Zagazig University Medical Journal
www.zumj.journals.ekb.eg

https://doi.org/10.21608/zumj.2024.234154.2873 Volume 30, Issue 1.6, September 2024, Supplement Issue
Manuscript ID:ZUMJ-2312-3038 (R1) DOI:10.21608/zumj.2024.254130.3038.

ORIGINAL ARTICLE

Response to Different Inspiratory Flow Patterns in Acute Respiratory Distress Syndrome Patients

Mohammed MN Abozaid, Adel Salah Ahmed, Sara MS Saadawy*, TarekHamdy Hassan.
Chest Department, Faculty of Medicine, Zagazig university, Egypt.

Corresponding author*
Sara MS Saadawy

E-mail:
Sarasaadawy@gmail.com

Submit Date 08-12-2023
Revise Date 31-12-2023
Accept Date 19-01-2023

ABSTRACT

Background: Acute respiratory distress syndrome (ARDS) is defined as acute hypoxemic breathing problems with bilateral infiltrates on chest imaging that cannot be described adequately by heart failure or excessive fluid retention. The present work aimed to provide better oxygenation and an improvement in terms of gas exchange of ARDS patients by adjusting flow patterns.**Methods:** This prospective cohort study was conducted on 30 cases with ARDS, regardless of the cause. All cases in the study were subjected to medical history taking, general examination, complete local chest examination, portable chest x-ray or CT (on admission and as ordered by ICU staff), laboratory investigations, electrocardiography, echocardiography, continuous monitoring of pulse oximetry, and recording of ventilator parameters as ventilator data and ABGs after one hour of each flow pattern tabulation.**Results:**There was no substantial variance between the two ventilation modes for any of the parameters, including pH, PaO2, PCO2, HCo3, and Spo2 ($p > 0.05$). Among the patients, 26.67% showed improvement and 73.34% died. Among the complications, pneumothorax occurred in 6.67% of cases. In comparison, acute renal failure affected 10%, gastrointestinal bleeding, and acute kidney injury (AKI) each occurred in 3.33% of cases, and ventilator-associated pneumonia (VAP) was observed in 16.67% of cases.**Conclusion:** The present investigation examined how varied IF waveforms are performed during regulated mechanical breathing in cases with ARDS. Our findings revealed no substantial variations in arterial oxygenation and partitioned respiration mechanics between the decelerating and square waveforms.

Keywords: Flow Patterns, Acute Respiratory Distress Syndrome, Ventilator Synchrony.

INTRODUCTION

During acute respiratory failure (ARF), the primary support of the respiration process is mechanical ventilation (MV), which is an incredibly invasive positive pressure [1]. Acute respiratory distress syndrome (ARDS) is defined by the presence of bilateral infiltrates and reduced blood oxygenation upon an initial insult. ARDS is a clinical illness characterized by sudden hypoxemia (PaO2:FiO2 ratio 300) and bilateral lung opacities that are not entirely attributed to heart failure or excess volume [2-4].

Flow curves depict the speed as well as the direction of both inhalation and exhalation in chronological order. They can be affected by respiration mechanics, the individual's effort, and the settings and type of ventilation [5].The most common types of MV are pressure- and volume-controlled ventilation, which are distinguished correspondingly by a square and a slowing-down flow waveform. Currently, the clinical value of various

inspiratory flow (IF) waveforms is unknown [6].

When modifying the ventilator setting, the data offered by the flow curves throughout MV, such as respiration mechanics, the case's effort, and patient-ventilator relationships, is beneficial. If physicians can adequately monitor and interpret the flow curve data, it can be an effective diagnostic and medication technique at the bedside. Paying attention to specific waveforms is crucial to enhance case outcomes [5]. The present work aimed to provide better oxygenation and an improvement in terms of gas exchange of ARDS patients by adjusting flow patterns.

METHODS

This prospective cohort study was conducted from February 2023 to July 2023 in the respiratory intensive care unit of the Zagazig University Hospitals.

Inclusion criteria:All patients fulfilled the criteria of ARDS according to Berlin criteria [7]: timing, radiographical changes, and origin of edema (no evidence of LVF or fluid overload).Severity based on the PaO2/FiO2 ratio on 5 cm of positive end-expiratory pressure (PEEP). The three categories of ARDS are PaO2/FiO2 200-300 (mild), 100-200 (moderate), and ≤100 (severe) [8].

Exclusion criteria:Patient with age less than 18 years, intubation due to arrhythmias, hypothermia, and myocardial infarction.

Sample size:30 cases of ARDS, regardless of the cause.

All participants were subjected to the following: medical history taking for diagnosis and identification of the etiology of ARDS and comorbidity conditions from patients' relatives. General examination stresses other system affection as cardiac and neurological disorders. A complete local chest examination was done on admission, portable chest x-ray or CT (on admission and as ordered by ICU staff) and Laboratory investigations.

***Laboratory investigations*:**

Complete blood count (CBC), liver function tests (LFT) (SGOT, SGPT, serum bilirubin, serum albumin, and total serum protein), Kidney function tests (KFT), random blood sugar, CRP, and arterial blood gases.

Electrocardiography (ECG) and echocardiography to exclude heart failure that mimics bilateral infiltration of ARDS cases. Continuous monitoring of non-invasive blood pressure, pulse oximetry, urine output, and central venous pressure. Serial daily recording of ventilator parameters as tabulation of serial ventilator data so as long the cases stay on MV, including PEEP, TV (in mL/kg PBW), inspired oxygen fraction (FIO2), end-inspiratory plateau pressure (Pplat), and total respiratory rate (f). Reports of patients' complications, including (organ failure or pulmonary complications). The endpoint of the study is the discontinuation of MV, patient death, or the need for change to another strategy. The ventilatory strategy is the lung protective ventilation strategy, according to Thompson and Bernard [9].

The protocol of the ARDS clinical network MV [9] was utilized. The prediction of weight was evaluated as males: =50 +2.3 (Height (inches) -60). Females: = 45.5 +2.3 (Height (inches) -60). Tidal volume was 8 ml/kg PBW

at first and then decreased by 1 ml/kg at intervals ≤ 2h until TV reached 6 ml/kg PBW. TV & RR were adjusted to achieve target pH Pplat goals. I: E ratio goal: recommend that inspiration duration be ≤ expiration duration. Set the flow rate as low as possible to elevate the time of inspiration with the following oxygenation improvement and continue monitoring intrinsic PEEP (Total PEEP-Extrinsic PEEP). Flow waveform: during one h of MV with a respiratory rate, set tidal volume, and PEEP, the cases received in a decelerating IF waveform as the default flow waveform, the first set of measurements were collected, including Pmean, Pplat, PIP, and vital signs were recorded, then after 1hour square flow waveform was received the second set of measurements were recorded. After 30 minutes in each phase, data on respiratory mechanics and gas exchange was obtained and examined.

Successive breaths were collected in the final two minutes of every observation period to gather the tidal volume, respiratory rate, and inspiratory highest flow. Total respiration time was obtained by dividing 60 seconds by the rate of respiration. A set of two end-inspiratory and end-expiratory pauses were done in the last 1 minute of every investigation session. The PaO2/FI O2 ratio after 30 minutes of equilibration in a specific ventilation mode (VCV) was the primary outcome metric. Plateau pressure (Pplat), SpO2, mean airway pressure (Pmean), minute ventilation (MV), tidal volume (TV), arterial carbon dioxide partial pressure (PaCO2), and peak inspiratory pressure (PIP) were additional outcome variables. Heart rate, diastolic, systolic, and mean arterial pressures (MAP) were among the hemodynamic factors measured. Adverse outcomes were defined as significant hemodynamic instabilities (MAP15% of baseline), new onset arrhythmias, pneumothorax, or endotracheal tube displacement.

Ethical and administrative considerations: Written informed consent was collected from all cases' relatives to participate in the study. This investigation was performed after the approval of the Institutional Review Board (IRB#10277). The study was conducted according to Declaration of Helsinki.

STATISTICAL ANALYSIS

Data was analyzed statistically with IIBM SPSS, version 23.0 (IBM Corporation, Armonk, New York). Quantitative data were described utilizing the mean, standard deviation, and range, while qualitative data were expressed using the number and percentage. The t-test was used to compare two groups of normally distributed variables. When applicable, the Chi-square test was employed to compare percentages of categorical variables. The Pearson correlation coefficient (r) is a method of determining the degree and direction of a linear association between two variables. All of the tests were two-sided. A p-value < 0.05 is considered significant.

RESULTS

The mean age of the subjects is 60.93±19.44 years; 70% are male, and 30% are female. The subjects also have various co-morbidities, with the most common being hypertension (46.67%), followed by diabetes mellitus (26.67%). The most common cause of ARDS

was pneumonia affecting (30%), followed by sepsis (20%) and trauma (20%). Aspiration, acute pancreatitis (Table 1).

There were no statistically significant differences observed between the two flow patterns in terms of flow rate ($p = 0.262$), fraction of inspired oxygen (FIO2, $p = 0.326$), tidal volume (TV, $p > 0.99$), respiratory rate (RR, $p > 0.99$), positive end-expiratory pressure (PEEP, $p > 0.99$), pressure support (PS, $p > 0.99$), peak airway pressure (P-Peak, $p = 0.027$), plateau pressure (Ppl, $p = 0.587$), inspiratory-to-expiratory ratio (I/E, $p = 0.218$), and mean airway pressure (Pmean, $p = 0.384$)(Table 2).

The mean PaO2/FIO2 was 142.92 ± 58.5 (16.67%) cases suffered from mild ARDS, 17 (56.67%) cases suffered from moderate ARDS, and 8 (26.67%) cases suffered from severe ARDS. The total mortality rate was 22 (73.34%) cases, with 1 (3.34%) case from the mild, 13 (43.33%) cases from the moderate ARDS, and 8 (26.67%) cases from the severed ARDS (Table 3).

Significant differences were observed in several parameters between the two-time points. The pH increased significantly from 7.26 ± 0.13 to 7.34 ± 0.12 ($p = 0.015$), indicating improved acid-base balance. The partial pressure of oxygen (PaO2) also significantly increased from 54.39 ± 17.74 to 69.39 ± 26.86 ($p = 0.015$), suggesting improved oxygenation. While there were no statistically significant differences in partial pressure of carbon dioxide (PCO2), bicarbonate (HCo3), and oxygen saturation (Spo2) between the initial and after one-hour ABGs of square patter, the trends in pH and PaO2 indicate positive changes in respiratory and acid-base parameters following 1 hour of square flow waveform (Table 4).

There were no statistically significant differences observed between the two flow waveforms for any of the parameters, including pH ($p = 0.443$), partial pressure of oxygen (PaO2, $p = 0.591$), partial pressure of carbon dioxide (PCO2, $p = 0.611$), bicarbonate (HCo3, $p = 0.739$), and oxygen saturation (Spo2, $p = 0.612$) (Table 5).

Among the patients, 26.67% survived and 73.34% died. The complications among the included subjects: pulmonary complication was pneumothorax occurred in 6.67% of cases, while extra-pulmonary complications were acute renal failure affected 10%, gastrointestinal bleeding, and acute kidney injury (AKI) each occurred in 13.33% of cases, and ventilator-associated pneumonia (VAP) was observed in 16.67% of patients. Additionally, other complications like arrhythmia, bleeding, and refractory shock were recorded (Table 6).

Before the patients received different flow waveforms, the comparison revealed that only heart rate (HR) exhibited a statistically significant difference (83.7 ± 18.66 in "Improved" vs. 95.9 ± 22.41 in "Did Not Improve," $p = 0.15$). However, this difference was not significant. For all other parameters, including oxygen saturation (SPO2) ($p = 0.802$), mean arterial pressure (MAP) ($p = 0.486$), central venous pressure (CVP) ($p = 0.919$), and respiratory rate (RR) ($p = 0.452$), no significant disparities were observed between improved and not improved prior the

patients receive different flow waveform. Similarly, after 1 hour of each flow waveform, none of the parameters exhibited significant differences between improved and not improved patients, as indicated by the p-values (HR p = 0.47, SPO2 p = 0.45, MAP p = 0.367, CVP p = 0.758, RR p = 0.618) (Table 7).

A substantial negative correlation was observed between improvement and male gender (r = -.617**, p-value = 0.0003), WBCs (r = -.576**, p-value = 0.0009), CRP (r = -.499**, p-value = 0.005), as well as Decelerating flow pattern RR (r = -.375*, p-value = 0.0411), square flow pattern RR (r = -.375*, p-value = 0.0411), and Square pattern after 1 hour PH (r = .405*, p-value = 0.0265). Conversely, there was a significant positive correlation between death and male gender (r = .617**, p-value = 0.0003), WBCs (r = .576**, p-value = 0.0009), CRP (r = .499**, p-value = 0.005), Decelerating flow pattern RR (r = .375*, p-value = 0.0411), square flow pattern RR (r = .375*, p-value = 0.0411), and S ABG after 1 hour PH (r = -.405*, p-value = 0.0265). No significant correlation was observed with any other parameters (Supplementary Table 1).

Table (1): Demographic data, comorbidity, and causes of ARDS among included subjects.

Demographic data	Value (N = 30)
Age	60.93 ± 19.44
Sex	
• **Male**	21 (70%)
• **Female**	9 (30%)
Occupation	
• **Working**	29 (96.67%)
• **Not Working**	1 (3.33%)
Marital status	
• **Married**	25 (83.33%)
• **Single**	4 (13.33%)
• **Widow**	1 (3.33%)
Comorbidity	
PCI with stent	1 (3.33%)
DM	8 (26.67%)
HTN	14 (46.67%)
IHD	1 (3.33%)
AF	1 (3.33%)
Recurrent cholangitis	1 (3.33%)
HCV	1 (3.33%)
Cancer colon, colostomy	2 (6.67%)
Becker myopathy	1 (3.33%)
CKD	1 (3.33%)
Hepatic disease	1 (3.33%)
Hemorrhagic stroke 3 years ago	1 (3.33%)
MND	1 (3.33%)
Tracheostomy	1 (3.33%)
Old ischemic stroke	1 (3.33%)

Demographic data	Value (N = 30)
ALS	1 (3.33%)
Epilepsy	1 (3.33%)
Multiple strokes	2 (6.67%)
Causes of ARDS	
Pneumonia	9 (30%)
Sepsis	6 (20%)
Acute Pancreatitis	2 (6.67%)
Trauma	2 (6.67%)
Aspiration	4 (13.33%)
drug overdose	3 (10%)
BI lower limb ischemia	1 (3.33%)
Blood transfusion	1 (3.33%)
Sever COVID	1 (3.33%)
Pulmonary embolism	1 (3.33%)

PCI - Percutaneous Coronary Intervention, DM - Diabetes Mellitus, HTN - Hypertension, IHD - Ischemic Heart Disease, AF - Atrial Fibrillation, HCV - Hepatitis C Virus, CKD - Chronic Kidney Disease, MND - Motor Neuron Disease, ALS - Amyotrophic Lateral Sclerosis

Table (2): The initial ventilatory parameters used at the beginning of each flow pattern.

	Decelerating (N = 30)	Square (N = 30)	P. value
Flow rate	44.8 ± 11.87	42.8 ± 11.04	0.262
FIO2	57.17 ± 25.82	56.83 ± 25.44	0.326
TV	472 ± 39.34	472 ± 38.44	>0.99
RR	14.63 ± 2.75	14.63 ± 2.75	>0.99
PEEP	10.37 ± 3.44	10.37 ± 3.44	>0.99
PS	12.27 ± 3.2	12.27 ± 3.2	>0.99
P-Peak	23.6 ± 7.12	26.77 ± 8.32	0.027
Ppl	19.1 ± 7.85	18.7 ± 7.44	0.587
I/E	1/2.86 ± 3.13	1/3.78 ± 1.74	0.218
Pmean	10.98 ± 3.1	10.46 ± 4.11	0.384

TV - Tidal Volume, RR - Respiratory Rate, PEEP - Positive End-Expiratory Pressure, PS - Pressure Support, P-Peak - Peak Airway Pressure, Ppl - Plateau Pressure, I/E - Inspiratory-to-Expiratory Ratio, Pmean - Mean Airway Pressure.

Table (3): Severity of ARDS according to Berline criteria and mortality rate.

PaO2/FIO2 (mean ± SD)	142.92 ± 58
Severity of ARDS (n %):	
• **Mild**	5 (16.67%)
• **Moderate**	17 (56.67%)
• **Severe**	8 (26.67%)
Mortality rate (n %):	
• **Mild grade mortality**	1 (3.34%)
• **Moderate grade mortality**	13 (43.33%)
• **Severe grade mortality**	8 (26.67%)
Total Mortality rate (n %)	22 (73.34%)

Table (4): Comparison between Initial ABG and after one hour in square flow pattern.

S	Initial ABG	Square ABG after 1 hour	P. Value
PH	7.26 ± 0.13	7.34 ± 0.12	0.015
PaO2	54.39 ± 17.74	69.39 ± 26.86	0.015
PCO2	90.29 ± 176.98	43.06 ± 17.35	0.175
HCo3	25.71 ± 9.7	22.34 ± 7.28	0.195
Spo2	85.71 ± 8.46	0.88 ± 0.16	0.551

ABG - Arterial Blood Gas, PH - Potential of Hydrogen, PaO2 - Partial Pressure of Oxygen, PCO2 - Partial Pressure of Carbon Dioxide, HCo3 - Bicarbonate, Spo2 - Oxygen Saturation.

Table (5): Comparison between Decelerating pattern and square pattern ABG after 1 hour of each flow pattern.

D/S	Decelerating ABG after 1 hour	Square ABG after 1 hour	P. Value
PH	7.35 ± 0.14	7.34 ± 0.12	0.443
PaO2	73.38 ± 39.99	69.39 ± 26.86	0.591
PCO2	41.78 ± 11.66	43.06 ± 17.35	0.611
HCo3	22.71 ± 8.25	22.34 ± 7.28	0.739
Spo2	86.54 ± 13.56	0.88 ± 0.16	0.612

ABG - Arterial Blood Gas, PH - Potential of Hydrogen, PaO2 - Partial Pressure of Oxygen, PCO2 - Partial Pressure of Carbon Dioxide, HCo3 - Bicarbonate, Spo2 - Oxygen Saturation.

Table (6): Complications and Fate among included subjects.

Pulmonary:	
Pneumothorax	2 (6.67%)
Extrapulmonary:	
Acute renal failure	3 (10%)
GIT bleeding	1 (3.33%)
AKI	1 (3.33%)
VAP	5 (16.67%)
Arrhythmia	1 (3.33%)
DIC	1 (3.33%)
Refractory shock	1 (3.33%)

Fate of patients: Value (N = 30)	
length of hospital stays (Day)	13.3 ± 5.28
Survived	8 (26.67%)
Died	22 (73.34%)

AKI - Acute Kidney Injury, VAP - Ventilator-Associated Pneumonia, DIC – Disseminated Intravascular Coagulopathy.

Table (7): Comparison of vital signs between Square and Decelerating pattern before and after one hour among improved and did not improve.

	Improved (N = 8)	Did Not Improved (N = 22)	P. Value
Decelerating Pattern			
Vitals just before			
HR	83.7 ± 18.66	95.9 ± 22.41	0.15
SPO2	94.5 ± 3.47	94 ± 5.72	0.802
MAP	79 ± 18.35	83.98 ± 18.14	0.486
CVP	10 ± 2.4	10.2 ± 5.89	0.919
RR	19.9 ± 6.44	22.15 ± 8.13	0.452
Vitals after 1 hour			
HR	83.2 ± 22.05	91.42 ± 31.69	0.47
SPO2	95.9 ± 2.47	93.65 ± 9.06	0.45
MAP	89.2 ± 20.5	82.65 ± 17.37	0.367
CVP	10 ± 2.71	10.65 ± 6.28	0.758
RR	19.1 ± 5.95	20.45 ± 7.32	0.618
Square Pattern			
Vitals just before			
HR	84.5 ± 20.55	94.25 ± 19.92	0.221
SPO2	94.2 ± 5.79	93.45 ± 7.15	0.776
MAP	84.7 ± 21.3	81.73 ± 18.48	0.696
CVP	9.7 ± 2.79	10.8 ± 6.2	0.599
RR	18.8 ± 5.22	20.8 ± 6.91	0.428
Vitals after 1 hour			
HR	85 ± 19.85	91.96 ± 29.78	0.513
SPO2	95.7 ± 4.57	93.42 ± 8.07	0.418
MAP	87.5 ± 19.15	88 ± 19.79	0.948
CVP	10.1 ± 2.23	10.2 ± 6.23	0.961
RR	19.9 ± 5.95	20.65 ± 8.51	0.805

HR (heart rate), SPO2 (oxygen saturation), MAP (mean arterial pressure), CVP (central venous pressure), and RR (respiratory rate).

Supplementary Table (1): Correlation between fate and pattern with other parameters.

	Fate				Pattern			
	Improved		Died		S		D	
	r	P. value	r	P. value	r	P. value	r	P. value
Demographic data								
Sex								
Male	-.617**	0.0003	.617**	0.0003	-0.26245	0.1612	0.262445	0.1612
Female	.617**	0.0003	-.617**	0.0003	0.262445	0.1612	-0.26245	0.1612
Laboratory								
WBCs	-.576**	0.0009	.576**	0.0009	0.053259	0.7798	-0.05326	0.7798
CRP	-.499**	0.005	.499**	0.005	0.242131	0.1974	-0.24213	0.1974
VENTILATOR PARAMETERS (D)								
RR	-.375*	0.0411	.375*	0.0411	-0.29348	0.1155	0.293479	0.1155
Vitals just before (D)								
RR	-.375*	0.0411	.375*	0.0411	-0.29348	0.1155	0.293479	0.1155
Vitals just before (S)								
PH	.405*	0.0265	-.405*	0.0265	0.061785	0.7457	-0.06178	0.7457

WBCs: white blood cells, CRP: C reactive protein, RR: Respiratory rate.

DISCUSSION

The most common types of MV are pressure- and volume-controlled ventilation, which have a square and a decreasing flow waveform, respectively. Currently, the clinical value of various IF waveforms is unknown [1]. To accomplish this task, 30 ARDS cases (newly admitted) were enrolled. Data at the onset of diagnosis was evaluated upon them. The ARDS Network protocol of MV was conducted and admitted from February 2023 to Juley 2023. There were about 21 males and nine females.

In our study, the subjects have various co-morbidities, with the most common being hypertension (46.67%), followed by diabetes mellitus (26.67%). In total, 30 cases showed no significant difference between survived and dead cases except in hypertension.In the current investigation, we employed a common MV to provide VCV to sedated and paralyzed ARDS patients: two alternative flow waveforms (square and decelerating) were tested.The theoretical benefits from various inspiratory patterns of flow are likely to be associated with variations in the distribution of ventilation into the lung, leading to a theoretical decrease in inhalation airway peak pressure, improved lung recruitment, and decreased mechanical power and CO2 elimination [10].

The theoretical benefits from various inspiratory patterns of flow are likely to be associated with variations in the distribution of a decelerating trend that provides the most flow at the start of a breath when case flow demand is often highest. When applied, this flow pattern may increase patient/ventilator synchronization and aid cases who require high IF rates. This flow pace also can lower PIP when compared to a continuous flow waveform form. When PIPs approach their upper limits, this could have serious consequences. A decelerating flow waveform pattern has also been demonstrated to improve gas distribution and oxygenation status by increasing mean airway pressure [5]. The peak inspiratory flow (PIF) is equivalent to the IF when the square waveform is supplied. In contrast, the IF is near nil at the

conclusion of inspiration for decelerating, sinusoidal waveforms, and trunk decelerating [1]. Regarding ventilatory machines, we used the Bennett machine. It had more alarms, options, and pre-programmed settings, which impact the type of breath that is delivered to cases and clinician familiarity with ventilator machines.

In this investigation, the inspiratory time it stayed constant based on the ventilator employed. As a result, the exact gas volume was provided throughout the same inspiratory period of use when switching between flow waveforms. As in Chiumello*et al*. [1], the effects of varied forms and IF throughout the same inspiratory duration were investigated.The mean PIP was (23.6 ± 7.12) (cm.H2O) in the decelerating pattern, and the square pattern was (26.77 ± 8.32) (cm.H2O).In our study of 30 patients, as regards low tidal volume according to lung protective strategies, the mean tidal volume in all days of studied ARDS patients was 472 ± 39.34 ml.In this study, Compliance with the ARDS Network ventilatory regimen was preserved, with minimal tidal volume based on estimated body weight and protection against high plateau pressure, which led to a better outcome. The mean plateau pressure was 19.1 ± 7.85 cm H2O among decelerating flow, and the mean plateau pressure among square flow patients was 18.7 ± 7.44 cm H2O.With the harmony of Warner and Patel. [11] who stated that plateau pressures < 30 mm Hg were correlated with a reduced mortality rate than conventional tidal volume using plateau pressures < 50 mm Hg.

In a study by De Matos *et al*. [12], the inspiratory plateau pressure of roughly 40 cm H2O almost certainly resulted in considerable levels of stress/strain. Nevertheless, mortality rates were not more significant than predicted, considering the severity of the condition, and no severe extrapulmonary or pulmonary complications were observed. Furthermore, the chance for recruitability was significantly elevated than the stated average of roughly 13%.The decelerating pattern of flow causes an elevation in mean lung capacity and, as a consequence, a rise in intrapulmonary pressure, which is comparable to positive end-expiratory pressure [13]. It is commonly believed that decelerating waveforms result in higher mean airway pressures and alveolar recruitment with reduced PIF. In contrast, constant airflow ramps result in reduced inspiratory duration and higher PIF [5].Furthermore, the decelerating waveform had the most minor peak and plateau pressures. These gains in gas exchange and lung mechanics occurred without a negative impact on hemodynamics [14].

Substantial variances were detected in several parameters between the two time points. The pH elevated significantly from 7.26 ± 0.13 to 7.34 ± 0.12 (p = 0.015), indicating improved acid-base balance. The partial pressure of oxygen (PaO2) also significantly increased from 54.39 ± 17.74 to 69.39 ± 26.86 (p = 0.015), suggesting improved oxygenation. While there were no substantial variances in partial PCO2, bicarbonate (HCo3), and SpO2 between the initial and square ABG measurements, the trends in pH and PaO2 indicate positive changes in respiratory and acid-base parameters following 1 hour of square ventilation.There were no substantial variances detected between the two ventilation modes for any of the parameters, including pH (p = 0.443), PaO2 (p = 0.591), partial PCO2 (p = 0.611), HCO3 (p = 0.739), and SpO2 (p = 0.612).Davis *et al*. [10] assumed that decelerating waveform modestly increased oxygenation from 75 to 85 mmHg, most likely as a result of a more significant airway pressure, which might have assisted alveolar recruitment. In a similar study by Al-Saady *et al*. [14], in 14 individuals who used a decelerating waveform instead of a square waveform, alveolar oxygenation increased.

If flow drops throughout the lung inflate, the majority of the VT is given early in inspiration, permitting the fresh gas to have an extended stay in the alveoli and encouraging gas diffusion. Furthermore, with significant time constants, the reducing pattern offers excess time for gas to enter the alveoli [13]. During the inspiratory phase, airway pressure remains at a greater level, while with a steady flow, gradually, the

pressure elevates to the predetermined volume [13]. Yang and Yang [15] found that changing the rate of inspiration had no impact on arterial pH or oxygenation. Paco2 showed no variation between sine waveforms and constant squares. The decelerating waveform, on the other hand, generated much greater P(A-a) O2 and substantially reduced Paco2 than either sine waveforms or the constant square.According to Polese *et al*. [13], several researchers have explored the impact of IF waveform on ABG with inconsistent results. In anesthetized animals and individuals with ARF, a decelerating flow profile was observed to improve PaO2 much more than other inflation flow waveforms. During pressure-controlled ventilation, the same was discovered in individuals with ARF.

The present study was in line with Davis *et al*. [10], who showed that Pressure- and volume-controlled ventilation with a decelerating flow waveform both provided improved oxygenation at less PIP and higher airway pressure than volume-controlled ventilation with a square flow waveform.In another study, Roth *et al*. [16] showed that when compared to continuous IF ventilation, decelerating IF ventilation had no benefit in terms of gas exchange, alveolar recruitment, or inspiratory pressures.The increased FiO2 with increased PEEP can be explained by the fact that our mortality rate is high at 73.34%, and there were worsening cases; we used high FiO2 in our cases because we applied maximum allowable PEEP to guard against decreased COP or worsening shock so FiO2 increased to fulfill more oxygenation to tissues with increased PEEP and decreased flow rate.

There were no substantial variances detected between the two ventilation modes for any of the parameters, including pH ($p = 0.443$), PaO2 ($p = 0.591$), PCO2 ($p = 0.611$), HCo3 ($p = 0.739$), and Spo2 ($p = 0.612$). Considering all of these potential advantages of the decelerating flow profile, neither this investigation nor that of Laviola *et al*. [17] noticed a substantial increase in PaO2 with changes to the IF waveform. Many processes were suggested to explain the putative potential of varied IF waveforms on ABG. First, a decelerating flow profile has the potential to result in a more consistent ventilation distribution in relation to perfusion [13].

This study investigated the IF rate in mechanically ventilated ARDS cases and showed that the IF rate was set to 44.8 ± 11.87 (l/min), which was the mean IF rate in all studied ARDS cases. The mean IF rate in survived ARDS patients was 45 ± 11.6 (l/min) and 44.7 ± 12.3 (l/min) in dead ARDS patients, with highly significant statistical difference between zero reading at the onset of ARDS and mean IF rate in all studied ARDS cases. This is in harmony with Chen *et al*. [18].The study of Chen *et al*. [18] showed that a leading cause of elevated flow resistance and reduced IF rate in ARDS cases was high PEEP utility. They determined that monitoring IF rate and flow resistance during MV could be beneficial in ARDS case management in the Surgical ICU.As this LPV procedure engaged more than just using a low tidal volume with a decelerating flow wave, the following guidelines should be followed: tidal volume size should depend on the predicted weight of the individual rather than actual body weight; tidal volumes must be consistently modified to preserve a plateau pressure of less than 30 cm H2O; a pH of 7.3 to 7.45; and a suitable combination of FiO2 and PEEP must be employed to attain adequate oxygenation [9].

Concerning the frequency of complications in ARDS patients, pneumothorax occurred in 6.67% of cases. In comparison, acute renal failure affected 10%, gastrointestinal bleeding, and AKI each occurred in 3.33% of cases, and VAP was observed in 16.67% of cases. Additionally, other complications like arrhythmia, bleeding, refractory shock, and various causes of ARDS, including pneumonia, sepsis, trauma, and others, were recorded. This is consistent with Stapleton *et al*. [19].The total mortality rate was 22 (73.34%) cases, with 1 (3.34%) case from the mild, 13 (43.33%) cases from the moderate ARDS, and 8 (26.67%) cases from the severed ARDS.Nevertheless, mortality

increased with increasing age. That is in contrast with the study of Navarrete-Navarro *et al*. [20], which stated that mortality decreased in both those over and under 65 years old after the LPV strategy.

In our analysis of 30 cases, the overall mortality rate (22 instances) was roughly 73.34%. Over the last two decades, investigations from the world's leading medical institutes have claimed that mortality has dropped to 30%, which may be attributed to advancements in both the specialized therapy of cases with ARDS and ICU cases general management.Bellani *et al*. [21] still report that mortality rates are high despite the implementation of protective lung ventilation strategies.Additional investigations conducted over the last 20 years have found that ARDS mortality has decreased, and the only medication demonstrated to be beneficial in terms of mortality is protective ventilation [22].Similarly, observational studies of ARDS over 15 years revealed a mortality decrease. Doyle *et al*. [23] assumed that 58% of ARDS cases had a hospital mortality rate, and Nucktonet *al*. [24] showed a reduced mortality rate in ARDS cases (42%). A study by Saleh *et al*. [25] revealed that the mortality rate reached 41%. Also, another study by Nafea *et al*. [26] reported a mortality rate reached 60% in ARDS patients.This result supposes that there was a relatively high mortality rate in our cases, while the intention for lung protective ventilation utilization was due to most of them having moderate to severe ARDS criteria, as their mean PaO2/FiO2 was 142.92 ± 58.5.

Limitations:

One limitation is that just one MV type was employed. Hence, the current results cannot be generalized to other ventilators. However, by using the same ventilator throughout the research, the chance of inter-patient heterogeneity due to technical factors is reduced. Another constraint is the one-hour duration of the stabilization time.

CONCLUSION

The present investigation examined how varied IF waveforms performed during regulated mechanical breathing in cases with ARDS. Our findings revealed no substantial variations in arterial oxygenation and partitioned respiration mechanics between the decelerating and square waveforms.

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Citation:

Abozaid, M., Ahmed, A., Saadawy, S., Hassan, T. Response To Different Inspiratory Flow Patterns in Acute Respiratory Distress Syndrome Patients. *Zagazig University Medical Journal*, 2024; (3109-3121): -. doi: 10.21608/zumj.2024.254130.3038