GUIDELINES
Neonatal jaundice: summary of NICE guidance

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This is one of a series of *BMJ* summaries of new guidelines based on the best available evidence; they highlight important recommendations for clinical practice, especially where uncertainty or controversy exists. Further information about the guidance, a list of members of the guideline development group, and the supporting evidence statements are in the full version on bmj.com.

Neonatal jaundice is one of the most common conditions needing medical attention in newborn babies. About 60% of term and 80% of preterm babies develop jaundice in the first week of life, and about 10% of breast fed babies are still jaundiced at age 1 month.[1] Neonatal jaundice is generally harmless, but high concentrations of unconjugated bilirubin may occasionally cause kernicterus (permanent brain damage). This is a rare condition (about seven new cases each year in the United Kingdom[2]) and sequelae include choreoathetoid cerebral palsy, deafness, and upgaze palsy. Jaundice can also be a sign of serious liver disease, such as biliary atresia, the prognosis for which is better if it is treated before age 6 weeks.[3] Early recognition of jaundice is vital for treatment of any underlying condition and for the appropriate use of phototherapy, which can safely control bilirubin concentrations in most cases.

This article summarises the most recent recommendations from the National Institute for Health and Clinical Excellence (NICE) on how to diagnose and treat jaundice in newborns up to 28 days old.[4]

Recommendations

NICE recommendations are based on systematic reviews of best available evidence and explicit consideration of cost effectiveness. When minimal evidence is available, recommendations are based on the Guideline Development Group's experience and opinion of what constitutes good practice.

Information for parents and carers

Offer parents or carers information about neonatal jaundice that is tailored to their needs and expressed concerns, taking care to avoid causing unnecessary anxiety; discuss verbally and back up the discussions with written information.

The information might include the following:
- The fact that neonatal jaundice is common, and reassurance that it is usually transient and harmless
- Risk factors for significant hyperbilirubinaemia (a rise in the serum bilirubin concentration to a level requiring treatment)
- How to check the baby for jaundice
- What to do if they suspect jaundice
- The importance of recognising jaundice in the first 24 hours and of seeking urgent medical advice
- The importance of checking the baby's nappies for dark urine or pale, chalky stools
- Reassurance that breast feeding can usually continue.

Care for all babies
- Check if babies have the following risk factors for developing significant hyperbilirubinaemia:
  - Visible jaundice in the first 24 hours of life
  - A gestational age of <38 weeks
  - A previous sibling who had neonatal jaundice needing phototherapy, and/or
  - A mother who intends to breast feed exclusively.
- Ensure babies with risk factors for significant hyperbilirubinaemia receive an additional visual inspection by a healthcare professional during the first 48 hours of life.
- Examine all babies for jaundice at every opportunity, especially in the first 72 hours.
- When inspecting for jaundice:
  - Check the naked baby in bright and preferably natural light
  - Examine the sclerae, gums, and blanched skin, in babies of all skin tones.

Babies with jaundice
Measure bilirubin concentrations in all babies with jaundice—do not rely on visual inspection alone to estimate the bilirubin concentration.

When measuring the bilirubin concentration:
- Use a transcutaneous bilirubinometer (a non-invasive device applied to the baby's forehead to measure bilirubin concentrations) in babies with a gestational age of 35 weeks or more and postnatal age of more than 24 hours
- If a transcutaneous bilirubinometer is not available, measure the serum bilirubin
- If a transcutaneous bilirubinometer measurement indicates a bilirubin concentration >250 µmol/l, check the result by measuring the serum bilirubin concentration
- Always use a serum bilirubin measurement to determine the bilirubin concentration in *(a)* babies with jaundice in the first 24 hours of life; *(b)* preterm babies less than 35 weeks' gestational age; and *(c)* babies receiving phototherapy
- Do not use an icterometer (a non-invasive device allowing comparison of skin colour to a reference strip).

Management of hyperbilirubinaemia
Use bilirubin concentration to determine management (see figure and the treatment threshold graphs in the NICE guidance[4]).

Age (hours)	Bilirubin level (µmol/l) at which the relevant action below should be taken			
0			›100	›100
6	›100	›112	›125	›150
12	›100	›125	›150	›200
18	›100	›137	›175	›250
24	›100	›150	›200	›300
30	›112	›162	›212	›350
36	›125	›175	›225	›400
42	›137	›187	›237	›450
48	›150	›200	›250	›450
54	›162	›212	›262	›450
60	›175	›225	›275	›450
66	›187	›237	›287	›450
72	›200	›250	›300	›450
78		›262	›312	›450
84		›275	›325	›450
90		›287	›337	›450
≥96		›300	›350	›450
	Repeat bilirubin measurement within 6-12 hours	Consider phototherapy (repeat bilirubin measurement within six hours)	Start phototherapy	Perform an exchange transfusion unless the bilirubin level falls below threshold while the treatment is being prepared

Consensus based bilirubin thresholds for the management of babies of 38 weeks or more gestational age with hyperbilirubinaemia

During phototherapy
- Using clinical judgment, encourage short breaks for breast feeding, nappy changing, and cuddles unless bilirubin concentrations are approaching the exchange threshold or are rising by more than 8.5 µmol/l per hour.
- Continue lactation and feeding support.
- Do not routinely give additional fluids or feeds.
  Maternal expressed milk is the additional feed of choice if available and when additional feeds are indicated.

Prolonged jaundice
Prolonged jaundice is jaundice that lasts more than 14 days in term babies and more than 21 days in preterm babies. In babies with prolonged jaundice:
- Look for pale, chalky stools and/or dark urine that stains the nappy
- Measure the conjugated bilirubin (as raised concentrations may indicate liver disease)
- Do a full blood count
- Determine the blood group of the mother and the baby and do a direct antigen test (in the baby); interpret the result and likelihood of Rh disease or other haemolytic disease, taking account of the strength of reaction and whether mother received prophylactic anti-D immunoglobulin during pregnancy

- Arrange a urine culture
- Ensure that routine metabolic screening (including screening for congenital hypothyroidism) has been done.
  Follow expert advice for babies with a conjugated bilirubin concentration of >25 µmol/l because this level may indicate serious liver disease.

Overcoming barriers
The guideline recommends that babies at risk of hyperbilirubinaemia be further assessed within 48 hours. As most healthy term babies are at home by this stage, this requirement for a home visit is also an opportunity to deliver breastfeeding support and other advice.

More widespread use of transcutaneous bilirubinometry, recommended as a first line test in assessing jaundice, has implications for community workers and commissioners or purchasers. A health economic review in the guideline's development showed that investment in these devices would be cost effective if only one or two cases of kernicterus a year were averted by their use.[4]

The use of phototherapy currently varies widely.[5] The guideline's treatment threshold graphs provide guidance on starting phototherapy and on considering exchange transfusion, with advice on when to measure the serum bilirubin concentration and on continuation and cessation of phototherapy.

proceedings in cases of kernicterus. This work is undertaken outside of NHS time. All other authors have had no relationships with companies that might have an interest in the submitted work; (3) No spouses, partners, or children with financial relationships that may be relevant to the submitted work; and (4) No non-financial interests that may be relevant to the submitted work.

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EASILY MISSED?

Biliary atresia

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This is a series of occasional articles highlighting conditions that may be commoner than many doctors realise or may be missed at first presentation. The series advisers are Anthony Harnden, university lecturer in general practice, Department of Primary Health Care, University of Oxford, and Richard Lehman, general practitioner, Banbury. If you would like to suggest a topic for this series please email us (easilymissed.bmj@bmjgroup.com).

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Previous Easily Missed articles
❯ Pulmonary embolism (*BMJ* 2010;340:c1421)
❯ Lichen sclerosus (*BMJ* 2010;340:c731)
❯ Constipation in people with learning disability (*BMJ* 2010;340:c222)
❯ Long QT syndrome (*BMJ* 2010;340:b4815)
❯ Ovarian cancer (*BMJ* 2009;339:b4650)

Biliary atresia is a rare (one in 17 000 in the United Kingdom[1]) but serious liver disorder that presents with jaundice in the first few weeks of life in apparently well infants. About 50 cases of biliary atresia occur each year in term babies who are born healthy and have usually had normal antenatal scans.[2] The lumen of the biliary tree is obliterated by an inflammatory cholangiopathy, which obstructs the flow of bile from the liver to the intestine, resulting in progressive liver damage. Early surgery to reconstruct the biliary tree may reduce further damage and prevent the need for liver transplantation.

Why is it missed?

The diagnosis of neonatal liver disease, including biliary atresia, may be missed because of confusion with either physiological jaundice or breast milk jaundice (which is common and is thought to occur in as many as 60% of normal term infants[3]). Physiological jaundice should last only for two to three days in term infants, whereas breast milk jaundice can last for as long as 12 weeks.[4] Both forms of jaundice are associated with a rise in unconjugated bilirubin.

Prolonged jaundice, which persists beyond 14 days in term infants and 21 days in preterm infants, requires further investigation for a pathological cause, even if the mother is breast feeding.

Most infants with biliary atresia will appear well in the first few weeks of life, and there is usually no family history of liver disease.

Why does this matter?

The restoration of bile flow from the liver to the bowel is essential to prevent further scarring of the liver. This

CASE SCENARIO

A mother presents to her general practitioner with her 3 week old son with mild jaundice; he is her first baby and fully breastfed. The mother was reassured. At the examination at age 8 weeks, the GP notices he has mildly icteric sclera and, on questioning, the mother states that his stools are cream coloured and his urine very yellow. The GP immediately refers the infant to the local paediatric unit, where further testing shows conjugated bilirubin concentrations of 120 μmol/l and leads to a final diagnosis of biliary atresia. The Kasai portoenterostomy carried out at age 9 weeks was unsuccessful, and he had a liver transplant at age 6 months.

requires a palliative Roux-en-Y portojejunostomy, known as a Kasai procedure. The earlier the Kasai procedure is carried out, the more likely it is to be successful (defined as a normal bilirubin concentration within 6 months of the procedure) with 60% of babies achieving good bile flow.[5] The diagnosis is made very late (beyond 100 days of age) in about 8% of cases[6]; when the diagnosis is this late, a Kasai procedure is unlikely to be successful owing to advanced liver damage or cirrhosis. In children who present late or in whom the operation is unsuccessful (the success rate decreases with increasing age at the time of the Kasai procedure[7]), liver transplantation within the first year of life is the only option.

How is it diagnosed?
Clinical features

On examination the baby will have icteric sclera but will usually appear well unless the diagnosis is delayed, when signs of chronic liver disease (hepatomegaly, excessive bruising, ascites, and splenomegaly) will become obvious. Biliary atresia can occur in preterm infants, and in 20% of all cases biliary atresia is associated with cardiac malformations, polysplenia, and situs inversus, which will be identified on ultrasound scan.[8] The baby may be excessively hungry as a result of the poor absorption of long chain fat (secondary to the lack of bile in the intestine) and the high catabolic demand of liver disease.

HOW COMMON IS IT?

Biliary atresia is the most common indication for liver transplantation in childhood

The younger the baby at the time of a Kasai portoenterostomy, the more likely the procedure is to be successful and avoid the need for liver transplantation

When the diagnosis of biliary atresia is made very late (beyond 100 days of age) children usually need a liver transplant in the first year of life

Investigations

In all term babies with prolonged jaundice examine the colour of the stool and urine.[9] During the neonatal period the colour of stools varies but will become gradually paler in infants with biliary atresia,[10] and the urine colour will be yellow (it is normally colourless). Refer the infant to a local paediatric unit for a split bilirubin blood test (measuring conjugated and unconjugated bilirubin). Infants with prolonged physiological jaundice or prolonged breast milk jaundice (in total, about 2% of live births) will have a rise in unconjugated (indirect) bilirubin, whereas those with liver disease such as biliary atresia will have a rise in conjugated (direct) bilirubin.

If the conjugated bilirubin is raised above normal (normal conjugated bilirubin <20 µmol/l) refer the infant to the local paediatric department for further liver function tests.

Refer all babies with suspected biliary atresia to a paediatric liver unit. A substantial rise in alkaline phosphatase and γ-glutamyltransferase, with a variable rise in alanine aminotransferase and aspartate aminotransferase, may suggest biliary atresia. The synthetic function of the liver (assessed by measuring albumin levels and prothrombin time) will be normal unless diagnosis is delayed, resulting in decompensated cirrhosis. Further testing in a specialist unit will include an ultrasound scan (showing a small gallbladder and hepatomegaly without biliary dilatation), a radionucleotide excretion scan or endoscopic retrograde cholangiopancreatography (confirming lack of flow of bile from the liver into the bowel), and a liver biopsy (showing biliary obstruction and fibrosis while excluding other conditions such as neonatal hepatitis). Intraoperative cholangiography is the definitive test for biliary atresia as it displays the abnormal biliary tree.[11]
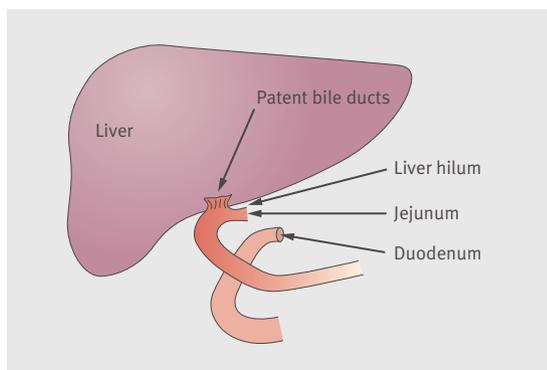
Kasai portoenterostomy with bowel attached to the liver hilum allowing unaffected patent bile ducts to drain into the intestine. Adapted from a line drawing supplied by Deirdre Kelly

How is it managed?

Children with biliary atresia initially need nutritional support with a high energy, high level, medium chain triglyceride feed such as Caprilon (SHS International) and oral supplementation with fat soluble vitamins (A, D E, and K) as biliary atresia impairs absorption of these vitamins.[11]

The Kasai portoenterostomy is usually performed at the same time as the intraoperative cholangiography and may restore the flow of bile (in 60% of cases) by removing the scarred proportion of the biliary tree and attaching a Roux-en-Y loop of bowel to the cut surface of the liver hilum so enabling small patent bile ducts to drain into the intestine (figure). Postoperative steroids are given to reduce inflammation and encourage bile flow. Postoperative complications include ascending cholangitis which is prevented by a rotating course of prophylactic antibiotics. Recurrent cholangitis is an indication for liver transplantation, and so prevention and prompt treatment is essential.[12] The presentation of ascending cholangitis is subtle but includes pyrexia, irritability, vomiting and abdominal pain with elevation of inflammatory markers and serum bilirubin.

All children should be followed up long term in a specialist liver centre so that complications (such as poor growth, vitamin deficiency, and the development of portal hypertension) and the need for liver transplantation can be monitored.

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A PATIENT'S JOURNEY
The war in my head

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This is one of a series of occasional articles by patients about their experiences that offer lessons to doctors. The *BMJ* welcomes contributions to the series. Please contact Peter Lapsley (plapsley@bmj.com) for guidance.

Linda Dauwerse, 29, had a brain haemorrhage at 14. She describes her journey to recovery through extracts from the diary she kept

"*I am in hospital because I had a stroke. I wanted to write often—so many things happened—but couldn't because I had to lie flat. Actually I do not realise how serious it was, because right now I feel much better.*"

I was 14 years old when I had a brain haemorrhage, and the following weeks were marked by mixed feelings. Sometimes I felt grateful and happy I was still alive. At other moments I felt sad about the loss of my daily routines, and scared. One night in hospital I was convinced I would die. The assistant physician came and sat next to me, which really helped. After admission the fear of dying returned regularly: "*I do not want to die yet. Really I don't. I love life so much. No, I cannot die yet. I am terrified of it. Terribly scared.*" Death was frequently on my mind, and sometimes I felt anxious and lonely, but I did not talk about these feelings.

Unrealistic hope

Fortunately a doctor in Paris was able to treat me, promising a full recovery. He said that after embolisation the AVM (arteriovenous malformation) that had caused the brain haemorrhage would be gone. This made me feel immensely hopeful: "*I will go to Paris tomorrow. Hopefully everything will turn out fine . . . I must be healthy again.*" However, the first embolisation was not completely successful. After the second one, a large part of the AVM was still there. So, my family and I felt disappointed and had to get used to the idea that the recovery might take longer. The whole process was frustrating: "*It drives me crazy. Easily said, 'she should become completely healthy again.' But how do I know this promise will be realised? I just want to be able to have fun again.*" One year after the brain haemorrhage I had the first stereotactic radiosurgery to treat the rest of the AVM. The effects would not be seen until two years after the treatment, so in the meantime I just had to wait and see what happened.

Losing my sport, losing myself

In the first year after the brain haemorrhage I was especially upset because I was told not to do sport any more. My family and friends were more concerned about my life and future. In their nightmares my parents said farewell and buried me. For me, the bad news was that I could no longer play handball because the exercise would put too much pressure on my head.

I felt powerless in directing my life. Life without sport was difficult, and at one point I decided to play handball

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A DOCTOR'S PERSPECTIVE

A cerebral arteriovenous malformation (cAVM) is a vascular clump made up of abnormal blood vessels that are a mixture of true arteries and veins. The central nidus is fed by one or several arteries and drained by one or more major draining veins. There is no intervening capillary bed, and owing to the lack of normal resistance to flow, the arteriovenous shunts transmit arterial pressure to the accommodating veins, causing hypertension in anomalous vessels. The nidal vessels can be extremely fragile and are prone to bleeding.

Cerebral arteriovenous malformations are generally believed to arise during embryonic or fetal development. The genetic transmission patterns of such malformations, if any, are unknown. These malformations are not thought to be inherited, except in the context of a specific hereditary disease, such as hereditary haemorrhagic telangiectasia (Rendu-Osler-Weber syndrome). A new case of arteriovenous malformations may become manifest in 1 per 100 000 persons a year. The total number of cases of symptomatic arteriovenous malformations at any time in the population has been estimated to be at least 18 per 100 000 but is as high as 6 per 1000 (0.6%) in hospital based, postmortem studies.

About two thirds of cerebral arteriovenous malformations are detected only after rupture. Others may become manifest by migraine-like headache or seizures, or occasionally by progressive neurological deficit if the malformations are very large. The incidence peak is at age 30-35 years. The average annual risk of haemorrhage in the subgroup with no previous haemorrhage is about 2%. The rebleeding rate in the subgroup presenting with haemorrhage is between 6% and 18% in the first year. Over the next few years, this rate seems to decrease to 3-4% a year. A previous haemorrhage is a strong and consistent risk factor for further bleeding.

Treatment may be considered because of the high complication rate of haemorrhage: up to 18% mortality and 16-55% neurological deficit. Cerebral arteriovenous malformations are considered suitable for four treatment options, alone or in combination: observation only, surgical excision, endovascular embolisation, and stereotactic radiosurgery. The high risks of rebleeding favour microsurgical excision in patients with previous haemorrhage. Radiosurgery may be the first choice in patients whose cerebral arteriovenous malformation has never bled or when surgery is unsuitable because of location, architecture, or size of the malformation. Although radiosurgery is a non-invasive procedure, with mortality and morbidity rates lower than those associated with open surgery, the obliteration of the malformation is delayed. In 50-95% of cases, obliteration occurs only after two to three years, with the obliteration rate and the timing of the obliteration being highly dependent on the volume of the vascular clump. Endovascular embolisation (occlusion of the blood supply to the malformation with coils, particles, or glue introduced by a radiographically guided catheter) can be helpful before surgery or radiosurgery but is rarely successful alone. For each individual patient a careful balancing of risks is necessary before treatment advice can be given.

John G Wolbers, consultant neurosurgeon and senior lecturer

WHAT CAN MEDICAL PROFESSIONALS DO?

Keep on asking and talking

- To reduce feelings of loneliness, look for implicit worries or problems by asking questions such as: What is important in your life? Can you still do the things you like to do? What kind of alternatives do you have? What do you worry about?
- Talk about death and fears

Keep on listening

- Show an interest in the person and in matters other than medical symptoms
- Listen to the message behind the initial message. Doctors should involve family and friends as these people can help the doctor to understand the patient's symptoms (for example, parents might say that their daughter is always sitting in her room and responding agitatedly if they ask something) and thereby understand the unexpressed needs of the patient
- Pay attention to psychosocial consequences and lower the threshold for seeking assistance (both during acute and after care phases) by providing a contact phone number and information about how to exchange experiences with other patients (in this case, other teenagers with health problems, who may have had similar experiences)

Keep on repeating

- Repeat information about outcomes; do not create false hope and be honest about possible negative side effects

Keep on doing something to try to help

- Help patients and their families and friends:
  - To anticipate new situations
  - To cope with the dynamics of recovery and restoring balance
  - To deal with decision making processes: do not just provide information; listen also to concerns and provide help with difficult decisions
- Make real contact and pay attention to personal needs
- Treat teenagers as partners in care: ask them how they see the situation and search with them for goals and solutions
- Regularly reassess previous medical advice, especially if it has an impact on daily life (for example, advice about exercise levels). Be proactive in telling the person about any changes in your insight or knowledge

again. I went to a training session, came home and told my parents I wanted to start again. But they felt responsible and anxious that I might suffer another brain haemorrhage, and although I felt angry, I gave up the idea of doing sport at that time.

Sometimes my emotions seemed completely empty: "*I don't know what to write because I don't feel anything. I am empty and afraid. Before, I really had inspiration to*

HELPING HANDS DURING MY JOURNEY

Friends and family being present—Family and friends were there for me, unconditionally, listening to me, sometimes confronting me. They supported my personal development and facilitated leading a "normal" life

Boyfriend accepting me—My boyfriend likes me the way I am. He takes me seriously, really listens, and helps me to deal with difficulties

Music—Music helped me to express (unconscious) feelings, to find out what really mattered to me

Exercise—Running, cycling, and walking help to put things in perspective, to create a little distance, which helps me focus on the things that really count

Psychologist—My psychologist helped me to accept myself, to deal with difficult situations, and to improve my life management skills

Attention to needs—The nurse who enabled us to go to the sea and sleep in a hotel instead of the hospital understood our personal needs and our determination to minimise the impact of the treatment

Involvement in decision making process—Dr Wolbers' attitude was open. He involved us in making decisions about treatment, with shared responsibility. And he always gave us "space"—time to consider and to stop treatment if we wanted to

write. Now I don't. I don't feel anything." Music helped me to express my feelings, and my anger found resonance when I listened to the words "*In your head, in your head, zombie, zombie. They are still fighting*" in a song by the Irish band the Cranberries. To me the war in the song was going on in my own head. If I was sad I listened to the words of another song, "*There is no need to argue any more. I gave all I could, but it left me so sore. And the thing that makes me mad, is the one thing that I had. I knew, I would lose you.*" For most people this song might be related to losing someone you love. For me it expressed how I felt; I had lost myself.

"Leave me alone!"

Those days I was not easy to be around. Most of the time my family and friends helped me by being there for me unconditionally, but tensions arose. My parents were worried and wanted to support me, but I didn't always accept their help. I was rebellious and angry. When my mother asked how I was, I responded: "*Leave me alone, stop it, just act normal!*" I loved her, but I felt she limited my freedom and interfered too much. I was the centre of attention, whereas I just wanted to pick up my life as it was before. People were paying attention to me, I felt miserable, alone, and misunderstood: "*I cannot play handball any more, which makes me hate myself because I will become fat. I have fights with my parents, and even my friends do not always understand me. I hate myself as I am now. I wish everything will be just as it was before.*"

After the first stereotactic treatment we were told that the AVM was still there. This came as a complete surprise. We had been convinced I would be cured and did not expect to hear this. We felt betrayed; we had always relied on the doctors and their decisions and had not taken into account that the results might be less positive. I cried all the way back home and shouted: "*I do not want to live like this any more.*" This car journey was a turning point in my disease process. I did not want the illness to dominate my life any longer.

My initial denial of the new situation developed into a fight. One weapon I used to regain control over my life was to eat small portions of healthy food and take strenuous exercise (against my doctor's initial advice). Later, I wondered whether I would have been the same difficult teenager if I had not had the brain haemorrhage.

Gaining control over my life again

Psychosocial support was not a part of the regular treatment. Our general practitioner paid little attention to anything other than the physical aspects of my illness. For example, when I had difficulty breathing she prescribed an inhaler for asthma, instead of exploring other causes such as anxiety. I felt I was seen as a patient, rather than a person. After three years of struggling, my parents contacted the general practitioner because they were very concerned about my wellbeing. Counselling from a psychologist helped me to understand that I still had control over my life, but my parents and sisters had to find a way to cope themselves.

When I was 18 I decided I wanted to live on my own because I didn't want to fight any more with the people I loved. I wanted to broaden my horizons and felt an enormous inherent power to do so. It was hard to convince my parents, but eventually they accepted my choice.

Next time I'm admitted to hospital, my goal is to stay there for as little time as possible, and not to lie flat if it's not necessary. For example, the day before my last stereotactic treatment (in 2006) my boyfriend and I went to the seaside and slept in a hotel instead of the hospital. That felt really good because I was still free to direct my own life.

I am happy that I received good medical treatment, and the AVM is now almost completely gone. My family and friends were always there for me and helped to deal with difficulties, transitions, and developing my new identity. My boyfriend helped me to become confident and accept myself. He loves me because of my experiences, not despite them. These days I experience hardly any consequences of the brain haemorrhage. I am now a PhD student, and in my job I am accepted as a person, and the development of positive qualities is emphasised. This kind of recognition and acceptance helps me to grow.

Since this article was written, JGW has confirmed that a recent angiogram seems to show that LD is now free of arteriovenous malformations (because the shunt, which is so characteristic of an arteriovenous malformation, has gone).

CORRECTIONS AND CLARIFICATIONS

Short term and intermediate term comparison of endarterectomy versus stenting for carotid artery stenosis: systematic review and meta-analysis of randomised controlled clinical trials
The corresponding author's email address was incorrect in this paper by Pascal Meier and colleagues (*BMJ* 2010;340:c467, print publication 27 February, p 459). The correct address is hgurm@med.umich.edu.

Edward Jenner's statue
An editorial by Gareth Williams (*BMJ* 2010;340:c1582, print publication 27 March, pp 665-6) wrongly said that Edward Jenner's statue used to occupy the currently empty "fourth plinth" in Trafalgar Square in London; in fact it was originally on a separate plinth in that square. The error was picked up and repeated by Fiona Godlee in her Editor's Choice in the same issue.

A memorable patient
In this Filler article by Stuart J Fergusson (*BMJ* 2010;340:b5403, print publication 8 May) we inserted the wrong publication details for article citation (year, volume, and elocator). When citing the article, use: *BMJ* 2010;340:b5403.

Is underdiagnosis the main pitfall when diagnosing bipolar disorder? Yes
One of the authors of this Head to Head article by Daniel J Smith and Nassir Ghaemi supporting the proposal that bipolar disorder is underdiagnosed (*BMJ* 2010;340:c854, print publication 27 March, pp 686-7) has told us that he should have declared a competing interest. Nassir Ghaemi currently has a research grant from Pfizer.

Access to antimalarial therapy: accurate diagnosis is essential to achieving long term goals
In this Analysis article by Heidi Hopkins, Caroline Asiimwe, and David Bell (*BMJ* 2009;339:b2606, print publication 8 August 2009, pp 324-6), we published a blood film whose caption states that the film shows "red blood cells infected with malaria parasites." Regrettably it does not show this clearly as the original image was cropped and printed at too low a resolution.

Effects of dietary intervention and quadriceps strengthening exercises on pain and function in overweight people with knee pain: randomised controlled trial
The authors of this paper by Claire M Jenkinson and colleagues have told us of a small error in their analysis (*BMJ* 2009;339:b3170, print publication 12 September 2009, pp 606-9). In the statistical analysis section of the Methods, the penultimate sentence in the print version (p 607) should have read: "The time course of treatment effects was estimated by a generalised estimating equations linear model incorporating time × treatment interactions with an unstructured correlation matrix to allow for the repeated measurements nature of the data." As a consequence, in the primary outcomes section of the Results (p 608), the effect of exercise at 24 months should have been given as −0.91 (−1.60 to −0.23; P = 0.009) [not −0.91 (−1.66 to −0.17; P=0.016), as was stated]—that is, slightly more significant.

Debendox in early pregnancy and fetal malformation
We have just been alerted to a decimal point that has been missing in a percentage for almost 30 years. The third sentence of this letter by D M Fleming and colleagues (*BMJ* 1981;283:99-101, print publication 11 July 1981, pp 99-101) should have said that 1.3% [not 13%] of the 620 women given Debendox delivered a malformed infant.

Maternal caffeine intake during pregnancy and risk of fetal growth restriction: a large prospective observational study
During some recent new analysis of the data for this study by the CARE Study Group, the authors noticed an inconsistency in the data recording for caffeine intake (*BMJ* 2008;337:a2332, print publication 6 December 2008, pp 1334-8). This meant that a small proportion of women had been wrongly allocated zero caffeine intake for instant coffee even though they might have consumed instant coffee. The authors have recalculated the corrected total caffeine values, resulting in small changes to the results. In the table, the adjusted odds ratios (95% confidence intervals) for the risk of fetal growth restriction according to caffeine intake averaged over pregnancy should read: for caffeine intake 100-199 mg/day, 1.1 (0.8 to 1.5) [not 1.2 (0.9 to 1.6)] ; 200-299 mg/day, 1.3 (0.9 to 1.8) [not 1.5 (1.1 to 2.1)]; ≥300 mg/day, 1.5 (1.0 to 2.1) [not 1.4 (1.1 to 2.0)]; and the P value for the test for trend (0.02) remains the same. In the figure the "best-fitting" curve remains closely similar to that in the published version. And the mean percentage of caffeine is now 21% from coffee and 58% from tea (not 14% and 62% respectively, as given in the main text). These revised results do not change the interpretation of the findings or the strength of association. Readers requiring more detailed information should contact the authors (Professor Janet Cade, j.e.cade@leeds.ac.uk).

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