acid formation after the second hour in the case of the mature and gravid proglottides. In the case of the head portion, the decrease in acid formation was noticed after the first half hour. The acid formation was the same in an intact portion of worm as in an equal adjacent portion when divided into several groups of proglottides.

The acid formation is practically the same either in presence of a 95% oxygen mixture or in absence of oxygen. The table shows the result of a typical experiment, performed in rectangular vessels by the improved method of Warburg.[2]

TABLE I. *Mature Proglottides.*

min.	QO_2	$Q\,\frac{O_2}{CO_2}$	$Q\,\frac{N_2}{CO_2}$
30	—2.8	+4.2	+4.0
30	—2.8	+3.9	+3.9
30	—2.0	+3.6	+3.9

QO_2 = cmm. of O_2 consumed per mg. of dried worm per hour.

$Q\,\frac{O_2}{CO_2}$ = cmm. of extra CO_2 liberated per mg. per hour in oxygen.

$Q\,\frac{N_2}{CO_2}$ = cmm. of CO_2 liberated per mg. per hour in nitrogen.

It has been found that lactic acid and fatty acids, in almost equal quantities make up the majority of the acids produced in the metabolism of the sheep tapeworm.[4,5] Small amounts of succinic acid[5] and oxalic acid[6] have also been reported.

5809

A Correlation Study of Vasomotor and Muscle Tonus Response to Sympathetic Ganglionectomy.

J. DEWEY BISGARD. (Introduced by L. R. Dragstedt.)

From the Department of Surgery, Division of Orthopedics, University of Chicago.

The theory of dual motor innervation of striated muscle suggested by the histological studies of Boeck[1] and supported by the physiological observations of DeBoer[2] and Langelaan[3] has led to considerable

[4] Tischer, O. A., unpublished work.

[5] Brandt, T., *Verhandl. d. deutsch, zoolog. Gesellschaft*, 1929.

[6] Loeper, M., and Tonnet, J., *Comp. rend. Soc. de biol.*, 1931, **106**, 716.

[1] Boeke, J., *Brain*, 1921, **44**, 1.

[2] Boer, S. de, *Folia Neuro-biol.*, 1913, **7**, 378.

[3] Langelaan, J. W., *Brain*, 1915, **38**, 235.

experimental investigation and controversy. Assuming this theory and the functional hypothesis that the sympathetic nervous system was responsible for the maintenance of plastic tone and that the medullated or sometic fibers served only to initiate contractile tone, Hunter[4] and Royle[5] proceeded experimentally, and later clinically, to give these studies clinical application. They interrupted the sympathetic innervation of one extremity of animals upon which an upper motor neurone (decerebrated spasticity) type of paralysis had been or was subsequently produced, and reported hypotonicity of the muscles of the sympathectomized side. However, no one has been able to duplicate their results experimentally or clinically (Von Lochem excepted).

Recently, Wilkinson[6] has demonstrated quite conclusively that striated muscle tissue receives no sympathetic innervation. Cobb,[7] Tower,[8] Kanavel and Davis,[9] Kuno,[10] and others were unable to show any constant difference in muscle tone as a result of sympathetic deinnervation. If the animals were observed from time to time the normal (control) extremity would show diminished tone as frequently as the operated side.

In an attempt to correlate the definitely recognized interruption of vasomotor control to any alteration in muscle tone (if obtainable) these classical experiments were repeated and frequent skin temperature determinations made. These readings have been tabulated for 2 animals. All the methods commonly used to produce hypertonicity were used. Strychnine was used for 16 observations on 10 animals; parathyroid tetany was induced in 2 animals; ether rigidity 34 times in 20 animals; decerebrated rigidity in 4 animals and spinal cord section in 2 animals.

From 14 animals (10 dogs and 4 goats) the lumbar ganglia 2, 3, 4, and 5 and their corresponding trunks were removed on the left side. In 6 animals (4 dogs and 2 goats) the left stellate ganglia were removed. This latter group all developed typical Horner's syndromes. In most instances the sympathetic chains were exposed on both sides so as to preclude an error in interpretation of results caused by the trauma of the operative approach. Muscle tone

[4] Hunter, J. I., *Surg. Gynec. and Obstet.*, 1924, **39**, 721.

[5] Royle, N. D., *Surg. Gynec. and Obstet.*, 1924, **39**, 701.

[6] Wilkinson, *Med. J. Australia*, 1929.

[7] Cobb, S., *Physiol. Rev.*, 1925, **5**, 518.

[8] Tower, S. S., *Am. J. Physiol.*, 1926, **78**, 462.

[9] Kanavel, A. B., Pollock, L. J., and Davis, L. E., *Arch. Neurol. and Psych.*, 1925, **13**, 197.

[10] Kuno, Y., *J. Physiol.*, 1914, **49**, 147.

was measured by the maintenance of position against gravity, by the resistance offered to passive extension and flexion and a comparison of reflexes. With the exception of one dog there appeared to be no constant difference in muscle tone on the 2 sides; comparing the normal and sympathectomized extremities. This one animal, showing evidence of marked vasomotor reactions in the unoperated extremity was observed in ether rigidity 5 times; in strychnine rigidity twice and decerebrated rigidity once. During all these observations diminished muscle tone was manifested quite consistently in the sympathectomized extremity. While in the decerebrated state, ligatures were placed around the axillary vessels on both sides and within 5 minutes both extremities relaxed and no difference in tone could be demonstrated.

These observations and the observations made by Tower of constant hypotonicity in 2 of her series of 15 decerebrated animals would seem to indicate that any alteration of muscle tone (whenever demonstrable) is dependent upon the alteration of the blood flow through the muscle tissue resulting from the removal of vasomotor control.