Do not hesitate and publish negative results and look for long-term results!

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Glaucoma disease will be diagnosed more often in the future. The detection of the disease is becoming easier and carried out earlier by new technologies with higher sensitivity by artificial intelligence. This results in an earlier start of a longer lasting therapy during the lifetime as people age. So the question arises: For how long can an eye tolerate the use of preservatives, antifibrotics like Mitomycin-C or implants inside?

Luckily there are many therapeutic options to choose from: topically used eye-drops, lasers and various surgical procedures. All of these 3 options allow you to target the production and/or the outflow of aqueous humour to reduce the intraocular pressure (IOP). Depending on the age and life expectancy of the patient and stage of the disease, a stepwise treatment is usually performed: beginning with eye-drops or Laser and ending up with incisional surgery. As a pre-step to incisional surgery, different MIGS (minimal invasive glaucoma surgeries) are used more often and elegantly, especially in combination with cataract surgery. The million-dollar question remains: what is the most safe, effective and long-acting method with few adverse effects and reasonable costs for the specific and individual eye and the quality of life of the patient?

Many systemic diseases like cancer use the 5-year survival rate as a criterion of success of the therapy. Using glaucoma therapies, we distinguish between complete success (IOP < 18 mmHg without additional drugs) and qualified success (IOP < 18 mmHg with additional drugs). Long-term results as 5 years should be the standard in publications. Six-month or 1-year results are preliminary and might be too short to judge the influence of antifibrotics (possibly causing a sweating bleb, resulting in blebitis or endophthalmitis many years after the surgery) or implanted devices to tissues of the eye.

There have been several publications showing that the implantation of glaucoma shunt devices (like Ahmed or Baerveldt) may correlate with loss of endothelial cells [1], especially in eyes with a penetrating keratoplasty [2]. The reasons are not well defined, possibly blinking and movements of the eye or of the tube or excessive rubbing are causative.

Most useful and informative publications are about negative results, which can be found in this issue of Graefe's Archives. The preliminary results of the implantation of the STARflow (iSTAR) implant were very good. After 1 year, it appeared to be safe and effective [3]. But after 2 years, the IOP was not lowered sufficiently in 28 eyes (70%) and 5 eyes (12.5%) showed signs of endothelial cell loss [4]. Quite similar, but with a delay, were the results of the CyPass implant (Alcon): not yet after 2 years, but after 5 years, some patients had less endothelial cells (1.931 cells/mm² vs 2.189 cells/mm² in control eyes) and the product was voluntarily withdrawn [5]. Both implants are placed suprachoroidally and maybe the constant pulsative action of the blood flow of the choroid lead to movements of the implants and to this loss of endothelial cells.

One could argue: we can repair endothelial cell loss elegantly by DMEK procedure. But this would be an additional surgery with an additional risk profile. Like a fiction story: you receive a new hip-prosthesis, but after a certain time, you experience pain again. The resulting constrained posture leads to an arthrosis of your knee-joint. And the doctor tells you: we can repair this easily; we will implant a new knee prosthesis. All which you can perform in eyes with problematic stents: Cutting the length of the stent or explanting it, it will always be an additional surgery with a certain risk.

Therefore, judge short-time results as preliminary, look for further information and data like adverse effects besides the reduction of the IOP, check the retinal nerve fibre thickness and the progression of the visual field and ask about the quality of life. Maybe we should prefer surgeries with treatment or removal of the pathologically changed tissue, the trabecular meshwork.

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