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Doppler Echocardiographic Changes in Respiratory Diseases

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1. Introduction

The heart is one of the most important organs which can be affected by several systems including respiratory system. The in common function of both heart and lung is to provide tissues with oxygen and to get rid of the Carbon dioxide. So; they work in harmony and interact in a proper way with each other. This interaction may be neural, mechanical and/or humeral. The neural effects are either central through the brain stem centres in medulla oblongata or peripheral through baro-reflexes or lung-stretch reflexes. Being together inside the thoracic cavity; the heart can be affected by lung inflation and deflation. This is called mechanical heart–lung interactions (Figure 1).
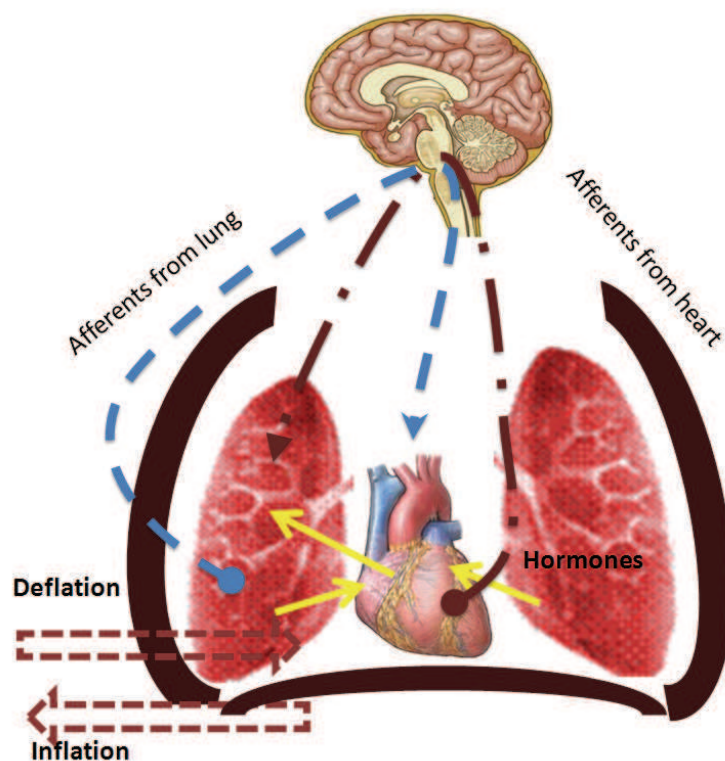

Fig. 1. The heart-lung interaction; neural, mechanical and humoral

The lung may act as an endocrine gland secreting certain hormones or substances that can affect heart functions forming what is known as humoral heart–lung interaction. The lung helps in deactivation of 5-hydroxytryptmine, acetylcholine, norepinephrine, bradykinin and prostaglandins E1, E2 and F2. It produces angiotensin converting enzyme that helps in activation of angiotensin I to angiotensin II and helps in synthesis of prostaglandins E1, E2. All these humoral products can affect the heart in a way or another. Atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) are two polypeptide hormones secreted by the atrial cardiocytes. The ANP is well known to have a bronchodilating effect more prominent in the larger central airways than in the peripheral airways. The lung also supplies the cardiac muscle with the needed oxygen and the heart supplies the respiratory muscles with oxygenated blood. So; respiratory pump failure may occur if the heart cannot supply the needed requirement. These heart-lung interactions occur all the time with every breath and every cardiac cycle. However; their effects are subtle under normal circumstances but become significant in certain pathological situations *(Said 2001)*.

2. Respiration induced changes in echocardiography

Respiration can bring on physiological changes of cardiovascular haemodynamics. These changes are mostly related to changes in intra-thoracic and intra-abdominal pressure which can affect both systemic and pulmonary venous return. Respiration can also affect the intra-pericardial pressure which in turn affects the filling of the four cardiac chambers. Respiration can affect all the modalities of the echocardiographic examination. It can affect the image quality as well as the measurements in Two-Dimensional echocardiography, M-mode and Doppler studies.

2.1 The image quality

The respiration may affect the image quality of the echocardiographic study especially when visualizing the posterior wall of the left ventricle (LV). Even normal respiration may have undulating, distorting, and blocking effect during echocardiographic examination of the heart; particularly the posterior wall of LV to the extent that the patient may need to hold his breath in order to obtain clear echoes (*Fenichel et al 1976*).

These effects are due to both physiological and anatomical changes that occur with respiration. The physiological changes are due to pressure variations (intra-thoracic, intrapericardial and intra-abdominal), changes in systemic and pulmonary venous return, diminished LV filling pressure during inspiration, pericardial constraint, ventricular interdependence and variation in the angle of incidence of the transducer beam. The anatomical changes are due to the diaphragmatic movements, lung inflation and with interference and dropping out of the lateral shadows together with longitudinal rotation and posterior motion of heart. Inspiration increases the antero-posterior diameter of the chest, and the lungs expand particularly anteriorly, and fill the space previously occupied by the heart. However; sub-costal view is an exception where the heart images become clearer with inspiration. However with deep inspiration; the abdomen becomes too tense to allow the transducer to be placed under the sternum. So; half a breath is preferred during sucostal examination of the heart. Lung deflation by expiration (particularly in steep left lateral position with left arm up to spread the ribs) can improve the image quality especially with the parasternal and often apical view of the heart. Left lateral rotation of the patient will deflate the left lung and avoid the pulmonary interference and improve the clarity of

echocardiograms. Improving the quality of image can be done by using device-driven systems, which can acquire signal averaging of echocardiographic frames over multiple cardiac cycles (*Ginghina et al 2009, Lang 2005, Klingler 1989*)

2.2 Two-D and M-mode measurements

The respiration does not only affect the image quality but also can alter the measurement taken during 2-D and M-mode study. There is an inspiratory decrease in LV end diastolic dimensions as measured by M-mode due to reduction of the LV filling during inspiration. Respiration also can change the position of the heart relative to the location of the echo transducer which can cause measurement error due to anatomical translation and rotation of the heart, which may change with respiratory phases. There may be a significant inspiratory decrease of LV diastolic dimension while the inspiratory decrease of LV mean systolic dimension is usually insignificant. The echocardiography-derived inspiratory decrease in stroke volume is about 16% (*Brenner et al 1978*). This reduction in the LV dimensions during inspiration may be due actual reduction of LV stroke volume as a result of decrease preload due to decrease diastolic filling, increased after load (due to increased impedance to LV emptying) or due to the anatomical medial rotation of the short axis of the heart during inspiration. There is an existed consistent relation between the respiratory excursion and the resultant displacement of the recorded echoes (*Fenichel et al 1976*).

	Findings in deep Inspiration
Anterior echoes	Increased especially with deep inspiration
Posterior echoes	Displaced posteriorly (about 2 cm). The echogenicity may decreased or even eliminated.
Septum	Can be seen displaced posteriorly just before and after the appearance of dense mass tissue
Posterior wall of LV	There is blunting of its systolic anterior motion and decreased its pulsation but still can be identified.
RV end-diastolic volume	Increased
LV end-diastolic volume	Decreased
Total end-diastolic heart volume	Unchanged
Mitral leaflets	Echos are displaced posteriorly but the opening and closing slopes remain the same
Aortic root and semilunar valves	Posterior deviation up to 1 cm
Posterior wall of the left atrium	Posterior deviation parallel to the aortic root.
Tricuspid valve	Displaced posteriorly

Table 1. The changes in M-mode and 2-D measurements observed during deep inspiration

In a Study done in 1992, the effects of the quiet respiration and body position on RV size and function using 2-D and M-mode echocardiography were observed in 15 healthy children. All the end-diastolic and end-systolic echocardiographic dimensions, areas, and volumes increased slightly but significantly with inspiration. RV ejection fractions were found to be significantly higher during inspiration, as were stroke volume indices. The study also found that RV dimensions increased from supine to left lateral decubitus position (*Norgård and Vik-*

Mo 1992). On the other hand a previous study done in 1979 found that the changes observed in RV and LV diemensions during respiration were opposite in direction and approximately equal in magnitude, so that total internal diastolic cardiac dimension remained essentially constant (*Lendrum et al 1979*).

These respiration-related changes in the cardiac measurements should be considered while doing serial follow up measurements of LV functions during various hemodynamic interventions. These changes should also be considered in doing any correlation between echocardiographic dimensions and other methods of assessing LV function. To obtain more accurate measurement during two-dimensional quantitation; the American Society of Echocardiography recommended to obtain the image during quiet or suspended respiration (at end-expiration). However, many patients may not be able to follow even careful instructions. If the images are to be obtained during held end-expiration; the patient is advised to avoid Valsalva manoeuvre, as it can degrade image quality and produces undesired physiologic changes that disrupt the basic measurements. Table 1 showed the 2-D and M-mode findings observed in deep inspiration

2.3 Doppler measurements

During inspiration; the blood flow increases in the right side of the heart and decreases in the left side because of the decreased flow out of the pulmonary veins into the left atrium and left ventricle. The reverse occurs during expiration. These respiratory-induced changes in the intra-cardiac blood flow were reflected in the Doppler estimated trans-mitral and trans-tricuspid blood inflow as well as the trans-pulmonary and trans-aortic blood outflow.

Respiration does not only affect the intracardiac blood inflow and outflow; but also affecting the intracardiac pressures. The right atrial pressure which is needed to estimate the systolic right ventricular, pulmonary artery pressures and left atrial pressure; can be expected by the changes in the diameter of the IVC during inspiration. Respiratory variation in SVC systolic forward flow may be a useful Doppler flow index for expecting the systolic pulmonary pressure and to assess the severity of pulmonary hypertension. During inspiration the E/A ratio of transmitral flow may be reduced but of no significance in healthy subjects. However this effect may become significant in certain cardiac diseases as in coronary artery diseases and abnormal LV relaxation pattern (*Ginghina et al 2009*).

2.3.1 Mitral and tricuspid inflow

The normal changes in the intra-thoracic pressure occurring during normal breathing have insignificant effects on the trans-mitral and trans-tricuspid flow. However these changes become significant with strained breathing. *Uiterwaal et al 1989* found that the maximum velocity during early diastole of right ventricle (VmaxE) and during atrial contraction (VmaxA) were significantly higher during inspiration than during expiration. They found the reverse on the mitral side; as the VmaxE and VmaxA were significantly lower during inspiration than during expiration. *Tsai et al* 1998 found that early diastolic peak flow velocity and flow velocity integral, the ratio of early/late diastolic peak flow velocity, and the ratio of early/late diastolic flow velocity integral at end-inspiration were significantly lower than those at end-expiration. The mitral peak E velocity decreased by 4-9% while the A wave remains unchanged. On the other hand the Tricuspid peak E velocity increased 15% while the A wave increased by 10% which maintain E/A ratio to be unchanged.

Riggs and Snider 1989 found reduction of early diastolic LV filling parameters in normal children (decreased E wave by 8 %) while active atrial emptying (peak A velocity and

A/total area ratio) may remain unchanged with respiration. This led to marked reduction of transmitral E/A ratio (by 14%). The effect was marked on the right side of the heart. The tricuspid peak E increased by 26% while peak A velocities increased by 18%. The E/A ratio remained unchanged. *Yuan et al 2004* quantitatively investigated the effect of the different intra-thoracic pressure on the blood flow velocities across the four cardiac valves. They found significant difference from the transvalvular velocities recorded during spontaneous respiration with the velocities recorded during various intrathoracic pressures. They concluded that the respiratory intrathoracic pressure changes may cause change in the velocity across the valves. However *Riggs et al 1989* showed that the inspiratory changes in Tricuspid valve Doppler indexes had less marked changes in neonates. This could be due to the faster heart and respiratory rates plus the reduced right ventricular compliance. To minimize the effect of respiration on the trans-mitral and trans-tricuspid flow velocities, the patient is advised to hold breathing in shallow end-expiration.

2.3.2 Aortic and pulmonary outflow

Ferreira T et al 1990 found significant higher peak early velocity in the inflow tract and in the maximal velocity in the outflow tract of the LV during expiration. There were also no significant changes in the time intervals observed in their study. *Buda et al 1979* studied the effects of Valsalva and Müller manoeuvres on LV functions. They observed that the negative intra-thoracic pressure may affect LV functions by increasing LV transmural pressures and consequently after load. The same observation was documented by *Buda et al 1981* who showed that deep inspiration decreased the LV outflow tract (LVOT) gradient and decreased the LV ejection time in patients with muscular subaortic stenosis. They linked this decrease in the pressure gradient across LVOT to the increasing LV afterload through an increase in LV transmural pressure as a result of the negative intra-thoracic pressure caused by the deep inspiration. *Weyrnan et a1 1973* studied the specific effects of respiratory movements on the recorded echocardiogram. They found an influence of respiratory changes on the pulmonary arterial pressure and its pulmonary valvular echoes.

2.3.3 Inferior vena cava and hepatic veins

The inferior vena cava and the hepatic veins flows are best recorded from the subcostal sagittal view; where the forward flow is away from the transducer (below the baseline) and the retrograde flow is towards the transducer. This flow is continuous with one large peak in systole (S wave) which coincides with relaxation of the right atrium and the descent of tricuspid annulus during RV systole. There is another large peak during diastole (D wave) which occurs during the rapid filling phase of ventricular diastole. A third wave (A wave) occurs in some normal subjects due to reversed flow secondary to right atrial contraction. There is a fourth wave in the hepatic flow called V wave which is due to reverse flow in late systole (*Reynolds et al 1991; Lee et al 2007*). The respiration induces marked changes in Doppler velocity curves of hepatic veins. During inspiration there is a significant increase in both normal forward systolic (S) and diastolic (D) flow velocities as well as retrograde A velocity (*Ginghina et al 2009*). During expiration, there is decreased diastolic flow and increased reversals. The RA pressure can be determined by the respiratory variation in inferior vena cava diameter observed through the subcostal window; where the diameter of the IVC decreases in response to inspiration with minimal size observed at end inspiration (*Kircher et al 1990*). However, the position of the patient must be considered, as the largest diameter is measured in the right lateral position, becomes intermediate in the supine

position, and the smallest diameter is measured in the left lateral position. For an adult; the normal IVC diameter is less than 1.7 cm and there is a 50% decrease in the diameter when the RA pressure is normal (0–5 mm Hg). When the IVC diameter is dilated (>1.7 cm) with normal inspiratory collapse (>50%), the RA pressure is suggestive to be mildly elevated (6–10 mm Hg). However when the inspiratory collapse is less than 50%, the RA pressure is usually between 10 and 15 mm Hg. If the IVC is dilated without any collapse; this suggests markedly elevation of RA pressure (greater than 15 mm Hg). With intravascular volume depletion, the IVC is small (usually < 1.2 cm) with spontaneous collapse (*Ginghina et al 2009*).

2.3.4 Superior vena cava

The superior vena cava flow can be best recorded from the suprasternal notch or subcostal view. The flow has the same wave like the IVC flow. It lacks the A wave of hepatic flow. However; the effect of respiration on SVC flow waves is less marked than in the hepatic flow; perhaps because of the pressure differences in the abdominal and thoracic cavities with respiration (*Reynolds et al 1991, Lee et al 2007*). Respiration makes appropriate fixed pulsed wave Doppler trace sampling to be difficult due the continuous movement of pulsed Doppler sample volume as a result of the change in heart position and diaphragmatic movement. Errors due to respiratory movement during Doppler estimation must be considered and manoeuvres that minimize the effect of respiration should be tried (*Kircher et al 1990*). The following measures may help to minimize the effect of respiration on Doppler studies:

1. Holding breathing at end expiration. Even patients with heart failure can do multiple holding of their breathing for 6 seconds.
2. Avoidance of angle of incidence by parallel positioning.
3. Taking the average velocities during multiple consecutive 3-5 quality tracing.

3. Respiratory maneuvers that may help in diagnosis of cardiac diseases

3.1 Valsalva maneuver

It is performed by attempting forceful expiration against a closed airway when the mouth is closed and the nose is pinched. There are 4 phases occurring during this maneuver. In the first phase; there is rise of the blood pressure coincide with onset of straining and increase the intra-thoracic pressure. The second phase coincides with the decrease in venous return and consequent reduction of stroke volume and pulse pressure and increase in heart rate. In the third phase, there is a release of straining; allowing re-expansion of the pulmonary vessels and the aorta causing increase of the pulmonary blood flow and cardiac output starts to increase. The last phase; there is marked increase of venous return which increase the cardiac output and leads to blood pressure overshoot (in normal heart) and return of the heart rate to the baseline. Performing Valsalva maneuver during echocardiography can help in diagnosis and evaluation of certain cardiac disorders e.g. aortic and pulmonic stenosis as well as tricuspid regurgitation (*Stoddard et al 1993*). It is also helpful in diagnosis of hypertrophic cardiomyopathy, and mitral valve prolapse (MVP). In patients with obstructive hypertrophic cardiomyopathy and mild or dynamic LV outflow tract obstruction; Valsalva maneuver can unmask latent gradients/to increase LVOT gradient specially during the straining phase of Valsalva maneuver. During this phase, the preload, end-diastolic LV volume and after load decreased. So the systolic anterior motion (SAM)

occurs earlier in systole, and mitral-septal contact lasts longer and LV outflow tract gradient increases (*Ginghina et al 2009*).

Valsalva maneuver helps also to augment the diagnosis of mitral valve prolapse, as it increases the intensity of mitral regurgitation associated with MVP and it makes regurgitation to start earlier in systole due to reduction of left ventricular volume. Patent foramen ovale is another cardiac disease in which Valsalva maneuver can help diagnosis. It can be reliably detected with contrast echocardiography or by using agitated saline through either trans-thoracic or trans-esophageal echocardiography. During Valsalva maneuver, the atrial shunting from right to left will be enhanced and begin during the third (release) phase. However, *Stoddard et al 1993* found that cough test is superior to the Valsalva maneuver in delineating a patent foramen ovale during contrast trans-esophageal echocardiography. Valsalva maneuver can be used for better assessment of the cardiac functions. The transmitral Doppler flow can be used to assess the diastolic functions of LV. There are small changes (<15%) in transmitral peak flow velocities that occur with spontaneous respiration. Valsalva maneuver was found to be effectively unloading the heart and unmask an impaired relaxation pattern and high filling pressures in patients with a baseline pseudonormal flow pattern (*Yuan et al 2007*).

3.2 Mûller maneuver

The Mûller maneuver is used to augment tricuspid regurgitation and is rarely used during echocardiographic examination. It is performed by holding the breathing in inspiration while the nose is closed and the mouth is sealed for 10 seconds. This exaggerated inspiratory effort, will increase the negative pressure in the chest and lungs is made very subatmospheric. This will augment the right-sided filling and hence augment the tricuspid regurgitation (*Buda et al 1981*).

4. Echocardiography in various respiratory conditions

Table 2 summarizes the indications of echocardiography in respiratory diseases.

a. Congenital diseases:
1. Choanal atresia
2. Congenital mal-development of the lung: Agenesis, hypoplasia, and dysplasia of lung
3. Congenital hiatus hernia or diaphragmatic hernia
4. Chest wall deformity as in Pectus excavatum; Pectus carinatum; Kyphoscoliotic heart disease
5. Situs inversus e.g. kartagener syndrome
6. Total anomalous pulmonary venous connection
7. Partial anomalous pulmonary venous connection

b. Acquired:
1. Traumatic chest conditions: Pulmonary insufficiency following trauma and surgery; Unspecified injury of heart with open wound into thorax; Contusion of heart with open wound into thorax; other trauma).
2. Obstructive sleep apnea (adult) (pediatric)
3. Reactive airway diseases (Asthma & COPD)
4. Pulmonary Vascular disorders (Aneurysm of pulmonary artery; arteriovenous fistula of pulmonary vessels (Contrast Echocardiography)).

5. Pulmonary embolism
6. Primary pulmonary hypertension, follow-up of pulmonary artery pressures in patients with pulmonary hypertension to evaluate response to treatment,
7. Swelling, mass, or lump in chest, malignancy.
8. Patients being considered for lung transplantation or other surgical procedure for advanced lung disease to exclude possible cardiac disease.
9. Acute respiratory infections: pneumonia especially if associated with empyema or septic shock
10. Chronic pulmonary infection: tuberculosis, fungal disease
11. ICU patient: Respiratory failure, mechanically ventilated patient, exacerbated reactive airway disease; Acute cor pulmonale and for routine re-evaluation of right ventricular function in patients with cor pulmonale, Iatrogenic pulmonary embolism and infarction, acute pulmonary edema; and chest pain: if associated with: hemodynamic instability, unexplained hypotension in Intensive Care or emergency settings;

Table 2. The indications of Echocardiography in respiratory conditions

4.1 Congenital respiratory diseases
4.1.1 Choanal atresia
Choanal atresia results from the persistence of the bucconasal membrane, which separates the nasal cavity and the nasopharynx in the early embryological development period. It may be unilateral or bilateral. Unilateral cases are often diagnosed later in life with unilateral nasal obstruction and discharge, while bilateral atresia almost always present with respiratory emergency. It may be found as isolated anomaly or associated with other defects as seen in CHARGE association. The most common cardiac lesions associated with choanal atresia are PDA and/or VSD, singly or in combination. Over half of the patients with choanal atresia may have multiple cardiac anomalies e.g. Endocardial cushion defect, conotruncal anomalies, or fallot's tetralogy. (*Zagnoev et al 1981*)

4.1.2 Congenital mal-development of the lung
Lung agenesis is defined as complete absence of lung tissue, carina, main bronchus and pulmonary vasculature while in lung aplasia there are a carina and main bronchus without lung tissue. In pulmonary hypoplasia there is bronchial underdevelopment associated with reduced amount of lung tissue. About 50% of patients with lung agenesis have other congenital anomalies especially cardiac anomalies. The incidence is being greater in those with right-sided agenesis. The congenital heart anomalies that may be associated with lung agenesis have a broad range, from simple isolated congeital cardiac defects like atrial septal defcts to extreme dextroversion and displacement of the heart. Tricuspid atresia; pulmonary artery sling; and total anomalous pulmonary venous drainage are among the recorded congnital heart diseases associated with lung agenesis. Some cardiac malformations (eg, tetralogy of Fallot, and scimitar syndrome) may lead to pulmonary hypoplasia. Echocardiography alone may be not enough for detailed diagnosis. Cross-sectional imaging modalities such as magnetic resonance imaging (MRI) and computed tomography (CT) and pulmonary angiography may be needed for accurate determination of anomalous vasculature, particularly in the presence of coexisting lung agenesis (*rzykowski et al 2007*).
Bridging bronchus is a malformation in which an aberrant bronchus arising from the left main stem bronchus to supply the right middle and /or lower lung lobes. Bridging

bronchus may be associated with many malformations like cardiovascular (a sling-like left pulmonary artery and left-sided obstructive lesions including coarctation of the aorta), skeletal, genitourinary and abdominal malformations.

4.1.3 Congenital hiatus hernia or diaphragmatic hernia

Congenital diaphragmatic hernia (CDH) is protrusion of the abdominal viscera into the thoracic cavity due to absence of the diaphragm, or through a hole in it. This can occur on the left or right side, but is most common on the left. Hiatus hernia occurs when part of the stomach is present the thoracic cavity through the esophageal hiatus of the diaphragm. Cardiovascular malformations are common in cases with congenital diaphragmatic hernia (25-50%). Ventricular septal defect (VSD), atrial septal defect (ASD), conotruncal defects such as tetralogy of Fallot, transposition of great vessels, or aortic coarctation are among the cardiac anomalies seen in such cases. Hypoplastic left heart syndrome, is sometimes seen in cases with left-sided CDH. Dextroposition of the heart (e.g., the heart is shifted into the right chest) is also seen in left-sided CDH. Pulmonary hypertension and pulmonary hypoplasia are common complications of CDH. (*Tonks et al 2008*)

Echocardiography is needed immediately after diagnosis of CDH. It is helpful to exclude any associated congenital heart defect. It also helps in assessing cardiac functions and determining any reduction of the LV mass. Increased pulmonary vascular resistance is an almost universal finding in CDH. Determination of pulmonary artery pressures is essential as many surgeons prefer to operate when echocardiography has shown normal pulmonary artery pressures which are maintained for at least 24 to 48 hours (*Bosenberg et al 2008*). Echocardiographic findings of increased pulmonary artery pressure are flattening of the intraventricular septum, presence of tricuspid regurgitation, and/or right-to-left or bidirectional shunting at the ductal level. Estimation of RV pressure can be done by estimation of tricuspid regurgitation jet. Patency of the ductus allows RV to decompress and prevents right heart failure when the pressure becomes suprasystemic (*Bohn 2000*).

4.1.4 Chest wall deformity

There is a wide range of congenital chest wall deformities. The most frequent ones are pectus excavatum (more than 90% of congenital chest wall deformities), pectus carinatum and kyphoscoliosis. Pectus excavatum (Funnel chest) is caused by an abnormality of connective tissue, which results in depression of the sternum. It is sometimes associated with Marfan and Ehlers Danlos syndromes. Pectus carinatum (Pigeon chest) is thought to be associated with rickets, severe childhood asthma; congenital heart diseases as in ventricular septal defect (VSD) or with scoliosis. Kyphoscoliosis may be due to neuromuscular, congenital vertebral deformity or may be idiopathic. The usual cardiac manifestations presented in chest wall deformity are pain, dyspnea, and palpitation. ECG may show incomplete right bundle block, right anterior fascicular block, right axis deviation, or LV hypertrophy (*Kara et al 2009*).

Echocardiography may be indicated in patients with chest wall deformity to evaluate cardiac compression and to exclude associated cardiac abnormalities. In pectus excavatum, the sternum depressed the right atrium and right ventricle, interfering with diastolic filling of these structures. Mitral valve prolapse is a common finding and usually presented with chest pain and palpitations. Evaluation of the aortic root is specially needed if there is suspicion of Marfan syndrome as aortic root dilatation is a common finding. Evaluation of the cardiac function particularly in the sitting, or upright, position is of utmost importance.

No significant impairment to cardiac function could be observed when the patient with pectus excavatum is lying flat especially in mild to moderate cases (*Colombani 2009*). However, unless the patient is symptomatic, echocardiography is not mandatory in the workup of patients with pectus excavatum.

Echocardiography may be considered if congenital heart disease is suspected in cases with pectus carinatum. In a study performed by *Iakovlev et al 1990, it was found* that 97% out of 70 children with pectus carinatum had echocardiographically documented MVP. Some of those patients had hemodynamic and cardiodynamic changes as well as decreased myocardial contractility. These abnormalities were more frequently observed in the patients with pigeon chest.

In Kyphoscoliosis, there is increased incidence of dilatation of the ascending aorta, dilatation of the pulmonary artery, bicuspid aortic valve, subaortic septal hypertrophy, and MVP with or without mitral regurgitation. Kyphoscoliosis complicated with chronic hypoxemia, may induce both functional and anatomic changes in the pulmonary vascular bed. Chronic hypoxemia and reduction in the pulmonary vascular bed due to small lungs in kyphoscoliotic patient may cause cor pulmonale as an end result. Myocardial noncompaction of the ventricular myocardium which is a rare congenital cardiomyopathy characterized by excessively protrusive trabeculae and deep trabecular recesses in one or more segments of the ventricle was observed in some cases. Echocardiography shows trabeculations and deep intratrabecular recesses along the ventricular cavity in the apical and lateral segments of LV. Colour Doppler imaging shows blood flow in these recesses. Dilatation of LV and LA may be noted with LV global hypokinesia. Eustachian valve may be thikened in some cases which may appear as mass in RA and need transesophageal echocardiography for better evaluation. There is an increased incidence of MVP in patients with skeletal abnormalities (*Velibey et al 2010*).

4.1.5 Situs inversus e.g. kartagener syndrome

Kartagener's syndrome is a very rare congenital malformation due to abnormal ciliary motility or immotile cilia with impaired mucociliary transport. So; the ciliated epithelium situated in the airways, brain ventricles, oviducts, and vasa efferentia of the testes all may be affected. The classic syndrome is a triad of situs inversus, bronchiectasis, and sinusitis which was first described by Kartagener 1904 (*Leigh et al 2009*). Situs inversus is present only in 47.7% of cases so some authors prefer to name this syndrome as *the immotile cilia syndrome or the dyskinetic cilia syndrome*. Heterotaxy (situs ambiguous) is present in about 6% of cases with Kartagener syndrome. However, the prevalence of CHD with heterotaxy was noted to be 200-fold higher in primary ciliary dyskinesia than in the general population.

Diagnosis is usually confirmed by studies of ciliary function and ultrastructure, and by immunohistochemistry of cilia and by measurements of nasal nitric oxide. Isolated dextrocardia is almost always associated with other cardiac anomalies, which are often serious. When accompanied by situs inversus, serious cardiac malformations are less common, but described. Echocardiography allows identification of the cardiac malformations that may be associated with Kartagner syndrome. Among these cardiac malformations are: tetralogy of Fallot, L-transposition of the great arteries, aortic coarctation, subpulmonic stenosis, right or left atrial isomerism, atrial septal defect, common atrium, atrioventricular septal defect; ventricular septal defect, double outlet right ventricle,

left ventricular outflow tract obstruction, bilateral superior vena cava, inferior vena cava drainage via azygous or via hemiazygous. (*Kennedy et al 2007*)

4.2 Acquired respiratory diseases
4.2.1 Traumatic chest conditions
Pulmonary insufficiency may occur following cardiac trauma and surgery. Unspecified injury of heart may occur with open wound into thorax. Contusion of heart may present with open thoracic wound. Though traumatic myocardial contusion or traumatic pericardial lesions are usually well tolerated and the clinical findings are transient; the sequelae of the cardiac trauma may be serious and difficult to be recognized. Blunt chest trauma might lead to cardiac injury ranging from simple arrhythmias to lethal conditions such as cardiac rupture. Therefore, a careful evaluation of every traumatized individual for cardiovascular injury is essential so that the more serious complications will be recognized and treated effectively. *Parmley et al 1958*; found that the most commonly encountered cardiac lesion at necropsy was myocardial rupture of a septum or a chamber wall. *Bjørnstad et al 2009* described a case with coronary artery dissection and acute myocardial infarction following blunt chest trauma.

Blunt trauma is uncommonly followed by intracardiac valve injuries but it does occur specially with car accidents. The valve most commonly affected is the tricuspid. *Hasdemir et al 2010* described occurrence of severe tricuspid regurgitation and second-degree Mobitz II atrioventricular block in a 68 years old woman due to blunt cardiac trauma secondary to car accident. Echocardiography could demonstrate rupture of tricuspid chordae tendinae or papillary muscles with prolapse of the valve cusps into the right atrium during systole and remarkable tricuspid regurgitation. Close follow-up may suffice in some patients with stable hemodynamic conditions, and regression of tricuspid regurgitation can be expected during follow-up. Mutilating mitral valve lesion secondary to cardiac trauma can induce mitral incompetence. The most common site of lesion is the papillary muscles (PM), followed by the chordae and then the mitral valve leaflets. The clinical course can be indolent or devastating, and most often requires urgent or delayed surgical treatment, either with mitral valve repair or replacement (*Pasquier et al 2010*). When the aortic valve is traumatically injured, it usually has a tear or avulsion on the cusp or on a commissure as well. The injury is often combined with trauma to the ascending aorta (*Kan & Yang 2005*).

4.2.2 Obstructive sleep apnea (adult & pediatric)
Obstructive sleep apnea (OSA) is a common but under-diagnosed condition that increases the risk of cardiovascular morbidity and mortality. Repetitive episodes of upper airway narrowing and/or occlusion; characteristic of OSA may lead to significant hypoxemia and cyclical alterations of arterial oxygen saturation. These episodes cause oxygen desaturation in response to apnoea, followed by the resumption of oxygen saturation during hyperventilation leading to a phenomenon called hypoxia/reoxygenation injury due to alteration of the oxidative balance through the induction of excess oxygen-free radicals, quite like in the sequelae of ischemia/reperfusion injury. These acute cardiovascular (CV) stressors together with swings in intrathoracic pressure, and central nervous system (CNS) arousals are potentially forming the basis for heightened CV risk in individuals with OSA (*Marrone et al 1998*). The development of pulmonary hypertension (PHT) and right heart dysfunction are well-known complications of OSA. Hypoxemia and hypercarbia-induced respiratory acidosis, which results from the apnoea episodes, are potent mediators of pulmonary vasoconstriction that can lead to reversible and irreversible chronic changes in

the pulmonary vasculature. It is likely that production of various neurohumoral factors in response to hypoxemia and respiratory distress may further promote PHT, right ventricular (RV) dysfunction and consequent impairment of systemic cardiac output (*Blum & McGowan 2004*).

Adenotonsillar hypertrophy is being the most common cause for upper airway obstruction and sleep apnoea in paediatric patients. Many studies showed the cardiac changes in children with OSA. *Kirk et al 2010* showed that all the Caucasian children with OSA included in their study had nocturnal systolic systemic hypertension and half of them had diurnal systolic systemic hypertension as well. However, they found no echocardiographic findings of LVH and/or RV hypertrophy. On the other hand; *Weber et al 2007* found dilatation of the RV systolic and diastolic diameters with significant reduction of RV functions in children with OSA. They also found reduction of the LV functions but without significance. The same finding was also documented by *Biltagi et al 2008* who found that there was significant reduction of both systolic and diastolic function of both RV and LV in the children with OSA and higher clinical score than in control children and children with OSA and lower clinical score. There was also a positive correlation between the echocardiographic finding of impairment of the cardiac functions with the level of the inflammatory breath markers and also with the clinical score of OSA. They also found that the Tissue Doppler is more sensitive to discover the impairment of the cardiac function than the conventional Doppler. The pulmonary pressure in the children with OSA and higher clinical score was significantly more than the control and children with OSA and lower clinical score. These findings were also confirmed by *Attia et al 2010* who found that the Tissue Doppler imaging can detect the subtle, subclinical changes in cardiac performance that occur in OSA due to adenotonsillar hypertrophy. They also found that these changes were reversible after surgical treatment.

Cardiac affection is well documented in adults with OSA. Atherosclerosis, secondary hypertension, atrial fibrillation, conduction disorders, coronary artery disease, congestive heart failure (CHF), pulmonary hypertension, stroke, and cardiac death; all are documented to occur in adults with OSA. *Romero-Corral et al 2007* found that echocardiographic examination documented impaired RV and LV functions and increased left atrial volume in cases with moderate to severe OSA. *Koshino et al 2010* explain these cardiac changes by the occurrence of negative intrathoracic pressure during apnea. They found that RV and LV longitudinal deformation was significantly reduced during the Müller maneuver. *Cioffi et al 2010* also documented occurrence of high prevalence of concentric LV hypertrophy in Moderate to severe OSA. They related these changes to the increase in myocardial end-systolic stress, venous return and sympathetic activity. Early recognition of RV dysfunction before development of pulmonary arterial (PA) hypertension is important for preventing further progression to heart failure and even death. *Shanoudy et al 1998* found by contrast transesophgeal echocardiography; an increased prevalence of PFO in adults with OSA which indicates that the pathophysiology of OSA may predispose to the maintenance of patency of a foramen ovale.

4.2.3 Bronchial asthma & COPD

Bronchial asthma (BA) is a common chronic inflammatory condition affecting the airways. Bronchial asthma does not affect only the lung but affects other organs including the heart. Even with mild cases, subclinical cardiac dysfunction can be documented and the severity of cardiac affection is parallel to the severity of the disease. Diastolic dysfunction of the RV was

the earliest hemodynamic change in BA. RV hypertrophy and dilation and LV diastolic dysfunction were observed in severe BA. Echocardiography detected RV systolic and diastolic dysfunction in a considerable percent of asthmatic children even with mild cases. Left ventricular dysfunction is usually detected in severe asthmatic cases. Tissue Doppler Echocardiography was found to be more sensitive in detecting cardiac dysfunction than conventional Doppler. However, these cardiac dysfunctions may be reversible especially in acute cases (*Peng et al 2006, Zeybek et al 2007*). The occurrence of supraventricular tachycardia in BA is related to presence of interventricular septal hypertrophy, LV dysfunction, and increased PAP (*Chicherina et al 2007*). Asthmatic medications can also affect the echocardiographic findings. Chronic administration of theophylline may cause a slight increase in percent fractional shortening, outflow peak velocity and atrial contribution to ventricular filling in the asthmatic children as compared to normal though these findings were found to be insignificant (*Aoki et al 1994*).

The cardiac manifestations of chronic obstructive pulmonary disease (COPD) are numerous. RV dysfunction and pulmonary vascular disease are well known to complicate the clinical course of COPD and correlate inversely with survival. Although RV dysfunction and PH are common in COPD; the increase in mean pulmonary artery pressures tends to be mild to moderate. The manifestations of pulmonary hypertension in patients with COPD are usually subtle and are often obscured by the manifestations of the lung disease. However, there is a dramatic increase in pulmonary pressure observed during exercise, nocturnal desaturations and acute exacerbations. Despite that the cardiac catheterization remains the "gold standard" for the measurement of pulmonary arterial pressures, but recent studies showed that continuous wave Doppler echocardiography is able to detect the increase in pulmonary pressure and is sufficiently sensitive to detect changes in pulmonary arterial pressure. Presence of progressive worsening of diastolic and systolic function of the LV is an additional factor aggravating hemodynamic compromise in patients with COPD which should be kept in mind when choosing the appropriate therapy (*Falk et al 2008, Higham et al 2001, Fisher et al 2009, Strutynskiĭ et al 2010*).

4.2.4 Pulmonary vascular disorders

Pulmonary artery (PA) aneurysm is a rare clinical condition, either congenital or acquired. It is defined as PA dilation greater than 4 cm. Large PA aneurysm can cause airway obstruction and compromise. Two-D echocardiography appears to be a useful non-invasive technique in the recognition of PA aneurysms. It can localize the site; the type; single or multiple; and the size of PA aneurysm. The fusiform aneurysms may reach a huge size, which may make it impossible to be imaged completely by a single 2-D sector plane and can cause displacement of the left atrium (*Bhandari & Nanda 1984*).

However; accurate diagnosis and evaluation of PA aneurysms may be difficult without angiography, computed tomography (CT) and MRIs. Associated cardiac lesions may be detected by echocardiography. Pulmonary hypertension is rarely found in some patients with aneurysm of the main pulmonary artery. However, severe pulmonary hypertension may be the cause of the aneurysm. Thrombus may be found inside the aneurysm with its characteristic echocardiographic features of having a laminated appearance, variably increased echodensity compared with surrounding tissues, stagnation caused by low blood flow state in the aneurysm and lack of obvious tumor features, such as narrow stalk and origin from PA wall. Echocardiography may detect associated lesions like atrial septal defects, mitral stenosis, aortic coarctation etc (*Güler et al 2003*).

Pulmonary arterio-venous fistula (PAVF) is a rare vascular anomaly. In PAVF, there is a direct communication between the PA branches and the pulmonary vein, without intervening pulmonary capillary bed. PAVFs are found in approximately 15–20% of patients with Rendu-Osler-Weber (ROW) disease. The M- mode and Two-D echocardiography may be normal and show normal intracardiac anatomy with no evidence of atrial or ventricular septal defect. However, if PAVF is suspected a contrast echocardiography showed be performed. After the injection of the contrast rapidly into the right antecubital vein; the right atrium and ventricle will be opacified but the left heart chambers will remain free of contrast until three cardiac cycles (3 s) after that the contrast will be seen in the left heart side. These findings are compatible with a right-to-left shunt at the pulmonary vasculature level and not intracardiac. (*Roolvink 2004*)

4.2.5 Pulmonary embolism

Pulmonary embolism (PE) is a common and serious disease. The prognosis depends on the speed of diagnosis and initiation of therapy. Since 1990; a large number of diagnostic tests and strategies have been evaluated for PE. Echocardiography has certain criteria to improve the diagnosis of acute PE. Presence of RV hypokinesis and dilation (without RV wall hypertrophy, and RV diameter becomes equal to or larger than the diameter of LV), tricuspid regurgitation velocity >2.7 m/sec without inspiratory collapse of the inferior vena cava, paradoxical septal movements and widening of pulmonary artery diameter together with the clinical suspicion of PE will increase the sensitivity of echocardiography to diagnose PE. Transesophageal echocardiography can show the central pulmonary arteries and may show a thrombus in the dilated segment of pulmonary artery. However, the echocardiography may fail to diagnose PE in about half of the cases. Despite that, echocardiography is one of the preferred first diagnostic tool to diagnose a patient with suspected PE (*Miniati et al 2001*).

4.2.6 Primary pulmonary hypertension (PPH)

Primary pulmonary hypertension (PPH) is a disease of unknown origin. It is characterised by a progressive increase in PA pressures. Despite that the invasive measurement of pulmonary vascular resistance (PVR) by right heart catheterisation remains the gold standard method to evaluate PVR, but echocardiography is still a good screening tool of patients with PH. It can estimate PVR using the ratio of peak tricuspid regurgitant velocity (TRV) to the RV outflow tract time-velocity integral (TVI rvot) or to the LV outflow tract time-velocity integral (TVI lvot). TRV/TVI rvot and TRV/TVI lvot was reported to be correlated significantly with invasively-determined PVR. Echocardiography can also be used to follow-up of PA pressures in patients with pulmonary hypertension to evaluate response to treatment. (*Roule et al 2010*)

4.2.7 Mass in chest

A wide variety of intra-thoracic masses can simulate primary intrinsic cardiac diseases. They can present with various cardiovascular manifestations; an abnormal heart shadow in the X-ray or unusual echoes in close proximity to the heart. Echocardiography can show unusual anterior wall echo which can be misinterpreted to be aneurysmal dilatation of either the RV outflow tract or pulmonary artery. M-mode echocardiography can differentiate between solid and cystic masses by their "sonolucency," which reflects the changes in gain settings. At high gain, the solid masses will "fill-in" with echoes, whereas a cystic structure will not.

An example of cystic masses in the anterior mediastinum is the thymic cyst which may appear as a cystic structure displaced by the great vessels during systole. Contrast echocardiography can help to know whether the cyst originates from the RV or from extra cardiac mediastinal structures (*Child et al 1975*).

In posterior mediastinal mass a wide strong echo, posterior to the posterior LV wall can be seen especially with tumours. An example of posterior mediastinal mass which presents with acute cardiovascular events is hiatus hernia. Hiatus hernia can hinder the sonographic configuration of the cardiac anatomy and can simulate the appearance of a left intra-atrial mass or a posterior mediastinal structure on transthoracic echocardiography. Contrast and transesophageal echocardiography are very helpful for better evaluation and to exclude the intracardiac nature of the mass. Other posterior mediastinal masses are esophageal carcinomas and hematomas, or dissecting aneurysms of the ascending aorta (*Koskinas et al 2008*). Some mediastinal masses can restrict the cardiac filling due to myocardial or pericardial infiltration. Decreased LV compliance impairs left atrial emptying and diminishes anterior mitral valve diastolic closure rate. Diminished ventricular filling may be observed due to decreased venous return as a consequence to compression of vena cava by the mass. So; when the prominent echo in front or behind the heart is seen and no primary cardiac disease can be detected; a mediastinal mass should be suspected and further studies are needed (*Child et al 1975*).

4.2.8 Patients being considered for lung transplantation or other surgical procedure for advanced lung disease to exclude possible cardiac disease

Patients with advanced lung disease need a detailed preoperative echocardiographic examination especially in patient with end stage lung disease (ESLD) who is in need for lung transplantation. Pulmonary hypertension is often detected in those patients. Echocardiography can detect abnormal left atrial filling, abnormal LV relaxation and geometry, RV enlargement, ventricular septal displacement and LV diastolic dysfunction (*Jastrzebski et al 2007*). Increased systolic pulmonary artery pressure, is a significant risk factors for death of patient with ESLD while being on the waiting list. A decrease of ejection fraction below 50% may indicate lower survival (*Jastrzebski et al 2005*).

4.2.9 Acute respiratory infections

There are a number of cardiac complications that occur in cases with pneumonia. Purulent pericarditis, Cardiac tamponade, pulmonary embolism and endocarditis are rare complications but reported specially with *Streptococcus pneumonia*. Congenital heart disease is the predisposing factor in about 22% of cases with recurrent pneumonia in children (*Al-Sabbagh et al 2008, Owayed et al 2000*).

4.2.10 Chronic pulmonary infection/inflammation

Tuberculous pericarditis continues to be a problem in both developed and developing countries. The echocardiography has certain characteristic features of tuberculous pericarditis. There may be pericardial thickening and calcification, intrapericardial fibrin strands, exudative coating with a tendency to form adhesions and in some instances constriction. There may be patchy deposits with ''fibrinous'' strands criss crossing the pericardial space. Echocardiographic evidence of cardiac tamponade is more common in tuberculous pericardial effusion. Organization of the pericardial tissue may form large pericardial mass or abscess which may obstruct RV free wall (*George et al 2004*).

Sarcoidosis is a granulomatous disease which may affect lungs and may be complicated with pulmonary hypertension. It may develop secondary to granulomatous involvement of pulmonary veins manifesting clinically as pulmonary veno-occlusive disease, extrinsic compression by mediastinal or hilar adenopathy, cardiac involvement including systolic or diastolic dysfunction, increased production of vasoactive endothelin-1 and downstream effects of hypoxemia. Presence of PH is an important risk factor affecting survival. Doppler echocardiography appears to be useful screening tool in the context of sarcoidosis with PH (*Alhamad et al 2010*). Fungal endocarditis has increased in incidence during the last 2 decades and may complicate fungal pulmonary disease especially in presence of prothetic valve or congenital heart disease. Echocardiography is extremely useful to diagnose Candida endocarditis because fungal endocarditis is frequently associated with large vegetations that are easily observed on standard echocardiograms. However, transthoracic echocardiography is less sensitive than transesophageal echocardiography but less invasive. Echocardiography can detect vegetations and intracardiac thrombi which are the most common types but are still rare. It may also demonstrate pericardial effusion, myocardial abscesses, associated myocarditis or pericarditis (*Ellis et al 2001*).

4.2.11 Pulmonary ICU patient

In the chest intensive care setting; echocardiography is a valuable and indispensable bedside diagnostic tool which has the advantage of providing very useful hemodynamic information in a matter of minutes. It is used to establish rapid diagnoses and assessment, to serially monitor the therapeutic interventions and to expect the prognosis in pulmonary/critical care patients with cardiopulmonary dysfunction. It can be used together with clinical assessment and other tools to assess the patient's hemodynamic. It is inevitable and appropriate that the pulmonary intensive care physician should know and have the skills to perform ICU echocardiography. Echocardiography is a useful diagnostic tool in cases with profound hypotension or shock or in whom hypotension or shock fails to respond to the standard treatment. It can confirm the cardiac cause of shock. Global biventricular function, LV myocardial kienetic status (Hypokinetic, hyperkinetic or normokinetic; regional and global) and valve status can be evaluated with standard methods keeping into consideration the amount of inotropic and vasoactive medications givin, the degree of LV filling (by assessement of IVC size); pulmonary vascular resistance and presence of extrinsic compression e.g. cardiac tamponade or effect of mechanical ventilation (*Kaplan & Mayo 2009*).

In septicemia, echocardiography can play a crucial role in the management of the septic ICU patient both by excluding cardiac causes for sepsis, and by monitoring and guiding management of the patient hemodynamics. Despite that septic shock is classically considered as hyperdynamic state, but sepsis can reduce myocardial contractility and ventricular functions and can decrease cardiac output. So, global evaluation of the cardiac functions is of paramount importance. The sepsis-induced cardiomyopathy is classically observed in children and adult patients with meningococcemia. Echocardiography can determine the cardiac source of sepsis e.g. infective endocarditis. It is to be noted that TEE is more sensitive than TTE to detect of small vegetations. However; echocardiography alone cannot be used to make diagnosis of endocarditis and must be used with the other diagnostic criteria. (*Price et al 2008*)

Echocardiography can detect pleural effusion and can differentiate left pleual effusion from the pericardial effusion. With subcostal 2-D echocardiography, right pleural effusion

appears as echo-free mass that is contiguous to the RA but not to any other cardiac chamber. It is also bounded inferiorly by the smooth round surface of the liver. The location of the descending thoracic aorta on 2-D echocardiography serves as a valuable landmark in localizing the pericardial-pleural interface, thereby differentiating pericardial from pleural effusions (*D'Cruz 1984*). However, the right-sided pleural effusion is more difficult to be detected than the left because there is no acoustic window. TEE can detect the echo-free space created by pleural fluid, as well as the appearance of adjacent atelectatic lung. TEE also could be used to quantify the size of effusions. *Howard et al 2011*; could estimate the volume of the pericardial effusion using TEE by applying the following formula:

$$V = 4.5 \cdot CSA(max)(3/2)$$

Where V is the expected pleural effusion volume in milliliters while the CSA(max) is the maximum cross-sectional area in centimeters squared of the pleural effusion recorded by transesophageal echocardiography. Pleural effusion in ICU patient can cause echocardiographic artefacts (called cardiac-mass lung' artefact,) which may give the impression of an intracardiac mass and could be mistaken with mobile components. In presence of hemodynamically unstable patient with significant pleural effusions; echocardiography must be performed to evaluate the cardiac function and to exclude presence of associated pericardial effusion (*Karabinis et al 2008*).

Echocardiography in pulmonary ICU allows immediate evaluation of patients with cardiopulmonary failure, to establish initial diagnosis and serial examinations may be performed to guide ongoing management. It also can evaluate the cardiac function both systolic and diastolic that can be compromised. Echocardiography may not be needed routinely in all patients with respiratory failure, but it is particularly useful when a cardiac cause of acute respiratory failure is suspected. Presence of LV dilation, regional or global myocardial wall motion abnormalities, and/or severe mitral regurgitation are present in cases with cardiogenic pulmonary oedema. On the other hand; a normal heart size and normal systolic and diastolic function in a patient with pulmonary oedema would suggest Adult Respiratory Distress Syndrome (ARDS). Estimation of PA pressure and evaluation of RV function are needed in chronic respiratory failure (*Vieillard-Baron et al 1999*)

Mechanical ventilation may cause potentially detrimental consequences for systemic venous return produced by an increase in pleural pressure. It also decreases RV after-load due to increased positive end expiratory presurre (PEEP), incresed lung volume or both. It also decreases radius of interventricular septum in diastole leading to its leftward shift with impeding filling of the LV and decreased LV cardiac output. However, the effect on the LV is insignificant. On the other hand, continuous positive airway pressure (CPAP) can improve LV afterload by reducing transmural LV pressure (*Huemer et al 1994*). TTE appears as a sensitive noninvasive method which accurately detects changes in central haemodynamics induced by changes in breathing pattern. It is a powerful tool to assess RV function, especially if acute cor pulmonale is a concern, as well as to estimate LV function (*Vieillard-Baron et al 1999*).

Weaning patients from the ventilator remains a crucial issue. TTE helps to identify patients at high risk of weaning failure. TTE findings which expect difficult weaning include: increase of LA pressure, appearance/worsening of mitral regurgitation new/worsening regional wall motion abnormalities, decreased LVEF, shortened deceleration time of mitral Doppler E wave, and increased E/E' ratio (E' is the the maximal velocity of its displacement

of the lateral portion of the mitral annulus during early diastole) and reduced tricuspid annular TDI systolic and diastolic velocities. (*Caille et al 2010*)

5. Conclusion

Echocardiography plays a crucial role in diagnosis, monitoring and follow up of many respiratory disorders.

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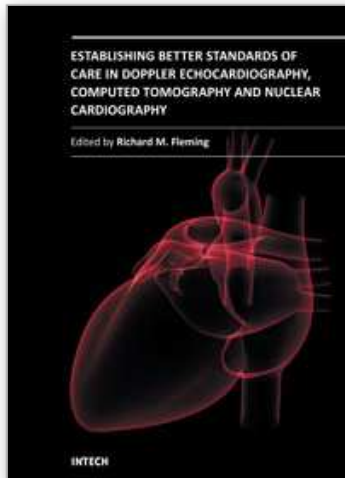
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