

ANAPHYLACTOID REACTIONS TO SNAKEBITE

SIR,—We have retrospectively studied 74 cases of envenomation by foreign snakes in Switzerland from 1967 to 1984. 42 of these incidents were in seventeen patients, and 7 of these 42 (18%) led to potentially life-threatening anaphylactoid reactions (table). There had been no reaction to the first bite; all happened after a second, third, or fourth bite.¹ All seven patients were given corticosteroids. The remaining 32 incidents (in thirty-two patients) were life-threatening in ten patients of whom one died.

SYMPTOMS OF ALLERGY IN SEVEN PATIENTS WITH REPEATED BITES FROM DIFFERENT VENOMOUS SNAKES

Previous bites*	Present bite*	Latency until symptoms	Symptoms†
Viperinae (×3), <i>V russelli</i> <i>V berus</i> , <i>V ammodytes</i> , <i>Cerastes</i>	<i>A contortrix</i>	A few minutes	U, D
<i>V ammodytes</i>	<i>V ammodytes</i>	35 min	U, D, AO, H
<i>V ammodytes</i> , <i>Dendroaspis</i> sp	<i>V ammodytes</i>	30 min	U, AO
Viperinae (×6)	Crotalinae	2 h	U, D
Viperinae (×6), <i>C durissus</i>	<i>C durissus</i>	20 min	U, H
Viperinae (×6), <i>C durissus</i>	<i>C viridis</i>	?	U
Viperinae (×6), <i>C durissus</i> , <i>C viridis</i>	<i>S miliarius</i>	“Very quick”	U, D, AO

*V = *Vipera*, A = *Agkistrodon*, C = *Crotalus*, S = *Sistrurus*.
†U = urticaria, D = dyspnoea, AO = angioneurotic oedema, H = hypotension.

Ellis and Smith² and Robert³ have emphasised the importance of anaphylactoid reactions to snakebite, and Kern⁴ showed that bites from different species could provoke cross-sensitisation. Reid and Theakston⁵ found that 78% of the Waorani tribe had antibodies against all locally prevalent *Bothrops* species. 4.9% of deaths in this tribe were due to snakebites, but the exact cause of death was not specified.

Our findings suggest that anaphylactoid reactions to snake venom, which can never be managed as promptly as they should be, are an underestimated contributing factor to fatal snakebites in endemic regions, apart from the toxicity of the venom itself.

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ANTIBIOTIC PROPHYLAXIS AND STERNAL WOUNDS

SIR,—Your Sept 28 editorial rightly stressed the lack of consensus over prophylactic antibiotic regimens in cardiac surgery (apart from the continuing value of beta-lactamase stable penicillins) and the high incidence of sternal wound infection after coronary artery bypass grafting. However, the striking effect of saphenous vein grafting on the organisms infecting the sternal wound^{1,2} was not mentioned.

Sternal infection after valve replacement was found to be uncommon^{1,2} and predominantly staphylococcal, but infection after coronary artery bypass was often due to gram-negative organisms. One group¹ used broad-spectrum prophylaxis with penicillins and gentamicin, and the other² used flucloxacillin, sometimes with amoxycillin. Few previous reports have considered the effects of saphenous vein harvest on the flora of sternal wound infections. For example, Palmer et al³ found that only surgical complications and prophylactic cephalosporins were associated with enterobacter mediastinitis after a variety of open heart

operations, but made no comment on the fact that all but one of the eight infected cases had had coronary artery bypass surgery. Coronary artery bypass may require prophylaxis different from that for valve replacement, but disinfection and depilation of the graft donor site is probably very important. Broad-spectrum prophylaxis with some agents encourages coliform infections;⁴ we² found that fewer gram-negative sternal infections resulted if saphenous vein harvest was restricted to the lower leg, but were unable to eliminate the possibility that this effect was due to differences in prophylaxis.

In their study of endotoxin immunity, postoperative fever, and gram-negative infection Freeman and Gould⁵ did not specify the proportion of saphenous vein graft surgery performed. Contamination of the graft from the skin of the leg and groin has been suggested to be the source of gram-negative sternal wound infection after coronary artery bypass surgery,^{1,2} and this sternal wound flora could be an alternative source for endotoxin. We note the low rate of postoperative gram-negative colonisation of Freeman and Gould's patients when flucloxacillin prophylaxis was used,⁴ but suspect that skin contamination at the time of surgery may be more important. Thus postoperative fever might respond to improved skin disinfection rather than requiring bowel decontamination or anti-endotoxin. Although circulating endotoxin may indeed originate directly from an atonic colon after any operation, it seems unlikely that haematogenous spread of bowel coliforms seeds the sternal wound after coronary artery bypass grafting but not after valve replacement. Any evaluation of postoperative shock, fever, or gram-negative infection should take account of the numbers of saphenous vein graft procedures and of differences in prophylaxis used in the study groups.

Sternal wound infections with *Staphylococcus epidermidis* are difficult to prove, but we agree with Bor et al⁶ that this organism may be a pathogen at the sternotomy site, which provides a fertile mix of sawn bone, haematoma, wire sutures, and bone wax. We isolated multiply-resistant *S epidermidis* repeatedly in moderate or heavy growth from seven of seventeen minor sternal infections.² These wounds often broke down superficially, had surrounding inflammation, and oozed serosanguineous fluid, sometimes for weeks. They usually responded to local antiseptics or irrigation, but oral antibiotics were required in two cases.

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ANTIBIOTIC-INDUCED NEUTROPENIA

SIR,—Your Oct 12 editorial emphasises toxic depression of granulopoiesis as the major cause of antibiotic-induced neutropenia. Neftel et al^{1,2} found β -lactam antibodies in several patients treated with these antibiotics, whether or not they were neutropenic, and concluded that antibody formation and neutropenia were not related. However, our studies provide evidence for antibody-mediated destruction of neutrophils as a significant factor in antibiotic-induced neutropenia.

Using a serological technique similar to the one used to demonstrate an immune mechanism for penicillin-induced haemolytic anaemia^{3,4} we found penicillin/cephalosporin-dependent IgG antibodies reacting with neutrophils of patients with penicillin/cephalosporin-induced neutropenia.^{5,6} The pathological significance of these antibodies was supported by the