follow-up clinic the results obtained were not far removed from those in healthy adults. The twenty patients with clinical syndromes following surgery for peptic ulcer—that is, recurrent peptic ulceration, afferent or efferent loop hold-up, dumping syndromes, or malabsorptive states—showed evidence of leucocyte ascorbic acid depletion. Patients with duodenal ulcer on admission for surgical management were found to have values similar to those in the subjects with clinical syndromes following operation.

Our findings indicate that it is in these patients with symptoms following gastric surgery that ascorbic acid depletion may be expected.—We are, etc.,

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Elastic Band Injuries

SIR,—I have read with interest the paper by Mr. Ian Kitchin and others (22 April, p. 218) on the production of artifact ulcers and bone lesions by elastic bands. It is not unusual for mental defectives to place elastic bands over their teeth and to leave them in situ. The elastic band moves slowly towards the apex of the teeth, and as it does it strips the mucoperiosteum from the bone, resulting in loose hypertrophic tissue which can be lifted from the bone with a probe. I have seen two such cases, which healed satisfactorily after removal of the elastic band and periodontal therapy.—I am, etc.,

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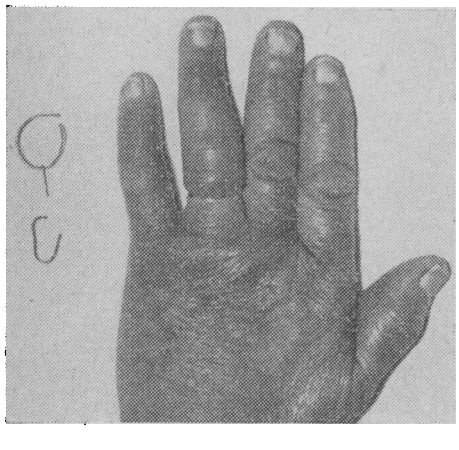
SIR,—I read with interest the article published in your journal last week (22 April, p. 218) entitled "Artifact Ulcers and Bone Lesions Produced by Elastic Bands." In it the authors state ". . . but for the band to have been lost and recovered at operation must be rare indeed. An extensive search of the literature has revealed no such case. . . ."

I admit that the two cases I reported[1] were not, as far as I was aware at the time, deliberately self-inflicted, but they did illustrate the damage caused by elastic bands hidden under wedding rings in just such a way as the first case reported in the current article. By a remarkable coincidence, my two patients both presented on the same morning in Lewisham Hospital Casualty Department.



It was only because I had found an elastic band under the ring of the first patient that I deliberately searched for one in the second. Both patients had presented with a swollen ring finger apparently due to a tight wedding ring. The photograph shows the patient's finger after removal of wedding ring and elastic band, both of which are shown alongside.

I admit that the cases reported by Dr. I. D. Kitchin, Dr. C. McGibbon, and Dr. R. H. Seville were deliberately self-inflicted injuries and involved bone, which mine did not, but I felt I could not let their comments pass without drawing attention to my report of four years ago.—I am, etc.,

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1 Thurston, J. G. B., *Brit. med. J.*, 1963, 1, 542.

Accidental Hypothermia

SIR,—Dr. C. W. Thomson (1 April, p. 51) has described some of the hazards of shivering and their treatment, and suggests these might be relevant to the problem of rewarming following accidental hypothermia. While it would certainly be important to control shivering occurring at this time, it seems probable that the cause of hypoxia and respiratory acidosis in this condition should be sought for other than in shivering.

Of 51 patients with accidental hypothermia[1–4] shivering was stated to be absent in 48, present in two, and slight in one. Of another 32 patients[5] it was stated that "the shivering response to cold is usually abolished" below 32.2° C. (90.0° F.). In 21 of these patients rectal temperature was below this level. Emslie-Smith[6] described E.C.G. changes associated with accidental hypothermia, including changes similar to those referred to by Dr. Thomson, but noted that shivering was not often evident clinically in his eight patients. This suggests that the coarse interference on the E.C.G. referred to by Dr. Thomson might not necessarily presage the onset of shivering in accidental hypothermia.

References to shivering during rewarming following accidental hypothermia are scanty, though Rees[1] reported its absence in an 86-year-old man at 27.8° C. (82° F.) and during rewarming to 32.2° C. (90.0° F.). Since various observers have commented specifically on the absence of shivering during accidental hypothermia, it might be supposed that its appearance during rewarming would occasion comment. Presumably, therefore, shivering is mostly absent also during rewarming. In neonates and infants shivering is absent during accidental hypothermia and subsequent rewarming.[7] In 71 episodes of induced hypothermia of 30–198 hours at 29–33° C. (84.2–91.4° F.), and during rewarming shivering was mostly absent except during active cooling or in association with a large fall in ambient temperature.[8] In dogs kept at 23–24° C. 73.4–75.2° F.) for up to 12 hours shivering was avoided during rewarming by immersing the animals in a water bath at 40° C. (104° F.) until body temperature reached 35° C. (95° F.).[9]

Since patients may remain alert and rational during eight days of hypothermia at 30° C. (86° F.), it is unlikely that uncomplicated prolonged hypothermia of this degree will be associated with serious disturbance of ventilation or acid-base balance. The following *p*H, P_{CO_2}, and P_{O_2} values for arterial blood relate to two mildly drowsy patients at 30.1° C. (86.2° F.) and 29.3° C. (84.7° F.) (rectal temperature) after 95 and 91 hours of induced hypothermia, respectively: 7.43, 35, 56, and 7.46, 28, 41, corrected for temperature and *p*H.[8]

Certainly, as indicated by the interesting references provided by Dr. J. D. Whitby (1 April, p. 51), the high mortality associated with active rewarming following prolonged hypothermia has been recognized for more than 25 years. During this latter period, however, prolonged *induced* hypothermia has been introduced into clinical practice. Conclusions based on hypothermia induced under control conditions of anaesthesia and maintained at comparatively fixed body temperatures, in conscious subjects, must have an especial validity for determining the effects of prolonged hypothermia and rewarming, while detracting nothing from the skill of earlier observers obliged to base their opinions on more complex and less controlled situations.

I am indebted to Dr. Knowle O'Brien, The Brompton Hospital, London, for the blood gas analyses.

—I am, etc.,

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6 Emslie-Smith, D., *Lancet*, 1958, 2, 492.
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Uterine Perforation with I.U.C.D. During Puerperium

SIR,—The letter of Mr. Wilfrid G. Mills in your columns (18 February, p. 427) brings me to record two cases of uterine perforation by contraceptive devices. In this country all but a small percentage of devices used are the Lippes loop.

Case 1.—A woman aged 31 was fitted with a loop 6 weeks after her second confinement. An hour later she experienced severe abdominal pain and the device was therefore removed. The pain persisted and she was thought to have salpingitis and this was treated with tetracyclines.

Ten days later severe pain returned and she was referred. Examination revealed a cystic mass in the right adnexa, some 6 inches in diameter. At laparotomy 20 ounces of old blood was removed from the right broad ligament. Both ovaries and tubes appeared normal. Careful examination of the uterus showed no obvious perforation but an area of bleeding just above the level of the internal os was ligated on the right side.

Case 2.—A woman aged 23 years was delivered normally after severe pre-eclampsia. A Lippes loop was inserted 8 weeks after delivery. She was later found to be pregnant again and in view of her previous history termination was carried out. However, attempts at removal of the loop resulted in the nylon threads breaking off on firm traction. *X*-ray showed the loop to be within or near the uterus. She was then referred. Examination under anaesthesia confirmed that no loop was present within the uterine cavity.

At laparotomy the loop was found lying between the vesical peritoneum and the uterus, its distal end imbedded in the uterine wall.

It would seem that the leading end of the loop is most likely to perforate the uterus, more so if pushed out of the inserter too quickly. A useful precaution would be to