Differentiation and Evaluation of Dyspnea

Hideo Ueda and Shigeo Koike

The Second Department of Internal Medicine,
University of Tokyo, Tokyo

1. Definition and Mechanism of Dyspnea

Clinically, dyspnea is defined as difficult and labored breathing. Differentiation and evaluation of dyspnea are practically difficult, because, like pain, dyspnea is precisely a complex of subjective symptoms.

The regulation of ventilation is so much complex as shown schematically in Fig. 1. When the balance between input and output information of the regulation system of ventilation in the central nervous system is inappropriate, dyspnea may occur. Therefore, multiple causes are involved in the occurrence of dyspnea.

2. Practical Methods of Differentiation and Evaluation of Dyspnea

Physician must differentiate the cause and evaluate the severity of dyspnea as quickly and precisely as possible, because frequently, urgent and adequate treatment for dyspnea is needed, though the mechanism of dyspnea is not fully understood. In this regard, the pneumotachography and arterial blood gas analysis are extremely useful, because both methods are accurate and can be easily applied even severely dyspnoeic patients.

In this study, volume of flow of air (V̇) and electronically integrated tidal volume (V̇E) were recorded by the pneumotachograph in dyspnoeic patients of various cases (Fig. 2). Ventilation was also measured with Wright respirometer. Arterial blood sample was taken anaerobically from the brachial or femoral artery and analyzed by IL-Meter (Type-113), which is capable to measure arterial oxygen tension (Pao₂), arterial carbon dioxide tension (Paco₂) and arterial pH (pHa).

In order to demonstrate the change in these values of the blood gas analysis, “Paco₂-pHa-Pao₂” nomogram was constructed as shown in Fig. 3. In relation to the change in Paco₂ and pHa, Paco₂-pHa nomogram is divided into 6 areas by the normal value of Paco₂ (40 mm.Hg) and pHa (7.4), and normal buffer line. Paco₂-Pao₂ nomogram is

Fig. 1. Scheme of respiratory control system.

Input information to regulation system of ventilation in central nervous system

Output information from regulation system of ventilation in central nervous system
divided by normal values of $\text{Paco}_2$, $\text{Pao}_2$ (85 mm.Hg) and by ideal blood gas line (with assumption of alveolar-arterial oxygen tension difference; 0, barometric pressure; 760 mm.Hg, $\text{RQ}=0.8$). In this nomogram, these values of arterial blood in individual can be shown by 2 points and a connecting bar parallel to the abscissa. By this nomogram alterations in alveolar ventilation, acid-base balance and hypoxemia can be easily analyzed and evaluated.

(1) Cardiac Dyspnea

Panel A in Fig. 2 shows $\dot{V}$ and $V$ curves of a patient with mitral stenosis, who was orthopnoeic. The $V$ curve appears normal except for increased respiratory frequency, in other words, there is no elongation of expiration. The $V$ curve reveals that the tidal volume 0.47 L., namely respiration is not shallow. The values of the arterial

![Diagram of volume of flow of air ($\dot{V}$) and tidal volume ($V$).](image)

Fig. 2. Curves of volume of flow of air ($\dot{V}$) and tidal volume ($V$).

- **A**: Mitral stenosis (in orthopnoea)
- **B**: Asthma bronchiale (in attack)
- **C**: Chronic pulmonary emphysema
- **D**: Chronic cor pulmonale
blood of this patient is represented by I (solid circle) in nomogram A of Fig. 3, which indicate respiratory alkalosis (alveolar hyperventilation) and slight hypoxemia. Here must be pointed out that the difference between measured $\text{Pao}_2$ in this case and that on ideal blood gas line which corresponds to measured $\text{Paco}_2$ is over 20 mm.Hg.

In the same nomogram, I, II (solid circle) represent the values in a patient with aortic insufficiency, who was in the attack of cardiac asthma. Respiratory alkalosis and markedly decreased $\text{Pao}_2$ are noted, but there is no elevation of $\text{Paco}_2$. This is

**Nomogram A**

![Nomogram A](image)

**Nomogram B**

![Nomogram B](image)

**Fig. 3.** "$\text{Paco}_2$-$\text{pHa}$-$\text{Pao}_2$" nomogram.

Nomogram A

- **Area 1:** Respiratory acidosis with metabolic compensation
- **Area 2:** Metabolic alkalosis with respiratory compensation
- **Area 3:** Respiratory and metabolic alkalosis
- **Area 4:** Respiratory alkalosis with respiratory compensation
- **Area 5:** Metabolic acidosis with respiratory compensation
- **Area 6:** Respiratory and metabolic acidosis
- ●: Cardiac disease (I & II)
- ○: Pulmonary disease (III, IV, V & VI)

Nomogram B

- ➔: Amyotrophic lateral sclerosis
- ●: Hepatic coma
- △: Uremia
characteristic to cardiac dyspnoea and one of the important differential points. The patient with chronic cor pulmonale or chronic obstructive lung disease, on the other hand, shows usually respiratory acidosis.

(2) Pulmonary Dyspnoea
Panel B in Fig. 2 shows the V and V curves of a patient with bronchial asthma (case B). The V curve reveals that tidal volume is by no means shallow. The important points in the V curve are the expiratory initial peaked flow and the elongation of expiration. The blood values of this patient are shown by III (open circle) in the nomogram A of Fig. 3. There are a slight increase in Paco₂ and a decrease in Pao₂.

In the same nomogram, IV represents the values of a patient with chronic pulmonary emphysema. V and VI represent the values of two patients with chronic cor pulmonale. It may be said that the more the increase in Paco₂ and the more greater the difference between measured Pao₂ and that on ideal blood gas line, the more severe the pulmonary disease is. The tracings C and D in Fig. 2 show the V and V curves of a patients with pulmonary emphysema and a patient with chronic cor pulmonale. There are pronounced elongation of inspiration, but expiratory initial peaked flow almost disappeared. Actual respiratory distress was more severe in the pulmonary emphysema than in the cor pulmonale, although the degree of hypercapnea and hypoxemia in the pulmonary emphysema are milder than in the cor pulmonale.

(3) Respiratory Muscle Atrophy
Open circle with arrow in nomogram B of the Fig. 3 represents the values of a patient with amyotrophic lateral sclerosis and the arrow represents the shift of values along the normal buffer line and ideal blood gas line during the course of illness.

(4) Metabolic diseases
Solid circle in the nomogram B of Fig. 3 is the values of a patient with hepatic coma. Measurement with a respirometer showed tidal volume was 0.8 L. and respiratory frequency was 30/min. Open triangles in the same nomogram represent the arterial values of a patient with uremia, who showed marked hyperventilation.

4. Summary
1. V curves are significantly different in cardiac patients and in patients with chronic obstructive lung diseases. But tidal volume and respiratory frequency are not so useful for differentiation of dyspnoea, but from the change of minute ventilation, the change of alveolar ventilation can be clinically evaluated.

2. "Paco₂-pHa-Pao₂" nomogram revealed its usefulness in differentiation and evaluation of dyspnea. Cardiac patients usually show respiratory alkalosis. On the contrary, the patients with chronic obstructive lung diseases show respiratory acidosis. On the evaluation of dyspnea, especially in lung diseases, the greater the difference between measured Pao₂ and ideal blood gas line, the more severe the disease is as shown by dotted area in Fig. 3. In the respiratory muscle paralysis, frequent check of the decrease of ventilation and increased Paco₂ are needed. In the metabolic diseases the change of pHa is important in differentiation and evaluation of diseases.

(Practical procedures of the evaluation on 11 patients with dyspnoea were demonstrated by the motion picture in this symposium.)