Inaccuracy of Oxygen Electrode Systems

To the Editor.—In the letter of Andersen et al. 1 concerning the article by Dueck et al., 2 a nomogram for the correction of measured $P_{O_2}$ values is recommended. Andersen et al., however, do not make clear the restriction to be imposed on the use of this nomogram. The nomogram, as constructed by Radiometer, corrects exclusively those changes in $P_{O_2}$ values that occur during the stay of the sample within the ABL 1 measuring system.

Many sources of error may contribute to the difference between the $P_{O_2}$ actually present in the arterial blood and the value given by a certain apparatus (fig. 1). These include: A) During sampling a syringe (glass or plastic), mostly containing a heparin solution with a certain $P_{O_2}$ in the dead space, is filled with a certain amount of blood having the blood $P_{O_2}$ at the tip of the sampling needle at that moment. B1) If any air bubbles are in the syringe directly after the sampling, these are either expelled or not. B2) Samples are stored for variable periods at different temperatures between 0 and 30 C. B3) During transportation for variable periods, further changes in temperature may occur. C1) At the moment of introduction some technicians flush the measuring system with a part of the sample; others introduce the sample according to the instruction manual. C2) The measuring system contains gas or a buffer solution with a $P_{O_2}$ that differs from the sample $P_{O_2}$. This contamination gives rise to the so-called memory effect. During the stay of the sample in the thermostatted measuring circuit, metabolism further decreases $P_{O_2}$. At a given moment, when equilibrium is attained between sample and electrode, electrode $P_{O_2}$ is assumed to be sample $P_{O_2}$.

The errors introduced by factors A–B3 are particularly variable, due to many unknown factors. Some of these factors could at least be standardized: dead space $P_{O_2}$ and oxygen capacity; dead space/sample volume ratio; storage and transportation temperatures; time between sampling and introducing the sample into the measuring circuit. Such standardization would lead to better precision, but would not correct for changes in $P_{O_2}$ values due to metabolism (which is not totally blocked by the addition of NaF to the heparin), and due to diffusion. So the magnitude of errors A–B3 will remain unknown. Error C1 can be prevented by following the instruction manual. In that case only, the presented nomogram gives a correction for errors C1–C2. Moreover, it holds only for the Radiometer ABL1 (which was not actually used by Dueck et al.). Of course, similar nomograms could be calculated for other electrodes and machines. Presumably, these will have different slopes and intercepts.

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**Fig. 1. Flow chart for blood-gas measurements.**

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In reply:—Oeseburg and Kwant emphasize that many sources of error exist in the determination of blood P02 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 P02 values. Therefore, the purpose of our report1 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 P02 values, reaching deviations of more than 20 per cent at P02 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 P02 electrode due to the polarographic reduction of halogenated hydrocarbons. 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.

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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 man4 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