I.U.D.s, such as those releasing progestogens, copper ions, or any other potential anti-implantation agent.—We are, etc.,

Max ELSTEIN
Karen FERRER
University of Southampton


Sir.—It is refreshing to see interest in the mechanism of action of the intrauterine contraceptive device and its potential hazards expressed in Dr. D. Wolters’s letter (8 January, p. 112). However, his concern about impregnated I.U.D.s and his representation of inert I.U.D.s such as the Dalkon shield as an end point in development requires comment. His example of the Dalkon shield as an “unimpregnated” device is unfortunate, since it contains over 40% of its weight as barium sulphate.

The use of gold, more noble metals than copper in I.U.D.s in the first half of this century was never shown to cause any increase in the incidence of malignancy in the male and female genital tracts. The manufacture of some “unimpregnated” I.U.D.s allows the formation of ridges on which cells can lodge and persist beyond their normal cycle, a situation analogous to the known carcinogenic effect of implantation.

The relation between the area of endometrium in contact with the I.U.D. and its effectiveness may be more subtle than Dr. Wolters’s letter would imply. There is evidence that “inert” I.U.D.s can not only stimulate uterine macrophages to attack the conceptus but can also stimulate an increased circulating immunoglobulin concentration. —I am, etc.,

R. M. PEARSON
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Removal of Extraneous Saf-T-Coil through Laparoscope

Sir.—A 20-year-old nulliparous woman married for twelve months was referred to the hospital for contraceptive advice. She had previously been using the contraceptive pill but after three months amenorrhoea from August 1971 she was changed to Volidin 21 and after a further six months’ amenorrhoea was referred to hospital. Prior to starting contraception she had a normal regular menstrual cycle. She was advised to discontin- uate the pill and was commenced on an intrauterine device, being strongly motivated against barrier methods of contraception.

Under general anaesthesia the vaginal findings were: normal vulva and vagina, retroverted small mobile uterus, and no abnormality was detected in the adnexae. The cervical os was healthy and dilated with ease to Hegar No. 7. The uterine sound was passed 4 in. (10 cm) and minimal curettages were obtained which showed mucus only on microscopy. A Saf-T-Coil 33-6 was inserted without difficulty.

The following morning she complained of tenderness over the lower abdomen but there was no rebound tenderness on palpation. The vital signs were satisfactory and vaginal examination confirmed tenderness in the right fornix. The coil loops were not seen on speculum examination and the coil could not be felt with a uterine sound. X-ray showed the coil in the right iliac fossa. Examination and laparoscopy under general anaesthesia with endotracheal intubation were then undertaken.

At operation, the pelvic findings were as previously noted and no coil was found on curretage. Laparoscopy showed a perforation of the right uterine cornua which was not bleeding but old blood was noted in the pouch of Douglas. The Saf-T-Coil was lying on loops of bowel in the right iliac fossa and was removed without difficulty through a suprapubic incision, holding the stem of the Saf-T-Coil with the Palmer forceps. The patient recovered without complication and was discharged home 48 hours later.

Leventhal et al.1 describe laparoscopic removal of intrauterine contraceptive devices following uterine penetration. They recommend minimal Trendelenburg position to avoid the possibility of the device being displaced into the upper abdominal cavity. Their experience was with four Lippes loops, and the case was quite suitable for laparotom- scopy as a suitable alternative to laparotomy should this complication arise.—We are, etc.,

R. S. LEDWARD
C. HEALEY
RONALD EADE
Royal Sussex County Hospital, Brighton, Sussex


Contact Dermatitis from Xerumenex

Sir.—We would like to report a case of contact dermatitis from Xerumenex ear wax solvent.

A man aged 43 developed bilateral acute otitis externa with spread of the dermatitis to the face, neck, and upper chest. There was marked swelling of the eyelids. The day before the onset of the eruption he had instilled Xerumenex once into each ear. It had probably been left in overnight. The manufacturers suggest that Xerumenex should be flushed out of the ears 15-30 minutes after application.

Readings at 48 and 96 hours of 24 standard patch tests were negative. A patch test with one drop of Xerumenex gave a strong reaction 3 cm in diameter at 96 hours. Xerumenex did not induce a pro-dermatitogenic surfactant. The active ingredient is triethanolamine polyolypeptide oleate-condensate 10%. The patient was also tested against the other ingredients of Xerumenex and against triethanolamine, which is a known skin sensitizer. There was a strong reaction to triethanolamine polyolypeptide oleate-condensate 1%, and no reaction to chlorbutanol 1%, propylene glycol 10%, or to triethanolamine 10%. On routine patch testing of eight patients against Xerumenex three produced slight reactions and one a moderate reaction. Of six patients tested to 1% triethanolamine polyolypeptide oleate-condensate 1% one produced a minimal reaction.

We thank Mr. A. W. Morrison and Dr. Harvey Baker for permission to report this case and Dr. Etain Cronin for the triethanolamine.

—We are, etc.,

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Ganglion Cysts of Bones

Sir.—The structure and pathogenesis of ganglia occurring around joints described in your leading article entitled "Ganglion Cysts of Bones" (13 November, p. 748) are inconsistent with our recent ultrastructural studies3 on ganglia of the wrist. Your leading article refers to the suggestion of some workers5 that "there is first a proliferation of synovial cells, but cells resembling fibroblasts are virtually absent from the wall of ganglia of the wrist. It is also misleading to say that "The dense fibrous capsule encloses cysts containing a viscous, jelly-like fluid," for although such a statement is of course justifiable on the grounds that the wall contains collagen fibres, it contains virtually no fibroblasts.

In discussing the pathogenesis of ganglia your leading article mentions, among others, a view that the ganglion originates from an out-pouching of synovial membrane through a defect in a joint capsule or a tendon sheath, and then states that this can seldom be substantiated by dissection but that possibly the original connexion underwent attenuation during the evolution of the lesion. One would expect that if ganglia did arise from synovial membrane they would be lined by synovial cells, but they are not. Ultrastructural study of the synovial cell is quite characteristic, and indeed two types, designated A and B, are known to occur in the synovial intima of all species studied to date.4 Our studies show that ganglia of the wrist have no obvious cellular lining but occasionally cells or groups of cells do occur in this region. These cells, however, bear no resemblance whatsoever to synovial cells or fibroblasts. Almost all the cells in the wall of the ganglion and the occasional ones lining the surface wall and mucoid content resemble smooth muscle cells, for they contain numerous characteristic myofilaments along which typical focal densities can be frequently demonstrated. Variants of this cell type, which besides containing myofilaments also contain aundant rough endo- plasmic reticulum and Golgi1 complex, were also found in our study. The situation here is somewhat analogous to that seen in the subintimal layer where two light microscopists had believed that fibroblasts were present but electron microscopy has shown the presence of cells, called multifunctional mesenchymal cells, containing myofilaments and variable amounts of rough endoplasmic reticulum and Golgi apparatus. In our experience the difference between the cells in ganglia and arterial wall is quite striking, and we believe that the cells resembling smooth muscle seen in the