apply, often to a lesser extent, across the range of patients with epilepsy. As we improve our services for this vulnerable group, the lessons learnt can only benefit people with epilepsy as a whole.

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1 Appleton RE, Chadwick DW, Sweeny A. Managing the teenager with epilepsy: paediatric to adult care. *Seizure* 1997;6:27-30.
2 Besag FM. Modern management of epilepsy: adolescents. *Baillière's Clin Neurol* 1996;5:803-20.
3 Timmings PL, Richens A. Juvenile myoclonic epilepsy. *BMJ* 1992;305:4-5.
4 Kurtz Z, Tookey P, Ross E. Epilepsy in young people: 23 year follow up of the British national child development study. *BMJ* 1998;316:339-42.
5 Ventureyra EC, Higgins MJ. Complications of epilepsy surgery in children and adolescents. *Pediatr Neurosurg* 1993;19:40-56.
6 Biale Y, Lewanthal M. Effect of folic acid supplementation on congenital malformations due to anticonvulsant drugs. *Eur J Obstet Gynaecol Reprod Biol* 1984;18:211-6.
7 Elmazar MMA, Thiel R, Nau H. Effect of supplementation with folinic acid, vitamin $B_6$ and vitamin $B_{12}$ on valproic acid induced teratogenesis in mice. *Fundamental and Applied Toxicology* 1992;18:389-94.
8 Preston J. *Epilepsy and the young adult*. London: Epilepsy and the Young Adult,1996.
9 Thompson P, Oxley J. Social aspects of epilepsy. In: Laidlaw J, Richens A, Chadwick DW, eds. *A textbook of epilepsy*. 4th ed. Edinburgh: Churchill Livingstone, 1993:661-704.

# Dying from heart failure: lessons from palliative care

*Many patients would benefit from palliative care at the end of their lives*

Increasing interest and research into the care of the dying over the past 25 years have resulted in better symptom control, psychological support, and choice for people dying from cancer and their families.[1] Little attention has been paid, however, to patients with other life threatening diseases, such as AIDS, neurological conditions, respiratory failure, and heart failure. Palliative care, with its emphasis on the care of patients whose prognosis is limited, on quality (not quantity) of life, and on a multidisciplinary approach, may benefit patients other than those with cancer. One such group is patients dying from heart failure.

Heart failure is the only major cardiovascular disease with increasing prevalence, incidence, and mortality. Incidence and prevalence both increase dramatically over the age of 75 years—up to 43.5 and 190 per 1000 population respectively.[2] With age adjusted mortality from cardiovascular disease declining and the size of the elderly population growing, the absolute number of individuals living with compromised cardiac function is expected to increase dramatically over the next few decades.[3] Modern treatments for heart failure slow but do not arrest progression of the disease. Despite the wealth of therapeutic advances, quality of life in chronic heart failure is poor[4] and discomfort and distress often worse than in cancer.[5]

In the United Kingdom only one study has investigated symptoms in terminal heart disease: the regional study of care for the dying.[6] This was a population based retrospective survey of a random sample of people dying in 20 English health districts in 1990. People who died from heart disease, including heart failure, had experienced a wide range of symptoms, often distressing and often lasting more than six months.[7] In addition to dyspnoea, pain, nausea, constipation, and low mood were common and poorly controlled. At least one in six had symptoms as severe as those in patients with cancer managed in hospices or by palliative care services. Although many were thought to have known that they were dying, open communication with health professionals was rare.[8]

In the United States the SUPPORT study included 263 patients with heart failure.[9] It showed severe symptoms in the last three days of life in patients with heart failure: 65% were breathless and 42% had severe pain. Forty percent of these patients received a major treatment intervention in the last three days of life, suggesting that doctors had not recognised the closeness of death. A salutary finding was that intervention by specially trained nurses to enhance decision making and improve patient care had no impact on symptom control or other outcome measures.

In heart failure, as in most diseases, the first step towards symptom control is optimisation of treatment of the underlying disease. The regional study of care of the dying suggests that this is not enough. The need for improved symptom control and greater emphasis on quality of life has been recognised,[10 11] but research into and provision of services for care of patients with end stage heart disease have been neglected.[12] Nurse practitioners have been advocated to help with patient management and may be effective.[12] Several trials are underway, but these may be premature since the needs of these patients have not been defined. Cost effective, appropriate, and acceptable services for these patients cannot be developed in the absence of good information on what their needs are and when to intervene to improve their lives.

The findings of the SUPPORT study suggest that the use of resources for the care of patients with heart failure will need to be imaginative to be effective. Anecdotal evidence exists that palliative care teams have managed patients with heart failure successfully using the same approach that helps cancer sufferers, but conventional hospice and specialist palliative care services could be overwhelmed by heart disease. Indeed, different models of care may be needed since patients with heart failure are more prone to sudden death than patients with cancer and do not necessarily have a clearly defined terminal phase. Specialist heart failure nurses may founder if they work in isolation. Palliative care is recognising the need to take stock of other terminal illnesses. Now is the time to collaborate and accelerate this change.

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1 Higginson I. Palliative care: a review of past changes and future trends. *J Pub Health Med* 1993;15:3-8.
2 Cowie MR, Mosterd A, Wood DA, Deckers JW, Poole-Wilson PA, Sutton GC, et al. The epidemiology of heart failure. *Eur Heart J* 1997;18:208-25.
3 Bonneaux L, Barendregt JJ, Meeter K, Bonsel GJ, van der Maas PJ. Estimating clinical morbidity due to ischemic heart disease and congestive heart failure: the future rise of heart failure. *Am J Pub Health* 1994;84:20-8.
4 Stewart AL, Greenfled S. Hays RD, Wells K, Rogers WH, Berry SD, et al. Functional status and well-being of patients with chronic conditions. Results from the medical outcomes study. *JAMA* 1989;262:907-13.
5 Hinton JM. The physical and mental stress of dying. *Q J Med* 1963;32: 1-21.
6 Addington-Hall JM, McCarthy M. Regional Study of Care for the Dying: methods and sample characteristics. *Palliative Med* 1995;9:27-35 .
7 McCarthy M, Lay M, Addington-Hall JM. Dying from heart disease. *J R Coll Physicians* 1996;30:325-8.
8 McCarthy M, Addington-Hall JM, Lay M. Communication and choice in dying from heart disease. *J R Soc Med* 1997;90:128-31.
9 Lynn J, Teno JM, Phillips RS, Wu AW, Desbiens N, Harrold J, et al. Perceptions by family members of the dying experience of older and seriously ill patients. *Ann Intern Med* 1997;126:97-106.
10 Dargie HJ, McMurray JJV. Diagnosis and management of heart failure. *BMJ* 1994;308:321-8.
11 Linden B. Severe heart failure: a focus on the quality of care. *Nursing Times* 1995;91:38-9.
12 Rich MW, Beckham V, Wittenberg C, Leven CL, Freedland KE, Carney RM. A multidisciplinary intervention to prevent the readmission of elderly patients with congestive heart failure. *N Engl J Med* 1995;333:1213-4.


# Smoking and stroke: a causative role

*Heavy smokers with hypertension benefit most from stopping*

Stroke remains the third leading cause of death in most Western countries and is second only to myocardial infarction as a cause of cardiovascular death. Many epidemiological studies have established cigarette smoking as an important risk factor for stroke. Until recently, however, this relation was based on observational studies and the effects of smoking were thought to be synergistic with hypertension, diabetes mellitus, glucose intolerance, age, hypercholesterolaemia, and pre-existing cardiovascular disease.[1] Now we have definite evidence that smoking itself has a direct causal effect on stroke.

The relation between smoking and atherosclerosis was observed as early as 1908 by Buerger, who noted severe distal ischaemia among young male addicted smokers.[2] The earliest report associating stroke and extracranial arterial disease is credited to Gowers, who in 1875 showed left carotid artery occlusion in a patient with right hemiplegia and loss of sight in the left eye.[3]

The Framingham Heart Study was among the first to assess these the relation of smoking to type of stroke, number of cigarettes smoked, and the effect of stopping.[4] It concluded that smoking made a significant independent contribution to the risk of stroke generally and to brain infarction specifically. The relative risk of stroke in heavy smokers (>40 cigarettes/day) was twice that of light smokers (<10 cigarettes/day), and the risk of stroke increased with the number of cigarettes smoked; cessation lowered the relative risk ratio to that of a non-smoker.[4] This reduction in risk ratio was significant by two years after stopping and had reached the level of a non-smoker at five years.[4] In a meta-analysis of 32 separate studies, Shinton and Beevers showed that cigarette smoking independently contributed to the incidence of stroke: the greatest risk was of subarachnoid haemorrhage, followed by cerebral infarction.[5]

Heavy smokers have a relative risk of stroke 2-4 times greater than non-smokers.[1 4] The large cohort study of 22 071 US male physicians showed that heavy smokers (>20 cigarettes/day) had a relative risk of total non-fatal stroke of 2.71 and of fatal stroke of 1.46 ($P<0.05$).[6] The British Regional Heart Study showed a relative risk of of 3.7 in all current smokers.[1]

Howard et al showed increased thickness of the intima-media wall of the carotid artery in smokers compared with non-smokers, particularly among people aged over 60.[7] Differences in mean maximum intima-media wall thickness in the internal carotid artery between current and non-smokers were greater than the change expected over 10 years for a person who has never smoked.[8]

The association between the number of cigarettes smoked and the increase in the risk of stroke remains inconclusive. Some authors suggest a linear relation, particularly in smokers of more than 20 cigarettes a day and older people.[4 6 7–9] A dose-response relation between pack years of smoking and carotid artery disease, measured by increased carotid artery intima-media wall thickness, has been shown.[7 8] An association may exist between passive smoking and intimal hyperplasia, potentially increasing the risk of stroke.[7 10] Although the dose-response relation is unclear, stopping smoking does reduce the incidence of stroke. Both the Framingham Heart Study[4] and the Nurses Health Study[11] showed a normalised risk ratio five years after cessation. Also after five years there was no further benefit.[1] Tell et al, however, showed that risk reduction was dependent on the quantity of cigarettes smoked before stopping: light smokers (<20 cigarettes/day) reverted to normal values, but heavy smokers retained twice the incidence of stroke as non-smokers.[8] Secondary pipe or cigar smokers still have an increased risk similar to that of light smokers,[1] so switching to a pipe or cigars confers little benefit.

Former smokers have a decreased prevalence of clinically significant (>50%) internal carotid artery stenosis (7.3%) than current smokers (9.5%),[8] this difference being greatest in older people.[7] No significant relation exists between carotid artery wall thickness and years since quitting smoking.

The relative risk of stroke among hypertensive smokers is five times that among normotensive smokers, but 20 times that of normotensive non-smokers. Pharmacological treatment of hypertension in mildly hypertensive smokers is much less effective in reducing the incidence of smoke than in mildly hypertensive non-smokers, supporting smoking as a causal agent.[12]

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