

Sudden cardiac death in apparently normal young adults

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MOST PEOPLE WHO DIE suddenly from cardiac disease are elderly and develop symptoms before the fatal event. A few apparently fit and healthy young adults die suddenly and unexpectedly from cardiac disease — often, but not always, during intense physical activity. Detecting young adults with potentially lethal underlying cardiac abnormalities is an important aspect of screening before enrolment in pilot training, military service and participation in sport.

In the literature, “young” is arbitrarily defined as an age of 35 years or less. Beyond this age, coronary artery disease is the commonest cause of sudden unexpected cardiac death. Coronary artery disease is still a relatively common cause of death even among young adults. Stary demonstrated fatty streaks in the coronary arteries of children as young as 10 years,¹ and Nissen et al, using intracardiac ultrasound, detected evidence of significant coronary atherosclerosis in the arteries of more than 50% of donor hearts (average age, 26 years) when these were screened before transplantation (personal communication, S Nissen, Department of Cardiology, Cleveland Clinic, Ohio).

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy is a relatively common genetic malformation of the heart affecting almost one in 500 of the population.^{2,3} About half those affected inherit the disorder, which is autosomal dominant with variable penetrance; the others are thought to have had a spontaneous mutation. The disorder is characterised by varying degrees of left ventricular hypertrophy, often with disproportionate thickening of the interventricular septum. The abnormal septal thickening frequently leads to dynamic obstruction of the left ventricular outflow tract, resulting in symptoms of chest pain, syncope

Synopsis

- ◆ Sometimes, apparently healthy young adults have cardiac conditions that carry a risk of sudden death.
- ◆ Beyond the age of 35 years, coronary artery disease is the commonest cause of sudden cardiac death, and it is relatively common even in younger people.
- ◆ Other causes include hypertrophic cardiomyopathy, arrhythmic syndromes, concussion of the heart, aortic dissection, viral myocarditis and congenital aortic valve stenosis.
- ◆ Screening tests may not reveal asymptomatic cardiac conditions, but a family history of sudden deaths or cardiac disease will often provide the diagnostic clue.
- ◆ Identifying asymptomatic cardiac disease may lead to treatment and to exclusion from military service and other strenuous activities that may be associated with sudden cardiac death.

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and dyspnoea. Some people with this condition, however, remain asymptomatic.

Hypertrophic cardiomyopathy is frequently reported to be the commonest cause of sudden unexpected cardiac death in young adults.⁴ Although a number of young people with this condition die suddenly, in some studies it was an uncommon cause of death,⁵ possibly because subjects were identified by screening programs and subsequently excluded from undertaking vigorous physical activity. It is also possible that the importance of hypertrophic cardiomyopathy as a cause of sudden unexpected cardiac death has been overstated by repeated reporting of the same cases in the literature.

The thickening of the interventricular septum that occurs with hypertrophic cardiomyopathy causes a dynamic obstruction across the left ventricular outflow tract. The severity of the gradient across the outflow tract is related to symptoms, but not to the patient’s prognosis. Factors linked to a poor prognosis for patients with hypertrophic cardiomyopathy are a family history of sudden unexpected death, symptoms of syncope, and the presence of ventricular tachycardia found on Holter monitoring (continuous electrocardiographic monitoring). There are several genotypes of this disorder, and if several family members are affected then a subject’s genotype can be mapped and an estimate of prognosis can be made.⁶ Some individuals with hypertrophic cardiomyopathy have extensive myocardial fibre disarray on histological examina-



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I Causes of sudden cardiac death in young adults (age \leq 35 years)

Hypertrophic cardiomyopathy	Coronary artery disease
Arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD)	Coronary artery anomalies
Ventricular pre-excitation syndromes	Long QT syndromes
Concussion of the heart (commotio cordis)	Brugada syndrome
	Viral myocarditis
	Congenital aortic valve stenosis
	Aortic dissection (Marfan syndrome)

tion but little in the way of hypertrophy, and yet these patients may be at high risk of sudden death. Certain genotypes have delayed phenotypic expression, with some patients being in their 60s before exhibiting structural change in the heart.⁷

There is no definite evidence that treatment of the condition alters prognosis, but symptoms are usually managed with β -blockers. Verapamil has also been used to treat symptoms and is beneficial in non-obstructive forms of the condition because of its negative inotropic effect, which results in left ventricular dilatation and a decrease in left ventricular pressure. There have been problems with the use of verapamil for patients with the obstructive form of hypertrophic cardiomyopathy: the vasodilatory properties of verapamil may override the negative inotropic effect, resulting in systemic vasodilatation with an increase in the gradient. The use of this drug has been associated with sudden death in some instances.^{8,9} For severely symptomatic patients, surgical myomectomy, sometimes coupled with mitral valve replacement, or, more recently, septal reduction by alcohol ablation,¹⁰ can be performed.

Coronary artery anomalies

The ostia of the coronary arteries are normally situated in the right and left coronary sinuses of the aortic root. Among congenital cardiac lesions, anomalous origin of a coronary artery is relatively common but those anomalies which carry an increased risk of sudden death are fortunately rare. For certain specific anatomical variants the risk of sudden unexpected cardiac death may be high.^{11,12}

High risk anatomical variants include origin of the left coronary artery from the pulmonary artery and origin of the left coronary artery from the right coronary or non-coronary sinus of the aortic root where the vessel courses between the aortic and pulmonary artery roots. A right coronary artery arising from the left coronary sinus was thought to carry less risk of sudden unexpected cardiac death, but it is now known that this condition can also cause angina, syncope, ventricular fibrillation and occasionally sudden death.¹³

Patients with coronary anomalies may die suddenly. If they present with symptoms and the abnormality is found then surgical correction may be possible.

Arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD)

This is a disease of unknown cause which can present as a cardiac arrhythmia, sudden death and, rarely, cardiac failure (as the left ventricle is usually spared at least until late in the disease process). Basso et al suggested that ARVD may be a form of myocarditis,¹⁴ as lymphocytic infiltrates can be found within the affected myocardial tissue. They also concluded that the term cardiomyopathy should replace dysplasia, as there seems to be a progressive damage/repair process rather than a congenital abnormality.

There is, however, evidence that in some cases ARVD may be a genetically determined disorder;¹⁵ a viral or environmental agent may possibly act in a genetically predisposed individual to cause the condition.

Typically, a young patient will present with syncope and be found to have ventricular tachycardia. Those who die suddenly are found at autopsy to have a variable amount of the right ventricular myocardium replaced by fat.

The diagnosis of ARVD is often difficult. Physical examination is frequently normal, but in some patients a widely split second heart sound may be heard as a result of right ventricular dysfunction. The resting electrocardiogram is abnormal in 70% of cases. T wave inversion in the chest leads is the commonest finding.¹⁶ The presence of a so-called epsilon wave, a characteristic notch in the secondary R wave of the right precordial leads, is, if present, said to be almost pathognomonic of ARVD.¹⁷ The notch is the result of prolongation of the QRS complex as a consequence of slow conduction through the diseased right ventricular free wall. There may be anatomical changes in the right ventricle, including right ventricular enlargement, multiple outpouchings and right ventricular dysfunction, but minor changes are easily missed on echocardiography. Cine magnetic resonance imaging is the most useful test for establishing the anatomical diagnosis, as this may best demonstrate the subepicardial infiltration of fat.

Endomyocardial biopsy will be abnormal if affected areas are sampled, but the disease process is often patchy and usually affects the right ventricular free wall first, sparing the septum, the usual site for taking biopsy specimens.

Programmed electrical stimulation is indicated for ARVD, particularly as catheter ablation techniques to prevent ventricular tachycardia¹⁸ have proven useful in the treatment of some patients.

Treatment with antiarrhythmic drugs, particularly sotalol, has been tried with variable success.¹⁹ An automatic implantable cardioverter defibrillator is often the most appropriate therapy,²⁰ but given that the right ventricular wall is often very thin, implantation of defibrillation leads in this chamber may be ineffective. Consideration may need to be given to positioning the leads in the left ventricle, and surgery (myocardial resection; cryosurgical ablation of the arrhythmogenic area; transplantation) has been performed in refractory situations.

With increasing awareness of this condition it may appear

that ARVD is more prevalent than previously thought. Some patients who have endstage dilated cardiomyopathy are found to have biopsy evidence of ARVD.

Long QT syndromes

These are inherited disorders of the sodium and potassium ion channel genes of the myocardium that prolong the QT interval on the resting electrocardiogram (QT interval corrected for heart rate [QTc] ≥ 0.48 second in females and ≥ 0.45 second in males) and produce T wave abnormalities, particularly T wave alternans. Individuals with this disorder frequently develop a characteristic ventricular tachycardia called torsade de pointes which may cause syncope or cardiac arrest (Box 2).

At least six genotypes with over 120 gene mutations have been associated with long QT syndromes, but as yet no specific gene for the syndrome has been determined.¹³ Syncope occurs in about two-thirds of gene carriers, with sudden death in 10%–15% of untreated patients.²¹

As well as the inherited forms of the condition, certain individuals can acquire long QT syndrome if they are exposed to some metabolic abnormalities, for example hypokalaemia, or to certain drugs.

There is an increased risk of drug-induced torsade de pointes in women who have had recent bradycardia or hypokalaemia.²² Susceptible individuals may have a mutation with a “silent” form of long QT syndrome, where the patient remains asymptomatic until exposed to certain conditions or drugs which further impair repolarisation. A normal QTc therefore does not exclude long QT syndrome, and up to 12% of affected individuals may have normal electrocardiograms.²¹

The principal treatment for this congenital condition is high dose β -blocker therapy. Sodium channel blockers such as procainamide and flecainide or potassium administration, depending on the particular ion channel defect, have been used, as well as pacing and automatic implantable cardioverter defibrillators. Treatment is indicated for all symptomatic patients as well as asymptomatic children and young adults (below 40 at diagnosis). Patients older than 40–45 years, if asymptomatic, may not need to be treated; their risk of sudden unexpected cardiac death is low but not zero. Clearly, precipitating agents must be avoided for all patients with congenital long QT syndrome.

Brugada syndrome

This was originally described by Pedrou and Josep Brugada in 1991 in eight otherwise healthy patients with sudden and aborted cardiac death who had “right bundle branch block and persistent ST elevation in V1–V3”.²³ The QT intervals were normal in these patients; it has been found subsequently that right bundle branch block is not an integral part of the syndrome.²⁴ ST segment elevation in the right precordial leads in the absence of ischaemia, electrolyte or metabolic disorders, pulmonary, inflammatory or nervous system disease may identify subjects with Brugada syndrome. Idiopathic ST segment elevation occurs in about 2.5% of individuals, but is confined to the right precordial leads in less than 1% of all cases of ST segment elevation. In this syndrome the ST elevation is characteristically downsloping and not accompanied by reciprocal ST depression.

Brugada syndrome appears to be a familial primary electrical disease caused by a defect in an ion channel gene. A particularly strong link has been found between Brugada syndrome and sudden unexpected death, particularly in South East Asian males.²⁵ Death often occurs during sleep and autopsy findings are usually negative. The syndrome is complicated because the electrocardiographic manifestations of Brugada syndrome may transiently normalise, leading to underdiagnosis. Provocation with sodium channel blockers (procainamide and flecainide) can unmask the ECG abnormality.

The syndrome is consistent with autosomal dominant inheritance with variable expression (similar to ARVD), but the relationship of Brugada syndrome to ARVD is unclear.²⁴ There is a high incidence of familial clustering.

In patients with Brugada syndrome, programmed electrical stimulation almost always induces malignant ventricular tachycardia or ventricular fibrillation with aborted sudden death or syncope. Implantable cardiac defibrillators are the only effective treatment and are indicated for symptomatic individuals with the syndrome and for those who are usually asymptomatic if malignant arrhythmias are induced by electrophysiological studies.²⁴

Ventricular pre-excitation syndromes

Many young people have episodes of paroxysmal supraventricular tachycardia, for which the underlying anatomical sub-

2 Torsade de pointes



3 Medications that may prolong the QT interval

Generic name	Brand name	Generic name	Brand name	Generic name	Brand name
Antiarrhythmic drugs		Tricyclic antidepressants		Antimalarials and antiprotozoals	
quinidine	Kinidin	amitriptyline	Amitrol, Endep, Tryptanol	chloroquine	Chlorquin
procainamide	Pronestyl	clomipramine	Anafranil, Placil	halofantrine	
disopyramide	Norpace, Rythmodan	desipramine	Pertofran	mefloquine	Lariam
amiodarone	Aratac, Cordarone X	dothiepin	Dothep, Prothiaden	pentamidine	
sotalol	Cardol, Sotacor	doxepin	Deptran, Sinequan	quinine	Quinate, Biquinate, Myoquin, Quinsul, Quinocotal, Quinbisul
bretylum		imipramine	Tofranil, Melipramine		
Antibiotics		nortriptyline	Allegron	Gastrointestinal drugs	
erythromycin	Eryc, Emu-V, Illosone, EES, E-Mycin, Erythrocin	trimipramine	Surmontil	cisapride	Prepulsid
clarithromycin	Klacid	Antipsychotic drugs		Miscellaneous	
trimethoprim-sulfamethoxazole	Bactrim, Septrin, Resprim	thioridazine	Aldazine, Melleril	amantadine	Symmetrel
Antihistamines		chlorpromazine	Largactil	probutal	Lurselle
astemizole	Hismanal	pimozide	Orap	tacrolimus	Prograf
terfenadine	Teldane	haloperidol	Serenace	vasopressin	Pitressin
		lithium	Lithicarb	Illicit drugs	
		chloral hydrate		cocaine	

strate is usually an accessory conduction pathway between the atria and the ventricles. These so called pre-excitation syndromes are a heterogeneous group of disorders. The best known is the Wolff–Parkinson–White syndrome. When patients with this condition present with rapid supraventricular tachycardia, conduction usually proceeds down the normal His–Purkinje conduction system antegradely and then returns to the atrium retrogradely via the accessory pathway to complete the circuit. In some patients the accessory pathway is also capable of conducting antegradely from atria to ventricle, sometimes at very rapid rates.

Patients with Wolff–Parkinson–White syndrome have a propensity to develop atrial fibrillation and, in the event of pre-excited atrial fibrillation (with conduction to the ventricle via the accessory pathway), they may develop very rapid ventricular rates and (rarely) ventricular fibrillation.

Concussion of the heart (commotio cordis)

There have been several case reports where a blow to the precordial area, often without undue force, has resulted in the sudden death of the recipient. Hockey, baseball and lacrosse players are particularly susceptible to such injuries. Link et al demonstrated in a swine model that a blow to the chest wall which coincides with the T wave results in ventricular fibrillation 90% of the time.²⁶ If the blow falls on the QRS complex, heart block or asystole occurs 30% of the time. A

4 ECG tracing from a patient with Brugada syndrome



blow timed elsewhere in the cardiac cycle causes ST segment elevation on the subsequent ECG complex; the significance of this is unclear.

If ventricular fibrillation occurs and lasts for more than four minutes without defibrillation, successful resuscitation is unlikely. This raises the question as to whether defibrillators should be available at large public gatherings and, if so, who should be trained in their use.

Aortic dissection: Marfan syndrome

Marfan syndrome is an autosomal dominant generalised abnormality of connective tissue, but there is variable phenotypic expression in subsequent generations.

The archetypal marfanoid patient, a tall, thin, shortsighted basketballer with aortic regurgitation, would be easily recognised. It is important to remember, however, that this is a generalised disorder of connective tissue affecting many body systems apart from the heart. Within the heart mitral regurgitation may be more common than aortic regurgitation.²⁷

In one study of 257 patients followed for 30 years, 72 patients died at an average age of 32 years, and in 80% of these the cause of death was aortic dissection or rupture.²⁸

In view of the risk of aortic dissection, subjects with Marfan syndrome should receive close follow up, including serial echocardiography. Medical treatment consists of vigorous control of hypertension, usually with β -blockers. Surgical treatment of the dilated aortic root is recommended, even in asymptomatic individuals with Marfan syndrome, as the risk of dissection increases significantly as aortic diameter increases.

Current guidelines for surgery are (1) aortic root diameter >55 mm, or (2) positive family history of aortic dissection and aortic root diameter >50 mm or (3) aortic root growth >2 mm/year.²⁹

Myocarditis

Myocarditis, or inflammation of the myocardium, can be caused by any one of a heterogeneous group of conditions and may be an acute or chronic process. The end stage of the inflammatory process is thought to result in severe global impairment of cardiac contraction, but some individuals who die suddenly with no evidence of any other cause are found to have histological evidence of myocarditis at autopsy.

Congenital abnormalities of the left ventricular outflow tract (LVOT)

The most common site of stenosis is at the aortic valve level, but supravalvular and infravalvular stenoses are well recognised. The major symptoms of congenital LVOT obstruction are angina, syncope and symptoms related to congestive cardiac failure. Many patients with significant degrees of stenosis may remain asymptomatic. Sudden death, usually during

exercise, may occur, but in previously asymptomatic individuals with aortic stenosis this is rare.³⁰

Screening

Fortunately, sudden cardiac death in apparently normal young adults is an uncommon event but always seems a particularly poignant tragedy which, in developed countries at least, usually receives considerable publicity. Screening asymptomatic young adults for potential causes of sudden death remains controversial; it is performed before individuals are enrolled in the Armed Forces and certain other occupations. Several sporting codes also require medical screening before participation.

Many of the causes of sudden unexpected death discussed in this article do not cause abnormal physical findings and the subjects may be asymptomatic. Screening should pay particular attention to the subject's family history, particularly if there are any relatives who have experienced sudden unexpected death. A resting electrocardiogram can reasonably be expected to be a cost effective screening tool, but routine echocardiography for individuals with no symptoms and no physical findings is not recommended.

It remains to be proven that appropriate screening of children and young adults to identify potential cardiac disorders will result in a reduction of sudden cardiac deaths, but experience from screening programs undertaken in Italy is encouraging.⁵

Wider availability of cardiac defibrillators in airport lounges, on aircraft and at sites of large public gatherings may save individuals who develop malignant cardiac arrhythmias and who would otherwise almost certainly die. The great majority of these individuals would not be young and would have underlying coronary disease.

Recent progress in understanding the molecular basis for cardiac repolarisation will aid in further understanding of arrhythmias and help identify new therapies. This progress may extend to a better understanding of the causes of sudden death, not only in young and apparently healthy adults but also in patients with heart failure, cardiac hypertrophy and myocardial infarction, and even in sudden infant death syndrome, where an association with abnormal repolarisation has been suggested.³¹

References

1. Stary HC. Evolution of atherosclerotic plaques in the coronary arteries of young adults [abstract]. *Arteriosclerosis* 1983; 3: 471A.
2. Maron BJ, Gardin JM, Flack JM, et al. Prevalence of hypertrophic cardiomyopathy in a general population of young adults: echocardiographic analysis of 4111 subjects in the CARDIA Study. *Circulation* 1995; 92: 785-789.
3. Spirito P, Seidman C, McKenna W, Maron BJ. The management of hypertrophic cardiomyopathy. *N Engl J Med* 1997; 336: 775-785.
4. Maron BJ, Shirani J, Poliac LC, et al. Sudden death in young competitive athletes. Clinical, demographic, and pathological profiles. *JAMA* 1996; 276: 199-204.
5. Thiene G, Basso C, Corrado D. Is prevention of sudden death in young athletes feasible? *Cardiologia* 1999; 44: 497-505.
6. Bonne G, Carrier L, Richard P, et al. Familial hypertrophic cardiomyopathy — from mutations to functional defects. *Circulation Res* 1998; 83: 580-593.

7. Mimura H, Bachinski LL, Sangwatanaroj S, Watkins H, et al. Mutations in the gene for cardiac myosin-binding protein C and late-onset familial hypertrophic cardiomyopathy. *N Engl J Med* 1998; 338: 1248-1257.
8. Epstein SE, Rosing DR. Verapamil: its potential for causing serious complications in patients with hypertrophic cardiomyopathy. *Circulation* 1981; 64: 437-441.
9. Perrot B, Canchin N, Terrier de la Chaise A. Verapamil: a cause of sudden death in a patient with hypertrophic cardiomyopathy. *Br Heart J* 1984; 51: 352-354.
10. Lakkis NM, Nagueh SF, Kleiman NS, et al. Echocardiography-guided ethanol septal reduction for hypertrophic obstructive cardiomyopathy. *Circulation* 1998; 98: 1750-1755.
11. Wesselhoeft H, Fawcett JS, Johnson AL. Anomalous origin of the left coronary artery from the pulmonary trunk. *Circulation* 1968; 38: 403-425.
12. Levin DC, Fellows KE, Abrams HL. Hemodynamically significant primary anomalies of the coronary arteries. Angiographic aspects. *Circulation* 1978; 58: 25-34.
13. Roberts WC. Major anomalies of coronary arterial origins seen in adulthood. *Am Heart J* 1986; 111: 941-963.
14. Basso C, Thiene G, Corrado D, et al. Arrhythmogenic right ventricular cardiomyopathy. Dysplasia, dystrophy or myocarditis? *Circulation* 1996; 94: 983-991.
15. Nava A, Thiene G, Canciani B, et al. Familial occurrence of right ventricular dysplasia: a study involving nine families. *J Am Coll Cardiol* 1998; 12: 1222-1228.
16. Laurenceau JL, Lienhart JF, Malergue MC, et al. Echocardiographics dans le syndrome ventricule droit papayrace. *Arch Mal Coeur Viass* 1979; 72: 258-262.
17. Ananthasubramaniam K, Khaha F. Arrhythmogenic right ventricular dysplasias/cardiomyopathy: review for the clinician. *Prog Cardiovasc Dis* 1998; 41: 237-246.
18. Fontaine G, Frank R, Rougier I, et al. Electrode catheter ablation of resistant ventricular tachycardia in arrhythmogenic right ventricular dysplasia: experience of thirteen patients with a mean follow up of 45 months. *Eur Heart J* 1989; 10 Suppl D: 74-81.
19. Leclercq J-F, Coumel P. Characteristics, prognosis and treatment of ventricular arrhythmias and right ventricular dysplasia. *Eur Heart J* 1989; 10 Suppl D: 61-67.
20. Trappe H, Klein H, Fieguth H, et al. Clinical efficacy and safety of the new cardioverter defibrillator systems. *PACE* 1993; 16: 153-158.
21. Vincent GM, Timothy K, Fox J, Zhang L. The inherited long QT syndrome: from ion channel to bedside. *Cardiol Rev* 1999; 7: 44-55.
22. Benton RE, Sale M, Flockhart DA, Woosley RL. Greater quinidine-induced QTc interval prolongation in women. *Clin Pharmacol Ther* 2000; 67: 413-418.
23. Brugada P, Brugada J. A distinct clinical and electrocardiographic syndrome: right bundle branch block, persistent ST segment elevation with normal QT interval and sudden cardiac death [abstract]. *Pacing Clin Electrophysiol* 1991; 14: 746.
24. Gussak I, Antzelevitch C, Bjerregaarde P, et al. The Brugada syndrome: clinical electrophysiologic and genetic aspects. *J Am Coll Cardiol* 1999; 33: 5-15.
25. Nademanee K, Veerakul G, Nimmannit S, et al. Arrhythmogenic marker for the sudden unexplained death syndrome in Thai men. *Circulation* 1997; 96: 2595-2600.
26. Link MS, Wang PJ, Pandian NG, et al. An experimental model of sudden death due to low energy chest-wall impact (commotio cordis). *N Engl J Med* 1998; 338: 1805-1811.
27. Phomphutkul C, Rosenthal A, Nadas A. Cardiac manifestation of Marfan syndrome in infancy and childhood. *Circulation* 1973; 47: 587-596.
28. Murdoch JL, Walker BA, Halpern BI, et al. Life expectancy and causes of death in the Marfan syndrome. *N Engl J Med* 1972; 286: 804-808.
29. Groenink M, Lohuis TAJ, Tijssen JGP, et al. Survival and complication free survival in Marfan's syndrome: implications of current guidelines. *Heart* 1999; 82: 499-504.
30. Hossack KF, Heutze JM, Lowe JB, Barratt-Boyes BG. Congenital valvular aortic stenosis. Natural history and assessment of operation. *Br Heart J* 1980; 43: 561-573.
31. Roden DM, Spooner PM. Inherited long QT syndromes: a paradigm for understanding arrhythmogenesis. *J Cardiovasc Electrophysiol* 1999; 10: 1664-1683. □

Behind the scenes

The DHS–DVA Medical Advisory Panel

SINCE FEDERATION, IT has been the policy of the Australian nation to provide health care for service men and women who have been on operational deployments both during and after their period of service. Health care for service personnel is supplied by the Defence Health Service; after their return to civilian life, veterans are cared for by the Department of Veterans' Affairs.

The Defence Health Service–Department of Veterans' Affairs Medical Advisory Panel meets twice a year. Chaired by the Surgeon General, ADF, the Advisory Panel brings together the senior decision makers and advisors from the Defence Health Service, the health care and services branch of the Department of Veterans' Affairs and the National Repatriation Medical Authority. The Authority is an independent statutory authority established by the Commonwealth Government to advise on health matters relating to Australia's past military deployments.

The objectives of the Advisory Panel's work are to facilitate a seamless transition of health care from uniformed service to post-service civilian life and to maximise the health of veterans. The Advisory Panel considers operational-specific diseases and disorders, and works towards the care of future veterans by providing proactive advice in relation to current operational deployments.



The Advisory Panel meeting on 26 May 2000. Front row (l–r): Colonel Robert Millar; Dr Graeme Killer, AO; Major General John Pearn, Surgeon General ADF; Mr Geoffrey Stonehouse, Head, Division of Healthcare, Department of Veterans' Affairs.