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PARTITION OF THE VENOUS RETURN TO THE HEART

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The experiments described in this paper were undertaken to supplement knowledge about the relative rates of blood flow in the superior and inferior venae cavae. During the investigations some of the factors which modified the rates of flow in the venae cavae were examined and the rates of flow in some other veins were measured.

METHODS

Experiments were performed on dogs (9–21 kg) anaesthetized with chloralose (0·1 g/kg intravenously). Briefly, the principle of the method was to feed blood into the right atrium at a controlled and measured rate, and to measure the venous return flow in the superior vena cava (SVC), inferior vena cava (IVC) or other veins. The arrangement of the apparatus and preparation is shown in Fig. 1.

Blood was drained from the veins by right-angled glass cannulae connected to outflow tubes which led to the flow recorders. The openings of these outflow tubes were set at the level of entry of the SVC into the right atrium.

The flow recorders (Fig. 2) were a modification of the type described by Stephenson (1949). In the original form described by Stephenson, there was a back pressure into the vein as the flow increased and there was also a tendency for the calibration of the recorder to be upset because of a gradual gas exchange between the venous blood and the air in the flow recorder. These two faults were eliminated by introducing an air break between the outflow tube and the recorder and by arranging a by-pass arm so that at any time the uninterrupted venous flow could be diverted from the recorder while the zero position was being re-set. By using tubes of various diameters at the bottom of the recorder the same recorder could be used for different ranges of flow. The ranges usually employed for different veins were: IVC, 250–1300 ml./min; SVC, 150–800 ml./min; femoral and jugular veins, 10–120 ml./min.

After passing through the flow recorders the blood was collected into a Perspex trough and returned to the main reservoir. This reservoir was fitted with a level gauge connected to a volume recorder so that alterations in the distribution of blood between the reservoir and the animal could be measured.

Blood was pumped from the reservoir through a flow recorder to an input reservoir from which the heart was fed. The height of the blood in the input reservoir was kept constant by an overflow tube, and the input to the heart was regulated by a screw clip on the tube leading to the right atrium. The blood pump was a double Dale-Schuster pump with a valve chamber modified so that an output of up to 2–3 l./min could be obtained (Coleridge & Hemingway, 1953).

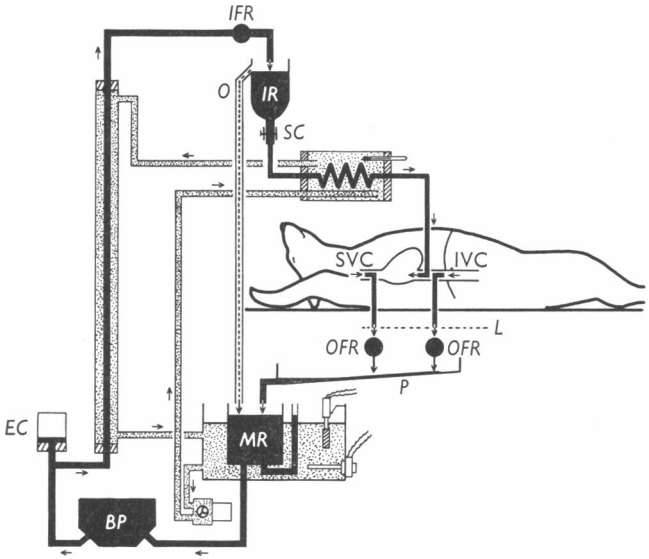

Fig. 1. Apparatus and preparation. Solid black indicates blood in external circuit; stippling represents water-jackets fed by a pump from the water-bath. *OFR*, outflow recorders; dotted line *L*, openings of outflow tubes set at level of right atrium; *P*, Perspex trough; *MR*, main reservoir; *BP*, blood pump; *EC*, elasticity chamber; *IFR*, inflow recorder; *IR*, input reservoir; *SC*, screw clip controlling constant level in reservoir; *O*, trickle overflow back to the main reservoir.

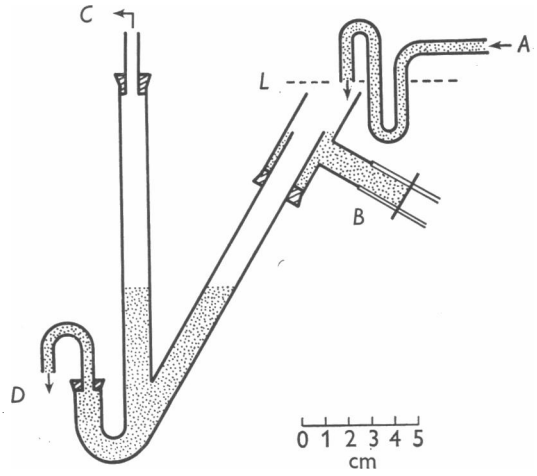

Fig. 2. Modified Stephenson flow recorder. *A*, flow from vein, *L*, opening of tube set at level of entry of SVC into the right atrium; *B*, by-pass with clip permitting diversion of venous outflow while zero position being re-set; *C*, connexion to volume recorder; *D*, outlet tube.

At the beginning of each experiment the perfusion apparatus was primed with heparinized blood (5000 i.u./l. Pularin, Evans Medical Supplies, Ltd.). The preparation was established as follows. The trachea was cannulated and the animal ventilated with a Starling 'Ideal' pump. The chest was opened by splitting the sternum or by resecting the 5th right rib. The SVC was dissected free distally for 2–3 cm from the pericardium, and the IVC was cleared between the diaphragm and the pericardium. Care was taken to avoid damaging nerves in the neighbourhood of the veins. Heparin was injected intravenously in an initial dose of 500 i.u./kg and a maintenance dose of 100 i.u./kg was given every 30 min.

Cannulation of the venae cavae and the establishment of an inflow to the heart were undertaken in three stages, with as little interference as possible with venous flow. In *Stage I* the IVC was ligated and cannulated just above the diaphragm and the IVC flow was directed to a flow recorder. Blood was pumped back to the animal through an external jugular vein at a rate which kept the level of the main reservoir constant. To prevent venous congestion during cannulation of the IVC, the blood returning to the IVC was drained temporarily to the main reservoir, through polythene tubing introduced through a femoral vein, and was then pumped back to the animal via the external jugular vein. In *Stage II* a cannula was tied into the heart end of the IVC and connected to the input reservoir. The input to the external jugular vein was then switched to the input reservoir and the screw clip on the tube leading to the heart was adjusted to keep the input reservoir just overflowing. In *Stage III* a cannula pointing headwards was inserted into the SVC just above the point of entry of the azygos vein, which was tied. As soon as the SVC flow was established through a recorder, the pump was adjusted to keep the volume of blood in the main reservoir constant. No attempt was made at any time to adjust the cardiac inflow to set the arterial blood pressure to a particular height.

In three animals the total hepatic flow was measured by cannulating the IVC immediately above the diaphragm; a cannula immediately below the diaphragm collected the flow from the trunk, the kidneys and the hind limbs. In other experiments flow in an external jugular, a femoral or the azygos vein was also measured.

Arterial blood pressure was recorded with a Hg manometer from a T cannula in the common carotid artery. Right atrial pressure was recorded with a saline manometer.

RESULTS

Measurements in the initial period

After a preparation had been established the output of the pump was adjusted so that the animal was neither gaining blood from, nor losing blood to, the main reservoir. In twenty-four experiments under these conditions the sum of the separate venous outflows (which included neither coronary nor bronchial flows) ranged from 710 to 1455 ml./min (mean 1105). Or expressed as l./min/m^2, for convenience of comparison with the findings of others, the total outflows ranged from 0·91 to 2·62 l./min/m^2 (mean 1·85; S.D. 0·47). The size of the total venous return appeared to be determined, to some extent, by the skill and experience gained in making the preparations, because highest values were obtained towards the ends of the series of experiments with both methods of thoracotomy.

The average initial mean arterial blood pressure in the twenty-four preparations was 96 mm Hg (range 45–155; S.D. $\pm$ 29·4).

The SVC flow in the different preparations ranged between 8 and 45 ml./min/kg (mean 27); the IVC flow varied from 29 to 93 ml./min/kg (mean 62).

The SVC given as a percentage of the total venous outflow ranged between 19·8 and 47·2% with a mean of 30·4% (S.D. 7·1%). The measurements of flow in the left femoral vein, the right external jugular vein and the azygos vein are given in Table 1.

TABLE 1. Summary of rates of flow from different veins

Vein	No. of preparations	Flow (ml./min/kg body wt.)	Flow as % of total SVC flow	Flow as % of total IVC flow	Flow as % of total venous flow
SVC	24	27·0 (8–45)	—	—	30·4
Total IVC	24	62·0 (29–93)	—	—	69·6
Hepatic	3	43·9 (37·1–50·0)	—	58·4	43·1
Abdominal IVC	3	31·3 (25·6–37·1)	—	41·6	30·8
Azygos	1	3·1	—	—	3·3
Ext. jugular (one)	2	5·5	17·5	—	5·3
Femoral (one)	9	2·3 (0·8–5·7)	—	3·5	2·4

TABLE 2. Measurements of hepatic, SVC and IVC flows in three preparations. The figures are the means of the various measurements and the ranges are given within the brackets

Preparation ...	1	2	3
Weight (kg)	11·5	12·0	10·5
Number of sets of measurements	8	7	8
Mean art. B.P. (mm Hg)	118 (100–135)	133 (117–140)	107 (100–120)
Total venous flow (ml./min)	1193 (1100–1385)	1183 (1150–1240)	1088 (1055–1130)
SVC flow (ml./min)	332 (260–460)	282 (260–300)	293 (245–340)
Hepatic flow (ml./min)	505 (450–575)	547 (525–575)	444 (390–490)
Abd. IVC flow (ml./min)	356 (295–395)	354 (330–380)	351 (290–390)
Total IVC flow (ml./min)	861 (835–925)	901 (880–940)	795 (755–865)

Blood flow through the liver

Three preparations were set up in which hepatic flow was measured and observations were made in each preparation at intervals over a period of about 2 hr. A summary of the observations is given in Tables 1 and 2. The total venous return was maintained within 5% of the mean value for most of the observations. On average, the hepatic flow was 43·1% of the total venous flow and 58·4% of the total IVC flow. Expressed in terms of body weight the mean hepatic flow was 43·9 ml./min/kg of body weight; or, taking the weight of the liver as 2·35% of body weight (Brody & Kibler, 1941), 188 ml./min/100 g liver.

The relationship of the size of the total venous return and of the arterial blood pressure to the relative rates of flow in the SVC and the IVC

No clear relationship was found between the size of the total venous return and the relative rates of flow in the two venae cavae in the initial period. However, the proportion of blood passing through the SVC appeared to vary inversely with the height of the arterial blood pressure in the initial period, as is shown in Fig. 3. Summarizing the measurements from all the experiments: in twenty-four in which initial arterial blood pressure ranged from 45 to

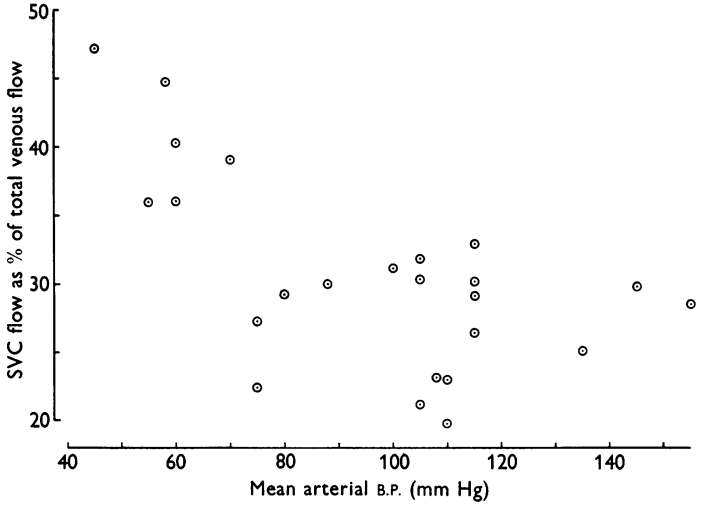

Fig. 3. Relationship between the mean arterial blood pressure and the proportion of the total venous return flowing through the SVC during the initial periods of twenty-four experiments.

155 mm Hg, the proportion of total flow which passed through the SVC varied from 19·8 to 47·2% (mean 30·4). In six experiments in which the arterial blood pressure was 70 mm Hg or lower, the SVC flow was 36·0–47·2% (mean 40·5). In eighteen experiments in which initial arterial blood pressure was higher than 70 mm Hg, the SVC flow ranged from 19·8 to 33·0 % (mean 27·3) of the total.

In a number of experiments (fifty-one sets of observations in thirteen preparations), after a steady state had been obtained initially, the effects produced by altering the rate of inflow to the heart were observed. Mean arterial blood pressure always rose when the cardiac inflow was increased. Hence, in view of the correlation between arterial pressure and the relative caval flows already described, it was expected that the changes in heart output might also be associated with alterations in the ratio SVC:IVC flow. However, in about one half of the experiments the ratio did not noticeably change. But in the other half, the proportion of the total flow passing through the SVC

decreased as the output was increased and the measurements showing the relationship between SVC flow and the total venous outflow in four such experiments are plotted in Fig. 4.

There were other circumstances in which the ratio SVC:IVC flow was inversely related to the arterial blood pressure. When observations were continued for some time after the initial period, many preparations showed a progressive deterioration in their general condition, characterized by a fall in mean arterial blood pressure and a gradual uptake of perfusate from the main

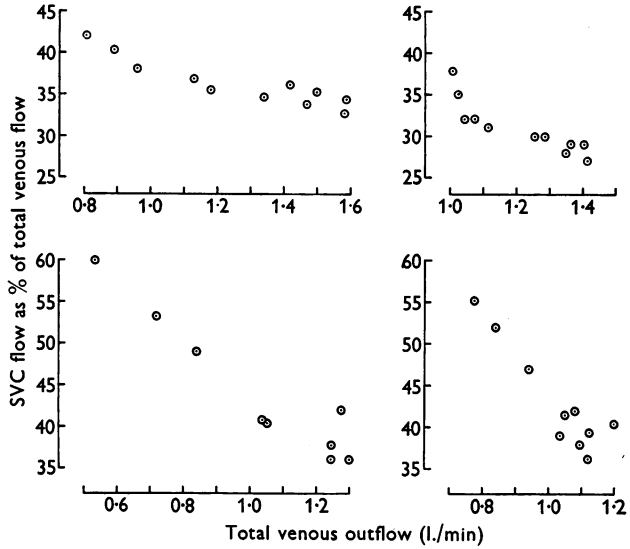

Fig. 4. Measurements from four experiments to show alteration in the SVC flow (as a percentage of total venous flow) as inflow to the heart was varied.

reservoir. Such changes are well known to occur in this type of preparation (in the dog, Wégria, Rojas & Wiggers, 1943; Huggins & Smith, 1952; Daly, Eggleton, Hebb, Linzell & Trowell, 1954: in the cat, Grundy & Howarth, 1957). But in addition, a striking feature of these changes was a diversion of blood flow to the head end of the animal as the arterial blood pressure fell. This sequence of events is illustrated by the results from one experiment in which, over a period of 35 min, mean arterial blood pressure fell from 105 to 40 mm Hg and 780 ml. of blood was transferred from the reservoir to the animal. The fall in blood pressure probably occurred as a result of a decrease in the total peripheral resistance, because during this period the input to the heart was maintained constant for as long as possible at 800 ml./min. At the end of the period the proportion of the total venous return passing through the SVC had risen from 21 to 51%.

The observations from four similar experiments in which arterial blood pressure fell progressively, although a constant cardiac inflow was maintained,

are shown in Fig. 5. The partition of the venous return with the deviation in favour of the SVC flow is shown at different arterial pressures as they occurred during the course of each experiment.

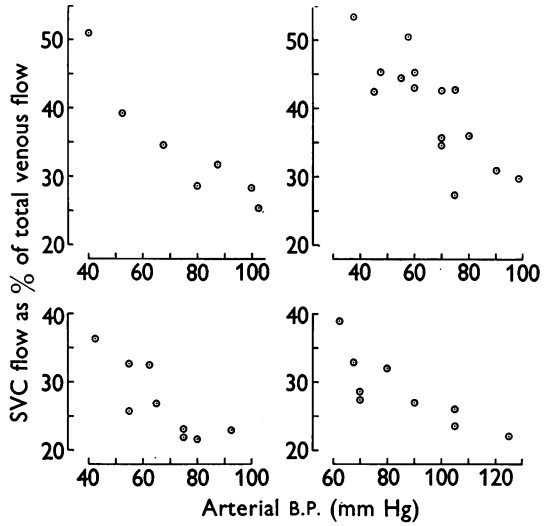

Fig. 5. Results from four preparations in which arterial blood pressure gradually fell and the proportion of blood returning via the SVC gradually increased. Cardiac inflow maintained within 10% of the original value in each experiment.

Effects produced by anoxia on the distribution of caval blood flows

Preparations which were being ventilated with room air, or with oxygen, were changed to a mixture of equal parts of nitrogen and air or, more usually, to nitrogen. Periods of anoxia ranging from 40 sec to 6 min 40 sec were induced in fifteen experiments on nine dogs before resuming ventilation with air or oxygen.

If the period of anoxia were prolonged the heart dilated, the right atrial pressure rose and the inflow to the heart could not be kept at the original rate. But in the majority of the experiments it was possible to maintain the cardiac inflow at the original rate for 1–2 min after inducing anoxia, by adjustment of the clip on the inflow tube.

In general, for as long as the cardiac inflow was maintained, the effects produced included a rise in arterial blood pressure, an increase, both absolute and proportional, in SVC flow, and a decrease in IVC flow. Although the heart accelerated in most cases during the first few seconds of anoxia, bradycardia usually ensued when arterial pressure began to rise. The resistance of the region drained by the IVC increased in all the experiments and of that drained by the SVC in nine of the experiments.

The flow through the SVC was proportionately increased in all the experi-

ments; but some of them were more satisfactory than others for analysis of the redistribution of the caval flows because the cardiac inflow was held steady within 10% of the original rate for the whole, or a considerable part, of the period of anoxia. In these experiments, depicted in Fig. 6, it can be seen that the alterations in SVC flow in different experiments varied considerably. In one case the SVC flow changed only from 42 to 44% of the total flow, but in two others it changed from 23 to 47% and from 38 to 64% respectively.

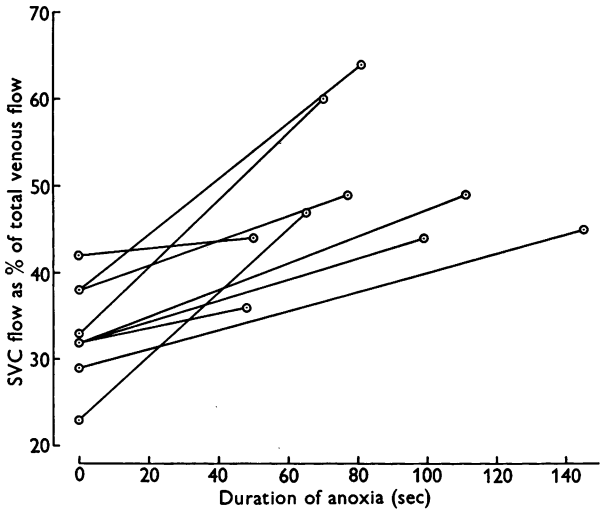

Fig. 6. Showing the effect of anoxia on the proportion of the total venous return flowing through the SVC in experiments in which cardiac inflow was held steady within 10% of the original rate.

The flow through the IVC was correspondingly reduced and in some of the experiments (not included in Fig. 6) in which the period of anoxia was prolonged and the cardiac inflow had to be reduced the effects produced on the IVC flow were dramatic. In some cases, as is shown in Table 3, the IVC flow was reduced to zero. (The IVC flow recorder did not record accurately rates of flow of less than 250 ml./min, but the outflow through the IVC recorder was seen to have stopped completely.) In both experiments there was a large relative increase in the SVC flow and there was also an increase in femoral vein flow.

Although as anoxia progressed there was always, eventually, a decrease in the IVC flow, in the initial stages of seven of the experiments there was a transient increase in the IVC flow (Fig. 7) of up to 1 min duration without any corresponding reduction in SVC flow; so that, for a short period, total outflow exceeded total inflow. In the experiment depicted in Fig. 7 the increase in the rate of flow gave an additional net outflow of 40–50 ml. over what might

have been expected from the rate of flow immediately before anoxia was induced. This blood represented, of course, a diminution in the total volume contained in the animal, and an increase in the volume held in the main reservoir.

TABLE 3. The effects produced by anoxia on SVC, IVC and femoral vein flows

Expt.	Duration of anoxia	Mean art B.P. (mm Hg)	Venous flow (ml./min) SVC	IVC	Femoral	Total	SVC flow (% of total flow)	Femoral flow (% of total flow)
1	Control	92	300	595	35	930	32	3·8
	1 min 39 sec	200	400	465	50	915	44	5·5
	3 min 16 sec	43	290	0	55	345	84	16·0
2	Control	130	450	730	10	1190	38	0·8
	1 min 17 sec	183	555	515	70	1140	49	6·1
	4 min	190	515	200	45	760	68	5·9
	6 min 45 sec	181	420	0	50	470	89	11·0

Flow measured from one femoral vein; other femoral vein used to record pressure in IVC.

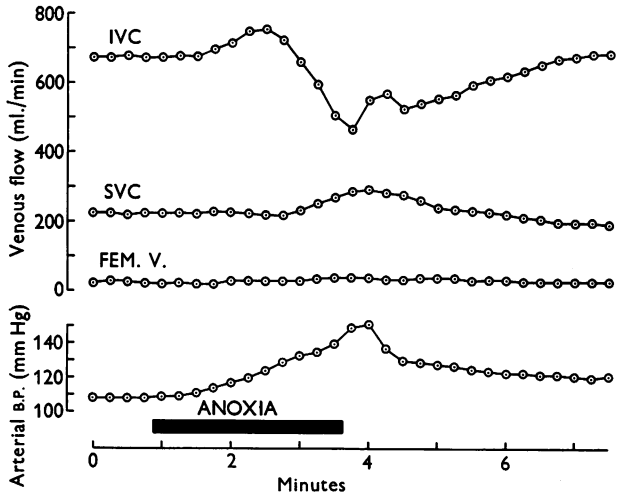

Fig. 7. Dog, 10 kg. Effect of anoxia on SVC, IVC and femoral vein flows and arterial blood pressure. Note particularly the temporary increase in IVC flow in the early stages of anoxia. During this period the reservoir gained 40 ml. of blood from the animal.

Effects produced on venous flow by occlusion of the common carotid arteries

Thirteen experiments were performed to determine whether any alterations in the actual or relative caval flows occurred when both common carotid arteries were occluded and the inflow to the heart from the pump was kept constant. In all the experiments the flow through the SVC was reduced (mean reduction, 13% of the original rate) and the IVC flow was increased. But there was no clear-cut evidence that total venous return was altered, even temporarily, by carotid occlusion; in six experiments out of the thirteen there

was a mean increase of 30 ml./min (about 2·5% of the original total flow) and in seven experiments there was a mean decrease of 12 ml./min (about 1%).

The results of three experiments in which the hepatic and abdominal IVC flows were separately collected and measured are given in Table 4. Although the SVC flow was reduced when the carotid arteries were occluded the total venous return was practically unchanged and the increase in the IVC flow was confined to the regions which were drained by the hepatic veins. Although the total IVC flow increased, the peripheral resistance of the region drained by the IVC also increased in eleven of the experiments.

TABLE 4. Effects on venous flow produced by occlusion of the common carotid arteries

Expt.		Mean art. B.P. (mm Hg)	Venous flow (ml./min) SVC	Hepatic	Abd. IVC	Total IVC	Total venous flow
1	Control	116	350	490	390	880	1230
	Carotids clamped	138	250	605	390	995	1245
2	Control	100	320	500	395	895	1215
	Carotids clamped	138	220	590	390	980	1200
3	Control	140	300	530	330	860	1160
	Carotids clamped	157	280	565	325	890	1170

DISCUSSION

When the preparation had been established the immediate aim was to bring the inflow to the heart and the venous return into such a relationship that there was no shift of blood from the animal to the reservoir or vice versa, rather than by setting it to give a predetermined value of cardiac output or of arterial blood pressure. It was of interest, therefore, to compare the total venous outflows under these conditions with the cardiac outputs measured by others in intact anaesthetized dogs. Kenney, Neil & Schweitzer (1951) give the results of different workers who employed the direct Fick principle; the mean was 2·85 l./min/m^2. The initial total venous return in our experiments was, on average, 35% less than this. But our measurements excluded coronary and bronchial flows. Moreover, it is well recognized that there is a reduction in cardiac output when the thorax is opened and artificial ventilation instituted; Daly, Lambertsen & Schweitzer (1953) found a reduction in output of 35–46%. It is within the limits of these 'subnormal' cardiac inflows that the rates of flow in the SVC, IVC and other large veins have been measured and some of the factors influencing flow have been investigated.

Caval flows

Few direct measurements have so far been made to assess simultaneously the relative rates of blood flow in the venae cavae; although observations have

been made of the variations in caval flow during the cardiac and respiratory cycles (for references see Brecher, 1956). Most previous workers have made measurements either in one vessel in a particular preparation (Burton-Opitz, 1921; Eckstein, Graham, Liebow & Wiggers, 1947; Feldman, Rodbard & Katz, 1948), or serially in different veins by transferring the flow-measuring device from one site to another (Blalock & Levy, 1937; Levy & Blalock, 1937). But Grundy & Howarth (1957) have recently reported simultaneous measurements of SVC and IVC flows in the cat in a preparation almost identical with ours.

The results obtained by Burton-Opitz indicated that the SVC flow of the cat was 43% of the combined caval flows; Grundy & Howarth's figures give a mean of 31% of the total venous return. Blalock & Levy (1937) found the flow in the SVC in the unanaesthetized dog to be 32·5% of the cardiac output measured by the direct Fick method. The proportion of the total caval flow, 30·4%, which has been found to return through the SVC in the present series of experiments, with measurements made simultaneously in different veins, is in good agreement with these observations of Blalock & Levy. There is less agreement with the results of Feldman *et al.* (1948), who measured the SVC and IVC flows separately in different dogs. Although they did not refer directly to the partition of the venous return between the two venae cavae, their data (tables 1 and 2 in their paper) indicated that the SVC carried approximately 43% of the combined caval flows during the initial period; however, in their experiments the vagi had been cut.

Hepatic flow

Measurements of liver blood flow in the dog *in vivo* have been made by the application of thermostromuhrs to the hepatic vessels, by the collection of the flow from the hepatic veins into the IVC and, indirectly, by measuring the rate of extraction from the blood and excretion by the liver of bromsulphalein (BSP). Some of the many measurements of liver flow obtained by these different methods are presented in Table 5. For purposes of comparison the different flows have been given in ml./min/kg body weight. It should be pointed out, however, that some workers are not in favour of relating liver flow to body weight, because they find that liver flow is variable and changes as the liver stores or releases blood (Grindlay, Herrick & Mann, 1941).

With the exception of the results of Blalock & Mason (1936), the direct measurements of liver flow given in Table 5 were made on dogs with open chests. The mean flows in the different series of experiments ranged from 25·3 to 33·0 ml./min/kg; our flows were higher than this (mean, 43·9 ml./min/kg). Our results agreed more closely with the estimates of liver flow made by the BSP method on dogs with the chest intact (29·5–48·6 ml./min/kg) but, in general, our flows are higher than the majority of hepatic flows given in

Table 5 and measured by a variety of methods. The reason for our higher flows is not clear, particularly since our results showed that deterioration in the general condition of the animal was accompanied by an increase in SVC flow at the expense of flow from the vascular bed drained by the IVC, hence it might have been expected that a reduction in liver flow would have been associated with the extensive operative procedures used in our experiments. Nevertheless, we do not wish to emphasize this difference, because our observations were made on only three preparations.

TABLE 5. Hepatic blood flow in dogs determined by various methods

Authors	Methods	Hepatic flow (ml./min/kg body wt.)
Grab, Janssen & Rein (1929)	Thermostromuhr	25·3
Blalock & Mason (1936)	Measurement of venous outflow	27·0
Grindlay, Herrick & Mann (1941)	Thermostromuhr	26·0
Selkurt (1954)	Measurement of venous outflow	33·0
Werner & Horvath (1952)	BSP clearance	42·0
Heineman, Smythe & Marks (1952)	BSP clearance	31·0
Pratt, Burdick & Holmes (1952)	BSP clearance	48·6
Smythe, Heinemann & Bradley (1953)	BSP clearance	29·5
Casselman & Rappaport (1954)	BSP clearance	37·0
Present results	Measurement of venous outflow	43·9

Flow in other veins

Only a few measurements of azygos flow in the dog seem to have been made with the venae cavae unoccluded. Andreasen & Watson (1952) report one measurement of 18 ml./min in a dog which probably weighed about 10 kg. A measurement of azygos flow has also been reported by Mott (1953); fig. 2 in her paper indicates a flow of about 15 ml./min in a dog weighing 3 kg. The rate of flow which we measured in a dog of 9·1 kg was about twice this.

Burton-Opitz (1902, 1903) found that the blood flows in the external jugular vein and the femoral vein of the dog were, on average, 11 and 3·6 ml./min/kg respectively; the corresponding flows in our experiments were 5·5 and 2·3 ml./min/kg. The larger flows noted by Burton-Opitz may be due to his observations having been made on intact dogs in which the cardiac outputs were probably higher than in the present experiments.

Effects produced by changes in arterial blood pressure and inflow to the heart

The changes in the partition of the caval flows which were produced by deliberate alterations in the inflow to the heart, or which were associated with spontaneous changes in the arterial blood pressure, can be conveniently considered together. In the initial period the proportion of the total flow

passing through the SVC in the different preparations appeared to vary inversely with the height of the arterial blood pressure. Similarly, in deteriorating preparations when arterial blood pressure fell although a constant inflow was maintained to the heart, SVC flow increased relatively. In other words, when arterial blood pressure was low there was evidence for an exclusion of blood from the regions drained by the IVC, presumably by vasoconstriction. Again, when the inflow to the heart was deliberately reduced the changes in the pattern of the venous return were similar to those described as occurring immediately after haemorrhage (Blalock & Levy, 1937; Eckstein *et al.* 1947; Wiggers, 1950). The results of Eckstein *et al.* and Wiggers suggested that after haemorrhage the measured reduction in the IVC flow was proportionately greater than the reduction in the cardiac output assessed from the arterial pressure pulse contour. Our results confirmed this and, moreover, the simultaneous measurements of SVC and IVC flows made it possible to demonstrate a relative increase in the SVC flow, although the absolute rate of flow fell as the cardiac output was reduced by diminishing the input from the pump.

Effects produced by anoxia

An even more striking reduction in IVC flow was shown when the preparations were submitted to anoxia. The effects produced were similar to those described by Feldman *et al.* (1948), but the controlled heart input and the simultaneous measurements of SVC and IVC flows in our experiments made it possible to amplify their conclusions. Because of the experimental conditions, cardiac output was not increased when anoxia was induced; nevertheless, the flow in the SVC was increased both absolutely and relatively during anoxia. This diversion of flow was due mainly to the rapid increase in resistance of the region drained by the IVC rather than to a dilatation in that associated with the SVC, although in some cases there was a fall in the peripheral resistance calculated for the SVC. The nature of the observations did not permit the identification of any particular region where vasodilatation occurred.

The increased rate of flow from the IVC during the initial stages of anoxia was similar to that observed by Feldman *et al.* (1948) and, in the intact animal, would be likely to represent an increase in the rate of the venous return, and therefore, in the output of the heart. An increase in the cardiac output in anoxia has been described by Harrison & Blalock (1927) and Harrison, Wilson, Neighbours & Pilcher (1927). The transient increase in the rate of venous outflow which was observed might have been caused by a constriction of veins. Alexander (1954) reported that venoconstriction is induced by anoxia.

Effects produced by carotid occlusion

Because the ability to produce an increased venous return had been demonstrated it was thought that further information about the effects of carotid occlusion on cardiac output might be elicited. The conflicting opinions on these effects were reviewed by Kenney *et al.* (1951) who, in their own experiments which refuted those of Charlier & Philippot (1947), could not find any substantial alteration in cardiac output after occlusion of both carotid arteries, stimulation of the carotid sinus nerve or changes in pressure in the perfused carotid sinus. Since their work, Leusen, Demeester & Bouckaert (1956) have claimed that occlusion of the common carotid artery caused an increase in cardiac output, although the increase was not as great as that which followed the reduction of pressure in an isolated sinus. Moreover, Rashkind, Lewis, Henderson, Heiman & Dietrick (1953), employing a preparation somewhat similar to the present one, obtained a fall in total venous return on stimulation of the carotid sinus nerves.

Constriction of veins in response to carotid artery occlusion has been demonstrated (Gollwitzer-Meier & Schulte, 1931; Alexander, 1954). But our experiments do not give any clear-cut indication of an increase in venous return such as might be caused by a constriction of veins. One reason for this may be that the arbitrary setting of the openings of the venous cannulae to the height of the right atrium did not impose sufficient distension upon the veins to enable optimum demonstration of venous capacity effects to be made. Furthermore, the vasomotor reactions may not have been as great as those in an animal which had been submitted to less stress by operation. Finally, as demonstrated by Chungchareon, Daly, Neil & Schweitzer (1952) there is uncertainty also, particularly in the dog, about the extent and the duration of the hypotension induced in the carotid sinus by occlusion of the common carotid artery.

SUMMARY

1. A preparation (dog) with open thorax is described in which the inflow to the heart was regulated and measured, and cardiac output controlled.

2. The rates of blood flow in the venae cavae, hepatic, external jugular, azygos and femoral veins were measured.

3. When the preparation was newly established and the circumstances were as similar as possible to those of the original circulation, the flow through the SVC was about one-third of the total venous return.

4. When the inflow to the heart was reduced the flow decreased proportionately more in the IVC than in the SVC.

5. When the animal was exposed to anoxic conditions the venous flow through the SVC increased both absolutely, if the cardiac inflow was maintained constant, and relatively, even if the cardiac inflow was reduced. In

some cases there was an increase in the venous return during the initial stages of anoxia.

6. After occlusion of the common carotid arteries there was no definite indication of any change in the total venous return.

7. Gradual deterioration of the preparation was accompanied by a diversion of blood flow to the vascular bed drained by the SVC.

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