tral terminal" and recording the potential between it and a chest lead (fig. 1). The "central terminal" consisting of RA, LL, and LA electrodes from the patient are connected through 5,000-ohm resistors and in turn connected to the RA terminal. The chest electrode is attached to the LA terminal of the monitor and serves as the exploring electrode. The RL lead is a ground that serves to reduce common mode interference.[4] In addition, the ground lead may be combined with one of the central terminal electrodes (see dashed line on fig. 1). This is advantageous because it reduces from five to four the number of electrodes, reducing the cost of disposable electrodes and increasing reliability while only slightly increasing susceptibility to interference. This alteration should be compatible with any three-lead EKG monitoring system.

We have used this modification in our operating room and find that the system is easily applied and satisfactory.

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(Accepted for publication February 29, 1980.)

Anesthesiology
53:180–181, 1980

Physostigmine Reversal of Diazepam

To the Editor:—Two reports dealing with the effects of physostigmine on the state of consciousness after administration of diazepam appeared in the September 1979 issue[1] and in its supplement.[2] The conclusions drawn in the two reports seem to be in contradiction.

It does not surprise me that the results are confusing. It seems that the authors have paid little attention to the fact that benzodiazepines primarily appear to facilitate the GABA-transmitter system, which has been found, so far, in the central nervous system (CNS) only. On the other hand, physostigmine is a pure cholinesterase inhibitor, poorly soluble in water, which can easily penetrate into the brain, as opposed to, for example, prostigmine or pyridostigmine. It will exclusively affect cholinergic transmission within the CNS through the increase in the concentration of acetylcholine.

Clinical signs of central cholinergic blockade vary greatly. Among others, coma, somnolence, short-term memory loss, excitation and agressiveness can be seen after administration of centrally active anticholinergic drugs or after different anesthetic agents. Enflurane and cyclopropane are well known for causing postanesthetic excitation. All of these behavioral changes were studied and described as a central anticholinergic syndrome (CAS) by Longo.[3]

We have used physostigmine in patients recovering from anesthesia for six years. It became obvious that physostigmine can cause arousal in comatose patients after the administration of diazepines.[4] Nevertheless, we observed that improvement of the latter condition was not of the same quality as in patients where anticholinergic drugs were the cause of changes in the conscious state. These observations were confirmed by others.[5,6] The most striking results were obtained in comatose patients after administration of flunitrazepam. Physostigmine caused an apparent arousal, leaving the short-term memory loss unchanged.[7]

There are many possible explanations for the positive results of physostigmine in the comatose states of benzodiazepine origin. Anticholinergic agents may have been used simultaneously; diazepines also may partly block central cholinergic synapses, or, most probably, inhibit the cholinergic system indirectly by facilitating the inhibitory GABA-transmitter system. Bearing in mind the well-known interdependence

of all central transmission systems, I favor the latter explanation. With regard to the two conflicting reports, this implies that there was a central anticholinergic syndrome in all cases where physostigmine proved to be effective. No positive effect was observed only in cases without central cholinergic block.

According to our experience based on more than two thousand cases in which physostigmine was administered, we consider a dosage of 0.04 mg/kg body weight to be optimal. It has proven to be effective and safe; moreover, we have used physostigmine as a differential diagnostic agent in comatose states of unknown origin. Prophylactic atropine administration was not necessary. Whenever an anticholinergic agent was needed (for example, because of simultaneous decurarization), methylatropine bromide or glycopyrrolate was administered, because they do not penetrate the CNS. In most cases, slow injection of physostigmine (1 mg/min) prevented nausea, which can accompany its administration. An absolute protection from nausea can be achieved by previous administration of an antiemetic.

We would like to point out that one should beware of expressions such as "aspecific central analeptic" effects of physostigmine, which are sometimes used.[6] It is necessary to realize that physostigmine possesses exclusively a cholinesterase-inhibitory action, and should thus be administered only for well-defined indications. This has been confirmed by the use of an alternative drug to physostigmine: galanthamine hydrobromide.[8] Full consideration of the pharmacology of physostigmine and careful study of the indications will lead to exclusively positive results.

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(Accepted for publication February 29, 1980.)

Errata

An error appeared in the article, "Intravenously Administered Lidocaine Prevents Intracranial Hypertension during Endotracheal Suctioning" (ANESTHESIOLOGY 52:516–518, 1980). The bar graph, figure 1, on page 517, is incorrectly labelled. The solid bars should indicate lidocaine treatment and the cross-hatched bars should indicate saline placebo treatment.

An error appeared in the article, "Chloroprocaine *vs* Bupivacaine for Lumbar Epidural Analgesia for Elective Cesarean Section" (ANESTHESIOLOGY 52:488–491, 1980). Table 1 includes a time for uterine incision to delivery (UD), which should be expressed in seconds as opposed to minutes. (There are a number of other time measurements included in the table, all of which are in minutes.)