Impact of congenital colour vision deficiency on education and unintentional injuries: findings from the 1958 British birth cohort

P Cumberland, J S Rahi and C S Peckham

*BMJ* 2004;329;1074-1075; originally published online 1 Oct 2004; doi:10.1136/bmj.38176.685208.F7

Updated information and services can be found at:
http://bmj.com/cgi/content/full/329/7474/1074

These include:

References
This article cites 4 articles, 1 of which can be accessed free at:
http://bmj.com/cgi/content/full/329/7474/1074#BIBL

4 online articles that cite this article can be accessed at:
http://bmj.com/cgi/content/full/329/7474/1074#otherarticles

Rapid responses
13 rapid responses have been posted to this article, which you can access for free at:
http://bmj.com/cgi/content/full/329/7474/1074#responses

You can respond to this article at:
http://bmj.com/cgi/eletter-submit/329/7474/1074

Email alerting service
Receive free email alerts when new articles cite this article - sign up in the box at the top left of the article

Topic collections
Articles on similar topics can be found in the following collections

- Ophthalmology (1074 articles)
- Child health (5670 articles)
- Screening (epidemiology) (2619 articles)
- Screening (public health) (2621 articles)
- Sociology (2921 articles)

Notes

To order reprints follow the “Request Permissions” link in the navigation box

To subscribe to *BMJ* go to:
http://resources.bmj.com/bmj/subscribers
Impact of congenital colour vision deficiency on education and unintentional injuries: findings from the 1958 British birth cohort

P Cumberland, J S Rahi, C S Peckham

Congenital colour vision defects (CVD) are common, inherited (most commonly X linked), non-progressive, and untreatable disorders.1 2 Screening children for these disorders is established practice in the United Kingdom, primarily so that those affected can be advised about occupational preclusions.3 Population based work on the broader impact of colour vision defects is, however, limited.

Participants, methods, and results

We investigated the association between CVD and education and unintentional injury in the 1958 British birth cohort.3 4 Despite attrition, people remaining were representative of the original sample, including with respect to colour vision status. The latter was assessed in 12 534 children aged 11 years using the Ishihara test,1 with CVD being the inability to identify all 24 plates. Corrected distance acuity was measured with Snellen charts. We analysed educational, perceptual, and motor skills tests done at 7, 11, and 16 years1 4 together with highest educational qualification by 33 (none, below O level or equivalent, O level or equivalent, A level or equivalent, or higher). We converted education test scores to z scores5 and assessed the effect of CVD with multivariate linear regression. We analysed unintentional injuries requiring hospital care by CVD status and sex.

Overall, 431 of 6422 boys (6.7%; 95% confidence interval 6.1% to 7.3%) and 68 of 6112 girls (1.1%; 0.8% to 1.4%) had CVD. The distribution of corrected visual acuity did not vary by colour vision status ($\chi^2$ trend, $P = 0.12$). Birthweight, social class at birth, family size, and parental education, all associated with education, were accounted for in the present analysis, although not associated with CVD.1 4

At 7 years, CVD and mathematics and reading scores were not significantly associated, after adjustment for age at testing and factors described above (table). At 16, after additional adjustment for prior test scores, children with CVD scored higher than those without; but the small differences, although statistically significant, were functionally unimportant (0.08 standard deviations; 0.002 to 0.16; $P = 0.05$ for mathematics and 0.07; 0.002 to 0.14; $P = 0.04$ for reading). There were no significant differences, by colour vision status, for boys or girls, in scores for “copy a design” or “draw a man” at 7 years. Highest educational qualification and colour vision status were not associated for either men or women ($\chi^2$ trend, $P = 0.07$ and $P = 0.61$).

Risk of unintentional injury did not differ significantly (table). Overall, 8.9% (8.2% to 9.6%) of females and 19.2% (18.2% to 20.2%) of males had road injuries as a driver by 33 years; people with CVD reported fewer unintentional injuries ($P = 0.08$ and $P = 0.05$). At 33 years, 30% (28.9% to 31.2%) of men reported unintentional injuries in the workplace, without any increased risk in those with colour vision defects ($P = 0.293$).

Comment

Increasing use of colour in education has raised concerns for children with CVD, but robust evidence is lacking.3 Our findings indicate that affected children do as well as their peers educationally, during school and subsequently. Although the use of colour has increased since the early schooling of the subjects of this study, only a minority with severely impaired colour vision would be potentially disadvantaged and any limitation would depend on the specific environment as well as the individual's abilities.

That unintentional injuries were no more common among those with CVD supports current standards for driving in the United Kingdom (in which CVD is not a preclusion) and also indicates that normal colour vision is not a prerequisite for safe working in many occupations or environments.

Most people with colour vision defects develop effective adaptive strategies and behaviours, and they use other clues, such as a colour's saturation, to deal with any potential limitations in their professional and personal lives.1 At a population level, congenital CVD confers no functional disadvantage in relation to educational attainment and unintentional injury. This challenges the rationale for and the value of population screening for these disorders.

JR has a joint appointment within the Division of Epidemiology, Institute of Ophthalmology, London EC1V 9EL. We thank the Centre for Longitudinal Studies (Institute of Education), National Birthday Trust Fund, National Children's Bureau, City University Social Statistics Research Unit, and the Data Archive distributor, SN:3138, Colchester, for archived data. We thank Angie Wade for comments on a previous draft.

This article was posted on bmj.com on 1 October 2004: http://bmj.com/cgi/doi/10.1136/bmj.38176.685208.F7
Lofexidine (BritLofex, Britannia) is an α 2 agonist used for opioid detoxification.\(^1\) We report one case of an increased QT interval after one dose of 0.4 mg lofexidine.\(^2\)

A 44 year old white opiate dependent female enrolled in a protocol to assess the safety of taking daily lofexidine with daily methadone. She had no known drug allergies, took no other drugs, and had never taken lofexidine before. An electrocardiogram before starting methadone showed sinus rhythm (70 beats/min) and QT/QTc 428/449 ms (fig). She was then given lofexidine with her daily dose of methadone. She had no known drug allergies, took no other drugs, and had never taken lofexidine before.

An electrocardiogram before starting methadone showed sinus rhythm (70 beats/min) and QT/QTc 428/449 ms (fig). She was then given lofexidine with her daily dose of methadone, which was followed by brief hypotension (88/55 mm Hg) with mild drowsiness. Four hours after taking lofexidine, an electrocardiogram showed sinus bradycardia (58 beats/min), QT/QTc 612/601 ms, and blood pressure 149/88 mm Hg (fig). The participant was otherwise asymptomatic. Within 24 hours, QT/QTc was 372/432 ms. Laboratory work found no abnormalities. Echocardiography showed mild mitral regurgitation.

Three independent cardiologists interpreted that although there was some QT prolongation (QT range 510–560 ms; QTc range 501–560), the computer overestimated the interval because of U waves.

Increased QT interval with lofexidine has been seen in animals, but only at very high doses;\(^3\) no clinically important changes have been seen in humans. Although high doses of methadone have been associated with changes in QT and arrhythmia,\(^4\) the woman in this case had a normal QT interval while on methadone. The temporal relationship and lack of other causes indicate that the combination of lofexidine and methadone perhaps was the precipitant of this self limited change in QT.

In response, Britannia reported this event to the United Kingdom Committee on Safety of Medicines and added a warning to the summary of product characteristics (see www.lofexidine.co.uk). Before initiating lofexidine, clinicians may want to screen patients who might be at risk for repolarisation abnormalities.

Funding: National Institutes of Health, National Institute on Drug Abuse, Intramural Research Program, provided all funding. Britannia Pharmaceuticals Limited provided lofexidine.

Competing interests: None declared.

Contributors: JSR designed the study; PC did the analysis; and PC, JSR, and CSP interpreted the findings and wrote the paper. JSR is guarantor.

Ethical approval: Institute of Child Health’s Research Ethics Committee.


(accepted 15 June 2004)
doi 10.1136/bmj.38176.685208.F7

---

**What is already known on this topic**
Congenital colour vision defects are common, non-progressive, and untreated disorders, for which screening is done so that affected children can be informed about occupations which require normal colour vision.

**What this study adds**
At a population level, colour vision defects confer no functional disadvantages in relation to educational attainment or unintentional injury—challenging the rationale for and value of screening.

---

**What is already known on this topic**
Little population based work exists on the broader functional impact of these disorders.

**What this study adds**
From a population perspective, there are no functional disadvantages in relation to educational attainment or unintentional injury—challenging the rationale for and value of screening.

---

**Drug Points**
**QT interval increased after single dose of lofexidine**

John Schmittner, Jennifer R Schroeder, David H Epstein, Kenzie L Preston

Lofexidine (BritLofex, Britannia) is an α 2 agonist used for opioid detoxification.\(^1\) We report one case of an increased QT interval after one dose of 0.4 mg lofexidine.\(^2\)

A 44 year old white opiate dependent female enrolled in a protocol to assess the safety of taking daily lofexidine with daily methadone. She had no known drug allergies, took no other drugs, and had never taken lofexidine before.

An electrocardiogram before starting methadone showed sinus rhythm (70 beats/min) and QT/QTc 428/449 ms (fig). She was then given lofexidine with her daily dose of methadone. She had no known drug allergies, took no other drugs, and had never taken lofexidine before.

An electrocardiogram before starting methadone showed sinus rhythm (70 beats/min) and QT/QTc 428/449 ms (fig). She was then given lofexidine with her daily dose of methadone, which was followed by brief hypotension (88/55 mm Hg) with mild drowsiness. Four hours after taking lofexidine, an electrocardiogram showed sinus bradycardia (58 beats/min), QT/QTc 612/601 ms, and blood pressure 149/88 mm Hg (fig). The participant was otherwise asymptomatic. Within 24 hours, QT/QTc was 372/432 ms. Laboratory work found no abnormalities. Echocardiography showed mild mitral regurgitation.

Three independent cardiologists interpreted that although there was some QT prolongation (QT range 510–560 ms; QTc range 501–560), the computer overestimated the interval because of U waves.

Increased QT interval with lofexidine has been seen in animals, but only at very high doses;\(^3\) no clinically important changes have been seen in humans. Although high doses of methadone have been associated with changes in QT and arrhythmia,\(^4\) the woman in this case had a normal QT interval while on methadone. The temporal relationship and lack of other causes indicate that the combination of lofexidine and methadone perhaps was the precipitant of this self limited change in QT.

In response, Britannia reported this event to the United Kingdom Committee on Safety of Medicines and added a warning to the summary of product characteristics (see www.lofexidine.co.uk). Before initiating lofexidine, clinicians may want to screen patients who might be at risk for repolarisation abnormalities.

Funding: National Institutes of Health, National Institute on Drug Abuse, Intramural Research Program, provided all funding. Britannia Pharmaceuticals Limited provided lofexidine.

Competing interests: None declared.

Contributors: JSR designed the study; PC did the analysis; and PC, JSR, and CSP interpreted the findings and wrote the paper. JSR is guarantor.

Ethical approval: Institute of Child Health’s Research Ethics Committee.


(accepted 15 June 2004)
doi 10.1136/bmj.38176.685208.F7