



Plasma Lp(a) lipoprotein concentrations in four study groups. Boxes represent median and middle quarters of Lp(a) lipoprotein concentration and whiskers represent lowest and highest quarters

Other studies of alcohol consumption and Lp(a) lipoprotein cholesterol concentrations have dealt with differences between men and women, analysed alcohol intake qualitatively,⁵ and compared heterogeneous groups—that is, non-drinkers together with those who drink regularly on three or less days a week. To our knowledge, ours is the first study to show a relation between moderate alcohol consumption and Lp(a) lipoprotein concentrations. We conclude that low Lp(a) lipoprotein concentrations may be one factor explain-

ing low mortality and retarded progression of coronary artery disease in social drinkers.

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Contributors: MP participated in data analysis, writing the paper, and a discussion of the core ideas. KK discussed the study hypothesis and core ideas, and participated in data analysis and writing the paper. AOR participated in the study design, patient investigations, and data collection. MJS discussed the study hypothesis and core ideas, and participated in writing the paper. ML participated in the study design, data collection, and writing the paper. AR participated in the study design, statistical analysis, and writing the paper. YAK was the principal investigator; he initiated and coordinated the formulation of the primary study hypothesis, designed the protocol, discussed core ideas, and participated in data interpretation and writing the paper. YAK will act as guarantor of the study.

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Drug points

Postural hypotension induced by paroxetine

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Antidepressant prescribing in elderly people is influenced by side effects and the patient's physical state.¹ The high rate of falls and fractures in this age group may relate to antidepressant induced postural hypotension.² Tricyclic antidepressants and monoamine oxidase inhibitors may produce postural hypotension,³ so treatment with selective serotonin reuptake inhibitors is often preferred in older patients. We report a case of postural hypotension induced by paroxetine.

A 75 year old woman who had had coronary artery bypass grafting six months previously was prescribed paroxetine for depression. The starting dose of 10 mg was increased to 20 mg after 14 days, but her other treatment (quinine bisulphate, fluvastatin, and temazepam) was unchanged. She continued to take paroxetine for 6 days, when she became dizzy and developed marked postural hypotension (blood pressure 170/90 mm Hg while lying and 90/60 mm Hg while standing). Physical examination and investigations, including a short tetracosactin test, gave normal results. Paroxetine treatment was discontinued and her postural hypotension resolved. She agreed to a rechallenge test with paroxetine at a reduced dose of 10 mg. Again, she developed dizziness and postural hypotension (blood pressure 140/90 mm Hg while lying and 110/

60 mm Hg while standing), which resolved on withdrawal of the drug.

To our knowledge, the only published report of postural hypotension associated with paroxetine relates to its increasing trimipramine concentrations when prescribed with trimipramine.⁴ At the time of writing, 43 cases of postural hypotension associated with paroxetine had been reported to the Committee on Safety of Medicines (personal communication). Other selective serotonin reuptake inhibitors have been reported to exacerbate syncope.⁵ Dizziness is cited on the datasheet for paroxetine, though not in relation to postural hypotension.

We suggest that postural hypotension should be considered if dizziness develops. The size of the postural fall in blood pressure seems to be dose related, and the dose should be reduced or drug treatment discontinued.

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