given (one 6150 anti-factor Xa IU subcutaneous injection daily). Despite emergency caesarean section for placental abruption, the fetus (570 g, 30 cm) died. No fetal malformation was found. The placenta showed numerous small focal ischaemic necrosis.

A 27-year-old woman (gravida 4, para 2) was admitted for uterine bleeding at 31 weeks' gestation. She had had a deep venous thrombosis 6 years earlier after a normal delivery. The patient’s sister had a stillbirth at 34 weeks. APC resistance was shown (APC ratio 1.65). The patient was kept on low-molecular-weight heparin prophylaxis (3075 anti-Xa IU; nadroparin, one subcutaneous injection daily). At admission fetal death caused by placental abruption and pregnancy-induced hypertension were found. After the stillbirth subarachnoidal haemorrhage was shown in the baby (1500 g, 44 cm) and the placental surface was coated by blood clots. In all patients the postabortal and postpartum courses were uneventful.

There is evidence that APC resistance in symptom-free women might contribute to midtrimester fetal loss.1 Our three cases indicate that APC resistance in women after clinical presentation of thrombosis comprises fetal outcome more severely than in symptom-free conditions because, despite thromboprophylaxis, the resistance may result in placental abruption, and fetal death. Nevertheless, our experiences suggest that the dose of low-molecular-weight heparin recommended for thromboprophylaxis should be increased during the pregnancy of the thrombophilic patient who has had thrombosis; this may reduce the risk to the fetus, just as in those mothers with antiphospholipid antibodies who were given full-dose heparin therapy2 after previous fetal loss.

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Drought and malaria retreat in the Sahel, West Africa

Sir—The Sahel is a belt of dry savanna lining the southern border of the Sahara. Since 1970, rainfall has decreased by nearly 30% in this region with severe droughts in 1972, 1983, and 1991–92. The impact of dryness on malaria, endemic in the Sahelian belt, has been studied in Senegal (the Niayes) and the Niger Republic (Niger Valley, Zinder, Diffa).

The Niayes are marshy areas, located in north-west Senegal. Mean annual rainfall decreased from 684 mm (1931–60) to 427 mm (1980–89), and to 259 mm (1992) (fig.). The rainy season shortened from 6 to 4 months. The same was observed in Niger, where the rainfall at Niamey decreased from 640 mm (1950–69) to 457 mm (1981–90). The environment has changed drastically under the combined impact of drought and population growth. Numerous trees were cut for domestic use and stayed unreplace due to the drought. In Niger and Senegal, marshes with emergent vegetation (cattail) were larval habitats of Anopheles funestus, a major malaria vector. Then, these habitats became dry even after the rainy season, although their soil retained some moisture allowing vegetable cultivation. As a consequence of environmental changes this vector almost disappeared, comparing data recorded before 1970 and after 1988. In Senegal, An funestus accounted for 66% of night-landing collections in 1967 with an average of 33 bites per man per night and a sporozoite index between 1-2 and 3-1.% In 1991, this mosquito was no longer captured.1 In 1995, despite heavy rains, its populations did not recover because larval habitats were not restored. In Niger, An funestus was also one of the main
Malodorous wounds

Sn—Mills and colleagues report (Nov 9, p 1282) a man who pricked his finger and smelled putrid for 5 years. Clostridia were isolated repeatedly and could not be eradicated by prolonged courses of antibiotics despite very good in-vitro susceptibilities.

A 58-year-old woman presented to the Department of Internal Medicine at Karl Franzens University with a 35×15 cm large skin ulcer beneath the right axilla which developed over a 4-year period after lung surgery. The ulcer was very malodorous, and anaerobic gram-positive rods were isolated (Bacteroides fragilis and Clostridium spp.). Despite repeated antibiotic treatment the ulcer did not decrease in size, odour did not diminish, and anaerobic bacteria persisted. Multiple repeated smears for acid-fast bacteria and mycobacterial cultures were negative. Finally, after five repeated biopsies the histological work-up revealed granulomas consistent with tuberculosis. Acid-fast bacilli were not seen. The ulcer healed promptly under tuberculostatic triple therapy. We have also reported another patient with salmonella sepsis recurring eight times despite prolonged treatment with different antibiotic regimens that were highly active in vitro. Salmonelles were eradicated with unusually high doses of intravenous ceftriaxone plus ciprofloxacin after the eighth recurrence of sepsis. Finally tuberculosis of the liver was diagnosed by PCR and histological work-up of biopsy material, and the patient recovered fully with appropriate triple therapy.

In both our cases the main bacteria isolated caused superinfections and persisted after antibiotic treatment despite high sensitivity in-vitro. Tuberculosis was the underlying disease in both patients, possibly leading to an immunological so-called blind spot for other bacteria. Because of the non-suppressive inflammation Mills and colleagues report (which did not subside with a broad spectrum of commonly used antibiotics), the chronic course of the disease, and the fact that atypical mycobacteria can be frequently isolated from chickens, mycobacterial infection should be considered in their patient. For the management of the patient we suggest examination of biopsy specimens for granulomas, and even in the absence of granulomas antimycobacterial therapy seems indicated.

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