Comparison of $^{85}$Krypton and $^{133}$Xenon Cerebral Blood Flow Measurements before, during, and following Focal, Incomplete Ischemia in the Squirrel Monkey

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ABSTRACT

A comparison of regional cerebral blood flow measurements made with beta- and gamma-emitting isotopes revealed good correspondence in areas of normal perfusion and reactive hyperemia but poor correspondence in areas of focal ischemia. After middle cerebral artery occlusion at normocapnia, there was a 65% reduction in regional cerebral blood flow from $1.40 \pm 0.27 \text{ ml/g min}^{-1}$ to $0.49 \pm 0.10 \text{ ml/g min}^{-1}$ in monkeys studied with $^{85}$Kr but only a 27% reduction in regional cerebral blood flow from $0.84 \pm 0.09 \text{ ml/g min}^{-1}$ to $0.61 \pm 0.08 \text{ ml/g min}^{-1}$ in monkeys studied with $^{133}$Xe. The lack of correlation within areas of focal, incomplete ischemia was attributed to an impairment of isotope delivery to the area of ischemia coupled with the inherent lack of spatial resolution of determinations made with $^{133}$Xe. This finding may partly explain the numerous discrepancies in experimental and clinical studies of the effects of alterations in the arterial partial pressure of $CO_2$ on regional cerebral blood flow in areas of ischemia; it may also explain the failure of such studies to reflect the true severity of focal ischemia.

KEY WORDS
cerebral autoregulation
luxury perfusion
compton's scatter
look-through phenomenon
reverse steal

Clinical and laboratory investigators generally agree that use of $^{133}$Xe provides good correspondence and documentation for variations in regional blood flow in the normal brain caused by alterations in the level of arterial carbon dioxide tension ($PCO_2$) (1-4), "luxury perfusion" (5-8), and vasoparalysis in areas of ischemia with a loss of autoregulation (5-8). However, conflicting data have been reported for the severity of reduction in (4-6, 9) and the influence of altered arterial $PCO_2$ on (4-6, 10) regional cerebral blood flow in areas of infarction.

The present study was undertaken in a proved model of focal, incomplete ischemia (11, 12) to determine if these discrepancies are due to the lack of spatial resolution inherent in techniques that depend on a gamma emitter. Errors from the "look-through" phenomenon (6, 8) and Compton's scatter (13-15) have been cited by knowledgeable workers in this field but have not been documented in laboratory models.

Methods

LABORATORY PREPARATION

The surgical technique, methodology of cerebral blood flow measurements, injection of indicators into an isolated right internal carotid artery system, and recording of systemic parameters in this laboratory preparation of focal, incomplete ischemia in the squirrel monkey have been previously described in detail (11, 12). In the present study, the transorbital approach (16, 17) was substituted for the retro-orbital approach. Two groups of monkeys, anesthetized with sodium pentobarbital (20 mg/kg, ip) and separated according to the indicator used to determine regional cerebral blood flow, i.e., $^{85}$Kr or $^{133}$Xe, were studied. In the $^{85}$Kr group, a right frontotemporoparietal craniectomy and dural resection allowed recordings to be made from a brain protected by Saran Wrap. In the $^{133}$Xe group, craniectomy was unnecessary. Recordings in both groups were from identical right frontoparietal areas, and the protocol for each experiment was identical in the two groups except for the indicator used. Determinations of regional cerebral blood flow were obtained at 20-minute intervals at variable levels of arterial $PCO_2$, for 90 minutes prior to occlusion of the right middle cerebral artery, for 70 minutes during occlusion, and for 30 minutes after release of occlusion (Figs. 1 and 2). Arterial $PCO_2$ was varied by altering the concentration of inspired
CEREBRAL BLOOD FLOW IN FOCAL ISCHEMIA

FIGURE 1
Measurements of regional cerebral blood flow (rCBF) obtained with $^{85}$Kr (solid line) and $^{133}$Xe (broken line) before, during, and after focal ischemia induced by temporary occlusion of the middle cerebral artery (MCA) in the squirrel monkey as calculated by kinetic analysis. Variations in flow are recorded before, during, and after ischemia with alterations in arterial carbon dioxide tension ($P_{\text{a},\text{CO}_2}$) and mean arterial blood pressure (MABP). Reduction in mean arterial blood pressure with hypocapnia in this preparation could have minimized a reverse steal. During ischemia, the values obtained with $^{133}$Xe did not reflect the severity of flow reduction measured in the $^{85}$Kr group. Regional cerebral blood flow continued to vary in response to alterations in arterial $P_{\text{a},\text{CO}_2}$ in the $^{133}$Xe group but did not change significantly throughout the period of ischemia in the $^{85}$Kr group. Failure to accurately reflect the significant reduction in regional cerebral blood flow during ischemia and the persistence of CO$_2$ responsivity in the monkeys studied with $^{133}$Xe is attributed to look-through phenomenon and Compton's scatter. Asterisk indicates mean values ± SE obtained during spontaneous respiration.

FIGURE 2
Measurements of regional cerebral blood flow (rCBF) obtained with $^{85}$Kr (solid line) and $^{133}$Xe (broken line) before, during, and after focal ischemia induced by temporary occlusion of the middle cerebral artery (MCA) in the squirrel monkey as calculated by initial slope analysis. In this preparation, initial slope analysis correlated closely to the gray matter flow of exponential analysis ($r = 0.98$). The artifact of the look-through phenomenon could not be eliminated by methods of analysis using only the fast component of the $^{133}$Xe washout curve. Asterisk indicates mean values ± SE obtained during spontaneous respiration. See Figure 1 for definition of other abbreviations.
kinetic analysis also includes a contribution from white matter. The more complex calculations involved in exponential analysis were not performed for two reasons. (1) Regional cerebral blood flow calculated by the initial slope formula correlates very closely with the value for gray matter flow obtained by exponential analysis ($r = 0.98$) (18, 20) and (2) the relative contributions of the two components (gray matter [Wg] and white matter [Ww]) derived from exponential analysis vary as much as 30% during successive injections, despite relatively constant systemic blood pressure and arterial PCO$_2$ levels (18). Correspondence of changes was excellent between the two techniques (the values obtained by the initial slope technique were higher than those obtained by kinetic analysis in both groups of monkeys). Although regional cerebral blood flow measured by both techniques is reported in the present paper, conclusions were based primarily on the data obtained by the kinetic analysis, reflecting our confidence in the superior reliability of this technique.

Arterial partial pressure of CO$_2$–regional cerebral blood flow response curve for measurements of $^{133}$Xe before middle cerebral artery occlusion (solid line) indicated a normal-appearing curve (kinetic analysis). After middle cerebral artery occlusion (broken line), measurements of regional cerebral blood flow (rCBF) continued to vary directly with alterations in arterial carbon dioxide tension (Pa$_{CO_2}$), indicating a failure of this indicator to reflect the true degree of ischemia in the area immediately under the probe (look-through phenomenon). Monkeys breathing spontaneously prior to controlled ventilation (open circle) were also excluded from the plotted curve in this group (see Fig. 3). Values are means ± SE.

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### Arterial Partial Pressure of Carbon Dioxide–Regional Cerebral Blood Flow Response Curve

The normal physiological response of cerebral blood flow to variations in arterial PCO$_2$ was determined for monkeys studied using $^{85}$Kr (Fig. 3) and $^{133}$Xe (Fig. 4) prior to and after right middle cerebral artery occlusion. These curves were created from the mean values of regional cerebral blood flow determinations at 20, 30, 40, and 60 torr and represent 20 separate regional cerebral blood flow determinations in each group. Regional cerebral blood flow values in spontaneously breathing monkeys (arterial PCO$_2$, 33 torr) were not included in the curves. Only monkeys demonstrating a physiological response to alterations in arterial PCO$_2$ and thereby representing reliable preparations were included in this analysis (21).

### Calibration of the Geiger-Müller Probe

Several unsuccessful attempts to calibrate the Geiger-Müller probe with equipment available in our laboratory were made. In each case, calibration could not be performed because a point source of beta particles with sufficient potency to penetrate the walls of the water beaker was not available. However, based on data provided by the manufacturer (Electronics, Optical, Nuclear Corporation) and the Department of Health, Education and Welfare (22), a figure of the field of view...
of the argon-quenched Geiger-Müller tube was constructed. Only the activity coming from a discrete volume of tissue 2.5 mm in depth with side scatter limited to approximately 1° is detected (Fig. 5). This depth is slightly greater than that reported previously by other investigators (23-25), but it still corresponds closely to their results.

**ISORESPONSE CURVE FOR $^{133}$Xe**

The isoresponse curve for the detection of the gamma photon of $^{133}$Xe by the detector used in this study was determined (13). The detector was placed perpendicular to the 2-mm side wall of a beaker filled with water. A discrete point source of $^{133}$Xe was placed directly in front of the probe in the central portion of the field of view of the probe, and the maximal counting rate was determined. The point source was moved through the field of view of the scintillation detector, and the maximal counting rate was determined at 5-mm intervals. The lower level discriminator was set at 75 kev so that only the 81-kev photopeak and a small portion of Compton's scatter was detected. This setting corresponds to that used during the experimental procedure. Areas with relatively equal levels of counts were then plotted, and an isoresponse curve was obtained (Fig. 6). The average coronal diameter of the squirrel monkey brain underlying the probe was 33.8 mm. The scalp, calvarium, and dura mater accounted for 3 mm of the distance between the face of the collimator and the brain. The sagittal plane of the brain was located 22 mm from the opening of the collimator. Thus, the 50% line of the isoresponse curve passed 3.0 mm across the sagittal plane into the left cerebral hemisphere. However, a significant amount of indicator probably did not perfuse the left hemisphere, and even if it did the error introduced would be similar only to that created by the activity originating in the right parasagittal area supplied by the right anterior cerebral artery to the nonischemic tissue adjacent to the territory of the right middle cerebral artery.

**DETERMINATION OF COMPTON'S SCATTER**

The total activity detected by the thallium-activated sodium iodide probe also contained Compton's scatter photons. To quantify the contribution of Compton's scatter to total activity and, hence, to more clearly define the physical limits of anatomic resolution of the $^{133}$Xe technique, a study was performed according to the method of Potchen et al. (14, 15). A glass cylinder with a glass cone inside that duplicated the theoretical truncated cone was placed over the $^{133}$Xe probe (Fig. 7). The cylinder outside the cone was filled with a saline solution of $^{133}$Xe (3 ml $^{133}$Xe/125 ml saline), and the activity was measured at 5-kev intervals from 25 kev to 120 kev with air in the central cone. This measurement represented background activity with negligible Compton's scatter. The central cone was then filled with saline to simulate Compton's scatter comparable with that of tissue, and determinations of the activity were repeated. Finally, the central cone was filled with saline containing the same concentration of $^{133}$Xe used in the exterior chamber, and this activity was also determined. The contribution of Compton's scatter was determined by subtracting the values obtained with air in the central cone from those obtained when water filled the central cone. There was significant activity contributed by Compton's scatter (Fig. 8). There was also a 13-kev shift in the peak activity detected when the central cone was filled with water. A 75-kev lower window eliminated most of the activity contributed by Compton's scatter (Fig. 8). However, of the total activity above 75 kev recorded in this model with both compartments filled with $^{133}$Xe, 12.5% was still due to Compton's scatter.
Results

STABILITY AND RELIABILITY OF LABORATORY PREPARATION

Loss of blood during the entire experimental procedure was less than 3 ml; transfusion of blood was never necessary. Based on the respiratory rate and the degree of spontaneous movements, none of the monkeys included for final analysis was in an excessively deep plane of anesthesia. It was never necessary to supplement the original dose of anesthetic. Serial hematocrit levels performed throughout the experiment demonstrated no hemodilution or evidence of significant loss of blood. Core body temperature was maintained at 36.5°C throughout the experiment by means previously described for this preparation (11, 12). Monkeys in which a physiologic response to alterations in arterial PCO₂ could not be demonstrated prior to occlusion of the middle cerebral artery were excluded from the study. In these preparations, the cause of the impaired response was not always apparent. However, in two of the monkeys, it was considered to be a secondary effect of the craniectomy without gross evidence of cortical damage, in two other monkeys, it was assumed that minute amounts of air entered the injecting system used to deliver the radioactive indicator, and in three monkeys, transient hypotension and hypoxia occurred with curarization and institution of mechanical ventilation.

REGIONAL CEREBRAL BLOOD FLOW DETERMINATIONS

Alterations in regional cerebral blood flow caused by changes in arterial PCO₂ before, during, and after occlusion of the middle cerebral artery are summarized for both groups by kinetic analysis (Fig. 1) and initial slope analysis (Fig. 2). Regardless of the method of calculation, the degree of flow reduction measured with ⁸⁵Kr far exceeded that measured with ¹³³Xe during ischemia. Mean regional cerebral blood flow remained nearly constant throughout the period of ischemia despite alterations in arterial PCO₂ when ⁸⁵Kr was used as the isotope by both methods of analysis, although values obtained with the initial slope technique were higher than those obtained with kinetic analysis. However, when ¹³³Xe was used as the isotope, mean regional cerebral blood flow continued to vary directly with arterial PCO₂ throughout the period of ischemia regardless of the method of analysis (kinetic analysis range 0.28 ± 0.04 ml/g min⁻¹ to 0.83 ± 0.11 ml/g min⁻¹, initial slope range 0.28 ± 0.05 ml/g min⁻¹ to 1.30 ± 0.21 ml/g min⁻¹). Examination of individual arterial PCO₂-regional cerebral blood flow response curves during ischemia in the ⁸⁵Kr group revealed one monkey with a definite increase in flow during hypercapnia (0.53 ml/g min⁻¹ to 0.82 ml/g min⁻¹, arterial PCO₂ 25 torr to 56 torr, and mean arterial

Determination of Compton’s scatter using the scintillation counter employed for measurements of ¹³³Xe in the monkeys indicated a significant contribution from Compton’s scatter that could not be excluded.

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blood pressure 107 mm Hg to 119 mm Hg) and one monkey with an increase in flow in response to hypocapnia (0.65 ml/g min⁻¹ to 0.90 ml/g min⁻¹, arterial PCO₂ 36 torr to 24 torr, mean arterial blood pressure 105 mm Hg to 99 mm Hg). These two monkeys were the only ones in the ⁸⁵Kr group in which regional cerebral blood flow changed significantly in response to alterations in arterial PCO₂ during the period of ischemia. The changes in regional cerebral blood flow measured in the first monkey could have been secondary to increased perfusion pressure, and those in the second monkey might represent an "inverse steal" syndrome. We attribute the apparent continued physiological response to alterations in arterial PCO₂ during ischemia as measured with ¹³³Xe to the look-through phenomenon. This inherent lack of spatial resolution of ¹³³Xe-regional cerebral blood flow studies may explain previous observations of increased flow within focal areas of ischemia in response to hypercapnia (4, 10).

ARTERIAL PARTIAL PRESSURE OF CARBON DIOXIDE-REGIONAL CEREBRAL BLOOD FLOW RESPONSE CURVES

The shapes of the arterial PCO₂-regional cerebral blood flow response curves (kinetic analysis) for studies performed using ⁸⁵Kr and ¹³³Xe were similar prior to middle cerebral artery occlusion (solid lines, Figs. 3 and 4); the higher flows measured with ⁸⁵Kr reflect essentially gray matter flow. During ischemia (broken lines, Figs. 3 and 4), there was no significant change in regional cerebral blood flow in response to alterations in arterial PCO₂ in monkeys studied with ⁸⁵Kr, whereas a direct positive correlation was found in monkeys in which ¹³³Xe was used as the isotope (values obtained by kinetic analysis showed less change than values obtained by the initial slope technique). For reasons not clear to us, routinely there was a modest increase in regional cerebral blood flow when monkeys were placed on controlled ventilation compared with base-line levels obtained with monkeys breathing spontaneously at the same level of arterial PCO₂.

Discussion

COMPARISON OF BETA AND GAMMA MEASUREMENTS

The Geiger-Müller probe recorded beta particles originating from very superficial layers of the brain and did not detect significant activity from a depth greater than 2.5 mm. This situation is consistent with data on the characteristics of the indicator that are accepted by most workers in the field (23–25). As previously indicated, counts were recorded through Saran Wrap, since the dura in this preparation has been demonstrated to represent a significant barrier to this indicator (Fig. 5) (11, 24).

Examination of the theoretical truncated cone (Fig. 7), the measured isoresponse curve (Fig. 6) for the detection of gamma activity, and the information relative to possible contamination from Compton’s scatter (Fig. 8) indicates the potential error introduced by the lack of spatial resolution for a gamma-emitting indicator. Figures 1 and 2 demonstrate the inability of regional cerebral blood flow determinations with ¹³³Xe, using the instrumentation of the present study, to accurately reflect the severity of blood flow reduction in regions of very severe ischemia regardless of the method used to calculate regional cerebral blood flow. This finding confirms previous observations by Paulson and co-workers (13) on problems related to the look-through phenomenon.

ARTERIAL PARTIAL PRESSURE OF CARBON DIOXIDE-REGIONAL CEREBRAL BLOOD FLOW RESPONSE CURVES

The predictable shape of the arterial PCO₂-regional cerebral blood flow response curves for monkeys studied with ⁸⁵Kr and ¹³³Xe indicated the reliability of these measurements in nonischemic brain prior to occlusion of the middle cerebral artery. The measurements of each preparation’s response to alterations in arterial PCO₂ prior to occlusion of the middle cerebral artery and the exclusion from study thereafter of all monkeys failing to demonstrate a normal autoregulatory response ensured that these experiments were performed in reliable laboratory preparations. Lassen (21), Ingvar and Lassen (26), and others (27–29) have previously stressed the importance of such precautions in both laboratory and clinical studies.

⁸⁵Kr AND ¹³³Xe MEASUREMENTS OF CEREBRAL BLOOD FLOW

There was good correspondence between the two groups of monkeys during intervals in which there was no obstruction or impairment of the delivery of indicator into the region predestined for ischemia. Alterations in regional cerebral blood flow caused by
changes in arterial PCO₂ prior to occlusion of the middle cerebral artery and luxury perfusion (reactive hyperemia) were of similar magnitudes in both groups of monkeys. However, during ischemia, when the relative amount of indicator delivered to the area directly under the probe was markedly reduced, a significant artifact arose in the 133Xe group from lack of spatial resolution. To detect the true reduction of flow in a focal area of ischemia using a gamma emitter, the concentration of indicator in the region to be measured must be equal to that in the underlying and adjacent areas. When regional cerebral blood flow measurements are performed during carotid endarterectomy, the indicator is delivered to the brain prior to carotid artery occlusion, and the relative reduction of flow is accurate unless there has been an intracranial occlusion to prevent the indicator from arriving in the area to be measured (30-32). However, with an intracranial occlusion, such as that which results from an embolus, before the injection of 133Xe, a representative amount of 133Xe may not arrive in the area of focal ischemia, and counts arriving under the probe may originate from deeper or adjacent tissue and contribute to the clearance curve measured by the probe (6, 8, 32).

Multiple small probes may partly reduce this artifact, and the probes can be collimated so that they record from a relatively small volume of tissue (9, 10, 13). Collimation may narrow the theoretical cone of tissue within the field of view of the detector, but depth resolution remains a function of the linear absorption coefficient for the gamma photon of 133Xe. Problems related to Compton's scatter also may result (13-15), and they are especially important for gamma photons whose energy is below 1.0 mev (33). Compton's scatter involves the interaction between an incident photon and an orbital electron in which only a part of the energy of the photon is imparted to the electron (34). The photon emerges from the interaction with a new direction and reduced energy (34). The effect of Compton's scatter on the counts detected using 133Xe can be approximated in phantom studies (13-15). Depending on the characteristics of the scintillation detectors and the photopeaks measured, studies reported previously have shown that between 35% (13) and 55% (14) of the total activity seen by the probe may not come from tissue within the truncated cone underlying the probe. Compton's scatter in effect increases the volume of tissue from which gamma photons are counted and may introduce a considerable error in the determinations of the volume and the severity of focal ischemia. These factors introduced a major error in measurements of regional cerebral blood flow in a patient with acute occlusion of the middle cerebral artery from a cardiac source prior to intracranial embolectomy (32).

CRITICAL CEREBRAL BLOOD FLOW

There is excellent correlation between the work of Boysen (30) and that of Sundt and coworkers (32) relating to continuous electroencephalograms and cerebral blood flow measurements during carotid endarterectomy. A cerebral blood flow of 18 ml/100 g min⁻¹ is required to sustain a normal electroencephalogram for short periods (32, 35); this value agrees with that required for maintenance of normal carbohydrate metabolism (36). Invariably, flow reductions below this level produce evidence of cerebral ischemia as shown by changes in the electroencephalogram (32). These measurements are free of the artifacts of the look-through phenomenon as previously discussed, because the indicator is delivered to the brain and the artery is occluded in the neck only after counts are identified. Therefore, true regional cerebral blood flow is reflected. On occasion, values as low as 5 ml/100 g min⁻¹ have been found during carotid occlusion (prior to placing a shunt); such values are much lower than those reported in most clinical studies in patients with infarction (4-8, 37, 38). Our measurements were performed under halothane anesthesia with a moderate degree of induced hypertension; nevertheless, they reflect the amazing ability of the brain to compensate for severe reductions in flow up to a point of critical perfusion. Comparison of these critical levels of flow with regions of focal ischemia found in clinical studies using 133Xe in patients with acute and chronic infarctions (4-8, 37, 38) indicates the significant possibility that the true severity of regional flow impairment has not been identified because of the problems of the look-through phenomenon and Compton's scatter. This possibility does not mini-
mize the importance of such investigations, but it does indicate the necessity of understanding the possible contribution of counts from underlying or adjacent perfused tissue. The more sophisticated instrumentation and techniques for measurement currently under development in which the relative number of counts is also considered may partly circumvent this obstacle.

Steal and Reverse Steal

The failure to more clearly document steal and reverse steal in our laboratory preparation does not indicate that they do not occur. Waltz (29) has previously suggested that the paradoxical response to hypercapnia (intracerebral steal) is related to the volume of tissue involved, the severity of ischemia, and the time delay between the ischemic episode and regional cerebral blood flow measurement (39). Previously, it has been demonstrated (40) that the squirrel monkey has a large area of infarction after permanent occlusion of the middle cerebral artery. In animals with a lesser degree of ischemia such as the cat, these paradoxical responses to hypercapnia have been documented (29). In other experimental preparations with relatively smaller areas of ischemia, some investigators (10, 41, 42) have demonstrated by other techniques of cerebral blood flow measurement an increase in cerebral blood flow after an increase in arterial PCO₂. The inability to demonstrate more commonly the paradoxical responses to hypercapnia and hypoxia in clinical situations is probably inextricably related to the limitations of spatial resolution imposed by the use of a gamma indicator and to the temporal relationship of the onset of the ischemic deficit and the regional cerebral blood flow measurement. Autoradiographic measurements (43) have indicated the profound variability of flow within discrete regions in and adjacent to the area of ischemia; these variations range from 3 ml/100 g min⁻¹ in the core area of infarction to more than 100 ml/100 g min⁻¹ in marginal zones of hyperemia (44). It is unlikely that this flow pattern in ischemic areas will be accurately detected by current techniques of external counting.

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