A 54-year-old man was diagnosed with Crohn's colitis in 1988. Although Crohn's disease was controlled with mesalazine (3 g per day), pyoderma gangrenosum appeared on the right leg in April, 1993. Topical corticosteroid failed. Colchicine 1 mg per day was started in August, 1993. The skin lesions disappeared within 3 months and colchicine was stopped in September, 1994. No relapse occurred during the 5-month follow-up without colchicine.

Colchicine exerts its anti-inflammatory effect by inhibiting synthesis of neutrophil microtubules. The decreased phagocytosis and chemotactism of neutrophils might explain the efficacy of colchicine in pyoderma gangrenosum. When classic local therapy fails, colchicine may be a good alternative for these lesions during inflammatory bowel disease, before recourse to high-dose corticosteroid or colon surgery.

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Nicotine for pyoderma gangrenosum

SIR—Signorelli et al[1] pointed out the beneficial properties of nicotine on intestinal obstruction reported by two investigators in the 18th century. The authors conclude that Nicot's herb (tobacco) had been doomed to a mixed reception throughout three centuries: "found guilty by cardiologists and respiratory physicians" and "acquitted by gastroenterologists". Nicotine, not smoking, could be acquitted by dermatologists too.

We attempted to treat a 44-year-old woman, who had long-standing and intractable pyoderma gangrenosum on her legs, with nicotine chewing gum. Pyoderma gangrenosum is well known to be associated with ulcerative colitis, which has been reported to have responded well to nicotine treatment (gum[2] and transdermal patch[3,4]). We began nicotine gum three tablets (2 mg per tablet) daily and the skin lesions consisting of erythemas, pustules, ulcers, and crusts cleared up within 3 weeks. Experimental withdrawal of nicotine gum for 2 weeks resulted in recurrence of ulcerations. The gum was reintroduced and the lesions cleared up again within 2 weeks.

Hence we as dermatologists hope for beneficial effects of nicotine in some skin diseases.

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AIDS in Manchester, 1959?

SIR—In July, 1990, we co-authored a letter[1] to *The Lancet* describing preliminary investigations undertaken on fixed paraffin-wax-embedded tissues from a young man who died in 1959 with a clinical syndrome that had all the hallmarks of AIDS.[2,3] Following application of the then relatively new polymerase chain reaction (PCR) to this tissue in a blind controlled study it was concluded that HIV proviral DNA was present in certain samples.

In July, 1992, we were approached by Dr David Ho, Director, Aaron Diamond AIDS Research Center, New York, in his capacity as a member of a committee investigating the possible link between the 1957 polio vaccine trials in the Congo and the AIDS epidemic. Ho requested sequence data and, if possible, samples of our material. These were sent. Ho's findings were finally published in *Nature* on April 6.[4]

It appears that Ho's findings have confirmed our own view (from sequencing work done in the two years after publication) that the HIV DNA detected was from a relatively "modern" strain. More interestingly, he suggests that tissue from more than one individual might have been examined. In view of the dilemma highlighted by these findings and in accordance with good laboratory practice we favour further investigation by a third party in an attempt to resolve the issues. Accordingly, arrangements have been made for the Forensic Science Service, Wetherby, Yorks, to perform DNA fingerprinting on residual material held in Manchester and Ho has been invited to return his own samples to this same laboratory. We understand that he is prepared to do so. Subsequently, we anticipate that HIV PCR will be used to determine the presence or absence of HIV sequences in the tissue. The results of these studies will be published.

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HCV-associated liver cancer

SIR—De Mitri and colleagues (Feb 18, p 413) examined liver tissues from patients with liver cancer associated with hepatitis C virus (HCV) for the presence of HCV and hepatitis B virus (HBV). They used PCR to amplify the HBV genome with primers specific to the pre-S, S, X, and C genes, and reverse transcription (RT) PCR to detect the replicative intermediates (negative strands) of HCV. There are potential difficulties associated with these methods.

The integration of HBV DNA in liver tumours, although clonal with each tumour, is random, frequently subgenomic, and does not contain an intact open-reading frame.[1] Southern blot of nucleic acid extracted from liver tissues and hybridisation with a complete HBV DNA probe, albeit less sensitive, can detect integrated fragments of all sizes. PCR, however sensitive, will give false-negative results if the selection of primers used does not cover the integrated sequences.

The validity of the detection of HCV-negative strands has also been considerably debated.[2-4] Earlier correspondence in *The Lancet* highlighted the occurrence of false-positive detection of HCV-negative strands.[4] Our experience is