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Prevalence and Risk Factors of Pneumothorax among Patients in Pediatric Intensive Care Unit

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ABSTRACT

Background: Pneumothorax should be considered a medical emergency and requires a high index of suspicion and prompt recognition and intervention. **Aim of the work:** was to study the prevalence and risk factors of pneumothorax among children in Tanta PICU. **Subjects and Methods:** Sixty pediatric patients aged from 2 to 180 months (29 males and 31 females) admitted to PICU, Tanta University Hospital and were divided into 3 groups: *Group 1*: 20 patients on P-CMV. *Group II*: 20 patients on HFOV: *Group III*: 20 non-ventilated patients. Each group was further divided into two subgroups: Subgroup I (SGI): without pneumothorax, Subgroup II (SGII): with pneumothorax. All patients were subjected to scoring systems for Pediatric Risk for Mortality (PRISM III) and Sequential Organ Failure Assessment (SOFA). They were also monitored for [pulse oximetry, blood pressure, oxygenation index, oxygenation saturation index, lung mechanics (compliance and resistance), ventilation parameters **(**HFOV and P-CMV) and trans-esophageal Doppler].

Results: Incidence of pneumothorax in patients on P-CMV is higher than who on HFOV and non- ventilated group. The significant risk factors of pneumothorax were HR, RR, pH, PCO2, HCO3, PaO2, SaO2, OI, MAP, FIO2, PIP, CO, CI, SVRI, SOFA and PRISM III score. X-ray showed equal results to CT chest in the diagnosis of pneumothorax without the disadvantage of exposure to high radiation accompanied the use of CT. **Conclusion:** Pneumothorax in P-CMV patients occur with higher ventilatory settings. Most cases of pneumothorax have underlying lung disease as pneumonia and ARDS. Mortality rate is higher among patients with pneumothorax.

Keywords: Pneumothorax, Pediatric, Intensive Care Unit, Risk factors

INTRODUCTION

Pneumothorax is defined as the accumulation of air between the visceral and parietal pleura that leads to partial or complete collapse of lung [(1)].

Ironically, the higher incidences of pneumothorax in ventilated neonates with mild increase among those receiving CPAP and dramatic increase with mandatory modes of ventilation have been addressed by only few studies [(2)].

Aim of the work was to study the prevalence and risk factors of pneumothorax among children in Tanta PICU.

PATIENTS AND METHODS

This prospective study was carried out during the period from July 2017 to November 2018. Cases were selected from Pediatric Intensive Care Unit (PICU), Tanta University Hospital. It included 60 pediatric patients aged from 2 to 180 months (29 males, 31 females) and were divided into **3** groups:

Group 1: 20 patients on P-CMV.

Group II: 20 patients on HFOV.

Group III: 20 non-ventilated patients. Each group was further divided into two subgroups: Subgroup I (SGI): without pneumothorax, Subgroup II (SGII): with pneumothorax.

Inclusion criteria: All patients admitted to PICU aged from 2 months to180 months.

Exclusion criteria: Patients with congenital lung disease.

Ethical approval: Written informed consent was obtained from the parents or guardians of each child.

The study was approved by the Ethics Committee of Faculty of Medicine, Tanta University.

Collection of data: All the studied patients were subjected to the following on the first day of admission to Tanta PICU**:**

1. **Detailed history taking with special emphasis on**:
- Demographic data: name, age, sex, socio-economic status.
- Cause of PICU admission.
- Initial symptoms: date of onset of symptoms and its duration and course of illness.
- Cardiac symptoms as dyspnea, cyanosis, edema, palpitation.
- Pulmonary symptoms as: wheezes, respiratory distress and cough.

2. **Thorough clinical examination including vital signs with especial emphasis on:**
- Anthropometric measurements: weight, height and vital data.
- Cardiac and respiratory systems, e.g.: heart sounds, murmurs, breath sounds and additional sounds.

3. **Routine Laboratory Investigations**

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- Complete Blood Count (CBC).
- C - reactive protein (CRP).
- Arterial blood gases (ABGs) sample taking from arterial blood [*blood gas analyzer Stat Profile ® pHOx ® Series plus nova biomedical UK: Innovation house Aston lane South, Runcan, Cheshire WA7, 3FY, UK*]
- Plain x-ray (*posterior anterior view, lateral view*)
- CT chest if possible.

4. Scoring systems for patients:

- **Pediatric risk for mortality (PRISM) III scoring** was obtained from each patient immediately on admission [(3)].
- **Sequential organ failure assessment (SOFA) scoring** was obtained from each patient 48 hours after admission [(4)].

5. Monitoring: All the patients are monitored for:

A) Noninvasive investigation:

- Pulse oximetry (*Bedside monitor, BSM-4113K, Nihon Kohden Corp., Tokyo, Japan*) over full-perfused area (*mostly over digits of upper limbs*) to continuously monitor SaO2.
- Blood pressure (*Bedside monitor, BSM-4113K, Nihon Kohden Corp., Tokyo, Japan*).

B) Oxygenation Index[(5)]: ($F_iO2 \times MAP / PaO2) \times 100$

- OI<4 (at risk), OI 4-7.9 (*mild hypoxemia*), OI 8-15.9 (*moderate hypoxemia*), ≥5 -10 (*20%mortality rate*), ≥10 (*50% mortality rate*), OI >16 (*severe hypoxemia*), ≥ 40 (*need for ECMO*). MAP: Mean airway pressure.

C) Oxygenation Saturation Index: (FiO2 × MAP/SaO2) × 100

D) Lung mechanics:

- Compliance; C = ΔV /ΔP
- Resistance; Raw=P plat/peak inspiratory flow rate [(6)].

E) Ventilation parameters e.g.:

1- HFOV: MAP, ΔP, Frequency and FiO2 Device: Ventilation was accomplished using a [*Fabian HFOV" "ACUTRONIC" Medical Systems AG Fabrik im Schiffli 8816 Hirzel / Switzerland*].

2-P-CMV: FiO2, TI, PIP, RR and PEEP. Ventilation was accomplished using a Raphael color ventilator, [*Model X1, Hamillton medical, Hamilton Medical AG, CH- 7403Rhazuns, Switzerland*].

F) *Trans-Esophageal Doppler* [(7)].

- Monitoring of: [Stroke volume - Stroke volume index - Cardiac output - Cardiac index - Systemic vascular resistance -Systemic vascular resistance index]
- **Principal of assay** [(7)]**:**
  - CardioQ-ODM manufactured by Deltex Medical of Chichester UK was used **[***Cardio Q ODM, Model No 9051-6935, DELTEX MEDICAL LTD PO19 8 TX UK, 2008***]**.
  - Pediatric Doppler probes were used: (*KDP72*) Doppler Probe (*Product Code: 9081-7001) 72-hour pediatric oral Doppler probe***.**

Statistical analysis

Statistical analysis was done using IBM SPSS version 23. Data were expressed as mean ± SD, range, median, frequency and percentage and were analyzed using the following tests:[*independent student "t" test - chi-square (χ^2) test - ANOVA with repeated measures - Fisher's Exact or Monte Carlo correction - McNemar-Bowker - Mann Whitney test'6 and Wilcoxon signed ranks test*] to assess the significance of difference in the levels between different parameters. $P < 0.05$ was accepted as significant. *Spearman coefficient* to correlate between two abnormally quantitative variables

RESULTS

Laboratory assessments of the measured parameters are presented in the following tables and figures:

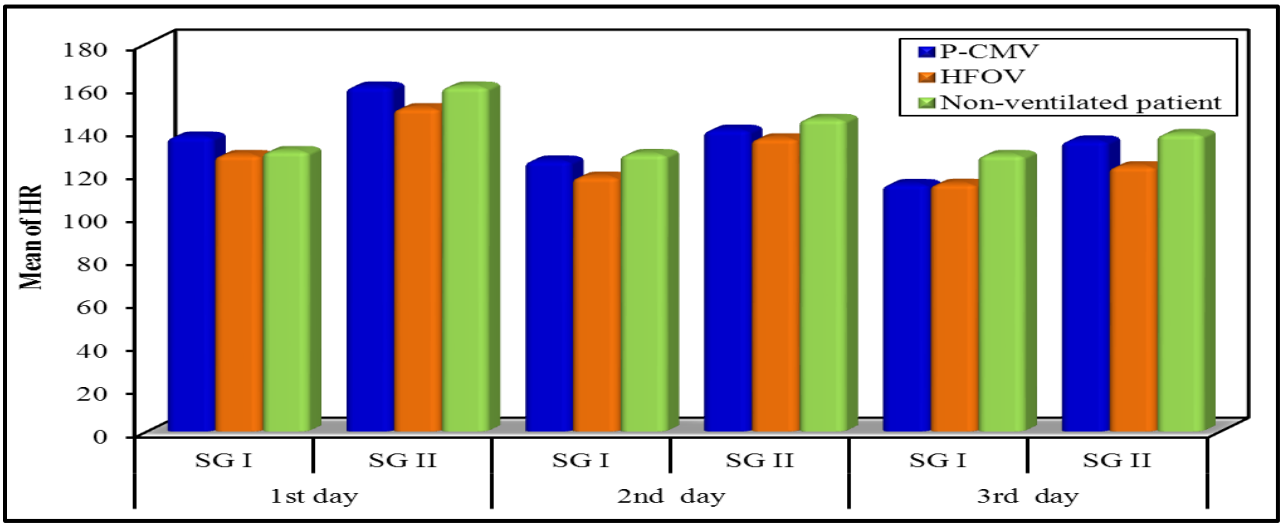

Figure (1): Comparison of heart rate (bpm) between the studied groups.

- **Figure (1)** showed heart rate (bpm) distribution between the studied groups.
  - Regarding P-CMV group
    - There was statistically significant increase in 1st and 2nd days compared to 3rd day in SG I.
    - There was statistically significant increase in 1st compared to 3rd day in SG II.
    - There was statistically significant increase in SG II compared to SG I in 1st day
  - Regarding HFOV group:
    - There was statistically significant increase in 1st compared to 3rd day in SG I.
    - There was statistically significant increase in 1st compared to 3rd day in SG II.
    - Otherwise, there was no statistically significant difference between studied periods or groups.
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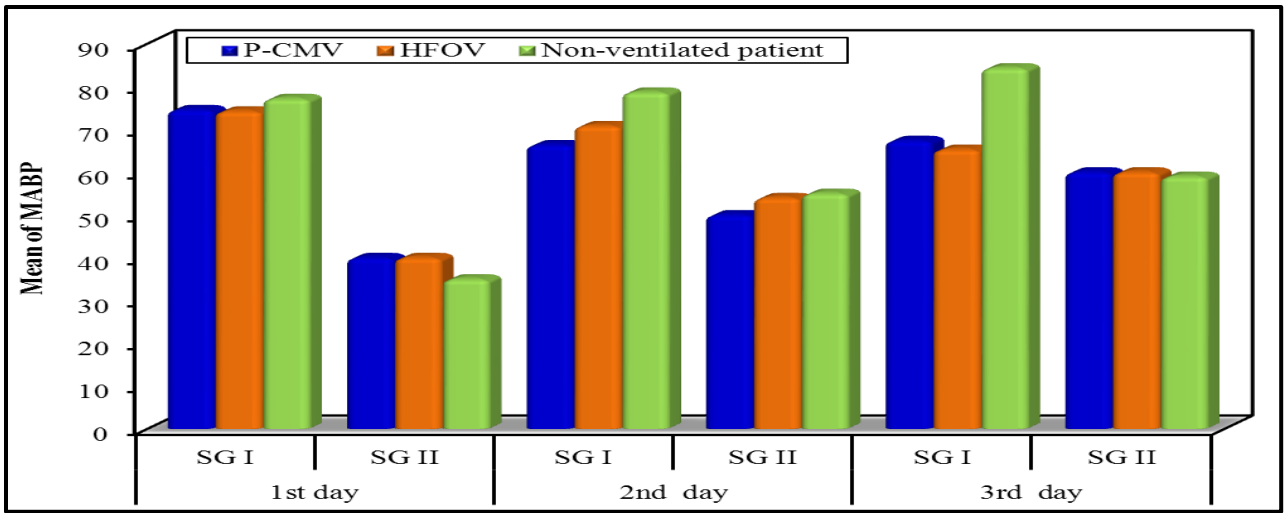

Figure (2): Comparison of mean arterial blood pressure (mmHg) between the studied groups.

- **Figure (2)** showed the mean arterial blood pressure in the studied groups.
  - Regarding P-CMV group
    - There was statistically significant decrease in 1st day compared to 3rd days in SG II.
    - There was statistically significant decrease in SG II compared to SG I in 1st day.
  - Regarding HFOV group
    - There was statistically significant increase in 1st and 2nd compared to 3rd day in SG I.
    - There was statistically significant decrease in SG II than SG I in 1st day.
  - Regarding non-ventilated group
    - There was statistically significant decrease in SG II than SG I in 1st and 3rd days.
    - Otherwise, there was no statistically significant difference between studied periods and groups.

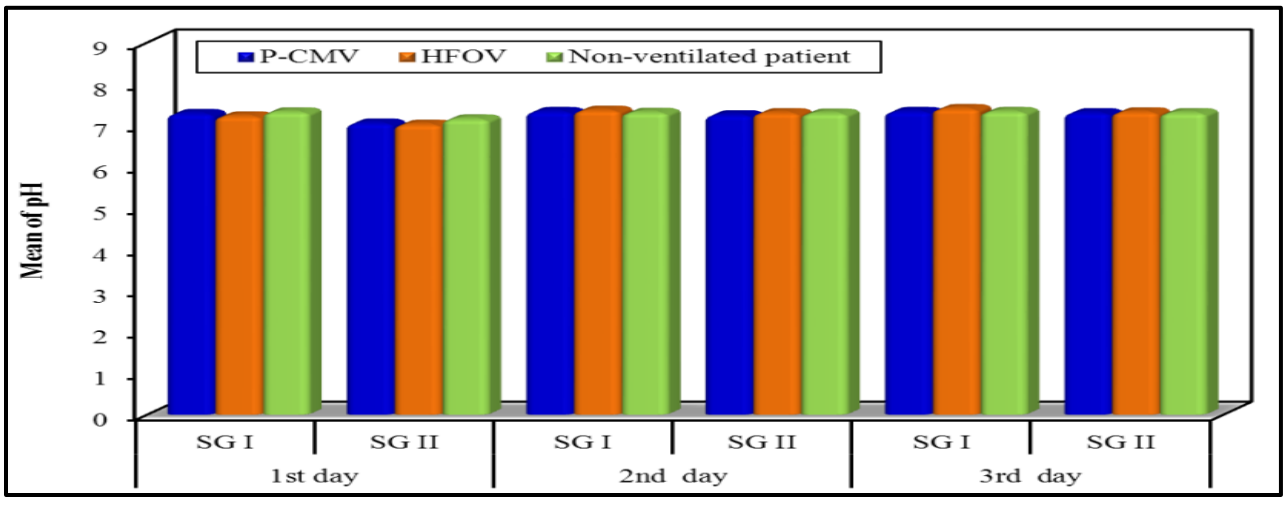

Figure (3): Comparison of pH between the studied groups.

- **Figure (3)** showed pH distribution in the studied groups.
  - Regarding P-CMV group
    - There was statistically significant decrease in 1st day compared to 2nd and 3rd days in SG II.
    - There was statistically significant decrease in SG II than SG I in 1st, 2nd and days.
  - Regarding HFOV group
    - There was statistically significant decrease in 1st compared to 2nd and 3rd days in SG I.
    - There was statistically significant decrease in 2nd compared to 3rd day in SG I.
    - There was statistically significant decrease in SG II compared to SG I in 1st day.
  - Regarding non-ventilated group
    - There was statistically significant decrease in SG II compared to SG I in 1st day.
    - Otherwise, there was no statistically significant difference between studied periods and groups.
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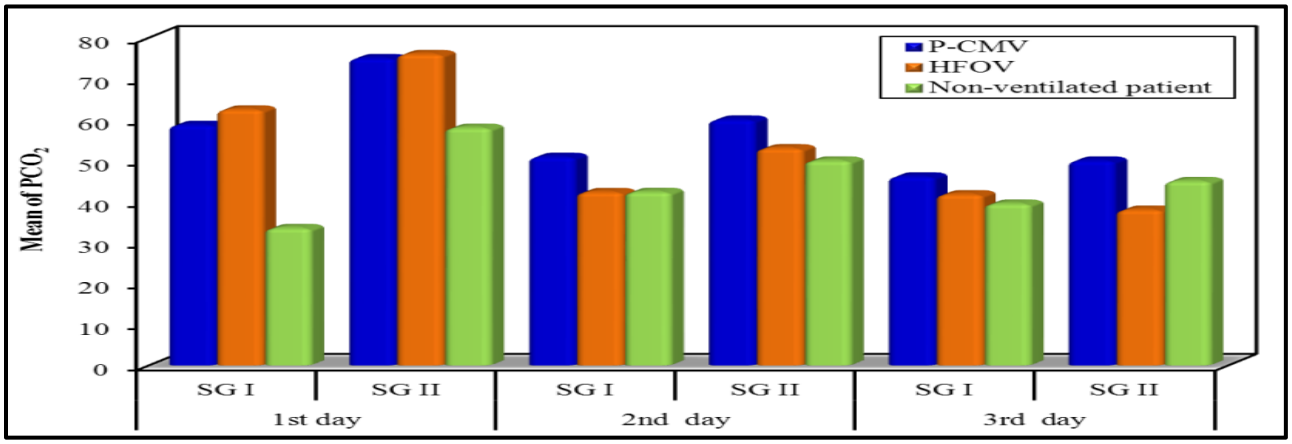

Figure (4): Comparison of partial pressure of arterial carbon dioxide (mmHg) between the studied Groups.

- **Figure (4)** showed partial pressure of arterial carbon dioxide (mmHg) distribution in the studied groups.
  - Regarding P-CMV group
    - There was statistically significant increase in 1st compared to 3rd day in SGI.
    - There was statistically significant increase in 1st compared to 2nd and 3rd days in SG II.
    - There was statistically significant increase in 2nd compared to 3rd day in SG II.
    - There was statistically significant increase in SG II compared to SG I in 1st day.
  - Regarding HFOV group
    - There was statistically significant increase in 1st compared to 2nd and 3rd days in SGI.
    - There was statistically significant increase in 1st compared to 3rd day in SG II.
  - Regarding non-ventilated group
    - There was statistically significant increase in SG II compared to SG I in 1st day.
    - Otherwise, there was no statistically significant difference between studied periods or groups.

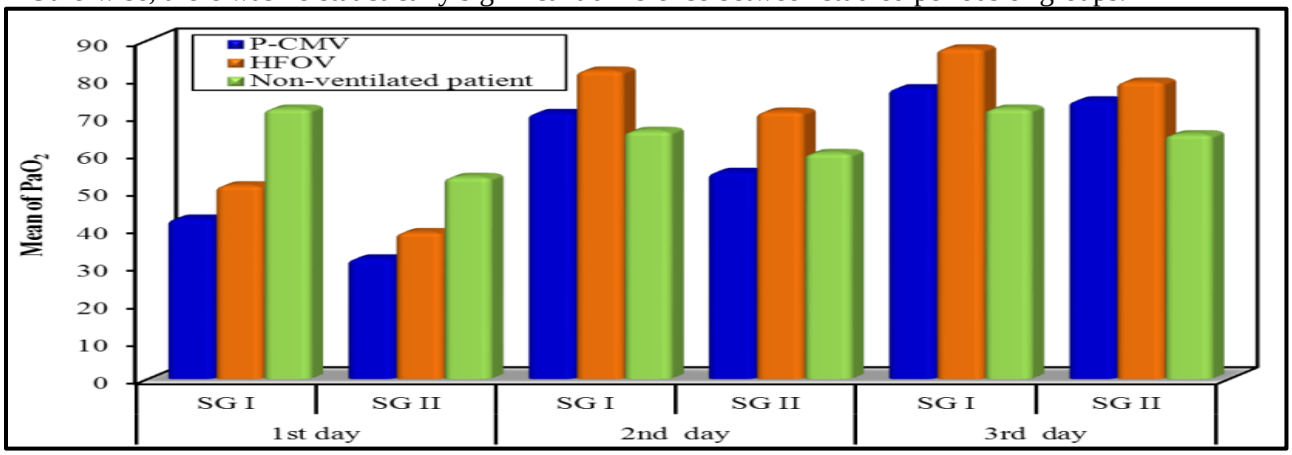

Figure (5): Comparison of partial pressure of arterial oxygen (mmHg) between the studied groups.

- **Figure (5)** showed partial pressure of arterial oxygen (mmHg) distribution in the studied groups.
  - Regarding P-CMV group
    - There was statistically significant decrease in 1st day compared to 2nd and 3rd days in SG I.
    - There was statistically significant decrease in 2nd compared to 3rd day in SG I.
    - There was statistically significant decrease in 1st and 2nd days compared to 3rd day in SG II.
    - There was statistically significant decrease in SG II compared to SG I in 2nd day.
  - Regarding HFOV group
    - There was statistically significant decrease in 1st day compared to 2nd and 3rd days in SG I.
    - There was statistically significant decrease in 2nd day compared to 3rd day in SG I.
    - There was statistically significant decrease in 1st day compared to 3rd day in SG II.
    - There was statistically significant decrease in SG II compared to SG I in 1st day.
    - Otherwise, there was no statistically significant difference between studied periods or groups.

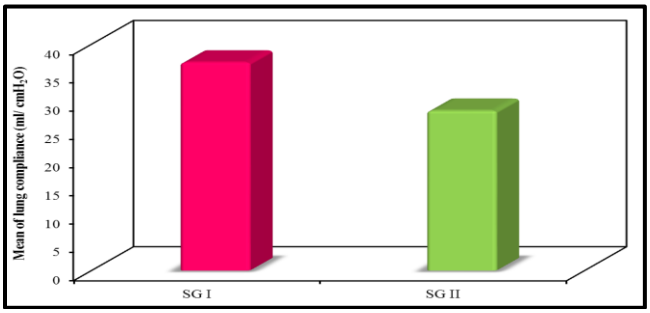

Figure (6): Comparison of Lung Compliance (ml/ cmH2O) Between the Studied Groups in Pressure controlled mandatory ventilation.

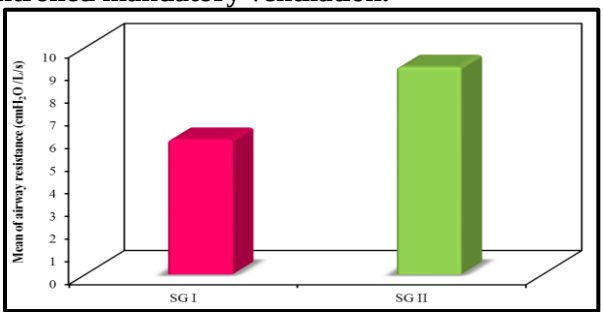

Figure (7): Comparison of Airway Resistance (cmH2O /L/s) Between the Studied Groups in Pressure controlled mandatory ventilation.

- **Figure (6,7)** showed lung mechanics distribution in the studied Groups.
  - Regarding lung compliance: There was statistically significant decrease in SG II compared to SG I.
  - Regarding airways resistance: There was statistically significant increase in SGII compared to SGI.

Table (1): Comparison of peak inspiratory pressure (cmH_2O) between the studied groups in pressure controlled mandatory ventilation

	PIP	1st day	2nd day	3rd day	F_2	p_2
P-CMV (n = 20)	**SG I (n=49)**	19.43 ± 5.18	18.0 ± 5.11	17.57 ± 5.08	2.945	0.070
	SG II (n=11)	29.0 ± 4.82	26.0 ± 4.65	23.0 ± 3.90	5.510	0.066
	t	**3.858***	**3.286***	**2.327***		
	p	**0.001***	**0.004***	**0.032***		

Cm H2o: centimeters Water, P-CMV: Pressure controlled mandatory ventilation, PIP: Peak Inspiratory Pressure
SG I: without pneumothorax SG II: with pneumothorax
t: Student t-test F_2: F test (ANOVA) with repeated measures
p: p value for comparing between SG I and SG II
p_2: p value for comparing between the three periods

- **Table (1)** showed the peak inspiratory pressure in the studied groups
  - There was statistically significant increase in SG II compared to SG I in 1st, 2nd and 3rd day.

Table (2): Comparison of positive end expiratory pressure (cmH_2O) between the studied groups in pressure controlled mandatory ventilation

	PEEP	1st day	2nd day	3rd day	Fr	p_2
P-CMV (n = 20)	**SG I (n=49)**	5.0 ± 0.0	5.14 ± 0.53	5.14 ± 0.53	2.000	0.368
	SG II (n=11)	6.0 ± 1.26	6.33 ± 1.03	6.50 ± 1.22	2.000	0.368
	U	**21.0**	**17.0***	**16.50***		
	p_1	**0.091**	**0.041***	**0.033***		

CmH2o: centimeters Water, PEEP: Positive End Expiratory Pressure, P-CMV: Pressure controlled mandatory ventilation.

- **Table (2)** showed the positive end expiratory pressure in the studied groups
  - Regarding P-CMV group:
    - There was statistically significant increase in SG II compared to SG I in 2nd and 3rd day.

- There was no statistically significant difference between studied periods.

Table (3): Comparison of stroke volume index (L/min/$m^{2)}$ between the studied groups

	SVI	1st day	2nd day	3rd day	Fr	p_2
P-CMV (n = 20)	**SG I (n=49)**	19.70 ± 9.19	22.90$^{\#}$ ± 10.63	21.81$^{\#}$ ± 9.15	10.302*	0.006*
	SG II (n=11)	13.0 ± 15.03	17.01 ± 15.05	19.0$^{\#}$ ± 15.02	12.0*	0.002*
	U	**16.0***	**20.0**	**24.0**		
	P	**0.033***	**0.076**	**0.153**		
HFOV (n = 20)	**SG I (n=49)**	18.13 ± 6.57	21.35 ± 8.63	21.22 ± 8.0	3.250	0.197
	SG II (n=11)	14.0 ± 5.56	19.0 ± 11.17	20.0 ± 11.01	0.667	0.717
	U	**12.0**	**20.0**	**22.50**		
	P	**0.179**	**0.616**	**0.765**		
U		**5.0**	**7.0**	**8.0**		
p_1		**0.381**	**0.714**	**0.905**		

HFOV: High Frequency Oscillatory Ventilation, P-CMV: Pressure controlled mandatory ventilation, SV: Stroke Volume index

- **Table (3)** showed the stroke volume index (L/min/$m^{2)}$ in the studied groups
  - Regarding P-CMV group:
    - There was statistically significant decrease in 1st compared to 2nd and 3rd days in SG I.
    - There was statistically significant decrease in 1st compared to 3rd day in SG II.
    - There was statistically significant decrease in SG II compared to SG I in 1st day.
    - Otherwise, there was no statistically significant difference between studied periods or groups.

Table (4): Comparison of systemic vascular resistance index (dyn.s/cm^5/m^2) between the studied groups

	SVRI	1st day	2nd day	3rd day	Fr	p_2
P-CMV (n = 20)	**SG I (n=49)**	2347.0 ± 1511.6	2099.6 ± 905.2	2105.1 ± 896.1	2.178	0.337
	SG II (n=11)	4213.8 ± 1936.9	2888.0$^{\#}$ ± 960.5	2888.0$^{\#}$ ± 960.4	11.474	0.003*
	U	**15.0***	**23.5**	**24.0**		
	P	**0.026***	**0.130**	**0.153**		
HFOV (n = 20)	**SG I (n=49)**	2639.6 ± 1521.9	2141.4 ± 927.3	2011.4 ± 822.5	3.745	0.154
	SG II (n=11)	3102.0 ± 794.1	2900.0 ± 955.1	2154.0 ± 302.3	4.667	0.097
	U	**9.0**	**13.0**	**22.0**		
	P	**0.093**	**0.216**	**0.765**		
U		**6.0**	**9.0**	**3.0**		
p_1		**0.548**	**1.000**	**0.167**		

HFOV: High Frequency Oscillatory Ventilation, P-CMV: Pressure controlled mandatory ventilation, SVRI: Vascular Resistance Index

Table (4) showed the systemic vascular resistance index (dyn.s/cm^5/m^2) in the studied groups

- Regarding P-CMV group:
  - There was statistically significant increase in 1st compared to 2nd and 3rd days In SG II.
  - There was statistically significant increase in SG II compared to SG I in 1st day.
  - Otherwise, there was no statistically significant difference between studied periods or groups.

Table (5): Prevalence of pneumothorax in the studied groups

Total prevalence	18% (11/60)
PCMV	30% (6/20)
HFOV	15% (3/20)
Non-ventilated	10% (2/20)

HFOV: High Frequency Oscillatory Ventilation, P-CMV: Conventional Mandatory Ventilation.

- **Table (5)** showed the prevalence of pneumothorax in the studied groups
  - In the present study, the prevalence of pneumothorax in Tanta PICU was 18 %.[(P-CMV (30%), HFOV (15%) and Non –ventilated (10%)].

DISCUSSION

Studies addressing the association of pneumothorax to various ventilation strategies are scarce. Such strategies with high incidence of pneumothorax include high PIP and MAP, active expiratory reflex, administration of bag and mask ventilation, endotracheal tube displacement, and an increase in clinical interventions, long TI, and HFOV [(8)] .

The current study as regarding HR in P-CMV group showed that there was increase in 1st and 2nd days compared to 3rd day in SG I. Also, there was increase in 1st compared to 3rd day in SG II. Too, there was increase in SG II compared to SG I in 1st day. In HFOV there was increase in 1st compared to 3rd day in SG I and SG II. This was in accordance with **Hsu and Sun** [(9)] who reported that, patients with pneumothorax present with tachycardia, chest pain, tachypnea, agitation, hypotension, cyanosis or consciousness change. Tachycardia is the most common finding. **Aslan *et al.*** [(10)] reported that although tachycardia and hypotension were observed in neonates with pneumothorax prior to thoracentesis, the severity or stage of pneumothorax was not stated. Likewise**,** **Waisman *et al.*** [(11)] who reported that pneumothorax caused a gradual decrease in MABP with an increase in

HR. This may be explained by that pneumothorax was associated reduction of arterial blood pressure.

The current study showed that regarding MABP in P-CMV there was decrease in 1st compared to 3rd day in SG II. Also, there was decrease in SG II compared to SG I in 1st day. In HFOV; there was increase in 1st and 2nd compared to 3rd day in SG I. Also, there was decrease in SG II than SG I in 1st day. In non-ventilated; there was decrease in SG II in 1st and 3rd days. This was in accordance with **Waisman *et al.*** [11] who reported that pneumothorax caused a gradual decrease in MABP with an increase in HR. Also, **Temesvári *et al.*** [12] who created a unilateral pneumothorax by injecting 60 mL/kg of air in newborn piglets, HR remained unchanged but MABP significantly reduced, whereas both HR and MAP reduced in bilateral pneumothorax model. This may be explained by that pneumothorax reduces arterial blood pressure and narrows pulse pressure by obstructing venous return and reducing cardiac output.

The present study showed that regarding pH in P-CMV there was decrease in 1st day compared to 2nd and 3rd days in SG II. Also, there was decrease in SG II than SG I in 1st, 2nd days. In HFOV; there was decrease in 1st compared to 2nd and 3rd days. Also, there was decrease in 2nd compared to 3rd day in SG I. Too, there was decrease in SG II than SG I in 1st day. In non-ventilated; there was decrease in SG II compared to SG I in 1st day. This was in accordance with **Liu *et al.*** [13] who noticed that during the development of pneumothorax, there were significant decreases in arterial pH. This may be explained by that the development of pneumothorax causes hypercapnia and decrease in pH. This was in contrast with **Ogata *et al.*** [14] who reported that, pH and PCO2 did not change significantly after pneumothorax.

The current study showed that regarding PaCO2 in P-CMV there was increase in 1st compared to 3rd day in SG I. Also, there was increase in 1st compared to 2nd and 3rd days in. Too, there was increase in 2nd compared to 3rd day in SG II. Likewise, there was increase in SG II compared to SG I in 1st day. In HFOV***:*** there was increase in 1st compared to 2nd and 3rd days in SG I. Also, there was increase in 1st compared to 2nd day in SG II. In non-ventilated: There was increase in SG II compared to SG I in 1st day. This was in accordance with **Ozer *et al.*** [15] who noticed that, the development of a pneumothorax with ensuing hypoxia and hypercapnia is a potentially life-threatening condition and 30% of the infants in the present study died in NICU. This was in contrast with **Ogata *et al.*** [14] who reported that, PCO2 did not change significantly after pneumothorax. However, **Liu *et al.*** [13] noticed that during the development of pneumothorax, there were significant decreases in arterial pH, PO2, and bicarbonate level, and significant increases in base deficit. Arterial PCO2 did not change significantly. With the progression of pneumothorax, PaO2 and SaO2 changed and followed by base deficit and bicarbonate level with significant acidemia with decreased arterial pH. Though, **Waisman *et al.*** [11] reported that PaCO2 decreased from 38.1 ± 4.2 mmHg at baseline to 32.9 ± 3.4 mmHg (29.2 ± 12.0 min after PTX onset). Thereafter, the PaCO2 increased back to 37.7 ± 4.8 mmHg, (49.8 ± 13.7 min after PTX onset). This may be explained as during the development of pneumothorax, hypoxia occurs due to an imbalance of ventilation and perfusion, in addition to the development of lung hypoventilation and increasing intrapleural pressure.

The present study showed that regarding PaO2 in P-CMV: there was decrease in 1st compared to 2nd and 3rd days in SG I. Also, there was decrease in 2nd compared to 3rd day in SG I. Too; there was decrease in 1st and 2nd compared to 3rd day in SG II. Likewise, there was decrease in SG II compared to SG I in 2nd day. In HFOV: There was decrease in 1st compared to 2nd and 3rd days in SG I. Also, there was decrease in 1st compared to 3rd day in SG II. Also, there was decrease in SG II compared to SG I in 1st day. This was in accordance with **Ogata *et al.*** [14] who reported that, PaO2 decreased significantly in 17 infants with pneumothorax. Also, **Norris *et al.*** [16] evaluating 12 patients diagnosed with pneumothorax, 9 patients (75%) had a PO2≤80 mm Hg, and 2 patients, who were both diagnosed with secondary pneumothorax, had a PO2≤55 mm Hg. Too, **Waisman *et al.*** [11] reported that there was decreases PaO2 came very late in all cases. This may be explained by that during the development of pneumothorax, hypoxia occurs due to an imbalance of ventilation and perfusion; in addition to the development of lung hypoventilation and increasing intrapleural pressure. The present study showed that regarding SaO2 in P-CMV: there was decrease in 1st compared to 2nd and 3rd days in SG I. Also, there was decrease in 1st compared to 2nd and 3rd days in SG II. Too, there was decrease in SG II compared to SG I in 3rd day. In HFOV: there was decrease in 1st compared to 3rd day in SG II. Regarding non-ventilated: there was decrease in 1st and 2nd compared to 3rd day in SG I. This in accordance with **Aslan *et al.*** ([10]) who found a significant decrease in SaO2 when pneumothorax developed in mechanically ventilated neonates. Also, **Waisman *et al.*** [11] found that there was decreases in SaO2 and PaO2 came very late in all cases. In six experiments the SaO2 dropped below 90 % only after 46.6 ± 11.3 min after pneumothorax onset. This may be explained by that during the development of pneumothorax, hypoxia occurs due to an imbalance of

ventilation and perfusion, in addition to the development of lung hypoventilation and increasing intrapleural pressure.

The present study showed that regarding OI in P-CMV: there was increase in 1st compared to 2nd and 3rd days in SG I. Also, there was increase in 1st compared to 3rd day in SG II. Too, there was increase in SG II compared to SG I in 1st, 2nd and 3rd days. In HFOV: There was increase in 1st compared to 2nd and 3rd days in SG I. There was increase in 2nd compared to 3rd day in SG II. There was increase in SG II compared to SG I in 2nd and 3rd day. The present study showed that regarding OSI in P-CMV; there was increase in SG II compared to SG I in1st and 2nd days. In late HFOV**,** there was ***increase*** in 1st day in SG II compared to SG I. This was in accordance with **Chen *et al.*** [17] who perform retrospective analysis on the 23 neonates with pneumothorax who received HFOV. Of the 23 cases, 19 cases were treated by HFOV as soon as they were diagnosed with pneumothorax, and 4 cases were

treated by HFOV after the occurrence of pneumothorax during (CMV) (CPAP) ventilation. Another 23 neonates with pneumothorax who received CMV in the same period were selected as controls. Both groups showed significantly decreased OI. This may be explained by that pneumothorax associated with decrease oxygenation with increase FiO2 needs. The present study showed that regarding lung compliance, there was decrease in SG II compared to SG I. Regarding airways resistance, there was there was increase in SGII compared to SGI. This is in accordance with **Salihoglu *et al.*** [18] who reported that there was decrease in lung compliance and increase in airway pressure when pneumothorax occurs during laparoscopic surgery. This may be explained by that pneumothorax affects mechanical function of the lung by impairing full expansion of lung and restriction of lung volume.

The present study showed that regarding FiO2 in P-CMV: there was increase in 1st and 2nd compared to 3rd day In SG I. Also, there was increase in 1st and 2nd compared to 3rd day in SG II. Too, there was increase in SG II compared to SG I in 1st, 2nd and 3rd day. In HFOV: there was increase in SG II compared to SG I in 1st day. This in accordance with **Shih *et al.*** [19] who demonstrated that a higher FiO2 (60% to 100%) is able to significantly shorten the resolution time of pneumothorax. Also, **Zierold *et al.*** [20] found that supplemental FiO2 (40% and 60%) accelerated the resolution of pneumothorax by 1.5- fold and 2.8-fold, respectively. This may be explained by that the administration of a high concentration of oxygen may reduce the total pressure of gases in pleural capillaries by reducing the partial pressure of nitrogen. This should increase the pressure gradient between the pleural capillaries and the pleural cavity, thereby increasing absorption of air from the pleural cavity.

The present study showed that regarding PIP: there was increase in SG II compared to SG I in 1st, 2nd and 3rd day. Increase in airway pressure and PIP following pneumothorax. After the placement of the chest drain, PIP decreased. Also, **Ellsbury *et al.*** [21] who reported that: It is most likely that the absence of high-PIP, the very short TI and small VT applied at higher frequencies may result in a rapid decrease of air leak, as showed by in an animal model of pneumothorax. Also, **Malek *et al.*** [22] reported that one of the factors in development of pneumothorax is the artificial ventilation setting. An increased risk of pneumothorax was associated with maximal PIP.

The present study showed that regarding **PEEP**: There was increase in SG II compared to SG I in 2nd and 3rd day. This was in accordance with **Chiche *et al.*** [23] who applied the 5cmH2O PEEP method in 5 pneumothorax cases during laparoscopic surgery. They reported that arterial blood-gas values improved, whereas raw and air pressure were decreasing. However**, Klinger *et al.*** [24] reported that decreasing the risk of pneumothorax requires intensive control of ventilation, including optimizing PEEP and minimizing PIP. This may be explained by that application of 5cmH2O PEEP has been suggested as an alternative means for a chest drain. It has been proposed that by using the PEEP method, the lungs can expand and supply enough gas exchange and the consequences can be seen in arterial blood gas samples and in the values of MV parameters. The present study showed that regarding SV, in P- CMV, there was decrease in 1st compared to 2nd and 3rd days in SG I. Also, there was decrease in 1st and 2nd compared to 3rd day in SG II. Also, there was decrease in SG II compared to SG I in 1st day. In HFOV: There was decrease in 1st compared to 3rd day in SG II. The present study showed that regarding SVI in P-CMV: there was decrease in 1st compared to 2nd day in SG I. Also, there was decrease in 1st compared to 3rd day in SG II. Also, there was decrease in SG II compared to SG I in 1st day. This in accordance with **Gustman *et al.*** [25] who reported that HR and SV showed a tension-dependent increase then decrease, respectively, consistent with the observed failure of cardiac output to change significantly. This may be explained by that pneumothorax cause increase in intrathoracic pressure and decrease in venous return and preload. The present study showed that regarding *CO* in P-CMV: there was decrease in 1st compared to 3rd day in SG II. Also, there was decrease in SG II compared to SG I in 1st day. In HFOV: There was decrease in 1st compared to 3rd day in SG II. Too, there was increase in SG II group compared to SG I in 1st day. This was in accordance with **Cournand et al.**

[26] who found there was a decrease in cardiac output while the subject had pneumothorax, partial or complete. This in contrast with **Gustman *et al.*** [25] who demonstrated that the maintenance of cardiac output, even in the presence of a large tension pneumothorax. This may be explained by that pneumothorax cause increase in intrathoracic pressure and decrease in venous return consequently decrease in CO. The present study showed that regarding **CI** in **HFOV:** there was decrease in SG II compared to SG I in 1st day. This in accordance with **Beards and Lipman *et al.*** [27] who reported that when hemodynamic measurements were performed in their pneumothorax cases, there was a marked decrease in cardiac index. Also, **Gustman *et al.*** [25] observed that sheep with tension pneumothorax that were breathing spontaneously showed no decrease in the cardiac index, whereas the cardiac index diminished significantly in those which were on MV. This may be explained by the decrease in CO that occurs with pneumothorax.

The present work showed that regarding SVR in P-CMV, there was increase in SG II compared to SG I in 2nd day. Regarding SVRI in P-CMV: there was increase in 1st compared to 2nd and 3rd days In SG II. Also, there was increase in SG II compared to SG I in 1st day. This in accordance with in accordance with **Connolly *et al.*** [28] who found that patients with pneumothorax whom all the hemodynamic and blood gases measurements were measured; there was increase in central venous, pulmonary artery pressures, and decrease of the cardiac output, consistent with the pneumothorax but maintaining the blood pressure at the expense of increased systemic vascular resistance. This may be explained by that pneumothorax cause decrease in MABP and CO.

The present study showed that regarding ***SOFA score*** there was increase in SG II compared to SG I in P-CMV, HFOV and non-ventilated groups. Regarding ***PRISM*** there was increase in SG II compared to SG I in P-CMV, HFOV and non-ventilated groups. This in accordance with **Ogata *et al.*** [14] who reported that pneumothorax is a life-threatening condition associated with a high incidence of mortality and morbidity among neonates. **de Lassence *et al.*** [29] Also reported that pneumothorax was found to be an independent predictor of mortality during MV and was associated with a significant increase in the ICU length of stay, hospital stay and mortality in all mechanically ventilated patients. This may be explained by that pneumothorax is life threatening condition associated with a high incidence of mortality and morbidity.

The present study showed that regarding univariant analysis it was significant in HR, RR, pH, PCO2, HCO3, PaO2, SaO2, OI, MAP, FIO2, PIP, CO, CI, SVRI, SOFA and PRISM III score. Ironically, multivariant analysis showed non-significance. This may be explained by the small number of the studied cases. This was in accordance with, **El-Nawawy *et al.*** [30] who found that PIP, PaCO2, FiO2, MAP and HCO3 were significant risk factors for the occurrence of pneumothorax. The present study showed that regarding the prevalence of pneumothorax in the studied groups in Tanta PICU was 18 %.(PCMV (30%), HFOV (15%) and Non –ventilated (10%). This was in accordance with, **El-Nawawy *et al.*** [30] who found that in his study; the prevalence of pneumothorax in Alexandria PICU during the 5-year study was 10.4%.

In conclusion: Incidence of pneumo-thorax in patients on P-CMV was higher than whom on HFOV and non- ventilated group. Pneumothorax in P-CMV patients occur with higher ventilatory settings. The significant risk factors of pneumothorax in our study were HR, RR, pH, PCO2, HCO3, PaO2, SaO2, OI, MAP, FIO2, PIP, CO, CI, SVRI, SOFA and PRISM III score. Mortality rate was higher among patients with pneumo-thorax.

REFERENCES

1. **Kuo P, Nong B, Huang Y *et al.* (2018):** Primary spontaneous pneumothorax in children: A literature review. Pediatric Respirology and Critical Care Medicine, 2(2): 25-31.
2. **Garcia-Munoz Rodrigo F, Urquia Marti L, Galan Henriquez G *et al.* (2017):** Perinatal risk factors for pneumothorax and morbidity and mortality in very low birth weight infants. The Journal of Maternal-Fetal & Neonatal Medicine, 30(22):2679-2685.
3. **Pollack M, Holubkov R, Funai T *et al.* (2016):** The pediatric risk of mortality score: update 2015. Pediatric critical care medicine: a journal of the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies, 17(1): 2-9.
4. **Jones A, Trzeciak S and Kline J (2009):** The Sequential organ failure assessment score for predicting outcome in patients with severe sepsis and evidence of hypoperfusion at the time of emergency department presentation. Critical Care Medicine, 37(5): 1649.
5. **Kao H, Lai T, Hung H *et al.* (2013):** Sequential oxygenation index and organ dysfunction assessment within the first 3 days of mechanical ventilation predict the outcome of adult patients with severe acute respiratory failure. The Scientific World Journal, 2013(3):43-26.
6. **Rimensberger P, and Bachman T (2014):** It is too early to declare early or late rescue high-frequency oscillatory ventilation dead. JAMA Pediatrics, 168(9): 862-863.
7. **Penny J, Anthony J, Shennan A *et al.* (2000):** A comparison of hemodynamic data derived by pulmonary artery flotation catheter and the esophageal Doppler monitor in preeclampsia. American Journal of Obstetrics and Gynecology, 183(3): 658-661.

8. **Ethawi Y, Mehrem, A, Minski J *et al.* (2016):** High frequency jet ventilation versus high frequency oscillatory ventilation for pulmonary dysfunction in preterm infants. https://www.researchgate.net/.../302033091_High_frequency_jet_ventilation_versus_hig
9. **Hsu C and Sun S (2014):** Iatrogenic pneumothorax related to mechanical ventilation. World Journal of Critical Care Medicine, 3(1): 8-14.
10. **Aslan A, Karagüzel G, Gura A *et al.* (2006):** Lung re-expansion and urinary lipid peroxidation in neonatal pneumothorax. European Journal of Pediatric Surgery, 16(05): 307-311.
11. **Waisman D, Faingersh A, Levy C *et al.* (2013):** Transient decrease in PaCO 2 and asymmetric chest wall dynamics in early progressing pneumothorax. Intensive Care Medicine, 39(1): 137-145.
12. **Temesvári P, Karg E, Bódi I *et al.* (2001):** Impaired early neurologic outcome in newborn piglets reoxygenated with 100% oxygen compared with room air after pneumothorax-induced asphyxia. Pediatric Research, 49(6): 812-819.
13. **Liu, J, Joynt C, Tymafichuk C *et al.* (2009):** Sequential changes of hemodynamics and blood gases in newborn piglets with developing pneumothorax. Pediatric Pulmonology, 44(1): 70-75.
14. **Ogata E, Gregory G, Kitterman J *et al.* (1976):** Pneumothorax in the respiratory distress syndrome: incidence and effect on vital signs, blood gases, and pH. Pediatrics, 58(2): 177-183.
15. **Ozer E, Ergin A, Sutcuoglu S *et al.* (2013):** Is pneumothorax size on chest x-ray a predictor of neonatal mortality? Iranian Journal of Pediatrics, 23(5): 541-545.
16. **Norris R, Jones J, Bishop J (1968).** Respiratory gas exchange in patients with spontaneous pneumothorax. Thorax, 23(4): 427-433.
17. **Chen D, Huang X, and Li X (2012):** Clinical application of high-frequency oscillatory ventilation for the treatment of neonatal pneumothorax. Chinese Journal of Contemporary Pediatrics, 14(7): 499-501.
18. **Salihoglu Z, Demiroluk S, Demirkiran O *et al.* (2008):** The effects of pneumothorax on the respiratory mechanics during laparoscopic surgery. Journal of Laparoendoscopic & Advanced Surgical Techniques, 18(3): 423-427.
19. **Shih C, Tsai W, Huang S *et al.* (2012):** Effects of high concentration oxygen treatment on traumatic pneumothorax in adult rabbits. Chin J Physiol, 55(3): 178-83.
20. **Zierold D, Lee S, Subramanian S and DuBois J (2000):** Supplemental oxygen improves resolution of injury-induced pneumothorax. Journal of Pediatric Surgery, 35(6): 998-1001.
21. **Ellsbury D, Klein J, and Segar J (2002):** Optimization of high-frequency oscillatory ventilation for the treatment of experimental pneumothorax. Critical Care Medicine, 30(5): 1131-1135.
22. **Malek A, Afzali N, Meshkat M , Yazdi N (2011):** Pneumothorax after mechanical ventilation in newborns. Iranian Journal of Pediatrics, 21(1): 45. -50
23. **Chiche J, Joris J and Lamy M (1994):** PEEP for treatment of intraoperative pneumothorax during laparoscopic fundoplication. Britch Journal of Anesthesia, 72: 20-25.
24. **Klinger G, Ish-Hurwitz S, Osovsky M *et al.* (2008):** Risk factors for pneumothorax in very low birth weight infants. Pediatric Critical Care Medicine, 9(4): 398-402.
25. **Gustman P, Yerger L and Wanner A (1983):** Immediate cardiovascular effects of tension pneumothorax. American Review of Respiratory Disease, 127(2):171-174.
26. **Cournand A, Bryan N, and Richards D (1935):** Cardiac output in relation to unilateral pneumothorax in man. The Journal of Clinical Investigation, 14(2): 181-189.
27. **Beards S, Lipman J (1994):** Decreased cardiac index as an indicator of tension pneumothorax in the ventilated patient. Anesthesia, 49(2): 137-141.
28. **Connolly J (1993):** Hemodynamic measurements during a tension pneumothorax. Critical Care Medicine, 21(2): 294-295.
29. **de Lassence A, Timsit J, Tafflet M *et al.* (2006):** Pneumothorax in the intensive care unit: incidence, risk factors, and outcome. Anesthesiology, 104(1): 5-13.

El-Nawawy A, Al-Halawany A, Antonios M , Newegy R (2016): Prevalence and risk factors of pneumothorax among patients admitted to a Pediatric Intensive Care Unit. Indian Journal of Citical Care Medicine. Indian journal of Critical Care Medicine, 20(8):453 -458.