CARDIAC CHANGES DURING VEANING OF MECHANICALLY VENTILATED CHRONIC OBSTRUCTIVE PULMONARY DISEASED (COPD) PAT IENTS

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ABSTRACT

Introduction: Weaning trials are performed to assess the readiness of mechanically ventilated patients to sustain spontaneous ventilation. Spontaneous breathing increases basal metabolism and weaning increases cardiopulmonary demand. The most critical time for patients with chronic obstructive pulmonary disease (COPD) mechanically ventilated for acute respiratory failure is the weaning period . Myocardial ischemia is potentially the most important cause of reduced contractility during weaning. This myocardial ischemia might occur as a consequence either of a reduction in myocardial oxygen supply or of an increase in myocardial oxygen demand or both. A reduced myocardial oxygen supply may result

from weaning induced hypoxaemia and tachycardia.

Aim of the work: The aim of this work was to monitor the cardiac changes during weaning of COPD patients by non invasive methods in an attempt to detect occult lethal events.

Patients and Methods:

This prospective study included 40 male COPD patients mechanically ventilated for acute on top of chronic respiratory failure, with mean age of 61.45±9.17 years. Four modes of weanin were used random-ly:sychronized intermittent mandatory ventilation SIMV) mode (11 patients), pressure support ventilation (PSV) mode (11patients), continous positive airway pressure (CPAP) mode (9 pa-

tients) and T piece mode (9 patients). All patients were subjected to plain chest X-ray, serum electrolytes and myocardial enzymes and Holter ECG monitoring during weaning and for 24 hour after disconnection of the patient from the ventilator(within one week after weaning). Monitoring of heart rate, systolic, diastolic and mean pressure and heart ratesystolic blood pressure product (RPP) during the weaning peroid and for 24 hour after patient disconnection from ventilator (within one week after Thirty three patients had weaning). successful outcome of weaning and seven patients died during weaning.

Results: There was a statistically significant increase in heart rate (HR) and heart ratesystolic blood pressure product (RPP) during weaning compared to that at post weaning. Also, there was a statistically significant increase in cardiac enzymes during weaning compared to that at post weaning. The results of twenty four hour continuous ECG monitoring (Holter's monitor) showed a statistically significant increase in ischemic-ST-segment depression, ischemic-ST-segment elevation and tachycardic episodes during weaning compared to that at post weaning. On the other hand, there was no difference of statistical significance in rhythm changes during weaning compared to that at post weaning.

Conclusions: Weaning process has a stressful effect on the cardiovascular system.

INTRODUCTION

Weaning trials are performed to assess the readiness of mechanically ventilated patients to sustain spontaneous ventilation (1). Insofar, as spontaneous breathing increases basal metabolism (oxygen consumption [VO2]) compared to that on full mechanical ventilatory support, weaning increases cardiopulmonary demand (2). The most critical time for patients with chronic obstructive pulmonary disease (COPD) mechanically ventilated for acute respiratory failure is the weaning period (3). Myocardial ischemia is potentially the most important cause of reduced contractility during weaning. Thus, myocardial ischemia might occur as a consequence either of a reduction in myocardial oxygen supply or of an increase in myocardial oxygen demand or both (4). A reduced myocardial oxygen supply may result from weaning induced hypoxaemia and tachycardia (5).

Aim Of The Work:

The aim of this work was to monitor the cardiac changes during weaning of COPD patients by non invasive methods in an attempt to detect occult lethal events.

PATIENTS AND METHODS

This prospective study was carried out at ICU of Mansoura Emergency Hospital, and included 40 male COPD patients mechanically ventilated for acute on top of chronic respiratory failure with mean age of 61.45 ±9.17 years. Four modes of weaning were used randomly:-

- Synchronized intermittent mandatory ventilation (SIMV) mode (11 patients)
- pressure support ventilation (PSV) mode (11patients),
- continous positive airway pressure (CPAP) mode (9 patients)
- 4. T piece mode (9 patients).

Thirty three patients had successful outcome of weaning and seven patients died during weaning.

Patients were excluded from this study when there is one or more of

the following:

- 1. Ischaemic heart disease.
- 2. Systemic hypertension.
- 3. Rheumatic heart disease.
- 4. Cardiomyopathies.
- 5. Renal impairement
- 6. Diabetes mellitus.
- Respiratory failure from causes other than COPD e.g. head trauma, cerebrovascular disease....etc.

All patients were subjected to:

- a. Thorough history taking and clinical examination.
- b. Investigations: CBC, SGOT, SGPT, cardiac enzyme (total CPK, CPK MB, LDH), serum bilirubin, arterial blood gases (ABGs), serum electrolytes, ECG and chest-X ray.
- c. Twenty four hour continuous ECG monitoring (Holter's monitor) during weaning and for 24 hour after disconnection of the patient from the ventilator (within one week after weaning).
- d. Heart rate, blood pressure (systolic, diastolic and mean BP which equal 2 X(diastolic + systolic)/3) and heart rate- systolic blood pressure product (RPP) were monitored every hour during the period of weaning and for 24 hour after disconnection from ventilator (within one week after weaning). The RPP increases with

myocardial work and is an index of myocardial perfusion requirement .(6) Calculations of the mean values were done.

RESULTS

This prospective study included 40 male COPD patients with mean age 61.45 ±9.17 years (table 1&2).

Complications developed in 5 patients (12.5%) during mechanical ventilation (2 patients (5%) developed pneumothorax, one patient (2.5%) developed right lower lobe pneumonia and 2 patients (5%) developed gastrointestinal bleeding). (table 2).

The results of haemodynamic monitoring data showed a statistically significant increase in heart rate (HR) and heart rate -systolic blood pressure product (RPP) during weaning compared to that at post weaning. But, no difference of statistical significance in systolic, diastolic and mean blood pressure during weaning

compared to that at post weaning (table 3).

Our results showed a highly statistically significant increase in total CPK, CPK MB and a LDH during weaning compared to that at post weaning (table 4).

The results of twenty four hour continuous ECG monitoring (Holter's monitor) showed a statistically significant increase in ST-segment depression, ST-segment elevation and tachycardic episodes during weaning compared to that of post weaning. (table 5). On the other hand, there was no difference of statistical significance in rhythm changes during weaning compared to that at post weaning (table 6).

The results of serum electrolytes showed no difference of statistical significance in serum sodium, serum potassium and serum HCO3 during weaning compared to that at post weaning (table 7).

Table (1): Age and sex distribution of the studied patients .

	Age distribution				Sex distribution			
No	is the	The District of the state of		Males		Females		
	Minimum	Maximum	Mean ± SD	No.	%	No.	%	
40	45	81	61.45±917	40	100	0	0	

Table (2): Clinical data of the studied patients .

	No.	%
Presenting clinical data:		
•Cyanosis	40	100
Disturbed level of consciousness	- 11	27.5
•Coma	- 29	72.5
Oedema LL	12	30
Congested neck veins	22	55
Tachypnea	40	100
• Tachycardia	28	70
• Chools	1 0	15
•Smoking habit	40	100
Complications during mechanical ventilation:		
• Pneumothorax	2	5
Right lower lobe pneumonia (VAP).	1	2.5
• Hematemesis	1	2.5
•Melena	1	2.5
Total	5	12.5
Therapy		
Theophylline	40	100
Digitalis	22	55
B2 stimulant	24	60-
Steroids	12	30
Antibiotics	40	100

Table (3): Haemodynamic monitoring data at the onset of ventilation and during weaning versus post weaning in all studied patients.

	H.R. beat/min M±SD	SBP mm Hg M±SD	DBP mm Hg M±SD	MBP mm Hg M±SD	RPP mmHgb/m inx10 ⁻³ M±SD
Post weaning No. = 33	91.15 ± 9.5	118.24 ±10.62	68.66 ± 8.36	83.72 ± 7.15	10.79 ± 1.65
During weaning No. = 40	102.1 ±13.41 P<0.001	118.32 ±10.95 P>0.05	66.95 ± 10.57 P>0.05	85.25 ± 9.44 P>0.05	12.09 ± 2.04 P<0.05

H.R. = Heart rate. SBP = Systolic blood pressure. DBP = Diastolic blood pressure. MBP = Mean blood pressure. RPP = Heart rate -systolic blood pressure product.

Table (4): Total CPK, CPKMB and LDH at the onset of ventilation and during weaning versus post weaning in all studied patients.

	Total CPK	CPKMB	LDH
	(I.U/L.)	(I.U./L.)	(I.U./L.)
	M ± SEM	M±SEM	M ± SEM
Post weaning No. = 33	117.6 ± 19.9	13.09 ± 4.35	307.82 ± 23.39
During weaning	296 ± 43.86	32.11 ± 4.26	374.7 ± 18.09
No. = 40	P<0.001	P<0.001	P<0.05

Table (5): ST-segment deviation and tachycardic episodes /24 hour during weaning versus post weaning in all studied patients.

C.2008.00	ST-segment depression episodes/24 hour M± SEM	ST- segment elevation episodes/24 hour M± SEM	Tachycardic episodes /24 hour M± SEM
Post weaning No. = 33	.87 ± .53	6.33 ± 1.27	3.72 ± .92
During weaning No. = 40	4.53 ± 1.09 P<0.05	14.23 ± 2.46 P<0.05	12.65 ± 3.45 P<0.05

Table (6): Premature atrial and ventricular beats, atypical run episodes, pauses and couplets episodes / hour during weaning versus post weaning in all studied patients.

	Premature atrial beats M± SEM	PVBs M± SEM	Atypical run episodes M± SEM	Pauses M± SEM	Couplets episodes M± SEM
Post weaning No. = 33	2.45 ± 1.07	7.6 ± 1.89	1.54 ± .43	.15 ± .088	1.3 ± .55
During weaning No. = 40	2.87 ± 1.1 P>0.05	12.4 ± 3.5 P>0.05	2.2 ± .79 P>0.05	.27 ± .1 P>0.05	4.42 ± 1.97 P>0.05

PVBs = premature ventricular beats.

Table (7): Serum electrolytes during weaning versus post weaning in all patients.

the singula	Serum sodium (Meq/L) M± SEM	Serum potassium (Meg:L) M± SEM	Serum HCO3 (Meq/L) M± SEM
Post weaning No. = 33	128.13 ± 2.81	4.83 ± .27	30.25 ± 3.4
During weaning No. = 40	128.78 ± 2.81 P>0.05	4.98 ± .26 P>0.05	29.98 ± 4.02 P>0.05

DISCUSSION

Patients are weaned from mechanical ventilation when the underlying disease improves, and extubated when they are assessed to be capable of sustaining spontaneous ventilation after mechanical ventilation is discontinued.(7) The resumption of spontaneous breathing after mechanical ventilation imposes several loads on the cardiorespiratory system including: increase venous return thus increasing ventricular preload (8,9), increase left ventricular afterload (9,10) and increases total body oxygen consumption by roughly about 15 to 20%.(3,4) Additionally, anxiety associated with weaning / resistive breathing through the endotracheal tube may increase catecholamins, thus further increasing myocardial work (afterload, preload, chronotropy, and inotropy. (11)

The most critical time for patients with chronic obstructive pulmonary disease (COPD) mechanically ventilated for acute respiratory failure is the weaning period .(2)

Myocardial ischemia is potentially the most important cause of reduced contractility during weaning. This myocardial ischemia might occur as a

consequence either of a reduction in myocardial oxygen supply or of an increase in myocardial oxygen demand or both(12). In COPD patients the onset of myocardial ischemia during weaning was suggested by the concomitant presence of acute left ventricular dysfunction and left entricular regional wall motion abnormalities detected during angioscintigraphic examination.(11) Cardiac arrhythmias are common in patients with respiratory failure from chronic obstructive pulmonary disease .(13).

The aim of this work was to recintor the cardiac changes during weaning of COPD patients by non invasive methods in an attempt to detect occult lethal events.

This prospective study included 40 COPD male patients with mean age 61.45 ±9.17 years. They were divided into four groups according to the mode of weaning: SIMV group (11 patients), PSV group (11patients), Tpiece group (9 patients) and CPAP group (9 patients).

Clinical data:

In 1995, The American Thoracic Society,(14) reported that major risks associated with assisted positive pressure ventilation include ventilator associated pneumonia (VAP), pulmonary barotrauma, and laryngotracheal complications associated with intubation and / or tracheostomy.

The incidence of pneumothorax in the present study was 5 %. This is in accordance to Peters & Sako, 1998 (15) who reported that the overall incidence of pneumothorax during mechanical ventilation is about 5 percent and the incidence increased if patients have chronic pulmonary disease

In this study the incidence of VAP was 2.5 %. Langer & associates, 1987 (16) reported that the incidence of pneumonia in mechanically ventilated patients rose from (5%) in patients receiving one day of respiratory assistance to (68.8%) in patients mechanically ventilated for more than 30 days. Trouillet & associates , 1998 (17) reported that the incidence of VAP is high , ranging from 7 % to more than 40 % .

Similarities and differences in pulmonary complications between our study and the above mentioned studies might be attributed to differences in duration of ventilation, severity of

the disease, mode of ventilation and mode of weaning in every study.

The incidence of gastrointestinal bleeding in this study was 5 %. This is in accordance to MacNee, 2000 (18) who had reported that the incidence of gastrointestinal bleeding appears to be lower, around 9%, in patients with COPD. Also, Susan, 1994 reported that clinically significant bleeding occured less frequently in 8% of mechanically ventilated patients with hypotension or sepsis. (19).

Hurford & & associates., 1991 (20) reported that the physiologic changes associated with the transfer from mechanical ventilation (MV) to spontanous ventilation (SV) were relatively minor. Heart rate increased 6% diastolic and mean arterial pressure increased 14% and 9%, respectively, whereas systolic pressure did not change.(20)

In this study, there was a significant increase in heart rate at the onset of ventilation and during weaning compared to that at post weaning. But, no significant changes in SBP, DBP and mean BP during weaning compared to that at post weaning (Table 3). This is in agreement with

Richard & associates ., 1994(21) who had demonstrated that weaning was associated with a significant increase in HR (P<05) without significant change in SBP during weaning of patients with COPD. Also, Gilbert & associates , 1974(22) noted an increase in heart rate to occur within 5 minutes after patients were placed on spontaneous ventilation. Also, Lemaire & associates , 1988(11) found that after 10 minutes of spontaneous ventilation there was a significant increase in heart rate and systemic arterial blood pressure.

On the other hand, Teboul and Richard, 1991(12) reported that catecholamine release related to emotional stress induced by disconnection from the ventilator and / or hypercapnia during spontaneous ventilation may elevate SBP.

Changes in the heart rate in this study may be explained according to Sahn & associates., 1976(23) who reported that on disconnection of COPD patients from the ventilator, these patients can rapidly develop evidence of sympathetic nervous system hyperactivity (tachycardia, systemic hypertension, sweating) (23).

Chatila & associates., 1996(1) reported that the resumption of spontaneous breathing increased myocardial demand, signaled by a 12% increase in the heart rate -systolic blood pressure product (RPP). The 12% increase in the RPP suggests that myocardial work roughly tracked the projected increase in total body oxygen consumption related to weaning. The increase in RPP tended to be associated with ischemia during weaning.

The results of the present study revealed significant increase in RPP at the onset of ventilation and during weaning compared to that at post weaning (Table3). This finding could be explained according to Srivastava & associates , 1999(24) who had demonstrated that RPP for all the studied patients increased significantly during weaning from $11.9 \pm .4$ to $13.5 \pm .5$ mm Hg x beats / min x 103.

This result signifies that RPP is a good noninvasive parameter for monitoring cardiac stress during mechanical ventilation.

* Cardiac enzymes data :

Myocardial infarction causes intracellular enzymes to escape into the blood stream, where increased concentrations can be detected and so aid the diagnosis. Most laboratories measure creatine kinase (CPK) and its cardio-specific isoenzyme (MB), SGOT and lactic dehydrogenase (LDH). It is important to recognize that non of these enzymes with exception of CPK-MB, is specific to the heart (25).

The results of the present study revealed a statistically significant increase in CPK, CPKMB and LDH at the onset of ventilation and during weaning compared to that at post weaning (table 4).

Up to our knowledge, no available literatures concerning the changes in serum myocardial enzymes during weaning of COPD patients.

The increase in CPKMB in our studied patients indicates myocardial stress during weaning of COPD patients. It could be considered of value in monitoring cardiac status during mechanical ventilation.

* Holter's monitoring data :

The results of long term 24 hour continuous ECG monitoring detected by Holter's monitor revealed a signifi-

cant increase of ischemic STsegment elevation and depression episodes / 24 hour during weaning compared to that at post weaning (Table 5). This is in accordance to Sakr, 1999(26) who had demonstrated that there was a statistically significant increase of ST- segment depression episodes detected by Holter's monitor in patients with respiratory failure from COPD. Also. Chatila& associates ., 1996(1) had demonstrated that 6 out of 93 patients (6.5%) experienced ischemia during weaning of their studied medically ill patients from mechanical ventilation (MV). However, ischemia was detected more frequently in (10%) of patients with a precedent history of coronary artery disease (CAD). Similarly, Rasanen & associates, 1984(27) in a study of acute myocardial infarction complicated by respiratory failure had demonstrated that 6 out of 12 patients experienced ischemia during weaning from MV. Also, Hurford & associates, 1991(20) had demonstrated that 7 out of 15 MV- patients developed acute regional alterations of myocardial perfusion, as detected by quantitative thallium 201 myocardial scintigraphy, when breath spontaneously despite unchanged ECGs (20). Abalos & associates, 1992(28) in a study of myocardial ischemia during the weaning period in patients with acute respiratory failure and CAD had demonstrated that 5 out of 62 patients had ECG changes of acute ischemia during weaning from MV after noncardiac surgery. Also, Srivastava & associates, 1999(24) had demonstrated that 8 out of 83 patients showed electrocardiographic evidence of ischemia during weaning.

On the other hand, Lemaire& associates, 1988(11) found a relatively low incidence of weaning induced ischemia events to explain the onset of left ventricular dysfunction in 15 patients with COPD and pre-existing heart disease.

The results of the present study revealed a statistically significant increase in tachycardic episodes / 24 hour, detected by Holter's monitor, during weaning compared to that at post weaning (Table 5). This is in accordance to Hurford & associates , 1991(20) who had demonstrated that hemodynamic and ventilatory changes associated with discontinuing MV were sufficient to increase myocardial oxygen demand (evidenced by the increased heart rate, arterial blood pressure and left ventricular cavity

size during spontaneous ventilation). Also, Richard& associates ., 1994(21) had demonstrated that weaning was associated with a significant increase in HR.

The results of the present study revealed insignificant increase in premature atrial beats, (PAB), premature ventricular beats (PVB), atypical run episodes, pauses and couplets episodes / hour detected by Holter's monitor during weaning compared to that at post weaning (Table 6). This is in accordance to Sakr, 1999 (26)who also found, in his study, insignificant association between PVBs and PABs and respiratory failure from COPD. Yet, incidence percentage of PVBs (62.2%) and PABs (93.5%) were higher in his studied patients with respiratory failure than patients without respiratory failure (26).

Incalzi & associates, 1990(29), in a study of cardiac arrhythmias and left ventricular function in respiratory failure from chronic obstructive pulmonary disease, had demonstrated that ventricular arrhythmias are quite common in patients affected by respiratory failure and COPD. Also, Shih & & associates, 1988(30) reported a correlation between PaCO2 and ventric-

ular arrhythmias in patients affected by COPD.

From this study we conclude the following:

- 1. Weaning process has a stressful effect on the cardiovascular system as evidenced by increased heart rate, heart rate-systolic blood pressure product, elevated cardiac enzymes and increased ST- segment depression and elevation episodes.
- 2. Occult cardiac ischemia should be considered during weaning of COPD patients from mechanical ventalation. Continuous ST-segment monitoring by Holter's monitor and serial cardiac enzymes determination may be useful in detecting occult ischemia in selected patients.
- 3. Non invasive monitoring such as heart rate-systolic blood pressure product and continous ECG monitoring may be useful in detecting occult lethal events, minimizing the risk of ventilator induced complications and determining the patient's readiness for weaning.

From this study we recommend the following:

 It is important to consider the diagnosis of myocardial ischemia during weaning of mechanically ventilat-

ed COPD patients.

- Further studies investigating the effect of anti-ischemics on the outcome of weaning specially in those who experienced difficulty to be liberated from mechanical ventilation.
- Strict monitoring of cardiac changes during weaning of mechanically ventilated COPD patients should be done for prevention of occurrence of lethal events.

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التغيرات القلبية في مرضى السدة الرئوية المزمنة أثناء الفطام من التغيرات القلبية في مرضى الصناعي

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أقسام الأمراض الصدزرية والأشعة والباطنة العامة - كلية طب المنصورة

- يتم عمل الفطام من التنفس الصناعى لمعرفة مدى إستجابة المريض المعتمد على جهاز التهوية الميكانيكية للاعتماد على نفسه بعملية التهوية الذاتية .
- مع بداية التنفس الذاتى يزيد إحتياج الجسم للأكسچين نتيجة زيادة عمليات الأيض، وهذا يزيد العب، على القلب لزيادة ضخ الدم لمواكبة إحتياج الجسم ولذلك فإن أخطر مرحلة يمر بها المريض المعتمد على جهاز التهوية الميكانيكية هى مرحلة الفطام مما قد يؤدى إلى قصور أداء القلب وضخ الدم أثناء عملية الفطام.
- كان الهدف من هذه الدراسة هى متابعة التغيرات القلبية أثناء فطام مرضى السدة الشعبية المزمنة من جهاز التهوية الميكانيكية بطرق غير نفاذية لمعرفة بعض تغيرات أو تأثيرات بالقلب تمكن أن تكون مهمة للمريض.
- إشتملت هذه الدراسة على عدد ٤٠ مريضاً بالسدة الشعبية المزمنة على جهاز التهوية المبكانيكية لعلاج الفشل التنفسي الحاد وكان متوسط العمر ٢١ر٦٥ عاما.
 - تم تقسيم المرضى عشوائياً إلى أربعة مجموعات حسب طريقة الفطام المستخدم :
 - ١- التهوية المنقطعة الحتميه المتوافقة (١١ مريضاً)
 - ٧- التهوية باستخدام الضغط المساعد (١١ مريضاً).
 - ٣- والفطام بإستخدام أنبوبة T (٩ مرضى).
 - ٤- الفطام باستخدام الضغط الإيجابي المستمر (٩ مرضي) .

وقد تم عمل الآتى لكل مريض: أشعة على الصدر، الكترولينات بالدم، انزيمات القلب، ورسم قلب متواصل لمدة ٢٤ ساعة (هولتر) أثناء الفطام وبعده (خلال الأسبوع الأول من الفطام)، وقد تم متابعة النبض وضغط الدم، حاصل ضرب سرعة النبض في الضغط الانقباضي لكل مريض أثناء وبعد الفطام وقد تم فطام ٣٣ مريضا بنجاح وتوفى سبعة مرضى أثناء الفطام.

أظهرت النتائج مايلي :

- زيادة في سرعة النبض وحاصل ضرب النبض في الضغط الانقباضي أثناء فترة الفطام مقارنة ببعد الفطام .
- زياده ذات دلالة إحصائية في نسبة إنزيمات القلب (إنزيم الكرياتين فوسفوكايناز الكلى والجزء الخاص بالقلب وانزيم اللاكتيت داى هيدروجيناز) أثناء فترة الفطام عنه بعد الفطام.
- زيادة ذات دلالة إحصائية في إنخفاض الفاصل ST ، وكذلك إرتفاع الفاصل ST ونوبات من زيادة ضربات القلب أثناء فترة الفطام عنه بعد الفطام.

الاستنتاج:

عملية الفطام من التنفس الصناعى لها تأثير ضار على القلب ولابد من متابعة حالة القلب وذلك عن طريق المتابعة المتواصلة لرسم القلب (المونيتور) وكذلك تكرار عمل تحاليل أنزيمات القلب أثناء فترة الفطام ولمدة أسبوع بعد فصل المريض من جهاز التهوية الميكانيكية .

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