Title
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Publication Date
1989

Peer reviewed|Thesis/dissertation

The Validity of the Pulse Oximeter in Critically Ill Hemodynamically Compromised Patients

by

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THESIS

Submitted in partial satisfaction of the requirements for the degree of

MASTER OF SCIENCE

in

Nursing

in the

GRADUATE DIVISION

of the

UNIVERSITY OF CALIFORNIA

San Francisco

Acknowledgments

The author wishes to acknowledge the nursing staff of the Surgical Intensive Care Unit at the Veterans Administration Medical Center, Palo Alto, California for their assistance with data collection during the research study that forms the basis for this thesis. The text of this thesis has been submitted for publication in Heart & Lung.

The Validity of the Pulse Oximeter in Critically Ill Hemodynamically Compromised Patients

Joni Louise Dirks

The purpose of this descriptive study was to determine the correlation between in vivo arterial oxygen saturation (Sa02) as measured by the pulse oximeter (NSa02) and oxygen saturation derived from in vitro measurements (Sa02-calc.) obtained by arterial blood gas analysis (ABG) across the available range of saturations in critically ill, hemodynamically compromised patients. Previous studies have documented the validity of the pulse oximeter in healthy subjects and in a variety of patient populations. Questions remain about the validity of pulse oximetry data in patients who are hypotensive or vasoconstricted. Valid information in this patient population is essential to nurses who must act quickly to counteract life-threatening hypoxemic episodes. The convenience sample consisted of 18 subjects who met the following criteria: (1) status post surgery within one week; (2) indwelling arterial line and pulmonary artery catheter as a part of prescribed therapy; (3) hypotension, defined as a mean arterial pressure (MAP) $\leq$ 70 torr and/or vasoconstriction, defined as a systemic vascular resistance (SVR) > 1300 dynes/sec/cm5; (4) core body temperature $\geq$ 36° C, and (5) no lipid transfusions for 24 hours prior to the study. Measurements of NSa02 and Sa02-calc. were recorded on each subject whenever ABGs were obtained for a period of 48 hours after entry into the study. Hemodynamic variables were recorded with each Sa02 measurement. A total of 282 data points were recorded, with a range of 12-21 observations per subject. The relationship between NSa02 and Sa02-calc. within individual subjects was evaluated using Pearson r correlations. The correlation between NSa02 and Sa02-calc. for individual subjects ranged from r = .42-.81 (p<.05). The correlation between these two parameters was higher for the group of subjects who were predominantly normotensive (r = .69, p<.05) than for those who were hypotensive (r = .51, p<.05). A higher individual correlation was found in subjects when data obtained under conditions of "low quality signal" were excluded from the analyses. The NSa02 measurements were less than or equal to the in vivo measurements in 77% of the data points recorded, making it a safe means to follow trends in oxygenation in patients in the intensive care unit.

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The Validity of the Pulse Oximeter in Critically Ill Hemodynamically Compromised Patients

Critically ill patients exhibit a broad spectrum of respiratory problems. Hypoxemia is the primary cause of the morbidity and mortality due to respiratory dysfunction.[1] The prevalence of respiratory dysfunction makes early detection of hypoxemia a fundamental concern for intensive care nurses. A valid means of evaluating oxygenation status is needed. Early hypoxemia is difficult to diagnose in the clinical setting on the basis of signs and symptoms. Its onset is often heralded by subtle compensatory changes in respiratory rate and cardiac rate and rhythm and, in the critical care setting, the use of drugs or presence of severe illness may inhibit these normal compensatory mechanisms.[2]

Traditionally, oxygen measurements obtained from the analysis of arterial blood gases have served as the standard in the assessment of a patient's oxygenation status. Classically, the partial pressure of oxygen (p02) in arterial blood was used to define hypoxemia.[3] Over the last decade, advances in technology have made it possible to monitor oxygenation noninvasively with a pulse oximeter. The pulse oximeter provides a different measure of oxygenation, the oxygen saturation of hemoglobin in arterial blood (Sa02). Unfortunately this instrument was adopted in clinical practice before adequate information was available to the clinician regarding its possible limitations.[4] The validity

of the measurements of the pulse oximeter need to be established in selected critical care patient populations. Studies are lacking to support the use of pulse oximetry in hemodynamically compromised patients. The purpose of this study was to determine the correlation between in vivo arterial oxygen saturations as measured by the pulse oximeter and oxygen saturation derived from in vitro measurements via arterial blood gas analysis in critically ill, hemodynamically compromised adult surgical patients.

Review of the Literature

Oxygen Measurement

Measurement of arterial oxygenation saturation is a relatively new phenomenon. Until the 1940's clinicians depended on cyanosis to estimate the adequacy of oxygenation. But in 1946 researchers found that cyanosis was an unreliable indicator of hypoxemia.[5] Since the invention of the Clark electrode in 1953, arterial blood gas (ABG) analysis has served as the gold standard for assessing the status of oxygenation in patients.[6] ABGs provide the clinician with a measurement of the p02 of arterial blood, as well as the partial pressure of carbon dioxide (pC02) and arterial pH. Sa02 is either measured directly with a co-oximeter, or more commonly, estimated from the standard oxyhemoglobin dissociation curve. ABGs are intermittent measurements, invasive, require frequent blood sampling, and in many settings the results are not immediately available. Despite

these disadvantages, arterial blood gases are a definitive physiological indicator of hypoxemia.[3]

The pulse oximeter is widely used to avoid some of the difficulties posed by ABG analysis of oxygenation.[7] A number of research studies have been conducted to evaluate the pulse oximeter. These studies have provided reliability and validity information on the instrument itself and uncovered potentially intervening variables which may modify measurements in clinical practice. In 1986, at an international symposium held to consider the practical utilization of the pulse oximeter, pulmonary authorities from 21 countries concurred that further testing of the oximeter was needed to delineate its limitations and range of accuracy.[8]

Research Review

Studies on the pulse oximeter have been conducted in the laboratory setting, using healthy volunteers as subjects.[9-13] These studies have shown a strong correlation between in vivo measurements of Sa02 obtained with the pulse oximeter and in vitro measurements obtained with a co-oximeter($r \geq .90$) in controlled laboratory experiments. Generalization of these findings to the intensive care setting is limited by the use of healthy subjects.

Several studies of the oximeter have been conducted in the intensive care setting.[10,14-17] In the one study that specifically addressed the effects of hemodynamics on pulse

oximeter accuracy, researchers found a moderate correlation between in vivo and in vitro measurements of Sa02 in unstable critical care subjects (r = .57, p<.005).[16] They also reported that extremes of systemic vascular resistance led to overestimation of the Sa02 by the pulse oximeter. Other variables found to interfere with the accuracy of oximeter readings include hypothermia and hypotension,[15] intralipid infusions,[18] dark skin pigmentation,[19] and elevated levels of carboxyhemoglobin.[20]

Related research in the field of noninvasive blood pressure monitoring has shown that measurement of arterial pressure in the finger may be hampered by insufficient blood flow and peripheral vasoconstriction.[21,22] Such instruments are similar to the pulse oximeter in that proper functioning depends on a pulsatile signal, as demonstrated by a plethysmographic waveform. Researchers have found discrepancies between direct measurements of intraarterial pressure and those measured noninvasively on the finger during periods of drug-induced or physiologically mediated vasoconstriction.[21] Because the pulse oximeter depends on adequate pulsatile flow to the extremities, its functioning also may be affected adversely by vasoconstriction and low flow states.

In vivo measurements of Sa02 obtained with the pulse oximeter correlate well with in vitro measurements in the upper levels of saturation, in both laboratory studies and a

limited number of clinical trials. The validity of the pulse oximeter may be enhanced if the clinician compensates for the effect of intervening variables (carboxyhemoglobin, skin pigmentation, intralipid infusions, and hypothermia). Questions remain, however, regarding the validity of this instrument in the critical care population, especially in patients who are hypotensive or vasoconstricted. Such questions must be addressed since hypotension and vasoconstriction commonly occur in critically ill patients as a result of disease processes and treatment modalities. Critically ill patients could benefit markedly from an accurate method of continuous oxygenation status monitoring.

Method

Design

A descriptive, correlational design was used in this research study to determine the correlation between in vivo and in vitro measurements of Sa02 over the available range of saturations in selected subjects. The study protocol was approved by the institutional committee on Human Research.

Sample

The sample was selected from patients admitted consecutively to a nine-bed surgical intensive care unit, located in a West Coast university affiliated teaching hospital. All subjects met the following criteria: (1) postoperative from a surgical procedure within one week, (2) indwelling arterial line and pulmonary arterial catheter as a

part of prescribed medical therapy, (3) core temperature of $\geq$ 36° C, (4) no lipid transfusions within 24 hours prior to the study, (5) and hypotension (a mean arterial pressure $\leq$ 70 torr) and/or vasoconstriction (a systemic vascular resistance > 1300 dynes/sec/cm5). The parameters for defining hypotension and vasoconstriction were based on previous research describing hemodynamic patterns for critically ill patients.[23] The convenience sample consisted of 18 postoperative patients who met all study criteria. In the sample there were 17 men and one woman with a mean age of 64.2 years, a range of 41 to 88 years. Operative procedures included coronary artery bypass surgery, mitral or aortic valve replacement, and major abdominal surgery (Table 1). Thirteen of the subjects reported a history of smoking prior to admission. All of the subjects received vasoactive drugs of some type during the course of the study. The various intravenous drugs received by subjects in this study included nitroglycerin, sodium nitroprusside, dopamine, epinephrine, norepinephrine, dobutamine, trimethaphan, and amrinone.

Instruments

The Biox 3700 (Ohmeda, Boulder, CO) pulse oximeter was used in this study to obtain noninvasive measurements of oxygen saturation (NSa02). This instrument measures pulse rate and amplitude as well as oxygen saturation via a finger or ear probe that contains two light emitting diodes (one red and one infrared) and one photodiode.

Pulse oximetry works by placing a pulsating arterial bed between a light source and a photodetector. The expansion and contraction of the pulsating vascular bed modifies the amount of light detected, and results in a plethysmograph waveform.[12] The NSa02 is determined by variations in the absorbance of the two wavelengths of light. Oxygenated hemoglobin absorbs a large amount of the infrared wave, but not much of the red wave. Deoxygenated hemoglobin absorbs more of the red wave and less of the infrared. Changes in the absorbances of the red and infrared light produce a ratio of oxygenated to deoxygenated hemoglobin which is then calculated and displayed by the pulse oximeter as an Sa02 percentage.

The Biox 3700 graphically displays a plethysmographic waveform along with a digital display of the patient's pulse rate and NSa02. If the signal strength is half scale or less, the alarm message "Low Quality Signal" appears above the graphic display to indicate that the NSa02 and pulse rate reading my be invalid due to unreliable data. The manufacturer suggests that this data be disregarded and that probe placement be checked or an alternate test site be selected to obtain an adequate signal. When used according to the manufacturer's instructions, the Biox 3700 reports Nsa02 within 1.5-2.4% accuracy when compared to the Sa02 measured via co-oximeter.[24]

Arterial blood gas samples were analyzed with the Corning

178 Automatic pH/Blood Gas Analyzer (Corning Limited, Essex, England). This machine directly measures pH, pC02, and p02 of three milliliter blood samples, using electrodes that have been calibrated with known gaseous or aqueous standards. The Corning 178 calculates oxygen saturation (Sa02-calc.) for each sample with a standard equation contained in its microprocessor. The Corning 178 is both accurate and precise when operated according to the manufacturer's instructions. The company reports 0.7-1.2% standard deviation from the mean in trials using p02 values between 28.4-107.0 torr.[25] The Corning 178 automatically performs two-point calibrations every two hours, and is recalibrated by the blood gas technician after every third specimen.

Procedure

Patients who met protocol criteria were entered into the study consecutively as they were admitted to the intensive care unit. Demographic data and pertinent patient characteristics were obtained by the investigator via chart review. Physiological data were collected by staff nurses who had been trained individually to use the study's protocol. The researcher observed each staff nurse who participated in data collection to promote uniformity in obtaining blood gases according to the established hospital procedure.

All subject's were continuously monitored with a 5-lead electrocardiogram (EKG), mean arterial pressure (MAP),

pulmonary artery pressures and central venous pressure via Siemens monitors (Siemens Medical Systems, Inc., Danvers, MA). Pressure transducers were recalibrated every four hours, with the fourth intercostal space in the mid-axillary line as the zero reference point. Cardiac output was measured via the pulmonary artery catheter using the thermodilution technique. Ten milliliters of room temperature injectate were used for each of three measurements, which then were averaged and the mean recorded. SVR was calculated with the following formula:

$$SVR = (MAP - CVP) \times 80 \div \text{cardiac output}$$

The Biox 3700 oxisensor was applied to the subject's ear or finger, using the appropriate probe, and adjusted for continuous display of the NSa02 according to the operator's manual. If a "low quality signal" was obtained, another probe site was selected. If only low quality signals were obtained, this information was recorded on the data collection sheet. The NSa02 measurements were recorded directly from the Biox 3700 display.

The calculated Sa02 (SA02-calc.) was derived from an arterial blood gas sample obtained from the arterial line within one minute of the NSa02 reading. Following a three milliliter discard sample, blood gases were drawn from the arterial line in a heparinized syringe. Air bubbles were expelled, the specimens were capped, placed on ice, and sent to the lab for analysis within 20 minutes. The

results of the blood gas were recorded on the data sheet along with the subject's core temperature, heart rate as displayed by both the EKG and the pulse oximeter, most recent cardiac output and SVR measurements, and present intravenous medications. All data points were recorded when the heart rate via EKG and the pulse rate via oximeter were within ± 3 beats per minute. This procedure was repeated each time an ABG was obtained for a 48 hour period or until the patient's arterial line was removed, whichever came first.

Data Analysis

Descriptive statistics were used to describe the sample and the physiological variables measured in this study. The Pearson product moment correlation was used to evaluate the relationship between NSa02 and Sa02-calc. within individual subjects. Separate analyses were done on the subset of data points that occurred when the SVR was greater than 1300 dynes/sec/cm5 and for data obtained when the "Low Quality Signal" alert was in effect. The number of data points obtained varied for each subject: mean values of NSa02, Sa02-calc., MAP, & SVR were used to make comparisons between groups. Mean values were used to evaluate the correlation between NSa02 and Sa02-calc. across the sample and within the subsets of subjects who were hypotensive and normotensive. Paired *t* tests were used to evaluate the differences between NSa02 and Sa02-calc. within individual subjects. The alpha

for statistical significance for the correlations and *t* tests was set a priori at $p<.05$.

Results

A total of 282 data sets were recorded for the 18 subjects studied, with a range of 12-21 observations per subject (Figure 1). The range of values for NSa02 measurements was 89-100% and for Sa02-calc. 90-99% across all data recorded. Individual mean values for the hemodynamic and oxygenation variables studied are presented in Table 2.

The correlation between NSa02 and Sa02-calc. for individual subjects ranged from $r = .42-.81$ ($p<.05$) and remained similar when subjects were divided into hypotensive ($r = .42-.79$, $p<.05$) and normotensive ($r = .44-.81$, $p<.05$) groups (Table 3). The correlation between NSa02 and Sa02-calc. across each of these groups was higher for subjects who were predominantly normotensive ($r = .69$) than for those who were hypotensive ($r = .51$). The correlation between NSa02 and Sa02-calc. for all subjects in the group as a whole was $r = .61$.

Only 3 subjects experienced periods when their systemic vascular resistance was greater than 1300 dynes/sec/cm5. There were only two to three data points for each of these subjects under this condition and therefore no valid correlations could be derived. Eight subjects had data recorded when the "low quality signal" alert was in effect, indicating that the NSa02 data may be unreliable due to a

weak signal. Of the 40 low quality data points recorded, 27 (68%) occurred during periods of hypotension (i.e. MAP $\leq$ 70 mmHg). A separate correlation between NSa02 and Sa02-calc. was computed for these subjects, excluding the low quality data points. A statistically significant individual correlation was found in each of five subjects, and in all cases was higher than the correlations that included low quality data (Table 4).

The noninvasive measurements of saturation (NSa02) were less than or equal to the in vivo measurements (Sa02-calc.) in 217 of 282 measured pairs of data (77%). In ten of the 18 subjects the individual mean NSa02 was consistently lower than the individual mean Sa02-calc.. This difference was statistically significant ($p<.05$) in all ten subjects as analyzed by paired t test (Table 5). These results indicate that clinically the pulse oximeter is more likely to provide information that is falsely low in comparison to in vitro measures of Sa02.

All of the subjects received vasoactive drugs at some time during the course of the study. The value of NSa02 remained stable within each subject across varying dosages of these vasoactive medications. An analysis of the direct effect of these drugs on the functioning of the pulse oximeter was limited by subject's individual hemodynamic responses to the medications and wide variations in the combinations of drugs received by subjects in this study.

Such drugs are used clinically to maintain an optimal cardiac output and systemic vascular resistance, as reflected by the mean arterial blood pressure. Because adequate perfusion pressure is needed for proper functioning of the pulse oximeter, vasoactive drugs may have indirectly affected NSa02 readings via their effect on cardiac output, systemic vascular resistance, and mean arterial pressure.

Discussion

Noninvasive measurements of oxygen saturation measured via the pulse oximeter correlated moderately with in vitro measurements of Sa02 in this sample of hemodynamically compromised patients. The overall correlation (r = .61, p< .05) approximated that obtained in a previous study conducted with a similar sample.[16] While the correlation was higher in subjects who were predominantly normotensive (r = .69, p<.05), it still remained lower than correlations previously reported for the critical care population.[10, 14-15, 17]

Study Validity

The potential intervening variables of lipids and hypothermia were controlled for in this study. Because all of the subjects in this study were Caucasian, this study did not evaluate the effect of skin pigmentation on the accuracy of oximetry readings. High levels of carboxyhemoglobin could have influenced the findings and was not measured in this study. Although thirteen of the subjects studied had smoked

in the past, none had smoked for at least three days prior to their surgery. Carboxyhemoglobin has a half-life of approximately five hours, and should not have been present in a quantity that would have affected Sa02 validity.[3]

Other researchers have suggested that the use of vasoactive medications in individuals may have some affect on the correlation between NSa02 and Sa02-calc.[15,16] While all of the subjects received vasopressors and/or vasodilators in various quantities, it was unlikely that these agents had a significant effect on individual correlations in this study, since the value of the NSa02 remained stable as these drugs were discontinued. The stability of the individual correlations may be explained by the observation that medications were titrated to keep the systemic vascular resistance within normal limits, as evidenced by the small number of data points obtained when the SVR was elevated. Thus the degree of vasoconstriction achieved with such medications may not have been high enough to influence the pulse oximetry readings obtained in this study.

Implications for Practice

The results of this study have several implications for clinical use of the pulse oximeter. The data indicate that the correlation between measurements of oxygen saturation obtained with the pulse oximeter and those obtained from arterial blood gases is weaker when critically ill patients are hypotensive than when they are normotensive. Therefore,

the NSa02 obtained via the pulse oximeter should not be relied upon as an absolute measurement of oxygen saturation in this patient population. Rather, the trend in oxygen saturation provided by the pulse oximeter could be more informative.

Data obtained under "low quality signal" conditions were included in this study because in clinical practice this information is often used when an adequate signal cannot be obtained. Correlations between invasive and noninvasive measures of Sa02 were stronger in these hemodynamically compromised subjects when low quality data were excluded indicating that such information should be used cautiously, if at all.

It is also clinically significant that in the majority of data sets the NSa02 was equal to or less than the Sa02-calc. Such "false negative" information could provide a safe margin of error in clinical interpretation of oximetry readings. That is, clinicians could set parameters for acceptable NSa02 values in individual patients (i.e. "maintain NSa02 $\geq$ 93%") with the assurance that the oximeter would not overshoot the true Sa02 and provide falsely elevated values. Interpretation by clinicians of lower rather than higher values would allow corrective measures to be taken before dangerous hypoxemic states were reached.

Even with these limitations, pulse oximetry measurements could be used to follow trends in oxygenation, alerting the

clinician to sudden changes in oxygenation status. The oximeter could be used to monitor patients during procedures associated with episodes of hypoxemia, such as nasotracheal suctioning, chest physiotherapy, and ventilator weaning. The additional information provided by oximetry could be used in conjunction with subjective and objective indicators of hypoxemia to determine the need for obtaining arterial blood gases. The pulse oximeter could thus augment, not replace, traditional assessments of hypoxemia.

Implications for Future Research

This study needs to be replicated with a larger sample of critically ill patients and an increased number of data points for each individual subject to obtain information that can be generalized to large numbers of critically ill patients with various diagnoses. More data is needed during periods of vasoconstriction to establish the stability of the correlation between NSa02 and Sa02-calc. under these conditions, since vasoconstriction often occurs in critical care patients during episodes of hypovolemic or cardiogencic shock. Clinical research is needed to establish the correlation between NSa02 and in vitro Sa02 at saturations less than 90%, as little research has been conducted in the critical care setting to establish the accuracy of the pulse oximeter in this range. Sudden decreases in a patient's NSa02 may indicate severe physiologic dysfunction, but this data is often discarded by the clinician as potentially

inaccurate. Also future research needs to address optimal methods of obtaining pulse oximetry signals, to decrease the amount of "low quality signal" data obtained in clinical practice.

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Table 1

Description of sample by age, smoking history, and surgical procedure

Subject	Age	Smoking history	Surgical procedure
1	41	yes	abdominal surgery
2	88	no	coronary artery bypass
3	65	yes	mitral valve replacement
4	70	yes	abdominal surgery
5	60	yes	mitral valve replacement
6	72	yes	aortic/mitral valve replacement
7	67	no	coronary artery bypass
8	42	yes	aortic valve replacement
9	78	yes	mitral valve replacement
10	63	yes	coronary artery bypass
11	70	yes	coronary artery bypass
12	63	yes	coronary artery bypass/ aortic valve replacement
13	74	no	aortic valve replacement
14[a]	69	no	abdominal surgery
15	69	yes	coronary artery bypass
16	46	yes	coronary artery bypass
17	60	no	coronary artery bypass
18	59	yes	coronary artery bypass

[a]all were male except subject 14

Table 2

Mean (+ standard deviation) hemodynamic and saturation values for individual subjects

Subject(#[a])	MAP[b]	(±SD)	SVR[c]	(±SD)	NSa02	(±SD)	Sa02-calc.	(±SD)
1 (17)	67.5	(±4.0)	280	(±48)	93.9	(±1.3)	95.3	(±1.5)
2 (19)	69.9	(±6.2)	1220	(±300)	96.7	(±1.9)	96.5	(±1.6)
3 (16)	67.2	(±3.8)	588	(±56)	95.9	(±2.1)	96.6	(±1.5)
4 (16)	68.2	(±7.3)	1069	(±796)	96.6	(±1.8)	97.6	(±1.2)
5 (20)	66.6	(±6.0)	522	(±452)	98.5	(±1.5)	97.4	(±1.0)
6 (18)	68.2	(±7.6)	833	(±113)	94.6	(±1.2)	96.7	(±1.4)
7 (15)	67.3	(±5.0)	782	(±104)	95.9	(±1.7)	96.3	(±1.7)
8 (19)	63.0	(±5.3)	486	(±135)	95.5	(±2.3)	95.9	(±2.3)
9 (13)	60.6	(±5.3)	594	(±71)	94.3	(±2.6)	96.9	(±1.7)
10 (16)	69.1	(±4.1)	650	(±166)	94.9	(±2.5)	97.3	(±1.6)
11 (12)	77.2	(±5.3)	745	(±116)	95.9	(±1.6)	97.3	(±1.0)
12 (12)	73.7	(±7.6)	789	(±117)	95.2	(±2.1)	95.6	(±1.8)
13 (12)	76.4	(±6.2)	865	(±176)	98.2	(±1.3)	97.8	(±1.0)
14 (17)	75.6	(±10.5)	872	(±124)	94.6	(±2.0)	94.1	(±2.3)
15 (12)	75.3	(±5.9)	726	(±156)	94.5	(±1.2)	97.0	(±1.4)
16 (15)	75.1	(±5.7)	1094	(±257)	94.5	(±2.2)	95.3	(±1.8)
17 (12)	73.2	(±5.0)	712	(±99)	94.3	(±3.2)	96.1	(±2.6)
18 (21)	81.2	(±11.1)	604	(±187)	94.0	(±2.0)	95.2	(±2.0)

[a]# = number of data points [b]mean arterial pressure measured in mm Hg
[c]systemic vascular resistance measured in dynes/sec/cm5

Table 3

Correlations between NSa02 and Sa02-calc. for hypotensive and normotensive individuals

Hypotensive MAP[a] ≤ 70 mmHg				Normotensive MAP[a] > 70 mmHg			
Subject	#[b]	r	p	Subject	#[b]	r	p
1	17	.61	<.01	11	12	.48	<.05
2	19	.43	<.05	12	12	.81	<.01
3	16	.59	<.01	13	12	.44	<.05
4	16	.79	<.01	14	17	.45	<.05
5	20	.65	<.01	15	12	.46	<.05
6	18	.45	<.05	16	15	.71	<.01
7	15	.68	<.01	17	12	.69	<.01
8	19	.75	<.01	18	21	.65	<.01
9	13	.42	<.05				
10	16	.75	<.01				
		group r = .51				group r = .69	
		p<.05				p<.05	

[a]mean arterial pressure measured in mm Hg

[b]# = number of data points per subjects

Table 4

Correlations between NSa02 and Sa02-calc. in subjects with and without "Low Quality Signal" (LQS) data points

Subject	excluding LQS data		for total data	
	r*	#[a]	r*	#[a]
2	.49	12	.43	19
3	.63	11	.59	16
7	.87	12	.68	15
14	.77	13	.45	17
18	.68	17	.65	21

*$p < 0.05$

[a]# = number of data points per subject

Table 5

Differences between mean NSa02 and Sa02-calc. (NSa02 < Sa02-calc.) by t test

Subject	#[a]	t	df	p
1	17	4.741	16	<.01
4	16	3.337	15	<.01
6	18	5.916	17	<.01
9	13	3.875	12	<.01
10	16	6.012	15	<.01
11	12	3.218	11	<.01
15	12	6.268	11	<.01
16	15	1.977	14	<.05
17	12	2.682	11	<.05
18	21	3.225	20	<.01

[a]# = number of data points per subject

Figure 1

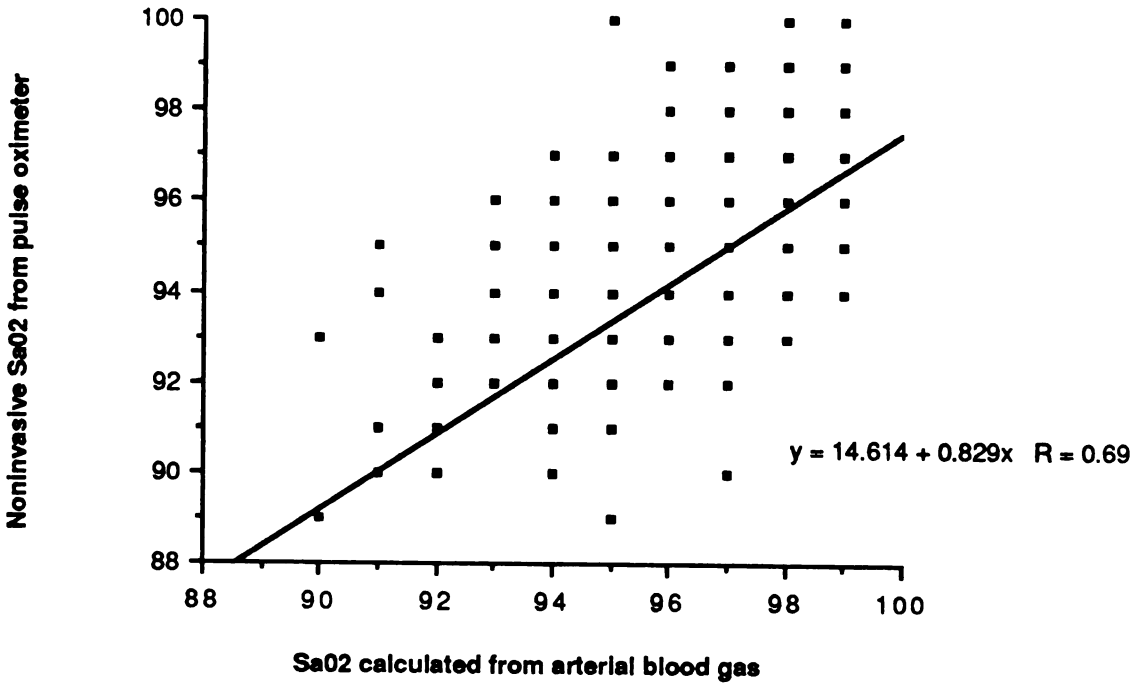
Noninvasive Sa02 from pulse oximeter
100
98
96
94
92
90
88
88
90
92
94
96
98
100
y = 14.614 + 0.829x R = 0.69
Sa02 calculated from arterial blood gas

Figure Caption

Figure 1.

Scatterplot of data points correlating NSa02 and Sa02-calc. for all subjects (n=18).