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Central Venous Oxygen Saturation in Shock:

A Study in Man

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In two groups of patients, oxygen saturation was determined in blood drawn simultaneously from several locations: superior vena cava ($S_{SVC}O_2$) (central venous blood); right atrium ($S_{RA}O_2$); right ventricle ($S_{RV}O_2$); pulmonary artery ($S\bar{v}O_2$); systemic artery ($S_{ART}O_2$); and inferior vena cava ($S_{IVC}O_2$). Values in 29 patients with cardiac indexes of 3.34 ± 0.18 were compared with comparable values in 15 patients in shock (hemorrhagic, late septic, and neurogenic) who had cardiac indexes of 1.71 ± 0.05. In the patients with shock, the mean $S_{SVC}O_2$ was significantly greater than the mean $S\bar{v}O_2$ ($P < 0.001$) and the correlation coefficient was $r = +0.73$. However, there was better correlation between $S_{RA}O_2$ and $S_{RV}O_2$ and $S\bar{v}O_2$ ($r = +0.95$ and $r = +0.97$). The reversal of the normal relationship between $S_{SVC}O_2$ and $S\bar{v}O_2$ in the shock state is compatible with the thesis that blood flow to the splenic and renal area is decreased, thus the lower $S_{IVC}O_2$ and $S\bar{v}O_2$. Ten patients in the shock group were restudied after their shock states had improved with therapy. The changes in $S\bar{v}O_2$ ($r = +0.81$), $S_{RV}O_2$ ($r = +0.70$), and $S_{RA}O_2$ ($r = +0.68$) are much more reliable indexes of cardiac output in the shock patient than the change in $S_{SVC}O_2$ ($r = +0.27$). (Key words: Shock; Mixed venous oxygen saturation; Central venous oxygen saturation.)

MIXED VENOUS OXYGEN SATURATION, $S\bar{v}O_2$, or pulmonary artery oxygen saturation is a useful index of the adequacy of effective systemic perfusion or tissue oxygenation because it is directly proportional to cardiac output when arterial oxygen content and tissue oxygen consumption remain constant. There has been much emphasis on the usefulness of central venous oxygen saturation, $S_{SVC}O_2$ (superior vena caval sample), in assessing hemodynamic impairment in patients with acute myocardial infarction.[1] The basic assumption is that $S_{SVC}O_2$ is an accurate reflection of $S\bar{v}O_2$. However, this was true only when the patient was not in shock or heart failure. When shock or heart failure was present with myocardial infarction, there was very poor correlation between values of $S_{SVC}O_2$ and $S\bar{v}O_2$.[2] The purpose of the clinical study was to determine: 1) whether there was any correlation between $S_{SVC}O_2$ in critically ill patients in shock and patients not in shock, all without myocardial infarction, heart failure, or arrhythmia; and 2) whether changes in $S_{SVC}O_2$ or $S\bar{v}O_2$ reflect changes in the cardiodynamic state or cardiac output as the shock state improves clinically.

Methods

Critically ill patients admitted to the Intensive Care Unit were separated into two groups. One group consisted of 29 patients having no clinical and hemodynamic evidence of shock (12 were being treated for barbiturate overdosage, eight for flail chest, four for cerebral concussion, five for postoperative respiratory failure) and the other group included 15 patients in shock with mean arterial pressures of less than 55 mm Hg and urinary output less than 10 ml per hour for two consecutive hours. Seven of these patients had late septic shock, six had hemorrhagic shock, and two had neurogenic shock. All shock patients were without

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myocardial infarction, heart failure or arrhythmia. None of the seven patients in late septic shock had body temperatures higher than 38.6 C or laboratory evidence of hyperdynamia when first seen. All postoperative respiratory failure patients had severe obstructive airway disease for which they had undergone lobectomy or pneumonectomy. Severe trauma to the head was the cause of neurogenic shock in two patients. In every subject, a 36-inch Portex epidural catheter (Portex Ltd., Hyth-Kent, England) with a square tip orifice was inserted percutaneously into the right basilic vein and advanced into the right atrium, right ventricle, and pulmonary artery while pressure and the electrocardiogram were monitored. The catheter was connected to a 500-ml saline-solution bottle containing 1,000 units of Na–heparin for irrigating the catheter and for keeping the catheter patent through a continuous microdrip for 24–72 hours. In addition, a large radiopaque Bardic intravenous catheter 24 inches long was inserted percutaneously or via a cutdown into the left median cubital vein, and the tip was advanced to the superior vena cava. The accurate positioning of this was confirmed by roentgenography. A polyethylene catheter was placed in the femoral artery using the Seldinger technique for sampling of arterial blood and the catheter was filled with heparinized saline solution between studies. In five patients in shock and nine patients not in shock, another radiopaque Bardic catheter was placed in the inferior vena cava via a cutdown in the femoral vein and the position of the tip of the catheter at the level of the diaphragm was confirmed by roentgenography. This catheter was kept open by continuous intravenous infusion of fluid. All patients in both groups were ventilated on assisted–controlled ventilation with the Bennett MA-1 respirator through an endotracheal tube or a tracheostomy tube. A tidal volume of 12 ml/kg body weight was used and deadspace was added to maintain a Pa_{CO_2} of 35–42 mm Hg. A Pa_{O_2} of 80 mm Hg or higher was maintained with inspired oxygen concentrations of 20–60 per cent except in one patient in shock who needed 100 per cent inspired oxygen for a satisfactory Pa_{O_2}. Body temperature was monitored with an esophageal probe and a telethermometer (Yellow Springs Instrumentation Co., Yellow Springs, Ohio).

Systemic and pulmonary arterial pressures were recorded continuously with Statham P23G transducers attached to a Model 5 Polygraph (Grass Instrument Co., Quincy, Mass.). While the Portex catheter was being advanced, 2-ml blood samples were taken from superior vena cava (SVC), right atrium (RA), right ventricle (RV), and pulmonary artery ($\bar{v}$); similar samples were taken from the systemic artery (ART) and inferior vena cava (IVC) into 2-ml heparinized glass syringes for P_{O_2}, P_{CO_2}, *p*H, and oxygen saturation determinations in duplicate samples. Blood gas tensions and *p*H were determined with Radiometer O_2, CO_2, and *p*H electrodes at 37 C and the blood values were corrected to body temperature. Base excess or deficit was determined using the Siggaard-Andersen alignment nomogram, and appropriate amounts of sodium bicarbonate were given intravenously to patients in shock so that the base deficit was no greater than 4 mEq/l before each study. Oxygen saturation was measured by the American Optical micro-oximeter immediately after withdrawal of samples from different sites. The micro-oximeter was calibrated to an accuracy of ±0.5 per cent in the 45–80 per cent oxygen saturation range against that determined by the Van Slyke method. Duplicate samples of saturation determination showed an average difference of 0.5 per cent. Samples of arterial and venous blood were taken prior to the determination of cardiac output. Cardiac output was computed from Cardio-Green (indocyanine green) dilution curves, after injection into the pulmonary artery, and also by the direct Fick method in five patients in shock and eight patients not in shock whose arterial P_{O_2}'s were greater than 80 mm Hg during ventilation with air (inspired O_2 20.9 per cent). The difference between the two methods was minimal and not significant statistically. Four of the 15 patients in shock (two late septic, one hemorrhagic, and one neurogenic) were studied twice in the 6–12-hour periods while they were in shock. Eight patients of the "non-shock" group (five barbiturate overdosage and three flail chest) were studied twice during a 24–48-hour period. Ten patients of the shock group (five

TABLE 1. Oxygen Saturation and Cardiodynamic Data—Patients in Shock

Age (Years)	Type of Shock	O_2 Saturation (Per Cent)						Pulmonary Artery Pressure		Q(l/min)	Cardiac Index
		Central Venous	Right Atrium	Right Ventricle	Pulmonary Artery	Systemic Artery	Interior Vena Cava	mm Hg	Mean		
53	Septic	59.0	51.5	53.0	51.5	96.5	44.7	26/10	15	2.9 2.7(F*)	1.58
61	Septic	68.7	58.0	63.0	60.0	96.0	53.9	28/16	21	3.9	1.90
		68.0	—	56.0	56.5	93.5		30/17	22	3.3	1.62
45	Hemorrhagic	68.5	61.0	60.5	60.0	96.8		27/12	19	3.9 3.8(F)	1.36
63	Neurogenic	78.8	69.0	67.5	68.0	99.4		26/10	17	3.1	1.62
		72.3	—	62.0	63.5	97.8		25/12	16	3.9	2.04
35	Hemorrhagic	71.2	63.5	62.0	61.2	100.0		34/18	24	3.5	1.82
33	Hemorrhagic	71.0	62.5	60.5	60.5	98.9	52.4	18/9	14	3.3	1.65
31	Hemorrhagic	67.0	56.0	57.5	54.5	97.2	43.7	25/14	18	3.3	1.58
78	Septic	58.5	52.0	51.0	50.0	99.5		55/20	38	2.7	1.51
57	Septic	60.0	—	52.0	48.0	100.0		38/15	24	2.9 3.2(F)	1.61
60	Hemorrhagic	63.2	53.5	56.0	54.5	99.6	48.9	26/10	17	3.2	1.65
59	Hemorrhagic	59.5	53.0	50.5	51.0	100.0		18/8	12	2.7	1.43
		62.5	—	46.0	44.5	100.0		20/8	12	2.6	1.35
51	Neurogenic	65.5	59.0	56.5	53.0	98.7		16/8	12	3.7	1.95
57	Septic	63.3	57.0	56.5	58.5	99.4		26/15	19	3.3 2.9(F)	1.89
67	Septic	69.0	60.3	58.0	56.0	100.0		38/14	23	4.0 3.9(F)	1.93
		67.8	—	61.4	59.5	97.7		35/10	20	3.7 4.0(F)	1.71
39	Septic	61.5	—	54.5	53.0	96.8		35/17	24	3.0 2.8(F)	1.76

* F = determined by the Fick principle.

late septic, four hemorrhagic, one neurogenic) were restudied after therapy and clinical improvement with increased mean arterial blood pressures and urinary output.

Results

The data and statistical analysis for the two groups of patients are shown in tables 1, 2, and 3. The 29 patients in the "non-shock" group had a mean cardiac index of 3.34 ± 0.18 and a mean esophageal temperature of 37.1 ± 1.2 C, and each patient had a mean arterial blood pressure of more than 72 mm Hg. Fifteen patients in the shock group had a mean cardiac index of 1.71 ± 0.05 and a mean esophageal temperature of 37.0 ± 1.4 C, and each had a mean arterial blood pressure of less than 55 mm Hg.

The relationship between all values of $Ssvc_{O_2}$ and simultaneously-determined $S\bar{v}_{O_2}$ in the two groups of patients is illustrated in figure 1. Mean $Ssvc_{O_2}$ of patients in shock was significantly greater than mean $S\bar{v}_{O_2}$ ($P < 0.001$), whereas mean $Ssvc_{O_2}$ of patients not in shock was significantly less than mean $S\bar{v}_{O_2}$ ($P < 0.01$). The correlation between each simultaneously-determined $Ssvc_{O_2}$ and $S\bar{v}_{O_2}$ amounted to $r = +0.73$. There was no significant difference between the mean $S_{RA}{}_{O_2}$ and the mean $S\bar{v}_{O_2}$, or the mean $S\bar{v}_{O_2}$'s in the two groups. Furthermore, there was good correlation between $S_{RA}{}_{O_2}$, $S_{RV}{}_{O_2}$, and $S\bar{v}_{O_2}$, with correlation coefficients of +0.95 and +0.97, respectively, in shock patients and +0.96 and 0.98, respectively, in patients not in shock. The $Ssvc_{O_2}$ was greater than the $Sivc_{O_2}$ in every shock patient, whereas the $Ssvc_{O_2}$ was less than the $Sivc_{O_2}$ in every "non-shock" patient, as illustrated in figure 2.

Results in the ten shock patients restudied as their shock states improved with therapy are shown in table 4. The mean changes in $Ssvc_{O_2}$ and $S\bar{v}_{O_2}$ determined simultaneously and the corresponding mean increase of cardiac output in l/min are shown in figure 3. The mean increase in cardiac index was $1.17 \pm$

TABLE 2. Oxygen Saturation and Cardiodynamic Data—Patients Not in Shock

Age (Years)	O_2 Saturation (Per Cent)						PA Pressure		Q̇(l/min)	Cardiac Index
	Central Venous	Right Atrium	Right Ventricle	Pulmonary Artery	Systemic Artery	Inferior Vena Cava	mm Hg	Mean		
29	68.0	72.0	72.5	71.5	99.7		18/8	14	5.6	3.06
	66.0	68.5	68.0		98.9	68.0	18/9	14	4.9 4.7(F*)	2.62
55	69.5	75.0	75.5	73.0	97.7		25/12	17	6.8	3.82
43	73.0	—	76.0	76.7	99.6		18/8	12	5.8	3.43
	68.5	—	72.0	71.0	97.9	69.5	18/8	12	5.2	3.09
29	62.0	68.0	67.5	66.5	96.7	68.0	25/14	18	7.2	3.87
39	70.0	76.5	76.0	74.5	98.4		30/16	21	5.9	3.55
43	71.0	74.5	76.0	74.5	100.0		20/9	14	5.2 5.0(F)	3.04
31	71.8	—	75.0	74.0	98.5	76.5	25/10	16	5.7 5.4(F)	3.26
63	67.0	70.5	70.6	69.5	94.9		25/10	15	5.9 6.1(F)	3.40
	72.0	—	74.5	75.5	97.1		28/12	18	6.7	3.79
43	70.0	74.0	73.4	72.4	100.0		30/16	20	8.2	4.34
	72.5	—	75.0	76.5	98.7		27/14	19	6.7 6.2(F)	3.42
43	62.0	—	65.5	64.5	97.9		28/15	19	7.3	3.94
	64.5	68.5	68.5	69.0	99.1		25/12	16	6.8	3.68
61	64.5	68.5	69.0	68.0	97.7	69.0	28/15	20	6.1	3.44
27	70.0	75.5	75.0	74.0	100.0		18/10	13	5.9	3.27
	70.0	—	72.5	73.0	98.6		18/8	12	5.3	2.95
49	68.5	73.5	73.0	72.0	97.9		26/10	15	5.7	3.32
47	69.0	—	74.0	72.5	98.4		19/10	13	5.9	3.37
63	69.5	73.0	72.0	72.5	98.2	73.5	25/12	18	6.8	3.58
35	65.5	—	69.5	68.0	100.0		22/10	15	6.1	3.37
33	67.8	73.0	73.5	72.5	98.2		24/10	14	6.4	3.45
15	70.5	74.5	73.5	74.0	100.0	76.5	18/8	12	6.9	3.64
	72.0	74.5	75.0	75.5	99.6		20/8	13	5.2	2.74
31	72.5	—	76.0	76.5	98.0		24/12	16	6.9	3.65
28	71.0	74.5	75.5	76.0	100.0	76.5	18/8	12	7.0	4.00
	70.5	75.0	75.0	—	99.1		20/8	13	5.8	3.32
78	60.5	—	64.0	63.5	96.9		26/14	19	4.5	2.70
29	72.0	76.5	75.5	76.0	100.0		19/10	13	5.9	3.11
57	66.5	71.5	70.5	70.0	99.7		38/15	24	5.8	3.26
66	66.5	69.5	70.0	70.5	98.8	72.0	22/10	14	6.1	3.51
51	68.3	—	71.5	72.5	99.5		18/10	13	5.9	3.39
41	68.0	73.5	72.0	72.5	99.0		18/8	12	6.7	3.68
47	65.0	—	71.5	71.5	98.2		20/9	13	5.7 5.2(F)	3.22
58	65.0	71.5	—	70.0	100.0		24/10	14	6.5 6.9(F)	3.62
67	67.3	—	73.0	71.5	100.0		26/12	16	5.9 5.8(F)	3.45

* F = determined by the Fick principle.

0.18. There was no significant change of $S_{SVC_{O_2}}$, but the increases of $S_{RA_{O_2}}$, $S_{R\bar{V}_{O_2}}$, and Q̇ (cardiac output) were significant at $P < 0.001$. In every patient there was excellent correlation between increase of $S_{\bar{V}_{O_2}}$ and increase of Q̇ ($r = +0.81$) as the shock state improved. On the other hand, the correlation between the change of $S_{SVC_{O_2}}$ and the increase of Q̇ was very poor ($r = +0.27$). The correlations between increases of $S_{RA_{O_2}}$ and $S_{RV_{O_2}}$ and increase of Q̇ were +0.68 and +0.70, respectively.

TABLE 3. Statistical Analyses of Data

Simultaneous Measurements	Number of Measurements	Per Cent Oxygen Saturation (Mean ± SD)	r	P Value for Comparison of Mean
Group I				
Central venous (SVC)—$\bar{v}$	35	SVC 68.3±3.2 $\bar{v}$ 72.1±3.2	+0.88	<0.01
Right atrium—right ventricle	23	RA 72.7±2.6 RV 72.2±2.5	+0.96	<0.5
Right atrium—$\bar{v}$	23	RA 72.7±2.6 $\bar{v}$ 71.9±3.0	+0.96	<0.5
Right ventricle—$\bar{v}$	35	RV 72.4±2.8 $\bar{v}$ 72.1±3.2	+0.98	<0.5
Group II				
Central venous (SVC)—$\bar{v}$	19	SVC 66.1±5.3 $\bar{v}$ 56.0±5.6	+0.73	<0.001
Right atrium—right ventricle	13	RA 58.2±5.2 RV 57.4±4.7	+0.96	<0.5
Right atrium—$\bar{v}$	13	RA 58.2±5.2 $\bar{v}$ 56.7±4.9	±0.95	<0.5
Right ventricle—$\bar{v}$	19	RV 57.1±5.1 $\bar{v}$ 56.0±5.6	+0.97	<0.5

Discussion

Goldman and co-workers described the clinical usefulness of measurements of $Ssvc_{O_2}$ in monitoring patients with myocardial infarction in the coronary care unit.[1] They found that patients with $Ssvc_{O_2}$ values of 60 per cent or less showed evidence of either heart failure or shock or a combination of the two. This find-

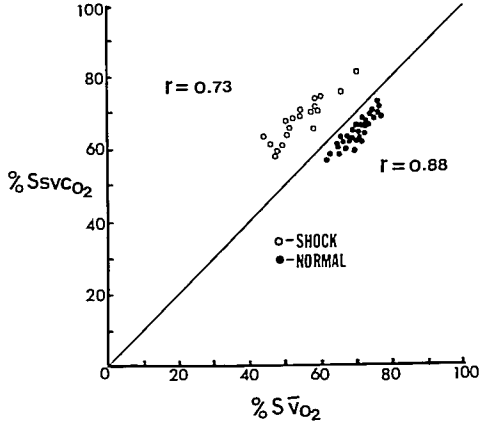

FIG. 1. Relationship between $Ssvc_{O_2}$ and Sv_{O_2} in the two groups of patients (shock and "non-shock").

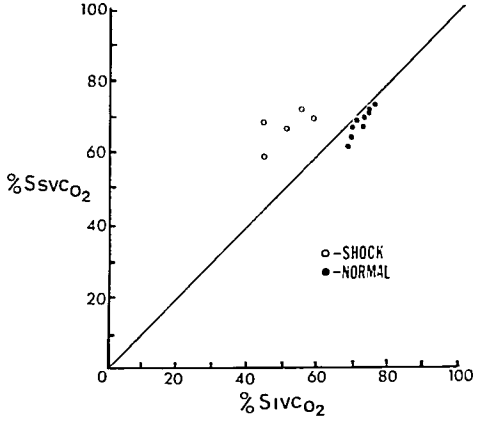

FIG. 2. $Ssvc_{O_2}$ and $Sivc_{O_2}$ in shock patients and "non-shock" patients.

ing is not too surprising, since decreased cardiac output would be expected to result in greater extraction of oxygen by peripheral tissue and therefore, an abnormally low oxygen saturation in the venous blood returning to the heart. The assumption that $Ssvc_{O_2}$ is an accurate reflection of $S\bar{v}_{O_2}$ was true only when the patient was not in cardiogenic shock or heart failure. Scheinman has shown that there was poor correlation between $Ssvc_{O_2}$ and $S\bar{v}_{O_2}$ when shock and heart failure were present in patients with acute myocardial infarction.[2] Our findings substantiate those of Scheinman, and indicate that $Ssvc_{O_2}$ proved to be an accurate reflection of $S\bar{v}_{O_2}$ in seriously ill patients without shock, arrhythmia, or acute myocardial infarction. $Ssvc_{O_2}$ was slightly lower than $S\bar{v}_{O_2}$ in 29 patients not in shock,

TABLE 4. Changes in Oxygen Saturation and Cardiac Output—Shock Group

Age (Years)	Diagnosis	Δ Per Cent Oxygen Saturation				Δ*Q̇ (l/min)
		Central Venous	Right Atrium	Right Ventricle	Pulmonary Artery	
53	Septic	+2.5	+10.5	+11.7	+12.5	+1.0
45	Hemorrhagic	−0.5	+ 8.5	+10.8	+ 9.5	+0.8
31	Hemorrhagic	+0.5	+12.5	+13.5	+14.5	+1.4
57	Septic	+1.5	+17.0	+17.0	+18.5	+1.8
60	Hemorrhagic	+2.5	+12.5	+11.5	+14.5	+2.1
59	Hemorrhagic	+3.5	+17.5	+18.0	+19.5	+2.2
51	Neurogenic	−1.0		+15.0	+15.5	+1.3
57	Septic	+2.2	+14.5	+15.5	+16.5	+2.2
39	Septic	+2.5		+16.0	+15.5	+2.2
67	Septic	−1.0	+15.5	+16.5	+18.5	+2.3
MEAN ± SD		+1.3 ± 0.8	+13.6 ± 2.9	+14.6 ± 2.4	+15.5 ± 2.8	+1.7 ± 0.5

* Δ = change.

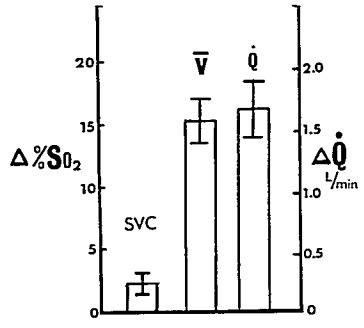

FIG. 3. Mean changes in $S_{SVC_{O_2}}$, $S\bar{v}_{O_2}$, and cardiac output in ten shock patients restudied as their shock states improved.

and this confirmed the report of Barratt-Boyes and Wood.[3] This finding is due to the higher $S_{IVC_{O_2}}$ resulting from the highly-saturated renal venous effluent.[4] A reversal of the normal relationship, in that $S_{SVC_{O_2}}$ was considerably higher than $S\bar{v}_{O_2}$, was found in every patient in shock. In five shock patients, the $S_{IVC_{O_2}}$ was much lower than the $S_{SVC_{O_2}}$; this was compatible with the thesis that the low-output state is attended by redistribution of blood flow away from the splenic, renal, and mesenteric bed toward the cerebral and coronary circulation. Forsyth demonstrated that the distribution of cardiac output to brain, heart, and adrenal gland was progressively increased at the expenses of the skin, spleen, and pancreas in the unanesthetized monkey during 50 per cent induced bleeding.[5] There might have been some degree of myocardial insufficiency in patients of the shock group, but our data did not show extreme low $S\bar{v}_{O_2}$ values such as those reported by Scheinman[2] since his patients in cardiogenic shock were actually sicker. All patients referred to the Intensive Care Unit with septic shock in our study were in the period of low cardiac output, maximum hypotension, and metabolic acidosis, in spite of compensatory peripheral vasoconstriction. These patients presumably progressed from the initial stage of increased cardiac output and peripheral dilatation to the stage of low cardiac output.

The use of $S_{SVC_{O_2}}$ as a guide to change in cardiac output must be interpreted cautiously, especially in critically ill patients in shock. Our data show that changes in $S_{RA_{O_2}}$, $S_{RV_{O_2}}$, and $S\bar{v}_{O_2}$ are much more reliable indexes than change in $S_{SVC_{O_2}}$ when the cardiodynamic state improves with clinical therapy in shock patients. Sufficient mixing usually takes place in the right ventricle and right atrium so that $S_{RA_{O_2}}$ or $S_{RV_{O_2}}$ provides an excellent reflection of $S\bar{v}_{O_2}$ and can be used in cardiodynamic monitoring of critically ill patients. Repeated measurement of $S\bar{v}_{O_2}$, $S_{RV_{O_2}}$, or $S_{RA_{O_2}}$ can be important in following the cardiodynamic changes in patients in shock, providing there is no significant alteration in oxygen consumption.

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