Thenar oxygen saturation during weaning from mechanical ventilation: an observational study

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ABSTRACT Our aim was to determine whether thenar tissue oxygen saturation (StO_2), measured by noninvasive near-infrared spectroscopy, and its changes derived from an ischaemic challenge are associated with weaning outcome.

Our study comprised a prospective observational study in a 26-bed medical–surgical intensive care unit. Patients receiving mechanical ventilation for >48 h, and considered ready to wean by their physicians underwent a 30-min weaning trial. StO_2 was measured continuously on the thenar eminence. A transient vascular occlusion test was performed prior to and at the end of the 30-min weaning trial, in order to obtain StO_2 deoxygenation and reoxygenation rates, and estimated local oxygen consumption.

37 patients were studied. Patients were classified as weaning success (n=24) or weaning failure (n=13). No significant demographic, respiratory or haemodynamic differences were observed between the groups at inclusion. Patients who failed the overall weaning process showed a significant increase in deoxygenation and in local oxygen consumption from baseline to 30 min of weaning trial, whereas no significant changes were observed in the weaning success group.

Failure to wean from mechanical ventilation was associated with higher relative increases in deoxygenation after 30 min of spontaneous ventilation.

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Introduction

Discontinuation of ventilatory support in critical care medicine can be a difficult challenge in about one-third of patients [1]. Failure to wean from mechanical ventilation (MV) is difficult to predict [2]. Furthermore, failed extubation is associated with increased hospital mortality, prolonged intensive care unit and hospital stays, and increased need for tracheostomy [3].

As spontaneous ventilation is a form of cardiovascular stress test, failure to wean from MV often reflects cardiovascular insufficiency owing to the increased oxygen cost of breathing [4, 5]. Normally, any increased oxygen cost of breathing is met by an increased respiratory muscle blood flow, which, in flow-limited states, will divert blood flow away from other tissues, such as the splanchnic and the peripheral circulation [6], and may lead to their hypoperfusion [7–11].

Recently, skeletal muscle oxygen saturation on the thenar eminence (StO_2) measured noninvasively using near-infrared spectroscopy (NIRS) has been proposed as an early and reliable measure of hypoperfusion states [12–14]. This proposal is based on the idea of the peripheral and splanchnic circulation shunting in low flow states, where blood flow is diverted from less-vital to more-vital areas, such as the heart, brain and respiratory muscles. In addition to reporting steady-state StO_2 [15–17], functional monitoring using a transient total vascular occlusion allows for further assessment of cardiovascular state. The dynamic vascular occlusion test (VOT) creates the novel StO_2 parameters deoxygenation rate (DeO_2) and, upon removal, reoxygenation rate (ReO_2), which improve the predictive value of StO_2 in certain critically ill populations [18, 19].

We postulated that NIRS monitoring would demonstrate significant hypoperfusion of peripheral skeletal muscle in patients during unsuccessful attempts to wean from MV. The purpose of this study was to analyse whether StO_2 and its VOT-derived changes can be useful in predicting weaning outcome in critically ill patients.

Material and methods

This prospective observational study was conducted in a 26-bed medical–surgical intensive care unit at a university hospital (Hospital de Sabadell, Barcelona, Spain). This study was approved by the Institutional Review Board at the Hospital de Sabadell. Informed consent was obtained from either the patient or their next of kin prior to the study initiation.

We included adult patients (aged $\geqslant$18 years) receiving invasive MV for $>$48 h and considered ready to wean by their physicians according to the following criteria: partial or complete recovery from the underlying cause of acute respiratory failure; adequate gas exchange, as indicated by an arterial oxygen tension of $>$60 Torr (7.99 kPa) and an inspiratory oxygen fraction (FIO_2) of $<$0.4, with a positive end-expiratory pressure of $<$5 cmH_2O; a core temperature of $<38^{\circ}$ C; haemoglobin $>$8 $g \cdot dL^{-1}$; and no further need for vasoactive and/or sedative agents.

Exclusion criteria were trauma in both upper limbs, and haematoma or skin lesions at the thenar eminence that could hinder placement of a NIRS sensor probe. Patients with an altered level of consciousness that could lead to central hypoventilation and/or impaired secretion management were also excluded.

Study protocol

After inclusion, patients underwent a weaning trial (WT) for 30 min, defined as assisted spontaneous breathing with continuous positive airway pressure of 5 cmH_2O, or a T-tube trial, according to their medical team. Patients were in a semi-recumbent position, and the FIO_2 was kept constant during the trial.

The evaluation criteria for WT failure were defined as the presence of one or more of the following during the 30-min trial: a respiratory rate of $>$35 $breaths \cdot min^{-1}$ for $\geqslant$5 min; an arterial oxygen saturation measured by pulse oximetry $<$90%, heart rate $>$140 $beats \cdot min^{-1}$ or sustained increase or decrease in heart rate of $>$20%; a systolic blood pressure $>$180 mmHg or $<$90 mmHg; increased anxiety; and diaphoresis. The decision to remove the endotracheal tube was made independently of the study investigators at the end of a successful WT by their attending physicians, who did not have access to the specific StO_2 data. Weaning success was defined as a patient remaining free of mechanical ventilatory support for $>$24 h after passing the WT. Weaning failure was defined as either failure to pass the WT (WT failure) or reinstitution of MV within 24 h of extubation (extubation failure) (fig. 1).

Methods

Demographic data, age, sex, diagnosis and days on MV, were collected. Haemodynamic, respiratory and oxygenation variables were monitored continuously and recorded just before starting and at 30 min into the WT. Heart rate and mean systemic arterial pressure (mAP) were recorded by routine bedside monitoring (Monitor Intellivue MP 70; Phillips Medizinsystems, Boeblingen, Germany). Respiratory rate, tidal volume, minute ventilation, FIO_2 and arterial oxygen saturation measured by pulse oximetry were recorded at start

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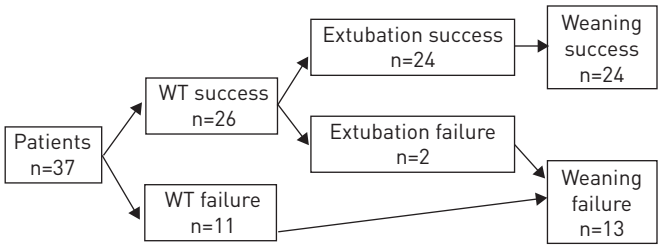

FIGURE 1 Flow chart of patients recruited in the study. Patients who were extubated without reinstitution of mechanical ventilation (MV) within 24 h were considered to be a "weaning success" (n=24). Patients who failed either the weaning trial (WT) or were extubated but required reinstitution of MV within 24 hours were considered to be a "weaning failure" (n=13).

and 30 min into the WT (Monitor Intellivue MP 70; Phillips Medizinsystems). Arterial blood gas analysis was performed before and 30 min into the WT (ABL 700 series; Radiometer Medical, Copenhagen, Denmark). StO_2 was recorded continuously using the InSpectra 650 Tissue Spectrometer (Hutchinson Technology, Hutchinson, MN, USA). The StO_2 15-mm optical surface probe was placed on intact skin on the thenar eminence; it was never placed adjacent to the site of radial artery cannulation. The InSpectra Tissue Spectrometer also measures relative haemoglobin concentration in the NIRS field of view, which is presented as the tissue haemoglobin index (THI).

Vascular occlusion test

The VOT was performed as previously described by GÓMEZ *et al.* [18]. Briefly, a blood pressure cuff is placed proximal to the hand on the forearm and rapidly inflated to 40 mmHg above systolic pressure and kept inflated until StO_2 decreases to 40%. Then the cuff is rapidly deflated and the rate of increase in StO_2 noted. The resulting DeO_2 and ReO_2 slopes are reported as change in oxygensaturation over time. We performed a VOT at the beginning of the WT and again at 30 min into the WT, giving DeO_2 and ReO_2 data paired for each time-point. Using the DeO_2 slope and the THI values at the beginning and at the end of the VOT, we also calculated the NIRS-derived thenar muscle oxygen consumption ($nirV'O_2$), as described by the equation of SKARDA *et al.* [20]: $nirV'O_2=(DeO_2\ slope)^{-1}/((THI_{start}+THI_{end})/2)$. Relative changes in StO_2-derived parameters were calculated as the quotient between values at 30 min and baseline. Absolute StO_2 and VOT-derived variables were obtained automatically using the InSpectra Research Software® v4.01 (Hutchinson Technology).

Analysis

A descriptive analysis was performed. The Kolmogorov–Smirnov test was used to verify the normality of distribution of the studied variables. Continuous variables are presented as median values with interquartile ranges. The Wilcoxon test was used to analyse changes over time in the paired variables. For continuous variables, the Mann–Whitney U-test and, for categorical variables, Fisher's exact test were used to compare failure and success groups. Data were analysed using the SPSS 17.0 Software (Chicago, IL, USA). Statistical significance was defined as $p<0.05$ (two-tailed test).

Results

37 patients were studied. Patient baseline characteristics are summarised in table 1. 26 patients succeeded at the WT and were extubated. However, two of the patients who were extubated required instauration of MV within 24 h (extubation failure). 11 patients failed the WT (fig. 1). No baseline differences in demographic, haemodynamic and respiratory variables were observed when comparing success and failure groups (table 1), except for lower baseline heart rate in those patients who failed the weaning process (median 78 (interquartile range 71–91) *versus* 90 (77–95); p=0.04).

The evolution of the main studied variables is shown in table 2. After 30 min of WT, both patients who succeed and patients who failed the overall weaning process showed a significant increase in respiratory rate and heart rate, with no other changes in their haemodynamic, respiratory and oxymetric parameters.

StO_2 variables

No differences were observed in baseline StO_2 parameters between the two groups (table 2). When analysing the evolution of NIRS-derived parameters, patients who succeeded and patients who failed showed different patterns. The weaning success group showed no significant changes in steady-state StO_2, DeO_2, ReO_2 and $nirV'O_2$ after 30 min of WT, whereas the weaning failure group showed a significant decrease in their DeO_2 slope (from -11.2 (-16.9– -7)%·min^{-1} to -13.7 (-18.7– -7.9)%·min^{-1}; p=0.04) (fig. 2), as well as an increase in $nirV'O_2$ (from 92 (76–146) to 141 (93–212) U; p=0.04) after 30 min of WT. Changes in local haemoglobin content (THI) were not different between the success and failure groups (from 9.9 (8.1–11.9) U

TABLE 1 Patient baseline characteristics prior to the weaning trial

	All patients	Weaning success	Weaning failure	p-value#
Subjects n	37	24	13	
Age years	71 (56–79)	71 (58–79)	73 (48–79)	1
Sex male/female	24/13	16/8	8/5	0.7
Pre-existent comorbidities				
Sleep apnoea	4 (11)	4 (17)	0 (0)	0.6
COPD	10 (27)	7 (29)	3 (15)	
Coronary disease	10 (27)	6 (25)	4 (31)	
CHF	11 (30)	7 (29)	4 (31)	
No previous disease	15 (41)	8 (33)	3 (15)	
Aetiology of ARF				
Septic shock	20 (54)	12 (50)	8 (62)	0.6
Acute heart failure	7 (19)	5 (20)	2 (9)	
Trauma	5 (14)	4 (17)	1 (8)	
Other	5 (14)	3 (13)	2 (15)	
Time on MV days	6 (4–10)	5 (4–10)	6 (4–11)	0.5
Heart rate beats·min^{-1}	88 (74–95)	90 (77–95)	78 (71–91)	0.04
Systolic BP mmHg	126 (113–137)	125 (117–137)	127 (109–145)	0.9
mAP mmHg	80 (71–89)	80 (76–89)	80 (69–88)	0.6
Respiratory rate breaths·min^{-1}	18 (17–21)	18 (16–21)	19 (17–23)	0.1
$P_{a}O_2/F_{I}O_2$	262 (229–341)	276 (240–317)	243 (209–380)	0.8
$S_{p}O_2$ %	97 (95–98)	97 (95–98)	97 (95–98)	0.2
Tidal volume mL	460 (390–500)	467 (385–500)	440 (400–502)	0.7
PEEP cmH_2O	5 (5–5)	5 (5–5)	5 (5–6)	0.2
$F_{I}O_2$ %	30 (28–35)	30 (30–35)	30 (28–32)	0.2
pH	7.48 (7.42–7.49)	7.47 (7.41–7.5)	7.48 (7.44–7.5)	0.7
$P_{C}O_2$ Torr	35 (30–39)	35 (29–39)	36 (32–40)	0.5
Base deficit mmol·L^{-1}	2.6 (-2.3–5)	2.1 (-2.5–5)	3.9 (-1.3–5.4)	0.5
Haemoglobin mg·dL^{-1}	9.1 (8.4–10.1)	8.9 (8.3–9.9)	9.2 (8.7–11.4)	0.3
$S_{cv}O_2$ %	65 (55–71)	66 (51–71)	61 (57–76)	1

Data are presented as median (interquartile range), n/n or n (%), unless otherwise stated. COPD: chronic obstructive pulmonary disease; CHF: congestive heart failure; ARF: acute respiratory failure; MV: mechanical ventilation; BP: blood pressure; mAP: mean arterial pressure; $P_{a}O_2$: arterial oxygen tension; $F_{I}O_2$: inspiratory oxygen fraction; $S_{p}O_2$: arterial oxygen saturation measured by pulse oximetry; PEEP: positive end-expiratory pressure; $P_{C}O_2$: carbon dioxide tension; $S_{cv}O_2$: central venous oxygen saturation. #: between weaning success and failure.

to 10.5 (8.5–12.9) U, $p<0.01$, in the success group; and from 10.1 (7.8–11.1) U to 10.4 (7.7–13) U, $p=0.03$, in the failure group).

The DeO_2 ratio (represented as the ratio of DeO_2 at 30 min to baseline DeO_2) was significantly higher in the failure group (1.03 (0.92–1.22) in the success group *versus* 1.27 (1.12–1.49) in the failure group; $p<0.01$) (fig. 3). In addition, the nirVO_2 ratio (the increase in nirVO_2 during the WT, represented as the ratio of nirVO_2 at 30 min to baseline nirVO_2) was also higher in the failure group (1.1 (0.8–1.3) in the success group *versus* 1.24 (1.21–1.66) in the failure group; $p=0.02$).

Discussion

The main observation of the present study is that higher relative increases in StO_2, DeO_2 rate and local skeletal muscle oxygen consumption (nir$V'O_2$) after a 30 min WT are associated with failure to wean from MV.

Changing from positive-pressure ventilation to spontaneous breathing determines an increase in the work of breathing and, thus, an increase in the oxygen demand of the respiratory muscles. If the metabolic demand of transitioning from MV to spontaneous ventilation cannot be met by increasing cardiac output, either because cardiovascular reserve is limited, the work cost of breathing excessive, or both, then the cardiovascular system addresses these excessive demands by increasing sympathetic tone, which tries to maximise cardiac output [21], while redistributing blood flow away from the periphery and splanchnic circulation to the respiratory muscles [22, 23]. Such increased sympathetic activity also increases tissue metabolic rate. Accordingly, blood flow redistribution and increased tissue oxygen consumption could coexist in patients during a failed WT.

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TABLE 2 Characteristics at baseline and at 30 min of weaning trial

	Weaning success		Weaning failure	
	Baseline	30 min	Baseline	30 min[#]
Subjects	24		13	
Heart rate beats·min^{-1}	90 (77–95)	95 (81–100)*	78 (71–91)[¶]	91 (85–98)*
Systolic BP mmHg	125 (117–137)	127 (119–141)	127 (109–145)	120 (113–157)
mAP mmHg	80 (76–89)	81 (76–89)	80 (69–88)	83 (72–94)
Respiratory rate breaths·min^{-1}	18 (16–21)	24 (17–27)*	19 (17–23)	35 (28–36)*
S_{PO_2} %	97 (95–98)	96 (94–98)	97 (95–98)	95 (90–96)
Tidal volume mL	467 (385–500)	450 (370–540)	440 (400–502)	285 (237–326)*,[¶]
pH	7.47 (7.41–7.5)	7.45 (7.39–7.48)	7.48 (7.44–7.5)	7.45 (7.4–7.47)
P_{CO_2} Torr	35 (29–39)	37 (30–41)	36 (32–40)	39 (34–43)
Base deficit mmol·L^{-1}	2.1 (-2.5–5)	1.1 (-1.5–3.6)	3.9 (-1.3–5.4)	3 (-2.1–4.8)
S_{tO_2} %	79 (75–84)	81 (76–84)	77 (73–83)	75 (72–88)
THI U	9.9 (8.1–11.9)	10.5 (8.5–12.9)*	10.1 (7.8–11.1)	10.4 (7.7–13)*
DeO_2 %·min^{-1}	-12.4 (-16.4– -9.8)	-12.7 (-16.5– -10.5)	-11.2 (-16.9– -7)	-13.7 (-18.7– -7.9)*,[¶]
ReO_2 %·min^{-1}	185 (144–252)	205 (137–293)	217 (181–309)	261 (222–365)
nir$V'O_2$ U	109 (90–158)	114 (66–168)	92 (76–146)	141 (93–212)*
Respiratory rate/V_T breaths·min^{-1}·L^{-1}		49 (33–68)		**133 (85–169)**[¶]
S_{tO_2} ratio		1.0 (0.96–1.05)		0.99 (0.97–1.03)
DeO_2 ratio		1.03 (0.92–1.22)		**1.27 (1.12–1.49)**[¶]
ReO_2 ratio		1.06 (0.88–1.25)		1.08 (0.92–1.7)
THI ratio		1.05 (1.02–1.19)		1.04 (0.98–1.07)
nir$V'O_2$ ratio		1.1 (0.8–1.3)		**1.24 (1.21–1.66)**[¶]

Data are presented as n or median (interquartile range). BP: blood pressure; mAP: mean arterial pressure; S_{PO_2}: arterial oxygen saturation measured by pulse oximetry; P_{CO_2}: arterial carbon dioxide tension; S_{tO_2}: thenar tissue oxygen saturation; THI: tissue haemoglobin index; DeO_2: deoxygenation slope; ReO_2: reoxygenation slope; nir$V'O_2$: thenar muscle oxygen consumption; V_T: tidal volume; S_{tO_2} ratio: S_{tO_2} at 30 min/S_{tO_2} at baseline; DeO_2 ratio: DeO_2 at 30 min/DeO_2 baseline; ReO_2 ratio: ReO_2 at 30 min/ReO_2 baseline; THI ratio: THI at 30 min/THI baseline; nir$V'O_2$ ratio: nir$V'O_2$ at 30 min/nir$V'O_2$ baseline. [#]: n=11, as explained in fig. 2b. *: $p<0.05$ *versus* baseline value; [¶]: $p<0.05$ *versus* weaning success at the same time point. p-values in bold represent statistical significance.

Assessing blood flow redistribution in the splanchnic bed *via* monitoring splanchnic tissue hypoxia has proven to be a useful tool to predict weaning outcome [9, 11]. Despite some initial promising results [8–11], gastric tonometry was not evaluated in large studies. Furthermore, this technique did not become a routine standard of care due to its technological requirements and difficult application at the bedside. As occurs with the splanchnic area, noninvasive monitoring of the peripheral circulation might add potential benefits to cardiovascular performance assessment. In our study, we hypothesised that presumed cardiovascular overload of excessive cost of breathing would be detected by noninvasive regional evaluation of skeletal muscle oxygenation using NIRS technology.

As we already expected, we did not observe significant changes in S_{tO_2} during the spontaneous breathing trial in any of the two groups. Although steady-state S_{tO_2} has been proposed as a marker of hypoperfusion states, especially in haemorrhagic shock [24], our group and others have already demonstrated the lack of sensitivity of the absolute S_{tO_2} value [15, 16, 25], suggesting that absolute steady-state S_{tO_2} might not detect less severe degrees of tissue hypoperfusion. We presumed that during the WT there would not be enough blood flow redistribution to be detected by static NIRS measurements.

However, dynamic NIRS-derived variables created by an ischaemic challenge, DeO_2 and ReO_2, provide more information about tissue wellness, exploring the local metabolic rate and the endothelial integrity, respectively. These novel parameters have been studied mostly in trauma and septic-shock patients, were they have consistently shown prognostic implications, independently from other cardiovascular parameters [26, 27].

In our set of patients, the relative increase in DeO_2 during a 30 min WT was associated with weaning failure. DeO_2 is a dynamic parameter that depends on the local O_2 supply–demand relationship and mirrors local oxygen utilisation. Increases in DeO_2 might be explained by two different, and cumulative, mechanisms: 1) by a local supply–demand dependency in low or inadequate flow states, such as blood flow redistribution; and 2) by an increased metabolic rate. During spontaneous breathing, if there is an increase in respiratory muscle demand, leading to sympathetically mediated peripheral vasoconstriction and blood

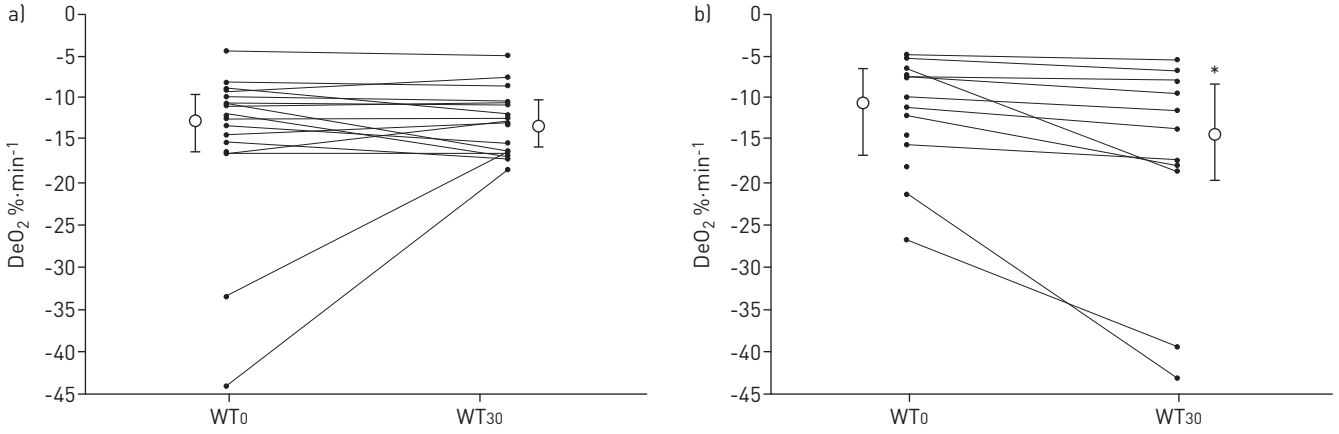

FIGURE 2 Deoxygenation (DeO_2) slope at baseline weaning trial (WT0) and 30 min after beginning the WT (WT30) for patients who a) succeeded or b) failed the overall WT. DeO_2 values for each patient (closed circles), and the median (interquartile range) value of DeO_2 at baseline and after 30 mins of WT (open circles) are shown. Two patients in the weaning failure group (shown in b) did not complete the WT, requiring reinstitution of mechanical ventilation, because of increased respiratory efforts, sweating and arterial oxygen desaturation. *: $p<0.05$, when comparing paired DeO_2 at 30 min and baseline DeO_2.

flow redistribution, inadequate local flow could lead to increased local oxygen extraction. In such cases, in addition to higher DeO_2 rates, one would expect to see reduced THI values in the failure group, as compared with the successfully weaned patients, reflecting lower tissue haemoglobin content in blood flow redistribution conditions. Indeed, BARTELS *et al.* [17] demonstrated that thenar eminence THI decreased significantly during blood flow redistribution induced by simulated hypovolaemia (*i.e.* lower body negative pressure). In our study, however, we did not observe any differences in THI behaviour during the WT between the two groups. Although, as occurs with absolute StO_2, THI might be not sensitive enough to detect mild decreases in local blood flow, during the spontaneous breathing trial, both groups showed a THI increase suggesting, if any, an increase in cardiac output in response to the reinitiation of spontaneous ventilation. Therefore, the observed increase in DeO_2 in the failure group might be mostly related to local oxygen consumption. This hypothesis was also supported by nir$V'O_2$ evolution, an estimation of local oxygen consumption that corrects DeO_2 for the local haemoglobin content [20]. This finding might reflect increased sympathetic outflow trying to compensate for the inadequate cardiovascular response, as supported by other studies that showed a significant increase in plasma catecholamine levels during WT, especially in failure to wean patients [28–30].

In summary, in the present observational study, higher DeO_2 and nir$V'O_2$ during WT were associated with weaning failure. Our findings suggest that these functional parameters might detect increased local oxygen consumption secondary to increased sympathetic tone in failure to wean patients.

Study limitations

Some limitations of the present study should be acknowledged. First, the study was carried out in a single centre. Although we expect that similar patients should behave similarly, weaning approaches may vary

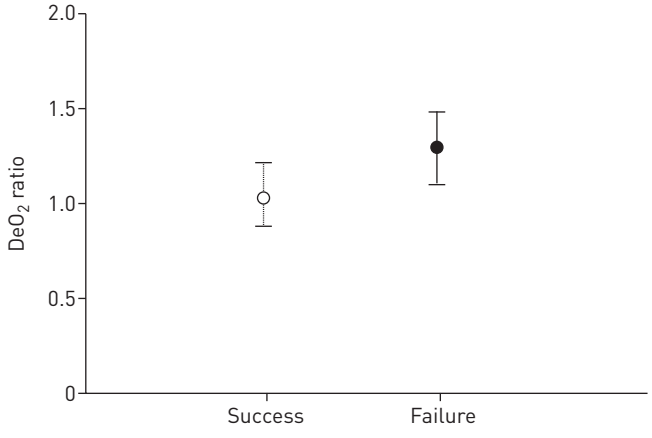

FIGURE 3 Deoxygenation ratio (DeO_2) for weaning success and failure. DeO_2 for the weaning success and failure groups, represented as median (interquartile range), were statistically different with $p<0.01$.

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across centres, thereby degrading the predictive value of these StO_2-derived parameters. Thus, this study needs to be duplicated across other centres. Secondly, our pilot study had a small sample size, and only 13 patients presented weaning failure (with only two extubation failures). It would be especially interesting to evaluate the predictive utility of these parameters in preventing extubation failure, which could not be addressed in our pilot study. Clearly, a larger study would allow refined investigation into the interactions of processes and calibration of the predictive parameters that were primarily identified in this study. Thirdly, we did not determine the cause of weaning failure in patients who were considered to fail. We merely identified that they did fail. We can expect different behaviours of StO_2 parameters in patients who fail because of limited cardiovascular reserve from patients who fail because of upper airway obstruction and/or impaired secretions management. This issue must be taken into account in future studies exploring weaning failure.

Conclusions

Relative changes in StO_2 VOT-derived DeO_2 slopes and local oxygen consumption after 30 min of a WT were associated with weaning outcome. Thus, StO_2 changes derived from a VOT might be a useful clinical tool to predict weaning outcome.

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