

## SOME COMPLICATIONS OF LENS EXTRACTION

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### SUMMARY

*The operation of lens extraction for cataract has been practised for many years. About 4% of cases develop some major complication and a few of these are reviewed, namely vitreous loss and its sequelae and management, macular oedema, the 'flat' anterior chamber, postoperative glaucoma and postoperative infection.*

*The treatment of each complication is briefly outlined.*

Surgical treatment of cataract dates back more than 3 000 years. Records of the couching operation are found in Hindu medical writings, long before the Christian era. The Roman writers, Galen and Celsus, speak of cataract surgery in the time of Herophilus, in 300 BC. They re-

ferred to the couching procedure, which was practised in Europe, until Jacques Daviel performed the first planned lens extraction on 8 April 1745. In 1753 he published accounts of 115 cases, of which 100 were successful; three years later, there were only 50 failures in his series of 434 cases.<sup>15</sup>

Compared with Daviel's failure rate of 11%, we can expect a major complication in 4% of all cases.<sup>41</sup> The possible complications of cataract surgery are innumerable. The literature on the subject is vast, though often repetitive. I have selected a few common or important complications as the theme of this article.

The ultimate failure is enucleation of the eye. Blodi has estimated that 1.3% of eyes will be lost at some stage,

following intracapsular extraction, and 2.0% after extracapsular extraction.<sup>8</sup>

#### VITREOUS COMPLICATIONS

We have all, in the words of Prof. Derrick Vail, 'seen vitreous presenting, beading, oozing, flowing and cascading out of newly opened eyes'.<sup>41</sup> The loss of formed vitreous at operation, shattering for the surgeon, is also one of the most serious mishaps for the eye. In its wake come a host of sequelae.

Six per cent of eyes which have lost vitreous at operation, will be lost within one year. Of the remainder, 18% will have no useful vision a year after operation, and 23% after 2 years. Three years after surgery only 20% of such eyes will have a visual acuity of 6/18 or better.<sup>40</sup>

#### Incidence of Vitreous Loss

The incidence of vitreous loss varies widely in reported reviews (Table I) from 17.3% down to 0.9%. A review of our cases at Groote Schuur Hospital for 1967 gave an incidence of 14.1%. Derrick Vail states that the incidence should not exceed 3%.<sup>41</sup> If one takes the reports from Table I, published after 1950, the average incidence of vitreous loss is found to be 5.7%.

TABLE I. INCIDENCE OF VITREOUS LOSS IN CATARACT EXTRACTION\*

Author	Year	Percentage loss
Daviel .. .. .	1753	11.6
De Wecker .. .. .	1869	5.4
Pagenstecher .. .. .	1881	13.7
Jullundar Smith .. .. .	1910	7-8
Vail (snr) India .. .. .	1910	7
Vail (snr) Ohio .. .. .	1910-1925	8.4
Parker .. .. .	1921	11.3
Dunphy .. .. .	1927	8.3
Elschnig .. .. .	1926-1931	3-48
Guillaumat <i>et al.</i> .. .. .	1934	16.24
Guillaumat <i>et al.</i> .. .. .	1939	5.29
Sinclair .. .. .	1939	17.3
Kirby .. .. .	1950	6.0
Little .. .. .		7.1
Vail (jnr) .. .. .	1925-1942	12.7
Vail (jnr) .. .. .	1946-1961	3.7
Knighton .. .. .	1949	0.9
Cinotti .. .. .	1952	1.57
Townes <i>et al.</i> .. .. .	1952	6.5
Heath .. .. .	1961	7.0
Awasthi .. .. .	1965	6.0

\*From Vail.<sup>41</sup>

When the incidence rises above 3-4% one should take a long, critical look at the surgery, for many of the causes of vitreous loss are avoidable. As Kipling has said:

So when the buckled girder  
Lets down the grinding span,  
The blame for loss, or murder,  
Is laid upon the man.  
Not on the stuff—the man!

#### Causes of Vitreous Loss

The causes of vitreous loss are well known and fall into three groups:

(a) *External pressure.* Taut eyelids, the retracting speculum, the surgeon's expressor and the pull of inadequately paralysed ocular muscles, may all exert an external, distorting pressure on the eye. None of these factors are unavoidable.

(b) *'Internal' pressure.* Choroidal haemorrhage, and choroidal congestion, by competing with the vitreous for the space available within the scleral sac, may exert an expulsive force from within the eye. Apart from not opening abnormally hard eyes, little can be done to avoid the catastrophe of choroidal haemorrhage. However, one can minimize choroidal congestion, which is a reflection of raised venous pressure in an open eye.

(c) *Vitreous pathology.* One cannot avoid vitreous pathology, but one can take steps to reduce the likelihood of vitreous loss in such cases.

#### Prevention of Vitreous Loss

There are certain warning signs of likely vitreous loss. Failure to take these signs seriously may lead to unnecessary difficulty. In this regard we must beware of:

- (i) the myopic eye and the prominent eye;
- (ii) the eye with previous iridocyclitis or glaucoma;
- (iii) the cataracta brunescens;
- (iv) the fellow eye of a previous 'vitreous loss eye'; and above all,
- (v) the case of the subluxed or dislocated lens.

Briefly, the precautions one may take against likely vitreous loss are:

- (a) Minimize external pressure on the eye, by the use of lid sutures rather than a speculum for retraction; by the performance of a canthotomy and the avoidance of a retrobulbar injection (this will necessitate the use of general anaesthesia).
- (b) Provide external support for the eye, by suturing a Flieringa ring to the episclera at 8 points.
- (c) Soften the eye pre-operatively by the intravenous injection of acetazolamide 500 mg and gentle, but sustained digital pressure on the globe.
- (d) Dehydrate, and so shrink the vitreous body, by the use of an osmotic diuretic, given by intravenous infusion over a period of 30 minutes, starting 1½ hours pre-operatively (urea or mannitol at the rate of 1.0-1.5 g/kg body-weight or isosorbide at the rate of 2.0 g/kg).
- (e) In an effort to reduce further the volume of the vitreous body, but at the same time retain an intact hyaloid face, some advocate the aspiration of vitreous through the pars plana, before opening the eye.<sup>36</sup>
- (f) Do a sliding extraction, without expression, for which procedure the cryo-extractor is the instrument of choice.
- (g) Use silk sutures from the outset, as catgut sutures will not hold their knots if vitreous comes forward and lubricates the thread.

#### Sequelae of Vitreous Loss

Following the loss of vitreous, over half (57.3%) of the eyes will develop vitreous opacities.<sup>40</sup> A third (34.4%) will have excessive or prolonged iridocyclitis, which often responds poorly to treatment, and aids and abets many other complications.<sup>40</sup> Secondary pupillary membranes develop in 10% of these cases.<sup>40</sup>

The frequency of aphakic glaucoma rises fourfold after vitreous loss. Incarceration of vitreous in the corneoscleral wound is a major problem, and the common feature in the pathogenesis of a number of other complications,

Among these, an undrawn pupil is the most classical sign of vitreous incarceration.

Interposition of vitreous between the wound edges may lead to imperfect healing, wound leak and cystoid scars. By acting as a tissue bridge incarcerated vitreous provides a route of access to epithelial cells, which invade the anterior chamber in 2.3% of cases.<sup>40</sup>

Touching of the back of the cornea by an intact hyaloid surface leads to interference with endothelial cell function, and raises the incidence of corneal complications such as persistent striate keratopathy, oedema and bullous keratopathy, and accelerated corneal dystrophy to 6.0%.<sup>40</sup>

After lens extraction the vitreous body always shifts forward, and some degree of posterior vitreous detachment is invariable. Adhesion of the vitreous body to the corneoscleral wound, and subsequent surface fibroblastic response and contraction, pull the vitreous yet further forward. As a result, traction on posteriorly situated structures occurs, and may account for vitreous haemorrhage, of choroidal origin in 11.3%; postextraction papilloedema; postextraction macular oedema, macular cysts and holes; and aphakic retinal tears and detachment which occurs in 7.2% of cases with vitreous traction (*cf.* 2.0% of all cases).

#### Management of Vitreous Loss

The loss of fluid vitreous—truly, not euphemistically 'fluid'—requires no special treatment, and is a less serious complication than the loss of formed vitreous.

Once vitreous is lost, it is lost. The first thing to do when this complication overtakes one, is to complete the extraction as carefully, and as calmly, as your shattered nerves will allow. The second, is to avoid the temptation to tie your sutures hurriedly, and go and recover over a cup of tea and a cigarette! Meticulous attention must be given to the wound closure, and in particular to the freeing from it of all vitreous. It is the vitreous left in, or adherent to the wound, which is going to cause trouble—not that vitreous which has been lost.

The extension of a peripheral iridectomy into a sector iridectomy, perhaps combined with an inferior sphincterotomy, will avoid the complication of an updrawn pupil. However, it will not achieve the vital objective of freeing the vitreous from the wound. In fact, it may make it more difficult to do so, by removing a potential barrier from between the vitreous and the corneoscleral wound. More definitive surgery is required if useful vision is to be salvaged from a good percentage of these 'vitreous loss' eyes. There are a few alternative approaches to the problem, which will now be outlined.

(a) *Wound closure first.* The pre-placed sutures may be tied immediately, and steps then taken to clear the incarcerated vitreous from the wound. It is essential to use sufficient wound sutures to ensure perfect wound closure. Having closed the wound, externally prolapsed vitreous is picked up by allowing it to adhere to a piece of dry surgical sponge, and cut off flush with the wound edge.<sup>41</sup>

A curved iris retractor or spatula is then introduced horizontally, across the anterior chamber. The point should follow the angle closely, and be kept well back so as to pass behind the incarcerated vitreous band, and between it and the iris. The blade is then swept downwards, across the iris and pupil, breaking the vitreous strand free from the wound. This procedure is made easier

if the anterior chamber is temporarily overfilled with air. This has two advantageous effects: the vitreous band is placed slightly on stretch, and is rendered more visible. Two or three attempts may be required to completely free all vitreous from the wound—but freed it must be. Success will be indicated by the resumption of a round shape and central position by the pupil.<sup>40</sup>

Once the vitreous has been freed a miotic should be instilled. Pilocarpine or eserine<sup>30</sup> or acetylcholine<sup>30</sup> may be chosen. Following the loss of vitreous, air should always be used to reform the anterior chamber.

(b) *Handling of the prolapsed vitreous before wound closure.* Alternatively we may decide to clear the anterior chamber and wound of vitreous, before tying the sutures.

If a Flieringa ring has already been sutured to the episclera, gentle forward lift upon it may cause the vitreous to plop back into the eye. This seldom happens, but is worth a try. Failing this—and it usually does fail—one is faced with the task of removing vitreous from the forbidden area of the anterior chamber and wound. There are two basic approaches to this task: one can either cut vitreous off the front, or suck it out from the back!

Cutting off the anterior, prolapsed vitreous necessitates a big incision, and an open-sky technique. Using a dry surgical sponge, the vitreous is systematically and repeatedly picked up, and cut off with scissors. This must be persisted with until all vitreous in front of the iris has been removed.

The alternative is to aspirate vitreous.<sup>30</sup> A large-bore needle, marked at 10 mm and 15 mm is passed backwards into the vitreous body. The marked needle enables one to judge with confidence the depth to which the needle has entered, which should be between 10 mm and 15 mm. By slow suction 1 m.-2 ml of vitreous is aspirated, upon which the anterior, prolapsed vitreous retracts into the eye.

Both these procedures appear drastic. However, it cannot be overemphasized that it is not the vitreous which is lost which is so important. It is the vitreous which becomes fixed to the corneoscleral wound which leads to so much subsequent trouble.

#### Postoperative Vitreous Herniation—and Postoperative Vitreous Face (Hyaloid) Rupture

Some measure of vitreous herniation through the pupil and iridectomy is usual after lens extraction. This bulge of vitreous may become large, and reach the cornea. If the hyaloid face is intact, it will interfere with endothelial cell function, and overlying corneal oedema will result. Should the vitreous come into contact with the corneoscleral wound postoperatively, it may adhere to it. This stickiness of the wound may persist for up to 3 months after operation. The occurrence of postoperative adhesion of the vitreous to the wound is usually insidious but may lead to symptoms of photophobia and lacrimation, with signs of a mild iritis. Corneal oedema and glaucoma may follow.

Spontaneous rupture of the hyaloid may occur at any time postoperatively. It happens after 33% of intracapsular extractions, usually in the first 6 postoperative weeks.<sup>38</sup> A small rupture is symptomless, but if it occurs suddenly, with vitreous spilling into the anterior chamber, a considerable amount of pain, with evidence of an iridocyclitis,

may result.<sup>20</sup>

Adhesions of vitreous to the wound in the postoperative period may cause persistent symptoms of a mild iridocyclitis for many months. Peaking of the pupil and the other sequelae of vitreous traction may all follow.

#### *Later Management of Vitreous Incarceration*

All cases of vitreous incarceration should be corrected as soon as possible. Little has shown that 21% of cases with vitreous incarceration develop macular lesions within 3 years of operation.<sup>25</sup> By comparison, only 5.0-7.3% of eyes which have lost vitreous, but which do not have vitreous stuck in the wound, show the same changes.

#### MACULAR OEDEMA

The development of macular oedema following an apparently perfect lens extraction is a great disappointment and occurs in about 2.0% of all cases. Following vitreous loss, and particularly if associated with vitreous wound adhesions, the incidence rises considerably.

Fine adhesions have been shown to exist between the vitreous and the retina, in the macular region, by Grignolo.<sup>26</sup> Traction upon these may well be the cause of macular oedema, macular cyst and hole formation.<sup>20,21,28</sup> However these vitreoretinal connections are not easily demonstrated, and others have questioned their existence and their significance, suggesting that hypotony and inflammatory changes are more likely causative factors.<sup>22</sup>

The leakage of oedema fluid into the macular region is well shown on fluorescein angiography.<sup>31</sup> This complication of macular oedema usually occurs within the first 6 months after operation, and is quite often preceded by a spontaneous rupture of the hyaloid face, which may otherwise have passed unnoticed.<sup>24,32,43</sup>

Treatment is unsatisfactory and the visual prognosis poor, though variable. Some resolve spontaneously. Maumane claims good results with oral prednisone in doses of 100 mg/day.<sup>31</sup> However Gartner, and others, are not much impressed with the results of steroid therapy.<sup>23</sup>

#### THE FLAT ANTERIOR CHAMBER

The anterior chamber, if not surgically reformed at the completion of the extraction, does so spontaneously in 4-8 hours. It should always have reformed by the time of the first dressing at 24 hours.

Many reform the anterior chamber on the table as a matter of routine. If this is done, air should not be used as a general rule. However, air should be used following vitreous loss, in cases of corneal dystrophy and where previous keratoplasty has been performed. Barraquer considers a sector iridectomy to be an indication for air, so as to try and separate the vitreous and wound.<sup>2</sup> If the eye is very soft, saline should be used to fill it up, and only sufficient air introduced to reform the anterior chamber, leaving a small meniscus of fluid all round the periphery of the bubble.

#### *Delayed Reformation of the Anterior Chamber*

Delay in the reformation of the anterior chamber is due to wound leak. The leak may be obvious, and associated with other features of a gaping wound. In other cases it may not be easily detected, particularly if a limbus-based flap has been used. These eyes are always soft, and may

show features of hypotony, such as retinal and choroidal oedema, and choroidal detachment.

*Treatment.* If the wound leak is apparent it should be repaired forthwith, by the placement of additional sutures. If no leak is seen, the eye should be padded for 48 hours. By the end of the third day many eyes will have reformed their anterior chambers, and the dilemma will have been solved.

If the anterior chamber is still flat, and a cause has become apparent, it should be dealt with surgically. If the cause remains hidden, the pupil should be dilated with phenylephrine and cyclopentolate, the eye padded and bandaged, and oral dosage with acetazolamide 250 mg *q.i.d.* commenced. If the anterior chamber is still flat 5 days after operation active intervention is essential to avoid permanent anterior synechiae and possible secondary glaucoma.

The wound should be explored surgically. Any badly placed or excessively deep sutures should be removed, and any wound gaps closed by additional sutures. A cyclodialysis track is then formed, and through it a cannula is passed into the anterior chamber permitting air to be injected. An existing choroidal detachment should be drained simultaneously.

If the anterior chamber is allowed to remain flat for periods in excess of 5 days, the incidence of aphakic glaucoma shows an alarming increase. After 8 'flat' days the incidence is 12% and after 10-12 'flat' days it is 44%!<sup>28</sup>

#### *Late Loss or Shallowing of the Anterior Chamber*

Late loss or shallowing of the anterior chamber, subsequent to its initial reformation, may be due to wound leak, or to pupil block; it may occur suddenly or gradually.

1. Sudden loss of the anterior chamber, 5-10 days after extraction often accompanied by some pain and hyphaema, is due to wound disruption, and consequent aqueous leak.<sup>17,38</sup> The leak may be intermittent, and is thus not excluded by a negative fluorescein test.<sup>23,17</sup> Unless the wound disruption is severe, these cases are best treated conservatively by double padding and bed rest for 24-48 hours.

2. Gradual shallowing or loss of the anterior chamber in the later stages of healing is not due to wound leak, but to a developing pupil block.<sup>14,18,33-35,42</sup> As a result of the impedance of forward aqueous flow a pressure gradient develops across the hyaloideo-iris barrier, thrusting it forward and so reducing the depth of the anterior chamber. For this block to occur the pupil and iridectomy must be applied, perhaps even lightly adherent, to a relatively impervious vitreous face. Postoperative iritis makes iris-hyaloid adhesions more likely: it also alters in some way, perhaps by surface fibroblastic proliferation, the permeability of the vitreous to aqueous.<sup>34</sup>

Initially the intra-ocular tension in these eyes is low or normal, but as the iris is pushed forward, the drainage angle becomes occluded, and glaucoma develops. It is important to recognize, and treat, these cases of pupil block, before the pressure begins to rise.

Occasionally air, used to reform the anterior chamber, gets behind the iris and pushes it forward. To make this air come forward is sometimes a simple matter of positioning of the patient. At other times it may prove impossible to get the air to return to the anterior chamber.

In such circumstances one may try turning the patient prone, upon which the air tends to 'float' towards the posterior pole of the eye, allowing the anterior chamber to deepen.<sup>6</sup> This is particularly useful in cases in which an air bubble is causing pupil block.

#### POSTEXTRACTION GLAUCOMA

Glaucoma develops after from 2% to 7% of all cases of lens extraction, usually within 3 months of operation. Glaucoma developing many months, or a year later, is probably a previously unrecognized glaucoma simplex.

A transient rise of pressure is quite normal following lens extraction, one-quarter of cases showing a quite appreciable rise. The use of alpha-chymotrypsin increases the frequency and height of this postoperative ocular hypertension. The incidence rises from 23.6% to 72.5%, and the tension may reach 50 mmHg.<sup>1,4,27</sup>

The peak of this rise is around the second to fifth day; the pressure is often well on its way down by the end of the first week, and is always back to normal at the end of 3 weeks.

Histological evidence of swelling and degeneration of endothelial cells has been shown.<sup>19</sup> All the side-effects of alpha-chymotrypsin are dose related, and are very much less frequent if a 1/10 000 dilution is used.

Aphakic glaucoma falls into two broad groups:

(a) Firstly, there are those with a shallow anterior chamber, extensive peripheral, anterior synechiae and angle closure. Pupil block plays an important role in the pathogenesis of this group.

(b) Secondly, there is a deep-chambered group, unassociated with pupil block, but in which the trabecular drainage system has become clogged. The causes of this trabecular clogging are many, and include: inflammatory cells and debris, blood cells and fibrin, lens material and phagocytes, iris pigment, vitreous gel, and ingrowing epithelial cells. All these, and probably other factors, may serve to block the trabecular meshwork, and so impede aqueous outflow.

#### Management of Aphakic Glaucoma

The management of the two types of aphakic glaucoma is somewhat different.

(a) *Treatment of pupillary block glaucoma.* In pupillary block glaucoma, with its shallow anterior chamber and angle closure, the approach is largely a mechanical one. If diagnosed early, the energetic use of phenylephrine 10% and cyclopentolate drops, or the subconjunctival injection of Mydracaine, may break the pupil block. If this treatment succeeds in breaking the cycle of pupil block and shallowing of the anterior chamber, it will do so quickly, and the chamber will be seen to have deepened. One may then observe the patient, to see if sufficient of the angle is undamaged by synechiae to permit adequate aqueous drainage. If mydriasis fails to break the pupil block, one must not procrastinate. A new passage for the aqueous from posterior to anterior chamber must be made, and the sooner the better.

There are two possible approaches:

1. Iridotomy or iridectomy: one may make a new hole in the iris—by simple transfixion iridotomy, or more preferably by formal basal iridectomy. This procedure will correct the pupil block in a large proportion of these cases,

but not in all. Following severe iritis, and very particularly after an endophthalmitis, the iris becomes widely adherent to the hyaloid face, which in turn becomes altered, and impervious to aqueous.<sup>27</sup>

2. Vitreous discision: in the cases which do not respond to iridectomy (by deepening of the anterior chamber) one should proceed immediately (under the same anaesthetic) to vitreous discision. This creates a new track for the aqueous, forwards into the anterior chamber.<sup>27</sup> Vitreous discision is an atraumatic procedure, simpler to perform than iridectomy, and is perhaps the better primary procedure, rather than limiting it to those cases which fail to respond to an iridectomy.

Delay in the relief of pupil block angle-closure will lead to extensive adhesions in the angle. In such cases a further procedure may well become necessary to control the intra-ocular pressure. Most authorities regard cyclodialysis as the procedure of choice in this type of case. At the conclusion of the dialysis, the anterior chamber should be reformed with air, introduced through the dialysis track.<sup>28</sup>

(b) *Treatment of deep-chamber aphakic glaucoma.* The treatment of deep-chamber aphakic glaucoma should be medical initially. The powerful anticholinesterase inhibitors such as phospholine iodide tend to be more effective in these cases than either pilocarpine or eserine, and are better tolerated in the aphakic eye than they are in the phakic eye. Acetazolamide may have to be used to supplement the effect of the miotics.

Concomitant iridocyclitis should be treated energetically with topical corticosteroids. If it is considered necessary to dilate the pupil periodically, so as to avoid posterior synechiae, reliance should be placed on phenylephrine 10% and the powerful, long-acting cycloplegics should be avoided.

Failure of medical therapy to adequately control the glaucoma becomes an indication for surgery. We have been using cyclodiathermy in these cases; the results have been rather disappointing, as is the case with so much of glaucoma surgery. Most writers in this field state that cyclodialysis is the procedure of choice in aphakic glaucoma.

#### POSTOPERATIVE INFECTION

The occurrence of an intra-ocular infection following a lens extraction is a catastrophe for the patient, and a bitter experience for the surgeon.

#### Incidence of Postoperative Infection

The eye has always shown a degree of resistance to infection greater than that of most other organs. Even in the days before aseptic surgery the incidence of post-operative endophthalmitis was only about 10% (Table II). With modern aseptic surgical techniques, no more than

TABLE II. INCIDENCE OF INFECTIVE OPHTHALMITIS FOLLOWING CATARACT EXTRACTION

Author	Period	No. of cases	Incidence
Berens and Bogart <sup>7</sup>	1921-1938	6 137	0.75%
Callahan <sup>21</sup>	1921-1952	18 563	0.5%
Burns <sup>9</sup>	1956-1958	2 695	0.19%
Locatcher-Khorazo & Gutierrez <sup>22</sup>	1945-1955	7 666	0.077%
Allen <sup>2</sup>	1950-1956	9 651	0.12%
Allen <sup>2</sup>	1950-1964	20 000	0.11%

about 3 cases of infective endophthalmitis should be expected in every thousand cases. However, one has only to have one bad day, when the irrigating solutions become contaminated, for the statistics to be completely altered.

**Bacteriology of Postoperative Endophthalmitis**

*Staphylococcus aureus* is the most frequent offender, and usually derives from the patient's own skin, hair follicles or nose, or from the surgical team. *Pseudomonas aeruginosa* comes in second place, and commonly reaches the eye from some eyedrop or irrigating solution.

Many other organisms have been incriminated from time to time, and some of the more frequent offenders are as follows:<sup>35</sup>

Gram-positive—*Staph. aureus*, *Staph. albus*, *Diplococcus pneumoniae* (pneumococcus), *Strep. haemolyticus*, *Strep. viridans*, *B. subtilis*, *B. megatherium*, *Clostridium perfringens* (*B. welchii*); Gram-negative—*Ps. aeruginosa* (*B. pyocyaneus*), *Proteus vulgaris* (*B. proteus*), *Klebsiella pneumoniae* (*B. friedlanderii*), *Escherichia coli* (*B. coli*), *Aerobacter aerogenes*, other coliform bacteria and *Neisseria catarrhalis*.

**Mycology of Postoperative Endophthalmitis**

In recent years fungi have assumed an increasing role in postoperative infection. The list of causative fungi shown in Table III is not an exhaustive list, but serves to illustrate the variety of fungi which may be responsible. However, the point of great interest is that these fungi

TABLE III. CAUSATIVE FUNGI IN 17 CASES OF FUNGAL ENDOPHTHALMITIS\*

	No. of cases
Cephalosporium sp. . . . .	7
Volutella sp. . . . .	4
Neurospora citophila . . . . .	1
Hormodendrum . . . . .	1
Hyalosporus . . . . .	1
Cephalosporium or penicillium . . . . .	1
Cephalosporium or hyphas . . . . .	1
Hyalopus bogolepofi . . . . .	1

\*From Theodore.<sup>35</sup>

are not generally regarded as pathogens. Some alteration in host susceptibility is thus of particular importance in the development of mycotic endophthalmitis. The use of corticosteroids is one important factor. Certain antibiotics may also encourage fungal growth, most notably streptomycin, the tetracyclines and neomycin.<sup>35</sup>

Traumatic surgery and prolonged fiddling increase the chances of infection. A mixture of lens material and vitreous appears to provide a superlative culture medium.

**Clinical Presentation of Postoperative Endophthalmitis**

Bacterial endophthalmitis usually manifests in the first 48 postoperative hours. If *Pseudomonas aeruginosa* is the culprit, things move quickly, but *Staphylococcus aureus* may be held back by topical antibiotics, and so take 4 or 5 days to show its hand. There is considerable chemosis, pain and an intense iritis, with a hazy cornea; the vision usually falls off rapidly.

Mycotic infections, on the other hand, never manifest early; usually they become apparent in the second, third or fourth week after operation. Occasionally they may take months to declare themselves.

The differential diagnosis rests between infection—

bacterial or mycotic—and iridocyclitis, usually with retained lens products. The chief clinical features are summarized in Table IV.

TABLE IV. CHIEF CLINICAL FEATURES OF ENDOPHTHALMITIS AND IRIDOCYCLITIS

	Bacterial endophthalmitis	Mycotic endophthalmitis	Lens induced iridocyclitis
Time of onset	Early: 24 - 48 hrs (up to 1 week)	Late: 8 - 14 days (up to 3 months)	Intermediate: 6 - 12 days
Tempo	Rapid—usually (slowed by antibiotics)	Slow—usually (speeded by steroids and antibiotics)	Variable—may be rapid in 'sensitized' patient
Pain and redness	+++	+	±
Chemosis	+++	+	±
Cornea	Hazy	Clear	Clear KP
Anterior chamber	Flare ++ Cells ++ Hypopyon Exudate	Flare + Cells + Hypopyon transient Exudate, usually inferior	Flare + Cells + No hypopyon No exudate, but fluffy lens material
Vitreous	Cells ++	'Cottonwool' satellite deposits	Lens material in front of vitreous
Membrane formation	++	+++	+
Vision	Often loose PL	Retain PL	

KP = keratic precipitates; PL = perception of light.

**Treatment of Infective Endophthalmitis**

Rational treatment is based on definitive diagnosis. This is often not made in these cases, and certainly cannot be waited for. However, every effort must be made to identify the particular intruder. Swabs should be taken from the lids and conjunctiva.<sup>35</sup> Theodore is very definitely in favour of paracentesis of the anterior chamber, to permit aqueous examination and culture.<sup>35</sup> He makes a small stab incision, almost through the cornea, a little inside the limbus, and then uses a thin hypodermic needle on a syringe to pierce the remaining cornea, and enter the anterior chamber. The aqueous sample should then be placed directly onto bacterial and fungal culture media, and a smear made for Gram's stain and microscopic examination.

Energetic treatment must begin immediately a bacterial endophthalmitis is suspected. One is faced with such a plethora of antibiotics, that no single regimen can be said to be the best. Our current policy is given below, and has yielded good results as far as cure of the infection and retention of the globe are concerned. Sight is seldom preserved in this tragic major complication.

Penicillin remains the antibiotic of choice for susceptible Gram-positive organisms. We use large doses of sodium penicillin, given by continuous intravenous infusion. Between 20 million and 40 million units are given per day.

While recognizing the dangers of chloramphenicol, we use it because it is effective against most Gram-positive and Gram-negative bacteria, and enters the eye better than most other antibiotics. A dose of 2.0 g (up to 3.0 g in very severe cases) is given daily for not longer than 7 days.

At the same time as commencing systemic antibiotics, a subconjunctival injection of Soframycin 500 mg is given, and repeated daily for 3 days.

If the response to treatment is poor, or if a penicillinase-producing *Staphylococcus aureus* is identified, then Fucidin should be added to the regimen. Fucidin has been shown to penetrate the eye in therapeutic concentration and is

effective against all strains of *Staphylococcus aureus in vivo*.<sup>2</sup> Its activity is not inhibited by the presence of blood or pus. A loading dose of 1.0 g is given and followed by 500 mg every 8 hours.

Penicillinase-producing staphylococci should alternatively be attacked with methicillin or cloxacillin, given in doses of 12.0 g per day, intramuscularly or intravenously.

Suspicion of pseudomonas infection, often one of the most fulminating types, should be handled by the subconjunctival injection of 15 - 30 mg of Colimycin (colistine).

Therapy of mycotic infections is a much more difficult matter. The diagnosis is often made late, by which time extensive invasion of the ocular tissues has occurred. The only antifungal agent of any value in these cases, amphotericin B, penetrates the eye poorly, and has problems of local irritation and severe systemic toxicity. In fact, so toxic is amphotericin B that it should not be used on the off-chance, but only on very clear-cut indications. A close watch must be kept on renal function.

#### *Use of Corticosteroids in Infective Endophthalmitis*

The use of steroids in infective endophthalmitis has been much debated. Given early, they may well exacerbate the infection, particularly if *Pseudomonas aeruginosa* or the fungi are to blame. Once there is definite evidence that the infection is under control, the use of steroids may reduce the degree of intra-ocular inflammatory response, and thus improve slightly the usually hopeless visual prognosis.

#### REFERENCES

1. Acers, T. E. (1965): Trans. Amer. Acad. Ophthal. Otolaryng., **69**, 1022.  
2. Allen, H. F. (1959): Trans. Amer. Ophthal. Soc., **57**, 377.

3. Allen, H. F. and Mangiaricini, A. B. (1964): Arch. Ophthal., **72**, 454.  
4. Barraquer, J. (1962): *La Extraccion Intracapsular del Cristalino*. Madrid.  
5. *Idem* (1968): *Proceedings of the First South African International Ophthalmological Symposium*, p. 227. Durban: Butterworth.  
6. *Idem* (1968): *Ibid.*, p. 251.  
7. Berens, C. and Bogart, D. (1938): Amer. J. Surg., **42**, 39.  
8. Blodi, F. C. (1965): Int. Ophthal. Clin., **5**, 257.  
9. Burns, R. P. (1959): Amer. J. Ophthal., **48**, 519.  
10. Castroviejo, R. (1959): *Ibid.*, **48**, 397.  
11. Callahan, A. (1953): Arch. Ophthal., **49**, 212.  
12. Chadwick, A. J. and Jackson, B. (1969): Brit. J. Ophthal., **53**, 26.  
13. Chandler, P. A. (1954): Trans. Amer. Acad. Ophthal. Otolaryng., **58**, 382.  
14. *Idem* (1961): *Ibid.*, **59**, 96.  
15. Duke-Elders, S. (1969): *System of Ophthalmology*, vol. XI, p. 251. London: Henry Kimpton.  
16. *Idem* (1969): *Ibid.*, p. 721.  
17. Dunnington, J. H. (1956): Arch. Ophthal., **56**, 639.  
18. Etienne, R. (1967): Ann. Oculist. (Paris), **200**, 729.  
19. Fanta, H. (1963): Klin. Mbl. Augenheilk., **142**, 1011.  
20. Gärtner, J. (1962): *Ibid.*, **141**, 481.  
21. *Idem* (1963): *Ibid.*, **142**, 769.  
22. Gass, J. D. and Norton, E. W. D. (1966): Trans. Amer. Ophthal. Soc., **64**, 232.  
23. Grignolo, A. (1952): Arch. Ophthal., **47**, 760.  
24. Irvine, S. R. (1953): Amer. J. Ophthal., **36**, 599.  
25. Jaffe, N. S. and Light, D. S. (1966): Arch. Ophthal., **76**, 54.  
26. Kirsch, R. E. and Steinman, W. (1954): Amer. J. Ophthal., **37**, 657.  
27. Kirsch, R. E. (1964): Arch. Ophthal., **72**, 612.  
28. Kronfeld, P. C. in Fasanella, R. M., ed. (1957): *Management of Complications in Eye Surgery*, p. 194. Philadelphia: W. B. Saunders  
29. Locatcher-Khoraz, D. and Gutierrez, E. (1956): Amer. J. Ophthal., **41**, 981.  
30. Maumanee, A. E. (1959): Highlights in Ophthalmology, **3**, 276.  
31. *Idem* (1968): Trans. Ophthal. Soc. U.K., **88**, 529.  
32. Nicholls, J. V. V. (1956): Arch. Ophthal., **55**, 595.  
33. Reese, A. B. (1949): Amer. J. Ophthal., **32**, 933.  
34. Shaffer, R. N. (1954): Trans. Amer. Acad. Ophthal. Otolaryng., **58**, 217.  
35. Swan, K. C. (1957): Arch. Ophthal., **58**, 126.  
36. Swan, K. C. and Campbell, L. (1964): *Ibid.*, **71**, 43.  
37. Theodore, F. H. (1963): Eye, Ear, Nose, Thr. Monthly, **42**, 60.  
38. *Idem* (1965): *Complications after Cataract Surgery*. Boston: Little, Brown & Co.  
39. Tolentino, F. I. and Schepens, C. L. (1965): Arch. Ophthal., **74**, 781.  
40. Vail, D. T. (1965): Amer. J. Ophthal., **59**, 573.  
41. *Idem* (1968): *Proceedings of the First South African International Ophthalmological Symposium*, p. 275. Durban: Butterworth.  
42. Villaseca, A. (1954): Arch. Ophthal., **52**, 25.  
43. Welch, R. B. and Cooper, J. C. (1958): *Ibid.*, **59**, 665.