CONSTRICTIVE PERISALPINGITIS

A PRELIMINARY REPORT ON ANOTHER POSSIBLE CAUSE OF NON-PATENCY OF THE PROXIMAL PORTION OF THE FALLOPIAN TUBE WITH A SHORT NOTE ON A MORE CONSERVATIVE APPROACH TO TUBAL PLASTIC SURGERY*

LOUIS RESNICK, M.D. (CAPE TOWN), F.R.C.O.G., Senior Lecturer in Obstetrics and Gynaecology,
University of Cape Town

In spite of the fact that non-patency of the proximal $\frac{1}{2}$ -1 inch of the fallopian tube might be remedied by complicated and often spectacular tuboplastic operations, it is the general experience of gynaecologists that women who have had such operations conceive infrequently, especially when associated obliteration of the fimbriated end of the tube had been present. However, even if pregnancy is not attained, operation is more than justified in many patients for its beneficial effects. These include the relief of debilitating symptoms such as chronic pelvic pain, irregular and often profuse menstruation, and invalidism, and the restoration of morale.

The purpose of this short communication is to draw attention to the possibility, in the light of recent experience, of occasionally being able to overcome proximal-tube non-patency without having to resort to major plastic procedures, where such non-patency was not due to primary endosalpingitic blockage. Alternatively, a less serious diagnostic operation is performed to establish whether such non-patency is due to either (i) constriction of the narrowest portion of the tube by peritoneum which is the seat of perisalpingitic inflammatory change, with or with-

out chronic tubal spasm in addition; or (ii) primary endosalpingitic blockage.

CAUSES OF NON-PATENCY OF PROXIMAL THIRD OF THE FALLOPIAN TUBE

These may be divided into 2 main groups, viz: (1) inflammatory, and (2) non-inflammatory.

1. Inflammatory Changes

There is no doubt that these are the most important contributory factors in the causation of tubal non-patency, affecting the ampullary end more often than the proximal tube. Inflammatory change manifests most commonly as a perisalpingitis, or infection from without the tube, rather than endosalpingitis or infection primarily from within the tube.

Perisalpingitis usually follows postabortal and puerperal infection from the lower genital tract; less often it follows operations such as curettage or cauterization of the cervix. Pyogenic organisms are usually responsible, and the common method of spread of infection is by way of the lymphatics and blood vessels of the parametrium and broad ligament, affecting primarily the peritoneum enveloping the fallopian tube. Interstitial involvement may occur with little, if any, encroachment on the tubal

^{*} From the Somerset Hospital Unit of the Department of Obstetrics and Gynaecology, University of Cape Town.

mucosa. Ampullary blockage from inversion of the fimbriae is a frequent end result. The prognosis for childbearing is therefore not as bad as with primary endosalpingitic infection, particularly following antibiotic therapy. Perisalpingitis due to coliform infection from gastro-intestinal sources such as the appendix, colon and bladder, and blood-borne perisalpingitis from childhood illnesses such as scarlet fever or measles, is less commonly seen. Perisalpingitis of tuberculous origin, and salpingitis isthmica nodosa, which affects the proximal tube and manifests itself as a nodular inflammatory thickening, are both very uncommon in this institution.

Endosalpingitis used to be attributed only to gonococcal disease with primary involvement of the lower genital tract, spreading to the uterine mucosa and then to the tubal mucosa. In recent years it has been reported that such infection occurs less frequently than was previously believed, although it is still relatively common. In the Somerset Hospital unit such infection in the non-White is still frequent, however. Mucosal damage, with gross tubal destruction and distortion in the form of pyosalpinx, hydrosalpinx, tubo-ovarian abscess, and even pelvic abscess, is frequently seen in untreated cases and is commonly accompanied by later organic blockage of both the proximal and distal portions of the tube. Future conception is therefore highly unlikely, especially in untreated cases. Tuberculous endosalpingitis may also destroy the fallopian tube, but not infrequently the ampullary end is found to be patent.

2. Non-inflammatory Causes

These occur less frequently than inflammatory causes and rarely affect the proximal tube. They include:

- (i) Foreign bodies in the tubal lumen, such as mucus plugs, may produce temporary occlusion, but they are usually amenable to treatment with insufflation.
- (ii) Angulations of the fallopian tube. These may be caused by adhesions, ovarian cysts, intraligamentary cysts or fibroids, or may follow suspension operations involving the shortening of the round ligaments.
- (iii) Developmental tubal defects, as manifested by elongated or convoluted organs or absence of one or both tubes (not infrequently in association with other manifestations of hypogenitalism) are causes of non-patency.
- (iv) Endometriosis of the tube is a rare cause of obstruction of the tubal lumen.
- (v) Previous operations on the tube, such as salpingectomy (partial or total) for ectopic pregnancy, or sterilization, will cause non-patency.

RECENT LITERATURE

Greenhill,1 in a first major review from the USA, reported unfavourably on tuboplastic operations for infertility because only 1 live baby was born for every 22.5 such operations performed. With the advent of antibiotics and cortisone therapy, together with improvements in technique, including the use of polythene accessories, successes in procuring pregnancy immediately followed. In 1956 Greenhill2 recorded data of 2,113 tubal operations, and noted a pregnancy rate of 1 in 5 operations, with a living child being delivered for every 6½ operations performed.

Siegler and Hellman^a similarly analysed the results of 2,285 operations on the fallopian tube from the data of 734 USA surgeons, and noted 378 live births, or 1 live birth for every 6 operations. Salpingolysis and salpingostomy accounted for 80% of such procedures, from which 56% of the live births took place. Resection and anastomosis were carried out in only 8% of patients, and no more than 2% of live births occurred.

Johnstone recorded that, following 27 tuboplastic resectionimplantation operations, 8 women (29.6%) conceived, but

only 5 living children were produced.

Peel⁵ reported on 48 patients who had had plastic tubal operations in the presence of pelvic inflammatory disease; 8 conceived and had successful pregnancies. He stressed that obstruction, owing to obliteration of the fimbriae after ascending salpingitis, produced universally bad results.

Fuensalida, Ernst and Bravo^s noted that only 5% of 40 women who had undergone salpingostomy, with creation of abdominal ostia, subsequently conceived. After 13 tubouterine implantations, 23% conceived. The same authors reviewed 964 salpingostomies performed by 30 surgeons, with a pregnancy rate of only 8.5%, suggesting that this procedure has only a limited application in correcting the tubal factor in infertility. in infertility.

Green-Armitage⁷ reported a pregnancy rate of 42.2%, with 36.1% going to term, in a series of tuboplastic operations. He injected cortisone into the tube after opening the blocked ampullary portion. He also noted fibrous occlusion of the tube at the cornua and/or in the first 1/2-inch of the isthmus, with a normal outer two-thirds of the tube, in 43 out of 50 consecutive operations on young women with a history of abortion before or in the early years of marriage. The tube was implantable after resection in all 43 instances, with a 40% success rate.

Shirodkars recorded 140 tubal implantations with a 90% patency rate after 4-5 years, and a 35% pregnancy rate, in postabortal and congenital cornual blockage. Occlusion of the fimbriated portion lessened the chances of conception.

Moore-White noted the results of 83 tubal operations as follows: (i) after salpingolysis and ventral suspension in 15 patients, 14 (93.3%) conceived; (ii) in 38 patients who had bilateral salpingostomy with or without lysis, 11 had patent tubes and 6 (15.8%) conceived; (iii) 16 women had tubal resection and implantation, and 8 (50%) became pregnant; (iv) in 14 women with infantile tubes, 7 conceived after operation. Polythene accessories and antibiotics were not used.

Pous Puigmacía¹⁰ analysed 3,368 patients who had been subjected to operations on the tubes for infertility. Approximately 20% of these women conceived subsequently.

Palmer¹¹ reported on 396 personally performed operations for infertility due to tubal factors, without using polythene tubing. Again, as with other operations, the results for salpingostomy were poor; only a 15% success rate being achieved for conception. His best results occurred after salpingolysis (46%) and tubal implantation (36%).

NON-PATENCY IN SOMERSET HOSPITAL

In the non-White Somerset Hospital gynaecological unit of the University of Cape Town pelvic infection is rife, mainly owing to the high incidence of septic abortion, and less frequently to venereal and other infections. Not only is chronic tubo-ovarian inflammation commonly manifest as palpable swellings on vaginal examination, but it is also frequently revealed on salpingography as quiescent unilateral or bilateral hydrosalpinges. Chronic ill-health and invalidism associated with infertility, resulting from the above pathology, is found in the bulk of the patients attending the Somerset Hospital outpatient clinics.

Abdominal operation for the relief of pain not responding to repeated conservative measures, has been undertaken frequently in Somerset Hospital during the past 3 years. Similarly, infertility was treated in the presence of nonpatent fallopian tubes due to pelvic sepsis. The aim was to preserve tubal and ovarian function, even in patients with grossly distorted hydrosalpinges and chronic tuboovarian abscesses. Results, as far as return to normal health is concerned, have been gratifying. Pregnancy rates have not been equally good, mainly because of the high incidence of fimbrial destruction, even though the proximal tube might have been patent. In fact, in many patients, even with marked hydrosalpinx formation, proximal-tube patency was noted with retrograde insufflation. When the proximal tube was non-patent, as verified by insufflation, great difficulty was often encountered in determining whether such obstruction was due to peri- or endosalpingitis, even after peritubal adhesions had been severed.

A simple procedure has been devised, by means of which the above types of non-patency could be differentiated without immediately resorting to major plastic operations such as excision and implantation.

Rationale of the Operation

When non-patency of the proximal ½-1 inch of the fallopian tube was due to perisalpingitic blockage, it was observed in a number of instances that the diameter of the tube was considerably reduced, almost thread-like in appearance, solid and fibrous in consistency, and non-patent with retrograde insufflation of air. It was surmised that such diminution in diameter could be due to chronic inflammatory change in the enveloping tubal peritoneum, contraction of which might compress and constrict this, the narrowest section of the tube. Blockage and interference with peristaltic contractions might therefore occur. A form of 'constrictive perisalpingitis' is believed to be present—a condition analogous to that which occurs in

constrictive pericarditis, where cardiac function is inhibited to a greater or lesser degree by a thickened, contracted, chronically inflamed pericardial sac, but can be restored to normal or near-normal after excision of the sac. It can also be suggested that inflammation of the enveloping peritoneum of the tube in its proximal section could initiate chronic spasm in that area with concomitant non-patency.

Such explanations of the cause of tubal blockage were substantiated later when division of the enveloping peritoneum resulted in restoration of patency, with return of peristaltic contractions to this portion of the tube. That some degree of inflammatory change in the muscular wall might, in addition, produce obstruction of the tubal lumen with or without spasm, was further verified following incision and 'splitting' of this layer, as performed in the Ramstedt operation for pyloric stenosis, patency being restored in 4 cases.

The Operation

The affected fallopian tube was mobilized by freeing all peritubal adhesions, and patency of the proximal portions was tested by retrograde insufflation of air if necessary, after first creating an artificial ostium in the ampulla (Fig. 1). Patency was noted after a free flow of air had escaped into the uterine cavity with a distinct gurgle or gush. Marked distension of the distal two-thirds of the tube with insufflation, and failure of passage of air into the uterus, even under considerable pressure, denoted obstruction in the proximal portion, which was then exposed between 2 pairs of tissue forceps. A

longitudinal incision through the peritoneum only was made in its anterior superior aspect (Fig. 2) so that the proximal $1 - 1\frac{1}{2}$ inches of the musculature of the tube was exposed. Bleeding of a troublesome nature was not encountered. and any slight bleeding was controlled when necessary by clamping and ligature. Air pressure in the distal tube was maintained throughout the above procedure.

In the first of 4 patients so treated, all of whom had associated fimbrial obstruction in addition, a remarkable alteration in the gross appearance of the tube in the constricted area was observed. Through the gap in the incised

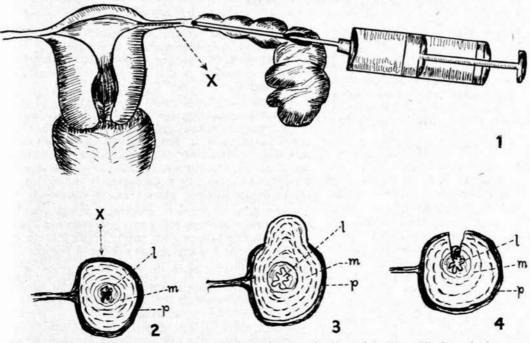


Fig. 1. Uterus and fallopian tube (hydrosalpinx), showing the line of incision (X) through the enveloping peritoneum, with retrograde insufflation of air through an artificially made ostium.

Fig. 2. Proximal tube showing line of incision (X) into enveloping peritoneum (tubal lumen closed).

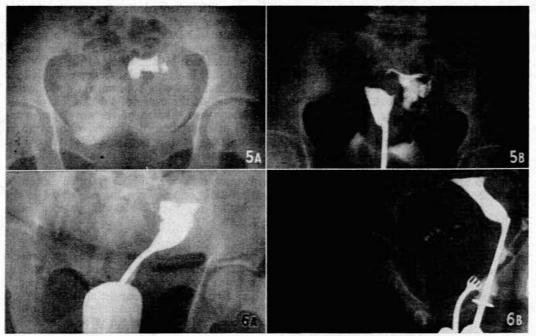
1=lumen, p=peritoneum, m= musculature.

Fig. 3. Pouting of tube through gap in peritoneum, with increase in diameter, and patent lumen.

Fig. 3. Pouting of tube through gap in peritoneum, with increase in diameter, and patent lumen. (Key as in Fig. 2.)

Fig. 4. Incision and 'splitting' of tubal musculature down to tubal mucosa (note patent lumen).

(Key as in Fig. 2.)



Figs. 5 and 6. Hysterosalpingograms on two patients who had constrictive perisalpingitis with gross bilateral hydrosalpinx and proximal tube blockage which was confirmed by retrograde air insufflation at operation. Patency was achieved with distal salpingostomy and incision of the proximal tube peritoneum. (In each, plate A was taken before operation and plate B 3 months after operation.)

peritoneum the tube bulged and pouted with an increased diameter and a resumption of the normal texture, in contradistinction to its earlier solid fibrous consistency and thread-like appearance (Fig. 3). Immediately after the entire constricted portion had been 'freed', a peristaltic contraction wave was seen to occur and air was heard to escape into the uterine cavity with a characteristic gush, accompanied by collapse of the distally ballooned out two-thirds of the tube. Repeated insufflations attained easy passage. No attempt was made to reperitonealize the exposed area, and X-ray salpingography 3 months later showed patency.

It was further observed that, should such a procedure fail to establish patency, without pouting of the tube through the incised peritoneum, a form of Ramstedt operation might overcome non-patency, possibly because some degree of interstitial inflammation, with or without chronic tubal spasm, was causing obstruction. Commencing at the distal end of the constricted portion of the tube, a longitudinal incision was made into the muscularis, which was then split with fine-pointed scissors until the mucosa was exposed (Fig. 4). Again, throughout the above procedure, insufflation of air was maintained in the distal non-obstructed portion of the tube. In 3 patients so treated, immediate patency was restored, as shown by the passage of air, even though the entire muscularis was not split. In a fourth patient patency was established after exposure of the tubal mucosa. X-ray salpingography 3 months later confirmed patency. Troublesome bleeding was also not encountered in these 4 patients, and the exposed area was not reperitonealized.

In selected cases, when patency was not established,

excision with tubouterine implantation was performed over a polythene tube.

Because of the common finding of multiple retention cysts of the ovaries in association with chronic pelvic infection, wedge resection, and with it ventral suspension, was carried out almost as a routine. Postoperative antibiotic therapy was prescribed in all patients for 10 days and X-ray salpingography was done 3 months later. Complications were not encountered.

Figs. 5 and 6 show examples of successful operations

on the tubes proved by salpingography 3 months after operation.

SUMMARY AND CONCLUSIONS

A further possible cause of non-patency of the proximal $\frac{1}{2}$ -1 inch of the fallopian tube is described. It is the constriction of this portion of the tube by chronically inflamed peritoneum, and it has been called 'constrictive perisalpingitis'.

A simple operation to prove this contention is described. In this the enveloping peritoneum is divided, and this relieves the non-patency. If this procedure fails to give the required relief, a further simple operation can be performed. In this the tubal musculature is incised and 'split' over the blocked area (as in the Ramstedt operation for pyloric stenosis). In those patients in whom this procedure was carried out, patency was re-established.

These minor operations provide a simple means of differentiating between endo- and perisalpingitic blockage of the fallopian tube, and an equally simple form of relief for non-patency of the proximal portion of the tube.

I wish to thank Prof. J. T. Louw for his interest in this subject and his helpful criticism, and Dr. G. J. Joubert, Medical Superintendent of Somerset Hospital, for permission to investigate the patients and to publish this paper.

REFERENCES

- 1. Greenhill, J. P. (1937): Amer. J. Obstet. Gynec., 33, 39.
 2. Idem (1956): Ibid., 72, 516.
 3. Siegler, A. M. and Hellman, L. M. (1956): Fertil. and Steril., 7, 170.
 4. Johnstone, J. W. (1955): J. Obstet. Gynaec. Brit. Emp., 62, 410.
 5. Peel, J. H. (1956): Amer. J. Obstet. Gynec., 71, 706.
 6. Fuensalida, S., Ernst, A. and Bravo, A. (1958): Bol. Soc. chil. Obstet. Ginec., 23, 198.
 7. Green-Armitage, V. B. (1959): J. Obstet. Gynaec. Brit. Emp., 66, 32.
 8. Shirodkar, V. N. (1960): Contributions to Obstetrics and Gynaecology. London: Livingstone.
 9. Moore-White, M. (1960): Int. J. Fertil., 5, 237.
 10. Pous Puigmacia, L. (1960): Rev. esp. Obstet. Ginec., 114, 3.
 11. Palmer, R. (1960): Proc. Roy. Soc. Med., 53, 357.