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alphabetical order: a, b, c, d, e, f,

g, h, i, j, k, l, m, n, o, p, q, r, s, t,
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advantage of a lower center of
gravity, which allows them
to. Effect of prolonged adrenaline
infusion on mitochondrial
function and metabolism. beta-
Adrenergic blockade with
propranolol does not influence
the development of the hypoxic
ventilatory response. This
indicates that beta-adrenergic
stimulation is only indirectly

involved in stimulating ventilation during hypoxia. The hypothesis is that there is a direct beta-adrenergic effect on ventilatory muscle metabolism.

The authors investigated this possibility by infusing epinephrine for 4-8 days into the arterial circulation of dogs.

Ventilatory response to hypoxia was recorded by plethysmography.

Phosphorylcreatine kinase activity and oxygen uptake by

isolated mitochondria were measured to monitor energy metabolism of the dogs' ventilatory muscles. Prolonged, steady-state, hypotensive doses of epinephrine do not increase oxygen uptake by isolated mitochondria.

Phosphorylcreatine kinase activity in the ventilatory muscles, a measure of ATP availability, increases during hypoxia for 2-3 days and then decreases when prolonged

infusion is continued. In acute experiments hypoxia alone increases phosphorylcreatine kinase activity. Propranolol does not influence the initial increase in phosphorylcreatine kinase activity observed in response to hypoxia. Results indicate that prolonged infusion of adrenaline is not associated with a direct metabolic effect on ventilatory muscles. The ability of epinephrine to induce a positive ventilatory response to hypoxia is

probably due to an indirect, beta 3da54e8ca3

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