LETTERS TO
THE EDITOR

Scope
Heart welcomes letters commenting on papers published in the journal in the previous six months. Topics 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 July 1999 issue of Heart (page 116).

Management of scorpion sting

EDITOR—We read with great interest the haemodynamic pattern in patients with scorpion envenomation.1 We have studied this acute time sensitive medical emergency since 1976 and have tried various regimens including antiscorpion venom.2 Since the advent of prazosin (1983–84)—an α-adrenergic blocker which acts as an antidote to venom—the mortality of scorpion sting victims is less than 1%.3

In the pre-prazosin era (1961–83) a fatality rate of 25–30% was reported from western India,4 acute pulmonary oedema causing death. We were therefore surprised by the 25% mortality reported by Karnad,5 and that two victims died on the third day of hospitalisation; we have similar findings in 1978–82—that is, before prazosin treatment.6

We have reported that the severity of scorpion sting depends on the victim’s age, the season, and the time between sting and administration of prazosin. The symptoms following the sting are hypotension, tachycardia, pulmonary oedema, and shock (autoimmune storm).7 We believe that the transport of Karnad’s patients to the nearest major hospital, and their delayed admission to their department, seven of eight had acute pulmonary oedema owing to a delay in reaching a tertiary care hospital.8 Scorpion venom inhibits angiotensin converting enzyme (ACE), resulting in accumulation of bradykinin, which is implicated in the development of pulmonary oedema and acute reversible pancreatitis.9 Captopril (an ACE inhibitor) has a similar action resulting in hypotension resistant to dopamine agonists.10 Bradykinin further enhances noradrenaline (norepinephrine) release by a presynaptic mechanism.11

Alpha receptor stimulation plays a major role in the pathogenesis of pulmonary oedema.12 It causes hyperkalaemia and hyperglycaemia (inhibition of insulin secretions). Angiotensin II stimulates α-adrenergic receptors in the myocardium and hypoxia results in coronary spasm as well as accumulation of free radicals and free radicals injurious to myocardium leading to cardiac arrhythmias and sudden death.13

At a general hospital at Mahad we successfully treated 658 cases of severe scorpion sting with oral rehydration, oral prazosin, and oxygen and umifenpyrim: 362 (55%) had hypertension, 178 (27%) had pulmonary oedema, 118 (18%) had tachycardia; no patient had lethal arrhythmias. Seventeen patients who died on the way to Mahad had been referred 24 hours after being stung while 13 cases admitted with multiorgan failure and were moribund and comatose; they died 30–60 minutes after admission despite treatment with dopamine, nitroprusside, oxygen, or an inhalation–inhaled–inhaled-drip. The remaining 56 cases with massive pulmonary oedema recovered after treatment with intravenous sodium nitroprusside.

We travelled throughout western Maharashtra where Mesobuthus tamulus scorpions flourish, and taught all peripheral doctors how to treat scorpion sting victims. There has not been any deaths over the past two years due to scorpion sting reported from this region. Moreover, only two patients had massive pulmonary oedema, which was treated with intravenous sodium nitroprusside. Captopril failed to correct haemodynamic abnormalities in two cases who had cardiac arrhythmias. These two patients would have been saved in the two patients treated with captopril who died in Karnad’s report.7 As a potent inhibitor of phosphodiesterase, prazosin causes accumulation of cGMP, a second messenger of nitric oxide, in vascular endothelium and myocardium, inhibits the formation of inositol triphosphate, and activates venom inhibited calcium dependent potassium channels.

Thus prazosin reverses both inotropic (hypertension), and hypokinetic (pulmonary oedema, hypotension, tachycardia) phases evoked by scorpion envenoming.

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Editor—The study by Karnad3 on the haemodynamic patterns encountered in scorpion envenomation raises important concerns. Karnad claims that the mechanism of pulmonary oedema observed in scorpion envenomation had not previously been established as pulmonary arterial wedge pressure (PAWP) had not been measured. However, the haemodynamic mechanism of pulmonary oedema in scorpion envenomation was suggested in animal studies as early as 19804 and confirmed by numerous series in human subjects. All of these series performed a haemodynamic study and PAWP was therefore not surprised with Karnad’s findings and observe that they are consistent with our previous reports suggesting similar effects of the Indian red scorpion Mesobuthus tamulus and the yellow scorpion of North Africa (Androctonus australis).

We are particularly concerned by Karnad’s interpretation of the haemodynamic records in the envenomed patients and the treatment strategy he suggests. A dynamic, echocardiographic, and angioscintigraphic studies have shown that severe scorpion envenomation impairs left and right ventricles to the same extent.5 Echocardiographic studies showed that LV systolic function might be deeply depressed with a mean LV fractional shortening as low as 12%6. Regarding the right ventricle, in eight patients we recorded a mean RV ejection fraction of 24%.7 In fact, severe scorpion envenomation evokes acute heart failure which tends to recover in a few days. The heart failure might be concealed in some patients by the simultaneous hypovolaemia that occurs in envenomed patients as a consequence of vomiting and sweating. Hence, the patterns II, III, and IV described by Karnad should not be disregarded differently from the multiple facets of the same and only haemodynamic feature that is the profile of acute heart failure (patterns II and IV) that might be mitigated by simultaneous hypovolaemia (pattern III). Moreover, the three reported patients who had simultaneous hypovolaemia exhibited a worsening in their pulmonary oedema with fluid infusion, suggesting an exaggerated increase in PAWP resulting from altered LV function.

Although attractive from a pathophysiological standpoint, Karnad challenges the usefulness of inotropic drugs and claims that they improve only transiently the circulatory failure observed in scorpion envenomation with no effect on mortality. We are unaware of any study specifically analysing inotropic drugs on this issue. Nevertheless, owing to the clearly established haemodynamic pattern of severe scorpion envenomation and on the basis of a pathophysiological approach, we have usually treated envenomed patients exhibiting pulmonary oedema and/or peripheral circulatory failure with dobutamine. The physiological effects were as expected, those usually observed in the treatment of heart failure: an increase in cardiac output as a consequence of an increase in stroke volume with enhanced LV performance, a substantial decrease in PAWP, and an increase of arterial pressure.

Finally, Karnad suggests that RV failure occurs late in the terminal phase of scorpion envenomation and combines with pre-existing LV failure to produce severe cardiogenic shock. This speculation is not supported by Nouira et al who used a modified Swan-Ganz catheter equipped with a fast response thermostance.8 This study showed that scorpion envenomation evokes simultaneous impairment of the LV as well as the RV, the latter being depressed to the same extent as the former. Infusion of dobutamine enhances RV as well as LV performance.

In conclusion, scorpion envenomation kills thousands of patients in developing coun-
tries. First line clinicians need comprehensive and meaningful insights regarding the pathophysiology and valuable treatments of this dreaded accident. The clearer and simpler the message on this issue the better the effect in daily clinical practice.

2 Gueron M, Adolphi RJ, Grupp LL, et al. Hemo
dynamic and myocardial consequence of scorpion venom. Am J Cardiol 1980;45:979-86.
5 Nouriya S, Abroug F, Haguida H, et al. Right ventricular dysfunction following severe scorre

These letters were shown to the author, who replies as follows:

The efforts of Bawaskar and Bawaskar in popularising prazosin treatment in western India are truly commendable, and the effect on mortality following scorpion envenomation is impressive. They cite their personal experience with captopril in scorpion envenomation, which they state has been beneficial. However, their hypothesis that prazosin may be effective in the treatment of scorpion envenomation is not supported by the available evidence.

In India, scorpion envenomation occurs almost exclusively in rural areas, and is particularly common in the coastal regions of western India where Dr Bawaskar’s hospital is located. Patients stung by scorpions are likely to be in poor condition, especially if envenomation is mild, explaining why 18% of patients had tachycardia alone and 55% had hypertension. Pulmonary oedema, resulting from more severe envenomation, was seen in 27% of patients. In contrast, our experience is from a tertiary referral centre in Bombay. Most patients treated in our unit were referred from rural areas 80 to 150 km away, 6–36 hours after the sting. Moreover, those patients did not improve and required acute receiving treatment at primary care centres. Consequent to this referral pattern, a greater proportion of our patients had severe envenomation and presented late—18 of 31 patients treated in our unit with captopril in the past 10 years had pulmonary oedema with hypoten
tion. Four patients (all had severe pulmo
nary oedema with hypotension) died. In Dr Bawaskar’s series, 178 patients with pulmo
nary oedema were treated with prazosin and 30 (17%) died. This is not significantly different from the 22% mortality in our experience with captopril.

I agree with Abroug and colleagues that patterns II, III, and IV described in my paper are facets of the same underlying abnormal
ity. For this reason, they were all grouped under the category of predominant myocardial effects. Haemodynamic abnormalities in patterns II and III differ only in terms of the patients’ fluid balance, but the clinical features of the two patterns are so different as to need separate discussion. Pattern II is characterised by severe pulmonary oedema and mild or no hypotension. Pattern III is seen in dehydrated patients and manifests as severe hypertension, with little or no pulmo
nary oedema.

Abroug et al have, in a previous study, shown that left ventricular ejection fraction measured by echocardiography was severely depressed (26%) following scorpion enveno
mation. They showed a threefold improve
ment to 75% during recovery. In another study, they assessed right ventricular function using a pulmonary artery catheter; right ven
tricular ejection fraction was 24% following scorpion envenomation. They suggested that right ventricular function could be improved by treatment with prazosin. However, these results are equivocal, and further studies are needed to confirm their findings.

Our patients with pattern II showed severely raised PAWP and subnormal left ventricular stroke work index, while right ventricular stroke work index and right atrial pressures were normal, suggesting that the left ventricle was more affected than the right. In pattern IV, however, as well as right ventricular stroke work indices were severely depressed and both PAWP and right atrial pressures were grossly raised suggesting that this was a more severe deranged condition than could be treated with captopril. Abroug et al mention that they have been using dobutamine in scorpion envenomation with good results. Their patients also received antivenin, which is not available in India. It may be possible that antivenom favourably alters the response to inotropic catecholamines. The experience of Bawaskar and Bawaskar suggests that inotropic treatment given to patients who have not received antivenom does not decrease mortality. Although no controlled studies exist, studies using historical controls treated conventionally, including inotropic drugs, have shown that vasodilators are beneficial in the treat
ment of cardiovascular manifestations of scorpion envenomation. Vasodilators like prazosin, calcium channel blockers, ACE inhibitors, and nitrates have been used. However, whether any one of them is superior has not been studied. Currently available evidence indicates that these are equally effective and the suggestion that prazosin may have been effective in patients who did not improve with captopril as suggested by Bawaskar can only be consid
ered speculative.


Letters

Is BNP ready for use in clinical practice?

Editor,—Richards et al suggest that brain natriuretic peptide (BNP) measured 24–96 hours after acute myocardial infarction (AMI) is a powerful independent indicator for subsequent development of left ventricular failure and death.1

In their multivariate analysis the site (ante
rior v inferior) and type (Q wave v non-Q wave) of infarction do not appear to have been included. The important prognostic value of these indicators has been established.1 One might expect the anterior infarcts (39% of their study population) to demonstrate greater ventricular dysfunction, higher BNP concentrations, and a poorer prognosis than the inferior infarcts (31%). A similar relation may exist between type of infarct and outcome, although figures for each type are not given. It would be interest
ing to see whether BNP is still a powerful prognostic variable if site and type of AMI were included in their analysis.

Two further confounding variables that may have weakened the association between BNP and outcome are the timings of the radionuclide ventriculography and blood sampling (1–4 days after AMI). Assessment of ventricular function in the first 24–48 hours after AMI could have led to an overestima
tion of damage due to the phenomenon of myocardial stunning.2

The time course of BNP shows a peak at 16 hours followed by a significant decline in the next 48–72 hours. A narrower and standardised time window for ventriculography and venesection may have improved the correlations.

It would have been useful to know the area under their receiver operating characteristic (ROC) curve for BNP, which is highly relevant in assessing the true value of a test.3 They demonstrated a negative predictive value of 100% for BNP at a threshold of 20 pmol/l, but they used different thresholds for looking at the sensitivity and specificity and predictive power for left ventricular ejection fraction and left ventricular failure (25 and 33 pmol/l, respectively). Although these values were derived from their ROC analyses, it is difficult to envisage how a BNP result should then be interpreted in clinical practice.

If the additional prognostic value of BNP is confirmed once site and type of infarct are incorporated into this analysis BNP would be a useful test. However, the data from AMI are not included in the study, and it is difficult to envision how the BNP result would then be interpreted in clinical practice.

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BNP and its prohormone derivative (N-
terminal proBNP) offer exciting prospects for non-invasive assessment of myocardial func
tion and outcome following infarction. Rich
ards et al have clearly demonstrated the prog
nostic superiority of BNP over other neurohumoral markers thus supporting their initial hypothesis. Their concluding statement, however, suggests that plasma BNP “could reasonably be included in the routine clinical workup of a patient following myocardial infarction” seems premature.
To alter measurement method or timing simply to improve the correlation of BNP with LVEF are likely to be unproductive.

The statement by Dr Khan “The time course of BNP shows a peak at 16 hours followed by a significant decline in the next 48–72 hours” does not take into account the conflicting nature of the literature. The pattern of BNP change is dependent on the severity of infarction, the exact nature of the BNP assay employed, and its degree of cross-reactivity with pro-BNP 1–108 or its N-terminal deleted metabolites. In our hands the time profile shows a plateau between 24 and 72 hours and hence our election of a 1–4 day sampling window.

Dr Khan’s statement that it is “difficult to envisage how a BNP result should then be interpreted in clinical practice” is disingenuous. Our published paper clearly points out that plasma BNP of less than twofold the upper limit normal within 1–4 days postinfarction has 100% negative predictive value for an ejection fraction of < 40% four months after MI. Our report also makes it very clear that the postinfarct BNP measurement will allow better risk stratification and therefore better targeting of surveillance in the follow up period.

The most important and insightful part of Dr Khan’s letter is the proposal that BNP measurement method or timing should be used to favor the routine use of plasma BNP as a substitute for measurement of left ventricular ejection fraction. However, the prognostic value of BNP is strong and, at the very least, early postinfarct BNP measurements will allow better risk stratification and therefore better targeting of surveillance in the follow up period.

Transfusion associated graft versus host disease

Editor,—Ayha et al reported a case of transfusion associated graft versus host disease (TA-GVHD) in a non-immunosuppressed patient resulting from blood transfusion after coronary artery bypass grafting (CABG). They concluded that this devastating complication of transfusion is probably underreported. There is no doubt that diagnosing this condition needs a high index of suspicion because its manifestations can be seen in other more common conditions such as sepsis. Moreover, histological diagnosis needs specialist expertise in tissue typing.

We report another patient with TA-GVHD acquired following elective four-vessel CABG and perioperative transfusion of a total of six units of blood. A 68 year old man was admitted three weeks after surgery with a seven day history of skin rash, breathlessness, cough, and expectation of brown sputum. He had an extensive erythrodemic maculopapular eruption, oral thrush, tachycardia, hypotension, general chest crepitations, and mild hepatomegaly. His condition worsened progressively with development of profound pancytopenia, disseminated intravascular coagulation, metabolic acidosis, hepatitis, and acute renal failure. His bone marrow was aplastic and skin biopsy showed monocytic infiltration with cellular apoptosis supporting the clinical diagnosis of TA-GVHD. Treatment with high dose steroids, ciclosporin, and OKT3 was initiated but despite maximum support he died within seven days of readmission.

Although TA-GVHD does occur in immunocompetent individuals who had blood transfusion following other more frequent following cardiopulmonary bypass surgery. This type of major surgery appears to induce a transient immunodeficiency state, the mechanism of which is poorly understood and likely is to be multifactorial. This surgery, use of fresh blood with more viable lymphocytes, immunosuppressive effect of multiple transfusions, and transient reduction in interleukin 2 postoperative systemic lymphocyte transformation following

First the transfused units may contain an insufficient number of immunocompetent lymphocytes; second, some recipients may be able to destroy the donor lymphocytes on the basis of subtype differences in the class I or other minor histocompatibility antigens between the donor and recipient. Finally, it is possible, and quite likely, that some cases have remained unrecognised and unreported.

We hope that our publication in Heart of two cases of TA-GVHD after cardiac surgery will heighten awareness of this complication of transfusion, and result in full investigation and reporting of all suspected cases to SHOT (serious hazards of transfusion). It is necessary to establish the true incidence of TA-GVHD in cardiac surgery so that cost–benefit analysis and informed review of guidelines for the irradiation of cellular blood products for this indication can be completed.

This letter was shown to the authors, who reply as follows:

Ghrew et al report a patient with TA-GVHD following elective four-vessel CABG and perioperative transfusion of six units of blood. It would be interesting to know in this case the freshness of the transfused blood and whether shared haplotypes between the donor(s) and recipient was a feature in addition to the transient immunomodulatory effect of cardiopulmonary bypass.

The Oxford report re-emphasises that TA-GVHD as a complication of cardiopulmonary bypass surgery is seen in Europe. The association was first made in Japan, where the reported frequency of one way matching or sharing HLA haplotype in a non-first degree relative ranges from 1:312 to 1:256 in immunocompetent individuals.1 First the transfused units may contain an insufficient number of immunocompetent lymphocytes; second, some recipients may be able to destroy the donor lymphocytes on the basis of subtype differences in the class I or other minor histocompatibility antigens between the donor and recipient. Finally, it is possible, and quite likely, that some cases have remained unrecognised and unreported.

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