Airway Occlusion Pressure ($P0_{.1}$) — A Useful Predictor for the Weaning Outcome in Patients with Acute Respiratory Failure —

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Twenty-five patients who required mechanical ventilatory support (MVS) after major surgery or severe burns were studied to determine whether airway occlusion pressure ($P_{0.1}$) is a clinically useful indicator to predict the success or failure of the weaning trial. A total of 33 weaning trials were attempted on these patients. Of the 33 trials, 24 were followed by successful weaning and 9 by failure. Although the success group, when compared with the failure group, had a lower respiratory rate ($P < 0.001$), a lower minute ventilation ($P < 0.001$), a higher maximal voluntary ventilation to minute ventilation ratio ($P < 0.01$) and a higher forced vital capacity ($P < 0.05$), no threshold values separated the success from the failure group. The alveolar-arterial P_{O_2} gradient, with an $F_{I_{O_2}}$ of 1.0, in weaning success and failure showed no statistical difference. In contrast, all patients in the success group had a $P_{0.1}$ of less than 3.5 cmH_2O and those in the failure group had a $P_{0.1}$ of greater than 3.5 cmH_2O ($P < 0.001$). We conclude that $P_{0.1}$ is a clinically superior indicator for discontinuing MVS in patients with acute respiratory failure. (Key words: airway occlusion pressure, acute respiratory failure, mechanical ventilation, weaning)

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For successful weaning from mechanical ventilatory support (MVS), some indicators as to arterial oxygenation, alveolar ventilation, and the mechanical properties of the respiratory system and respiratory muscles are commonly used[1,2]. However, these are not always valid to predict a successful weaning outcome[2–4].

Airway occlusion pressure ($P_{0.1}$) is the pressure developed at the trachea during the first 0.1 seconds of inspiratory effort against an occluded airway[5]. $P_{0.1}$ has been found to reflect an increase or a decrease in respiratory neuromuscular activity[5,6] and has been suggested to be a useful predictor for successful ventilator weaning in patients with chronic obstructive lung disease (COPD)[7,8]. However, there are few observations in patients with acute respiratory failure (ARF)[9]. The purpose of this study was to examine whether $P_{0.1}$ was a clinically useful indicator to predict the success or failure of the weaning trial in patients with ARF.

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Methods

During about a 2-month period, a weaning trial was performed on 27 adult patients who required MVS after major surgery or severe burns. Of these patients, 25 consented to participate in the present study. They were conscious and understood the nature of the study. Ten were females and 15 males

with a mean age of 58 years (range, 18 to 82 years).

Six of the patients received esophagogastrectomy (one complicated with postoperative pneumonia and sepsis); 5 received coronary artery bypass graft (one complicated with perioperative infarction); 4 received mitral and/or aortic valve replacement; 2 received repair of thoracic aortic aneurysm and 6 received other thoracoabdominal surgical procedures (one complicated with postoperative pneumonia). The remaining 2 patients had severe burns and were complicated with inhalation injury. All were hemodynamically stable and recovering from the critical condition which had necessitated MVS. No patients had COPD or impaired function of the respiratory muscles due to neuromuscular disease.

Each patient was evaluated at our routine morning conference with regard to undergoing a weaning trial. The decision whether to try weaning or not was primarily based on clinical assessment and the patient's ability to generate a forced vital capacity (FVC) of at least 10 $ml \cdot kg^{-1}$ body weight or greater. In some of the patients, FVC was a little bit lower than 10 $ml \cdot kg^{-1}$ body weight, but a weaning trial was still judged clinically appropriate if a patient's general physical condition was good.

The study was conducted in a semirecumbent position. The lead II of ECG and systemic arterial pressure were monitored continuously. A radial or dorsalis pedis arterial line was used to obtain arterial blood samples. All blood gas tensions and pH were measured with a Corning 168 pH/blood gas analyzer. Just prior to each weaning trial, arterial blood gases, with an ordinary F_{IO_2} over 0.2, were measured to calculate the ratio of the arterial oxygen pressure to the fraction of inspired oxygen (Pa_{O_2}/F_{IO_2}). Then, the patients were ventilated with pure oxygen for 15 min, for calculating the alveolar-arterial oxygen tension difference ($A\text{-}aD_{O_2}$) with an F_{IO_2} of 1.0.

FVC, minute ventilation (MV) and maximal voluntary ventilation (MVV) were measured during a brief period off the ventilator by using a Wright respirometer (Medishield, U.K.). Spontaneous respiratory rate (RR) was measured at the time of the measurement of MV. Tidal volume (V_T) was calculated from MV and RR. The maximal voluntary ventilation to minute ventilation ratio (MVV/MV) was calculated by dividing MVV by MV.

Airway pressure was monitored through a small catheter (2.0 mm ID and 2.7 mm OD) inserted into the endotracheal tube through the side port of a tube connector. The tip of the airway pressure catheter was placed 10 cm distal from the top of the endotracheal tube. The proximal side of the airway pressure catheter was connected to a pressure transducer (P23ID, Statham Instruments, U.S.A.) and airway pressure was continuously recorded on a polygraph (Nihon Koden, Japan) at a speed of 50 $mm \cdot sec^{-1}$. Prior to the measurement of $P_{0.1}$, MVS was discontinued and the patients were placed on the T-piece breathing circuit to allow spontaneous respiration. One minute later, the breathing circuit was disconnected from the connector of the endotracheal tube. Then, the orifice of this connector was tightly occluded with the tip of the finger. The occlusion was started at the end-expiratory phase and was maintained during the first 0.4 – 0.6 seconds of the inspiratory phase. Because of its shortness, it did not disturb the patients and was not noticed by most of them.

Careful attention was paid to the following points: (1) the patients could neither see nor hear the occlusion; (2) the timing of the occlusions was varied enough so that patients could not predict when the occlusions would occur. At least 5 measurements of $P_{0.1}$ were obtained, and the mean value was used for analysis.

After all the measurements were made, it was attempted to wean the patients from MVS. Most patients were directly placed on the T-piece breathing circuit with a large balloon reservoir[10]. Three to 5 cmH_2O of continuous positive airway pressure (CPAP) were applied if clinically needed. For some patients, intermittent mandatory ventilation (IMV) or pressure support ventilation (PSV)

was used as a weaning process. However, IMV or PSV was not used longer than 4 hr. These patients were also placed on the T-piece breathing circuit at the end of the weaning process. The completion of the weaning process was defined as extubation or, for patients with a tracheostomy, the discontinuation of CPAP.

The weaning trial was terminated when the patients complained of severe dyspnea and/or developed anxiety, agitation or diaphoresis, or when acidemia (pH < 7.3) and/or progressive hypercapnea (Pa_{CO_2} > 50 mmHg) developed. Failure to wean was concluded if the patient was placed back on MVS within 24 hr because of respiratory deterioration.

Data and statistical analysis: Of the 25 patients studied, 21 were followed by successful weaning and 4 by failure. In the latter 4 patients who failed to be weaned, a weaning trial was repeated on a different day. As a result, a total of 33 weaning trials were attempted in this study. The weaning trials were grouped as those that were followed by success (n = 24) and those that were followed by failure (n = 9). Group means and standard deviations were calculated. The Student's unpaired t-test was used in the statistical analysis between the 2 groups. $P < 0.05$ was considered significant.

Results

Figure 1 shows individual data of FVC, MV, MVV and MVV/MV divided into the weaning success and failure groups. The mean values of FVC and MVV/MV in the success group were significantly greater than in the failure group (19.4 ± 7.6 versus 12.4 ± 4.6 $ml \cdot kg^{-1}$; $P < 0.05$ and 3.9 ± 2.2 versus 1.5 ± 0.4 times; $P < 0.01$, respectively). The mean value of MV in the success group was significantly lower than in the failure group (6.1 ± 3.1 versus 11.5 ± 3.1 $l \cdot min^{-1}$; $P < 0.001$). However, for all 3 indicators, the wide distribution of individual values showed overlap of bordering values between the success and failure groups. Therefore, a threshold value, separating the success from the failure group, was impossible to determine. The mean values of MVV did not show any statistical difference between the success and failure groups (0.37 ± 0.15 versus 0.27 ± 0.13 $l \cdot kg^{-1} \cdot min^{-1}$).

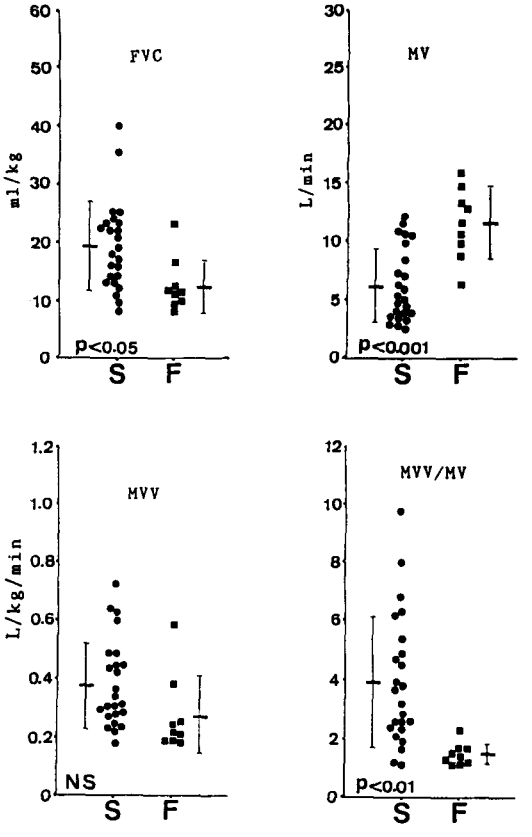

Fig. 1. Individual data of forced vital capacity (FVC), minute ventilation (MV), maximal voluntary ventilation (MVV) and the maximal voluntary ventilation to minute ventilation ratio (MVV/MV) in the weaning success group (S) and in the failure group (F).

Figure 2 shows individual data of RR, V_T, A-aD_{O_2}, and Pa_{O_2}/FI_{O_2} in the success and failure groups. The mean value of RR in the success group was significantly lower than in the failure group (17.4 ± 6.8 versus 29.0 ± 5.1 breaths/min; $P < 0.001$). However, no threshold value separated the success from the failure group, for this indicator either. The mean values of V_T, A-aD_{O_2} and Pa_{O_2}/FI_{O_2} were not different between the success and failure groups (6.7 ± 2.3 versus

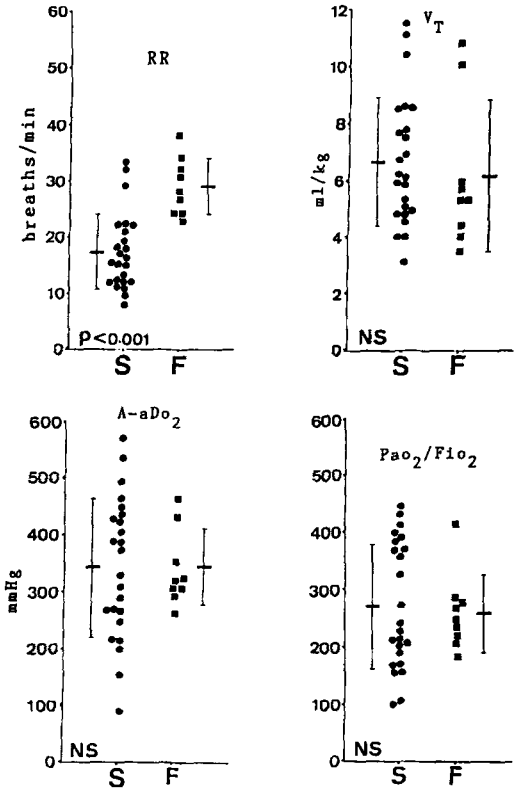

Fig. 2. Individual data of spontaneous respiratory rate (RR), tidal volume (V_T), alveolar-arterial oxygen tension difference with an FI_{O_2} of 1.0 (A-aDo2), and the ratio of the arterial oxygen pressure to the fraction of inspired oxygen (Pa_{O_2}/FI_{O_2}) in the weaning success group (S) and failure group (F).

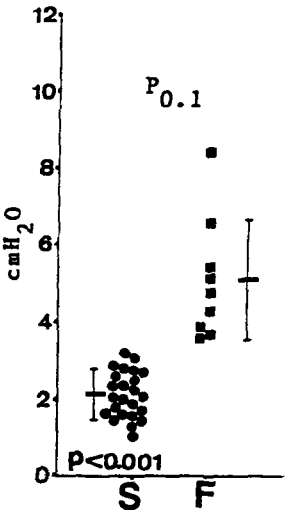

Fig. 3. Individual data of $P_{0.1}$ in the weaning success group (S) and failure group (F).

6.2 ± 2.7 ml·kg^{-1}, 341.5 ± 122.9 versus 340.6 ± 65.1 mmHg and 271.0 ± 109.4 versus 260.6 ± 66.6, respectively).

Figure 3 shows individual data of $P_{0.1}$ in the success and failure group. The mean value of $P_{0.1}$ in the success group was significantly lower than in the failure group (2.2 ± 0.6 versus 5.1 ± 1.5 cmH_2O; $P < 0.001$). All 24 successfully weaned patients had a $P_{0.1}$ of less than 3.5 cmH_2O, whereas all 9 patients who failed to wean had a $P_{0.1}$ of greater than 3.5 cmH_2O.

Discussion

The present study suggests that, in patients with ARF unrelated to neuromuscular disease, $P_{0.1}$, RR, MV, FVC and MVV/MV may serve a predictive function with regard to weaning outcome whereas MVV, V_T, A-aDo$_2$ and Pa_{O_2}/FI_{O_2} may not. Above all, since the $P_{0.1}$ value was clearly different between those patients who succeeded and those who failed to wean, $P_{0.1}$ appears to be more accurate for predicting weaning failure or success than the conventional indicators in patients with ARF.

Measuring $P_{0.1}$ as an index of respiratory center function was first introduced by Whitelaw et al. in 1975[5]. The high $P_{0.1}$ value implies that the activities of the neural inspiratory drive and the inspiratory muscles are high[5,6]. In patients with COPD, it has been found that $P_{0.1}$ is elevated during acute failure and decreases substantially to values equal to what they are in the stable state with an improving respiratory status and recovery[6]. These results suggest that the respiratory centers adapt their activity to the level of mechanical impairment of the respiratory system during the period of acute failure[11]. In other words, a high $P_{0.1}$ implies that a high neuromuscular drive is required to maintain adequate alveolar ventilation against the high mechanical impedance of the respiratory system[11].

A high $P_{0.1}$ was a consistent finding among our patients who failed to wean. Because a high $P_{0.1}$ implies that the high mechanical impedance of the respiratory system is sustained[11], it may lead to inspiratory muscle fatigue, resulting in weaning failure. Similar to our results, 3 other groups of investigators have reported that a high $P_{0.1}$ was predictive of weaning failure[7–9].

Among a group of 16 patients with COPD, Murciano et al.[7] found that 5 patients who had a persistently high $P_{0.1}$ (7.1 ± 2.4 cmH_2O) required reintubation and the 11 who showed a decrease in $P_{0.1}$ (4.7 ± 1.8 cmH_2O) before extubation were successfully weaned. In 11 patients with COPD and 9 with ARF, Herrera et al.[9] found that the mean $P_{0.1}$ values were high in patients who required MVS and were low in patients who were extubated without complication. They suggested that the $P_{0.1}$ of 4.2 cmH_2O separated patients who could and could not be successfully weaned. Recently, in a group of 12 patients with COPD, Sassoon et al.[8] found that 5 patients who failed to wean had a higher $P_{0.1}$ (8.0 ± 0.4 cmH_2O) than the 7 whose weaning was successful (4.0 ± 0.5 cmH_2O). They suggested that the $P_{0.1}$ of 6.0 cmH_2O separated patients who could and could not be successfully weaned.

Our threshold value separating success from failure was lower than those of the foregoing 2 studies[8,9]. Since the inspiratory muscle performance decreases with increasing lung volume due to the length-tension relationship, the difference in lung volume between the subjects of the studies may affect the absolute values of $P_{0.1}$[12]. However, this cannot account for the differences between the results of the foregoing studies on patients with COPD and those of our study on patients with ARF. In general, patients with ARF have a less functional residual capacity than those with COPD[11]. Therefore, a higher $P_{0.1}$ may be generated in patients with ARF if the activity of the neural inspiratory drive is at the same level. One possible explanation for this discrepancy may be offered by the difference in the underlying pulmonary disease. According to the study of Aubier et al.[6], the $P_{0.1}$ values in the normal subjects are lower than those with COPD in a stable state. Since the ordinary $P_{0.1}$ values are different between patients with or without COPD, the threshold values separating success from failure may differ if the subjects' underlying pulmonary disease differs. We suggest that our threshold value of 3.5 cmH_2O should be used only in patients with ARF, free from COPD.

Incidentally, in contrast to the above 3 studies and ours, Montgomery et al.[13] reported that simple measurement of $P_{0.1}$ did not have the predictive ability of weaning outcome. However, in their study, the subjects were composed of not only ARF but COPD, in whom the ordinary $P_{0.1}$ might be different. Their failure to predict the weaning outcome by simple measurement of $P_{0.1}$ might be due to having made no distinction between ARF and COPD.

During the last 15 years, various studies have been carried out to find the most reliable indicator for successful weaning. However, conventional indicators are not always accurate in predicting the ability of patients to wean from MVS[2–4]. For example, on a series of 47 ventilated patients with a variety of illness, Tahvanainen et al.[3] reported that conventional indicators such as FVC, MVV, MV, MVV/MV, V_T, RR and peak negative pressure did not predict the need for reintubation. Morganroth et al.[4] also reported that for 11 patients requiring prolonged MVS, FVC, MV, RR, V_T and peak negative pressure were not useful in judging the ability to wean. In the present study, some of the mean values of conventional indicators appeared to separate patients who could and could not be weaned. However, overlaps of values between the success and failure groups were observed, suggesting that the indicators might not be accurate for predicting weaning outcome.

The drawback of some of these conventional indicators, such as FVC, MVV, MVV/MV and peak negative pressure, is that they require the patient's good cooperation. In addition, tests of maximum effort, such as FVC and peak negative pressure, provide limited information regarding res-

piratory muscle fatigue[2]. A high MV may indicate the presence of increased dead space ventilation and/or increased CO_2 production. However, a low or normal MV does not always indicate the absence of increased dead space and/or increased CO_2 production. A low or normal MV may arise from decreased respiratory center drive, a structural abnormality of the lungs or of the thoracic cage, or respiratory muscle dysfunction. Moreover, a high RR may be an evidence of respiratory distress[14,15]. However, tachypnea is a nonspecific sign and may arise from an illness unrelated to the lungs.

In contrast, an advantage of $P_{0.1}$ is that it is independent of the patient voluntary cooperation[5]. Since $P_{0.1}$ represents the central respiratory drive that is activated when the lungs are mechanically impaired, it is not surprising that the patients with a high $P_{0.1}$ are susceptible to respiratory muscle fatigue[7,11].

In conclusion, we have shown that $P_{0.1}$ is a clinically useful indicator for predicting the weaning outcome in patients with ARF unrelated to neuromuscular disease. Since $P_{0.1}$ is a simple, noninvasive and highly reproducible technique, we recommend routine measurements of $P_{0.1}$ along with the conventional indicators. Measurements of $P_{0.1}$ will offer not only an information about the level of mechanical impairment of the respiratory system but also a more accurate prediction of the weaning outcome in patients with ARF.

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