Syncope and profound bradycardia associated with intrauterine contraceptive procedures

Aisling Baird,1 Jane Dickson,2 Mary Jensen,3 Martin Talbot4

Abstract
There has been recent interest in this Journal concerning the occurrence of profound bradycardia with impaired consciousness during insertion of intrauterine contraceptive devices or systems. Questions have been raised regarding the requirement for medication for reversal of the condition, the role of the nurse practitioner in the light of this, and the effects upon sexual and reproductive health care service delivery. We present three cases where this condition affected patients under our care and suggest that although very infrequent, it is important. Medication for treatment and staff trained to administer it should always be available.

Introduction
Bradyarrhythmia and syncope during insertion of an intrauterine contraceptive device (IUD) or system (IUS) are recognised complications and are thought to be due to stimulation of the vagus nerve. Although this is not a common occurrence, women undergoing the procedure may rapidly become profoundly unwell. The guidance on intrauterine contraception of the UK Faculty of Sexual and Reproductive Healthcare (FSRH) states that the availability of appropriate emergency medication, including atropine, during IUD/IUS insertion is essential, and this is a service standard for resuscitation in sexual health services.1 2

In a retrospective chart review of 545 patients seen over a 3-year period, Farmer and Webb reported that bradycardia [pulse <60 beats per minute (bpm)] occurred in 1.8% of IUD insertions.1 They mentioned earlier ECG studies that had estimated the occurrence of bradycardia as 32%, although this was in an era of more inflexible, rigid devices, specifically the Dalkon Shield, and referred to other studies reporting syncope in 2.1% of insertions. In recent correspondence in this Journal, Gormley and Eady drew attention to the rarity of the condition and called for evidence that a doctor must be present for all insertions, yet offered only anecdotal evidence as to the lack of need for atropine.4 Hollingworth used personal experience and that of her colleagues to query the FSRH guidelines on resuscitation in sexual health services and specifically the availability of atropine.5 Mansour conducted a poll of over 70 inserters, none of whom had ever needed to use atropine – the implication being that simple first aid measures should be sufficient to reverse the condition.6 Mehigan believed the FSRH guidance to be “unrealistic”.7 Gormley and Eady championed the preparedness of the woman and the universal use of local anaesthesia; however, there is little evidence to support the assumption that pain is part of the pathogenesis of this condition.

Dickson et al. responded to these communications in 2011, setting out the origin of the Faculty standards.8 Specifically, and pertinent to our communication, they discussed the Resuscitation Council guideline that intravenous atropine should be administered if adverse signs such as shock or syncope are present. We now report three cases of the condition in patients under our care, in whom conservative measures were ineffective but who responded successfully to the administration of atropine.

Case histories
Case 1
This 35-year-old woman, para 3, presented for IUD insertion. She was well and had no medical or gynaecological problems. On the morning of the procedure she had not had breakfast. Preliminary examination was normal. An IUD was inserted without difficulty, with no analgesia. Immediately following insertion, she became pale, sweaty, vacant and unresponsive [Glasgow Coma Scale (GCS) 10]. Her pulse was 60 bpm and thready. She was laid flat and her legs elevated. This produced no response. Atropine sulphate 600 µg was administered intravenously,

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1Consultant in Sexual and Reproductive Health, Liverpool Community Health, Central Abacus, Liverpool, UK
2Community Specialist, Contraception and Sexual Health, Greenwich Community Health Services, Oxleas NHS Foundation Trust, London, UK
3Associate Specialist in Sexual and Reproductive Health, Centre for Contraception and Sexual Health, Victoria Health Centre, Glasshouse Street, Nottingham, UK
4Consultant Physician in Genitourinary Medicine/HIV, Department of Genitourinary Medicine, Sheffield Teaching Hospitals NHS Foundation Trust, Royal Hallamshire Hospital, Sheffield, UK

Correspondence to
Dr Aisling Baird, Liverpool Community Health, Central Abacus, Citrus House, 40–46 Dale Street, Liverpool L2 5SF, UK; aisling.baird@liverpoolch.nhs.uk

Received 28 August 2011
Accepted 22 December 2011
following which she became responsive (GCS 15) and her pulse rose to 60 bpm.

Case 2
This 46-year-old woman, para 3, was referred to a specialist clinic for replacement of her IUS. She reported symptoms suggestive of the menopause with hot flushes and difficulty sleeping. Her IUS had been in situ for 6 years following a “difficult fitting”. When questioned about the difficulty she simply said that she had not been very well afterwards. Preliminary examination was normal. Her pre-procedure pulse was 60 bpm and blood pressure (BP) was 104/70 mmHg. The internal os was stenosed but the fundus was sounded successfully and the cavity measured 7 cm. The IUS ‘snagged’ at the internal os on traction, but was then removed easily.

At this point the patient had a vasovagal episode and the replacement procedure was abandoned. She initially recovered spontaneously and was able to communicate. Her pulse fluctuated between 42 and 48 bpm. Her BP was normal. Simple first aid measures were continued. At one point her pulse returned to 60 bpm. Her head was raised for her to have a sip of water, at which point she collapsed again, her pulse falling to 36 bpm. Oxygen therapy was commenced and 200 µg atropine sulphate was administered intravenously. She was now completely unresponsive, GCS 3, her pulse remained 36 bpm and the heart sounds were very faint. There had been no response to the initial small dose of atropine. After 10 minutes another 200 µg atropine was administered, following which her condition improved, GCS 14 and pulse 60 bpm. She was taken to hospital where she only required observation for a short time.

Case 3
This 40-year-old nulliparous woman attended for IUS insertion. Preliminary assessment was unremarkable. Judd-Allis forceps were applied to the anterior cervical insertion. Preliminary assessment was unremarkable. Her pre-procedure pulse was 60 bpm and blood pressure (BP) was 104/70 mmHg. The internal os was stenosed but the fundus was sounded successfully and the cavity measured 7 cm. The IUS ‘snagged’ at the internal os on traction, but was then removed easily.

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Discussion
Atropine is an alkaloid from the plant Atropa bella-donna and is a competitive antagonist to acetylcholine. It blocks muscarinic receptors in the autonomic nervous system, thus counteracting the effects of vagal stimulation. In healthy individuals this results in a modest tachycardia, since it is the parasympathetic nervous system that is blocked, rather than the sympathetic

...rehabilitation. It blocks muscarinic receptors in the autonomic nervous system, thus counteracting the effects of vagal stimulation. In healthy individuals this results in a modest tachycardia, since it is the parasympathetic nervous system that is blocked, rather than the sympathetic stimulation immediately and after 1 minute her pulse increased to 72 bpm. She recovered fully within 30 minutes and was allowed home.

Competition interests Dr Jane Dickson is the current Vice-Chair of the FSRH Clinical Standards Committee.

Provenance and peer review Not commissioned; externally peer reviewed.

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J Fam Plann Reprod Health Care published online January 17, 2012

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