BARORECEPTOR CONTROL OF REGIONAL HAEMODYNAMICS DURING HALOTHANE ANAESTHESIA IN THE DOG

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SUMMARY

Regional haemodynamic control by the cardiovascular baroreceptors was examined in dogs anaesthetized with 1 + MAC of halothane in oxygen (1%). The open-loop relationships between carotid sinus pressure (CSP) and regional haemodynamics in the iliac, renal, mesenteric, aortic and coeliac beds were examined before vagotomy, following vagotomy and following thoracotomy. Around the carotid sinus reflex set point, the ratio of the reflex decrease in systemic arterial pressure to an increase in CSP (reflex gain) was \(-0.744 \pm 0.089\) (mean \(\pm\) SEM): the latter increased to \(-1.275 \pm 0.093\) following vagotomy. Reflex resistance changes were greatest in the renal bed and least in the coeliac bed, reflecting blood flow homeostasis which was well preserved in the renal bed but minimal in the coeliac bed. Thoracotomy in the dogs in which vagotomy had been performed resulted in no significant changes in the dependent variables studied. It is concluded that, in these dogs anaesthetized with 1 + MAC of halothane, baroreceptor control of regional pressure flow relationships is well preserved.

The purpose of this study was to examine the state of the baroreceptor control of regional haemodynamics during halothane anaesthesia in the dog.

A variety of anaesthetic agents have been studied with respect to their interaction with several aspects of baroreceptor physiology. Such agents include chloralose (Brown and Hilton, 1956), sodium pentobarbitone (Vatner, Franklin and Braunwald, 1971), Althesin and thiopentone (MacKenzie et al., 1976), ketamine (McGrath, MacKenzie and Millar, 1975), methohexitone (Skovsted, Price and Price, 1970) and cyclopropane, ether and halothane (Biscoe and Millar, 1966). The data from such studies cannot be interpreted without a careful study of the experimental conditions (Bagshaw and Cox, 1975) and the methods used to examine the baroreceptor system. Similarly, a knowledge of the particular experimental model is essential to the critical evaluation of an anaesthetic agent, such as halothane, which may be used in clinical practice (Beaton, 1959; Millar and Biscoe, 1965; Bristow et al., 1969).

Many studies of halothane have examined either a single relationship such as that between carotid sinus pressure and carotid sinus nerve activity (Millar and Biscoe, 1965), or employed a closed-loop method, such as bilateral carotid artery occlusion (Beaton, 1959) or the reflex increase of heart period following a pharmacological pressor response (Bristow et al., 1969). The former, while important, do not necessarily give information about the overall control characteristics. Bilateral carotid occlusion is a unidirectional stimulus at one level of intensity which is attenuated rapidly by blood flow entering the cephalic end of the carotid tree via the Circle of Willis. The relationship of mean arterial pressure and heart rate may not necessarily be indicative of overall cardiovascular baroreceptor control. Other experimental variables are introduced because the cardiovascular effects of halothane are dependent upon the dose and the duration of administration and are affected by other variables such as P\(\text{CO}_2\), P\(\text{O}_2\) and pH (Deutsch et al., 1962; Biscoe and Millar, 1966; Smith and Smith, 1972; Steffey et al., 1974; Vatner and Smith, 1974).

The purpose of this study was to examine overall baroreceptor cardiovascular reflex control employing a physiological perturbation to the carotid baroreceptors, and studying a comprehensive range of dependent variables which might be relevant to anaesthetic practice.

METHODS

Eight mongrel dogs of either sex, with an average weight of 26.3 ± 1.9 kg were studied. The dogs were premedicated with morphine sulphate 0.1 mg/kg i.m., atropine sulphate 0.5 mg i.m. and anaesthesia was induced with sodium thiamylal 250 mg i.v. The
trachea was intubated with a cuffed endotracheal tube and positive pressure ventilation was performed using a constant-volume ventilator (Ventimeter). Halothane in oxygen in concentrations sufficient for surgical anaesthesia was given during the experimental preparation. Following this, the inspired halothane concentration was reduced to 1+ MAC (1%) and the animal was allowed to reach a steady state before data collection (approximately 2 h free from surgical stimulation). Minute ventilation, measured with a Dräger volumeter, was adjusted to maintain arterial Pco₂ tension between 4.7 and 5.3 kPa. End-tidal carbon dioxide was measured using an infra-red analyser (Godart). Periodic blood-gas tensions and pH were measured using an appropriate electrode. If any metabolic acidosis occurred it was corrected using sodium bicarbonate. Sensible and insensible fluid losses were replaced with balanced electrolyte solution (Normosol-R) and 0.5 N saline in 5% dextrose solution to maintain a control venous pressure of about 0.4–1.1 kPa with a urinary output of at least 1 ml.kg⁻¹.h⁻¹.

**Experimental preparation**

Initial pressure monitoring was recorded from a catheter in a side branch of the right femoral artery. The dog was then turned to its right side and an electromagnetic flow probe (Statham) was placed on the ascending aorta through the left third intercostal space. The chest was closed in layers around a chest tube, the pneumothorax evacuated and the lungs hyperinflated. The carotid sinuses were then isolated and prepared for perfusion through a midline cervical incision; the details of this procedure have been described in detail previously (Bagshaw, Iizuka and Peterson, 1971). At this stage of the preparation blood still perfused the sinuses via the common and internal carotid arteries. During the preparation of the carotid sinuses for isolation, cuff-type electromagnetic flow probes (Statham type Q) were placed on the right iliac, left renal, cranial mesenteric and coeliac arteries through an abdominal midline incision. Mechanical zero flow was achieved using pneumatic occlusion devices (Rhodes Medical Instruments) placed distal to each flow probe. Blood flow to the hind paws was restricted by ligatures. Arterial pressures were recorded from: the femoral artery via a muscular side branch, ascending aorta via a catheter tip transducer (Statham SF-1) passed retrograde through the right common carotid following carotid sinus isolation, abdominal aorta via the thoraco–abdominal artery and the carotid sinus pressure via the perfusing system. A control venous pressure was obtained via the right external jugular vein.

Arterial and venous pressures were measured with strain-gauge transducers (Statham P23 Db or Gb). The arterial catheters were 20-cm polyethylene (i.d. 1.15 mm). Static pressure calibrations were made using a precision mercury barometer system (Hass Instruments). The amplitude response of the dynamic catheter–manometer characteristics was found to be flat within ±5% up to 20 Hz with a phase shift of approximately 2° at this frequency.

**Experimental protocol**

Following the abdominal surgery, closed-loop pressures and flows were recorded on magnetic tape (Sangamo Model 471) and displayed on a polygraph (Brush 260). Carotid sinus isolation was then completed and perfusion with an oxygenated physiological salt solution was started. A degree of back bleeding always occurred from vasa vasorum and blood vessels of similar size; however, this was never sufficient to modulate the perfusing pressures. The perfusion pulse pressure was set at 40 mm Hg with a frequency of approximately 70 cycles/min (1.17 Hz): these variables were kept constant throughout the experiment. Mean carotid sinus pressure was then adjusted to be equal to the mean systemic pressure, giving a value which we called the closed-loop operating pressure, that is, the pressure around which the carotid sinus reflex operates in the intact animal. After the cardiovascular variables had reached a steady state, carotid sinus pressure was decreased to a mean pressure of about 50 mm Hg from the closed-loop operating pressure and the experimental variables recorded on analog tape in the steady state. This was repeated for carotid sinus pressures to about 250 mm Hg in increments of 30–50 mm Hg, returning to a mean pressure of about 50 mm Hg. This procedure was repeated following bilateral vagotomy and again following bilateral thoracotomy in the vagotomized dogs. The latter consisted of opening the left chest tube to air and placing a similar tube through a small incision in a corresponding area of the right chest. Pressure and flow zeros were taken at each phase of the experiment. Figure 1 shows an example of the data recording.

**Data analysis**

At each mean carotid sinus pressure for each experimental condition, pressure and flow signals
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CSP-50 CSP-91 CSP-116 CSP-142 CSP-177 CSP-220

Fig. 1. Example of on-line records of haemodynamic variables at different mean carotid sinus pressures. Variables in descending order are carotid sinus pressure (CSP), femoral artery pressure ($P_f$), aortic flow ($Q_a$), coeliac artery flow ($Q_c$), mesenteric artery flow ($Q_m$), renal artery flow ($Q_r$) and iliac artery flow ($Q_i$). All pressures are in mm Hg and all flows are in litre.min$^{-1}$.

were converted to digital form over 4–6 cardiac cycles as has been described previously (Cox, 1970). Mean aortic pressure for each experiment and each experimental condition was plotted against the mean carotid sinus pressure. A line of identity was drawn and its intersection with the above relationship was taken as the closed-loop operating pressure (CLOP), that is, the set point of the reflex around which arterial pressure is controlled in the intact animal (fig. 2). The relationships shown in figure 2 do not represent actual experimental data and are purely for illustrative purposes. Figures 3–9 represent the mean values of eight experiments. The gain of the carotid sinus reflex is the slope of a pressure-pressure relationship such as the one shown in figure 2, so that the slope at CLOP is the operating gain of the reflex—a measure of the ability of the carotid sinus to maintain the systemic arterial pressure at pressures around the reflex set point. Other haemodynamic variables co-existing at CLOP constitute the closed-loop operating resistances and flows, as illustrated in the lower part of figure 2 for aortic resistance. The slope of a line relating two dissimilar variables gives the sensitivity of one variable to changes in the other as opposed to the gain which is a relationship between two variables having identical units. Thus the slope at CLOP in the lower portion of figure 2 gives the sensitivity of total peripheral resistance (aortic resistance) to carotid sinus pressure. Because significant dog-to-dog variation with respect to the operating characteristics of the carotid sinus control of regional haemodynamics was found, all the data were standardized before averaging as follows. The value of each haemodynamic variable was divided by its operating point value. For example, if the coeliac blood flows at successive carotid sinus

![Graph](https://via.placeholder.com/150)

Fig. 2. A hypothetical relationship between aortic pressure and carotid sinus pressure (upper) and the corresponding aortic resistance and carotid sinus pressure (lower) to illustrate carotid sinus gain and sensitivity respectively. $P_{op}$ represents operating pressure and $AR_{op}$ represents operating resistance.
pressures were 390, 370 and 343 ml and the coeliac blood flow at the corresponding CLOP was 314 ml, then the above standardized coeliac flows would be 1.242, 1.178 and 1.092 respectively. Haemodynamic variables are expressed at carotid sinus pressures of 0.4-1.8 in increments of 0.2. The values at these CSP were obtained by three-point interpolation. Standardized values from the eight experiments were averaged at the incremental values of CSP. Finally, each haemodynamic variable resulting from increasing carotid sinus pressures was averaged with the corresponding value for decreasing carotid sinus pressures. Statistical significance between the various operating characteristics was assessed using a paired data analysis. All of the numerical analysis procedures were performed automatically on a digital computer (IBM 360/185).

RESULTS

Operating characteristics

The values of each of the dependent variables considered at the closed-loop operating point are summarized in table I, together with the sensitivities of these variables to changes in carotid sinus pressure at the operating point. These operating point values are given for the experimental condition of intact vagi, divided vagi and following a bilateral thoracotomy in the vagotomized dogs.

Bilateral vagotomy produced a significant ($P<0.05$) increase in the closed-loop operating pressure with no significant changes in cardiac output or heart rate. All five regional operating resistances increased following vagotomy, being significant in the case of the renal and mesenteric beds and the total peripheral resistance: the latter only for a paired analysis.

| Table I. Control, following vagotomy and following thoracotomy operating point values and sensitivities to changes in carotid sinus pressure |
|---|---|---|
| **Operating point values** | **Vagi intact** | **Vagi cut** | **Chest open and vagi cut** |
| AP (kPa) | $13.33 \pm 0.27$ | $15.47 \pm 0.4^*$ | $14.53 \pm 0.4^*$ |
| $Q_{aa}$ (ml.min$^{-1}$) | $2639 \pm 227$ | $2449 \pm 199$ | $2246 \pm 179$ |
| HR (beat.min$^{-1}$) | $135 \pm 7$ | $147 \pm 5$ | $149 \pm 6$ |
| $R_{ma}$ (dyne.s.cm$^{-1}$) | $3354 \pm 299$ | $4042 \pm 275^*$ | $4080 \pm 256^*$ |
| $R_m$ (dyne.s.cm$^{-1}$) | $19715 \pm 1034$ | $21137 \pm 1284$ | $21696 \pm 1475$ |
| $R_n$ (dyne.s.cm$^{-1}$) | $17375 \pm 1144$ | $21419 \pm 1793^*$ | $21262 \pm 1842^*$ |
| $R_{na}$ (dyne.s.cm$^{-1}$) | $21456 \pm 1780$ | $26691 \pm 1823^*$ | $26208 \pm 2043^*$ |
| $Q_{ma}$ (ml.min$^{-1}$) | $77006 \pm 8777$ | $103275 \pm 12532$ | $104801 \pm 13471$ |
| $Q_{ma}$ (ml.min$^{-1}$) | $424 \pm 35$ | $477 \pm 38$ | $421 \pm 33$ |
| $Q_{ma}$ (ml.min$^{-1}$) | $483 \pm 37$ | $472 \pm 42$ | $439 \pm 38$ |
| $Q_{ma}$ (ml.min$^{-1}$) | $406 \pm 39$ | $366 \pm 26$ | $347 \pm 25$ |
| $Q_{ma}$ (ml.min$^{-1}$) | $121 \pm 12$ | $114 \pm 14$ | $109 \pm 17$ |

| **Operating point sensitivities** | | | |
| AP | $-0.744 \pm 0.089$ | $-1.276 \pm 0.093^*$ | $-1.142 \pm 0.073^*$ |
| $Q_{ma}$ | $-0.219 \pm 0.064$ | $-0.357 \pm 0.074$ | $-0.282 \pm 0.063$ |
| HR | $-0.254 \pm 0.050$ | $-0.184 \pm 0.027$ | $-0.164 \pm 0.021$ |
| $R_{ma}$ | $-0.547 \pm 0.081$ | $-0.956 \pm 0.090^*$ | $-0.904 \pm 0.085^*$ |
| $R_m$ | $-0.042 \pm 0.092$ | $-0.378 \pm 0.108$ | $-0.241 \pm 0.097$ |
| $R_{ma}$ | $-0.211 \pm 0.068$ | $-1.009 \pm 0.094^*$ | $-0.971 \pm 0.136^*$ |
| $R_n$ | $-0.779 \pm 0.128$ | $-1.455 \pm 0.143^*$ | $-1.250 \pm 0.143^*$ |
| $R_{na}$ | $-0.657 \pm 0.184$ | $-1.254 \pm 0.303$ | $-0.959 \pm 0.248$ |
| $Q_{ma}$ | $-0.742 \pm 0.099$ | $-0.886 \pm 0.116$ | $-0.914 \pm 0.120$ |
| $Q_{ma}$ | $-0.551 \pm 0.081$ | $-0.355 \pm 0.073^*$ | $-0.257 \pm 0.079$ |
| $Q_n$ | $-0.047 \pm 0.131$ | $-0.334 \pm 0.049$ | $+0.046 \pm 0.086$ |
| $Q_{na}$ | $+0.035 \pm 0.167$ | $+0.041 \pm 0.234$ | $-0.291 \pm 0.167$ |

$AP =$ mean arterial pressure; $Q_{aa}$ and $R_{aa} =$ ascending aorta flow and resistance; HR = heart rate; $Q_m$ and $R_m =$ coeliac artery flow and resistance; $Q_{ma}$ and $R_{ma} =$ mesenteric artery flow and resistance; $Q_n$ and $R_{na} =$ renal artery flow and resistance; $Q_{ja}$ and $R_{ja} =$ iliac artery flow and resistance. * Significantly different from the corresponding value for intact vagi ($P<0.05$). All values are represented by the mean ± 1 SEM.
Vagotomy produced no significant changes in the regional operating flows. At the operating point, dependent variable sensitivity to changes in carotid sinus pressure (CSP) was increased significantly by vagotomy in the case of aortic pressure, total peripheral, mesenteric and renal resistance, and mesenteric flow.

Following bilateral thoracotomy in the dogs in which vagotomy had been performed, changes in operating point values and their sensitivities were minimal and the measurements that were significantly different from control following vagotomy remained so following thoracotomy (P<0.05). However, thoracotomy per se produced no significant changes in any of the experimental variables.

Figure 3 shows standardized aortic pressure, resistance and flow together with heart rate as a function of changes in standardized CSP. These relationships as in all other figures are shown for control conditions, following vagotomy and following thoracotomy. All four variables showed the characteristic inverse relationship which was accentuated by vagotomy in the case of aortic pressure and total peripheral resistance. At increments of standardized CSP of 0.2 significant differences (P<0.05) between control and vagotomy, and control and thoracotomy are indicated by (*) and (‡) respectively. At CSP values greater than the operating point an increasing CSP produced less change in heart rate following vagotomy compared with control values. The corresponding slopes or sensitivities of the relationships shown in figure 3 are illustrated in figure 4. With the exception of cardiac output and heart rate before vagotomy the sensitivities varied in a bell-shaped fashion with changes in CSP. Peak sensitivities occurred at or around the operating point. Vagotomy increased significantly aortic pressure gain and sensitivity of total peripheral resistance, particularly at lower CSP values.

Standardized coeliac, mesenteric, renal and femoral resistances also showed inverse relationships to CSP although this was minimal in the coeliac bed with the vagi intact (fig. 5). These relationships were accentuated by vagotomy in all four beds, particularly at CSP values less than the operating point with some attenuation following thoracotomy in the vagotomized dogs. Regional resistance sensitivities showed a bell-shaped relationship to CSP particularly in the renal and mesenteric beds where, again, peak sensitivity occurred at or around the operating point.
point (fig. 6). In all four beds, vagotomy tended to increase sensitivity particularly at the lower CSP's.

In considering the relationship of bed blood flow to CSP, more bed-to-bed variation occurred (fig. 7). Iliac blood flow remained essentially unchanged with a changing CSP for all three experimental conditions. Coeliac flow decreased inversely with CSP; however, renal and mesenteric blood flow tended to remain constant or increased up to the operating point and then decreased. Vagotomy tended to accentuate these relationships in the renal and mesenteric beds, maximum flow occurring at or around the operating point. Figure 8 shows the corresponding flow sensitivities to CSP changes. Peak coeliac flow sensitivity occurred around the operating point, whereas operating flow sensitivity was minimal in the renal and iliac beds and to a lesser extent in the mesenteric bed. Similar relationships persist following division of regional blood flow by the appropriate cardiac output as seen in figure 9.

**DISCUSSION**

In the dog anaesthetized with 1+ MAC of halothane in oxygen, the cardiovascular baroreceptors have considerable ability to effect changes in the systemic arterial pressure. The effectiveness or gain is greatest in the region of the closed-loop operating...
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Figs. 8 and 9. Variation of regional flow sensitivity with standardized carotid sinus pressure. Open circles represent data before vagotomy, solid circles following vagotomy and crosses following thoracotomy. Vertical bars represent ±1 SEM at the closed-loop operating points.

pressure, that is the set point of the reflex, thus assuring maximum baroreceptor compensation to perturbation of pressure in this region. Reflex changes in pressure appear to be effected primarily by changes in total peripheral resistance, reflex changes in cardiac output being consistent but small. However, cardiac output and peripheral resistance did not change independently and some interaction must contribute to the changes seen.

The considerable potentiation of reflex aortic pressure and resistance changes by vagotomy suggests the considerable influence of the aortic or cardiopulmonary baroreceptors, or both, at the concentrations of halothane studied. Furthermore, the above potentiation was primarily at the lower carotid sinus pressures, suggesting that the aortic baroreceptors were still acting as high pressure receptors (Donald and Edis, 1971).

The relatively low sensitivity of heart rate to changes in CSP both before and after vagotomy are similar to those seen in earlier studies using the animal anaesthetic chloralose (Bagshaw, Iizuka and Peterson, 1971). The greatest heart rate sensitivity occurred at high carotid sinus pressures before vagotomy, which may reflect the slower heart rates in that condition. This tends to confirm our impression that heart rate control has characteristics different from other dependent variables such as pressure or resistance.

The ability of the baroreceptors to effect regional resistance changes showed considerable bed-to-bed variation, being minimal in the coeliac and mesenteric beds and highest in the renal bed: the iliac resistance changes being intermediate in magnitude. Similarly, vagotomy had differential effects, being greatest in the mesenteric and renal beds compared with the coeliac and iliac beds. The minimal changes in reflex coeliac resistance reflect the significant passive changes in coeliac blood flow, particularly around the reflex set point.

The experimental increases in CSP are accompanied by reflex decreases in systemic arterial pressure following inhibition of post-ganglionic sympathetic nerve activity to the heart and peripheral vasculature (Heymans and Neil, 1958). Thus, in the coeliac bed this decrease in pressure is accompanied by profound decreases in blood flow even when the vagi are intact and the aortic baroreceptors are active. However, following vagotomy and the negation of the aortic baroreceptor effects, the magnitude of the coeliac blood flow reduction is very similar. Thus, assuming that there are no other sites of significant baroreceptor activity, one can conclude that a decreasing systemic pressure in the intact closed loop is still accompanied by significant decreases in coeliac blood flow, that is, there is minimal baroreceptor homeostasis with respect to coeliac blood flow under
the conditions of this investigation. This has been found by several investigators using a variety of experimental conditions. For example, inspired halothane concentrations of 1–2% superimposed upon 70% nitrous oxide in oxygen and an indeterminate amount of pentobarbitone produced a 54% decrease in hepatic blood flow (Thulin, Andreen and Irestedt, 1975).

Conversely, the decreases in systemic arterial pressure following increases in CSP are accompanied by minimal changes in iliac and renal blood flow, particularly below the operating point. This relationship is relatively unchanged by the withdrawal of the aortic baroreceptors following vagotomy. Consequently, a decrease in arterial pressure during 1 MAC of halothane in the intact animal with maximal activation of the aortic and carotid baroreceptors, would be expected to change renal blood flow little, particularly around the reflex set point. This certainly seems to be the case in the study of Vatner and Smith (1974), who found that renal blood flow did not change significantly from awake control conditions for end-tidal halothane concentrations of 1% and 2%. The reflex blood flow changes seen in the regional beds under open-loop conditions are, of course, subjected to other homeostatic mechanisms, the most significant of which is autoregulation. However, the degree to which autoregulation contributes to the above changes cannot be deduced from this study. The regional blood flow changes are essentially unaltered when expressed as a percentage of cardiac output suggesting that the changes do not reflect merely reflex changes in cardiac output.

In considering the differential effects of vagotomy upon regional resistance as described above, differences were significant compared with pre-vagotomy values at all carotid sinus pressures for the coeliac, mesenteric and renal beds. The decreasing resistance and systemic arterial pressure accompanying the increase in carotid sinus pressure was diminished significantly by the aortic baroreceptors, particularly at the greater systemic arterial pressures (low CSP). Therefore, in the intact animal, with the carotid and aortic baroreceptors acting in concert, presumably there would be significant baroreceptor compensation for any direct depression of vascular smooth muscle in the regional beds by concentrations of halothane employed in this study.

Many cardiovascular studies which measure cardiac output directly using electromagnetic flow probes are performed in open-chest dogs. Chest closure following flow probe placement with subsequent bilateral thoracotomy was carried out in this study to observe whether thoracotomy per se had any effect upon cardiovascular baroreceptor control. At this time bilateral thoracotomy had been performed. At the inspired concentrations of halothane studied, bilateral thoracotomy in the vagotomized dogs produced no significant changes in any haemodynamic variable when compared with the post-vagotomy controls.

In conclusion, an anaesthetic agent could alter baroreceptor haemodynamic control by modulating any or all of the various modalities of the baroreceptor control loop. These include, for example, the baroreceptors themselves, the afferent nerves, central mechanisms, efferent sympathetic mechanisms and the end-organ smooth muscle. Thus, halothane, if given in sufficient amounts with or without additional basal anaesthetic agents, under a variety of experimental conditions, could depress each or all of the above reflex limb components (Hughes, 1973). Unfortunately, there are no comparable awake controls to which the experimental values reported here can be compared. However, a study has been performed in a different group of dogs anaesthetized with chloralose (Cox and Bagshaw, 1975). This produced maximum open-loop reflex gains which were less than in the present study both before and after vagotomy. In the amounts used, chloralose has been shown to remain, or even potentiate, the baroreceptor reflexes (Brown and Hilton, 1956). Therefore, in the dog anaesthetized with 1+ MAC of halothane in oxygen, both carotid and aortic baroreceptor control of peripheral haemodynamics appeared to be well preserved, at least compared with similar studies using chloralose. It is conceivable that, in spite of the back bleeding into the carotid sinuses, the halothane tension in the isolated sinuses was less than at other sites of the baroreceptor control loops. Such possible differences are unlikely to produce significant local modulation of the baroreceptors (Millar and Biscoe, 1965). Certainly no significant depression of the intact aortic baroreceptors occurred, as judged by the reflex effects of vagotomy. Systemic pressure reflex changes were effected primarily by changes in the peripheral vascular resistance with small reflex changes in heart rate and cardiac output. Active regional differential control was found to be intact. Reflex resistance changes were greatest in the renal bed and least in the coeliac bed. Significant modulation by the aortic baroreceptors in the coeliac, mesenteric and renal beds was observed. Little blood flow homeostasis
was present in the coeliac bed. However, renal (kidney) and iliac (muscle) blood flow was well preserved during reflex depression of systemic arterial pressure.

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REFERENCES


CONTROLE DE L’HEMODYNAMIQUE REGIONALE PAR LES BARORECEPTEURS PENDANT UNE ANESTHESIE PAR L’HALOTHANE EFFECTUEE SUR DES CHIENS

RESUME
Le contrôle de l’éméodynamique régionale par les barorecepteurs cardiovasculaires a été étudié sur des chiens anesthésiés à l’aide de 1 MAC (concentration alvéolaire minimale) d’halothane dans l’oxygène (1%). Les relations de la boucle ouverte qui existent entre la pression sinus carotide (CSP) et l’éméodynamique régionale des lits iliaques, rénaux, mesenteriques, aortiques et coeliaques ont été observées avant vagotomie, après vagotomie et après thoracectomie. Autour du point fixe du réflexe sinus carotide, le rapport entre la diminution de la pression artérielle systémique et l’augmentation de la CSP (augmentation du réflexe) a été de −0,744 ± 0,089 (moyenne ± erreur type des moyennes); cette dernière augmentant à −1,275 ± 0,093 après la vagotomie. Les variations de la résistance du réflexe ont été plus fortes dans le lit rénal et moins fortes dans le lit coeliaque, reflétant l’hémostase du débit sanguin qui a été bien conservée dans le lit rénal, mais qui a été minimale dans le lit coeliaque. La thoracectomie des chiens sur lesquels on avait effectué la vagotomie n’a pas donné de variations importantes dans les variables dépendantes que l’on a étudiés. Il en a été conclu que sur ces chiens anesthésiés à l’aide de 1 MAC d’halothane, le contrôle par les barorecepteurs des relations régionales débit pression est bien préservé.

REGIONALE HÄMODYNAMISCHE KONTROLLE DURCH BAROREZEPTEOREN WAHREN HALOTHANNARKOSE BEI HUNDEN

ZUSAMMENFASSUNG
Regionale hämodynamische Kontrolle durch die kardiovaskulären Barorezeptoren wurde bei Hunden untersucht, die mit 1+ MAC Halothan in 1% Sauerstoff narkotisiert...
waren (MAC = minimale alveolare Konzentration). Die Beziehungen zwischen Karotis-Sinusdruck (CSP) und regionaler Hämodynamik im Darm-, Nieren-, Mesenterial-, Aorten- und Bauchgebiet wurden untersucht, und zwar vor und nach der Vagotomie, und nach der Brustraumeröffnung. Rund um den Karotis-Sinusreflexpunkt betrug das Verhältnis des Reflexabfalls im systemischen arteriellen Druck zu dem Anstieg in CSP (Reflexgewinn) — 0,744 ± 0,089 (Mittel + SEM): letzterer Wert stieg nach der Vagotomie auf —1,275 ± 0,093. Änderungen des Reflexwiderstandes waren stärker im Nierengebiet und am schwächsten im Bauchgebiet, was eine Blutstrom-Homeostase erkennen lässt, die im Nierengebiet gut erhalten war, aber nur minimal im Bauchgebiet. Eine Brustraumeröffnung bei Hunden, die einer Vagotomie unterzogen worden waren, führte zu keinen wesentlichen Änderungen der untersuchten abhängigen Variablen. Es wird festgestellt, dass bei auf diesen Weisen narkotisierten Hunden die Barorezeptor-Kontrolle der regionalen Druck-Blutflussverhältnisse gut erhalten bleibt.

REGULACION BARORRECEPTORA DE LA HEMODINAMICA REGIONAL DURANTE LA ANESTESIA CON HALOTANO EN EL PERRO

La regulación hemodinámica regional mediante los barorreceptores cardiovasculares fue examinada en perros anestesiados con 1 + CAM de halotano en oxígeno (1%). Las relaciones de asa abierta entre la presión (PSC) del seno carotídeo y la hemodinámica regional en los lechos ilíaco, renal, mesentérico, aórtico y celiaco fueron examinadas antes y después de vagotomía, y tras toracotomía. En torno al punto fijo de reflejo del seno carotídeo la proporción del descenso del reflejo en la presión arterial sistémica a un aumento en la PSC (aumento de reflejo) fue — 0,744 ± 0,089 (media ± ETM): esta última aumentó a —1,275 ± 0,093 tras la vagotomía. Cambios en la resistencia del reflejo fueron mayores que en ninguno otro en el lecho renal, y de valor mínimo en el lecho celiaco; reflejando homeostasis del flujo sanguíneo que estaba bien preservada en el lecho renal pero que era mínima en el lecho celiaco. Toracotomía en los perros sometidos a vagotomía no produjo cambios significativos en las variables dependientes estudiadas. Se concluye que, en estos perros anestesiados con 1+ CAM de halotano, la regulación barorreceptora del flujo sanguíneo regional mediante las interrelaciones se halla bien preservada.