A 62-year-old sports teacher was admitted to the emergency room with progressively severe headaches, vomiting, left homonymous hemianopsia, weakness of the left arm and leg, and clouding of consciousness progressing over a few hours. The initial electrocardiogram (ECG) revealed new onset of atrial fibrillation with a ventricular rate of 170 bpm (Figure 1G). A computed tomography (CT) brain scan with contrast enhancement (Figure 1A) confirmed the presence of a large right lobar haemorrhage ~6 cm in diameter with some mass effect.

Figure 1  (A) Contrast CT brain scan on hospital admission showing a large right lobar intracerebral haemorrhage ~6 cm in diameter with some mass effect. (B) Magnetic resonance imaging brain scan on day 4 after admission showing subacute right lobar intracerebral haemorrhage that appears to be a liquid separated from a clot and blood breakdown products. (C) Left ventricular angiogram on day 6 after admission: the left and right coronary arteries reveal no significant coronary artery disease. (D) Reduced uptake of $^{123}$I-MIBG SPECT and 18F-FDG PET in the apical area. $^{99m}$Tc-MIBI SPECT indicated normal perfusion within this region. (E) Left ventricular angiogram on day 6 after admission: end-systolic and end-diastolic frame of left ventricular angiography exhibiting apical lateral wall dyskinesis and basal hypercontractility. Left ventricular ejection fraction 40%. (F) Transthoracic echocardiogram on day 4 after admission: end-systolic and end-diastolic frame of left ventricular echocardiography exhibiting apical lateral wall dyskinesis and basal hypercontractility. Left ventricular ejection fraction 29%. (G) Electrocardiogram on admission: atrial fibrillation with a ventricular rate of 170 bpm. (H) Electrocardiogram on day 4 after admission showing novel T-wave inversion in leads II, III, aVF, V$_1$ - V$_6$, and downsloping ST-segment depression. (I) Scheme of regional wall motion from the traced endocardial LV contours at end-diastole and end-systole from left ventricular angiogram on day 6 after hospital admission.
During conservative treatment, the patient’s symptoms resolved within 3 days and his neurological signs also improved quickly. A magnetic resonance imaging brain scan (Figure 1B) revealed no further increase of the lesion. However, a follow-up ECG now exhibited novel T-wave inversion in leads II, III, aVF, V1–V6, and downsloping ST-segment depression (Figure 1H). Furthermore, serum levels of cardiac troponin T (0.18 ng/mL) were elevated. The ECG displayed markedly impaired left ventricular (LV) function (left ventricular ejection fraction 29%) (Figure 1F). In view of these diagnostic findings and moderate dyspnea, the patient was referred for cardiac catheterization. Coronary angiography revealed insignificant coronary artery disease (Figure 1C). Left ventricular angiography confirmed a depressed ventricular function with an unusual pattern of wall motion abnormalities, characterized by an apical and lateral balloon-like dyskinesis and basal hypercontractility (Figure 1E and I). Single-photon emission computed tomography (SPECT) revealed that myocardial iodine-123 metaiodobenzylguanidine (123I-MIBG) uptake and 18F-fluoro-deoxy-glucose (18F-FDG) were reduced in the akinetic LV area, whereas technetium-99 m methoxyisobutylisonitrile (99mTc-MIBI) SPECT indicated normal perfusion within this region (Figure 1D).

The association between intracerebral hemorrhage and cardiac dysfunction, reflected by ECG-changes, arrhythmia, and elevations of cardiac markers, is well known. However, the underlying pathophysiological mechanisms still remain unclear. Recently, the syndrome of ‘Takotsubo cardiomyopathy’, ‘apical ballooning’ or ‘broken heart syndrome’, characterized by a transient apical ballooning of the LV during states of exaggerated sympathetic activation, has been more and more recognized. Thus, both entities may provide a uniform pathophysiology, characterized by excessive cardiac sympathetic nervous discharge.

Conflict of interest: none declared.

CASE REPORT
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Is coronary vein angioplasty necessary to provide cardiac resynchronization in selected patients? A case report

Maciej Sterliński*, Cezary Sosnowski2, Dariusz Zająć1, Witold Rużyłło2, and Hanna Szwed1

1II Coronary Artery Disease Department, Institute of Cardiology, Spartan ska 1, 02-637 Warsaw, Poland; and 2I Department of Haemodynamics, Institute of Cardiology, Spartan ska 1, 02-637 Warsaw, Poland

*Corresponding author. Tel: +48 223434050; fax: +48 228449510. E-mail address: msterlinski@poczta.onet.pl

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Cardiac resynchronization therapy (CRT) has become a recommended method for patients with congestive heart failure (CHF) and cardiac dyssynchrony. In some cases, CRT implantation procedure can be complicated because of anatomic and technical reasons. Some reports describe balloon angioplasty of stenotic heart veins as a method to achieve the target vessel. We present a case of a 58-year-old male with permanent atrial fibrillation and CHF who was referred for CRT. During the implantation of the pacemaker, the diaphragmatic obstacle in coronary sinus (CS) has been passed after many attempts using a balloon catheter with no inflation. The aim of the report is to discuss, in short, the real necessity of venous angioplasty in the CS bed during CRT implantation.

Keywords Cardiac resynchronization therapy; Venous angioplasty

Introduction
Cardiac resynchronization therapy (CRT) has become a recommended method by the guidelines for selected patients with pharmaco-resistant congestive heart failure (CHF). 1 In some cases, CRT implantation procedure is difficult because of many unexpected reasons. In highly specialized centres, the utility of this procedure fell from 15% to 5–6% during the last few years, 2 because of new improved methods and some interventional cardiology techniques being adapted. In some reports balloon angioplasty of stenotic heart vein has been described as a method to achieve the target vessel. 3 The aim of this paper is to discuss the real necessity of such procedures.

Case report
A 58-year-old male with permanent atrial fibrillation and metabolic syndrome was referred to our hospital, owing to pharmaco-resistant CHF class III NYHA, to be considered for CRT. A borderline ECG and echocardiographic criteria