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Effect of Hyperpyrexia Produced by Baths upon the Intracranial Pressure in Epileptics.

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Hyperpyrexia produced by baths is now frequently utilized in many neurologic disorders.[1] Epileptics, when mouth temperatures are raised to 39.5°C. or higher, are especially liable to convulsive attacks while in the bath. The question of changes in intracranial pressure was investigated.

In attempting to get direct information on the subject, the method of Stevenson[2] *et al* for measuring changes of intracranial pressure seemed the most available. Their method consists of utilizing the cerebral hernia following subtemporal decompression. Their apparatus consists of a closed air tambour system under a positive pressure of 12 mm. of mercury. One tambour was strapped over the brain hernia. The other was attached to a pointer which registered on a smoked drum the changes in intracranial pressure.

For our purpose, when the patient was subjected to high temper-

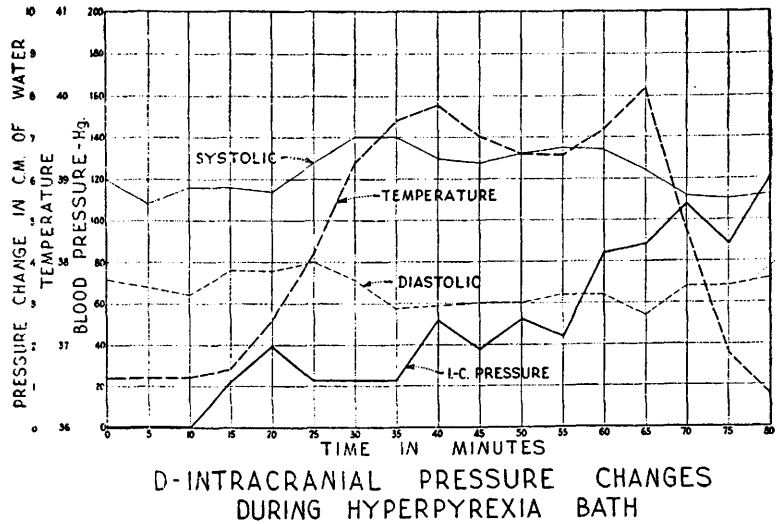

CHART A.

Effect of hyperpyrexia produced by baths upon intracranial pressure in epileptics.

[1] Mehrtens and Pouppirt, *Arch. Neurol. and Psy.*, 1929, **22**, 700.

[2] Stevenson, Lewis, Christensen, B. E., and Wortis, S. Bernard, *Am. J. Med. Sciences*, 1929, **178**, 663.

ature with consequent involuntary movements, it seemed desirable to modify the above method. The tambour, strapped over the hernia, was attached to a closed manometer system and the pressure changes read directly on the U tube of the manometer. The patient was placed in the continuous bath at 37°C., a control observation made for 20 minutes, then the hot water in the tub was raised to 41°C. until the mouth temperature of the patient was raised to 40°C. This mouth temperature was maintained about 20 minutes.

Five such observations were made on 2 epileptic patients. Chart A shows a gradual rise in intracranial pressure as the mouth temperature increased, but not accompanied by a corresponding increase in blood pressure.

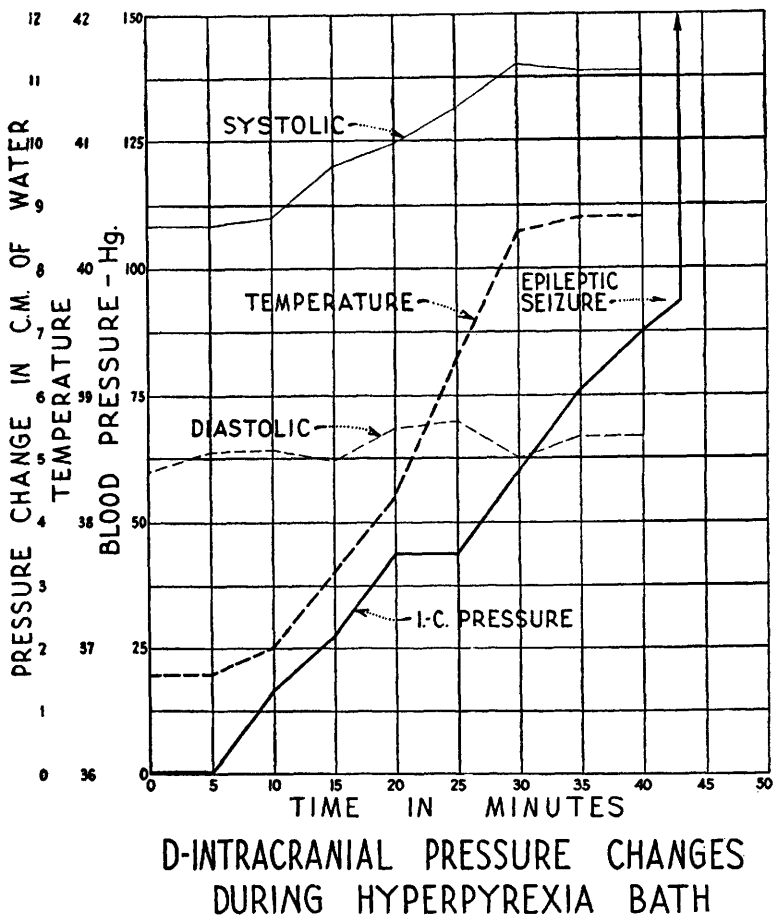

CHART B.

Effect of hyperpyrexia produced by baths upon intracranial pressure in epileptics.

Chart B shows a condition—occurring twice in one series—in which the intracranial pressure increased rapidly upon applying hyperpyrexia, and ended in an epileptic convulsion.

Conclusions. Hyperpyrexia produced by baths raises the intracranial pressure in epileptics, and perhaps in normal individuals. The hyperpyrexia frequently results in an epileptic seizure, occurring as the intracranial pressure rapidly rises. The seizure may be related to this rapidly increased intracranial pressure. Normal individuals never develop convulsions when subjected to such hyperpyrexia.

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Survival of Bacteriophage.

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The interval of time over which bacteriophage filtrates maintain their activity is of general biologic interest. Enzymes demonstrate their activities after many months. Immune substances retain their activities for at least more than 4 years. Ultramicroscopic viruses retain their activity under certain circumstances for several years. Ballantyne[1] has shown the survival of bacteria in water for at least 32 months. Anaerobic organisms have been recovered from old cultures after more than 14 years. The plague bacillus has remained viable for 10 years. Cultures of *Leptospira icteroides* and *L. icterohemorrhagiae* may show motility after 2 years and 4 months (Hadley, P.). Pure cultures of molds have yielded active spores after longer periods. The bacteriophage has been demonstrated to retain its activity for more than 5 years.

The present brief note is based on tests of a bacteriophage filtrate originally recovered by d'Herelle. It was sent to Dr. F. G. Novy in 1921, and was rejuvenated by Dr. Philip Hadley against the Shiga dysentery bacillus for which it was said to be specific. The properties were specifically studied[2] during the fall and winter of 1922-23, during which time several filtrates were sealed in tubes and stored in the dark at room temperature. The menstruum was a beef infusion broth, presumably pH 7.2 ± 0.2, used for all of the work

[1] Ballantyne, E. N., *J. Bact.*, 1930, **19**, 303.

[2] Marshall, M. S., *J. Infect. Dis.*, 1925, **37**, 126.