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Inappropriate use of computed tomography chest scanning in hospital patients

Askin Gunes, Lloyd J Ridley and Graham Simpson

TO THE EDITOR: Computed tomography (CT) of the chest is superior to chest x-ray as an imaging modality of the lungs, mediastinum, pleura and the chest wall,¹ and its use is increasing for a range of diagnostic and therapeutic applications.² There are clear indications for the appropriate use of chest CT, and adherence to these can reduce cost, workload, procedure-related complications and radiation exposure. Our group recently analysed referrals for chest CT from general practice, and found that the scan was clinically helpful in only 12%, and inappropriate in 68%.³ We thus examined the indications for ordering CT of the chest, and the associated outcomes in hospital inpatients, who had been referred for chest CT by general physicians.

Two respiratory physicians retrospectively reviewed the clinical files, the CT request form, and previous and current imaging of 47 consecutive non-surgical patients admitted to Cairns Base Hospital between 1 January and 1 July 2005. One illustrative patient's case is described in the

Box. The impact of the chest CT on the patient's clinical outcome was assessed. We used the imaging guidelines of the Royal Australian and New Zealand College of Radiologists (RANZCR) as the standard for evaluating appropriate ordering of chest CT.⁴

Overall, chest CT was appropriately ordered in 26 of 47 patients (55%). The correct type of scan (contrast, non-contrast or high resolution) was requested for 38 of the 47 patients (81%). In 25 of the 26 appropriately ordered scans (96%), the patient's physicians had compared the CT scan with previous chest x-rays and recorded this in the file; this was done for only 11 of the 21 inappropriately ordered scans (52%; $P = 0.001$).

Further useful information that had not been detected by other means was obtained from the CT scan (compared with chest x-ray alone) in 26 of 47 patients (55%, comprising 25 of 37 [68%] in the subgroup in whom the CT had been ordered appropriately and one of 10 [10%] in the group ordered inappropriately; $P = 0.01$). Management was changed as a result of CT scanning in 19 of 47 patients (40%): 18/26 (69%) in the appropriately ordered CT group and 1/21 (5%) in the inappropriately ordered CT group ($P = 0.001$). The correct type of CT scan led to

a higher incidence of change in management (18 of 38 patients; 47%; $P = 0.046$).

We encourage all doctors to use the RANZCR guidelines, or web-based imaging pathways such as those developed by Royal Perth Hospital <www.imagingpathways.health.wa.gov.au> to ensure better clinical practice.

Acknowledgements: We thank the staff of Cairns Base Hospital Radiology Department and Rabia Khan, Tropical Public Health Unit Network, Queensland Health, for her guidance with the statistical analysis.

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An illustrative case of acute respiratory illness from the study

A 41-year-old woman with a past history of asthma was admitted to hospital with moderately severe right-lower-lobe pneumonia. She responded to antibiotic and bronchodilator therapy and was discharged on Day 6 with no complications. During her admission she had five chest x-rays and high-resolution computed tomography (HRCT) of the chest to rule out empyema; all of these showed consolidation with a small effusion. In outpatient follow-up, she had two further chest x-rays and HRCT of the chest repeated once during Week 3 because of "slowly resolving" shadows.

Assessment and comment:

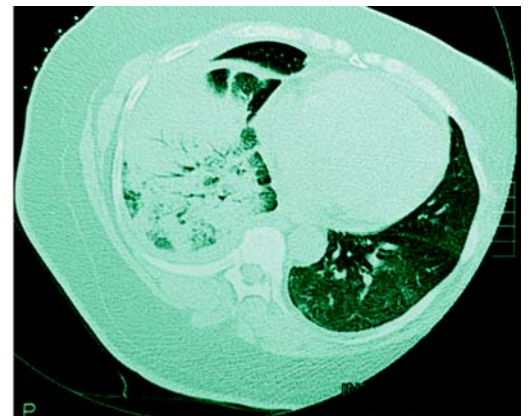
The imaging guidelines of the Royal Australian and New Zealand College of Radiologists⁴ recommend that further imaging is indicated for clinical deterioration, complications, or slow recovery. Thus, this patient did not need computed tomography (CT) scanning, as none of these criteria were met. Had CT been indicated, conventional CT, and not HRCT, would have been the correct choice. A repeat chest x-ray with a lateral view at discharge and at 6 weeks would have been the appropriate management in this patient.



Chest x-ray, Day 1.



Chest x-ray, Week 3.



High-resolution chest CT, Day 1. ◆

Unexpected benefits of bethanechol in adults with cerebral palsy

Warwick J Carter

TO THE EDITOR: Bethanechol is a parasympathomimetic agent similar to acetylcholine that is known to be a selective stimulant of smooth muscle in the gastrointestinal tract and urinary bladder. It is normally used to treat non-obstructive urinary retention and has not previously been known to have any effect on skeletal muscle.

Adults with cerebral palsy usually slowly deteriorate over the years, with gradually increasing muscle tone, worsening speech, mobility difficulties and a loss of independence. There has been no change in their management for decades.

While working in a residential facility for adults with cerebral palsy, we serendipitously found that bethanechol significantly reduced the muscle spasticity in a patient for whom it was initially used to treat micturition difficulty.

Seven other patients who were wheelchair-bound with cerebral palsy were then progressively given bethanechol in increasing doses. All patients and/or their carers were advised that the medication was being used experimentally, and all consented to participate in a clinical trial. The results are summarised in the Box.

In all patients, bethanechol treatment was ceased for a week once the clinical benefits had been established, and all deteriorated during that week.

None of the patients suffered any detectable side effects from the use of bethanechol, but many were already taking a proton-pump inhibitor that may have protected them from any gastrointestinal adverse effects.

A synergistic interaction between bethanechol and another medication (eg, diazepam) was excluded as an explanation for the results obtained, as no other medication was common to all patients.

Bethanechol's effect seems to be long-lasting, as the first patient has now been using it for 6 months with no deterioration in his improved muscle tone.

A MEDLINE search revealed no studies in which bethanechol had been used as a treatment for cerebral palsy. Although our sample was very small, the fact that every patient improved indicates that a larger trial of bethanechol for cerebral palsy is warranted.

Acknowledgements: I would like to thank physiotherapist Diane Josephson and registered nurse Lynne Roche for their assistance with the study.

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Desflurane-induced acute liver failure

Marcus W Chin, Dolores B Njoku,
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and Nickolas Kontorinis

TO THE EDITOR: It has been well established that traditional inhalational anaesthetic agents can cause mild and sometimes fulminant liver failure.¹ However, while newer inhalational agents are a theoretical cause of hepatotoxicity, such cases have rarely been reported.^{2,3}

We describe desflurane-induced acute liver failure in a 53-year-old woman with achalasia, hypertension, type 2 diabetes mellitus and hyperlipidaemia. She underwent a Heller myotomy for treatment of the achalasia in late 2004. During anaesthesia, desflurane was administered (1.2 minimum alveolar concentration [MAC]) via a Datex-Ohmeda Aestiva/5 anaesthesia delivery system (GE Healthcare, Sydney, NSW). After the operation, her serum alanine aminotransferase (ALT) concentration peaked at 943 U/L (reference range, <35 U/L). This was attributed to antibiotic toxicity. As the initial myotomy was inadequate, the surgery was repeated 10 days later with desflurane (0.9 MAC) anaesthesia. The patient developed acute liver failure 96 hours after surgery (serum ALT level, 11 600 U/L; pH, 7.06; international normalised ratio, 3.7) and died despite supportive management. A postmortem examination confirmed massive hepatic necrosis and significantly elevated trifluoroacetyl chloride-specific IgG4 antibodies (optical density, 0.585; reference range, <0.233) — consistent with an inhalational agent being the cause of the necrosis.

There are few similar cases of desflurane-induced acute liver failure in the literature to date^{2,3} and none, to our knowledge, in Australia. Fulminant hepatic necrosis induced by halothane, the original offending agent, occurs in about one in 35 000 adults. This is thought to be immune-mediated and appears to be directly correlated with the metabolism of the anaesthetic, catalysed by cytochrome P450 2E1, to trifluoroacetylated hepatic proteins. The altered protein is seen as “non-self”, generating an immune response that, on re-exposure, leads to inflammation and cellular death.⁴

Desflurane is metabolised to inorganic fluoride and trifluoroacetyl chloride. However, due to a lower blood:gas partition coefficient and its resistance to degradation (as a result of replacement of chlorine by fluorine at the α -carbon position), desflu-

Clinical outcomes for eight patients with cerebral palsy after treatment with bethanechol

Sex (age in years)	Diagnosis	Final daily dose of bethanechol*	Clinical effects
M (41)	Ataxic and spastic quadriplegia	60 mg	Reduced muscle spasm, improved joint movement and speech, improved sense of wellbeing
F (53)	Ataxic and spastic quadriplegia	60 mg	Improved arm movement and speech, looser muscle tone, more relaxed
F (47)	Spastic quadriplegia, kyphoscoliosis	60 mg	Able to abduct legs from previously clamped closed position, loss of leg spasm pain, improved speech, muscle spasm induced by touch eliminated
M (68)	Spastic quadriplegia, dysphagia	60 mg	Less stiffness, speech clearer, easier for carers to move, improved sense of wellbeing
M (58)	Rigid spastic quadriplegia	60 mg	Less limb muscle spasm, improved arm and trunk movement, markedly improved speech
F (44)	Spastic quadriplegia, epilepsy	60 mg	Improved arm and leg movement, easier to roll
M (49)	Spastic quadriplegia, athetosis	30 mg	Chronic spasmodic jerks ceased completely, speech better, able to play carpet bowls better, back extension improved
M (68)	Spastic quadriplegia, kyphoscoliosis	60 mg	Less muscle pain, less back spasm, easier for carers to lift, felt happier and more relaxed

* Given orally in three divided doses.

rane is metabolised by hepatic enzymes to a lesser extent than halothane, enflurane and isoflurane.⁴ Thus, the degree of hepatic metabolism appears to be related to the potential for hepatic injury, as seen clinically.

Evidence for immune-mediated, allergic sensitisation continues to emerge. Identification of IgG4 antibodies, the rarest and most IgE-like immunoglobulins, strongly suggests an allergic component in the pathophysiology of this disease.⁵

Although hepatotoxicity is a rare complication of the newer inhaled volatile agents, it may have devastating consequences. Anaesthetic agents should be considered in the differential diagnosis of hepatotoxicity, especially in the context of extreme elevation of serum transaminases, suggesting the presence of massive hepatic necrosis. In this case, postoperative ALT elevation was attributed to antibiotic — rather than desflurane — toxicity, with disastrous results following re-exposure, a scenario that might have been prevented if recognised earlier.

A full incident report was made at the tertiary hospital involved, and the death was reported to (and examined by) the coroner. The main recommendation made from the case was that inhalational agents should be avoided in the setting of hepatitis.

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Laparoscopic repair of gastric volvulus secondary to transverse colon diaphragmatic hernia

Kevin Ooi and Christophe Berney

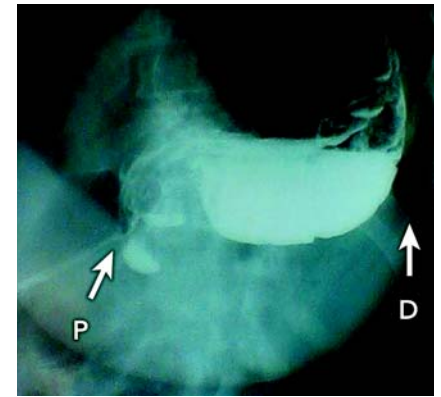
TO THE EDITOR: Gastric volvulus is rare but has been reported increasingly due to greater frequency of upper gastrointestinal tract investigations. Depending on the rotation axis, gastric volvulus can be classified as organoaxial, mesenteroaxial or mixed type. We report a case of laparoscopic mesh repair of a mesenteroaxial gastric volvulus secondary to a transverse colon diaphragmatic hernia.

A 50-year-old woman presented with a 10-year history of intermittent epigastric pain and vomiting. Symptoms persisted despite multiple investigations over the years and treatment with proton-pump inhibitors and prokinetic agents. She described weight loss and intolerance to solid food, but her medical history was unremarkable. Gastroscopy revealed an unusual stomach configuration and difficulty was experienced in intubating the pylorus. A barium x-ray showed no gastric herniation, but the stomach had an unusual appearance (Box, A). Manometry studies showed normal gastric muscle activity.

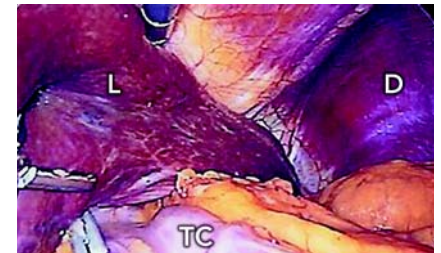
The patient underwent a laparoscopy, which revealed a mesenteroaxial intra-abdominal gastric volvulus secondary to the presence of a section of transverse colon caught in a diaphragmatic hernia adjacent to the oesophagus (Box, B). The colon was reduced and the hernia sac excised (Box, C). The defect in the diaphragm was subsequently closed, and a dual-layered prosthetic mesh was laid over the repaired area. The stomach was repositioned by anterior gastropexy. The patient's recovery was uneventful and she was discharged on a fluid diet 3 days after surgery. At 4-month review, she was well and a follow-up abdominal computed tomography scan showed no abnormalities.

Reports of isolated colonic hiatal hernia are rare.^{1,2} This case was interesting as it was associated with an intra-abdominal gastric volvulus that presented with chronic symptoms, despite most cases of mesenteroaxial volvulus presenting acutely. Barium studies from 19 patients with colonic herniation through the oesophageal hiatus showed that these hernias were invariably associated with herniation of the stomach, which was partially volvulated in many cases.³ These patients were mostly older women, and did not present in an emergency setting. With growing use of laparoscopic surgery, patients benefit from a minimally invasive approach,

Diagnosis and repair of a gastric volvulus



A: Barium x-ray of stomach, showing two air-fluid levels that give the impression of an "upside-down" stomach of mesenteroaxial rotation; pylorus (P) and diaphragm (D) are shown.



B: Herniated transverse colon (TC) tracking under the liver (L) and into a hernia of the diaphragm.



C: Diaphragmatic sac adjacent to the oesophagus, revealed by reducing the colon; oesophagus (O) and stomach (S) are shown. ♦

decreased pulmonary and wound complications, and faster postoperative recovery. Several authors have reported favourable outcomes after performing laparoscopic diaphragmatic hernia repairs and gastropexy.^{2,4,5} Our case demonstrates the feasibility of laparoscopic repair of a gastric volvulus secondary to a transverse colon diaphragmatic hernia.

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Bicycle handlebar injuries in Western Australia: from imprints to abdominal wall hernias

Parshotam K Gera, Andrew P Barker, Ian Gollow, Jillian Orford, Sue Wicks and Liz Whan

TO THE EDITOR: In bicycle accidents, direct impact with the bicycle handlebar can cause serious abdominal injuries. These injuries occur not only in high-speed collisions, where the rider is thrown from the bicycle, but also in low-speed crashes, where the bicycle handlebar strikes the rider in the abdomen or pelvic region.¹

We retrospectively reviewed all children who presented to Princess Margaret Hospital for Children with abdominal bicycle handlebar injuries from January 2002 to July 2007. The patients were identified from the emergency department trauma database; 60 boys and 10 girls were identified, aged 5–15 years.

Significant injuries (defined as injuries to the liver, spleen, kidney, pancreas, small bowel, stomach or urinary bladder) were noted in 25 of the 70 patients (36%), and 15 of the 70 patients (21%) required surgery. Twenty-one patients (30%) had handlebar imprints on the abdomen (Box, A), and 17 of them (81%) had significant injuries. Traumatic abdominal wall hernia (TAWH) was present in three patients (Box, B).

The odds of a significant injury were 21.8 times higher (95% CI, 5.8–82.1) for patients with handlebar imprints than for those with no handlebar imprints. Computed tomography (CT) was the main method of diagnosis of significant injury, and there was a statistically significant association between handlebar imprints and a positive CT scan result, defined as evidence of a solid or hollow viscus injury (2-sided Fisher's exact test, $P = 0.01$). Of those patients who underwent CT scanning, 89% of those with handlebar

Injuries caused by bicycle handlebars in children



A: Handlebar imprint on abdomen.



B: Traumatic abdominal wall hernia, caused by handlebar injury, with omentum protruding through the defect. ♦

imprints (16/18) had a positive CT scan, compared with 36% of those with no handlebar imprints (4/11). The odds of a positive CT scan were 14 times higher (95% CI, 2.1–95.1) for patients with handlebar imprints than for those with no handlebar imprints.

Similar rates of significant injury resulting from impact with handlebars have been reported previously.² TAWH was first described in 1906,³ and 31 cases of handlebar-related TAWH in children have been reported to date, excluding our cases.^{4,5} TAWH is produced by sudden application of blunt force to the abdomen that does not penetrate the skin, but is strong enough to disrupt muscle and fascia. Surgical repair is usually required to prevent complications.⁵

Children with handlebar imprints should be observed closely, and assessed by CT scan and treated surgically as indicated. They should be encouraged to use protective gear, such as handlebar padding, helmets and protective clothing, when riding bicycles.

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In the long run, skills are as good as pills for attention deficit hyperactivity disorder

Alison Poulton and Ralph KH Nanan

TO THE EDITOR: We read with interest Rey's interpretation of the Multimodal Treatment Study of Children with Attention Deficit Hyperactivity Disorder (MTA).¹ The MTA was a large randomised study comparing the impact of stimulant medication, behavioural treatment, a combination of the two, and standard community care on attention deficit hyperactivity disorder (ADHD).² The treatment phase lasted 14 months, during which the children taking medication showed more improvement than the other groups. Participants were then allowed to change their treatment and, at 36-month follow-up, the outcomes in all groups were similar. Rey concluded that, if stimulant medication is not associated with sustained improvement, its place in the treatment of ADHD is limited.

This conclusion overlooks two important points. First, the greater initial improvement in symptoms of ADHD associated with stimulant medication might be important both clinically and socially. The second point is the expected impact of a relatively brief intervention: is it really plausible that an independent effect of 14 months of controlled treatment will be detectable after a further 22 months of self-selected management? The observation that the 14-month treatment phase becomes progressively less relevant as time passes is perhaps not altogether unexpected.

In the unmedicated group, the 23% non-compliance rate during the 14-month treatment phase indicated greater dissatisfaction with treatment than the 10% non-compliance in the medication groups ($P < 0.005$, χ^2 test). This could imply a parental preference for more immediate relief of symptoms, even if it involves their child taking medication. Parental preference can be accommodated if

the family and treating physician discuss and agree on a treatment plan, adjusted to optimise functioning. Far from indicating a diminished role for medication, the evidence from the MTA study suggests that the clinical approach would involve most individuals with ADHD being treated with stimulant medication at some stage.

Stimulant medication treats symptoms; it is not curative. It is likely that the role of stimulant medication in the treatment of ADHD decreases as children mature. However, temporary relief of symptoms can be highly valuable for affected children and their families.

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1 Rey JM. In the long run, skills are as good as pills for attention deficit hyperactivity disorder [editorial]. *Med J Aust* 2008; 188: 133-132.

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Joseph M Rey

IN REPLY: Poulton and Nanan question my statement that the role of psychostimulant medication in attention deficit hyperactivity disorder (ADHD) becomes less prominent when the 3-year results of the Multimodal Treatment Study of Children with Attention Deficit Hyperactivity Disorder (MTA) are taken into account.¹ Studies such as the MTA that report dramatic short- to medium-term improvement have, in my experience, increased practitioners' expectations and reliance on these medications. Their clinical use has gradually widened to preschool-aged children and to the, so far, poorly validated inattentive and impulsive-hyperactive subtypes of ADHD. I observe this increasing the pressure on parents — not necessarily from clinicians — to use stimulants through an emphasis on the consequences of non-treatment, such as underachievement and conduct problems.

The 3-year follow-up of the MTA brings the early findings into perspective: a carefully titrated medication regimen produces no better results 3 years later than behavioural treatment and standard community care.² In that sense, the role of stimulants versus other interventions has shrunk, and conscientious practitioners will inform parents and children of these findings when examining treatment options. My editorial did not query the many short-term benefits

of stimulants but raised questions about when, and for how long, they should be used.

Poulton and Nanan rightly emphasise that stimulants are a "symptomatic" treatment. Further, stimulants increase the ability to concentrate and be on task whether or not individuals meet criteria for ADHD.³ This is further compounded because ADHD, like intellectual disability in the case of intelligence, represents the extreme of a dimension of behaviour.⁴ The boundary between illness and non-illness depends on where you draw the line, not on qualitative differences. However, there is no good tool to measure ADHD, unlike intelligence, and assessment depends on clinicians' thoroughness and skill, and on informants, who may or may not be reliable.

The situation with ADHD is also similar to that for nocturnal enuresis, another disorder that lessens with increasing age, although it may persist. While behavioural treatment (the bell and pad alarm) is effective for enuresis, families and clinicians prefer using medication, even though the latter is "symptomatic" treatment and potentially harmful.⁵ This may be an alternative explanation for the higher non-compliance rate in the non-medicated group: behavioural treatments place more demands on parents, children and schools than a pill.

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