Mouth Pressure Curve on Abrupt Interruption of Airflow during Forced Expiration

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Ohya, N., Huang, J., Fukunaga, T. and Toga, H. Mouth Pressure Curve on Abrupt Interruption of Airflow during Forced Expiration. Tohoku J. exp. Med., 1988, 155 (1), 103-104——The mouth pressure curve after abrupt interruption during forced expiratory maneuver was investigated to evaluate the collapsing state of the airway downstream to the choke point. Immediately after the airflow interruption at the mouth by means of the electromagnetic valve, the mouth pressure suddenly increased (1st phase), followed by a slower rise (2nd phase) within about 100 msec until the pressure reaches the alveolar pressure. The pleural and alveolar pressures remained constant during this process. It was evidenced that, from point of view of mean flow, the airflow flowed at a rate of $V_{\text{max}}$ through the choke point during the second phase. Thus, it is strongly suggested that the choke point remained at the same point during the 2nd phase. From these results, the 2nd phase of the mouth pressure is expected to represent the specific characteristics for the downstream airway.

The behavior of the airway downstream to the choke point was investigated by analyzing the changes of mouth pressure on abrupt interruption and restoring at the mouth during forced expiration.

The mouth pressure ($P_{\text{ao}}$) was measured with a differential pressure transducer (Model P-3000S, Copal, Tokyo), the airflow using a Fleisch-type flowmeter (Type 3, Hewlett Packard, Andover, MA, USA), and a differential pressure transducer (Model TP-602T, Nihon Kohden Kogyo, Tokyo). The pleural pressure was estimated using an esophageal catheter, of which frequency characteristics including the transducer is flat up to 20 Hz.

The behavior of the mouth pressure revealed to be two phasic, i.e., step-functional phase (1st phase) which is only due to kinetic energy conversion of airflow to pressure, and a slower 2nd phase (2nd phase) (Fig. 1C). During the 2nd phase, no change in the pleural pressure was observed (Fig. 1D), and a possibility of contribution of gas compression in the airway was excluded theoretically. The results of the series of $V_{\text{supramax}}$ (Fig. 1B), which was observed on abrupt restoring of airflow before the mouth pressure reaches the level of the

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alveolar pressure, indicated that from point of view of mean flow measured at the mouth, the air was continuing to flow with \( V_{\text{max}} \) from upstream segment through the choke point during the 2nd phase. According to Dawson and Elliott (1977), this fact means also the choke point is preserving the same point, that is, the disturbance occurred in the downstream cannot be transmitted upward through the choke point during the 2nd phase.

In conclusion, with abrupt interrupting and restoring the airflow at the mouth during forced expiration, the airway wall downstream to the choke point is undergoing change of the state ranging collapsing to relaxing. Its physical characteristics will be reflected in changes in the 2nd phase of the mouth pressure.

**Reference**