Anesthesiology
53:179, 1980

In reply:—Dr. Kushins' interest in our suggested guidelines is commendable, but his understanding of our meaning, as well as those of the authors he cites, is not complete. A careful reading of our article does not support his statement that we "ignore the significant contributions that beta blockers can make." Nor does the reference he makes to the Koch-Weser paper, in which propranolol is mentioned only in passing in three sentences on page 213.[1] In Dr. Kushins' next sentence, he refers to the Brodsky and Bravo paper[2] as an example where "Other authors have used beta blockers in combination with either alpha antagonists or other vasodilators," whereas Brodsky and Bravo did not use beta blockers at any time in the case they described. Next, Dr. Kushins quarrels with the use of hydralazine as a substitute for clonidine and suggests that beta blockers are a useful adjunct to hydralazine. Here, we don't disagree. If we gave hydralazine to a patient and produced "unwanted reflex events" that could be rationally treated with beta blockers, we would certainly do so. The final point about potentiation of nitroprusside by beta blockers is true but trivial.

Our suggested plan for withdrawal of drugs from hypertensive patients receiving both propranolol and clonidine was meant only for the rare patient who cannot continue taking both drugs to, and including, the morning of a surgical procedure and resume oral drug intake that evening. Such patients are rare. If there is a better way to manage them than the plan we suggested, we would like to learn of it. Dr. Kushins has not presented one. However, we thank him for his interest in ours.

DAVID L. BRUCE, M.D.
Chief, Anesthesiology Service
VA Medical Center, Long Beach

Professor
Department of Anesthesiology
University of California, Irvine
Irvine, California

REFERENCES

1. Koch-Weser J: Hypertensive emergencies. N Engl J Med 290: 211–214, 1974
2. Brodsky JB, Bravo JJ: Acute postoperative clonidine withdrawal syndrome. ANESTHESIOLOGY 44:519–520, 1976

(Accepted for publication February 29, 1980.)

Anesthesiology
53:179–180, 1980

Precordial Electrocardiographic Lead (V_5) Capability by Simple Modification of EKG Monitor Leads

To the Editor:—Recently, monitoring of the V_5 electrocardiographic lead has been advocated for patients with coronary-artery disease undergoing surgical procedures.[1–3] This lead is purported to be the most sensitive for detecting electrocardiographic changes of myocardial ischemia.[1] We propose a simple modification of standard three-lead EKG monitor leads to provide a precordial (V_5) capability.[4] This inexpensive modification can be performed by anyone skilled at soldering using three 5,000-ohm resistors and short lengths of 3-mm and 8-mm heat-shrink tubing* for strain relief.

The precordial lead is obtained by creating a "cen-

* Available from Radio Shack, P.O. Box 2625, Fort Worth, Texas 76101.

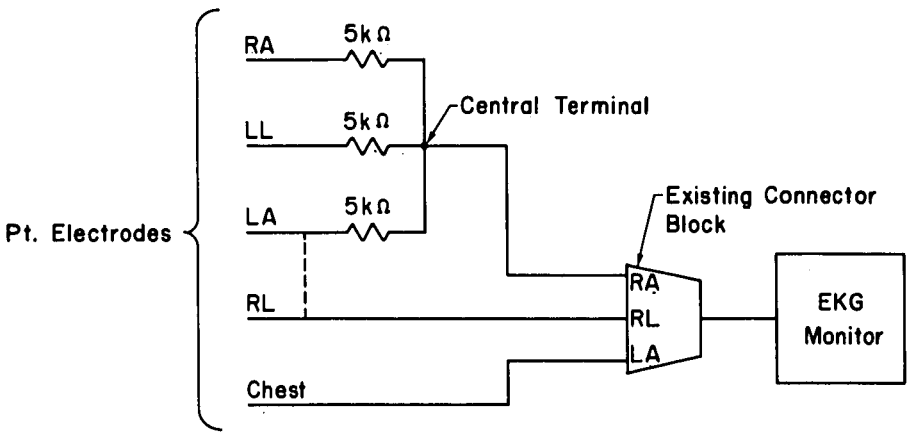

FIG. 1. Lead modification to provide a precordial electrocardiographic lead.

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.

ALAN F. NAGEL, M.D.
Resident

DUKE B. WEEKS, M.D.
Associate Professor

Department of Anesthesia
Wake Forest University
Bowman Gray School of Medicine
300 South Hawthorne Road
Winston-Salem, North Carolina 27103

REFERENCES

1. Kaplan JA: Monitoring of myocardial ischemia during anesthesia. Refresher Courses in Anesthesiology 7:155–168, 1979
2. Kaplan JA, Spincer BK: The precordial electrocardiographic lead (V_5) in patients who have coronary-artery disease. ANESTHESIOLOGY 45:570–74, 1976
3. Roy WL, Edelist G: Myocardial ischemia during non-cardiac surgical procedures in patients with coronary-artery disease. ANESTHESIOLOGY 51:393–397, 1979
4. Cromwell L, Weibell FJ: Biomedical Instrumentation and Measurements. Englewood Cliffs, N. J., Prentice-Hall, 1973, pp 88–93

(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