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CASE REPORT
Cardiac imaging

Evaluating physiology of a human heart during ventricular tachycardia: new insights of mechanical alterations via beat to beat strain analysis—case report

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Background Ventricular tachycardia can be triggered by myocardial ischaemia. These often fatal events are nearly always accompanied by a significant circulatory depression. However, the exact mechanical alterations and mechanisms of adaption during such arrhythmia episodes are still unknown.

Case summary We report on a 71-year-old male patient with a distinct cardiovascular risk profile, recurrent incidences of dizziness and palpitations. A Holter electrocardiogram was performed showing multiple episodes of ventricular tachycardia. He was immediately transferred to our hospital for further monitoring and diagnostics. During echocardiography, one of these episodes could be recorded with a four-dimensional (4D) probe in triplane acquisition mode and strain analysis was done. Afterwards, a heart catheter examination was performed. A one-vessel coronary heart disease was diagnosed and treated with three drug-eluting stents. The burden of non-sustained ventricular tachycardia (nsVT) significantly reduced post-procedure. During the follow-up, new episodes of nsVT occurred after 6 weeks, which were treated by electrophysiological examinations.

Discussion Modern 4D echocardiography machines offer the possibility to visualize the entire heart simultaneously. Thus ventricular arrhythmias can be evaluated using off-line strain analysis. This technology allows new real-time insights into the human heart showing compensatory mechanisms to overcome stressful episodes, such as ventricular tachycardia.

Keywords Ventricular tachycardia • Strain • Echo • Triplanar acquisition • Case report

Learning points

- New insights into the mechanism of contraction during ventricular tachycardia.
- Triplanar echocardiography with speckle tracking is a very versatile method for clinical and scientific use.

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Introduction

Ischaemic heart diseases and resulting cardiac arrhythmias are the leading cause of death in industrialized nations.[1] Our medical infrastructure tries to counteract this with specialized centres such as chest pain units or hospitals linked in 'heart attack networks'. Ischaemic heart diseases are often complicated by ventricular arrhythmias. Based on electrophysiological exams it was possible to gain a clear view of the origin and the electrical pathways of ventricular tachycardias.[2] However, most of the latest state of the art imaging methods are not able to track rapid motions of a heart during tachycardic events in multiple views simultaneously. In contrast to this, modern echocardiography systems with fast processing and phased array probes are able to record the movement of a patient's heart in 'real time' during such episodes. By chance, a ventricular tachycardia was recorded using a triplanar acquisition mode with high image quality. Based on this data a beat-to-beat strain analysis was performed, providing novel insights into the changes of myocardial contraction over a full cycle of a non-sustained ventricular tachycardia (nsVT).

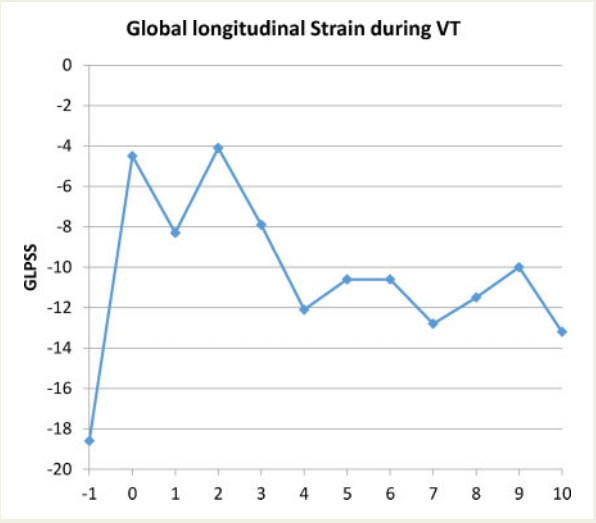

Figure 1 The global longitudinal peak systolic strain is shown. Starting with the basal value during sinus rhythm ('-1'). The triggering ventricular extrasystole is marked as beat '0'. The following beats are the single beats of the non-sustained ventricular tachycardia. The strain is deteriorated by the ventricular extrasystole, but global contraction recovers partially during the ongoing non-sustained ventricular tachycardia.

Timeline

Day 0	Dizziness and palpitations
Day 20	Holter electrocardiogram (ECG) with recurrent episodes of non-sustained ventricular tachycardia (nsVT)
Day 21	Heart catheterization: culprit lesion in the left anterior descending artery with 70–90% stenosis
	Therapy with three drug-eluting stents without significant stenosis afterwards
	Intensifying the bisoprolol therapy and starting with magnesium
Day 22	In the following Holter ECG only one nsVT is recorded
	Back up therapy with 'life vest'
Day 50	Reappearance of the same nsVT
	First attempt of catheter ablation of the LVOT focus (endocardial approach) fails
Day 52	Implantation of an implantable cardioverter-defibrillator
Day 86	Second attempt of catheter ablation (endocardial approach) fails
Day 94	Third catheter ablation (epicardial approach), successful so far
	Initiating therapy with sotalol

Case presentation

Our patient, a 71-year-old man, was admitted to our emergency department. He initially presented to his general practitioner with dizziness and palpitations, and was urgently transferred to hospital when his Holter electrocardiogram (ECG) demonstrated hundreds of episodes of nsVT. The patient's past medical history demonstrated a high cardiovascular risk profile, and included diabetes mellitus and hypertension. He had also previously been diagnosed with blue toe syndrome and peripheral arterial occlusive disease, for which he had previously been administered prostavasin therapy. No cardiac disease was known so far. A clinical examination, including a cardiovascular examination, was performed and was unremarkable. No relevant laboratory abnormalities were present. Continuous ECG monitoring and a 12 channel Holter ECG were performed, to detect the origin of the arrhythmia (Supplementary material online, *Figure S1*). Multiple monomorphic, nsVTs were documented. The origin of the triggering ventricular extrasystole (VES) was septal. Based on the hospital's own localization algorithm (adapted from[3]), the left ventricular outflow tract (LVOT) aortic cusp region was the most likely origin of the nsVT. In a first step an echocardiography was performed. During this exam, an episode of an nsVT was recorded, including the triggering VES. All other values during sinus rhythm (SR) were normal (except a borderline left ventricular global longitudinal peak systolic strain and an aortic sclerosis) (see Supplementary material online, *Table S1*). During coronary angiography, one-vessel disease was revealed with a 70% obstruction of the left anterior descending artery and a 90% obstruction of the first diagonal branch. Both lesions were treated using in total three drug-eluting stents. After this procedure, no residual obstruction was left. Antiarrhythmic medication was optimized by intensifying the existing bisoprolol therapy and adding magnesium. During the three-channel 24 h Holter ECG after intervention only one further nsVT was documented. After a short monitoring period, the patient was supplied with a LifeVest® and was discharged. In the follow-up, the nsVTs reappeared after 6 weeks and the patient had to be sent to a specialized centre. In total, three electrophysiological studies were performed. Two endocardial

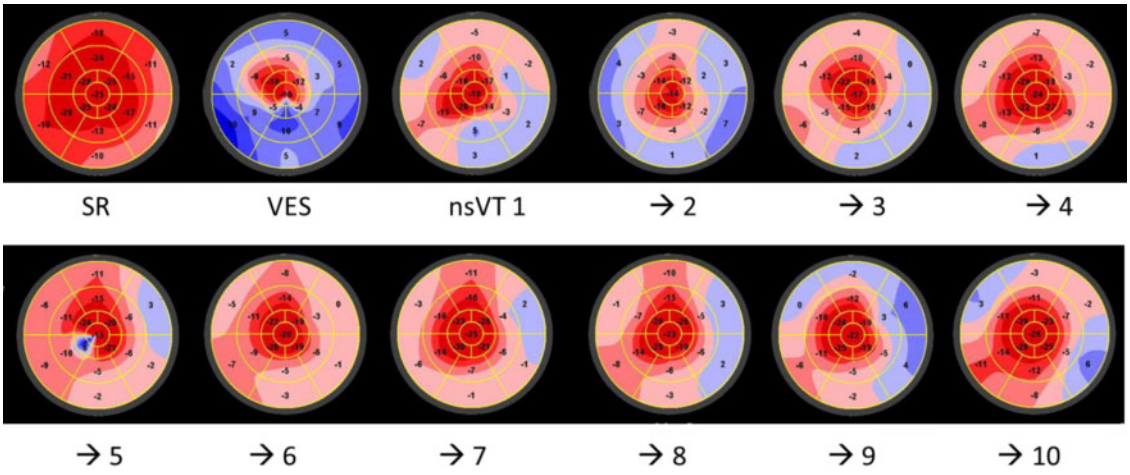

Figure 2 During sinus rhythm, there was a slight strain reduction in the basal segments. The ventricular extrasystole showed a chaotic strain pattern. During ongoing ventricular tachycardia, the longitudinal peak systolic strain recovers in the apical part of the heart.

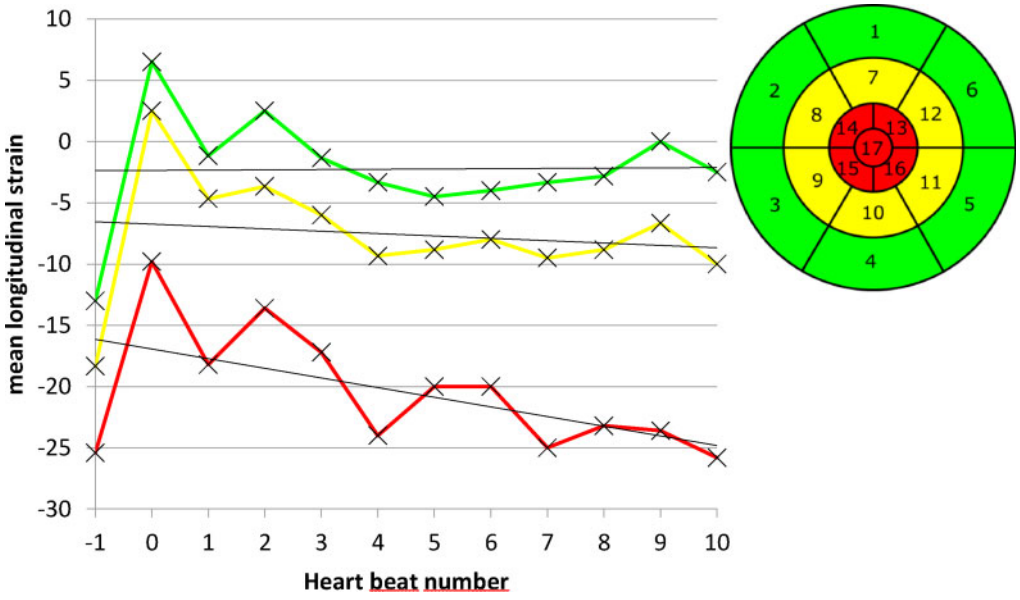

Figure 3 The mean longitudinal strain of the basal (green), the middle (yellow), and the apical part of the heart (red). The normal rhythm is marked as beat '-1', the ventricular extrasystole as beat '0'. The ventricular tachycardia is represented by the beats '1' to '10'. The black lines show the linear regression of each area. Finally, only the apex recovers.

approaches failed to terminate the arrhythmia. Finally, the arrhythmogenic LVOT area was treated by an epicardial ablation and an antiarrhythmic medication with sotalol instead of bisoprolol.

Discussion

Non-sustained ventricular tachycardias are a common consequence of ischaemic heart disease. New imaging techniques enable recording all three apical standard views of a heart simultaneously. Thanks to this new technology, fascinating insights into the pathophysiology of the heart were gained. In this case, high-quality echocardiographic movies of a ventricular tachycardia with a four-dimensional (4D) probe (GE vivid 95; 4Vc Probe, Supplementary material online, *Figure S2*) were captured. In the following analysis, every single beat was analysed using the AFI speckle tracking algorithm from GE providing longitudinal strain values. In the following images, the first beat always represents the normal SR (or beat

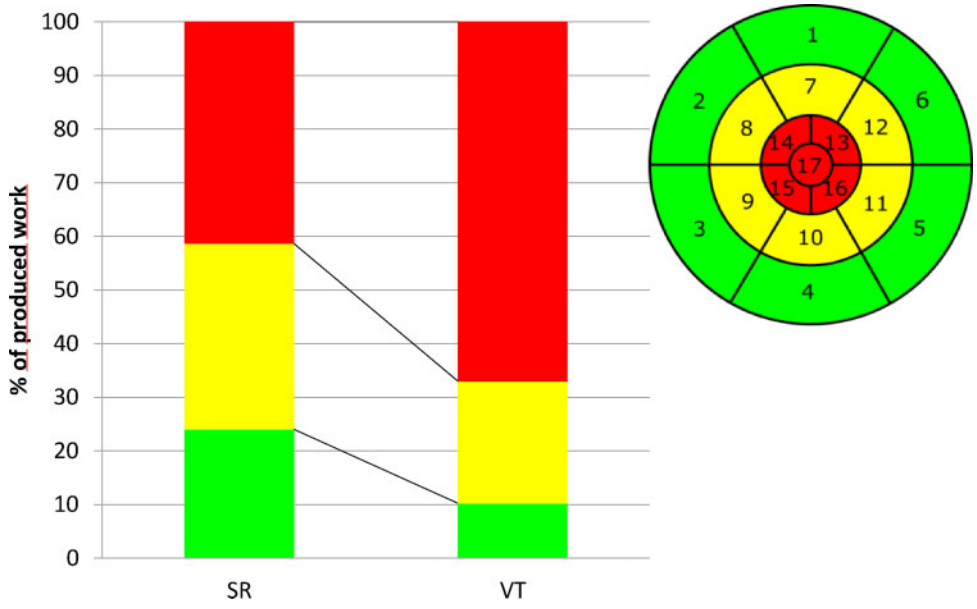

Figure 4 Redistribution of the local myocardial work. The numbers represent the percentage of the total work. During ventricular tachycardia, most of the work is done by the apical parts of the heart (red). Whereas the basal (green) and the middle part (yellow) only produce about one-third of the achieved work.

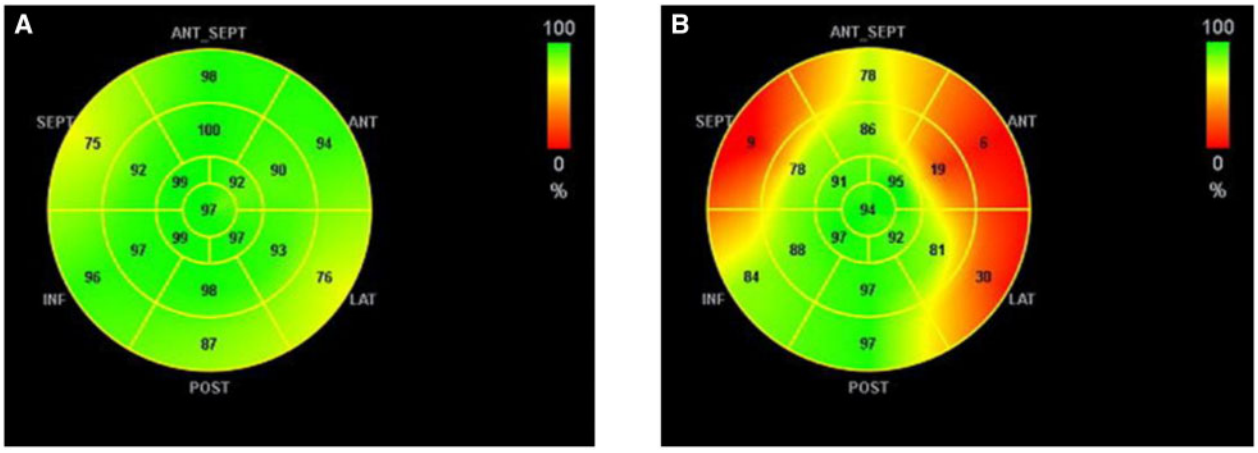

Figure 5 The distribution of myocardial work efficiency. During normal sinus rhythm, the efficiency is high and homogenously distributed (*A*). The ventricular tachycardia only allows an efficient work mainly in the apex (*B*).

'-1'). The second beat shows the triggering VES (or beat '0'), followed by the beats of the ventricular tachycardia in a chronological order (beat 1–10). The first observation was (as expected) a chaotic longitudinal contraction of the myocardium during the extrasystole. The result was a breakdown of the global longitudinal strain. It should be highlighted that strain values recovered during the ongoing nsVT but never reached baseline values (*Figure 1*).

When evaluating in detail, *Figure 2* shows the contraction concentrating progressively at the apex of the heart. In the following, the different segments of the heart are analysed individually. An ASE 17 segment bull's-eye model[4] was used to subdivide the heart in the basal ring (segment 1–6), the middle ring (segment 7–12), and the apex (segment 13–17). During SR there had already been a slight basoapical gradient in strain analysis. This gradient intensified

significantly during nsVT (*Figure 3*). During ongoing ventricular tachycardia, the apical areas slowly reached the same strain as during SR. In contrast, the basal segments did not recover. The GE Vivid e 95 ultrasound system is also able to analyse myocardial work, wasted work, and constructive work with underlying pressure curve templates.[5] An exact definition of the time intervals with the help of valves opening and closing events is crucial for this method. During nsVT these necessary timing events correlated with the recorded image intervals only in one beat. No invasive blood pressure measurement was performed during this standard examination, thus only relative myocardial work values could be analysed. The results of the basal, mid, and apical parts were compared. A significant redistribution of the myocardial work was detected during the tachycardia from the basal parts towards the apex. Here 67% (before 41%; $P < 0.05$) of the contractile work was done. The middle parts only contributed 23% (before 35%; $P < 0.05$) and the base of the ventricle only did 10% (before 24%; $P < 0.001$) of the produced work (*Figure 4*). Furthermore, it is possible to calculate the work efficiency. This pressure independent value is calculated by dividing the amount of constructive work by the sum of constructive and wasted work.[6] As expected the work efficiency was distributed homogenously during SR (*Figure 5A*). The efficiency dropped in the basal part during ventricular tachycardia. The apical parts still worked very efficiently (*Figure 5B*). In summary, the basal parts operated with a low output and low efficiency during nsVT, whereas in contrast, the apical parts did most of the work with relatively good efficiency during nsVT.

Conclusion

Four-dimensional echocardiography is the only imaging technique available to record fast arrhythmias in triplane, in real-time, and with high framerates. When capturing such an abnormal rhythm, important insights are gained into the adaption of the heart and how it overcomes such a stressful event. The apical parts appear to take over the majority of the longitudinal contraction work, but it is not clear whether this redistribution is the result of a general mechanism. It might also be possible that it differs depending on the exit point or pathway of the nsVT. It is also possible that the localization of the underlying structural heart disease may cause this distribution; indeed, such patterns are also observed in other diseases such as cardiac amyloidosis and end-stage hypertension.[7] So far no underlying pathophysiological mechanism is known which explains this common phenomenon. Further research is necessary to clarify whether there are different strain patterns in different nsVT patients. It is also unclear which mechanism is responsible for the apparent recovery of the apical parts. It will likely be possible in the near future to record hearts in 4D with a high temporal and spatial resolution with upcoming techniques like high frame rate echocardiography. This will enable further evaluation of pathological and contractile mechanisms of ventricular tachycardias which may lead to novel diagnostic algorithms.

Lead author biography

Dr. med. Georg Wedekind works at the Clinic for Cardiology and Vascular Medicine at the Alb Fils Kliniken in Göppingen. He studied human medicine at the Ludwig-Maximilians University of Munich and was active in basic research in Munich and Würzburg for several years. His clinical and academic interests include non-invasive cardiac imaging with a focus on 4D echocardiography and strain analysis.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

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Conflict of interest: none declared.

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