No change in bleeding and clotting time could be found in humans 10 days after daily administration of the stated iproniazid doses. In all the five humans receiving an iproniazid dose of 5–8 mgm./kgm., a rise in the number of platelets was seen. The absolute and relative number, as well as the morphology of the other blood cells, was not altered by the drug.

There is a possible connexion between the increase in the number and the 5-hydroxytryptamine content of platelets. It has been shown that 5-hydroxytryptamine administered by itself causes a rise of the platelet number also[11,12]. The mechanism which leads to this increase is not yet known.

It is conceivable that certain pharmacological actions of iproniazid are mediated by the increased 5-hydroxytryptamine in platelets. This might be the case, for example, for certain effects of iproniazid on the heart, since the cardiovascular system is relatively sensitive to 5-hydroxytryptamine[13].

A. Pletscher
A. Bernstein

Medical Research Department of
F. Hoffmann-La Roche and Co., Ltd.,
Basle, and the
Department of Internal Medicine,
University of Basle.
Jan. 17.

[1] Udenfriend, S., Weissbach, H., and Bogdanski, D. F., *Ann. N.Y. Acad. Sci.*, **66**, 602 (1957).
[2] Pletscher, A., *Helv. physiol. Acta*, **14**, C76 (1956); *Experientia*, **12**, 479 (1956); *Schweiz. med. Wschr.*, **87**, 1532 (1957).
[3] Pletscher, A., *Experientia* (in the press).
[4] Zeller, E. A., Barsky, J., Fouts, J. R., Kirchheimer, W. F., and van Orden, L. S., *Experientia*, **8**, 349 (1952).
[5] Zeller, E. A., Barsky, J., Berman, E. R., and Fouts, J. R., *J. Lab. Clin. Med.*, **40**, 965 (1952).
[6] Zeller, E. A., and Barsky, J., *Proc. Soc. Exp. Biol.*, **81**, 459 (1952).
[7] Schayer, R. W., *Proc. Soc. Exp. Biol.*, **84**, 60 (1953).
[8] Viollier, G., Quiring, E., and Staub, H., *Helv. Chim. Acta*, **36**, 724 (1953).
[9] Pletscher, A., *Experientia*, **12**, 479 (1956).
[10] Udenfriend, S., Weissbach, H., and Clark, C. T., *J. Biol. Chem.*, **215**, 447 (1955).
[11] Steiner, F. A., and Hedinger, C., *Experientia*, **12**, 109 (1956).
[12] Steiner, F. A., *et al.*, *Experientia* (in the press).
[13] Shore, P. A., Symposium on Iproniazid, New York, Nov. 1957, discussion remark.

Two Inhibitory Fibres forming Synapses with a Single Cell

Drs. A. S. V. Burgen and S. W. Kuffler claim[1] that the dual inhibitory regulation of one cell by two neurons with distinctly different physiological performance which they found in the stretch receptor of the lobster (presumably *Homarus vulgaris* ?) is a phenomenon which has not been found elsewhere.

It has also been found in the limbs of decapod crustaceans. The opener and stretcher muscles are innervated by branches of the same single motor axon and also by the same 'common' inhibitor axon. In order that the two muscles may function independently they also receive in some species 'specific' or 'true' inhibitor axons. Thus the muscle fibres receive a double inhibitory nerve supply. Wiersma[2] showed that there are distinct physiological differences between the two (see also refs. 3 and 4). The ratios of frequency of inhibitory to that of excitatory stimulation for just complete inhibition are different in the two cases. Also, the specific inhibitor in crabs can give supplemented (α-inhibition) whereas the common one cannot. Hoyle and Wiersma[5] have studied these phenomena with the aid of intracellular recording electrodes.

G. Hoyle

Department of Zoology,
The University,
Glasgow. Jan. 3.

[1] *Nature*, **180**, 1490 (1957).
[2] Wiersma, C. A. G., Biol. Symp., **3**, 159 (1941).
[3] Wiersma, C. A. G., and Helfer, R. G., *Physiol. Zool.*, **14**, 296 (1941).
[4] Wiersma, C. A. G., and Ellis, C. H., *J. Exp. Biol.*, **18**, 223 (1942).
[5] Hoyle, G., and Wiersma, C. A. G. (in preparation).

Erythropoiesis in Nephrectomized Dogs

The striking disappearance of erythroblasts from the bone marrow of bilaterally nephrectomized dogs, kept alive by peritoneal dialysis, has been reported[1]. Erythroblastopænia is likewise observed in patients with acute renal failure, but does not occur in cases of chronic uræmia, in spite of similar non-protein nitrogen blood-levels in the two situations. These observations suggest that the kidney produces a specific erythropoietic factor. The present study with iron-59 demonstrates the abolition of erythropoiesis following bilateral nephrectomy.

Seven bilaterally nephrectomized and seven intact dogs were studied, Unilateral nephrectomy was performed initially, the remaining kidney being removed 15–30 days later.

Dogs were kept alive by two or three daily peritoneal washings, according to Grollman's procedure[2]; 72 hr. following the second nephrectomy, 5 μc. of iron-59, previously incubated in 20 ml. of plasma, were given intravenously. Blood samples were collected every 45 min. during the first three hours and afterwards every two days.

The curves of disappearance of iron-59 from the plasma of the normal dogs and of the nephrectomized ones are shown in Fig. 1. The average time at which half the activity initially present had disappeared ($T1/2$) in the nephrectomized dogs was 279 min., as compared with 89 min. in normal dogs.

The plasma iron turnover-rate was also decreased in the nephrectomized group: 0·09–0·49 mgm./kgm./day (mean, 0·27) against 0·25–0·92 (mean, 0·64) in the control group.

In the control group, incorporation of iron-59 into erythrocytes was between 60 and 80 per cent of the injected dose and was maximal between the third and the eighth day. Incorporation was definitely

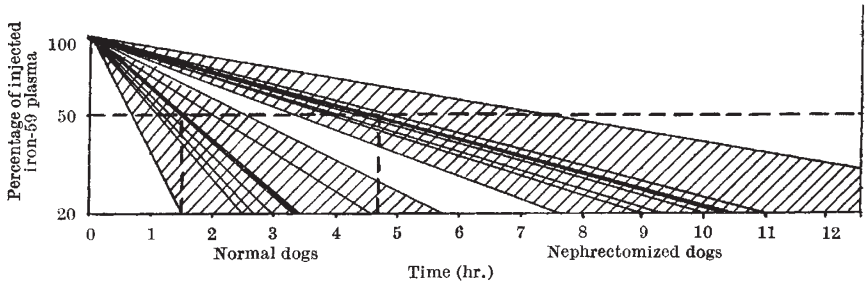

Fig. 1. Disappearance of iron-59 from plasma in normal and nephrectomized dogs. ——, Mean in each case. (The points are plotted on semi-logarithmic paper.)