Anesthesiology
71:355–361, 1989

Intraoperative Glucose Infusion and Blood Lactate: Endocrine and Metabolic Relationships during Abdominal Aortic Surgery

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The hypothesis that increased intraoperative blood lactate depends both on intraoperative glucose supply and inadequate tissue oxygenation occurring during surgery was tested in anesthetized patients undergoing infrarenal abdominal aortic surgery. Twenty surgical patients received either Ringer's solution or 5% glucose solution for intraoperative volume loading. Arterial blood lactate, arterial glucose, hemodynamic variables, insulin, glucagon, cortisol, epinephrine, and norepinephrine were determined preoperatively and intraoperatively. There were no significant changes in hemodynamic values, glucagon, norepinephrine, and epinephrine compared with control values in both groups. Oxygen consumption decreased only during aortic clamping. Cortisol and lactate increased significantly 10 min after aortic clamping until the end in both groups. Glucose 5% solution infusion resulted in significantly greater blood lactate accumulation and significantly greater blood glucose and insulin levels, whereas there were no changes in the patients receiving Ringer's solution. From control until aortic clamping, lactate and glucose were significantly correlated with each other in both groups; after aortic clamping until the end of the procedure, the correlation remained constant in patients in the Ringer's group, whereas no relationship could be demonstrated in those in the glucose group. The authors conclude that intraoperative glucose administration increases intraoperative blood lactate and that blood lactate accumulation depends both on glucose supply and tissue oxygen deficit. Furthermore, none of the hemodynamic metabolic and endocrine factors were reliable for assessing tissue perfusion and metabolic demands during surgery. (Key words: Anesthetics, intravenous: fentanyl. Endocrine: metabolic response to surgery. Hormones: cortisol; epinephrine; glucagon; insulin; norepinephrine. Metabolism: blood lactate; glucose; oxygen consumption.)

CURRENTLY, during surgery, decreased whole body oxygen consumption and increased blood lactate are assumed to signify increases in metabolic demands,[1] whereas increased glucose and endocrine responses (insulin, glucagon, cortisol, epinephrine, and norepinephrine increases) are assumed to reflect surgical stress.[2–5] In a context of cardiac hemodynamic monitoring and oxygen transport monitoring, blood lactate determinations provide a simple means for rapidly assessing the degree of accumulated oxygen deficit and tissue perfusion.[1,6]

Hyperglycemia-associated intracellular lactic acidosis occurs during intraoperative use of glucose[7]; recent animal studies have shown that hyperglycemia existing prior to a severe hypoxic event will enhance the ischemic damage because of lactic acid accumulation.[8] Abdominal aortic surgery is associated with lactate production.[9] This study, performed in patients undergoing abdominal aortic repair, was designed to test the hypothesis that elevated intraoperative blood lactate levels depends both on intraoperative glucose supply and inadequate tissue oxygenation due to reduced tissue perfusion following aortic clamping and associated hypothermia. Further, we intended to evaluate which factor (hemodynamic, metabolic, or endocrine) might prove reliable for assessing and monitoring tissue perfusion and metabolic demands during surgery.

Materials and Methods

PATIENTS

Twenty patients undergoing infrarenal abdominal aortic surgery were included in this study. Institutional approval was obtained, and all patients gave informed consent. All patients were ASA physical status 2 or 3 and were in good nutritional condition. Patients with diabetes, with low cardiac output syndrome, or receiving beta-adrenoreceptor blocking drugs were excluded. Before induction of anesthesia, patients were randomly assigned to one of two groups with ten patients in each group to receive either Ringer's solution or 5% glucose solution during surgery.

ANESTHESIA

After an overnight fast, all patients received hydroxyzine (100 mg) and dipotassic chlorazepate (50 mg) as oral medication 2 h before general anesthesia. In all patients anesthesia was induced with fentanyl (30 μg/kg) and was maintained with incremental doses of fentanyl iv and with 50% nitrous oxide in oxygen. Pancuronium (0.1 mg/kg initially) was administered to permit nasotracheal intubation and neuromuscular blockade (by incremental doses of 0.03 mg/kg). Ventilation of the lungs was controlled with a tidal volume of 10 ml/kg and a respiratory rate of 10 cycles/min.

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Received from Universite Lyon-Sud, Centre Hospitalier Lyon-Sud, and Centre de Medecine Nucleaire, Lyon, France. Accepted for publication April 17, 1989. Supported in part by grants from INSERM, Contract No. 486024.

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TABLE 1. Anthropomorphic Characteristics, Duration of Major Surgical Events, Volume of Fluid, and Total Glucose Supply

	Ringer's Group (n = 10)	Glucose Group (n = 10)	*P* Values
Sex distribution (M/F)	8/2	7/3	
Age (yr)	63 ± 7.1	60.2 ± 8.7	NS
Weight (kg)	66.8 ± 5	59.5 ± 4	NS
Duration of surgery (min)	225 ± 38	238 ± 51	NS
Duration of aortic cross-clamping (min)	52 ± 16	57 ± 21	NS
Volume of fluid (ml)	4,050 ± 595	4,166 ± 807	NS
Glucose (g)	0	170–230	<0.05

Hemodynamic Measurements and Glucose Administration

Before induction of anesthesia, a 7-Fr triple lumen thermodilution catheter was inserted under local anesthesia into the right pulmonary artery, and a cannula was inserted into the left radial artery. Mean arterial pressure (MAP) and pulmonary artery wedge pressure (PAWP) were directly measured *via* a transducer. Heart rate (HR), and ECG (V5) were continuously monitored. Cardiac output and core temperature (CT) were measured every 10 min. Cardiac index (CI), left ventricular stroke work index (LVSWI), and systemic vascular resistance index (SVRI) were calculated using standard formulas. During surgery to maintain hemodynamic stability throughout clamping and unclamping of the aorta, volume loading and nitroglycerin were administered[10]; the rate and volume of fluid were adjusted to maintain optimal PAWP: optimal PAWP corresponds to the best cardiac performance obtained during a rapid volume loading[11] and was determined before induction of anesthesia after control supine values were determined. No vasopressor or inotropic agents were given. In both groups, intraoperative blood loss was replaced by blood volume for volume: patients with blood cell transfusion greater than 800 ml were excluded.

Metabolic Measurements

At the time of hemodynamic measurements, oxygen delivery (D_{O_2}), oxygen consumption (V_{O_2}), and left-right shunt (Q_s/Q_t) were calculated[12] from usual parameters (p_{O_2}, p_{CO_2}, S_{O_2}, and hemoglobin), which were determined from arterial and mixed venous blood samples.

Arterial glucose was determined by the hexokinase-glucose 6 phosphate dehydrogenase method (DuPont Instruments aca) with coefficients of variation of 1.4% for tested concentrations. Arterial lactate was determined by an enzymatic method using the oxidation of lactate to pyruvate (DuPont Instruments aca) with coefficients of variation of 5.6%.

Hormonal Measurements

From arterial samples, insulin, glucagon, and cortisol were analyzed by radioimmunoassay using commercial kits (Glucagon Kit Biodata, Milana, Italy; Insulin Kit Sorin Biomedica, Saluglia, Italy) or a method previously described.[13] Coefficients of variation were 10.6% for tested concentrations for insulin, 5.8% for glucagon, and 6% for cortisol.

Norepinephrine and epinephrine were analyzed by high performance liquid chromatography (HPLC) and electrochemical detection.[14] The limits of sensitivity were 50 pg/ml: coefficients of variation were 10% for norepinephrine and 9.8% for epinephrine.

Methods

Hemodynamic and metabolic measurements were made, with the patients at rest 15 min before induction of anesthesia (control supine, CS), 10 min after tracheal intubation (TI 10 min), 30 min after surgical incision (SI 30 min), 10 min after aortic clamping (AC 10 min), 30 min after aortic clamping (AC 30 min), 10 min after aortic declamping (AD 10 min), and 15 min after the end of surgery (END 15 min). Hormonal measurements were performed at CS, SI 30 min, AC 10 min, AD 10 min, and END 15 min.

Data Evaluation

Means (±SEM) were plotted against time. The measurements during surgery were tested for significance in relation to the control resting values prior to anesthesia. In each group, comparisons with control supine values were made using a two-way analysis of variance. Where indicated, a modified *t* test was used to identify significant differences using critical values of *P* according to the method of Bonferroni.[15] A value of $P < 0.05$ was regarded as significant. The statistically significant differences between the two groups in hemodynamic, metabolic, and hormonal values were evaluated by the Mann-Whitney U-test. In each group, a stepwise multiple linear regression technique tested the relationships between blood lactate concentrations and the hemodynamic, metabolic, and hormonal values. In both groups, relationship between blood lactate and blood glucose concentrations from control until aortic clamping was compared with relationship between blood lactate and blood glucose concentrations from control until end of surgery, and was made using least-squares regression analysis. A probability of less than 0.05 was considered significant.

Results

The anthropomorphic characteristics of the patients and the durations of aortic clamping and of surgery are given in table 1. There was no significant difference be-

TABLE 2. Hemodynamic Data

Group	Control Supine	Tracheal Intubation 10 min	Skin Incision 30 min	Aortic Clamping 10 min	Aortic Clamping 30 min	Aortic Declamping 10 min	End 15 min
Heart rate (beats/min)							
Ringer's	66 ± 17	70 ± 11	93 ± 17	85 ± 9	84 ± 14	87 ± 11	85 ± 10
Glucose	70 ± 15	63 ± 14	85 ± 17	87 ± 14	85 ± 10	87 ± 10	86 ± 12
Pulmonary artery wedge pressure (mmHg)							
Ringer's	10 ± 5	10 ± 5	10 ± 6	10 ± 7	12 ± 5	14 ± 7	10 ± 6
Glucose	11 ± 5	10 ± 5	12 ± 6	13 ± 6	14 ± 4	14 ± 4	12 ± 5
Mean arterial pressure (mmHg)							
Ringer's	104 ± 17	85 ± 11	99 ± 20	94 ± 6	102 ± 12	100 ± 14	100 ± 16
Glucose	104 ± 18	87 ± 20	96 ± 23	97 ± 10	96 ± 16	103 ± 12	95 ± 8
Cardiac index ($l \cdot min^{-1} \cdot m^{-2}$)							
Ringer's	3.1 ± 0.3	3 ± 1.2	4.2 ± 0.8	3.6 ± 0.9	3.6 ± 1	4.1 ± 1.7	3.9 ± 1.2
Glucose	2.8 ± 0.9	2.6 ± 1.2	4.5 ± 2	3 ± 1	2.9 ± 1	4 ± 1.3	3.6 ± 1
Systemic vascular resistance index ($dyn \cdot s \cdot cm^{-5} \cdot m^{-2}$)							
Ringer's	2,279 ± 566	2,024 ± 741	1,678 ± 625	1,834 ± 483	2,110 ± 631	1,869 ± 578	1,904 ± 515
Glucose	2,755 ± 997	2,439 ± 957	2,094 ± 1415	2,433 ± 894	2,439 ± 724	2,081 ± 1153	2,059 ± 650
Left ventricular stroke work index ($g \cdot m \cdot m^{-2}$)							
Ringer's	56 ± 13	43 ± 8	55 ± 18	48 ± 8	54 ± 7	63 ± 14	58 ± 15
Glucose	49 ± 11	43 ± 15	58 ± 20	45 ± 9	43 ± 16	55 ± 15	45 ± 11
Left–right shunt (%)							
Ringer's	24 ± 18	29 ± 11	31 ± 7	36 ± 12	33 ± 11	34 ± 11	29 ± 14
Glucose	19 ± 8	29 ± 15	27 ± 11	29 ± 4	23 ± 5	23 ± 5	22 ± 10

N = 10 in each group. All values are mean ± SEM. There were no significant changes within the intergroup.

tween the two groups of patients. The quantity of fluid both groups received was not different. A total of 170–230 g of glucose was infused in the glucose group. Hemodynamic data are shown in table 2. There was no significant difference between the two groups. Metabolic data are shown in table 3. No changes and no significant difference between the two groups occurred in D_{O_2}.

Compared with control period, V_{O_2} decreased signifi-

TABLE 3. Metabolic Data

Group	Control Supine	Tracheal Intubation 10 min	Skin Incision 30 min	Aortic Clamping 10 min	Aortic Clamping 30 min	Aortic Declamping 10 min	End 15 min
Oxygen delivery ($ml \cdot mn^{-1} \cdot m^{-2}$)							
Ringer's	530 ± 137	484 ± 131	615 ± 99	527 ± 95	518 ± 83	632 ± 162	632 ± 172
Glucose	458 ± 135	412 ± 204	517 ± 146	446 ± 111	438 ± 135	646 ± 76	569 ± 138
Oxygen consumption ($ml \cdot min^{-1} \cdot m^{-2}$)							
Ringer's	110 ± 30	68 ± 35	76 ± 20	62 ± 25*	66 ± 16*	75 ± 17	97 ± 35
Glucose	106 ± 39	62 ± 34	70 ± 18	58 ± 25*	62 ± 11*	88 ± 38	110 ± 27
Arterial lactate (mM)							
Ringer's	0.86 ± 0.36	0.7 ± 0.36	1.1 ± 0.34	1.9 ± 0.56*	1.8 ± 0.14*	1.85 ± 0.3*	1.85 ± 0.4*
Glucose	1.24 ± 0.57	1.15 ± 0.6	1.8 ± 0.67	2.24 ± 0.8*	3.21 ± 1.1*‡	4.9 ± 0.58*§	4.15 ± 0.8*§
Arterial glucose (mM)							
Ringer's	5.9 ± 2	5.8 ± 2.2	6.2 ± 1.7	7.9 ± 1.56	7.5 ± 1.4	8.4 ± 2.8	8.6 ± 2.7
Glucose	11.7 ± 6.4‡	18.3 ± 4.8‡	26 ± 4.9*‡	28.8 ± 3.3*‡	26.2 ± 6.7*§	21.4 ± 6.2§	21 ± 6.5§
Core temperature (° C)							
Ringer's	36.3 ± 0.2	35.6 ± 0.4*	35.1 ± 0.3†	34.6 ± 0.1†	34.5 ± 0.1†	34.4 ± 0.2†	34.6 ± 0.5†
Glucose	36.2 ± 0.4	35.4 ± 0.3*	34.8 ± 0.3†	34.6 ± 0.1†	34.5 ± 0.1†	34.2 ± 0.3†	34.3 ± 0.4†

N = 10 in each group. All values are mean ± SEM.

* $P < 0.05$ and † $P < 0.01$, significant differences from control in each group: two-way analysis of variance (Bonferroni).

‡ $P < 0.05$ and § $P < 0.01$, compared with patients receiving Ringer's solution (Mann-Whitney U-test).

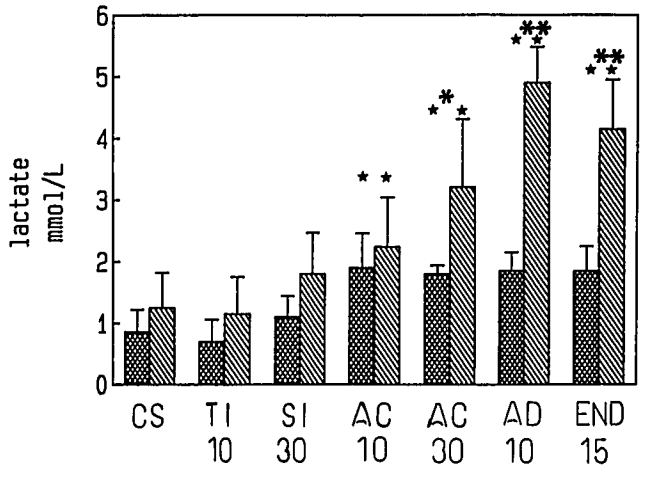

FIG. 1. Changes in plasma lactate concentrations. Ringer's group ▨; Glucose group ▧; CS control supine; T1 10 = 10 min after tracheal intubation; SI 30 = 30 min after skin incision; AC 10 = 10 min after aortic clamping; AC 30 = 30 min after aortic clamping; AD 10 = 10 min after aortic declamping; END 15 = 15 min after the end of surgery. *$P < 0.05$ *versus* control in each group. *$P < 0.05$ and **$P < 0.01$, compared with Ringer's group.

cantly during aortic clamping without any difference between the groups and rose in both groups to control values after aortic declamping.

Ten minutes after aortic clamping, blood lactate increased significantly in both groups. Thirty minutes after aortic clamping, lactate levels were significantly higher in the patients in the glucose group ($P < 0.05$) compared with those in the Ringer's group: this difference persisted ($P < 0.01$) after aortic declamping (fig. 1).

Arterial glucose did not vary in patients receiving Ringer's solution. In those in the glucose group, after skin incision, glucose rose significantly. The difference between the two groups occurred from control supine (fig. 2). Core temperature decreased significantly in both

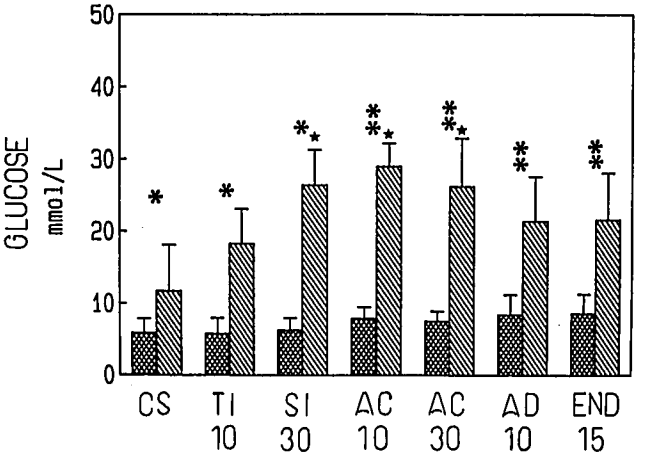

FIG. 2. Changes in plasma glucose concentrations. Same legend as in figure 1.

groups of patients without any significant difference between the groups. Hormonal data are shown in table 4. Compared with control values, no changes occurred in either group in glucagon, norepinephrine, and epinephrine.

Insulin after aortic clamping in the glucose group increased significantly. There was no change in insulin in patients in the Ringer's group. A significant difference between the groups was noted after aortic clamping ($P < 0.05$).

Following aortic clamping, cortisol increased significantly in both groups of patients without any difference between the two groups. Relationships between blood lactate concentrations and hemodynamic, metabolic, and hormonal values are shown in table 5. Lactate and core temperature were significantly correlated in both groups. Lactate and glucose were significantly correlated in patients receiving Ringer's solution. Lactate and insulin were significantly correlated in patients receiving glucose 5% solution. Hemodynamic values and other metabolic and hormonal values were independent variables for lactate in both groups.

Plasma lactate and glucose concentrations were significantly correlated in both groups from control until aortic clamping ($r = 0.62$, $P < 0.01$ in patients receiving Ringer's; $r = 0.44$, $P < 0.02$ in patients receiving glucose); from clamping until the end of surgery the correlation persisted in patients in the Ringer's group ($r = 0.50$, $P < 0.01$), whereas in patients receiving glucose ($r = 0.02$, $P > 0.05$) the correlation was absent (fig. 3).

Discussion

Hemodynamic Steady State

During infrarenal abdominal aortic surgery, cross-clamping of the aorta may be associated with severe hemodynamic alterations,[16] including altered acid–base status and increases in lactate concentrations.[9] Aortic cross-clamping and unclamping have been shown to be particularly hazardous events; hypertension, myocardial ischemia, and arrhythmias have been reported to occur after application of the aortic cross-clamp and severe hypotension with subsequent myocardial, cerebral, and renal ischemia are described following removal of the cross-clamp.[17,18]

In this study, the judicious use of volume loading, rapid-acting vasodilator, and high-dose opioid anesthesia partly avoided these severe consequences.[10,19–21]

Attenuated Surgical Stress

Surgical stress, assessed by endocrine response and plasma catecholamine levels, was minimized in these patients. No changes in glucagon or catecholamines oc-

TABLE 4. Hormonal Data

Group	Control Supine	Skin Incision 30 min	Aortic Clamping 10 min	Aortic Declamping 10 min	End 15 min
Insulin (mU/l)					
Ringer's	32.8 ± 20	24.6 ± 4.5	22.9 ± 4.7	23.6 ± 1.1	18.7 ± 10
Glucose	26.5 ± 15.7	47.7 ± 27	61.6 ± 22*‡	114 ± 37.3*‡	132 ± 55†‡
Glucagon, (pg/ml)					
Ringer's	94.5 ± 68	127.6 ± 38	130 ± 22	137 ± 32.6	147 ± 58
Glucose	128 ± 69	113.2 ± 73	120 ± 55.5	101.1 ± 45	103.4 ± 47.7
Cortisol (nmol/l)					
Ringer's	291 ± 161	438 ± 197	1,039 ± 289*	929 ± 165*	1,154 ± 282*
Glucose	231 ± 198	325 ± 256	753 ± 181*	693 ± 143*	975 ± 195*
Epinephrine (pg/ml)					
Ringer's	399 ± 317	455 ± 184	530 ± 234	572 ± 351	320 ± 203
Glucose	252 ± 126	227 ± 81	274 ± 98	252 ± 105	240 ± 53
Norepinephrine (pg/ml)					
Ringer's	606 ± 551	666 ± 541	895 ± 135	1,694 ± 1,500	759 ± 223
Glucose	429 ± 321	489 ± 253	532 ± 300	586 ± 264	493 ± 258

N = 10 in each group. All values are mean ± SEM.
* $P < 0.05$ and † $P < 0.01$, significant differences from control in each group: two-way analysis of variance (Bonferroni).
‡ $P < 0.01$, compared with patients receiving Ringer's solution (Mann-Whitney U-test).

curred in either group. Glucose and insulin responses to surgery were also avoided, as seen in the Ringer's group. Cortisol and lactate increased in both groups only after aortic clamping in the two groups, *i.e.*, after at least 90 min of surgery. These data agree with those of previous studies of patients anesthetized with a high dose of opioid.[22,23] At the same time inhibition of hypothalamic–pituitary function by high-dose fentanyl anesthesia was partially overcome after aortic clamping. Blood catecholamine concentrations that were not significantly different from control supine values differed, however, from that obtained by Normann *et al.*,[4] probably because of a different anesthetic technique and measurement method.

Inadequate Tissue Oxygenation

In spite of hemodynamic steady state and steady oxygen transport, inadequate tissue oxygenation occurred in the two groups following aortic clamping as reflected by decreased V_{O_2} and increased blood lactate. Inadequate tissue oxygenation was in part due to reduced vascular bed by clamping and reduced oxygen extraction associated with hypothermia.

Decreased V_{O_2} did not imply decreased oxygen needs, especially as lactate increased, confirming the hypothesis of Waxman *et al.*[1] that intraoperative decreased V_{O_2} had been attributed to decreased metabolic demands caused by anesthetic agents and hypothermia.[24] Moreover, when increased intraoperative lactate was observed, it suggested that the decreased V_{O_2} reflected tissue oxygen utilization rather than oxygen needs. A recent experimental study in dogs has demonstrated that anesthetic agents can increase V_{O_2}, which is greater during anesthesia than in either a drowsy or sleeping state.[25]

Blood Lactate Accumulation

Lactate is produced in cells in the course of oxidation of glucose or glycogen by the anaerobic glycolysis or the hexose monophosphate shunt: it is the dead end in the glycolytic process. Lactate can be produced even under fully aerobic conditions.[26]

During abdominal aortic surgery with cross-clamping of the aorta, decreased oxygen delivery (regional vascular obstruction), reduced oxygen uptake (lower core temperature), and decreased lactate uptake (by skeletal mus-

TABLE 5. Correlations Between Blood Lactate Concentrations and Hemodynamic, Metabolic, and Hormonal Values: Stepwise Selection for Lactate

	Step	Dependent Variable	Coefficient	SE	R^2	F(1,67)	Significance Level
Ringer's group*	2	Glucose	0.1160	0.0318	0.2007	13.305	0.07
		Core temperature	−0.2848	0.1076	0.1167	7.002	1.04
Glucose group†	2	Insulin	0.0101	0.0030	0.1777	11.453	0.15
		Core temperature	−0.5765	0.1657	0.1859	12.099	0.11

* Mean squared error = 0.6192; $R^2 = 0.3503$; $R = 0.5919$.
† Mean squared error = 1.3575; $R^2 = 0.3895$; $R = 0.6241$.

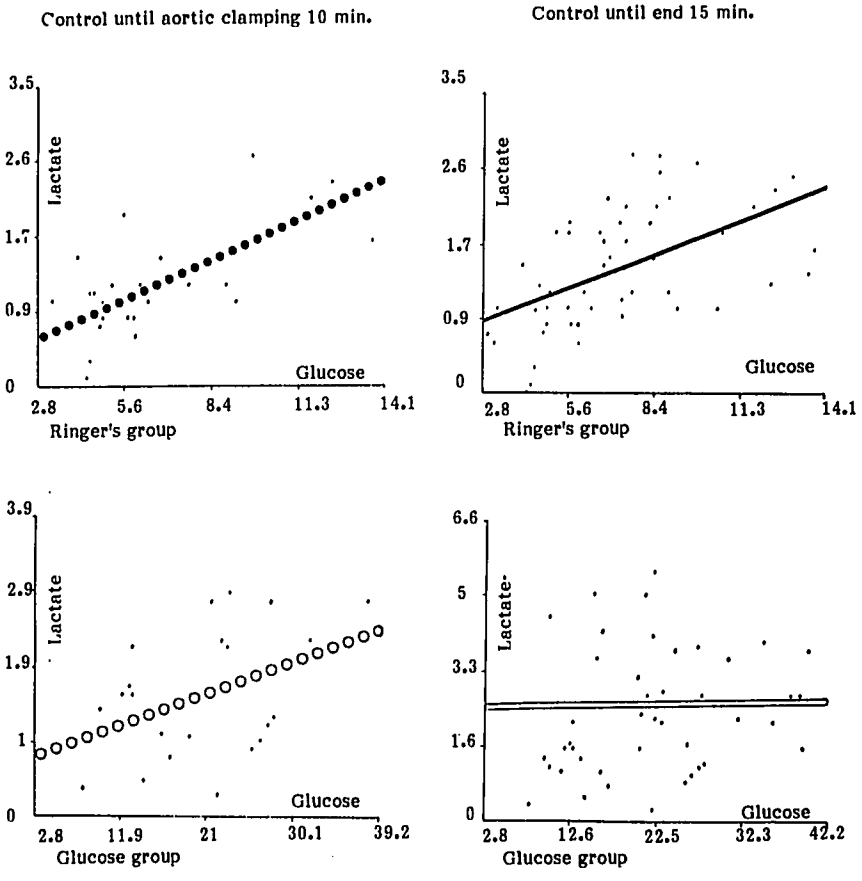

FIG. 3. Correlations between plasma lactate and plasma glucose concentrations. The ●●● line represents the regression line ($r = 0.62$; $P < 0.01$) for Ringer's group from control until aortic clamping. The ■ line represents the regression line ($r = 0.50$; $P < 0.01$) for Ringer's group from control until the end of surgery. The ○○○ line represents the regression line ($r = 0.44$; $P < 0.02$) for glucose group from control until aortic clamping. The □ line represents the regression line ($r = 0.02$: NS) for glucose group from control until the end of surgery.

cle, liver, and myocardium;[6]) resulted in an augmented amount of blood lactate. The major site of lactate production and extraction is exercising skeletal muscle.[27,28] During general anesthesia, paralyzed muscles cannot have any active contraction but maintain basal metabolism, which is altered in areas distal to the aortic clamp (pelvis and legs). The other major sites of lactate production and removal are the liver[29] and the heart.[30] Partial and varying splanchnic ischemia due to aortic clamping may augment hepatic lactate production. In this study, heart performance remained constant; therefore, cardiac lactate production did not increase whole body blood lactate. General anesthesia interfered with glucose metabolism and lactate[31,32]; however, high-dose fentanyl anesthesia produced only slight changes in lactate response.[23] The role of the sympathoadrenal system in induced glycogenolysis and lactate production was not decisive in this study (no significant changes in catecholamines).[33]

Finally, with regard to inadequate tissue oxygenation, this study verified in both groups that in the presence of ischemia or hypoxia (aortic clamping), oxidative metabolism of glucose was impaired and glycolysis with its end product of lactate increased.[8,34,35]

In addition, this study noted that intraoperative glucose infusion induced a rise in blood lactate, insulin, and glucose as reflected by the difference between patients receiving glucose and Ringer's solution. The exogenous glucose supply gave rise to augmented endogenous insulin after a delay due to fentanyl anesthesia.[36] After the aortic clamping, the absence of a positive correlation between lactate and glucose in patients receiving glucose showed that the imbalance between oxidative metabolism of glucose and glycolysis provoked by aortic clamping ischemia was worsened by glucose infusion.

This study supports the hypothesis that intraoperative glucose infusion, with associated ischemia, provokes greater blood lactate accumulation than intraoperative infusion of Ringer's solution, and that the intraoperative blood lactate level depends both on glucose supply and inadequate tissue oxygenation occurring during aortic surgery.

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