graduates of medical schools outside the United States or Canada remains just a little more than 50 per cent, about the same as in the past several years.

As of this date, 5,688 certificates have been awarded to physicians, and of these Diplomates, 5,444 are living. Approximately 2,460 candidates are in some state of the examination process.

### Future Examinations

A record number of 832 candidates participated in the written examination given on July 14, 1972. The next scheduled written examination will be offered at a number of locations in the United States and, by special arrangement, overseas, on July 13, 1973. Oral examinations are scheduled for October 1972 in Houston, Texas, and for April 1973, in Tampa, Florida.

Robert T. Patrick, M.D.
*Secretary-Treasurer*
*The American Board of Anesthesiology, Inc.*
*100 Constitution Plaza*
*Hartford, Connecticut 06103*

# *Literature Briefs*

*Literature Briefs were submitted by Drs. Leonard Brand, Lee H. Cooperman, L. H. Cronau, B. C. Dalton, B. B. Das, M. I. Gold, P. Hallowell, E. Lowenstein, H. Rackow, E. Salanitre, and M. J. Strong. Briefs appearing elsewhere in this issue are part of this column.*

## Circulation

**HEMODYNAMICS OF UREMIC ANEMIA** Forty uremic patients on a program of maintenance hemodialysis had cardiac output, intra-arterial blood pressure, and heart rate measured, and systemic vascular resistance, cardiac index, and stroke index calculated. Important findings were that systemic vascular resistance and stroke index were normal in both normotensive and hypertensive patients. When hematocrit was increased without changing blood volume, cardiac index decreased and arterial blood pressure and systemic vascular resistance increased linearly. As hematocrit decreased, these changes reverted to the previous values. The authors conclude that the basic cause of hypertension in chronic renal disease is an inappropriately increased peripheral vascular resistance; that the high-cardiac-output state in uremia is predominately due to anemia, and that this may actually protect patients from the effects of otherwise-devastating hypertension. (*Neff, M. S., and others: Hemodynamics of Uremic Anemia, Circulation 43: 876–883, 1971.*) Abstracter's comment: These are excellent data. The authors believe that the increase in resistance with an increase in hematocrit is related to the fact that severe anemia is associated with inadequate oxygen delivery to the tissues, producing peripheral vasodilation, which is abolished by increasing hematocrit. That acute normovolemic anemia normally produces a decrease in calculated SVR, and that the finding of a normal SVR in the face of anemia is markedly abnormal, receive no comment. The effects of blood viscosity over a large hematocrit range are also ignored. Therefore, the interpretation of the excellent data appears inadequate.