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In reply:—Oeseburg and Kwant emphasize that many sources of error exist in the determination of blood P_{O_2} and that these may be related to blood sampling, sample handling, and the measuring system. However, even with the most meticulous sampling and handling techniques, there will be differences between observed and actual P_{O_2} values. Therefore, the purpose of our report[1] was twofold: first, to point out that the measuring system introduces errors specific to each particular system; second, to show how it is possible to eliminate these errors of a measuring system by applying a standardized reference method based on blood tonometry using a well-defined reference system, and transforming the results onto a nomogram. We did not intend to produce a complete record of possible errors.

As can be seen from our nomogram, the inaccuracy of the oxygen analyzer increases with increasing blood P_{O_2} values, reaching deviations of more than 20 per cent at P_{O_2} levels of more than 500 torr. This is to a certain extent dictated by the shape of the oxyhemoglobin-dissociation curve. The remarks of Oeseburg and Kwant concerning the different slopes and intercepts of such nomograms suggest that they will be linear, but in fact the ABL 1 nomogram is nonlinear.

An additional topic of current interest should be mentioned to complete the subject. It is of particular concern to anesthesiologists that halothane may have a considerable effect on the stability of the P_{O_2} electrode due to the polarographic reduction of halogenated hydrocarbons.[2] It appears that with the ABL 1 system even a single exposure to blood containing halothane, 1 per cent, results in a gradual upward drift in the electrode calibration, and that this effect may persist for several hours.[3]

MORTEN M. BRINKLØV, M.D.
POUL K. ANDERSEN, M.D.
DAG B. STOKKE, M.D.
PETER HOLE, M.D.
Department of Anaesthesia
Odense University Hospital
DK-5000, Odense C, Denmark

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Hypothermia and Neuromuscular Blockade

To the Editor:—Like Ham *et al.*,[1] we have observed and reported the prolongation of nondepolarizing neuromuscular blockade when the temperature of muscle is decreased.[2,3] In control experiments we have also demonstrated, in both man[4] and dog,[5] that hypothermia alone will produce a decrease in the indirectly elicited twitch response (fig. 1), an effect that is antagonized by edrophonium. This clearly demonstrated that hypothermia to less than 32 C in man and 29 C in the dog critically decreased acetylcholine mobilization and release, which is fundamental to neuromuscular transmission. The effect of cold on acetylcholine mobilization has been demonstrated to be biphasic, with a transient initial increase followed by a marked diminution.[6] Temperatures at which this failure occurs vary according to the species studied, being lower in hibernating animals and amphibians than in the higher species of mammals.[7] It is probable that this failure of acetylcholine mobilization is the cause of the increased synaptic delay time that occurs during hypothermia.[8] It is most probable, therefore, that it is this critical decrease in the margin of safety of neuromuscular transmission that results in the prolongation of the effect of the nondepolarizing relaxants during hypothermia, an effect that will be exacerbated by the decrease in renal clearance observed by Ham *et al.*, but was unlikely to have contributed to the prolongation of block observed in our