International Journal of Cardiology 191 (2015) 110–113

Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Review

"Left ventricular filling pressure(s)" — Ambiguous and misleading terminology, best abandoned

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ARTICLE INFO

Article history:
Received 6 April 2015
Accepted 30 April 2015
Available online 1 May 2015

Keywords:
Left atrial pressure
Left ventricular end diastolic pressure
Left ventricular filling pressure
Left ventricular filling pressures

ABSTRACT

The use of the terms "left ventricular filling pressure" and "left ventricular filling pressures" is widespread in the cardiology literature, but the meanings ascribed to these terms have not been consistent. Left ventricular end-diastolic pressure (LVEDP) and mean left atrial pressure (LAP) cannot be used interchangeably as they will often differ in magnitude in the presence of cardiac disease and they also have different clinical significance. LVEDP is the best pressure to use when considering left ventricular function, whereas mean LAP is the most relevant pressure when considering the tendency to pulmonary congestion. The mean LAP is also the most relevant pressure for determining whether pulmonary hypertension has a left heart (post-capillary) component. If only a left ventricular pressure tracing is available then a technique to measure the mean left ventricular diastolic pressure is the best option for estimating the mean LAP. If only right heart pressures are available then the pulmonary artery end-diastolic pressure will provide a reasonable estimate of LVEDP, but only when the heart and pulmonary circulation are normal. If there is mitral valve disease, left ventricular disease or pulmonary hypertension the LVEDP cannot be estimated from right heart pressures. The problem of the ambiguity of "filling pressure (s)" is readily solved by the abandonment of this term and the use of either LVEDP or mean LAP as appropriate.
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Use of the terms "left ventricular filling pressure" and "left ventricular filling pressures" is widespread in the cardiology literature and clinical practice and thus, presumably their current use is generally accepted. However, as will become clear, over the last 20 years there has not been a consistent interpretation of these terms, nor presumably much appreciation that having a consistent interpretation is of any importance. Essential principles related to the understanding and interpretation of the various left heart pressures, and in particular the differences between the left ventricular (LV) end-diastolic pressure (LVEDP) and the mean left atrial (LA) pressure (LAP), were presented in an editorial by Dr. Rahimtoola in 1973 [1]. Although the differences between LVEDP and mean LAP have important implications for both diagnosis and treatment in all types of left ventricular disease, as well as pulmonary hypertension, the principles described by Dr. Rahimtoola appear to have been forgotten. It is therefore not before time that the physiology and pathophysiology underlying the various left heart pressures is updated.

Understanding that there is a contribution from left atrial (LA) contraction to left ventricular (LV) filling began with William Harvey, progressed substantially with Gessell [2–4] in the early 20th century, advanced further in the 1960s with several landmark studies by Braunwald [5,6] and then again in the 1970s with studies by Rahimtoola [7,8]. In the normal cardiovascular system at rest, pressures in the pulmonary artery, left atrium and left ventricle are essentially equal at end-diastole [5,9]. Mean LA pressure (LAP) is also similar to LA end-diastolic pressure and mean LV diastolic pressure is similar to LV end-diastolic pressure (LVEDP). Given that all these pressures are nearly equal, it was not unreasonably suggested that they could be referred to together as LV filling pressures [1]. However, the limitation of the term LV filling pressures becomes apparent once there is any type of cardiac disease, in which case the mean LAP and the LVEDP cannot be assumed to be the same. Thus, in mitral stenosis there is a diastolic pressure gradient between the left atrium and left ventricle and the LAP can be higher than the LVEDP. In contrast, and a more common cause of a difference between LAP and LVEDP, a strong atrial contribution to LV filling can occur in patients with LV disease and this can result in a LVEDP which is considerably higher than the mean LAP [6–8]. In Braunwald's original comparison of LVEDP and mean LAP in patients with LV disease the LVEDP exceeded the mean LAP in all patients, with the pressure difference ranging from 1 to 18 mm Hg and averaging 9 mm Hg [6]. This study provided clear evidence that LA contraction could elevate the LVEDP while permitting the mean LAP to remain at a lower level. Indeed, the LVEDP could be >20 mm Hg while the mean LA pressure still remained within the normal range (<12 mm Hg). That LA contraction not only increases the LVEDP but can also provide

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http://dx.doi.org/10.1016/j.ijcard.2015.04.254
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a substantial component of LV filling in LV disease was demonstrated by Rahimtoola [8]. The importance of the LA contribution to LV filling in both LV disease and aging has subsequently been confirmed in a number of studies using either nuclear or echocardiographic techniques.

That the mean LAP and the LVEDP can differ in magnitude is of particular importance because the pathophysiological significance of these pressures is not the same. LVEDP provides an estimate of LV end diastolic volume (LVEDV), the latter being an important determinant of stroke volume and stroke work via the Frank–Starling mechanism. Therefore, with the important limitation that an initially linear relationship of LVEDP with LVEDV flattens out at high pressures [10,11], an increase in LVEDP will generally be associated with an increase in LVEDV and thus an increase in LV output [12]. On the other hand, the pulmonary circulation "sees" the LAP throughout the cardiac cycle and many of the manifestations of LV failure result either directly or indirectly from elevation of mean LAP, and of the associated elevation of pressures in the venous and capillary beds proximal to the left atrium. Therefore, LA contraction mediated elevation of LVEDP has the advantage of increasing preload of the left ventricle, but at the same time minimizing effects on the flow and pressure in the pulmonary circulation which would occur if there was a simultaneous increase in mean LAP. Moreover, while an increase in LVEDP may be a marker of LV disease (whether or not there is a reduction of LV ejection fraction), it is elevated mean LAP rather than elevated LVEDP which is likely to be the best guide of whether dyspnea might be related to pulmonary congestion, and therefore the best predictor of a symptomatic benefit from diuretic therapy. In contrast, a diuretic-induced reduction in LVEDP when mean LAP is not elevated may have adverse consequences as it may also be associated with a reduction of LVEDV and hence (via the Frank–Starling mechanism) a decrease in LV stroke volume and a reduction in perfusion of major organs.

There has been investigation in the past of other pressures measured in the pulmonary circulation as potential predictors of LVEDP. In particular, the pulmonary arterial end diastolic pressure (PAEDP) was considered promising because there is functional continuity between the pulmonary artery and left ventricle during the normal diastolic period which should permit equilibration of these pressures. However, while there may be a reasonable correlation between the PAEDP and the LV pre (a) pressure (the LV pressure immediately prior to atrial contraction) [13], this relationship is of less clinical significance, as in the setting of a significant LA contribution to LV filling the LV pre (a) pressure will not adequately reflect either the LVEDP or the LVEDV. Moreover, it has been shown that the PAEDP does not provide an accurate estimate of LVEDP in patients with chronic LV disease, that it fails to accurately reflect acute alterations in LVEDP and that it is particularly misleading if there is accompanying pulmonary hypertension due to elevation in pulmonary vascular resistance [7,14,15]. Of the pressures obtainable from left heart catheterization, the mean LV diastolic pressure provides the best estimate of the mean LAP in patients with LV disease as it closely mirrors the LAP during diastole and thus at least partly accounts for the LA A and V waves [16]. LAP is most commonly assessed by measurement of the pulmonary artery wedge pressure (PAWP) and although there are limitations of PAWP as a surrogate for LAP, the mean PAWP and mean LAP are very similar except at high levels of LAP (>30 mm Hg) [17]. There are also technical limitations of PAWP which can affect individual cases. In particular, it may be difficult or even impossible to obtain a wedge position in severe pulmonary hypertension and it is possible for a damped PA trace to be misinterpreted as a PAWP trace, resulting in overestimation of LAP.

In studies using Doppler echocardiography to estimate intracardiac pressures it has been common over the last 20 years to use the term "left ventricular filling pressures" as the aim of the non-invasive estimation [18–46]. However, as discussed above, in the circumstances of possible LV disease this general term is not specific as it does not distinguish between LVEDP, mean LAP or the other LV pressures which can be invasively measured. The ambiguous use of the term LV filling pressures is demonstrated in the above echocardiographic studies, where it has been used to refer to LVEDP [21,33,35,43], LV pre (a) pressure [22,26,28,40], mean LV diastolic pressure [27,34], directly measured mean LAP [20,37], mean PAWP [19,23–25,29,31,32,36,39,42,44,45] or as a general term for a number of the above pressures [18].

Given that mean LAP and LVEDP may diverge in the presence of cardiac disease and also have different clinical significance, it is important to appreciate that various echocardiographic variables have theoretical advantages for the assessment of these different pressures. The most reliable echocardiographic technique for the specific assessment of LVEDP uses the difference in the duration of the pulmonary venous and mitral Doppler flow signals due to LA contraction [18,47–49]. When the left atrium contracts there should, under normal circumstances, be greater forward flow into the left ventricle than backward flow into the pulmonary veins. If the pulmonary venous A-wave is increased in either velocity (>35 cm/s) or duration (>30 ms longer than mitral A-wave duration) this suggests that LV a-wave pressure is increased, in which case the LVEDP is also likely to be elevated. There are Doppler variables which will be more closely related to the mean LAP than the LVEDP because they reflect the early diastolic transmitral gradient, an important component of which is the level of the LAP at the time of mitral valve opening. While there are studies which have reported positive correlations of mean LAP with the transmitral E and E/A [18–20,50,51], it is well recognized that there are limitations in using either of these Doppler variables as a predictor of mean LAP as both E and E/A decrease with LV myocardial disease and aging. As a way of addressing the difficulties related to a U shaped relationship of E with myocardial dysfunction, the E/e′ ratio has been proposed as a means of estimation of mean LAP [23], although it is often in an indeterminate range [27] and has not always provided accurate predictions [39,41,52].

A recent study of 580 patients with pulmonary hypertension who had undergone simultaneous right and left heart catheterization and had a PAWP ≤ 15 mm Hg reported that more than 50% of the patients had LVEDP values > 15 mm Hg [53]. The authors concluded that PAWP frequently underestimates LVEDP, that it is poorly calibrated to LVEDP, and that it has only a moderate ability to discriminate between patients with normal or elevated LVEDP. A concerning thing about this conclusion is that it was not realized that all these points about the relationship between LAP and LVEDP were known in the 1960s (see above) and more importantly, that they are not valid concerns about mean LAP but represent genuine and physiological important differences between the two pressure measurements. While the authors considered that use of the LVEDP instead of PAWP could have avoided misclassification of patients as having a diagnosis of pulmonary arterial hypertension when they actually had left heart related pulmonary hypertension, this conclusion is also concerning as it appears to be a misunderstanding of the means by which LV dysfunction can cause pulmonary hypertension. It is in fact the elevation of mean LAP which leads to pulmonary hypertension, initially by a direct backward effect on the pulmonary circulation in all patients, but in a proportion of patients via an additional pulmonary vasoconstrictive component [54,55]. A genuine isolated increase in LVEDP (i.e. without elevation of LAP) cannot be the cause of pulmonary hypertension, although it may be a marker of an individual who is susceptible to developing elevation of LAP in the future.

In the evaluation of pulmonary hypertension by catheterization there are other considerations in the interpretation of the data. Both LAP and LVEDP can decrease with dehydration (e.g. fasting) and the use of diuretics, but following an acute reduction of LAP the PA pressure may not fall to same extent, even if it is pulmonary hypertension secondary to left heart disease, as there can be a delay in resolution of the pulmonary vasoconstrictive component [55,56]. Left heart catheterization may still have a role in the assessment of severe pulmonary hypertension if a PAWP cannot be obtained, but despite the recommendation in the European Society of Cardiology/European Respiratory Society guidelines [57], a LVEDP > 15 mm Hg should not be used in isolation

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