

Nunn J F, Bergman N A & Coleman A J. Factors influencing the arterial oxygen tension during anaesthesia with artificial ventilation.

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During anaesthesia with paralysis and artificial ventilation, oxygen consumption was 10 percent below basal, and physiological dead space was 36 percent of tidal volume. Mean alveolar/arterial  $pO_2$  gradient was 52 mm Hg with inspired oxygen concentration in the range 21-30 percent and 145 mm Hg at 98 percent oxygen. This corresponded to a mean shunt of 10 percent but correlated positively with age. [The SCJ® indicates that this paper has been cited in over 145 publications.]

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Before 1964 almost nothing was known about the effect of anaesthesia on the oxygenation of arterial blood. This was largely because of difficulty in the measurement of arterial  $pO_2$  in the presence of nitrous oxide. Absorption techniques (such as the Riley bubble) were impossible to use, and indirect derivation from saturation was too insensitive at high levels of  $pO_2$ . All of this changed when the polarograph became a practical possibility. Long before this device became available commercially, I had built an electrode as described by my former colleagues J.M. Bishop and A.C. Pincock,<sup>1</sup> fusing the platinum cathode into lead glass and grinding the surface flush by hand. We had carefully established the accuracy of our system in the presence of anaesthetics, and I had already undertaken a study of arterial oxygenation during anaesthesia with spontaneous breathing.<sup>2</sup>

In 1964 I was working for the Medical Research Council with Tony Coleman, who later

became professor of anaesthesia in Durban, South Africa. Happily, we were joined by Norman Bergman, then on sabbatical, who was later made professor and chairman of anaesthesiology at Portland, Oregon. In the course of a very busy year, we undertook one of the first studies of the factors influencing arterial  $pO_2$  during anaesthesia with paralysis and artificial ventilation.

Patients were studied at the Royal Postgraduate Medical School where I held an honorary contract. We confirmed our earlier measurements of oxygen uptake<sup>3</sup> and physiological dead space,<sup>4</sup> measured the alveolar/arterial  $pO_2$  gradient, estimated the venous admixture, and studied the effects of sustained positive airway pressure, long before the concept of positive end-expiratory pressure (PEEP) had become fashionable. We clearly demonstrated that alveolar/arterial  $pO_2$  gradient and venous admixture were increased far above the normal values for conscious subjects, although the effect was directly related to the age of the patient. We showed that sustained airway pressure up to 30 cm of water had no significant effect on the gradient, and we defined the inspired oxygen concentration that was required to ensure a satisfactory arterial  $pO_2$  in the anaesthetised patient.

Our conclusions have stood the test of time, but there has been much work by us and others to determine the precise causes of the changes that we observed. Before the paper was published, N.A. Bergman, A.J. Coleman, and I had dispersed to separate continents.

I suspect that our paper has been so frequently cited because it was one of the first to provide the essential physiological data on which the oxygenation of an anaesthetised, paralysed, and ventilated patient could be based. The most important recent contribution to the field is by L. Bindslev and his colleagues.<sup>5</sup>

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2. Nunn J F. Factors influencing the arterial oxygen tension during halothane anaesthesia with spontaneous respiration. *Brit. J. Anaesth.* 36:327-41, 1964. (Cited 115 times.)
3. Nunn J F & Matthews R L. Gaseous exchange during halothane anaesthesia: the steady respiratory state. *Brit. J. Anaesth.* 31:330-40, 1959. (Cited 60 times.)
4. Nunn J F & Hill D W. Respiratory dead space and arterial to end-tidal  $CO_2$  tension difference in anesthetized man. *J. Appl. Physiol.* 15:383-9, 1960. (Cited 165 times.)
5. Bindslev L, Hedenstierna G, Santesson J, Gottlieb I & Carvallhas A. Ventilation-perfusion distribution during inhalation anaesthesia. *Acta Anaesthesiol. Scand.* 25:360-71, 1981. (Cited 20 times.)