AN ESTIMATION OF THE HEPATIC BLOOD FLOW AND SPLANCHNIC OXYGEN CONSUMPTION IN HEART FAILURE

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Research Article

AN ESTIMATION OF THE HEPATIC BLOOD FLOW AND SPLANCHNIC OXYGEN CONSUMPTION IN HEART FAILURE[1]

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That the circulation of the liver is disturbed in heart failure is well known to every clinician. A tender, enlarged liver is a frequent and at times the only evident manifestation of cardiac failure. The disturbances in the circulation leading to hepatic enlargement have been difficult to investigate because of the lack of methods to measure hepatic blood flow and cardiac output in patients with congestive heart failure. The recent introduction of the procedure of catheterization of the hepatic veins has provided means for the estimation of hepatic blood flow and splanchnic oxygen consumption of man[3] (1, 2, 3). The cardiac output can be measured in the same study by right heart catheterization. These advances in methodology have made possible investigation of the effects of alteration of the cardiac output on the circulation of the liver.

When the cardiac output is inadequate to meet the total metabolic needs of the body, the various viscera do not participate equally in the reduction of blood supply. This has been shown clearly for the kidney by Merrill (4) where a 50 per cent reduction in cardiac output may be accompanied by as much as an 80 per cent reduction in renal blood flow. Thus, under circumstances where the total cardiac output is below normal, the blood flow of each viscus in turn must be assayed. Most patients with symptoms and signs of chronic heart failure, which prove to be persistent at rest and on a normal diet, have subnormal cardiac outputs

(5) and therefore provide clinical material for study of the effect of an inadequate cardiac output on hepatic blood flow. This report deals with the data which have been collected on a group of 13 of such patients with heart failure of various degrees and of various etiologies. The group by selection contains no patients, such as those with severe anemia or thyrotoxicosis, in whom heart failure develops in the face of a normal or supernormal cardiac output. Studies of the latter type of patient will be reported in a future communication. The results in the patients with cardiac failure are compared with the data on 14 subjects without any or significant disease who were studied under similar experimental conditions. Part of these control data have been presented in a previous publication (3); control cases studied since then have yielded concordant results.

METHODS

The patients with cardiac failure had all fasted for a period of 12 hours or longer prior to the study. Effort was made throughout the procedure to avoid reactions of apprehension on the part of the patients. In a few individuals phenobarbital 0.1 gm. was administered orally about one hour before the beginning of the study. An estimate was made in each subject of the amount of anxiety produced in response to the entire procedure.

The intravenous catheter was introduced as described in a previous publication (3). In most instances an hepatic vein, usually one of the right veins, was catheterized first for the estimation of hepatic blood flow and splanchnic oxygen consumption; upon the completion of these measurements the catheter was withdrawn into the right heart for the collection of the mixed venous blood sample for determination of the cardiac output. The mixed venous sample was obtained in most instances from the pulmonary artery, but in some, for technical reasons, blood from the right ventricle or right atrium near the tricuspid valve was used. In a few patients the cardiac output was measured before the determination of the liver blood flow rather than following it.

The oxygen content of the blood samples was determined by the method of Van Slyke and Neill. Analyses were performed in duplicate and were required to check

[3] The splanchnic area as used in this paper constitutes the liver plus those viscera drained by the portal vein. The splanchnic blood flow is equal to the total hepatic blood flow. The splanchnic oxygen consumption, however, equals the hepatic oxygen consumption plus the oxygen consumption of the various portal viscera.

within 0.1 volume per cent. The oxygen and carbon dioxide contents of the expired air were measured by the method of Haldane. Atrial pressures were recorded by a sensitive Hamilton manometer and mean pressures determined by planimetric integration, the point of zero reference being 5 cm. posterior to the fourth left costochondral junction. Peripheral venous pressures, when measured, were obtained using a saline manometer. Arterial blood samples were withdrawn from a femoral artery through an inlying needle.

The liver blood flow was estimated by the bromsulphalein method of Bradley et al. (2, 3). The bromsulphalein in the serum was determined by means of a Lumetron photelometer at a wave length of 575 mμ. As a rule, 1 ml. of serum was diluted with 10 ml. of 1/50 N sodium hydroxide in 0.9 per cent sodium chloride solution. The color developed under these circumstances is stable for at least one hour. All determinations were made in duplicate. Care was exercised to prevent hemolysis of blood samples prior to the separation of their sera.

Plasma volume determinations were done on every patient upon the completion of the measurements of hepatic blood flow. The ten-minute Evans blue (T-1824) technique was used. The plasma volume is utilized in calculating hepatic blood flow in the presence of a rising or falling arterial serum concentration of bromsulphalein (2). No data are included if the bromsulphalein concentration was changing at a rate exceeding 0.0002 mgm. per ml. of serum per min.

RESULTS

The data on the 13 patients with various kinds of heart failure are given in Tables I and II. Some sets of data are incomplete in one or more items for technical reasons. Most of the patients had only minor or no anxiety during the procedure. Edema and serous effusions were disregarded in the determination of body weight for

TABLE I

Subject	Sex	Diagnosis*	Age	Surface area	Estimated degree of anxiety	Atrial pressure†	Hematocrit	Total blood volume	Blood volume—devia. from predicted normal	Average BSP concentration in arterial serum	Hepatic extraction of BSP	Total removal rate of BSP	BSP clearance
			yrs.	sq. M.	0 = none to 4+ = marked	cm. of water	per cent	ml.	per cent	mgm. per 100 ml.	per cent	mgm. per min.	ml. per min. per sq. M.
J. G.	M	Undet.	63	1.57	1+	0	32.2	5560	+21	1.56	28	2.09	85
L. B.	M	HCVD	52	1.64	2+		52.5	6020	+26	2.54	49	1.94	46
J. S.	M	HCVD	60	1.72	0	15	47.5	5970	+19	2.43		3.57	85
B. S.	F	HCVD	59	1.56	0	(13)	43.5	5870	+47	4.23	23	4.68	72
J. R.	M	RHD-AS	44	2.14	2+	(28)	46.5	7980	+28	3.04	11	2.41	37
T. J.	M	RHD-MI	40	1.68	±	25	45.7	6380	+30	2.36	17	1.32	34
L. S.	F	HCVD	41	1.61	3+		31.4	4500	+12	1.36	19	2.21	101
J. D.	M	Undet.	48	1.75	0	43	44.3	7950	+55	2.03	16	1.32	37
E. W.	F	Undet.	48	1.39	2+	30	38.5	4070	+16	2.22	16	1.99	64
F. B.	M	RHD-MS	50	1.71	1+	(11)	41.4	6060	+21	2.07	35	2.93	82
J. A.	M	SHD-AI	52	1.81	1+	0	43.7	7050	+34	0.81	46	2.01	137
C. T.	M	MYO. INF.	61	1.63	0	0	49.2	6870	+44	0.78	42	1.89	149
E. B.	F	RHD-MS	41	1.55	2+	16	41.3	4820	+23	7.69	8	3.51	30
Cardiacs: mean									29	2.55	26	2.45	74
st. dev.‡									13	1.79	14	0.97	45
S. E.§									3.6	0.50	4.0	0.27	13
Controls: mean									3	1.61	50	5.82	233
st. dev.‡									15	0.75	15	1.47	71
S. E.§									4.3	0.20	4.0	0.39	19

* HCVD   hypertensive cardio-vascular disease
 RHD   rheumatic heart disease
 SHD   syphilitic heart disease
 MYO. INF. myocardial infarction, old
 Undet.   heart failure of undetermined etiology
† Figures in parentheses are antecubital venous pressures.

AI   aortic insufficiency
MS   mitral stenosis
MI   mitral insufficiency
AS   aortic stenosis

‡ standard deviation, $s = \sqrt{\dfrac{\Sigma X^2 - \dfrac{(\Sigma X)^2}{n}}{n-1}}$.

§ standard error $= \dfrac{s}{\sqrt{n}}$.

TABLE II

Subject	Oxygen consumption	Metabolic rate—devia. from normal basal	Oxygen satur. of arterial blood	Oxygen content of arterial blood	Arterial-mixed venous oxygen diff.	Arterial-hepatic venous oxygen diff.	Cardiac index	Hepatic blood flow	Hepatic blood flow as percent. of cardiac output	Splanchnic oxygen consump.	Splanchnic oxygen consump. as percent. of total oxygen consump.
	ml. per min. per sq. M.	per cent	per cent	vol. per cent	vol. per cent	vol. per cent	liters per min. per sq. M.	ml. per min. per sq. M.	per cent	ml. per min. per sq. M.	per cent
J. G.	166	+33	88	12.0	6.1	8.8	2.7	460	17	41	25
L. B.	185	+44	90	19.6	8.4	12.8	2.2	200	9	26	14
J. S.	155	+25	97	19.1	10.2	11.0	1.5				
B. S.	160	+36	89	16.9	8.2	9.1	2.0	530	27	48	30
J. R.	206	+56	92	16.7	7.7	9.6	2.7	680	25	65	32
T. J.	161	+22	89	16.1	7.7	9.2	2.1	370	18	34	21
L. S.	124	+ 2	92	9.6	5.9	5.9	2.1	800	38	47	38
J. D.	117	−13	89	16.0	8.0	8.8	1.5	420	29	37	32
E. W.	162	+34	96	14.9	6.5	7.7	2.5	650	26	50	31
F. B.	110	−14	96	18.0	7.2	8.0	1.5	390	25	31	29
J. A.	140	+ 9	96	17.0	5.3	6.2	2.6	540	21	33	24
C. T.	148	+19	97	19.7	5.5	5.5	2.7	680	25	37	25
E. B.	131	+ 7	92	17.6	6.0	6.6	2.2	700	32	46	35
Cardiacs:											
mean	151	20			7.1	8.4	2.2	535	24	41	28
st. dev.*	27	21			1.4	2.0	0.5	170	7	11	7
S. E.†	7.5	5.8			0.4	0.6	0.14	49	2.0	3.2	2.0
Controls:											
mean	153	13			3.9	4.5	4.1	850	20	38	24
st. dev.*	16	10			0.5	0.8	0.5	170	4	10	8
S. E.†	5.3	3.3			0.2	0.2	0.17	45	1.3	2.9	2.7

* standard deviation.   † standard error.

the estimation of surface area. However, only two patients (L. S. and J. D.) had edema or effusions of such degree as might significantly affect calculations.

As compared to normal individuals (2, 3), relatively higher concentrations of bromsulphalein were obtained in cardiacs from a given dose of the dye per minute. The 13 patients obtained a mean concentration in arterial serum of 2.55 ± 1.79 mgm.[4] per 100 ml. from an average infusion rate of 2.5 ± 0.97 mgm. per min. The 12 control subjects showed a mean arterial concentration of 1.61 ± 0.75 mgm. per 100 ml. from an average infusion rate of 5.82 ± 1.47 mgm. per min. The percentage extraction of bromsulphalein ([arterial serum concentration − hepatic venous serum concentration] ÷ [arterial serum concentration]) varied widely but in general was again subnormal in the patients with heart failure, they

[4] Throughout this paper in the expression of ± values, the standard deviation, s, is used, where

$$s = \sqrt{\frac{\Sigma X^2 - \frac{(\Sigma X)^2}{n}}{n-1}}$$

averaging 26 per cent extraction as compared to 50 per cent for the controls. As the percentage extraction varies inversely with the arterial concentration, the control subjects with their lower average arterial concentration of bromsulphalein would be expected to have a higher percentage of extraction. However, it seems that the difference in arterial concentration does not account for the difference in extraction between the controls and the cardiacs. From our data, the control subjects at an arterial concentration of 2.55 mgm. per 100 ml. (the average arterial concentration for the group with heart failure) would have a predicted extraction of 37 per cent. This is to be compared with the 26 per cent extraction found in the heart failure group at that average arterial concentration.

The data on the removal of bromsulphalein by the liver of heart failure, in comparison with the normal liver, are better expressed in terms of bromsulphalein clearance ([total removal rate of BSP in mgm. per min. per sq. M.] ÷ [concentration of BSP in mgm. per ml. of arterial serum] = ml. of arterial serum cleared completely of

BSP per min. per sq. M.). In our series of 14 subjects without significant disease the bromsulphalein clearance ranged from 152 to 365 ml. per minute per sq. M. with a mean of 233 ± 71 ml. The cardiac patients gave clearance of from 30 to 149 ml. per min. per sq. M. for a mean of 74 ± 45 ml.

It has been shown (2) that bromsulphalein in the blood stream is protein-bound and that dye will be lost into the urine in the presence of proteinuria. Only three of the present series of cardiac patients (B. S., J. R., and J. D.) had proteinuria by the usual qualitative tests at the time of the measurement of hepatic blood flows. These proteinurias were all of moderate degree and it does not seem that the amount of dye lost into the urine would seriously affect the determinations.

Oxygen consumptions in the group with cardiac failure ranged from 110 to 206 ml. per min. per sq. M. of body surface. The corresponding metabolic rates varied between minus 13 and plus 56 per cent of the normal basal with a mean of plus 20 per cent. In this respect the patients with cardiac failure did not differ significantly from the controls who had a mean metabolic rate under the conditions of this study of plus 13 per cent of the normal basal.

None of the patients demonstrated a marked degree of unsaturation of the arterial blood for oxygen. The patients with more severe degrees of heart failure, however, did have moderate unsaturation.

The values for hematocrit and oxygen content of arterial blood in Tables I and II show that only two patients (J. G. and L. S.) had significant anemia. It is unlikely that the degree of anemia in subject J. G. played any great part in altering his circulatory measurements, as Brannon et al. (6) have found that depression of the hemoglobin to below 7 g. per 100 ml. is necessary before one gets a significant effect on total cardiac output. In the case of L. S., however, it cannot be assumed that her anemia may not have affected the data. It is noted that L. S. had the highest hepatic blood flow of any patient with heart failure. It has been observed (7) that severe degrees of anemia in the absence of heart failure are associated with elevated hepatic blood flows, which

in general are proportionate to the elevation in total cardiac outputs due to the anemia.

All patients with cardiac failure had elevated arterial-mixed venous oxygen differences, the values ranging from 5.3 to 10.2 volumes per cent and the mean being 7.1 ± 1.4 volumes per cent. The control subjects by comparison gave differences of from 3.3 to 4.7 volumes per cent with a mean of 3.9 ± 0.5 volumes per cent. Arterial-hepatic venous oxygen differences corresponded, the cardiacs showing elevated values varying from 5.5 to 12.8 volumes per cent for a mean of 8.4 ± 2.0 volumes per cent. The control group had differences of from 3.8 to 5.8 volumes per cent with a mean of 4.5 ± 0.8 volumes per cent. Thus all the patients with cardiac failure, with the exception of one (C. T.) who had only mild failure, showed decidedly elevated arterial-hepatic venous differences. In the cardiacs, as in normal persons (3), the arterial-hepatic venous difference in general exceeds the arterial-mixed venous difference by a small amount.

Every patient with heart failure had a subnormal cardiac index (cardiac output in liters per min. per sq. M. of body surface), particularly when it is remembered that the nature and duration of the circulatory measurements reported here tend to increase the circulation somewhat above the basal. The control group had cardiac indices between 3.3 and 4.9, the patients with heart failure indices from 1.5 to 2.7. The means of these determinations are for the controls 4.1 ± 0.5, and for the cardiacs 2.2 ± 0.5.

The estimated hepatic blood flows vary over a considerable range in both the control and heart failure groups. This is probably explainable, at least in part, when it is remembered that the flow through only one portion of the liver is being determined, and that the flows as estimated from several areas of the same liver under the same conditions may show considerable variation (2). Furthermore, several of the patients in the heart failure group had low extraction percentages for bromsulphalein. Under this circumstance, the usual technical errors in measuring the dye concentrations in arterial and hepatic venous bloods have a considerably greater influence on the calculated hepatic blood flow. The hepatic blood flows for the patients with heart failure ranged from 200 to 800 ml. per min. per sq. M. of body

surface; the controls gave values of from 600 to 1160 ml. per min. per sq. M. The average values for the cardiacs are 535 ± 170 ml. and for the controls 850 ± 170 ml. This overall difference is statistically significant ($p < 0.01$). When the values for cardiac output and hepatic blood flow in each patient are correlated, it is found that, in both the control group and the series with heart failure, the hepatic blood flow represents a fairly constant percentage of the cardiac output. Among the cardiacs the hepatic blood flow made up on the average 24 ± 7 per cent of the cardiac output; in the controls it comprised 20 ± 4 per cent. Three patients with heart failure show considerable disproportion between the two values. Patient L. B., who had severe heart failure at the time of study, had a very low hepatic blood flow and at the same time an extreme hepatic arterio-venous oxygen difference. He is the only patient in our experience to manifest a much more marked reduction in liver blood flow than in cardiac output. Whether this does occur with any frequency in severe heart failure can only be told by the study of more patients. Patient L. S., with an arterial oxygen content of only 9.6 volumes per cent, had a high liver blood flow in proportion to her cardiac output. One can only conjecture concerning the effect of her considerable anemia in explaining this discrepancy. Patient E. B. also had an hepatic blood flow which was proportionately large for her cardiac output. E. B. had a chronically enlarged liver, and palpable spleen and, as can be seen in Table I, a marked retention of bromsulphalein, 7.69 mg. per 100 ml. of arterial serum from a moderate dose of 3.51 mg. of dye per minute. At her extraction percentage of bromsulphalein of only 8 per cent, the measurement of liver blood flow is subject to considerable error. Thus the figure of 700 ml. per min. per sq. M. is open to question. It seems likely that E. B. had chronic liver disease out of proportion to that usually encountered in congestive heart failure; possibly this liver disease may have locally affected hepatic blood flow (8). In only one other patient in the series, (J. D.), were there clinical indications of the existence of liver disease which was out of proportion to the degree and duration of the cardiac decompensation.

The splanchnic oxygen consumption (hepatic blood flow × hepatic A-V oxygen difference) in the patients with heart failure ranged from 26 to 65 ml. per min. per sq. M. as compared with the control range of 26 to 53 ml. The corresponding mean values are, for the cardiacs, 41 ± 11 ml. and, for the controls, 38 ± 10 ml. When the splanchnic oxygen consumptions for the two groups are expressed as percentages of the respective total oxygen consumptions, again essentially identical values are obtained, 28 ± 7 per cent for the cardiacs and 24 ± 8 per cent for the controls.

DISCUSSION

Before discussing what implications can be drawn from the above data, several precautions must be sounded in connection with the interpretation of such data. Firstly, circulatory measurements as have been reported in this paper have such inherent variations as to show quite wide discrepancies even among similar individuals (9). Secondly, any measurements made by the procedure of catheterization of one of several hepatic veins are measurements by sampling and again considerable variation among similar subjects is to be expected. Lastly, our studies by their very nature could not be performed under consistently basal conditions, the element of anxiety to the procedure being particularly variable. It must be stressed therefore, that comparison of any one case with another might well lead to erroneous conclusions, and that only groups of cases should be compared. Thus the mean values reported here are the pertinent figures.
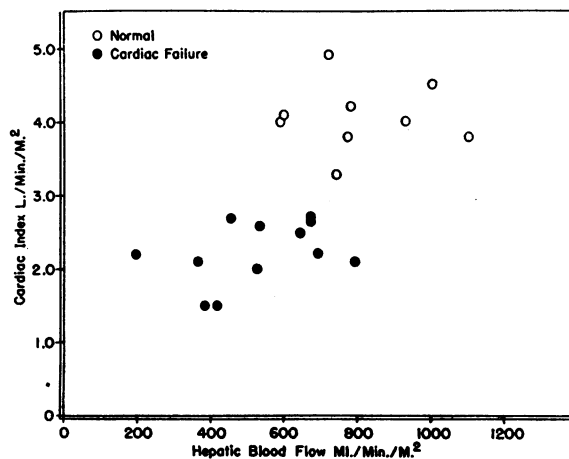

FIG. 1. THE RELATIONSHIP OF HEPATIC BLOOD FLOW TO CARDIAC OUTPUT IN CONTROL SUBJECTS AND PATIENTS WITH HEART FAILURE
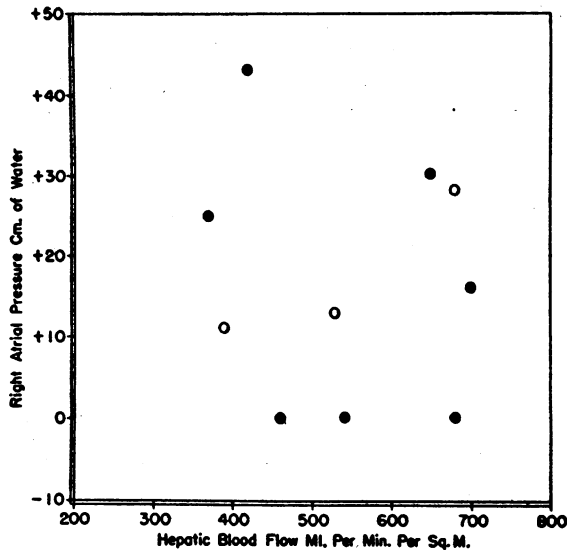
FIG. 2. HEPATIC BLOOD FLOW AT VARIOUS LEVELS OF RIGHT ATRIAL PRESSURES (BLACK DOTS) AND PERIPHERAL VENOUS PRESSURE (OPEN CIRCLES)

The above data indicate that in cardiac failure there is a decrease in hepatic blood flow, and that this decrease is roughly proportionate to the decrease in total cardiac output (Figure 1). Mechanisms by which this decrease in flow might be brought about are (a) a decreased inflow of blood due to a decrease in total cardiac output, (b) vasoconstriction of the afferent vessels to the liver, similar to that presumed for the kidney in heart failure (4), (c) an increased resistance in the hepatic capillary bed as from edema or other pathologic changes in the liver itself, and (d) impairment to outflow of blood from the liver because of an elevation of hepatic venous pressure as a part of the general elevation of venous pressure found in heart failure. The finding that the mean hepatic blood flow in heart failure expressed as a percentage of the total cardiac output, 24 ± 7 per cent, is the same as for the control subjects, 20 ± 4 per cent, is of particular significance in that it indicates that there is no selective reduction of the liver blood flow. Thus possibilities (b) and (c) above are eliminated as accounting, by themselves at least, for the reduced flow. Examination of Tables I and II and Figure 2 shows that there is poor correlation between the level of hepatic blood flow and the corresponding right atrial or antecubital venous pressure. For example, patient E. W. had an hepatic blood flow within the lower

normal range, 650 ml. per min. per sq. M., in the presence of a right atrial pressure of 30 cm. of water. Several patients do show reduced hepatic blood flows in conjunction with elevated pressures. However, both phenomena may be parallel manifestations of severe heart failure and are not necessarily directly interrelated. It seems, then, that the liver blood flow in heart failure is governed, in large part at least, by the level of the cardiac output. The liver receives its usual percentage of the total output, in this respect differing strikingly from the kidney which undergoes a marked and disproportionate reduction in blood flow.

It has been considered that the so-called central necrosis of liver lobules found in heart failure is a manifestation of an increased hepatic venous pressure. The finding of similar lesions in severe anemia and anoxemia (10) without elevation of venous pressure throws doubt on the above correlation. Our finding of a marked unsaturation of hepatic venous blood in cardiac failure is in agreement with the hypothesis that central necrosis may be related to a low oxygen concentration in the blood bathing those liver cells which lie farthest from the afferent blood supply.

The individual in heart failure with a reduced cardiac output maintains a normal body oxygen consumption by an increased extraction of oxygen from that blood supplied to the various viscera, that is, by an increased arterial-mixed venous oxygen difference. This is demonstrated for our
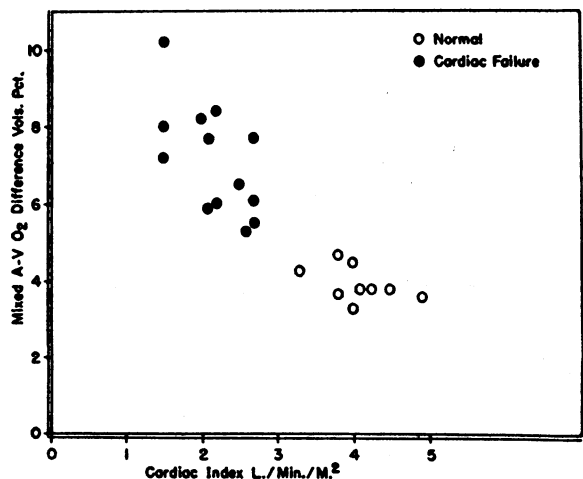

FIG. 3. RELATIONSHIP OF MIXED ARTERIO-VENOUS OXYGEN DIFFERENCE TO THE CARDIAC OUTPUT IN CONTROL SUBJECTS AND PATIENTS WITH HEART FAILURE

group of patients with cardiac failure in Figure 3. The moderate reduction in blood supply which the liver suffers in heart failure is accompanied by a similar compensatory increase in extraction of oxygen from that blood supplied to it (Figure 4). This increase in arterial-hepatic venous oxygen difference allows the body to maintain, *under conditions of rest and fasting,* a normal splanchnic oxygen consumption. It seems reasonable, under the conditions of our study, that the splanchnic oxygen consumption, which is the oxygen
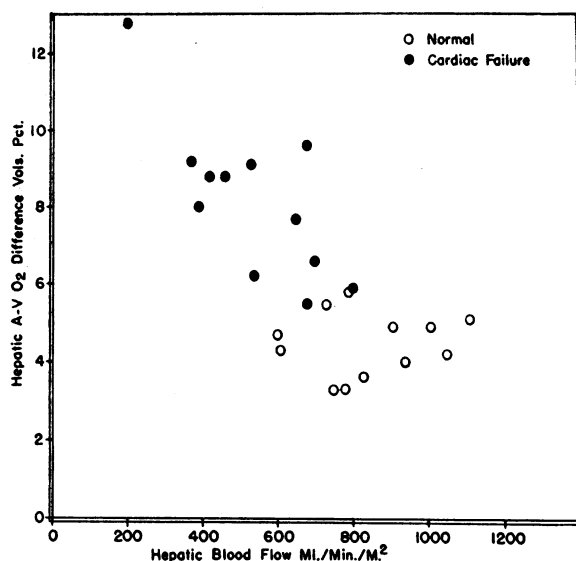

FIG. 4. RELATIONSHIP OF HEPATIC ARTERIO-VENOUS OXYGEN DIFFERENCE TO HEPATIC BLOOD FLOW IN CONTROL SUBJECTS AND PATIENTS WITH CARDIAC FAILURE

consumption of the gut, pancreas and spleen as well as the liver, is reflective of the true hepatic oxygen consumption. The finding of a normal splanchnic oxygen consumption, then, is in agreement with the clinical observations that the liver of heart failure maintains most of its function quite well, *e.g.,* the formation of plasma proteins. One awaits with interest, however, studies as to the effects of various stresses, such as exercise and the intake of foodstuffs, on the liver blood flow and oxygen consumption. Several of our patients (*e.g.,* J. S., J. B.), with cardiac failure had such large arterial-hepatic venous oxygen differences that little further increase in splanchnic oxygen consumption could be accomplished without an increase in blood flow. Since many in-

dividuals with severe heart failure are incapable of an increase in cardiac output so as to be able to augment hepatic blood flow (11), an increase in the latter could only be provided, if at all, by a diversion of blood from some other portion of the body. The liver of heart failure, then, when under stress, might well find its supply of oxygen and other metabolites inadequate to meet normal metabolic demands.

SUMMARY

1. The hepatic blood flow in a group of 13 patients with cardiac failure, as estimated by the bromsulphalein method, varied from 200 to 800 ml. per min. per sq. M. of body surface with a mean of 535 ± 170 ml. The hepatic blood flows in a group of 14 controls studied under similar circumstances ranged between 600 to 1160 ml. for a mean of 850 ± 170 ml. Statistical analysis shows the difference between the means of the two groups to be highly significant.

2. The mean cardiac indices for the two groups were 2.2 for the cardiacs and 4.1 for the controls. The hepatic blood flows expressed as percentages of the total cardiac outputs averaged 24 per cent for the cardiacs and 20 per cent for the controls.

3. The arterial-hepatic venous oxygen differences for the patients with heart failure varied from 5.5 to 12.8 volumes per cent, as compared to a range of 3.3 to 5.8 volumes per cent in the controls. Mean values were 8.4 volumes per cent for the cardiacs and 4.5 volumes per cent for the controls.

4. The average splanchnic oxygen consumption in heart failure was 41 ml. and for the controls 38 ml. per min. per sq. M. of body surface. These values represent 28 per cent and 24 per cent, respectively, of the total oxygen consumption.

CONCLUSIONS

1. The hepatic blood flow in cardiac failure is moderately reduced, and in proportion to the reduction in total cardiac output.

2. This reduction in liver blood flow is compensated by an increase in arterial-hepatic venous oxygen difference so as to provide, under conditions of rest and fasting, a normal splanchnic oxygen consumption.

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