disease, the laparoscopy results in combination with the direct inspection of the interior of the tube via falloposcopy are required. The information gleaned from these two diagnostic procedures is interpreted based upon the prior information we have on the histology of the tubal wall. This prior microsurgical experience allows us to single out those patients with fibrotic occlusions who are best treated by surgery; (iii) for patients with nodular or pseudo-occlusion GnRH analogues have been found useful for treating the tubal wall. This benefit can be followed and evaluated by repeat falloposcopy as a marked improvement in the ease of passage of the catheter; (iv) it is important to recognize that tubal patency is not the goal for patients with nodular or pseudo-occlusion; once tubal patency is re-established, further treatment modalities (ART) have to be discussed in order to achieve acceptable pregnancy rates. Questions for the future remain as to which type of procedure, HMG with IUI or GIFT, would be most beneficial for treated nodular and pseudo-occlusion in light of the risk of tubal pregnancies.

More importantly, it is hoped that in consideration of such work as we have presented, current diagnostic techniques will be re-evaluated and patients will no longer be judged solely by the spilling of dye or radioactivity from the tubes. Instead, patients will be referred to and studied by the underlying pathology and ultimate origin of their tubal disease and once classified, be directed toward more appropriate therapies.

References


Wiedemann, R., Sterzik, K., Gombusch, V. et al. (1996) Beyond recanalizing proximal tubal occlusion (PTO) the argument for further diagnosis and classification Hum. Reprod., 11, 986–991


The diagnosis and treatment of proximal tubal disease

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Our discussion on the diagnosis and treatment of proximal tubal disease is in response to the preceding piece by Wiedemann et al. (1996a). In contrast to their thesis, ours on purpose addresses the treatment of proximal tubal ‘disease’ and not of proximal tubal ‘obstruction’ since we consider proximal tubal disease as a continuum with tubal obstruction of varying severity representing only the end of a spectrum. Based on this concept of proximal tubal disease, we agree with Wiedemann et al. on some of their concepts but have considerable doubts about others.

The authors are correct in stating that experienced tubal surgeons were initially sceptical about some of the early reports on successful tubal catheterization procedures. However after considerable initial scepticism, one of the most prominent surgeons in the US (De Cherney, 1987), was quickly convinced otherwise once given the opportunity to observe such procedures hands-on. As a participant in this first hands-on session one of these authors witnessed this expert in the successful performance of a radiologically-guided unilateral tubal catheterization procedure after previously observing only one (the contralateral) such catheterization in the same patient. This experience (now almost 10 years ago) is highly symbolic for the introduction (or lack thereof) of tubal catheterization procedures into the routine clinical practice of our profession. Here was a patient with long-standing infertility due to bilateral cornual obstruction. She had failed attempts of selective salpingography (SS), a highly important procedure to the concept of proximal tubal disease (which we will be discussing in more detail), and a tubal surgeon, inexperienced in tubal catheterization, had repaired her Fallopian tube in <10 min without surgery and without anaesthesia, with basically no risk and at dramatically lower cost. Who can be surprised that this surgeon has become a convert. What is astonishing, however, is how few of his colleagues have followed in the decade since. Why tubal catheterization procedures have attracted so few converts in all that time has remained somewhat perplexing. One cannot but wonder whether some widely distributed misconceptions have not profoundly contributed to this lack of acceptance. Unfortunately, Wiedemann et al. continue to perpetuate some of these in their preceding piece. Here are a few examples: (i) as many before them, these authors repeatedly allude to the concept of the ‘false-positive’ tubal occlusion. Such a concept can historically be found in the literature since the advent of X-ray hysterosalpingography (HSG) and is
usually ascribed to 'tubal spasm'; (ii) gynaecoradiologically contrast studies can be performed incorrectly. For example, excessive injection pressures may lead to the occurrence of iatrogenically induced cornual spasm. In the absence of technical incompetence there is, however, no such thing as a 'false positive' gynaecoradiological study. If a properly performed HSG leads to intermittent tubal occlusion, whether due to tubal spasm or some other pathophysiology, the intermittent occurrence of an occlusion, with alternating periods of patency does not denote a 'false positive' during the obstruction phase; it denotes significant proximal tubal disease.

This has to be the obvious conclusion from the long-term follow-up of the Multicenter Transcervical Balloon Tuboplasty Study (MTBTS), briefly alluded to by Wiedemann et al. in a very different context (Gleicher et al., 1993). What this study demonstrated was that patients with intermittent proximal tubal occlusion, if left alone without intervention by either SS or tubal catheterization, simply did not conceive (Figure 1). Intermittent tubal obstruction is therefore clearly a severe enough stage of tubal disease to prevent pregnancy from occurring. To consider such a finding as a 'false positive' simply does not make sense if we consider pregnancy potential as the endpoint for basically every diagnostic tubal evaluation.

This brings us to another major misconception. The literature equates tubal patency with functional tubal capacity to achieve pregnancy. This is once again a categorically wrong concept. Tubal patency as a simple diagnostic parameter has, in fact, only very limited sensitivity and specificity to predict the pregnancy potential of a tube (Gleicher et al., 1992; Karande et al., 1995a). Wiedemann et al. acknowledge as much in their communication by emphasizing that proximal tubal disease involves a spectrum of conditions. They then, however, make without any supporting data a quantum leap by suggesting that falloposcopy could aid in 'more reliably' diagnosing tubal pathology. More reliably than what?

Falloposcopy is a very interesting research instrument (Kerin et al., 1990). However, clinically, despite obvious industry interest to the contrary, it still appears to be an (expensive) procedure in search of an application. Current falloscopes have only the capability to look forward. One has to wonder about the clinical value in seeing a tubal obstruction, or seeing intratubal adhesions directly. A single SS, performed in <5 min under radiological control, will recanalize at least 75% of obstructions (Capitanio et al., 1991). Amongst the remaining cases, ~85% will be recanalized within another 5 min (at the most) if a tubal catheter is passed through the SS catheter into the tube (Confino et al., 1990). Who then needs the cumbersome, time-consuming and costly salpingoscopy, which after all this effort still has to be followed by SS and/or tubal catheterization if, in fact, an obstruction is encountered?

Wiedemann et al. are claiming that salpingoscopy provides them with a superior selection mechanism for their various choices of therapy. Our argument is that they are complicating a very simple clinical situation: the tube is either patent or not. If not, let us treat patients as outlined above. Pregnancy rates after SS and tubal catheterization respectively, are excellent (Gleicher et al., 1993). We are unaware of any data in the literature that would suggest that the addition of falloposcopy will add pregnancy potential to the above outlined tubal catheterization protocol, which clearly is simpler and less costly. Therefore, why do it?

All of this is not meant to denigrate their recent argument in another publication (Wiedemann et al., 1996b) that the diagnosis of tubal conditions beyond patency should be actively pursued. In fact, we fully agree with this statement. Where we disagree with Wiedemann et al. is how this further evaluation should be conducted.

If one accepts the premise that the ultimate endpoint for every diagnostic tubal evaluation is the capability to predict a tube's capacity to allow pregnancy to occur, then any further tubal diagnosis beyond patency has to address this tubal function and not tubal anatomy. We are unaware of any data that would allow such a functional prediction of tubal status via the use of falloposcopy. In contrast, oviducts can be functionally assessed by evaluating tubal perfusion pressures (TPPs) during SS. It would exceed the framework of this brief communication to provide the reader with a more detailed description of how TPPs are obtained. For this purpose, we refer to a number of recent publications (Gleicher et al., 1992;
Proximal tubal occlusion

Figure 3. Diagnostic and therapeutic algorithm for proximal tubal disease using a gynaecoradiological approach. Modified from Karande and Gleicher (1996).

Kalande et al., 1995a,b). Both the work at our centre and the studies by Afnan et al. (1995) in the UK quite clearly suggest, however, that an elevated TPP, which persists after tubal wire-guide cannulation, is indicative of decreased tubal wall compliance and is associated with extremely poor pregnancy rates (Figure 2). The evaluation of TPP thus, in contrast to falloposcopy, does provide a truly functional assessment of the Fallopian tube. Moreover, this technique is simple, minimally invasive, minimally time consuming and highly cost effective in determining the direction of future care.

We have also demonstrated that elevated TPPs are highly associated with the presence of (mild) endometriosis (Karande et al., 1995b). Our functionally poor prognosis patients may therefore very well coincide with the so-called ‘true PTO cases’ in the scheme of things described by Wiedemann et al. In fact their ‘non-nodular’ (fibrotic) occlusions are very likely the same as those cases reported by us to have reached tubal catheterization without successful reduction of TPP below ~350 mm Hg. Pregnancy rates are awfully low in such cases, and patients clearly should be quickly advanced towards IVF (Gleicher et al., 1994). Their ‘nodular occlusions’ are likely our endometrosis cases with elevated TPPs and with varying pregnancy chances, depending on the level of elevation in TPPs. We, in fact, have some preliminary anecdotal evidence that the treatment of elevated TPPs with either danazol or GnRH analogues will reduce TPPs and, by definition, therefore improve pregnancy rates (V.Karande and N.Gleicher, unpublished data). Finally, their ‘pseudo occlusions’ are probably those patients who successfully achieve patency with SS and demonstrate normal TPPs. These patients have excellent pregnancy chances, as widely reported in the literature.

In other words, Wiedemann et al. (1996a) and ourselves may not be as far apart on a conceptional level as it may appear from the outset. We, however, perceive their diagnostic algorithm as especially complex and therefore, costly. If one combines this with the fact that our diagnostic algorithm concomitantly also represents a therapeutic approach, the simplicity and cost effectiveness of our approach should become obvious.

Conclusion

Figure 3 summarizes our diagnostic/therapeutic algorithm for proximal tubal disease. The figure will be self explanatory. Since the introduction of gynecoradiology as a diagnostic and therapeutic concept into our routine practice, we have basically stopped the performance of diagnostic laparoscopies. As a consequence, our utilization of laparoscopies overall has decreased by ~75%. A recently performed study by a local insurance carrier demonstrated that this did not affect our pregnancy rates adversely in comparison to other practitioners with a traditional surgical utilization pattern. It reduced treatment cost per clinical pregnancy achieved, however, by >40% (VanderLaan et al., 1996).
In today’s economic climate this is obviously a major consideration, which in combination with the relative case of clinical performance and low risk of the gynecological approach towards proximal tubal diseases, should lead to a much wider utilization of this approach amongst practitioners in the field.

References

Table I. Results of sonographic evaluation of isthmus tubal patency with Echovist R 200 versus hysterosalpingography

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<tr>
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<th>Echovist R 200</th>
<th>Hysterosalpingography</th>
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<tbody>
<tr>
<td>Proximal patency</td>
<td>63</td>
<td>2</td>
</tr>
<tr>
<td>Proximal occlusion</td>
<td>10</td>
<td>22</td>
</tr>
<tr>
<td>Not assessable</td>
<td>3</td>
<td>0</td>
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Proximal tubal obstruction—is there a best way to treat it?

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Tubal infertility is still the dominant cause of infertility in up to 40% of all infertile couples, and in 10–25% of these cases proximal tubal obstruction is found (Segars et al., 1990). Whereas distal tubal occlusion is usually the result of pelvic inflammatory disease (PID), the etiology of proximal tubal occlusion (PTO) is still a matter for debate. Complete transmural fibrosis, nodular occlusion like salpingitis isthmica nodosa (SIN) or rare endometriotic lesions can be diagnosed as well as mild forms of obstructions associated with mucous plugs, cornual synechiae or simple tubal spasms (Duphny, 1994). Furthermore, pathological evaluation of resected proximal tubal segments fails to show anatomical evidence of occlusion in up to 60% of cases (Sulak et al., 1987). Consequently, the most appropriate approach to successfully treat infertility caused by PTO differs widely and is basically dependent on the aetiology of the cornual block.

Diagnosis of PTO

A thorough work-up of the underlying tubal disease is a prerequisite for counselling of the patient. Traditional diagnostic tools including hysterosalpingography and laparoscopy allow accurate diagnosis of tubal patency in most cases (Kerse and Vandervellen, 1973). Both methods show identical results in 55–75% of cases (Ismajovich et al., 1986; Fayez et al., 1988; Adelusi et al., 1995). In order to confirm peritubal adhesions, a hydrosalpinx or nodular proximal pathology, invasive laparoscopy is unavoidable.

However the diagnostic capability of simple dye-spilling techniques is very limited in cases where tubal filling fails and a proximal stop is assumed. Hysterosalpingography (HSG) may misdiagnose proximal tubal obstruction in ~50% of cases (Novy et al., 1988) and proximal tubal occlusion is overdiagnosed in 42% of patients by HSG (World Health Organization, 1986). Karande et al. (1995) clearly demonstrated that accuracy and prognostic value of HSG can be further improved by selective salpingography and recording of perfusion pressure. Recently, sonographic evaluation of tubal patency has further extended the spectrum of diagnostic tools for PTO allowing a clear visualization of the intramural and isthmic region (Deichert et al., 1989). In comparison to standard hysterosalpingography, we have found a specificity of 96.9% and a sensitivity of 68.8% by using the echogenic Echovist 200 for sonographic evaluation of isthmic tubal patency (Table I; Korell et al., 1996b). In evaluation of the proximal tube this technique might gain greater value, if Doppler techniques are used to assess the isthmic flow.

In order to confirm PTO or to rule out false positives, transcervical tubal cannulation was introduced. Based on our own experience with different catheter systems (Korell et al., 1995) and our data from transvaginal gamete intra-Fallopian transfer (GIFT) (Strowitzki et al., 1993), we found that blind transvaginal tubal cannulation can be easily performed after some training in the majority of cases. However, even in the