THE VELOCITY FACTOR IN CARDIAC WORK.

By C. LOVATT EVANS.

(*From the Institute of Physiology, University College, London.*)

It was shown in a recent paper by Matsuoka and the present writer (1) that in the computation of the work of the heart the velocity factor $\frac{mV^2}{2g}$ is often of considerable importance, and when large outputs are being dealt with forms a considerable percentage of the total work of the heart. In the calculations which were dealt with in that communication the velocity taken for these estimations was the mean velocity at the narrowest point close to the aortic orifice, in this case at the opening of the glass cannula inserted into the ascending aorta and having a diameter of 5 mm. at its narrowest point. It is this mean velocity which has hitherto been used in all calculations of cardiac work. Prof. Starling has suggested to me that it is not the mean blood velocity in the ascending aorta or at the aortic orifice which should be taken as a basis for calculation, but the velocity during the period of ventricular expulsion only. Since the velocity at the aortic orifice during the period of expulsion is also not uniform, but rises to a maximum and then declines again to zero, it would seem that for accurate calculation the velocity to be taken could be best arrived at by a mathematical treatment of cardiometer curves. But for present purposes such refinements as this would seem to be superfluous, since the velocity would be liable to an indeterminate amount of further variation owing to alteration of the sectional area of the orifice by stretching during maximal phases of pressure, as well as from other circumstances.

It will therefore be sufficient for present purposes to assume that the mean velocity in systole varies inversely as the duration of the period of expulsion. With the conditions which prevailed in the experiments already referred to, the period of systolic output would occupy not more than three-eighths of the cardiac cycle[1]. The mean velocity

[1] Calculations from curves taken by Patterson, Piper and Starling (2) from hearts working under similar conditions indicate that it might be somewhat less than this, but the higher figure is taken in order to avoid over-estimating the effect now under consideration.

during expulsion will therefore be $2\frac{2}{3}$ times greater than the mean aortic velocity, and in accordance with this the expression $\frac{mV^2}{2g}$ in all the previous calculations where V represents the mean aortic velocity should be multiplied by the square of 8/3 or by approximately 7 to arrive at the correct value. The work formula for both ventricles, when the rate of the heart does not greatly depart from the normal value which it has for the isolated organ, now becomes $W=7\left(\frac{QR}{6}+\frac{wV^2}{g}\right)$ instead of that previously given[1], V representing the mean aortic velocity.

The effect of thus multiplying the velocity factor of both ventricles by 7 is to raise it to an order of magnitude comparable with that of the resistance (QR) expression; with low arterial pressures and high outputs in fact, it may happen with hearts under the conditions of our experiments that the greater part of the heart's energy is used in imparting velocity to the blood. When the output is small, say less than eight times the heart-weight per minute, the velocity factor is still small enough to be negligible. In Table I are given the values of the

TABLE I. Values of $\frac{7wV^2}{g}$ for dog's heart with aortic cannula of 5 mm. diameter.

Output l. p. hr	Mean velocity m. p. sec.	$\frac{7wV^2}{g}$ kg.m. per hr (both ventricles)
3	0·042	0·0038
12	0·169	0·253
36	0·51	6·72
48	0·67	15·4
60	0·845	30·7
72	1·02	53·7
84	1·18	84·2
96	1·35	125·0
108	1·52	179·0
120	1·69	245·0

[1] The formula previously given was $7\frac{QR}{6}+\frac{wV^2}{g}$. In cases where the relative duration of systole is much altered, the new formula given above must be modified to meet the altered conditions, and now becomes $W=7\frac{QR}{6}+\frac{w(VC)^2}{gE^2}$, where C = duration of cardiac cycle, and E = duration of period of expulsion. A further correction which might in some cases be of importance and lead to further modification of the formula, is introduced in cases where there is much difference between the sectional areas of the pulmonary and aortic orifices. The pulmonary orifice is usually held to be 3 or 4 mm. wider than the aortic, which is in the male human heart about 24 mm. in diameter *post mortem*; owing to the differences in pressure on the two sides of the heart, it is doubtful however whether this difference, or any difference at all, obtains during life, and this discrepancy is therefore not taken into further account here.

velocity expression for possible ranges of output of dog's hearts as used in the series of experiments with Matsuoka, and in which a cannula of known diameter was inserted into the aorta immediately beyond the valvular orifice. In Fig. 1 these results are shown graphically (*B*) and may be compared with the work done in overcoming the arterial resistance (*A*) at a pressure of 120 mm.; the total work done in kinetic and

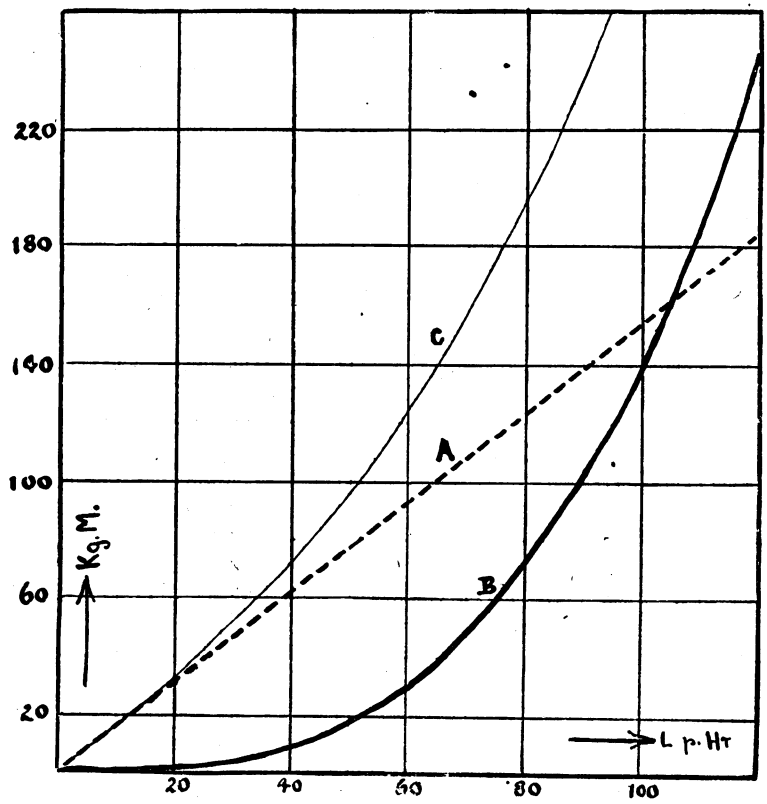

Fig. 1. Curves showing the relative importance of the kinetic and static factors when the arterial pressure is 120 mm. A = values of QR. B = values of $\frac{wV^2}{g}$ when V represents velocity during systolic output. C = total work done ($A + B$). The velocity factor, at first negligible, rapidly outstrips the resistance factor, as the output is raised.

static forms together is also shown (*C*). It will be noted that at outputs of about 104 l. per hr. the values of the two modes of disposal of energy have become equal, while at still greater outputs the velocity factor rapidly outstrips the other.

Effect of the foregoing considerations on previous results. The effect of this enhanced importance of the velocity component on the results of previous experiments is to modify the amounts of work done by the hearts, and to raise the efficiencies of hearts working at high outputs.

TABLE II. Recalculations from the experiments bearing the same numbers given in *Journ. of Physiol.* **49**. 402–403. 1915. The periods are here grouped together as shown in Column I and the average results are shown in the other columns.

Periods	Arterial pressure	c.c. O_2 per hour	Output l. p. hr	Kg.m. work done p. hr	Kg.m. energy used p. hr	% efficiency
Exp. 7.						
1, 2, 3	80 mm.	179	8·7	11·9	370	3·3
4, 5, 6	,,	185	16·3	20·7	383	5·5
7, 8	,,	212	27·8	37·1	437	8·5
9, 10	,,	287	52·0	81·6	593	13·8
11	,,	649	92·0	218·0	1343	16·3
12	,,	175	16·4	20·3	363	5·6
Exp. 8.						
1, 2	,,	116	6·4	7·8	240	3·3
3, 4	,,	129	18·9	24·0	267	9·0
5	,,	247	53·4	87·0	509	17·1
Exp. 9.						
1, 2	,,	97	14·6	18·2	201	9·0
3, 4, 5	,,	158	29·7	39·9	328	12·1
6, 7	,,	201	42·3	62·1	416	15·0
8, 9	,,	281	70·8	137·0	584	23·5
10, 11	,,	520	91·4	217·0	1078	20·4
Exp. 10						
1, 2		140	17·0	21·2	291	7·3
3, 4		159	33·8	46·7	329	14·2
5, 6	,,	299	81·0	175·0	620	28·2
7, 8	40 mm.	98	17·0	11·0	202	5·5
9	80 ,,	119	16·8	20·7	247	8·4
10, 11	120 ,,	174	16·8	31·0	360	8·6
12	160 ,,	250	16·8	40·7	517	7·9
13	,,	334	37·7	99·0	693	14·3
Exp. 11						
1	100 mm.	230	37·9	64·7	476	13·6
2	140 ,,	410	63·1	166·0	850	19·6
3	180 ,,	517	73·9	259·0	1070	24·2
Exp. 12.						
1	100 mm.	213	20·4	32·2	442	7·3
2	126 ,,	334	67·2	163·0	690	23·6
3	134 ,,	636	100·2	333·0	1310	25·5

The results of all those experiments in which the outputs were low are not altered. In the paper by Matsuoka and the present writer, the chief alterations are in Exps. 7 to 12, the results of which, corrected in the way already described, are given in an abbreviated form in Table II.

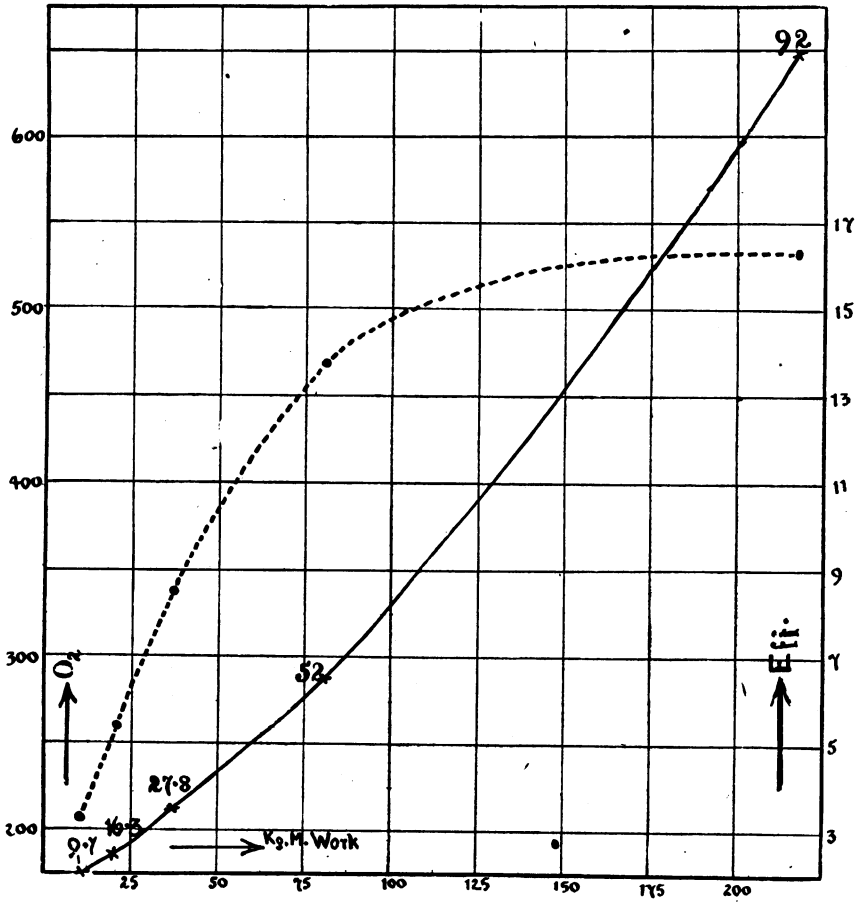

Fig. 2 (Exp. 7). Effect of varied output on oxygen usage (c.c. per hr—continuous line) and on % efficiency (dotted line). The figures on the oxygen curve indicate the hourly output in litres.

The results of Exp. 7 are shown graphically in Fig. 2 which should be compared with Fig. 3 of the former paper. It will be noted that the mechanical efficiency now calculated shows a continuous increase with increasing output, the rise being at first rapid and then more slow. The

efficiency in some cases, however, still shows a falling-off when the output is raised beyond a certain limit. This is the case in Exp. 9, graphically represented in Fig. 3, and is presumably due to over-stretching of the fibres of the ventricular wall.

The main conclusions in the paper with Matsuoka on the effect of

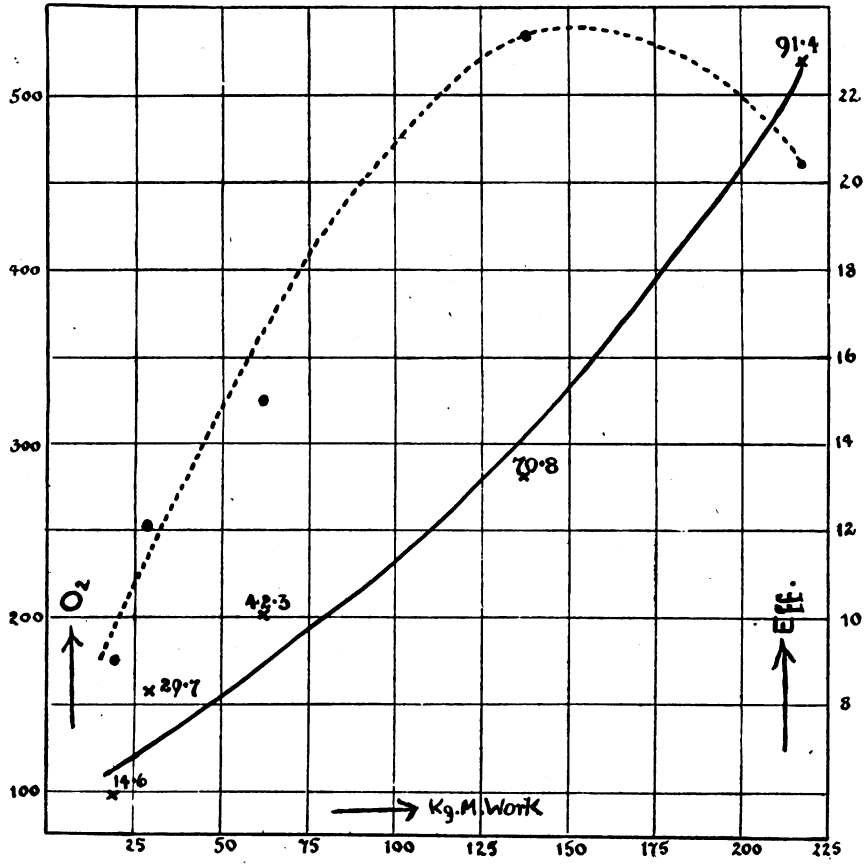

Fig. 3 (Exp. 9). Type of efficiency curve showing first a rise and then a fall of efficiency as the output increases. Curves and figures as in Fig. 2.

various conditions on the performance of the heart are not altered except as regards some of the quantitative results given. Thus the statement there made with regard to the maximum efficiency of the heart under the conditions of these experiments was that it was about 20 %; we now see that this figure has been often exceeded (Exps. 9, 10,

11 and 12) and the efficiency obtainable with optimum conditions probably lies in the neighbourhood of 30 %, *e.g.* 28·2 % in Exp. 10.

Applications to the human heart. In the absence of certain knowledge of the effective diameters of the aortic and pulmonary orifices in the human heart during life, it is hardly possible to make reliable calculations regarding the work done in giving velocity to the blood, since the work varies inversely as the square of the diameter of the outlet. It is moreover reasonable to suppose that in any individual heart these apertures, even when considered at the same phase of systole, are not constant in diameter, but vary according to the pressure within. With large outputs therefore, under conditions which prevail in the intact body, the accompanying rise of pressure would tend to relieve the work done in this respect.

Little is also known with regard to the velocity of the blood in the human aorta. If either the mean velocity at the aortic orifice, or the sectional area of the orifice itself were known, it would be possible to calculate the other from our knowledge of the volume output of the heart, which is known with a reasonable approach to accuracy from the investigations of Krogh(3) and others.

Nicolls(4) has calculated that the velocity of output past the aortic valves is ten times the mean velocity in the ascending aorta, and amounts to 320 cm./sec., but this calculation was made from premises which are no longer tenable. I have calculated that when the output is 60 c.c. per beat, and the aortic entrance has a diameter of 2·5 cms., and the time of output lasts for 0·3 sec., that the systolic velocity of output is about 40 cms. per second; these are data applicable to the normal heart when the body is at rest, and as is pointed out in every text-book of physiology, the kinetic energy given to the blood under these circumstances is a negligible fraction of the whole energy expenditure. Let us now consider a case where a large output is being dealt with, say the output of 21 litres per minute found in one case by Krogh and Lindhard(3); if the arterial pressure was low, say 100 mm. and the pulse rate and period of blood-expulsion were the same as obtained with the body at rest, the maximum velocity of blood would be about 196 cm./sec., and the velocity expression would amount to nearly 20 % of the total work of the heart. But these conditions do not obtain, for with such a large output the ventricle spends relatively longer in systole. Using our formula $W=\frac{7QR}{6}+\frac{w(VC)^2}{gE^2}$ for the calculation, we obtain the following result:

Assume the arterial pressure to be 130 mm., the duration of the cardiac cycle to be 0·5 sec., the period of expulsion to be 0·25 sec., then we have as the work of both ventricles, per min.

$$W = \frac{7 \times 21 \times 1{\cdot}7}{6} + \frac{21 \times (\cdot 71 \times 0{\cdot}5)^2}{9{\cdot}8 \times (0{\cdot}25)^2} \text{ kg.m.}^1$$

$$= 41{\cdot}3 + 4{\cdot}3 \text{ kg.m. per minute.}$$

i.e. the expenditure of energy in kinetic form is still only about 9·5 % of the total. The advantage of the altered time-relations is evident.

Still clearer are the effects of a stenosis of the aortic opening. Let us suppose that the orifice be narrowed down to a diameter of 1 cm. In such a case the pulse rate is always slowed and a longer time is spent in systole, and these circumstances lead to a reduction of the blood velocity. Nevertheless, even under conditions of bodily rest, the velocity factor may easily represent half of the total work of the heart[2].

The velocity factor in hearts under the influence of adrenalin. It has been shown that the gaseous metabolism of the heart when under the influence of adrenalin is greatly increased (5), and Patterson has shown (6) that the duration of the isometric period is greatly shortened, being often reduced to half its normal value, while the time per minute spent in systole is also reduced. The great developments of endocardiac pressure in both ventricles is probably a result of this increase in the velocity of the blood expelled, and there seems little doubt that this factor plays a big part in determining the increase in the gaseous metabolism.

Influence of heart rate on the efficiencies. It must not be forgotten that in all these experiments we are dealing with the denervated heart, and that the heart as it beats in the intact animal would beat at rates

[1] The figure 1·7 represents the arterial pressure in terms of a column of blood: 0·71 is the mean velocity for the entire cycle, and is arrived at as follows:

$$\text{Sectional area of aortic orifice} = \pi \left(\frac{2{\cdot}5}{2}\right)^2 = 4{\cdot}9 \text{ sq. cm.}$$

21 l. per min. = 350 c.c. per sec.

$$\text{Velocity per second} = \frac{350}{4{\cdot}9 \times 100} = 0{\cdot}71 \text{ metre.}$$

The actual velocity of output is now about 140 cm. per second under the conditions assumed.

[2] Let us suppose that the output per beat in such a heart is 60 c.c. and the time of output is 0·5 second (this is certainly as long as it would ever be likely to be). The mean systolic velocity amounts to 152 cm./sec. and the distribution of the work done per heart beat is as follows:

$$QR = \cdot 06 \times 1{\cdot}3 = \cdot 078 \text{ kg.m.}$$

$$wV^2/2g = \frac{\cdot 06 \times (1{\cdot}52)^2}{9{\cdot}8 \times 2} = \cdot 071 \text{ kg.m.} = 48\,\% \text{ of total work.}$$

which, except in the case of the highest outputs, would be lower than those obtained with the use of the heart-lung preparation.

It is possible, by making certain assumptions, to allow provisionally for these differences, and to get an idea of what would be the approximate metabolism in cases where the rate resembled that in the normal animal. Thus, if we assume that (as is certainly the case for different rates due to slight differences of temperature), the gaseous exchanges are directly proportional to the rates, and if we further assume that the pulse rate steadily increases from the normal intact rate when the output is low, to the denervated rate when the maximum output is reached, then we shall find that the efficiencies at the lower outputs are considerably increased. Thus the following table shows such a recalculation in the case of Exp. 9. It is quite probable that the effect of this alteration in pulse rate is rather under- than over-estimated in this case.

TABLE III.

	Unadjusted figures (Exp. 9)			Figures adjusted for rates		
Output p. hr	Pulse rate p. m.	Oxygen usage c.c./hr	Efficiency %	Pulse rate p. m.	Oxygen usage c.c./hr	Efficiency %
14·6	145	97	9·0	100	67	13
29·7	145	158	12·1	110	120	16
42·3	145	201	15·0	115	159	19
70·8	145	281	23·5	135	262	25
91·4	145	520	20·4	145	520	20·4

SUMMARY.

It is shown that the energy expended in imparting velocity to the blood is very considerable, both under experimental conditions in animals and in the human heart during exercise.

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