Title: CAPILLARY PERFUSION DURING ISOFLURANE AND ENFLURANE

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Introduction: Recent experiments suggested that the volatile anesthetics Isoflurane (I) and Enflurane (E) induce shunt-perfusion at the capillary level in the healthy myocardium of dogs (2). The direct assessment of the myocardial microvascular bed, however, is impeded by the movement of the heart due to the cardiorespiratory cycles. In addition, though the general anesthesia produces significant changes in the microcirculation (3), detailed and direct investigations with I and E are rare. We therefore studied the effects of I and E on capillary blood flow in an in-vivo muscle preparation.

Methods: Transparent aluminium access chambers were implanted into the dorsal skinfold of healthy male Syrian Golden hamsters (body weight: 70-80 g) during pentobarbital anesthesia (1). For monitoring of systemic pressures and heart rate, for blood gas sampling and for injection of dyes and drugs, catheters were advanced into v. cava superior and into the right carotid artery. At least after 48-72 h recovery from microsurgery and anesthesia preparations demonstrating an intact muscle microcirculation (4) were used for the acute experiments only (n=10).

One day prior to the acute experiment hamsters of the inbred strain were exsanguinated and red cells labeled with fluorescein-isothiocyanate (FITC). 0.3 ml of this suspension (hematocrit 50%) was injected intravenously. Assuming that only rheological normal behaving FITC-red cells were still recirculating after 30 min, microcirculatory measurements were started after this period. For that purpose the awake animals were immobilized in a transparent plastic tube and the skin muscle microcirculation visualized by incident fluorescence videomicroscopy. The whole capillary network supplied by one terminal arteriole was recorded via a low-light-level TV-camera on videotape. Anesthesia was induced by I (n=5) or E (n=5) and the animals tracheotomized, intubated and ventilated mechanically to maintain arterial blood gas values at baseline levels. Videorecordings of the identical microvessels were repeated after 1 MAC I or E for 1 h. Endexspiratory I- and E-concentrations were measured by a multigas-analyzer and MAC was determined for each animal by standardized tail-clamping. 1 MAC for I was 1,3 vol% and for E 1,6 vol%. During anesthesia 1,3 ml/100 g b.w. homologous blood was transfused. Temperature in the preparation area was controlled continously and adjusted to 32° C by a feedback-controlled heating-system. Arterial blood gases were controlled prior to and at the end of the videorecordings. Vessel diameter (d), red cell velocity (v) and red cell flux (F) were analyzed offline frame-to-frame at magnifications of 1200x.

Results: With I and E arterial pressure decreased significantly: I:101 vs 79 mmHg; E:99 vs 75 mmHg. Arterial blood gases did not change significantly as compared to baseline values. Changes in capillary perfusion with I and E are given in fig. 1 and 2. The upper part shows the red cell fluxes in single capillaries and the lower one the corresponding absolute change with I and E. Changes are given in a rank order. In the awake animals the number of capillaries not perfused by red cells was 10 % (I-group) and 20% (E-group). During anesthesia this proportion increased to 35 % (I) and to 30% (E). F of the total capillary network was reduced by 50% (I) and by 30 % (E). Terminal arterioles (d; 19-10 um) were dilated with I and E by 10 % and precapillaries (d; 9-3,5 um) as well by 25 and 10 %, respectively. v in terminal arterioles and precapillaries decreased by 40 and 27 % with I. Corresponding values for E were -4 % and 24 %. v in capillaries was reduced by 9 % (I) and increased by 20 % (E).

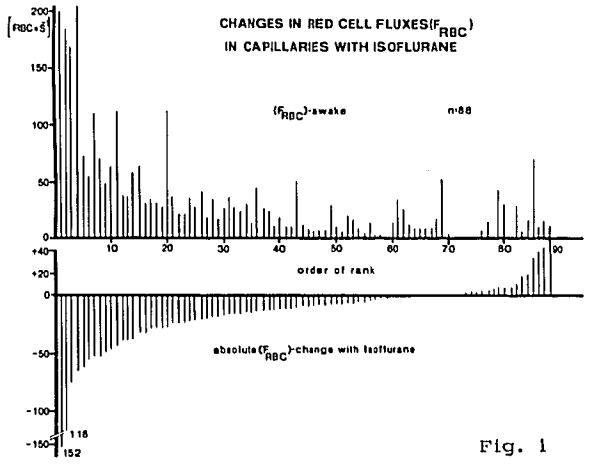

Fig. 1

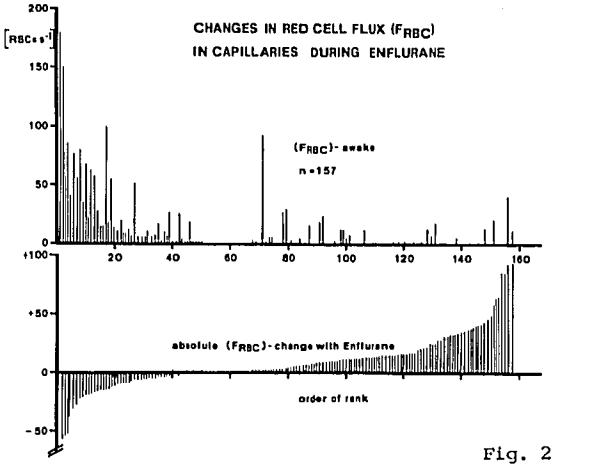

Fig. 2

Summary and conclusions: Although both anesthetics induced marked microcirculatory changes, no significant differences between I and E were measured in this chronic striated muscle preparation. I and E induced a vasodilation of terminal arterioles and precapillaries as compared to the awake state. The functional capillary density decreased markedly. This was accompanied by a reduction of the capillary red cell flux, i.e. nutritional capillary flow. With decreasing capillary perfusion a redistribution of flow occurs within the capillary network as demonstrated in fig. 1 and 2. This possibly indicates preferential channel perfusion. Decisive conclusions, however, can only be drawn after exact topographical network analysis of the microangioarchitecture and its correlation to microhemodynamic changes.

References:

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