MODERATE HYPOTHERMIA INDUCES A PREFERENTIAL INCREASE IN PANCREATIC ISLET BLOOD FLOW IN ANAESTHETIZED RATS

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Short title: Hypothermia and blood flow

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ABSTRACT

The aim of the study was to characterize the effects of induced moderate hypothermia on splanchnic blood flow, with particular reference to that of the pancreas and the islets of Langerhans. We also investigated how interference with the autonomic nervous system at different levels influenced the blood perfusion during hypothermia. For this purpose, hypothermia (body temperature 28°C) was induced by external cooling, whereas normothermic (37.5°C) anaesthetized Sprague-Dawley rats were used as controls. Some rats were pretreated with either propranolol, yohimbine, atropine, hexamethonium or a bilateral abdominal vagotomy. Our findings suggest that moderate hypothermia elicits complex, organ-specific circulatory changes, with increased perfusion noted in the pylorus, as well as the whole pancreas and the pancreatic islets. The pancreatic islets maintain their high blood perfusion through mechanism involving both sympathetic and parasympathetic mediators, whereas the increased pyloric blood flow is mediated through parasympathetic mechanisms. Renal blood flow was decreased and this can be prevented by ganglionic blockade and is also influenced by β-adrenoceptors.

Key words: Hypothermia – Islet blood flow – Pancreatic blood flow – Renal blood flow – Intestinal blood flow – Adrenal blood flow
INTRODUCTION

Mild to moderate hypothermia is used in clinical medicine to benefit from the imposed decrease in tissue metabolism and oxygen consumption (16, 28, 44). Thus, hypothermia will allow for more extended time periods of surgery without hypoxic damage to tissues (43, 45). Another condition when induced hypothermia might be of relevance is during harvesting of organs for transplantation from brain dead donors, *i.e.* patients with abnormal body temperature regulation (53). In addition to this, cold preservation solutions are flushed through the organs intended for implantation, leading to profound changes in flow distribution (47).

Hypothermia *per se* is associated with changes in blood flow in several organs, most commonly a decrease. This can be due to the decreased metabolism in itself, by affecting neural control and transmission (16, 28, 29, 44) or to changes in blood viscosity and hematocrit (27). The pancreatic islet vasculature responds differently from that of most other organs upon stimulation or inhibition of adrenoceptors. Thus, activation of \( \beta_2 \)-adrenoceptors stimulates insulin release but decreases islet blood flow (22, 25), whereas inhibition of \( \alpha_2 \)-adrenoceptors or all \( \beta \)-adrenoceptors increases islet blood flow (26). However, islet blood flow has not been studied during hypothermia.

Further, moderate hypothermia consistently reduces renal blood flow (9, 10). The effects on splanchnic blood flow are more controversial, and both increased and decreased blood perfusion have been reported (13, 27). In previous studies in anaesthetized rats moderate hypothermia, *i.e.* body temperatures within the range of 25-30°C (16), led to an unchanged (27), a reduced (19, 38) or increased blood flow (39). In combination with hypothermic cardiopulmonary bypass procedures there were no changes or decreases in intestinal mucosal blood flow and total pancreatic blood flow (31, 34, 37, 40, 42). In view of these divergent
results it is likely that several factors, including choice of experimental animal, anaesthesia etc. influence the measurements.

It has been known for long that cooling induces a sympathetically mediated generalized vasoconstriction in skin, fat and skeletal muscle (18, 46). It can be speculated that this may cause a redistribution of blood favoring the splanchnic vascular beds. It should be noted in this context that many steps of catecholamine synthesis and release (5, 41, 51) are affected by hypothermia. Furthermore, cooling may in itself alter the affinity of different drugs for their receptors (7, 54). It has been reported that there is an increased potency of noradrenaline as a constrictor at a body temperature of 28°C compared to normothermia (2). Furthermore, studies on adrenoceptors during hypothermia have demonstrated a hypersensitivity in $\beta_1$-receptors, but not $\beta_2$, (54), and that $\alpha_1$-adrenoceptor mediated vasoconstriction is attenuated, whereas that in response to $\alpha_2$-adrenoceptors is unaffected (12). It should be noted that both $\alpha$- and $\beta$-adrenoceptors are desensitized in hypothermia associated with cardiopulmonary bypass (46).

In view of the findings referred to above we wanted to further characterize the effects of induced moderate hypothermia on splanchnic blood flow, with particular reference to that of the pancreas and the islets of Langerhans. To further address the issue on whether the nervous system, and especially so sympathetic nerves (14), are involved in the hypothermia-induced blood flow changes we used pretreatment with different pharmacological agents and surgical vagotomy before the induction of hypothermia.

**MATERIALS AND METHODS**

**Animals**: Adult, male Sprague-Dawley rats weighing approximately 320 g from a local breeding colony (Biomedical Centre, Uppsala University, Uppsala, Sweden) were used in all experiments. The animals had free access to pelleted rat food (Type R3; Ewos AB,
Sollentuna, Sweden) and tap water until the night before the experiments, when food was withdrawn. The animals were divided into twelve groups. Two groups received each pretreatment as outlined below and one group in each pair was subjected to hypothermia, while the other group was investigated at normothermia. The experiments were reviewed and approved by the Uppsala municipal court animal research ethics committee.

**Surgical preparation and induction of hypothermia:** All animals were anaesthetized with an intraperitoneal injection of thiobutabarbital (Inactin™; Research Biochemicals, Natick, MA, USA; 120 mg/kg body weight), placed on an operating table with access to thermal pads programmed to maintain the body temperature at 38°C (control animals) or 28°C (hypothermic animals). After tracheostomy, polyethylene catheters were inserted into the ascending aorta, via the right carotid artery, and into the left femoral artery and vein. The catheter in the femoral vein was used to continuously infuse saline (6 ml/kg body weight/h) throughout the experiments, and the aortic catheter was used to monitor mean arterial blood pressure (MAP) by a transducer (PDCR 75/1; Druck Ltd., Groby, Leics., UK).

Hypothermia was induced by placing plastic gloves filled with ice around the body of the animals. Care was taken to keep the plastic dry at all times. After 10-15 min the body temperature had dropped to 28°C, and was then maintained at this level by the thermal pad preset to this value. Registration of body temperature was made by a thermistor probe inserted into the descending colon. Body temperature was kept at 28°C or >37°C for at least 20 min before blood flow measurements were made.

**Pretreatment of animals:** Before being subjected to cooling or control procedures, but after induction of anesthesia, the animals were treated according to one of the protocols outlined below. All pharmaceutical agents (from Sigma Chemicals Co., St. Louis, MO unless given otherwise) were dissolved in saline and given as a bolus iv injection (1 ml/kg body weight) after surgical preparation, but before commencing hypothermia induction Atropine, a
parasympatolytic acetyl choline muscarinic receptor blocker (10 mg/kg body weight); hexamethonium, which blocks nicotinic acetyl choline signalling in autonomic ganglia (10 mg/kg body weight); propranolol, a non-selective β-adrenecptor antagonist (10 mg/kg body weight); yohimbine, an α₂-adrenoceptor antagonist (2.5 mg/kg body weight); abdominal vagotomy, which was performed by isolating and dividing the vagus nerves directly below the diaphragm thus parasympathetically denervating all abdominal organs (17). Control animals received only saline.

**Blood flow measurements:** The experiments were performed according to a protocol previously described in detail (24). Briefly, after a stable induction of body temperature had been achieved (see above), 1.5-2.0 x 10⁵ non-radioactive microspheres (E-Z Trac™; ITM Products, San Diego, CA, USA) with a mean diameter of 10 μm were injected during 10 sec via the catheter placed with its tip in the ascending aorta. Starting 5 sec before the microsphere injection, and continuing for a total of 60 sec, an arterial blood sample was collected from the catheter in the femoral artery at a rate of approximately 0.30 ml/min. The exact withdrawal rate was determined in each animal by weighing the sample. After obtaining the reference sample, another blood sample was drawn for measurement of hematocrit, blood glucose and serum insulin concentrations. After the animals had been killed the pancreas and adrenal glands as well as samples from the pylorus, duodenum, ileum, colon and left kidney were removed, blotted and weighed. Samples from the kidney was obtained by cutting a thin slice through the kidney at a right angle to the hilar region Tissue samples were then treated with a freeze-thawing technique to visualize the microspheres as previously described (23). The blood flow values were calculated according to the formula \( Q_{\text{org}} = Q_{\text{ref}} \times \frac{N_{\text{org}}}{N_{\text{ref}}} \) where \( Q_{\text{org}} \) is organ blood flow (ml/min), \( Q_{\text{ref}} \) is withdrawal rate of the reference sample (ml/min), \( N_{\text{org}} \) is number of microspheres present in the organ and \( N_{\text{ref}} \) is number of microspheres in the
reference sample. A difference <10% in blood flow values between the adrenal glands was used to confirm adequate mixing of the spheres in the circulation.

**Measurements of blood glucose and serum insulin concentration:** Arterial blood samples were obtained after securing the reference blood sample and later analyzed for blood glucose concentrations with a blood glucose meter (Medisense®; Svenska Medisense AB, Stockholm, Sweden) and serum insulin concentrations with ELISA (Rat Insulin ELISA®; Mercodia AB, Uppsala, Sweden) with rat insulin (Novo Nordic, Bagsværd, Denmark) as a standard.

**Statistical calculations:** All values are given as means ± SEM. Probabilities (P) of chance differences between the groups were calculated by Student's T-test between the normothermic and hypothermic saline treated animals for each organ. Differences between treatments were tested within either the normothermic or the hypothermic groups for each organ by ANOVA and paired t-tests with Bonferroni’s correction. Statistics were performed using R version 2.1.0 (49).

**RESULTS**

Of the total of 82 animals used in this study 6 were excluded, 3 due to uneven microsphere distribution and 3 because of uncontrolled shivering during hypothermia. Body temperature was almost exactly 28ºC in all animals in the hypothermic group (variation <0.2ºC) and 37.5ºC (variation <0.3ºC) in the normothermic group. Blood glucose concentrations were not affected by hypothermia per se (Table 1). The β-adrenoceptor blocker propranolol increased while vagotomy decreased blood glucose in both normothermic and hypothermic rats. Yohimbine lowered blood glucose in hypothermia, but had no effect in normothermia. Neither hexamethonium nor atropine pretreatment affected blood glucose. Serum insulin concentrations remained unchanged after hypothermia (Table 1). Hexamethonium increased serum insulin in normothermia, but had no effect in hypothermic rats.
Mean arterial blood pressure was not affected by hypothermia, but decreased after pretreatment with atropine, hexamethonium or propranolol in normothermic as well as in hypothermic rats (Table 1). Vagotomy or yohimbine elicited no change in MAP. Hematocrit values were increased in all hypothermic animals, except those pretreated with hexamethonium, where the value was lower (Table 1).

Pancreatic blood flow increased during hypothermia (Figure 1), and so did islet blood flow (Figure 2). Pancreatic blood flow was otherwise affected only during propranolol-pretreatment of hypothermic rats where a diminished blood flow was seen when compared to hypothermic control rats (Figure 1). Islet blood flow was increased by propranolol- and hexamethonium-pretreatment in normothermic rats (Figure 2). Administration of hexamethonium during hypothermia further augmented the islet blood flow increase, whereas no increase in islet blood flow was seen in hypothermic rats pretreated with atropine, propranolol, yohimbine or vagotomy (Figure 2).

Pyloric blood flow increased during hypothermia (Figure 3), a phenomenon which was prevented by all experimental interventions. However, both hexamethonium administration and vagotomy markedly decreased pyloric blood flow in both normo- and hypothermic rats. Duodenal blood flow was unaffected by hypothermia and showed no reaction to pharmacological interventions whereas vagotomy decreased blood flow in hypothermia (Table 2). Ileum and colon showed no changes in blood flow during hypothermia and their blood perfusion was unaffected by any of the given treatments (Table 2).

Hypothermic control animals had a lower renal blood perfusion than the corresponding control rats (Figure 4). Propranolol administration had no effect in normothermic rats, and did not affect the decrease in RBF during hypothermia.
Hypothermia decreased adrenal blood flow in all groups. In hexamethonium-treated rats there was no discernable difference between blood flow in the normothermic and hypothermic rats (Table 2).

**DISCUSSION**

We found that moderate hypothermia increased pyloric, total pancreatic and islet blood flow, whereas renal and adrenal blood flow were decreased and the other splanchnic organs had an unaffected blood flow.

The non-specific $\beta$-adrenoceptor antagonist propranolol increased islet blood flow in normothermic animals when compared to normothermic controls and the $\alpha_2$-adrenoceptor antagonist yohimbine produced a higher islet blood flow in normothermia, although the difference did not attain statistical significance. Hexamethonium also produced a strong increase of islet blood perfusion in normothermia in contrast to previous results in our hands in other rat strains (21). This may, at least partially, reflect the use of different rat strains. Total pancreatic blood flow in normothermia, on the other hand, was unaffected by any of the pretreatments given.

All pretreatments, besides hexamethonium, prevented the hypothermia-induced increase in total pancreatic blood flow. The hypothermia-induced islet blood flow increase was similarly prevented by all pretreatments, also in this case with the exception of hexamethonium. It should, however, be noted that especially the islet blood flow is already increased by several of the pretreatments given, and increased hypothermia-induced sympathetic stimulation may not be able to further add to this. With regard to total pancreatic blood flow this may be the case in propranolol-treated rats, since the flow value was decreased when compared to control hypothermic rats. In view of this, it seems as if both the parasympathetic and sympathetic nervous system may be involved in the increase in pancreatic and islet blood flow observed
during moderate hypothermia. Thus, it is a truly multifactorial response which is elicited in these animals.

In view of the blood flow responses to inhibition of sympathetic or parasympathetic nerves it is puzzling that the ganglionic blocker hexamethonium actually increases the blood perfusion. It can be speculated that during these functionally denervated conditions a shift in the balance between locally produced vasoconstrictors and vasodilators occurs. We have previously shown that pancreatic islets are extremely sensitive to both nitric oxide (48) and endothelin-1 (30), and to what extent hypothermia affects the effects of these substances certainly merits further investigation.

With regard to the blood flow to other parts of the gastrointestinal canal pyloric blood flow was increased by hypothermia, but no change was seen in the duodenum, ileum or colon. This is in contrast to some other studies in which a decreased (13, 42) or increased (39) mucosal intestinal or gastric blood flow, respectively, was observed during hypothermia. However, other studies have demonstrated an unchanged mucosal blood perfusion (27). The colon and ileum responded little to pretreatment, while the duodenum and pylorus both showed a decreased blood flow after vagotomy and the pylorus responded strongly also to hexamethonium, which is in line with earlier results (17). Thus, the major effects of hypothermia seem to be on the pylorus and pancreatic islets. From a teleological point of view this can be explained by the fact that ingestion of cold food should still be able to increase gastric and islet activity to facilitate digestion, and thereby also increase blood flow. Pyloric blood perfusion seems to be increased mainly through the parasympathetic system, whereas islet blood flow is stimulated through the sympathetic system.

The blood glucose concentrations were only marginally affected by any of the treatments given, and is unlikely to affect any of the observed blood flow values (20). There is a discrepancy between the changes in blood glucose and serum insulin in hexamethonium-
pretreated hypothermic rats, where insulin increases while blood glucose is unchanged. This is likely to reflect changes in insulin sensitivity caused by the increased sympathetic activity, or a direct release of glucose from skeletal muscle and/or liver.

Reduced basal insulin secretion in combination with blunted glucose-induced insulin secretion and constant or increased glucagon secretion leads to glucose intolerance in hypothermia (19, 32). The same pattern is observed in hibernating squirrels where somatostatin and pancreatic polypeptide has also been shown to be decreased (4). Somatostatin is known to inhibit both glucagon and insulin release (36, 52) and shows equal efficiency during shivering thermogenesis and control conditions in dogs. The combined evidence of increased glucagon and decreased insulin in rats in combination with decreased somatostatin in other models leads us to believe that pancreatic somatostatin secretion is decreased and that the imbalance between induced insulin and glucagon secretion seen in hypothermia and the slight decrease in insulin we show in non-stimulated conditions is not somatostatin related. This is consistent with our previous finding that the somatostatin analogue octreotide decreases islet blood flow (11), whereas the present results show an increase in hypothermia.

The present findings of a decreased renal blood flow during moderate hypothermia confirm previous results (9, 10). In the present study pretreatment with hexamethonium abolished the down regulation of renal blood flow in hypothermia, suggesting that the nervous system is involved in this response. However the results are somewhat ambiguous since the hexamethonium treated normothermic rats show a decreased blood flow at a level comparable with the saline treated hypothermic animals without attaining statistical significance. Propranolol elicited a renal blood flow decrease in hypothermic animals beyond what was seen in saline treated animals. This is compatible with recent results showing a beta-receptor mediated dilation in isolated renal afferent arterioles (50) and that they may be sensitized in
hypothermia (54). Most of these effects are likely to emanate from circulating catecholamines, since denervation does not affect hypothermia-induced decrease in renal blood flow (10). Indeed, previous studies in human kidneys have shown that intra-renal \(\alpha\)-adrenoceptors mediate a tonic renal vasoconstriction (1, 15, 35).

The adrenal glands demonstrated a marked attenuation of blood flow during hypothermia and this response was unaffected by administration of any of the tested substances. However, propranolol markedly increased adrenal blood flow in normothermic rats. The regulation of adrenal medullary and cortical blood flow is complex and mediated through both neural and metabolic mechanisms (6). The contribution of various transmitters in this context is controversial, and conflicting results have been presented (3, 6). The results in the present study suggest that \(\beta\)-adrenoceptors are involved in this process, but this awaits further experimental confirmation.

Blood pressure was, as expected, lowered by propranolol, atropine and hexamethonium (33). However, the changes in the vascular conductance of the studied organs mimicked those of the measured blood flow values (data not shown).

Hematocrit affects blood viscosity and is thereby an important factor in determining blood flow. The effect seen in the hypothermic animals in this study is in agreement with out earlier results (9, 10). We have previously calculated that this difference causes a 10% increase in viscosity (8) which would have minor effect on the results of the present study. Hexamethonium-pretreated rats did not demonstrate any increase in hematocrit, which may be due to an effect on the precapillary vasoconstriction.

We conclude that moderate hypothermia elicits complex and largely organ-specific circulatory changes, with increased perfusion noted in the pylorus as well as the whole pancreas and the pancreatic islets. The pancreatic islets maintain their high blood perfusion through mechanisms involving both sympathetic and parasympathetic mediators, whereas the
increased pyloric blood flow is mediated through parasympathetic mechanisms. Renal blood flow was decreased and we have provided evidence that this can be prevented by ganglionic blockade and that it is influenced by β-adrenoceptors.

ACKNOWLEDGEMENTS

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REFERENCES


**LEGENDS**

**Table 1:** Blood glucose, serum insulin, MAP and hematocrit values during normothermia (37.5°C) or hypothermia (28°C) in animals pretreated with an intravenous injection of either saline, atropine, hexamethonium, propranolol or yohimbine or surgical sub-diaphragmatic vagotomv. Values are means ± SEM for 7-8 experiments. $ denote P<0.05 between saline treated normothermic and hypothermic animals, * denote P<0.05 compared to saline treated normothermic control animals and # denote P<0.05 compared to saline treated hypothermic controls.

**Table 2:** Blood flow values for the duodenum, ileum, colon and adrenals during normothermia (37.5°C) or hypothermia (28°C) in animals pretreated with an intravenous injection of either saline, atropine, hexamethonium, propranolol or yohimbine or surgical sub-diaphragmatic vagotomy. Values are means ± SEM for 7-8 experiments. # denote P<0.05 compared to saline treated hypothermic controls.

**Figure 1:** Total pancreatic blood flow in normothermic (grey bars) and hypothermic (28°C; black bars) rats pretreated with an intravenous injection of either saline, atropine, hexamethonium, propranolol or yohimbine or surgical sub-diaphragmatic vagotomy. Values are means ± SEM for 7-8 experiments. $ denote P<0.05 between saline treated normothermic and hypothermic animals and # denote P<0.05 compared to saline treated hypothermic controls.

**Figure 2:** Pancreatic islet blood in normothermic (grey bars) and hypothermic (28°C; black bars) rats pretreated with an intravenous injection of either saline, atropine, hexamethonium, propranolol or yohimbine or surgical sub-diaphragmatic vagotomy. Values are means ± SEM for 7-8 experiments. $ denote P<0.05 between saline treated normothermic and hypothermic animals, * denote P<0.05 compared to saline treated normothermic control animals and # denote P<0.05 compared to saline treated hypothermic controls.
Figure 3: Pyloric blood flow in normothermic (grey bars) and hypothermic (28°C; black bars) rats pretreated with an intravenous injection of either saline, atropine, hexamethonium, propranolol or yohimbine or surgical sub-diaphragmatic vagotomy. Values are means ± SEM for 7-8 experiments. $ denote P<0.05 between saline treated normothermic and hypothermic animals, * denote P<0.05 compared to saline treated normothermic control animals and # denote P<0.05 compared to saline treated hypothermic controls.

Figure 4: Renal blood flow in normothermic (grey bars) and hypothermic (28°C; black bars) rats pretreated with an intravenous injection of either saline, atropine, hexamethonium, propranolol or yohimbine or surgical sub-diaphragmatic vagotomy. Values are means ± SEM for 7-8 experiments. $ denote P<0.05 between saline treated normothermic and hypothermic animals and # denote P<0.05 compared to saline treated hypothermic controls.
Table 1. Physiological characteristics of the different groups.

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Group</th>
<th>Blood glucose</th>
<th>Serum Insulin</th>
<th>MAP</th>
<th>Hct</th>
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<tr>
<td></td>
<td></td>
<td>mmol/L</td>
<td>ng/mL</td>
<td>mmHg</td>
<td>%</td>
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<td>Normothermia</td>
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<td>1.68 ± 0.21</td>
<td>115 ± 3</td>
<td>44.9 ± 0.6</td>
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<td></td>
<td>Hypothermia</td>
<td>6.8 ± 0.5</td>
<td>1.34 ± 0.17</td>
<td>110 ± 5</td>
<td>48.3 ± 1.2 $</td>
</tr>
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<td>Normothermia</td>
<td>6.3 ± 0.1</td>
<td>3.13 ± 0.30</td>
<td>79 ± 3 *</td>
<td>46.2 ± 0.6</td>
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<td>7.3 ± 0.1</td>
<td>1.51 ± 0.12</td>
<td>72 ± 3 #</td>
<td>49.2 ± 0.6</td>
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<td>Hexamethonium</td>
<td>Normothermia</td>
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<td>4.17 ± 0.29 *</td>
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<td>7.4 ± 0.5</td>
<td>1.69 ± 0.17</td>
<td>76 ± 2 #</td>
<td>44.6 ± 0.3 #</td>
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<td>Propranolol</td>
<td>Normothermia</td>
<td>6.9 ± 0.1 *</td>
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<td>1.77 ± 0.18</td>
<td>81 ± 10 #</td>
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<td>Normothermia</td>
<td>4.4 ± 0.1 *</td>
<td>3.45 ± 0.76 *</td>
<td>111 ± 6</td>
<td>44.9 ± 0.8</td>
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<td>Hypothermia</td>
<td>5.1 ± 0.1 #</td>
<td>1.18 ± 0.32</td>
<td>89 ± 2</td>
<td>46.8 ± 0.8</td>
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<td>Yohimbine</td>
<td>Normothermia</td>
<td>5.2 ± 0.1</td>
<td>2.73 ± 0.23</td>
<td>129 ± 4</td>
<td>44.0 ± 0.5</td>
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<td>4.8 ± 0.2</td>
<td>2.26 ± 0.29</td>
<td>124 ± 2</td>
<td>45.6 ± 0.5</td>
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## Table 2. Blood flow of different parts of the intestine and the adrenal glands.

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<th>Treatment</th>
<th>Group</th>
<th>Duodenal flow (ml/(min*g))</th>
<th>Ileal flow (ml/(min*g))</th>
<th>Colonic flow (ml/(min*g))</th>
<th>Adrenal flow (ml/(min*g))</th>
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<td>Saline</td>
<td>Normothermia</td>
<td>1.55 ± 0.34</td>
<td>0.73 ± 0.10</td>
<td>0.58 ± 0.15</td>
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<td>1.87 ± 0.13</td>
<td>1.18 ± 0.31</td>
<td>0.63 ± 0.09</td>
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<td>2.51 ± 0.18</td>
<td>0.86 ± 0.10</td>
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<td>31.7 ± 4.9</td>
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<td>Normothermia</td>
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<td>1.77 ± 0.36</td>
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<td>Normothermia</td>
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<td>1.00 ± 0.31</td>
<td>0.55 ± 0.10</td>
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<td>Normothermia</td>
<td>0.55 ± 0.14</td>
<td>1.11 ± 0.23</td>
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<td>0.49 ± 0.10</td>
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<td>1.38 ± 0.30</td>
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<td>Normothermia</td>
<td>2.28 ± 0.46</td>
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<td>Hypothermia</td>
<td>1.59 ± 0.19</td>
<td>0.54 ± 0.11</td>
<td>0.41 ± 0.05</td>
<td>15.7 ± 1.6</td>
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