

Fortnightly review

Faecal incontinence

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Faecal incontinence affects men and women of all ages. Daily or weekly episodes occur in about 2% of the adult population and in about 7% of healthy independent adults over the age of 65.¹⁻⁴ The disorder causes great personal disability and has a high financial cost. In the United States, more than \$400m each year is spent on faecal incontinence appliances alone, and faecal incontinence is the second commonest reason (more common than dementia) for requesting placement in a nursing home.⁵ A third of elderly people in retirement homes or hospital are incontinent for stool.

Issues related to faecal incontinence are especially relevant with a growing population of elderly people. Advances in understanding and treatment have led to improved treatments and a good outcome for most patients. Much progress has been brought about by the recent development of accurate and simple techniques for imaging the anal sphincter muscles.

This review focuses on advances in the understanding and treatment of faecal incontinence. It is not a systematic review but is based on a choice of areas that I feel are of greatest recent importance, supplemented by a Medline search using the keywords "faecal incontinence."

Symptoms, clues, and investigations

Passive faecal incontinence or soiling—that is, faecal leakage without awareness—is usually associated with dysfunction of the smooth muscle tissue of the internal anal sphincter or with impacted stool in the rectum. Faecal urgency and urge incontinence are generally related to dysfunction of the striated muscle tissue of the external anal sphincter or to high bowel pressures and a normal sphincter, seen in patients with irritable bowel syndrome or diarrhoea from other causes.⁶

Advances in sphincter imaging and physiological testing

The introduction of anal endosonography as a way of imaging accurately the anal sphincter muscles has enabled accurate identification of the lesions causing faecal incontinence and has allowed rational planning of treatment. Magnetic resonance imaging with an anal coil can be helpful in investigating para-anal structures or when the ultrasound findings are equivocal.

Endosonography has largely replaced electromyography, although the latter may be useful when scarring makes interpretation of ultrasonography diffi-

Summary points

Anal incontinence affects 2% of all adults and 7% of all healthy adults over the age of 65

Manometry, sensory testing, and ultrasonography allow accurate characterisation of sphincter abnormality in most patients

Faecal incontinence is commonly caused by structural damage during childbirth and anal surgery; neurological disease and previously corrected congenital anorectal malformations may cause faecal incontinence and constipation

Loperamide or codeine phosphate are often safe and effective when symptoms are mild, infrequent, are not caused by faecal impaction with overflow, or where no easily correctable lesion is present

Behavioural techniques may help where there is no structural sphincter damage and may be useful as an adjunct to other treatments

Some structural damage to the anus may be repaired by simple surgery, and new operative techniques may help patients with complex sphincter injury or degenerate sphincter muscles

cult or in patients who have had an imperforate anus. Pudendal nerve latency testing is less valuable than was previously believed, as most patients have identifiable structural damage or muscle degeneration rather than neuropathy.

Clinical presentation

Faecal incontinence after childbirth

Structural damage to the anus and new bowel symptoms are common after vaginal delivery.⁷ Anal endosonography has shown that 30% of women have occult structural damage affecting one or both anal sphincter muscles after their first vaginal delivery.⁸ Around a third of these women develop anal incontinence or urgency. Incontinence is usually for gas, but in a few women it is for solid or liquid stool. Of

Advances in faecal incontinence

- Better understanding of the long term disability associated with congenital anorectal disorders
- Less emphasis on pelvic neuropathy and more attention to the newly recognised degenerative sphincter myopathy and to the frequent and dramatic nature of structural damage during childbirth
- Development of new surgical procedures
- Recognition of the value of "alternative" behavioural therapies in patients with no structural sphincter damage

the 0.5% to 1% of women who have a recognised third degree tear during vaginal delivery, 85% have residual structural sphincter damage and half of these have symptoms despite a primary repair at the time of delivery.⁹ The commonest risk factors for damage during childbirth are the use of forceps, a large baby, a long second stage of labour, and occipitoposterior presentation of the fetus.

Occult anal sphincter damage can exist even though the perineum is intact, and digital examination may be misleading about muscle strength and sphincter integrity.¹⁰ For these reasons manometric assessment of function, together with endosonographic assessment of structure, is necessary to identify the abnormality.

Faecal incontinence after surgery

In a referral centre, sphincter damage after anal surgery is the commonest cause of incontinence after obstetric trauma. Incontinence may be unavoidable—for example, after complex anal fistula surgery. It may also occur as an unexpected complication of a simple operation—for example, after internal anal sphincter damage during haemorrhoidectomy (figs 1 and 2). Lastly, incontinence may occur when the sphincter is divided as part of surgical treatment—for example, after division of the lower internal anal sphincter in chronic anal fissure. In one series of 829 patients who had a sphincterotomy for chronic anal fissure, 35% had

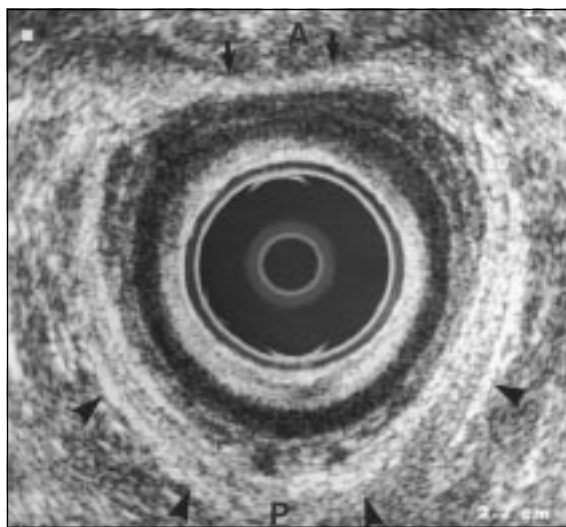


Fig 1 Ultrasound scan showing a normal anal canal. The ultrasound probe is central; the dark ring is the internal anal sphincter and the white structure surrounding it is the external anal sphincter muscle (arrows indicate outer limits). A = anterior, P = posterior

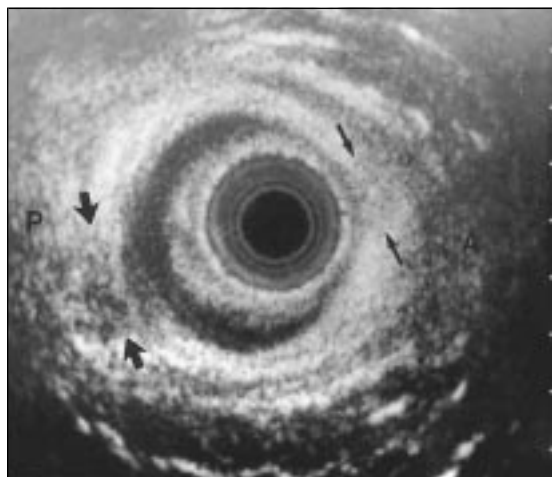


Fig 2 Ultrasound scan showing the anal canal of a 50 year old woman with passive faecal soiling. There is anterior (A, thin arrows) damage to the internal and external sphincter muscles related to childbirth and posterior (P, thick arrows) damage to the internal and external sphincter muscles related to haemorrhoidectomy

lack of control for flatus, 22% soiled, and 5% had major episodes of incontinence.¹¹ The recent finding that treatment with topical glyceryl trinitrate or other nitric oxide donors can lower anal pressure and cause fissure healing suggests that pharmacological therapies will become first line treatment for chronic anal fissure.^{12 13}

Internal sphincter dysfunction

Structural damage is the commonest cause of sphincter weakness¹⁴; where this is absent, isolated degeneration of the smooth muscle of the internal anal sphincter is the commonest cause of soiling.¹⁵ This condition affects both men and women, often in middle age. The resting anal pressure is low, and endosonography often shows that the internal sphincter is thin and fibrotic. Pudendal nerve damage, previously believed to be responsible for most cases of idiopathic faecal incontinence, is rare. Weak or structurally damaged smooth muscle tissue of the internal anal sphincter cannot be treated surgically. Most of these patients respond to treatment with loperamide.

Patients with progressive systemic sclerosis¹⁶ or chronic idiopathic intestinal pseudo-obstruction, and those who have had radiotherapy for cervical or other pelvic neoplasms, may develop degeneration and fibrosis of the internal anal sphincter leading to passive faecal incontinence. The problem is sometimes compounded by diarrhoea.

Neurological disease

Many patients with neurological disease have faecal incontinence that may impair their quality of life as much as the primary disorder. In these patients, any non-neurological components such as damage during childbirth, side effects of drugs, or coexistent behavioural disorders need to be identified.

Faecal incontinence occurs in about 50% of patients with multiple sclerosis, and in 25% it occurs at least weekly.¹⁷ In a series of patients with spinal injuries, 61% had faecal incontinence, and in 11% it occurred at least weekly.¹⁸ Patients rated their bowel symptoms not far below the loss of mobility in terms of distress.

Faecal incontinence also occurs in patients with long-standing neuropathy secondary to diabetes mellitus.

Congenital disorders

Faecal incontinence is a major social disability in 90% of patients with spina bifida, and about 50% soil regularly.¹⁹ Bowel programmes with regular bowel evacuation triggered by reflex can lead to improved continence in some.²⁰ We are now seeing the long term results of surgery in infancy or childhood for congenital disorders. Incontinence occurs in 50% to 80% of adolescents and adults treated for anal atresia,²¹ regardless of whether the operation was a posterior sagittal anorectoplasty or a "pull through" procedure.²² Faecal incontinence can be a considerable source of distress; in one study 20-30% of 160 children and adolescents with congenital malformations had emotional problems.²³ In 60 children who had been operated on for Hirschsprung's disease a mean of nine years previously, 53% had serious faecal soiling and 27% less severe soiling, and the prevalence did not seem to decrease with age.²⁴

Disorders in children and adolescents

Children with a normal anal sphincter can pass stool inappropriately (encopresis), or they may leak faeces as a result of faecal impaction with overflow, which usually requires disimpaction and treatment with laxatives. When laxatives are used as the main treatment, biofeedback does not offer additional benefit,²⁵ but it may be a useful alternative primary treatment. Biofeedback aims to teach the patient pelvic floor coordination and also incorporates disciplined toiletting behaviour. In one study, almost half the children who had failed with laxative treatment recovered with biofeedback.²⁶ Constipation in most children will resolve with time, but a small group with a widely dilated rectum and recurrent faecal impaction remain constipated as adolescents and young adults. While the cause may be behavioural in some of these children, others may have a poorly defined neuromuscular disorder of the distal gut. They require faecal disimpaction and long term laxative treatment.²⁷

Miscellaneous causes of faecal incontinence

- Rectal prolapse is usually associated with faecal incontinence, and although this improves with prolapse repair, it often persists to some degree because of internal anal sphincter weakness.²⁸ External rectal prolapse is not always evident
- Faecal urgency and incontinence are hidden symptoms of inflammatory bowel disease. Rectal inflammation and irritability may mean that the problem is as great for patients with distal colitis as for those with extensive colitis. Long term results of the ileoanal reservoir have shown that 10-20% of patients have some leakage during the day and up to 25% have some leakage at night, but gross incontinence is uncommon
- Unwanted anal penetration can cause internal, and occasionally external, damage to the anal sphincter and should be asked about when there is no obvious cause for sphincter disruption²⁹

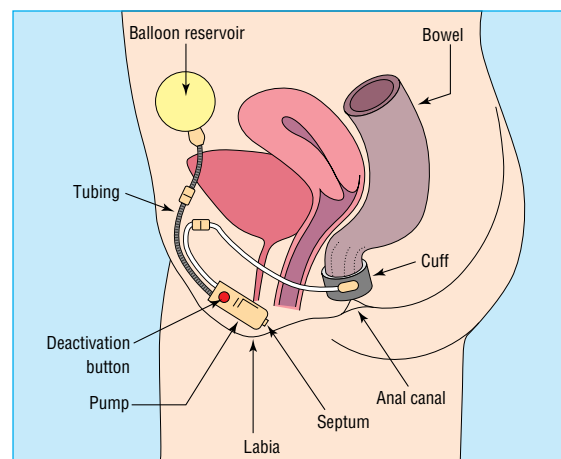


Fig 3 Artificial anal sphincter consisting of fluid reservoir, valve, and cuff which is placed around the anal canal.

Advances in surgical treatment

Simple surgical repair

Many patients with a single sphincter tear from obstetric trauma, and some with discrete damage from other types of injury such as motor accidents and after fistula surgery, can be cured by surgical repair. In 80% of patients with obstetric damage, anterior overlap repair of the external sphincter resolves symptoms, and benefit is seen in middle aged women as well as those who present soon after childbirth.³⁰

Complex surgery

Muscle transposition—When the anal sphincter muscles are damaged irreparably, other striated muscles such as the gracilis or gluteus can be surgically "wrapped" around the anal canal to increase resting tone. The most tested of these operations is the gracilis neosphincter, which requires chronic electrical stimulation to maintain active muscle contraction. In a study of 52 patients, 73% were continent after a median follow up of two years. The procedure was associated with improved bowel frequency, ability to postpone defecation, less anxiety, and improved quality of life.³¹ The gracilis neosphincter has also been used in patients undergoing abdominoperineal excision for low rectal cancer. The continence rate after this procedure is high, although the complication rate in one series was approximately 40%.³² The operation has a high failure rate in patients with congenital abnormalities, probably because these patients have abnormal hind gut function. Although the gracilis neosphincter procedure has good results in expert hands,³¹ the learning curve for surgeons is steep and the operation is associated with many pitfalls and high morbidity.³³

Artificial sphincter—An alternative to the use of transplanted striated muscle is a synthetic sphincter device. This consists of a silastic inflatable cuff, a valve which allows the cuff to deflate for a few minutes for the purpose of defecation, and a reservoir (fig 3). The artificial urinary sphincter has been implanted around the anal canal, but a purpose designed anal device is currently undergoing evaluation. These devices may be best for patients with anal leakage, rather than those with faecal urgency and high bowel pressures.

Sacral nerve stimulation—Electrical stimulation of sacral nerve roots has been reported to restore continence in patients with structurally intact muscles.³⁴ An electrode inserted into the S3 sacral foramen provides low grade stimulation via a chronic stimulator implanted under the anterior abdominal wall. This technique does not stimulate the motor nerves sufficiently to cause contraction, but it is thought to act by facilitating sphincter contraction and changing the sacral reflexes.

Antegrade colonic irrigation—Some patients with extensive sphincter damage, neurological disease, previous anorectal abnormalities, spina bifida, or sacral agenesis have poor control over bowel function. Daily enemas to empty the distal bowel, followed by loperamide to prevent soiling, are sometimes effective, but if they are not, colonic emptying can be achieved by antegrade colonic irrigation. The appendix is brought out to the skin and is used for catheter entry—the so called antegrade continence enema (ACE) procedure. This technique has been used mainly in children, and although it is effective initially, long term problems include stenosis, the need for refashioning, and resistance to laxative or irrigation.³⁵ An alternative means of antegrade colonic irrigation is via a transverse colonic conduit incorporating an intussusception valve and cutaneous aperture.³⁶

Colostomy—When other surgical measures have failed or are inappropriate and the patient has uncontrolled incontinence, the creation of a colostomy can still provide major benefit to some.

Advances in non-surgical treatment

Drugs

When symptoms are mild, infrequent, and not due to impaction with overflow, treatment with loperamide or codeine phosphate is safe and often very effective. These drugs can benefit patients with soiling or passive leakage and those with urge incontinence. Loperamide is safe, can be titrated to achieve relief of symptoms, and can be used as required or continuously. Both drugs reduce motility of the large bowel and increase absorption, making the stool more formed.

Direct sphincter electrical stimulation

Although anecdotal reports about direct transcutaneous electrical stimulation of the sphincter have been positive, there are no studies in which the cause of incontinence has been well defined and patients have been followed up for a long period. Given the success of this treatment in urinary incontinence, however, it deserves further evaluation.

Biofeedback for faecal incontinence

Similar criticisms can be levelled at the studies on biofeedback retraining. Most of the 40 published studies describing the results of pelvic floor biofeedback for faecal incontinence suggest an improvement rate of 60-70%.³⁷ However, selection criteria were poor, the sphincter abnormalities not well defined, and the follow up periods short. Biofeedback aims to condition patients to be more sensitive to a stimulus distending the rectum and to heighten the voluntary striated muscle sphincter response, with the patient watching

or listening to a feedback signal of the level of external sphincter contraction. We do not yet know which patients respond best to this type of treatment.

Behavioural retraining

In intellectually disabled people, a behavioural programme with prompted sitting on the lavatory, rewards for evacuation, and attention to stool consistency using fibre supplements or a bulking agent can produce “normal” bowel function.³⁸

Conclusion

An awareness of the high prevalence and predisposing factors for the development of faecal incontinence, together with a multidisciplinary approach to treatment, can lead to relief from this distressing symptom in most patients.

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Lesson of the week

Unusual case of choriocarcinoma occurring 12 months after delivery

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Choriocarcinoma should be considered in women of childbearing age with acute haemorrhage occurring even years after a normal birth

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Choriocarcinoma is a malignant tumour that arises from trophoblastic epithelium; it has an incidence of 0.05 to 0.23 per 1000 live births.¹ The most important risk factor, a history of hydatidiform mole, is present in only half of cases, and many cases occur after a normal full term pregnancy.² We report the case of a young woman with choriocarcinoma who presented with an intraperitoneal haemorrhage from splenic metastases. She also developed multiple pulmonary tumour emboli and haemorrhage from tumour infiltration. She is the first patient in the United Kingdom, so far as we are aware, to survive ventilation for this disease.

Case report

Twelve months before she presented with choriocarcinoma this 32 year old woman had given birth by normal vaginal delivery to a boy, the second of two children. At both 3 and 6 months before presentation she had had two transient ischaemic episodes (attributed to protein S deficiency) affecting the right middle cerebral artery. Shortly after beginning warfarin treatment for these attacks she had a left parieto-occipital haematoma; it was evacuated but she was left with right hemiparesis and she developed epilepsy after surgery. The bone flap was not replaced.

Two months after surgery she was admitted to hospital after several days of upper abdominal discomfort and nausea. She was mildly tachycardic, normotensive, and tachypnoeic. She had a diffusely tender abdomen and smooth hepatomegaly of 4 cm. Laboratory analysis showed modest neutrophilia, slightly raised serum urea, γ -glutamyl transaminase, and alkaline phosphatase concentrations. Within hours she became anaemic and went into shock. She subsequently needed a blood transfusion. She had no external blood loss, and upper gastrointestinal endoscopy gave normal results.

The next day abdominal ultrasonography and aspiration identified free intraperitoneal blood, numer-

ous focal liver lesions, and a subcapsular splenic haematoma. A pelvic ultrasound scan was normal. Angiography showed the liver lesions to be vascular, and a vascular pelvic lesion was thought to be either the normal uterus or an ovarian tumour. The next day multiple focal masses in the lungs that had not been seen in a chest x ray film and another mass in the right kidney were identified on computed tomography. The splenic lesion was confirmed as the source of continuing haemorrhage. Vascular metastases from an

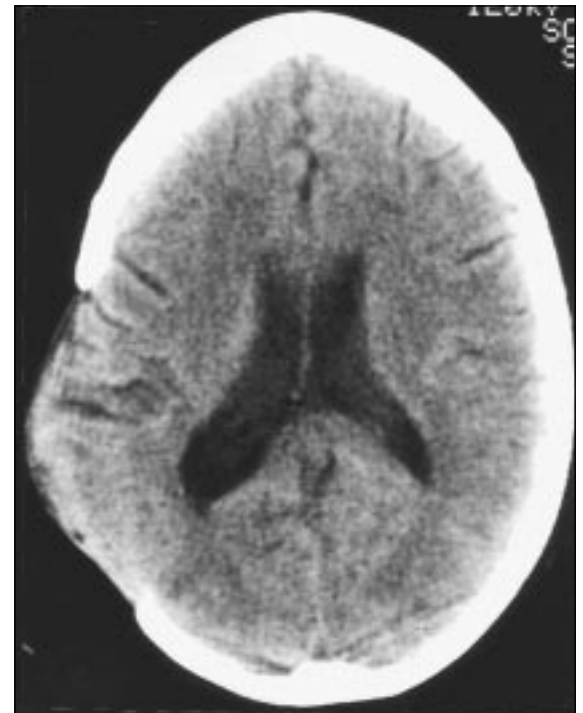


Fig 1 Computed tomogram showing extracranial extrusion of right cerebral hemisphere as result of cerebral oedema. Extrusion is through site of evacuation of previous intracranial haematoma, which was not covered with bone flap after surgery

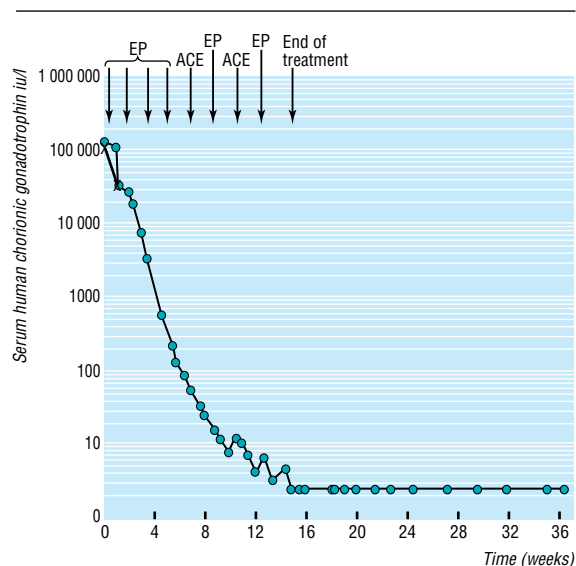


Fig 2 Serum concentrations of human chorionic gonadotrophin during drug treatment in patient with gestational choriocarcinoma. Doses were adjusted to account for grossly deranged liver function (EP=etoposide, cisplatin; ACE=dactinomycin, cyclophosphamide, etoposide)

undetermined primary or multiple haemangioma were considered the likely diagnosis, and she was transferred to the regional liver unit for further tests.

Biopsy of the liver guided by ultrasonography was initially unsuccessful. She developed haemoptysis and sudden opacity of the right lung in a chest x ray film. Bronchoscopy showed multiple bleeding lesions in the main bronchi. The diagnosis of choriocarcinoma was confirmed on biopsy of the liver guided by computed tomography and by serum concentrations of β human chorionic gonadotrophin of 20 000 IU/l. The patient was transferred to London for treatment.

By the time of transfer she had developed respiratory failure as a result of extensive pulmonary infiltration by the tumour, pulmonary oedema, and pleural effusion. She also had ascites and grossly abnormal hepatic function. Once respiratory function was stabilised, chemotherapy was started with reduced doses of etoposide and cisplatin to minimise the risk of the acute tumour lysis syndrome. Within a few days, however, her respiratory function had deteriorated as a result of tumour embolisation and pulmonary infarction and she required admission to the intensive care unit for ventilation by continuous positive airways pressure. By then she was grossly jaundiced as a result of both deteriorating hepatic function and intravascular haemolysis from coagulopathy. After two courses of etoposide and cisplatin her respiratory and hepatic functions began to improve. However, her coagulopathy proved difficult to control, and she developed extensive intrapulmonary haemorrhaging, which necessitated intubation and ventilation. While she was being ventilated she developed gross cerebral oedema, which would have proved fatal were it not for the expansion afforded by the absent bone flap (fig 1). Her stay in the intensive care unit was further complicated by the adult respiratory distress syndrome, Gram negative septicaemia, and pseudomembranous colitis. After six weeks and four courses of chemotherapy her coagulopathy had resolved, and her respiratory and

liver functions were improving sufficiently to permit extubation and transfer to the ward.

She began treatment with dactinomycin, cyclophosphamide, and etoposide alternating with etoposide and cisplatin every two weeks. The chemotherapy regimen was selected to avoid the potential hepatotoxicity of methotrexate, which is difficult to administer in cases of gross ascites because it is excreted renally. Serum concentrations of human chorionic gonadotrophin returned to normal after three cycles (fig 2). The patient had another pulmonary embolism, for which she was treated with low molecular weight heparin. She also developed transient cranial diabetes insipidus. Renal tubular dysfunction induced by the cisplatin caused persisting hypomagnesaemia which led to problems in controlling her epilepsy. Additionally, malabsorption compounded her electrolyte disturbances and her weight dropped to 44 kg. She gradually improved, though persisting hepatic dysfunction and ascites (probably due to portal hypertension from scarring around previous hepatic metastases) remained a problem.

Review of the results of coagulation studies from the time of her transient ischaemic attacks showed a modest reduction in both protein S and factor VII concentrations which could be explained by early liver dysfunction. More recent testing for thrombophilia showed normal results.

More than a year after diagnosis computed tomography showed persisting large intrahepatic cysts, at least one of which later ruptured intraperitoneally (fig 3). Her liver function has since improved considerably, and she has now returned to normal activity. Concentrations of human chorionic gonadotrophin remain below detection thresholds, which indicates continuing remission.

Discussion

This case illustrates the need for prompt diagnosis and awareness of the protean manifestations of choriocarcinoma. Choriocarcinoma may form brain emboli, which cause cerebral ischaemia⁵ and possibly intracere-

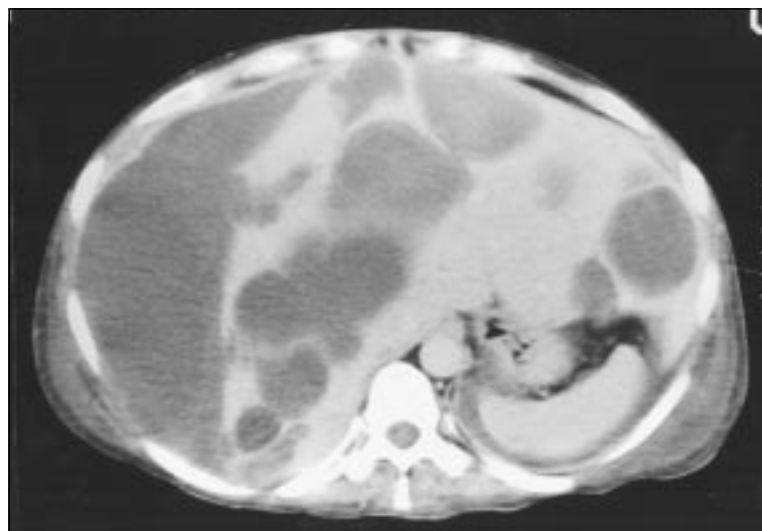


Fig 3 Computed tomogram of abdomen more than one year after diagnosis of choriocarcinoma. Darker areas show multiple large, intrahepatic, fluid filled cysts at site of previous hepatic metastases

ebral haemorrhage, especially in patients who are not taking anticoagulants. Unfortunately, the clot that had been evacuated from our patient was not available for retrospective histological examination. Her neurological problems, however, were probably the early signs of disseminated malignancy that went unrecognised rather than an independent disease process. We believe that the transient ischaemic attacks were caused by cerebral tumour emboli; computed tomography gave negative results, and the coagulation disturbance was minimal at that stage. The massive haemorrhage that occurred later could have been caused by a combination of cerebral metastasis and a deficiency in vitamin K dependent factors that was aggravated by treatment with warfarin.

The primary symptoms of choriocarcinoma are gynaecological in only 50% to 60% of postpartum cases; the most common symptoms are dyspnoea, neurological symptoms, and abdominal pain.³ The delay between birth and presentation may exceed that in our case: intervals of several years have been recorded.³ Choriocarcinoma metastasises readily—especially to lung, brain, and liver—and it can present with widely disseminated disease. The metastases are highly vascular, and cerebral and intestinal haemorrhage are recognised presentations, though splenic bleeding has been reported only rarely.^{6,7} Haemorrhage may be massive and life threatening, making early tumour diagnosis and vigorous support critical. Diagnosis is ideally on the basis of raised concentrations of human chorionic gonadotrophin and appropriate histology (to exclude other tumours that produce human chorionic gonadotrophin, such as those of the lung and stomach). Adequate tissue may

be difficult to obtain, so diagnosis may have to be on the basis of an appropriate clinical setting and high concentrations of human chorionic gonadotrophin. Serial testing of serum concentrations is essential in monitoring treatment and confirming remission. Monitoring of human chorionic gonadotrophin concentrations should be continued for life as late recurrences may occur.

Choriocarcinoma should be considered in any woman of reproductive age with widespread vascular lesions, metastases of unknown origin, or cerebral or intra-abdominal haemorrhage. Serum concentrations of human chorionic gonadotrophin should be measured in all such patients.⁴ Cure rates are high with chemotherapy even in widely disseminated disease, but treatment of choriocarcinoma after a live birth is associated with higher mortality. Eliminating the disease becomes more difficult as the time between pregnancy and the onset of treatment increases.²

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When I use a word...

Spurious words

Lexicographers don't always get it right. James Boswell tells how a woman challenged Samuel Johnson to explain why, in his dictionary of 1755, he had defined "pastern" as "the knee of a horse." [Johnson amended this error in the subsequent abridged version of 1756, in which pastern is correctly defined as "that part of the leg of a horse between the joint next the foot and the hoof."] Johnson's reply was typically robust: "Ignorance, madam, pure ignorance."

Among the almost 300 000 entries in the second edition of the *Oxford English Dictionary* a tiny minority (fewer than 400) are distinguished by being completely enclosed in square brackets. These rarities are spurious words, which James Murray originally included in the first edition to correct errors that had been perpetrated in other dictionaries—a sort of one-upmanship on the editor's part. Most of these spurious words arose through misprints or through the errors or misreadings of copyists or translators. For example, epidemic for epidemic, owing to the similarity between x and p in early handwriting.

Some, however, have interesting stories. For instance, dentize, defined as "to cut new teeth," was a misreading of "dentire" in *Sylvia* (1626), in which Francis Bacon recounted how "They tell a tale of the old Countesse of Desmond, who lived till she was seven-score yeares old, that she did Dentire, twice, or thrice; Casting her old Teeth, and others Comming in their Place." An unlikely tale? Well, the term "third dentition" has been used to

describe the eruption of teeth after the loss of all permanent teeth. Ooë (*Okajimas Fol Anat Jap* 1969;46:243-51) referred to some reported cases (attributed, however, to delayed eruption of supernumerary teeth) and also described a histological structure that he proposed was the precursor of a potential third dentition in man. Still, "third dentition" is more commonly used to describe false teeth.

A well known example in modern times of a spurious scientific word is "dord," defined in *Webster's New International Dictionary* as "density." The error was explained by the editor of *Webster's Third New International*, Philip Gove, in an article in *American Speech* (1954;29:136-8). An expert had sent the publishers a definition slip bearing the words "D or d, cont/density." "Cont," short for continued, meant that this would be one in a series of entries for the abbreviation "D," but it was misinterpreted to mean that the "D or d" should be read as a continuous string. And so "dord" was created. Gove thought it a pity that it was spurious, "for why shouldn't dord mean density?"

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