Pulmonary Compliance in Patients with Periodic Breathing

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A study of pulmonary compliance in patients with periodic breathing was made. The elastic properties of the lung were found to be normal in some patients and grossly decreased in others. Two types of periodic breathing mechanisms were found: in one there are similar variations of tidal volumes and unchanged pulmonary compliance; in the other a constant fluctuation of the intraesophageal pressure associated with the variations in tidal volume.

Cheyne, a professor in Dublin, reported a description of the breathing of one of his patients. "For several days 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 a minute during which there were thirty acts of respiration." This was the first clinical description of periodic breathing. Thirty-six years later Stokes described similar breathing in a patient of his own. "For more than two months before his death, this singular character of respiration was always present, and so long would the periods of suspension be, that his attendants were frequently in doubt whether he was not actually dead. Then a very feeble, indeed barely perceptible inspiration would take place, followed by another somewhat stronger, until at length high heaving, and even violent breathing was established, which would then subside till the next suspension. This was frequently a quarter of a minute in duration..." Twenty-four years later Traube advanced the first noteworthy theory of the mechanism of Cheyne-Stokes breathing. He described 2 types of Cheyne-Stokes breathing, namely that with an intracranial lesion in the presence of an intact heart, and that with cardiac disease but no change in the brain. He believed that a diminished arterial blood supply to the brain was the pathogenic factor common to both types, this decreased cerebral blood supply causing a diminished respiratory center activity.

Since then, the condition of periodic breathing has been the subject of numerous reports. However, in the investigation of the abnormal state, attention must be given to the original description of a definite period of apnea in which the length of the cycle must be 1 minute. There has been a tendency to call all forms of periodic breathing, Cheyne-Stokes, without regard to the description found in the first reports. Christie and Hayward suggested that the type of periodic breathing found in patients with left ventricular failure was due to a waxing and waning pulmonary congestion. They based this opinion on observations of measurements of intrapleural pressures and tidal volumes made on anesthetized cats and rabbits. They found that although the tidal volume underwent cyclic changes, the intrapleural pressure remained constant. These findings could only be interpreted as being due to cyclic changes in the mechanical properties of the lungs. If the origin was of involvement of the respiratory center, then the intrapleural swings should also show similar cyclic changes.

Eyster has pointed to the differences in the two forms of periodic breathing. He observed when depression of the respiratory center occurs, the blood pressure rises during the apneic phase and falls during the hyperpnea, while the reverse is found in patients with periodic breathing associated with cardiovascular disease. It has been noted clinically that the cycle is longer in

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periodic breathing associated with cardiovascular disease, and that the fluctuations in resting end-expiratory level do not occur in the central type. The response to oxygen inhalation is also of interest for the periodic breathing of cerebral origin will be abolished, whereas that due to cardiovascular disease usually will not be affected, or it may, in fact, be exaggerated. Carbon dioxide inhalations will usually abolish the periodic breathing associated with cardiac disease, but will not affect that associated with a cerebral disturbance.

That changes in the activity of the respiratory center and prolongation of the circulation time are important factors in the production of Cheyne-Stokes breathing has been shown by recent studies.9–8

This report stems from a study of the mechanical properties of the lungs in patients with periodic breathing in whom an attempt was made to distinguish 2 mechanisms, a peripheral and a central type for periodic breathing. An attempt was also made to assess the role of the waxing and waning of pulmonary congestion as suggested by Christie and Hayward in their study.

METHODS

The patients were all ill enough to make their cooperation difficult. All patients had to be studied either in the sitting position or semi-recumbent in bed. Those patients in whom satisfactory records for tidal volume were not obtainable were rejected from consideration in this study. Intra-esophageal pressures were obtained with an air-filled balloon connected with a Statham gage similar to the previously described method.9 Tidal volume was obtained by electronic integration of the air flow obtained through a pneumotachograph. All records were made simultaneously and recorded by photography of an oscillographic trace.9 Breath-by-breath records of tidal volume and intra-esophageal pressures were obtained throughout several cycles of periodic breathing in each patient.

The elastic behavior of the lungs may be stated in terms of compliance, an expression that relates changes in volume to changes in transpulmonary pressures when there is no flow of air:

\[
\text{Pulmonary compliance} = \frac{\Delta \text{ volume, L.}}{\Delta \text{ pressure, cm. H}_2\text{O}}
\]

*Electronics for Medicine Recorder.

By convention, in human subjects the ratio is determined from the end-expiratory relaxation volume (functional residual capacity).

Five patients with congestive heart failure who had Cheyne-Stokes respiration and whose studies were satisfactory were used in this study.

CLINICAL SUMMARIES

S. O., a 55-year-old white man, was admitted to the hospital complaining of exertional dyspnea, paroxysmal nocturnal dyspnea, orthopnea, weakness, and fatigue. He had had a posterior myocardial infarction 6 months prior to admission, shortly after which the above symptoms began and progressed. His blood pressure was 94/80 mm. Hg, the pulse was 112 per minute and regular, the respirations were 40 per minute and of the Cheyne-Stokes type. The cervical veins were distended and bilateral basilar pulmonary rales were heard. The heart was enlarged to percussion and a gallop rhythm was present. The second pulmonic sound was louder than the second sound heard at the aortic area. The liver was enlarged and tender. The venous pressure was equivalent to 120 mm. of saline and the circulation (Decholin) was 36 seconds. Little variance in compliance was present throughout the cycle of his periodic breathing.

W. G. was a 27-year-old obese Negro man who gave a long-standing history of hypertensive cardiovascular disease. He was admitted in congestive heart failure. His weight was over 300 pounds. The blood pressure was 240/170, the pulse was 108 per minute and regular. The respirations were 40 per minute and of the Cheyne-Stokes type. Pulmonary rales, an enlarged heart with gallop rhythm and an enlarged tender liver with dependent edema were present. The pulmonary compliance was 0.05 L. per cm. of water throughout the entire Cheyne-Stokes cycle.

A. K. was a 45-year-old white woman with a history of rheumatic heart disease from an early age. Six months before admission there were several embolic episodes. Congestive heart failure for many years had been controlled by digitalis and salt restriction. At the time this patient was studied recent recurrences of cerebral embolization were present. There was aphasia and a left-sided hemiplegia. Classical signs of congestive heart failure were present. There was marked distention, pulmonary rales, and an enlarged liver with peripheral edema. Atrial fibrillation was present. The apical rate was 140 per minute. Pulmonary compliance was 0.10 L. per cm. of water. There was an absence of variation and pressure fluctuations following tidal volume changes.

E. D. was a 75-year-old white woman with a history of hypertension for many years. Nine months before admission a cerebral vascular accident had occurred. A left-sided hemiparesis re-
Progressive dyspnea, orthopnea, and left chest pain required her admission to the hospital. The blood pressure was 180/120; the pulse was 120 per minute and was regular. The thyroid gland was palpable. There were signs of severe congestive heart failure with a gallop rhythm being present. The pulmonary compliance was decreased ranging from 0.07 L per cm to 0.06 L per cm. of water. This record showed a cyclic fluctuation during the phases of periodic breathing, becoming more decreased during apnea. The intraesophageal pressure fluctuations remained constant during the phase (figs. 1 and 2).

I. D. was a 55-year-old white woman who had hypertension and congestive heart failure for 2 years before admission. Digitalis and diuretics...
were needed for left-sided failure. Her symptoms had progressively become worse for the past 6 months. On physical examination the blood pressure was 150/105, the pulse 100 per minute and regular. Distended cervical veins and bilateral rales at the bases of the lungs were present. The heart was found enlarged on percussion; the liver was palpable 6 cm. below the costal margin. Pretibial edema was found. Periodic breathing was present. The venous pressure was equivalent to 130 mm. of saline and the circulation time was 35 seconds. The pulmonary compliance was the lowest found among these patients, being 0.025 to 0.03 L per cm. of water. In the records the fluctuation of the esophageal pressure remained constant throughout the cycle of periodic breathing, even during the phase of apnea. This was evidence of the force applied being ineffective to produce a change in lung volume in this phase of the cycle.

RESULTS

Five patients of those studied had tracings satisfactory and complete enough to be analyzed. Two men and 3 women comprised the group. All had classical Cheyne-Stokes breathing. The period of apnea lasted 15 to 28 seconds and the cycle length was 40 to 69 seconds. In 3 patients, who were the only ones to whom aminophylline was administered after completion of the study, the periodic breathing was abolished.

The data obtained from the 5 patients are presented in figure 1. This is a graph of the average pulmonary compliance during a cycle of periodic breathing for each patient. Each curve is the mean curve of 2 to 3 measurements of separate cycles of periodic breathing for each patient. It will be seen that in 3 patients (S.O., W.G., and A.K.) no changes in the mechanical properties of the lungs during periodic breathing occurred even though 2 of them demonstrated an increased rigidity of the lungs. A.K. had a normal value for pulmonary compliance. In all these patients the pressure and volume tracings waxed and waned together. This type of trace indicates a periodic variation in the forces applied to the lungs, presumably central in origin due to cyclic variations in the respiratory center. The remaining 2 patients, however, as is seen in figure 1, did not show the periodic fluctuation in the pressure record and there was considerable variation in compliance. In patient A.D. there was a continuing pressure fluctuation during apnea, and the lung compliance became progressively less compliant for this period and became more compliant during hyperpnea. The simultaneous record of volume and pressure is shown in figure 2. This finding was identical to that found by Christie and Hayward in laboratory animals.4 Patient E.D. also showed a periodic fluctuation in pulmonary compliance of the same type. In all these patients except A.K. there was increased lung rigidity. Three other patients with periodic breathing also had normal pulmonary compliance values. However, because of incomplete studies they were excluded from the final results. The record of S.O. is seen in figure 3.

Ear oximeter studies were made in 3 patients but are not pertinent to this discussion; they are mentioned only to state that the findings were similar to those of Gilmore and Kopelman, where the highest oxygen saturations are recorded during apnea and the lowest at hyperpnea.7

DISCUSSION

Although 3 of these 5 patients gave no evidence of the waxing and waning pulmonary congestion suggested by the studies of Christie and Hayward,4 2 patients definitely did. The 3 patients all were severely ill and had marked evidence of pulmonary congestion and heart failure. In these patients the intrasophageal pressure records were compatible with extrapulmonary cyclic changes, namely from the respiratory center, and thus are of the central type of Cheyne-Stokes breathing.9 The 2 patients I.D. and E.D. did, however, show the pattern reported by Christie and Hayward.4 This finding is the peripheral type of Cheyne-Stokes breathing referred to by Traube,3 where the intrasophageal pressure fluctuations remain constant in the presence of a tidal volume exhibiting periodic breathing. The suggestion of Christie and Hayward was that this was due to pulmonary
congestion waxing and waning. Unfortunately no hemodynamic traces were recorded by us. In these patients the nonelastic resistance was elevated fourfold in one and threefold in the other. It is possible that other mechanisms may contribute to this second type of periodic breathing than that suggested by Christie and Hayward. It is known that with pulmonary congestion surface tension plays a significant role in decreasing pulmonary compliance. A liquid-air interface in the alveolar units produces a lag of the change in volume to the applied transpulmonary pressure. This effect has been found to be most marked in pulmonary edema. The intrapleural pressure may, because of this effect, not be great enough to effect inflation. But this factor alone cannot explain the reinflation that follows the apneic phase. Greene observed 2 types of Cheyne-Stokes respiration: in one there was an unchanging end expiratory level and in the other there was a fluctuating level of the resting respiratory level. This observation was thought to distinguish the peripheral from the central type of periodic breathing. But progressive increase in airflow resistance was not evident from our study, and this abnormality, by itself, is insufficient to explain the periodic breathing with continuing intrapleural fluctuations. Inertness of the lung, if profound, might similarly become a factor, but the situation as found in periodic breathing does allow this to become a helpful explanation. In experiments on normal subjects simulating periodic breathing not a single instance was found of continuing intraesophageal pressure fluctuations during voluntary apnea.

Wilson in experiments with newborn rabbits, in spite of vigorous respiratory efforts and great diaphragmatic contractions, detected only negligible tidal flow. In dogs, while investigating the intrapleural pressures at different points in the chest, Fahri noted no change in tidal volume at the time changes in pleural pressure occurred. Furthermore, these changes in pressure occurred even after the dogs had been killed, in some instances as long as 30 minutes after death. Neither of these separate observations in newborn rabbits and in dogs was explained. We also have no sound explanation to offer for the phenomenon. The possibility of soft tissue obstruction arises as an explanation for the peripheral type. But attention to the obstruction of the pharynx and laryngeal areas did not seem a factor in preventing the occurrence of tidal volume.

The effect of aminophylline, by its bronchodilatory action and by affecting the geometric arrangement of the bronchi, may actually reduce intrapulmonary factors as surface tension and air-flow resistance. Its other pharmacologic effects also must operate in abolishing periodic breathing.

**Summary**

The elastic properties of the lung have been measured in 5 patients with periodic breathing. The pulmonary compliance was found to be normal in some patients with periodic breathing and grossly decreased in others.

Two types of periodic breathing were found: first, in 3 patients the intraesophageal pressure record showed variation similar to the tidal volume, without change in compliance during the different phases of the cycle of periodic breathing; second, in 2 patients there was a constant fluctuation of the intraesophageal pressure record and of the force applied, which was accompanied by variations in the tidal volume record during the different phases of the cycle (from apnea to hyperpnea).

These findings support the conclusion that there is a periodic breathing due to periodic fluctuations in the activity of the respiratory center and that there is another type that is due to peripheral factors. The peripheral factors are not well defined.

**Summario in Interlingua**

Le qualitates elastic del pulmon esseva mesurate in 5 patientes con respiration periodic. Illos se monstrava normal in certe patientes con respiration periodic e grandemente diminuite in alteres.

Esseva trovate 2 typos de respiration peri-
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odie. (1) In 3 patientes le registration del pression intraesophaghe exhibiva variationes simile al variationes del volumine de aere currente, sin alteratione de elasticitate durante le diverse phases del cyclo de respiration periodic. (2) In 2 patientes il habeva un fluctuation constante del pression intraesophaghe e del fortia applicate, e isto esseva acompaniate de variationes in le volumine del aere currente in le curso del diverse phases del cyclo (ab apnea a hyperpnea).

Iste constatationes supporta le conclusion que il existe (1) un typo de respiration periodic causate per fluctuationes in le activitate del centro respiratori e (2) un altere type que es causate per factores peripheric. Iste factores peripheric non es ben definite.

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