viewed as a narcotic (Ebbecke, *Pflüg. Arch. ges. Physiol.*, 1935, **236**, 648). In our experiments the reversal of drug narcosis was always manifested before harmful effects of pressure became apparent. Following compression at 5,000 psi, gradual recovery in tap water at normal pressure often occurred, but some of the individuals died. Narcotized animals that were not subjected to pressure always recovered after transfer to tap water.

The basic mechanism through which temperature, pressure, and various drugs act on luminescence has been considered at some length (reviewed by Johnson, *Adv. Enzymol.*, 1947, **7**, 215). Heretofore, there has been no direct evidence that the same theory, specifically with reference to the influence of hydrostatic pressure on narcosis, applies in higher organisms, although other parallels have been found with reference to the influence of temperature. These relations support the general implications of the fundamental theory, and invite further study with various aquatic animals and narcotic agents.

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Erratum

In my recent paper "Concerning the Theory of Photoconductivity in Infrared-sensitive Semiconducting Films" (*Science*, 1950, **111**) page 685, line 7, column 2, should read ". . . to essentially pictures (1) or (2). . . ." rather than ". . . pictures (*10*) or (*5*). . . ." On page 687, line 5, column, 1, the words "fixed," "positive," and "electrons" should be deleted.

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Pancreatic Changes after Injection of Intermediary Fat Metabolites

It has already been reported (Nath, M. C., and Brahmachari, H. D. *Nature*, Lond., 1944, **154**, 487; and Nath, M. C., and Brahmachari, H. D. *Nature*, Lond., 1946, **157**, 335) that intermediary fat metabolites are responsible to a great extent for the onset of diabetic symptoms. Recently it has been found (Nath, M. C., and Brahmachari, H. D. *Nature*, Lond., 1948, **161**, 18; and Nath, M. C., and Brahmachari, H. D. *Indian J. med. Res.*, 1949, **37**, 71) that the guinea pigs injected with these substances show hypersecretion of insulin in the first stage. The animals begin to lose the activity of their pancreatic insulin after treatment with intermediary fat metabolites for about two months. A stage is reached in 70 days when the potency of the pancreatic insulin comes down to half the normal value.

This hypothesis has found support from A. Lazarow, who believes the effect of injected ketone bodies might account for the increased fasting blood sugar levels observed when men are placed on a high fat diet (*Physiol. Rev.*, 1949, **29**, 48); and the increasing demand for more and more insulin which results might increase the sensitivity of beta-cells to degeneration.

This prompted us to undertake histological examination, at different stages, of pancreatic cells from animals injected with β-hydroxy butyric acid (Na salt) in gradually increasing doses as mentioned hereafter.[1] The results of preliminary observations are indicated here:

First stage—The animals (rabbits) were killed on the 27th day.

1. The area of the islets of Langerhans increases; there are relatively fewer islets in a particular microscopic field in comparison with the normal pancreas.
2. There seems to be an increase in the number of cells in the islets as shown by their being very tightly packed.
3. The nuclei of these cells are large and appear to be active, as shown by staining with hemotoxylin.
4. However, the cells of pancreatic acini do not show any deviation from the normal.

Second stage—The animals were killed on the 53rd day of the experiment.

1. Islet cells do not show the close packing present in the first stage. There is a great amount of intercellular space.
2. The nuclei of these islet cells show distinct signs of degeneration. They take less stain and are therefore less clearly defined. The islet as a whole appears dull in contrast to the deep-staining cells of the pancreatic acini.
3. In at least one islet there is an invasion of the pancreatic blood capillary.
4. A curious feature is that the acinar cells do not seem to be affected at all and are normal.

These findings confirm the hypothesis, started by two of the authors in 1944 (Nath, M. C., and Brahmachari, H. D. *Nature*, Lond., 1944, **154**, 487) that keto acids might first stimulate the pancreatic islet cells and later cause lesions after fatigue through excessive work.

Further studies on detailed investigations are in progress.

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[1] The total number of animals used in the experiment was 12, of which 2 were killed after the 1st stage and an equal number after the 2nd stage. The remaining animals were used for glucose tolerance tests and other observations. The weight range of the animals selected was between 1.8 and 2.1 kg each, and the injections were given intramuscularly in the leg muscle every day after giving food. The initial daily dose of injection was about 50 mg per kg, which was increased by 7.5 mg per kg per week.

[2] The authors are grateful to Drs. K. Krishnamurti and M. A. Moghe for their kind interest and for the facilities they offered.

The Donora Episode—A Reply to Clarence A. Mills

There are many misleading statements in Dr. Mills' note regarding the U. S. Public Health Service's Bulletin 306 on the Donora episode (*Science*, 1950, **111**, 67). Neither of us has any connection whatever with the steel mill and zinc plant in the Donora area, or with the Public Health Service, but we wish to reply to Dr. Mills.