CARDIOVASCULAR MEDICINE

Sudden adult death syndrome and other non-ischaemic causes of sudden cardiac death

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Objective: To evaluate non-atherosclerotic cardiac deaths in the UK population aged over 15 years including elderly patients and to highlight the concept of the structurally normal heart in sudden death.
Methods: Pathological data were collected prospectively for sudden adult deaths referred by UK coroners.
Results: 453 cases of sudden death from 1994 to 2003 (278 men (61.4%) and 175 women (38.6%), age range 15–81 years) were reviewed. Males predominated in both age groups ($\leqslant$ 35 years, $>$ 35 years). More than half of the hearts (n = 269, 59.3%) were structurally normal. In the other 40.7%, cardiac abnormalities were noted, which included: (1) cardiomyopathies (23%) such as idiopathic fibrosis, left ventricular hypertrophy, hypertrophic cardiomyopathy, dilated cardiomyopathy, and arrhythmogenic right ventricular dysplasia; (2) inflammatory disorders (8.6%) including lymphocytic myocarditis and cardiac sarcoidosis; (3) non-atheromatous abnormalities of coronary arteries (4.6%); (4) valve diseases; and (5) miscellaneous and rare causes.
Conclusion: The concept of the structurally normal heart in sudden death and the need for histological examination to detect underlying disease is highlighted. Relatives need to be referred for cardiological and genetic screening in cases of normal hearts found at necropsy.

Sudden adult cardiac death is caused by ischaemic heart disease in the vast majority of cases.[1] In previous UK series this cause ranged between 59–86% of sudden death cases in the community.[2 3] Sudden unexpected cardiac death in the community in which no cause can be found at a coroner's postmortem examination is increasingly recognised. The proportion of unexplained deaths in one of the earliest studies in Wandsworth in 1988 was 3.4%.[2] In the first national prospective study of sudden death, funded by the British Heart Foundation in the early 1990s, a very similar figure of 4.1% of unexplained deaths was reported after detailed examination by three cardiac pathologists.[3] Both these studies advocated identifying these cases by a name, the sudden adult death syndrome (SADS), to highlight the problem and deal with it in a similar fashion to sudden infant death and to study the aetiology systematically. The concept of the morphologically normal heart in sudden death is of major importance with the emergence of the molecular channelopathies such as the long QT or Brugada's syndrome giving rise to lethal cardiac arrhythmias in the past 15 years.[4] Non-ischaemic causes of sudden cardiac death are of major importance because they often include genetic diseases, such as hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy, and arrhythmogenic right ventricular dysplasia (ARVD).[5] After the initial British Heart Foundation study we acted as a referral centre for sudden cardiac death cases from coroners throughout the UK and have now completed an analysis of the cases referred for a pathological opinion, often because the referring pathologist did not find a cause of death or was uncertain of the cause of death. The patients were all older than 15 years, and atherosclerotic coronary artery disease (coronary artery stenosis, with or without acute or old myocardial infarction or fibrosis) was excluded as a cause of death. This is the first pathological study to evaluate non-atherosclerotic cardiac deaths in the UK population aged over 15 years including elderly patients.

METHODS

From January 1994 to April 2003, all cases of sudden adult cardiac deaths referred to us from coroners throughout the UK were entered on a prospective database. All the patients had been well until their sudden death with no history of heart disease, apart from the congenital cases and a history of hypertension (elicited from the general practitioner's notes after left ventricular hypertrophy (LVH) was reported pathologically). Toxicology (reports were provided by the coroner) was negative for all patients. Details concerning other diseases were obtained from the coroners once they had been in contact with patients' general practitioners. For this study, ischaemic heart disease cases were excluded. Histological sections from the myocardium (51% of cases, between 2–10 sections for each case), a single myocardial transverse section of both ventricles (2%), or whole hearts (47%) were referred by pathologists with permission obtained from the next of kin. The patient's age, sex, weight, and height (when provided), heart weight, thicknesses of the left and right ventricles, and overall description were recorded. Where a patient's height and weight were not available, normal cut off parameters for heart weight were 500 g for men and 400 g for women and for left ventricular thickness, 15 mm. Selected sections were stained with elastic van Gieson to assess fibrosis.

Histological criteria were as follows: for HCM, myocyte disarray, interstitial fibrosis, and vascular changes of small arterioles (thickening of the wall); for ARVD, fat and fibrosis throughout the wall of the right ventricle with or without chronic inflammatory infiltrate; for LVH, hypertrophied myocytes with no fibrosis; for idiopathic fibrosis, widespread fibrosis in the left ventricle with no evidence of ventricular wall thinning; and for dilated cardiomyopathy, a thin walled

Abbreviations: ARVD, arrhythmogenic right ventricular dysplasia; HCM, hypertrophic cardiomyopathy; LVH, left ventricular hypertrophy; SADS, sudden adult death syndrome

left ventricle with fibrosis throughout the left ventricle with no coronary artery disease. The final pathological diagnosis was noted and results were categorised as follows: (1) normal heart (with or without associated diseases); (2) cardiomyopathies; (3) myocarditis; (4) non-atheromatous abnormality of the coronary arteries; (5) valvar diseases; and (6) other abnormalities including disorders of the conduction system.

RESULTS

We retrieved 453 cases of sudden cardiac death from the database for 278 men (61.4%) (median age 32 years (range 15–81)) and 175 women (38.6%) (median age 31 years (range 15–75)). Two hundred and twenty three (49.2%) were aged ⩽ 35 years (75 women, 148 men) and 230 (50.8%) were aged > 35 years (table 1).

Normal hearts

Hearts were found to have normal macroscopic and microscopic appearances in 269 cases (59.3%; 162 men, 107 women), indicating true SADS. Patients 35 or younger (53.5%) and patients older than 35 (46.5%) (table 1) were equally distributed. Males predominate in both age groups. In this group of patients with morphologically normal hearts, 13% had reported diseases known to predispose to sudden death. These were epilepsy (n = 11), alcoholic fatty liver (n = 9), anorexia (n = 5), asthma (n = 4), diabetes mellitus (n = 4), and schizophrenia (n = 3).

Cardiomyopathies

Abnormalities of the myocardium were found with certainty in 107 patients, accounting for 24% of all cases (table 2). Tissue sampling from seven patients (as described below) was insufficient for definite diagnosis. Males predominated in both age groups. LVH and idiopathic diffuse cardiac fibrosis made up 55% of the total cardiomyopathy group. LVH without fibrosis or disarray was the most common abnormality, found in 31 patients, with males predominating in both age groups. Associated diseases found in 14 patients were hypertension (n = 9), including four patients (four males, three aged less than 35) reported to be of Afro-Caribbean origin, and aortic stenosis (n = 5). Idiopathic diffuse left ventricular cardiac fibrosis was similarly seen in 29 patients (6.4% of the whole series, 27% of the cardiomyopathy group). The majority were men (18 of 29 cases) with equal distribution in both age groups. In 15 of 29 cases, the heart was macroscopically normal with no evidence of thinning or scarring. In the remaining 14, the heart was hypertrophied. No history of hypertension was obtained for these patients. HCM was the third most common disease of the myocardium in 28 cases. While 23 patients had the classic hypertrophied left ventricle, in five the heart was macroscopically normal. Males also predominated with this disease (21 of 28 cases). Cases were equally distributed between both age groups but more women had the diagnosis after the age of 35. Interestingly, one patient had associated diffuse lymphocytic myocarditis.

ARVD was observed in 10 patients, six males and four females, and six of 10 cases of ARVD were diagnosed in the ⩽ 35 age group. Seven were described as having fatty replacement of the right ventricle macroscopically, one had a thin right ventricle wall, and two appeared macroscopically normal. ARVD was seen in association with diffuse lymphocytic myocarditis in one patient. Seven additional cases with fatty infiltration of the right ventricle were referred, but we had only limited material for all cases (slides only, no whole hearts) and ARVD could not be diagnosed with certainty because of this limitation. Dilated cardiomyopathy was diagnosed in nine cases, with a female predominance (seven of nine). In the > 35 age group, all patients were female. In retrospect we found five patients had associated heavy alcohol intake, one died after childbirth, one had diabetes mellitus, one had acute thyrotoxicosis, and one died after chemotherapy. For one patient, a positive family history for dilated cardiomyopathy had been reported.

Table 1 Age and sex distribution of the cohort and constitutively normal hearts

	⩽35 years	>35 years	Total
Overall			
F	75	100	175 (38.6%)
M	148	130	278 (61.4%)
Total	223	230	453
Normal hearts (59.3%)			
F	52	55	107 (39.8%)
M	92	70	162 (60.2%)
Total	144	125	269

F, female; M, male.

Myocarditis

Thirty nine patients had myocarditis (two already mentioned in association with HCM and ARVD) accounting for 8.6% of the studied hearts (table 3). Lymphocytic myocarditis, which was shown by lymphocytes surrounding necrotic myocytes in at least two foci in each block of tissue examined, was diagnosed in 24 patients and most of these had macroscopically normal hearts in which histological analysis showed the cause of death. The majority of cases were found in patients aged ⩽ 35 (16 of 24) and of those, 12 were male. Granulomatous myocarditis, in which there were well formed granulomas without any eosinophilic infiltrate (cardiac sarcoidosis), was observed in 10 patients, all aged > 35, with a female predominance (eight of 10). Toxic myocarditis was diagnosed in five patients by an infiltration of predominantly macrophages and eosinophils in the interstitium (attributed to drugs in two cases and thyrotoxicosis in one case).

Non-atheromatous abnormality of the coronary arteries

Non-atheromatous coronary artery diseases were observed in 21 patients (4.6%) (table 4). Anomalous coronary arteries were observed in six patients with both coronary arteries arising from the same coronary ostium in five cases and an absent right coronary artery in one case. Five of six patients were aged ⩽ 35 years. Coronary spasm was diagnosed in six patients, where regional ischaemic damage including contraction band necrosis (three of six) or regional acute transmural or chronic myocardial infarction (three of six) was observed in association with normal coronary arteries. Cases were found in both age groups. Bridging of the left anterior descending coronary artery by muscle was systematically looked for and observed in four patients, three being in the ⩽ 35 year age group. The bridge varied from 20–40 mm in length and 2–5 mm in depth. Coronary artery vasculitis was found in three patients (one associated with an inflammatory pseudotumour of the kidney with IgG paraproteinaemia, one case of eosinophilic vasculitis with possible Churg-Strauss syndrome, and one case of giant cell arteritis associated with giant cell aortitis). Both cases of spontaneous dissection of the coronary arteries were found in males.

Valvar abnormality

Valvar abnormalities as a cause of death were nine floppy mitral valves, one cleft mitral valve, aortic stenosis with bicuspid aortic valve in four cases and degenerative trileaflet aortic valve in one case.

Table 2 Abnormality of the myocardium with a diagnosis of certainty by age and sex

	No	Percentage of group	Percentage of cohort	⩽35 years		>35 years	
				M	F	M	F
LVH	31	28%	6.8%	11	0	15	5
IF	29	27%	6.4%	8	5	10	6
HCM	28	26%	6.2%	13	2	8	5
ARVD	10	9%	2.2%	4	2	2	2
DCM	9	8%	1.9%	2	3	0	4
Total	107	100%	24%	38	12	35	22

ARVD, arrhythmogenic right ventricular dysplasia; DCM, dilated cardiomyopathy; HCM, hypertrophic cardiomyopathy; IF, idiopathic fibrosis; LVH, left ventricular hypertrophy.

Other abnormalities

Conduction system

The conduction system was examined in seven cases. There was one case of fat and fibrosis in the atrioventricular node of a 43 year old woman who had a documented history of arrhythmias during life (paroxysmal supraventricular tachycardia, atrial fibrillation, and complete heart block), one case of tuberous sclerosis in a 41 year old woman with multiple ventricular lipomata and a lipoma in the atrioventricular node, and two cases of lipomatous hypertrophy of the interatrial septum, one associated with fatty infiltration of the atrioventricular node. In three cases of cardiac sarcoidosis, epithelioid and giant cell granulomas were found in the atrioventricular node.

Congenital heart disease

There were six cases of surgically corrected complex congenital heart diseases (two repaired atrial septal defects with right ventricular hypertrophy and LVH, one repaired tetralogy of Fallot, one repaired ventricular septal defect with right ventricular hypertrophy, two more complex cases with dextrocardia in one patient and corrected atrioventricular septal defect in another). All these patients had been operated on many years previously and there was no indication of clinical deterioration before sudden death. At necropsy, apart from the surgical corrections and congenital anomalies, there were no specific new findings to explain the sudden death in each case. The pathologists had referred them because of their complexity and surgical correction.

Miscellaneous

Very rare causes of sudden death were idiopathic thrombotic thrombocytopenic purpura associated myocardial necrosis (n = 2), lipoma in the left coronary ostium (n = 1) causing obstruction of the left main stem, small benign tumour (atrioventricular nodal mesothelioma) (n = 1), and metastatic adenocarcinoma to the myocardium (n = 1).

DISCUSSION

A substantial proportion of people experience sudden death as the first and only clinical expression of underlying coronary artery disease and pathologists are familiar with this in their coronial practice. However, there are few large reports on sudden cardiac deaths due to non-atheromatous or to no discernable cause. Reports of non-atheromatous causes of sudden cardiac death tend to concentrate on a limited age group of patients younger than 35–40, including athletes.[6–9] The current study differs in that it explored sudden cardiac death in a wider age group (15–81 years) and described a large proportion of macroscopically and microscopically normal hearts and non-atheromatous causes of sudden cardiac death in which pathologists were uncertain of the cause of death or wished for confirmation of the diagnosis.

Almost 60% of our patients had a macroscopically and microscopically normal heart with an equal distribution between patients 35 and younger and patients older than 35, emphasising that all age groups and not just the young or athletes can die suddenly. The male predominance is also remarkable. This group is much larger than that of the Italian study of 273 young patients who died suddenly (⩽ 35 years old), of whom 16 had a normal heart, giving a rate of 6%.[8] A 21% rate of normal hearts in unexplained death was observed in a Swedish study[9] in the same age group. In a French study in 1996 of 1000 sudden death necropsies of adults under 65 years of age, 12.3% had normal hearts.[10] In an American series of 14–40 year olds, 15.8% had normal hearts.[6] We studied all our cases less than 40 years for comparison and showed that 60% had a normal heart. The prevalence of hearts with ''no finding'' decreased with age in the study of Virmani *et al*[7] in 2001: 30% in the 14–20 age group, 21% in 21–30 age group, and 9% in the 31–40 age group. In our series, almost half of the normal hearts were from patients older than 35 years, a finding not established in the literature, which has in the past emphasised sudden death in younger age groups. The high proportion of normal hearts in the present study may be explained by the selective and biased referral pattern by pathologists who found nothing at necropsy or were uncertain of the cause of death; another explanation may be the poor sampling in some cases (as seen for seven cases of suspected ARVD) and the fact that slides rather than whole hearts were referred in 51% of cases with limited sampling of the right ventricle.

Table 3 Inflammatory diseases of the myocardium by age and sex

	⩽35 years		>35 years		
	M	F	M	F	Total
Lymphocytic myocarditis	12	4	1	7	24
Granulomatous myocarditis	0	0	2	8	10
Toxic myocarditis	1	0	1	3	5

Table 4 Non-atheromatous abnormality of the coronary arteries by age and sex

	⩽35 years		>35 years		
	M	F	M	F	Total
Anomalous coronary arteries	4	1	1	0	6
Spasm	2	2	1	1	6
Bridging of the LAD	2	1	1	0	4
Vasculitis	0	2	1	0	3
Spontaneous dissection	1	0	1	0	2

LAD, left anterior descending coronary artery.

Sudden death with a morphological normal heart is a very important negative finding at necropsy.[3] Genes have been identified for several disorders responsible for arrhythmias and sudden death. These genes all encode ion channels and are referred to as channelopathy genes. The proteins that regulate electrical activity are not detectable morphologically at the time of postmortem examination. A diagnosis can only be made by ECG investigation during life.[11] The occurrence of sudden death with a normal heart should therefore prompt referral of close relatives to a specialist cardiologist for genetic screening. In a recently completed study of 147 first degree relatives of 32 people who died of SADS, 109 (74%) underwent cardiological assessment; the diagnoses were inherited cardiac disease in seven (22%) of the 32 families and the long QT syndrome in four.[12] This study emphasises that it is important to screen families for genetic conditions in sudden cardiac death cases. However, although we systematically advise genetic screening of relatives in cases of sudden adult death with a morphologically normal heart, one limitation of the current study is the lack of genetic results or information of family history in this group of patients with structurally normal hearts. We need to study these cases in more detail.

The proportion of patients with sudden death with a normal heart and associated diseases was 13% in our series. Sudden death has also been reported in epilepsy without clinical evidence of status epilepticus,[13] in asthma without clinical evidence of status asthmaticus,[14] in anorexia,[15] in schizophrenia,[16] and in patients with alcoholic fatty liver without an alcoholic cardiomyopathy.[17] As arrhythmias have been documented during life in patients with these conditions, cardiac arrhythmias are thought to be the mode of death. This 13% rate is by no means accurate and is probably an underestimate due to lack of detailed clinical data in each case. We need to study the clinical history of all these cases further.

Normal hearts macroscopically can show microscopic abnormalities as our study also highlights. In the Italian study[8] among the 28% of the hearts that were macroscopically normal, histological examination of 79% disclosed concealed pathological substrates, as also described in a recent French study (of 1930 unexplained sudden deaths, 200 had pathological evidence of ARVD[18]), emphasising that histological examination and sampling are essential even in normal appearing hearts.

All types of cardiomyopathies including HCM, dilated cardiomyopathy, and ARVD have an underlying genetic mutation in many cases. Cardiomyopathies were responsible for sudden cardiac death in nearly 25% of our patients, a significant proportion. The diagnosis of cardiomyopathies can be difficult in view of their variation in phenotypic expression. HCM can present with sudden death and a macroscopically normal heart as this study also shows. The male predominance with this entity in both age groups is emphasised and also that older patients with this condition can die suddenly.

LVH was male predominant and affected the older age group in our study. Note the link with hypertension in nine cases, which emphasises that detailed study of general practitioners' records of sudden deaths may disclose underlying causes as well as racial differences. Although LVH is accepted as a cause of sudden death, its definition is controversial and should be ideally related to the body mass index of the deceased. In current routine coronial work, the weight and height of the deceased may not be recorded and the heart weight is assessed in isolation. One of the drawbacks of the current study is the absence of body mass index data in most cases and the evaluation of LVH on arbitrary cut off points, as these values may be normal in tall and overweight people.[19]

Normally we do not check the conduction system in sudden cardiac death. Abnormalities including abnormal conduction bundles, fibrosis, and fatty infiltration have been described but their role in causing death is controversial.[20] In the current study the conduction system was examined only if clinically indicated, in particular if the patient was know to have arrhythmias during life, and this yielded positive findings. Interestingly the Italian study reported 24 cases of conduction system diseases and most of the patients had clinical evidence of arrhythmias during life.[8]

Many other studies confirm anomalous coronary arteries and bridging as a cause of sudden death.[21 22] The role of coronary artery spasm is more controversial but has been linked to sudden death and survival after cardiac arrest.[23] Vasculitis can affect the coronary arteries locally. This emphasises that detailed study of all of the coronary arterial system is essential in cases of sudden cardiac death. Lastly, we included congenital heart disease because it has an established link to fatal arrhythmias in the absence of symptoms and clinical deterioration in which no new findings are seen.[24]

Conclusion

The non-atheromatous causes of sudden cardiac death present a diagnostic challenge for coroners and pathologists. A normal heart is an important negative finding in the investigations, as it warrants referral of living relatives to a specialist cardiologist and genetic screening. As macroscopically normal hearts can have microscopic disease, histological analysis is mandatory to make a specific diagnosis. Today, with many questioning the role of the necropsy in modern medicine,[25] this study emphasises its importance and central role in helping families come to terms with death in a previously healthy relative. Detailed and precise cardiac findings will be important for these families in terms of health screening, genetic counselling, health insurance, and treatments such as drugs and implantable defibrillators.

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IMAGES IN CARDIOLOGY

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Left subclavian artery and left anterior descending coronary artery stenoses: obstacles to the left internal mammary artery

A 77 year old man, having previously undergone triple vessel coronary artery bypass (CABG) surgery with a left internal mammary graft (LIMAG) to the left anterior descending (LAD), saphenous vein grafts (SVG) to the obtuse marginal (OM) and circumflex (Cx) branches, presented with an anterior acute coronary syndrome. Coronary angiography revealed an occluded Cx-SVG graft and patent OM-SVG. There was considerable difficulty in selectively intubating the LIMAG; suboptimal images suggested complete occlusion of a relatively small calibre LAD beyond the insertion of the LIMAG (panel A). A subsequent pullback pressure gradient (60 mm Hg) across the left subclavian artery (LSCA) and arch aortogram revealed a significant LSCA stenosis (panel B). At this stage the LSCA stenosis was dilated with an 8 mm diameter balloon by a vascular radiologist, enabling selective catheterisation of the LIMAG and enhanced visualisation of the LIMAG-distal LAD (panel C). It was then apparent that there was subtotal occlusion of the LAD with minimal antegrade flow. In light of the clinical presentation, the LAD was recanalised by percutaneous transluminal coronary angioplasty (PTCA) and stenting via the LIMAG (panel D). The patient has remained asymptomatic since. This case highlights the importance of pursuing the acquisition of selective images of all vessels at the time of coronary angiography particularly in CABG patients since, in this instance, discovery and then treatment of the LSCA stenosis undoubtedly improved flow and hence visualisation of the LIMAG-LAD system. This certainly facilitated successful PTCA of the culprit LAD lesion which led to symptom resolution.

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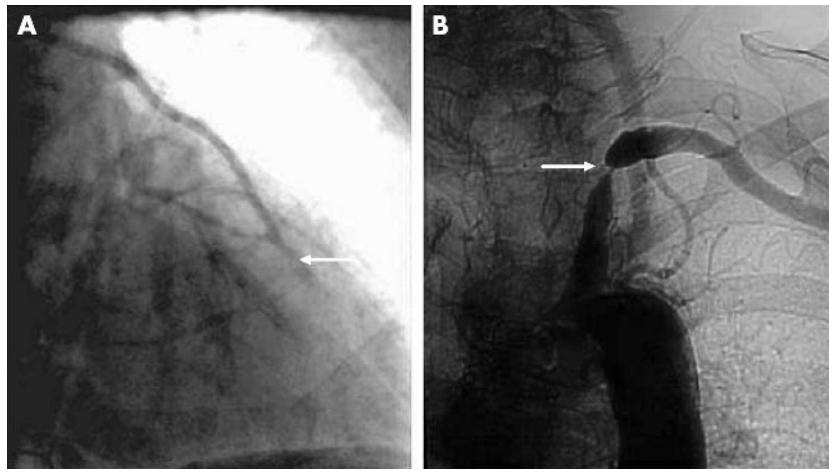
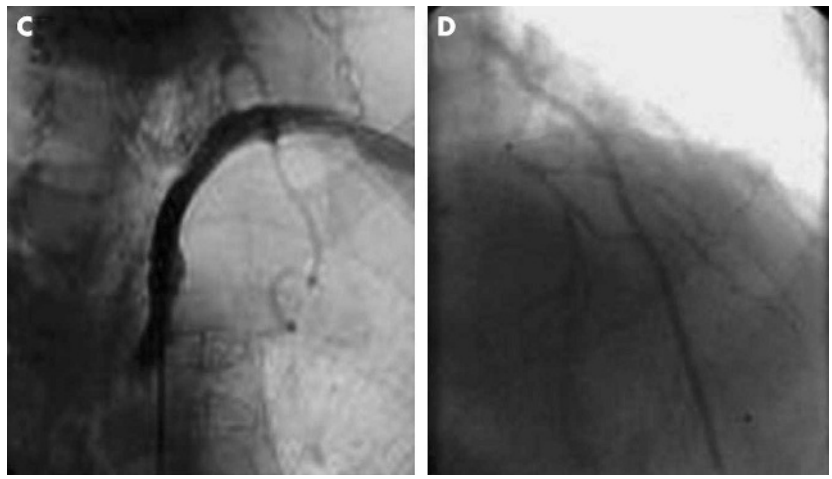
We, the authors, wish to state that there are no competing interests/no conflicts of interest



(A) Initial appearance of the left internal mammary graft-left anterior descending (LIMAG-LAD) system with a non-selective injection suggesting an occluded LAD beyond the graft anastomosis (arrow). (B) Subsequent arch aortogram demonstrating a significant flow limiting left subclavian artery (LSCA) stenosis (arrow).



(C) Successful balloon dilatation of the LSCA stenosis. (D) Final injection of the LIMAG following coronary angioplasty and stenting of the LAD.