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EFFECTS OF STIMULATION OF AORTIC CHEMORECEPTORS ON ABDOMINAL VASCULAR RESISTANCE AND CAPACITANCE IN ANAESTHETIZED DOGS

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SUMMARY

1. Dogs were anaesthetized with chloralose, ventilated artificially, and the regions of the aortic arch and carotid sinuses were isolated vascularly and perfused with blood. The abdominal circulation was isolated vascularly, perfused at constant flow and drained from the inferior vena cava at constant venous pressure. Changes in vascular resistance were determined by calculating changes in abdominal aortic perfusion pressure, and changes in capacitance by integrating the changes in venous outflow.

2. Stimulation of aortic body chemoreceptors, either by changing the aortic arch perfusate from arterial to venous blood at constant perfusion pressure or by injection of sodium cyanide into the aortic arch, resulted in an increase in abdominal vascular resistance and a decrease in abdominal vascular capacitance.

3. After both cervical vagosympathetic trunks had been cut, stimulation of aortic chemoreceptors no longer resulted in resistance or capacitance responses.

4. These results indicate that stimulation of aortic chemoreceptors, like carotid chemoreceptors, results in reflex constriction of both resistance and capacitance vessels in the abdominal circulation.

INTRODUCTION

The cardiovascular responses from stimulation of carotid chemoreceptors are now well described. These consist of negative inotropic and chronotropic responses of the heart (e.g. Hainsworth, Karim & Sofola, 1979) and constriction of resistance and capacitance vessels (e.g. Hainsworth, Karim, McGregor & Wood, 1983). There is much less information on the cardiovascular responses from aortic chemoreceptors. The responses that have been described are not invariably the same as those from carotid chemoreceptors. For example, we showed recently that stimulation of aortic chemoreceptors with venous blood, unlike stimulation of carotid chemoreceptors, usually results in positive inotropic and chronotropic responses of the heart (Karim, Hainsworth, Sofola & Wood, 1980). Daly, Hazzledine & Howe (1965) reported that stimulation of aortic chemoreceptors results in an increase in total systemic vascular resistance. However, there has been no study so far of the responses of resistance or capacitance vessels in individual vascular beds.

The abdominal circulation is the region which is probably of greatest importance in respect of vascular capacitance responses (see Hainsworth & Linden, 1979). The reflex responses of abdominal vascular capacitance to stimulation of carotid baroreceptors or stimulation of splanchnic sympathetic nerves are only slightly less than the responses of the whole body (Shoukas & Sagawa, 1973; Hainsworth & Karim, 1976; Karim & Hainsworth, 1976). Because of the potentially important role of the abdominal circulation, we undertook this study in which resistance and capacitance responses in this region were studied in response to stimulation of the vascularly isolated aortic chemoreceptors.

METHODS

Dogs (weighing 20–33 kg) were anaesthetized with chloralose (100 mg kg^{-1}, Cambrian Chemicals Ltd., Croydon) infused through a catheter inserted, under local anaesthesia (2% amethocaine) through a saphenous vein, so that the tip of the catheter lay in the inferior vena cava. Surgical anaesthesia was maintained by further infusions of chloralose (10 mg kg^{-1}) at approximately 20 min intervals. A mid-line incision was made in the neck. The trachea was cannulated and the lungs were ventilated by positive pressure with 40% oxygen in nitrogen by means of a Starling Ideal pump. The pump rate was 18 strokes min^{-1} and the stroke volume approximately 17 ml. kg^{-1}. When the pleura was opened an expiratory resistance was applied by placing the expiratory outlet from the pump under 3 cm of water. The expiratory outlet was occasionally occluded for three or four pump strokes to expand collapsed lung.

The method of isolation of the abdominal circulation and the measurement of resistance and capacitance have been described previously (Hainsworth & Karim, 1976; Karim & Hainsworth, 1976). Briefly, the abdomen was not opened but was isolated vascularly by cutting or tying all structures above the diaphragm except the sympathetic trunks. The hind limbs were excluded from the perfusion by use of strong nylon snares tightened at their upper ends.

The common carotid arteries and all their main branches were dissected free, with particular care being taken to avoid damage to the sinus nerves. Threads were placed loosely round all these vessels. The aorta was mobilized by tying and cutting the upper and lower four pairs of intercostal arteries. The left subclavian artery was dissected for about 1 cm, and loose soft threads were placed round the brachiocephalic artery and between the ascending aorta and pulmonary artery. During this dissection particular care was taken to avoid damage to branches of vagus and sympathetic nerves. The perfusion circuit (capacity about 1·5 l.) was filled with a mixture of approximately 2 parts of blood, obtained from the animal about 2 weeks prior to the experiment, and 1 part of Krebs–Ringer solution. The haematocrit values of the stored blood and the dog's blood on the days of the experiments were $48 \pm 2{\cdot}2\%$ and $36 \pm 1{\cdot}7\%$ respectively. The animal was given heparin (500 i.u. kg^{-1} followed by 50 i.u. kg^{-1} every 30 min, I.V.) then the perfusion circuit (Fig. 1) was connected to the animal. Temporary bypasses were first connected between the proximal ends of a femoral artery and a carotid artery and between a femoral vein and a jugular vein, to allow perfusion of the abdomen during cannulation procedures. A curved stainless-steel cannula was inserted into the aortic arch to conduct the aortic blood flow into an arterial reservoir to which a constant pressure head was applied and from which the blood was distributed to the various parts of the circuit. A perfusion line connecting the arterial reservoir and the descending aorta delivered blood to the thoracic structures through the intact middle four or five pairs of intercostal arteries. The abdominal vascular bed was perfused at constant flow through a cannula inserted into the descending aorta immediately above the diaphragm. The abdominal blood drained, through a steel cannula inserted into the inferior vena cava, into a venous reservoir. This maintained inferior vena caval pressure constant. The blood from the venous reservoir was pumped into an external jugular vein. During cannulations of the aorta (2–3 min) the pressure in the abdominal aorta never fell below 5 kPa. After the circuit had been connected the abdominal circulation was perfused at constant flow through the abdominal aorta and the bypasses were clamped. The perfusion flow was adjusted so that the abdominal aortic pressure was approximately the same as systemic arterial pressure. The head was perfused by pumping blood through cannulae inserted into the distal end of the left subclavian artery and the proximal end of the left common carotid artery.

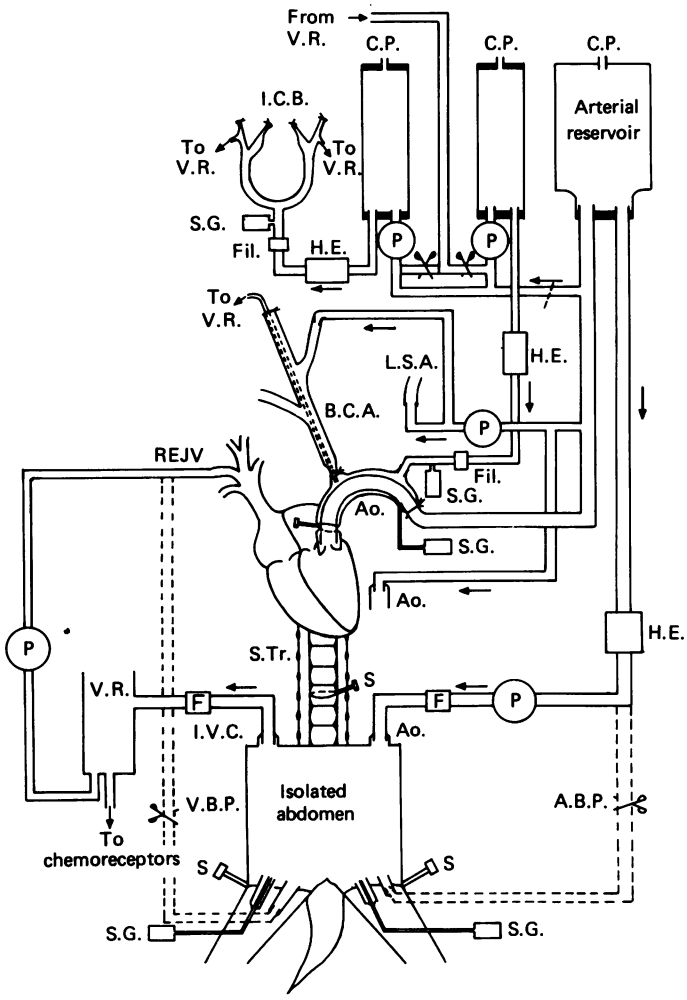

Fig. 1. Diagram of the experimental preparation. Blood entering the aortic arch passed through a curved cannula to be distributed to various parts of the circuit. Pressure in the aortic cannula and thoracic aorta was controlled by an arterial reservoir. Blood was pumped at constant pressure into the carotid arteries and drained into a venous reservoir from cannulae in the lingual arteries. The aortic pouch was perfused through the left subclavian artery and drained into the venous reservoir through a cannula in the brachiocephalic artery. The abdominal circulation was perfused at constant flow through the aorta immediately above the diaphragm and drained into a reservoir from the inferior vena cava. The cerebral circulation was perfused at constant flow through cannulae in the distal end of the left subclavian artery and the central end of the left common carotid artery. Symbols: A.B.P., temporary arterial bypass; Ao, aorta; B.C.A., brachiocephalic artery; C.P., constant pressure (provided by pressurized bottles); F, flow transducers; Fil., blood filters; H.E., heat exchangers; I.C.B., isolated carotid bifurcations; I.V.C., inferior vena cava; L.S.A., left subclavian artery; P, pumps; R.E.J.V., right external jugular vein; S, snares; S.G., strain gauges; S.Tr., sympathetic trunks; V.B.P., temporary venous bypass (only open during time of connexion of cannula to inferior vena cava); V.R., venous reservoir.

A pouch of the aortic arch, containing the arteries to most of the aortic chemoreceptors, was created by tying the aorta on to the cannula about 1 cm distal to the origins of the coronary arteries. The pouch was perfused through a cannula tied in the central end of the left subclavian artery and drained through a polythene cannula passed through the right common carotid artery to the brachiocephalic artery where it was secured by a tie.

A Y-shaped cannula was inserted into the cephalic ends of the common carotid arteries and the vascularly isolated carotid bifurcations were perfused at a constant non-pulsatile pressure. Blood was drained from the region through cannulae tied into the lingual arteries. The temperature of the blood perfusing the carotid, aortic arch and abdominal circulations was maintained at 38 °C by means of heat exchangers. The blood perfusing the chemoreceptors passed through filters (pore size 150 μm) to prevent emboli reaching the chemoreceptors.

The effectiveness of the vascular isolation of the carotid bifurcations, the aortic arch and the abdomen was tested by stopping the arterial inflow to each region and noting the perfusion pressure. In all cases the perfusion pressures fell to less than 1·5 kPa.

Blood pressures were recorded using Statham (P23Gb) transducers connected to cannulae inserted in the carotid arteries, in the central end of the left subclavian artery (aortic pouch pressure), through a femoral artery into the abdominal aorta (abdominal perfusion pressure), in the aortic cannula (systemic arterial pressure) and in the inferior vena cava through a catheter inserted via a femoral vein. Zero pressures were recorded at the ends of the experiments with the catheter tips free in air. The signals from the transducers passed to Elcomatic (EM760) amplifiers and were recorded on photographic paper using a direct-writing ultraviolet light oscillograph (SE Laboratories, model 2000). Mean pressures were obtained by use of R–C networks with time constants 2 sec incorporated in the amplifiers.

Blood flows into and out of the abdomen were recorded on the oscillograph using Biotronex Flowmeters (BL 410) with cannulating transducers. The flowmeters were calibrated at the ends of the experiments using the animal's own blood, and zero flows were checked periodically during the experiments by stopping the perfusion through the flow transducers and diverting it through bypasses (not shown in Fig. 1).

Arterial blood gases and pH were measured frequently during each experiment using standard glass electrode systems (Corning model 165). Oxygen was added to the inspired air if arterial P_{O_2} fell below 10 kPa. Arterial P_{CO_2} and pH were corrected, when necessary, to 4·6–6·0 kPa and 7·30–7·40 units respectively by adjusting the stroke volume of the respiratory pump and intravenous infusions of molar sodium bicarbonate. The actual values of blood gases and pH are listed in the Results.

A thermistor probe (Yellow Springs Instruments) was used to record oesophageal temperature which was maintained at 37–39 °C by use of heating elements under the operating table.

During the experimental period, spontaneous respiratory movements were prevented by the I.V. administration of suxamethonium chloride (0·5 mg kg^{-1} every 15 min). Chloralose infusions were continued at the same rate, and after about 1 hr suxamethonium chloride was stopped long enough for the level of anaesthesia to be assessed.

When all measured variables had reached steady states we confirmed that the preparation responded to stimulation of aortic baroreceptors by increasing aortic pouch pressure in a large single step. During all tests of stimulation of chemoreceptors the pressures in the aortic pouch and the carotid sinuses were held constant. Aortic body chemoreceptors were stimulated either by perfusing the aortic pouch with venous blood or by injecting sodium cyanide at various doses dissolved in NaCl solution (0·15 M). During venous perfusion, records were taken until a steady state was obtained (about 2 min). Following sodium cyanide injections, the outflow from the pouch was collected in a beaker and records continuously obtained for about 1 min.

Responses of abdominal aortic perfusion pressure were taken to be the steady state, or the peak changes compared with the means of control values during arterial perfusion before and after the period of stimulation. Capacitance responses were determined by integration, using a planimeter, of the area enclosed by the inferior vena caval outflow trace and a line either joining the means of the steady values before and during venous perfusion or continuing at the same level as that occurring before sodium cyanide injections (see Hainsworth & Karim, 1976).

All values reported are means $\pm$ one standard error of the means.

RESULTS

In all tests of baroreceptor and chemoreceptor stimulation ($n = 56$) performed in eleven dogs the mean value of abdominal aortic inflow was 887 ± 55 ml. min^{-1}, vena caval outflow 877 ± 56 ml. min^{-1} and inferior vena caval pressure $0{\cdot}51 \pm 0{\cdot}08$ kPa. Carotid sinus pressure was held constant at $17{\cdot}7 \pm 0{\cdot}44$ kPa and, except during aortic baroreceptor tests, the aortic pouch was perfused at $21{\cdot}9 \pm 0{\cdot}6$ kPa.

Responses from large step increases in aortic arch pressure

In eleven tests in eleven dogs an increase in aortic pouch pressure from $11{\cdot}8 \pm 1{\cdot}8$ kPa to $35{\cdot}7 \pm 2{\cdot}3$ kPa resulted in a decrease in abdominal perfusion pressure of $2{\cdot}8 \pm 0{\cdot}61$ kPa from $12{\cdot}7 \pm 1{\cdot}8$ kPa ($-20{\cdot}8 \pm 2{\cdot}2$ %; $P < 0{\cdot}001$). There was also a transient decrease in vena caval outflow which on integration signified an increase in the volume in the abdominal circulation of $15{\cdot}8 \pm 4{\cdot}1$ ml. ($0{\cdot}62 \pm 0{\cdot}15$ ml. kg^{-1}; $P < 0{\cdot}01$). Systemic arterial pressure (changes in which were reduced by the arterial reservoir) decreased from $12{\cdot}2 \pm 1{\cdot}2$ kPa to $10{\cdot}8 \pm 1{\cdot}1$ kPa ($P < 0{\cdot}01$) and heart rate decreased by $23{\cdot}6 \pm 8{\cdot}0$ beats min^{-1} from $174{\cdot}7 \pm 15{\cdot}0$ beats min^{-1} ($P < 0{\cdot}02$).

Responses to perfusion of aortic chemoreceptors with venous blood

The values of P_{O_2}, P_{CO_2} and pH, determined from samples of the blood perfusing the aortic pouch during each test ($n = 18$), were $10{\cdot}1 \pm 0{\cdot}78$ kPa, $5{\cdot}1 \pm 0{\cdot}29$ kPa, $7{\cdot}40 \pm 0{\cdot}02$ units during arterial perfusion and $3{\cdot}9 \pm 0{\cdot}20$ kPa, $6{\cdot}5 \pm 0{\cdot}51$ kPa, $7{\cdot}33 \pm 0{\cdot}02$ units during venous perfusion respectively.

In eighteen tests in eight dogs, stimulation of aortic chemoreceptors with venous blood resulted in an increase in abdominal perfusion pressure and a transient increase in vena caval outflow. In eleven tests aortic perfusion pressure reached a new steady level, but in seven of the tests it did not reach a steady state and the peak response obtained 166 ± 8 sec from the start of venous perfusion was analysed. The mean response of perfusion pressure, calculated from the averages of all tests performed in each dog, was an increase of $1{\cdot}1 \pm 0{\cdot}4$ kPa from $11{\cdot}2 \pm 1{\cdot}5$ kPa ($+10{\cdot}6 \pm 4{\cdot}2$ %; $P < 0{\cdot}01$). Vascular capacitance decreased by $22{\cdot}5 \pm 3{\cdot}2$ ml. ($0{\cdot}88 \pm 0{\cdot}09$ ml. kg^{-1}; $P < 0{\cdot}001$). Systemic arterial pressure, changes in which were partly buffered by the arterial reservoir, increased by $2{\cdot}0 \pm 1{\cdot}2$ kPa and heart rate increased by $2{\cdot}4 \pm 1{\cdot}7$ beats min^{-1}.

The averages of one to three responses obtained in each dog are listed in Table 1. The responses varied quantitatively, not only between dogs but within individual dogs. Fig. 2 shows contrasting responses obtained from different dogs. In the upper traces perfusion with venous blood resulted in a larger than average capacitance response with only a small change in perfusion pressure, whereas the lower traces show a very small change in capacitance but a large change in perfusion pressure.

Responses to injection of sodium cyanide into the aortic pouch

In eight dogs, including five of those in which aortic chemoreceptors were stimulated with venous blood, the responses to injection into the aortic pouch of sodium cyanide were studied. The sodium cyanide was dissolved in 0·2 ml. NaCl solution (0·15 M). Injection of 0·2 ml. NaCl solution had no effect.

TABLE 1. Responses to stimulation of aortic body chemoreceptors with venous blood

Dog	Systemic arterial pressure (kPa) Control	Change	Abdominal blood flow (ml. min^{-1})	Perfusion pressure (kPa) Control	Change	Capacitance change (ml.)
1	10·3	0	1169	8·1	+1·4	−32
2	8·7	+0·81	360	12·5	+0·5	−20
3	12·5	+2·9	484	20·7	+3·3	−17
4	10·5	+0·4	727	12·1	−0·3	−11
5	11·8	+0·5	767	9·4	+0·3	−12
6	11·3	+0·5	1157	10·7	+0·6	−27
7	9·6	+0·5	1422	8·6	+0·5	−36
8	18·2	+10·2	403	7·4	+2·6	−25
Mean	11·6	+2·0	811	11·2	±1·1	−22·5
S.E.	±1·0	±1·2	±141	±1·5	±0·4	±3·2
P	—	> 0·05	—	—	< 0·05	< 0·001

Control values were obtained during arterial perfusion of the aortic pouch before and after each test of venous perfusion. Change indicates response to venous perfusion. Probabilities calculated from paired *t* test. Results are of averages for all tests performed in each dog.

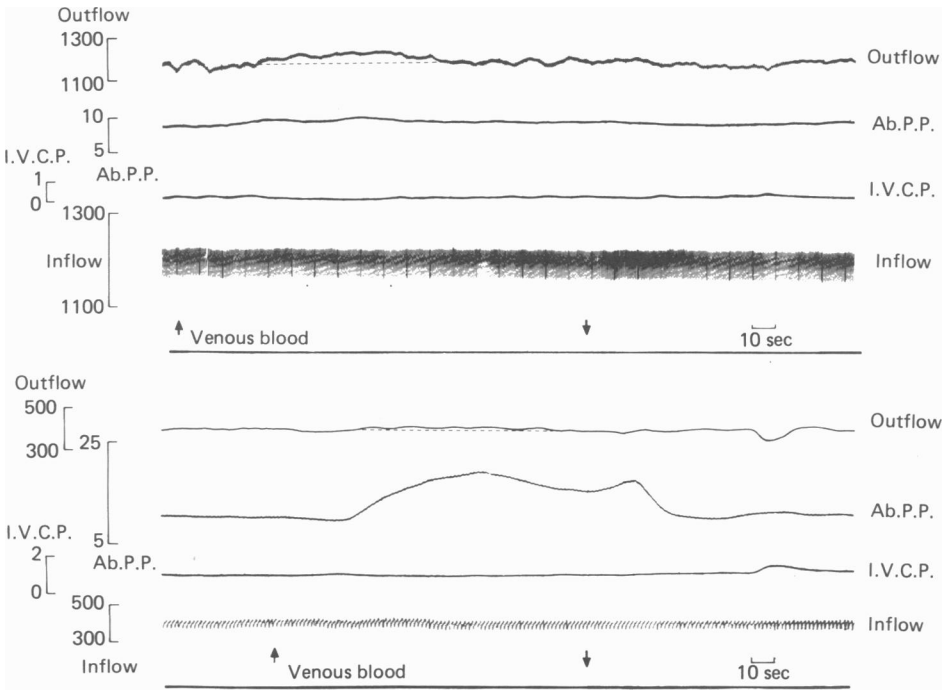

Fig. 2. Responses to stimulation of aortic chemoreceptors with venous blood. Traces obtained from two dogs. Symbols: Outflow, flow from inferior vena cava; Ab.P.P., abdominal perfusion pressure; I.V.C.P., inferior vena caval pressure; Inflow, abdominal aortic perfusion flow. Arrows indicate times of changing to venous perfusion and back to arterial perfusion. The areas enclosed by outflow traces and dotted lines were used to calculate changes in abdominal blood volume. In upper traces there was a decrease in abdominal capacitance of 54 ml. but an increase in perfusion pressure of only 0·5 kPa. In lower traces capacitance decreased by only 16 ml. but perfusion pressure increased by 4·1 kPa. Traces of aortic pouch, carotid and systemic arterial pressures have been removed from this Figure.

Injection of 2·5 mg of sodium cyanide, in twelve tests, resulted in an average peak increase in aortic perfusion pressure of $5{\cdot}0 \pm 1{\cdot}5$ kPa from $10{\cdot}8 \pm 1{\cdot}3$ kPa ($+56{\cdot}6 \pm 11{\cdot}3\%$; $P < 0{\cdot}01$). Since the injection was given as a bolus, the response was always transient. There was also an initial transient increase in vena caval outflow signifying expulsion of blood from the abdomen and this was followed by a decreased

TABLE 2. Responses to stimulation of aortic body chemoreceptors with sodium cyanide (2·5 mg).

	Systemic arterial pressure (kPa)		Perfusion pressure (kPa)		
Dog	Control	Change	Control	Change	Capacitance change (ml.)
2	7·0	+1·6	10·4	+2·9	−19
3	17·1	+0·2	15·2	+4·0	−27
5	16·5	+0·4	8·2	+12·6	−74
6	7·7	+0·2	8·6	+1·0	−21
7	10·0	+3·3	8·0	+2·5	−50
9	7·4	+0·2	18·4	+0·7	0
10	16·2	+1·0	17·8	+14·8	0
11	11·5	−0·1	6·9	+ 0·8	−10
Mean	11·7	+0·85	11·7	+4·9	−25·1
S.E.	±1·5	±0·40	±1·7	±2·0	±9·0
P	—	< 0·05	—	< 0·05	< 0·05

Responses are of averages of all tests performed in each dog. Control values were obtained during normal arterial perfusion of the aortic pouch. Change indicates peak responses following injection of sodium cyanide. Probabilities calculated from paired *t* test.

outflow signifying retention of blood. The average initial expulsion of blood was calculated to be $30{\cdot}2 \pm 7{\cdot}6$ ml. ($1{\cdot}2 \pm 0{\cdot}31$ ml. kg^{-1}), ($P < 0{\cdot}005$). There were no secondary responses due to sodium cyanide reaching other chemosensitive areas until the effluent from the pouch, which was collected in a beaker, was re-infused.

An example showing transient increases in arterial perfusion pressure and in vena caval outflow, in response to injection of 2·5 mg sodium cyanide, is shown in Fig. 3 and the results averaged from eight dogs are listed in Table 2.

Responses of heart rate were studied in five of the dogs. The most consistent finding was that there was a decrease in heart rate counted over the 10 sec period during which response of perfusion pressure was maximal. In three of eight tests heart rate responses were biphasic. In two tests, heart rate increased by 9 and 6 beats min^{-1} during the first 15 sec of the vascular responses, and in the other test it increased by 13 beats min^{-1} 40–60 sec from the onset of the response, when the resistance response was declining.

In eleven tests in six dogs injection of 6·25 mg sodium cyanide (dissolved in 0·5 ml. NaCl solution resulted in a mean peak increase in aortic perfusion pressure of $7{\cdot}6 \pm 2{\cdot}4$ kPa from $12{\cdot}2 \pm 1{\cdot}2$ kPa ($+61{\cdot}5 \pm 17{\cdot}8\%$; $P < 0{\cdot}01$). There was also a transient increase in vena caval outflow resulting in a calculated decrease in capacitance of $40{\cdot}5 \pm 10{\cdot}5$ ml. ($1{\cdot}7 \pm 0{\cdot}4$ ml. kg^{-1}; $P < 0{\cdot}01$).

Effects of bilateral vagotomy on responses from aortic chemoreceptors

In four dogs, the responses to stimulation of aortic chemoreceptors with sodium cyanide (6·25 mg) were compared before and after cutting both vagosympathetic trunks in the neck. With the vagi intact there was an average increase in perfusion pressure of $63 \pm 23\%$ and a decrease in capacitance of $35{\cdot}6 \pm 13{\cdot}2$ ml. ($1{\cdot}3 \pm 0{\cdot}4$ ml. kg^{-1}). After vagotomy both responses were abolished.

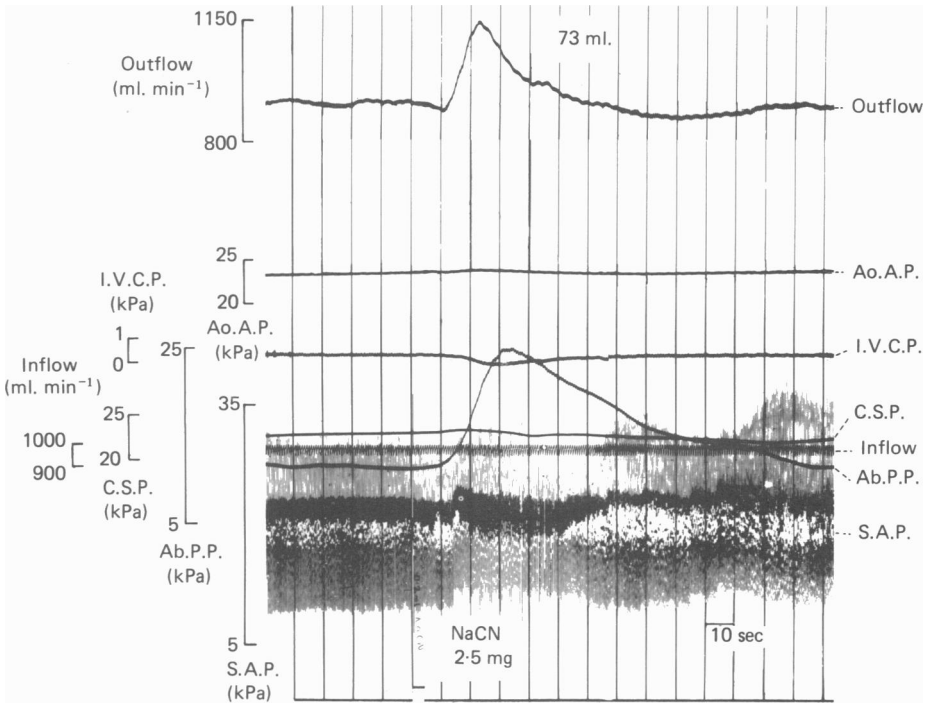

Fig. 3. Responses to stimulation of aortic chemoreceptors with a bolus injection of 2·5 mg sodium cyanide. Symbols: Outflow, flow from inferior vena cava; Ao.A.P., aortic arch pressure; I.V.C.P., inferior vena caval pressure; C.S.P., carotid sinus pressure; Inflow, abdominal aortic flow; Ab.P.P. abdominal perfusion pressure; S.A.P., systemic arterial (aortic cannula) pressure. Following the injection of sodium cyanide into the aortic pouch perfusate there were transient increases in abdominal perfusion pressure and in venous outflow. The initial capacitance response was calculated to be 73 ml.

DISCUSSION

The technique of creating a pouch of the aortic arch effectively isolates the perfusion to most of the aortic chemoreceptors. However, those chemoreceptors which are perfused from the brachiocephalic artery and from the left coronary artery (Comroe, 1939; Howe, 1956; Coleridge, Coleridge & Howe, 1970) would not be stimulated by the present technique. It is unlikely that the stimulus would have affected any adjacent structures because we showed that the aortic pouch was

effectively isolated vascularly. The only structures other than chemoreceptors to be exposed to the stimulus would be the aortic baroreceptors and is it unlikely that these would have been responsible for the responses (see Karim *et al.* 1980).

The perfusion circuits were filled with a mixture of heparinized blood, which had been taken from the animal about two weeks earlier, and Krebs–Ringer solution. This technique was adopted to avoid the necessity of diluting the animal's blood with a volume of fluid about two thirds of the estimated blood volume. Following the withdrawal of blood, the volume was replaced with dextran solution (Dextraven 150; Fisons) and an injection of iron sorbitol (2 ml. Jectofer, Astra) was given I.M. The haematocrit of the dogs at the time of the experiment was, on average, 36 %.

The pressure perfusing the aortic arch was held constant throughout the experiments at an average value of 21·9 kPa. We used this rather high value because, owing to the dead space in the circuit and the aortic pouch and the high resistance to blood flow from the pouch, the latency of the responses to the perfusion with venous blood was otherwise rather long. The delay in the reflex responses from stimulation of aortic chemoreceptors may also be partly due to the long response time of aortic chemoreceptors (Lahiri, Nishino, Mulligan & Mokash, 1980).

The physiological stimulus of the aortic chemoreceptors is perfusion with hypoxic, hypercapnic and acidaemic blood and this was provided by changing the perfusate to venous blood. However, possibly because of the relatively small number of chemoreceptors stimulated in this preparation, the responses to venous perfusion were frequently small and slow in onset. For this reason we also tested the effect of stimulation with sodium cyanide. This, although unphysiological, is known to provide an intense stimulus to chemoreceptors and, in our preparation, resulted in responses which were larger and of a more rapid onset than those caused by venous blood.

The vascular responses were studied in the abdominal circulation which we showed to be effectively isolated from the rest of the circulation. Because aortic flow and inferior vena caval pressure were constant we were able to regard the changes in perfusion pressure as providing an index of changes in vascular resistance. Also, since there was no flow into the region other than the constant flow perfusion of the aorta, we were able to regard changes in outflow from the inferior vena cava as indicating changes in abdominal vascular volume which, under conditions of constant flow and constant venous pressure, can be regarded as being due to active capacitance responses (Hainsworth & Linden, 1979).

The results obtained from this study indicate that stimulation of aortic chemoreceptors results in constriction of both resistance and capacitance vessels in the abdominal circulation. These experiments do not, however, provide information on the relative contribution of the various organs within the abdomen to the over-all response because we were not able to study this with the abdomen unopened. Furthermore, because the over-all response was small, it is unlikely to be very useful to attempt to analyse its components.

The responses to stimulation of aortic chemoreceptors with venous blood were smaller than the responses obtained in previous experiments to stimulation of aortic or carotid baroreceptors (Hainsworth & Karim, 1976; Karim, Hainsworth & Pandey, 1978). However, in the present experiments the responses to aortic baroreceptor

stimulation were also small, which may be related to the relatively high pressure perfusing the carotid baroreceptors (see Karim *et al.* 1978). The finding that capacitance responses were small does not imply that they are necessarily of little importance. The preparation involved very extensive dissection, particularly in the thorax, and it is possible that some damage to chemoreceptors or their nerves may have occurred. Furthermore, it is likely that larger responses would have been obtained if the chemoreceptors perfused by the brachiocephalic and left coronary arteries had also been stimulated.

There is little previous information on the vascular responses from stimulation of aortic chemoreceptors. Braunwald, Ross, Kahler, Gaffney, Goldblatt & Mason (1963) reported that systemic hypoxia results in an increase in total systemic vascular resistance and a decrease in vascular volume. However, the mechanisms responsible for these responses were not examined in detail. Daly *et al.* (1965) localized the stimulus to aortic chemoreceptors and reported an increase in total systemic vascular resistance and an increase in the volume of blood in the perfusion circuit, which indicated that there was a reduction in intravascular blood volume. However, the regions responsible for the resistance change are not known and it is not known whether the volume changes represented true capacitance responses. Studies of responses to injection of chemicals such as sodium cyanide or nicotine into the ascending aorta (e.g. Comroe & Mortimer, 1964; Iizuka, Mark, Wendling, Schmid & Eckstein, 1970) are not conclusive since, even when delay coils are inserted in the carotid arteries, it is not possible to be certain which structures are actually being stimulated and it is possible that some of the drug may reach carotid chemoreceptors through collateral blood vessels. The present study provides the first conclusive evidence that stimulation of aortic chemoreceptors constricts resistance and capacitance vessels in the abdominal circulation.

In the present investigation the responses of heart rate to stimulation of aortic chemoreceptors with venous blood obtained were small and variable. This differs from our recent publication (Karim *et al.* 1980) in which we reported that there was a significant increase in heart rate. However, in the present study the operational trauma was greater and the initial heart rates were faster, which may have prevented further increases from occurring in some experiments. Stimulation with sodium cyanide resulted in a decrease in heart rate, although this was frequently preceded or followed by tachycardia. This is difficult to explain and may be related either to a different intensity of stimulation or to stimulation of a different population of receptors.

In conclusion, we have shown that resistance and capacitance vessels in the abdominal circulation constrict in response to stimulation of aortic chemoreceptors. These responses are thus qualitatively similar to those obtained from stimulation of carotid chemoreceptors.

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