J. Physiol. (1967), **190**, *pp.* 229–240
With 3 text-figures
Printed in Great Britain

SOME OBSERVATIONS ON 'AUTOREGULATORY ESCAPE' IN CAT INTESTINE

BY ROSEMARY BAKER AND D. MENDEL
From the Department of Physiology, King's College, Strand, London, W.C. 2

(*Received* 12 *December* 1966)

SUMMARY

1. 'Autoregulatory escape' (Folkow, Lewis, Lundgren, Mellander & Wallentin, 1964*a*) in which intestinal blood flow escapes from the constrictor influence of splanchnic nerve stimulation, was investigated in the cat. Measurements were made of intestinal blood flow, movements and portal venous pH and oxygen content.

2. 'Autoregulatory escape' was observed with sympathetic nerve stimulation and noradrenaline infusion. It did not occur during haemorrhage.

3. The recovery of flow during noradrenaline infusion could not be explained by changes in intestinal motility or loss of effect of noradrenaline due to changes in portal venous hydrogen ion concentration.

4. During a noradrenaline infusion there was a reduction in oxygen consumption associated with a fall in blood flow. During the period of 'autoregulatory escape', however, oxygen consumption increased. The significance of these observations is discussed.

INTRODUCTION

It has been reported by Folkow *et al.* (1964*a*) that stimulation of the splanchnic nerves in the cat resulted in a reduction in intestinal blood flow followed by a partial recovery of flow in spite of continued stimulation. They have termed this the period of 'autoregulatory escape' from the constrictor fibre influence. In an attempt to study the distribution of blood flow to the intestinal wall during 'autoregulatory escape', Folkow, Lewis, Lundgren, Mellander & Wallentin (1964*b*) injected India ink into the intestinal circulation of the cat and compared the amount of ink in successive segments of the intestine at different times during splanchnic nerve stimulation. They found that there was a gross reduction in the amount of ink in the mucosa during the period of maximum reduction in flow and slightly more ink during the period of escape. It was suggested that escape was associated with the opening up of submucosal shunts.

It is known from the work of Cohnheim (1907–8) that the oxygen consumption of intestinal smooth muscle is small compared with that of glandular tissues. Since a redistribution of blood flow from mucosa to smooth muscle is said to occur with sympathetic nerve stimulation (Folkow *et al.* 1964*a*), it was of interest to see if there were associated changes in oxygen consumption which could support this concept.

Apart from Folkow's suggestion that 'autoregulatory escape' might be related to the opening up of shunts, the mechanism is not clear. It is not known, for example, if it is associated only with sympathetic nerve stimulation or whether it also occurs when intestinal blood flow is reduced by other means. There is no information whether bowel movements are involved or whether there is loss of sensitivity to sympathetic amines due to alterations in local pH (Bygdeman, 1963). These matters have been the subject of the present work.

METHODS

Cats weighing between 2 and 3 kg were used. They were anaesthetized with intraperitoneal injection of sodium pentobarbitone (Nembutal, Abbott Laboratories Ltd—42 mg/kg body weight).

Operative procedure. Following tracheostomy, one common carotid artery was dissected clear of surrounding tissue. A mid-line incision was made in the abdomen and haemostasis secured. The superior mesenteric artery was identified. The sympathetic nerves in the sheath around it were carefully separated and preserved for stimulation during the experiment. The main mesenteric artery usually divided into three branches, a large central branch which was prepared for cannulation, and two smaller ones, one on each side of the central branch. The splenic vein, femoral artery, femoral vein and, when necessary, the superior mesenteric vein were also prepared for cannulation. At the end of the operative procedure, the animals were heparinized ('Pularin', Evans Medical Ltd., 5000 units/ml–0·1 ml./kg body weight).

Blood flow. Arterial blood flow to the intestine was measured by means of a density flowmeter (Dawes, Mott & Vane, 1953). In this method, which is based on the stromuhr principle, the time taken for a known quantity of fluid to travel between two electrodes is determined. The basic design of the apparatus was unchanged, but the circuit was altered by using two double triode valves controlling two multi-contact relays instead of the larger number of relays used in the original apparatus. The time taken for the fluid to travel between two electrodes was recorded on a millisecond stop-clock (Venner Electronics Ltd. Type TSA 3314) which was started automatically when the fluid reached the first and stopped when it reached the second electrode. When the apparatus was calibrated against known flow rates it was accurate to within $\pm 3\%$ over the range 1–40 ml./min. The pressure drop across the flowmeter was 1 mm Hg at the highest rates of flow.

The flowmeter was inserted between the carotid artery and the large branch of the main mesenteric trunk. The carotid connexion to the flowmeter was first made by a polythene cannula (internal diameter 1·5 mm) and the larger central branch of the mesenteric artery was then cannulated distally. To maintain blood flow in the intestine during this procedure, the main mesenteric truuk was left unoccluded and a ligature tied on the large central branch immediately distal to the division of the main trunk. Sufficient blood was carried by the two small branches to cause a retrograde flow of blood from the large branch when it was incised. After cannulation, the main trunk was occluded, care being taken not to involve the sympathetic nerves. At the end of the experiment dye was injected into the

superior mesenteric artery cannula and the injected area removed and weighed. The duodenum was not injected.

Venous outflow from the intestine was measured by passing the blood from a cannula in the superior mesenteric vein through a photo-electric drop recorder. The outflow was returned to the femoral vein. Each drop of blood was signalled by the deflexion of a galvanometer recording on an infra-red 12 channel recorder (SE Laboratories).

Pressure measurement. Perfusion pressure was determined by means of an S.E. Laboratories manometer attached to a side arm of the arterial inflow. Portal venous pressure was recorded by means of a Statham PG 23B from a catheter in the splenic vein. All pressures were recorded on the S.E. Laboratories recorder. Blood pressure compensation was obtained by allowing the animal to bleed into or receive blood from a reservoir maintained at a known pressure. Resistance was calculated from the expression arterial inflow pressure − portal venous pressure/blood flow.

Bowel movements. Ligatures were placed round the duodenum and lower end of the ileum so that the intestine being studied was a closed system. A small incision was made in the wall of the intestine and a flanged cannula filled with saline was inserted and tied in position. The pressure changes within the lumen were recorded by a Statham PG 23B.

Noradrenaline infusion. Solutions of −noradrenaline (Levophed 2–10 μg/ml.) were delivered to the superior mesenteric arterial inflow via a side arm at a rate of 0·5 ml./min from a constant infusion machine.

Nerve stimulation. The mesenteric nerves were placed over a pair of electrodes and square wave pulses (9 V, 3 msec) were delivered at various frequencies from a Palmer square wave stimulator.

pH. Samples of arterial blood were obtained from the cannulated femoral artery. Portal venous blood was obtained from the splenic vein cannula which was inserted as far as the junction of the vein with the superior mesenteric vein, its position being identified both during cannulation and at the end of the experiment. pH was measured on an Electronic Instruments Vibron Blood pH meter.

Oxygen determination. Samples of arterial and venous blood were obtained as for the pH determinations. Enough blood (about 1·5 ml.) was withdrawn to clear the splenic cannula of stagnant blood and then the sample was slowly taken whilst the flow was being measured. In preliminary experiments during the resting state blood taken in this manner did not affect the flow. Small gastric, pancreatic and duodenal branches of the portal vein were ligatured in some experiments to minimize the risk of withdrawing blood from areas other than the one under consideration. The percentage saturation of the blood with oxygen was measured on a Haemoreflector (Kipp and Zonen Mo 1) and the haemoglobin content was determined by Haldane's method. The haemoreflector was calibrated before each group of readings. The oxygen consumption was calculated from the arterio–venous O_2 difference × blood flow.

RESULTS

Resting flows. In nine cats the range of blood pressure at the beginning of each experiment was 80–120 mm Hg (average 90 mm Hg) and the range of flows was 6–32 ml./min (average 17 ml./min). The average weight of the perfused intestine was 105 g.

Sympathetic nerve stimulation. The effect of stimulating the post-ganglionic sympathetic fibres to the intestinal vessels on intestinal blood flow was determined in eleven experiments on two cats. A bundle of sympathetic fibres surrounding the main mesenteric artery was supported

on electrodes and resting flows determined for a period of 5 min. The nerves were stimulated with square wave pulses of 9 V and 3 msec duration at frequencies between 5 and 20 impulses/sec for periods of up to 10 min. Blood flow was measured repeatedly before, during and after the period of stimulation. A typical result is shown in Fig. 1*A*. It can be seen that there was an initial rapid decrease in flow followed by a period of recovery of flow ('autoregulatory escape', Folkow *et al.* 1964*a*) which persisted for the duration of the stimulus. After the period of stimulation flows returned to near the initial levels. Similar results were obtained in all experiments. During the period of stimulation the minimum flow values observed ranged from 28 to 82 % of the initial flow and returned during

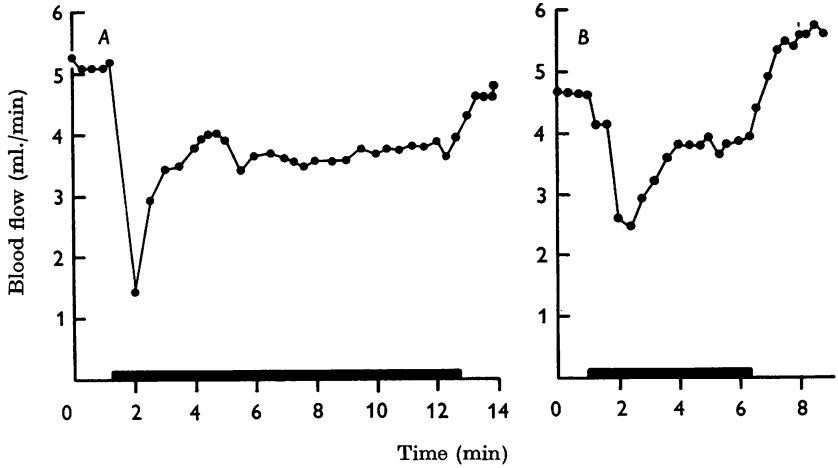

Fig. 1. (*A*) The effects of splanchnic nerve stimulation on intestinal blood flow. Black bar—nerve stimulation. 9 V, 3 msec, 20 impulses/sec. (*B*) The effects of noradrenaline infusion on intestinal blood flow. Black bar—noradrenaline infusion into superior mesenteric artery. 2 μg/min.

the period of 'autoregulatory escape', to between 59 and 94 % of the initial flow. The reduction in flow was greatest with the higher rates of stimulation but there was no relation between the frequency of stimulation and the extent of the recovery in flow. Flow exceeded the initial levels in two of the experiments after stimulation was stopped. Individual stimulations lasted from 3·5 to 10 min, the maximum reduction in flow occurring after 0·5–2·5 min and escape after 2·0–4·0 min.

Effect of infusions of noradrenaline. The observation that partial recovery of flow ('autoregulatory escape') during mesenteric nerve stimulation occurred at low frequencies as well as at high frequencies made it unlikely that fatigue of the nerve endings was the cause. In order to verify

this the effect of infusion of noradrenaline into the mesenteric arterial bed was examined.

The concentration of noradrenaline ranged from 2 to 10 μg/ml. delivered at a rate of 0·5 ml./min. Fifteen infusions were carried out on 7 cats and a typical result is shown in Fig. 1*B*. It can be seen that the response was similar to that obtained with splanchnic nerve stimulation in that there was a rapid decrease in flow followed by a period of recovery whilst the infusion was maintained. The recovery of flow was observed in thirteen out of the fifteen experiments. The minimum flow values ranged from 0 to 78 % of the initial flow and returned to between 51 and 87 % of the initial level. In the two experiments where no escape occurred there was complete cessation of flow.

Increase of flow above the initial level was seen in twelve out of fifteen experiments after stopping noradrenaline infusion, in contrast to two out of eleven after nerve stimulation. After noradrenaline the increase in flow over the initial flow ranged from 3 to 74 % of the initial flow (average 22·3 %).

In order to standardize the conditions, noradrenaline infusions were used to obtain more information about 'autoregulatory escape'.

Effect of bowel movement. It is known that peristalsis can augment intestinal blood flow (Sidky & Bean, 1958) and the possibility was investigated that changes in bowel activity could be responsible for 'autoregulatory escape' during noradrenaline infusion. Bowel movements were recorded during eight infusions in three of the cats previously described. In three experiments, bowel movements were absent throughout and 'autoregulatory escape' was seen. In the remaining five experiments movements were present initially and in four of these they were inhibited by noradrenaline. Flow 'escaped' before the return of movements in three experiments and movements returned before the 'escape' of flow in the fourth experiment. In the experiment where bowel movement was not inhibited, 'escape' of flow was still seen. There was no change in tonus in any of these experiments. It was clear then that changes in bowel activity could not explain the phenomenon of 'autoregulatory escape'.

Oxygen consumption of the small intestine. Oxygen determinations were carried out on portal vein blood immediately before and during infusion of noradrenaline. A total of ten infusions were studied in five cats. the average percentage saturation of the arterial blood during resting conditions was 97·9 % and that of the venous blood was 52·3 %.

Table 1 shows the results and the calculated oxygen consumptions. It can be seen in six out of nine observations there was a marked decrease in the percentage saturation of portal blood with oxygen during the period of reduced blood flow. There was only a small change, or no change, in three

experiments. In every case the oxygen consumption of the gut was markedly decreased. During the period of 'autoregulatory escape', however, oxygen consumption increased but remained below the initial level. In order to exclude the possibility that arterial inflow might not be a valid basis for the calculation of oxygen consumption, similar measurements were made during three infusions in two cats when portal outflow was measured directly by cannulating the superior mesenteric vein and allowing blood to pass through a drop counter before returning to the femoral vein. During one infusion arterial inflow and venous outflow were both measured, reduction and 'escape' of flow being recorded simultaneously in

TABLE 1. Changes in arterial inflow, portal venous oxygen content and oxygen consumption during ten noradrenaline infusions. (*a*) Before infusion; (*b*) during period of reduced flow; (*c*) during autoregulatory escape

Cat no.	Dose of noradren. ug/min.	Blood flow (ml./min)			(%) saturation of venous blood			Oxygen consumption (ml./min)		
		a	*b*	*c*	*a*	*b*	*c*	*a*	*b*	*c*
24	3	15.3	1·5	6·0	72	53	65	0·66	0·11	0·34
25	1	5·9	3·3	4·7	43	40	46	0·53	0·31	0·40
—	2	5·9	0·9	—	64	56	—	0·32	0·06	—
27	2	20·0	11·8	14·2	47	38	38	1·22	0·85	1·02
—	3	18·7	7·2	10·6	45	—	32	1·18	—	0·84
—	3	15·5	4·5	9·6	47	32	46	0·95	0·36	0·59
—	3·5	17·9	4·4	7·9	51	32	37	1·00	0·35	0·58
—	4	16·2	2·4	—	46	39	—	1·00	0·17	—
29	5	12·8	7·5	10·5	61	59	58	0·74	0·46	0·66
30	5	22·5	9·3	12·0	47	47	37	1·73	0·71	1·10

artery and vein. In addition to this, occlusion of the arterial inflow resulted in cessation of the outflow from the superior mesenteric vein. In the other two infusions arterial inflow was not recorded, but noradrenaline again produced a fall in venous outflow of a similar magnitude to that reported for the arterial circulation and changes in portal vein, oxygen content and oxygen consumption were also similar. For example, a reduction in venous outflow from 18·2 to 5·3 ml./min was accompanied by a change in oxygen consumption from 1·22 to 0·44 ml./min.

Noradrenaline and pH. It has been shown by Bygdeman (1963) that a fall in pH is accompanied by a decreased sensitivity of the blood vessels to noradrenaline. In order to determine whether this could be the cause of 'autoregulatory escape' the pH of the venous blood was measured during three of the infusions previously described. The pH values in the three experiments were 7·36, 7·21 and 7·19 before the infusions were begun and 7·31, 7·21 and 7·22 respectively during the period of maximum reduction in flow. It seems unlikely, therefore, that pH changes are responsible for the production of 'autoregulatory escape'.

Effect of acute haemorrhage on intestinal blood flow

In view of the 'autoregulatory escape' observed with noradrenaline infusion and sympathetic nerve stimulation, it seemed of interest to see if a similar phenomenon would occur with haemorrhage. The effect of haemorrhage on intestinal blood flow and oxygen consumption was measured in four experiments. Following blood flow determinations at about 90 mm Hg the blood pressure was rapidly lowered to 50 mm Hg (by allowing the animal to bleed into a reservoir at a controlled pressure) and maintained at this level for 4–6 min. In each animal 'autoregulatory

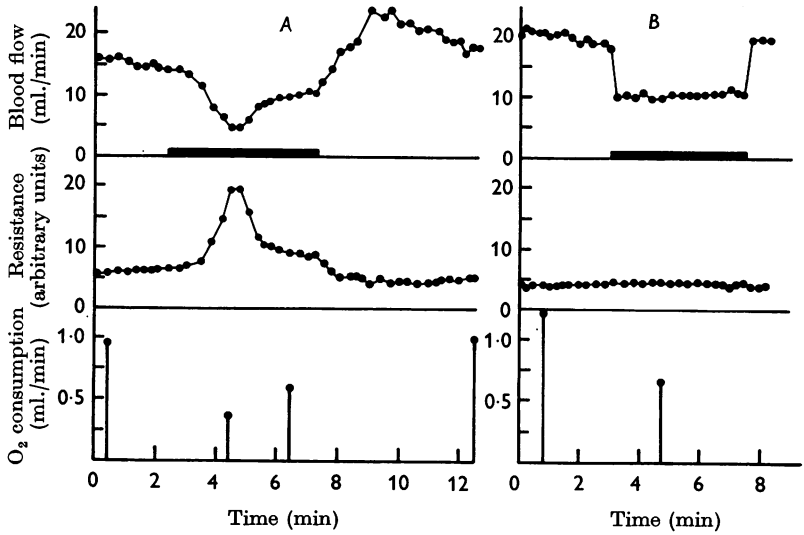

Fig. 2. (*A*) The effects of noradrenaline infusion and haemorrhage on intestinal blood flow, resistance and oxygen consumption. (*A*) Black bar—noradrenaline infusion—3 μg/min. (*B*) Black bar—haemorrhage—B.P. 50 mm Hg.

escape' had been demonstrated during a previous infusion of noradrenaline. In Fig. 2 a typical response during haemorrhage is contrasted with that during noradrenaline infusion. It can be seen that although there were appreciable reductions in flow in both experiments there was no escape of flow after haemorrhage and yet, in both experiments, oxygen consumption was reduced.

The failure to 'escape' after haemorrhage was somewhat surprising and the possibility was investigated that the low perfusion pressure prevented 'escape' from occurring. In two experiments, however, noradrenaline infused when the blood pressure was 50 mm Hg resulted in a decrease in flow, followed by 'escape'. A possible explanation of the failure of intestinal blood flow to 'escape' after haemorrhage could be that the

sympathetic activity to the intestinal arterial bed was not increased. This was supported by the observation that there was very little change in resistance (see Fig. 2) during haemorrhage in contrast to the increase in resistance with noradrenaline. The blood flow through the intestinal vessels was also reduced in three experiments by gradual occlusion of the inflow with a screwclip whilst the systemic blood pressure was held constant. The response was similar to that of haemorrhage, there being no 'escape' from the reduced blood flow but a reduction in oxygen consumption.

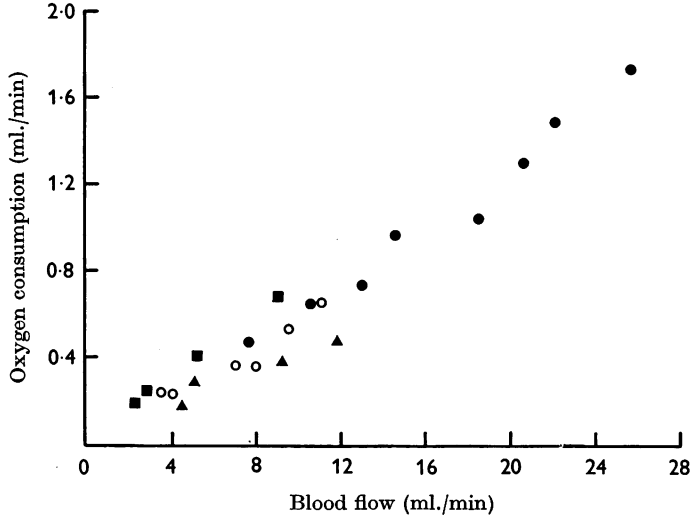

Fig. 3. The relationship between intestinal blood flow and oxygen consumption. ● Noradrenaline infusion. ■ Inflow occlusion with screwclip. ○ Haemorrhage before Rogitine. ▲ Haemorrhage after Rogitine.

In one experiment adrenergic blockage was carried out with 2-(N-*p*-tolyl-N-*m*-hydroxyphenylamino methyl)-imidazoline (Rogitine, Ciba Laboratories Ltd.—1 mg/kg body weight) and the blood pressure lowered rapidly to 50 mm Hg by bleeding. The results obtained were similar to those during haemorrhage before blockade in that there was no 'escape' of flow and oxygen consumption was reduced.

In all these experiments in which a decrease in intestinal blood flow was produced there was a reduction in oxygen consumption. Figure 3 shows the changes in oxygen consumption from one cat in which the flow was altered by noradrenaline, occlusion of the arterial inflow and haemorrhage before and after Rogitine. It can be seen that oxygen consumption and flow are clearly related and that the method by which the flow was altered did not affect this relation.

DISCUSSION

The values which we obtained for resting blood flow through the bowel were variable and ranged from 6 to 32 ml./min (average 17 ml./min) at an average blood pressure of 90 mm Hg. Our results are comparable with those of Barcroft & Shore (1912) who measured flow in the portal vein by direct collection, the flows in eleven cats ranging from 9·6 to 28·6 ml./min (average 20 ml./min). However, our flow values are lower than those reported by Folkow, Lundgren & Wallentin (1963) who found venous outflows of 40–60 (ml./min) per 100 gram tissue at a pressure of 100 mm Hg from a loop of denervated intestine and also lower than the results recently reported by Greenway & Lawson (1966) who obtained flows ranging from 33 to 77 ml./min (average 53 ml./min) by means of an electromagnetic flowmeter around the superior mesenteric artery. The reason for this discrepancy is not clear but cannot be attributed to high resistances in our catheter system or to inadequate volume of perfusion from the carotid artery.

In the present work we have used arterial flow to calculate oxygen consumption. This may be criticized on the grounds (1) that the inflow need not be the same as the venous outflow and (2) that the portal blood sampled might not represent venous blood drained only from the territories perfused by the cannulated branch of the superior mesenteric artery. In two cats in which superior mesenteric venous outflow was measured directly it was clear that occlusion of the arterial inflow caused an immediate cessation of venous outflow. It is therefore unlikely that there was an appreciable non-intestinal contribution to the portal blood. The validity of our oxygen consumption determinations is further supported by the observation that similar reductions in oxygen consumption were obtained when the venous outflow was measured directly.

In the above experiments, 'autoregulatory escape', as described by Folkow *et al.* (1964*a*) was well substantiated. It occurred both during sympathetic nerve stimulation and noradrenaline infusion and therefore it cannot be argued that the observations of Folkow *et al.* (1964*a*) were due to exhaustion of the sympathetic nerves. The mechanism responsible for the escape is not clear, but we have eliminated a number of possibilities. Sidky & Bean (1958) have shown that decreased tonus and increased motility in the intestine augment blood flow, but we have excluded these as factors in the production of 'escape'. The possibility was investigated that escape could be due to decreased sensitivity to noradrenaline associated with a fall in pH. Bygdeman (1963) has shown that, in kidney and muscle circulations at least, the peripheral vasoconstrictor effect of noradrenaline decreased as the degree of acidosis increased. This could not

be responsible for our results as there was little change in pH in the venous blood.

It is evident that 'escape' is peculiar to noradrenaline infusion and splanchnic nerve stimulation as it was not observed during haemorrhage or obstruction of the arterial inflow with a screw clip. At first sight the failure of flow to 'escape' during haemorrhage is surprising. The calculated resistance to flow during haemorrhage, however, showed very little increase when the blood pressure was lowered to 50 mm Hg. This agrees with the findings of Selkurt & Brecher (1956), Levy (1958) and Friedman (1961) who also found little increase in resistance in the intestine during haemorrhage in the dog. Thus it would appear that during haemorrhage there was very little vasoconstrictor influence from which the flow could escape.

The oxygen consumption values of 1·1–1·3 (ml./min) per 100 gram found by Barcroft & Shore (1912) are comparable with our average initial level of 0·91 ml./min. They found, however, that oxygen consumption was relatively constant over a wide range of flows when they compared different cats.

When noradrenaline was infused into the intestinal circulation there was at first a reduction in flow which was associated with a fall in oxygen consumption. If it is accepted that the major uptake of oxygen occurs in the mucosa, it is likely therefore that during this period there was reduction in mucosal flow as suggested by Folkow *et al.* (1964*b*). It was clear, however, that during the period of 'autoregulatory escape' there was an increase in oxygen consumption. One explanation of this phenomenon may have been that the mucosa was once again being perfused with blood, which would not be in accord with Folkow's observation that during 'autoregulatory escape' it is possible to have a return of blood flow in the intestine without increased perfusion of the mucosa. On the other hand, if Folkow's concept is correct the increase in oxygen consumption during 'auto regulatory escape' must be taken to mean that increased perfusion of smooth muscle leads to an increased oxygen utilization which is not related to intestinal movement. In favour of the suggestion that the mucosa is again being perfused during 'autoregulatory escape' is the observation of Grim & Lindseth (1958) that in the dog intestine only 4% of the intestinal flow is carried by arteriovenous anastamoses. It seems uulikely, therefore, that such channels could carry 'escape' flows of the magnitude found in the present experiments.

When blood flow to the intestine was reduced, either by noradrenaline infusion, by haemorrhage or by restriction of the arterial inflow there was a fall in oxygen consumption. Selkurt & Brecher (1956) also found a decreased oxygen consumption accompanying decreased flow during

haemorrhage in dogs. They suggested that when the oxygen saturation of venous blood reaches a low critical level, areas of the splanchnic bed where flow is markedly reduced will receive blood in which the level of saturation is so low that no further oxygen extraction can occur. Although the average percentage saturation of our portal venous blood was 44 % when blood flows were minimal, it may be that if an extensive arteriovenous shunt existed the blood draining the mucosa would be more reduced than this. We cannot, therefore, exclude this possibility although the small shunt values reported by Grim & Lindseth (1958) make it unlikely.

There are two other possibilities. First this observation could be accounted for if there was a patchy shut-down of mucosal perfusion when intestinal blood flow was reduced such that some areas of mucosa were normally perfused and others devoid of blood. This would explain the relatively constant arteriovenous oxygen difference, the linear relationship between blood flow and oxygen consumption and the hyperaemia which was sometimes seen after noradrenaline infusions. If this were to be the case, the observation that the linear relationship between blood flow and oxygen consumption was not disturbed after Rogitine suggests that such a redistribution of blood would not be sympathetically controlled. Secondly, it may be that the demand by the intestine for oxygen is determined by the flow. Our present experiments do not help in distinguishing these possibilities.

REFERENCES

BARCROFT, J. & SHORE, L. E. (1912). The gaseous metabolism of the liver. *J. Physiol.* **45**, 296–306.

BYGDEMAN, S. (1963). Vascular reactivity in cats during induced changes in the acid-base balance of the blood. *Acta physiol. scand.* **61**, Suppl. 222, 1–65.

COHNHEIM, O. (1907–8). Die Arbeit der Darmmuskeln. *Hoppe-Seyler's Z. physiol. chem.* **54**, 461.

DAWES, G. S., MOTT, J. C. & VANE, J. R. (1953). The density flow meter, direct method for the measurement of blood flow. *J. Physiol.* **121**, 72–79.

FOLKOW, B., LUNDGREN, O. & WALLENTIN, I. (1963). Studies on the relationship between flow resistance, capillary filtration coefficient and regional blood volume in the intestine of the cat. *Acta physiol. scand.* **57**, 270–283.

FOLKOW, B., LEWIS, D. H., LUNDGREN, O. MELLANDER, S. & WALLENTIN, I. (1964*a*). The effect of graded vasoconstrictor fibre stimulation on the intestinal resistance and capacitance vessels. *Acta physiol. scand.* **61**, 445–457.

FOLKOW, B., LEWIS, D. H., LUNDGREN, O., MELLANDER, S. & WALLENTIN, I. (1964*b*). The effect of the sympathetic vasoconstrictor fibres on the distribution of capillary blood flow in the intestine. *Acta physiol. scand.* **61**, 458–466.

FRIEDMAN, J. J. (1961). Mesenteric circulation in haemorrhagic shock. *Circulation Res.* **9**, 561–565.

GREENWAY, C. V. & LAWSON, A. E. (1966). The effects of adrenaline and noradrenaline on venous return and regional blood flows in the anaesthetised cat with special reference to intestinal blood flow. *J. Physiol.* **186**, 579–595.

GRIM, E. & LINDSETH, E. O. (1958). Distribution of blood flow to the tissues of the small intestine of the dog. *Minn. Med.* **30**, 138–145.

LEVY, M. N. (1958). Influence of levarterenol on portal venous flow in acute haemorrhage. *Circulation Res.* **6**, 587–591.

SELKURT, E. E. & BRECHER, G. A. (1956). Splanchnic haemodynamics and oxygen utilisation during haemorrhagic shock in the dog. *Circulation Res.* **4**, 693–704.

SIDKY, M. & BEAN, J. W. (1958). Influence of rhythmic and tonic contraction of intestinal muscle on blood flow and blood reservoir capacity in dog intestine. *Am. J. Physiol.* **193**, 386–392.