TABLE 1

PREDICTED AND ACTUAL DIAGNOSIS OF BLUE COMB IN 1952*

Predicted	Amherst, Mass.	Waltham	Conn.	N. H.	R. I.
Jun 6–12		4, 10, 13	3		
15–20	20, 23, 23	17, 23	23	20	
28–30	30	25, 25, 26, 30		30	
Jul 3–7	8, 9	3, 7, 8, 10, 10			
13–20	14	14, 14, 14, 15		15, 15, 16	
24–30	29, 30	30		17, 25, 28	
Aug 6	7, 8	6, 8, 11			
12–19	19		12, 15, 21, 21	13	23
Sep 3	2	3, 3, 4, 5	2		
11–14	9, 12	16			11
20	24, 26, 26	22, 26	19, 22, 23		
29	29				
Oct 7	1, 10	2			
16	22	14	16, 20		
29		28, 30			

* Dates in first column represent days when diagnoses were predicted. Dates in other columns represent outbreaks diagnosed.

accompany leukosis or Newcastle disease, or heavy egg production. The pathologist might separate the acute and the subacute phase, or use a different nomenclature.

The result of the survey was promising, as the five laboratories received 71 cases or in within 2 days where temperature had been 78° F or higher. Of 31 other cases, 6 occurred during January to April, and 9 from October to December. Prediction accuracy was not good in 1951, as it was thought that fowl might become acclimatized to hot weather in July and August.

In 1952 a similar prediction was made. Prediction accuracy was better during the hot months, but the influence of cold weather stress needs further review. The results of the prediction for June to October (Table 1) serve to encourage the further study of climatic influence on the development of blue comb in poultry.

Manuscript received July 6, 1953.

The Effect of a Noxious Stimulus in Man on the Antidiuretic Activity of the Blood

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In both man and dog, exposure to a noxious stimulus results in a marked inhibition of the diuresis induced by the ingestion of a water load (*1*). This antidiuretic response to a noxious stimulus is diminished in the absence of the neurohypophysis (*2*). Consequently, it has been postulated that the antidiuretic response is dependent upon the secretion of an antidiuretic hormone by the neurohypophysis. Direct evidence of such secretion into the circulation is not available.

With the development of a relatively simple, sensitive, and precise procedure for the assay of antidiuretic substances (ADS) in the blood plasma (*3*) it became possible to determine directly the response of the blood ADS to a noxious stimulus.

Kelsall has demonstrated that a short period of ischemic pain in the arm may inhibit the diuresis produced by a water load in man (*4*). Accordingly, healthy men were given 1000 ml of tap water and the urine voided every 10 min was replaced with an equal volume of water in order to maintain a constant water load. When the rate of urine excretion exceeded a volume of 10 ml/min for 2 consecutive periods, a venous blood sample was drawn. Shortly thereafter, the circulation through the right arm was occluded by a sphygmoma-

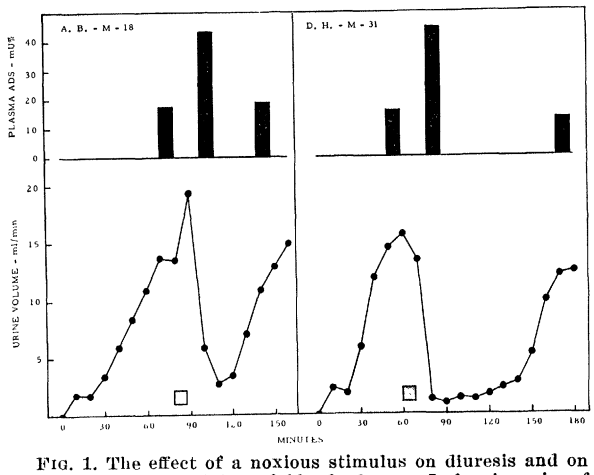

FIG. 1. The effect of a noxious stimulus on diuresis and on the antidiuretic activity of blood plasma. Ischemic pain of an arm was produced during the period marked by the shaded area.

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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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Manuscript received June 19, 1953.

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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Received September 23, 1953.

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