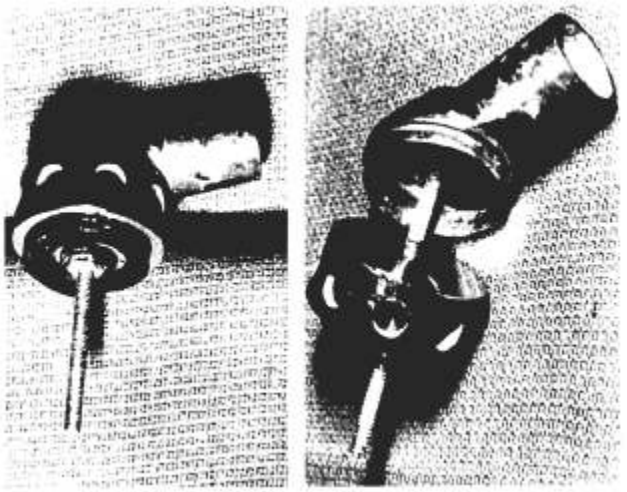

FIG. 1. Exhaust connector assembled correctly (*left*) and incorrectly (*right*).

to an exhaust connector,* which is part of a nonrecirculating ventilating system mounted on the operating room wall. This exhaust connector consists of two metal parts transfixed by a threaded bolt (fig. 1), by which it is attached to the wall-mounted extraction grille. When correctly assembled, exhaust anesthesia gases are free to spill out through the lateral holes of the system and be taken away in the air conditioning ducts. The particular unit in question was installed incorrectly, with the metal component which has the lateral holes being assembled in reverse position, resulting in total obstruction. The unit is supplied preassembled by the manufacturer; just where the misassembly occurred is not known. The defect was immediately discovered when the reservoir bag in the anesthesia circuit became increasingly tense during the first few breaths of an anesthetic. The scavenging line was disconnected from the pop-off valve, which immediately removed the risk of pulmonary barotrauma. An immediate search for the cause of high resistance in the scavenging line indicated that the problem was in the exhaust connector.

A safety interface, located distal to the popoff valve and designed to prevent positive or subatmospheric pressure in the anesthesia circuit, is essential to prevent accidents due to mechanical obstruction of scavenging lines. The exhaust connector, the source of the obstruction reported above, is widely used in North America, and attention is hereby drawn to a potentially lethal situation that may arise by incorrectly assembling the parts.

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* Exhaust connector 219-1384-800 for nonrecirculating ventilating system, Ohio Medical Products, Madison, Wisconsin.

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Anesthesiology
51:366–368, 1979

Hypotension with Anesthesia in Disulfiram-treated Patients

To the Editor:—Over the past year, we have observed four cases of acute, serious hypotension (greater than 50 per cent decrease from baseline blood pressure) following tracheal intubation (2) or skin incision (2) in chronically alcoholic men receiving long-term (a year or more) disulfiram (Antabuse®) therapy. The patients ranged in age from 25 to 65 years, were all undergoing nonthoracic elective procedures, and manifested no other major systemic disease. In all cases, the observed hypotension was acute, followed stable periods of normotension, and closely followed stimulating events during general anesthesia, such as laryngoscopy, tracheal intubation, or incision of the skin. The anesthetic techniques in all cases included induction with thiopental, 3 mg/kg, succinylcholine, 1.0–1.5 mg/kg, for endotracheal intubation, and maintenance with either halothane (1) or enflurane (3) and nitrous oxide, 40–50 per cent, in oxygen. In all cases, the hypotension responded to discontinuance of the inhalational anesthesia and vigorous fluid therapy alone (1) or in combination with phenylephrine (2) or ephedrine (1).

Because our observations could not be completely explained by anesthetic overdose with or without some

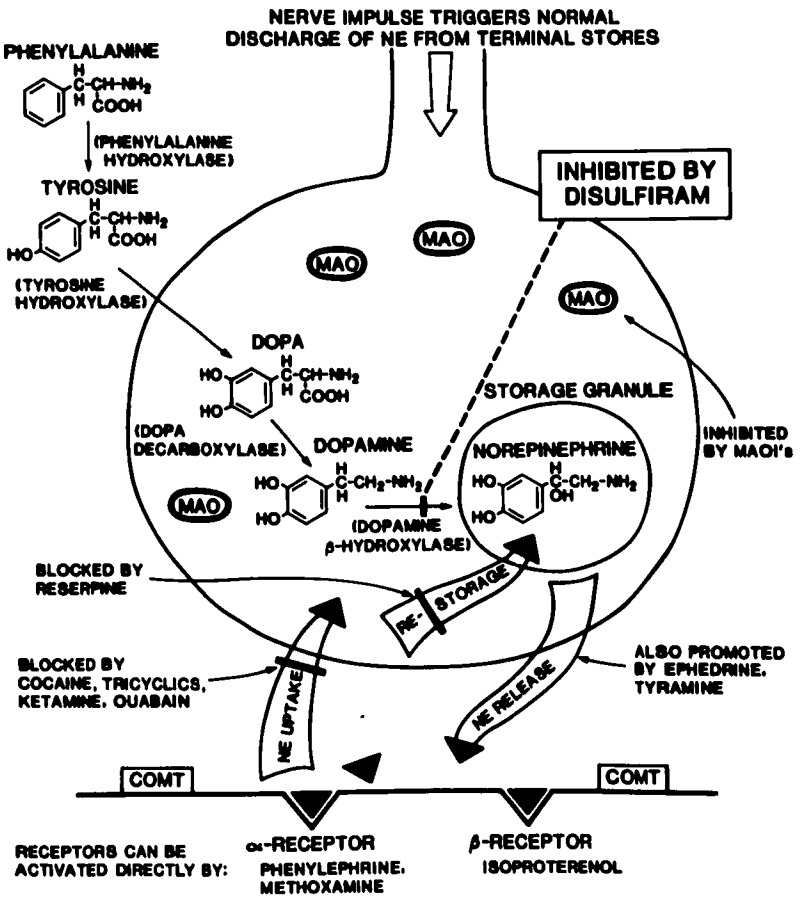

FIG. 1. Schematic diagram of an adrenergic nerve terminal depicting norepinephrine (NE) synthesis, storage in mobile granules, release by nerve action potentials or drug displacement, distribution to postsynaptic receptors, and metabolic disposition[4] via 1) neuronal reuptake and restorage (53 per cent), 2) extraneuronal enzymatic inactivation by catechol-o-methyl transferase (COMT) (40 per cent), or 3) intraneuronal enzymatic deamination by mitochondrial monoamine oxidase (MAO) (7 per cent).[5] The site of inhibition of dopamine β-hydroxylase by disulfiram is shown. MAOI = monoamine oxidase inhibitor.

fasting dehydration, we reviewed the pharmacodynamics of disulfiram, the common denominator in all cases (fig. 1). Disulfiram inhibits the enzyme aldehyde dehydrogenase, which in turn precipitates an acute acetaldehyde syndrome when ethanol in any form is ingested.[1] Another less well-known pharmacologic action of disulfiram is its inhibition of dopamine β-hydroxylase, with a subsequent dose-dependent decrease in norepinephrine synthesis and storage in adrenergic nerve terminals.[2–4] This concurrent inhibition of dopamine β-hydroxylase has been suspected of producing cardiovascular collapse during severe acetaldehyde reactions.[1]

Acute hypotension following periods of stability during anesthesia in patients receiving long-term disulfiram maintenance may be possible via depletion of the postganglionic adrenergic neurotransmitter, norepinephrine. Immediate therapy should consist of discontinuance of anesthesia with potent inhalational agents, fluid administration, and, when necessary, administration of direct-acting α sympathomimetics (*e.g.*, phenylephrine and methoxamine), which bypass adrenergic terminals to act directly on postsynaptic receptors.[4,5] One patient's hypotension did, however, respond promptly to ephedrine and fluid therapy. Since the main action of ephedrine is indirect, through release of norepinephrine,[5] we do not recommend its use for patients who may be chronically depleted of norepinephrine. We are now engaged in controlled prospective studies of urinary catecholamine metabolites in chronically alcoholic men being treated by use of psychotherapy alone or in combination with short- or long-term disulfiram maintenance. At present, we would suggest that careful monitoring of these patients and appreciation of the pharmacologic mechanisms involved will allow for rational anesthetic management without discontinuance of beneficial drug therapy.

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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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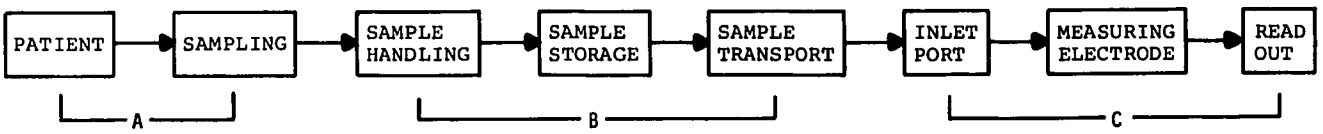

FIG. 1. Flow chart for blood-gas measurements.