

Fallopian Tube Microsurgery

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When the fallopian tubes are blocked, sperm can't reach the ovulated egg. Infertility is inevitable and it's complete (sometimes referred to as sterility, meaning that time alone will never overcome the infertility). Tubes can be blocked from birth (congenital tubal obstruction, though this is rare), from inflammation (salpingitis), and from intentionally cutting, tying or clipping them (for sterilisation).

After inflammation, tubes can be partly blocked, usually taking the form of peritubal adhesions (fine sheets of scar tissue like plastic-wrap enveloping the tubes and ovaries), in which case there is subfertility: pregnancy is not impossible but it's likely to take longer than it should. Partial blockages can also trap an egg that does get fertilised, sometimes causing an ectopic pregnancy. More complicated combinations of one or two ovaries with tubes that are not completely blocked are common and special experience may be needed to interpret the likely impact on fertility.

I will discuss here the microsurgical and the so-called 'non-invasive' ways of treating blocked tubes. I will also talk about peritubal adhesions: how to stop them forming or, if they're being treated, how to stop them growing back. For really seriously damaged fallopian tubes, beyond help with microsurgery, IVF will be needed (and this is why IVF was first invented).

The Fallopian Tubes

Each *fallopian tube* is about ten to twelve centimetres long and consists of an outer, wide, thin-walled part, the *ampulla*, and an inner, much narrower, more muscular part, the *isthmus*. The isthmus

connects to the narrowest part of all, the *interstitial segment*, which passes for one to two cm through the wall of the uterus to join the cavity of the uterus. The opening at the outermost part is the *fimbrial end*, the delicate, finger-like processes of which adhere to the ovulating egg (in its sticky, mucus-like, *cumulus*).

The tube is lined by two kinds of cells: *secretory cells*, which produce mucus, glucose and other substances important for nourishing the egg and embryo; and *ciliated cells*, which bear tiny hair-like structures, the *cilia*, beating in the direction of the uterus and carrying the still-sticky cumulus down the ampulla, towards the junction between the ampulla and the isthmus.

It's at the fallopian tube's *ampullary-isthmic junction* where fertilisation takes place. The fertilised egg stays at the ampullary-isthmic junction for two to three days before the tubal isthmus loses its dense secretions, and the muscle in its wall relaxes, enabling the early embryo, now rid of its cumulus cells, to reach the uterus. The hormone that causes these changes in the tube is *progesterone*.

Investigations

The tests used to diagnose and to evaluate the severity of tubal disease are: *hysterosalpingography* (an x-ray of the uterus and tubes, which requires the radiologist to carry out

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a vaginal examination, at which a special liquid that is dense to x-rays is injected through the cervix) and *laparoscopy*. There are also *salpingoscopy* and *falloposcopy*, used to assess the lining of the tube (salpingoscopy is done through the wide end of the tube at laparoscopy; falloposcopy is done with a very fine, flexible scope through the cervix and the uterus into the narrow part of the tube). Carefully planned microsurgery may require all of these investigations if there's to be a realistic chance of success.

Salpingitis and Other Causes of Tubal Blockage

••• *Salpingitis* means inflammation of the fallopian tube (the Latin for which is *salpinx*). Inflammation may reach the tube from within, i.e. from the uterus, as with sexually transmitted diseases. These STDs include chlamydia and gonorrhoea. The salpingitis that follows can be without symptoms (or clinically silent and unsuspected, as is often the case with chlamydia), or it can be accompanied by *peritonitis* (inflammation of the *peritoneal cavity*), with pain, tenderness, fever and a vaginal discharge. These symptoms should lead to an urgent need to see a doctor, often with admission to hospital for treatment with intravenous antibiotics.

••• Inflammation can also reach the tube from its outside, by the spread, for example, of inflammation from a neighbouring organ such as the appendix. When this happens, most of the damage is on the outside, often sparing the delicate structure of the inside the tube. The tube and ovary can become separated by new tissue – referred to as *peritubal adhesions* if they surround the opening of the tube, and *peri-ovarian adhesions* if they enclose the ovary.

••• Think of an *adhesion* as a sheet of scar tissue. If you shaved off the skin over the tips of your two index fingers and held

your finger-tips together long enough an adhesion would grow between them to join them. So it is when the organs in the abdomen and pelvis lose their single-cell-thick lining and continue to lie close together; they become joined by an adhesion.

••• The same can happen when scarring of the tube follows surgical operations in the pelvis for other conditions, resulting in 'postsurgical' adhesions.

••• Adhesions can also block the outer end of the tube as a result of *endometriosis*, especially when the neighbouring ovary is affected. Rather less often, the tube can be blocked by endometriosis of its wall, usually in the middle part of the tube, a position different to what we see resulting from salpingitis.

••• In the best of all these cases, the tube itself is spared from harm and it's the plastic-wrap-like peritubal adhesions, enveloping tubes and ovaries and stopping egg pick-up by the fimbrial end of the tube. Microsurgery can then be particularly helpful.

••• If the infection is repeated, or goes untreated, the inflammation becomes chronic (termed *chronic salpingitis*). All of the tube's normal functions are disrupted: the hair-like cilia are lost, the muscle of the tube's wall is disorganised, the secretions needed to nourish the embryo are spoiled, and the tube itself nearly always gets blocked. The blockage can be mostly at the outer, fimbrial end (resulting in a *hydrosalpinx*), or mostly at the inner end, close to the uterus, involving the isthmus and/or the interstitial segment of the tube. Depending on how much damage there is, and where the damage is, some function can remain, enough for the tube to work if the blockages are removed with microsurgery.

••• Ideal for microsurgery too is when the tubes have been intentionally blocked in a sterilisation operation that needs

to be reversed. The operation is a *tubal anastomosis*. Pregnancy rates should exceed 90 per cent within 12 months of surgery, provided the operation is done skillfully, and either the father of the previous children is still the partner or the new partner is known to be fertile.

Preserving the tube's potential after salpingitis is most likely with a blockage in its isthmic or interstitial part; the microsurgery to correct it is the same as that carried out for reversal of most sterilisation operations, namely a tubal anastomosis.

Pregnancy rates of up to 70 per cent – almost as good as after sterilisation-reversal – can be expected.

Fimbrial blockages are more damaging; the microsurgery operation to reverse it is a *salpingostomy*. If there's still a small opening in the fimbrial end, the damage to the tube is typically a lot less than if the tube's completely blocked; the microsurgery for this situation is called *fimbriolysis*. When the tube is blocked at both its ends there's usually disruption along the tube's whole length and microsurgery rarely helps. Evaluating the tubes for microsurgery is a very, very specialised field. Before I make a final decision on whether or not to recommend an operation I often want to know what the tube looks like inside (by hysterosalpingography, falloposcopy and salpingoscopy) and outside (by laparoscopy), as mentioned above.

Carrying out a salpingostomy that gives the maximum chance of success, given the damage the tube has suffered, requires the surgeon to have had a great deal of experience with tubal microsurgery.

The most must be made of what fimbrial structures remain. Afterward the ultimate result, though, is out of the surgeon's

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hands. The scarring and thickening of the tube's wall, the loss of the fine hairs on the cells that propel the egg, the coexistence of other places of blockage along the tube – all come from the damage previous salpingitis can cause. This damage continues to reduce the tube's ability to transport eggs, sperm and embryos after a technically successful salpingostomy leaves the tube open and in reasonably close to its ovary.

On average, only five to ten per cent of women per year have a pregnancy that leads to a baby after salpingostomies. The results are better if the lining of the tube has been well preserved. The results are worse than this for women with badly damaged tubes and for women who've had a previously unsuccessful attempt at the operation.

For many women, one cycle of IVF gives a better chance of pregnancy than a year of waiting after salpingostomies.

The operations can, with the exception of tubal anastomosis, be done at an open operation, or *laparotomy*, or at *laparoscopy*. Special skills are needed in either case. Laparoscopy has the advantage of a short hospital stay, but the operation is technically much more difficult and usually takes longer. Less drying out of tissues occurs compared with laparotomy, but unexpected difficulties with the operation are less readily dealt with. Laparotomy is a more major procedure in terms of recovery, but it's more accurate and it's often quicker. The balance between the two has not been decided.

Preventing Adhesions

If you've read this far, you'll know well that adhesions are a menace to fertility. They can also cause pain from cramping of the intestines and obstructing the process of ovulation.

The hunt for ways of stopping adhesions forming or reforming after operations has been accompanied by some research, a lot of speculation, and a bucketful of snake-oil – magic remedies that are all promise and don't deliver the goods. The way adhesions form is interesting. Just briefly, the only way of stopping them growing back after they're cut out is to operate very carefully, with microsurgery, to carry out a laparoscopy eight to ten days after the microsurgery, detaching adherences before they become adhesions, and perhaps to use one of the new adhesion-barriers made from woven, dissolvable material.

Adhesions or healing, a delicate balance.

When the coating of the peritoneal cavity, the serosa, is injured – as it inevitably is to some extent at any pelvic operation – the blood on its surface soon clots. This superficial clot causes nearby organs to stick together, at least temporarily. Normally the clot dissolves in about six hours. The wandering cells of the peritoneal cavity, the *macrophages*, form a protective coat over the raw and exposed peritoneal tissues. Any blood clot that's still left three days after the operation begins to form scar tissue. If there's no blood clot left by three days after operation, there'll be no adhesions.

There's still some doubt about whether the macrophages themselves change into new peritoneal serosal cells or whether the underlying peritoneal cells produce the new serosal cells. It doesn't matter much. The important point is that the new serosa lining the peritoneum is fully formed by eight days after operation – except for those areas where blood clot was left undissolved, and which by eight days is already well on the way to forming scar tissue between organs such as the tube, the ovary, the uterus, and the intestines. This scar tissue is, then, the adhesion. Prevent the scar tissue forming and you prevent

adhesions. The more often the scar tissue (the adhesions) have been operated on, the more likely they are to regrow – and to be tough, thick adhesions rather than delicate, filmy adhesions.

Surgeons have long contrived to influence the outcome of peritoneal healing in favour of repair instead of scarring. To be sure, if we were to use drugs to completely stop blood from clotting or scar tissue from forming we could stop adhesions, but for someone who's recovering from an operation neither of these extreme manoeuvres is acceptable. Surgeons in the 1940s, for example, showed that anticoagulants were effective in stopping adhesions only if they were kept going for dangerously long times after the operation and in dangerously large amounts.

We need something that sticks around locally for the three days encouraging the clot to dissolve, or which keeps all the surfaces apart for the eight days it takes all the serosa to reform. For now, it's just the second option we have the means for.

Early laparoscopy

Eight to ten days after the microsurgery, we can do a laparoscopy. This is after the serosa has healed. At the laparoscopy we push apart any early points of adherence between the tubes and ovaries or other organs. This separates the very early scar tissue, on its way to forming an adhesion, and allows a few days for islands of new serosal cells to have another chance at linking up and completing the job. Several different groups of infertility surgeons around the world, including Sweden, Holland, the United States, as well as my own work in Australia, have shown that carrying out such an early ('second-look') laparoscopy is effective in diminishing eventual adhesion formation.

Enough 'third-looks' have been done to know that adhesions are usually much reduced and can often be prevented altogether.

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Surgical adhesion barriers

There are several products on the market that can be used at operation to form a barrier between the pelvic organs to stop them from sticking.

The best known barrier is Interceed (made by Johnson & Johnson). The material is woven from fibres of modified cellulose and, after being placed over abdominal surfaces the serosa of which is likely to have been damaged, dissolves in about eight days into simple sugar molecules (which are then absorbed by the body and metabolised). In the meantime, the cloth keeps the covered surfaces apart while the serosa reforms. Controlled trials have shown Interceed to be effective in reducing or (sometimes) preventing adhesions, but an adhesion-free result is not guaranteed.

If adhesions are going to grow back, they do so straight away. A surgeon who tells you that you've got just six months to get pregnant after he or she has 'treated' adhesions is misleading you badly. It's an unfair and self-serving thing to say. Sure, if you do get pregnant so quickly the surgeon probably did a good job and gets your thanks; but if you're not pregnant in that time the surgeon's made it your fault! You had six months and you didn't try hard enough! The fact is that adhesions, if they're going to grow back, do so straight after the operation, probably before you've even thought about having sex. By not regularly doing early laparoscopies, most surgeons cannot know how often their operations fail to relieve adhesions.

Tubal Canalisation

Dr Amy Thurmond, a radiologist (or x-ray doctor) from Portland, Oregon, became famous in the 1990s by advocating the use of a catheter or a probe made of wire to push through and so to open a blockage in the isthmus or the interstitial segment of the tube, close to the uterus.

The procedure is done vaginally, through the cervix and the cavity of the uterus. In practice a variety of tubal catheters and probes can be made use of for this procedure (and it's automatically done if you have a *falloposcopy*). The catheter or wire needs to be brought to the point at which the tube enters the cavity of the uterus. This can be done either at hysteroscopy (so that the opening of the tube is actually seen while the catheter is passed into it) or at x-ray, while a hysterosalpingogram is attempted. The procedure is moderately painful, more so than with the usual hysterosalpingogram, though the pain should be brief.

Dr Thurmond has not always been able to reproduce the good results she's had at home with this technique in other countries. This discrepancy means that the patients with tubes apparently blocked this way are not equally suitable for the procedure. There seems little doubt that her approach works well for cases of 'tubal spasm' or with blockages from 'plugs of secretion'. But there's doubt that these apparent blockages are in fact real or permanent blockages. I've found the technique especially useful when a tubal

anastomosis has for some reason become blocked. Note that it is not a procedure that's suitable for blocks at the outer part of the tubes.

Being a new form of treatment there's still debate among infertility specialists about the proper place of tubal canalisation procedures. Undoubtedly part of the problem in evaluating it is that many of the cases treated this way by some radiologists have not been properly investigated first. I personally doubt that true tubal blockages, especially if they extend over a centimetre or more, are best treated this way instead of with microsurgery.

This article has been abridged from:

Getting Pregnant, A Compassionate Resource for Overcoming Infertility and Avoiding Miscarriage

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