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Echocardiographic insights into cardioembolic stroke: identifying risk factors and sources

Rola echokardiografii w diagnostyce kardiogennej przyczyny udaru niedokrwiennego mózgu

Short title: Echocardiography in Embolic Stroke

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Abstract

Stroke is one of the leading global causes of death and long-term disability. Approximately 15–40% of ischaemic strokes have a cardioembolic origin. These are associated with worse prognosis, higher propensity for early and late recurrences, and increased mortality compared to other types of ischaemic strokes. The echocardiographic examination is a crucial diagnostic tool for determining both the aetiology of the stroke and appropriate secondary prevention. Early implementation of causative treatment reduces the risk of disability and decreases long-term patient care costs. This article focuses on discussing high-risk heart diseases associated with cardioembolic sources, such as atrial flutter and fibrillation, coronary artery disease, cardiomyopathies, the presence of artificial heart valves, infectious endocarditis, intracardiac masses, and aortic atherosclerosis. Transthoracic and transoesophageal echocardiography play a significant role in diagnosing these conditions. The aim of this review is to discuss optimal diagnostic methods for potential sources of cardioembolism and to explore new potential predictive factors for stroke occurrence in echocardiographic studies.

Keywords: ischaemic stroke, echocardiography, cardioembolic stroke, secondary prevention

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Introduction

Cerebral strokes, ranking second after ischaemic heart disease, are among the leading causes of death worldwide and the most common cause of long-term disability [1]. Each year, approximately 17 million people experience a stroke globally, with an estimated 73,900 cases of ischaemic stroke in Poland. Ischaemic strokes (IS) account for about 80% of all strokes.

Various mechanisms can lead to a cerebral stroke [2]. To determine the probable aetiology of IS, the TOAST classification (Trial of Org 10172 in Acute Stroke Treatment) is commonly used, which distinguishes five subtypes of strokes:

- stroke due to large artery atherosclerosis,
- stroke due to small artery occlusion,
- cardioembolic stroke,
- stroke of other determined aetiology,
- stroke of undetermined aetiology [3, 4].

Approximately 15–40% of IS have a cardioembolic origin, known as cardioembolic infarctions (CEI) [5]. These strokes typically result from the migration of embolic material including blood clots, tumour fragments, or vegetation [1].

Cardioembolic strokes have a worse prognosis and a higher risk of early and late recurrence compared to other stroke types [6]. Early recurrences are typically defined as occurring within 90 days after the last stroke episode [7].

Patients with cardioembolic strokes require statistically longer hospitalization and are more likely to suffer from permanent disabilities, resulting in increased healthcare costs. The cost of acute care for cardioembolic stroke patients may be approximately 40% higher than that for patients with non-cardioembolic stroke [8].

Early recognition of the cardioembolic aetiology and implementing secondary prevention measures are crucial to minimize the risk of recurrence. Establishing the potential source of embolism and ruling out possible causes of the stroke are essential in diagnosing cardioembolic stroke [5, 9]. Echocardiographic evaluation plays a significant role in diagnosing potential embolic sources, enabling early initiation of preventive measures against recurrent cardioembolic events.

Use of echocardiography

To assess the structural and functional aspects of the heart in the acute phase of an ischaemic stroke (IS), echocardiographic examinations are recommended, including both transthoracic echocardiography (TTE) and in specific cases transoesophageal echocardiography (TEE). TTE is

usually the first-line imaging method. However, in cases where there is suspicion of cardioembolic stroke with an undetermined source or when TTE imaging is inconclusive due to challenging conditions, TEE is employed. It is considered a semi-invasive procedure, typically well-tolerated with local anaesthesia of the throat and oesophagus or, alternatively, performed under sedation [10]. TEE is regarded as the gold standard for imaging the interatrial septum and assessing the left atrial appendage (LAA) for potential embolic material. TEE allows for better visualization of intracardiac structures, valvular apparatus, and the thoracic aorta compared to TTE. It is particularly useful in patients suspected of having infective endocarditis or dysfunction of prosthetic heart valves [11, 12]. In daily practice, TTE and TEE should be considered as complementary methods.

The recommendations for performing echocardiographic examinations in patients with IS are somewhat ambiguous. The 2016 guidelines from the American Society of Echocardiography suggest routine TTE as a screening method for potential cardiac sources of embolism in all stroke patients, with TEE reserved for selected cases, such as suspected endocarditis [13].

During the European Stroke Organization Karolinska Stroke Update Conference in 2018, TTE was recognized as the first-choice imaging method for heart assessment in patients with acute ischaemic stroke (AIS), with TEE recommended only for patients with embolic strokes of undetermined source to further evaluate PFO and aortic atherosclerosis [14].

According to the 2021 Guideline for the Prevention of Stroke in Patients with Stroke and Transient Ischaemic Attack from the American Heart Association and American Stroke Association, TTE is the preferred method for searching for embolic material in the left heart chamber, while TEE is recommended to detect left atrial (LA) thrombus, aortic atheroma, prosthetic valve abnormalities, native valve abnormalities, atrial septal abnormalities, and cardiac tumours [15].

Potential sources of embolism detected by echocardiography

Cerebral cardiogenic stroke is characterized by significant heterogeneity. Heart diseases detected during echocardiographic examination can be classified into high-risk and low-risk factors (Table 1) [15, 16].

Atrial fibrillation

Unrecognized atrial fibrillation (AF) is the main cause of cardioembolic strokes. The incidence of this arrhythmia increases with age and reaches 5–15% in patients over 80 years old [18]. In the NOMED-AF study conducted on the Polish population, the prevalence of AF over 65 years old was found to be as high as 19.2% [19]. Even a short, five-minute episode of AF (which can be completely asymptomatic) is estimated to increase the risk of stroke by 2.8 times [20]. In a study conducted by Nguyen et al. [19], the prevalence of LAA thrombus in TEE in patients after an

ischaemic stroke with sinus rhythm was 6.7%, with the most prevalent aetiology being paroxysmal AF identified later on in Holter ECG monitoring.

AF is associated with LA enlargement and impaired LA contractility, which leads to blood flow stasis and facilitates the formation of thrombus (most commonly in the LAA which can cause embolism). TEE is the gold standard for detecting thrombus located in the LAA. It also can evaluate LA dysfunction, such as the presence of spontaneous contrast or reduced LAA emptying velocity, which may accompany or precede thrombi formation [14].

Mitral valve stenosis

Progressive mitral valve stenosis contributes to changes in atrial architecture, leading to increased pressure in the left atrium, its enlargement, and increased wall tension, which can result in the development of AF and the formation of thrombi.

The most common type of mitral stenosis is degenerative mitral stenosis (DMS) primarily caused by mitral annular calcification (MAC) [21, 22] (Figure 1). Excessive tension on the mitral annulus necessitates higher pressure for valve closure, leading to microtraumas in the valve. Sites of damage are associated with an increased risk of degenerative calcifications [23, 24]. Numerous studies have shown that MAC is an independent risk factor associated with a higher risk of stroke [23].

In patients with rheumatic mitral valve stenosis and sinus rhythm, the risk of stroke is 8%, but in the presence of AF, increases it to 31.5%. Similarly, for patients with rheumatic heart disease and mitral regurgitation, the risk of stroke is 7.7% with sinus rhythm and 22% with AF [11, 25].

Echocardiography is the primary diagnostic test for suspected mitral stenosis; the quantitative and qualitative assessment of the defect is necessary for surgical qualification and in the choice of appropriate treatment valve repair or replacement.

Intracardiac thrombi associated with local or global myocardial dysfunction

In the context of global or segmental left ventricular (LV) dysfunction, blood flow deceleration may lead to intraventricular thrombus formation. Furthermore, congestive heart failure, reduced left ventricular ejection fraction, and a history of myocardial infarction (MI) not only increase the risk of atrial fibrillation (AF) but also increase the risk of thromboembolism according to the CHA₂DS₂-VASc score. The most common cause of impaired contractility is undoubtedly coronary heart disease, especially MI. Other causes include dilated cardiomyopathies of various aetiologies (genetic, inflammatory, toxic) (Figure 2).

In cases of MI, blood flow deceleration is related to myocardial damage and the occurrence of segmental myocardial dysfunction, which can be assessed using echocardiography. In acute coronary syndrome, the incidence of LV thrombi is approximately 5–15% despite appropriate

treatment [11]. LV thrombi are more common after extensive MI, particularly those involving the anterior wall of the heart, including the apex. The development of a post-infarction aneurysm represents the highest risk for thrombus formation. Mobile thrombi are associated with a higher risk of stroke compared to mural thrombi [11].

It is estimated that approximately 2.5% of patients experience an ischaemic stroke within a month after MI, and approximately 10% will experience it within the following 6 years [6, 26]. In dilated cardiomyopathy (DCM), blood stasis also occurs, leading to thrombus formation in 1.7–18% of patients [27]. However, according to Moulson et al. [28] the routine prophylactic use of warfarin in addition to dual antiplatelet therapy following an anterior STEMI with a decrease in LVEF and wall motion abnormalities, appears to result in no mortality benefit or reduction in stroke rates but may increase the frequency of major bleeding.

Transthoracic echocardiography allows the detection of LV thrombi with a sensitivity of 95% and a specificity of 85–90% [11]. In cases where visualization of the LV apex is limited on TTE, the use of contrast-enhanced echocardiography is recommended for a more accurate assessment [11]. In case of further diagnostic uncertainty, cardiac magnetic resonance is perceived as the gold standard.

Infective endocarditis

Infective endocarditis (IE) is associated with bacteraemia, which leads to endothelial damage of the endocardium and the formation of bacterial vegetations, most commonly located on heart valves (Figure 3). Both the increased coagulability accompanying the inflammation and the detachment of fragments from the bacterial vegetations can lead to embolic complications.

The risk of IS is highest before antibiotic therapy and during the first two weeks of treatment, with an incidence of approximately 15–20% in patients with IE [6].

According to the 2023 ESC guidelines for the treatment of infective endocarditis after ischaemic stroke, immediate surgical intervention is recommended in the presence of indications such as heart failure, uncontrolled infection, abscess, or persistent high risk of embolism if the patient is not in a coma and intracranial bleeding has been excluded [29]. Urgent surgery is also indicated in patients with vegetations > 10 mm on the mitral or aortic valve, who have experienced at least one embolic event despite appropriate antibiotic therapy [30].

Echocardiography is the method of choice for the diagnosis and monitoring of IE [31]. When suspecting IE, TTE should be performed first, and if the presence of vegetation is not confirmed, especially under suboptimal imaging conditions, TEE is indicated. The sensitivity of detecting vegetation on native valves and prosthetic valves is approximately 70% and 50% for TTE, respectively, and 96% and 92% for TEE, respectively [32].

Marantic endocarditis

Marantic endocarditis is a rare form of non-infective endocarditis manifested by thromboembolic disease [6]. It is distinguished by the presence of non-infectious vegetations on heart valves, most commonly involving the mitral and aortic valves, composed of platelet and fibrin deposits [33]. It is often associated with advanced malignancies, such as pancreatic, lung, and ovarian cancer, as well as debilitating conditions like autoimmune diseases. Particularly, patients with pancreatic cancer are at an increased risk of stroke and should be closely monitored, especially within the first 6 months of diagnosis [34].

Mechanical heart valve thrombosis

The risk of thromboembolism in patients with mechanical valves is associated with infective endocarditis or thrombosis of the prosthetic valve.

In patients with mechanical valves, the formation of thrombi within the valves should be suspected in every patient with stroke. The risk of ischaemic stroke in patients with mechanical heart valve receiving anticoagulation therapy is about 2-4% per year, whereas, without such treatment, the risk is significantly higher (12–22%) so oral vitamin K antagonists (VKA) for lifelong anticoagulation therapy in this group is recommended [6, 35].

In cases of suspected valve thrombosis, both TTE and TEE should be performed to assess valve morphology, the peri-valvular region, and the flow across the valve, as well as to estimate the transvalvular gradient. The detection of a large thrombus on the prosthetic valve may be an indication for surgical treatment [36].

Intracardiac Tumours

Primary intracardiac tumours are very rare — based on autopsy studies, their estimated frequency is about 0.02%. Neurological complications of cardiac tumours, usually embolic stroke or transient ischaemic attack (TIA) occur in about 12–45% of patients and are mostly associated with the presence of LA myxoma. Even in 80% of patients, neurological complications may represent the initial symptom of a cardiac tumour [37].

Myxomas are considered fragile tumours that tend to fragment easily. Embolic stroke is a complication of myxoma in about 16.8% of patients. The primary method for detecting myxoma is echocardiography, and the recommended approach is surgical treatment [38].

Atherosclerosis of the Aorta

The other possible cause of ischaemic stroke described in the literature is atherosclerosis of the descending aorta. The primary tool for assessing potential atherosclerotic plaques in the aorta is

TEE. TTE can be used as a complementary method, helpful in visualizing the distal part of the ascending aorta and the proximal part of the aortic arch.

Detected atherosclerotic plaques can be divided into two groups: simple plaques - with a thickness of the inner aortic wall < 4 mm, and complex plaques, which are larger, calcified with ulceration and/or mobile elements. It is estimated that atherosclerosis of the aorta increases the risk of stroke up to four times [11].

Persistent Foramen Ovale (PFO) and Atrial Septal Aneurysms (ASA)

The occurrence of PFO is estimated at approximately 20–25% of the adult population based on autopsy studies [11]. In the absence of accompanying symptoms, it is not considered a pathology.

In individuals under 55 years with cryptogenic stroke, PFO is found significantly more frequently than in the general population — up to 61% of patients [6].

PFO is often associated with ASA (approximately 60–70% of ASA cases have PFO), which are diagnosed when the septum is displaced or deviated towards the right or left atrium by at least 10 mm from the midline. The coexistence of PFO and ASA significantly increases the risk of stroke [6]. There are a few case reports of acute stroke preceded by documented passage of a thrombus through a PFO confirming the mechanism of paradoxical embolism.

In cases of suspected PFO, performing a TTE with contrast is recommended, and in negative or inconclusive results, TEE is advised. The RoPE scale (Risk of Paradoxical Embolism score) is used for clinical assessment of the likelihood of PFO being the cause of IS. Percutaneous PFO closure is indicated when the RoPE score is ≥ 7 points [39].

Echocardiography in predicting ischaemic stroke

New predictive factors for IE, especially in the context of paroxysmal AF, are still being sought. Recently, there has been an increasing number of reports on the use of left atrial enlargement (LAE) in stratifying the occurrence of ischaemic stroke. Previous studies have shown that atrial enlargement ($> 34 \text{ mL/m}^2$) increases the risk of cardioembolic stroke. Several recent studies have shown that LAE may be associated with recurrent cardioembolic and cryptogenic strokes and be a significant predictor of recurrent events in patients with cardioembolic stroke [40].

The exact mechanism responsible for the link between LAE and stroke recurrence is not fully understood, but it is suspected that atrial enlargement increases the risk of thrombus formation within LAA [41].

Another parameter related to IS is LA strain. Strain is a quantitative parameter using acoustic marker tracking techniques that describes atrial function in different cycles of its work. LA strain analysis can predict adverse AF events with greater sensitivity than LA volume [42]. It has also been observed that LA strain is a better predictor than the volume of adverse clinical events in individuals with AF [43].

Conclusions

TTE and TEE are integral parts of the diagnostic process in patients after a stroke. TTE is a non-invasive examination that can be performed at the patient's bedside, allowing for its application in a wide range of patients. It enables the assessment of heart morphology and function and often helps to detect the direct source of embolism. In cases where the TTE image is inconclusive, the evaluation may be expanded with TEE. Since TEE is a semi-invasive procedure that requires local anaesthesia, it is usually not the first-line examination but rather a complement to TTE in selected cases.

The echocardiographic examination helps determine the aetiology of stroke and in guiding appropriate measures for secondary prevention. Due to the high recurrence rate of strokes, implementing adequate secondary prophylaxis reduces the risk of subsequent incidents, thereby lowering the costs of long-term patient care.

Additional information

Author contributions

AW — concept/design, drafting article, critical revision of article; AN — drafting article, editing; MBO — critical revision of the article, preparation of figures and tables; WK — critical revision of the article; WB — critical revision of the article, approval of the article.

Conflict of interests

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Streszczenie

Udary mózgu są jedną z głównych przyczyn śmierci na całym świecie, a także jedną z wiodących przyczyn długotrwałej niesprawności. Około 15-40% udarów niedokrwiennych ma podłoże sercowo-zatorowe. Wiążą się one z gorszym rokowaniem, większą skłonnością do nawrotów wczesnych i późnych oraz większą śmiertelnością niż inne typy udarów niedokrwiennych. Badanie echokardiograficzne jest ważnym elementem diagnostycznym zarówno w ustaleniu etiologii udaru jak i właściwego postępowania w jego prewencji wtórnej. Wdrożenie leczenia przyczynowego na wczesnym etapie pozwala zmniejszyć ryzyko niepełnosprawności i zmniejsza koszty długotrwałej opieki nad pacjentem. Ten artykuł skupia się na omówieniu chorób serca związanych z dużym ryzykiem wystąpienia zatorowości sercowopochodnej takich jak: trzepotanie i migotanie przedsionków, choroba niedokrwienna serca, kardiomiopatie, obecność sztucznych zastawek serca, infekcyjne zapalenia wsierdza, obecność mas wewnątrzsercowych oraz miażdżycy aorty. Istotną

rolę w diagnostyce tych chorób stanowi badanie echokardiograficzne przezklatkowe i przezprzełykowe. Celem tej pracy przeglądowej jest omówienie optymalnych metod diagnostycznych potencjalnych źródeł zatorowości serc pochodnej, a także omówienie nowych, potencjalnych czynników predykcyjnych wystąpienia udaru mózgu w badaniu echokardiograficznych.

Słowa kluczowe: udar niedokrwienny, echokardiografia, udar kardiogeny, prewencja wtórna

References

1. Król W, Żarek A, Wilk A, et al. Transthoracic echocardiography in the assessment of cardiogenic causes of ischaemic stroke. *Neurol Neurochir Pol.* 2019; 53(2): 156–161, doi: [10.5603/PJNNS.a2019.0016](https://doi.org/10.5603/PJNNS.a2019.0016), indexed in Pubmed: [31020990](https://pubmed.ncbi.nlm.nih.gov/31020990/).
2. Abreu TT, Mateus S, Correia J. Therapy implications of transthoracic echocardiography in acute ischemic stroke patients. *Stroke.* 2005; 36(7): 1565–1566, doi: [10.1161/01.STR.0000170636.08554.49](https://doi.org/10.1161/01.STR.0000170636.08554.49), indexed in Pubmed: [15947277](https://pubmed.ncbi.nlm.nih.gov/15947277/).
3. Camen S, Haeusler KG, Schnabel RB. Cardiac imaging after ischemic stroke or transient ischemic attack. *Curr Neurol Neurosci Rep.* 2020; 20(8): 36, doi: [10.1007/s11910-020-01053-3](https://doi.org/10.1007/s11910-020-01053-3), indexed in Pubmed: [32607785](https://pubmed.ncbi.nlm.nih.gov/32607785/).
4. Adams HP, Bendixen BH, Kappelle LJ, et al. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. *Stroke.* 1993; 24(1): 35–41, doi: [10.1161/01.str.24.1.35](https://doi.org/10.1161/01.str.24.1.35), indexed in Pubmed: [7678184](https://pubmed.ncbi.nlm.nih.gov/7678184/).
5. Gąsiorek P, Banach M, Maciejewski M, et al. Stroke as a result of cardioembolism-characteristic features in the context of diagnostic methods and secondary prevention. *Folia Cardiol.* 2018; 13(1): 21–28, doi: [10.5603/FC.2018.0004](https://doi.org/10.5603/FC.2018.0004).
6. Arnautu SF, Arnautu DA, Lascu A, et al. A review of the role of transthoracic and transesophageal echocardiography, computed tomography, and magnetic resonance imaging in cardioembolic stroke. *Med Sci Monit.* 2022; 28: e936365, doi: [10.12659/MSM.936365](https://doi.org/10.12659/MSM.936365), indexed in Pubmed: [35729858](https://pubmed.ncbi.nlm.nih.gov/35729858/).
7. Moroney JT, Bagiella E, Paik MC, et al. Risk factors for early recurrence after ischemic stroke: the role of stroke syndrome and subtype. *Stroke.* 1998; 29(10): 2118–2124, doi: [10.1161/01.str.29.10.2118](https://doi.org/10.1161/01.str.29.10.2118), indexed in Pubmed: [9756592](https://pubmed.ncbi.nlm.nih.gov/9756592/).
8. Winter Y, Wolfram C, Schaeg M, et al. Evaluation of costs and outcome in cardioembolic stroke or TIA. *J Neurol.* 2009; 256(6): 954–963, doi: [10.1007/s00415-009-5053-2](https://doi.org/10.1007/s00415-009-5053-2), indexed in Pubmed: [19252783](https://pubmed.ncbi.nlm.nih.gov/19252783/).
9. Arboix A, Oliveres M, Massons J, et al. Early differentiation of cardioembolic from atherothrombotic cerebral infarction: a multivariate analysis. *Eur J Neurol.* 1999; 6(6): 677–683, doi: [10.1046/j.1468-1331.1999.660677.x](https://doi.org/10.1046/j.1468-1331.1999.660677.x), indexed in Pubmed: [10529755](https://pubmed.ncbi.nlm.nih.gov/10529755/).
10. Lipiec P, Bąk J, Braksator W, et al. [Transesophageal echocardiography in adults - guidelines of the Working Group on Echocardiography of the Polish Cardiac Society]. *Kardiol Pol.* 2018; 76(2): 494–498, doi: [10.5603/KP.2018.0052](https://doi.org/10.5603/KP.2018.0052), indexed in Pubmed: [29457626](https://pubmed.ncbi.nlm.nih.gov/29457626/).

11. Danese A, Mugnai G, Prevedello F, et al. The role of echocardiography in the embolic stroke of undetermined source. *J Cardiovasc Med (Hagerstown)*. 2020; 21(8): 547–555, doi: [10.2459/JCM.0000000000001023](https://doi.org/10.2459/JCM.0000000000001023), indexed in Pubmed: [32628421](https://pubmed.ncbi.nlm.nih.gov/32628421/).
12. Hahn RT, Abraham T, Adams MS, et al. Guidelines for performing a comprehensive transesophageal echocardiographic examination: recommendations from the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. *J Am Soc Echocardiogr*. 2013; 26(9): 921–964, doi: [10.1016/j.echo.2013.07.009](https://doi.org/10.1016/j.echo.2013.07.009), indexed in Pubmed: [23998692](https://pubmed.ncbi.nlm.nih.gov/23998692/).
13. Saric M, Armour AC, Arnaout MS, et al. Guidelines for the use of echocardiography in the evaluation of a cardiac source of embolism. *J Am Soc Echocardiogr*. 2016; 29(1): 1–42, doi: [10.1016/j.echo.2015.09.011](https://doi.org/10.1016/j.echo.2015.09.011), indexed in Pubmed: [26765302](https://pubmed.ncbi.nlm.nih.gov/26765302/).
14. Schnabel RB, Camen S, Knebel F, et al. Expert opinion paper on cardiac imaging after ischemic stroke. *Clin Res Cardiol*. 2021; 110(7): 938–958, doi: [10.1007/s00392-021-01834-x](https://doi.org/10.1007/s00392-021-01834-x), indexed in Pubmed: [34143285](https://pubmed.ncbi.nlm.nih.gov/34143285/).
15. Kleindorfer DO, Towfighi A, Chaturvedi S, et al. 2021 guideline for the prevention of stroke in patients with stroke and transient ischemic attack: a guideline from the american heart association/american stroke association. *Stroke*. 2021; 52(7): e364–e467, doi: [10.1161/STR.0000000000000375](https://doi.org/10.1161/STR.0000000000000375), indexed in Pubmed: [34024117](https://pubmed.ncbi.nlm.nih.gov/34024117/).
16. Secades S, Martín M, Corros C, et al. Diagnostic yield of echocardiography in stroke: should we improve patient selection? *Neurologia*. 2013; 28(1): 15–18, doi: [10.1016/j.nrl.2012.03.002](https://doi.org/10.1016/j.nrl.2012.03.002), indexed in Pubmed: [22608679](https://pubmed.ncbi.nlm.nih.gov/22608679/).
17. Zhang L, Harrison JK, Goldstein LB. Echocardiography for the detection of cardiac sources of embolism in patients with stroke or transient ischemic attack. *J Stroke Cerebrovasc Dis*. 2012; 21(7): 577–582, doi: [10.1016/j.jstrokecerebrovasdis.2011.01.005](https://doi.org/10.1016/j.jstrokecerebrovasdis.2011.01.005), indexed in Pubmed: [21367623](https://pubmed.ncbi.nlm.nih.gov/21367623/).
18. Wytyczne dotyczące postępowania u chorych z migotaniem przedsionków. *Kardiolog Pol*. 2010; 68(supl. VII): 487–566.
19. Kalarus Z, Średniawa B, Mitreǵa K, et al. Prevalence of atrial fibrillation in the 65 or over Polish population. Report of cross-sectional NOMED-AF study. *Kardiolog Pol*. 2023; 81(1): 14–21, doi: [10.33963/KP.a2022.0202](https://doi.org/10.33963/KP.a2022.0202), indexed in Pubmed: [36043418](https://pubmed.ncbi.nlm.nih.gov/36043418/).
20. Miyasaka Y, Barnes ME, Gersh BJ, et al. Secular trends in incidence of atrial fibrillation in Olmsted County, Minnesota, 1980 to 2000, and implications on the projections for future prevalence. *Circulation*. 2006; 114(2): 119–125, doi: [10.1161/CIRCULATIONAHA.105.595140](https://doi.org/10.1161/CIRCULATIONAHA.105.595140), indexed in Pubmed: [16818816](https://pubmed.ncbi.nlm.nih.gov/16818816/).
21. Steiner DK, Søgaard P, Jensen M, et al. Risk of stroke or systemic embolism in patients with degenerative mitral stenosis with or without atrial fibrillation: A cohort study. *Int J Cardiol Heart Vasc*. 2022; 43, doi: [10.1016/j.ijcha.2022.101126](https://doi.org/10.1016/j.ijcha.2022.101126), indexed in Pubmed: [36237964](https://pubmed.ncbi.nlm.nih.gov/36237964/).
22. Abramowitz Y, Jilaihawi H, Chakravarty T, et al. Mitral annulus calcification. *J Am Coll Cardiol*. 2015; 66(17): 1934–1941, doi: [10.1016/j.jacc.2015.08.872](https://doi.org/10.1016/j.jacc.2015.08.872), indexed in Pubmed: [26493666](https://pubmed.ncbi.nlm.nih.gov/26493666/).
23. Sud K, Agarwal S, Parashar A, et al. Degenerative mitral stenosis: unmet need for percutaneous interventions. *Circulation*. 2016; 133(16): 1594–1604, doi: [10.1161/CIRCULATIONAHA.115.020185](https://doi.org/10.1161/CIRCULATIONAHA.115.020185), indexed in Pubmed: [27142604](https://pubmed.ncbi.nlm.nih.gov/27142604/).
24. Silbiger JJ. Anatomy, mechanics, and pathophysiology of the mitral annulus. *Am Heart J*. 2012; 164(2): 163–176, doi: [10.1016/j.ahj.2012.05.014](https://doi.org/10.1016/j.ahj.2012.05.014), indexed in Pubmed: [22877801](https://pubmed.ncbi.nlm.nih.gov/22877801/).

25. Ahmad S, Wilt H. Stroke prevention in atrial fibrillation and valvular heart disease. *Open Cardiovasc Med J*. 2016; 10: 110–116, doi: [10.2174/1874192401610010110](https://doi.org/10.2174/1874192401610010110), indexed in Pubmed: [27347228](https://pubmed.ncbi.nlm.nih.gov/27347228/).
26. Ibanez B, James S, Agewall S, et al. ESC Scientific Document Group. 2017 ESC Guidelines for the management of acute myocardial infarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST-segment elevation of the European Society of Cardiology (ESC). *Eur Heart J*. 2018; 39(2): 119–177, doi: [10.1093/eurheartj/ehx393](https://doi.org/10.1093/eurheartj/ehx393), indexed in Pubmed: [28886621](https://pubmed.ncbi.nlm.nih.gov/28886621/).
27. Kim AS. Evaluation and prevention of cardioembolic stroke. *Continuum (Minneapolis)*. 2014; 20(2 Cerebrovascular Disease): 309–322, doi: [10.1212/01.CON.0000446103.82420.2d](https://doi.org/10.1212/01.CON.0000446103.82420.2d), indexed in Pubmed: [24699483](https://pubmed.ncbi.nlm.nih.gov/24699483/).
28. Moulson N, LaHaye SA, Bertrand OF, et al. Prophylactic warfarin post anterior st-elevation myocardial infarction: a systematic review and meta-analysis. *Cardiovasc Revasc Med*. 2017; 18(8): 559–564, doi: [10.1016/j.carrev.2017.05.002](https://doi.org/10.1016/j.carrev.2017.05.002), indexed in Pubmed: [28501493](https://pubmed.ncbi.nlm.nih.gov/28501493/).
29. Habib G, Lancellotti P, Antunes MJ, et al. ESC Scientific Document Group. 2015 ESC guidelines for the management of infective endocarditis: the task force for the management of infective endocarditis of the European Society of Cardiology (ESC). Endorsed by: European Association for Cardio-Thoracic Surgery (EACTS), the European Association of Nuclear Medicine (EANM). *Eur Heart J*. 2015; 36(44): 3075–3128, doi: [10.1093/eurheartj/ehv319](https://doi.org/10.1093/eurheartj/ehv319), indexed in Pubmed: [26320109](https://pubmed.ncbi.nlm.nih.gov/26320109/).
30. Vahanian A, Alfieri O, Andreotti F, et al. Joint Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology (ESC), European Association for Cardio-Thoracic Surgery (EACTS). Guidelines on the management of valvular heart disease (version 2012). *Eur Heart J*. 2012; 33(19): 2451–2496, doi: [10.1093/eurheartj/ehs109](https://doi.org/10.1093/eurheartj/ehs109), indexed in Pubmed: [22922415](https://pubmed.ncbi.nlm.nih.gov/22922415/).
31. Binkiewicz-Orluk M, Konopka M, Sierakowska-Sitkiewicz K, et al. Multimodality imaging in a diagnostic and therapeutic process of a patient with infective endocarditis. *J Ultrason*. 2020; 20(80): e61–e66, doi: [10.15557/JoU.2020.0010](https://doi.org/10.15557/JoU.2020.0010), indexed in Pubmed: [32320549](https://pubmed.ncbi.nlm.nih.gov/32320549/).
32. Habib G, Badano L, Tribouilloy C, et al. European Association of Echocardiography. Recommendations for the practice of echocardiography in infective endocarditis. *Eur J Echocardiogr*. 2010; 11(2): 202–219, doi: [10.1093/ejechocard/jeq004](https://doi.org/10.1093/ejechocard/jeq004), indexed in Pubmed: [20223755](https://pubmed.ncbi.nlm.nih.gov/20223755/).
33. Asopa S, Patel A, Khan OA, et al. Non-bacterial thrombotic endocarditis. *Eur J Cardiothorac Surg*. 2007; 32(5): 696–701, doi: [10.1016/j.ejcts.2007.07.029](https://doi.org/10.1016/j.ejcts.2007.07.029), indexed in Pubmed: [17881239](https://pubmed.ncbi.nlm.nih.gov/17881239/).
34. Savarapu P, Abdelazeem B, Isa S, et al. Cancer-Related non-bacterial thrombotic endocarditis presenting as acute ischemic stroke. *Cureus*. 2021; 13(5): e14953, doi: [10.7759/cureus.14953](https://doi.org/10.7759/cureus.14953), indexed in Pubmed: [34123650](https://pubmed.ncbi.nlm.nih.gov/34123650/).
35. Vahanian A, Beyersdorf F, Praz F, et al. ESC/EACTS Scientific Document Group. 2021 ESC/EACTS Guidelines for the management of valvular heart disease. *Eur Heart J*. 2022; 43(7): 561–632, doi: [10.1093/eurheartj/ehab395](https://doi.org/10.1093/eurheartj/ehab395), indexed in Pubmed: [34453165](https://pubmed.ncbi.nlm.nih.gov/34453165/).
36. Kuligowska-Jakubowska M, Neubauer-Geryk J, Bieniaszewski L. Leczenie przeciwzakrzepowe u pacjentów po zabiegach kardiochirurgicznych Anticoagulant therapy in patients after cardiac surgery treatment. *ChSiN*. 2010; 1(7): 14–22.

37. Long Y, Gao C. Brain embolism secondary to cardiac myxoma in fifteen Chinese patients. *ScientificWorldJournal*. 2014; 2014, doi: [10.1155/2014/718246](https://doi.org/10.1155/2014/718246), indexed in Pubmed: [24737987](https://pubmed.ncbi.nlm.nih.gov/24737987/).
38. Rosário M, Fonseca AC, Sotero FD, et al. Neurological complications of cardiac tumors. *Curr Neurol Neurosci Rep*. 2019; 19(4): 15, doi: [10.1007/s11910-019-0931-1](https://doi.org/10.1007/s11910-019-0931-1), indexed in Pubmed: [30806824](https://pubmed.ncbi.nlm.nih.gov/30806824/).
39. Kent DM, Ruthazer R, Weimar C, et al. An index to identify stroke-related vs incidental patent foramen ovale in cryptogenic stroke. *Neurology*. 2013; 81(7): 619–625, doi: [10.1212/WNL.0b013e3182a08d59](https://doi.org/10.1212/WNL.0b013e3182a08d59), indexed in Pubmed: [23864310](https://pubmed.ncbi.nlm.nih.gov/23864310/).
40. Shaikh Q, Ahmed B, Ahmed M, et al. Left atrial volumes and associated stroke subtypes. *BMC Neurol*. 2013; 13: 149, doi: [10.1186/1471-2377-13-149](https://doi.org/10.1186/1471-2377-13-149), indexed in Pubmed: [24139054](https://pubmed.ncbi.nlm.nih.gov/24139054/).
41. Quan W, Yang X, Li Y, et al. Left atrial size and risk of recurrent ischemic stroke in cardiogenic cerebral embolism. *Brain Behav*. 2020; 10(10): e01798, doi: [10.1002/brb3.1798](https://doi.org/10.1002/brb3.1798), indexed in Pubmed: [32783327](https://pubmed.ncbi.nlm.nih.gov/32783327/).
42. Ozturk U, Ozturk O. Assessment of left atrial function by strain in patients with acute ischemic stroke left atrial function and acute stroke. *Rev Assoc Med Bras (1992)*. 2021; 67(1): 71–76, doi: [10.1590/1806-9282.67.01.20200303](https://doi.org/10.1590/1806-9282.67.01.20200303), indexed in Pubmed: [34161491](https://pubmed.ncbi.nlm.nih.gov/34161491/).
43. Saha SK, Kiotsekoglou A. Value of speckle tracking echocardiography for prediction of stroke risk in atrial fibrillation: Time to spare a stare outside the box? *Echocardiography*. 2018; 35(5): 589–591, doi: [10.1111/echo.14005](https://doi.org/10.1111/echo.14005), indexed in Pubmed: [29744921](https://pubmed.ncbi.nlm.nih.gov/29744921/).

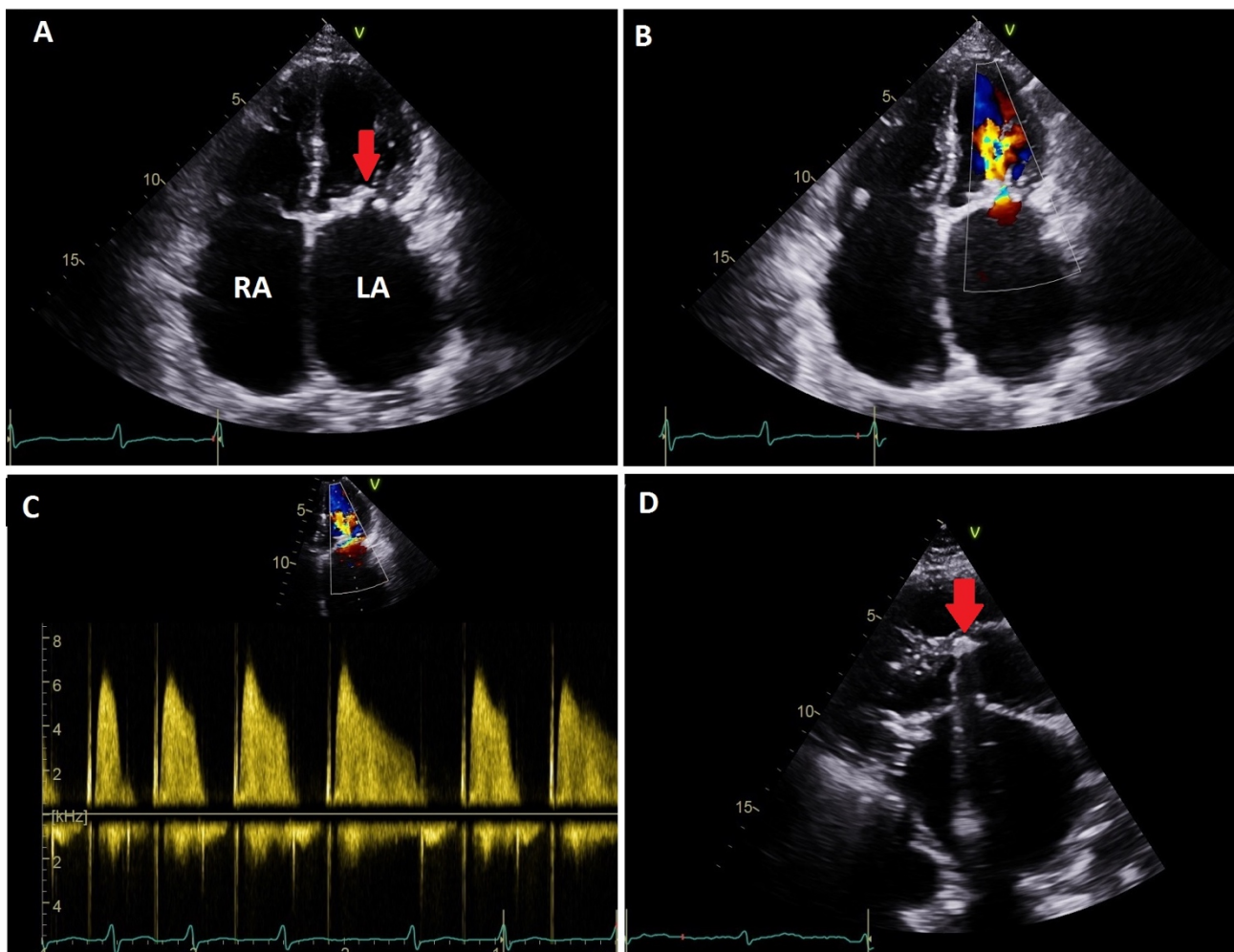


Figure 1. Transthoracic echocardiographic examination of a female patient with several risk factors for cardiac embolism (atrial fibrillation, significant mitral stenosis, aortic valve calcifications): A — Four-chamber apical view, 2D presentation: significantly enlarged atria (LA — left atrium, RA — right atrium), marked with a red arrow indicating massive calcifications of the mitral valve leaflets. B — Four-chamber apical view, colour Doppler: "candle flame" sign — typical for stenotic acceleration of flow across the mitral valve. C — Four-chamber apical view, continuous wave Doppler: flow spectrum across the valve typical for mitral stenosis in a patient with atrial fibrillation. D — Short-axis parasternal view, 2D presentation: The red arrow indicates calcification within the aortic valve leaflets, causing an acoustic shadow.

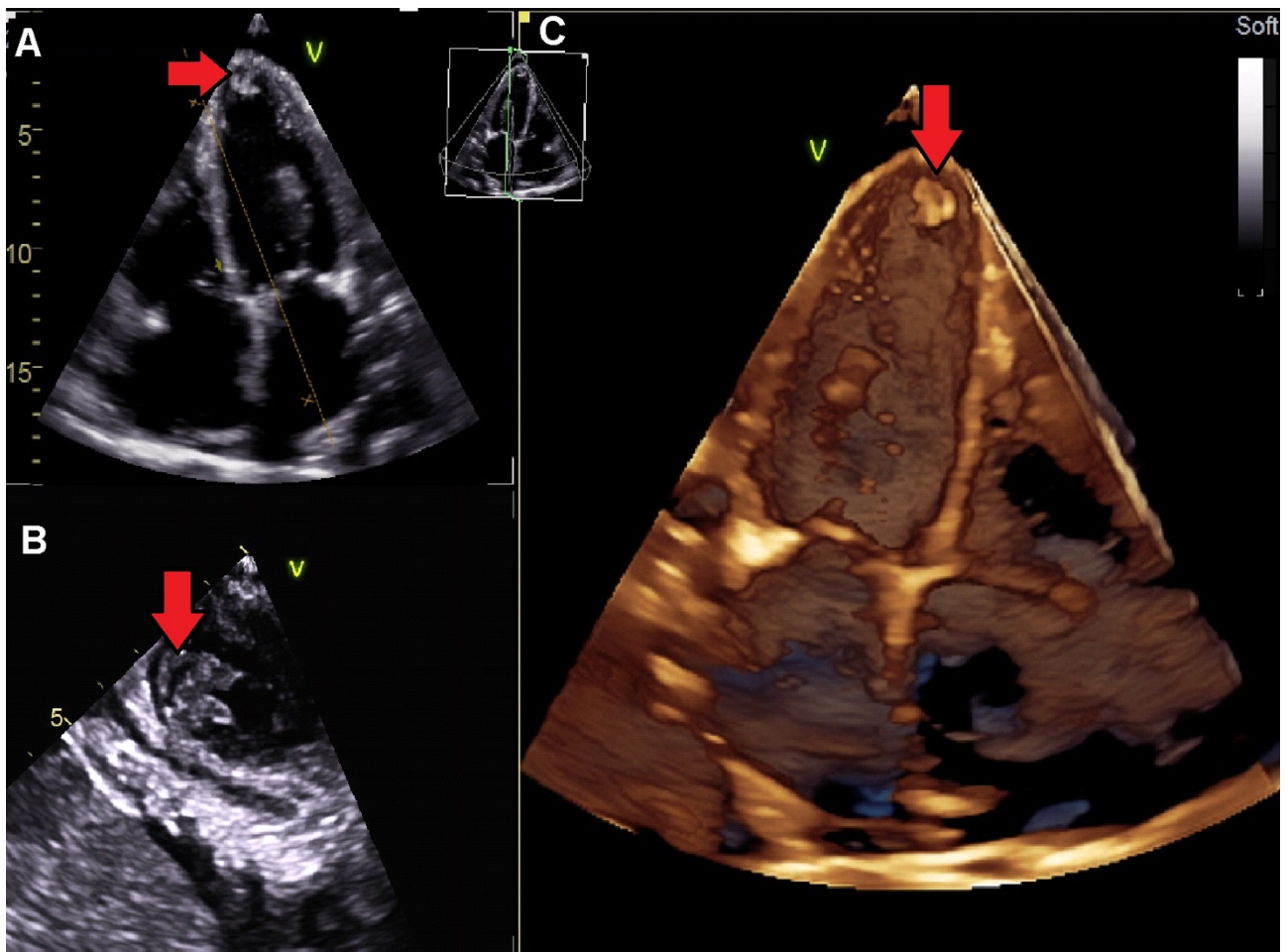


Figure 2. Transthoracic echocardiographic examination in a patient with suspected Transient Ischaemic Attack (TIA) after a previous heart attack involving the apex, with unknown timing: A — Four-chamber apical view. A red arrow indicates an additional structure of variable echogenicity within the area of impaired contractility (apical akinesia with endocardial thinning), corresponding to a thrombus. B — Short-axis parasternal view at the level of the apex. C — Modified three-chamber view, 3D presentation.

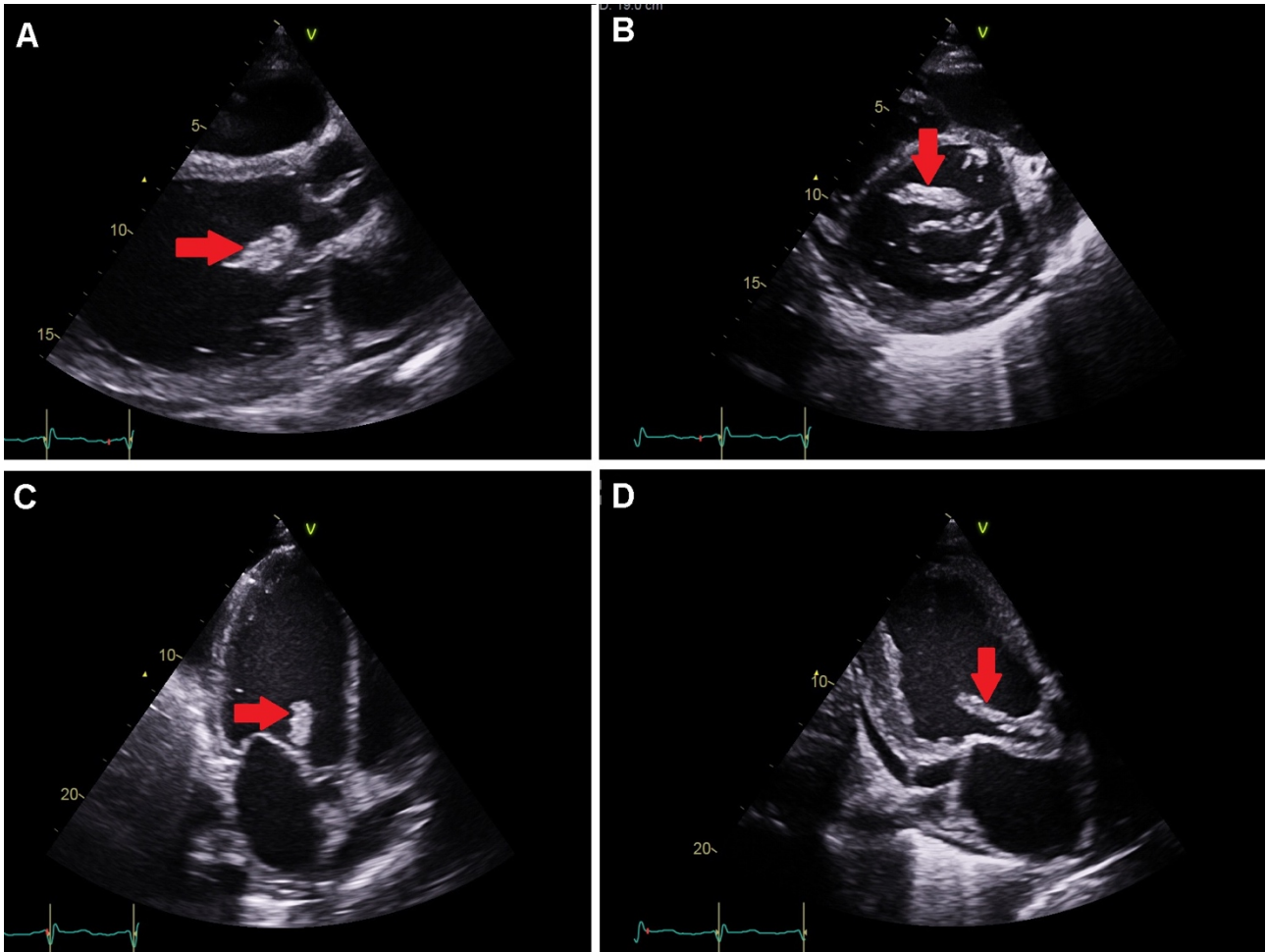


Figure 3. Transthoracic echocardiographic examination, 2D imaging: A — Long-axis parasternal view, B — Short-axis parasternal view, C, D — Apical views C — Three-chamber, D — Modified. A red arrow indicates a structure with variable echogenicity corresponding to bacterial vegetation.

Table 1. Classification of Cardiac Origin Emboli based on the American Society of Echocardiography 2016 Guidelines¹³.

HIGH RISK	LOW RISK
<p>Intracardiac thrombi:</p> <ul style="list-style-type: none"> • Atrial arrhythmias (thrombi in atrial appendage) <ul style="list-style-type: none"> ○ Atrial fibrillation (valvular) ○ Atrial fibrillation (non-valvular) ○ Atrial flutter • Ischaemic heart disease <ul style="list-style-type: none"> ○ Recent MI ○ Chronic myocarditis, especially with left ventricular aneurysm • Non-ischaemic cardiomyopathies • Prosthetic heart valves and devices 	<p>Potential intracardiac thrombi precursors:</p> <ul style="list-style-type: none"> • Spontaneous contrast (without atrial fibrillation) • Left ventricular aneurysm without thrombus • Mitral valve prolapse
<p>Intracardiac vegetations:</p> <ul style="list-style-type: none"> • Infective endocarditis related to native valves • Infective endocarditis related to prosthetic valves • Infective endocarditis not associated with valves 	<p>Intracardiac calcifications:</p> <ul style="list-style-type: none"> • Mitral annular calcification • Calcified aortic valve stenosis

<p>Intracardiac masses:</p> <ul style="list-style-type: none">• Myxoma• Papillary fibroelastoma	<p>Valvular anomalies:</p> <ul style="list-style-type: none">• Chordae tendineae• Lambl's excrescences
<p>Aortic atherosclerosis:</p> <ul style="list-style-type: none">• Thromboembolism• Cholesterol crystal embolization	<p>Defects in the septum and their anomalies:</p> <ul style="list-style-type: none">• Patent foramen ovale (PFO)• Aneurysm of the atrial septum (ASA)• Atrial septal defect (ASD)