



HAL
open science

Takotsubo syndrome during induction of general anaesthesia

M. Jabaudon, Marie Bonnin, F. Bolandard, S. Chanseume, C. Dauphin, J. E Bazin

► **To cite this version:**

M. Jabaudon, Marie Bonnin, F. Bolandard, S. Chanseume, C. Dauphin, et al.. Takotsubo syndrome during induction of general anaesthesia. *Anaesthesia*, 2007, 62 (5), pp.519 - 523. 10.1111/j.1365-2044.2007.05061.x . hal-01919309

HAL Id: hal-01919309

<https://hal.science/hal-01919309v1>

Submitted on 12 Nov 2018

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

CASE REPORT**Takotsubo syndrome during induction of general anaesthesia****M. Jabaudon,¹ M. Bonnin,¹ F. Bolandard,¹ S. Chanseau,² C. Dauphin² and J. E. Bazin³**

1 Specialist Anaesthetist and 3 Professor of Anaesthesia and Intensive Care, Department of Anaesthesia and Intensive Care, Hôtel-Dieu, CHU Clermont-Ferrand, France

2 Specialist Cardiologist, Department of Cardiology and Vascular Diseases, Hôpital Gabriel-Montpied, CHU Clermont-Ferrand, France

Summary

A 77-year-old female was admitted in our hospital for uterine prolapse surgery. She developed ventricular tachycardia during induction of general anaesthesia and after initial symptomatic measures, she was transferred to the coronary care unit. Heart failure persisted and electrocardiographic changes mimicking acute myocardial infarction appeared. Coronary angiography was normal and left ventriculography revealed akinesis of the apical region of the left ventricle and apical ballooning during systole, with relative sparing of the base of the heart. Complete recovery of left ventricular function occurred 8 days after the initial onset of symptoms. A diagnosis of Takotsubo syndrome was made on the basis of consistent clinical and laboratory findings, typical echocardiography and angiography findings, and reversible course. This case emphasises the importance of being aware of uncommon causes of cardiac dysfunction in stressful situations, especially during induction of general anaesthesia.

Correspondence to: Dr Matthieu Jabaudon

E-mail: matthieujabaudon@yahoo.fr

Accepted: 13 February 2007

Takotsubo cardiomyopathy, or transient left ventricular apical ballooning syndrome, was first described in Japan as acute systolic heart failure caused by transient left ventricle apical akinesis [1]. This syndrome is usually triggered by stressful situations, and mainly affects elderly female patients [2]. The peri-operative period is well-known to induce stress in patients, and this diagnosis should be considered when a patient presents with left ventricular dysfunction or electrocardiographic changes mimicking acute myocardial infarction in stressful situations [3, 4], especially during the induction of general anaesthesia. We report the case of a 77-year-old female with Takotsubo syndrome that revealed itself during the induction of general anaesthesia.

Case report

A 77-year-old female was admitted to our hospital for vaginal repair of uterine prolapse. She had a history of

facial neuralgia treated by clonazepam, venous insufficiency, previous laparotomy under general anaesthesia and bilateral cataract surgery under peribulbar anaesthesia. There were no anaesthetic complications during these procedures. Pre-operative evaluation including electrocardiography was unremarkable, arterial pressure was 150/80 mmHg, and cardiopulmonary auscultation was normal. The patient decided against spinal anaesthesia, and it was therefore decided to administer a target-controlled infusion (TCI) of propofol, combined with regional anaesthesia (bilateral paracervical and pudendal nerve blocks). Before transfer to the operating room, the patient received 50 mg hydroxyzine. In addition to standard monitoring, anaesthesia depth monitoring was performed during surgery to measure bispectral index (BIS). Her arterial pressure was 195/92 mmHg, and heart rate was 70 beats.min⁻¹. Oxygen saturation was 95% with the patient breathing air. Pre-oxygenation was administered until the expired oxygen reached a fraction of 0.9.

Sufentanil 5 µg was then injected intravenously and propofol was started with a target plasma concentration of 2 µg.ml⁻¹. A few seconds later, the heart rate suddenly increased to 135 beats.min⁻¹, with regular sinus tachycardia. Isolated and runs of premature ventricular beats first appeared, followed by unstable ventricular tachycardia. By this time, the patient had received 112 mg propofol. Ventricular tachycardia ended spontaneously; however, the patient remained in shock, with severe hypoxaemia (lowest saturation 42%). The heart rate suddenly decreased to 40 beats.min⁻¹, prompting intravenous administration of 1 mg atropine. Orotracheal intubation and ventilation with 100% oxygen improved saturation. The BIS value was 0. Surgery was cancelled and it was decided to wake the patient. After awakening, neurological examination was normal. Her arterial pressure decreased from 103/64 to 70/38 mmHg during awakening and intravenous administration of ephedrine (to a total of 15 mg) provided transient and moderate improvement. An electrocardiogram revealed sinus rhythm; the heart rate was 100 beats.min⁻¹, with a QS pattern in leads V₁ and V₂, and a rapidly upsloping ST segment in the anterior and apical precordial leads (Fig. 1). As hypotension and tachycardia remained uncontrolled, an intravenous infusion of noradrenaline was started with increasing doses up to 3 µg.kg⁻¹.min⁻¹, resulting in a rapid rise in blood pressure to 103/71 mmHg. Plasma electrolytes were measured and found to be normal (urea 4.7 mmol.l⁻¹, creatinine

74 µmol.l⁻¹, sodium 138 mmol.l⁻¹, potassium 4.3 mmol.l⁻¹, chloride 105 mmol.l⁻¹, glucose 7.1 mmol.l⁻¹, calcium 2.25 mmol.l⁻¹).

The patient's trachea was extubated, and spontaneous ventilation through a facemask with a 6 l.min⁻¹ oxygen flow resulted in an oxygen saturation of 98%. The patient was transferred to the coronary care unit. Haemodynamic parameters were stable with noradrenaline support, but impairment of tissue perfusion persisted. Electrocardiography was unchanged. On initial echocardiography, the left ventricle was of normal size and non-dilated, with a large antero-apical hypokinetic zone, and hypercontraction of the basal segment of the ventricle. The left ventricle ejection fraction was estimated at 30%. Mild mitral regurgitation was revealed, and systolic pulmonary arterial blood pressure was 48 mmHg. It was decided to switch from noradrenaline to dobutamine (7 µg.kg⁻¹.min⁻¹). Because of the suspicion of acute myocardial infarction, antiplatelet treatment was started with acetylsalicylic acid (500 mg daily), together with enoxaparin (50 mg twice daily), amiodarone (200 mg three times a day), and pravastatin. Inverted T waves appeared in leads V₁ to V₆ on electrocardiography, 12 h after admission to the coronary care unit (Fig. 2). Simultaneously, a systolic murmur, grade 3/6, was heard on the left side of the sternum. There was no chest pain. Troponin Ic levels increased to 0.71 µg.l⁻¹ at 4 h and 0.46 µg.l⁻¹ at 11 h after the episode of ventricular tachycardia (normal values < 0.03 µg.l⁻¹). Creatinine phosphokinase levels were

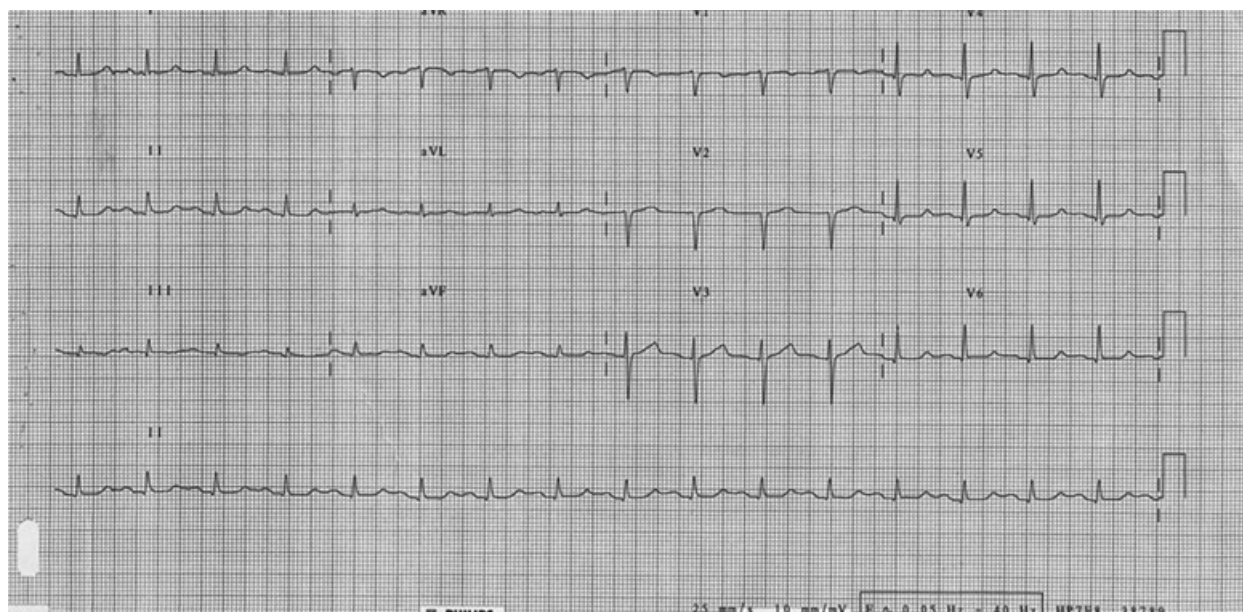


Figure 1 Electrocardiography performed in the postoperative care unit, showing regular sinus cardiac rhythm, heart rate 100 beats.min⁻¹, QS pattern in precordial leads V₁ and V₂, and rapidly upsloping ST segment in the anterior and apical precordial leads.

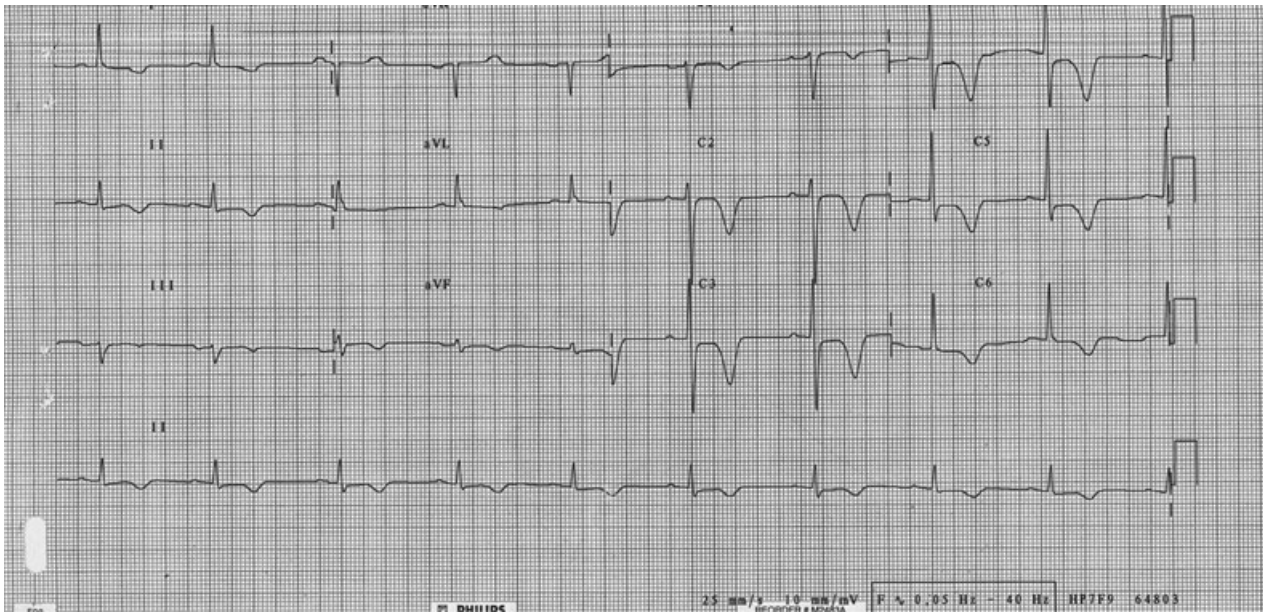


Figure 2 Electrocardiography performed 12 h after admission to the coronary care unit, showing inverted T waves in leads V₁ to V₆.

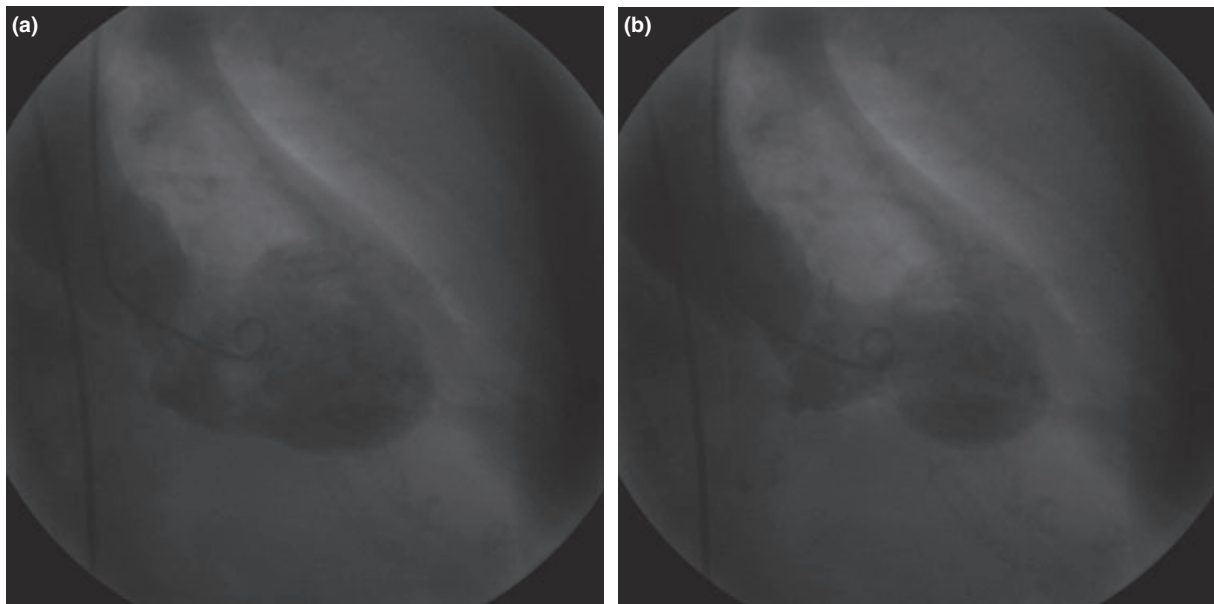


Figure 3 Left ventriculography (a) during diastole and (b) during systole showing akinesis of the distal anterior, apical, and basal regions, and typical apical ballooning during systole, with relative sparing of the basal segment of the left ventricle.

normal, as was coronary angiography. However, left ventriculography revealed akinesis of the distal anterior, apical, and basal regions of the left ventricle, severe systolic dysfunction and apical ballooning during systole, with relative sparing of the base of the heart (Fig. 3). The ejection fraction was 49%. Echocardiography was repeated under dobutamine infusion and persistent apical akinesis was present, with systolic

anterior movement of the mitral valve. The pressure gradient in the left ventricle was 119 mmHg, with typical Doppler flow findings (dynamic outflow obstruction tract). The dobutamine infusion was discontinued, and bisoprolol was started, increasing progressively to 10 mg.day⁻¹. Further laboratory investigations (thyroid function, adrenal cortex function and lipid metabolism) were normal.

Echocardiographical assessment of the left ventricular function 8 days after the initial onset of symptoms revealed complete recovery, and the patient was discharged from the cardiology department on the same day. A diagnosis of Takotsubo syndrome was made on the basis of consistent clinical and laboratory findings, typical echocardiographical and angiographical findings, and the reversible course.

Discussion

Most reports of Takotsubo syndrome are from Japan. Takotsubo cardiomyopathy has been reported during pre-, per-, and postoperative periods but to our knowledge our case is the first observation seen during the induction of general anaesthesia [5–11]. Transient left ventricular apical ballooning syndrome is a recently recognised entity that mimics acute myocardial infarction without coronary stenosis. It results in a profound, reversible left ventricular dysfunction and occurs after sudden emotional stress. This disorder mainly affects elderly female patients (mean age 67 years) [2]. A diagnostic score has been proposed with two major and three minor items [12]. Major items are reversible left ventricular apical ballooning and electrocardiographic changes mimicking acute myocardial syndrome. Minor items are emotional stress, moderate elevation of cardiac enzymes, and chest pain [13]. Our patient had no chest pain, but all other items were present.

Despite the increasing awareness of acute stress-induced myocardial dysfunction, the pathological mechanisms of Takotsubo syndrome remain unclear. Some findings suggest the activation of the neurovegetative system, resulting in exaggerated sympathetic activation [14]. During the acute phase, elevated plasma catecholamine levels are higher in patients with stress cardiomyopathy than in patients with acute myocardial infarction. Unfortunately, these laboratory examinations could not be performed in our case. Catecholamine overload may cause or worsen an intraventricular pressure gradient, with dynamic left ventricle outflow resulting in myocardial ischaemia, as encountered in hypertrophic obstructive cardiomyopathy [15]. Intraventricular pressure gradients have already been described in Takotsubo cardiomyopathy. In our patient, intraventricular pressure gradient rose to 119 mmHg, probably as the result of dobutamine infusion. The increase was higher than in other reported cases [2, 15–17]. The reversibility of pressure gradient under β -antagonist treatment, when appropriate, is well documented [15–17]. The initial choice of haemodynamic support with noradrenaline infusion is perhaps debatable as Takotsubo syndrome is related to exaggerated sympathetic activation, and increased catecholamine intake such as

noradrenaline infusion could worsen symptoms [18]. As left ventricle dynamic obstruction plays a central role in the pathogenesis of Takotsubo syndrome, therapy such as β -antagonists or α_1 -agonists should be aimed at reducing this pressure gradient to improve symptoms [17]. Nevertheless, noradrenaline infusion increased our patient's blood pressure, and at least did not worsen symptoms. β -antagonist treatment, once started, resulted in the complete resolution of symptoms.

Takotsubo syndrome is a rare disorder that is probably often misdiagnosed, and it is suspected of being responsible for 1–2% of admissions for acute myocardial infarction, depending on the country [2, 18]. The peri-operative state is known to induce stress in patients, but there are few documented reports of the onset of Takotsubo syndrome during this period [5–9]. Further data are therefore needed to assess the anaesthetic management of this disorder. However, establishing a good relationship with the patient and administering pre-operative anxiolytic drugs are two measures that could readily be taken. Serious complications have been reported, including left ventricle wall rupture, arrhythmias with repolarisation dispersion, long Q-T interval, torsade de pointe, and conduction abnormalities with third degree atrioventricular block [4, 19, 20]. Fatal outcomes in patients with Takotsubo syndrome have been observed during the initial phase of the disease [2]. However, the prognosis is good among patients who survive the initial severe heart failure without complication, and recurrence seems unusual [2, 11, 21, 22].

The pathogenesis and prevalence of Takotsubo syndrome remain unclear, despite the increasing awareness of acute stress-induced myocardial dysfunction and the increasing number of documented reports. The best therapeutic approach has yet to be established, but β -antagonists are the treatment of choice during the acute phase. Specialised investigations in a coronary care unit setting are needed to diagnose a clinical course such as Takotsubo cardiomyopathy. Nevertheless, this diagnosis should be considered when a patient presents with acute onset of chest pain, electrocardiographic changes mimicking acute myocardial infarction, acute heart failure, or ventricular arrhythmias during the induction of general anaesthesia, and more generally during the whole peri-operative period.

References

- 1 Satoh H, Tateishi H, Uchida T, et al. Takotsubo-type cardiomyopathy due to multivessel spasm. In: Kodama K, Haze K, Hon M, eds. *Clinical Aspect of Myocardial Injury: from Ischemia to Heart Failure*. Tokyo: Kagakuhyouronsya Co., 1990: 56–64.

- 2 Tsuchihashi K, Ueshima K, Uchida T, et al. Transient left ventricular apical ballooning without coronary artery stenosis: a novel heart syndrome mimicking acute myocardial infarction. *Journal of the American College of Cardiology* 2001; **38**: 11–8.
- 3 Matsuoka K, Okubo S, Fujii E, et al. Evaluation of the arrhythmogenicity of stress-induced 'Takotsubo Cardiomyopathy' from the time course of the 12-lead surface electrocardiogram. *American Journal of Cardiology* 2003; **92**: 230–3.
- 4 Denney SD, Lakkireddy DR, Khan IA. Long QT syndrome and torsade de pointes in transient left ventricular apical ballooning syndrome. *International Journal of Cardiology* 2005; **100**: 499–501.
- 5 Takigawa T, Tokioka H, Chikai T, et al. A case of undiagnosed 'takotsubo' cardiomyopathy during anaesthesia. *Masui* 2003; **52**: 1104–6.
- 6 Takayama N, Iwase Y, Ohtsu S, et al. 'Takotsubo' cardiomyopathy developed in the postoperative period in a patient with amyotrophic lateral sclerosis. *Masui* 2004; **53**: 403–6.
- 7 Mizutani K, Okada M. A case of intraoperative repeated coronary artery spasm with ST-depression. *Masui* 2002; **51**: 1114–6.
- 8 Jensen JB, Malouf JF. Takotsubo cardiomyopathy following cholecystectomy: a poorly recognized cause of acute reversible left ventricular dysfunction. *International Journal of Cardiology* 2006; **106**: 390–1.
- 9 Gavish D, Rozenman Y, Hafner R, et al. Takotsubo cardiomyopathy after general anaesthesia for eye surgery. *Anesthesiology* 2006; **105**: 621–3.
- 10 Otomo S, Sugita M, Shimoda O, et al. Two cases of transient left ventricular apical ballooning syndrome associated with subarachnoid hemorrhage. *Anesthesia and Analgesia* 2006; **103**: 583–6.
- 11 Lentschener C, Vignaux O, Spaulding C, et al. Early postoperative tako-tsubo-like left ventricular dysfunction: transient left ventricular apical ballooning syndrome. *Anesthesia and Analgesia* 2006; **103**: 580–2.
- 12 Abe Y, Kondo M. Apical ballooning of the left ventricle: a distinct entity? *Heart* 2003; **89**: 974–6.
- 13 Lipiecki J, Durel N, Decalf V, et al. Ballonisation apicale transitoire du ventricule gauche ou syndrome du tako-tsubo. A propos de 10 nouveaux cas. *Archives des Maladies du Coeur et des Vaisseaux* 2005; **98**: 275–80.
- 14 Wittstein IS, Thieman DR, Lima JA, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. *New England Journal of Medicine* 2005; **352**: 539–48.
- 15 Villareal RP, Achari A, Wiansky S, et al. Anteroapical stunning and left ventricular outflow obstruction. *Mayo Clinic Proceedings* 2001; **76**: 79–80.
- 16 Kyuma M, Tsuchihashi K, Shinshi Y, et al. Effect of intravenous propranolol on left ventricular apical ballooning without coronary artery stenosis (apical cardiomyopathy): three cases. *Circulation Journal* 2002; **66**: 1181–4.
- 17 Penas-Lado M, Barriales-Villa R, Goicolea J. Transient left ventricular apical ballooning and outflow tract obstruction. *Journal of the American College of Cardiology* 2003; **42**: 1143–4.
- 18 Ohtsuka T, Hamada M, Kodama K, et al. Neurogenic stunned myocardium. *Circulation* 2000; **101**: 2122–4.
- 19 Marboeuf P, Ennezat PV, Gonin X, et al. Syndrome de tako-tsubo et obstruction intraventriculaire gauche: mécanisme de pérennisation? A propos d'un cas. *Archives des Maladies du Coeur et des Vaisseaux* 2006; **99**: 69–72.
- 20 Akashi YJ, Tejima T, Sakurada H, et al. Left ventricular rupture associated with Takotsubo cardiomyopathy. *Mayo Clinic Proceedings* 2004; **79**: 821–4.
- 21 Bybee KA, Prasad A, Barsness GW, et al. Clinical characteristics and thrombolysis in myocardial infarction frame counts in women with transient left ventricular apical ballooning syndrome. *American Journal of Cardiology* 2004; **94**: 343–6.
- 22 Akashi YJ, Musha H, Kida K, et al. Reversible ventricular dysfunction takotsubo cardiomyopathy. *European Journal of Heart Failure* 2005; **7**: 1171–6.