In 4 experiments in this laboratory it was found that the corpuscles constitute 47.3% of the blood volume. This agrees fairly well with the mean value of 45.21% found by Powers, Bowie and Howard on a series of twenty-five dogs.[9]

Assuming that the corpuscles constitute 45.2% of the blood volume, calculation from the above data gives a value of 3.34 for the ratio

$$\frac{\text{plasma lactate concentration}}{\text{corpuscle lactate concentration}}$$

While the finding is chiefly of qualitative interest, owing to the statistical inadequacy of the data, it is clear that there is a marked inequality in the distribution of lactate between the corpuscles and plasma of the dog.

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Dilatation of the Heart by Amytal.

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Amytal, prepared as for surgical anesthesia, was added to the venous reservoir of an open-circuit heart-lung-preparation. The preparation was made under pure ether anesthesia, and in each case the experiment was postponed for at least 45 minutes for the effects of the ether to wear off. Most of the blood was defibrinated and the remainder rendered incoagulable by the addition of 25 mgm. heparin. The venous pressure and artificial resistance pressure were maintained constant; heart volume changes were recorded by a float recorder connected to a Jerusalem and Starling cardiometer. The useful (non-coronary) output of the heart was measured several times before and after each dose by collection in a graduated cylinder for 10 second periods. At the conclusion of each experiment the quantity of blood in the reservoir, cannulae and apparatus was measured and the heart and lungs were weighed and measured by displacement. The resulting estimate of quantity of tissue is probably accurate to within 10%.

[9] Powers, J. H., Bowie, M. A., and Howard, I. M., *Am. J. Physiol.*, 1930, xcii, 665.

TABLE I.

	Exp. No.	Dose	Diastolic vol. after dose	Useful output cc. per min.	Rate	Stroke volume
			Amytal			
Dog 1	1	18 mgm./kilo	+	360 cc. No change	No change	Inc.
	*2	27 " / "	++	360-300	" "	"
	*3	45 " / "	+++	300-275	" "	"
Dog 2	4	50 " / "	+	280-264	" "	"
	*5	50 " / "	++	280-188	" "	"
Dog 3	6	58 " / "	++++	264-66-0 Heartblock	" "	"
Dog 4	7	70 " / "	+++	547-360-180	" " (120-120)	Dim.
	8	70 " / "	Very slight increase	210-60	No change	"
Dog 5	9	20 " / "	+	No change	(No venous pulsation)	
	10	50 " / "	++++	Flow stopped	(Venous pulsation)	
		Washed carefully—adrenalin—almost complete recovery				
	11	75 " / "	++++	Flow stopped	(Venous pulsation)	
			Ether			
Dog 6	12	2 breaths	+++	250-210	No change	Inc.
	13	3 "	++++	Dim.	130-119	"
	14	3 "	++++	"	120-60	"
	15	Greater than 3 breaths	+++++	250-60	No change	"

* Before these experiments blood was drained from venous reservoir and discarded; fresh blood added to wash out tissue, and discarded; more fresh blood added for experiment.

The table gives a record of our experiments comparing the effects of amytal with those of ether, the former being expressed in mgm. amytal per kilo of tissue (heart, lung, blood), the latter in the number of breaths during which a wad of cotton soaked in ether was plugged into the intake pipe of the artificial respiration pump.

Fig. 1 shows a typical cardiometer tracing, recording the result of a dose of 27 mgm. amytal per kilo. With this slight dose, as with all other doses of amytal, save in one case where the heart was revived by adrenalin, no recovery was observable during at least half an hour although fresh blood was substituted twice. According to Eddy[1] such a dose has no anesthetic effect; on the contrary the sensitivity to pain is heightened.

Examination of the results in the table shows that after a very small dose of amytal (18 mgm. per kilo) an increase in diastolic volume takes place although the venous pressure be maintained prac-

[1] Eddy, N. B., *J. Pharm. and Exp. Therap.*, 1928, xxxiii, 43.

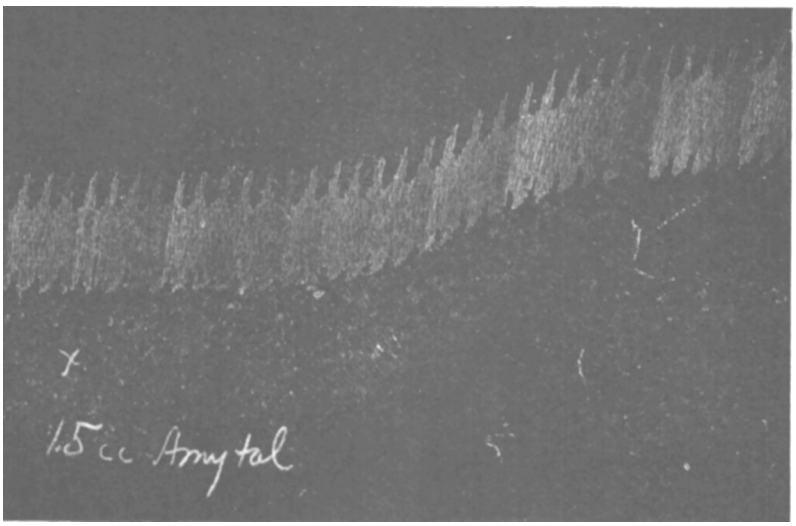

FIG. 1.

tically constant. Presumably dilatation is necessary to enable the heart to maintain its useful output. A greater dose causes a greater dilatation which, however, is accompanied by a diminution in useful output, since the dilatation is apparently not sufficient to compensate for the effect of the drug. This diminution in output may be observed with a dose as small as 27 mgm. per kilo, which is about half that commonly used for anesthetic purposes. It should, however, be remembered that in a whole animal under anesthesia a rise in venous pressure is possible and would provide further scope for compensation, so that there might be no diminution of heart output. In one experiment after a small dose, we raised the venous pressure, and found that compensation was perfect.

A dose of ether of 3 breaths produced the same effects in a more marked degree than the usual anesthetic dose of amytal, but with ether the effects rapidly passed off (*i. e.*, diastolic volume had returned to normal within 3 minutes). Ether in large doses sometimes produced a marked diminution in heart rate. With amytal we were able to observe a change in rate only in the one case where we used so large a dose as to produce heart block and death. Eddy[1] found a diminution of heart rate in intact cats with all doses of amytal from 25 mgm. per kilo up.

Accurate quantitative determinations of stroke volume have not yet been made, but in all cases save one, an increase of stroke volume was observed to accompany the diminished useful output. This

suggests that one action of the drug may be a relaxation of the coronary system, though if an increase in coronary flow takes place, we have no indication whether this is in response to some change in the myocardium or is itself the primary effect of the drug. We have observed marked venous pulsation when the outflow is almost completely stopped, but since increasing the venous pressure will compensate for considerable diminution in flow without producing venous pulsation, we do not believe that valvular incompetence plays any part in the diminution in flow except with abnormally large doses, in which case the flow is almost *nil*.

Conclusion. In a heart-lung-preparation amytal increases diastolic volume and decreases useful outflow.

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Changes in Temperature of the Lower Extremities Following the Induction of Spinal Anesthesia.*

SELLING BRILL AND LESTER B. LAWRENCE. (Introduced by C. D. Leake.)

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We were interested to determine whether or not spinal anesthesia would cause vasodilatation and increased blood flow in the lower extremities with a resulting rise in temperature, and whether or not such effects of blocking the sympathetic nerves would be of aid in selecting patients for surgery of the sympathetic nervous system.[1]

Temperature readings were taken with the thermo-electric apparatus designed by Bazett and McGlone,[2] which contains loop and needle thermocouples allowing accurate surface and tissue temperature measurements to be made on human subjects. Surface readings were taken from the plantar surfaces of the big and little toes of both feet and from the palmar surfaces of the index, middle and ring finger of one hand. Tissue temperatures were noted by inserting the needle thermocouple from 1 to 2 cm. into the first and fourth metatarsal spaces of both feet. One or more readings were taken before induction of spinal anesthesia and frequent observations were made for 40 or more minutes afterward. Procaine hydrochloride

* Aided in part by a grant from the Purington Research Fund.

[1] This problem arose from certain suggestions of Prof. H. C. Naffziger.

[2] Bazett, H. C., and McGlone, F., *J. Lab. and Clin. Med.*, 1927, xii, 9, 913.