INTRODUCTION

Asthma

Global burden of asthma

Globally, morbidity and mortality associated with asthma have increased over the last 2 decades where the prevalence of the disease is greatest, about 8% of African population [1], and 5% of general population in the united state of America.[2] The number of patients with asthma who seek medical attention is increasing and accurate non invasive tests to detect significant secondary cardiac affection leading to decreased functional status of the heart and increased mortality, is necessary and could aid in the clinical management of these patients. [3]

Epidemiology

235–300 million people worldwide are affected by asthma as recorded in world health organization 2011.[4,5] and approximately 250,000 people die per year from the disease.[5] Rates vary between countries with prevalences between 1 and 18%.[5] It is more common in developed than developing countries.[5] One thus sees lower rates in Asia, Eastern Europe and Africa.[6] Within developed countries it is more common in those who are economically disadvantaged while in contrast in developing countries it is more common in the affluent.[5] The reason for these differences is not well known.[5] Low and middle income countries make up more than 80% of the mortality.[7]

While asthma is twice as common in boys as girls,[5] severe asthma occurs at equal rates.[8]In contrast adult women have a higher rate of asthma than men,[5] and it is more common in the young than the old.[6]

Global rates of asthma have increased significantly between the 1960 and 2008[9,10] with it being recognized as a major public health problem since the 1970.[6] Rates of asthma have plateaued in the developed world since the mid-1990 with recent increases primarily in the developing world.[11]

- Undoubtedly, asthma is a very common condition and currently, according to Asthma in UK: [12] 5.4 million people in the UK receive treatment for asthma: 1 in 10 children and 1 in 12 adults.
- Gender differences - there is a male preponderance in childhood with a reversal in early adulthood. Certain trends in the prevalence of asthma seem apparent over time.[13]

Definition:

Asthma is a chronic inflammatory disorder of the airways characterized by an obstruction of airflow, bronchial hyperresponsive as variable disease day and night difference which may be completely or partially reversed with or without specific therapy.[2]

Pathophysiology

Obstruction of the lumen of a bronchiole by mucoid exudate, goblet cell metaplasia, and epithelial basement membrane thickening in a person with asthma.(5)

Asthma is the result of chronic inflammation of the airways which subsequently results in increased contractability of the surrounding smooth muscles. This among other factors leads to bouts of narrowing of the airway and the classic symptoms of wheezing. The narrowing is typically reversible with or without treatment. Occasionally the airways themselves change.(5) Typical changes in the airways include an increase in eosinophils and thickening of the lamina reticularis. Chronically the airways smooth muscle may increase in size along with an increase in the numbers of mucous glands. Other cell types involved include: T lymphocytes, macrophages, and neutrophils. There may also be involvement of other components of the immune system including: cytokines, chemokines, histamine, and leukotrienes among others.(6)

Classification

Table (1): Clinical classification of asthma

Clinical classification (≥ 12 years old) (14)

Severity	Symptom frequency	Night time symptoms	%FEV_1 of predicted	FEV_1 Variability	SABA use
Intermittent	≤2/week	≤2/month	≥80%	<20%	≤2 days/week
Mild persistent	>2/week	3–4/month	≥80%	20–30%	>2 days/week
Moderate persistent	Daily	>1/week	60–80%	>30%	Daily
Severe persistent	Continuously	Frequent (7×/week)	<60%	>30%	≥twice/day

Asthma is clinically classified according to the frequency of symptoms, forced expiratory volume in one second (FEV_1), and peak expiratory flow rate.(14)Asthma may also be classified as atopic (extrinsic) or non-atopic (intrinsic), based on whether symptoms are precipitated by allergens (atopic) or not (non-atopic).(15) While asthma is classified based on severity, at the moment there is no clear method for classifying different subgroups of asthma beyond this system.(16)

Although asthma is a chronic obstructive condition, it is not considered as a part of chronic obstructive pulmonary disease as this term refers specifically to combinations of disease that are irreversible such as bronchiectasis, chronic bronchitis, and emphysema.(17)Unlike these diseases, the airway obstruction in asthma is usually reversible; however, if left untreated, the chronic inflammation from asthma can lead the lungs to become irreversibly obstructed due to airway remodeling.(18) In contrast to emphysema, asthma affects the bronchi, not the alveoli.(19)

Environmental control

Environmental factors play an important role in the origin and persistence of asthma, severity of airflow obstruction and the ability to achieve adequate asthma control. Viral infections and other 'triggers' should be avoided whenever possible.

Approximately 25% of adult asthma cases are estimated to be work related.[20] Furthermore, 10% to 15% of adult asthma may be caused by an occupational agent.[21,22] Therefore, it is crucial to identify work-related asthma by completing a thorough medical and occupational history, and performing the appropriate investigations. [23]

Diagnosis in adults

Current guidelines emphasise that the diagnosis of asthma is a clinical one, based on typical symptoms and signs, and a measurement of airflow obstruction for which spirometry is the preferred initial test. Ascribe high, intermediate or low probability of asthma based on this assessment to determine the use of further investigations or treatment trials.[24]

CXR

CXR is remarkably normal, even in very severe asthma. It should not be used routinely in the assessment of asthma but consider CXR in any patient presenting with an atypical history or with atypical findings on examination.[24]

Spirometry

Spirometry is the measurement of flow rates and volumes during a forced expiratory manoeuvre to determine the forced expiratory volume in 1 second (FEV_1) and the forced vital capacity (FVC). The ratio FEV_1/FVC is a measure of air flow obstruction. Such tests must be performed by standardized methods, as described by the European and American Thoracic Societies. [25]

Effect of asthma on the right ventricle:

The rise in negative pressure during inspiration and positive pressure during expiration which exceeds the normal right ventricular preload and after load cycle may cause an increase in intrathoracic pressure.[26] Excessive respiratory effort in asthma may lead to increased intrathoracic pressure and lung hyperinflation, which causes an increased after load for the right ventricle.[27-28] Chronic lung disease may contribute to an increased pulmonary arterial resistance, causing disturbance in right ventricular systolic and diastolic function. Theoretically, a change in right ventricular compliance will take place due to the rise in pulmonary vascular resistance, causing changes in right ventricular diastolic filling parameters. This will cause substantial changes in cardiac performance.[29-34]

In bronchial asthma recurrent exposure to hypoxemia is one of the mechanisms besides others leading to sustained pulmonary vasoconstriction and narrowing of the pulmonary vasculature. Consequently, Pulmonary hypertension develops leading to right heart enlargement with ventricular hypertrophy, and impaired cardiac function, known as corpulmonale.[35]

Complications

Inadequate control of asthma leads to much morbidity and poor quality of life.[36] Complications mostly relate to acute exacerbations:

- Pneumonia
- Pneumothorax
- Pneumomediastinum
- Respiratory failure and arrest
- Death
- Cardiac arrhythmias

A common feature of deaths from asthma is that the patient and/or the medical staff have underestimated the severity of the attack. Patients frequently have adverse psychosocial factors that interact with the ability to judge or manage their disease, leading to late presentation. A confidential enquiry from the east of England concluded that in two-thirds of asthma deaths, medical management failed to comply with national guidelines. It is suggested that 'at-risk' asthma registers in primary care may improve recognition and management of high-risk patients. [37-38]

Anatomy and physiology of right ventricle

Macroscopic Anatomy of the RV

In the normal heart, the RV is the most anteriorly situated cardiac chamber and lies immediately behind the sternum. In the absence of transposition of great arteries, the RV is delimited by the annulus of the tricuspid valve and by the pulmonary valve. As suggested by Goor and Lillehi[39] RV can be described in terms of 3 components: (1) the inlet, which consists of the tricuspid valve, chordae tendineae, and papillary muscles; (2) the trabeculated apical myocardium and (3) the infundibulum, or conus, which corresponds to the smooth myocardial region [39,40] (Figure 1). In the study of congenital heart disease, this division seems to be more practical than the traditional division of the RV into sinus and conus components.[40] Additionally, the RV can also be divided into anterior, lateral, and inferior walls, as well as basal, mid, and apical sections. [41]

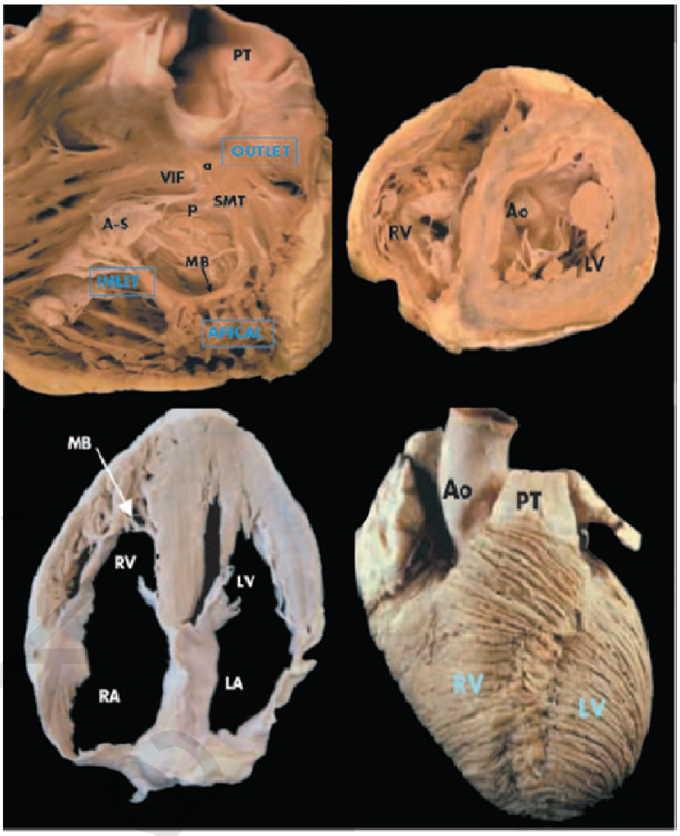

Figure (1): A, The inlet, trabeculated apical myocardium and infundibulum of the RV. The tricuspid and pulmonary valves are separated by the ventriculoinfundibular fold (VIF). B, Short-axis plane of the RV demonstrating its crescentic shape. C, The 4-chamber anatomic plane of the heart showing the moderator band (MB) and the more apical insertion of the tricuspid valve. D, Superficial muscle layer of the RV (dissection by Damian Sanchez- Quintana, University of Extremadura, Spain). SMT indicates septomarginal trabeculation with its anterior (a) and posterior(p) arm; A-S, anterosuperior leaflet of the tricuspid valve; PT, pulmonary trunk; Ao, aorta; RA, right atrium; and LA, left atrium. [39,40]

Three prominent muscular bands are present in the RV: the parietal band, the septomarginal band, and the moderator band. The parietal band and the infundibular septum make up the crista supraventricularis.[42] The septomarginal band extends inferiorly and becomes continuous with the moderator band, which attaches to the anterior papillary muscle. [42] When abnormally formed or hypertrophied, the septomarginal band can divide the ventricle into 2 chambers (double-chambered RV). [40]

Another important characteristic of the RV is the presence of a ventriculoinfundibular fold that separates the tricuspid and pulmonary valves. In contrast, in the LV, the aortic and mitral valves are in fibrous continuity[39,40] (Figure 1).

The shape of the RV is complex. In contrast to the ellipsoidal shape of the LV, the RV appears triangular when viewed from the side and crescent shaped when viewed in cross section. [41]

The shape of the RV is also influenced by the position of the inter ventricular septum. Under normal loading and electrical conditions, the septum is concave toward the LV in both systole and diastole. [41]

In the mature child and adult, the volume of the RV is larger than the volume of the LV, whereas RV mass is approximately one sixth that of the LV. [43]

Myofiber Architecture of the RV

The ventricles are not composed of a single muscle layer but rather of multiple layers that form a 3-dimensional (3D) network of fibers. [40]

As described by Ho and Nihoyannopoulos [40] the RV wall is mainly composed of superficial and deep muscle layers. The fibers of the superficial layer are arranged more or less circumferentially in a direction that is parallel to the atrioventricular (AV) groove (Figure 1). [40,44]

These fibers turn obliquely toward the cardiac apex on the sternocostal aspect and continue into the superficial myofibers of the LV. [40,44]

The deep muscle fibers of the RV are longitudinally aligned base to apex. In contrast to the RV, the LV contains obliquely oriented myofibers superficially, longitudinally oriented myofibers in the subendocardium, and predominantly circular fibers in between. This arrangement contributes to the more complex movement of the LV, which includes torsion, translation, rotation, and thickening. [40,44]

The continuity between the muscle fibers of the RV and LV functionally binds the ventricles together and represents the anatomic basis of free ventricular wall traction caused by LV contraction. This continuity also contributes, along with the interventricular septum and pericardium, to ventricular interdependence.[45] The morphological features that best differentiate anatomic RV, LV, or indeterminate ventricle include the following: (1) the more apical hinge line of the septal leaflet of the tricuspid valve relative to the anterior leaflet of the mitral valve; (2) the presence of a moderator band; (3) the presence of more than 3 papillary muscles; (4) the trileaflet configuration of the tricuspid valve with septal papillary attachments; and (5) the presence of coarse trabeculations. [40,42]

Physiology

The primary function of the RV is to receive systemic venous return and to pump it into the pulmonary arteries. Under normal circumstances, the RV is connected in series with the LV and is, therefore, obligated to pump on average the same effective stroke volume.

Mechanical Aspects of Ventricular Contraction:

RV contraction is sequential, starting with the contraction of the inlet and trabeculated myocardium and ending with the contraction of the infundibulum (approximately 25 to 50 ms apart).[44] Contraction of the infundibulum is of longer duration than contraction of the inflow region. [44]

The RV contracts by 3 separate mechanisms: (1) inward movement of the free wall, which produces a bellows effect; (2) contraction of the longitudinal fibers, which shortens the long axis and draws the tricuspid annulus toward the apex; and (3) traction on the free wall at the points of attachment secondary to LV contraction. Shortening of the RV is greater longitudinally than radially.[46] In contrast to the LV, twisting and rotational movements do not contribute significantly to RV contraction. Moreover, because of the higher surface-to volume ratio of the RV, a smaller inward motion is required to eject the same stroke volume. [41]

RV Hemodynamics:

Under normal conditions, the RV is coupled with a low impedance, highly distensible pulmonary vascular system. Compared with the systemic circulation, pulmonary circulation has a much lower vascular resistance, greater pulmonary artery distensibility, and a lower peripheral pulse wave reflection coefficient. [44]

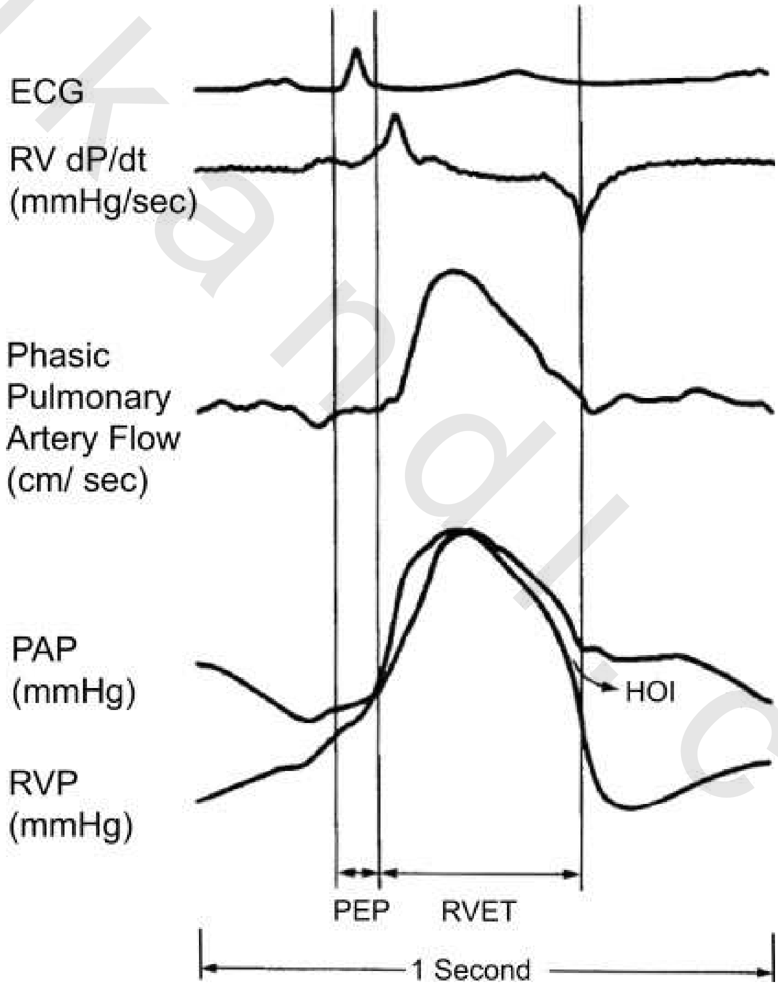

Figure (2): Simultaneously recorded ECG, RV analog signal of pressure development (dP/dt), phasic pulmonary artery flow, pulmonary artery pressure (PAP), and RV pressure (RVP) in the human subject. PEP indicates pre ejection period; RVET, RV ejection time; and HOI, hangout interval. [48]

Under normal conditions, right-sided pressures are significantly lower than comparable left-sided pressures. (47) RV pressure tracings show an early peaking and a rapidly declining pressure in contrast to the rounded contour of LV pressure tracing (Figure 2).(48) RV isovolumic contraction time is shorter because RV systolic pressure rapidly exceeds the low pulmonary artery diastolic pressure. A careful study of hemodynamic tracings and flow dynamics also reveals that end-systolic flow may continue in the presence of a negative ventricular-arterial pressure gradient (Figure 2).(44,48) This interval, which is referred to as the hangout interval, is most likely explained by the momentum of blood in the outflow tract. (48)

Cardiodynamics

RV systolic function is a reflection of contractility, after load, and preload. RV performance is also influenced by heart rhythm, synchrony of ventricular contraction, RV force interval relationship, and ventricular interdependence. (49–52) Significant valvular regurgitation or shunt physiology should always be considered because they can decrease effective cardiac output. (44)

The complex relationship between RV contractility, preload, and after load can be better understood with the help of pressure-volume loops. Pressure-volume loops depict instantaneous pressure-volume curves under different loading conditions. For the LV, Suga et al(53) showed that the end-systolic pressure-volume relationship can be approximated by a linear relationship. The slope of this relationship is referred to as ventricular elastance. Because of its relative load independence, many investigators consider ventricular elastance as the most reliable index of contractility.(54)

Interestingly, despite having markedly different ventricular geometry and hemodynamics, many studies showed that the RV also follows a time-varying elastance model (Figure3). (55,56) Because of the different shape of RV pressure-volume curves, maximal RV elastance better reflects RV contractility than does the end-systolic elastance commonly used in LV pressure-volume interpretation. (56)

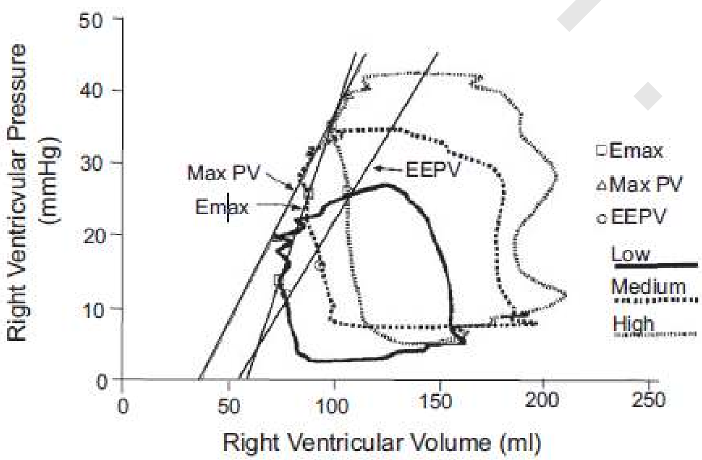

Figure (3): Pressure–volume loops of the RV under different loading conditions. The slope of maximum time-varying elastance (Emax), maximum pressure-volume ratio (Max PV), and end ejection pressure/volume (EEPV) are displayed on the graph.(55,56)

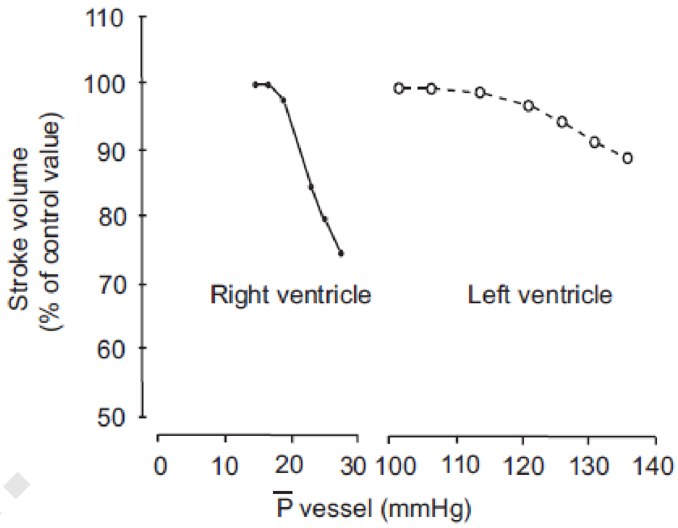

Figure (4): The response of the RV and LV to experimental increase in after load. [57,58]

RV after load represents the load that the RV has to overcome during ejection. Compared with the LV, the RV demonstrates a heightened sensitivity to after load change (Figure 4).[57,58] Although in clinical practice, pulmonary vascular resistance (PVR) is the most commonly used index of after load, PVR may not reflect the complex nature of ventricular after load.[44]

RV preload represents the load present before contraction. Within physiological limits, an increase in RV preload improves myocardial contraction on the basis of the Frank Starling mechanism. Beyond the physiological range, excessive RV volume loading can compress the LV and impair global ventricular function through the mechanism of ventricular interdependence. [58] Compared with LV filling, RV filling normally starts before and finishes after. RV isovolumic relaxation time is shorter, and RV filling velocities (E and A) and the E/A ratio are lower. The respiratory variations in RV filling velocities are, however, more pronounced. [41,59]

Many factors influence RV filling, including intravascular volume status, ventricular relaxation, ventricular chamber compliance, heart rate, passive and active atrial characteristics, LV filling, and pericardial constraint. [60]

Assessment of the RV

Evaluation of RV structure and function in patients with cardiopulmonary disorders is an essential component of clinical management. Although there have been significant improvements in cardiac imaging, many factors contribute to the challenges of RV assessment. These include (1) the complex geometry of the RV; (2) the limited definition of RV endocardial surface caused by the heavily trabeculated myocardium; (3) the retrosternal position of the RV, which can limit echocardiographic imaging windows; and (4) the marked load dependence of indices of RV function. [41]

Cardiac magnetic resonance imaging (**MRI**) is increasingly used as a standard tool in the evaluation of RV structure and function. MRI is the most accurate method for assessing

RV volume. With careful attention to detail, diastolic and systolic volumes can be determined and used to calculate ejection fraction. In addition, MRI flow studies are used to estimate forward flow through semilunar valves and AV valves, which allows accurate calculation of regurgitant fractions, cardiac output, and shunt fraction. In the future, MRI could also have a potential role in assessing the physiological characteristics of pulmonary arterial flow.

Radionuclide-based techniques provide reliable and geometrically independent assessments of RVEF.

Cardiac catheterization provides direct hemodynamic data and allows accurate assessment of pulmonary vascular resistance. Pulmonary angiography and coronary angiography can further delineate important anatomic and functional characteristics. Compared with CT angiography, pulmonary angiography may be limited in its assessment of proximal lamination but has a relative benefit in assessing distal obstruction. Analysis of RV function by pressure volume loops is useful because it quantifies various determinants of RV function such as RV elastance, dP/dt, ventricular compliance, stroke work, and preload recruitable stroke work. Currently, the conductance catheter is the most frequently used method to construct pressure-volume loops. This catheter contains a high-fidelity pressure sensor and up to 12 electrodes to measure RV electrical conductance, from which instantaneous RV chamber volume is determined. [61]

Echocardiogarphic Assessment of RV

Conventional echocardiography

RV Dimensions

RV dimension is best estimated at end-diastole from a right ventricle focused apical 4-chamber view as shown in figure (5). Diameter ≥ 35 mm at the mid level indicates RV dilatation. Similarly, longitudinal dimension ≥86 mm indicates RV enlargement.[62]

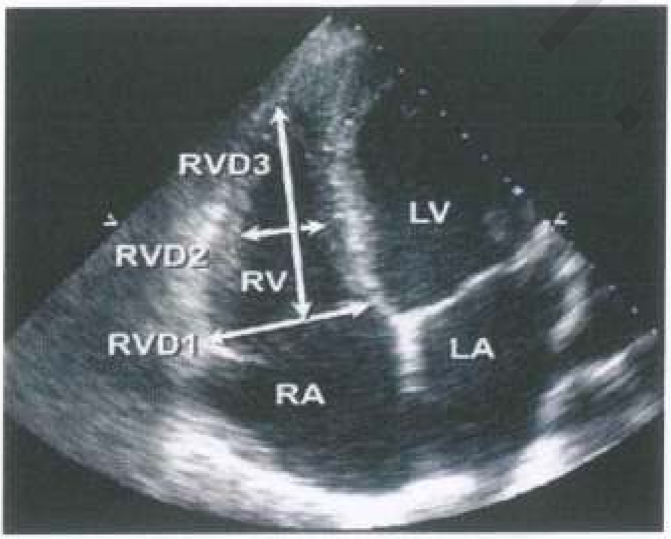

Figure (5): Right ventricle—focused apical 4-chamber view showing measurement of the dimensions.[62]

RVOT fractional shortening

RVOT-FS another measure of RV global systolic function, is calculated as the percentage of fall in right ventricular outflow tract diameter in systole with respect to that in diastole. [63]

Lindqvist et al. [63] found that RVOT fractional shortening correlates well with longitudinal function, pulmonary pressure gradient, and RV- right atrial (RA) pressure gradient.

Although it only assesses systolic performance of the outflow tract, its combination with long axis measurements and transtricuspid Doppler analysis should provide a comprehensive evaluation of the right ventricular performance. [63]

RV Diastolic Dysfunction

The parameters used to assess RV diastolic function are essentially the same as those used to assess the left side. Those that have been most validated are Doppler velocities of the transtricuspid flow (E, A, and E/A), tissue Doppler velocities of the tricuspid annulus (E', A', E'/A'), deceleration time, and IVRT [62]

Measurement of RV diastolic function should be considered in patients with suspected RV impairment as a marker of early or subtle RV dysfunction, or in patients with known RV impairment as a marker of poor prognosis.

Transtricuspid E/A ratio, E/E' ratio, and RA size have been most validated and are the preferred measures.

Grading of RV diastolic dysfunction should be done as follows; tricuspid E/A ratio ≤ 0.8 suggests impaired relaxation, a tricuspid E/A ratio of 0.8 to 2.1 with an E/E' ratio >6 or diastolic flow predominance in the hepatic veins suggests pseudonormal filling, and a tricuspid E/A ratio >2.1 with a deceleration time <120 ms suggests restrictive filling. [62]

TAPSE(Tricuspid annulus plane septal excursion)

TAPSE is a method to measure the distance of systolic excursion of the RV annular segment along its longitudinal plane, from a standard apical 4-chamber window. TAPSE represents longitudinal function of right ventricle in the same way as mitral annular plane systolic excursion by Doppler tissue imaging does with the left ventricle. It is refered that the greater the descent of the base in systole, the better the RV systolic function. As with other regional methods, it assumes that the displacement of the basal and adjacent segments in the apical 4-chamber view is representative of the function of entire right ventricle, an assumption that is not valid in many disease states or when there are regional RV wall motion abnormalities. TAPSE is usually acquired by placing an M-mode cursor through the tricuspid annulus and measuring the amount of longitudinal motion of the annulus at peak systole. [62]

In the initial validation study by Kaul et al, [64] TAPSE correlated strongly with radionuclide angiography, with low interobserver variability. It has also been validated against biplane Simpson RV EF and RV fractional area shortening. [65,66] In a study of 750 patients with a variety of cardiac conditions, compared with 150 age-matched normal controls, a TAPSE cutoff value < 17 mm yielded high specificity, through low sensitivity to distinguish abnormal from normal subjects. [67]

Advantages: TAPSE is simple, less dependent on optimal image quality, and reproducible, and it does not require sophisticated equipment or prolonged image analysis.

Disadvantages: TAPSE assumes that the displacement of a single segment represents the function of a complex 3D structure. Furthermore, it is angle dependent, and there are no large-scale validation studies. [62]

Myocardial performance index

The MPI, or Tie index, is a global estimate of both systolic and diastolic function of the right ventricle. It is based on relationship between ejection and non ejection work of the heart. The MPI is defined as the ratio of isovolumic time divided by ET, or [(IVRT +IVCT)/ET].

The measure remains accurate within a broad range of heart rates, [68] through the components should be measured with a constant R-R interval to minimize error. Although the MPI was initially thought to be relatively independent of preload, this has been questioned in more recent studies. In addition, the MPI has been demonstrated to be unreliable when RA pressure is elevated (eg. RV infarction), as there is a more rapid equilibration of pressures between the RV and RA, shortening the IVRT and resulting in an inappropriately small MPI. [69]

The right-sided MPI can be obtained by two methods: the pulsed Doppler method and the tissue Doppler method. In the pulsed Doppler method, the ET is measured with pulsed Doppler of RV outflow (time from the onset to the cessation of flow), and the tricuspid valve closure-opening time is measured with either pulsed Doppler of the tricuspid inflow (time from the end of transtricuspid A wave to the beginning of the transtricuspid E wave) or continous Doppler of the TR jet (time from the onset to the cessation of the jet). These measurements are taken from different images, and one must therefore attempt to use beats with similar R-R intervals to obtain a more accurate RIMP value. In the tissue Doppler method, all time intervals are measured from a single beat by pulsing the tricuspid annulus. As was demonstrated for the LV MPI,[70,71] it is important to note that the correlation between both methods is modest and that normal values differ on the basis of the method chosen.

The MPI has prognostic value in patients with PHT[68], and changes in MPI correlate with change in clinical status in this patient group [72] It has also been studied in RV infarction, hypertrophic cardiomyopathy, and congenital heart disease. [73-78]

The MPI has been measured in healthy individuals and in normal control subjects in 23 studies with > 1000 subjects. The upper reference limit is 0.40 by pulsed Doppler and 0.55 by tissue Doppler.

Advantages: This approach is feasible in a large majority of subjects both with and without TR, the MPI is reproducible, and it avoids the geometric assumptions and limitations of complex RV geometry. The pulsed tissue Doppler method allows for measurement of MPI as well as S`,E' and A', all from a single image.

Disadvantages: The MPI is unreliable when RV ET and TR time are measured with differing R-R intervals, as in atrial fibrillation Moreover, it is load dependent and unreliable when RA pressure is elevated.

Tissue Doppler Echocardiography

TDE has three modalities: spectral pulsed wave Doppler, two dimensional, and M mode colour Doppler.

Spectral Doppler

Spectral pulsed TDE has the advantage of online measurements of velocities and time intervals and an excellent temporal resolution. According to the Doppler principle, tissue velocities moving toward the transducer are positive, whereas velocities moving away from the transducer are negative. Since the wall moves whereas the sample volume is fixed, the spatial resolution is poor and myocardial layers cannot be separately analysed. [79]

Colour Doppler

In colour TDE, red encodes wall motion towards the transducer (positive velocities), whereas blue encodes wall motion away from the transducer (negative velocities). On each side of the scale, the brightest shades correspond to the highest velocities.

Colour images require digital acquisition and storage for off-line post-processing analysis. In contrast to spectral Doppler, endocardial and epicardial layers can be separately analysed. Peak and mean velocities, time velocity integral, and regional time intervals can be measured in each myocardial segment, in each myocardial layer, and in each phase of the cardiac cycle. [79]

Strain and Strain Rate

Strain is a measure of tissue deformation and is defined as the change in length normalized to the original length as illustrated in (figure 6). The rate at which this change occurs is called strain rate. Deformation in a 1-dimensional object, such as a thin bar, is limited to lengthening or shortening. [80]

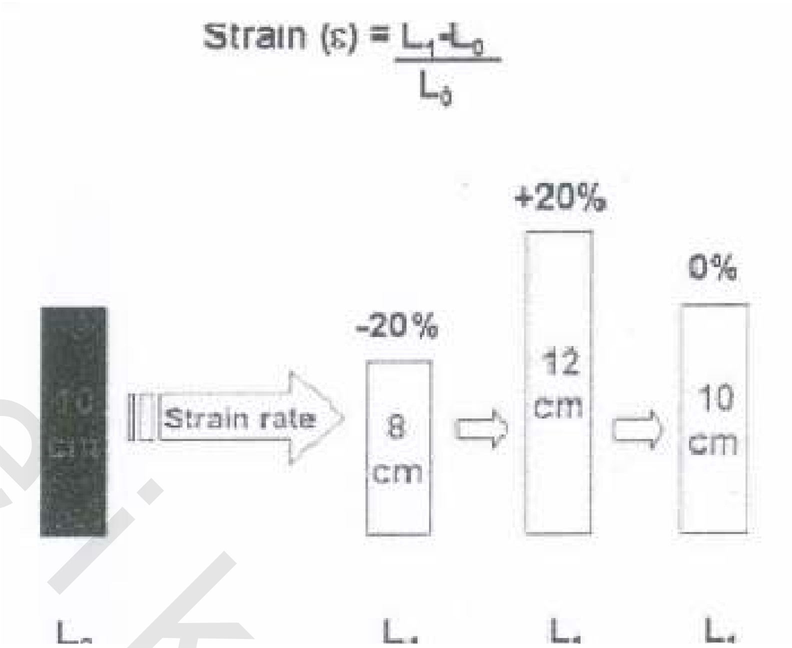

Figure (6): Strain is the percentage of change from the initial length[80]

Strain is how much the bar is shortened or lengthened relative to its original length (ie, reduction to half its original length is -50% strain, and an increase to one third longer is -33% strain). Strain rate is the speed at which this change occurs. Strain rate and strain are kind of shortening velocity and shortening fraction, respectively. [81]

By TDI, strain rate is the difference in velocity between 2 points along the myocardial wall (velocity gradient) normalized to the distance between the 2 points as shown in figure (7). [82]

The heart shortens and lengthens in the longitudinal direction, it thickens and thins in the radial direction, and it shortens and lengthens in the circumferential direction figure (8). [81]

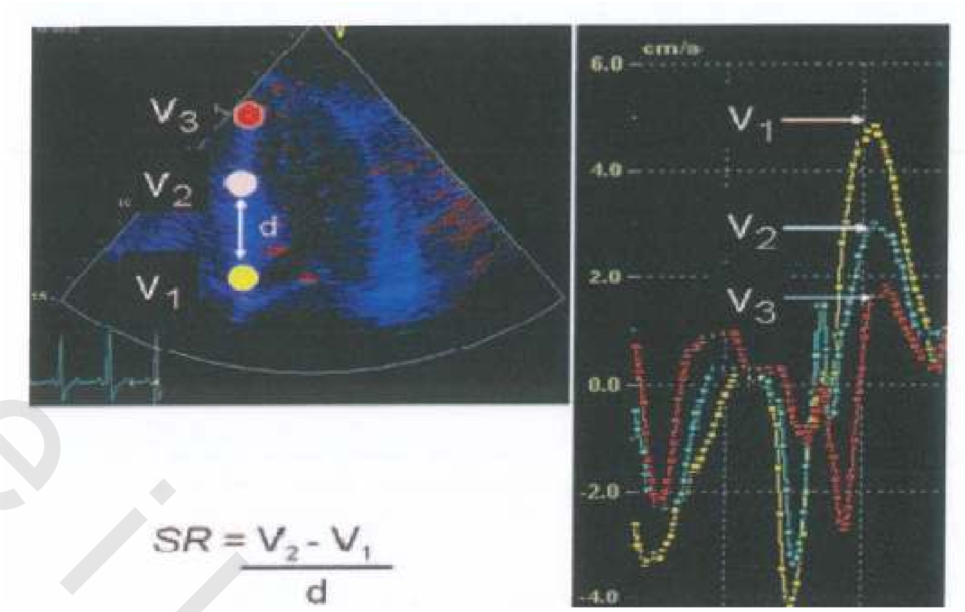

Figure (7): Strain rate is the difference in velocity between two points along the myocardial wall (velocity gradient) normalized to the distance between the two points.[82]

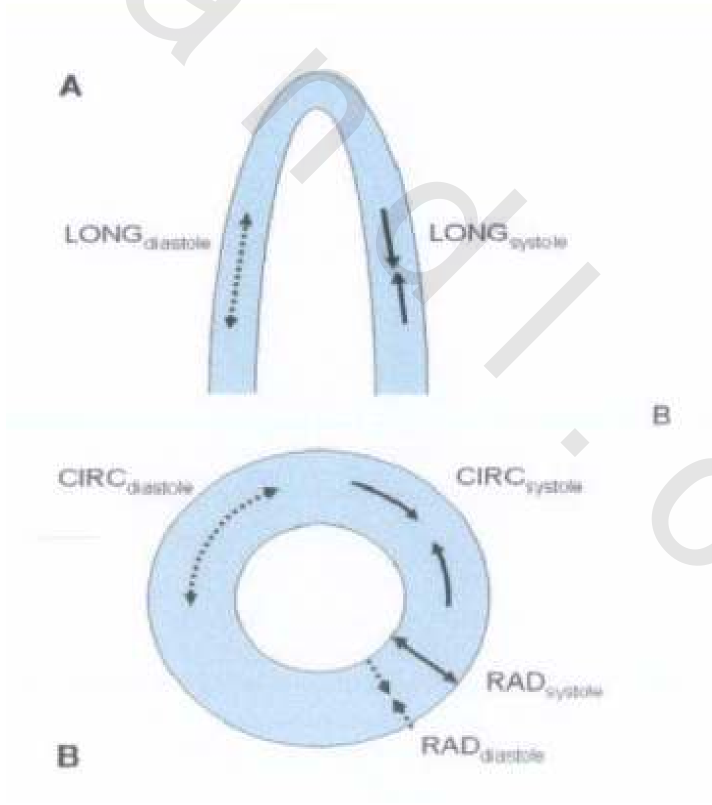

Figure (8): Graphic representation of the principal myocardial deformations: longitudinal (A), radial and circumferential (B) [81]

Doppler tissue imaging of the RV

Among the most reliably and reproducibly imaged regions of the right ventricle are the tricuspid annulus and the basal free wall segment these regions can be assessed by pulsed tissue Doppler and color-coded tissue Doppler to measure the longitudinal velocity of excursion. This velocity has been termed the RV S or systolic excursion velocity S <10 cm/s should raise the suspicion for abnormal RV function, particularly in a younger adult.(62) Assessment of the mid and apical ventricular free wall velocities is discouraged in the routine echocardiographic studies, because there is a lower rate of obtaining adequate signals. (83)

Because the interventricular septum does not exclusively reflect RV function, it should not be used alone to assess the right ventricle. (62)

Strain and strain rate values have been studied in a number of conditions affecting the right heart, including arrhythmogenic RV dysplasia, (84) pulmonary embolism, (85) PH, (86) systemic disease affection, (87) and amyloidosis. (88-90)

There is a lack of normative data for strain and strain rate measures of the right ventricle, and most values in normal subjects represent small groups of patients representing control arms in pathologic studies. (62)

Mean value of 2D peak systolic strain rate at the RV base is in one study including 61 patients is 1.62 (1.50-1.74). (62)

Mean value of 2D peak strain at the RV base in five studies including 183 patients is 28 (25-32)%. (62)