University of Wisconsin School of
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Milwaukee, Wis

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MISUSE OF THE TERM “SUBCLAVIAN ACCESS”

To the Editor:

In a recent issue of the *Journal*, there was an article by Ramlawi and colleagues¹ entitled, “Direct aortic and subclavian access for transcatheter aortic valve replacement: Decision making and technique.” I was enthusiastic to read the article because the title potentially indicated a description of a novel access for transarterial aortic valve implantation through direct puncture for access of the subclavian artery. I was a bit disturbed to realize that Ramlawi and colleagues¹ do not actually use or expose the subclavian artery, but in fact the axillary artery. Per anatomic definition, the subclavian artery extends to the lateral border of the first rib, where it becomes the axillary artery, which in turn becomes the brachial artery after passing the lower margin of the major teres muscle. Both from their figure and the description (incision in the deltopectoral groove), it is obvious that Ramlawi and colleagues¹ use the axillary artery as access site and not the subclavian artery. They depict the same approach as vascular surgeons use when performing axillofemoral bypass grafting.² The usual access to the subclavian artery is by a supraclavicular incision, as used in deviation surgery before implantation of endovascular aortic arch grafts.³ The message is that we should be accurate in the use of medical terms

when reporting on anatomic structures with precisely defined names.

Sven M. Almdahl, MD, PhD
Department of Cardiothoracic and
Vascular Surgery
University Hospital of North Norway
Tromsø, Norway

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Reply to the Editor:

We appreciate Almdahl's contribution. His letter describes concern about arterial access nomenclature, defining subclavian artery access as supraclavicular and stating that our approach, which is infraclavicular, is axillary rather than subclavian.

From an anatomic standpoint, the subclavian, axillary, and brachial arteries represent a continuum starting from the point of emergence of the subclavian artery in the chest and extending to the terminal branching of the brachial artery in the antecubital fossa. Three anatomic territories are limited by the inferior border of the first rib and that of the teres major muscle. Further divisions into thirds of the subclavian and axillary arteries are defined relative to the scalenus anterior and the pectoralis minor muscles, each passing anterior to the second portion of the respective artery of interest.

The subclavian artery can in fact be accessed through either a

supraclavicular or an infraclavicular approach. With the latter approach, the infraclavicular portion of the subclavian artery is accessed through an incision in the Mohrenheim fossa, through the pectoralis major fibers. The deltopectoral fascia is accessed cranial and medial to the pectoralis minor muscle, exposing the area of the vessel between the lateral border of the scalenus anterior and the medial border of the pectoralis minor. This fully exposes the third part of the subclavian artery, transitioning into the first part of the axillary artery. A clear distinction between these zones bears little importance for the procedural technique, and individual surgeons are encouraged to choose the puncture point with which they are most comfortable.

We described our technique as an infraclavicular yet still a distal subclavian puncture, and this has been similarly described by a number of other authors.¹⁻⁴ This same infraclavicular approach has also been described for subclavian access for balloon pump insertion.⁵ Almdahl's point is very well taken. Axillary access also describes how some surgeons would opt to access the vessel. For future use, we believe that the most anatomically descriptive nomenclature for the infraclavicular approach would be the term *subclavian/axillary access*, as described in a few reports.^{6,7}

We again thank Almdahl for his enriching contribution.

Basel Ramlawi, MD
Kareem Bedeir, MBChB, MS
Michael Reardon, MD
Methodist DeBakey Heart and
Vascular Center
Houston, Tex

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THE AORTIC WALL WITH BICUSPID AORTIC VALVE: IMMATURE OR PREMATURELY AGING?

To the Editor:

We read with interest the study by Grewal and coworkers¹ suggesting a maturation defect of the aortic wall in patients with bicuspid aortic valve (BAV). A number of recent publications have addressed the pathobiology of BAV-associated aortopathy; however most of them just vaguely concluded that BAV and tricuspid aortic valve must have different mechanisms driving aortic dilatation. Grewal and coworkers¹ are commendable for their effort to define the nature of this difference.

Their data, however, are partially in contrast with previous studies, with which they failed to perform a critical comparison. We have reported² increased α -smooth muscle actin messenger RNA expression and only mildly and nonhomogeneously increased overall protein levels, with subintimal areas of α -smooth muscle

actin-positive cell loss. This relative stability of the amount of α -smooth muscle actin (in contrast with Grewal and coworkers' data¹), coupled with smoothelin decrease (not absence, unlike in Grewal and coworkers' results¹) was consistent with other studies both of samples from patients and of experimental models of aneurysm (discussed in our own article²) and suggests a loss of contractile-phenotype smooth muscle cells and the emergence of a differentiated myofibroblast line.

In our previously mentioned study,² we selected BAV and tricuspid aortic valve patients with aortic stenosis with a maximal diameter less than 4 cm and analyzed both the concavity and the convexity of the vessel to minimize confounding factors by distinguishing constitutive from stress-induced changes. Some of the authors among Grewal and coworkers have previously borrowed in their studies this protocol of sample retrieval from different sites of each aorta, first introduced by us,³ and confirmed that aortic wall changes are asymmetrically expressed with BAV, suggesting a role for long-standing flow and stress pattern alterations in their development. The change of smooth muscle cell orientation from circumferential to longitudinal direction that we observed in the convexity² is known to occur in vessels submitted to altered tensile strain. The undefined site of sampling in the study of Grewal and coworkers,¹ conversely, could explain these discrepancies.

Similarly, Grewal and coworkers¹ stated that the aortic wall specimens were obtained during full-root stentless implantation in patients with nondilated BAV,¹ suggesting that the BAV specimens were taken from the root (sinuses of Valsalva) instead of the ascending tubular tract proper. Where were the aortic specimens taken in the other patients, also considering that BAV aortopathy usually affects the ascending tract?

How was valve function in the 4 groups? Also, the mean diameters per subgroup were not listed.¹ Intimal thickness data¹ were at odds with previous investigations^{2,4}; could different diameters between BAV and tricuspid aortic valve subgroups explain this?

The paradigms of flow-induced remodeling share many mechanisms with the vascular aging process, including increased transforming growth factor- β receptor II signaling,⁵ which has been evidenced in aortopathies both with and without associated BAV.² Functionally, one of the early signs of arterial aging is impaired wall distensibility, which is known to occur in the BAV aorta, even before overt dilation. Thus with the currently available data, an hypothesis of defective wall maturation is hardly sustainable without ruling out the contribution of flow-induced remodeling. A conceptually opposite theory of premature aging of the aortic wall, prompted by altered biomechanical environment, could be drawn as well.

Amalia Forte, PhD^a

Alessandro Della Corte, MD, PhD^b

^aDepartment of Experimental Medicine

Second University of Naples
Naples, Italy

^bDepartment of Cardiothoracic and Respiratory Sciences
Second University of Naples
Naples, Italy

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