Table 1. Effect of Œstrous Cycle on Uterine Blood Flow

	% injected ^{42}K*	Mean uterine weight	% injected ^{42}K/g
Dicestrus (12)	0·31 ± 0·05 (*S.E.*)	172	1·8 ± 0·23
Procestrus (6)	0·30 ± 0·07	202	1·5 ± 0·27
Œstrus (6)	0·25 ± 0·05	253	1·0 ± 0·15†
Metacestrus (6)	0·22 ± 0·06	158	1·4 ± 0·24

* Per cent injected potassium-42 is taken to represent per cent of cardiac output delivered to the uterus.
† $P < 0·02$ compared to dicestrus.

must be concluded that the mechanism for binding tritiated-epinephrine is altered during the œstrous cycle.

Investigations with oophorectomized rats have indicated that the atrophic uterus receives 0·045 per cent of the cardiac output[4]. Administration of œstrogen resulted in a 3-fold increase in this fraction. In these experiments flow of blood through the ovarian artery, which is responsible for a portion of the uterine blood flow, had been cut off. The value obtained for the fraction of the cardiac output delivered to the uterus 4 h after œstrogen administration, 0·2 per cent, is compatible with that reported here. The changes noted with œstrogen administration were in a situation of almost total œstrogen deprivation and uterine atrophy. It is evident that the changes produced in delivery of blood to the uterus by cyclic fluctuations in œstrogen-levels are not nearly as marked as those observed after œstrogen administration to oophorectomized rats.

Irwin J. Kopin
Richard J. Wurtman

Laboratory of Clinical Science,
National Institute of Mental Health,
National Institutes of Health,
Bethesda, Maryland.

[1] Wurtman, R. J., Chu, E., and Axelrod, J., *Nature*, **198**, 547 (1963).
[2] Sapirstein, L. A., *Amer. J. Physiol.*, **193**, 161 (1958).
[3] Papanicolaou, G. N., *Atlas of Exfoliative Cytology* (Harvard Univ. Press, Camb., Mass., 1954).
[4] Kalman, S. M., *J. Pharmacol. Exp. Therap.*, **124**, 179 (1958).

Fœtal Myoglobin in the Urine of an Adult

A fœtal form of myoglobin has been shown in newborn animals[1] and human infants[2], and it has a different electrophoretic mobility from myoglobin in adults. This communication describes the presence of fœtal myoglobin in the urine of an adult with idiopathic myoglobinuria.

The patient was a 28-year-old female with a long history of muscle pain associated with the passage of dark urine. The diagnosis of myoglobinuria was established during an acute attack, although it was noted that her urinary myoglobin precipitated in 80 per cent saturated ammonium sulphate. Since fœtal myoglobin is not as soluble in ammonium sulphate as the adult protein[1], the presence of a fœtal form of myoglobin was suspected.

Fœtal myoglobin was prepared from psoas muscles of stillborn infants, and adult myoglobin was obtained from the psoas muscle of an adult who died of a non-myopathic disease. The muscle tissues were homogenized in the cold with distilled water and the cellular detritus centrifuged out. The two respective muscle liquors and the patient's urine were purified simultaneously by column chromatography, utilizing carboxymethylcellulose according to the method of Åkeson and Theorell[3]. Following elution from the column with phosphate buffer (*p*H 6·9), the myoglobins were reduced to the met-forms with potassium ferricyanide and then dialysed against distilled water. Electrophoresis was performed on cellulose acetate at 120 mV with barbital buffer (*p*H 8·6, ionic strength 0·1 M) for 110 min. Concurrently, samples of the patient's serum and hæmoglobin were subjected to electrophoresis. The resulting separations were stained with benzidine for iron, as well as with nigrosin for protein.

The results (Fig. 1) demonstrate the presence of a urinary iron-containing protein in the same electrophoretic region as purified fœtal myoglobin. In addition, the patient's urine had an iron-staining protein corresponding

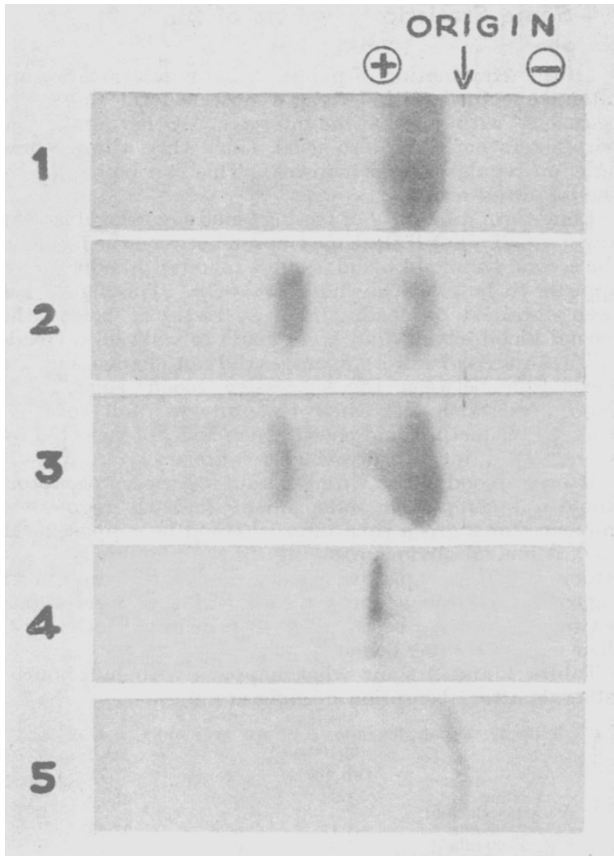

Fig. 1. Electrophoresis on cellulose acetate with barbital buffer (*p*H 8·6; 0·1 M ionic strength) and stained with benzidine. 1, Adult myoglobin; 2, fœtal myoglobin; 3, patient's urinary myoglobin; 4, patient's hæmoglobin; 5, patient's serum

in mobility to the adult myoglobin. Comparison of the stained areas by densitometry, to ascertain relative content of the two urinary proteins, showed 43 per cent fœtal and 57 per cent adult form. The serum and hæmoglobin patterns did not show iron-containing proteins in the same areas as either fœtal or adult myoglobin. The strips stained with nigrosin for protein gave the same patterns, except in the instance of the serum sample, which showed a normal pattern.

These findings indicate that during an attack of myoglobinuria this patient excreted a urinary protein which was about one-half fœtal and one-half adult myoglobin. Although muscle biopsy was not performed, it is presumed that this abnormal myoglobin originated in the patient's muscles, and that it represented persistence of the fœtal protein into adulthood. Further, it is speculated that this molecular defect may be responsible for the myoglobinuria.

This work was supported in part by Contract *Nonr*-222(51) (*NR* 105 156) between the Office of Naval Research, Department of the Navy, Washington, D.C., and the University of California School of Medicine, San Francisco. The views expressed are the private ones of the writers and are not to be construed as official or as necessarily reflecting the views of the Medical Department of the Navy or of the Naval Service at large.

F. L. Benoit
G. B. Theil
R. H. Watten

Clinical Investigation Center,
U.S. Naval Hospital,
Oakland 14, California.

[1] Jonxis, J. H. P., and Wadman, S. K., *Nature*, **169**, 884 (1952).
[2] Singer, K., Angelopoulos, B., and Ramot, B., *Blood*, **10**, 987 (1955).
[3] Åkeson, A., and Theorell, H., *Arch. Biochem.*, **91**, 319 (1960).