It is noteworthy that despite outward motion in 1 direction, the space occupied within the pericardium would still be reduced by the stroke volume (outer left ventricular volume = cavity volume + incompressible myocardial volume).

Carlsson et al. (2) extracted the longitudinal component to stroke volume by using a cylinder approach: a constant outer epicardial area multiplied by the longitudinal decent. Effectively, this approach incorporates circumferential shortening of every layer inside the epicardium, as well as wall thickening caused by both the longitudinal and circumferential shortening. We therefore argue that this theoretical volume exaggerates the pure longitudinal contribution. To perform what we believe is a fair investigation of their individual contribution to EF, circumferential shortening was fixed to a prescribed value, and the longitudinal shortening was varied, and vice versa. This perspective shows that circumferential shortening contributes more to EF, which is consistent with the following: 1) numerous studies showing loss of longitudinal shortening and still preserved EF; 2) circumferential shortening's quadratic impact on EF as opposed to only linear for longitudinal shortening; and 3) the good agreement of our model with measurements.

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Arterial Thromboembolism in Non-Hodgkin Lymphoma

In a recent paper, Navi et al. (1) showed that patients with cancer have an increased risk of myocardial infarction and ischemic stroke in the months after the cancer diagnosis. The study included almost 280,000 pairs of cancer (8 types, including non-Hodgkin lymphoma [NHL]) and matched noncancer patients. Due to the size and the methodologic rigor, this study settles a question that, as the authors point out, had received little attention.

NHL is a heterogeneous group, ranging from aggressive lymphomas that can threaten the patient's life in weeks to indolent lymphomas that require no treatment for years. In line with solid tumors, where the risk of venous thromboembolism is generally greater in clinically more aggressive cancers (2), the risk of venous thromboembolism is higher in high-grade lymphomas (3,4). Indeed, the effect of NHL subtype might be greater than that of tumor stage, also well-documented (4), and which Navi et al. (1) found to be relevant for the risk of arterial thromboembolism. Importantly, however, stage and NHL histology are not unrelated. Aggressive lymphomas are often symptomatic, leading the patient to seek medical help earlier, whereas indolent lymphomas are more often diagnosed in asymptomatic patients, already in stage IV. Therefore, the risk of arterial thromboembolism in aggressive NHL could be underestimated when not taking the histological subtype into account. Because the clinical approach to patients with aggressive and indolent lymphomas

TABLE 1 Classification of Aggressive Versus Indolent Non-Hodgkin Lymphoma According to the International Classification of Diseases for Oncology, 3rd Edition Histology Classification

Aggressive	Indolent
9590*,9591*,9596* 9597,9673,9675,9678-9680,9684,9687,9688,9700-9702,9705,9708,9709,9712,9714-9719,9724-9729,9735,9737,9738,9811-9818,9827,9837	9670,9671,9689,9690, 9691,9695,9698, 9699,9823

*Ideally, a sensitivity analysis would be performed excluding 9590, 9591, and 9596, given that those categories are unspecific, even though they are probably most likely to be used for aggressive non-Hodgkin lymphomas.

is very different, we kindly ask Navi et al. (1) to carry out this subgroup analysis if possible (**Table 1** shows a possible option to group patients according to the International Classification of Diseases for Oncology, 3rd edition, classification), because the knowledge gained would be invaluable and their study offers a truly unique opportunity to do so.

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Recurrent Late Bioresorbable Scaffold Thrombosis as a Presenting Symptom of Underlying Cancer

In a recent issue of the *Journal*, Navi et al. (1) demonstrated an increase in short-term risks for arterial thromboembolisms in patients with cancer. We would like to argue that this finding implies that arterial thromboembolisms can be the presenting symptom of an indolent cancer.

Herewith, we present the case of a 50-year-old man with stable coronary artery disease, who experienced 3 angiographically confirmed thromboses of bioresorbable scaffolds (2.5- to 3.5-mm diameter overlapping Absorb, Abbott Vascular, Abbott Park, Illinois) in his left anterior descending artery at 128, 129, and 242 days after implantation. Scaffold thromboses occurred despite treatment with a combination of aspirin, ticagrelor, and either coumarin (with an international normalized ratio in the therapeutic range at time of thrombosis) or therapeutic dose low-molecular-weight heparin. During this period of recurrent scaffold thromboses, the patient also developed multiple unprovoked deep vein thromboses, a pulmonary embolism, and a thrombus in the apex of the left ventricle. No abnormalities could be identified by extensive hematologic analysis, including lupus anticoagulant, anticardiolipin antibodies, antinuclear antibodies, β2-glycoprotein-I antibodies, protein S activity, protein C activity, antithrombin III activity, JAK2 mutations, factor II mutation, and factor V Leiden mutation. Further imaging, including abdominal computed tomography scans, raised the suspicion of a widespread metastasized cancer, the presence of which was confirmed by biopsy of a bone lesion and an esophageal tumor. Due to his deteriorating clinical condition, the patient was offered best supportive care. He died of a hemorrhagic stroke 1 month after the diagnosis of metastasized esophageal cancer was made.

Venous thromboembolism was identified as a common complication of cancer by Trousseau in 1865. Since then, the cancer-induced prothrombotic state leading to venous thromboembolism has been recognized as a well-known presenting symptom of underlying cancer (2). A similar mechanism may explain the observed increased risk of arterial thromboembolisms in patients with cancer (1). Because Absorb bioresorbable scaffolds provide a clear substrate for coronary thrombosis (3), we would like to create awareness among cardiovascular specialists for the occurrence and especially recurrence of late scaffold thrombosis as a potential presenting symptom of an underlying undiagnosed cancer.

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