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CAPACITANCE EFFECTS AND BLOOD RESERVOIR FUNCTION IN THE SPLANCHNIC VASCULAR BED DURING NON-HYPOTENSIVE HAEMORRHAGE AND BLOOD VOLUME EXPANSION IN ANAESTHETIZED CATS

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SUMMARY

1. These experiments were designed to measure how much blood is mobilized from or pooled in the liver, spleen and gastro-intestinal tract to compensate for a haemorrhage or infusion of blood.

2. Hepatic volume, splenic weight and intestinal volume were recorded in cats anaesthetized with sodium pentobarbitone. Whole blood was removed or infused at rates of 0·5–0·6 ml. $kg^{-1}.min^{-1}$ until 10 ml./kg (19% blood volume) had been removed or 18 ml./kg (34% blood volume) had been infused. These blood volume changes produced only small changes in arterial and portal pressures except after removal of 8 ml./kg (15% blood volume) when arterial pressure began to decrease rapidly.

3. With small haemorrhages of up to 4% blood volume, the liver contributed 16%, the gastro-intestinal tract 23% and the spleen a negligible proportion of the blood volume removed. With haemorrhages of 15% blood volume, the liver contributed 21%, the gastro-intestinal tract 22% and the spleen 19% of the volume removed; a total splanchnic contribution of 62%.

4. During infusions of 5–18 ml./kg (10–34% blood volume), the liver pooled 20%, the gastro-intestinal tract 40% and the spleen 6% of the volume infused; a total splanchnic contribution of 66%.

5. It is concluded that the splanchnic bed mobilizes or pools up to 65% of the volume of blood removed from or infused into the cats. The mechanisms responsible for this blood reservoir function are discussed. While several factors may be involved, it seems likely that a reflex regulation involving atrial receptors and the sympathetic innervation of the splanchnic capacitance vessels is of predominant importance.

INTRODUCTION

A blood reservoir may be defined as an area from which a significant volume of blood can be rapidly redistributed in a precise and controlled way to maintain cardiovascular homeostasis in response to stimuli such as postural changes or haemorrhage. An area which contains a significant proportion of the blood volume is usually but not necessarily a blood reservoir since a mechanism may not exist to mobilize this blood in a controlled way. Thus the distribution of blood volume and the distribution of blood reservoirs are not synonymous terms. Experimental data on the blood content and the blood reservoir function of various organs are scattered in the literature and we recently made a tentative tabulation of this data (Greenway & Oshiro, 1972). Subsequent work (C. V. Greenway, unpublished observations) showed that in cats, the gastrointestinal tract forms 6 % of the body weight and contains 10 % of the total blood volume instead of the 3 and 5 % respectively shown in the table. The corrected table is shown here (Table 1); the data on which it was based are quoted in the original paper.

TABLE 1. Regional distribution of blood volume; a tentative tabulation for the cat and dog (modified from Greenway & Oshiro, 1972)

Organ	Body wt. %	Total blood volume %	Total blood volume mobilized by sympathetic nerves %
Splanchnic organs			
Liver	3	14	6
Spleen	1	12	9
Intestine & stomach	6	10	4
Muscle	45	14	4
Adipose tissue	14	11	4
Lungs	1·5	10	?
Skin	3	2	1
Heart: Chambers	—	5	—
Muscle	0·5	1	—
Kidneys	0·5	1	—
Remainder	25·5	20	?
	100 %	100 %	28 %

It appears that some 28 % of the blood volume can be mobilized from the blood reservoirs if the sympathetic nerves supplying these capacitance vessels are simultaneously and maximally activated. This represents the maximum volume of blood which could theoretically be removed from an animal over a relatively short period of time without causing marked hypotension and disruption of cardiovascular homeostasis – the blood

volume reserve of Groom, Rowlands & Thomas (1965). However, simultaneous maximal activation of the sympathetic nerves to these reservoirs is unlikely to occur after small haemorrhages and in an experimental situation, anaesthesia and surgical preparation of the animal would be expected to further modify the results (Chien, 1967). On this basis, one might expect to be able to remove some 15–20% of the blood volume before hypotension develops (Groom *et al.* 1965). From Table 1, 6/28 or 21% of the volume removed would be mobilized from the liver, 32% from the spleen and 14% from the intestine, a total splanchnic contribution of 67% of the volume removed. However, this makes no allowance for an additional factor – reabsorption of extracellular fluid in skeletal muscle after haemorrhage. This might replace up to one third of the volume of blood removed (Kerr & Kirklin, 1970; Lundgren, Lundwall & Mellander, 1964).

Several earlier studies have shown decreases in splanchnic blood volume after haemorrhage but these were either not quantitative or involved marked hypotension (Alexander, 1955; Friedman, Frank & Fine, 1951; Glaser, McPherson, Prior & Charles, 1954; Johnson, 1960; Reynell, Marks, Chidsey & Bradley, 1955). The experiments by Brooksby & Donald (1971, 1972) tended to confirm the prediction for the total splanchnic bed of dogs but there are no data analysing the individual contributions of the splanchnic organs. In addition, we cannot predict from Table 1 the amounts of blood pooled in the splanchnic organs during an infusion of whole blood.

The experiments in this paper were designed to answer the following questions: (1) how much blood is mobilized from or pooled in each of the main splanchnic organs to compensate for blood lost or infused? and (2) what proportions of the volume of blood removed or infused do these contributions represent? Cats were used since many of the data in Table 1 were obtained in cats and control values of blood volumes and organ blood volumes are well established. Sodium pentobarbitone was used as the anaesthetic agent since it appears to cause minimal disturbance of the splanchnic vascular bed, it reduces rather than enhances reflex cardiovascular responses (thus our data underestimate rather than overestimate the significance of the splanchnic blood reservoirs) and many of the data on other responses to haemorrhage have been obtained on animals anaesthetized with this agent (these aspects were reviewed by Greenway & Stark, 1971). Changes in the volume of the liver, spleen and a piece of ileum were measured and they represent the total fluid mobilized or pooled in each organ; most of these organ volume changes are changes in blood content but the possible contributions of transcapillary fluid movements are discussed later. It was not possible to measure the changes in the volume

of the whole gastrointestinal tract and therefore changes in ileal volume were extrapolated on a weight basis. This extrapolation is discussed later. With this limitation, the experiments show the contributions of the hepatic splenic and intestinal blood reservoirs during haemorrhages of 0–20% of the blood volume and whole blood infusion of 0–35% of the blood volume. Changes in blood volumes of these magnitudes were chosen because they do not represent an overwhelming stimulus to the mechanisms which attempt to maintain cardiovascular homeostasis and they cause minimal changes in arterial and venous pressures, thus minimizing passive changes in regional blood volume.

METHODS

Thirty six cats (1·9–2·9 kg body weight; mean 2·4 kg) were anaesthetized by I.P. injection of sodium pentobarbitone (Abbott, 30 mg/kg). When reflex ear, limb and swallowing movements returned, supplementary doses of pentobarbitone (2 mg/kg) were given through a cannula in a forelimb cutaneous vein. The trachea was cannulated and mean arterial pressure was recorded from a femoral artery with a P23AC Statham pressure transducer. The abdomen was opened by a mid-line incision and mean portal pressure was recorded (P23BC Statham transducer) from a cannula inserted into the portal vein through a small branch from the appendix. All recordings were made on a Beckman Type R dynograph recorder. A cannula was inserted through a femoral vein so that its tip lay in the lower abdominal inferior vena cava; this cannula was used for infusion or withdrawal of blood.

In twelve cats (six subjected to haemorrhage, six to infusion), hepatic volume was recorded by the plethysmographic method previously described and evaluated (Greenway, Stark & Lautt, 1969; Greenway & Lautt, 1970). Briefly, the ligaments connecting the central and left lobes of the liver to the diaphragm were ligated and cut and the liver with the exception of the right lateral and caudate lobes was inserted into a Perspex plethysmograph. The vessels to and from the liver remained intact and passed through a 5 cm diameter aperture which was sealed with a plasticized hydrocarbon gel (Plastibase, Squibb). The plethysmograph was filled with Ringer–Locke solution at 37° C and connected to a float recorder which operated an isotonic transducer (Harvard Apparatus Co. Model 356). The pressure within the plethysmograph was adjusted to zero relative to the hilum of the liver. In each experiment, the recorded volume changes were multiplied by total liver weight, divided by the weight of the part of the liver within the plethysmograph and divided by the body weight, to convert the data into ml. change in liver volume/kg body weight. The means ($\pm$ S.E.) of these conversion factors were $85 \pm 2{\cdot}8$ g total liver weight, $69 \pm 2{\cdot}5$ g liver in plethysmograph and $2{\cdot}4 \pm 0{\cdot}04$ kg body weight.

In another twelve cats (six subjected to haemorrhage, six to infusion), splenic weight was recorded by the method previously described and evaluated (Greenway, Lawson & Stark, 1968). Briefly, the ligaments were tied and divided to allow mobilization of the spleen through the abdominal incision. The spleen was wrapped in gauze and polythene and placed on a cradle suspended from a force-displacement transducer (Grass Ft03C). The system was calibrated by weights and allowance was made for pedicle tension as stated in the original description of the method. In each experiment, the recorded weight changes were divided by the body weight (mean $2{\cdot}6 \pm 0{\cdot}12$ kg) to convert the data into g change in spleen weight/kg body weight.

In another twelve cats (six subjected to haemorrhage, six to infusion), the volume

of a section of ileum was recorded by plethysmography. The method was similar to that previously described (Folkow, Lundgren & Wallentin, 1963) except that the intestinal vein was left intact. Briefly, a length of ileum was separated from the remainder of the intestine by division between ligatures at each end. The mesentery was divided between ligatures down to the origin of the superior mesenteric artery and vein, thus providing a vascular pedicle to the separated loop. The loop was placed in a Perspex plethysmograph which was sealed with Plastibase. In these experiments, the tip of the portal pressure cannula lay in the mesenteric vein within the plethysmograph and this pressure was recorded before and after the plethysmograph was sealed. Great care was taken to avoid obstruction to the vein during sealing. The portion of the ileum in the plethysmograph represented 26% by weight of the total gastro-intestinal tract. In each experiment, the recorded volume changes were multiplied by the total weight of the stomach, intestine and colon and divided by the weight of intestine in the plethysmograph and the body weight to convert the data into ml. change in gasto-intestinal volume/kg body weight. The means (± S.E.) of these conversion factors were 123 ± 3·2 g total weight of the gastro-intestinal tract, 32 ± 1·5 g small intestine in plethysmograph and 2·2 ± 0·09 kg body weight.

After completion of the surgery, all animals were given 5 ml. 5% dextran in 0·9% w/v NaCl and the recorded variables were allowed to stabilize for 30–45 min. In the cats subjected to haemorrhage, blood was then removed at the rate of 0·50 ml. kg^{-1} min^{-1} into a glass syringe containing heparin (10 mg) and a magnetic stirring bar. In the volume expansion studies, fresh whole blood obtained from donor cats with heparin added (10 mg heparin /50 ml. blood), was infused at the rate of 0·56 ml. $kg^{-1}.min^{-1}$.

RESULTS

Organ volume changes in response to haemorrhage

Fig. 1 shows the experimental record in one cat subjected to haemorrhage. Arterial and portal pressures were well maintained until 8 ml./kg had been removed. Liver volume decreased steadily until this start of hypotension was reached and then it decreased more rapidly. This figure is included to show the type of record from which the following data were calculated (see Methods).

Hepatic volume was measured in six cats, splenic weight in six cats and intestinal volume in six cats. In all eighteen, blood was removed at the rate of 0·5 ml. $kg^{-1}.min^{-1}$. The effects of these haemorrhages on arterial and portal pressures were not significantly different between the three groups of cats ($P > 0·1$, t test for unpaired data) and the data have been combined for presentation. The mean control arterial pressure was 121 ± 6·0 mmHg (mean ± S.E.). This pressure decreased 13 mmHg during the first 16 min of haemorrhage but then decreased more rapidly (Fig. 2). Therefore, this point, when 8 ml./kg had been removed, was taken as the start of hypotension and the organ volume changes up to this point were analysed. The mean portal pressure during the control period was 8·6 ± 0·77 mmHg (mean ± S.E.). This had decreased to 7·3 ± 0·86 mmHg when 8 ml./kg had been removed. Removal of 8 ml./kg represents the removal of 15%

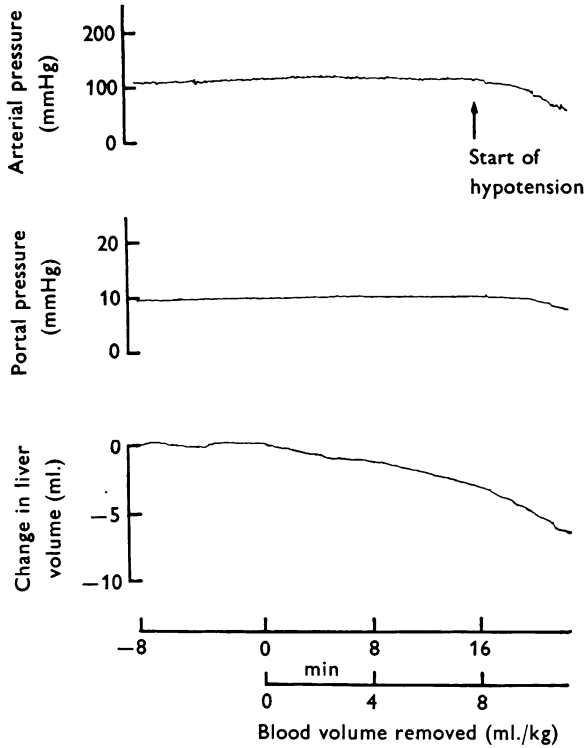

Fig. 1. Cat 2·6 kg, liver weight 66 g. Effects of haemorrhage on arterial pressure, portal pressure and hepatic volume.

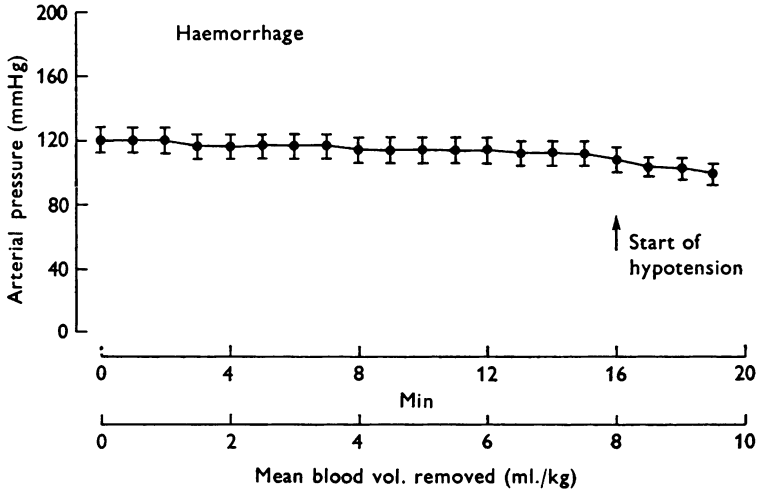

Fig. 2. The means (± S.E.) of the arterial pressure in the eighteen cats subjected to haemorrhage.

of the total blood volume of cats (mean blood volume 52 ml./kg; Farnsworth, Paulino-Gonzalez & Gregersen, 1960; Groom *et al.* 1965; Scott, 1972; C. V. Greenway, unpublished observations).

The effects of the haemorrhages on the hepatic, splenic and intestinal volumes/kg body weight are shown in Fig. 3. It can be seen that hepatic and intestinal volumes decreased steadily from the start of the haemorrhage. Splenic weight showed little change until 2·5 ml./kg had been removed but as the haemorrhage became greater the spleen decreased rapidly in size. To allow assessment of the role of these organs as blood

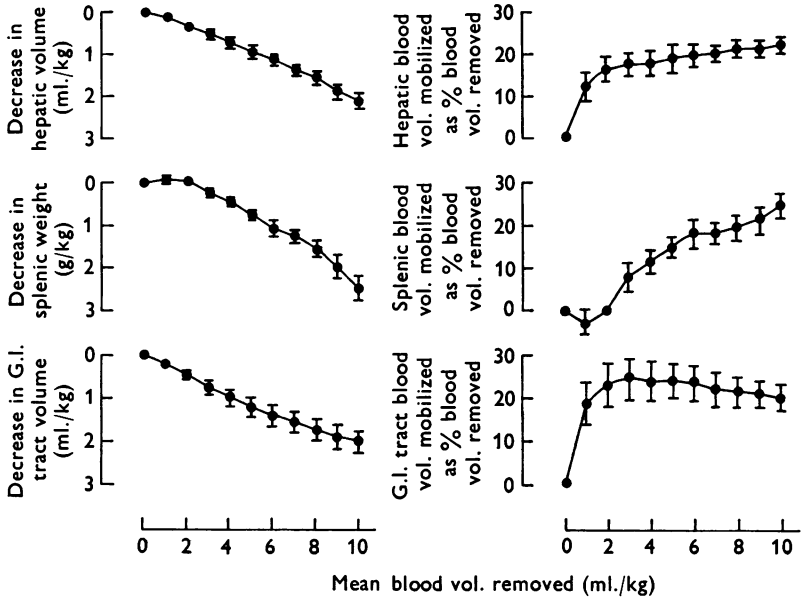

Fig. 3. The means (± S.E.) of the decreases in hepatic volume (six cats), splenic weight (six cats) and gastrointestinal volume (six cats) during haemorrhage and the means (± S.E.) of these values expressed as proportions of the volume of blood removed.

reservoirs, the volumes of blood mobilized from each organ are expressed as proportions of the blood volume removed from each animal (Fig. 3). It can be seen that the liver mobilized 16 % and the gastro-intestinal tract 23 % of the volume removed when the haemorrhage was 2 ml./kg; the spleen did not respond to these small haemorrhages. When 8 ml./kg was removed, the liver mobilized 21 %, the spleen 19 % and the gastro-intestinal tract 22 % of the volume removed. Thus the total splanchnic bed contributed 62 % of the volume of blood removed from the animal.

Since the liver contains 14 % of the blood volume of 52 ml./kg (Table 1),

it contains 7·3 ml./kg. After a haemorrhage of 8 ml./kg (15% blood volume), 1·67 ml./kg had come from the liver (Fig. 3). Thus the hepatic blood content was reduced by 23%. The spleen contains 12% of the blood volume representing 6·2 ml./kg. After a haemorrhage of 8 ml./kg, 1·54 ml. had come from the spleen and splenic blood content was reduced by 25%. The gastro-intestinal tract contains 10% of the blood volume representing 5·2 ml./kg. After a haemorrhage of 8 ml./kg, 1·72 ml. had come from the tract and its blood content was reduced by 33%.

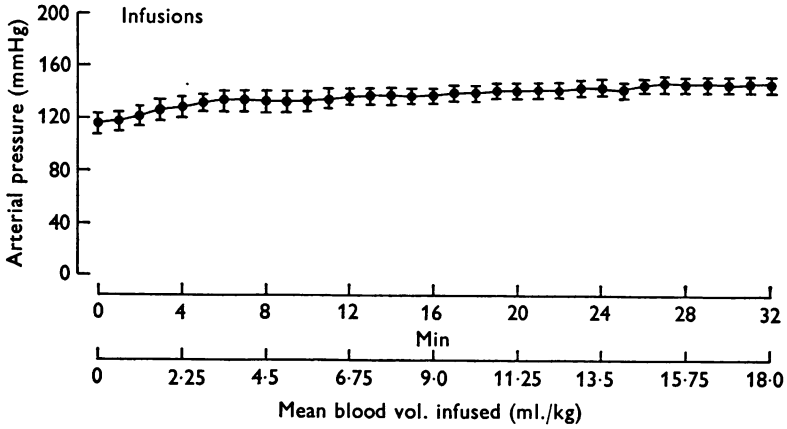

Fig. 4. The means (± S.E.) of the arterial pressures in the eighteen cats given infusions of whole blood.

Organ volume changes in response to infusions

Hepatic volume was measured in six cats, splenic weight in six cats and intestinal volume in six cats. In all eighteen cats, blood was infused at the rate of 0·56 ml. $kg^{-1}.min^{-1}$. The effects of these infusions on arterial and portal pressures were not significantly different between the three groups ($P > 0·1$, t test for unpaired data). The mean control arterial pressure was 116 ± 7·5 mmHg (mean ± S.E.) and this pressure increased steadily to 144 ± 8·7 mmHg after 18 ml./kg had been infused (Fig. 4). This volume represents an increase of 35% in the cat's total blood volume. The mean portal pressure during the control period was 7·9 ± 0·57 mmHg (mean ± S.E.). This increased to 8·8 ± 0·67 mmHg when 9·0 ml./kg had been infused and to 10·1 ± 0·77 mmHg when 18 ml./kg had been infused.

The effects of the infusions on the hepatic, splenic and gastro-intestinal volumes/kg cat weight are shown in Fig. 5. It can be seen that the hepatic and intestinal volumes increased steadily from the start of the infusion. Splenic weight increased later and to a smaller degree. To allow assessment of the role of these organs as blood reservoirs, the volumes of blood pooled

in each organ are again expressed as proportions of the blood volume infused (Fig. 5). It can be seen that the liver pooled some 20 % and the intestine 40 % of the infused volume when that volume was 8–18 ml./kg. The spleen did not respond to small infusions and pooled only about 6 % of larger infusions. It was thus much less able to pool blood than to mobilize it.

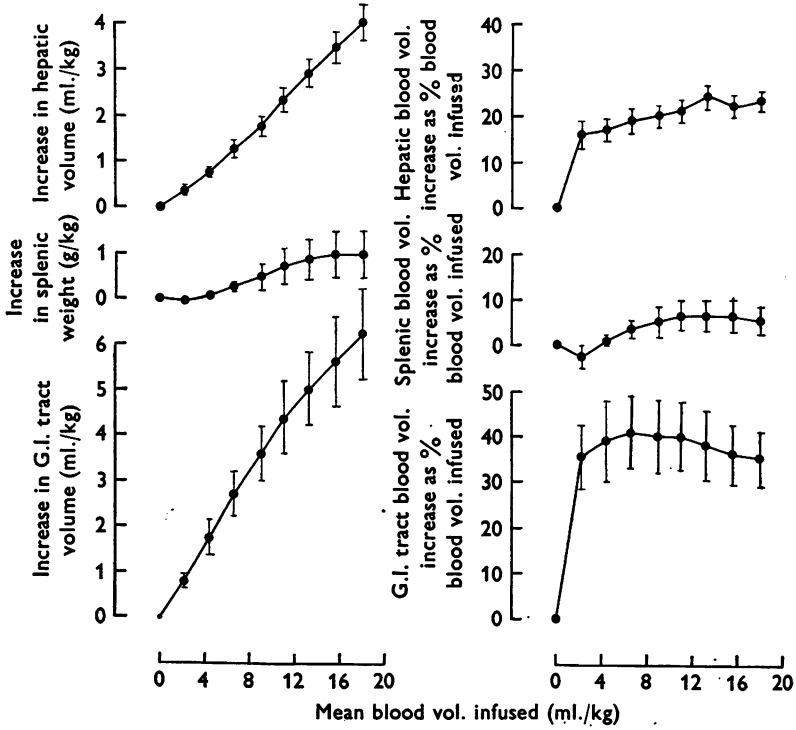

Fig. 5. The means (± S.E.) of the increases in hepatic volume (six cats), splenic weight (six cats) and gastrointestinal volume (six cats) during infusions of whole blood and the means (± S.E.) of these values expressed as proportions of the volume of blood infused.

Total splanchnic volume changes

The sum of the volume changes in the three organs represents essentially the total splanchnic volume changes. These changes during both haemorrhage and infusions are shown in Fig. 6. It can be seen that the splanchnic bed is able to mobilize or pool 50–70 % of the volume of blood removed or infused respectively.

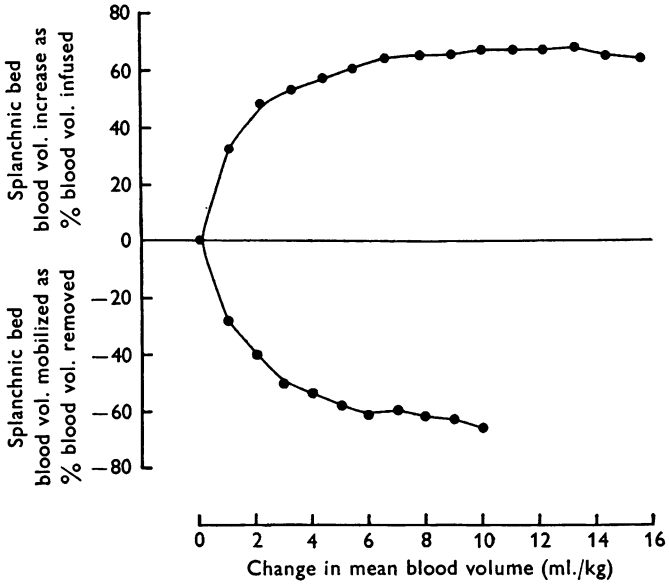

Fig. 6. The mean proportions which are contributed by the splanchnic vascular bed to the volume of blood infused into or removed from cats. These values were obtained by summing the contributions of the liver spleen and gastrointestinal tracts in Figs. 3 and 5.

DISCUSSION

The methods used in this study have been used previously and their advantages and disadvantages have been discussed (see references in Methods). Two aspects require further consideration in relation to the experiments presented here. First, the volume changes were recorded only in a small piece of ileum and these were extrapolated on a weight basis to the whole gastro-intestinal tract. We are not aware of any direct data in support of this extrapolation for the stomach but Hultén (1969) showed that the blood content of the colon was very similar to that of the ileum in cats. The extrapolation was used to estimate the total splanchnic blood reservoir function and to allow comparison of our results with those of Brooksby & Donald (1971, 1972). Their work involved equally important but different limitations and the similarity of the data (see below) suggests that both methods give reasonable estimates of the true values.

Secondly, the recorded volume or weight changes represent the total contribution by each organ in mobilizing or pooling fluid, be it whole blood, red cells, plasma or extracellular fluid. In the case of the spleen, the weight changes represent addition or removal of blood with a high haematocrit and a greater proportion of red cells than plasma is mobilized or

pooled. In the case of the liver, the volume mobilized during haemorrhage is probably entirely blood. No evidence of reabsorption of extracellular fluid was obtained previously during stimulation of the sympathetic nerves (Greenway *et al.* 1969), during reduction in hepatic venous pressure (Greenway & Lautt, 1970) or during infusions of a variety of drugs (Greenway & Lautt, 1972*b*). The volume pooled during infusions is likely to contain a component due to increased extracellular fluid although most of it is intravascular blood. Filtration of fluid does occur when hepatic venous pressure is increased (Greenway & Lautt, 1970). In these infusion experiments, portal pressure increased by only 2·2 mmHg by the end of the infusion. A transsinusoidal filtration rate of 0·06 ml. $min^{-1}.mmHg^{-1}$. 100 g liver (Greenway & Lautt, 1970) would result in an accumulated volume of about 1 ml./kg at the end of infusion; that is, about 25 % of the recorded volume change. In the gastro-intestinal tract, reabsorption and filtration respectively would be expected to occur during the haemorrhage and infusion. However, consideration of the portal pressure changes in relation to the work of Johnson & Hanson (1966) and Wallentin (1966) again suggests that such effects would be small relative to the total volume changes. After large haemorrhages with marked hypotension (45 mmHg), there was considerable extracellular fluid reabsorption from the splanchnic bed in splenectomized dogs (Marty & Zweifach, 1971). It is interesting that the transcapillary fluid movements in the liver and intestine tend to offset the red cell effects in the spleen, thus maintaining the haematocrit of the circulating blood. We have no quantitative data on the accuracy of this balance within the splanchnic bed of the cat but it merits further study.

Our results suggest that as the volume of blood lost or infused increases, the splanchnic bed mobilizes or pools respectively 50–70 % of the volume (Fig. 6). With small haemorrhages up to 2 ml./kg (4 % blood volume), the liver contributes 16 %, the gastro-intestinal tract 23 % and the spleen a negligible proportion of the volume removed. When these small volumes are removed, the small but readily mobilizable reservoirs are presumably most important, for example, the large veins (Kerr & Kirklin, 1970). With haemorrhages of 8 ml./kg (15 % blood volume), the splanchnic bed is clearly an important blood reservoir mobilizing 62 % of the removed volume. Another 15 % of the removed blood comes from the lungs (Magilligan, Oleksyn, Schwartz & Yu, 1972). The remaining 23 % of the blood is mobilized from other reservoirs (Table 1) and of these, skeletal muscle appears to be of major importance (Lundgren, Lundwall & Mellander, 1964).

Our data are in reasonable agreement with the data of Brooksby & Donald (1971). Their data were obtained in dogs anaesthetized with

chloralose, by summation of the excess of outflow over inflow to the splanchnic area. After a haemorrhage of 7·2 ml./kg over 2 min, their average decrease in arterial pressure was 10 mmHg as opposed to 13 mmHg in our experiments, and 54% of the volume removed was mobilized from the splanchnic bed as opposed to 62% in our experiments. This difference may be due to the slower rate of haemorrhage used in our experiments.

Several questions arise in relation to the mechanisms of the observed changes. The decreases in splanchnic blood content during haemorrhage could be due to active constriction of the capacitance vessels or to passive collapse due to decreased transmural pressure. This decreased transmural venous pressure could result from decreased arterial and venous pressures due to inability of the cardiovascular system to compensate for the haemorrhage, or it could result from the decreased organ blood flow due to arteriolar vasoconstriction, itself a compensatory response (Brooksby & Donald, 1972). In these experiments, the changes in arterial and portal pressures were minimal and the passive consequences of these changes would appear to be at most a small part of the observed responses. We have previously shown that during vasopressin infusion, marked splenic and intestinal vasoconstriction with a consequent reduction in portal flow do not alter hepatic or splenic volume, and changes in splenic flow due to partial occlusion of the splenic artery do not alter splenic weight (Greenway, Lawson & Stark, 1968; Greenway & Lautt, 1972*a*). Although we have no data on the gastrointestinal tract, we tend to believe that passive collapse of the veins secondary to reduced flow is likely to be of greater importance in this bed since intestinal flow shows a marked and sustained decrease (McNeil, Stark & Greenway, 1970). Nevertheless we conclude that the *major* factor causing mobilization of blood from the gastrointestinal tract, liver and spleen after small haemorrhages is vasoconstriction of the capacitance vessels. Brooksby & Donald (1972) favoured a more important role for passive collapse in their experiments but the design of their experiments favoured passive responses. In our experience, the 18 sec periods of nerve stimulation which they used will markedly overestimate the passive component since the flow response, and hence the passive component, is essentially complete within this time while the active capacitance response takes several minutes to become maximal. The obvious approach of repeating the haemorrhage after section of the splanchnic nerves is also full of pitfalls. As Brooksby & Donald (1972) showed and our experiments (unpublished observations) confirmed, after splanchnic nerve section a given haemorrhage causes a much more marked arterial and portal hypotension and the passive component of the response to haemorrhage is increased and over-estimated. This increased hypotension is itself strong evidence that the splanchnic nerves normally cause

important active capacitance responses after haemorrhage since their removal does not markedly affect flow responses to haemorrhage in cats (McNeill *et al.* 1970; Greenway & Stark, 1971). In skeletal muscle, the situation may be rather different and passive collapse secondary to flow reduction may be more important (Lesh & Rothe, 1969).

Humoral factors causing contraction of the splanchnic capacitance vessels are also unlikely to be of major importance. Slow haemorrhages of the type used in these experiments do not release catecholamines from the adrenal medullae (Regoli & Vane, 1966) and angiotensin and vasopressin have relatively small effects on the capacitance vessels (Greenway & Stark, 1970; Greenway & Lautt, 1972*a*). Thus it seems reasonable to conclude that the predominant pathway causing constriction of the splanchnic capacitance vessels is the sympathetic nerves which are well known to produce large decreases in blood content in the liver, spleen and intestine (Table 1).

The afferent pathways which cause activation of these nerves in response to haemorrhage are unlikely to be from arterial baroreceptors. Arterial pressure did not change markedly and although there may have been changes in pulse pressure, these baroreceptors do not cause marked responses in capacitance vessels (Hainsworth, Karim & Stoker, 1973; Lautt & Greenway, 1972; Pelletier, Edis & Shepherd, 1971). However, Brooksby & Donald (1971) did obtain some changes in splanchnic blood volume during large changes in carotid sinus pressure in dogs. Atrial pressure receptors may be of greater importance. These receptors have been shown to markedly alter their firing rate during haemorrhage (Gupta, Henry, Sinclair & von Baumgarten, 1966) and they are known to cause other responses to haemorrhage such as renin release (Hodge, Lowe, Ng & Vane, 1969), tachycardia and arteriolar vasoconstriction (Pelletier *et al.* 1971; Paintal, 1973). Direct evidence of their role in the splenic contraction after small haemorrhages was presented by Pelletier *et al.* (1971).

Thus the available evidence suggests, as a working hypothesis, that mobilization of blood from the splanchnic region after haemorrhage involves active constriction of the capacitance vessels mediated by a sympathetic reflex from atrial pressure receptors.

The mechanism of the pooling of blood during the infusions is not as clear. In the normal, normovolaemic resting animal, there appears to be some basal sympathetic tone on the capacitance vessels as shown by the redistribution of blood volume from the pulmonary to the systemic vascular bed after administration of an adrenergic α-receptor blocking agent (Nickerson, 1970). This is increased in animals anaesthetized and subjected to surgery (Chien, 1967). Thus, a part of the pooling during

infusions could involve inhibition of tonic sympathetic activity to the capacitance vessels. The changes in arterial pressure were quite large while portal pressure changes were small, and the pressure in the inferior vena cava was not measured. Thus the role of passive distension is difficult to assess from our data and further experiments are required.

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