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Artificial insemination for all?

Artificial insemination by donor (AID) is available (mostly semiprivately) in some 22 centres in Britain, of which seven are in London. In 1976 (the most recent year for reliable figures) at least 700 pregnancies were achieved by AID. Nevertheless, the distribution of centres providing the service is uneven—for example, there is none in the south or north-east of England or in Wales. The AID subcommittee of the Royal College of Obstetricians and Gynaecologists has called for new facilities to be set up to meet "a substantial and growing demand for AID," and has produced a booklet[1] for patients seeking advice about the treatment. The booklet, funded by the DHSS, gives succinct advice on such matters as confidentiality (which must be absolute), complications of pregnancy (the incidence of which is normal), and legitimacy (which remains a confused question, though the booklet advises that "babies born within a marriage are presumed to be legitimate"). If a couple decide to go ahead with AID, what problems can they expect, and what are the prospects for success?

The indication for AID (usually gross male subfertility or familial disease) must be carefully checked. All women undergoing AID should first be investigated to rule out any obvious bar to fertility,[2 3] but even then AID is no guarantee of conception. In recent published series the proportion of women who conceived has varied from 37% to 71% when frozen semen was used,[4–7] and from 42% to 84% with fresh semen.[8–12] (One report[13] from California gave a conception rate of 94% when clomiphene was used to induce ovulation: 17 women had 15 full-term pregnancies, including two sets of twins.) About half the patients who conceive do so within three cycles of starting treatment.[8 9 12] The side effects of treatment are few: gonorrhoea is one risk, but screening is possible even when fresh semen is used[14] and is easier still with frozen semen.

The debate about the ethics of AID[15–17] seems now to have been resolved, but one aspect of the treatment that should not be ignored is the psychological problems[8 11] that may occur in couples. Apparently unsuitable cases,[3] such as patients with unstable marriages, are usually refused treatment. Nevertheless, in some series[5 7] up to half of patients defaulted during six months of treatment, and couples should, perhaps, be offered better psychological support during a course of AID.[7] In some circumstances help given to a couple to accept and enjoy their childlessness may be preferable to helping persistence with a treatment that makes them uneasy. Follow-up studies[12] after successful pregnancy with AID are few, but we have no evidence that the incidence of marital breakdown rises. There is little information on the long-term welfare of the children.[15]

The source of semen remains a problem. Most centres still use the long-suffering medical undergraduate, although four years ago we drew attention[18] to disquiet among clinical deans over this practice. There is no consensus about how often a man's semen should be used successfully. In Denmark[6] the limit is 15 pregnancies, and in Britain a limit of 20 has been tentatively suggested—a figure which seems rather high.

Should AID be more widely available within the National Health Service? At present it is mainly provided by university departments of obstetrics—and by private practitioners, whose fees may be far from nominal. The service is not particularly expensive, but in theory it diverts NHS resources which could have been used for the care of the sick. Potentially about 3% of the population[3] could benefit from AID, but until more people know about the service and it has become more widely acceptable the full extent of demand will be impossible to assess.

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Pyomyositis

A recent review from Nigeria[1] reminds us that, while pyomyositis is mainly a disease of the tropics and subtropics, it may also be seen in Europe. The muscles most commonly affected are those in the gluteal or quadriceps group, but any large muscle may be infected. Usually the first symptoms are pain in the affected muscle, followed within the next week by fever, localised induration, and oedema. A minor degree of leucocytosis and eosinophilia (about 10%) is commonly present at this stage.

Symptoms gradually progress, so that between 10 and 20 days after the onset the muscle mass is definitely oedematous and tender. At this stage aspiration of the muscle will yield pus, which usually produces a growth of *Staphylococcus aureus*. If no effective treatment is given, the infection will progress until finally the whole muscle is destroyed and replaced by a bag of pus. The muscle abscesses are often multiple, and occasionally there may be evidence of more disseminated pyogenic infection

—empyema, lung abscesses, and even myocardial abscess.[2-4] Nevertheless, generalised septicaemia is rare.

The cause of pyomyositis remains a mystery. Trauma, various parasitic and viral infections, and nutritional deficiencies have all been suggested as antecedents. None of these possibilities has stood up to close scrutiny, and there is still no plausible unitary theory of aetiology. Two features that such a theory would have to explain are the pronounced male dominance in reported cases and the frequent concurrent eosinophilia.

There is no specific method of making the diagnosis before suppuration. In the early stages management is based on a presumptive diagnosis made from the clinical features. In view of the almost universal role of *Staph aureus*, the optimum treatment is a β-lactamase-resistant penicillin in large doses.[5] If suppuration has occurred and pus is obtained on aspiration, then drainage must be assured by an adequate surgical incision. Pus should always be sent to the laboratory for culture and determination of antibiotic sensitivities of the organism. Because of the occasional occurrence of pyogenic lesions in the chest a chest radiograph is essential.

Though pyomyositis may destroy a large volume of muscle, functional recovery is usually good. Furthermore, cosmetic defects are unusual: the cavity left by the destroyed muscle apparently soon becomes filled in, presumably because adjacent muscle hypertrophies to occupy the space.

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Six-month-old persistent vomiters

Nearly all infants bring some milk up after feeds, often with a belch, but some do it more than others. Factors which may persuade a mother to seek medical advice include her faith in her doctor, belief in the magic of medicine, normal anxiety, and her threshold of tolerance for the constant soiling of the baby's clothes and the ever-present sour smell. Her doctor has to decide whether the vomiting is within normal limits. He will be guided by the baby's weight and weight gain: there is less likely to be organic disease if the weight is normal in relation to the birth weight, the duration of gestation, and the build of the parents. If the weight gain is defective, the family doctor should be able to suspect or rule out the possibility of underfeeding and child abuse from his knowledge of the family.

More than anything else diagnosis will depend on the history. The vomiting may be due to giving the baby solid foods (such as raw apple, biscuits, or rusks) before he can chew[1] —average age 6 to 7 months, but later in a backward child. The baby may be ruminating—and this is diagnosed by the history and by observation. The baby, usually aged between 4 and 9 months, arches his back, contracts his abdominal muscles, and by rhythmical movements of the tongue, jaw, and pharynx brings up milk; some escapes from the mouth; with the remainder he may seem to gargle, the milk disappearing and reappearing in his throat, with an apparent feeling of satisfaction. He may not perform the act when asleep, when watched, or when he is distracted or interested in something. He may hasten the regurgitation by inserting his fingers into the back of the throat. The child may present as "failure to thrive," and rarely there is serious loss of weight; only when a careful history is taken will the diagnosis be made.

In 1907 Brockbank,[2] reviewing old stories of ruminators with horns growing from the forehead, suggested that psychological stress was a factor: many others subsequently ascribed rumination to emotional deprivation,[3-7] and there have been reports of favourable response to family psychotherapy[8 9] and aversive conditioning with an electric stimulation belt.[10] Fleisher[9] described an 8-month-old infant who before the diagnosis of rumination had been subjected to an *x*-ray examination of the skull, encephalogram, three barium meals, oesophageal manometry, endoscopy, treatment with propantheline, thickened feeds, soya milk, postural and other restraints, and threatened pyloroplasty or gastrojejunostomy—all causing a long, painful, costly delay in management. Treatment directed to reduction of emotional deprivation slowly but successfully cured the child.

Nevertheless, many paediatricians doubt whether all cases of rumination can be explained by psychological factors. The editor of the *1959-60 Year Book of Pediatrics*[11] commented, "We are not satisfied that the condition is due to emotional instability on the part of the mother. What do we know about the gastrointestinal tracts and the central nervous system of these patients?" At least some, probably many, ruminating infants have oesophageal reflux. Herbst *et al*[12], after describing three examples of ruminators with hiatus hernia, wrote that "although rumination has sometimes been considered a manifestation of psychiatric illness, the clinical features, anatomic and functional abnormalities, and therapeutic regimens advocated in the literature indicate that oesophageal disease, especially hiatus hernia, is the basic cause of the syndrome." Certainly the association is common: presumably the reflux makes it easier for the child to regurgitate and then, perhaps, emotional deprivation may somehow encourage rumination.

Hiatus hernia is often difficult to diagnose with certainty, but what is more important is the presence of reflux. Nevertheless, neither reflux nor emotional deprivation may be found in some ruminators, and there may be other factors. The rumination syndrome has been found, for example, in biotin-responsive propionic acidaemia.[13]

Persistent vomiting may be due to reflux or hiatus hernia without rumination. Symptoms may begin late (at several years of age) as a result of the development of oesophagitis and ulceration. The prime symptom which should alert the physician to the possibility of reflux with ulceration—and a symptom which should always be sought by direct questioning —is blood in the vomit. Occasionally reflux presents as a persistent cough, owing to inhalation of regurgitated material, causing patchy pulmonary consolidation.[14] Only on careful questioning will the persistent vomiting become apparent. Hiatus hernia occurs more often in children with cerebral palsy[15] and was found in 15 out of 20 persistent vomiters among 136 severely mentally defective children.[16] Hiatus hernia is sometimes found in association with abnormal head and neck movements, notably opisthotonos (Sandifer's syndrome).[17] When there is clear evidence of oesophagitis due to reflux, surgery may be indicated if the symptoms cannot be controlled by medical treatment, if the child is failing to thrive, or if there is a stricture.

Persistent vomiting should also suggest the possibility of