the wide range of activities discussed above.

Hopefully, in the future a few program directors will be anesthesiologists, but from the practical viewpoint, anesthesiologists undoubtedly will make their major contributions as specialty members of academic clinical pharmacology units. The structures of several large clinical pharmacology units have now been adapted to include clinical specialists as part-time faculty members. At Emory, for example, four departments are currently involved in the clinical pharmacology program: Medicine, Pharmacology, Pediatrics and Anesthesiology. An anesthesiologist with appropriate training would make a unique contribution in such an integrated research and educational program. For example, he would be the most appropriate person to teach the proper care of patients acutely depressed by drug overdosage. In addition, he could be the faculty member responsible for new drug research in the Anesthesiology Department, and for coordinating clinical pharmacology education in his department. Training for this role would be less extensive than that which is necessary to be a director of a clinical pharmacology unit, and would permit the anesthesiologist to continue to participate more actively in his specialty.

The extreme shortage of clinical pharmacologists in our academic institutions, the pharmaceutical industry, and the federal government was recently emphasized at a meeting on Clinical Pharmacology sponsored by the Drug Research Board of the National Academy of Science and the National Research Council. Anesthesiologists have a unique contribution to make in clincial pharmacology, and more should choose clinical pharmacology as a career. A list of training programs may be obtained by writing to Dr. Ellsworth Cook, Executive Secretary, The American Society For Pharmacology and Experimental Therapeutics, 9650 Rockville Pike, Bethesda, Maryland 20014.

LEON I. GOLDBERG, PH.D., M.D.
Department of Medicine and
Department of Pharmacology
(Clinical Pharmacology Program)
Emory University School of Medicine
Atlanta, Georgia 30322

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Drugs

ANGIOTENSIN AND MYOCARDIAL FUNCTION Although angiotensin has been available as a vasopressor for several years, its clinical use has been limited, partly due to the controversial evidence regarding its clinical pharmacology, but more specifically because of its effect on myocardial function. The myocardial effect of angiotensin was studied in anesthetized dogs. Arterial, left ventricular, and coronary sinus catheters were introduced percutaneously without a thoracotomy and left ventricular performance was studied before and during an intravenous infusion of angiotensin. The findings were: myocardial contractility was reduced by 24 per cent, while heart rate, stroke volume, left ventricular coronary blood flow and left ventricular oxygen consumption did not change significantly; left ventricular end-diastolic and end-systolic volumes increased; stroke work did not increase uniformly; the stroke work-to-fiber length ratio declined. The authors concluded that: 1) angiotensin has a negative inotropic effect on the intact myocardium; 2) the unchanged myocardial oxygen consumption is due to a balance between declining contractility and increasing wall tension; and 3) the therapeutic use of angiotensin may be dangerous when contractility is already compromised. (*Frank, M. K., et al.: The Effect of Angiotensin on Myocardial Function, Amer. J. Physiol. 218: 1267–1272, 1970.*)