periods and on the learning of new and old habits. The means for the various groups are as follows:

Older habits

Elevated maze, pre-shock, .33, post-shock, 6.0
Alley maze, pre-shock, .83, post-shock, 4.83

Newer habits

Elevated maze, pre-shock, .50, post-shock, 11.00
Alley maze, pre-shock, .50, post-shock, 15.33

The conclusions from the above results seem to point to a more severe impairment of the newer and less overtrained habits. This statement is bolstered by the small size of the differences between the various habits during the pre-shock test period. Using Fisher's T test to determine the significance of the obtained differences we find that neither of the comparisons between the pre-shock tests are of statistical reliability. The P values for these differences are between .7 and .9.

The P values for the comparisons between the new and old habits in terms of post-shock differences are .075 for the elevated maze and less than .01 for the alley maze. In other words, the insulin shock seems definitely to impair the learning of a recently acquired alley maze habit and probably to impair the learning of a recently acquired elevated maze habit. No definite impairment can be found for the learning of older habits either on the elevated or alley maze.

TABLE I

EFFECT OF INSULIN SHOCK ON ERROR SCORES OF RATS

Elevated maze (old habit)			Alley maze (old habit)		
Subject number	Pre-shock errors	Post-shock errors	Subject number	Pre-shock errors	Post shock errors
1	0	8	7	1	3
2	1	6	8	1	6
3	1	9	9	1	4
4	0	5	10	0	4
5	0	5	11	2	10
6	0	3	12	0	2
(new habit)			(new habit)		
7	0	5	1	1	10
8	1	15	2	0	12
9	0	6	3	1	11
10	0	18	4	0	24
11	0	12	5	0	16
12	2	10	6	1	19

Each entry under "errors" represents the sum of 20 trials.

In the psychiatric conditions in which insulin shock has been employed, the symptoms are relatively recent cerebral manifestations as compared with the behavior acquired during the pre-morbid phases of the individual's development. These results with the experimental animal would seem to confirm our hypothesis of their relative susceptibility to metabolic shock applied to the cerebral patterns behind them. We are continuing our experiments and shall carry out similar ones with metrazol shock and electro-shock which also produce their therapeutic effects by the production of a cerebral anoxia.

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TOXIN FORMATION BY CERATOSTOMELLA ULMI

SINCE the discovery of the Dutch elm disease, caused by *Ceratostomella ulmi* (Schwarz) Buisman, in 1919, gums and tyloses have been observed commonly plugging xylem vessels of affected elm trees.[1,2] It has been assumed that wilting and decline of diseased elms are entirely the result of a "drouth" reaction, caused directly by the plugging of the vessels.

In the case of many vascular diseases of plants the concept that wilting and necrosis result primarily from plugging of xylem elements with hyphae, gums and tyloses has been largely replaced during the past twenty years by evidence that a toxin is the primary disease factor. Many of the fungi causing this type of disease, as well as other pathogenic fungi, have been shown to produce toxic substances which, on being transported upward in the vessels, can cause symptom appearance considerably in advance of the pathogen. Linden, Zenneck and Gunther[3] and Clinton and McCormick[4] have speculated that the symptoms produced by *Ceratostomella ulmi* might be the result of toxin production, but these authors did not present experimental evidence.

As the result of experiments begun here in 1940 it has been found that *C. ulmi* in culture produces a soluble toxic substance which is evidently the primary factor in the production of Dutch elm disease symptoms. This toxin is produced when the fungus is grown on a liquid nutrient solution containing yeast extract in addition to other nutrients. The fungus grows very poorly on the standard liquid synthetic media, but good mat formation is obtained when essential growth factors are provided by adding yeast extract, as in the following solution: KH_2PO_4 1.5 g., $MgSO_4 \cdot 7H_2O$ 1.0 g., $FeCl_3$ 0.01 g., asparagin 2.0 g., dextrose 30 g., Bacto yeast extract 2 g. and distilled water 1,000 cc.

Toxicity is tested by filtering off the fungus, after 12 to 25 days growth on the nutrient solution, injecting the sterile filtrate into small American elm trees, and testing its effect on various plant cuttings. When the toxin was freed of the fungus by Berkefeld filtra-

[1] M. G. Schwarz, *Meded. Phytop. Labor. Willie Comm. Scholt. Baarn*, 5: 74 pp., December, 1922.
[2] C. J. Buisman, *Tijdschr. Nederl. Heidemaatsch.*, 40: 338–345, October, 1928.
[3] G. V. Linden, L. Zenneck and Gunther, *Centralbl. Bakt. Abt.* II, 69: 340–351, February 28, 1927.
[4] G. P. Clinton and F. A. McCormick, *Conn. Agr. Expt. Sta. Bull.*, 389: 697–752, October, 1936.

tion and injected into healthy elm seedlings, typical symptoms of the Dutch elm disease appeared. Young leaves wilted and died, older leaves curled upward or developed necrotic spots; cell walls were discolored for long distances from the injection hole and dark gums appeared to plug the vessels. Tomato, elm, snapdragon and maple cuttings placed in tubes containing the filtrate from fungus cultures wilted severely, usually in from one to four hours. Uninoculated nutrient solution did not affect elm trees or plant cuttings, nor did control solutions adjusted to the pH of the fungus filtrate.

Five potted elms in the greenhouse and 10 elms three to four feet tall growing in nursery rows were injected with filtrate from a 25-day-old culture. Within three days the above-mentioned symptoms appeared on all trees. An average of 20 per cent. of the leaves were killed, and the average height to which the wood discoloration extended above the injection hole was 24 inches on five trees injected with 75 cc of filtrate; comparable figures for five trees injected with 200 cc of filtrate were: 29 per cent. leaf kill and 29 inches for the average height of discoloration. Tests on the time factor in relation to toxin production showed slight toxin formation after three days growth of the fungus and optimum production after a 12-day incubation period. As expected, toxin production is closely related to vigor of growth of the fungus culture.

Thermostability of the toxin after boiling for five minutes indicates that it is not enzymatic. The toxin is adsorbed by activated charcoal, is removed from aqueous solution by an excess of toluene and is antidoted by several organic chemicals.[5] Further work on the identity of the toxic substance is in progress.

Thus it has been demonstrated that symptoms similar to those of the Dutch elm disease can be reproduced by injecting a sterile filtrate from cultures of the fungus. This indicates that the same type of reaction takes place in the tree and that wilting and necrosis of leaves and discoloration of the wood are a response to the fungus toxin produced in the tree in the same way as they are to the fungus toxin produced in culture. As infection develops and more toxin is formed, gum and tylosis formation undoubtedly increases until the plugging of vessels becomes the important secondary disease factor. As a result of this plugging, water conduction may be shut off entirely and the tree dies unless new wood can be formed.

If, as these results indicate, *C. ulmi* is effective in killing elm trees primarily because of toxin production, several different approaches to attempted control of the disease by chemotherapy are indicated. As stated recently,[5] it may not be necessary to attempt to *kill* the pathogen by chemotherapy. This has been the prevailing endeavor in the past, but if the fungus toxin can be antidoted for a sufficient time by injection of a suitable chemical, the tree may outgrow and wall off the infection, particularly in a vascular disease of this type. Thus the chemical nature of the toxin becomes important in selecting chemicals which may affect it in the diseased plant. Chemotherapy, of course, can also attempt to affect the processes causing toxin production, by injecting chemicals with the aim of directly interfering with some phase of metabolism of the pathogen.

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SCIENTIFIC APPARATUS AND LABORATORY METHODS

A PHOTOELECTRIC MEMBRANE MANOMETER*

THE device to be described was designed for the purpose of making an accurate record of the pressure changes in the cardiovascular systems of various animals, especially the dog and turtle. Probably the most successful method has been used by Wiggers[1] and others, who have had good results with it in the dog. This method uses a membrane manometer similar to that shown, except that a complicated adjusting mechanism is necessary. The light source is a powerful carbon arc focused on the mirror, and the light reflected must be projected fifteen feet to a moving strip of photographic paper. This great distance is needed to provide sufficient optical magnification. Auricular pressures in the turtle would be almost impossible to record with this instrument. Because of the inconvenience of the method, its lack of sensitivity and the difficulty of making simultaneous cathode ray oscillograph records of the electrical changes on the surface of the heart it was decided to try to find some electronic means of pressure recording.

Several methods seemed promising, among them the variable resistance devices, on which Edison took out dozens of interesting patents in his search for a practical microphone. These devices were not sufficiently stable to return to the base line when the pressure was removed or had some other disadvantage. The piezoelectric pressure recorders will not record accurately a sustained pressure increase. Using the diaphragm as a condenser microphone has the same objection.

[5] J. G. Horsfall and G. A. Zentmyer, 17th Proc. Nat. Shade Tree Conf., pp. 7–15, 1941.

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[1] C. J. Wiggers, "The Pressure Pulses in the Cardiovascular System." New York: Longmans, Green and Company. 1928.