In Reply—We are grateful to Dr. Enk et al. for the comments, although we believe that they are based on misunderstandings and erroneous assumptions related to our article.¹ Their calculations are probably redundant since in the in vitro experiments precisely this, the very thing that they tried to calculate, was considered: the effective volumes delivered from the pressurized gas bottle were measured and compared to the volumes inferred from the flows displayed on the flowmeter. It was demonstrated that the volumes that were delivered differed from those that could be inferred from the preset flows (table 2). In addition, their calculations are incomplete, since they erroneously missed including the extremely high resistance of the transtracheal cannula and the manual respiration valve itself.

Another basic assumption of the authors of the comment is that a pressure-compensated flowmeter was not used, which indeed would have been an error, whereas in reality we used a pressure-compensated flowmeter (2M 85503; Dräger, Lübeck, Germany) but unfortunately did not specify this in the article. The nonpressure-compensated flowmeters are, contrary to what is claimed, rarely used anymore, if at all. Readily available pressure-compensated flowmeters have an error allowance of up to 10%. Higher-precision pressure-compensated flowmeters exist, but they also have their limits. Therefore when there is no need for them, as in our experiments, their use is not justified. If precise volume measurements are needed, and they could be performed, direct volume measurement should and would be employed.

The primary outcome measure in the in vitro experiment was the time to achieve, using the manual respiratory valve device that uses the venturi principle, a volume of 1 l, which was measured and not extrapolated from the flow (p. 252). The flow measurements, since they are quite unreliable, were merely used to have some control and not for a precise quantitative measurement of gas delivery (p. 253, column 2). Tables 2 and 3 display the measured values for time to 1 l so that the minute volume could be directly extrapolated if the expiration time with the valve is taken into account. The values given in these tables permit to verify the “effective flows.” For example (in table 3), if for the preset flow of 6 l/min (by the compliance of 0.1 l/mbar and resistance of 2 mbar · l⁻¹ · s⁻¹), 10.4 s are needed to achieve a lung volume of 1 l, then effective flow is 5.76 l/min and not 6 l/min. Or, if for the preset flow of 12 l per min, 6.3 s are needed to achieve the lung volume of 1 l, then the effective flow is 9.52 l/min and not 10 l/min. For the reasons of clarity, these and numbers of other calculations were not given in the article.

In the in vivo experiments, the primary outcome measure was the achieved gas exchange as expressed as oxygen and carbon dioxide partial pressures in the arterial blood and not the “exact” lung volumes. Indeed, we were not completely ignorant of the volumes. The knowledge of the effectively delivered volumes acquired from the in vitro experiments, simultaneous registration of the thorax expansion with plethysmography (in the preliminary experiments using a capacitive thoracic circumference measuring device, p. 253), correlation of those to the known volumes delivered by the mechanical respirator (during the 30-min periods of transition) and continuous tracheal pressures monitoring, permitted us to have some idea of the volumes achieved during transtracheal ventilation with the manual respiratory valve. The fact that by low lung volumes compliance is higher was beneficial for the in vitro experiments, since the target tidal volumes were very small (table 1) in comparison to the “1-Liter Test,” and the effect of compliance was certainly minimal in these experiments.

The “hyperventilation of the animals” was not a shortcoming of the study. The use of an exceptionally small transtracheal cannula and low oxygen flows permitted demonstrating that, under extremely unfavorable conditions, it is just possible to provide an adequate or sufficient gas exchange. In these experiments, the obtained carbon dioxide partial pressures of 63 mmHg at a steady state and the oxygen partial pressures much higher than physiologically necessary would certainly assure survival for at least 1 h, until a secure airway could be established. It is indeed quite intuitive that the normoventilation in an adult patient could be easily achieved by simply turning gas flow a bit higher.

The capacity of the valve to increase minute ventilation by increasing flow was not systematically measured. It could be calculated (from tables 2 and 3) that doubling the flow produced about 31.88% to 35% (with a 12- or 16-gauge cannula) increase of minute volume. With a 12-gauge cannula and flow of 12 l/min even by extremely low compliance, a volume of over 4 l is possible, and doubling or tripling that flow may achieve volumes which would be certainly sufficient for ventilation of an adult person.

These experiments demonstrated where the low limits of that method actually lie, and how feasible it would be to use the manual respiratory valve for transtracheal lung ventilation in clinically near-emergency situations with the material normally available in ambulances. The device that we presented as compared to other available devices for transtracheal emergency ventilation has just one additional feature: it provides an expiratory aid that shortens the expiration and can therefore deliver and remove, per minute, larger volumes of gas than the other devices. One such device that is common in Germany is the Enk Oxygen Flow Modulator.* Indeed, one of the authors of the present comment has a serious undisclosed conflict of interests concerning the devices for transtracheal ventilation, which is precisely the subject of the paper they discuss. Certainly if the authors of the comment had disclosed their conflict of interests the moral problem that overshadows their interesting comments would have easily been avoided.

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Reference


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