nometer cuff inflated to a pressure of 180 mm Hg and the subject instructed to make strong gripping movements of the hand. Ischemic pain was thereby induced. At the end of 9 min, the cuff was deflated, and the patient was permitted to void by the 10th min. Ten minutes later, a blood sample was drawn. A 3rd sample of blood was taken when the rate of urine excretion again exceeded 10 ml/min.

Figure 1 is illustrative of 2 experiments. In accord with Kelsall's observations, the production of ischemic pain of the arm results in a marked inhibition of diuresis within 10 min after the cessation of pain. The duration of the antidiuresis varies from individual to individual, as exemplified by the data depicted in Fig. 1.

The blood plasma was assayed for its antidiuretic activity and expressed in terms of equivalents of Pitressin/100 ml of plasma (*3*). Minimal quantities of ADS were found in the plasma prior to the production of pain. A significant increase in the antidiuretic activity of the plasma was noted by the time maximal inhibition of water diuresis occurred. The sample drawn after the restitution of the diuresis showed a return of the plasma ADS to its pretest concentration.

This study reveals that the antidiuretic response to a noxious stimulus such as ischemic pain of the arm is associated with an increase in the antidiuretic activity of the blood. The increase may be due to the secretion of the antidiuretic hormone of the neurohypophysis or to the release of a similar ADS from some other site. The probability that the hypothalamus is the site of origin of the ADS will be considered in a more comprehensive report.

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Comments and Communications

Histamine in Mast Cells

THE contention of James F. Riley (*1*) that mast cells function not only as "heparinocytes" but also as "histaminocytes" is supported by the observation that the histamine content of the blood is well correlated with the number of circulating mast cells (basophiles), but not with that of eosinophiles or neutrophiles. Table 1 shows this correlation in the blood of various species, Table 2 in the blood of leukemic patients. These observations are in keeping with the view that blood and tissue mast cells are functionally the same.

TABLE 1*

Species	Histamine γ %	Basophiles/ mm³	Eosinophiles/ mm³	Neutrophiles/ mm³
Cat	2–5	20	850	9500
Man	2–8	45	240	4000
Guinea pig	6–25	70	180	4100
Rabbit	100–500	450	400	4200

* Histamine values according to Code (*2*), leucocyte values according to Albritton (*3*). The disproportionally high histamine value of rabbits' blood is due to its high concentration in the platelets of this species (*4*).

TABLE 2*

Histamine γ %	Basophiles/ mm³	Neutrophiles/ mm³
132	3280	154.980
250	4987	187.508
375	4425	162.070
800	7040	112.640

* Calculated from data published by Valentine and Lawrence (*5*).

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On [the Interpretation of] the Use of Calomel Half Cells to Measure Donnan Potentials

IN a recent note under (substantially) the above title, Babcock and Overstreet (*1*) discuss the interpretation of potentials obtained by inserting salt bridges into a Donnan system. They conclude that "even" if one assumes equal transference of K^+ and Cl^- ions at the bridges, the potential should be zero. Since recently a correct and very satisfying discussion of this problem has been given by Overbeek (*2*) and the related problem of ion exchange membranes treated rigorously by Scatchard (*3*), I would like only to point out some fallacies in the reasoning of this note (*1*).

The assumption of equal transference of K^+ and Cl^- ions is not sufficient to prove that the potential of the cell is zero. The authors have explicitly assumed