

and ACTH are concentrated in the lung tissue following intravenous injection, and the possibility that the lung may play a part in the regulation of the circulatory level of such hormones has been entertained.

However, most of the work reviewed by Gillis concerns the role of the lung in regulating the plasma concentration of the vasoactive agents²⁻⁶ 5-hydroxytryptamine (serotonin), bradykinin, angiotensin, and the prostaglandins. The evidence of the modifying effect of passage through the lung is convincing and its specificity remarkable. The hypothesis proposed is appealing and gives a nice "sense of order" to an otherwise very confusing field.

Of course there are problems, both the technical ones of identifying and determining differences in concentration of the minute quantities involved, as well as those of assessing the physiologic significance of these phenomena. A primary consideration has to be the difficulty of defining the importance of 5-hydroxytryptamine, kinins, and the prostaglandins in homeostasis and defense reactions. They are found in many tissues and have been implicated individually or in concert as regulators of normal tissue blood flow and of fluid and electrolyte balance, instigators of thrombosis, inflammatory and antigen-antibody reactions, and modulators of cerebral nervous system activity and endocrine effects as second messengers comparable to cyclic adenosine monophosphate.

While there is considerable confusion about the relative influence of each agent, there is little doubt that they are involved in generalized responses such as hemorrhagic, septic, and traumatic shock, and transplantation reactions. The paper by Gillis is the first to bring to the attention of anesthesiologists the possibility that the lung is a critically important part of the system regulating the level of circulating vasoactive substances. The concept is thought-provoking and exciting, and it is tempting to hypothesize pathophysiologic consequences of an interaction with anesthetic drugs and techniques. Future work along the line suggested by Gillis will be awaited with great interest.

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References

1. Heinemann HO, Fishman AP: Nonrespiratory functions of mammalian lungs. *Physiol Rev* 49:1-48, 1969
2. Axelsson J: Catecholamine functions. *Ann Rev Pharmacol* 33:1-29, 1971
3. Garahini S, Valzelli L: Serotonin. New York, American Elsevier, 1965
4. Schachter M: Kallikreins and kinins. *Physiol Rev* 49:509-547, 1969
5. Haber E: Recent developments in pathophysiologic studies of the renin-angiotensin system. *N Engl J Med* 280:148-155, 1969
6. Weeks JR: Prostaglandins. *Ann Rev Pharmacol* 12:317-336, 1972

Obstetrics

FETAL ELECTROCARDIOGRAPHY Intrapartum fetal ECG monitoring was performed on 1,150 occasions with a transcervical intrauterine catheter. Endometritis was rare, and postpartum febrile morbidity could not be attributed directly to the techniques used. Three asymptomatic uterine perforations were encountered. Two of these were found incidentally at time of cesarean section; an additional suspected perforation was confirmed radiologically; but none was associated with additional complications. Fetal scalp abscesses or other major complications due to application of the electrodes were not seen. (Chan, W. H., Paul, R. H., and Toews, J.: *Intrapartum Fetal Monitoring—Maternal and Fetal Morbidity and Perinatal Mortality. Obstet. Gynecol.* 41: 7-13, 1973.)