JCI The Journal of Clinical Investigation

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J Clin Invest. 1964;43(2):193-200. https://doi.org/10.1172/JCI104904.

Research Article

Journal of Clinical Investigation
Vol. 43, No. 2, 1964

Determination of Tissue O_2 Tensions by Hollow Visceral Tonometers: Effect of Breathing Enriched O_2 Mixtures *

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The concentrations of O_2 and CO_2 in tissues (i.e., the extracellular tissue spaces) can be an index to the relationship between the blood flow and metabolism of an organ. Since the optimal performance of an organ is expected to occur between definable limits of the tissue O_2 and CO_2 tensions (1, 2), correct values for these gas tensions delineate the most suitable relationship between flow and metabolism during various physiological circumstances.

Numerous methods have been devised to measure respiratory gases in tissues, but each is hampered by artifacts that are likely to alter the gas tensions: polarographic needle electrodes implanted in tissues often rupture capillaries or compress blood vessels (3); subcutaneous gas depots are usually associated with extensive pathological changes in contiguous tissues (4, 5); and even a method equating the gas tensions of lymph to those of interstitial fluid results in variable values (6, 7). With these difficulties, it has, for practical purposes, been assumed that the respiratory gas tensions of the draining venous blood are reasonable estimates of the tissue gas tensions; however, comparisons of values for tissue and venous blood gas tensions in a single organ in order to validate this assumption have not been performed.

The present study investigates the hollow viscus as a convenient *in vivo* tonometer in which to equilibrate liquids until they assume the gas tensions of the surrounding tissues. We found that such organs as the urinary bladder act as very rapid and convenient tissue tonometers and that it is possible to obtain corresponding samples of venous blood draining the organ to compare tissue and blood gas tensions under various experimental conditions.

* Submitted for publication August 5, 1963; accepted October 8, 1963.

This investigation was supported by a research grant (RD-1315-M) from the Vocational Rehabilitation Administration, Department of Health, Education, and Welfare, Washington, D. C.

Methods

Twenty adult mongrel dogs were used in these experiments; of these, 15 females were chosen, since it was soon observed that the veins draining the female bladder were especially large and easily isolated. Each animal was anesthetized with pentobarbital (30 mg per kg of body weight), the trachea was intubated, and alveolar ventilation was maintained constant by a Bird mechanical respirator.

Through a mid-line suprapubic incision, the ureters were ligated, and a short polyethylene catheter was inserted through a needle hole in the avascular area at the dome of the bladder. Of the four veins usually draining the bladder, one was isolated at the base of the bladder and cannulated with fine polyethylene tubing; in many instances, the cannula could be inserted without obstructing the blood flow through the vein. Even when the vein was obstructed by the cannula, there was little evidence of congestion in the area drained by the vein; it appeared that the clearly evident vascular anastomoses drained blood from the obstructed vein into the several other bladder veins remaining patent. That the gaseous composition of any one bladder vein was similar to the others and represented the mixed venous blood draining the bladder was indicated by identical values for O_2 tension in blood aspirated by needle from two or three of the bladder veins in each of two dogs. After completion of the cannulations, the bladder was replaced in its normal position in the abdominal cavity, and the skin incision was closed. The femoral artery was cannulated in the usual manner with an indwelling needle. In four animals, it was also possible to catheterize the gall bladder through a similar avascular area in its dome. In these instances, the cystic duct was ligated, but it was not technically possible to obtain adequate samples of blood from the cystic vein.

Procedure. In both the urinary bladder and the gall bladder, it was observed that some agent was often present in the urine or bile that produced progressive decreases in the O_2 tension imposed on these materials after their removal from the dog. Accordingly, 0.8%

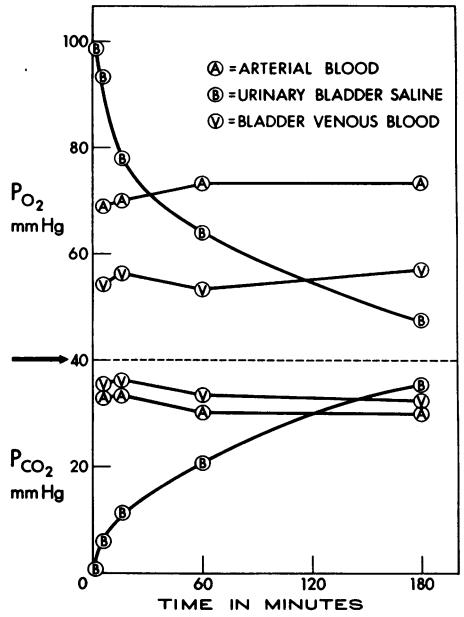

FIG. 1. CHANGES IN O_2 AND CO_2 TENSIONS (P_{O_2}, P_{CO_2}) OF SALINE INSTILLED IN THE URINARY BLADDER AND OF ARTERIAL AND VENOUS BLOOD. Each point represents the average of 11 dogs; 60 ml of saline was instilled in the bladder at time 0 with an average P_{O_2} of 99 mm Hg and an average P_{CO_2} of 0.

saline, rather than urine or bile, was used as the equilibrating liquid in both these organs.

In 16 of the 20 animals, only the urinary bladder was prepared. It was emptied of its urine and repeatedly flushed with saline until the contents no longer appeared to contain urine. The empty bladder was then filled with saline on which a known O_2 tension had been imposed by equilibrating with a gas of measured O_2 tension. The O_2 tension imposed on the saline was usually about 100 mm Hg. In 5 of these 16 dogs, 25 ml of saline was instilled; in the remaining 11 dogs, 60 ml of saline. Samples of the bladder saline, of arterial blood, and of bladder venous blood were taken 5, 20, 60, 90, and 180 minutes after the instillation of the saline, or until the gaseous composition of the bladder saline became constant. The effect of changes in the oxygenation of arterial blood was studied by altering the inspired gas mixture from room air to pure O_2. This procedure was imposed on the 11 animals with 60 ml of saline in the bladder after the gas tensions of the urinary bladder contents had stabilized. Samples of the urinary bladder saline and of arterial and venous blood were again taken periodically until changes in their gaseous composition ceased to occur.

In the four animals in which the gall bladder was also catheterized, a similar procedure was adopted. After flushes of normal saline, the gall bladder was filled with 25 ml of saline simultaneously with the filling of the urinary bladder. Samples of saline from the gall bladder were then taken along with the blood samples and the saline samples from the urinary bladder.

Several precautions were taken in the experimental procedure involving both the urinary bladder and the gall bladder: *1*) the samples of saline were limited to approximately 1 or 1.5 ml; in this manner, the volume of fluid in each of these hollow viscera did not change by more than 5 or 10 ml throughout the experiments; *2*) at the conclusion of each experiment, all the remaining urinary and gall bladder fluid was withdrawn in anaerobic fashion, so that periodic *in vitro* measurements of O_2 tension could be performed to determine the O_2 consumption of the bladder fluid itself; *3*) samples of arterial and venous blood from the urinary bladder were taken simultaneously while the bladder was full (about 60 ml of saline) and after the bladder was emptied, to determine the effects of distension of a hollow viscus on the vascular resistance to its perfusion.

Analytic technics and calculations. All blood and saline samples were drawn in an anaerobic fashion in greased syringes in which the dead space of the system was filled with a sodium heparin solution. Direct measurements of gas tensions were made by an O_2 electrode (8) and a CO_2 electrode (9) in steel and plastic cuvettes that were connected in series by rubber tubing and mounted in a thermostatic water bath. Stopcocks were so arranged that either samples or calibrating solutions could be pushed through the cuvettes in the same manner and at the same rate of flow. The calibrating solutions consisted of normal saline contained in tonometers through which bubbled gases of known O_2 and CO_2 tensions that had been previously saturated with water vapor. Both electrodes were calibrated before and after each unknown sample, and readings were made by a Beckman Spinco gas analyzer.[1] The gas tanks with which the calibrating solutions were equilibrated had previously been analyzed for their O_2 and CO_2 concentrations by the method of Scholander (10). Blood and fluid pH were measured by a glass electrode and the Radiometer potentiometric apparatus.[2] All measurements were made at 37° C.

The O_2 concentrations of blood were calculated from the O_2 saturations and hemoglobin concentrations. The O_2 saturations were obtained from the measured values for pH and O_2 tension by a standard canine oxyhemoglobin dissociation curve (11). The hemoglobin concentrations were estimated from the arterial hematocrits.

Results

Urinary bladder. The average changes in the respiratory gas tensions of the saline instilled in the bladder and the corresponding gaseous composition of arterial and venous blood are listed in Table I and Figure 1 for eleven dogs. In these animals, the initial volume of saline instilled was

[1] Beckman Instruments, South Pasadena, Calif.

[2] The London Co., Cleveland, Ohio.

TABLE I

*The relation between P_{O_2} and P_{CO_2} of arterial blood, bladder venous blood, and saline in the urinary bladder**

	Arterial				Bladder venous					Bladder contents	
	P_{O_2}	P_{CO_2}	pH	S_{O_2}	P_{O_2}	P_{CO_2}	pH	S_{O_2}	$Ca_{O_2}-C\bar{v}_{O_2}$	P_{O_2}	P_{CO_2}
	mm Hg	*mm Hg*		%	*mm Hg*	*mm Hg*		%	*ml/100 ml*	*mm Hg*	*mm Hg*
					5 minutes						
AV.	69	36	7.34	91	54	35	7.33	83	1.59	93	8
SD	9.2	11.7	0.13	5.4	8.8	12.8	0.12	5.0	1.10	11.9	2.7
SE	2.7	3.5	0.03	1.6	2.7	4.1	0.04	1.6	0.34	3.4	0.7
					20 minutes						
AV.	70	34	7.36	91	56	35	7.32	84	1.19	77	11
SD	8.1	5.3	0.15	5.1	6.7	11.9	0.15	5.7	0.76	11.4	6.6
SE	2.5	1.7	0.04	1.6	2.4	4.2	0.05	1.9	0.26	3.2	1.9
					60 minutes						
AV.	73	31	7.32	91	53	32	7.30	82	1.70	64	22
SD	6.6	8.2	0.09	2.6	4.9	8.7	0.08	2.7	0.80	13.9	4.7
SE	2.0	2.4	0.02	0.8	1.5	2.6	0.02	0.8	0.20	4.2	1.4
					180 minutes						
AV.	76	31	7.36	91	57	32	7.31	83	1.58	48	35
SD	8.5	7.7	0.10	2.4	4.9	8.4	0.10	2.4	0.76	8.3	8.2
SE	2.5	2.3	0.03	1.2	1.5	2.5	0.03	0.9	0.23	2.5	2.5
					240 minutes						
AV.	71				56					49	
SD	8.0				5.0					8.5	
SE	2.5				1.6					3.4	

* Times refer to minutes after instillation of saline in urinary bladder.

P_{O_2} = oxygen tension; P_{CO_2} = carbon dioxide tension; S_{O_2} = oxygen saturation; $Ca_{O_2} - C\bar{v}_{O_2}$ = oxygen concentration difference between arterial and venous blood; AV. = average of 11 values; SD = plus or minus 1 standard deviation; SE = plus or minus 1 standard error. As is customary, pH is expressed as the average of the pH values, rather than the average hydrogen ion concentration.

60 ml, and the average O_2 tension imposed on the saline before instillation in the bladder was 99 mm Hg. Within 5 minutes after instillation, the O_2 tension had decreased to 93 mm Hg, and within 3 hours, it had reached an average value of 48 mm Hg (SE, ± 2.5). Samples of bladder saline taken thereafter showed no further changes in gaseous composition. Figure 2 compares the rate of change in the O_2 tension of the saline when different volumes are initially instilled in the urinary bladder. Initial volumes of 25 ml require 1 hour to reach a final average value (48 mm Hg) from which no further changes occur, whereas initial volumes of 60 ml require 3 hours to reach a similar O_2 tension. This figure also demonstrates that when the progressive changes in the O_2 tension of bladder saline are plotted semilogarithmically, a nearly straight line is produced that describes the rate at which O_2 is removed from the saline. Table I also lists the O_2 tensions of arterial blood and venous blood draining the bladder during the experimental procedure. The O_2 tensions of arterial blood were so maintained that the arterial O_2 saturation was usually above 90%. The average O_2 tension of venous blood remained between 53 and 57 mm Hg, and 3 hours after the start of the experiment was 57 mm Hg (SE, ± 1.5). This value exceeded the average O_2 tension of saline in the bladder taken at a corresponding time by 9 mm Hg.

The changes in CO_2 tension are also shown in Table I. Initially the CO_2 tension of the saline was 0, and during the course of the experiments it rose to values exceeding those of both arterial and venous blood. At 180 minutes, the average CO_2 tension of saline reached 35 mm Hg and thereby exceeded the average CO_2 tension of bladder venous blood by 3 mm Hg. The average pH of both arterial and venous blood remained within a narrow range throughout these studies; arterial blood fluctuated between 7.32 and 7.36, and venous blood between 7.30 and 7.33.

Measurements of blood flow to the bladder were not directly made during these experiments, but were inferred from the arteriovenous differences in O_2 concentration. The average values ranged from 1.10 to 1.92 ml per 100 ml during the course of the experiments, but 5 minutes after the instil-

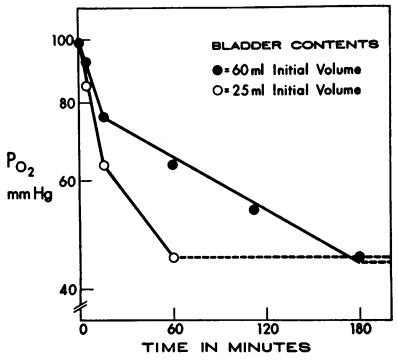

FIG. 2. MANNER IN WHICH THE INITIAL VOLUME OF SALINE INSTILLED IN THE URINARY BLADDER AFFECTS THE RATE AT WHICH THE O_2 TENSION (P_{O_2}) OF THE SALINE STABILIZES. The ordinate is scaled logarithmically. The curve describing the course of 60-ml initial volumes represents the average of 11 animals, and the curve for the 25-ml initial volume represents the average of 5 animals. The dashed lines are continuations of each curve after the stable O_2 tensions were achieved.

lation of saline (1.59 ml per 100 ml), they were, on the average, very similar to those at the conclusion of the experiment (1.52 ml per 100 ml).

The effect of changes in the volume of liquid contained in the bladder on the blood flow to the bladder is indicated by arteriovenous differences in O_2 concentrations, which are listed in Table II. The arteriovenous O_2 differences averaged 1.5 ml per 100 ml in the full bladder and 1.4 ml per 100 ml in the empty bladder. In no animal did the arteriovenous differences change by more than 10% (0.2 ml per 100 ml) during the experiment.

The effects of breathing 100% O_2 on the gaseous composition of the saline in the bladder and of the blood perfusing the bladder appear in Table III and are illustrated by Figure 3. The control values in this table are the same as those obtained 180 minutes after the instillation of saline; these were previously listed in Table I. Ten minutes after 100% O_2 was substituted for room air, the average arterial O_2 tension rose from 76 to 380 mm Hg. The O_2 tension of venous blood also rose on the average from 57 to 111 mm Hg and did not vary by more than 7 mm Hg during the remainder of the experiments. By contrast, the O_2 tension of the fluid in the bladder rose after 10 and 40 minutes by only 6 or 7 mm Hg; moreover, after 150 minutes it had again fallen to 50 mm Hg, a value which exceeded the control average by only 2 mm Hg.

The arteriovenous difference in O_2 concentration was also used in this portion of the experiment as an indicator of blood flow to the bladder. As listed in Table III, the initial control value averaged 1.58 ml per 100 ml, and the final value taken 150 minutes later was 1.67 ml per 100 ml, a change of only 7%. The maximal change in arteriovenous O_2 difference occurred after 10 minutes of breathing 100% O_2; even at this point, however, the decrease was only 24%. During this procedure, the CO_2 tension of both blood and saline in the bladder rose, on the average, by 4 mm Hg, while the average pH fell by 0.04 U.

TABLE II

*The effect of distension of the urinary bladder on the difference in oxygen concentrations between arterial and venous blood**

Dog no.	State	Arterial P_{O_2}	Venous P_{O_2}	$Ca_{O_2} - C\bar{v}_{O_2}$
		mm Hg	*mm Hg*	*ml/100 ml*
1	Full	68	60	1.3
	Empty	68	58	1.2
6	Full	64	50	2.1
	Empty	74	55	1.9
9†	Full	550	148	1.2
	Empty	550	170	1.1

* Abbreviations as in Table I.
† The bladder was emptied and filled while this animal was breathing pure O_2.

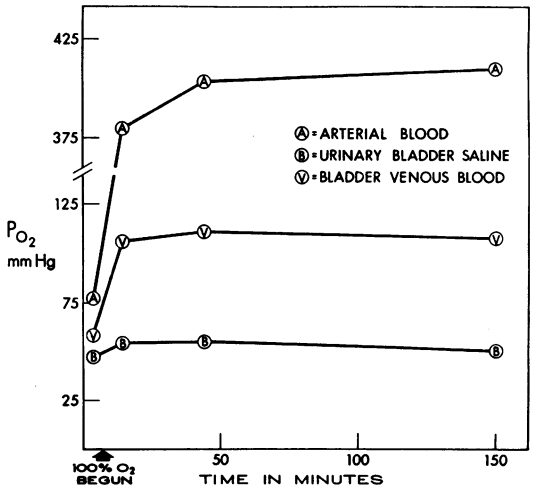

FIG. 3. EFFECT OF BREATHING PURE O_2 ON THE O_2 TENSIONS (P_{O_2}) OF ARTERIAL BLOOD, BLADDER VENOUS BLOOD, AND SALINE IN THE URINARY BLADDER. The points represents average values from the same 11 dogs illustrated in Figure 1. The arrow marks the point at which the breathing of 100% O_2 was begun.

TABLE III

*The effect of breathing pure O_2 on the gaseous composition of bladder tissues and blood in 11 dogs**

	Arterial			Bladder venous				Bladder contents	
	P_{O_2}	P_{CO_2}	pH	P_{O_2}	P_{CO_2}	pH	$Ca_{O_2}-C\bar{v}_{O_2}$	P_{O_2}	P_{CO_2}
	mm Hg	*mm Hg*		*mm Hg*	*mm Hg*		*ml/100 ml*	*mm Hg*	*mm Hg*
					Control				
Av.	76	31	7.36	57	32	7.31	1.58	48	35
SD	8.5	7.7	0.10	4.9	8.4	0.10	0.76	8.3	8.2
SE	2.5	2.3	0.03	1.5	2.5	0.03	0.23	2.4	2.5
					Breathing pure O_2, 10 minutes				
Av.	380	35	7.33	111	36	7.28	2.08	54	33
SD	62.2	10.5	0.05	65.6	11.3	0.05	1.13	15.6	7.9
SE	19.6	3.3	0.02	20.7	3.5	0.02	0.35	5.0	2.5
					Breathing pure O_2, 40 minutes				
Av.	410	36	7.32	118	37	7.28	1.81	55	35
SD	43.2	12.5	0.05	56.4	15.7	0.06	1.16	20.2	9.0
SE	13.6	3.9	0.02	17.8	4.9	0.02	0.37	6.4	2.8
					Breathing pure O_2, 150 minutes				
Av.	417	35	7.33	113	36	7.28	1.67	50	37
SD	29.2	11.6	0.06	56.3	13.0	0.06	1.08	19.7	8.6
SE	8.4	3.5	0.02	17.8	4.1	0.02	0.34	5.9	2.6

* Abbreviations as in Table I.

Finally, the serial measurements of the O_2 tension of saline removed from the urinary bladder at the end of the experiments and sealed anaerobically in a syringe were made at 30-minute intervals. Since these measurements demonstrated no decline in the O_2 tension of the saline ($\pm$ 3 mm Hg), little or no O_2 consumption or O_2 disappearance could be attributed to constituents which entered the saline during its residence in the bladder.

Gall bladder. The relation between the O_2 tension of saline in the urinary bladder and in the gall bladder is shown in Table IV. In each case, the listed values are from samples of saline taken from the urinary bladder and gall bladder at identical times, i.e., 150 minutes after the saline was first instilled in the organs. Moreover, the listed O_2 tensions were not different from values in samples taken at even later times. As indicated, in two animals, the values for the urinary bladder and gall bladder were identical, and, in two other animals, the O_2 tension of the gall bladder was 13 and 25 mm Hg lower than that of the urinary bladder.

Discussion

The present study utilized a liquid in a hollow viscus as an equilibrating system to measure the gas tensions in the tissues (i.e., the extracellular tissue spaces) immediately surrounding the liquid. We found that the liquid took on the O_2 and CO_2 tensions which were to be expected in tissues more swiftly than the equilibration time for subcutaneous gas pockets (5) and that the rapidity with which equilibration occurred was inversely proportional to the volume of liquid instilled in the bladder. Thus, large volumes of 60 ml achieved stable gas tensions in 3 hours, whereas a small volume of 25 ml required less than an hour for equilibration. By this technic, we have a convenient system whereby the gas tensions of some tissues can be obtained under a variety of physiological circumstances.

TABLE IV

*The relation between the P_{O_2} of saline in the urinary bladder and gall bladder**

Dog no.	Arterial blood	Urinary bladder contents	Gall bladder contents
	mm Hg	*mm Hg*	*mm Hg*
17	81	49	49
18	87	57	45
19	76	47	47
20	80	51	44
Mean	81	51	43

* Abbreviations as in Table I.

Relation between gas tension of tissue and blood. This system, utilizing the urinary bladder in particular, has also made it possible to compare the gaseous composition of tissues with those of the corresponding venous blood draining the tissues. In this respect, this method has an advantage over other technics of measuring tissue gas tensions such as those involving lymph or the subcutaneous gas pocket. In the present study, a comparison of these two values indicates that the O_2 tension of venous blood exceeds that of the tissues, on the average, by 9 mm Hg. This tendency of tissue O_2 tension to fall lower than the value of draining venous blood has been observed previously (6, 7). On the other hand, the O_2 tensions attributable to bladder tissues are not so low as those recorded from thoracic duct lymph (6). The reason for this difference is unclear; however, the present method measures only the values in two hollow viscera, whereas thoracic duct lymph measures the O_2 tensions of hollow viscera, liver, and muscle together.

This small O_2 pressure gradient between tissues and draining venous blood in the present study may have several possible explanations: *1*) The first explanation is based on the principle that the average O_2 tension of a unit of tissue does, indeed, fall lower than that of the blood in the venous end of the capillary serving this unit of tissue. The principle rests on calculations performed by Krogh (12) using measured values for the rate of diffusion of O_2 through excised tissues and using a model that features a capillary surrounded by a cylinder of tissue; in this model, the gradient between the venous blood O_2 tension and the average tissue O_2 tension is due to the preponderant contribution to the whole made by O_2 tensions of tissues farthest from the capillary; these distant tissues have the lowest O_2 tensions and yet comprise the largest bulk of the cylinder of tissue. *2*) The second explanation is based on the idea that the rate of diffusion of O_2 through tissues is greater than Krogh thought (13, 14) and that the average tissue O_2 tension is the same as that of blood at the venous end of the capillary; this explanation implies that some arterial blood is shunted through vessels across which gas exchange is limited. The mixture of capillary venous blood in gaseous equilibrium with tissues and arterial blood little modified by its passage through tissues would be expected to produce O_2 tensions in the mixed venous blood draining the organ which are higher than the average tissue O_2 tensions. *3*) The third possibility implies that the liquid in the bladder equilibrates mainly with epithelial cells of the mucosal surface whose O_2 tension may be lower than that of smooth muscle, whereas the blood in the veins reflects gas tensions from the entire thickness of the bladder wall. This possibility cannot be entirely excluded, even though the bladder in many experiments was so distended as to be transparent and even though its thickness did not appear to exceed 1 mm.

Regulation of tissue oxygen tension. In the present study the breathing of an enriched O_2 mixture resulted in the expected rise in the O_2 tension of arterial blood without markedly or consistently affecting the O_2 tension of the saline in the bladder. These observations are in contrast to other studies of tissue O_2 tensions during the breathing of enriched O_2 mixtures: in procedures involving measurements in skin and subcutaneous tissues by implanted electrodes or subcutaneous gas pockets (15–19), increases in tissue O_2 tension of 10 to 50 mm Hg have been recorded. Although the accuracy of such procedures may be hampered by difficulties in calibrating electrodes or the pathological changes in the subcutaneous pocket, these different observations during O_2 breathing may possibly be attributed to the different types of tissues being studied. Even the increases in tissue O_2 tensions observed in some of these studies are relatively small, however, when compared with the large increases in O_2 tension that are assumed to have occurred in the arterial blood.

The tendency to stabilize the tissue O_2 tension during changes in the inspired O_2 concentration has been previously noted for the brain; in this case, stabilization of the tissue O_2 tension appears to be accomplished by decreasing the total blood flow to the brain (20). In the present study, however, the breathing of pure O_2 did not affect the difference in the O_2 concentration between arterial blood and the venous blood draining the urinary bladder. Accordingly, it is not possible to attribute the constant tissue O_2 tension during the breathing of pure O_2 to decreases in the total blood flow to the bladder.

The most likely mechanism to explain the pres-

ent observations involves the shunting of arterial blood away from capillaries where gas exchange can occur to vessels where gas exchange is limited. The partition of urinary bladder flow between the gas exchanging capillaries and the vessels where gas exchange is limited may be estimated by a mixing formula analogous to that used for pulmonary blood flow (21). As in the lung, the mixing formulas applied to the systemic circulation will mathematically render blood flow to poorly exchanging vessels as though it were a smaller blood flow to nongas-exchanging vessels. In this formula, the amount of O_2 appearing in the venous blood of the bladder ($\dot{Q}v_{O_2}$) is equal to the amount of O_2 appearing in the blood at the venous end of the capillary ($\dot{Q}c_{O_2}$) plus the amount of O_2 in shunted blood (Qs_{O_2}):

$$Qv_{O_2} = Qc_{O_2} + Qs_{O_2},$$

or

$$Cv_{O_2} \times Qt = Ca_{O_2} \times Qs + Cc_{O_2} \times (Qt - Qs),$$

where Cv_{O_2}, Ca_{O_2}, and Cc_{O_2} are the concentrations of O_2 in venous, arterial, and end-capillary blood, respectively, and $\dot{Q}t$ and Qs are total and shunted blood flow, respectively.

In the present circumstance, the O_2 tensions of end-capillary blood, the bladder tissues, and the saline in the bladder are considered equal. When this relationship is applied to the average data obtained during the breathing of pure O_2, it is estimated that under this circumstance 76% of the blood flow to the bladder does not enter gas exchanging capillaries. By contrast, application of this relationship to the data obtained during ambient air breathing indicates that only 10% of blood flow to the bladder is shunted away from gas exchanging capillaries.

The anatomic basis in the urinary bladder for the shunting of blood away from the capillary bed into vessels that exchange substances poorly is uncertain. In fact, microscopic studies of the capillaries and other small vessels of the urinary bladder are not available in large systematic texts of anatomy (22) or even in specialized studies (23). On the other hand, a special anatomic shunt need not be required to fulfill the role of the physiologic shunts observed in the present study. A functional type of shunt is possible, in which the diversion of blood flow occurs from capillaries of small caliber to vessels of larger caliber. Such functional shunts have been described in the omentum (24, 25), where the transparent structure makes possible microscopic *in vivo* study which is not readily feasible in the wall of the urinary bladder.

The mechanism whereby abnormally high O_2 tensions regulated the partition of blood flow through the capillary bed of the bladder is also uncertain. The data of the present study do not suggest whether this mechanism is neurogenic and reflex in nature or whether it is a direct effect of O_2 on the smooth muscle of the small blood vessel. Regardless of mechanism, the present data indicate that at least in one organ, the urinary bladder, the tissue O_2 tension is prevented from rising during the breathing of high O_2 mixtures by selective diversions of blood from the capillary bed. These data also imply that during the process the exchange of other substances between tissues and capillaries may be impaired. Unfortunately, the large capacity of the saline in the bladder in the present method did not allow the accumulation of tissue CO_2 to be measured accurately as an indicator of impaired exchange across the capillary bed during these decreases in capillary blood flow.

Finally, the site from which changes in O_2 tension may act to effect changes in the distribution of blood flow is as uncertain as the over-all distribution of the various O_2 tensions within a tissue; the present method, like others, measures the integral of what may be many values for tissue O_2 tension in a single organ.

Summary

1) The O_2 and CO_2 tensions of tissues were estimated by sampling liquids that were instilled in hollow viscera and allowed to remain until they reached gaseous equilibrium with the surrounding tissue. This method proved to be a convenient *in vivo* system with which to study the gas tensions of tissues under various circumstances.

2) It was demonstrated that, under normal conditions, the O_2 tension of the tissues of the urinary bladder is lower than that of its venous blood and that, during the breathing of enriched O_2 mixtures, the difference in O_2 tension between tissues and venous blood becomes even greater.

These differences are attributed to the diversion of blood flow from capillaries to vessels where gas exchange is limited.

3) The inordinate diversion of blood from the capillary bed of the bladder during the breathing of an enriched O_2 mixture tended to prevent increases in the O_2 tension of the tissues. Implicit in this mechanism which curtails capillary blood flow, however, is the likelihood of impairing the exchange of other substances between blood and tissues.

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