$R.CO.NH_2$ $R.C(OH)=NH$

I II

clature used by Baeyer for the parallel case of isatin and its homologues.

It is clear that the term 'imine' should be confined to substances in which the $>C=N-$ is present, and that 'imide' should be reserved for derivatives of the enolic form of the amide structure and for the cyclic amides of dibasic acids. Substances of the structure R_1CONHR_2 should be termed 'substituted amides'. The term 'polyamide' for nylon is, therefore, correct.

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[1] *Nature*, **154**, 486 (1944).
[2] Wurtz, *Jahresb.*, 566 (1854).

IN reply to the above communications, I wish to say that the main point in my complaint is that in two closely allied fields of chemistry the connecting link –CONH– is being called by two different names, namely, 'amide' and 'peptide', and that the expression 'amide (or amido) group' is being used to cover both the connecting –CONH– and the terminal group $–CONH_2$, both of which are present in proteins.

For example, in a recent paper by P. J. Flory[1] dealing with three-dimensional polymers and the theory of gelation, in the section on protein gels he refers to "amide-amide hydrogen bonds" and quotes a paper by Myers and France. Reference to the latter paper[2] shows that these workers talk of hydrogen bond formation (with acetic acid) at "the loose ends of the salt-bridges after neutralisation has permitted them to separate", and of the "possibilities of hydrogen bond formation at each peptide link". They do not refer to 'amides' at all. When Dr. Astbury talks of an 'amide-hydroxyl' hydrogen bond in keratin, he means a side-chain link between the group $–CONH_2$ and OH.

Some of the correspondents also appreciate the difficulty. I am not an authority on organic nomenclature, and am willing to accept any ruling which would lead to clarity and be acceptable to the Chemical Society. As regards the past history of polypeptides and synthetic polyamides, this has not escaped my attention, but it does not of itself suggest how to deal with the future.

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[1] *J. Phys. Chem.*, **46**, 132 (1942).
[2] *J. Phys. Chem.*, **44**, 1113 (1940).

Endocrine Reaction to Tissue Injury

IT has been reported previously that tissue damage produces in animals a state of resistance to the lethal effects of a subsequent trauma[1,2]. Resistance, as detected by the decrease in post-traumatic mortality, was accompanied by the following functional changes: inhibition of the normal release of histamine from blood cells[3]; shortening of the bleeding time; and increase of the capillary resistance[4]. All changes could be transferred to normal animals by injection of the serum of traumatized animals. It was also shown that the substance present in the serum and responsible for the resistance was produced by the pituitary and acted through the adrenal cortex[4].

Further investigations have been carried out, using the shortening of bleeding-time as test. Trauma, as well as injection of serum from traumatized animals, reduces the mean bleeding-time by about 40 per cent in groups of guinea pigs and rats. In hypophysectomized, adrenalectomized or splenectomized animals, however, neither trauma nor the injection of traumatic plasma produces a shortening of bleeding-time.

The part played by various tissues was further tested by studying the action of tissue extracts on bleeding-time. Of eighteen tissues investigated, only the extracts of pituitary, adrenals and spleen shortened bleeding-time. The same effect was observed with purified products from these organs: corticotrophic hormone of the pituitary and whole cortical extract (synthetic desoxycorticosterone was inactive). A spleen extract was prepared which shortened bleeding-time in a dose of 0·02 μgm. per kgm. body weight. Chemical identification of the latter is being attempted.

It has also been shown that the pituitary hormone is without effect in adrenalectomized or splenectomized animals. Adrenal extract had no action in the absence of the spleen, but spleen extract was still active in animals deprived of pituitary, adrenals or spleen.

Selye[5] observed hypertrophy of the adrenals in the 'adaptation' phase of the 'alarm reaction' which can be elicited by tissue injury. It is also known that adrenalectomized animals are particularly sensitive to 'shock' conditions[6]. The intervention of the pituitary in protection against these conditions was suggested by Reiss, Macleod and Golla[7]. Perla and Marmorston put forward the idea that the spleen might play a part in the resistance to infections[8]; but the facts mentioned above supply probably the first experimental proof of an endocrine function of the spleen.

The results of the experiments reported here point to the existence of a physiological mechanism responsible for the resistance to lethal effects of trauma. The pituitary responds to tissue damage by the secretion of corticotrophic hormone, which determines the release of an adrenal product stimulating eventually the secretion of the splenic substance. The mode of action of the latter is not yet known; some of its effects suggest either a change in the reactions of the capillary wall, reducing perhaps the escape of fluid into the tissues, or the inhibition of the release of toxic substances from certain cells.

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[5] Selye, H., *Endocrinology*, **21**, 169 (1937).
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[7] Reiss, M., Macleod, L. D., and Golla, Y. M. L., *J. Endocrin.*, **3**, 292 (1943).
[8] Perla, E., and Marmorston, J., "The Spleen and Resistance" (London: Baillière, 1935).