.S.	58	58	22	—	29	19	0.048	9.6	—	Coronary thrombosis
		58	14	20	28	—	0.034	11.6	+	Heart failure
G.C.	59	30	7	11	23	—	0.22	2.5	—	Uræmia
C.A.	62	16	15	—	60	50	0.040	6.0	—	Chronic bronchitis Emphysema, hypertension Heart failure
		17	12	3	60	—				
M.J.	66	60	24	—	26	20	0.126	2.6	+	Coronary thrombosis
		56	14	22	26	—	0.084	2.8	—	Heart failure
C.B.	68	60	17	—	18	16	0.14	3.9	—	Malignant hypertension Cerebral thrombosis Heart failure (P.M.)
F.S.	69	40	26	—	30	24	0.053	7.0	+	28.10.54. Hypertension
		38	22	—	30	23	0.049	8.5	+	26.5.55. Heart failure
H.B.	74	65	22	—	28	18	0.061	6.3	+	Coronary thrombosis Heart failure (P.M.)
C.G.	74	60	26	—	28	21	0.108	4.8	—	Coronary thrombosis Heart failure

of apnoea lasting 10–22 seconds and a cycle length of 30–58 seconds. Five had periodic breathing with a cycle length of 38–65 seconds but no period of apnoea, while the other patient had a shorter cycle of 16–17 seconds with an apnoeic period of 3 seconds on occasions. Two of the patients with classical Cheyne-Stokes breathing did not always have apnoea, in some cycles there was no apnoea while in others apnoea lasted up to 22 seconds.

It will be seen from Table I that the mechanical properties of the lungs varied considerably from patient to patient, being within normal limits in some and showing a marked reduction in compliance and an increase in respiratory resistance in others. A simultaneous tracing of intra-oesophageal pressure and tidal volume in patient F.S. is shown in Fig. 1. It will be seen that the pressure and volume tracings wax and wane together indicating a periodic variation in the force applied to the lungs, presumably due to periodic changes in the activity of the respiratory centre. This periodic fluctuation in the pressure record was seen in every patient studied and moreover there was no

FIG. 1.—Tracing of intra-oesophageal pressure and tidal volume, showing periodic breathing in a patient with left ventricular failure.

consistent variation in the compliance and respiratory resistance measured from breath to breath throughout the cycle in the five patients in whom simultaneous tracings were obtained during periodic breathing.

A tracing of intra-oesophageal pressure in M.J. is shown in Fig. 2. In this patient there was a period of apnoea in some cycles but not in others and it will be seen that the respiratory rate and the pattern of the pressure tracing were essentially the same whether there was a period of apnoea or not. In the patient (C.A.) in whom oxygen saturation was measured, it was found to reach a maximum of 98 per cent during the apnoeic period and a minimum of 91 per cent at the time of maximum hyperpnoea. This finding is similar to that previously reported by Klein (1930).

DISCUSSION

If there is a form of periodic breathing due to waxing and waning pulmonary congestion, it should differ from that seen in patients with normal lungs and should not be associated with a period of apnoea, for apnoea must indicate a temporary cessation of activity of the respiratory centre.

We have found no evidence of waxing and waning pulmonary congestion in ten patients with heart disease and periodic breathing, in several of whom there was significant pulmonary congestion and no period of apnoea. In every patient the intra-oesophageal pressure tracings were compatible with cyclical changes in the activity of the respiratory centre and in no case was there evidence that the mechanical properties of the lungs changed from breath to breath throughout the cycle. We have not observed anything resembling the pattern reported by Christie and Hayward (1943) in animals in which the intra-pleural pressure fluctuations remained of constant amplitude while the tidal volume showed periodic breathing. It is, however, impossible to exclude the existence of such a form of periodic breathing.

FIG. 2.—Tracings of intra-oesophageal pressure in a patient with periodic breathing. The upper tracing shows no apnoeic period, while the lower tracing obtained several minutes later shows apnoea lasting 22 seconds.

SUMMARY

The mechanical properties of the lungs have been measured in ten patients with periodic breathing. The compliance of the lungs and the respiratory resistance varied, being within normal limits in some and grossly abnormal in others.

The intra-oesophageal pressure record in every case showed variations in the force applied to the lungs similar to those in tidal volume and there was no evidence that the mechanical properties of the lungs varied at different phases of the cycle of periodic breathing.

These findings support the conclusion that periodic fluctuations in the activity of the respiratory centre play an important part in the production of periodic breathing and indicate that waxing and waning pulmonary congestion was not a factor in the patients studied.

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REFERENCES

- Cheyne, J. (1818). *Dublin Hosp. Reports*, 2, 216.
 Christie, R. V., and Hayward, G. W. (1943). *J. Physiol.*, 102, 88.
 Dornhorst, A. C., and Leathart, G. L. (1952). *Lancet*, 2, 109.
 Gilmore, H. R., and Kopelman, H. (1954). *Brit. med. J.*, 2., 1439.
 Guyton, A. C., Crowell, J. W., and Moore, J. (1955). *Circulation*, 12, 717.
 Klein, O. (1930). *Gesellsch. inn. Med.*, 42, 217.
 Mead, J., McIlroy, M. B., Selverstone, N. J., and Kriete, B. C. (1955). *J. appl. Phys.*, 7, 491.
 Pryor, W. W. (1951). *Circulation*, 4, 233.
 Stokes, W. (1854). *Diseases of Heart and Aorta*. Hodges and Smith, Dublin.