

## SAFETY ALERTS

# Improving the safety of oxygen therapy in hospitals: summary of a safety report from the National Patient Safety Agency

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### Why read this summary?

Oxygen is used commonly and can save lives by preventing severe hypoxaemia.<sup>1</sup> However, serious harm is possible if oxygen is not managed properly and the risks are poorly understood by trainee doctors and others.<sup>2</sup> Underuse of oxygen can cause hypoxic organ damage, whereas overuse may harm neonates or cause hypercapnic respiratory failure in patients with chronic obstructive pulmonary disease.

From December 2004 to June 2009, healthcare staff reported 281 serious incidents relating to oxygen use to the National Patient Safety Agency in England and Wales. Among these incidents, poor oxygen management seems to have caused nine deaths and may have contributed to a further 35 deaths. A typical incident report reads: "Patient [has] known type 2 respiratory failure with a diagnosis of COPD [chronic obstructive pulmonary disease] and who had previously required BIPAP [bilevel positive airway pressure] to control hypercapnia, [and was] switched to 15 litres O<sub>2</sub> via face mask by nurse as he had low saturations, without the advice of a doctor. The patient was seen several hours later, GCS3 [Glasgow coma score 3], profound respiratory acidosis. Cardiac arrest and died that afternoon."

This summary is based on a safety report (known as a "rapid response report" or "RRR") from the National Patient Safety Agency on the safe use of oxygen use in hospitals, with key actions for staff.

### Problems identified by the National Patient Safety Agency

Problems included:

- Administration of oxygen without prescription or other written order
- Incorrect oxygen flow levels
- Failure to monitor and act on abnormal oxygen saturation levels
- Confusion of oxygen with medical compressed air (or other gas)
- Empty cylinders or other faulty or missing equipment.

The National Patient Safety Agency issued its RRR on the safe use of oxygen in hospitals in September 2009 (NPSA/2009/RRR06, [www.nrls.npsa.nhs.uk/oxygen](http://www.nrls.npsa.nhs.uk/oxygen)).

### What can we do?

The RRR recommended that hospitals minimise the use of oxygen cylinders on wards; check processes for using cylinders in transfer and emergency situations; make pulse oximeters available on wards; remove or cover medical air flow meters when not in regular use; and provide training for relevant staff.

The RRR also endorsed the British Thoracic Society's guidelines,<sup>3</sup> highlighting key messages for individual clinicians to make practice safer:

For individual clinicians, the RRR also recommended:

- Ask yourself if the patient needs oxygen (despite established practice, evidence does not support routine use of oxygen for non-hypoxaemic patients with stroke, myocardial infarction, or breathlessness)
- Prescribe oxygen according to target saturation rate: 94-98% for most acutely ill patients but 88-92% for those at risk of hypercapnic respiratory failure (such as patients with chronic obstructive pulmonary disease)
- Select the appropriate device: a nasal cannula administering medium dose oxygen for most patients; reservoir masks giving high dose oxygen therapy for critically ill patients; 28% venturi masks for high risk patients with chronic obstructive pulmonary disease and others needing low dose oxygen
- Record target saturation, oximetry results, method of delivery, and oxygen dose on bedside observation chart
- Check blood gases in all critically ill patients and if acidosis or hypercapnia is suspected (as oximetry provides no information on acidity levels or partial pressure of carbon dioxide)
- Monitor results and use established early warning "track and trigger" systems such as MEWS (modified early warning scores) to identify and rescue deteriorating patients<sup>4,5</sup>
- Be aware of colour coding of flow meters (in the United Kingdom, white indicates oxygen, black air) and remove air flow meters from the wall sockets when not in use
- In emergencies, give oxygen immediately and record later.

This is one of a series of *BMJ* summaries of recommendations to improve patients' safety, based on reports of safety concerns, incident analysis, and other evidence. The articles will highlight the risks of incidents that have the potential for serious harm and are not well known, and for which clear preventive actions are available.

To report adverse events to the National Patient Safety Agency, go to [www.nrls.npsa.nhs.uk/](http://www.nrls.npsa.nhs.uk/)

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Insertion of chest drains: summary of a safety report from the National Patient Safety Agency (2009;339:b4923)

National Patient Safety Agency: combining stories with statistics to minimise harm (2009;339:b4489)

Avoiding midazolam overdose: summary of a safety report from the National Patient Safety Agency (2009;339:b4459)

**What else do we need to know?**

Current clinical guidelines for use of emergency oxygen are available only for patients aged over 16 years. Evidence based guidance is needed on oxygen use in children and neonates, including appropriate target saturation ranges, and on particular challenges, such as recognising hypoxaemia in very young patients.

Although an international consensus statement for target oxygen saturation in critical care is available,<sup>6</sup> no UK evidence based guidelines on oxygen use in such units exist.

The RRR presents immediate steps to improve oxygen safety. In the long term, design solutions are needed to make it impossible to connect standard oxygen tubing to an air outlet. Ultimately, compressed air outlets on general wards may become redundant, with greater use of electrically driven and ultrasonic nebulisers and other new inhaler devices.

**How will we know when practice has become safer?**

A baseline national audit of 99 UK hospitals by the British Thoracic Society in 2008 (before issuing its guidelines) found that only 32% of patients who were receiving oxygen had any sort of written order for oxygen use and even fewer (10%) had a prescription with a target saturation range.<sup>7</sup> Provisional data from a repeat audit at the end of 2009<sup>7</sup> suggest that oxygen prescribing is improving. The deadline for hospitals to comply with the NPSA's recommendations on training and system changes is the end

of March 2010—further audits will be needed to monitor progress over time.

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**Competing interests:** All authors have completed the Unified Competing Interest form at [www.icmje.org/coi\\_disclosure.pdf](http://www.icmje.org/coi_disclosure.pdf) (available on request from the corresponding author) (URL) and declare they had: (1) No financial support for the submitted work from anyone other than their employer; (2) No financial relationships with commercial entities that might have an interest in the submitted work; (3) No spouses, partners, or children with relationships with commercial entities that might have an interest in the submitted work; (4) BRO'D co-chairs the British Thoracic Society Emergency Oxygen Guideline Group.

**Provenance and peer review:** Commissioned; not externally peer reviewed.

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**EASILY MISSED?****Long QT syndrome**

Dominic J Abrams,<sup>1</sup> Malcolm A Perkin,<sup>2</sup> Jonathan R Skinner<sup>3</sup>

Congenital long QT syndrome is a potential cause of avoidable sudden cardiac death. Affected individuals may have ventricular arrhythmias, leading to palpitations, syncope, and, if sustained, cardiac arrest.<sup>1</sup> The syndrome is inherited in an autosomal dominant fashion, with variable disease expression: those severely affected may die in fetal or neonatal life, but others remain asymptomatic throughout their life. At a cellular level, genetically encoded abnormalities in sodium and potassium ion channels within the cell membrane lengthen cardiac repolarisation, which manifests as a prolongation of the QT interval in the electrocardiogram. QT prolongation may be acquired secondary to certain medications, metabolic disturbance, cerebral injury, myocardial disease, and hypothermia—factors that may also unmask the congenital syndrome in a previously asymptomatic individual.

**Why is long QT syndrome missed?**

Syncope is highly prevalent in young adults. Among 394 students, 154 reported at least one episode of syncope.<sup>4</sup> In a young, fit adult (such as outlined in the scenario box), important differential diagnoses include the most common

and benign cause of syncope, neurocardiogenic (vasovagal) syncope,<sup>4</sup> as well as primary arrhythmias, cardiomyopathies, and structural heart disease that all warrant further cardiology evaluation. Cerebral hypoperfusion during arrhythmic syncope may manifest as myoclonic jerks or epileptic type movements,<sup>5</sup> leading to long QT syndrome often being misdiagnosed as epilepsy. In a recent study 39% of patients in a cohort with long QT syndrome had a delayed diagnosis after presentation with seizures or syncope, which were most frequently misdiagnosed as

**CASE SCENARIO**

A 19 year old female student consulted her general practitioner about two recent episodes of syncope, both of which occurred while playing hockey. Her team mates reported that she collapsed suddenly with little warning, recovering rapidly within 30 seconds without confusion. She was otherwise well, although she was taking erythromycin for an infected leg abrasion at the time of the events. As part of the routine evaluation for syncope, her general practitioner performed a 12 lead electrocardiogram, which showed a prolonged corrected QT interval of 510 ms.

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

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epilepsy but also as breath-holding attacks and vasovagal syncope. The time between presentation and diagnosis of long QT syndrome ranged from two months to 23 years, and in four cases another family member died before the correct diagnosis had been made.<sup>6</sup>

### Why does this matter?

In long QT syndrome, prolongation of the QT interval and appropriately timed ventricular ectopy (R on T phenomenon) leads to the development of torsades de pointes, a ventricular arrhythmia with a classic appearance in an electrocardiogram (figure 1). Torsades de pointes will often resolve spontaneously, although may degenerate to ventricular fibrillation, leading to cardiac arrest and potentially sudden cardiac death. Early identification of a patient with long QT syndrome and prompt, appropriate treatment are therefore critical to prevent cardiac events. All drugs that prolong the QT interval should be stringently avoided in patients with long QT syndrome (box). As the syndrome is an inherited condition, identification of other family members at risk is essential.

### How is it diagnosed?

The diagnosis is a clinical one based on electrocardiographic findings and clinical and family history.

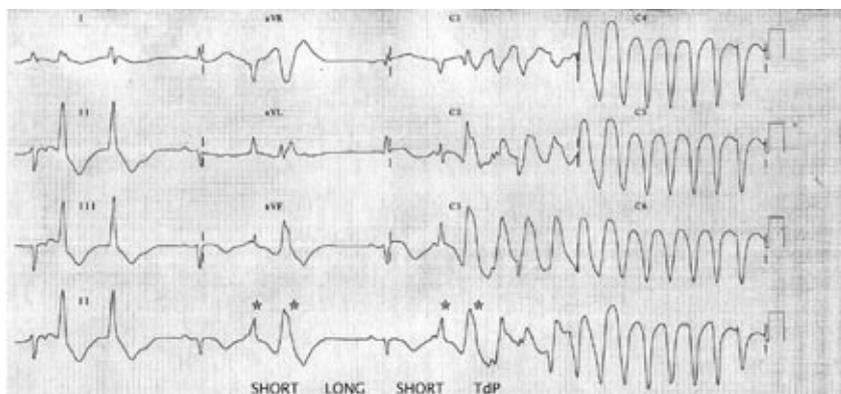
### Clinical features

#### History taking

An accurate history is essential, so ask patients to talk through the event(s) step by step, carefully describing their symptoms and timing. Seek witness statements where possible. Features that stand out are listed below.

- *Exertional syncope*—this should always raise alarm of a sinister cause. Three genetically and clinically distinct subtypes of congenital long QT syndrome exist:

- Long QT1 is associated with events during exercise, particularly swimming, with frequent symptoms but lower mortality
- Long QT2 events occur during emotional stress or auditory stimuli, especially waking from sleep (such as ringing telephones and alarm clocks)
- Long QT3 is associated with events at rest;



**Fig 1** | Initiation of torsades de pointes. A 12 lead electrocardiogram and rhythm strip (lead II) recorded from a patient with long QT syndrome and a corrected QT interval of 540 ms. The rhythm strip at the bottom of the image shows numerous ventricular extrasystoles (indicated by asterisks) creating alternating short, long, and short RR intervals initiating torsades de pointes (TdP)

### Commonly prescribed agents that prolong QT interval and should be avoided in patients with congenital long QT syndrome\*

#### Antiarrhythmics

- Amiodarone
- Sotalol

#### Antibiotics

- Erythromycin
- Clarithromycin
- Ciprofloxacin

#### Antihistamines

- Terfenadine

#### Antidepressants

- Fluoxetine
- Sertraline
- Amitryptiline

\*A comprehensive list is available at [www.azcert.org](http://www.azcert.org)

### HOW COMMON IS LONG QT SYNDROME?

The exact prevalence of long QT syndrome in the population is unknown as disease expression may be highly variable

Current population estimates range from 1 in 2000<sup>2</sup> to 1 in 3000<sup>3</sup>

Most general practices in the UK are likely therefore to have at least one patient with the condition

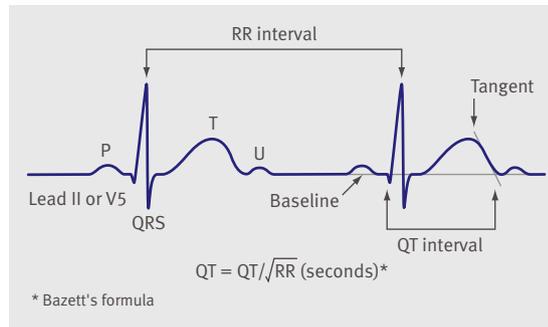
such events are less common but have a higher associated mortality.<sup>1</sup>

- *Vasovagal syncope*—this is most commonly triggered by a warm environment, prolonged standing, painful stimuli, or insufficient food intake.<sup>4</sup>
- *Sudden syncopal events*—as in the case scenario we describe, these are an atypical feature of vasovagal syncope, in which dizziness, sweating, and visual disturbances often precede loss of consciousness. In long QT syndrome patients may have rapid palpitations as the first symptom, whereas a compensatory sinus tachycardia after the onset of other symptoms may occur in benign syncope.
- *Rapid recovery*—after the syncopal event, rapid recovery without confusion or drowsiness is characteristic of cardiac syncope. Although epileptic-type movements secondary to cerebral anoxia may occur in long QT syndrome,<sup>5,6</sup> neurological features such as aura and postictal confusion are typically absent.
- *Drug history*—ask about any drugs used when symptoms were present as many commonly used agents (box) prolong the QT interval and may precipitate cardiac arrhythmias in patients with long QT syndrome.<sup>7</sup> Similarly, unexplained syncope in older patients should trigger a careful review of medication.
- *Family history*—ask specifically whether there is a family history of unexplained sudden death (including in young children), refractory epilepsy, or recurrent syncope

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## Easily Missed? articles

- Ovarian cancer (2009;339:b4650)
- Slipped capital femoral epiphysis (2009;339:b4457)
- Subarachnoid haemorrhage (2009;339:b2874)



**Fig 2 |** Measurement of the QT interval using the tangent technique. The QT interval is measured from the onset of the Q wave to the end of the T wave, defined as the intersection of the tangent to the steepest downslope of the T wave and the baseline. To adjust for the effects of heart rate, Bazett's formula is used to calculate the corrected QT interval (QTc) Adapted with permission from Postema et al<sup>2</sup>

### Clinical examination

Patients with long QT syndrome have a structurally and functionally normal heart, so a cardiac examination will yield a normal result. Any abnormal findings on cardiac examination suggest a structural or cardiomyopathic cause of syncope instead.

### Investigations

Measurement of the QT interval in the electrocardiogram is the primary investigation, with figures of >450 ms in males and >460 ms in females suggesting QT prolongation. The QT interval is calculated automatically and displayed on the electrocardiogram, but errors are common, and manual measurement is advised (figure 2). Inaccurate calculation of the QT interval<sup>2</sup> may lead to a false negative<sup>6</sup> or false positive diagnosis.<sup>8</sup> Measurement of the QT interval after exercise testing<sup>9</sup> or at times of bradycardia on 24 hour Holter monitoring may help in the identification of carriers of the long QT gene who have a normal QT interval on resting electrocardiographic evaluation.

Once long QT syndrome has been diagnosed definitively, genetic analysis for mutations in the genes encoding for cardiac ion channels may be helpful in confirming the subtype and in identifying all other affected family members. At present 65-70% of affected individuals will have an identifiable mutation in the known genes.

### KEY POINTS

Long QT syndrome is a familial condition associated with recurrent syncope and sudden cardiac death resulting from ventricular arrhythmias; it may be misdiagnosed as epilepsy

Triggers for arrhythmias may include medications that prolong the QT interval or subtype specific factors such as swimming and other exercise (long QT1), auditory stimuli and emotional stress (long QT2), and rest or sleep (long QT3) β blockers are usually highly effective, with implantable cardioverter defibrillators reserved for people deemed at high risk or refractory to medical treatment

Thoracoscopic left cardiac sympathectomy is highly effective and is useful if β blockers are not tolerated or an implantable cardioverter defibrillator is contraindicated

### How is it managed?

The mainstay of treatment is β blockers. These are most effective in the long QT1 subtype, in which the prognosis is excellent if patients avoid drugs that prolong the QT interval<sup>7</sup> and specific triggers such as competitive exercise.<sup>10</sup> Although β blockers are least effective in long QT3, they may still be beneficial.<sup>11</sup> Patients should continue taking β blockers at all times, including pregnancy and peripartum.<sup>12</sup> As in any long term treatment, non-compliance<sup>7</sup> and contraindications (such as asthma) provide particular challenges. In high risk patients (including those who have survived a cardiac arrest, those with a recent history of syncope or with a QT interval >500 ms, males under 18 years, or females over 18 years<sup>13 14</sup>), consider implantable cardioverter defibrillators in conjunction with β blockers. If β blockers are not tolerated, defibrillators are contraindicated, or symptoms persist despite optimal β blockade, thoracoscopic left cardiac sympathectomy is highly effective.<sup>15</sup> All patients should be given a list of medications that prolong the QT interval and be advised carefully of specific triggers.

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