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Editorials

Squeezing healthcare costs—every drop counts

Health care in the United States is in a crisis. At present, rising costs account for 13% of the gross national product making health care the third largest industry in the country, and there is no end in sight as costs spiral out of control.¹ For years we have been admonished as healthcare providers to control costs by improving productivity and flexibility, by adopting new appropriate technologies, and by using competitive market strategies to squeeze the fat out of the healthcare system. All of these innovations, it was assumed, would allow healthcare providers to continue to deliver high quality care while eventually cutting costs to the bone.² The promise of managed care as the big fix for US healthcare problems has not been realised. An analysis of managed care plan performance from 37 recently published peer reviewed studies reveals that on balance managed care has not improved the efficiency or quality of US health care and that some patients, specifically Medicare HMO enrollees with chronic health conditions, have received poor quality medical care as a result.³ By now it should be obvious to all of us that a big solution does not exist for our big problem.

It was, therefore, with great interest that I read the paper by Livingstone *et al* in this issue of the *BJO* (p 473) in which the authors propose that eye drops used on hospital wards in the British National Health Service may be used for 2 weeks instead of the 1 week mandated by the Department of Health. Adoption of this practice would lead to an annual savings to the NHS of £500 000. The specific question is whether or not eye drops become increasingly contaminated with potential ocular pathogens by extending their hospital life by 1 week. There is real cause for concern. Microbial contamination of eye drop solutions and dropper tips has been implicated as a cause of severe ocular infection in a number of patients.⁴⁻⁷ In addition, the chance of contamination is much more likely on a hospital ward than in a domiciliary setting.⁸ To answer the question, Livingstone *et al* cultured eye drop residues for bacteria and fungi from 341 samples after 7 days and from 295 samples after 14 days in the setting of a hospital ward. Not surprisingly, the incidence of microbial contamination was not statistically different between the two groups. The authors also found that the contaminating micro-organisms were similar in both groups; they were mostly associated with the skin and none of the micro-organisms isolated was highly pathogenic. In view of those interesting research findings there appears to be no laboratory

evidence to support the current practice of discarding eye drops on hospital wards after only 1 week because of the risk of contamination. As a result of this scientific study, an evidence based decision by the NHS may lead to a saving of £500 000 a year from the national healthcare budget.

This study by Livingstone *et al* should be used as a model for the evidence based rational decisions that need to be made regarding every aspect of the healthcare problem. The pressure to reduce burgeoning healthcare budgets in the United States and the UK has led to unpopular and at times irrational cost cutting measures. Healthcare “managers” with little understanding of the scientific approach to medical practice or the sanctity of the patient-physician relationship have been allowed to implement putative cost effective solutions in an attempt to solve the healthcare crisis. Since there is general agreement that this approach has not been effective, maybe it is time to try something else. The real need may be to implement many small painstaking solutions to fix the big seemingly insurmountable problem. Small evidence based solutions applied to every problem area of the healthcare system can result in cost savings and also in continued improvement in the quality of patient care. In implementing these small solutions physicians, researchers, nurses, and all healthcare workers should continue to be guided by the traditional principles: firstly, “do no harm” and secondly, “strive unceasingly to improve the quality of care”. Innovation, creativity, and dedication in solving the many small problems in the healthcare system will ultimately cure the big problem.

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Adhesion of IOLs to the posterior capsule

Posterior capsule opacification (PCO) occurs in up to 50% of patients within 5 years following extracapsular cataract extraction (ECCE) surgery.¹ Residual lens epithelial cells proliferate and migrate onto the posterior capsule where they may undergo phenotypic change to form contractile fibroblast-like cells which secrete extracellular matrix material. Massive proliferation results in multilayering of these cells on the posterior capsule and the formation of fibrotic membranes. Residual lens epithelial cells also form clusters of swollen Elschnig's pearls as a result of their failed attempts to form new fibre cells. These fibrotic membranes and/or Elschnig's pearls, if allowed to reach the visual axis, may cause significant visual loss as a result of the diffraction of light by the accumulated mass of cells and matrix.¹⁻³ In these cases, the patients once again have their vision compromised and often require a Nd:YAG laser posterior capsulotomy. This treatment is associated with the increased risk of a number of complications such as cystoid macular oedema and retinal detachment.^{1,3} Despite extensive research our understanding of the cell biology of PCO remains limited as does our ability to successfully prevent its formation. Interventions to reduce or prevent this opacification can be broadly divided into surgical and pharmacological.

Surgical approaches tried with varying success include altering the size of the anterior capsulorhexis or of the optic to determine whether the incidence of PCO reflects the location of the cut edge of the rhexis either on or off the optic.^{1-4,6} There is continuing debate about the validity of these approaches because results are so inconclusive. Attempts to remove lens cells include extensive polishing of the posterior capsule, cleaning of the anterior capsule with an ultrasound irrigating scratcher, infusion of water to induce lens cell lysis, and freezing the capsule with a cryoprobe to rupture lens cells.¹⁻³ Although the techniques used to remove residual cells have had varying degrees of success they are time consuming and not without their own ocular complications.

With our increasing understanding of cellular and molecular biology it is now possible to target the pathophysiological basis of PCO. Pharmacological agents, in particular antimitotic/antimetabolic drugs (for example, 5-fluorouracil, mitomycin C, colchicine, and daunomycin) and immunotoxins have been reported to significantly reduce the incidence of PCO.⁷ Unfortunately, these agents can cause significant ocular toxicity. More subtle approaches include the inhibition of lens cell attachment to the posterior capsule by blocking cell adhesion receptors and using the intraocular lens (IOL) itself as a drug delivery system.⁸⁻¹¹ Agents incorporated into IOLs include indomethacin, daunorubicin, thapsigargin, and the toxin, saporin, conjugated to fibroblast growth factor, all of which have been used successfully to inhibit migration/proliferation and/or induce cell death in the vicinity of the IOL. However, while these agents have shown some promise for therapeutic intervention in cell culture and animal models, their efficacy has yet to be proved in the clinic.

Perhaps an easier solution to PCO is simply to fashion the IOL so as to prevent the migration of cells into the visual axis. This can best be achieved by ensuring that the IOL is in immediate apposition to the anterior surface of

the posterior capsule; manufacturers have attempted a variety of lens optic designs (for example, biconvex or posterior convex, barrier ridge optics) to try and present a barrier to the migrating epithelial cells.^{12,13} A novel approach is to identify lens materials which will strongly adhere to the posterior lens capsule, thus obliterating any space or substrate for cell migration. Oshika and colleagues in this issue of the *BJO* (p 549) have assessed the adhesion characteristics of three intraocular lens materials both in vitro and in vivo. In vitro studies demonstrated that acrylic IOLs adhered three times more strongly to a collagen sheet than did PMMA IOLs with silicone IOLs demonstrating minimal adhesion. These observations were confirmed in vivo following IOL implantation in the rabbit. While lens cells could be observed throughout the posterior capsule in rabbits receiving PMMA and silicone IOLs, cell migration appeared to be blocked at the edge of the acrylic IOLs thus preventing cells from reaching the visual axis. The nature of the adhesive forces between the capsule and the acrylic IOLs remains to be determined. The dependency of IOL adhesion on the nature of the IOL material goes some way to explaining (a) the variable success rates of different IOLs in vivo, and (b) the reported low incidence of PCO using acrylic IOLs. Confirmation of the improved efficacy of acrylic IOLs in preventing PCO will be derived from appropriate clinical trials. In the meantime, Spalton and colleagues have reported that Acrysof lenses have an improved outcome but that this probably relates not to inhibition of migration but to regression after migration.^{14,15}

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