SELF ASSESSMENT QUESTIONS

Obstetric difficulties due to Graves’ disease

A Bhattacharyya, J D Wright, P A Vice

A 14 year old girl was referred to the paediatricians with symptoms of hyperthyroidism. She had a smooth diffuse goitre with dysthyroid eye disease (proptosis, lid lag, and lid retraction). Hyperthyroidism was confirmed biochemically (protein bound iodine 18.8, normal 5–8 µg). She was treated with carbimazole, 30 mg/day. Poor compliance resulted in inpatient care for treatment with carbimazole followed by subtotal thyroidectomy, two years later. Two years later she presented with a self limited episode of hyperthyroidism. Aged 22 years she was referred with eight weeks amenorrhoea when pregnancy was confirmed and terminated. She was floridly hyperthyroid (free thyroxine 36.6, normal 10–23 pmol/l, free triiodothyronine 14, normal 3–9 pmol/l; thyroid stimulating hormone (TSH) undetectable, normal 0.5–5 mU/l). She was treated with carbimazole for nine months with apparently good clinical response.

At 24 years, she was admitted in premature labour at 36 weeks, resulting in a stillborn infant (weight 1700 g, goitre of 4.4 g, and diffuse hyperplasia on histology). It was when thyroid microsomal antibody (TMA) was noted (thyroid weighing 6 g, and a goitre was noted (thyroid weighing 6 g, diffuse hyperplasia on histology). No obvious maternal thyroid problem had been noted during the pregnancy. Six months later an unplanned pregnancy was terminated at 10 weeks of gestation when she was proved to be hyperthyroid (free thyroxine 30 pmol/l). At 26 years, she had a first trimester spontaneous abortion. A further pregnancy at the age of 30 years without obvious thyroid disease was complicated by premature labour, resulting in an urgent caesarean section for fetal distress and a stillborn baby. This baby weighed 1870 g and a goitre was noted (thyroid weighing 6 g, diffuse hyperplasia on histology). It was when she was readmitted with wound infection that she was noted to be hyperthyroid and was referred to the Division of Endocrinology. She was clinically and biochemically hyperthyroid (free thyroxine 33 pmol/l and free triiodothyronine 11.3 pmol/l) with dysthyroid eye disease. Her TSH receptor antibody (TSHRAb) was 95 (normal 0–10 U/l), thyroglobulin antibody (TGA) was 1: 1 638 400 (normal being less than 1: 400 for both TGA and TMA). The patient wished to try for a future pregnancy. After discussion, it was agreed that she would receive an ablative dose of radioactive iodine (with contraceptive care after the dose), to render her hypothyroid and maintain her on thyroxine. The plan was successful and she became hypothyroid within three months of receiving radioactive iodine. She was euthyroid on 100 µg thyroxine daily.

Fifteen months after receiving radioiodine she conceived and was seen in the Department of Obstetrics by JDW. She was closely monitored throughout the pregnancy. TSH and thyroxine confirmed she remained euthyroid throughout the pregnancy. TSHRAb became undetectable in late pregnancy (table 1). Fetal monitoring confirmed an euthyroid state (no tachycardia) with normal growth and no goitre on the scan. Amniocentesis, done to assess the maturity of the fetal lungs (this is not the practice now) at 36 weeks, was complicated by antepartum haemorrhage. Emergency caesarean section resulted in a live baby (birth weight 3400 g, no goitre, normal thyroid function, and negative TSHRAb). She conceived again at 35 years, resulting in a further successful outcome (birth weight 3700 g). She was managed in the same way as the previous successful one (the TSHRAb remained in the normal range throughout the pregnancy), the baby was euthyroid, and there was no goitre. She was sterilised after the second childbirth.

Questions

(1) Would more aggressive monitoring of the maternal thyroid status and fetal condition in the unsuccessful pregnancies have led to a better outcome?

(2) Is it likely that the difference in the outcome of those unsuccessful pregnancies was related to the maternal uncontrolled thyroid state or to the circulating antibody levels?

(3) What are the three crucial factors for the successful outcome in this case?

Table 1  Showing the thyroid parameters, antibody status, and treatment during the first successful pregnancy

<table>
<thead>
<tr>
<th></th>
<th>Normal values</th>
<th>Preconception</th>
<th>First trimester</th>
<th>Second trimester</th>
<th>Third trimester</th>
<th>Two months after delivery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Free thyroxine (pmol/l)</td>
<td>10–23</td>
<td>12.9</td>
<td>17.5</td>
<td>15</td>
<td>21.9</td>
<td>15</td>
</tr>
<tr>
<td>Free triiodothyronine (pmol/l)</td>
<td>2–5.5</td>
<td>4.1</td>
<td>4.7</td>
<td>3.1</td>
<td>3.9</td>
<td>4.2</td>
</tr>
<tr>
<td>TSH (mU/l)</td>
<td>0.5–5</td>
<td>1.6</td>
<td>4.1</td>
<td>7.4</td>
<td>0.9</td>
<td>1.5</td>
</tr>
<tr>
<td>TSHRAb (U/l)</td>
<td>0–10</td>
<td>40</td>
<td>21</td>
<td>17</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Daily dose of thyroxine (µg)</td>
<td>100</td>
<td>100</td>
<td>200</td>
<td>200</td>
<td>200</td>
<td>100</td>
</tr>
</tbody>
</table>

Answers on p 669.

Preston Acute Hospital NHS Trust, UK: Department of Medicine (Division of Endocrinology)
A Bhattacharyya
P A Vice

Department of Obstetrics
J D Wright

Correspondence to:
Dr P A Vice, Royal Preston Hospital, Sharoe Green Lane, Preston PR2 9HT, UK
Patricia.Vice@patn.nhs.uk

Submitted 16 November 1999
Accepted 1 August 2000
A rare presentation of a common disease

H Patel, K S Hindle, G Tsavellas, A Huang

A previously well 77 year old man presented with acute left iliac fossa pain. He was pyrexial and tachycardic with localised abdominal guarding but there was no palpable mass or generalised peritonitis. Respiratory examination revealed tachypnoea with normal bilateral air entry and no tracheal deviation. Blood investigations showed a leucocytosis with raised serum C reactive protein and erythrocyte sedimentation rate. Serum amylase was normal and he was hypoxic with reduced oxygen saturation in the blood. His abdominal and erect thoracic radiographs are shown (figs 1 and 2). He also underwent abdominal computed tomography (fig 3).

Questions
(1) What are the radiographic findings?
(2) What is the most likely diagnosis?
(3) What are the treatment options?

Adrenal mass in a diabetic with hypergastrinaemia

H M S Elasha, D Devendra, S Travis, D Wilkins, P Newman, T J Wilkin

A 50 year old man originally presented in 1980 at the age of 31 with diarrhoea, weight loss, and abdominal pain. He was initially treated with steroids for presumed inflammatory bowel disease. About a month later he presented with severe abdominal pain, and a laparotomy revealed multiple jejunal perforations and a possible mass in the head of the pancreas. He made a good recovery during which time a nasogastric tube was in place for two weeks. A markedly raised serum gastrin level confirmed the diagnosis of Zollinger–Ellison syndrome. Other gut hormone levels were normal. A parathyroid adenoma was suspected on account of raised calcium and parathyroid hormone levels.

He underwent a parathyroidectomy, and since then his serum calcium has remained normal.
Biopsy of the nodule in the head of the pancreas confirmed a pancreatic islet cell tumour. He subsequently underwent total gastrectomy, distal oesophagectomy for a peptic oesophageal stricture, and pancreaticoduodenectomy in 1980.

His gastrin levels fell after surgery, but rose again sharply within the next two years.

He remained well until 1995, when he presented with weight loss, polyuria, and polydipsia. Diabetes mellitus was diagnosed and he was started on insulin therapy. At this time a computed tomography scan of his abdomen revealed a 4 × 2 cm right adrenal mass and a 1.5 cm nodule in the tail of the pancreas. An octreotide labelled scan was normal, and the adrenal mass was thought to be an incidental mass. Subsequent computed tomography indicated that the adrenal mass was growing in size, and was removed surgically in 1999. Histology was diagnostic of primary adrenal carcinoma.

Questions
(1) What other conditions besides Zohlinger-Ellison syndrome could cause a raised gastrin level?
(2) Why does hypercalcaemia sometime present with abdominal pain?
(3) How would you proceed to investigate whether the adrenal mass is functioning or just an incidental finding?
(4) Why did this man develop diabetes mellitus?
(5) Why is it important to screen his family?
(6) Why did he develop a distal oesophageal stricture?

Adult intussusception—an elusive diagnosis

W J Sotheran, M H Wise

An 81 year old woman presented, as an emergency, with a two day history of abdominal pain. The pain was intermittent and confined to the right upper quadrant. She had no other symptoms and normally enjoyed good health. Examination revealed tenderness in the right hypochondrium. Investigations revealed a mildly raised serum alkaline phosphatase. All other haematological and biochemical parameters were within normal limits. A provisional diagnosis of biliary colic was made.

Four days later, the patient experienced diarrhoea and vomiting. Her abdominal pain had increased and there was generalised abdominal tenderness. She developed a pyrexia of 38°C. An ultrasound scan demonstrated a distended gall bladder with stones and a dilated common bile duct (12.6 mm). Antibiotic treatment was started. The patient made little progress with continued diarrhoea and vomiting. Stool cultures and sigmoidoscopy were normal. Parenteral nutrition was started.

Over the next two days, the patient developed a palpable mass in the epigastrium. Repeat ultrasound showed a normal biliary tree, but a mass consistent with a necrotic pancreas with pseudocyst was imaged. Computed tomography of the abdomen and pelvis was requested. Computed tomograms are shown in figs 1 and 2.

Questions
(1) The images show a characteristic mass lesion anteriorly. What diagnosis is suggested by these findings?
(2) How may this condition present in adults?
(3) How should such cases be managed?
Acute respiratory failure in a middle aged woman

C McGuigan, G McDonnell, M Mirakhur, J I Morrow

A 61 year old woman presented with a one week history of headache, drowsiness, and shortness of breath. Initial examination revealed bilateral ptosis, which the patient stated had been present for three to four years before this admission. She also reported that her father had died suddenly, shortly after developing “drooping eyelids”. There was restriction in the range of eye movements in all directions. The remaining cranial nerves were intact.

In the limbs there was normal tone but mildly reduced power in all muscle groups, worse proximally. There was generalised areflexia. Plantar responses were flexor and there was no sensory deficit. Coordination was normal. There were no signs of meningism. On chest examination there was reduced air entry bilaterally and bronchial breathing in both lower zones. Cardiovascular and abdominal examinations were unremarkable.

There was a past medical history of pernicious anaemia, osteoporosis, partial thyroidectomy, and cholecystectomy. Drugs on admission were disodium etidronate and hormone replacement therapy.

Laboratory investigations included an arterial pH of 7.34, oxygen pressure 11.78 kPa, carbon dioxide pressure 7.1 kPa, and a base excess +5.1. Urea and electrolytes, creatinine, calcium, creatinine kinase, and lactate were all within normal limits. Full blood picture was unremarkable and cerebrospinal fluid examination was also normal. A chest radiography showed bilateral lower lobe collapse and consolidation. The electrocardiogram is shown in fig 1.

The patient’s condition deteriorated with increasing respiratory distress. Her respiratory rate rose to 32 breaths/min, repeat arterial blood gases indicated pH 7.30 kPa, oxygen pressure 9.9 kPa, and carbon dioxide pressure 8.9 kPa. The patient was transferred to intensive care for ventilatory support.

Questions

(1) What is the differential diagnosis for this clinical presentation?

(2) What does the electrocardiogram (fig 1) show and what is the significance of this?

(3) What other clinical findings would be useful to elicit?

(4) What further investigations would you like to perform?

Figure 1 Electrocardiogram.
Cor pulmonale: variation on a theme

O M P Jolobe, H J Schlayer, A Yates

A woman aged 89 was admitted with a three month history of exertional dyspnoea. She had had a previous admission, at the age of 86, for left lower lobe pneumonia, with coincidental atrial fibrillation.

On examination she was centrally cyanosed, normotensive, and had atrial fibrillation, pedal oedema, and raised jugular venous pressure to the angle of the jaw even when sitting up. A soft systolic murmur was audible at the left sternal edge.

Questions
(1) What investigations would help to identify the underlying cause of cardiac failure in this patient?
(2) Could there be a link between the atrial fibrillation and evolution of the changes in the QRS axis?

Rectal bleeding in a patient with portal hypertension

U K Sinha, S K Raha, W E Wilkins

A 77 year old woman presented for the first time in 1997 with severe haematemesis and melena. After initial resuscitation she underwent an emergency upper gastrointestinal endoscopy which showed bleeding oesophageal varices. She was treated with injection sclerotherapy.

She had a history of ischaemic heart disease but was stable on medication. She was not known to have any other medical problems.

Clinically she was pale but not jaundiced. There was mild ascites and prominent abdominal veins. The splenic tip was just palpable below the left costal margin. Her liver function tests were normal. There was no coagulation abnormality. Ultrasound scan of the abdomen showed normal hepatic architecture. The spleen was enlarged and there was evidence of ascites. Portal blood flow studies suggested portal hypertension secondary to thrombus in the portal vein. After discharge from the hospital she was kept under endoscopic surveillance. The variceal recurrences needed repeated ligation.

Three years later she had to be admitted again, this time for the investigation of bleeding per rectum. It was intermittent, self limiting, and small in amount. Her bowel habits were normal. Clinical examination revealed pallor, mild ascites, and prominent abdominal veins. Her spleen was palpable 2 cm below the costal margin. Figure 1 shows the finding on flexible sigmoidoscopy. Her barium enema was normal.

Questions
(1) What was the most probable cause of rectal bleeding in this woman?
(2) What is the prevalence of this condition in portal hypertension?
(3) What is the management?
A state of confusion

N Sofat, C S Higgins

A 66 year old right handed man was admitted via the accident and emergency department with confusion and weakness. His wife gave most of the history, saying he had not been able to express himself clearly for the last day and looked weak. He had no history of ischaemic heart disease, hypertension, hypercholesterolaemia, diabetes mellitus, or previous strokes. He smoked 10 cigarettes a day and drank alcohol only socially. He had been diagnosed with rheumatoid arthritis 20 years before. His drug history included diclofenac. On examination, the admitting doctor found that he had an expressive dysphasia and a right hemiparesis. He had a blood pressure of 140/70 mm Hg, pulse 80 beats/min and regular, both first and second heart sounds present with no added sounds and his chest was clear. His abdomen was soft and non-tender with no organomegaly. He had bilateral metacarpophalangeal joint swelling in his hands with ulnar deviation. On further neurological assessment he had an upper motor neurone right facial nerve palsy and the rest of his cranial nerves were normal. In his limbs, in addition to the right hemiparesis, he also had an ulnar nerve palsy in his left hand.

The patient was diagnosed as having had an acute stroke. Computed tomography the same day showed areas of hypodensity consistent with fresh infarctions in the temporal lobes bilaterally and also in the left frontoparietal lobe. He was started on 300 mg aspirin daily. His plasma lipids and glucose were found to be normal. Doppler examination of his carotid arteries and cardiac echocardiogram were also normal. He was transferred to the neurorehabilitation unit. Five days after his admission, he was noted to have a lesion in his left eye as illustrated in fig 1. He was also seen to have a lesion over his left lateral malleolus, shown in fig 2, which was very painful.

Questions
(1) What is illustrated in fig 1?
(2) What is the skin lesion illustrated in fig 2?
(3) What is the unifying diagnosis?
(4) What treatment would you now offer the patient?
(5) What is his prognosis?
Dysphagia in a patient with palmoplantar keratoderma

R Morgan

A 72 year old man was referred by his general practitioner with a four month history of dysphagia. Physical examination was unremarkable with the exception of his hands and feet which showed hyperkeratosis on the palms (fig 1) and soles of his feet. Gastroscopy showed severe extensive oesophagitis (grade III) confirmed with multiple oesophageal biopsies. He was started on a proton pump inhibitor (omeprazole) and rescoped three months later. He still had oesophagitis at this time, although macroscopically it had improved; repeat biopsies again showed oesophagitis. A further gastroscopy three months later showed complete resolution of his oesophagitis confirmed by repeated oesophageal biopsies. He remains well and asymptomatic on omeprazole and continues to be followed up.

Questions
1. What is the diagnosis?
2. How is it inherited?
3. What is the major complication?

Decreased sexual function in a young man

R A Fisken

A 36 year old professional man presented with an 18 month history of difficulty in achieving and maintaining an erection. He also described “lack of sex drive”, malaise, and non-specific ill health over several months. He was sometimes able to masturbate successfully but commented that his ejaculate was of small volume. Physical examination showed him to be anxious but he was otherwise normal except for the fact that the testes were smaller than expected (about 12 ml in volume) and soft.

Questions
1. What is the commonest cause of erectile dysfunction in a man of this age?
2. What features of the clinical presentation in this patient would be in favour of an organic cause for his problem
3. What baseline investigations would you undertake?

Answers
1. What is the commonest cause of erectile dysfunction in a man of this age?
Erectile problems in young men are, on average, more likely to result from psychological than physical causes, though a combination of the two may be present. The older the patient at presentation, the more likely is the disorder to have a mainly physical cause.

Q2: What features of the clinical presentation in this patient would be in favour of an organic cause for his problem
The reduction in libido, small ejaculate volume, and small, soft testes are all suggestive of hypogonadism.

Q3: What baseline investigations would you undertake?
In this case there is clear evidence of the need to measure serum testosterone, prolactin, follicle stimulating hormone (FSH), and luteinising hormone. In many clinics a baseline blood glucose and renal and liver function tests would also be requested.

The patient’s initial endocrine results are shown in table 1. Plain skull radiography and

Table 1 Patient’s endocrine results

<table>
<thead>
<tr>
<th>Testosterone (nmol/l)</th>
<th>1.6</th>
<th>&gt;10.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Free androgen index</td>
<td>4</td>
<td>&gt;14</td>
</tr>
<tr>
<td>FSH (U/l)</td>
<td>1.4</td>
<td>2.0–12.0</td>
</tr>
<tr>
<td>Luteinising hormone (U/l)</td>
<td>2.8</td>
<td>1.0–12.0</td>
</tr>
<tr>
<td>Prolactin (mIU/l)</td>
<td>1063</td>
<td>&lt;555</td>
</tr>
</tbody>
</table>
magnetic resonance imaging (MRI) were requested (figs 1 and 2).

The low serum testosterone and free androgen index confirm hypogonadism.

Questions

(4) Is this patient’s hypogonadism primary or secondary? Why?

(5) What is the abnormality seen on the skull radiography (fig 1)?

(6) What diagnosis is suggested by the MRI scan (fig 2)?

(7) At what age does this condition typically present?

(8) What treatment should this patient have for his primary disorder?

(9) What treatment should he have for his hypogonadism? Why?

Answers

Q4: Is this patient’s hypogonadism primary or secondary? Why?

This patient has secondary hypogonadism: his serum testosterone is very low but there is no compensatory increase in the secretion of FSH and luteinising hormone—in a man with primary testicular failure and a serum testosterone of this level one would expect the serum FSH and luteinising hormone to be greater than 30.

Learning points

- Erectile dysfunction in younger men is commonly caused by mainly psychological factors but organic factors should nevertheless be sought.
- The presence of loss of libido or of small, often soft, testes favours a diagnosis of hypogonadism.
- If the serum testosterone is low and the levels of FSH and luteinising hormone are low or normal then the patient has secondary hypogonadism due to pituitary or hypothalamic disease.
- Craniopharyngiomas may present in adulthood.

Q5: What is the abnormality seen on the skull radiography?

There is suprasellar calcification.

Q6: What diagnosis is suggested by the MRI (fig 2) scan?

The MRI scan shows a solid soft tissue mass in the suprasellar cistern. It does not appear to arise from the pituitary and is most likely to be a craniopharyngioma.

Q7: At what age does this condition typically present?

Craniopharyngioma is most commonly detected between the ages of 6 and 14 but may come to light in adults, even up to an advanced age.1 2 3

Q8: What treatment should this patient have for his primary disorder?

The patient will require a full pituitary assessment, including a combined pituitary function test. The lesion itself is compressing the optic chiasm and will require surgery followed by radiotherapy.

Q9: What treatment should he have for his hypogonadism? Why?

The patient should be offered a choice of forms of testosterone replacement, both for the sake of his sexual function and in order to preserve his muscle mass and prevent osteoporosis. Available treatments include intramuscular testosterone esters, oral testosterone undecanoate, transdermal testosterone, and testosterone implants.

Rectal bleeding in a patient with portal hypertension

U K Sinha, S K Raha and W E Wilkins

Postgrad Med J 2001 77: 665-666
doi: 10.1136/pmj.77.912.665a

Updated information and services can be found at:
http://pmj.bmj.com/content/77/912/665.2

These include:

Email alerting service
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Errata
An erratum has been published regarding this article. Please see next page or:
/content/78/915/62.2.full.pdf

Notes

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/
**LETTERS**

**Culture negative endocarditis: data from the national survey in Slovakia**

Millar et al in their interesting review discussed culture negative endocarditis. The mainstay of diagnosis of infective endocarditis is still conventional blood culture; however, blood culture may be negative in 1%–79% of all cases. The incidence of culture negative endocarditis has been increasing. This could be for a number of reasons. Prosthetic heart valves are prone to infection and in many of these cases the culture is negative. Many aetiological agents causing infective endocarditis may be fastidious in nature, such as the HACEK group of organisms or unusual and require specialised microbiological techniques.

Within our national survey of 180 cases in Slovakia, culture negative endocarditis appeared in 35 cases (19.5%), which is higher than that reported in the Netherlands (1%), the USA (5%), Sweden (12%), the UK (15%), France (18%), but lower than in Russia (26%) and Spain (37%–43%) and much lower than in India (53%–79%).

In univariate analysis comparing all cases (180) to culture negative (35 cases), prior endocarditis may be fastidious in nature, such as the HACEK group of organisms or unusual and require specialised microbiological techniques. The odds ratio was 2.45 (confidence interval 0.95 to 2.35) in the group with culture negative endocarditis, which was 2.45 times higher than in culture positive endocarditis. Interestingly mortality was lower in culture negative endocarditis than among all cases (24.5% vs 44.4%, p<0.001). Millar et al in his excellent review analysed reasons for culture negative endocarditis. We found according to our experience one more risk factor, prior cardiac surgery. Probably, those undergoing cardiac surgery and receiving antibiotic prophylaxis (first generation cephalosporins/cefazolin in Slovakia) have lower death rates in endocarditis due to protective effect of antimicrobials for occurrence of infection.

**P Marks, M Gogova, V Kromery Jr**

North London Postgraduate Medical Centre, UK and University of Trnava, School of Public Health, Slovakia

**References**


**Table 1** Comparison of all cases with those with culture negative endocarditis (CNE); values are number (%)

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>All cases (n=180)</th>
<th>CNE (n=35)</th>
<th>Univariate analysis</th>
<th>Multivariate analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age less than 60</td>
<td>46 (25)</td>
<td>11 (31)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Male gender</td>
<td>123 (69.4)</td>
<td>20 (57)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Rheumatic fever</td>
<td>64 (35)</td>
<td>11 (31)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Malignancy</td>
<td>12 (6.7)</td>
<td>24 (5)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>11 (6.1)</td>
<td>3 (9.8)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Intravenous drug use</td>
<td>2 (1.1)</td>
<td>0</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Prior cardiac surgery</td>
<td>14 (7.8)</td>
<td>5 (14.3)</td>
<td>0.045 NS</td>
<td>NS</td>
</tr>
<tr>
<td>Prior endoscopy</td>
<td>8 (4.4)</td>
<td>1 (2.9)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Dialysis</td>
<td>8 (4.4)</td>
<td>2 (5.7)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Central venous catheter</td>
<td>6 (3.3)</td>
<td>3 (9.8)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Dental surgery &lt;96</td>
<td>37 (20.5)</td>
<td>3 (9.8)</td>
<td>0.045 NS</td>
<td></td>
</tr>
<tr>
<td>Tonsillitis or sinusis &lt;96</td>
<td>15 (8.3)</td>
<td>2 (5.7)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Duke’s criteria and localisation</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Definitive diagnosis</td>
<td>169 (93.9)</td>
<td>27 (78)</td>
<td>0.045 NS</td>
<td></td>
</tr>
<tr>
<td>Probable diagnosis</td>
<td>21 (11.6)</td>
<td>8 (23)</td>
<td>0.045 NS</td>
<td></td>
</tr>
<tr>
<td>Aortic damage</td>
<td>84 (46.7)</td>
<td>18 (52)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Mitral damage</td>
<td>85 (47.2)</td>
<td>14 (40)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Complications (embolus, heart attack, haemothrombosis)</td>
<td>36 (20.0)</td>
<td>17 (48)</td>
<td>0.001</td>
<td>0.024 OR 3.05</td>
</tr>
<tr>
<td>Right ventricular failure</td>
<td>11 (6.1)</td>
<td>3 (9.8)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Immunological phenomena</td>
<td>116 (64.4)</td>
<td>21 (60)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antibiotic only</td>
<td>120 (66.7)</td>
<td>20 (57)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Antibiotic plus surgery</td>
<td>60 (33.3)</td>
<td>15 (9.8)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Antibiotic &lt;21 days with surgery</td>
<td>35 (19.5)</td>
<td>7 (20)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Antibiotic &lt;21 days without surgery</td>
<td>31 (17.2)</td>
<td>5 (14)</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Outcome: death due to infection</td>
<td>40 (44.4)</td>
<td>9 (24.5)</td>
<td>0.001 NS</td>
<td></td>
</tr>
</tbody>
</table>

OR, odds ratio

---


**Ethical, professional, and legal obligations in clinical practice**

We wish to applaud Mr Gore on conducting sessions and writing about ethical, professional, and legal obligations in clinical practice. It is an area in which most doctors fail to get training at an earlier stage, and there is a case for other specialties to take heed from Gore’s series and conduct such educational exercises in their hospitals.

We agree with Gore that doctors tend to underestimate how willing people are to talk about their own death and, in fact, their resuscitation status. As doctors we tend to assume that this discussion with patients (where feasible) would upset them enormously and hence the reluctance to discuss it with them.

To find an answer to this dilemma, we conducted an interview based study in our district general hospital, where 70 inpatients on medical wards were interviewed to assess their knowledge of cardiopulmonary resuscitation and their views on getting involved in their “not for resuscitation” (NFR) decision. The group had equal number of male and female patients and equal number of patients below and above the age of 70 years. The results were very interesting and showed that majority (71%) of the hospital inpatients wished to get involved in the discussion related to their NFR decision. This view was similar among young and old patients. This sends a strong message that ethically we ought to involve mentally competent patients in their NFR decisions if the latter so wish.

We disagree with Gore that resuscitation be offered if it is specifically requested by a patient even if a successful resuscitation is unlikely. In patients in whom cardiopulmonary arrest clearly represents a terminal event in their illness, attempted resuscitation might be considered inappropriate. Neither patients nor their relatives can demand treatment that the health care team judges to be inappropriate. There are situations where medical reality and patient’s expectations in relation to their illness and NFR decisions do not match. In situations like these the healthcare team has the moral and legal responsibility to help their patients reach a decision in their best interest.

**A K Jain, I Chattopadhyay, A Kallat**

Department of Medicine for the Elderly, Wrexham Maelor Hospital, Wrexham LL13 7TX, UK; drolajain@netscapeonline.co.uk

**References**


www.postgradmedj.com

Author’s reply

I welcome the comments of Dr Jain and his colleagues. The apposite study which they conducted at Wrexham Maelor Hospital demonstrates the desire among patients, young and old, for involvement in NFR decisions. As doctors we must confront our own unease at discussing matters of resuscitation and death with patients.

I accept the authors’ reservations about my endorsement of compliance with a patient’s wish for cardiopulmonary resusciation in all cases. In many such cases cardiopulmonary resuscitation would be medically inappropriate, and it is indeed the responsibility of the healthcare team to counsel the patient accordingly. Nevertheless the series of discussion articles was geared towards education for junior medical staff, and I chose to keep the guidelines straightforward with an emphasis on patient autonomy. Certainly in any such situation one would expect a more senior member of the healthcare team to identify and address that mismatch between medical reality and patient/relative expectation. Counselling might then be offered in the hope of reaching consensus on the suitability of a NFR decision.

Intraoperative glove perforation

We read with interest the paper by Thomas et al concerning single versus double gloving in protection against intraoperative skin contamination from glove perforation. We note that one of the methods used to detect glove perforation was the water leak method. Although we accept that the water leak test is an acceptable method we believe that it is not as sensitive as the electrical conductance test as demonstrated by Sohn et al.

Interim results from an ongoing study yielded 211 sterile and non-sterile gloves used during venepuncture or wound closure in our emergency department. We identified nine glove perforations with the water leak test and 22 with the electrical conductance test. All water leak positives were also electrical conductance test positive. This study supports the work by Sohn et al. We believe that Thomas, Agarwal, and Metha may have underestimated the incidence of glove perforation in their study group.

R McLaughlin, B McNicholl, J Barton Emergency Department, Royal Victoria Hospital, Grosvenor Road, Belfast, BT12 6BA, UK; remclaughlin@tinyworld.co.uk

References


Authors’ reply

The water load test, as per the criteria established by the American Society for Testing and Materials, is one of the methods approved by law for testing integrity of latex gloves. Recent studies have shown that the electrical conductance test has a higher sensitivity than the water load test in detecting smaller glove perforations. The study by Sohn et al cited by McLaughlin and colleagues shows similar findings, although the number of patients testing false negative with the electrical conductance test has not been mentioned.

Both these tests, however, have an inherent disadvantage as they overstend the gloves, thus negating the viscoelastic, self-sealing properties of latex and aggravating the potential permeability to fluid/electrical impulses. It may be more appropriate if these tests are conducted with the gloves distended with liquid only up to the appropriate size; the significance of the glove perforation can then be assessed by the number of bacteria/quantity of water that can pass through the perforation in a fixed period of time.

In the ongoing study described by McLaughlin et al, there were 22 perforations in 211 gloves used in minor surgical procedures, indicating that one in five minor surgical procedures will result in perforated gloves (one pair of gloves for each procedure). This emphasises the point made by our article that even in minor surgical procedures, single gloving alone will not provide adequate protection.

Reference


CORRECTION

Cor pulmonale: variation on a theme

We regret that an error occurred in the above paper by Jolobe, Schlayer, and Yates (Postgrad Med J 2001;77:665, 675–7). The final diagnosis should have read “Cor pulmonale resulting from pulmonary tumour embolism.”