IRREGULARITY OF LUNG VENTILATION STUDIED BY A SINGLE BREATH METHOD

T. S. Tsuzmer and R. S. Vinitskaya

UDC 612.21-087.7

The N_2 concentration curve during a single quiet expiration after inhalation of pure O_2 is a suitable criterion of the regularity of lung ventilation. Evidence of irregularity is given by a decrease in the N_2 concentration at the beginning of the alveolar phase, an increase in the rise in N_2 concentration from the beginning to the end of the alveolar phase, and also an increase in the ratio between the rise in concentration and the duration of the alveolar phase.

The distribution of the inspired gas between the large number of alveoli in the lungs and its mixing with the alveolar gas cannot be absolutely regular even in the healthy organism because of differences in the anatomical and physiological conditions of ventilation in different segments of the lungs. Under pathological conditions this irregularity is increased. The rate of change in composition of the inspired air can be used as a measure of the uniformity of ventilation [4-6, 9].

The method of determining the regularity of ventilation by analysis of the N_2 concentration during a single expiration after inhalation of O_2 [7, 8] is based on simultaneous recording of the N_2 concentration and the expired volume during deep expiration (not less than 1250 ml). At the same time, it has been shown that the N_2 concentration, measured at the end of normal expiration after inhalation of O_2 can be equated with the mean alveolar N_2 concentration. Accordingly, to detect irregularity of ventilation during quiet breathing, it was decided to analyze not a forced, but an ordinary single expiration.

EXPERIMENTAL METHOD

The model A-1 SKTB nitrometer (Medfizpribor, Kazan', USSR) [10] was used for the analysis. Single breaths recorded during determination of the functional reserve capacity [3] were analyzed. In all cases the second expiration from the beginning of O_2 inhalation was used because it is more stable than the first.

After inhalation of O_2, it is mixed with the N_2 present in the air filling the lungs. During expiration, the dead space is first emptied, and mixed gas from the alveoli then begins to be expelled (the alveolar phase).

The beginning of the alveolar phase was conventionally defined as the point of inflection of the curve (the point a), i.e., as the moment of a sharp decrease in the rate of rise of the N_2 concentration (Fig. 1). As experiments with graphic differentiation of the initial part of the curves for a single expiration at 0.2-sec intervals [1] showed, the point of inflection in healthy and sick subjects lies in the first 0.4 sec after the beginning of elevation of the curve (this time includes the inertia time of the instrument).

The end of the alveolar phase and beginning of inspiration are denoted by the point b. The increase in N_2 concentration in the alveolar phase is defined as ΔFN_2 and is measured on the curve by means of a stencil. By multiplying ΔFN_2 by the atmospheric pressure on the day of investigation (subtracting the water vapor pressure, 47 mm Hg) the increase in partial pressure of N_2 in the alveolar phase is obtained in millimeters of mercury (ΔFN_2). This index, together with its ratio with the duration of the alveolar

Laboratory of Physiology, A. V. Vishnevskii Institute of Surgery, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR A. A. Vishnevskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 72, No. 9, pp. 23-26, September, 1971. Original article submitted February 5, 1971.

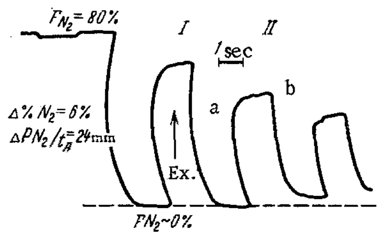

Fig. 1

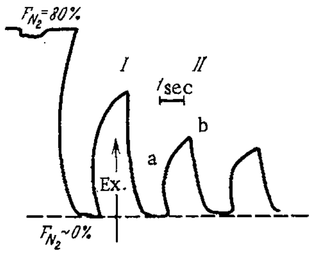

Fig. 2

Fig. 1. Changes in nitrogen concentration in a healthy subject: a = 49% N_2; b = 55% N_2; ΔPN_2 = 42.8 mm Hg; t = 1.8 sec; $\Delta PN_2/t$ = 23.8 mm Hg/sec; Ex. = expiration.

Fig. 2. Changes in nitrogen concentration in patient with bronchiectasis: a = 23% N_2; b = 42% N_2; ΔPN_2 = 71 mm Hg; t = 1 sec; $\Delta PN_2/t$ = 71 mm Hg/sec; Ex. = expiration.

TABLE 1. Characteristics of Changes in Nitrogen Concentration during Single Quiet Expiration in Healthy Subjects and Patients (mean values and limits of variation)

Subjects	Concentration at point a (in %)	Concentration at point b (in %)	Increase in alveolar phase (in mm Hg), ΔPN_2	Duration of alveolar phase (in sec), t	$\Delta PN_2/t$ (in mm Hg/sec)	RV/FRC (in %)
Healthy (n = 18)	51.9 (40–60)	57.3 (43–69)	36.6 (21–71)	1.86 (1.0–3.3)	20.6 (8.9–33.6)	24.8 (16.3–41.6)
Patients with bronchiectasis and lung abscesses (N = 25)	47.4 (30–63)	54.6 (38–66)	51.8 (21–107)	1.49 (0.9–3.0)	38.7 (12–71)	19.6 (11–41)
Patients with tumors of the lungs (N = 45)	45.8 (22–68)	54.3 (35–69)	60.6 (21–140)	1.17 (0.6–2.6)	54.9 (12–140)	18.9 (8–49)
Patients after lobectomy (N = 2)	— (31–54)	— (46–62)	— (53–106)	— (1.6–1.7)	— (31–60)	— (31–40)

phase (t) between the points a and b ($\Delta FN_2/t$) was used to express the irregularity of ventilation qualitatively. By this method the increase in N_2 in the alveolar gas was correlated, not with the volume, but with the time parameters of expiration. This is convenient when the apparatus used does not allow the concentration of the gas and the respiratory volume to be recorded concurrently.

RESULTS AND DISCUSSION

The hypophesis that changes in the N_2 concentration during a single quiet expiration reflect the irregularity of ventilation was tested in 18 healthy persons, in whom the irregularity of gas mixing was slight, and in 72 patients with diseases of the lungs.

Typical curves for a single exhalation of N_2 are shown in Figs. 1 and 2. In the healthy subject the horizontal plateau of the alveolar phase is clearly defined, but in the patients with bronchiectasis the increase in N_2 concentration continued until the end of expiration.

In patients with bronchiectasis and lung abscesses, the value of ΔPN_2 was 15.2 mm Hg higher than in the healthy subjects, in patients with tumors of the lung it was 24 mm Hg higher, and in patients after lobectomy it was more than twice as high (Table 1). In addition, in the patients there was a well-marked decrease in duration of the alveolar phase associated with an increased rate of respiration. In the patients of these same groups the alveolar phase was shorter than in the healthy subjects by 0.37, 0.69, and 0.26-0.16 sec, respectively.

The increase in ΔNP_2 evidently arises from several causes.

1. The N_2 concentration at the beginning of the alveolar phase (point a) in the patients was on the average much lower than in the healthy subjects; when the N_2 was flushed from the lungs at the beginning of the second expiration in those segments of the lungs which were emptied first (the anatomical dead space and the well-ventilated segments), the value of N_2 was lower in these patients than in the healthy subjects.

2. The decrease in N_2 concentration at point b was not always proportional to the decrease at the point a, and in most cases was below it. This was responsible for the high value of ΔPN_2 in the alveolar phase.

In each individual case no direct connection could be found between the degree of decrease of the point a and ΔPN_2. Lowering the point a did not always lead to an increase in ΔPN_2, and a high value of ΔPN_2 could be observed even without lowering of the point a.

One possible cause of the decrease in the N_2 concentration in the lungs at the beginning of the second expiration during inhalation of O_2 by the patients was an increase in the depth of respiration. To investigate the importance of this factor, the N_2 concentration at point a was compared with the ratio between the respiratory volume (RV) and the functional reserve capacity of the lungs (FRC). The degree of lowering of the point a was not directly dependent on the ratio RV/FRC. Furthermore, with equal values of RV/FRC, the point a was at a lower level in the patients than in the healthy subjects. This means that under conditions of equal ventilation, toward the beginning of the second expiration the N_2 concentration in the dead space and the well-ventilated segments of the lungs was lower in the patients than in the healthy subjects. Since the volume of the anatomical dead space did not differ significantly in the patients and the healthy subjects, only one conclusion was possible: the volume of the well-ventilated spaces of the lungs was smaller in the patients than in the healthy subjects, and accordingly, during inhalation of O_2 this volume was ventilated more rapidly in the patients than in the healthy subjects. The volume of the poorly ventilated spaces was relatively larger in the patients and it was ventilated more slowly, with a consequent increase in the value of ΔPN_2.

Proof that ΔPN_2 in fact reflects the irregularity of ventilation was also given by the following observation: in a healthy subject a voluntary increase in the depth of respiration causes no change in ΔPN_2. For instance, in a healthy person tested during quiet breathing, with RV = 650 ml and a respiration rate of 14/min, the value of RV/FRC was 20%, the N_2 concentration at point a was 49% and at point b 55%, and $\Delta PN_2 = 42.8$ mm Hg. A voluntary increase in RV to 1500 ml (with a rate of 9/min) led to an increase in RV/FRC to 53.5%. The concentration fell equally at points a and b, and there was no change in ΔPN_2.

The index $\Delta PN_2/t$ (the mean rate of rise in concentration in the alveolar phase) was increased in all the patients (Table 1). This is evidence of the informativeness of this index, which the writers previously suggested for use in the analysis of curves of CO_2 concentration during expiration and also for the investigation of a single exhalation of N_2.

However, it should be borne in mind that with an increase in the respiration rate and corresponding decrease in the duration of the alveolar phase (t) the ratio $\Delta PN_2/t$ may be normal. Consequently, the difference detected by analysis of the position of the point a and the value ΔPN_2 can disappear if the ratio $\Delta PN_2/t$ is used.

It can be concluded from the results described that changes in the N_2 concentration during a single quiet expiration after inhalation of O_2 can be used as a criterion of the presence of irregularity of lung ventilation. The increase in N_2 in the alveolar phase (ΔPN_2) and the partial N_2 pressure near the beginning of the alveolar phase (the position of point a) give some idea of the presence of spaces with different levels of ventilation and of their approximate relative volume.

LITERATURE CITED

1. R. S. Vinitskaya, T. S. Tsuzmer, and N. A. Koganova, in: New Instruments for Gas Analysis in Modern Medicine and Physiology [in Russian], Kazan' (1967), p. 158.

2. T. S. Tsuzmer, R. S. Vinitskaya, and N. A. Koganova, Éksper. Khir., No. 3, 22 (1967).
3. T. S. Tsuzmer and R. S. Vinitskaya, Grudnaya Khir., No. 4, 68 (1969).
4. M. S. Shneider, Ter. Arkh., No. 7, 3 (1964).
5. M. S. Shneider, Klin. Med., No. 5, 99 (1966).
6. A. Bouhuys and G. Lundin, Physiol. Rev., 39, 731 (1959).
7. J. H. Comroe and W. S. Fowler, Ann. Med., 10, 408 (1951).
8. W. S. Fowler, J. Appl. Physiol., 2, 283 (1949).
9. W. S. Fowler, Physiol. Rev., 32, 1 (1952).
10. J. C. Lilly, Am. J. Physiol., 161, 342 (1950).
11. T. Lundman, E. Orinius, and J. Stähle, Scand. J. Clin. Lab. Invest., 16, 332 (1964).