An open-loop analysis of the aortic arch barostatic reflex

JAMES L. ALLISON, KIICHI SAGAWA, AND MAMORU KUMADA
Department of Physiology and Biophysics, University of Mississippi School of Medicine, Jackson, Mississippi 39216

The obvious reason for this gap in knowledge is the surgical difficulty involved in opening the aortic reflex loop. No single animal preparation has been reported in which the aortic arch baroreceptor area was isolated and forced independently of systemic arterial pressure while both cardiac and vascular activities were maintained and studied in reasonably natural working condition. The small number of studies with isolated aortic arch preparations in the past were confined to the reflex effects on either heart rate (1, 10, 13, 28) or peak systolic pressure in the isovolumic left ventricle (28). Thus, the total buffer effect on arterial pressure could not be quantitated in these studies.

This paper reports our attempt to perform an open-loop analysis of the aortic arch barostatic reflex in the dog, using a preparation which allowed us to study differentially the simultaneous responses of cardiac pumping and total systemic vascular resistance to pressure variations in the isolated aortic arch. The results, in association with the preceding partial information on the reflex, yield a quantitative index of the overall transfer characteristics of the aortic baroreceptor reflex and aid in the analysis of overall circulatory regulation.

METHODS

General surgery. Experiments were performed on 100 mongrel dogs (ranging from 12 to 18 kg in weight) which were anesthetized with morphine sulfate (7 mg/kg) followed 30 min later by an intravenous administration of a mixture of chloralose (60 mg/kg) and urethane (600 mg/kg). This formula of anesthesia has been used by Daly et al. (7) and other investigators in studying the aortic reflex. The left femoral artery was cannulated and connected to a pressure transducer (Statham model P23AC) to record systemic arterial pressure (SAP). The carotid sinus nerves and the left and right common carotid arteries were isolated and loose ligatures were placed around each. The trachea was cannulated and positive pressure ventilation was instituted by a constant-volume respirator. To prevent hyperventilation, which is known to affect the baroreceptor reflexes (20), the respiratory rate and tidal volume were adjusted to the point where spontaneous respiration was barely observed. In some experiments, blood samples were taken periodically to measure blood PO₂, pH, and PCO₂ (IL gas analyzer). Ventilation was then further adjusted to produce an arterial PO₂ of 90 mm Hg (or greater) and a PCO₂ of approximately 40 mm Hg. The animal's
AORTIC BAROSTATIC REFLEX

Isolation of the aortic arch. Figure 1 illustrates the steps taken to isolate the aortic arch. The left subclavian artery and the descending aorta were exposed, and the first 4-5 intercostal arteries were ligated. To include the baroreceptors at the origin of the right common carotid artery, a ligature was placed around the right subclavian artery immediately proximal to the origin of the vertebral artery but distal to that of the common carotids. A loose ligature was placed around the ascending aorta about 1 cm distal to its cardiac junction. Care was taken in isolating these vessels to minimize the number of nerve fibers damaged. At this stage, priming blood for the perfusion system was obtained from a donor dog anesthetized with sodium thiopental (Surital, 60 mg/kg) and heparinized (3.0 mg/kg). The experimental animal was then administered heparin (3.0 mg/kg) intravenously.

The left atrial appendage was cannulated and connected to an overflow disc, from which excessive blood flowed into the reservoir. This partial left atrial bypass served two purposes: 1) to prevent overloading of the left ventricle during the ascending aorta cannulation and 2) to maintain mean left atrial pressure constant during the experiment.

The proximal part of the left subclavian artery was then cannulated with one tip of a T cannula projecting into the aortic arch lumen. The second tip was connected to a pressure transducer (Statham P23DC) to record the aortic arch pressure (AAP). The third tip was connected to a cannulated with one tip of a T cannula projecting into the brachiocephalic area were isolated from the systemic arterial perfusion. The major systemic arteries were perfused directly by this pump except for the right subclavian artery and its branches.

On completion of the above procedures, the following steps were followed in rapid succession. The overflow disc was lowered to bypass most of the blood flowing into the left atrium to the reservoir. The ascending aorta was clamped with a hemostat at a site a few millimeters proximal to the origin of the brachiocephalic. The systemic perfusion pump was turned on. The ascending aorta proximal to the hemostat was cut and a cannula type electromagnetic flow probe (EMF probe) was quickly inserted toward the heart. A compliance chamber (120 ml) was connected in series with the flow probe, and the level of the overflow disc was reelevated so that the left ventricle ejected 1–1.5 liters/min of blood into the compliance chamber. Outflow resistance from this chamber was regulated by a servo-controlled resistance regulator (Vacuum Industrial Application-type I) to maintain the mean chamber pressure at 100 ± 10 mm Hg. Misteakers and insufficient speed of cannulation of the ascending aorta caused a high rate of failure (79 preparations of 100 dogs) with resultant ventricular fibrillation or cerebral ischemia. The success rate rose markedly during the later experiments in this series as the speed and exactness of the cannulation technique were improved.

Perfusion of the systemic arterial bed by the constant-volume flow pump was started simultaneously with the clamping of the ascending aorta. A preliminary check of this pump showed that changes in the output pressure load from 0 to over 300 mm Hg had little effect on flow output (< ± 1%). The pulse pressure caused by this pump was so small that it was not necessary to extract the mean electrically for recording systemic arterial pressure. The temperature of the blood in the reservoir supplying the pump was maintained at 37 ± 1°C by a thermostat bath. The flow of the pump was adjusted to a level between 1 and 2 liters/min which was sufficient to produce an arterial pressure between 100 and 150 mm Hg.

With the above procedures, the aortic arch and brachiocephalic area were isolated from the systemic arterial perfusion. To confirm the responsiveness of the reflex center, the common carotid arteries were temporarily occluded and the pressor response was observed. Both carotid sinus nerves were then ligated. To test the completeness of the block, the common carotid arteries were again clamped temporarily. When there was no pressor response, the sinus nerves were assumed to be functionally blocked. Heart rate was measured by a cardiotachograph (Grass model 7P4A).

Input and output variables. AAP was varied by means of a servo-controlled reciprocating type infusion-withdrawal pump which was connected to the isolated arch at one end and to the reservoir at the other. A desired pressure setting and actual aortic arch pressure were fed to the servo amplifier, and the pump infused or withdrew arterial blood into or from the isolated aortic arch until aortic arch pressure equaled the given setting.
To measure the static and transient properties of the aortic arch reflex, step changes (usually 30 mm Hg) of aortic arch pressure were employed. The steady-state value of each variable was determined at different aortic pressures after the transient responses had disappeared.

In some dogs, the effects of pulsation of aortic arch pressure were studied. To produce rapid (60-120 cycles/min) pulsation in AAP, an additional piston pump (Harvard model 1401) was connected in parallel with the servo-controlled infusion-withdrawal pump. The piston pump superimposed quasinusoidal pressure variations on the constant mean level of aortic arch pressure.

The ascending aortic flow was recorded by an EMF probe and meter (Medicon K2000) and calibrated in vivo at the end of the experiment. Cardiac output was determined in one of two ways depending upon the particular experiment. In some experiments, the ascending aortic flow was electrically integrated per beat (Grass model 7P10A) and recorded as stroke volume. Cardiac output was then calculated as heart rate times stroke volume. In other experiments, the flow signal was meaned electrically, and stroke volume was calculated by dividing the recorded cardiac output by heart rate. Cardiac output or stroke volume, heart rate, systemic arterial pressure, and input aortic arch pressure were recorded on a pen recorder (Sanborn model 64-1300).

Treatment of chemoreceptors and definition of aortic barostatic reflex. Daly et al. (7) found that in the dog the functional aortic bodies were all situated on the ventral surface of the aortic arch, which corresponds to group III area of Howe’s classification (cited in ref 17). The blood is supplied to the aortic bodies via a small artery arising from the ventral surface of either the brachiocephalic or the ascending aorta. We ligated this artery during the isolation of the aortic arch in the hope that the chemoreceptors would be functionally damaged by ischemia. In fact, not a single preparation showed any change in the recorded hemodynamic variables in response to the elevation of aortic arch pressure from 0 to 60 or 90 mm Hg.

Unfortunately, there is no established method to destroy completely the chemoreceptors without simultaneously damaging the baroreceptors (2, 17). We attempted microsphere injection, or acetic acid injection, into the aortic arch in a few dogs. However, since there was danger of the microspheres or acetic acid reaching the coronary or brain circulation through small anastomoses, this procedure was not employed in the majority of the experiments. For this reason, the term aortic arch “barostatic” reflex is used here instead of “baroreceptor” reflex. Because the term is effect oriented, it does not specify the responsible receptor although we do not consider that the chemoreceptor reflex was actively participating in the cardiovascular responses observed in our preparations.

RESULTS

Step-Change Responses

As aortic arch pressure was raised in steps from 0 to 300 mm Hg and then lowered stepwise, responses were evoked in systemic arterial pressure, stroke volume, heart rate, and cardiac output with varying degrees of intensity at different regions of AAP. These responses consisted of two components: transient response and steady-state response. Among the multiple-output variables, the SAP response was usually more marked and consistent than the cardiac response.

Vascular response. The top row of the experimental recording shown in Fig. 2 exemplifies a typical SAP response to multiple-step changes of AAP. When AAP was raised from 90 to 120 mm Hg, a definite fall in SAP occurred with a subsequent return nearly to the previous SAP level. In response to the next step elevation of AAP, a greater transient fall in SAP occurred, followed this time by partial recovery toward the previous level. Similar SAP responses were seen repeatedly in the following step elevations of AAP, as shown in the upper half of Fig. 2. Likewise, when AAP was lowered in multiple steps, similar but directionally opposite responses were evoked: SAP indicated overshoots.
followed by various degrees of steady-state rise in response to step fall of AAP (lower half of Fig. 2.) Thus, the reflex appeared to have rate sensitivity as well as proportional sensitivity. SAP falls similar to the example in Fig. 2A were seen in 19 of 21 dog preparations during step elevations of AAP. The magnitude of the undershoot relative to that of the steady-state fall was variable from one step to another in each preparation and also from one dog to another. The undershoot was invariably prominent in relatively low regions of AAP, whereas significant overshoot was seen only in some preparations and in relatively high AAP regions. When both transients were present, the undershoot was usually far greater than the overshoot; the ratio of the magnitudes of the largest undershoot to the largest overshoot in individual preparations was 1.5:2. In fact, the example shown in the lower half of Fig. 2 exhibited the greatest overshoot of all the cases. Thus the rate sensitivity was very asymmetric, though not unidirectional, and the degree of the asymmetry depended on the range of input AAP, as was reported also for carotid sinus baroreceptor discharges (5, 26).

It was noted in all of the 19 preparations that the threshold AAP for the transient response was lower than the threshold pressure for the steady-state fall. In the example shown in Fig. 2, the threshold for the transient response was between 90 and 120 mm Hg, whereas that for a definite steady-state response was between 120 and 150 mm Hg.

A similar discrepancy was observed between the saturation levels of AAP for the transient and steady-state responses. The saturation AAP for the transient response was usually higher than that for the steady response, although the difference was difficult to detect in some preparations. Thus, the operating range of AAP for the rate-sensitive response was wider than that for the proportional response. Table 1 shows, among other data, the mean of the threshold and saturation AAPs from the 19 dogs.

As a measure of the sensitivity of the reflex control on the total peripheral resistance, the ratio of the change in systemic arterial pressure (ΔSAP) over the given change in aortic arch pressure (ΔAAP = usually 30 mm Hg) was determined in all the preparations. This ratio does not represent the total barostatic capacity (overall open-loop gain) of the reflex system because the ΔSAP does not include the component of pressure variation that would result from the reflex change in cardiac output if the heart were perfusing the systemic vascular bed. However, the ratio was the major fraction of the total barostatic gain as described below (see Discussion).

The top curves of Fig. 3 is an example of SAP responses plotted against AAP (same example as the top of Fig. 2). The second curves show plots of ΔSAP/ΔAAP. The solid line indicates steady-state gain, whereas the broken line shows the transient response gain defined as the ratio (transient fall in systemic arterial pressure measured from the level prior to the step input)/(ΔAAP). The steady-state gain increased gradually toward a peak value of 1.3 in a region of aortic arch pressure between 135 and 165 mm Hg and thereafter decreased to 0. An approximately linear waxing and waning of the transient gain was observed, the peak value being 0.8. On step lowering of AAP, a steady-state gain curve similar to that during positive step elevations was obtained but the transient gain curve was generally not superimposable on that for positive step inputs. Similar gain curves were obtained from the remaining 19 dogs.

Figure 4 shows the mean steady-state SAP responses from 21 preparations which were expressed in percent of the SAP at 0 AAP and plotted against AAP. The optimal AAP at which the maximal SAP fall occurred varied widely among individual dogs that this mean plot indicates an approximately linear slope of 30% reduction over the AAP range from 90 to 240 mm Hg. If we assume a

![Figure 4](https://example.com/figure4.png)
FIG. 4. Composite data from 20 experiments in which aortic arch pressure was varied in 30-mm Hg steps. Steady-state changes in systemic arterial pressure, heart rate, cardiac output, and stroke volume were calculated as percentages of their initial value at 0 aortic arch pressure and plotted as a function of aortic pressure. (Only one value is included at each aortic pressure from any single preparation.) Vertical lines represent SEM.

The control arterial pressure of 180 mm Hg (with the sinus nerves ligated and AAP at 0 mm Hg), the slope corresponds to an average gain of −0.3. The maximal gain, first measured in individual dogs and then expressed as the mean, was −0.51 ± 0.03 (SEM).

The temporal features of the transient responses were as follows: an average of 3 sec elapsed before any definite change in SAP took place. The total duration of the transient undershoot response ranged from 20 to 56 sec, the average duration being 26 sec. The duration of the overshoot response, occasionally noticed following step lowering of AAP, was generally shorter, the average being about one-half the duration of the undershoot response.

Cardiac responses: heart rate response. In 13 of 15 animals in which heart rates were recorded, the heart rate was found to be inversely related to the aortic arch pressure, as illustrated in the second curve of Fig. 4. In the remaining two animals no heart rate changes occurred, although there were pronounced vascular responses. In one animal there was no vascular response despite a tremendous heart rate response. Both the threshold and saturation level of AAP for the heart rate response were almost always higher than those for the SAP response (Table 1 and Fig. 4). In four animals the heart rate was still changing in response to a step AAP change from 270 to 300 mm Hg. In the study of Levy and associates (28) on the reflex control of heart rate, saturation levels above 300 mm Hg were not uncommon.

Transient characteristics of heart rate responses to step inputs were as follows: the latency or dead time was much shorter than the vascular dead time (usually 0.4–1 sec). There was very little overshoot response to a negative step, but a slight undershoot (usually 5–10 beats/min) was frequently observed during a positive step. The duration of the transient response to positive steps was from 14 to 16 sec, being much shorter than that of the transient fall in SAP.

Stroke Volume and Cardiac Output Responses

As AAP was raised stepwise and heart rate decreased, slight increases in stroke volume occurred, as illustrated in Figs. 2–4. These steady and transient increases in stroke volume paralleled the bradycardic responses over the relatively low AAP range, thus preventing a net change in cardiac output until AAP reached 150 mm Hg. Above this range of AAP, the stroke-volume increases became insufficient to offset the reduction in heart rate and cardiac output decreased with AAP. From threshold to saturation the cardiac output decrease amounted to about 15% while stroke volume increased about 14% (Fig. 4). Due to the reciprocal relationship between heart rate and stroke volume changes, the mean threshold for the cardiac output response appeared at a higher pressure than that for heart rate, being about 140 mm Hg.

Effects of Pulsation

In many studies on the carotid sinus reflex (11, 12, 27, 38), pulsation of the sinus pressure at a constant mean pressure was found to cause a fall in SAP, and this has been interpreted as a frequency-dependent rectification phenomenon due to unidirectional rate sensitivity of the reflex.

In about one-third of the preparations, the effect of high-frequency pulsations of aortic arch pressure on systemic arterial pressure was examined. The amplitude of the pulsa-
tion was usually ± 40 mm Hg and the frequency was 1.2 Hz. It was commonly seen that SAP responded to the sudden pulsation with a large transient fall followed by partial or sometimes complete recovery to the control. Figure 5 is an example in which two different amplitudes of pulsations (±20 and ±40 mm Hg) caused transient undershoots of systemic arterial pressure (about 15 mm Hg in both cases) and steady-state falls of 0 and 5 mm Hg. The time course of the transient response was similar to that seen after step elevations of AAP. At the cessation of pulsation, a smaller but definite overshoot response was recognized in some of the dogs. The magnitude of the steady-state fall was generally less than 15 mm Hg. The heart rate also decreased in response to the pulsation, without any transient change in some dogs (as in the example in Fig. 5) or with obvious under- and overshoot in others. In general, the heart rate response to aortic pulsation was much less than the SAP response with those frequencies of pulsation.

DISCUSSION

Since no previous investigation has examined the overall open-loop transfer characteristics of the aortic baroreceptor reflex system, there is no direct reference to judge the validity of the above data. However, we can compare our results with earlier partial information on the aortic arch reflex and much more complete information on the carotid sinus reflex. The comparison should help to reveal the approximate quality of the present results.

Our experiments showed that from threshold to saturation of the reflex the total reflex reduction of vascular resistance amounted to 34%, as opposed to the 15% reduction in cardiac output from the mechanically isolated left heart. On this basis it may be said that the major effector limb of aortic baroreceptor reflex control is systemic arterial resistance, and the reflex control on cardiac pumping action is relatively weak, particularly in the physiological pressure range. This has been shown repeatedly in studies of the carotid sinus reflex (6, 9, 24, 41).

However, the effects of the reflex on total systemic vascular capacitance, which, obviously, affect cardiac output were not investigated in this experiment. Thus, the relative magnitudes of cardiac output responses versus vascular resistance responses are not directly applicable to overall closed-loop circulatory regulation by the reflex.

A few dogs in the present group showed very pronounced cardiac responses while exhibiting little vascular response. This individual difference has also been observed in studies of the carotid sinus reflex, including those done with unanesthetized dogs. Olinstond and co-workers (32) reported that about one-half of their unanesthetized dogs responded to bilateral common carotid occlusion primarily with vasocostriction, whereas the other half responded with relatively stronger cardiac output response and weaker vasocostriction. In the experiments of Rushmer et al. (34), cardiac versus vascular responses to mild hemorrhage varied so markedly from dog to dog, in the conscious state, that the authors criticized the conventional concept of the baroreceptor reflex as a gross oversimplification. Thus the individual variability of the relative intensity of cardiac and vascular controls seen among our preparations is probably not an artifact due to anesthesia and/or surgery but to a physiological phenomenon, whatever the cause may be.

The total "barostatic gain" of the aortic arch reflex must be determined by the arterial pressure response which integrates all the output components of the reflex, such as changes in cardiac pumping, vascular resistance, and vascular capacitance. Such a gain was not determined in our experiment because the left ventricular pump and systemic vascular bed were mechanically uncoupled. This uncoupling was advantageous to study differentially the multiple outputs of the reflex, i.e., the controls on the heart and the peripheral resistance, without mechanical interactions between the two.

If we assume that the heart was perfusing the systemic vascular bed rather than the constant flow pump and that the cardiac and resistance vessels' responses were identical to those observed in the experiment, we can estimate an approximate barostatic gain of the reflex system. To do this, we first calculated the systemic arterial pressures expected at each aortic arch pressure between 0 and 790 mm Hg according to the following formula:

\[
\text{SAP}_i = \text{SAP}_{0} \times \frac{[R(\%)_i \cdot \text{CO}(\%)_i]}{100}
\]

where SAP\(_i\) is the expected systemic arterial pressure when aortic arch pressure was set at the \(i\)th value of six AAPs (120, 150, 180, 210, 240, and 270 mm Hg). SAP\(_{0}\) was assumed to be 180 mm Hg, considering the reported values of SAP after sinoaortic denervation in the dog. \(R(\%)_i\) and \(\text{CO}(\%)_i\) are the average resistance and cardiac output values at the \(i\)th value of aortic arch pressure, expressed in percent of the resistance and cardiac output when aortic arch pressure was 90 mm Hg.

From these values of SAP\(_i\), the difference \(\Delta \text{SAP}\) between two consecutive SAP\(_i\)s (e.g., SAP\(_{180}\) and SAP\(_{210}\)) was calculated and then divided by \(\Delta \text{AAP}\) (i.e., 30 mm Hg). The ratio \(\Delta \text{SAP}/\Delta \text{AAP}\), which indicate the barostatic gains of the reflex over each aortic arch pressure interval, are shown in Table 2, with the values of calculated SAP\(_i\)s. Although these barostatic gain values do not include the reflex control of the capacitance vessels and, therefore, are not truly total gains, they may still serve as a useful approximation of the overall barostatic gain of the reflex system as a function of the input pressure. The maximal total barostatic gain was found to be 0.66 at an aortic pressure range of 150 to 100 mm Hg.

The above calculation assumes no significant mechanical interactions between the heart and systemic vessels. This assumption appears justifiable as far as the interaction between the left ventricular pump and arterial pressure is concerned, since Sagawa (35) observed that only a negligibly small change in steady-state cardiac output occurred when both mean left atrial and aortic pressures were controlled and the aortic pressure was varied from 50 to 150 mm Hg, provided that the control cardiac output was relatively small (i.e., about 100 ml/kg body weight of the dog).
However, the assumption is obviously not justified regarding the interaction between the cardiac pump and the venous capacitance vessels. If the reflex control on overall venous capacitance is powerful enough to affect the cardiac filling, the total barostatic gain will be greater than the above values. Unfortunately, the reflex control of overall venous capacitance has never been quantitated, although positive evidence exists for carotid sinus reflex control on parts of the venous system (3, 39).

Several investigators have made indirect estimates of the overall aortic baroreflex gain from the studies of arterial pressure responses to various disturbances before and after vagotomy. Thus, Guyton and associates (16) estimated that the aortic reflex gain was approximately the same as that of the carotid sinus reflex. Sagawa and Watanabe (37) estimated it as -0.8 from the change in carotid sinus reflex gain after vagotomy. Later, Sagawa (36) estimated the gain at -0.6 from a separate study on posthemorrhagic hypotension with and without the vagus nerves. This latter estimate is very close to the present more directly estimated total barostatic gain.

Included in Table 3 is the result of our calculations of the vascular gain of the aortic arch baroreflex from one experimental recording which appeared in the report of Daly and co-workers (7) on their study of the aortic chemoreceptor reflex. The gain value coincides with that found in our experiment. Also included in Table 2 are the overall barostatic gains calculated from the experimental records of Nakayama (29) and Neil (30) on the right subclavian baroreceptor reflex. These values are much smaller than ours or Daly's.

Generally one tends to assume that the reflex gain will be underestimated in anesthetized conditions. That this is not always a justifiable inference was recently demonstrated by the work of Lamberti et al. (25), who showed that the gain of head level control of arterial pressure increased rather than decreased with anesthesia. Also, these authors estimated the overall gain of the trunk baroreceptor reflex as small as -0.3 (personal communication). Therefore, the seemingly small barostatic gain obtained in this study may well represent the approximate magnitude under physiological conditions. The above coincidence of the present gain value with our earlier estimates deduced from the closed-chest dog experiments seems to be indicative of quantitative significance of the present gain data.

The threshold and most sensitive regions of aortic arch pressure reported in this study appear fairly higher than those on the carotid sinus reflex. However, as Levy and associates (28) discussed, there is a wide range of reported values of these parameters for the carotid sinus reflex. The natural forcing pressure to the arterial baroreceptors is pulsatile, and with pulsation the operating range of the reflex is expected to extend toward the lower pressure region than that reported here (11). On the other hand, the high saturation level of input pressure in the aortic reflex seems to be a prominent feature which distinguishes it from the carotid sinus reflex.

There seems to be little doubt that the directionally asymmetric rate sensitivity of the reflex is a real property of the aortic baroreceptor reflex. A similar property has repeatedly been identified with the carotid sinus reflex (11, 14, 27, 38), and electroneurograms of the aortic nerve also gave evidence of it (18, 19, 22, 31). Glick and Cove11 (13) noted this property of the aortic reflex in heart rate responses. The possibility that the partial recovery of systemic arterial pressure to a step change of aortic arch pressure is due to a secondary negative feedback via unknown remaining reflexes seems rather unlikely in our study. In each preparation, we confirmed the absence of the SAP response to carotid occlusion after the sinus nerve ligature. Left cardiac or coronary baroreceptors (4, 16, 33) could not sense any pressure change in our preparation due to the mechanical uncoupling. Participation of the aortic chemoreceptor reflex was also unlikely, because changes in aortic arch pressure from 0 to 60 or 90 mm Hg, which must have caused a tremendous change in blood flow through the aortic bodies, elicited no significant response in any of the output variables.

Levy and co-workers (28) systematically studied aortic

<table>
<thead>
<tr>
<th>(\Delta A P, \text{mm Hg} )</th>
<th>(\text{SAP, mm Hg} )</th>
<th>Gain = (\Delta\text{SAP}/\Delta\text{AAP} ) at AAP</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>180</td>
<td>0 (45±45 mm Hg)</td>
</tr>
<tr>
<td>30</td>
<td>180</td>
<td>0.47 (105±15 mm Hg)</td>
</tr>
<tr>
<td>90</td>
<td>166</td>
<td>0.27 (135±15 mm Hg)</td>
</tr>
<tr>
<td>150</td>
<td>172</td>
<td>0.66 (165±15 mm Hg)</td>
</tr>
<tr>
<td>180</td>
<td>160</td>
<td>0.43 (195±15 mm Hg)</td>
</tr>
<tr>
<td>210</td>
<td>167</td>
<td>0.47 (225±15 mm Hg)</td>
</tr>
<tr>
<td>240</td>
<td>166</td>
<td>0.17 (255±15 mm Hg)</td>
</tr>
</tbody>
</table>

* Assuming control systemic arterial pressure to be 180 mm Hg at the origin of each interval. † Gain is defined as change in systemic arterial pressure (\(\Delta\text{SAP} \)) relative to change in aortic arch pressure (\(\Delta\text{AAP} \)). SAP was calculated using mean magnitude of reflex change in resistance and cardiac output from all experiments.

### Table 2. Calculated systemic arterial pressure at each aortic arch pressure*

<table>
<thead>
<tr>
<th>(\Delta\text{AAP}, \text{mm Hg} )</th>
<th>(\text{ AAP, mm Hg} )</th>
<th>(\Delta\text{SAP}/\Delta\text{AAP} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>180</td>
<td>0.47 (105±15 mm Hg)</td>
</tr>
<tr>
<td>30</td>
<td>180</td>
<td>0.27 (135±15 mm Hg)</td>
</tr>
<tr>
<td>90</td>
<td>166</td>
<td>0.66 (165±15 mm Hg)</td>
</tr>
<tr>
<td>150</td>
<td>172</td>
<td>0.43 (195±15 mm Hg)</td>
</tr>
<tr>
<td>180</td>
<td>160</td>
<td>0.47 (225±15 mm Hg)</td>
</tr>
<tr>
<td>210</td>
<td>167</td>
<td>0.17 (255±15 mm Hg)</td>
</tr>
</tbody>
</table>

* Assuming control systemic arterial pressure to be 180 mm Hg at the origin of each interval. † Gain is defined as change in systemic arterial pressure (\(\Delta\text{SAP} \)) relative to change in aortic arch pressure (\(\Delta\text{AAP} \)). SAP was calculated using mean magnitude of reflex change in resistance and cardiac output from all experiments.
arch reflex control of cardiac contractility using the isovolumically contracting left ventricle of the dog. They measured changes in heart rate and the peak systolic ventricular pressure with altered aortic arch pressure. The threshold pressure, saturation level of aortic arch pressure, and percent change in heart rate observed by these investigators agree well with our observations, as shown in Table 1. Although their data on the peak systolic pressure during isovolumic beats and our data on cardiac output against a mean pressure load of 100 mm Hg are not directly comparable, the percent reduction in both quantities is similar in magnitude. Thus their results and ours are complementary and provide an estimate of the aortic reflex control of cardiac performance. Undoubtedly more detailed information is needed, but it must be remembered that quantitative aspects of even carotid sinus reflex control of the heart have not necessarily been well agreed upon by different investigators (9, 17, 21).

Most of the previous open-loop analyses of the carotid sinus reflex (12, 14, 23, 38) have not differentiated between the various outputs of the reflex. Therefore, the major effector limb responsible for the rate-sensitive arterial pressure response has not been identified. The separate determinations of the reflex outputs in the present study demonstrate that the rate sensitivity exists both in the vascular and cardiac responses but is much more marked in the former than in the latter.

However, it should be emphasized that the present investigation did not evaluate aortic reflex control of systemic venous capacitance which would obviously influence cardiac output by altering venous return to the right and left hearts. Consequently, further investigation of the aortic reflex effects on total systemic vascular capacitance is needed in order to quantitatively define aortic regulation of total circulatory dynamics.

This research work was submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy for J. L. Allison at University of Mississippi. This work was supported, in part, by Public Health Service Grants HE-09644 and HE-10581 and, in part, by National Institutes of Health Training Grant 5714-GM-316.

All reprint requests should be sent to: J. L. Allison, University of Mississippi School of Medicine, Department of Physiology and Biophysics, 2300 North State Street, Jackson, Miss. 39216. His present address is Division of Biomedical Engineering, University of Virginia, Box 294, Medical Center, Charlottesville, Va. 22901.

Present address of K. Sagawa and M. Kumada: Department of Biomedical Engineering, Schools of Medicine and Engineering, Case Western Reserve University, Cleveland, Ohio 44106.

Received for publication 24 February 1969.

REFERENCES


