gested by Stewart[2] has recently received strong inferential support from the demonstration of localized antibody formation by McMasters and Hudack.[3]

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Influence of Ethyl Alcohol on Energy Metabolism of the Mammalian Heart.*

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(Introduced by Maurice B. Visscher.)

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The use of the older methods of studying the isolated heart led to contradictory conclusions regarding the effect of alcohol on cardiac contraction. Sulzer,[1] using the Starling heart-lung preparation, at present the most sensitive and reliable method for this purpose, found no stimulating effect in any concentration and showed that concentrations commonly reached in the human blood stream in alcoholic intoxication produce dilatation without an increase in the work of the heart. We have confirmed this effect and studied the changes in energy metabolism associated with it.

We have used a modified heart-lung preparation, which has been previously described.[2] In some experiments the diastolic volume was allowed to increase when alcohol was added (Table I), while in others (Table II) the external diastolic volume was maintained constant throughout the experiment by adjustment of the venous return. In all cases the venous pressure had to be lowered in order to keep diastolic volume constant after alcohol. A piston recorder was used for ventricular volume recording and the heart was held in a glass cardiometer, following the technique of Starling and Visscher.[3]

After a control period of 15 to 30 minutes, during which the volume output and oxygen usage remained constant, the desired

2 Stewart, F. W., *Am. J. Path.*, 1925, **1**, 495.

3 McMasters, P. D., and Hudack, S. S., *J. Exp. Med.*, 1935, **61**, 783.

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1 Sulzer, *Heart*, 1924, **11**, 141.

2 Peters, Rea and Visscher, PROC. SOC. EXP. BIOL. AND MED., 1934, **32**, 268.

3 Starling and Visscher, *J. Physiol.*, 1927, **62**, 243.

TABLE I.
Effect of Ethyl Alcohol on Energy Exchanges in the Heart.

Exp.	Estimated conc. of alcohol, %	Oxygen consumption cal./min.	Volume output cc./min.	Efficiency %
4/6/33	0	34.1	416	3.72
	.12	36.2	406	3.41
	.23	37.0	385	3.12
4/8/33	0	36.4	429	3.68
	.10	39.6	429	3.38
	.20	33.8	353	3.15
4/10/33	0	36.1	682	6.70
	.12	36.7	600	5.56
	.25	33.2	429	4.04
4/11/33	0	30.2	341	3.96
	.13	23.5	206	3.00
4/17/33	0	30.6	411	4.33
	.17	28.1	353	3.95
4/19/33	0	38.3	469	4.54
	.14	33.9	441	4.75
	.28	29.0	330	3.99
4/23/33	0	33.8	395	3.54
	.14	35.9	366	2.81
	.28	29.5	291	2.88
6/1/33	0	30.6	435	5.26
	.16	35.7	411	3.57
6/3/33	0	33.8	364	3.36
	.23	31.0	333	3.32
	—	34.5	333	2.98

TABLE II.
Influence of Alcohol on Energy Exchanges at Constant Diastolic Volume.

Exp.	Estimated conc. of alcohol, %	Oxygen consumption cal./min.	Volume output cc./min.	Efficiency %
6/13/33	0	30.9	750	9.04
	.31	14.19	84	2.79
6/14/33	0	28.5	600	7.13
	.16	13.3	188	4.36
6/16/33	0	27.1	545	7.21
	.25	18.8	50	.75
	—	17.5	23	.34
6/21/33	0	23.3	353	4.43
	.15	14.3	0	0
6/24/33	0	26.4	600	7.96
	.25	21.1	480	7.46

amount of alcohol was injected into the tubing between the stromuhr and the venous reservoir so as to insure thorough mixing before reaching the heart. Within 30 to 40 minutes after the injection the variables recorded had generally reached a new fairly constant level, and then in some experiments alcohol was injected again and the effect of the higher concentration determined. The concentration of alcohol injected was 20% except in Exp. 4/10/33,

in which 95% alcohol was added to the blood undiluted for purposes of comparison. The time of injection was generally 2 to 3 minutes.

The alcohol concentrations in per cent by weight were estimated from the amount injected, the volume of circulating blood, and the weight of the heart and lungs. The cardiac efficiencies are based on the external work of the heart only. A correction for the metabolism of the lungs was made in the experiments of Table I by subtracting 25% of the initial oxygen consumption values except in Exp. 4/10/33 where the output is large and a 20% correction was applied. The experiments of Table II are not corrected for lung metabolism.

When the ventricular volume was unrecorded (Table I) the oxygen consumption generally decreased or increased only slightly although the heart must have dilated. In Exp. 6/3/33 in which ventricular volume was recorded but not controlled, the oxygen consumption decreased slightly although the external diastolic ventricular volume increased 17 cc. immediately after injection and continued to increase. The efficiency changes are not large but show a tendency to a decrease.

In the experiments of Table II the oxygen consumption decreased at constant external diastolic volume. This decrease is definitely an effect of the alcohol as we have never obtained decreases this large in numerous preparations failing spontaneously.[4]

Since a decrease in oxygen consumption at constant external diastolic volume might be due to hydration of the myocardium, we determined the water content of 2 groups of ventricles (Table III) by drying samples at 105° to constant weight. One group comprised hearts sampled while working 45 minutes after injection of alcohol and 2 hours after completing the heart-lung preparation. The control group was sampled after working 2 hours. It appears that alcohol produced only very slight, if any, hydration. The effects on oxygen consumption are not explicable on the grounds of alteration in volume of muscle mass due to hydration.

TABLE III.

Water content of	
Alcohol treated hearts, %	Controls, %
79.2	79.4
79.1	79.2
78.1	
80.1	Av. 79.3
79.1	
Av. 79.1	

[4] Peters and Visscher, *Am. Heart J.*, in press.

The changes in external efficiency are the result mainly of the great decrease in external cardiac output. Since no measurements of coronary flow were made the actual muscle efficiency is not known. It would be unsafe to conclude that the muscle became very much less efficient after alcohol unless coronary blood flow was determined in cases where the external output decreased greatly. The important and unequivocal result of these studies is that the total energy output diminishes, at constant diastolic volume, under the influence of ethyl alcohol.