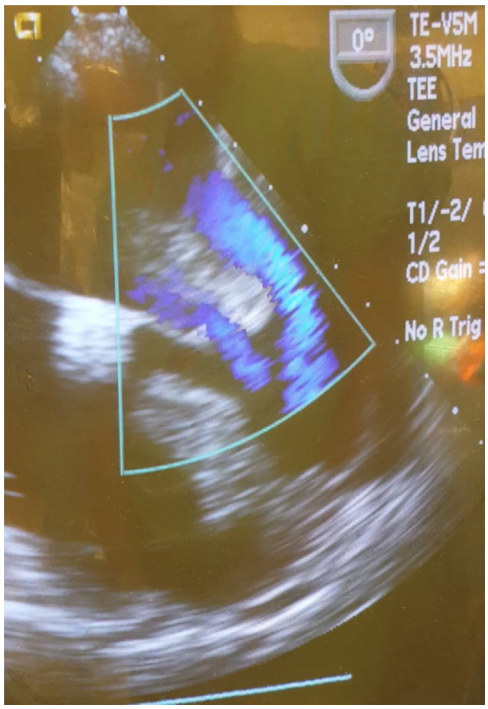


Fig 2. Transesophageal echocardiography showing severely limited flow into the left ventricle.

resolution of symptoms. In this case, the potential varying presentation of cardiac masses yielded a high-grade mitral stenotic lesion. If untreated, obstructive mitral stenosis will lead to left atrial enlargement, atrial fibrillation, pulmonary hypertension, right heart failure, and reduced cardiac output.[5] Understanding mitral stenosis helped guide the anesthetic plan for a successful surgical procedure. Maintaining preload and contractility while avoiding tachycardia and afterload reduction was paramount. This necessitated fluid administration to ensure normovolemia, and preinduction arterial cannulation to monitor potential induction-induced cardiovascular collapse without delay. Mass excision during CPB relieved this obstruction resulting in significant improvements in hemodynamic profile and subsequent symptomatology.

Surgical resection of malignant cardiac neoplasm is achievable in less than half of presenting patients. Surgery is rarely curative as many sarcomas have spread to other sites by the time diagnosis is obtained. Mean survival time is 7 months to 2 years.[3] Of the 11 cases mentioned above, 3 of the patients survived more than 11 months after surgery.[6]

Leiomyosarcoma is distinctive because metastatic lesions favor the pulmonary veins and left heart.[5] Definitive treatment is radical surgical resection followed by adjuvant radiation and/or chemotherapy. Postoperative chemotherapy should be considered because of possible incomplete resection. Select cases revealed estrogen and progesterone receptor proteins with a potential role for receptor-targeted therapies. For leiomyosarcoma the mean survival after surgery and adjuvant therapies is 6.8 months.[7] An optimal treatment regimen has yet to be defined.

Suggested conditions to resect lung metastases include that the primary lesion has been definitively treated, there are no other sites of metastasis, and the patient will be able to tolerate the procedure.[8] Although our patient did not meet these criteria, she was experiencing significant symptomatology, and therefore, surgical intervention was offered.

## References

1 Reynen K. Frequency of primary tumors of the heart (brief report). Am J Cardiol 1996;77:107.
2 McAllister HA, Fenoglio JJ. Tumors of the cardiovascular system In: Firminger HI, editor. Atlas of Tumor Pathology, 2nd Series, fascicle 15. Washington, DC: Armed Forced Institute of Pathology; 1978. p. 46–52.
3 Burke A, Virmani R. Tumors of the heart and great vessels. Washington, DC: Armed Forces Institute of Pathology; 1996.
4 Raaf HN, Raaf JH. Sarcomas related to the heart and vasculature. Semin Surg Oncol 1994;10:374–82.
5 Araoz PA, Eklund HE, Welch TJ, et al. CT and MR imaging of primary cardiac malignancies. Radiographics 1999;19:1421–34.
6 Funakoshi Y, Mukohara T, Kataoka T, et al. Left atrial extension of metastatic lung tumor via pulmonary vein: Report on the first case of Ewing sarcoma. Rare Tumors 2010;2:53.
7 Avakian S, Takada J, Mansur A. Giant obstructive left atrial myxoma resembling mitral valve stenosis. Clinics 2012;67:853–4.
8 Alexander J, Haight C. Pulmonary resection for solitary metastatic sarcoma and carcinoma. Surg Gynecol Obset 1947;85:129.

Michael Stuart Green, DO, MBA, FASA
Christopher Ryan Hoffman, DO
Scott Coleman, DO
*Drexel University College of Medicine*
*Hahnemann University Hospital*
*Department of Anesthesia*
*Philadelphia, PA*

https://doi.org/10.1053/j.jvca.2018.01.017

## Systemic Inflammation after Transcatheter Aortic Valve Implantation: A Prospective Exploratory Study

*To the Editor:*

Several studies showed that the systemic inflammatory response syndrome (SIRS) after transcatheter aortic valve implantation (TAVI) is associated with an increased risk of morbidity and mortality.[1–3] The pathogenesis of SIRS after TAVI is not clear. In one study patients with SIRS had more rapid ventricular pacing (RVP) runs and higher postprocedural lactate plasma concentrations compared with patients without SIRS.[1] RVP and other characteristics of the TAVI procedure, such as balloon-valvuloplasty and post-dilatation of the native

Table 1
Baseline Characteristics

| Variable | All (n = 39) | SIRS (n = 18) | No SIRS (n = 21) | p Value |
|---|---|---|---|---|
| Male, n (%) | 25 (64.1) | 13 (72.2) | 12 (57.1) | 0.328 |
| Age, y | 81 ± 6 | 79 ± 7 | 83 ± 5 | 0.068 |
| BMI, kg/m$^2$ (%) | 25.7 ± 3.9 | 26.7 ± 3.9 | 24.8 ± 3.7 | 0.134 |
| Comorbidity | | | | |
| Diabetes, n (%) | 14 (35.9) | 7 (38.9) | 7 (33.3) | 0.718 |
| COPD, n (%) | 8 (20.5) | 4 (22.2) | 4 (19.0) | 1.000 |
| Hypertension, n (%) | 23 (59.0) | 8 (44.4) | 15 (71.4) | 0.088 |
| Coronary artery disease, n (%) | 20 (51.3) | 9 (50.0) | 11 (52.4) | 0.882 |
| Atrial fibrillation, n (%) | 21 (53.8) | 11 (61.1) | 10 (47.6) | 0.399 |
| Previous myocardial infarction, n %) | 8 (20.5) | 6 (33.3) | 2 (9.5) | 0.112 |
| Previous cardiac surgery, n (%) | 19 (48.7) | 8 (44.4) | 11 (52.4) | 0.621 |
| Previous stroke, n (%) | 6 (15.4) | 4 (22.2) | 2 (9.5) | 0.387 |
| Peripheral artery disease, n (%) | 5 (12.8) | 2 (11.1) | 3 (14.3) | 1.000 |
| Chronic renal failure, n (%) | 13 (33.3) | 5 (27.8) | 8 (38.1) | 0.496 |
| Pulmonary hypertension, n (%) | 13 (33.3) | 6 (33.3) | 7 (33.3) | 1.000 |
| EuroSCORE | 18 (11-32) | 18 (11-32) | 18 (13-32) | 0.835 |
| STS score | 5.6 (3.7-8.6) | 4.6 (3.2-9.9) | 5.9 (4.6-8.6) | 0.549 |
| Normal LVEF, n (%) | 17 (43.6) | 5 (27.8) | 12 (57.1) | 0.065 |
| Hemoglobin, mmol/L | 8.0 ± 1.0 | 8.0 ± 1.1 | 8.0 ± 0.9 | 0.945 |
| Creatinine, μmol/L | 102 (80-128) | 104 (83-120) | 97 (77-131) | 0.813 |
| Medication use | | | | |
| *Preprocedural* | | | | |
| Statin, n (%) | 20 (51.3) | 8 (44.4) | 12 (57.1) | 0.429 |
| Steroids, n (%) | 11 (28.2) | 8 (44.4) | 3 (14.3) | 0.037 |
| B-blocker, n (%) | 24 (61.5) | 11 (61.1) | 13 (61.9) | 0.959 |
| Aspirin, n (%) | 16 (41.0) | 8 (44.4) | 8 (38.1) | 0.688 |
| Oral anticoagulation, n (%) | 18 (46.2) | 9 (50.0) | 9 (42.9) | 0.656 |
| *Postprocedural* | | | | |
| Statin, n (%) | 19 (48.7) | 7 (38.9) | 12 (57.1) | 0.256 |
| Steroids, n (%) | 10 (25.6) | 7 (38.9) | 3 (14.3) | 0.141 |
| Clopidogrel, n (%) | 25 (64.1) | 12 (66.7) | 13 (61.9) | 0.757 |
| Aspirin, n (%) | 14 (35.9) | 6 (33.3) | 8 (38.1) | 0.757 |

NOTE. Values are mean (standard deviation), median (interquartile range), or absolute number (percentage). The Student's t test or the Mann-Whitney U test were used to test for the association of continuous variables and SIRS for normally and non-normally distributed data, respectively. To test for the association of categorical variables and SIRS the Pearson chi-squared test was used, while the Fisher's exact test was used when the expected cell count was less than 5.
Abbreviations: BMI, body mass index; COPD, chronic obstructive pulmonary disease; LVEF, left ventricular ejection fraction; SIRS, systemic inflammatory response syndrome; STS, Society of Thoracic Surgeons.

aortic valve, interfere with normal cardiac function and may lead to tissue hypoperfusion, intestinal ischemia, and systemic inflammation. The aim of this exploratory study was to investigate whether SIRS after TAVI is associated with increased plasma concentrations of biochemical markers of inflammation and to assess the potential relation between RVP, intestinal ischemia, and postprocedural systemic inflammation.

This study was approved by the Medical Research Ethics Committees United (Research and Development Department, trial number NL45668.100.13, R13.030) and has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Written informed consent was obtained for this study in all patients.

The patients included in this study also took part in the randomized controlled Antiplatelet Therapy for Patients Undergoing Transcatheter Aortic Valve Implantation (POPular-TAVI) trial (ClinicalTrials.gov Identifier NCT02247128).[4]

SIRS was defined as the development of 2 or more of the following symptoms during the first 48 hours after TAVI and that lasted for 1 hour or more: leukocyte count $>12$ or $<4$ ($10^9$/L), respiratory rate $>20$ breaths/minute or $PaCO_2$ $<32$ mmHg, temperature $<36.0$ or $>38.0^{\circ}$C, heart rate $>90$ beats/minute.[5] The SIRS criteria respiratory rate and $PaCO_2$ were not assessed if a patient was mechanically ventilated. Also, the temperature criterion could only be satisfied after rewarming to normothermia.

Blood samples were drawn on the day of TAVI immediately after induction of general anesthesia and after 1, 3, 6, 24, and

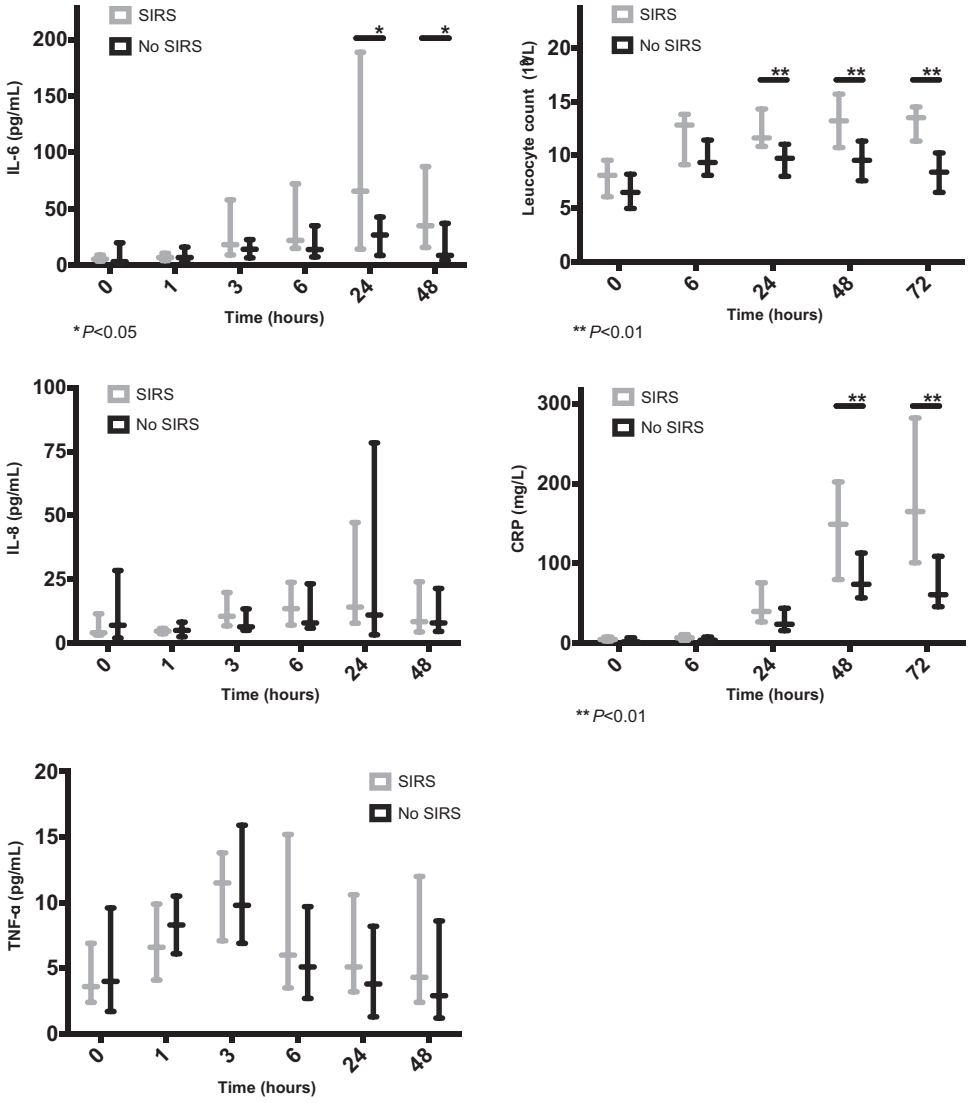


Fig 1. Plasma concentrations of biochemical markers of inflammation according to the occurrence of systemic inflammatory response syndrome (values are median and interquartile range).

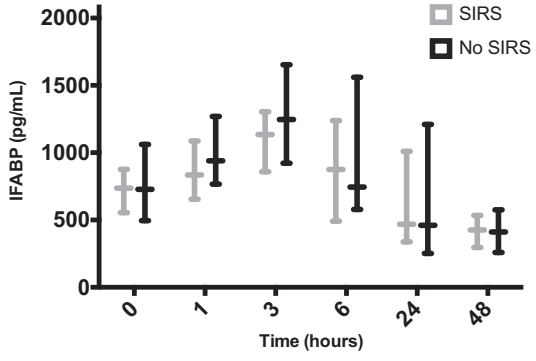


Fig 2. Plasma concentration of intestinal fatty acid binding protein according to the occurrence of systemic inflammatory response syndrome (values are median and interquartile range).

48 hours to measure plasma concentrations of interleukin (IL)-6, IL-8, tumor necrosis factor (TNF)-α, and intestinal fatty acid binding protein. Plasma concentration of C-reactive protein (CRP) and leukocyte count were determined on the day of TAVI after induction of anesthesia and after 6, 24, 48, and 72 hours.

The chi-square test or the Fisher's exact test were used to compare dichotomous variables between patients with and without SIRS. The Student's t test and the Mann-Whitney U test were used to compare independent continuous normally and non-normally distributed data between groups. A p value of $< 0.05$ was considered significant. For statistical analysis, IBM SPSS version 22 was used.

The study population consisted of 39 patients. Eighteen patients (46%) developed SIRS (Table 1). Plasma concentrations

Table 2
Periprocedural Characteristics

| Variable | All (n = 39) | SIRS (n = 18) | No SIRS (n = 21) | p Value |
|---|---|---|---|---|
| Approach | | | | |
| Transfemoral, n (%) | 29 (74.4) | 11 (61.1) | 18 (62.1) | 0.141 |
| Apical, n (%) | 10 (25.6) | 7 (38.9) | 3 (14.3) | |
| Procedural characteristics | | | | |
| Rapid pacing used, n (%) | 25 (64.1) | 11 (61.1) | 14 (66.7) | 0.718 |
| Rapid pacing runs, n | 1 (0-2) | 1 (0-2) | 1 (0-3) | 0.791 |
| Total pacing duration, sec | 13 (0-37) | 18 (0-40) | 13 (0-34) | 0.813 |
| Pre-dilatation, n (%) | 20 (51.3) | 8 (44.4) | 12 (57.1) | 0.429 |
| Post-dilatation, n (%) | 9 (23.1) | 6 (33.3) | 3 (14.3) | 0.255 |
| Intervention time, min | 106 ± 50 | 97 ± 24 | 114 ± 64 | 0.312 |
| PRBC transfusion, n (%) | 3 (7.7) | 2 (11.1) | 1 (4.8) | 0.586 |
| Number of PRBC, n | 0 (0-0) | 0 (0-0) | 0 (0-0) | 0.728 |
| Contrast, mL | 79 (60-126) | 80 (57-120) | 70 (60-138) | 0.835 |
| Vasopressor use, n (%) | 10 (25.6) | 6 (33.3) | 4 (19.0) | 0.465 |
| Inotropic use, n (%) | 10 (25.6) | 3 (16.7) | 7 (33.3) | 0.290 |
| Postprocedural hemoglobin | 6.9 ± 1.1 | 6.9 ± 1.1 | 6.9 ± 1.1 | 0.918 |
| Prosthetic valve type | | | | |
| Self-expandable, n (%) | 21 (53.8) | 12 (66.7) | 9 (42.9) | 0.201 |
| Non-self-expandable, n (%) | 18 (46.2) | 6 (33.3) | 12 (57.1) | |
| Outcome | | | | |
| AKI, n (%) | 5 (12.8) | 4 (22.2) | 1 (4.8) | 0.162 |
| Length of hospital stay, d | 6 (5-9) | 8 (6-12) | 6 (5-9) | 0.202 |
| 30-day mortality, n (%) | 4 (10.3) | 4 (22.2) | 0 | 0.037 |

NOTE. Values are mean (standard deviation), median (interquartile range), or absolute number (percentage). The Student's t test or the Mann-Whitney U test were used to test for the association of continuous variables and SIRS for normally and non-normally distributed data, respectively. To test for the association of categorical variables and SIRS the Pearson chi-squared test was used, while the Fisher's exact test was used when the expected cell count was less than 5. PRBC indicates packed red blood cell (during the first 48 hours after TAVI).
Acute kidney injury (AKI) was defined as an increase of $>26$ μmol or an increase in serum creatinine of 50% or more within 72 hours after TAVI.
Abbreviations: AKI, acute kidney injury; PRBC, packed red blood cells; SIRS, systemic inflammatory response syndrome.

of IL-6 and CRP and leucocyte count were increased in patients with SIRS on day 1, 2, and 3 (Fig 1). The intestinal fatty acid binding protein plasma concentrations in patients with and without SIRS are shown in Fig 2.

RVP was used in 25 (64%) patients. The number of rapid ventricular pacing, the number of rapid pacing runs, and the total pacing duration was similar in patients with and without SIRS (Table 2). Leukocyte count and plasma concentrations of IL-6, IL-8, and CRP in patients with and without RVP are shown in Figure 3.

This study confirms that the incidence of SIRS after TAVI is high and that SIRS is characterized by increased plasma concentrations of biochemical markers of inflammation.

We have found no evidence that RVP or intestinal ischemia is the catalyst for systemic inflammation. Although RVP causes tissue hypoperfusion, as was confirmed by the assessment of sublingual microvascular tissue perfusion in 42 patients undergoing TAVI, the duration of RVP (the average duration was 10 to 20 seconds in our study population) is probably not long enough to cause harm.[6]

Other causes of SIRS after TAVI remain to be elucidated. It is possible that systemic inflammation after TAVI is not a consequence of procedure-related characteristics but explained by genetic variability, postprocedural hypotension due to myocardial stunning, or oxidative stress and reactive oxygen species caused by supplemental oxygen therapy and subsequent hyperoxia.[7]

The results of this study are clearly exploratory due to its small sample size. In addition, assessment of tissue perfusion (using transcranial Doppler ultrasonography or Sidestream-Darkfield imaging) and monitoring cardiac output could have given us more insight in the exact role of RVP in causing tissue hypoperfusion and systemic inflammation.

In summary, SIRS after TAVI is characterized by increased plasma concentrations of biochemical markers of inflammation. We did not observe a relation among RVP, intestinal ischemia, and systemic inflammation. Other causes of systemic inflammation after TAVI deserve further study.

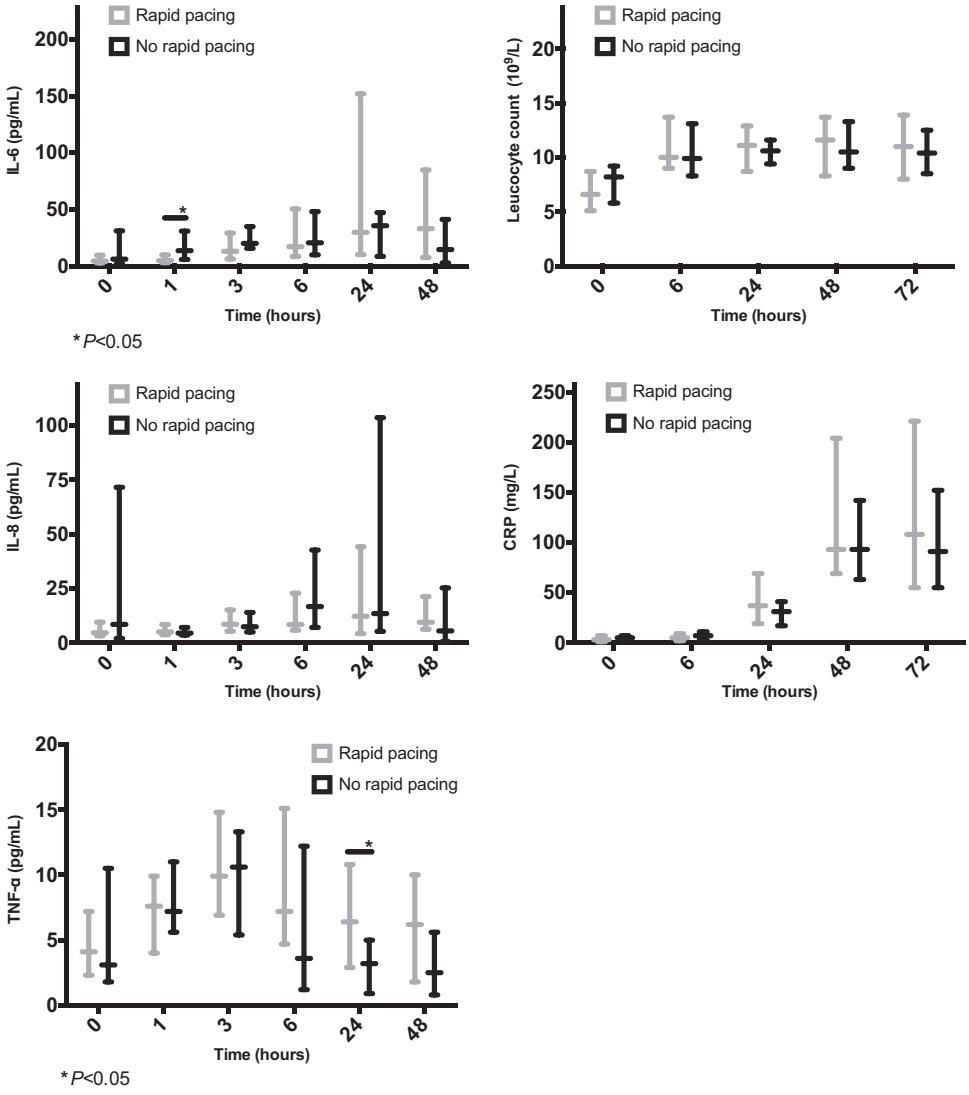


Fig 3. Plasma concentrations of biochemical markers of inflammation according to rapid ventricular pacing (values are median and interquartile range).

## Acknowledgments

We would like to thank Jedidja Baaij, Rozanne Tieman, Ben de Jong and Amber de Kroes for the collection of blood samples and performing assays.

## References

1 Sinning JM, Scheer AC, Adenauer V, et al. Systemic inflammatory response syndrome predicts increased mortality in patients after transcatheter aortic valve implantation. Eur Heart J 2012;33:1459–68.
2 Rettig TC, Rigter S, Nijenhuis VJ, et al. The systemic inflammatory response syndrome predicts short-term outcome after transapical transcatheter aortic valve implantation. J Cardiothorac Vasc Anesth 2015;29:283–7.
3 Lindman BR, Goldstein JS, Nassif ME, et al. Systemic inflammatory response syndrome after transcatheter or surgical aortic valve replacement. Heart 2015;101:537–45.
4 Nijenhuis VJ, Bennaghmouch N, Hassell M, et al. Rationale and design of POPular-TAVI: antiPlatelet therapy fOr Patients undergoing Transcatheter Aortic Valve Implantation. Am Heart J 2016;173:77–85.
5 Bone RC, Balk RA, Cerra FB, et al. ACCP/SCCM Consensus Conference Committee: Definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis. The ACCP/SCCM Consensus Conference Committee. American College of Chest Physicians/Society of Critical Care Medicine. 1992. Chest 2009;136:e28.
6 Selle A, Figulla HR, Ferrari M, et al. Impact of rapid ventricular pacing during TAVI on microvascular tissue perfusion. Clin Res Cardiol 2014;103:902–11.
7 Schwartz DA. The genetics of innate immunity. Chest 2002;121:62S–8S.

Thijs C.D. Rettig, MD, PhD*
Vincent J. Nijenhuis, MD†
Bob Meek MD‡
Sander Rigter MD*
Jurrien M. ten Berg, MD, PhD†
Bart Vlaminckx, MD, PhD‡
Wilton A. van Klei, MD, PhD§
Ewoudt M.W. van de Garde, PharmD||

Linda M. Peelen, PhD[§,#]
Peter G. Noordzij, MD, PhD[*]
[*]*Department of Anesthesiology*
*Intensive Care and Pain Medicine*
*St. Antonius Hospital*
*Nieuwegein, The Netherlands*
[†]*Department of Cardiology*
*St. Antonius Hospital*
*Nieuwegein, The Netherlands*
[‡]*Department of Medical Microbiology and Immunology*
*St. Antonius Hospital*
*Nieuwegein, The Netherlands*
[§]*Department of Anesthesiology*
*Intensive Care and Emergency Medicine*
*University Medical Center Utrecht*
*Utrecht, The Netherlands*
[‖]*Department of Clinical Pharmacy*
*St. Antonius Hospital*
*Nieuwegein, The Netherlands*
[#]*Department of Epidemiology*
*Julius Center for Health Sciences and Primary Care*
*University Medical Center Utrecht*
*Utrecht, The Netherlands*

https://doi.org/10.1053/j.jvca.2017.12.051

## Retained Paravertebral Catheter Fragment during Removal. A Rare Event without Damage for the Patient


*To the Editor:*

Epidural analgesia, which was considered the gold standard for analgesia after thoracic surgery, has been replaced in many institutions by paravertebral catheter blockade with infusion of local anesthetics into an extrapleural pocket associated with multimodal treatment.[1,2] Complications such as breakage of the catheter have been described with the epidural technique but are very rare in the case of paravertebral blocks.[3–6]

A 62-year-old female patient underwent a right lower lobectomy through a videothoracoscopic posterior approach with a conversion to a posterolateral muscle–sparing thoracotomy with rib spreading as a result of technical difficulties. At the end of the surgery, the operating surgeon inserted a paravertebral catheter under direct vision. According to our technique, the paravertebral catheter is inserted 1 space below the thoracotomy incision posteriorly by direct skin puncture without tunnelization under the skin, and the position of the needle into the extrapleural space is verified by direct vision. In case of videothoracoscopic approach the level of the most inferior incision is chosen and the catheter is advanced cranially to cover as many intercostal spaces as possible. The injection of 10 mL of saline creates the extrapleural pocket, and the catheter is inserted gently through the needle into the intercostal space until the tip reaches the paravertebral space. The catheter is then fixed to the skin by a dedicated adhesive tape and is connected to an elastomeric system for the continuous infusion of local anesthetics (ropivacaine 2 mg/mL plain solution).

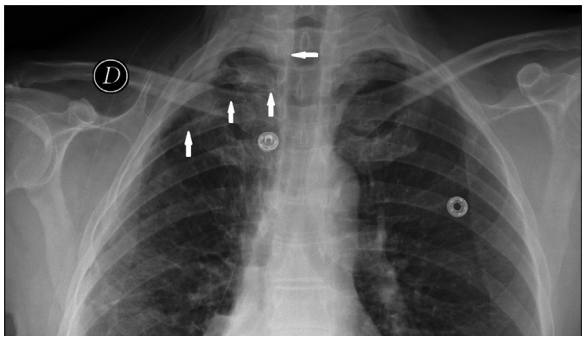


Fig 1. Chest radiography showing the position of the paravertebral catheter (*arrows*) before attempting its removal.

After an uneventful postoperative course it was decided to remove the catheter on the fourth postoperative day; however, removal was impossible because resistance was felt when pulling the catheter. A chest x-ray showing the position of the paravertebral catheter before attempting its removal is shown on Figure 1. A computed tomography scan was performed, showing the penetration site of the catheter into the extrapleural space at the level of the third intercostal space. The catheter was into the extrapleural space and its tip was located behind the right carotid artery (Fig 2). The catheter was patent; however, injection of saline could not release the resistance. During manipulations, the catheter finally was retrieved, but a small, 1-cm fragment was left in situ. A chest x-ray illustrating the retained fragment is shown on Figure 3. The patient was asymptomatic throughout this procedure. Surgery for the retrieval of the retained fragment was decided against, and the patient was discharged.

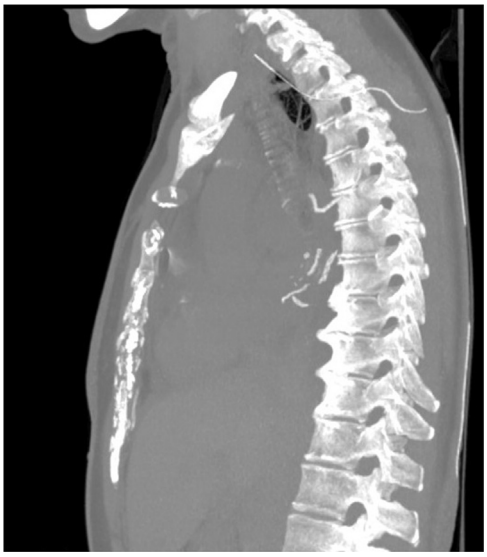

Fig 2. Computed tomography scan images showing the point of penetration of the catheter into the extrapleural space at the level of the third intercostal space and the high position of its tip.

V.J.N. and B.M. contributed equally to this study.