As we have said in a previous article,[2] the resistance of animals tested with a definite lethal dose is at least comparable.

We believe that these experiments can explain up to a certain point why micro-organisms recently isolated from the body, especially from the blood of the patient, are not always agglutinated by their specific serums, and that passage through ordinary culture media is necessary in order to demonstrate their agglutinative properties.

These experiments agree very well with the observations of Patrick[3] concerning the agglutination of *B. typhosus* isolated from the body of patients.

We cannot here speak of microbic mutation in the sense of H. de Vries, these antigenic modifications taking place with microbes heated to 60° C. as well as with living microbes.

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The rôle of the skeletal musculature in the maintenance of the asphyxial rise of blood pressure during bulbar anemia.*

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In an analysis of the cardio-vascular mechanism of the cat by means of the changes in blood pressure and pulse rate taking place under the conditions of medullary anemia caused by temporary ligation of the carotid, vertebral and subclavian arteries, it was considered desirable to ascertain, if possible, the extent of the rôle played by the skeletal musculature in the maintenance of the high pressures which the animals show.

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[2] Combiesco, D., *Compt. rend. Soc. de biol.*, 1924, xc, 752.

[3] Patrick, A., *J. Hyg.*, 1914, xiv, 163.

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After a control tracing had been taken, in which the anemic rise of blood pressure was obtained, curare was administered intravenously in doses of 1 mg. per 5 cc. of Ringer's solution. This dose was repeated, if necessary, every five minutes until failure of the respiratory mechanism, after which time artificial respiration was maintained. Intravenous injection of curare is followed by a fall in blood pressure, due to peripheral vasomotor depression. This action is apparently on the ganglion cells (Langley), but Sollmann[1] states that this effect on the circulation passes off within fifteen minutes, while the skeletal muscular paralysis persists. Accordingly, further occlusion was delayed for about fifteen minutes in order that the effects of muscular paralysis alone might be studied. Occlusions of the head arteries were then made as before. While the height of the anemic rise was fair—in some cases double that of the control pressure—the duration before the final fall to spinal level and the number of occlusions obtainable were markedly less than in the normal animal. This is shown in the following table:

Normal June 15, 1923.				Curare March 25, 1924.		
Occlusion number	Length of occlusion	Max. height of pressure	Control pressure	Length of occlusion	Max. height of pressure	Control pressure
	secs.	mm. Hg.	mm.		Control	
1	120	240	100	secs.	mm. Hg.	mm. Hg.
2	180	205	80	200	200	100
3	130	170	80		After curare	
4	160	185	80	90	130	60
5	160	200	75	85	120	65
6	165	230	75	100	120	50
7	170	190	75	95	110	45
8	110	190	70	50	60	30
Six more occlusions were obtained.				No further rise obtainable.		

Another striking feature of the reaction in the curarized animal is the increased latent period after occlusion of the head arteries, before the onset of the anemic rise. Normally, this occurs within one second after occlusion; in the curarized animal, the anemic rise does not begin until ten seconds or more have elapsed. For example, in the experiment of April 21, 1924, the tracing shows that for the control occlusion, one second elapsed between the time the head arteries were clamped off and the onset of the anemic rise, while in the second occlusion, after curare had been

[1] Sollmann, A Manual of Pharmacology, 1922, 383.

previously injected, this same period occupied 11 seconds; in the third occlusion, 11 seconds; and in the fourth and last occlusion, 15 seconds.

Pike[2] has shown that intravenous injection of curare after complete elimination of the medulla brings about a constant fall in blood pressure which is, in general, about equal to the fall observed after division of the dorsal roots of the spinal nerves. Langley[3] confirmed this observation in 1919. This fall is not to be confused with the effects immediately following injection of curare, as its duration persists as long as paralysis of the skeletal musculature. Tillie[4] has stated that the dose of curarin by which the vasomotor nerves are affected in 100 to 300 times as great as that which induces paralysis of the skeletal muscles.

Since it would appear, then, that under the conditions of these experiments, the skeletal musculature and not the vaso-motor mechanism is directly affected by curare, we may be justified in concluding that the skeletal musculature is responsible in some degree for the rapid initiation of the height of the anemic rise, and in a considerable degree for

1. The length of the latent period before response to occlusion.
2. The duration of the anemic rise of blood pressure.

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The influence of suprarenalectomy in rabbits on antitoxin (diphtheria) formation.

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Previous reports by Také and Marine on hemolysin formation in suprarenalectomized rabbits and by Jaffe and Marine on agglutinin (typhoid) formation in suprarenalectomized rats have shown that both hemolysin and agglutinin formation are increased in suprarenalectomized animals. The antibody titer may

[2] Pike, *Quart. J. Exp. Physiol.*, 1913, vii, 1.
[3] Langley, *J. Physiol.*, 1919, liii, 120.
[4] Tillie, *Arch. exp. Path. u. Pharmacol.*, 1890, xxvii, 1.