This relatively young patient with residual life expectancy of over 20 years, has severe high pressure POAG, confirmed by OCT and visual field, uncontrolled despite supra-maximal tolerated medical treatment (MTMT), on five medications. She is at extremely high risk of further visual loss that will severely reduce vision-related quality of life in her lifetime. The only relevant modifiable risk factor is her intra-ocular pressure (IOP) and she clearly needs lower IOP in both eyes, preferably in the single digits to low teens. While she has not undergone selective laser trabeculectomy this is very unlikely to have a sufficient effect for a patient on MTMT with low target pressures and would likely merely delay the definitive treatment - which should be surgical.

Considering her visually significant cataract combined phaco-MIGS in her left eye was an entirely reasonable but unfortunately unsuccessful first step, leading to urgent trabeculectomy that has achieved only modest success with mid-teens IOP even with three medications. This is highly likely to need further future glaucoma surgery (which would in my hands be a Paul Tube with high does MMC & intra-cameral Avastin). The failure of trabecular bypass surgery suggests that there might be post-Schlemm's canal limitations tp outflow while the limited trabeculectomy function implies a tendency to aggressive wound healing that is likely to limit success of any bleb-forming procedure, minimal or otherwise, such as trabeculectomy, or Preserflo microshunt.

A further consideration is that any stand-alone surgery which leaves her phakic will have to survive the impact of future cataract surgery.

The plethora of 'MIGS' surgeries, with or without concurrent cataract extraction, e.g., GATT, KDB, OMNI, Hydrus or iStent, would mostly still depend on functioning post-Schlemm's outflow routes. Ciliary body destructive procedures such as endo-cyclo photocoagulation or micro-pulse diode laser might achieve lower IOPs in the short-term but have limited evidence that they can provide the long-term control required here.

Traditional 'tube' surgery with posterior drainage to a reservoir via a glaucoma drainage device will bypass physiological out-flow (and thus any post-Schlemm's obstruction) and divert aqueous to a region with fewer tenons fibroblasts less prone to fibrotic capsule formation than is a trabeculectomy. Baerveldt or Ahmed tubes each have their advocates, with Baerveldt tubes likely to give slightly lower IOPs in the long-term. The newer un-valved 'PAUL' tubes have not yet been fully proven in randomised controlled trials yet already have evidence of safe and effective long-term IOP lowering and more predictable immediate day one IOPs. Avoiding such an immediate post-op IOP spike is important in a patient such as this who has significant existing nerve damage. The smaller tube diameter of the PAUL may also pose less threat of long-term corneal endothelial cell loss than larger traditional tubes and posterior drainage should be resistant to the inflammatory effects of later cataract extraction.

An alternative approach also avoiding post-Schlemm's obstructions would be to target supra-choroidal outflow routes. For example, the MINIject (iSTAR) device, inserted ab interno via a standard angle surgery approach, is a flexible porous sponge that gains access to the low-pressure supra-choroidal space without disturbing conjunctiva. The extent and

duration of IOP lowering, and the effects of concurrent or later cataract surgery, remain to be fully elucidated by randomised controlled trials.

On balance, because of the patient's previous failed surgeries, her reasonable vision, and the slightly greater risks of combining tube insertion with cataract surgery I would carry out a PAUL insertion first with later phacoemulsification. In view of her aggressive wound healing after trabeculectomy I would use 0.5mg/ml mitomycin C applied per-operatively via a sponge to the sub-tenons space for 3 minutes and intra-cameral Avastin given at the end of surgery.