It cannot be held that temperature characteristics are determined by the organism as a whole. The movements of heart and of gills, in *Limulus* embryos, show different μ values in the same individual at the same time; and the critical temperatures for the two activities do not coincide. This is also true in *Asellus*. The critical increments or temperature characteristics have, therefore, a specific, local significance.

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Measurements of Fatal Doses of Chloroform in the Brains of White Rats.

WILLIAM H. COLE.

From the Biological Laboratories of Clark University.

Qualitative tests. The aqueous extracts of the brains of frogs, mice, rats, guinea pigs, cats, and dogs, killed by inhaling chloroform, have all shown the presence of chloroform by the pyridine test,[1] while similar extracts from animals killed by electricity, illuminating gas, CO_2, blow on the head, and beheading, have all been negative for chloroform. In a series of 8 brains from rats killed by inhaling chloroform, the tests were made at the end of 1, 1½, 2, 3, 5, 6, 15 and 25 days respectively. In each case chloroform was indicated, in spite of the fact that considerable decay of the tissues had occurred. These results agree with those reported by Luedeking,[2] and Angiolani,[3] to the effect that chloroform in animal tissues persists unchanged for many days after death and decay.

Quantitative tests. The chloroform present in extracts of brains from rats killed by inhaling chloroform has been measured by the pyridine colorimetric method. Into a 1-liter dish containing a rat, a known amount of chloroform was sprayed. At the moment of death (cessation of respiratory movements) the rat was removed and its brain extracted. A dose of 0.1 cc. was sub-lethal in 35 minutes; 0.2 cc. caused death in an average of 11.2 minutes; 0.4 cc. in 5.4 minutes; 0.8 cc. in 4.3 minutes; 1.0 cc. in 3.5 minutes; 2.0 cc. in 2.5 minutes; 4.0 cc. in 1.6 minutes and 5.0 cc. or more in 1.5 minutes. The time of death bore no clear relationship to size of the animal. In Table I are given the amounts of chloroform found in brains of killed and anesthetized rats.

TABLE I.

The amounts of chloroform expressed as cc. per milligram of brain tissue (fresh), in rat brains following death and anesthesia by the inhalation of chloroform vapor.

Dosage: cc. in 1-liter dish.	Chloroform in brain tissue, cc. per mgm.
0.2	0.03780
0.4	0.03579
0.8	0.06810
1.0	0.07281
2.0	0.09136
4.0	0.08992
5.0 or more	0.09210
anesthesia, slight	0.01190
anesthesia, deep	0.03028

These results indicate that the amount of chloroform present in the brains of rats at the time of death varies directly with the dosage. In other words, the amount of chloroform in the brains of rats killed by chloroform is not constant. This fact may be explained by assuming that the immediate cause of death from chloroform is exerted on some tissue or organ other than the brain; or that the effect of chloroform on the brain is secondary in so far as death is concerned. Further experiments are being done to identify the tissue or organ whose poisoning by chloroform causes death.

[1] Cole, W. H., *J. Biol. Chem.*, 1926, lxxi, 173.

[2] Luedeking, C., *Am. Chem. J.*, 1886, viii, 358.

[3] Angiolani, P., *Chem. Centralbl.*, 1891, lxii, 1068.

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Galvanotropism of Roots.

A. E. NAVEZ. (Introduced by W. J. Crozier.)

From the Laboratory of General Physiology, Harvard University, Cambridge, Massachusetts.

Every one interested in plant irritability has always considered the galvanotropic response of roots as remarkable.

From previous work it could be deducted that for high densities of current or long exposures, anodic curvature (Elfving's curvature) is obtained.

For low densities of current or short exposure, cathodic curvature (genuine galvanotropic response) is obtained.