Anesthesiology
54:526, 1981

Low Failure Rate for Medical Gas Line Systems in United Kingdom

To the Editor:—In the Special Article, "Hazards of Hospital Bulk Oxygen Delivery Systems",[1] the authors state on page 507 that 29 patients died as the result of gas supply errors and malfunctions in the United Kingdom between 1964 and 1973.

I think it should be made clear that this number includes only 3 cases in which death occurred as a result of a mistake in the fixed part of a medical gas pipeline system, and that all of these occurred in 1965 in the same hospital as the result of one mistake. In the paper cited by the authors[2] it is, in fact, emphasized that the majority of the 29 cases occurred as the result of mistakes in the use of anesthetic apparatus in the operating room.

Errors in the construction and operation of the fixed parts of medical gas pipeline systems in the United Kingdom resulting in harm to patients have, fortunately, occurred very rarely.[3] This may be true because the United Kingdom is much smaller than the United States, or because the great majority of medical gas pipeline systems are supplied by one company. It may also be the result of the comprehensive guidelines on these systems published by the British Department of Health and Social Security since 1972, which are followed closely by engineers responsible for pipeline systems. Perhaps this is a field in which centralization of experience and regulation is an advantage.

It is to be hoped that the introduction in 1978 of a British Standard for terminal units, hoses, and connectors will lead to a similar improvement and reduction in complications from the "non-fixed" components of pipeline systems.

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(Accepted for publication December 16, 1980.)

Anesthesiology
54:526–527, 1981

Another Benefit of the Subatmospheric Alarm

To the Editor:—Many current anesthesia machines are equipped with subatmospheric pressure alarms to alert the anesthesiologist to the development of a subatmospheric pressure within the breathing circuit. Such subatmospheric pressure may be caused by malfunctioning of a suction scavenging system, by a patient attempting to breathe through a blocked breathing system, and by a patient attempting to breathe through a system with an inadequate fresh gas supply. Because pulmonary edema, atelectasis, and hypoxia can quickly develop when a subatmospheric pressure exists,[1] these alarms should be valuable in avoiding such iatrogenic problems. In addition, we recently became aware of another benefit of having such a subatmospheric pressure alarm.

During the blind passage of a nasogastric tube in an anesthetized patient with a cuffed endotracheal tube in place, we attempted to verify the position of the nasogastric tube by applying suction to it. Soon after the suction had been applied, the subatmospheric pressure alarm was actuated. As a result, we discontinued the suction to the nasogastric tube and immediately thereafter observed the alarm to deactivate. Further evaluation confirmed our suspicion that the nasogastric tube had been inadvertently passed into the trachea and that the actuated alarm signaled the period during which a subatmospheric pressure existed.

During the blind passage of a nasogastric tube, the inadvertent passage of the tube into the trachea is not uncommon. This event is often heralded by the onset of an airleak around the cuff of the endotracheal tube as the nasogastric tube is passed, by the escape of gas from the end of the nasogastric tube during the posi-

tive pressure phase of the ventilatory cycle, or by a characteristic noise heard through the esophageal stethoscope when suction is applied. Because a subatmospheric pressure as great as 450 torr may be generated by wall suction, quick detection of this problem is imperative. The subatmospheric pressure alarm appears to be a useful adjunct in detecting the problem when other means have not been decisive.

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(Accepted for publication December 16, 1980.)

Anesthesiology
54:527, 1981

Evidence for *In vivo* Biotransformation of Nitrous Oxide

To the Editor:—Dr. Linde and Dr. Avram[1] pose the question: "Can mammalian systems biotransform N_2O?" I think the answer must be yes. It is known that nitrous oxide can oxidize the cobalt moiety of vitamin B_{12} to its bivalent form *in vitro*.[2] It has been clearly established that methionine synthesis is inhibited when rats or humans breathe 50 per cent nitrous oxide for several hours,[3,4] and there can be little doubt that this must be due to oxidation of B_{12} occurring *in vivo* as it also happens *in vitro*. Demonstration of the biotransformation of nitrous oxide by this mechanism is difficult. Even if all the B_{12} in the body was oxidized (and this does not appear to be the case), the quantity of nitrous oxide required would still be much less than 1 ml, and the detection of this small amount of $^{15}N_2$ formed from $^{15}N_2O$ would be virtually impossible in view of the substantial background level of $^{15}N_2$ in the atmosphere.

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(Accepted for publication December 17, 1980.)

Anesthesiology
54:527–528, 1981

Asystole after Intravenous Dantrolene Sodium in Pigs

To the Editor:—The recommended method of administering dantrolene sodium for malignant hyperthermia (MH) crisis in patients is 1 mg/kg, iv, rapidly, then continuing to 10 mg/kg if needed. Animal studies report no appreciable effect on the heart at these doses in one study[1] and cardiac depression in other studies.[2,3]

We have had two 35-kg MH positive pigs die from intravenous dantrolene administered during an MH crisis. One pig received a 10-mg/kg dose as a bolus; the other pig received a total dose of 30 mg (0.43 mg/kg) of dantrolene prior to arrest. Both pigs were in sinus rhythm (rate 130–140) prior to the dantrolene, but developed severe sinus bradycardia followed by cardiac arrest from asystole during the dantrolene administration. The first pig may have been given dantrolene in too high a concentration and too rapidly, but the second pig had an abrupt asystole while the dantrolene was being titrated and arrested after 30 mg was given. Neither pig was collapsing