

## Two problems during one pacemaker implantation procedure: axillary vein spasm and subclavian vein compression, or ‘every cloud has a silver lining’

Dwa problemy jednej procedury CIED – spazm AV i supresja SV,  
a może „nie ma tego złego, co by na dobre nie wyszło”

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### Abstract

Cardiac implantable electronic device (CIED) implantation procedures have become an indispensable part of treating the clinical manifestations of arrhythmias and/or heart conduction disorders.

The first stage of CIED implantation involves the insertion of cardiac leads into the venous system via a cephalic vein cut down and/or axillary vein/subclavian vein (SV) puncture using special kits designated for this purpose. Similar techniques are used for central venous catheter (CVC) placement. Nonetheless, the course and effectiveness of this stage of the procedure may be affected by mediastinal vein anomalies, atypical venous morphometry and/or topography, reflex venospasm, and – in the case of the SV – the very fact of its coursing through the costoclavicular space.

The rare coexistence of several unfavourable factors and the degree of such anomalies may sometimes prevent the originally planned approach, which happened in the case presented here.

Key words: venography, venospasm, venipuncture, venous compression, subclavian vein, axillary vein, CIED, CVC, TOS

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### Introduction

The special tools used in obtaining access to the cardiovascular system during such procedures as cardiac implantable electronic device (CIED) implantation or central venous catheter (CVC) placement help introduce the cardiac leads/catheters without the necessity of venesection [1].

Nonetheless, despite the existence of alternative options, even this initial stage of the procedure may be hindered by difficulties in obtaining venous access. The risk of such difficulties increases in anomalies of the systemic veins of the mediastinum, considerably atypical venous morphometry and/or topography, venospasm, etc. [2–5].

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**Figure 1.** Contrast-enhanced radiographic images showing the morphometry and topography of the veins of both claviopectoral triangles (the left one in panel **A** and the right one in panel **B**) in the presented 59-year-old man, and their relationship to the bone structures of the chest. Both venography assessments were performed in posteroanterior (PA) views with the contrast agent administered through a cubital fossa vein. The differentiating characteristics of the drainage sites for the superficial vein (panel **A**) and the subclavian vein (SV) (panel **B**) have been marked with circles; namely, the circle in panel **A** marks the drainage site into a larger vein, which is not the left brachiocephalic vein (BCV), whereas the circle in panel **B** marks the drainage point of the right SV into the right BCV: **A.** The left side of the chest. An ineffective attempt at subclavian/axillary vein puncture under venographic guidance; the superficial vein (white arrowheads); the needle from the cardiac lead placement kit (black arrow); **B.** The right side of the chest. Contrast flow through the patent, lead-carrying veins: the axillary vein (AV) → subclavian vein (SV) → brachiocephalic vein (BCV) → superior vena cava (SVC)

In the case of using the SV, a possible compression of this vessel in the costoclavicular space may also produce a problem [6, 7].

In the case presented below, the failed attempt to establish venous access was due to several different phenomena of various aetiologies.

### Case report

A 59-year-old male farmer, with no significant medical history, was admitted to the hospital to receive a CIED due to his Morgagni–Adams–Stokes attacks resulting from a paroxysmal complete atrioventricular conduction block.

The initially planned approach to cardiac lead insertion was via the veins of the left claviopectoral triangle. However, a failure to locate the CV in the deltopectoral groove led to the use of the AV/SV puncture approach instead, also under venographic guidance. The contrast agent, which was administered into a cubital fossa vein, visualized only a single narrow superficial vein coursing in the left claviopectoral triangle towards the angle formed by the clavicle and the first rib (Figure 1A). The position of the vessel in a posteroanterior (PA) view alone, may have been misinterpreted as that of the AV, albeit an underdeveloped, anomalous, or spastic one.

Considering this unexpected situation, the patient's pacemaker implantation procedure was ultimately rescheduled for the following day, with the pacemaker ultimately

implanted on the right side of the chest. The cardiac leads were introduced via CV cut down, and the procedure itself was preceded by venography to assess the layout and accessibility of relevant veins (Figure 1B).

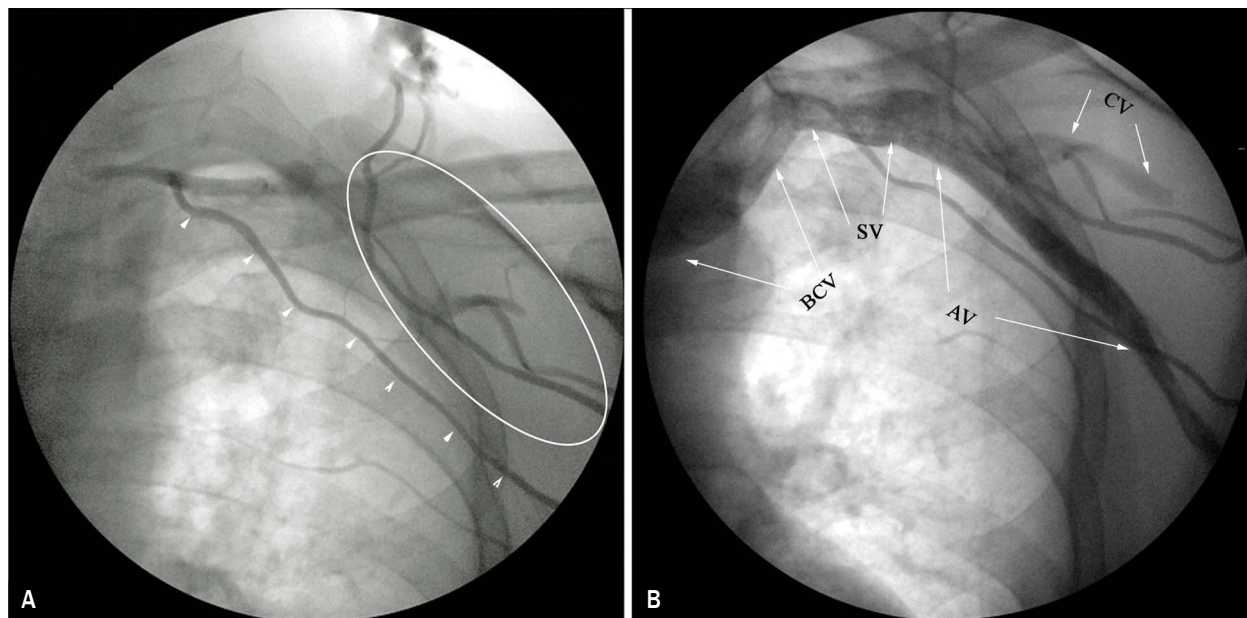
The phenomenon of superficial veins of the left claviopectoral triangle newly filled with the contrast agent (Figure 2A) in addition to just the single vein visualized during the first venography (Figure 1A), within the area for attempted access to the AV, indicates their previous reflex spasm in response to a traumatic stimulus.

Ultrasonography also showed a position-dependent SV compression by adjacent bone structures, which was due to their shifting location in space during movements in the left sternoclavicular joint (Figure 3).

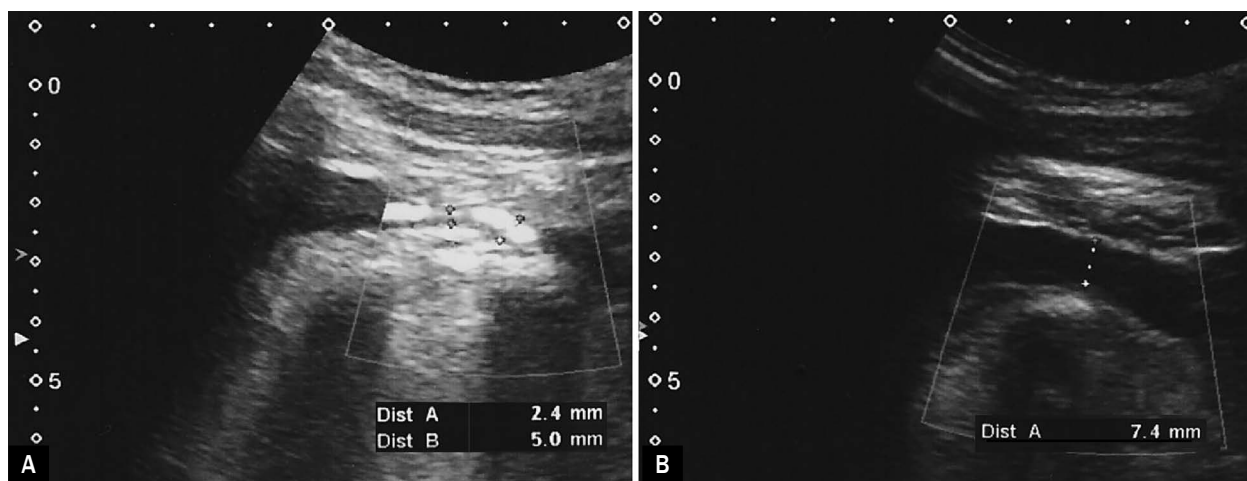
### Discussion

Classic CIED implantation procedures make use of the veins situated in the claviopectoral triangle (i.e. CV, AV, or SV) for cardiac lead insertion [1, 2, 4].

Forgoing the initially planned CV cut down due to a failure to locate the vessel in its typical anatomical position in the left deltopectoral groove was the key reason for changing the technique and using the left AV or SV puncture instead. Interestingly, the alternative technique also failed to produce the desired effect, and venography visualized only a single superficial vein on the thoracic wall within the left claviopectoral triangle (Figure 1A). Due to its



**Figure 2.** The venographic re-evaluation of the veins of the left infraclavicular region performed on the next day. The different numbers of contrast-enhanced vessels depend on the position of the upper limb (panels **A** and **B**); **A**. With the upper limb in the position typically adopted during the procedure (i.e., lying horizontally, adducted, along the torso), the flow of contrast is visible only in the superficial veins of the clavipectoral triangle; these include the vessel originally visualized (in Figure 1A, white arrowheads) and new, previously non-visualized veins (oval). The axillary vein (AV) and subclavian vein (SV) remain invisible, with no contrast enhancement of their lumen; **B**. A change in the upper limb position and the respective angle at the sternoclavicular joint reverses SV compression by adjacent bone structures (the clavicle and first rib) allowed the contrast to fill the AV and SV, as well as retrogradely fill the CV



**Figure 3.** Ultrasound images: **A**. The patient in a horizontal position with the left upper limb lying along the torso – subclavian vein (SV) compression in its middle segment to a minimum diameter of 2–5–3 mm, depending on the respiratory phase; **B**. During the Valsalva manoeuvre (inspiration), the SV diameter increases to 7 mm

course, as visualized via PA fluoroscopy, the vein could be initially mistaken for an atypical AV. However, this conclusion was contradicted by the fact that the vein in question drained to a vessel that was not the left brachiocephalic vein (BCV) (Figure 1A and 2B).

The presence of the left AV and SV was confirmed on venography conducted the following day, which visualized the actual topography and morphometry of those vessels (Figure 2B). A comparison of the number and characteristics of the left clavipectoral triangle vessels visualized

during the two venographic assessments (Figure 2A and 1A) suggests an episode of venospasm triggered by venous puncture and affecting not only the AV and SV but also veins of the chest wall (Figure 1A). It is possible that a similar reflex venospasm was the reason why the CV could not be found, with the manoeuvres involved in searching for the vessel being the traumatic trigger for the spasm in that instance (Figure 1A and 2B).

The shape of the costoclavicular space is also subject to individual variations. This is one of the causes of venous thoracic outlet syndrome (TOS), with the severity of the resulting symptoms reflecting the extent of compression of the following structures: the brachial plexus, the subclavian artery and/or vein. The isolated vascular form of TOS associated with SV compression is found in 3–5% of TOS patients. This form of TOS often leads to localized venous thrombosis referred to as Paget-Schroetter syndrome [6, 7].

Another reason behind the absence of visualized vessels in the case presented here may have been a temporary compression of the SV by the clavicle. This may have been exacerbated by the patient's position during the procedure since a subsequent ultrasound examination revealed a position-dependent nature of this compression (Figure 3A). This position-dependent occlusion of the SV lumen prevented the contrast agent to fill and visualize the AV (Figure 2A).

It is likely that the decision to forgo the procedure on the left side reduced the risk of potential complications due to SV compression, such as thrombosis secondary to endothelial injury by the cardiac leads positioned within the narrowed venous lumen and/or mechanical damage to the cardiac leads by the bone and muscle structures compressing the vein, ultimately leading to CIED dysfunction [8–10].

### Conflict of interests

The authors declare no conflict of interest.

### Streszczenie

Procedury implantacji wszczepialnych urządzeń do elektroterapii serca (CIED) stały się obecnie niezbędnym elementem terapii klinicznych następstw zaburzeń rytmu i/lub przewodzenia układu bodźcowo-przewodzącego serca.

Pierwszym etapem implantacji CIED jest wprowadzenie elektrod do układu żylno-sercowego – z wenesekcji żyły odpromieniowej i/lub nakłucia żyły pachowej/podbojczykowej (SV) przeznaczonymi do tego celu zestawami. Analogiczne techniki stosuje się do wprowadzenia kateterów podczas procedur CVC (*central venous catheter*). Niemniej na przebieg i efektywność tego etapu zabiegu mogą wpłynąć takie czynniki, jak obecność wad systemowych naczyń żylnych śródpiersia, morfometria i/lub topografia naczyń inna niż typowo obserwowana, żyłne odruchy spastyczne, a w przypadku SV – sam fakt jej lokalizacji w przestrzeni obojczykowo-żebrowej.

Zbieżność czasowa wystąpienia kilku niesprzyjających czynników i ich zaawansowanie może niekiedy wpłynąć na odstąpienie od realizacji procedury w miejscu pierwotnie zaplanowanym, podobnie jak w prezentowanym przypadku.

Słowa kluczowe: wenografia, spazm żylny, nakłucie żyły, ucisk żyły, żyła podbojczykowa, żyła pachowa, CIED, CVC, TOS

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### References

1. Bongiorni MG, Proclemer A, Dobreanu D, et al. Scientific Initiative Committee, European Heart Rhythm Association. Preferred tools and techniques for implantation of cardiac electronic devices in Europe: results of the European Heart Rhythm Association survey. *Europace*. 2013; 15(11): 1664–1668, doi: [10.1093/europace/eut345](https://doi.org/10.1093/europace/eut345), indexed in Pubmed: [24170423](https://pubmed.ncbi.nlm.nih.gov/24170423/).
2. Loukas M, Myers CS, Wartmann ChT, et al. The clinical anatomy of the cephalic vein in the deltopectoral triangle. *Folia Morphol (Warsz)*. 2008; 67(1): 72–77, indexed in Pubmed: [18335417](https://pubmed.ncbi.nlm.nih.gov/18335417/).
3. Steckiewicz R, Górko D, Świętoń EB, et al. Axillary vein spasm during cardiac implantable electronic device implantation. *Folia Morphol (Warsz)*. 2016; 75(4): 543–549, doi: [10.5603/FM.a2016.0027](https://doi.org/10.5603/FM.a2016.0027), indexed in Pubmed: [27830883](https://pubmed.ncbi.nlm.nih.gov/27830883/).
4. Yang HJ, Gil YC, Jin JD, et al. Novel findings of the anatomy and variations of the axillary vein and its tributaries. *Clin Anat*. 2012; 25(7): 893–902, doi: [10.1002/ca.22086](https://doi.org/10.1002/ca.22086), indexed in Pubmed: [22623347](https://pubmed.ncbi.nlm.nih.gov/22623347/).
5. Oginosawa Y, Abe H, Nakashima Y. Prevalence of venous anatomic variants and occlusion among patients undergoing implantation of

- transvenous leads. *Pacing Clin Electrophysiol.* 2005; 28(5): 425–428, doi: [10.1111/j.1540-8159.2005.09534.x](https://doi.org/10.1111/j.1540-8159.2005.09534.x), indexed in Pubmed: [15869675](https://pubmed.ncbi.nlm.nih.gov/15869675/).
6. Illig KA, Doyle AJ. A comprehensive review of Paget-Schroetter syndrome. *J Vasc Surg.* 2010; 51(6): 1538–1547, doi: [10.1016/j.jvs.2009.12.022](https://doi.org/10.1016/j.jvs.2009.12.022), indexed in Pubmed: [20304578](https://pubmed.ncbi.nlm.nih.gov/20304578/).
  7. Demondion X, Bacqueville E, Paul C, et al. Thoracic outlet: assessment with MR imaging in asymptomatic and symptomatic populations. *Radiology.* 2003; 227(2): 461–468, doi: [10.1148/radiol.2272012111](https://doi.org/10.1148/radiol.2272012111), indexed in Pubmed: [12637678](https://pubmed.ncbi.nlm.nih.gov/12637678/).
  8. Weiner S, Patel J, Jadonath RL, et al. Lead failure due to the subclavian crush syndrome in a patient implanted with both standard and thin bipolar spiral wound leads. *Pacing Clin Electrophysiol.* 1999; 22(6 Pt 1): 975–976, doi: [10.1111/j.1540-8159.1999.tb06829.x](https://doi.org/10.1111/j.1540-8159.1999.tb06829.x), indexed in Pubmed: [10392402](https://pubmed.ncbi.nlm.nih.gov/10392402/).
  9. Jacobs DM, Fink AS, Miller RP, et al. Anatomical and morphological evaluation of pacemaker lead compression. *Pacing Clin Electrophysiol.* 1993; 16(3 Pt 1): 434–444, doi: [10.1111/j.1540-8159.1993.tb01606.x](https://doi.org/10.1111/j.1540-8159.1993.tb01606.x), indexed in Pubmed: [7681195](https://pubmed.ncbi.nlm.nih.gov/7681195/).
  10. Said SAM, Ticheler CH, Stassen CM, et al. Possible complications of subclavian crush syndrome. *Neth Heart J.* 2005; 13(3): 92–97, indexed in Pubmed: [25696461](https://pubmed.ncbi.nlm.nih.gov/25696461/).