ABSTRACT

Dynamic arterial blood pressures were recorded by micropuncture of 99 cortical surface arteries and arterioles in cats anesthetized with pentobarbital or chloralose and urethane, using the Wiederhielm servocontrolled micropipette pressure system incorporating pipette tips less than 0.5μ in diameter. During these measurements, systemic blood pressure was varied by supplemental intravenous administration of barbiturate, by intravenous administration of norepinephrine, or by arterial hemorrhage and reinfusion. Pial arterial blood pressure varied systematically with the changes in systemic blood pressure, forming a basis for analysis and synthesis of the results. Measurements in individual vessels indicated that the range of pressures and of pressure responses of the cortical surface vessels was variable with both vessel size and prevailing end-expiratory gas CO₂ content. Pooled data indicated that 39% of the loss in pressure head occurred in arteries upstream from the largest (300μ or more in diameter) surface arteries, 21% between these arteries and surface arterioles 50μ or less in diameter, and 40% downstream from the surface arterioles at a systemic blood pressure of 50 mm Hg. At a systemic blood pressure of 180 mm Hg the upstream loss decreased to 33%, loss across the pial vessels decreased to 15%, and the downstream loss increased to 52%. Thus, the results indicated that the cerebrovascular response to changes in systemic blood pressure occurred predominantly downstream from the surface arterial vasculature.

KEY WORDS cerebrovascular resistance cerebrovascular control pressure autoregulation end-expiratory CO₂ arterial anastomoses
The Wiederhielm servocontrolled micropipette pressure system (3, 4) was used to measure blood pressure in cortical surface arteries and arterioles, and aortic and central venous blood pressures were recorded via polyethylene femoral catheters attached to strain-gauge pressure transducers. All pressures were zero-referenced to mid-thoracic level. Expiratory gas CO₂ was monitored with an infrared absorption gas analyzer while the cat was respired by a fixed-volume mechanical respirator. Bilateral pneumothoracotomies, cisternal cerebrospinal fluid drainage, and intravenous administration of gallamine triethiodide (Flaxedil) at an initial dose of 20 mg were used as necessary to reduce the effects of respiratory effort on intracranial volume and therefore on movement of the exposed brain surface. A heating pad was used to control rectal temperature at 37 ± 1°C throughout the experiment.

Surgical procedures consisted of reflecting the scalp, carefully removing a portion of the parietal calvarium with a trephine, liberal application of bone wax, and reflecting the dura to expose the cortical surface (pial) vasculature. Preparation took about 2 hours from the initiation of anesthesia. A more detailed discussion of the preparation is in press (5).

Polarized heat-filtered side illumination, using fiber optics, under a Zeiss operating microscope equipped with a 50-mm objective and an adjustable polaroid filter provided glare-free visualization of the operative field. Micropuncture pressure measurements were made using a pneumatic micromanipulator. The entire exposed cortical surface was continuously irrigated with warm Elliot's B solution (Travenol) throughout the experiment. An eyepiece micrometer was used to measure pial arterial diameters at 165x magnification. This measurement was probably not better than 20% for the smallest arterioles.

Variations in systemic blood pressure were induced by (1) gradual spontaneous changes in the depth of anesthesia, (2) intravenous infusion of norepinephrine bitartrate, (3) infusion of supplemental barbiturate anesthetic, or (4) rapid arterial hemorrhage or reinfusion of shed blood. In recordings in which systemic blood pressure was rapidly changing, data were collected at intervals of 1 or 2 seconds. As the rate of change in systemic blood pressure diminished, data were taken at progressively longer intervals until a steady state was reached. Steady-state measurements were made at 30- to 60-second intervals or longer. Durations of recordings from individual vessels ranged from a few seconds to nearly an hour. Those sections of the recordings where it was evident that brain surface movement was distorting the pial arterial pressure wave form (e.g., pipette was pushed against the vessel wall at the peak of respiratory movement) were disregarded. As a result of using this criterion, the measurements were weighted toward covering the widest range of systemic blood pressure, toward the more rapidly changing pressures, toward results from arteries and arterioles that were observed for the longest time, and toward those recordings having the highest pressure-tracking reliability. Occasionally, marked arterial or arteriolar constriction occurred at the site of puncture after 1.5-2 hours of cortical exposure. In this event a note was made on the chart, and the resulting records were disregarded; either the contralateral hemisphere was prepared or the experiment was terminated. Data were also discarded when there was bleeding at or near the puncture site.

Data were tabulated as systolic and diastolic pressures from the aortic and pial arterial pressure records, as mean venous pressure from the central venous pressure record, and as peak expiratory CO₂ in percent from the record of CO₂ content of the expired gas. Tabulated data were punched on digital data cards together with time after onset of forcing and identifying codes, as indicated in Table 1. The arithmetic mean of systolic and diastolic pressures was used in all subsequent calculations and graphs of the results.

Regression analysis was carried out in two phases. The first was a least-squares linear regression of pial arterial blood pressure on systemic blood pressure: \( P = a + bS \), where \( P \) = pial arterial blood pressure and \( S \) = systemic arterial blood pressure both expressed in millimeters of mercury. Effects of variations in peak expiratory CO₂ and other parameters on the pial arterial blood pressure were considered in the second phase of the analysis. To determine the effects of variations in expiratory CO₂, multiple regression analysis was used. The least-squares fit of data from individual vessels to the expression \( \bar{P} = a + bS + c[CO_2] \), where \([CO_2]\) is the peak concentration of CO₂ expressed in percent of expired gas. The mean square deviation of the data from this regression was then used to establish those cases in which adding the CO₂ term gave an improved fit over linear regression at the 90% confidence level or better (6, 7). Similarly, the least-squares fit to the expression \( \bar{P} = a + bS + c[CO_2] + dS[CO_2] \) was found to test the hypothesis that a change in CO₂ could affect the sensitivity of pial arterial blood pressure to systemic blood pressure. Terms of higher order were also calculated but found unnecessary by these criteria.
### Data Characteristics, Regression Analysis Results, and Coding for 36 Vessels

#### Initial vessel diameter (μm) | Number of points | Systemic pressure (mm Hg) | End-expiratory CO₂ (%) | Regression analysis coefficients | Mean square deviation | Codes
--- | --- | --- | --- | --- | --- | ---
100 | 14 | 91 | 155 | 4.0 | 5.1 | 9.3 | 0.51 | 39.0 | 0, 5
200 | 10 | 37 | 178 | 3.5 | 6.0 | -118.0 | 1.40 | 27.0 | -0.15 | 33.0 | 0, 1
250 | 10 | 77 | 129 | 3.0 | 3.5 | 85.0 | 0.70 | 22.0 | 20.0 | 0, 1
300 | 17 | 43 | 71 | 2.3 | 3.0 | 1.2 | 0.81 | 13.0 | 0, 1
330 | 15 | 88 | 114 | 4.3 | 4.4 | 22.0 | 0.44 | 2.4 | 0
350 | 19 | 83 | 166 | 3.4 | 4.3 | -15.0 | 0.68 | 4.9 | 0
400 | 10 | 46 | 156 | 3.7 | 3.8 | -3.5 | 0.72 | -2.5 | 7.4 | 0, 2
450 | 13 | 55 | 107 | 3.8 | 4.3 | -73.0 | 0.61 | 18.0 | 7.4 | 0, 2
500 | 16 | 67 | 145 | 3.5 | 4.1 | -13.0 | 0.78 | 100.0 | 0, 2
550 | 19 | 80 | 172 | 3.6 | 4.1 | 0.3 | 0.56 | 23.0 | 0, 2
600 | 36 | 84 | 141 | 2.6 | 5.1 | 3.3 | 0.76 | -6.1 | 190.0 | 0, 2
650 | 21 | 87 | 135 | 4.0 | 4.3 | -38.0 | 1.00 | 33.0 | 11
700 | 38 | 70 | 142 | 4.2 | 5.2 | 129.0 | 0.53 | 44.0 | 96.0 | 11
750 | 100 | 61 | 145 | 3.8 | 4.9 | -3.0 | 0.68 | 59.0 | 11, 14
800 | 21 | 80 | 146 | 4.0 | 4.4 | 42.0 | 0.65 | 11.0 | 15.0 | 0
850 | 13 | 75 | 153 | 3.8 | 4.5 | -17.0 | 0.68 | 15.0 | 0, 4
900 | 32 | 73 | 133 | 4.0 | 4.2 | -41.0 | 0.88 | 17.0 | 11.0 | 0, 4
950 | 19 | 115 | 181 | 4.0 | 4.6 | -103.0 | 0.70 | 21.0 | 39.0 | 0, 4
1000 | 24 | 114 | 202 | 3.9 | 4.6 | -67.0 | 0.76 | 13.0 | 18.0 | 0, 4
1050 | 28 | 46 | 169 | 4.2 | 4.6 | -20.0 | 0.75 | 9.5 | 0, 3, 4
1100 | 25 | 101 | 176 | 4.4 | 4.7 | -18.0 | 0.63 | 27.0 | 0, 4
1150 | 19 | 73 | 118 | 3.9 | 4.6 | 9.9 | 0.58 | 11.0 | 0
1200 | 18 | 64 | 124 | 3.5 | 4.3 | -28.0 | 0.81 | 12.0 | 0, 4
1250 | 40 | 89 | 186 | 4.0 | 4.8 | -41.0 | 0.78 | 7.1 | 8.1 | 0, 4
1300 | 33 | 83 | 121 | 3.9 | 4.6 | -44.0 | 0.92 | 12.0 | 0
1350 | 46 | 81 | 170 | 3.8 | 4.7 | 130.0 | 0.86 | 19.0 | 11.0 | 0, 4
1400 | 22 | 46 | 88 | 3.8 | 4.0 | -21.0 | 0.76 | 7.6 | 0
1450 | 26 | 81 | 132 | 4.0 | 4.3 | -14.0 | 0.82 | 15.0 | 0, 3, 4
1500 | 34 | 14 | 61 | 118 | 3.6 | 4.0 | -18.0 | 0.66 | 8.6 | 0, 3, 4
1550 | 160 | 71 | 203 | 3.6 | 5.2 | 78.0 | 0.55 | -14.0 | 32.0 | 0, 3, 4, 5
1600 | 71 | 76 | 145 | 3.6 | 5.8 | 65.0 | 0.39 | -10.0 | 40.0 | 0, 5

The range of systemic blood pressure was at least 30 mm Hg for these experiments. The coefficients of the expression $P = a + b \cdot S + c \cdot \left[CO_2\right] + d \cdot [CO_2]$ from the regression analysis, where $P$ is mean pial arterial blood pressure, $S$ is mean systemic blood pressure, and $[CO_2]$ is the percent of expired gas, are listed together with the mean square deviation from the regression (SE of the estimate). When no coefficient is listed that term did not contribute significantly at the 90% level. The codes listed in the last column indicate combinations of anesthetic used and method of changing systemic blood pressure during pressure recordings from that vessel. Codes 0–5 indicate pentobarbital anesthesia. Codes 11 and 14 indicate chloralose-urethane anesthesia. Codes 0 and 11 indicate spontaneous systemic blood pressure changes, probably due to slowly varying anesthetic level. Code 1 indicates that arterial hemorrhage and reinfusion were used first to lower and then to increase systemic blood pressure. Code 2 indicates that thiopental (a short-acting barbiturate) was given intravenously and code 3 that pentobarbital was given intravenously as supplemental anesthesia producing a transient decrease in systemic blood pressure. Finally, code 5 indicates that respiratory minute volume was varied to induce a change in end-expiratory CO₂ concentration.
A recording showing the responses which followed an intravenous infusion of norepinephrine. Systemic blood pressure measured via a catheter in the thoracic aorta and pressure measured in a 290µ surface artery increased over an interval of several seconds. This and similar recordings of concomitant increases and decreases of pressures due to changing levels of anesthesia, rapid infusion of supplemental barbiturate, and arterial hemorrhage and reinfusion as well as to norepinephrine infusions provided the relationships considered in this report.

Results

INDIVIDUAL VESSELS

Pressures in the pial surface arteries and arterioles varied systematically with systemic blood pressure, as indicated by the recording from a 290µ artery during an intravenous infusion of norepinephrine shown in Figure 1 and by the plot of data from similar recordings from a 200µ artery shown in Figure 2. This systematic correspondence between pressures formed a basis for data analysis.

Regression analysis of results from individual vessels was restricted to those vessels from which data covered a range of systemic blood pressure of at least 30 mm Hg. The histogram in Figure 3 shows the diameter distribution of the 99 vessels that were punctured. A bias toward smaller vessels is evident in the population of all arteries and arterioles punctured but is not evident in the population of vessels for which data were recorded over a systemic blood pressure range of at least 30 mm Hg.

Although the variability of data within a given vessel, as illustrated in Figure 2, was relatively small, variability of data among vessels was relatively large. Variation among regression lines fitted to different vessels was due primarily to differences in elevation rather than to differences in slope of the regression lines. A summary of the ranges of the data, the regression coefficients, and the mean square
Among the factors that might influence pial arterial blood pressure are arterial $\text{PCO}_2$, rates of change of the pressures, method of changing systemic blood pressure, and type of anesthetic used. These factors were considered through their influence on the relationship between systemic blood pressure and pial arterial blood pressure in individual vessels.

**END-EXPIRATORY CARBON DIOXIDE VARIATIONS**

In 18 vessels, including the $\text{CO}_2$ terms in the regression analysis gave a significant reduction in the mean square deviation from regression, indicating a significant effect of end-expiratory $\text{CO}_2$ on the sensitivity of pial arterial blood pressure to systemic blood pressure. These instances are indicated in Table 1 by coefficients in the $c$ and $d$ columns. The net effect of increasing $\text{CO}_2$ in 10 of the vessels was an increase in pial arterial blood pressure. The net effect in the other 8 was a decrease. No correspondence between the initial vessel diameter and the effect on pressure relationships due to alterations in end-expiratory $\text{CO}_2$ was evident.

These results therefore include a full range of possible influences of increasing $\text{CO}_2$ on pial arterial blood pressure. Although, qualitatively, the surface arterial vasculature dilated with increasing $\text{CO}_2$, pressures in the pial arterial system increased in about a fourth of the cases, stayed the same in about a half, and decreased in about a fourth. The magnitudes of the effects also covered a wide range, as indicated by the $c$ and $d$ coefficients. It must be mentioned, however, that in most cases these experiments involved $\text{CO}_2$ changes only as an inadvertent variable which usually occurred during changes in systemic blood pressure, i.e., as systemic blood pressure increased, end-expiratory $\text{CO}_2$ also increased and vice versa. As indicated in the listing of codes in Table 1, the respirator minute volume was purposely varied (code 5) in five instances. These intentional changes in respirator minute volume also induced changes in end-expiratory $\text{CO}_2$ which yielded a pattern of results (two increases, one unchanged, two decreases) similar to those from the inadvertent variations. These results were therefore included in the foregoing consideration.

**RATE OF PRESSURE CHANGE**

The rate of change of pial arterial blood pressure varied with changes in the rate of arterial bleeding and reinfusion and with the dosage and rate of administration of the barbiturates and norepinephrine. (The rates of change were as high as 8 mm Hg/sec, but most were lower than 2 mm Hg/sec.) This factor was considered by comparing the rate of pressure change over short intervals to the deviation of pial arterial blood pressure from the regression line. For example, if, due to significant differences in rates of adjustment of portions of the cerebral vasculature, there was a time lag in the adjustment of the vasculature upstream from the site of pressure measurement relative to that of the downstream vasculature, pressure measurements made during rapid increases in systemic blood pressure would fall markedly below the regression line. Similarly, during rapid decreases in systemic blood pressure, they would fall above the regression line. There were, however, no consistent deviations of sufficient magnitude to attribute a significant influence on pial arterial blood pressure to the rate of change of pressure.
MODE OF ALTERING SYSTEMIC BLOOD PRESSURE

Although there was little difference in the results with the different methods of altering systemic blood pressure, bleeding and reinfusion yielded somewhat higher pial arterial blood pressures than did either of the pharmacologic agents over the whole range of systemic blood pressures in all sizes of vessels.

ANESTHETIC DIFFERENCES

There was no apparent difference in the results with the two different anesthetics.

GROUPED VESSELS

The foregoing regression analysis based on data from individual vessels required that the data from each vessel cover a range of systemic blood pressure of at least 30 mm Hg. As indicated by the histogram in Figure 3, this meant neglecting the results from micropuncture of 63 of the 99 vessels. All the results, including those from the 63 vessels previously neglected, were considered by pooling the data. The four graphs in Figure 4 show the pial arterial blood pressures from the pooled data plotted as a function of initial vessel diameter over four representative ranges of systemic blood pressure. (Although not quantified in this study, vessels dilated with decreases and constricted with increases in systemic blood pressure.) These graphs further confirm that there was considerable variability in pial arterial blood pressure in vessels of the same or similar dimensions under similar systemic blood pressure heads. In the graph in the upper left corner, which shows results over the systemic blood pressure range from 20 mm Hg to 60 mm Hg, the pial arterial blood pressures showed a great deal of scatter: they appeared to cover essentially the same wide pressure range as did the aortic blood pressures and there was poor correspondence between vessel size and pressure. Over the progressively higher systemic blood pressure ranges represented in the other graphs,
the pressure drop between the aortic measuring site and the larger pial arteries became more clearly evident. Although correspondence between diameter and pressure was also poor in these graphs, there appeared to be progressively lower pressures in the smaller branches. Plotting the pooled data in this way emphasizes the high degree of variability of pressures in similar-sized vessels. However, because of the scatter and the lack of an evident relationship between vessel diameter and pressure, it is difficult to make generalizations about the possible role of the pial vasculature from these plots. Data were therefore separated into subgroups on the basis of vessel diameter and fitted with regression lines.

Results from this analysis are shown in Figure 5. The middle regression line is a fit to all 1816 data points from all 99 vessels. It emphasizes that pial arterial blood pressure was considerably lower than systemic blood pressure over the whole range of systemic blood pressure. The other two lines represent regression lines fitted to data from subgroups corresponding to the largest and the smallest surface vessels. The top line, representing the largest vessels (greater than 300 μ in diameter), indicates pressures higher than average for this population of vessels over the whole range of systemic blood pressure, and the bottom line, representing the smallest surface arterioles (less than or equal to 50 μ in diameter), indicates lower pressures. Furthermore, the slopes of the top and bottom regression lines are significantly different at the 99% level, indicating an increasing pressure drop across the surface arterial bed for increasing systemic blood pressure.

Although involving the risks inherent in statistical inferences from pooled data which include considerable variability, this analytic approach leads to simplification of the pressure data and allows generalization with regard to the potential role of different portions of the cerebral vasculature, as indicated in Figure 6. At a systemic blood pressure of 50 mm Hg, there was a substantial loss in pressure head (21%) across the pial arterial network, but the loss was almost twice as great across both the upstream arteries (39%) and the vasculature downstream from...

**FIGURE 5**

Regression lines fitted to data from all the vessels punctured, from those vessels greater than 300 μ in diameter, and from those vessels 50 μ or less in diameter. Standard errors are shown for each regression line. The top and bottom plots were used in computing the fractional pressure drops diagramed in Figure 6. Slopes of the top and bottom lines are significantly different at the 99% level, indicating an increased pressure drop across the surface arterial bed with increasing systemic blood pressure.

**FIGURE 6**

Per cents of pressure head lost across different portions of the cerebral vasculature from the aorta through the middle cerebral artery and back to the central venous pool at low (50 mm Hg) and high (180 mm Hg) systemic blood pressures. Average central venous blood pressure in these preparations was 4.2 mm Hg. Calculations are based on the top and bottom regression lines shown in Figure 5. Upstream refers to the pressure drop in the arteries upstream from the pial surface arteries, across to that between the largest pial surface arteries and the smallest surface arterioles, and downstream to the pressure drop between these arterioles and the central venous pool. The 90% confidence intervals are 39 ± 4, 21 ± 6, and 40 ± 5, respectively, at 50 mm Hg and 33 ± 1, 15 ± 2, and 53 ± 2, respectively, at 180 mm Hg. The differences between each of the pairs of values at the two systemic blood pressures is significant at the 90% level or better.
the cortical surface (40%). At a systemic blood pressure of 180 mm Hg, this pattern shifted significantly. The proportion of the pressure head lost across the downstream vasculature increased substantially to 52% of the total loss and that lost across the upstream arteries and across the pial arterial network decreased to 33% and 15%, respectively. These changes were significant at the 90% level or better.

**Discussion**

Blood flow measurement techniques have provided considerable information about regional and total changes in cerebral blood flow resulting from a variety of types of stimulation. These flow changes are primarily the result of alterations in cerebrovascular resistance, i.e., changes in the caliber of the vessels in the cerebrovascular bed. Little information is currently available concerning the segmental distribution of these resistance changes. Exposing the cortical surface vessels by methods such as those outlined by Forbes (2) allows direct observation and study of the brain surface (pial) segment of the cerebral vasculature. However, it has been difficult to assess the relative role of this segment of the cerebral vasculature in the control of cerebral blood flow without information about concurrent changes in other segments of the vasculature proximal and distal to the site of surface measurement. Diameter measurements of the pial vessels yields information about their responsiveness to various physiological forcings (1, 8-12), but the effect of these diameter changes on the magnitude and distribution of cerebral blood flow cannot be determined without relating these changes in diameter to changes in resistance and to other components contributing to resistance changes in the cerebral vasculature. Similarly, it is not possible to determine the segmental distribution of vascular resistance from measurements of blood pressure in a single pial artery or arteriole.

In an end-artery type of vascular bed, if it is assumed that flows are identical in parallel branches at corresponding levels of the vasculature and that the system may be analyzed through an equivalent series resistance network, then the ratio of upstream to downstream pressure drops can be equated to the ratio of upstream to downstream resistances. This resistance ratio determines the pressure at a measurement site under a given pressure head. Pressure is therefore dependent primarily on the caliber of vessels distant from the site of the pressure measurement and is largely independent of diameter changes at that site. In the highly anastomotic pial arterial bed (13) changes in local vascular resistance are probably accompanied by redistribution of blood flow through alternative channels, providing an additional complicating factor. Equating relative pressure drops to relative resistance is clearly not valid for pressure measurements from a single site in this type of bed. Only by measuring a large number of pressures at a variety of locations can the segmental distribution of resistance be inferred. The actual values of resistances in the network cannot be determined without concomitant measurements of blood flow through the resistive elements.

By measuring systemic arterial blood pressure in the aorta and venous blood pressure in the inferior vena cava in this study, we have included virtually the entire circuit traversed by blood moving from the heart to the brain and back to the heart. Using the entire circuit has the virtue of placing the contribution of the pial surface vasculature in perspective relative to the total resistance of the vasculature involved in determining cerebral blood flow.

The technique and limitations of the open-calvarium preparation used in this study are discussed in detail elsewhere (5). In spite of pH buffering and temperature control of the bathing solution, the milieu of the surface vessels is undoubtedly altered, especially with respect to Pco₂ and Po₂. Exposing the bathing solution to room air causes reduced Pco₂ and increased Po₂; these changes as well as possible undetected blood on the brain surface may produce significant vasoconstriction of the surface vasculature. This suggests that the estimation of pressure difference between the
largest and smallest arterial vessels of the pial vasculature, which ranged as high as 21% of the total pressure drop in this study, may be an overestimate.

The relatively large pressure drop across the arteries between the aorta and the arteries of the brain surface, as indicated in Figures 2, 4, and 5, has been reported in several other studies (8, 14-18). Based on anatomic measurements showing relatively long cerebral arteries and relatively short arterioles, theoretical hydrodynamic calculations taking into account the high blood flow to the brain led Fukasawa (19) to conclude that there was "a large pressure drop in the region of large arterial branches and a relatively insignificant role of the arteriolar region in providing a resistance to cerebral blood flow." The results from our micropuncture pressure measurements generally agreed with this prediction over the range of systemic blood pressures studied.

The brain surface arterial network is not only highly variable in terms of patterns of branching, with small arterioles sometimes arising directly from relatively large arteries, but the arterial network is richly anastomotic (13). Many of these anastomoses occur rather directly between surface arteries and small arterioles. These anatomic factors, with the inherent variable parallel blood flow and resistance pathways, probably contribute to the observed low correspondence between arterial diameter and micropuncture pressure measurements in this study (Fig. 4).

Dilation of pial arteries and arterioles in response to an increase in arterial Pco2 has been well documented. Wolff and Lennox (10) reported increases in pial artery diameters when arterial Pco2 was increased in cats. Raper et al. (11) reported similar but larger changes in surface arterioles less than 90μ in diameter. Arterioles less than 40μ in diameter yielded greater percent changes in diameter than did larger ones in their study. These results indicate that there is a gradient of responsiveness to CO2. Raper et al. deduced that significant changes in pressure occur in these vessels as a result of such a gradient of responsiveness. Kanzow et al. (20) reported that the 40% pressure drop they found between the aorta and pial arterioles 30-40μ in diameter in the cat middle cerebral artery did not vary appreciably either with inhalation of CO2 or with infusions of norepinephrine. From this observation they concluded that there were proportionately distributed resistance changes in the vasculature upstream and downstream from the pial arterioles. In about half the vessels in our study we also found no correlation between CO2 and pressure responsiveness, suggesting proportionate changes. However, in the other half of the vessels, responsiveness was either increased or decreased with increasing CO2 (Table 1). Among the vessels in the latter group there was no correspondence between vessel size and response.

Unfortunately the effects of factors such as the artery-arteriolar anastomoses in the pial vasculature preclude detection in our preparation of pressure gradient changes as distinct as those postulated by Raper et al. (11) with increasing CO2. For example, in an anastomotic network, dilation of a vessel at one location may yield either an increase or a decrease in pressure at another measurement site depending on the effect of the dilation on upstream and downstream resistance ratios and on resistance changes relative to those in the alternative parallel pathways. It is possible that the shifts in microvascular flow patterns implied by the variable pressure responses (Table 1) in the face of a gradient of vascular responsiveness to CO2 (Raper et al. [11]) may be involved in local metabolic regulation, as suggested by Wahl et al. (12).

As mentioned above, although it is hazardous to postulate resistance changes from pressure measurements in the pial arterial network where parallel pathways and anastomoses are abundant, by statistically considering pressure drops between the largest surface arteries and the smaller arterioles, it is possible to infer the relative potential contribution of the surface arterial vasculature to the resistance changes involved in cerebrovascular control. This relative contribution, as well as
that of the upstream and downstream vasculature, is indicated in Figure 6.

Since the fractional pressure drop across the vasculature downstream from the pial surface in our preparation increased with increasing systemic blood pressure while the fractional drop across the pial arterial network and across the arteries upstream from the surface decreased, it is likely that the resistance changes in response to pressure changes in this preparation were not proportional. Furthermore, although the proportion of pressure head lost across the upstream arteries was relatively large, it decreased with increasing systemic blood pressure, suggesting that the postulated vasoconstriction of the larger arteries (21) was overshadowed during the pressure response by a greater effective constriction of the parenchymal or other downstream vasculature, or both. Similarly, vasoconstriction in the pial surface arterial network (9, 22) was also apparently overshadowed by downstream effects in our preparations. Therefore, if the observed changes in segmental pressure drops represent part of an autoregulatory response distributed among different parts of the cerebral vasculature, these results indicate that the response occurred predominantly in the vasculature distal to the site of arteriolar penetration into the brain.

Acknowledgment

We are pleased to acknowledge the contributions of Dr. C. A. Wiederhielm, Dr. H. M. Shapiro, Mr. D. B. Lee, Mr. R. B. Heald, Mr. B. V. Weston, Mrs. M. Wade, Dr. M. Shaw, and Mrs. M. Siew.

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Circ Res. 1972;31:229-239
doi: 10.1161/01.RES.31.2.229

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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