LETTERS TO THE EDITOR

Scope

Heart welcomes letters commenting on papers published in the journal in the previous six months. Topics not related to papers published earlier in the journal may be introduced as a letter: letters reporting original data may be sent for peer review.

Presentation

Letters should be:

- not more than 600 words and six references in length
- typed in double spacing (fax copies and paper copy only)
- signed by all authors

They may contain short tables or a small figure. **Please send a copy of your letter on disk.** Full instructions to authors appear in the January 1999 issue of *Heart* (page 104).

Does aspirin treatment influence vascular resistance and fluid filtration in patients with congestive heart failure?

Sir—During recent years the beneficial effect of continued use of aspirin after acute myocardial infarction in patients with congestive heart failure has been questioned.[1] This is based on retrospective findings in some large clinical trials showing a reduced beneficial effect on mortality of angiotensin converting enzyme (ACE) inhibitors in patients treated with aspirin.[2 3] Moreover, improvement in central haemodynamic indices and pulmonary diffusion capacity in patients with congestive heart failure during short term ACE inhibitor treatment was impaired when aspirin was added,[4–6] though there was no interaction between antiplatelet treatment with ticlopidine and ACE inhibitor treatment.[6]

We retrospectively analysed data from a study on calf capillary fluid filtration and microvascular blood flow in 22 patients with congestive heart failure (20 men and two women; mean age 45 years (range 27 to 64); mean (SD) ejection fraction 22 (8)%; mean arterial blood pressure 88 (15) mm Hg) in New York Heart Association (NYHA) class II (n = 14), class III (n = 6), and class IV (n = 2), to see whether resting calf skeletal muscle blood flow and capillary filtration of water differed in six patients on long term treatment with 75–150 mg aspirin and 16 patients not on treatment with aspirin.

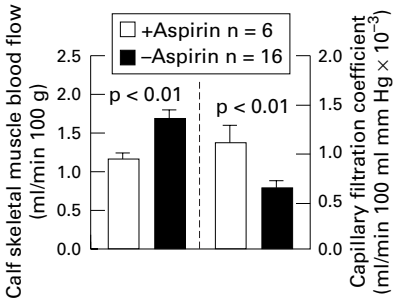

Figure 1 Skeletal muscle blood flow was reduced (left) and capillary filtration coefficient for water was increased (right) in patients with congestive heart failure on aspirin treatment compared with those not treated with aspirin. Data are means, error bars = SEM (Mann-Whitney U test).

To test the effect on heart failure alone and to avoid influence of atherosclerosis, we only included patients with idiopathic dilated cardiomyopathy. The groups compared did not differ with respect to NYHA class, ejection fraction, mean arterial pressure, sex, or age and all were treated with an ACE inhibitor.

Calf skeletal muscle blood flow was measured by the local ^{133}xenon washout method and capillary filtration by venous occlusion strain gauge plethysmography. Figure 1 shows that patients on aspirin treatment had lower resting skeletal muscle blood flow (and thus increased vascular resistance: 80 (20) mm Hg.min/ml in patients on aspirin treatment *v* 56 (17) mm Hg.min/ml not on aspirin treatment, $p < 0.01$) and a higher capillary filtration coefficient for water than patients not on aspirin treatment. There was no significant difference in calf skeletal muscle blood flow or capillary fluid filtration coefficient in relation to other medication.

Aspirin impedes platelet aggregation and blocks prostaglandin formation, including the formation of the vasoconstrictor and platelet aggregation inducer thromboxane A_2. Our data suggest that patients with congestive heart failure on long term treatment with aspirin and an ACE inhibitor have increased peripheral precapillary to postcapillary resistance, with increased afterload and increased susceptibility to oedema formation. These findings add to the growing evidence that aspirin may not be beneficial in patients with congestive heart failure on treatment with an ACE inhibitor. However, our study was small, retrospective, and non-randomised, and the subject is controversial as there are also data showing an absence of interaction between aspirin and ACE inhibitor treatment in congestive heart failure.[7] The data therefore need to be confirmed in a larger study specifically designed for the purpose before a deleterious effect of aspirin can be considered proven.

S GALATIUS
H WROBLEWSKI
J KASTRUP
The Heart Centre, The Rigshospital, Copenhagen, DK 2100 Copenhagen Ø, Denmark

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Brugada syndrome associated with an autonomic disorder

Sir,—I read with interest the case report from Japan of a patient with Brugada syndrome associated with an autonomic disorder.[1] The patient lost consciousness on two occasions, and ventricular fibrillation was documented in the cardiac catheterisation laboratory. But no mention was made of treatment, which should include implantation of an automatic cardioverter defibrillator.

The Brugada syndrome occurs not only in caucasian[2 3] but also in oriental people, including Japanese[1 4] and Chinese people.[5] It has recently been speculated that the sudden unexplained death syndrome (SUDS) in young Thai men and other Southeast Asians may have features similar to Brugada syndrome, including an intermittent right bundle branch block on ECG with pronounced ST elevation.[6] However, patients with SUDS die at night while asleep[6 7]; in contrast patients with Brugada syndrome do not have symptoms during sleep.[5]

The diagnosis of Brugada syndrome calls for implantation of an implantable cardioverter defibrllator[8] because of the high risk of sudden death, especially in young patients.[4] Even in initially asymptomatic patients, the incidence of ventricular fibrillation occurring later is high.[5] Therefore, all patients with Brugada syndrome should have an implantable cardioverter defibrillator as a definitive treatment.[2 3 5 8]

TSUNG O CHENG
Professor of Medicine, Division of Cardiology, George Washington University Medical Center, 2150 Pennsylvania Avenue NW, Washington, DC 20037, USA

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CORRECTION

Coexistence of mitochondrial DNA and β myosin heavy chain mutations in hypertrophic cardiomyopathy with late congestive heart failure. E Arbustini, R Fasani, P Morbini, et al. Heart *1998;***80***:548–58*

The affiliations for two authors of this paper were incorrect or omitted.

Dr P Banfi is affiliated with the Department of Neurology, Ospedale Maggiore, Lodi, Italy.

Dr G Comi is affiliated with Centro Dino Ferrari, Istituto di Clinica Neurologica, Università di Milano, IRCCS Ospedale Milano, Italy.

These errors are regetted.