

Oxygen Consumption of Post-Mortem Human Heart Muscle.*† (24310)

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(Introduced by Earl R. Loew)

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Excitability and contractility characteristics of post-mortem human heart muscle were previously reported by the present authors(1). This report is concerned with oxygen consumption of post-mortem human heart muscle.

Materials and methods. Human hearts were obtained from 1.8 to 8.4 hours after death. Two trabecular muscles of diameters less than 1 mm were removed from each left ventricle. One muscle preparation was positioned in clamps, placed in a constant temperature 37.5° bath and bathed continuously with oxygenated Ringer's solution. The muscle was kept under a constant resting tension of 1 g, and after a 30 minute to one hour equilibration period was stimulated at a frequency of 1/sec with 6 msec square wave pulses. Isometric developed tension was recorded. Details of the method of stimulation and recording, and composition of Ringer's solution used were as previously reported(2). The other muscle preparation from each heart was suspended in a modified Warburg flask in a vertical position. A freely hanging 1 g weight was attached to the lower end of the preparation. The muscle was not stimulated, and was under a constant resting tension of one g. The Warburg flask contained 6.9 ml of Ringer's solution and 0.1 ml of 20% KOH in a side well. Oxygen consumption, under a 100% O₂ gas phase, was determined by standard manometric technics, and is expressed as $\mu\text{l O}_2$ uptake/hr/mg dry weight of tissue

(QO₂). Oxygen consumptions are reported for only those preparations, 7 in number, whose mates from the same heart responded by contraction to electrical stimulation in the muscle bath. (5 preparations, obtained within the same time interval after death, did not respond).

Results. Table I lists the primary clinical diagnosis of the patient from whom each heart was obtained, interval from death of patient to placement of the muscle preparations in bath and Warburg systems, and QO₂'s and isometric tension development of the preparations from each heart. The mean QO₂ was 21.0 ± 2.5 (S.E.) and mean isometric tension development was 287 ± 137 mg. Tension is expressed simply in mg per preparation, since the preparations were approximately all the same size, (length: 5 mm; diameter: 0.7 mm; and weight: 0.6 mg, dry). There was no correlation between oxygen consumption of one preparation from an autopsied heart and capacity to develop tension by another preparation from the same heart, nor was there any correlation between oxygen consumption or tension development with the post-mortem interval.

It may be questionable whether the oxygen consumptions reported here represent respiratory rates for normal human heart tissue. The influence of the post-mortem anoxic interval on respiration is undetermined. Clinical condition of the patient at time of death may also have influenced the respiratory rate. In spite of these considerations mean oxygen consumption for post-mortem human ventricular tissue compares favorably with *in vivo* measurements for the normal human heart reported by others. For example, recently reported mean values are 21.84(3) and 22.00(4) (converting given ml O₂ utilization/min/100 g of tissue to QO₂'s). Our data therefore indicate that post-mortem human heart muscle is a functionally and metabolically active tissue, and may serve as a valuable adjunct to the study

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TABLE I. Oxygen Consumption of Post-Mortem Human Heart Muscle.

Clinical diagnosis	Post-mortem interval, hr	QO ₂	
		Prep. A	Isometric developed tension, mg Prep. B
Hydrocephalus (still-born infant)	1.8	23	90
Rheumatic heart disease, mitral stenosis	2.0	17	1020
Generalized arteriosclerosis, involvement of coronary arteries	3.5	12	30
Arteriosclerotic heart disease	4.1	17	190
Generalized arteriosclerosis, G-I bleeding	5.0	27	150
Carcinoma of esophagus	5.5	21	55
Malignant carcinoid	8.4	31	475
Mean		21.0 ± 2.6	287 ± 137

of human cardiac physiology and pathophysiology.

Summary. Oxygen consumption measurements were made on trabeculae carneae from 7 contractile human hearts, obtained 1.8 to 8.4 hours after death. Mean QO₂ was 21.0 ± 2.5, which compares favorably with mean *in vivo* oxygen utilization values reported for the normal human heart. There was no correlation between magnitude of the oxygen uptake and isometric tension developed by a

sister preparation from the same heart, nor with the post-mortem interval.

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Isolation of Partially Purified Properdin from Bovine Serum by Cold Ethanol Fractionation.* (24311)

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Properdin, a normal serum constituent which in conjunction with complement or complement-like substances and Mg++ constitutes the properdin system, was first reported to be a natural humoral defense mechanism by Pillemer and co-workers(1). A procedure for isolation and concentration of this euglobulin substance was described. The protocol involves the adsorption of properdin to zymosan and subsequent elution under suitable conditions(2). The use of the zymosan adsorption technic generally yields final properdin solutions of variable activity since zymosan preparations

vary in activity. Pillemer *et al.*(1) reported that properdin is found in serum Fraction III using the separation procedure described by Deutsch *et al.*(3). Pennell *et al.*(4) used method 6 of Cohn, *et al.*(5) and reported that 90-95% of total properdin activity of plasma was recovered in a subfraction of Fraction I, and the remaining activity appeared in Fraction III. Linder(6) has shown that the majority of properdin activity of human serum resides in the gamma globulin eluate when the serum constituents are separated electrophoretically. Experiments designed to determine the value of properdin as a therapeutic agent following exposure of animals to whole body x-irradiation made necessary the preparation of properdin solutions of high activity per unit volume in order that

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