Coronary Circulation Response
to Hyperoxia after Vagotomy and Combined
Alpha and Beta Adrenergic Receptors Blockade
in the Anesthetized Intact Dog*

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Received November 6, 1968

Summary. In closed-chest vagotomized dogs with alpha and beta adrenergic
receptors blockade, ventilation with 100 per cent oxygen at atmospheric pressure
did not modify the tension-time index nor the myocardial oxygen consumption.
However, coronary blood flow decreased and coronary resistance increased signifi-
cantly. A rise of the myocardial \( pO_2 \) was not likely to be primarily responsible for the
elevation of the coronary resistance. Since in the presence of a fixed oxygen con-
sumption the myocardial \( pO_2 \)-elevation would occur following the rise of the arterial
\( pO_2 \) whose direct effect on the vascular smooth muscle tone has been demonstrated
in vitro by other workers, it may be concluded that elevation of arterial \( pO_2 \) exerted
a direct constrictive action on the coronary vessels.

However, oxygen transport to the left ventricle remained commensurate to the
myocardial oxygen consumption. It is suggested that an additional mechanism
adjusted the elevated coronary resistance. Shifts of the myocardial \( pO_2 \), resulting
from transient imbalances between oxygen supply and demand, may be the
stimulus initiating the adjustment through changing release of vasodilator or vaso-
constrictor substances.

Results of this paper and those previously published indicate that autonomic
influences normally play a dominant role in the hyperoxia-induced reduction in
cardiac work and metabolism.

Key-Words: Blood Oxygen Tension — Coronary Blood Flow — Coronary

In the open-chest dog, ventilation with 100 per cent oxygen at one
atmosphere absolute pressure has been shown to induce a decrease in the
coronary blood flow [8,9,16,23] which can be accentuated by raising the
inspired oxygen pressure from one to three atmospheres [16]. By resorting

* This investigation was supported in part by a grant from the Fonds National
de la Recherche Scientifique of Belgium.

A preliminary report on these results was made at the XXIV International
to a radioactive inert gas method for coronary flow measurement [12, 19], we have recently confirmed this observation in the intact anesthetized dog artificially ventilated with pure oxygen at atmospheric pressure, under controlled baseline conditions as far as the arterial blood oxygen saturation and pH are concerned [15]. In particular, it was observed that, in addition to cardiac slowing, the hyperoxia-induced decrease in coronary flow and increase in coronary resistance were associated with a lowering of the tension-time index and a reduction of the myocardial oxygen consumption. Despite the elevated arterial blood oxygen content, oxygen transport to the left ventricle decreased in proportion to the reduction of the oxygen consumption of the left ventricle.

The mechanisms responsible for the elevation of the coronary resistance during hyperoxia are still unsettled. It is pertinent, however, to consider that oxygen breathing has been demonstrated in the normal man to result in a vagus-dependent decrease in heart rate, a rate-dependent decrease in cardiac output, an increase in mean arterial blood pressure and an increase in systemic resistance [5, 10]. Vagal activity has been evidenced by atropine-induced blockade of the changes in heart rate and cardiac output [5]. Cardiac slowing on oxygen breathing has been attributed either to a chemoreceptor reflex [5, 7] or to a baroreceptor reflex elicited by the elevation of the arterial blood pressure as a result of peripheral vascular constriction [10]. Supporting the baroreceptor reflex hypothesis is the finding of reduced urinary excretion of both adrenaline and noradrenaline [11]. However, the exact cause of the peripheral vascular constriction is still conjectural [10], although in vitro experiments on small isolated femoral arteries [4] and various isolated intestinal and vascular smooth muscle preparations [22] point to a direct effect of the elevated oxygen tension on the vascular smooth muscle tone.

The hyperoxia-induced elevation of the coronary resistance might thus be related to 1.—a direct effect of the high oxygen tension on the coronary vessels, and 2.—indirect effects resulting from the resetting of the autonomic nervous system toward vagal activity and sympatho-adrenal depression. The latter effects could be mediated by the accompanying reduction in cardiac work and metabolism leading to autoregulation of the coronary circulation.

Elimination of autonomic influences would thus be expected to offer some insight into the mechanisms involved.

**Methods**

Twenty adult mongrel dogs of either sex, ranging in weight from 17.6 to 46.0 kg, were sedated with morphine (2 mg per kg, subcutaneously) and anesthetized 30 min later with pentobarbital (20 mg per kg, intravenously). The trachea was cannulated through a midline cervical incision and intermittent positive pressure respiration