Evolution of Ventricular Interdependence Concept: Bernheim, Reverse-Bernheim Effect and Beyond!

Historically speaking, Bernheim is credited for hypothesizing the conceptual notion of ventricular interdependence. It was in 1910, a French physiologist, P. I. Bernheim reported patients with right heart failure wherein the necropsy revealed left ventricular (LV) hypertrophy with the inter-ventricular septum (IVS) bulging into the right ventricle (RV) [Figure 1]. He outlined his research as: *"*Venous asystole in hypertrophy of the left heart with associated stenosis of the RV,*"* whereby he postulated that LV hypertrophy is expected to compress the RV (which he termed as "stenosis"), leading to a diminished RV function.[1] This phenomenon was referred to as the Bernheim effect to characterize a syndrome of right heart failure without pulmonary congestion in the setting of diastolic left heart failure. The notion has been debated for over a century, considering the fact that a completely isolated diastolic RV failure is an uncommon mechanism of right heart failure phenotype, even in patients with left-sided heart failure.[2]

Despite the controversy emanating as a consequence of many researchers suggesting the inappropriateness of envisaging Bernheim syndrome as a realistic clinical entity,[3] Bernheim's initial description constituted the basis of subsequent comprehension of ventricular interdependence. Within a few years of Bernheim's elucidation, Henderson and Prince in 1914 studied an isolated cat heart preparation and depicted that pressure or volume overload of one ventricle detrimentally affects the output and function of the other.[4] However, ventricular interdependence was further elaborated upon by Dexter in 1956 with the description of the reverse-Bernheim effect.[5] In accordance with the reverse-Bernheim effect, RV hypertrophy and dilatation tend to shift the IVS leftward, resulting in decreased LV cavity, compliance, and contractile function or the ejection fraction [Figure 1]. This phenomenon has been described in the setting of both RV volume and pressure overload (pulmonary arterial hypertension) and over a wide range of severe pulmonary diseases.

The aforementioned phenomena are better delineated under the more physiological representation of ventricular interdependence. The continuity of muscle fibers of LV and RV, series circulatory arrangement, IVS, and the common pericardium associate the two ventricles functionally and form the basis of the contralateral impact on the size, shape, and the pressure-volume relationship. The majority of systolic ventricular interdependence is mediated by the IVS. However, the pericardium is a peculiarly important mediator of the diastolic ventricular interdependence.[6] Increased pericardial restraint in pathologies such as chronic constrictive pericarditis exaggerates the underlying ventricular interdependency which is further accentuated by augmented RV filling with inspiration.[7]

In context of the ventricular cross talk, the importance of the ventricular interactions is being increasingly realized in the present era of mechanical circulatory assist devices. On one hand, where RV dysfunction alters LV functional geometry, on the other, an LV assist device (LVAD) support induces counterproductive

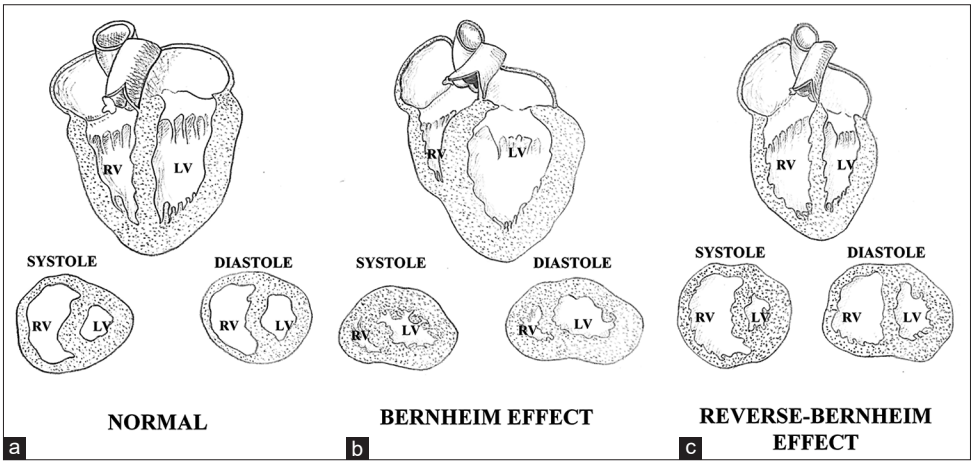

Figure 1: An illustration of the spectrum of ventricular interdependence. Depicting the normal concavity of the IVS toward the LV throughout the cardiac cycle (a) Outline of the original description of Bernheim effect wherein concentric LVH detrimentally affects the RV function owing to exaggerated rightward movement of the IVS (b) Reverse-Bernheim effect wherein RV pressure or volume overload tends to result in septal flattening and a consequential "D" shaped LV in setting of RV dysfunction (c). (IVS: Inter-ventricular septum, LV: Left ventricle, LVH: Left ventricular hypertrophy, RV: Right ventricle).

RV geometrical and functional alterations. This is attributable to the heavy reliance of the RV on the LV contribution to the septal function and the overall ventricular performance.[8] Following the institution of LVAD support, the LV is unloaded, and the combined effect of a diminished LV contractility and an elevated RV free-wall-septal distance has a detrimental impact on the RV function.[9] Moreover, the circulatory assistance augments the RV preload which is ineffectively handled by the declining RV performance. This necessitates the therapeutic measures aimed at preventing any accentuations in the RV afterload to alleviate any further deterioration of the RV function.

To conclude, the medical lexicon comprises terminologies that demonstrate an interesting history surrounding their original description although have laid the foundation of understanding of certain important clinical contexts. The elucidation of Bernheim and reverse-Bernheim effect is a representative example of the aforementioned fact, suggesting that there are enough parental factors that strongly bond the two ventricles together, which continue to be actively investigated till today under the formal categorization of ventricular interdependence.

Ethics clearance

The index manuscript is a Letter to the Editor, there are none ethical issues involved.

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Conflicts of interest

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