

present in the adjacent media.

Although the "spontaneous" lesions were also found in the stilbestrol-injected birds, they differed from those found in the controls in that they contained an abundant amount of lipid material some of which was cholesterol. Moreover, degenerative changes as well as calcification had occurred in the plaques of the stilbestrol-treated birds (Fig. 2).

In none of the control birds did the level of total lipids of plasma exceed 500 mg per

100 cc. Although considerable fluctuations occurred in the lipid level of the plasma of the stilbestrol-treated birds, values for total lipids well over 10,000 mg per 100 cc were commonly found; in a single bird the concentration of total lipid rose to 17,800 mg. Neutral fat accounted for most of the rise in total lipid. In the control birds the levels of total cholesterol in plasma did not exceed 130 mg, whereas in the injected group values above 500 mg were frequently observed.

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A Transitory Decrease in Glucose Tolerance Following Experimental Lesions in the Central Nervous System.*

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In the process of determining daily postoperative blood sugars in animals with major traumatic experimental lesions in the brain stem, it was noted that an immediate postoperative hyperglycemia seldom occurred,^{1,2} but in some instances a definite hyperglycemia appeared after the onset of postoperative feeding, *i.e.*, when blood sugar was determined 18 to 24 hours after the previous meal.

It seemed apparent that this postfeeding hyperglycemia was due to the presence of a decrease in the animal's tolerance for carbohydrates, the 18- to 24-hour hyperglycemia being the direct result of an excessive prolongation of the meal tolerance curve. For the purpose of ascertaining if this were true, the glucose tolerance in dogs has been followed for extended periods after major intracranial procedures.

Methods. The intracranial procedures consisted of (1) hemisection and transection of the midbrain and pons, (2) destruction of

the ventral tuberal portion of the hypothalamus, and (3) removal of the cerebellar vermis and the cerebellum in its entirety. Operative procedures were carried out under sodium pentobarbital anesthesia (30 mg per kg of body weight), and have been described in detail elsewhere.³⁻⁵

The glucose meal consisted of 2 g per kg of body weight, administered in a 20% aqueous solution by stomach tube. Tests were run 24 hours after the previous meal, and postoperative feeding was begun the day after operation, *i.e.*, 48 hours after the last preoperative meal. Blood sugar was determined by the Gibson modification of the Folin-Wu method.⁶

Results. Elevated and prolonged tolerance curves, ranging from hardly detectable (see Dog 3-C, Fig. 1) to very striking effects (see Dogs 5 and 4-C, Fig. 1) were routinely encountered following these procedures. This

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⁴ Keller, A. D., *J. Neurophysiol.*, 1945, **8**, 275.

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GLUCOSE TOLERANCE TESTS

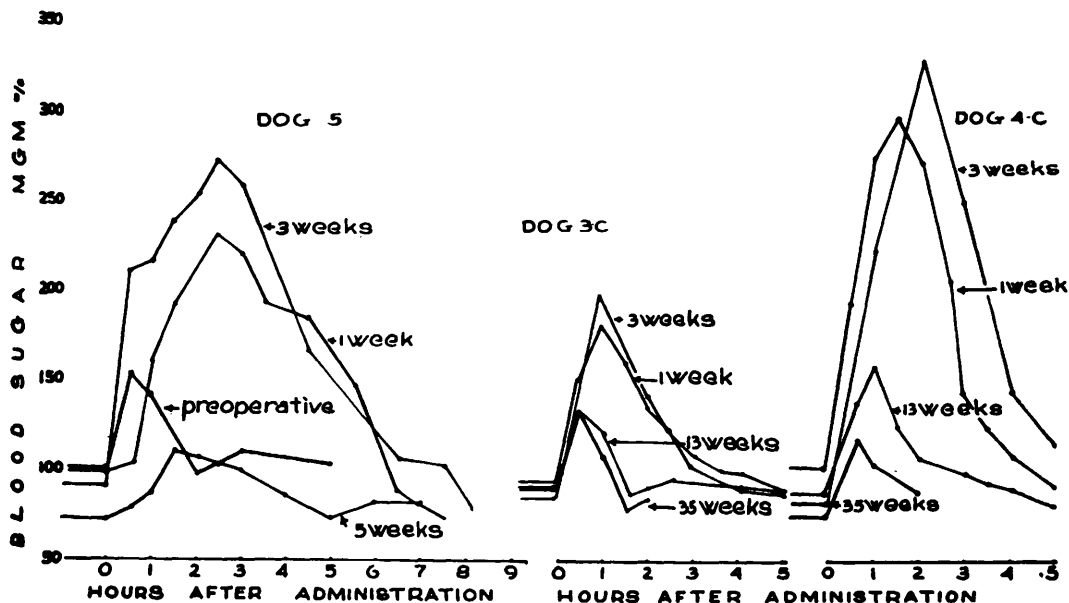


Fig. 1.

The operation in Dog 5 consisted in a double transection of the brain stem, one through the caudal extent of the diencephalon, the other through the pons. The operation in Dog 4C was a radical removal of the cerebellar vermis. The operation for Dog 3C was a radical removal of the entire cerebellum.

decrease in tolerance increased progressively for some time after operation, rapidly at first, then more slowly. After a maximum was reached, it was maintained for some time, after which it receded, ultimately returning to normal (see all animals, Fig. 1).

There was no certain indication that a lesion in any one part of the central nervous system was more prone to precipitate striking effects.

Discussion. The similarity of this phenomenon to starvation diabetes is at once apparent. The possibility that dietary factors may have enhanced the height and duration of the early postoperative tolerance curves in some instances is quite probable. However, the fact that the increased tolerance persists for a matter of weeks safely rules out dietary factors *per se* as being the primary causation variable.

The only as yet recognizable factor that was constant in all the instances where striking change in tolerance occurred was the di-

rect accessibility of the lesion debris to the ventricular cerebrospinal fluid.⁷ The close correlation between the duration of the disturbed tolerance and the time it takes for the organization of lesion debris may be significant.

The question is raised as to the possibility of the ultimate disturbance being a reversible impairment in liver function. This possibility is not too remote because drastic acute liver damage⁸ and hepatic cirrhosis⁹ have occasionally been encountered following experimental intracranial procedures.

Summary. A reversible decrease in glucose tolerance, variable in magnitude, has been encountered following major intracranial procedures in dogs.

⁷ Keller, A. D., and D'Amour, M. C., *Arch. Path.*, 1936, **21**, 185.

⁸ Keller, A. D., *Arch. Path.*, 1936, **21**, 127.

⁹ Graef, I., Negrin, J., and Page, Irvine H., *Am. J. Path.*, 1944, **20**, 823.