Comment

Our results confirm a higher early neonatal mortality in low risk babies born at night. This may be a result of staff's increased physical and mental fatigue during the night, when doctors in charge, at least in Germany, have usually worked through a complete day shift. Overreliance on less experienced staff may be another important reason for the higher risk of early neonatal death during the night. These phenomena are not specific to the NHS or the British population. Better designed shifts, resulting in shorter working hours or decreased workload with greater supervision by experienced staff at night, should be considered to reduce early neonatal mortality during the night.

Although our analyses are consistent with previously reported British results, some differences should be considered.[1 2] We used slightly different definitions of night and day because the hours of day shifts are different in Germany. Applying the time categorisation of the British studies yielded almost identical results. Because of the nature of our database only deaths occurring during labour or in the first seven days of life could be traced. There was some concern that babies born during the day are more likely to be preterm or high risk babies who have had induced births. We therefore restricted our study population rigorously. The assumption that a death was related to asphyxia relied solely on the obstetrician's documentation of morbidity and reasons for death, which could be prone to error. Nevertheless, a higher early neonatal mortality in general and a higher mortality related to asphyxia were seen. Additionally, for each deceased child each author reviewed all the information available from the register's database for other potential confounding factors. No alternative explanations for the reported relationship were found.

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Contributors: GH had the idea for the paper, performed statistical analysis, wrote the paper, and is the study guarantor. BM gave access to data, participated in performing statistical analysis, and commented on the draft. SS helped to write the paper. All authors reviewed the database for each deceased child.

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Correction

Systematic reviews and meta-analyses on treatment of asthma: critical evaluation

Several errors occurred in table 2 of this paper by Jadad et al (26 February, pp 527-40). Under the heading "Was bias in the selection of the studies avoided?" the number for "all" should be 14/30, not 15/30; under "Were the findings of the relevant studies combined appropriately?" the number for "all" should be 22/26, not 24/26; and under: "Were the conclusions made by the author(s) supported by the data provided?" the number for "peer reviewed journals" should be 14/38, not 14/14. The P values are the same.

Drug points

Atrial fibrillation associated with sumatriptan

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A 34 year old man with a history of migraine presented with palpitations after taking sumatriptan by nasal spray for a severe headache. Examination was unremarkable apart from atrial fibrillation, with a ventricular rate of 130 beats/min. He recalled having had a fast irregular pulse after taking sumatriptan previously, and this was confirmed from his case notes. He reverted to sinus rhythm spontaneously within 12 hours of admission. Thyroid function tests and a transthoracic echocardiogram gave normal results. Exercise stress testing showed no abnormalities.

Sumatriptan is a 5-hydroxytryptanine agonist active at 5-hyroxytryptanine 1B and 1D receptors. Chest tightness and pain are reported in up to 15% of patients and are presumed to be due to vasoconstriction of the coronary arteries.[1] At therapeutic plasma concentrations, sumatriptan does not reduce myocardial perfusion in healthy migraineurs.[2] Myocardial infarction has, however, been reported as a consequence of sumatriptan treatment for migraine and its use is contraindicated in patients with ischaemic heart disease.[1]

Sumatriptan is the drug most frequently reported to cause chest pain.[3] Atrial fibrillation associated with sumatriptan is uncommon. The Committee on Safety of Medicines has received six reports of atrial fibrillation associated with sumatriptan, which accords with data from the manufacturer. Naratriptan has been associated with one episode of atrial fibrillation. The Committee on Safety of Medicines has received no reports of arrhythmias with rizatriptan, but it has been associated with tachycardia and chest pain (data from manufacturers).

In this patient's case there was evidence of rechallenge, implicating sumatriptan as the cause of the atrial fibrillation, irregular tachycardia having been previously documented after sumatriptan use. The mechanism causing atrial fibrillation is uncertain. In this patient underlying coronary insufficiency seems unlikely. Sumatriptan has been shown to be without inherent tachycardic effect in in vivo preparations of denervated cat heart.[4] Therefore, we suggest that myocardial ischaemia secondary to coronary vasospasm could be a trigger for atrial fibrillation.

Competing interests: None declared.

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