part after informed consent. All probands were healthy nonsmokers and took no medication. All were low land dwellers, but experienced leisure time in the mountains. Lung function was normal in all subjects. Subjects were randomized to inhale their own expi-
ratatory gas or environmental air as placebo control after performing CPR at 171 m altitude in the University Clinic and on the follow-
ing day after cable car transport and 4 h of adaptation time at 3,000 m in a mountaineering resort. Similar to the Wenzel et al trial, subjects performed one-rescuer CPR after the guidelines of the American Heart Association for 10 min on a CPR manikin (Resus-
ci-Anne, Laerdal Medical Corp; Armonk, NY). Rescuers did not ventilate the manikin, but exhaled into gas collection equipment. The nose of the rescuers was blocked by a nose clip; subjects inhaled environmental air through a mouth piece. Expiratory air was collected in a 100-L bag via a valve. The bag was sealed after 10 min. Subjects were allowed to rest for 30 min. Then the value between the bag with the expiratory air was switched and subjects (now as “victims”) inhaled their own expiratory gas mixture for 10 min and expired into the environment. Capillary blood samples were obtained from the arterialized ear lobe before and after 10 min of inhaling expiratory gas. One investigator coached the subjects during the “victim phase” of the experiment to keep ventilation rate to 12 per minute. The next day, subjects crossed to the other study branch. In the placebo control, the same procedure was performed, but subjects inhaled from an identical bag filled with environmental air. Subjects were blinded to the content of the air bag. Blood samples were stored in an ice water containing cooling box and arterial oxygen tension (PaO$_2$) and arterial carbon dioxide tension (PaCO$_2$) analyzed within 2 h on a BGE Blood Gas Analyser (Instrumentation Laboratory: Milano, Italy). Statistically, direct comparison of the differences of blood gas analysis before and after inhalation of expiratory gas or environmental air was performed by t test. Data are expressed as means±standard deviations and 95% confidence interval (CI) of the mean pair differences are reported. Probability levels less than 0.05 were considered significant.

At 171 m altitude, after inhalation of expiratory gas for 10 min, PaO$_2$ decreased from 87.0±3.67 mm Hg to 78.2±2.17 mm Hg, a mean decrease in the paired differences of 8.8 mm Hg (95% CI 4.4 to 13.2, p<0.01). PaCO$_2$ increased from 37.2±1.3 to 44.8±1.92 mm Hg, a mean increase in the paired differences of 7.6 mm Hg (95% CI −9.99 to −5.21, p<0.01). At 3,000 m altitude, after inhalation of expiratory gas for 10 min, PaO$_2$ decreased from 72.2±1.3 mm Hg to 67.6±1.82 mm Hg, a mean decrease in the paired differences of 4.6 mm Hg (95% CI 2.29 to 6.91, p<0.01). PaCO$_2$ increased from 33.2±0.84 to 37.4±0.55 mm Hg, a mean increase in the paired differences of 4.2 mm Hg (95% CI −5.24 to −3.16, p<0.01). Direct comparison of the decreases in PaO$_2$ at both investigation altitudes showed no statistical difference (9.0±5.61 mm Hg vs 4.6±0.89 mm Hg, difference 4.4 mm Hg, 95% CI −1.46 to 10.26, p=NS). Direct comparison of the increases in PaCO$_2$ at both investigation altitudes showed a statistically elevated increase in PaCO$_2$ at 171 m altitude (−7.6±1.67 mm Hg vs −4.2±1.1 mm Hg, difference −3.4 mm Hg, 95% CI −5.46 to −1.34, p<0.01). At both investigation altitudes, PaO$_2$ and PaCO$_2$ did not change in the placebo test.

Resuscitation at moderate altitude in an alpine country is not rare. Hypothermic victims of avalanches frequently undergo prolonged preclinical resuscitation, sudden cardiac death among mountain hikers and downhill skiing is not utterly uncommon. Altitude tolerance and altitude adaptation depends on hyperventilation, which is induced by the carotid bodies, although this effect is counteracted partly by the negative feedback resulting from the reduction of PaCO$_2$. Although our small trial was carried out with healthy subjects and not with patients in cardiac arrest, our data at low altitude support the findings by Wenzel et al. PaCO$_2$ increased by 20.4% at low altitude but only by 10.2% at 3,000 m altitude after inhalation of expiratory gas. This effect is obviously due to the decreased CO$_2$ content of expiratory gas at moderate altitude due to altitude induced hyperventilation. Our data clearly indicate, that the danger of contributing to hypercarbia by mouth-to-mouth ventilation is less pronounced at moderate altitude than in the low lands.

**REFERENCES**

1 Hills M, Armitage P. The two-period cross over clinical trial. Br J Clin Pharmacol 1979; 8:7-20


To the Editor:

We are pleased to see an additional study of mouth-to-mouth ventilation from our colleagues in Vienna. Their study was elegant in design and took our study one step further by showing the effect of breathing exhaled gas on capillary blood gases.

Their study showed that at an altitude of 3,000 m (10,000 ft), breathing exhaled gas caused an average decline of PaO$_2$ to 67 mm Hg. Although the PaCO$_2$ increased at altitude, it was less than the increase near sea level, probably because of hypoxic hyperventilation at altitude.

Our concern with the use of mouth-to-mouth ventilation during cardiac arrest arises from an experimental model in which we found that hypercarbia or hypoxia independently had an adverse effect on resuscitation from cardiac arrest. Of the animals ventilated with 95% O$_2$ and 5% CO$_2$ or 10% O$_2$ and 90% N$_2$, only 1 of 5 in each group could be resuscitated compared with 6 of 8 animals ventilated with 85% O$_2$ and 15% N$_2$.

Although exhaled gas at high altitude contains less CO$_2$, it may still be undesirable for ventilation of cardiac arrest because of the lower oxygen concentration at altitude. We also wonder if the exhaled CO$_2$ concentration would be greater in individuals who are fully adapted to altitude.

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**REFERENCE**