It seems there is a semantic confusion in the definition of perimembranous VSD coming from the vague definition given by many authors using the term subarterial or subaortic VSD to describe perimembranous VSD. When the aorta is far away, it seems that there is difficulty in distinguishing perimembranous VSD from inlet VSD. The definition of the membranous septum is independent from the aortic annulus. It is defined by Anderson [1] as being in contact with the central fibrous body, next to the tricuspid valve. The two specimens shown in our article are true DORV non-committed VSD as stated by authors Bob Anderson and Bob Freedom [2]; the VSD are perimembranous VSD with inlet extension.

The difference with a Taussig-Bing is not due to the nature of the VSD that is also a perimembranous VSD (but with outlet extension) but to a greater distance to the pulmonary valve. As shown by Bob Freedom [2], in Taussig-Bing the VSD is immediately located beneath the pulmonary valve and located above the posterior limb of the trabecula septomarginalis. In DORV non-committed VSD, the VSD is distant from the pulmonary valve and located beneath the posterior limb of the trabecula septomarginalis. True muscular inlet or trabecular VSDs are possible, although we have not found any in our series. As stated by Belli [3], in DORV non-committed VSD, the distance from both arterial annuli should be greater than an aortic annulus diameter. In Taussig-Bing, the distance of the VSD from the pulmonary artery is less than an aortic valve diameter and in DORV non-committed VSD this distance is greater.

One consequence is that the VSDs in DOR non-committed VSD are frequently restrictive which we have never seen in Taussig-Bing. The VSD has to be enlarged anteriorly, as was done in all the cases published. There was no heart block in our series, confirming the perimembranous nature of the VSD. I consider that the VSD depicted by Barbero-Marcial [4] as muscular VSD are in fact mostly true perimembranous VSD. Notice also that an AVSD defect is in contact with the tricuspid annulus and is a perimembranous VSD with inlet extension.

Another difference in Taussig-Bing is the usual overriding of the pulmonary artery that is partly located on the left ventricle (now named DORV-TGA type). In DORV non-committed VSD, the vessels are very clearly 200% on the RV.

In DORV non-committed VSD hearts, there is an association with subpulmonary stenosis which is never the case in Taussig-Bing.

Finally, Taussig-Bing belongs more to the group of TGA VSD and is almost exclusively treated by arterial switch (the Kawashima operation being rarely indicated) and on the contrary, the DORV non-committed VSD can be treated differently by VSD tunnellingization to the aorta in favourable forms and by tunnellingization to the PA and ASO in the most severe forms.

References


Letter to the Editor

Bilateral harvesting of internal thoracic artery for coronary bypass: augmenting the risk for postoperative airway obstruction?

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Keywords: Complication; Coronary bypass; Internal mammary artery; Internal thoracic artery; Outcome; Vocal cord paralysis

You published two interesting papers on iatrogenic vocal cord dysfunction, recently by Hamdan et al. [1] and previously by Shafei et al. [2]. In the era of total arterial coronary revascularisation, another relevant pathological mechanism springs to mind:

It is known that trauma to the recurrent laryngeal nerve (RLN) compromises the posterior cricoarytenoid muscle (abductor of the ipsilateral vocal cord) and thus paralyses the latter structure in adduction. Unilateral vocal cord paralysis may manifest as hoarseness or other deficit in phonation. Bilateral paralysis causes life-threatening acute airway obstruction, estimated to 1.9% in all-comers to cardiac surgery by Shafei et al. [2].

It is of particular interest to the coronary surgeon that the RLN may be injured during the harvesting of internal thoracic (mammary) artery (ITA) [3], where it ‘hooks’ around the subclavian artery (SA).

Most of us are increasingly using bilateral ITAs [4], the right ITA being often mobilised up to its origin from the SA [5]. Bilateral dissection exposes both right and left RLNs to concurrent intraoperative injury.

Will this evolution in cardiothoracic practice increase the incidence of bilateral vocal cord paralysis in coronary surgery? Does the risk warrant further prospective evalua-
tion with a view to medico legal implications and informed consent?

In any case, we would consider prudent to keep this potential disaster mechanism in mind, in addition to the scenarios meticulously enumerated by Hamdan et al.[1].

References


Reply to the Letter to the Editor

Reply to Protopapas

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The author has raised two concerns: One regarding the possible increase in recurrent laryngeal nerve injury with the upcoming evolution in cardiothoracic surgery, where the internal thoracic artery is completely harvested bilaterally thus exposing both recurrent laryngeal nerves, second whether this potential increase in risk warrants further prospective evaluation in view of the medico legal implications.

Regarding the first concern, no doubt that bilateral usage of the internal thoracic artery for total arterial coronary revascularization can theoretically increase the incidence of vocal cord paralysis since both recurrent laryngeal nerves are exposed and dissected during the surgery. However, no study has compared the outcome of unilateral versus bilateral vocal cord paralysis in unilateral versus bilateral dissection of the mammary arteries following open heart surgery, most likely in view of the very small incidence [1].

Regarding the second concern, no study has confirmed the exact etiology behind the paralysis or paresis of the vocal cords following open heart surgery. Injury to the recurrent laryngeal nerve has been suspected but the exact mechanism of injury has never been specified or drawn, and this is mainly due to the difficulty in determining the site of injury and also due to the spontaneous recovery after few months [2]. True, dissection of the Internal thoracic artery at the level of the subclavian artery can expose the recurrent laryngeal nerve, however dissection and exposure of the recurrent laryngeal nerve are not enough to incriminate the harvesting of these vessels as an increase in the risk of vocal cord paralysis knowing that other mechanisms of injury such as traction on the major vessels and sternotomy are as likely to be responsible for the insult [3–5]. Hence I do not believe that this evolution in Cardiothoracic surgery should prompt the surgeons to change their technique nor to have medico-legal concerns. Maybe it will be informative to do a comparative study between two groups of open heart surgery patients operated on and anesthetized by the same team, whereby in one group one mammary artery is harvested and in the other two are harvested. If the results show that the incidence of vocal cord paralysis is higher in the second group, maybe further evaluation of the harvesting technique should be evaluated.

References


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