

Calculation of Coronary Blood Flow from Myocardial Clearance of Systemically Administered ^{133}Xe

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Clearance curves for arterial and coronary-venous blood were determined after systemic left-ventricle or pulmonary-artery injections of ^{133}Xe , paired with selective left-coronary-artery injections of ^{133}Xe in 20 dogs with closed chest. Coronary blood flows calculated from systemic and coronary-artery injections were comparable only when a correction was made for arterial recirculation of ^{133}Xe following the systemic injection ($r = 0.962$ for left ventricle and 0.932 for pulmonary artery, paired with coronary artery). Experiments in four other dogs verified that clearance of ^{133}Xe from the pulmonary circulation was only about 60%. The myocardium/blood ^{133}Xe partition coefficient, determined in vivo in ten dogs, agreed within 10% with that previously determined in vitro.

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Various procedures have been used to estimate coronary blood flow from the rate of clearance of inert diffusible gases from the myocardium. Their limitations have been reviewed (1,2). Either ^{85}Kr or ^{133}Xe has been injected into the coronary artery or the left ventricular chamber and the myocardial clearance rate determined from the decline in gamma emission detected precordially or in coronary-venous blood samples. With coronary-artery injections, clearance rates determined precordially correspond with rates determined from coronary-venous blood samples (3). Furthermore, the use of precordial gamma detection after coronary-artery injection of ^{85}Kr or ^{133}Xe has been substantiated independently by simultaneous ratemeter-measured coronary blood flow (4,5), and myocardial clearance determined from coronary-venous blood samples after systemic injection of ^{85}Kr into the left ventricle correlates with coronary bloodflow rates measured by nitrous oxide or ^{131}I -antipyrine (6).

These observations suggest that when its distribution is not overtly heterogeneous, coronary blood flow can be measured satisfactorily by ^{85}Kr or ^{133}Xe injected at either site. There are advantages in a technique that avoids coronary-artery injection. However, since the myocardial clearance could be influenced by continued introduction of indicator

into the myocardium from arterial blood, clearance of the gas from the blood by the lungs becomes critical with the larger injectate needed for systemic injection.

The experiments to be reported show that: (A) ^{133}Xe is poorly cleared by the lungs; (B) arterial recirculation after systemic ^{133}Xe administration causes falsely low coronary blood flow as calculated from coronary-venous clearance curves alone; and (C) when arterial recirculation is properly taken into account by simultaneously determined arterial and coronary-venous clearance curves, coronary blood flow determined after ^{133}Xe injection into either left ventricle or pulmonary artery agrees well with that obtained through coronary-artery injection. This report describes a technique for determining coronary blood flow by the inert-gas clearance method without coronary-artery injection. However, this modification will probably retain the limitations previously described for other clearance methods (1). In particular, our clearance techniques failed to discriminate between regions of uneven myocar-

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dial perfusion detectable by other methods (7,8), and in the presence of heterogeneous blood flow, they give falsely high values (9).

We also determined the ^{133}Xe partition coefficient between myocardium and blood in intact dogs. The value obtained validates within 10% the currently accepted value based on in vitro data (10,11).

METHODS

Procedures. Coronary blood flow was determined by the ^{133}Xe myocardial clearance technique (closed chest) in 20 mongrel dogs weighing approximately 20–30 kg each. The dogs were either anesthetized with pentobarbital, 20–30 mg/kg, or tranquilized with Acepromazine,* 1 mg/kg, followed by 30 mg of intramuscular morphine. Respiration was spontaneous. Via neck vessels, catheters were positioned with tips in the left ventricle, aortic arch, coronary sinus, and pulmonary artery. Through a femoral artery, a catheter was positioned at the ostium of the left coronary artery, with continuous pressure monitoring to ensure that the catheter did not obstruct coronary blood flow. To achieve a wide range of coronary bloodflow rates, coronary flow was increased in some experiments by atrial pacing or by isoproterenol or atropine administration and decreased in others by phlebotomy or propranolol administration.

Xenon-133, dissolved in saline, was administered as a 1-ml bolus. The bolus injection into the left ventricle (100 μCi) or pulmonary artery (300–400 μCi) was followed by a flush of 5–10 ml of saline. To minimize effects on coronary vascular resistance, coronary artery injections (5 μCi) were not flushed with saline. For paired coronary bloodflow determinations, a coronary artery injection preceded the other injections by 5 min, and hemodynamic steady-state conditions were verified by monitoring systemic arterial pressure and heart rate.

After ^{133}Xe injection, blood samples for radioassay were obtained simultaneously through two manifolds, one connected to the catheter in the coronary sinus and the other to the catheter in the aortic arch. Blood was withdrawn at a steady rate of 0.1 ml/sec, and 1.5-ml samples of arterial and coronary-venous blood were collected every 15 sec. The dead space from each catheter tip to manifold was 2.5 ml.

Xenon partition between myocardium and blood was investigated in ten other dogs anesthetized with pentobarbital. The trachea was cannulated, and respiration was controlled by a Harvard pump connected to a closed system spirometer containing a CO_2 absorber and 10 liters of O_2 . Four hundred microcuries of ^{133}Xe gas was injected into the spirometer, and paired arterial and coronary-venous blood samples were obtained at 2-min intervals for

10 min. The chest was then opened and an arterial blood sample and a 2–4-gm piece of left ventricular myocardium were taken as quickly as the heart could be exposed (2.5 min). The myocardial sample was quickly divided into three portions, two being sealed within 10 sec while the third was exposed to air for 1.5 min. Gamma emission was found lower in the third sample by a mean of only 11%, indicating that ^{133}Xe loss from the rapidly handled samples was probably negligible.

The concentration of ^{133}Xe in blood and myocardial samples was determined by scintillation counting in a well system.†

Calculations. The introduction of an indicator gas into the myocardium permits measurement of coronary blood flow (CBF) based on the Fick principle (5,12):

$$\text{CBF} = \frac{\Delta Q_i / \Delta t}{C_a - C_v}, \quad (1)$$

where C_a and C_v are the concentrations of the gas in the arterial and coronary venous blood, respectively, and Q_i is the quantity of gas in the myocardium. Although Q_i is not measured, it depends on the concentrations of gas in the myocardium (C_i) and the coronary sinus (C_v); the partition coefficient for this equilibrium, λ , is equal to C_i / C_v .

Further, $C_i = Q_i / V$, where V is the volume of myocardium containing Q_i . Then, by substitution in Eq. (1), we have

$$\text{CBF} = \frac{\lambda V}{C_a - C_v} \times \frac{\Delta C_v}{\Delta t}, \quad (2)$$

which in differential notation is

$$\text{CBF} = \frac{\lambda V dC_v / dt}{C_a - C_v},$$

whence,

$$\frac{dC_v}{dt} = \frac{\text{CBF}}{\lambda V} (C_a - C_v).$$

This differential equation is readily solved to give C_v as a function of time, provided the time variation of C_a is also known. However, if C_a is a constant fraction of C_v (i.e., if $C_a / C_v = \alpha$), then the solution of this equation is

$$C_v(t) = C_v(0) \exp \left[- \frac{\text{CBF}}{\lambda V} (1 - \alpha) t \right].$$

Thus, C_v exhibits a pure exponential decay. This has been observed for the period of washout used to measure flow (see Results). If $T_{1/2}$ is the time for C_v to decay to half its initial value, then

$$\text{CBF} = \frac{0.693 \lambda V}{T_{1/2} (1 - \alpha)}.$$

TABLE 1. CLEARANCE OF ^{133}Xe FROM THE BLOOD BY THE LUNGS

Experiment	Time after left-ventricle injection of ^{133}Xe (sec)				
	60-75	75-90	90-105	105-120	120-135
1	66%	58%	57%	54%	57%
2	68	58	51	55	55
3	67	64	59	57	60
4	4	66	54	64	69
5	20	51	67	69	77
6	40	59	55	61	65
7	56	55	59	64	64
Mean \pm s.d.	46 \pm 26%	59 \pm 5%	57 \pm 5%	61 \pm 5%	64 \pm 5%

Numbers represent percent ^{133}Xe cleared: (conc pulmonary art - conc aorta \div conc pulmonary art) \times 100%.

Moreover, since $\alpha = \text{Ca}/\text{Cv}$, $(1 - \alpha) = \text{Cv} - \text{Ca}/\text{Cv}$. Substituting in the foregoing equation, we have

$$\text{CBF} = \frac{0.693 \lambda V}{T_{1/2}} \times \frac{\text{Cv}}{\text{Cv} - \text{Ca}} \quad (3)$$

The volume of myocardium, for convenience, is defined as a specified weight/density, in this case, 100 gm/1.05. For xenon, the usual value assigned λ is 0.72. Therefore, we have

$$\text{CBF} = \frac{(0.72)(100)}{(1.05)} \times \frac{(0.693)}{(T_{1/2})} \times \frac{\text{Cv}}{\text{Cv} - \text{Ca}} \quad (4)$$

The CBF is expressed in ml/100 gm-min if $T_{1/2}$ is measured in minutes. The factor $\text{Cv}/(\text{Cv} - \text{Ca})$ corrects for arterial recirculation of the gas. Since coronary-venous xenon concentration decreased mono-exponentially, our correction of the coronary-venous clearance by the mean value of $\text{Cv}/(\text{Cv} - \text{Ca})$ when blood flow was determined is equivalent to correction of each Cv value by the simultaneous $\text{Cv}/(\text{Cv} - \text{Ca})$ value. If arterial recirculation of the gas is negligible ($\text{Ca} = 0$ and $\text{Cv}/(\text{Cv} - \text{Ca}) = 1$):

$$\text{CBF} = \frac{0.72 (100)(0.693)}{1.05 T_{1/2}} \quad (5)$$

RESULTS

Simultaneous blood samples from the aorta and pulmonary artery were taken every 15 sec after left ventricular injection of 100 μCi of ^{133}Xe in seven experiments using four dogs. During the interval between 75 and 120 sec after injection, the percent clearance of ^{133}Xe in transit through the pulmonary circulation ranged from 59 to 64% (Table 1).

To investigate the time limits of the exponential phase of decline in coronary venous ^{133}Xe concentration, blood samples were drawn at 15-sec intervals between 45 and 180 sec after left ventricle ^{133}Xe injection in five experiments on three dogs. Regression analysis of these coronary-venous curves re-

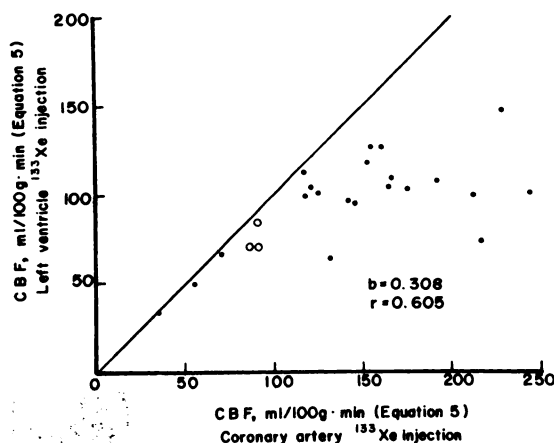


FIG. 1. Comparison of coronary blood flows (CBF) determined by ^{133}Xe injection through left ventricle and coronary artery. CBF was calculated from coronary-venous blood ^{133}Xe alone (Eq. 5). Line of identity is shown; b, regression coefficient; r, correlation coefficient.

vealed an exponential clearance between 60 and 180 sec ($r = 0.991-0.999$).

Gamma emission from the 45-60-sec sample fell below the exponential line. For all subsequent data to be presented, five paired arterial and coronary-venous blood samples were taken between 60 and 135 sec after left-ventricle or coronary-artery injections and, to allow for pulmonary transit, between 90 and 165 sec after pulmonary-artery injection. These time periods represent the earliest exponential phase of washout that would be mainly myocardial, and thus would minimize the contribution of xenon washout from fatty tissue.

Paired coronary-artery and left-ventricle ^{133}Xe injections, followed by simultaneous arterial and coronary-venous blood sampling, were performed in 21 experiments on ten dogs. After coronary-artery injection, gamma emission from arterial blood was less than 2% of that from coronary-venous blood, whereas after left-ventricle injection the percentage was 20-30%. When coronary blood flow was cal-

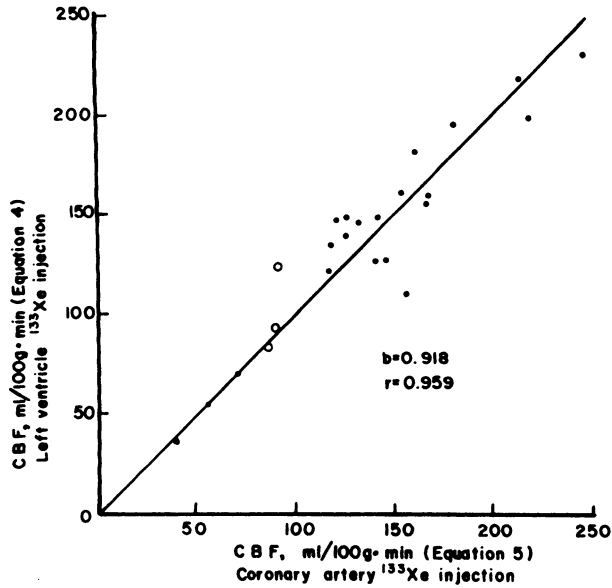


FIG. 2. Comparison of CBF determined by ^{133}Xe injection through left ventricle and coronary artery. CBF from left-ventricle injection was calculated by Eq. 4. CBF from coronary-artery injection was calculated by Eq. 5, since arterial ^{133}Xe was insignificant. Line of identity is shown.

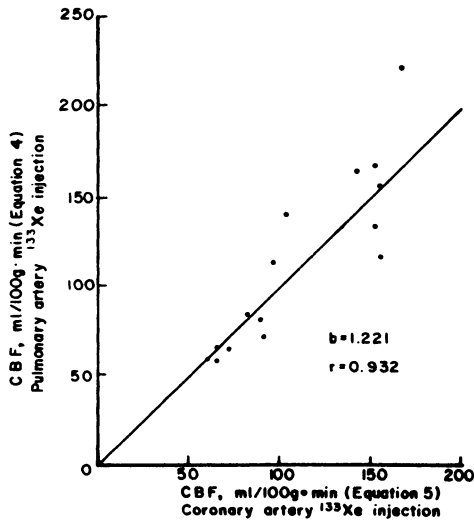


FIG. 3. Comparison of CBF determined by ^{133}Xe injection through pulmonary artery and coronary artery. CBF from pulmonary-artery injection was calculated by Eq. (4). CBF from coronary-artery injection was calculated by Eq. (5). Line of identity is shown.

culated from coronary-venous curves alone (Eq. 5), left-ventricle injection consistently gave lower values than did coronary-artery injection, especially when blood flow was high (Fig. 1), and the correlation between the results from the two injection sites was low ($r = 0.556$). The average CA-LV difference, D , was 42.6 with a s.d. of 41.9; the t-test yielded a value of 4.7 with a p value less than 0.001. On the other hand, when arterial recirculation of ^{133}Xe was

taken into account (Eq. 4), coronary blood flow calculated from coronary-artery and left-ventricle injections corresponded closely ($r = 0.962$) and scattered around the line of identity (Fig. 2). The average \bar{D} was -3.50 and s.d. = 7.60, giving a t value of 2.11, for which the p value lies between 0.05 and 0.02.

Paired coronary-artery and pulmonary-artery injections of ^{133}Xe were performed in 15 experiments on eight dogs. The correlation between paired coronary bloodflow values (Eq. 4) is illustrated in Fig. 3 ($r = 0.932$). The average $\bar{D} = -7.33$ and s.d. = 3.89, giving a t value of 1.89, for which the p value lies between 0.1 and 0.05.

Data for the partition of ^{133}Xe between left ventricular myocardium and blood in ten dogs are presented in Fig. 4. After 2 min of ^{133}Xe inhalation, the arterial blood concentration had reached 95% of the concentration after 10 min, just prior to the opening of the chest for myocardial biopsy. The slight progressive increase in blood xenon concentration between 2 and 10 min can be explained by decrease in reservoir volume due to O_2 consumption by the dog. The ratio of arterial to coronary venous concentration was essentially constant and near unity from 2 to 10 min, indicating near equilibrium between myocardium and coronary blood. The mean partition coefficient value of myocardium to arterial blood was 0.65 ± 0.04 (s.d.) when the myocardium was sampled at 12.5 min, compared to the previously reported in vitro value of 0.72 (10,11). Variations in hematocrit from 36 to 41 had no detectable effect on the partition coefficient.

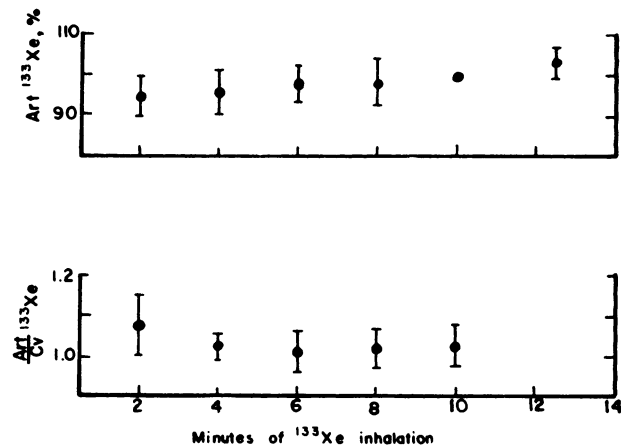


FIG. 4. Equilibration of arterial and coronary venous ^{133}Xe concentrations in experiments establishing myocardial/blood partition coefficient for ^{133}Xe . Concentrations of ^{133}Xe in arterial blood (Art ^{133}Xe) are shown at top of figure as percentages of the 10-min value. Xenon-133 arterial-to-coronary-venous concentration ratio (Art/Cv ^{133}Xe) is shown below. Mean values and s.d. are indicated.

DISCUSSION

Approximately 95% of an intravenous bolus injection of ^{85}Kr is cleared in transit through the pulmonary circulation (13). This observation is consistent with the insignificant gamma emission from arterial blood found 1–3 min after left-ventricle injection of ^{85}Kr (3), and under these circumstances coronary blood flow calculated from the coronary-venous clearance curve alone correlated with the flow calculated independently by antipyrine or N_2O methods (3).

Although it has been stated that the lungs clear xenon as efficiently as krypton (14), convincing data have not been published. Our experiments in dogs showed that only about 60% of the ^{133}Xe was removed from blood in transit from pulmonary artery to aorta (Table 1). We also found that gamma emission from arterial blood was typically about 20–30% of that from coronary-venous blood when myocardial clearance was determined 1–2 min after left-ventricle injection of ^{133}Xe . However, the coronary-artery dose required to achieve adequate myocardial and coronary-venous concentrations is small enough so that the concentration recirculating in the arterial blood (or even entering the pulmonary artery) is negligible and was found experimentally with coronary-artery injection. When ^{133}Xe is injected into the left ventricle, it is mixed systemically, and the dose required to yield satisfactory coronary-venous concentration is 20 times that for coronary-arterial injection. Under these circumstances, the concentration of ^{133}Xe in arterial blood continuing to enter the coronary arteries after 1–2 min is significant in relation to the myocardial and coronary venous concentrations, and coronary blood flow calculated from the coronary-venous clearance curve would be expected to be falsely low, as was found experimentally (Fig. 1). The higher the coronary blood flow, the less the difference between arterial and coronary-venous concentrations and, therefore, the greater the discrepancy (Fig. 1). When arterial recirculation was taken into account, however, (Eq. 4) left-ventricle and coronary-artery injections gave comparable values for coronary blood flow (Fig. 2). Unrecognized recirculation of ^{133}Xe may have contributed to the difficulty others have encountered in determining coronary blood flow from left-ventricle injection (14).

Incomplete removal of ^{133}Xe from the blood by the lungs requires the determination of gamma emission from arterial as well as coronary-venous blood. This property of ^{133}Xe technically discourages its use in determining coronary blood flow from blood clearance curves and could be expected to render precordial detection especially unreliable following

systemic injection. Incomplete pulmonary clearance, however, also enhances the flexibility of the use of ^{133}Xe . Since a large fraction escapes excretion in the pulmonary circuit, the ^{133}Xe can be injected into the venous side of the circulation by adjusting the dose to make up for the anticipated respiratory loss. Even with the required increase, the dose of 300 μCi is well below the maximum acceptable level. We found this procedure to give coronary bloodflow values that correlated well with values obtained by coronary-artery injection (Fig. 3). This technical modification avoids the more hazardous coronary-artery or left-ventricle catheterization and would facilitate determinations during muscular exercise, for example.

The limitations of these modifications are recognized. Uneven flow distribution and xenon washout from fat cannot be ruled out, but the portion of the washout curve we used is the earliest single-exponential phase seen. The fact that myocardial blood-flow measurements by this procedure agree closely with measurements by other methods (e.g., nitrous oxide washout), and the fact that the fat/blood partition coefficient for xenon is 7.94 (11), as compared to 0.72 for myocardium/blood, decrease the likelihood that xenon washout from fat is significant during the sample period.

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FOOTNOTES

- * Ayerst Laboratories, New York, N.Y.
- † Searle Radiographics, Des Plaines, Ill.

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