Fluid balance, sodium, potassium and hydrogen ion disturbances.

Disturbances of fluid balance and “electrolyte” concentrations occur in a wide variety of conditions and lead to significant morbidity and mortality. Globally the most serious problems are those of diarrhoea and dehydration. Up to 6 million deaths result annually from gastroenteritis and 80% of these are children aged less than two years.

Basic facts and figures.

Knowledge about the volume of fluids in the main body compartments is helpful in the understanding of clinical disorders and the calculations needed for appropriate treatment. In most sub-Saharan African settings measurement of plasma electrolytes is not available. However, to ignore electrolyte disturbances that we cannot measure will put our patients at risk. The figures that follow and in Tables 1 and 2 apply to an average normal adult NOT living in extreme environmental conditions or undertaking heavy exercise.

Total body fluid
40 litres

<table>
<thead>
<tr>
<th>Intracellular fluid</th>
<th>Extracellular fluid</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 litres</td>
<td>15 litres</td>
</tr>
</tbody>
</table>

[includes 3 litres of plasma in the intravascular space]

Plasma sodium 135 – 145 mM/litre, potassium 3.5 – 5.0 mM/litre, pH 7.36 – 7.42.
Intracellular sodium 4 mM/litre, potassium 150 - 160 mM/litre, pH 6.3 – 7.4.

<table>
<thead>
<tr>
<th>Fluid lost</th>
<th>Volume (mls.)</th>
<th>Sodium (mM)</th>
<th>Potassium (mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine*</td>
<td>1,500</td>
<td>120- 220</td>
<td>35 - 80</td>
</tr>
<tr>
<td>Stool</td>
<td>200</td>
<td></td>
<td>5 - 15</td>
</tr>
<tr>
<td>Insensible:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sweat**:</td>
<td>400</td>
<td>approx. 20</td>
<td>approx. 20</td>
</tr>
<tr>
<td>Respiratory:</td>
<td>400</td>
<td></td>
<td></td>
</tr>
<tr>
<td>**Totals</td>
<td>2,500</td>
<td>approx. 140 - 240</td>
<td>approx. 60 - 115</td>
</tr>
</tbody>
</table>

Table 1: Average fluid, sodium and potassium losses per 24 hours.
[*Urine: sodium 80 – 145 mM/litre, potassium 20 – 50 mM/litre, pH 4.8 - 7.5,
**Sweat: sodium approx. 45 mM/litre, potassium 10 mM/litre]

<table>
<thead>
<tr>
<th>Fluid secretion</th>
<th>Volume (mls.)</th>
<th>Sodium (mM/litre)</th>
<th>Potassium (mM/litre)</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salivary</td>
<td>500 - 1,500</td>
<td>10 - 25</td>
<td>15 - 40</td>
<td>6.0 - 7.0</td>
</tr>
<tr>
<td>Gastric</td>
<td>2,000 - 3,000</td>
<td>20 - 150</td>
<td>5 - 15</td>
<td>1.0 - 3.5</td>
</tr>
<tr>
<td>Pancreatic</td>
<td>300 - 1,500</td>
<td>140</td>
<td>6 - 9</td>
<td>8.0 - 8.3</td>
</tr>
<tr>
<td>Biliary</td>
<td>250 - 1,100</td>
<td>130 - 165</td>
<td>3 - 12</td>
<td>7.8</td>
</tr>
<tr>
<td>Small intestinal</td>
<td>1,800</td>
<td>80 - 150</td>
<td>2 - 8</td>
<td>7.5 - 8.0</td>
</tr>
<tr>
<td>Colonic</td>
<td>200</td>
<td></td>
<td></td>
<td>7.5 – 8.0</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>5,050 - 9,100</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Secretions per 24 hours of gastrointestinal fluids and their concentrations of sodium and potassium and pH.

From these data it is clear that the potential for fluid and electrolyte balance problems is great. The blood plasma volume is only 3 litres. The gastrointestinal tract is capable of secreting, under normal circumstances, up to about 9 litres of fluid containing about 1,000mM sodium and 150mM potassium. Most of this is reabsorbed. When stress is placed upon the gut (e.g. infection with cholera) the resulting losses may be enormous.
Dehydration: water and sodium balance.

The metabolism of water and sodium is so closely allied that it is best to discuss them together. The common causes of their imbalance are:

- Diarrhoea and / or vomiting,
- Inadequate water and sodium intake often in association with diarrhoea and / or vomiting,
- Inappropriate use of diuretics,
- Diabetes mellitus when an osmotic diuresis is caused by uncontrolled hyperglycaemia.

Other less common causes include:

- Renal losses as in salt-losing nephropathies (during diuretic phase of recovery from acute tubular necrosis and after relief of urinary tract obstruction) and diabetes insipidus.
- Adrenal cortical destruction leading to reduced renal tubular reabsorption of water and sodium.
- Diabetes insipidus: large volumes of dilute urine.

**Dehydration may be associated with normal plasma sodium (eunatraemia), low sodium (hyponatraemia) or high sodium (hypernatraemia) concentrations.** An hyponatraemic state may exist with iso-, hypo- or hypertonicity of the plasma. It is important to attempt a distinction between these especially when facilities for measuring plasma sodium and osmolality are not available. Help with this differentiation is presented in Table 3. Hyponatraemia is much more common than hypernatraemia. The clinical features (Table 4) of an hyponatraemic hypertonic state are not diagnostic but they should provide clues. They are more obvious and serious if the hyponatraemia has developed rapidly. The very young and very old and the alcoholic are at greatest risk:

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>Probable plasma sodium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anorexia, Nausea, Malaise</td>
<td>&lt;125 mM/litre</td>
</tr>
<tr>
<td>Headache, Lethargy, Confusional and agitated state</td>
<td>110 – 120 mM/litre</td>
</tr>
<tr>
<td>Convulsions, Coma, Death</td>
<td>&lt;110 mM/litre</td>
</tr>
</tbody>
</table>

Table 4: Clinical indicators to the degree of possible hyponatraemia.

Therefore if a patient has any of these features consider the following questions and strategy:

1. Have the above features appeared rapidly (over a few days).
2. Is he / she clinically dehydrated, oedematous or normally hydrated? See Table 3.
3. Keep a regular chart of body temperature, pulse rate and blood pressure (if possible include the sitting and standing readings).
4. Test urine for glucose and protein.
5. Measure the blood sugar.
6. Check haemoglobin and packed cell volume and compare with previous records: rising values suggest dehydration.
7. Keep an accurate fluid balance chart and weigh the patient daily.
8. Then consider the following three lists of causes to assist the differential diagnosis:

**Dehydrated patients:**
- Diarrhoea and / or vomiting, Severe heat exposure,
- Small bowel obstructions, Diuretic therapy,
- Burns, Diabetes mellitus,
- Trauma, Recovery phase (diuretic) of acute renal failure,
- Coma and reduced access to water,
**States of dehydration in relation to plasma sodium concentration**

<table>
<thead>
<tr>
<th>Eunatraemia</th>
<th>Hypotonaemia (&lt;130 mM/litre)</th>
<th>Hypernatraemia (&gt;150 mM/litre)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Isotonic plasma</strong>*</td>
<td><strong>Hypotonic plasma</strong></td>
<td><strong>Hypertonic plasma</strong></td>
</tr>
<tr>
<td>• Commonest.</td>
<td>• The patient losing fluids by vomiting and diarrhoea is hypovolaemic. Replacement using oral or intravenous fluids with low sodium concentrations (e.g. 5% glucose in water) leads to an hyponatraemic hypotonic plasma.</td>
<td>• In diabetic hyperglycaemic ketoacidosis (DKA) and hyperglycaemic hyperosmolar non-ketotic syndrome (HHNKS) the excess plasma glucose is the cause of the hypertonicity.</td>
</tr>
<tr>
<td>• Water and sodium are lost in equal proportions leading to hypovolaemia (low extracellular fluid volume).</td>
<td>• A similar situation is caused by diuretics (e.g. frusemide and thiazides).</td>
<td>• NB: The sodium concentration depends on the associated osmotic diuresis, water intake and shift of water from the intracellular space to the extracellular space and so eu- or hyper-natraemia may occur.</td>
</tr>
<tr>
<td>• Thirst, dry mucous membranes, dry axillae, reduced subcutaneous tissue turgor, sunken eyes, oliguria, postural hypotension.</td>
<td>• Water moves across cell membranes and so causes intracellular volume increase.</td>
<td>• Water has been lost to a greater extent than sodium.</td>
</tr>
<tr>
<td>• If not reversed: leads to anuria, severe hypotension, thready and fast pulse, cold peripheries and coma and death.</td>
<td>• Features of dehydration, lethargy. Convulsions uncommon.</td>
<td>• When gastrointestinal losses are replaced with fluids containing an excess of sodium the patient may become hypervolaemic. This causes severe thirst, lethargy, muscle weakness, irritability, confusion, convulsions (when plasma sodium &gt;165 mM/litre).</td>
</tr>
<tr>
<td>• Note similar picture also caused by DKA and HHNKS.</td>
<td></td>
<td>• Losses of large amounts of hypotonic fluid leads to an hypovolaemic state where there is lack of thirst. Dehydrated. Diabetes insipidus (may result from a previous head injury) leads to large volumes of dilute urine.</td>
</tr>
</tbody>
</table>

*Table 3: Features of eu-, hypo- and hyper-natraemic states.*

*An isonotic hyponatraemic state may arise. This is an artefact of measurement when there is a large amount of non-aqueous material (e.g. paraprotein) present; it is a technical and not a clinical problem

** A euvoalaemic (normal extracellular fluid (ECF) volume), hyponatraemic, hypotonic state may arise as the result of the syndrome of inappropriate antidiuretic hormone (SIADH) secretion. There is increased water retention by the renal tubules. The condition is found with bronchial carcinoma, pneumonia, advanced tuberculosis, cerebrovascular diseases and cerebral infections, and the use of chlorpropamide, carbamazepine, antidepressants and morphine. A severely ill patient (infection, trauma, malnutrition) may rapidly develop hyponatraemia and remain euvoalaemic.

A hypervolaemic (expanded ECF volume), hyponatraemic, hypotonic state is caused by an increase in water and sodium but the water excess is greater. Such situations arise in the oedema-producing conditions of congestive heart failure, liver cirrhosis, nephrotic syndrome and renal failure.
Oedematous patients:
Cardiac failure, Liver cirrhosis,
Nephrotic syndrome, Renal failure.

Normally hydrated patients:
Water overload, Steroid insufficiency.
Syndrome of inappropriate antidiuretic secretion,

The clinical features of the hypernatraemic hypertonic state are listed in Table 3. They are mainly the result of intracellular volume reduction, which has serious effects on cerebral tissue. The more acute the onset of sodium and water imbalance the more severe the symptoms. When hypernatraemia develops slowly there may be no clinical features or perhaps just a little drowsiness.

The management of these fluid and sodium disorders is that of the underlying problem (e.g. diabetes mellitus) and the working diagnosis of the mechanism. Here are some simple tips:

- If the patient is dehydrated and hypovolaemic then assess the probable volume of the losses and calculate the sodium (and potassium: see below) content depending on the source of the fluid loss. Replace these with careful clinical monitoring: pulse rate, blood pressure (lying and standing if possible), central venous pressure, fluid balance and body weight charts. A series of haemoglobin and packed cell volume measurements may also help: the values fall as the patient becomes hydrated but be aware of blood loss as another cause!!

- If the patient is considered to be hypervolaemic and/or oedematous then treat according to the underlying cause e.g. heart failure, nephrotic syndrome.

Potassium balance disorders.

The plasma potassium concentration is kept within the narrow range of 3.5 – 5.0 mM/litre. Many physiological mechanisms influence this. The concentration tends to be reduced by aldosterone, insulin, and adrenaline (including severe physical stress) and increased by glucagon. The balance of potassium and hydrogen ions is closely linked. If there is a metabolic acidosis (high hydrogen ion concentration and low pH) there is a reduction of potassium excretion by the renal tubules and intracellular potassium exchanges for extracellular hydrogen ions: hyperkalaemia results. When acidosis is corrected or alkalosis (low hydrogen ion concentration and high pH) arises then the opposite occurs and hypokalaemia may develop.

As with sodium imbalance there are no precise diagnostic clinical features but the situations where hypo- or hyper-kalaemia occur are well known. By having the latter in mind and considering the clinical features will assist management. It is all too easy to ignore the possibility of a potassium imbalance when the plasma levels cannot be measured. Such an approach has potentially serious consequences for the patient.

There are many causes of hypo- and hyper-kalaemia but only the most important ones are listed in Tables 5 and 6. The appearance of the clinical manifestations indicates a grave life threatening stage (Table 7). In medical practice in the Tropics the most important causes are gastrointestinal: a comparison of the effects of vomiting and diarrhoea follows:

- **VOMITING**

  Vomitus contains only 5 – 10 mM potassium/litre but much acid

  Acid loss

  Metabolic alkalosis

  Potassium loss from kidneys

  HYPOKALAEMIA

\[1\text{ Respiratory acidosis has far less effect on plasma potassium.}\]
• DIARRHOEA
  up to 100 mM potassium / litre

HYPOKALAEMIA
  associated with bicarbonate loss
  lessens the hypokalaemia

METABOLIC ACIDOSIS

<table>
<thead>
<tr>
<th>Non-renal</th>
<th>Renal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-acidotic</td>
<td>Probably acidic</td>
</tr>
</tbody>
</table>

Table 5: Causes of hypokalaemia.
[*usually the patients are alkalotic]

<table>
<thead>
<tr>
<th>Very poor renal function (GFR &lt; 10 – 20ml/minute)</th>
<th>Renal function normal or GFR &gt; 20ml/minute</th>
<th>Other (movement of potassium from ICF to ECF)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drugs: spironolactone, *amiloride, triamterine, angiotensin converting enzyme inhibitors. NB. non-steroidal anti-inflammatory drugs (e.g. indomethacin) lead to hyperkalaemia in about 25% patients in chronic renal failure.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 6: Causes of hyperkalaemia.
*[GFR: glomerular filtration rate. *may also cause a marked hyponatraemia.]

**Hypokalaemia (< 2.5 mM/litre)**

Weak hypotonic muscles.
Reduced or absent deep tendon reflexes.
Cramps.
Paralysis.
Tetany.
*Cardiac dysrhythmias.
Intestinal ileus.
Constipation.
Features of nephrogenic diabetes insipidus especially with chronic hypokalaemia.

**Hyperkalaemia (> 6.5 mM/litre)**

Muscle weakness.
Flaccid paralysis.
Paraesthesiae.
*Cardiac dysrhythmias.
Cardiac arrest and sudden death without warning.

Table 7: Clinical features of hypo- and hyper-kalaemia.
*[“There may be just more than usual extrasystoles causing a complaint of “palpitations” and leading to life-threatening ventricular tachycardia and / fibrillation.]
Digoxin and plasma potassium concentration.

An excess of digoxin in the plasma (as with accidental or deliberate self-poisoning) inhibits the Na-K ATPase pump of cell membranes and hyperkalaemia results. Conversely hypokalaemia increases the chance of digoxin toxicity even when the plasma digoxin concentration is within the so-called safe therapeutic range. The clinician should be aware of this risk when prescribing a combination of digoxin and a diuretic (e.g. bendrofluazide, frusemide) that causes a renal potassium loss.

Acidosis.

The terminology used for and the description of acid-base balance is often confusing. In acidosis the arterial pH is low unless compensatory physiological mechanisms act and bring the pH back to normal. Acidaemia indicates acidosis without compensation i.e. the pH remains low. Here the term acidosis will be used and only metabolic acidosis discussed.

It is important to recognise the clinical features that might suggest a metabolic acidosis and then to consider the differential diagnoses:

The clinical features that may result from any cause of metabolic acidosis:
♦ Deep sighing hyperventilation (Kussmaul respiration).
♦ Lethargy.
♦ Hypertension at first becoming hypotension when the pH falls below 7.1.
♦ Bradycardia.
♦ Cardiac extrasystoles (ventricular).

Important causes of metabolic acidosis:
♦ Diabetic ketoacidosis,
♦ Ketoadidosis may occur in the chronic alcoholic who has not eaten adequately, has had a session (“binge”) of drinking followed by vomiting.
♦ Shock from any cause especially trauma, haemorrhage and sepsis often associated with
♦ Severe hypoxia and / or severe anaemia.
♦ Liver failure.
♦ Renal failure.
♦ Poisoning with paracetamol, salicylate (“Aspirin”), methanol, ethylene glycol.
♦ Metformin and other biguanides especially given to a patient in renal or liver failure.

The treatment of potassium disturbances and metabolic acidosis.

It must be realised that the preceding discussion has been based on the assumption that no supportive biochemical measurements are available and conclusions are being made after careful clinical assessment. Early treatment and resuscitation of the dehydrated, hypovolaemic and shocked, patient will frequently prevent deterioration to the point where these disturbances become serious. The same applies to the prevention and adequate treatment of sepsis. Hyperkalaemic acidosis is always present in diabetic ketoacidosis. Normal saline infusions to correct dehydration will improve tissue perfusion and with insulin therapy will reduce the acidosis forcing potassium back into the cells. Subsequently hypokalaemia may develop.

Therefore, the general message is to treat or remove urgently the underlying causes of the “electrolyte” disturbances. Among these must be included iatrogenic causes: the important drugs potentially leading to hyperkalaemia are listed in Table 6. The diets of people living in the Tropics often contain fairly high levels of potassium e.g. kidney beans, spinach, tomatoes, sweet potatoes, bananas. Such foods should be reduced if hyperkalaemia is thought to exist but encouraged in the presence of hypokalaemia.

It would be unwise to treat suspected metabolic acidosis specifically with intravenous bicarbonate without laboratory measurements of potassium and pH but in most cases the problem will resolve if the advice above is followed. In some situations hyperkalaemia may be considered to be highly likely and a serious life threatening risk as for example in a patient with oliguric renal failure.
associated with a septic hypercatabolic state. Under these conditions the safest approach to reduce plasma potassium concentrations is to use the “glucose and insulin infusion” method:

♦ 25 – 50 grams glucose in one hour by intravenous infusion (i.e. 500ml 5% or 10% glucose) and
♦ 5 – 10 units soluble insulin at the 15th, and 45th minute of glucose infusion.

This should reduce the plasma potassium by about 1mM/litre in 30 minutes. Unfortunately the effect may last only two hours but this may give time to increase urine output by high intravenous doses of frusemide. A risk of glucose and insulin infusion is hypoglycaemia and caution is needed.

*** *** ***

From the Institute of Development Studies, University of Sussex, Brighton, UK.

Id21, based at the Institute of Development Studies, UK, has kindly offered to provide “id21 Health Highlights” that may be of particular value for Healthcare Professionals in Uganda. The web site is www.id21.org/health. "Views expressed are not necessarily those of DFID, IDS, Id21 or other contributing institutions. Unless stated otherwise articles may be copied or quoted without restriction, provided id21 and originating author(s) and institution(s) are acknowledged."

Good news at last – HIV rates fall in Uganda

Uganda was one of the first African countries to face the AIDS epidemic. Prevention programmes began in the late 1980s with strong political commitment. A study by the Medical Research Council Programme on AIDS in Uganda suggests that these efforts have produced a fall in HIV prevalence (the total number of people infected) and incidence (the number of people newly infected each year).

Studies suggest that HIV prevalence in Uganda has fallen from a peak of 29% in the late 1990's to around 8% now. However, little is known about the incidence of infection, which is the most reliable measure of epidemic trends. Researchers studied changes in incidence and prevalence among adults in 15 rural villages in south-west Uganda between 1989 and 1999.

Over the 10-year study period:

• 6,566 HIV-negative adults were tested twice or more times. This represents 31,984 person years at risk (PYARs). 190 became infected.
• HIV incidence fell from 8.0 to 5.2 per 1000 PYAR, with significant reductions in all population groups.
• The prevalence of HIV also fell significantly, especially among men aged 20-24 years (from 6.5 to 2.2 percent) and 25-29 years (15.2 to 10.9 percent) and women aged 13-19 years (2.8 to 0.9 percent) and 20-24 years (19.3 to 10.1 percent).
• Prevalence increased among older women, as infected women grew older.
• Prevalence rates were higher for women than men and also higher in migrants than in the general population.
• The median age of HIV-positive men rose from 32 to 35 years and of women from 26 to 30 years.

The researchers discuss factors that may have contributed to these changes:

• There is high awareness about HIV: 59% of adults had heard an HIV-related message in the past month. Sources include radio or newspaper (90%, friends or relatives (86%) and the study staff (79%).
• Uganda has also seen decreases in teenage pregnancy rates and increases in safer sexual practices, reported age at first sex, age at first marriage among women and reported condom use. This suggests that intensive behaviour change campaigns have been successful.
• Strategies to tackle sexually transmitted infections (STIs) can reduce the rate of new HIV infections. But there were no specific STI programmes in this population. Delays in seeking medical help and use of inappropriate treatment for STIs are still common.

More than half a million people have died from AIDS in Uganda since the epidemic began. These results give hope that behaviour change strategies can help to turn the tide.

**Contributor:** James Whitworth  
**Contact:** James Whitworth, Infectious Disease Epidemiology Unit, London School of Hygiene & Tropical Medicine, Keppel Street, London, WC1E 7HT, UK  
**Tel:** +44 (0) 207 299 4722; **Email:** jimmy.whitworth@lshtm.ac.uk

**Curbing mother to child transmission: testing pregnant women for HIV**

Testing and treating pregnant women for HIV reduces the likelihood of infecting the child. But do women want to know if they have HIV? Researchers with the Medical Research Council programme in Uganda investigated how rural women feel about counselling and testing for HIV during pregnancy. While most women are prepared to undergo testing, they are worried about confidentiality and the behaviour of medical staff.

Around 10% of pregnant women are HIV-positive in Masaka district, south-west Uganda. As many as four out of every ten babies born to HIV-positive mothers in Africa will be HIV-positive. Researchers ran discussion groups involving women whose average age was 23. The youngest were 14. The average number of children each woman has had is three, although some have had as many as nine. The majority were either pregnant or breast-feeding. Most did not know that HIV can be transmitted through breast milk. However they were aware that a baby could become infected during childbirth from the mother’s blood.

While most women are prepared to take an HIV test during pregnancy if it will protect their child, they are concerned about the stigma attached to HIV. They are also worried that:

• Maternity staff, particularly community midwives, will refuse to deliver the baby for fear of infection if the mother is HIV-positive.
• Midwives will gossip and their HIV status will become common knowledge
• Staff might even kill HIV-positive women to stem the spread of the epidemic
• They will be beaten or thrown out of the home when their husbands find out they are HIV-positive.

The acceptability of HIV testing and counselling during pregnancy could be improved by:

• increasing awareness in the community about HIV to reduce the stigma attached to being infected
• tackling husbands’ attitudes to their wives and HIV
• ensuring that medical staff will treat HIV-infected pregnant women well and respect their confidentiality, so they are reassured

**Contributor(s):** Robert Pool, S. Nyanzi & James Whitworth  
**Source(s):** ‘Attitudes to voluntary counselling and testing for HIV among pregnant women in rural south-west Uganda’, Aids Care 2001; 13 (5): 605-615, by R.Pool, S. Nyanzi and J. Whitworth.  
**Contact:** James Whitworth, Infectious Disease Epidemiology Unit London School of Hygiene & Tropical Medicine, Keppel Street, London, WC1E 7HT, UK  
**Tel:** +44 (0) 207 299 4722; **Email:** jimmy.whitworth@lshtm.ac.uk

**Casting the net – free bednets for pregnant Kenyan women**

Malaria prevention using insecticide-treated bednets (ITNs) can increase child survival, reduce illness among pregnant women and improve birth outcomes. But what is the best way to deliver ITNs to those at risk? Researchers from the Kenya Medical Research Institute, UNICEF
and the Kenyan Ministry of Health assessed the success of a scheme to distribute free ITNs to pregnant women.

Access to bednets and insecticides is poor in Kenya. Many people know about their benefit as a barrier to mosquitoes, but cannot afford them. Kenya’s National Malaria Strategy recommends providing free ITNs to vulnerable groups, such as pregnant women and their newborn children.

In 2001, UNICEF and the Government of Kenya brokered support to buy and distribute 70,000 nets and K-O TABs (deltamethrin) to pregnant women in 35 districts. To address concerns about sustainability and ‘leakage’, researchers tracked the fate of the nets. After 12 weeks of the programme:

- 92% of the nets had reached the target district headquarters.
- 53% had been distributed to pregnant women throughout the country. One-fifth had gone out to other people, with only 2,870 (4.1%) nets going astray at antenatal clinic (ANC) facilities.
- The cost of getting a net and K-O TAB to an ANC facility was US$3.81. The cost for each ITN received by a pregnant woman was US$5.26.
- Overall, a third of ANC attendees knew that the ITN service was available.

The researchers conclude that this approach to ITN delivery has advantages because it:

- uses an existing system, reducing delivery costs and simplifying logistics
- is equitable
- has the added benefit of strengthening ANC service delivery and use.

However, they emphasise the need for information on the importance of ITNs at all levels of the distribution process, including the women themselves. If free ITN distribution went to scale in Kenya, the target population (excluding Nairobi) would be around 1.44 million women. An information and education campaign would add US$0.19 to the cost of each ITN. The total cost of the delivery programme would approach US$5.8 million each year. It would be possible to saturate the entire country’s at-risk population within seven to 11 years. The researchers call for sustained donor commitment for this programme as cost recovery strategies do not ensure sustainability and can be logistically complicated, expensive and inequitable.

Contributors: Helen Guyatt, Marinus Gotink, Sam Ochola and Robert Snow
Contact: Helen Guyatt. Kenya Medical Research Institute/Wellcome Trust Collaborative Programme, PO Box 43640, Nairobi, Kenya. Fax: +254 2 711673; Email: hguyatt@wtnairobi.mimcom.net

Big risk? Malaria among pregnant women in low transmission areas

Pregnant women have an enhanced risk of malaria in regions where transmission rates are high. Is this also true in areas of low transmission? Researchers from the London School of Hygiene and Tropical Medicine tackled this question in Byumba District Hospital, Rwanda.

In low transmission areas, the entire population is at risk, but pregnant women are especially vulnerable, particularly during epidemics. Byumba District is at an altitude of 2,300 m. It has low levels of malaria transmission. The population includes non-immune returnees and refugees from the high altitude east Congolese region of Masisi.

Over a period of three years, the researchers found that:

- Heavy rainfall in February 1998 led to a malaria epidemic, which produced a fourfold increase in malaria admissions among pregnant women and a fivefold increase in maternal deaths from malaria.
- In 1998, 38% of pregnancy related admissions in Byumba Hospital were due to malaria. 71% of maternal deaths were malaria-related.
- Assuming pregnant women were as likely to be admitted as non-pregnant adults would lead to a prediction of 34, 40 and 25 malaria-related pregnant admissions in each of the three years studied. Actual admissions were 54, 198 and 67 respectively.
Pregnant women may be 1.6 – 4.9 times more likely to be admitted for malaria than other adults in low transmission areas.

These results show that malaria is an important cause of maternal illness and death in hospitals. Even in non-epidemic years, it causes a large proportion of maternal deaths. Neither the international Safe Motherhood Initiative nor the Roll Back Malaria Initiative fully address malaria in low transmission areas as a major contributor to maternal death and disease. The researchers recommend that these international initiatives should:

- acknowledge the importance of malaria as a cause of maternal death and a contributor to the disease burden in low transmission areas
- work together to apply the available control measures, such as insecticide-treated bednets, in epidemic situations
- investigate the usefulness of providing preventative anti-malarial drug treatment during pregnancy in low transmission areas.

Contributors: Asmus Hammerich, Oona Campbell and Daniel Chandramohan
Contact: Asmus Hammerich, German Technical Cooperation (GTZ) Technical Assistance Team, Indo-German Basic Health Project, West Bengal, 29 GN Block, Sector V, Bidhan Nagar, Calcutta, 700 091, India
Tel: +91 33 3574695/6; Fax: +91 33 35746957; Email: asmus_hammerich@yahoo.com

Maternal mortality in rural Gambia: levels, causes and contributing factors

Women are 75 times more likely to die as a result of pregnancy in sub-Saharan Africa than in developed regions. Reducing maternal mortality is therefore high on the international health agenda. But how effective are current efforts to improve maternal health in developing countries? What factors cause and contribute to maternal mortality? How can they be more successfully addressed? A demographic study by the Medical Research Council Laboratories in rural Gambia suggest that maternal mortality ratios are often reduced following the introduction of better obstetric care. The authors also suggest further action to improve maternal survival.

A Primary Health Care programme was introduced into the study area in 1983 with a strong mother and child health component. Antenatal clinics were also well established by this time. The researchers analysed all deaths among women aged 15 to 49 years in 40 rural villages and hamlets around Farafenni over a six-year period. They used previous estimates of maternal mortality in the area from 1982-3 and 1984-7 for comparison.

Of the 79 deaths recorded, 18 were classified as maternal. This gives a maternal mortality ratio of 424 per 100,000 live births suggesting a major improvement over the last 15-20 years. The study also found that:

- Haemorrhage is the most common cause of death.
- The village with the highest mortality is the most isolated in the study area.
- The lack of blood transfusion services at the referral level may contribute to nearly a third of maternal deaths.
- Other contributory factors include a low standard of care for obstetric referrals, poor quality primary care and a delay in starting the decision-making process.

Maternal mortality rates have continued to fall since the start of the maternal programme in the Farafenni area. Despite this, they are still 50 times greater than those in industrialised nations. The authors recommend that health policy-makers attempting to reduce maternal mortality levels further should:

- consider the introduction of the drug misoprostol to prevent postpartum haemorrhage - if this is proven to be safe and effective it could greatly increase the safety of home births
- improve the effectiveness of anti-anaemia programmes by intensifying malaria-prevention efforts among pregnant women

Contributors: Asmus Hammerich, Oona Campbell and Daniel Chandramohan
Contact: Asmus Hammerich, German Technical Cooperation (GTZ) Technical Assistance Team, Indo-German Basic Health Project, West Bengal, 29 GN Block, Sector V, Bidhan Nagar, Calcutta, 700 091, India
Tel: +91 33 3574695/6; Fax: +91 33 35746957; Email: asmus_hammerich@yahoo.com
• introduce campaigns to recruit more blood donors
• increase the accessibility and availability of high-quality essential obstetric services and primary healthcare
• implement community-based education to increase recognition of possible severe maternity problems and the need to take prompt action.

Contributors: Carine Ronsmans
Contact: Carine Ronsmans, Maternal Health Programme
Infectious Disease Epidemiology Unit, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, UK
Tel: +44 (0)20 79272190; Fax: +44 (0)20 7299 4720; Email: carine.ronsmans@lshtm.ac.uk

Is pregnancy good for your health? Evidence from Senegal

Maternal mortality is an important public health issue in developing countries. It is also widely recognised as a sensitive indicator of health system performance. But are all deaths during pregnancy directly due to child-bearing? What role do indirect causes play?

Research in Senegal suggests that very young and older women are at particular risk during pregnancy. By contrast, many women appear to be less susceptible to ill-health during and after pregnancy than women of a similar age who are not pregnant. The definition of maternal mortality has recently been expanded to include ‘indirect maternal deaths’. These are deaths from illnesses that may be aggravated by the physiological effects of pregnancy.

There is limited evidence to support the widely held view that pregnant women are more vulnerable to health risks. This study uses demographic surveillance information to examine the death rates and the causes of death of Senegalese women aged 15 to 50. It found that:

• Total death rates among all women increase with age, as expected.
• Among pregnant and recently-delivered women, those aged 45 - 49 have a death rate 18 times higher than women aged 35 - 39; those aged 15 - 19 are twice as likely to die as women aged 20 - 24.
• Between the ages of 20 and 44, pregnancy does not increase health risks.
• In fact, excluding deaths directly related to pregnancy, women aged 20 - 39 are less likely to die of other causes than non-pregnant women of the same age.

These striking findings contradict the usual assumptions about the vulnerability of pregnant women. Pregnancy may actually have a protective effect on women’s health. More work is needed to distinguish between direct or indirect causes of maternal mortality and deaths which are incidental to the pregnancy. The results are supported by the few similar studies of maternal mortality. The researchers suggest three possible explanations for this apparent protective effect:

• Pregnant women may be more concerned about their health and get better care during pregnancy.
• Physiological changes during pregnancy may directly protect women from severe diseases.
• Healthier women may be more likely to become pregnant.

Contributor: Carine Ronsmans
Contact: Carine Ronsmans, Maternal Health Programme, Infectious Disease Epidemiology Unit, London School of Hygiene and Tropical Medicine, Keppel Street, London WC1E 7HT, UK.
Tel: +44 (0)20 7927 2190; Fax: +44 (0)20 7299 4720; Email: carine.ronsmans@lshtm.ac.uk

Malaria in pregnancy – still high-risk after 20 years of research

Research two decades ago showed that malaria infection in pregnant Gambian women increased the health risks for mother and baby. A new study in Fajara shows that the situation
has not improved. Why have policy-makers failed to implement strategies to protect pregnant women?

Every year about 24 million African women become pregnant in areas where the risk of malaria is high. They are more likely than other adults to become infected and this increases the risk of poor pregnancy outcome.

This study involved 313 mothers and their babies at Bansang Hospital in the rural eastern area of the Gambia. This is the second largest referral hospital in the country, serving around 300 000 people. None of the women were taking drugs to prevent malaria infection. The study showed that:
• The mean age of these pregnant women is 23.7 years.
• The overall prevalence of placental malaria infection is 51%.
• Women are most susceptible to malaria infection in their first pregnancy.
• Malaria infection of the placenta is associated with premature delivery. Babies are four times more likely to have a low birth-weight. The average reduction in weight is 320g. Placental malaria also doubles the risk of stillbirth delivery.

These results emphasise the need for improved management of pregnancy and labour in the Gambia. Since the first study 20 years ago, several trials have produced recommendations on strategies to tackle maternal malaria. This serious public health problem may persist due to a lack of collaboration between scientists and policy-makers or because existing policy is not fully implemented.

The researchers suggest that policy-makers should:
• implement effective malaria control strategies for pregnant women, including the use of insecticide-treated bednets, and drugs to prevent and treat malaria
• ensure that control measures start as early as possible in pregnancy
• strengthen their working links with researchers
• conduct community-level research to guide programmes, and monitor and evaluate success.

Contributor: Brown Okoko
Contact: Brown Okoko, Medical Research Council Laboratories, PO Box 273, Banjul, The Gambia
Fax: +220 674089; Email: okokobrown@hotmail.com

Occupational hazard – protecting healthworkers from TB in Malawi

High rates of TB and HIV infection in sub-Saharan Africa increase the risk of healthworkers catching TB from their patients. In mid-1998, Malawi’s National Tuberculosis Control Programme produced guidelines for hospitals on TB control. Are hospitals sticking to the guidelines? Are they having any effect?

Each hospital received a copy of these guidelines and held a seminar on them for healthworkers. The guidelines include:
• rapidly diagnosing people with the most infectious form of TB infection in the lungs
• attempting to isolate infectious patients
• educating patients about cough hygiene
• offering confidential counselling and HIV testing to staff.

A year later, researchers from the Programme returned to all 40 district and mission hospitals to assess the impact of the guidelines. They found that:
• In over 90% of hospitals, ward staff say they have introduced a system of rapid sample collections. Laboratory workers say that they process these samples promptly.
• However, limited laboratory staff and facilities mean that only 35% of laboratories test samples five days a week.
• Over 70% of hospitals follow guidelines about ventilation and cough hygiene education.
• Hospitals are less successful in scheduling chest x-rays for quiet times and requiring TB patients to wear face masks.
• Introduction of the guidelines has not reduced the interval between admission and TB diagnosis, which is usually around four days, with treatment starting a day later.
• 3.2% of healthworkers were registered with TB in 1999. This is higher than the rate of 1.8% among 4,367 primary school teachers in the same year. 3.2% of healthworkers in 1999 is little different to the rate in 1996 (3.7%).

In settings where resources are very scarce, rapid diagnosis of infectious TB is one of the most important ways of reducing spread of the infection. So what can hospitals in poorer countries do to protect their staff from TB? Recommendations include:
• providing additional support for the hospitals beyond written documents and a one-off seminar
• appointing an individual or committee to be responsible for infection control procedures
• employing more laboratory staff to run tests on samples every day
• setting up occupational health and safety services, including confidential voluntary counselling and care for healthworkers with HIV
• allocating work away from general wards and TB wards for HIV-positive healthworkers.

Contributor: Anthony Harries
Contact: Anthony Harries, National Tuberculosis Control Programme, Community Health Science Unit, Private Bag 65, Lilongwe, Malawi. Email: adharries@malawi.net

TB decided… Patient choice of treatment strategies in South Africa

South Africa’s Northern Cape Province has around 547 new TB cases per 100,000 population each year. It may be impossible to provide directly observed treatment (DOT) for every TB patient in high burden settings. Are there effective alternatives to DOT? Should health facilities offer patients a range of treatment options?

The World Health Organisation (WHO) advises that all TB patients should receive their treatment under direct supervision by health workers. However, health facility-based DOT is often impractical due to the distances patients have to travel. Health workers often resort to giving patients their drugs to take home with them. But patients find it difficult to stick to self-administered therapy (SAT). An alternative is for members of the community, usually volunteers, to supervise patients’ treatment at home. These three options were compared.

The study involved 769 patients at 45 primary health care facilities - almost 20% of all TB patients registered in the province during this time. Patients were given three treatment options:
• Clinic-based DOT – they visit the clinic five days in every week.
• Community-based DOT – they visit a volunteer’s home five days each week.
• SAT – they receive a monthly supply of anti-TB drugs to take home.

Questionnaires and follow-up studies of participants showed that:
• 83% are new patients; the rest are receiving re-treatment.
• Just over half of returning patients are successfully treated compared with 70% of new patients.
• The choice of treatment delivery option does not affect the outcome for new patients. Treatment success is most likely for those who live in a rural area, have secondary education, know about the duration of treatment and do not live in a shack.
• Patients are more likely to have successful re-treatment if they are supervised. Success is not linked to TB drug side effects.
• Re-treatment patients who completed their previous treatment are just as likely as new patients to have a successful outcome.
• Re-treatment patients who opt for community-based DOT are almost ten times more likely to have a successful treatment outcome than those who receive SAT.

The successful treatment outcome reported for new patients in this study is below the WHO target of 85%, but is an improvement on rates in the province before the introduction of DOT. The study shows no added benefit of direct supervision for new patients. This suggests that other elements
The researchers highlight the importance of:
- providing treatment delivery options to patients on a voluntary basis
- telling patients how long treatment will last
- targeting treatment supervision at patients most likely to benefit from it.

**Contributors:** Samson Kironde and M Meintjies  

**Contact:** Samson Kironde, Sandown Mews East, 88 Stella Street, Sandown, Sandton, P.O.Box 652767, Benmore 2010, South Africa.  
Tel: +27 (0) 82 883 5450; Fax: +27 (0)11 506 9009; Email: skironde@hotmail.com

---

**World Health Organisation Information Office: Fact Sheets**  
*(all obtainable through the web site: http://www.who.int/inf-fs/en/)*

**Brucellosis**  
*(Fact Sheet N173, July 1997)*

Animal and human health are inextricably linked. People depend on animals for nutrition, socio-economic development and companionship. Yet animals can transmit many different diseases to humans. Diseases transmitted from animals to humans are termed zoonoses and some of them are potentially devastating.

Brucellosis is a zoonosis of both public health and economic significance in most developing countries. In many developed countries, the animal disease has been brought under control, which has led to a subsequent decrease in the number of human cases. The occurrence of the disease in humans is largely dependent on the animal reservoir. Where brucellosis exists in sheep and goats, it causes the greatest incidence of infection in humans.

**Causes**  
Six species of Brucella are currently presently known, of which *Brucella melitensis*, *Brucella suis* and *Brucella abortus* have public health implications. *Brucella melitensis* occurs more frequently than the other types in the general population and it is the most pathogenic and invasive species of *Brucella*, followed, in order, by *Brucella suis* and *Brucella abortus*.

**Transmission**  
Brucellosis is transmitted through contaminated and untreated milk and milk products and by direct contact with infected animals (cattle, sheep, goats, pigs, camels, buffaloes, wild ruminants and, very recently, seals), animal carcasses, and abortion materials. Worldwide, millions of individual are at risk, especially in developing countries where the infection in animals has not been brought under control, heat treatment procedures of milk (e.g. pasteurisation) are not routinely applied, and food habits such as consumption of raw milk and poor hygienic conditions favour human infection. In such conditions transmission of the infection to humans may frequently occur. Although the disease in animals has been brought under control in several industrialised countries, it occurs sporadically in individuals who acquire the infection abroad or by ingestion of unsafe animal products and in occupationally exposed groups (e.g. farmers, veterinarians, laboratory and slaughterhouse workers).

**Main clinical symptoms**  
The incubation period of brucellosis is usually one to three weeks, but sometimes may be several months. The illness may be mild and self-limiting or severe. It may have either a sudden or insidious onset and is accompanied by continued, intermittent, or irregular fever. The symptomatology of brucellosis is like that of many other febrile diseases, but with a marked effect on the musculoskeletal system evidenced by generalised aches and pains and associated with fatigue, prostration and mental depression. Urogenital symptoms may dominate the clinical presentation in some
patients. The duration of the disease can vary from a few weeks to several months and laboratory tests are needed to confirm the clinical diagnosis.

**Prevention** Brucellosis can be prevented in humans by controlling, or better, eliminating the disease in the animal population and avoiding consumption of raw milk and raw milk products. Brucellosis control programmes based on various strategies, including vaccination and/or test-and-slaughter of infected animals, have been successful in controlling the disease in animals in several countries, resulting in a drastic reduction in its incidence in the human population. Proper heat treatment of milk or milk products is important for effective prevention of brucellosis in humans. However, local customs, traditional habits and beliefs may impede the wide application of such measures. Health education should be intimately linked with all phases of prevention and control activities.

**Treatment** Antibiotics are effective against *Brucella*. However, *Brucella* is localised intracellularly like certain other micro-organisms (e.g., *Mycobacterium tuberculosis*), and requires the association of more than one antimicrobial for several weeks.

**Possible drug resistance** Antimicrobial resistant strains of *Brucella* are reported; however, their clinical implications are not yet fully understood. Some of the commonly-used antimicrobials for brucellosis treatment (i.e. Rifampicin and Streptomycin) are also first line drugs for the treatment of tuberculosis. The present worldwide occurrence of multi-drug resistant strains of pathogenic *Mycobacterium tuberculosis* poses the urgent question of an alternative treatment for brucellosis, using antimicrobial agents not employed for tuberculosis.

**Worldwide burden of the disease** Although human brucellosis is a notifiable disease in many countries, official figures do not fully reflect the number of people infected each year and the true incidence has been estimated to be between 10 and 25 times higher than what reported figures indicate. Cases very often remain unrecognised because of inaccurate diagnosis, and are thus treated as other diseases or as "fever of unknown origin". Animal brucellosis also poses a barrier to trade of animals and animal products and could seriously impair socio-economic development, especially of livestock owners, a most vulnerable sector in many rural populations. As an indication of the importance of this disease, plans to eliminate ovine, caprine and bovine brucellosis from the European Union were expected to receive over half of the total European Commission funding for animal diseases control measures in 1997.

Brucellosis in humans and animals is increasing in certain parts of the world, especially in developing areas of the Mediterranean Region, Middle East, western Asia and parts of Africa and Latin America. *Brucella melitensis* especially, being very pathogenic for human beings, constitutes a public health priority. Its recent emergence as a bovine pathogen in intensive dairy farms causes particular concern. A similar problem has also been reported where *Brucella suis* has become established in cattle. In Mediterranean and Middle East countries the annual incidence of brucellosis in people varies from less than 1 up to 78 cases per 100 000; however, over 550 cases have been reported from confined endemic areas where no animal control measures are applied. Up to 77 cases per 100 000 people have been reported from certain communities of south European countries in which animal control measures are mandatory. Reported cases largely underestimate the size of the problem. From a recent survey in a randomly selected human population of a country of the Arabic Peninsula, serological evidence of exposure to *Brucella* has been found to be close to 20%, with more than 2% of these having active disease. Similar figures may be expected from most countries in which the disease is endemic in the animal population. Higher seroprevalence of brucellosis should also be expected in occupationally exposed groups.

**WHO’s efforts to combat brucellosis** WHO has been involved in brucellosis work since its establishment and a number of programmes are underway to strengthen brucellosis surveillance activities at global level and to reduce the incidence of human brucellosis. In collaboration with the Food and Agriculture Organisation of the United Nations, Rome, Italy (FAO) and the Office international des Epizooties, Paris, France (OIE), WHO is promoting implementation of a regional control programme in Middle East countries. The Mediterranean Zoonoses Control Programme (MZCP) of WHO is co-ordinating a study on the evaluation of new treatment regimes for human brucellosis. The results of the pilot feasibility study were recently evaluated and the main study should begin by October 1997. WHO and the United Nations Development Programme (UNDP) are jointly assisting the Palestinian Authority to develop and implement a programme for the control of human and
animal brucellosis in the West Bank and Gaza Strip. In the Americas Region, the Pan-American Health Organisation and the Regional Office of WHO have launched an initiative for bovine brucellosis elimination from Latin American countries. WHO has also developed a number of educational material for the attention of travellers and consumers on dietary precautions, promoting, among others, heat treatment of milk and derived products. WHO is currently developing guidelines for integrated surveillance of brucellosis and promoting research on new brucellosis vaccines for both humans and animals.

The “Undefined and hidden burden” of mental health problems.
(Fact Sheet N° 218, Revised November 2001)

The Undefined Burden of mental problems refers to the economic and social burden for families, communities and countries. Although obviously substantial, this burden has not been efficiently measured. This is because of the lack of quantitative data and difficulties in measuring and evaluating.

The Hidden Burden refers to the burden associated with stigma and violations of human rights and freedoms. Again, this burden is difficult to quantify. This is a major problem throughout the world, as many cases remain concealed and unreported.

Undefined Burden
Mental illnesses affect the functioning and thinking processes of the individual, greatly diminishing his or her social role and productivity in the community. In addition, because mental illnesses are disabling and last for many years, they take a tremendous toll on the emotional and socio-economic capabilities of relatives who care for the patient, especially when the health system is unable to offer treatment and support at an early stage. Some of the specific economic and social costs include:

- lost production from premature deaths caused by suicide (generally equivalent to, and in some countries greater, than deaths from road traffic accidents);
- lost production from people with mental illness who are unable to work, in the short, medium or long term;
- lost productivity from family members caring for the mentally-ill person;
- reduced productivity from people being ill while at work;
- cost of accidents by people who are psychologically disturbed, especially dangerous in people like train drivers, airline pilots, factory workers;
- supporting dependants of the mentally ill person;
- direct and indirect financial costs for families caring for the mentally-ill person;
- unemployment, alienation, and crime in young people whose childhood problems, e.g., depression, behaviour disorder, were not sufficiently well addressed for them to benefit fully from the education available;
- poor cognitive development in the children of mentally ill parents, and the emotional burden and diminished quality of life for family members.

The Hidden Burden
Stigma can be defined as a mark of shame, disgrace or disapproval, which results in an individual being shunned or rejected by others. The stigma associated with all forms of mental illness is strong but generally increases the more an individual's behaviour differs from that of the 'norm'. Because of stigma, persons suffering from a mental illness are:

- often rejected by friends, relatives, neighbours and employers leading to aggravated feelings of rejection, loneliness and depression;
- often denied equal participation in family life, normal social networks, and productive employment;
- Stigma has a detrimental effect on a mentally ill person's recovery, ability to find access to services, the type of treatment and level of support received and acceptance in the community;
- Rejection of people with mental illness also affects the family and caretakers of the mentally ill person and leads to isolation and humiliation; and
- A major cause of stigma associated with mental illness are the myths, misconceptions and negative stereotypes about mental illness held by many people in the community.
The stigma can be reduced by:
- openly talking about mental illness in the community;
- providing accurate information on the causes, prevalence, course and effects of mental illness;
- countering the negative stereotypes and misconceptions surrounding mental illness;
- providing support and treatment services that enable persons suffering from a mental illness to participate fully in all aspects of community life;
- ensuring the existence of legislation to reduce discrimination in the workplace, in access to health and social community services.

Human rights violations
Persons experiencing mental problems are more vulnerable than others in their social dealings and, as a result, are at a relatively higher risk to have their human rights and freedoms violated. These include:
- the right not to be discriminated against (e.g., in access to health care, social services or employment);
- the right to liberty (e.g., not to have restrictions automatically imposed on freedom of movement through measures such as detention);
- the right to integrity of the person (e.g., not to be unduly subjected to mental or physical harm. Typical violations include treatment that ignores the requirement to obtain either the patient's informed consent or a surrogate decision-maker's, and sexual abuse);
- The right to control one's own resources (e.g., one should not be automatically removed on the mere grounds of status as a mental patient, but should be judged on his or her actual ability to manage resources).

Mental health legislation – a necessary requirement
General principles for mental health legislation to protect the rights of the mentally ill include:
- Respect for individuals and their social, cultural, ethnic, religious and philosophical values.
- Individuals’ needs taken fully into account. Individual’s need for health and social care must be assessed thoroughly. In particular, it is important to ensure that the views of an individual (and his or her carers) are considered. For this to happen, there must be close liaison between health, housing and social care services.
- Care and treatment provided in the least restrictive environment. In order to uphold this principle, legislation should be framed so that involuntary (formal) hospital admission is a last resort. This can be achieved through: clearly defined grounds for detention; procedural safeguards when the power to detain is used; an obligation to discharge when grounds for detention are no longer met; an independent review of the decision to detain.
- Provision of care and treatment aimed at promoting each individual’s self-determination and personal responsibility. It is vital that individuals are given the opportunity to exercise choice and make decisions about their own care and treatment. Legislation should aim to ensure that: treatment can be imposed only in strictly limited and clearly defined circumstances and must be the least restrictive alternative; where individuals are unable to make decisions for themselves, steps are taken to find out their wishes and feelings; clear information on treatment and detention is readily available; appropriate provisions for confidentiality are in force.
- Provision of care and treatment aimed at achieving the individual’s own highest attainable level of health and well-being. In addition, to issues of quality and continuity of care, this principle addresses the question of a “right” to treatment. It can also cover more general issues such as the requirement that the individual should be cared for properly in a safe environment and subject only to restrictions for reasons of his or her health or safety, or the safety of others. In this regard: there should be no restrictions on an individual’s contact with friends and family, except in rare and clearly defined circumstances; stringent safeguards from abuse, exploitation and neglect should be in place.

***   ***   ***
Multiple Choice Questions Quiz (19) (May - June 2002)  
(Answers)

A. The following are causes of a tachycardia:
1. Atrial fibrillation.  
   True
2. Atrial flutter.  
   True
3. Ventricular tachycardia.  
   True
4. Ventricular fibrillation.  
   False (there is no effective cardiac output)
5. Beta-blockade.  
   False (bradycardia)

B. The following may cause a sinus tachycardia:
1. Anaemia.  
   True
2. Hypothyroidism.  
   False (bradycardia)
3. Atropine.  
   True
4. Fever.  
   True
5. Constrictive pericarditis.  
   True

C. Hyperventilation may be the result of:
1. Metabolic acidosis.  
   True
2. Hypercapnia.  
   True
3. Pneumothorax.  
   False (tachypnoea)
4. Hypoventilation.  
   False (this is the opposite of hyperventilation)
5. Pain.  
   True

D. Carcinoma of the bronchus may cause:
   True
2. Cushing’s syndrome by metastasising to the adrenal glands.  
   False (causes hypoadrenalism)
   True
4. Empyema of the gall bladder.  
   False (may cause empyema thoracis)
5. Hypoglycaemia.  
   True (endocrine syndrome)

E. The following statements are true:
1. Bone marrow suppression by chloramphenicol is always reversible.  
   False
2. All tetracyclines are contraindicated in the presence of impaired renal function.  
   False (not doxycycline)
3. Chloramphenicol is metabolised mainly by the liver.  
   True
4. The effect of phenytoin is reduced by chloramphenicol.  
   False
5. Chloramphenicol may be given safely at all stages of pregnancy.  
   False (not in very late pregnancy)

F. These statements about tuberculosis (TB) are true:
1. Epididymitis is the commonest presentation of genital TB in the male.  
   True
2. In spinal TB it is uncommon for two adjacent vertebrae to be involved.  
   False
3. Abdominal TB is more common in females than males.  
   True
4. In the early stages of TB meningitis the fever is high and “swinging”.  
   False (low grade)
5. A patient given isoniazid is more likely to develop a peripheral neuropathy if he / she is malnourished.  
   True

G. These statements are true in relation to Ebola virus disease (EVB):
1. The Ebola and Marburg viruses are indistinguishable.  
   False (different antigens)
2. The incubation period of EVD is 7 – 14 days.  
   True
3. The onset of EVD is gradual.  
   False (sudden)
   True
5. Anti-Ebola convalescent serum has been shown to be of no value.  
   False

H. The following are true about schistosomiasis:
1. Schistosoma mansoni eggs have a terminal spike.  
   False
2. Periportal hepatic fibrosis is the most important complication of chronic infestation with S. mansoni.  
   True
3. Katayama syndrome (acute toxaemic schistosomiasis) caused by S. mansoni has an incubation period of 1 – 3 weeks.  
   False (3 – 7 weeks usually)
4. Praziquantel is effective against all schistosomal species affecting humans.  
   True
5. Haematemesis may be the presenting feature of hepatosplenic schistosomiasis.  
   True
I. These statements are true about malaria:
   1. The mean incubation period for Plasmodium falciparum (PF) malaria is 16 days. **False (13 days)**
   2. At autopsy, the brain of a patient dying from PF malaria shows small petechial haemorrhages mainly in the grey matter. **False (white matter)**
   3. Large cerebral haemorrhages and infarcts are rare in cerebral malaria. **True**
   4. PF malaria during pregnancy has little effect on the birth-weight of the baby. **False (reduces birth weight)**
   5. Five percent of children who survive cerebral malaria have a persistent neurological deficit. **False (10%)**

Results of MCQ Quiz (19) (April – May 2002)

The only entrant for this quiz was Dr. Charles Deo from Hoima Hospital: he obtained a net score of 13 points from a maximum of 45. So Dr. Deo wins the prize.

Multiple Choice Questions Quiz (21)

Here follows a set of MCQ’s on a variety of topics. If anyone wishes to send in a set of answers then it is suggested that a copy be made of the questions. The answers “True” (T), “False” (F) or “Don’t know” (DK) should be put clearly alongside and level with each question. Answers **must be sent to the address below and arrive before the 30th April 2003**. The answers to the MCQ’s will be in the May - June 2003 issue of the Newsletter. There will be a prize for the best set of answers. **Do ensure that your name and address is clearly stated.**

A. The following are causes of the carpal tunnel syndrome:
   1. Pregnancy.
   2. Rheumatoid arthritis.
   3. Acromegaly.
   4. Hypothyroidism.

B. Aspiration pneumonia
   1. May be due to oesophageal disease.
   2. Can lead to a lung abscess.
   3. Is usually due to pneumococci.
   4. Occurs more often on the right side than the left.

C. In acute osteomyelitis
   1. Infection usually begins in the epiphysis.
   2. The infarct becomes ensheathed by an involucrum.
   3. Cloacae may develop which allow pus to discharge through the involucrum.
   4. Infection is usually haematogenous (blood spread).

D. In syphilis
   1. The causative organism is Treponema pertenue.
   2. The incubation period is usually 17 – 28 days.
   3. The primary chancre is tender.
   4. A maculopapular rash is usual in the primary stage.

E. In chronic alcoholism
   1. Delirium tremens may be precipitated by a surgical operation.
   2. The cerebellar disturbance characteristically involves the arms.
   3. Epilepsy is a recognised associated feature.
   4. Dementia rarely occurs in longstanding cases.
F. After head injury there is an increased rate of:
   1. Depression.
   2. Dementia.
   3. Personality disorder.
   4. Suicide.

G. In miliary tuberculosis:
   1. The chest Xray may be normal.
   2. A negative tuberculin test excludes the diagnosis.
   3. The patient may have no symptoms except a mild fever.
   4. Tubercular meningitis is uncommon.

H. Alcohol
   1. Is metabolised by alcohol dehydrogenase.
   2. May be associated with gynaecomastia.
   3. In excess may cause red blood cell macrocytosis.
   4. In excess may cause a constrictive cardiomyopathy.

I. Recognised features of lead poisoning are:
   1. Abdominal pain.
   2. Diarrhoea.
   3. Convulsions.
   4. Deterioration of intellect.

J. In osteoarthritis the following are common:
   1. An ESR greater than 40 mm.
   2. Heberden’s\textsuperscript{2} nodes.
   3. Morning stiffness until mid-day.

[The marking system for this Self Assessment Questionnaire will be based on “+1” for a correct answer and “zero” for “don’t know and “-1” for an incorrect answer.]

Please insert your full name and address below:

........................................................................
........................................................................
........................................................................

and return to:

Dr. David Tibbutt,
"Perry Point",
4, Whittington Road,
WORCESTER,
WR5 2JU,
UK.

\textsuperscript{2} Strictly these should be called Heberden-Rosenbach nodes:

W. HEBERDEN (1710 – 1801) WAS AN English physician. He produced classic descriptions of angina, chicken pox and night blindness. He also observed that tuberculosis seemed to improve in pregnancy but not in the postpartum period. O. ROSENBACK (1851 – 1907) was a German physician.