CORA Cork Open Research Archive
Cartlann Taighde Oscailte Chorcaí

| | |
|---|---|
| Title | Creative consulting: more about placebos? |
| Authors | Reilly, David;Di Blasi, Zelda |
| Publication date | 2002-03 |
| Original Citation | Reilly, D., Di Blasi, Z., 2002. Creative consulting: more about placebos? Student BMJ, 10, pp.61-63 |
| Type of publication | Article (peer-reviewed) |
| Link to publisher's version | http://archive.student.bmj.com/issues/02/03/education/61.php |
| Rights | ©BMJ Publishing Group Limited. |
| Download date | 2024-04-19 16:02:06 |
| Item downloaded from | https://hdl.handle.net/10468/276 |

UCC
University College Cork, Ireland
Coláiste na hOllscoile Corcaigh

# Dead cert: a guide to death certificates

Death is a daunting thing. And to make it worse, certifying someone dead can be terrifying, as Jean Adams found out (see p 86). **Sabina Dosani** gives a step by step guide to one of those skills you just never seem to be taught at medical school

A death certificate, or more correctly, a certificate for registration of death, is the document used to register death. Without one, funerals cannot go ahead. Filling out your first death certificate can be a daunting task. It is important to learn how to do it correctly as errors may result in a delayed funeral and cause further distress to

MED A 909122

BIRTHS AND DEATHS REGISTRATION ACT 1953

MEDICAL CERTIFICATE OF CAUSE OF DEATH

Name of deceased

Date of death as stated to me ..... day of ..... Age as stated to me

Place of death

Last seen alive by me ..... day of

1 The certified cause of death takes account of information obtained from post-mortem.
2 Information from post-mortem may be available later.
3 Post-mortem not being held.
4 I have reported this death to the Coroner for further action.

a Seen after death by me
b Seen after death by another medical practitioner but not by me.
c Not seen after death by a medical practitioner.

CAUSE OF DEATH

The condition thought to be the Underlying Cause of Death should appear on the lowest completed line of Part I.

Approximate interval between onset and death

The death might have been due to or contributed to by the employment followed at some time by the deceased. ☐ Please tick where applicable

*This does not mean the mode of dying, such as heart failure, asphyxia, asthenia, etc. it means the disease, injury, or complication which caused death.

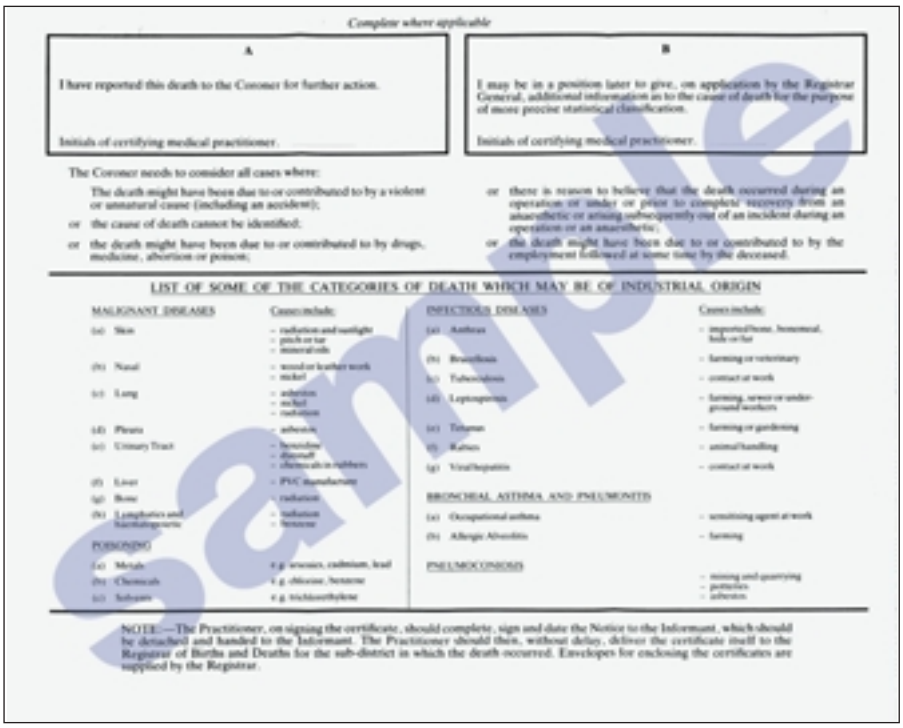
I hereby certify that I was in medical attendance during the above named deceased's last illness, and that the particulars and cause of death above written are true to the best of my knowledge and belief.

Signature ..... Qualifications as registered by General Medical Council

Residence ..... Date

For deaths in hospital: Please give the name of the consultant responsible for the above stated as a patient.

sample



*Complete where applicable*

A

I have reported this death to the Coroner for further action.

Initials of certifying medical practitioner

B

I may be in a position later to give, on application by the Registrar General, additional information as to the cause of death for the purpose of more precise statistical classification.

Initials of certifying medical practitioner

The Coroner needs to consider all cases where:

The death might have been due to or contributed to by a violent or unnatural cause (including an accident);
or the cause of death cannot be identified;
or the death might have been due to or contributed to by drugs, medicine, abortion or poison;
or there is reason to believe that the death occurred during an operation or under or prior to complete recovery from an anaesthetic or arising subsequently out of an incident during an operation or an anaesthetic;
or the death might have been due to or contributed to by the employment followed at some time by the deceased.

LIST OF SOME OF THE CATEGORIES OF DEATH WHICH MAY BE OF INDUSTRIAL ORIGIN

| MALIGNANT DISEASES | Causes include: | INFECTIOUS DISEASES | Causes include: |
|---|---|---|---|
| (a) Skin | – radiation and sunlight<br>– pitch or tar<br>– mineral oils | (a) Anthrax | – imported hides, horsehair, bones or hair |
| (b) Nasal | – wood or leather work<br>– nickel | (b) Brucellosis | – farming or veterinary |
| (c) Lung | – asbestos<br>– nickel<br>– radiation | (c) Tuberculosis | – contact at work |
| (d) Pleura | – asbestos | (d) Leptospirosis | – farming, sewer or underground workers |
| (e) Urinary Tract | – benzidine<br>– dimethyl<br>– chemicals in rubbers | (e) Tetanus | – farming or gardening |
| (f) Liver | – PVC manufacture | (f) Rabies | – animal handling |
| (g) Bone | – radiation | (g) Viral hepatitis | – contact at work |
| (h) Lymphatics and haematopoietic | – radiation<br>– benzene | BRONCHIAL ASTHMA AND PNEUMONITIS | |
| POISONING | | (a) Occupational asthma | – sensitising agent at work |
| (a) Metals | e.g. arsenic, cadmium, lead | (b) Allergic Alveolitis | – farming |
| (b) Chemicals | e.g. chlorine, benzene | PNEUMOCONIOSIS | |
| (c) Solvents | e.g. trichloroethylene | | – mining and quarrying<br>– pottery<br>– asbestos |

NOTE.—The Practitioner, on signing the certificate, should complete, sign and date the Notice to the Informant, which should be detached and handed to the Informant. The Practitioner should then, without delay, deliver the certificate itself to the Registrar of Births and Deaths for the sub-district in which the death occurred. Envelopes for enclosing the certificates are supplied by the Registrar.

sample

(Right)
How a death certificate looks, front (above) and back (below).

**Box 1: Cases which have to reported to the coroner**

- Violent deaths
- Deaths when a doctor has not attended in the previous 14 days
- Cause of death is unknown or uncertain
- Accidental death
- Doubtful stillbirth
- Deaths related to surgery or anaesthetic
- Deaths within 24 hours of admission to hospital

For some deaths, you can issue the certificate for registration but can alert the coroner or other authorities (such as a pension agency) by ticking a box (box A) on the reverse that further action may be required. Examples of this are given in box 2.

bereaved relatives.

A doctor may complete a certificate for registration of a death only if he or she has been in attendance on the deceased during the last illness and has seen the deceased within 14 days of death or after death. If no doctor meets these criteria the coroner is informed in England and Wales. In Scotland, it is the procurator fiscal who has a duty to investigate all sudden, suspicious, accidental, unexplained and unexpected deaths, and any death occurring in circumstances that would give rise to serious public concern.

There are four potential outcomes:

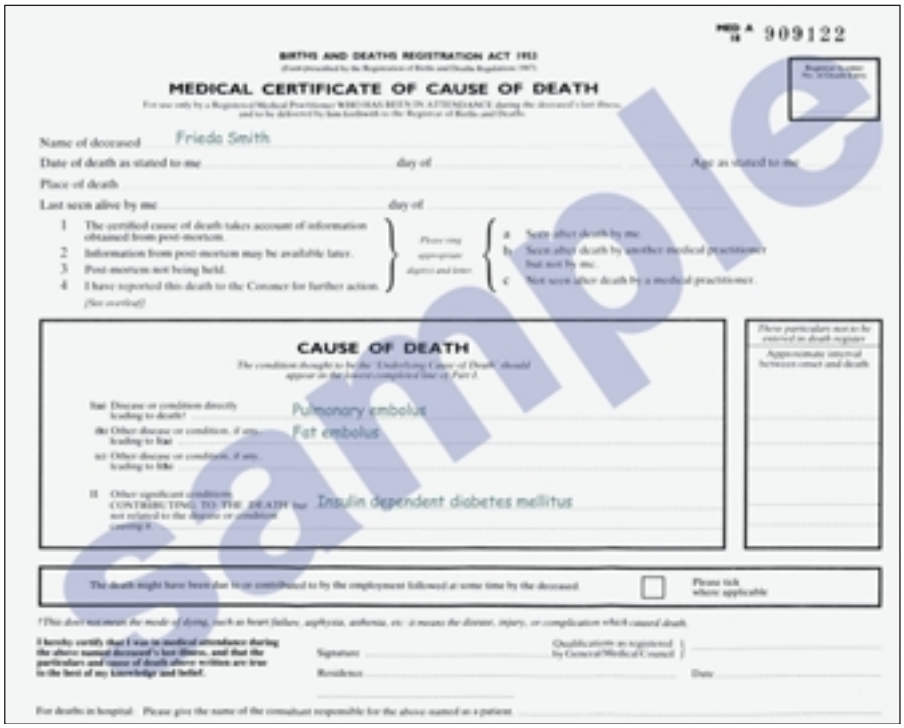
- An uncertified death
- Death certified by a doctor
- Death certified after a postmortem examination without an inquest
- Death certified after an inquest

## Uncertified death is rare

An uncertified death is rare. For instance, a dying patient attended before death by the GP who discussed this with his or her partners and then emigrated to Australia. No doctor fulfilling the criteria to complete the certificate would be available. If such a death is reported to the coroner or procurator fiscal, he or she may allow an uncertified death, without requesting a postmortem examination or inquest. After the Harold Shipman case this is very unlikely.

**Box 2: Cases where further action may be required**

- Death from an industrial disease
- Death of a person who was in receipt of an industrial pension
- Death by suicide
- Death by poisoning or drugs (including alcohol)
- Death as a result of illegal abortion
- Death from want, neglect, or exposure



909122

BIRTHS AND DEATHS REGISTRATION ACT 1953

MEDICAL CERTIFICATE OF CAUSE OF DEATH

Name of deceased Frieda Smith

Date of death as stated to me ..... day of ..... Age as stated to me

Place of death

Last seen alive by me ..... day of

1 The certified cause of death takes account of information obtained from post-mortem.
2 Information from post-mortem may be available later.
3 Post-mortem not being held.
4 I have reported this death to the Coroner for further action.
(See overleaf)

a Seen after death by me
b Seen after death by another medical practitioner but not by me
c Not seen after death by a medical practitioner

CAUSE OF DEATH

The condition thought to be the 'Underlying Cause of Death' should appear in the lowest completed line of Part I.

Pulmonary embolus
Fat embolus
Insulin dependent diabetes mellitus

These particulars are to be entered in death register

Approximate interval between onset and death

The death might have been due to or contributed to by the employment followed at some time by the deceased. Please tick where applicable

†This does not mean the mode of dying, such as heart failure, asphyxia, asthenia, etc. it means the disease, injury, or complication which caused death.

I hereby certify that I was in medical attendance during the above named deceased's last illness, and that the particulars and cause of death above written are true to the best of my knowledge and belief.

Signature ..... Qualifications as registered by General Medical Council

Residence ..... Date

For deaths in hospital: Please give the name of the consultant responsible for the above named as a patient

sample

Example 1

It is worth knowing where the death certificates are kept. Usually this is in a bereavement office, but smaller hospitals may have other arrangements. They are in

**Box 3: Information on the certificate for registration**

- Name of deceased
- Age
- Date of death
- Place of death
- Date last seen by the doctor issuing the certificate for registration
- This doctor can circle one or more of the following statements:
  - (a) This certificate takes account of information obtained from a postmortem examination
  - (b) Information from the postmortem examination may be available later
  - (c) A postmortem examination not being held
  - (d) I have reported this death to the coroner for further action
- There is also an option for one of the following statements:
  - (a) Seen after death by me
  - (b) Seen after death by a another medical practitioner but not by me
  - (c) Not seen after death by a medical practitioner
- Cause of death which is in two parts:
  - (1a) Primary cause of death ……(and duration, but this is not obligatory)
  - (1b) Due to ……(and duration, but this is not obligatory)
  - (1c) Due to ……(and duration, but this is not obligatory)
  - (2) Significant conditions not related to primary cause of death (can be more than one)
- You can tick a box if the death is related to employment. It is important to discuss the implications of this with your seniors as it may influence a widow's pension.
- Signature of doctor
- Date of issue
- Qualifications of doctor
- The name of the consultant responsible for the deceased if they died in hospital

MED A 909122

BIRTHS AND DEATHS REGISTRATION ACT 1953

MEDICAL CERTIFICATE OF CAUSE OF DEATH

Name of deceased Mabel Green

Date of death as stated to me ..... day of ..... Age as stated to me .....

Place of death

Last seen alive by me ..... day of .....

1 The certified cause of death takes account of information obtained from post-mortem.
2 Information from post-mortem may be available later.
3 Post-mortem not being held.
4 I have reported this death to the Coroner for further action.

a Seen after death by me.
b Seen after death by another medical practitioner but not by me.
c Not seen after death by a medical practitioner.

CAUSE OF DEATH

1(a) Disease or condition directly leading to death? Tumour lysis syndrome
(b) Other disease or condition, if any, leading to 1(a) Myeloma
(c) Other disease or condition, if any, leading to 1(b)
II Other significant conditions CONTRIBUTING TO THE DEATH but not related to the disease or condition causing it.

Approximate interval between onset and death

The death might have been due to or contributed to by the employment followed at some time by the deceased. Please tick where applicable

*This does not mean the mode of dying, such as heart failure, asphyxia, asthenia, etc: it means the disease, injury, or complication which caused death.

I hereby certify that I was in medical attendance during the above named deceased's last illness, and that the particulars and cause of death above written are true to the best of my knowledge and belief.

Signature ..... Qualification as registered by General Medical Council .....
Residence ..... Date .....

For deaths in hospital: Please give the name of the consultant responsible for the above named as a patient .....

Example 2

MED A 909122

BIRTHS AND DEATHS REGISTRATION ACT 1953

MEDICAL CERTIFICATE OF CAUSE OF DEATH

Name of deceased Arthur Pink

Date of death as stated to me ..... day of ..... Age as stated to me .....

Place of death

Last seen alive by me ..... day of .....

1 The certified cause of death takes account of information obtained from post-mortem.
2 Information from post-mortem may be available later.
3 Post-mortem not being held.
4 I have reported this death to the Coroner for further action.

a Seen after death by me.
b Seen after death by another medical practitioner but not by me.
c Not seen after death by a medical practitioner.

CAUSE OF DEATH

1(a) Disease or condition directly leading to death? Cerebrovascular accident (stroke)
(b) Other disease or condition, if any, leading to 1(a) Atherosclerosis (hardening of the arteries)
(c) Other disease or condition, if any, leading to 1(b) Hypertension (high blood pressure)
II Other significant conditions CONTRIBUTING TO THE DEATH but not related to the disease or condition causing it. Diabetes mellitus
Multi-infarct dementia

Approximate interval between onset and death

The death might have been due to or contributed to by the employment followed at some time by the deceased. Please tick where applicable

*This does not mean the mode of dying, such as heart failure, asphyxia, asthenia, etc: it means the disease, injury, or complication which caused death.

I hereby certify that I was in medical attendance during the above named deceased's last illness, and that the particulars and cause of death above written are true to the best of my knowledge and belief.

Signature ..... Qualification as registered by General Medical Council .....
Residence ..... Date .....

For deaths in hospital: Please give the name of the consultant responsible for the above named as a patient .....

Example 3 (see p 57)

a format similar to a large cheque book. There are three sections of paper, separated by perforations. The largest piece is the *certificate for the registration of death*; to the left is a *counterfoil* for hospital records that remains attached to the book; and on the far right is a *notice to informants*, summarising the information.

Ensure that you are authorised to complete the death certificate. As a house officer, ensure that you have looked after the patient within 14 days before death and you know the cause. If you are unsure about the cause of death read through the patient's notes and discuss them with your seniors. If the cause of death remains unclear the death will probably need to be referred to the coroner. Some deaths must be automatically referred to the coroner. Informing the coroner is compulsory in certain circumstances (see box 1).

Relatives appreciate it if you translate medical jargon, such as myocardial infarction and cerebrovascular accident into lay terms: "heart attack" and "stroke." It is good practice to put these lay terms in brackets after the medical terms on the counterfoil section.

## Example 1

Frieda Smith, a 78 year old retired school teacher, is registered blind secondary to her diabetic retinopathy and so does not see her dropped novopen on the floor. She falls over it, hurting herself and is unable to move. Sadly, she remains on the floor for three days before being discovered by a neighbour. You are the house officer on call at her local district general hospital. She has fractured her neck of femur and you are instructed to prepare her for theatre. However, she becomes suddenly dyspnoeic and you see that the electrocardiograph (ECG) shows sinus tachycardia. Her arterial blood gases suggest respiratory failure. A ventilation perfusion scan shows ventilation and perfusion mismatch. Mrs Smith has a cardiorespiratory arrest. You start cardiopulmonary resuscitation and put out a crash call. After 20 minutes' resuscitation, she remains unresponsive and the crash team decides to stop.

### *Frieda Smith's death certificate*

(1a) Pulmonary embolus (hours)

You can justify this because of the ECG, blood gas results, and scan result. Note that uninvestigated shortness of breath three hours before death alone would be no good.

(1b) Fat embolus (days)

From her fractured neck of femur

(2) Insulin dependent diabetes mellitus (20 years)

## Example 2

Mabel Green is 68 and was admitted after presenting with pain in her arms, legs, and ribs. Her bones are tender and skeletal *x* ray examinations show "punched out" osteolytic lesions. Bone marrow aspirate contains abundant plasma cells and your team diagnoses myeloma. Mrs Green is treated with chemotherapy but develops renal failure. She and her family ask that she should not be resuscitated in the event of a cardiac arrest. She dies suddenly, during one of your nights on call. You suspect she has perished from a hyperkalaemic cardiac arrest.

### *Mrs Green's death certificate*

(1a) Tumour lysis syndrome (two weeks)

(1b) Myeloma (five months)

Remember, you cannot give renal failure or cardiac arrest as a cause of death.

### Example 3

Arthur Pink is a 72 year old retired docker. Six months ago he was diagnosed with multi-infarct dementia and his wife Grace has been struggling to control his diabetes as he has become reluctant to cooperate with blood glucose monitoring. He has been hypertensive for 20 years but recently has been forgetting to take his antihypertensive medication. One morning he falls to the floor and Grace notices that he cannot move his left side. He is admitted to hospital where your team is on call. You diagnose a cerebrovascular accident. Despite treatment and secondary preventive measures, he dies four days later.

***Mr Pink's death certificate.***

In brackets is the sort of lay terminology that you might like to include for relatives.

(Ia) Cerebrovascular accident (stroke) Four days

(Ib) Atherosclerosis (hardening of the arteries) Five years

(Ic) Hypertension (high blood pressure) 20 years

(2) Diabetes mellitus (10 years)

Multi-infarct dementia (six months)

Sabina Dosani *senior house officer in psychiatry, St Thomas's Hospital, London*
s.dosani@medix-uk.com

All the scenarios in this article are fictitious and not based on any particular patient.

# Diet and bone health

In the sixth article in our series on nutrition, **Sarah Schenker** explains the importance of calcium and vitamin D

Diet is an important factor in forming healthy bones. The mineral calcium is obtained from the diet and deposited in bones and teeth. Vitamin D is required for this process.

A healthy diet providing adequate calcium at all stages of life, coupled with an active lifestyle, will help to ensure strong bones. This is particularly important during childhood, adolescence, and early adulthood when bones are developing. Peak bone mass is reached at the age of about 30 to 35. It is the stage at which the skeleton is strongest. After this age bone mass decreases. Optimising peak bone mass at skeletal maturity provides important protection against osteoporosis in later life.

Around 90-95% of peak bone mass is attained by the end of the second decade. Adolescence is a particularly critical period, with approximately 40% of peak bone mass in girls being laid down during this time. This process is under strong genetic control but other determinants include physical activity, especially weight bearing exercise, such as brisk walking, running, and climbing stairs, and nutritional factors, such as dietary calcium and blood levels of vitamin D.

## Osteoporosis

Osteoporosis is an increasing problem in the United Kingdom. It causes considerable pain and disability and costs the NHS in excess of £940m to treat each year.[1] It is a disease characterised by loss of bone mass and a deterioration in structural strength, in which the bones become fragile and susceptible to fracture, particularly at the hip, wrist, and spine. Osteoporosis most frequently affects older women who have gone through the menopause but it can affect men and younger women. One in three women and one in 12 men suffer from osteoporosis in the United Kingdom. Providing the hormones—in the form of hormone replacement therapy (HRT) that the body lacks, as a result of the menopause, can help prevent bone loss in women.

## Recommended intake of calcium

There is currently no international consensus as to exact recommendations for calcium intake. This is partly because some focus on meeting requirements, others for optimising bone density. Recommended intakes for young adults vary. Calcium requirements within the UK were reappraised in 1998 and the recommended intake of 700 mg of calcium a day for adults aged 19 to 50 was reaffirmed.[1] In contrast a United States report recommended 1000 mg a day.[2]

The National Diet and Nutrition Survey (NDNS) of people aged 4 to 18 showed that about 10% of older boys (11 to 18) and 20% of older girls had calcium intakes below the lower reference nutrient intake—that is, intakes that are likely to be inadequate.[3] In the NDNS survey of British adults aged 16 to 64 the average intake of those aged 16 to 24 was significantly lower than those aged 35 to 64 years.[4]

## Research findings

Intervention studies indicate that bone mineral density can be increased in the short term with increased calcium intake. In elderly people fracture rate is lower in those with a higher calcium and vitamin D intake.

Several randomised controlled trials have now investigated the link between calcium intake and bone mineral density. Bonjour and colleagues found a greater increase in bone mineral density among 8 year old girls taking an extra 850 mg of calcium compared with those on their normal diet.[5] This study also showed that the response to calcium supplementation depended on habitual calcium intake. Girls who had previously had the lowest calcium intake (<880 mg a day) benefited most from supplementation.

Research suggests that encouraging an increase in dairy food consumption could produce significant gains in bone density in children and adolescents. Cadogan and colleagues supplemented 80, 12 year old girls with an average of 330 ml of milk a day for 18 months.[6] Bone mineral density increased to a greater extent in the supplemented group compared with those who had no extra milk. Some of the benefit may be due to other nutrients besides calcium as milk contains several other nutrients which may be essential for bone growth, including protein, phosphorus, magnesium, zinc, and B vitamins.

A recent review concluded that an increase in calcium intake during growth increases bone mineral by approximately 1-5%, depending on the skeletal site measured.[7]

Despite good evidence for a short term benefit of calcium supplementation, it is not clear if this persists once the supplement is withdrawn. Short term increases in calcium or dairy food intake in children or adolescents may not be sufficient to sustain an increase in bone mass over several decades. Previous studies have found differences in bone mineral density to disappear 18 months to two years after the withdrawal of the calcium supplements.[8,9]

The ongoing Cambridge Bone Study is seeking to determine whether advising young people aged 16 to 18 to increase calcium intake (to 1000 mg a day), as well as increas-

**Jason and the argonauts discover that calcium and vitamin D help grow strong skeletons**

ing exercise levels, provides an effective means of optimising bone mineralisation. This study will also measure the effects 12 to 18 months after the end of the intervention.

## Dietary sources of calcium

As well as milk and dairy products, such as yoghurt and cheese, which are the major calcium providers in the diet, calcium is also obtained from bread (a statutory requirement exists in the United Kingdom that white flour should be fortified with calcium, iron, vitamin B1, and vitamin B2), pulses, green vegetables, dried fruits, such as apricots, nuts, and seeds, and the soft bones found in canned fish.

## Vitamin D

Vitamin D is important for the absorption of calcium. Bone loss may be significantly reduced in postmenopausal women whose diets are supplemented with 700 IU vitamin D daily. Other nutrients may also be important, such as magnesium, potassium, fibre, vitamin C, and zinc to be associated with a significantly higher lumbar spine bone mineral density.[10]

Vitamin D plays a vital role in calcium homeostasis and bone metabolism. Vitamin D insufficiency causes a change in serum free calcium which in turn stimulates parathyroid hormone secretion and mobilises calcium from bone. The clinical deficiency diseases rickets, which affects infants and children when bones are growing, and osteomalacia, which affects adults whose bone growth is completed, are rare in the United Kingdom, although cases are still sporadically reported.

But poor vitamin D status has substantial public health implications since it may be an adverse factor in developing osteoporosis. An adequate vitamin D status throughout childhood is likely to influence achieving peak bone mass.

### *Older people*

Supplementation with calcium and vitamin D in older men and women reduces bone loss at several sites and decreases rates of non-vertebral fractures.[11] Ensuring an optimal intake of both calcium and vitamin D is, therefore, an important strategy to maintain existing bone mass and reduce fracture in older people.

The NDNS of people aged 65 years and over found that approximately 98% had vitamin D intakes below the level recommended (the recommended nutrient intake for this age group is 10 µg a day).[12] A large proportion also had low vitamin D status (low plasma levels), particularly those living in institutions as they had little exposure to sunlight.

The NDNS of people aged 4 to18 years found a low vitamin D state in a significant proportion of those surveyed.[4] In both boys and girls, this problem increased with age. This may be linked with a reduction in the amount of time spent playing outside (thus exposure to sunlight).

### *Sources of vitamin D*

*Diet:* The best dietary sources of vitamin D are oily fish—for example, herring, mackerel, salmon, trout—fortified margarines and spreads, meat and meat products, and eggs. The vitamin can also be synthesised through the action of sunlight on the skin, and for most people this provides the major source. Vitamin D levels fall in winter as skin synthesis declines.

*Sunlight:* Older people are vulnerable to vitamin D insufficiency because the skin becomes less efficient at synthesising vitamin D with age as the epidermis thins. The amount of pigmentation in the skin also influences its capacity to synthesise vitamin D and those with darker skin require longer exposure to ultraviolet light. Certain ethnic groups in the United Kingdom are, therefore, vulnerable to vitamin D deficiency and advised to take a supplement. There are also several cultural characteristics among these groups that adversely affects vitamin D status, including wearing concealing clothes and excluding meat and fish from the diet.

Encouraging children to be more physically active should ensure sufficient sunshine exposure and achievement of optimal peak bone mass.

Increasing vitamin D intake from the diet or through supplementation is likely to be of benefit for vulnerable groups who rely on dietary sources as a means of achieving an adequate state, particularly during the winter months. In the United Kingdom these vulnerable groups include children from Asian communities, whose skin pigmentation reduces absorption of sunlight, those who wear clothes which fully conceal them, older people who are housebound or seldom go out, and those living in institutions.

The British Food Foundation is an independent charity, which raises funds from the food industry, the government, the EU Commission, and other sources.

Sarah Schenker *British Nutrition Foundation, London*
s.schenker@nutrition.org.uk

1 Department of Health. *Nutrition and bone health.* London: DoH, 1998.
2 National Institutes of Health Consensus Development Panel. Optimal calcium intake. *JAMA* 1994;272:1924-8.
3 Gregory J, Lowe C, Bates C, et al. *National diet and nutrition survey: young people aged 4 to 18 years.* London: HMSO, 2000.
4 Gregory J, Foster K, Tyler H, Wiseman M. The dietary and nutritional survey of British adults. London: HMSO, 1990.
5 Bonjour J-P, Carrie A-L, Ferrari S, Clavien H, Slosman D, Thientz G, et al. Calcium-enriched foods and bone mass growth in prepubertal girls: a randomized, double-blind, placebo-controlled trial. *J Clin Invest* 1997;99:1287-94.
6 Cadogan J, Eastell R, Jones N, Barker ME. Milk intake and bone mineral acquisition in adolescent girls: randomised, controlled intervention trial. *BMJ* 1997;315,1255-60.
7 Cashman KD, Flynn A. Optimal nutrition: calcium, magnesium and phosphorus. *Proc Nutr Soc* 1999;58:477-87.
8 Slemanda CW, Reister TK, Peacock M, Johnston CC. Bone growth in children following the cessation of calcium supplementation. *J Bone Miner Res* 1993;8:S154.
9 Lee WTK, Leung SSF, Leung DMY, Cheng JCY. A follow-up study on the effects of calcium-supplement withdrawal and puberty on bone acquisition of children. *Am J Clin Nutr* 1996;64:71-7.
10 New S. Fruit and vegetable consumption and skeletal health: is there a positive link? *Nutr Bull* 2001; 26:121-6.
11 Dawson-Hughes B, Harris S, Krall E, Dallal G. Effects of calcium supplementation on bone density in men and women 65 years of age or older. *N Engl J Med* 1997;337:670-6.
12 Finch S, Doyle W, Lowe C, Bates CJ, Prentice A, Smithers G, et al. *National diet and nutrition survey: people aged 65 years and over.* London: HMSO, 1998.

# Only one chance to get it right: end of life care

**Mari Lloyd-Williams**, **Sanjay Shah**, and **Idris Baker** explain the fundamentals of palliative care and supporting dying patients

End of life care is an art—it is a challenging and essential area of medical practice. There is no specialty without such responsibility at least from time to time.

Components of such care includes identifying that death is approaching; discussion with the patient and family and carers or both; the identification and management of distressing symptoms; support for family and carers and bereavement support; and, if we are to do it well, looking after ourselves.

## When are the last days?

In this article we refer to the last few days of life, when the final decline on the background of a progressive illness has begun—this is not always easy to identify and others' perspectives must be added to it. Nursing staff have longer periods of direct patient contact and their impression should be noted. Families (in which we include all "significant others" and not only relatives) and, when lucid, patients may perceive signs of imminent death. They may have no special knowledge of dying but have a unique insight and their impressions often add sensitivity to the identification of end of life events. It is less difficult to predict end of life in patients with incurable cancer than in other patients. Some of the most important indicators that life is coming to end are:

- Progressive and profound weight loss
- Profound weakness
- Impaired cognition
- Reduced oral intake and difficulty in taking oral medication

Sometimes there is a tendency for people to withdraw from a dying person, and professionals are not immune from this. This may be a fear of death or lack of confidence that they will know what to say or do. Very often, little needs to be said or done, but the act of not withdrawing, of continuing to go into the side room rather than guiding the ward round past the dying, is valuable in its own right.

## Broadening decision making and teamwork

One of the hallmarks of palliative care—and this should be applied equally to end of life care whatever the arena—is the involvement in decisions of patients and, where appropriate, families. Examples are decisions such as where the patient may wish to die and the use of possible life prolonging treatment and possibly futile interventions—for example, antibiotics. Most patients want doctors to discuss such issues. They may not be able to ask about it openly because of fear and anxiety or may think that they would be wasting the doctor's time. It is not unusual for families to withhold such information from the patients, as they fear its adverse impact. It would be useful again to allay their anxieties and explore reasons for such a request. It should be made clear that if a patient wants to know, it is a doctor's duty to give the patient honest information.

Effective care at the end of life is a multidisciplinary and multiprofessional task. Doctors and nurses are commonly the main professionals, but in some cases there is a need for others' involvement—for example, chaplains, social worker, physiotherapist, and possibly the specialist pallia-

Principles for managing symptoms

| Symptoms | Treatment | Comments |
|---|---|---|
| Pain | Paracetamol<br>Non-steroidal anti-inflammatory drugs<br>Adjuvant analgesics, such as anticonvulsants<br>Opioids<br>Steroids | |
| Dyspnoea | Opioids<br>Benzodiazepines<br>Oxygen<br>Complementary treatments | Look for easily and non-invasively reversible causes, such as bronchospasm |
| Nausea/vomiting | Metoclopramide<br>Cyclizine<br>Haloperidol<br>Methotrimeprazine<br>Steroids | Look for reversible causes, such as constipation, and in selected group hypercalcaemia or urine tract infections. Stop all non-essential drugs |
| Confusion | Haloperidol | Don't forget: urine retention and constipation can cause confusion in terminally ill<br>Review drug chart |
| Agitation/restlessness | Midazolam<br>Haloperidol<br>Methotrimeprazine | Again, remember urine retention and constipation |
| Noisy breathing | Hyoscine butylbromide<br>Glycopyrronium<br>Suction | It is frequently more distressing to carers, relatives, and fellow patients |
| Urinary incontinence/retention | Catheterisation | |
| Dry/sore mouth | Good oral hygiene<br>Oral balance gel<br>Frequent moistening of mouth<br>Frequent sips of water<br>Ice cubes<br>Artificial saliva<br>Vitamin C | Rule out oral thrush<br>Avoid temptation to use IV fluids to relieve this symptom |
| Extreme fatigue | Steroids | Very difficult to treat<br>In a highly selected group of patients, blood transfusion may be appropriate |
| Constipation | Bisacodyl suppositories<br>Stool softeners | |
| Terminal restlessness | Midazolam | |
| Psychological and emotional issues: fear, anxiety, loss of control, helplessness, hopelessness, feelings of guilt, grieving for loss of family | Reassurance<br>Discussion<br>Empathy<br>The art of "being" rather than "doing" or "saying"<br>Sedation | Role of chaplain and priest can be very important |

tive care team. In addition many areas now have community nursing care—for example, "hospital at home" or "hospice at home" which undertake care of patients who are dying in the community. Where many professionals are involved good communication is essential both between colleagues and with the patient and their family.

## Ongoing evaluation and symptom control

Patients' needs at the end of life can be complex. A marked proportion of patients (though not all) will develop new symptoms or worsening of existing symptoms. Patients may not mention these spontaneously due to exhaustion or the assumption that nothing can be done to help—for example, constipation. Full examination may cause discomfort so a detailed appropriate examination should be carried out. Investigations have little if any role to play in end of life care, and procedures such as repeated blood tests are unnecessary and distressing to patients.

Common symptoms include

- Pain
- Dyspnoea
- Nausea or vomiting
- Confusion
- Agitation or restlessness
- Noisy breathing
- Urinary incontinence or retention
- Dry or sore mouth
- Extreme fatigue

There are non-pharmacological and pharmacological treatments for virtually any problem. Pain caused by an unstable fracture in a dying patient unfit for surgery may be treated effectively by gentle immobilisation and analgesia.

Although drugs are a large part of the management of many symptoms at the end of life, it is as important to stop some drugs as it is to start others. Oral medications often become difficult to take and most long term drugs may be stopped—for example, a dying patient does not need drugs to lower their blood pressure, slow their heart rate, or lower their cholesterol levels. Often the best approach is to stop everything except what is clearly still needed.

General principles for the management of common and some other symptoms is outlined in the table.

Many patients are unable to swallow for a short period before death, and medication previously given orally must be given by a parenteral route—for example, in a syringe driver. Regular injections or suppositories are less well tolerated but subcutaneous injections can be used when needed as a back up. Syringe drivers for continuous infusion should be universally available and most of the drugs needed at the end of life can be given by this route. Drugs that can be administered using syringe drivers include diamorphine, haloperidol, cyclizine, metoclopramide, methotrimeprazine, midazolam, steroids, and hyoscine.

It is important to remember that for good symptom control in the last few days we need to assess, explain, reassure, treat, and review.

## Support for family and carers

Family and carers should be actively involved in caring for their loved ones if they so wish. Acknowledge their physical—for example, exhaustion—psychological, and emotional needs and help them to cope by listening and supporting and if appropriate giving them a chance to say "goodbye." Do not ignore children and elderly members of the family—they may seem fragile, but usually cope well if kept well informed and supported appropriately.

## After the death

Always show sympathy to relatives—they will remember this time vividly and many complaints to hospitals and trusts are made as a result of harsh or insensitive words at the time of a death. Listen to them patiently and be prepared to go through what has happened again. Explain about the administrative procedure—for example, that a death certificate will be issued and that they will be required to register the death before making any funeral arrangements. Relatives may feel utterly sad, numb, or elated and thankful that their relative's suffering is over. Remember to acknowledge that all such feelings are normal and that grief is a rollercoaster of emotions. Encourage the family to make and maintain contact with the primary care team who will possibly have known the family well for several years and be able to offer ongoing support.

Lastly, remember that care of the dying is not a passive, but an active and potentially exhausting yet a rewarding act. Some deaths will affect us more than others; shedding tears and feeling sad is not a failure for a doctor, but reminds us that we too are human.

Mari Lloyd-Williams *palliative care consultant and lecturer*, m.lloydwilliams@tesco.net

Sanjay Shah *specialist registrar in palliative care*,

Idris Baker *specialist registrar, LOROS Hospice, Leicester*

## Further reading

Fallon M, O'Neill B. *ABC of palliative care*. London: BMJ Publishing Group, 1998.

Twycross R. Symptom management in advanced cancer. 2nd ed. Abingdon: Radcliffe Medical Press, 1997.

National Council of Hospice and Specialist Palliative Care. *Changing gear: guidelines for managing the last days of life in adults*. NCHSPC,1997.

BRIAN HARRIS/LIGHTBOX

# Creative consulting: more about placebos?

In the fifth article in his series **David Reilly** is joined by **Zelda Di Blasi** to look at the placebo response

If placebos are inactive, what is it that produces the remarkable changes? Do not ask what the placebo can do but, "What can the thing that responds to the placebo do?" You will end up thinking about self healing and self destructive processes.

Just as you can work backwards from a successful recovery to study healing reactions,[1] you can consider what placebo reactions say about healing responses and better care. If placebo can provoke a change indirectly perhaps we can learn to do so directly, because we are the only possible pathway for placebo action. The implications of this pathway, between our consciousness and our biology, can be missing in academic debate about placebo,[2 3 4] but the discovery that people responding to placebo show definite brain scan changes can bring it back into focus.[5]

### Universal cultural features in healing rituals[24]

- The healer must have a coherent frame of reference or explanation for the origin and nature of the problem and how it can be helped—germs, curse, chi, etc
- A symbolic bridge is made, integrating the relationships and situation in terms of the culture and the healer's frame of reference
- The healer aims to activate the bridge (often subliminally) by persuading the client that the problem is explainable in the healer's frame of reference: "I can see you have cancer, curses, suppressed anger, irritable bowel, etc"
- Once cognitive consensus is achieved, emotional involvement is needed through attachment to the symbols and frame of reference. Clients have to be convinced they are possessed by a spirit, have depression, have food allergies, etc, and it will kill or damage them if not treated
- Techniques and guided symbols of reference are used—for example, prescription, exercise, diet, or exorcism. Now over time the patient can reframe the situation in the light of the healer's reference—for example, by talking about the medicine's effect or balancing the chakras
- The healed client has a new narrative and way of functioning. A story is necessary to explain what happened—the tablets worked, the spirit is gone, etc

## What can the placebo effect do?

Distress can be modified—as, for example, physical pain: in one study saline was as effective as morphine in 40% of people after surgery.[6] The same goes for emotional pain: about 70% of patients respond to placebo for depression.[7] So is the placebo effect all just in the mind? Does it only modify experience, not "objective" reality? When we blush when embarrassed is it "real"? It is as real as the quantitative electroencephalograph brain scans showing reduced prefrontal cortex activity in people responding to placebo antidepressants.[5]

## How many respond?

It is often misquoted that one in three people are placebo responders. This is based on Beecher's original work analysing 1082 cases where "35.2% responded." The range, however, was 18-52%.[8]. Reported rates vary from less than 10% to more than 70% according to circumstances and context. Everyone has self healing potential.

## Who responds?

There is probably no simple predictive measure of whether someone will or will not respond. People who respond to placebo have normal personalities, and those who do not have more rigid personalities, are suspicious, and sometimes do not respond to "ordinary medicine."[9] Maybe it is only the gullible who respond: medical students respond more than most. In a study on the effects of psychotropics, of 300 medical students 50% had psychological changes and 60% had physical effects. They identified the pink pills as stimulants and the blue pills as sedatives. They were all placebos.[10]

## What form?

The physical form of the intervention has an impact. Larger placebo capsules are viewed as stronger, and two are stronger than one. Injections produce larger effects than pills. Red capsules and yellow capsules tend to act as stimulants or antidepressants, blue capsules as sedatives, and white capsules as analgesics or narcotics.[11 12 13]

Symbolism has impact, and none more so than the ritual of surgery.[13] Surgeons made skin incisions in patients expecting to have their internal mammary artery tied to help blood flow to their heart, to help with their angina. A random selection of patients, however, never had the operation and the incision was sewn back up. All 18 non-treated patients had less angina six weeks afterwards, some had improved exercise electrocardiographs, and the effect lasted for years in some.[14]

## In which culture?

The advertisements, the packaging, and the hypnotic names carry messages—for example, "Welldorm" for insomnia and "Marvelon" as a marvellous contraceptive.[15] The brand name on that free plastic pen the drug representative gives to us has an effect. In one study, branded tablets were significantly more effective than unbranded tablets for the treatment of headaches.[16] A powerful message in one culture may be meaningless in another.

## Expecting what?

You tend to get what you expect. From aspirin placebo you get aspirin-like effects; from morphine you get morphine-like effects and side effects. This system has puzzling specificity, and placebos interact with drug action synergistically or disruptively. Bronchoconstriction due to atropine and other anticholinergics can be reversed by suggestion (with saline inhalation) and bronchoconstriction of suggestion can be blocked by ipratropium.[17] Your previous experience and learning all have an effect like Pavlov's dogs salivating to the sound of the feeding bell. Rats given repeated scopolamine injections show the same depressed behaviour when given later placebo injections.[18] If a prescription helped (say in reducing your anxiety), you might repeat what you "learned" to a later placebo version of the same treatment. So it is better to "anchor" the patient on their own self coping than on you or your treatments.

Drugs are modelled in the laboratory and tested in animals. It is naive, however, to expect only predictable "hard" pharmacological outcomes. They become an ingredient in a complex reactive system. This means it is also naive to believe that the real effect is that part left over after subtracting the placebo: one plus one might come out as four, and you might be one of the active ingredients.

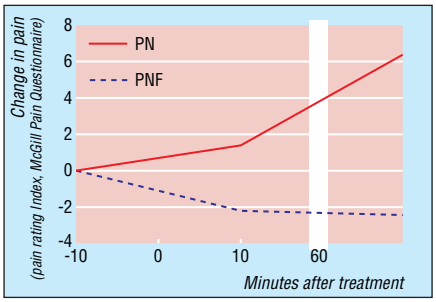
## From which carer?

Push the square peg of medicine into the round hole of healing and you end up with ugly words, like iatroplacebogenics, to explain that results are influenced by who does the caring and how.[19] The carer's personality and attitude, their warmth, empathy or hostility towards the patient, and their attitude towards the treatment (active enthusiastic or passive nihilistic) all affect outcome. A review of controlled trials found that doctors who adopted a warm, friendly, and reassuring manner were more effective than those whose consultations were formal and did not offer reassurance.[20] Good caring and a weak medicine can give a better outcome than poor caring and a strong medicine.[21] This means that the placebo arm of one study can sometimes have better results than the "active" arm of another. This causes endless confusion and complicates the search for evidence based practice.

Single blinded design is even less reliable, which brings us back to expectations. In a study of people having teeth removed, patients were told that they might get one of three injections for pain control that would make their pain better (fentanyl, a strong pain killer), worse (naloxone, an opiate antagonist that would make pain worse), or have no effect (placebo). The patient was "blind" but the dentist knew which group each patient was in.[22]

The graph shows the response to placebo in the two groups. When the dentists knew the patient had a chance of a "real" painkiller, the placebo was "activated" and was as distinct from the other placebo response as a "real" medicine from a dummy medicine. The carer knew there was a chance of receiving a "real" medicine and somehow transmitted this to patients, who then activated their own healing systems.

We can just as readily destroy such a reaction. Patients receiving eight weeks of placebo or antidepressants were improving equally until those taking placebo were told so.[5] Most deteriorated and ended up taking medications.[23]




**Response of patients when dentists knew they would get an injection with no chance of pain relief (PN, placebo or naloxone) or a 1 in 3 chance of pain relief (PNF, placebo, naloxone, or fentanyl [22]) (reprinted with permission from Elsevier Science (*The Lancet* 1985; i: 43))**

## Conclusion

So the treatment, its presentation, and its expected effects interact with patient factors blended with the carer and the context to affect healing systems. It seems that the best results are achieved when a patient has confidence in the carer, the institution, or the system of care, and when these are congruent with the patient's attitudes, beliefs, and expectations. Western medicine has often ignored or marginalised direct engagement with self healing (hypnotherapy is an important exception), but, as the box shows, anthropologists have seen that every human culture expresses these same dimensions, consciously or not.

David Reilly *consultant physician, Homeopathic Hospital, Glasgow*
davidreilly1@compuserve.com

David Reilly welcomes correspondence or questions on this series.

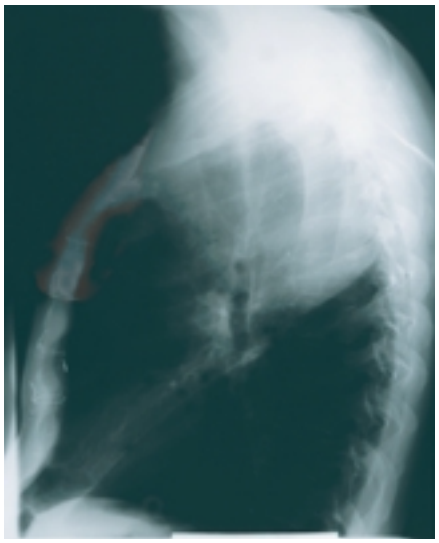
1 Reilly D. Creative consulting: what modifies a healing response? *studentBMJ* 2002;10:12-3. (January.)
2 Kienle GS, Kiene H. The powerful placebo effect: fact or fiction ? *J Clin Epidemiol* 1997;50:1311-8.
3 Kienle GS, Kiene H. The placebo effect: a scientific critique. *Complement Ther Med* 1998;6:14-24.
4 Hrobjartsson A, Gotzsche PC. Is the placebo powerless? An analysis of clinical trials comparing placebo with no treatment. *N Engl J Med* 2001;344:1594-602.
5 Leuchter AF, Cook IA, Witte EA, Morgan M, Abrams M. Changes in brain function of depressed subjects during treatment with placebo. *Am J Psychiatry* 2002;159:122-9.
6 Beecher HK. Increased stress and effectiveness of placebos and "active" drugs. *Science* 1960;132:91-2.
7 Kirsch I, Sapirstein G. Listening to Prozac but hearing placebo: a meta-analysis or antidepressant medication. In: Kirsch I, ed. *Expectancy, experience and behaviour*. Washington, DC: American Psychological Association, 1999.
8 Beecher HK. The powerful placebo. *JAMA* 1955;159:1602-6.
9 Lasagna L, Mosteller F, von Felsinger JM, Beecher HK. A study of the placebo response. *Am J Med* 1954;16:770-9.
10 Blackwell B, Bloomfield SS, Buncher CR. Demonstration to medical students of placebo responses and non-drug factors. *Lancet* 1972;13i:1-11.
11 Buckalew LW, Coffield KE. An investigation of drug expectancy as a function of capsule colour and size and preparation form. *J Clin Psychopharmacol* 1982;2:245-8.
12 De Craen AJM, Roos PJ, de Vries AL, Kleijnen J. Effect of colour of drugs: systematic review of perceived effect of drugs and of their effectiveness. *BMJ* 1996;313:1624-6.
13 Beecher HK. Surgery as placebo. *JAMA* 1961;176:1102-7.
14 Cobb L, Thomas GI, Dillard DH, Merendino KA, Bruce RA. An evaluation of internal-mammary-artery ligation by a double-blind technique. *N Engl J Med* 1959;260:1115-8.
15 Holm S, Evans M. Product names, proper claims? More ethical issues in the marketing of drugs. *BMJ* 1996;313:1627-9.
16 Branthwaite A, Cooper P. Analgesic effects of branding in treatment of headaches. *BMJ* 1981;282:1576-8.
17 Butler C, Steptoe A. Placebo responses: an experimental study of psychophysiological processes in asthmatic volunteers. *Br J Clin Psychol* 1986;25:173-83.
18 Herrnstein RJ. Placebo effect in the rat. *Science* 1962;138:677-8.
19 Shapiro AK. Iatroplacebogenics. *Int J Pharmacopsychiat.* 1969;2:215.
20 Di Blasi Z, Harkness E, Ernst E, Georgiou A, Kleijnen J. Influence of context effects on health outcomes: a systematic review. *Lancet* 2001;357:757-62.
21 Walach H. The efficacy paradox in randomised controlled trials of CAM and elsewhere: beware of the placebo trap. *J Altern Complement Med* 2001;7:213-8.
22 Gracely RH, Dubner R, Deeter WR, Wolksee PJ. Clinicians' expectations influence placebo analgesia. *Lancet* 1985;i:4.
23 Fox, M. Brain scan study shows how placebo aids depression. Washington: Reuters, 2002. www.forbes.com/newswire/2002/01/01/rtr467478.html (accessed 5 Feb 2002).
24 Helman H. Placebo and nocebos: the cultural construction of belief. In: Peters D, ed. *The placebo response: biology and belief*. London: Churchill Livingstone, 2001.

# Pancoast's apical bronchial carcinoma

## Case history

This 72 year old retired engineer, a former smoker, presented to his GP complaining of a recent onset cough, productive of sputum and containing "spots" of fresh blood. He also said that he had recently been more breathless than normal on walking to collect his morning newspaper.

On examination the left apex was dull to percussion and there were increased breath sounds over this area. There was also a reduction in chest expansion on the left side. His left eyelid was slightly droopy and the pupil seemed smaller on that side.



**Lateral chest radiograph**

### *Questions*

(1) What preliminary investigations would you request given the above clinical features?

The posteroanterior (PA) and lateral chest radiographs requested are shown below.

(2) What abnormalities are apparent on these films?

(3) What further imaging investigations may be required in the management of this condition?

(4) What are the main histological types of this condition?

(5) What is your diagnosis and what are the characteristic features of the associated syndrome?

### *Answers*

(1) The following first line investigations would be necessary given the symptoms in a former smoker in his 70s: a posteroanterior (PA) chest radiograph, sputum cytology, full blood count, urea and electrolytes, liver function tests. A lateral radiograph can help in localising a lesion within the lung.

(2) The PA chest radiograph shows a large, poorly defined radio-opaque area in the right upper lobe of the lung extending into the apex. This is further shown on the lateral radiograph.

Sternal wiring indicates a previous median sternotomy, with surgical clips projected over the heart consistent with a left internal mammary artery (LIMA) to left anterior descending (LAD) coronary graft. Such a medical history may be related to his smoking history.

(3) Thoracic computed tomography (CT) is appropriate to assess and stage the full extent of the lesion. This may precede or follow bronchoscopy with biopsies/brushings. CT imaging of the liver and adrenals is routinely undertaken in the patient with carcinoma of the lung to exclude intra-abdominal metastases.

Thoracic CT can also be useful in confirming that the lesion's features are consistent with bronchial malignancy. If alkaline phosphatase (from the above biochemical tests) is markedly raised or if "bony" pain is a further patient complaint a radioisotope bone scan should be considered to identify any metastatic bone disease. Clearly, this would further influence management.

(4) Bronchial carcinomas are typically divided into small cell lung cancers and non-small cell lung cancers because the histology determines the management and prognosis. Non-small cell lung cancers are further divided into large cell carcinoma, adenocarcinoma, and squamous cell carcinoma. Squamous cell carcinoma is the commonest bronchial carcinoma overall and is the one most associated with Pancoast's tumour.

(5) The diagnosis is an apical bronchial carcinoma, specifically a Pancoast's tumour. This is an apical bronchial carcinoma associated with ipsilateral Horner's syndrome. This is characterised by ptosis, miosis, enophthalmos, and ipsilateral anhydrosis. This is due to the invasion of the cervical sympathetic plexus by the encroaching tumour.

### Key terms

- Miosis—constriction of the pupil
- Enophthalmos—shrunken eye
- Anhydrosis—loss of sweating

Ian C Bickle, *final year medical student, Queen's University, Belfast*
medicine@totalise.co.uk,

Barry Kelly *consultant radiologist, Royal Victoria Hospital, Belfast*

## Further reading

Dicks E. Chest *x* rays made easy. *studentBMJ* 2001;9:10-2.(February 2001.)

Henry Khumrath Pancoast, 1875-1939, professor of radiology, University of Pennsylvania, Philadelphia.

Johann Horner, 1831-83, professor of ophthalmology, Zurich. He first described his syndrome in 1869.

# Will listening to Mozart make you smarter?

**Marion Simpson** investigates an unusual exam aid

If there is an easy way of doing a difficult task we will take it every time. So it is easy to see why the experiments carried out by Rauscher and colleagues in the University of California in 1993 caused such a stir in the popular press.1 They suggested that something as simple as listening to a Mozart piano sonata might improve your aptitude for complex tasks.

The authors based their claims on a series of experiments with 36 college students in which volunteers were subjected to 10 minutes of one of three conditions: silence, a relaxation tape designed to lower blood pressure, or a recording of Mozart's Sonata in D Major for Two Pianos, K448. After this period, participants' spatial reasoning skills were tested using a set of tasks which included pattern analysis and paper folding and cutting. When their performance was translated into IQ scores, the group which listened to Mozart displayed a mean nine point lead over the other two groups.

## Why was Mozart chosen?

The original theory behind the "Mozart effect" experiments was derived from a mathematical model of the cerebral cortex.[2] The same research group also subjected music of various types to mathematical analysis and proposed that certain features of Mozart's music meant that it would resonate with certain brain regions, notably those cortical areas involved with spatial reasoning, "priming" them so that they would be quicker and more efficient on future use. This idea of "use dependence" is a popular one in neuroscience, and the cellular mechanisms involved are a hot topic in current research.

## Controversy and misconception

The original results caused great excitement, generating wild ideas about the mind expanding powers of the composer. This was bound to lead to disappointment when the hypotheses were subjected to more stringent testing. Rauscher and colleagues never claimed that Mozart could enhance all aspects of brain function. Perhaps it was misleading to translate their subjects' scores on the spatial reasoning tasks into IQ ratings, but the authors did not suggest an effect on general intelligence. Even now, however, debate continues about the authenticity of the original results.[3]

Criticism has been directed at the small scale of the original study and the suitability of the controls used, and attempts to replicate the original findings have yielded varying degrees of success, depending on the test paradigms used.[4]

Other groups have suggested that enjoyment of the music and resultant arousal could explain the improvements seen,[3] although

studies designed specifically to examine the influence of musical preference have not supported this.[4] More recently, rats exposed to Mozart in the uterus have shown improved aptitude for navigation around a t-maze.[5] This is further evidence for an enhancement of spatial reasoning in the absence of conscious musical appreciation.

## Mozart as medicine

More recently, electroencephalography (EEG) in patients with epilepsy and those who were unconscious has shown changes in brain activity in response to Mozart. This argues against the enjoyment arousal theory, at the same time as suggesting interesting and novel avenues for treatment. EEG measurements taken while patients were having seizures have shown as much as a 41% reduction in epileptiform events while Mozart's K448 was being played, with a significant beneficial effect being retained after the music stopped.[6]

Similar results were found in both conscious and unconscious patients, arguing against simple relaxation or enjoyment.

One possible explanation is that features of Mozart's music induce patterns of neuronal activity which somehow disrupt those necessary for the seizure to proceed, "resetting" the brain's electrical activity. Certainly, various reports have been made of epileptic seizures being triggered by certain pieces of music.[7]

It is not implausible that other pieces could have a beneficial effect. Anecdotal evidence suggests that Mozart might be of benefit in other neurological conditions, notably Alzheimer's disease, stroke, and Williams syndrome.[8]

## The general consensus

Opinion is still divided on the evidence for the "Mozart effect," both in itself and as a potential neurological treatment. Its original proponents defend it vociferously, and are in the process of developing a mathematics teaching programme for primary school children, based on their neural model. Longer term studies with children have yielded encouraging results in terms of subsequent successes in maths after musical training,[9] but the precise explanation remains cloudy. Music seems to enhance self esteem and motivation to attend school, which could account for a proportion of the effect; thus the debate rages on.

So Mozart might not be able to turn us into a mathematical or any other type of genius, he might not be able to cure neurological diseases, but it certainly cannot do any harm.

My radio is now tuned to Classic FM and will certainly stay that way in the run up to exams. It's worth a try.

Marion Simpson *third year medical student, University of Edinburgh Medical School*
9810930@sms.ed.ac.uk,

1 Rauscher FH, Shaw GL, Ky KN. Music and spatial task performance. *Nature* 1993;365:611.
2 Leng X, Shaw GL. Towards a neural theory of higher brain function using music as a window. *Concepts in Neuroscience* 1991;2:29-58.
3 Chabris CF, Steele KM, Rauscher FH. Prelude or requiem for the "Mozart effect"? *Nature* 1999;400:826-8.
4 Newman J, Rosenbach JH, Burns KL, Latimer BC, Matocha HR, Vogt ER. An experimental test of "the Mozart effect": does listening to his music improve spatial ability? *Percept Mot Skills* 1995;81:1379-87.
5 Rauscher FH, Robinson KD, Jens JJ. Improved maze learning through early music exposure in rats. *Neurol Res* 1998;20:427-32.
6 Hughes JR, Daaboul Y, Fino JJ, Shaw GL. The "Mozart effect" on epileptiform activity. *Clin Electroencephalogr* 1998;29:109-19.
7 Sacks O. *An anthropologist on Mars*. New York: Knopf, 1995.
8 Shaw GL. *Keeping Mozart in mind*. London: Academic Press, 2000.
9 Rauscher FH, Shaw GL, Levine LJ, Wright EL, Dennis WR, Newcomb RL. Music training causes long-term enhancement of preschool children's spatial-temporal reasoning. *Neurol Res* 1997;19:2-8.