Combined posterior contusion and penetrating injury in the pig eye. I. A natural history study

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SUMMARY We produced a standard experimental posterior contusion injury in the pig eye which resulted in immediate intraocular haemorrhage and retinal whitening and folding without the formation of a retinal break. When combined with a standard posterior penetrating eye injury, traction retinal detachment began to develop after 2 weeks. We recognised a period of transition between the resolution of the acute effects of contusion and the onset of fibrocellular proliferation resulting from ocular penetration.

The damage from a penetrating eye injury results from a combination of the effects of contusion and laceration of ocular tissues. Posterior penetrating injuries with a major contusive component may result in far greater and more extensive damage than those with laceration alone.1 These injuries may be complicated by an indirect scleral rupture or by expulsive choroidal haemorrhage, with extrusion of intraocular contents.2 Similarly haemorrhage, said to originate from engorged choroidal vessels,3 may occur during early vitrectomy performed on severely injured eyes4-6 with little indication preoperatively of the impending haemorrhage. Clinical impressions, however, suggest that intraoperative haemorrhage is less likely to occur if vitrectomy is delayed for several days or weeks after a severe injury.

Previously developed experimental models of posterior penetrating injury have reproducibly resulted in vitreous traction and traction retinal detachment.7-9 However, in these models oculer penetration was achieved with a surgical type incision only, and thus no contusive element, no choroidal haemorrhage, and no intraoperative haemorrhage from the uveal tract were encountered during subsequent vitrectomies.9,10

In an attempt to achieve a greater degree of clinical relevance and the better to evaluate the timing of vitrectomy after severe eye injury we decided to investigate the role of contusion in posterior penetrating injuries. The purpose of this study was to record the natural history following a combined posterior contusion and penetrating injury to the pig eye.

Material and methods

The monkey eye was not considered suitable for the purposes of this study because its sclera is very thin11 and the impact necessary for significant ocular contusion would have resulted in scleral rupture. We selected the pig eye because its sclera is considerably thicker.12 The anatomy of the pig eye, including its vitreous,13 has been said to resemble that of the human eye in many respects, and the pig eye has been the subject of a number of studies of anatomical14-17 and functional18 changes following ocular contusion. The major disadvantage of the pig as an experimental animal is its rapid growth in size over the course of a project lasting several months.

DEVELOPMENT OF STANDARD CONTUSION INJURY

To produce a contusion injury we used lead pellets shot from a 0·22 calibre single shot pump pistol (Model 132, Benjamin High Compression Air Pistol, St Louis). The missile velocity (v) was measured on a specially constructed bench connected to an electronic counter (Hewlett Packard 5228) and the mass (m) of the missile determined by removing small slices from the tip of a 0·22 calibre magnum lead pellet. The measurements were done at the Department of Civil Engineering, University of Southern California.

By our definition the standard posterior contusion
Injury is an injury severe enough to produce intraocular haemorrhage but insufficient to result in retinal dialysis. The optimal impact energy required to produce such an injury was determined empirically both in 50 enucleated and in 20 live pig eyes. To produce a standard posterior contusion injury as described above an impact energy of 0-24 ft-lb (0-32 joule) was found satisfactory.

Thirty-three male and female domestic pigs (Sus scrofa), aged 6–10 weeks and weighing 25 to 60 pounds (11–27 kg), were premedicated with a mixture of ketamine, acepromazine, and atropine, followed 10 minutes later by an injection of fentanyl/droperidol (Innovar-Vet). Each animal was anaesthetised by a mixture of halothane, nitrous oxide, and oxygen administered via a nose cone. The pupil of the right eye was dilated with drops of 1% atropine sulphate, 10% phenylephrine, and 1% tropicamide. A retrobulbar injection of 4 ml of 2% lignocaine was given to supplement anaesthesia and to prevent retraction of the eye into the orbit. All surgery was performed in sterile conditions under an operating microscope.

A conjunctival peritomy was made temporally from 7 to 11 o’clock and a fixation suture was placed under the lateral rectus muscle. A standard contusion injury was produced with a modified lead pellet weighing 0-57 g travelling at a velocity of 33-3 m/s. (impact energy, Ke=0-32 joule) from a hand-held pistol. The pistol was held perpendicular to the sclera at a distance of 2-5 cm. The limbal area and the ciliary body including the pars plana were thus contused. After contusion the impact site was inspected by indirect ophthalmoscopy and scleral indentation to rule out retinal breaks.

We then performed a standard posterior penetrating injury consisting of an incision 8 mm long (from 8 to 10 o’clock) parallel to and positioned 2.5 mm from the corneoscleral limbus. The incision through the pars plana avoided the lens and the peripheral retina. The vitreous gel prolapsed and was abscissed after gentle pressure on the globe. At times difficulties were experienced in prolapsing vitreous because the vitreous cortex of a young pig is more solid than that of rabbits, monkeys, or humans. After the removal of 0·3 to 0·4 ml of vitreous, the wound was closed with 8–0 nylon sutures, with particular attention paid to good wound apposition. After wound closure the fundus, peripheral retina and area of the wound were examined with the indirect ophthalmoscope and by scleral indentation to confirm that no retinal damage had occurred. Anterior chamber paracentesis was then performed. In some eyes 0·5 ml of autologous blood, drawn immediately beforehand, was injected via a 25 gauge needle inserted under ophthalmoscopic control through the wound into the mid vitreous. In other eyes 0·5 ml of balanced salt solution was injected. The blood or balanced salt solution (BSS) was injected slowly under low pressure in order to prevent reflux through the wound. The conjunctiva was closed with interrupted 7–0 Vicryl sutures, and a subconjunctival injection of 20 mg of gentamicin was given.

All animals were followed up at weekly intervals with indirect ophthalmoscopy and B-scan ultrasound for up to 4 months after surgery. Findings were recorded by drawings, descriptions, and/or photographs. Two animals died of unrelated causes during follow-up.

In order to study the various components of a combined posterior contusion and penetrating injury the animals were divided into 3 groups (Table 1).

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of eyes</th>
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<tr>
<td>I</td>
<td>10</td>
</tr>
<tr>
<td>II</td>
<td>5</td>
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<tr>
<td>III</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>38</td>
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Ten eyes were subjected to a standard posterior contusion injury without penetration; 5 of these eyes were enucleated immediately after the injury and the other 5 were enucleated at 4 months (group I). In 10 other eyes a standard posterior penetrating injury without contusion was performed; 5 of these eyes had an intravitreal injection of autologous blood and 5 received an intravitreal injection of balanced salt solution (group II). All of these eyes were followed up for 4 months before enucleation.

Group III contained 18 eyes that underwent a combined posterior contusion and penetrating injury. Fifteen of these received intravitreal injection of blood and 3 control eyes received intravitreal injection of BSS. All eyes in this group were enucleated for histology at intervals between one day and 4 months after injury.

Immediately after enucleation all the eyes were fixed and examined carefully with the dissecting microscope and by routine light microscopy.

Results

**Group I: Standard posterior contusion injury**

Anterior segment. Less than one second after contusion the bulbar conjunctiva became intensely hyperaemic. The pupil constricted by 1 to 2 mm, and occasionally a small hyphaema developed within the
Combined posterior contusion and penetrating injury in the pig eye. I. A natural history study

Table 2  Retinal detachment following combined posterior injury

<table>
<thead>
<tr>
<th>Weeks</th>
<th>Anterior circumferential folds</th>
<th>Posterior radial folds</th>
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<tbody>
<tr>
<td></td>
<td>With blood</td>
<td>With BSS</td>
</tr>
<tr>
<td>1</td>
<td>++++</td>
<td>+++</td>
</tr>
<tr>
<td>1½</td>
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<tr>
<td>2</td>
<td>+</td>
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<td>5</td>
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<td>6</td>
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<td>10</td>
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<td>14</td>
<td>+*</td>
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</tr>
<tr>
<td>16</td>
<td>+*</td>
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</tbody>
</table>

*Forward roll of ora.
†Subtotal roll of ora.

next 5 minutes; this, however, resolved within the first week. Lens changes were not observed in any of the eyes studied.

A corneal epithelial abrasion of the temporal limbal area resulted from the injury. This was slow to heal and was frequently associated with prolonged stromal oedema that usually cleared within 3 weeks.

Vitreous. A small vitreous haemorrhage overlying the ora serrata was observed immediately after contusion; this slowly increased during the next 5 minutes. Over the following 2 weeks the blood became diffuse; it was resorbed a week later leaving clear vitreous with no membrane or band formation. Posterior vitreous detachment did not occur.

Retina. Immediately after contusion there was widespread whitening and wrinkling of the retina posterior to the impact site. This increased in intensity and extent over the next 5 minutes, often extending posteriorly to the equator. At the same time circumferential haemorrhagic retinal folds concentric with the injury site developed in the same area. These gradually decreased in height and disappeared during the second week of follow-up (Table 2).

An extensive area of pigment epithelial change was evident during the following week, with atrophy and clumping extending as far as the equator; this was particularly pronounced in the area where the retinal folds had been initially noted. Further anteriorly, pigment epithelial atrophy of the pars plana developed in the immediate vicinity of the impact site. The posterior retina appeared normal throughout the duration of the study.

GROUP II: STANDARD POSTERIOR PENETRATING INJURY

Anterior segment. After posterior penetrating injury without contusion there was mild conjunctival hyperaemia, but the anterior segment became quiet one week after the injury. The cornea and the lens remained clear.

Vitreous. In the eyes with blood injection the view of the posterior segment was obscured for 3 to 4 weeks. During this time the blood clot was at first situated in the anterior vitreous cavity but subsequently became dispersed through the vitreous gel. The clot was absorbed after 4 weeks, and, although the vitreous remained hazy, posterior vitreous separation was not evident. In 3 eyes transvitreal strands spanned the distance between the posterior retina and the site of the penetrating injury.

Retina. The retina became visible after 3 to 4 weeks; at that time, retinal folds were observed in the posterior pole in all but one of the eyes. These folds radiated from the disc towards the equator and remained present throughout the follow-up period, becoming less prominent in the later stages of the study. Extensive retinal detachment did not occur.

No visible vitreous or retinal abnormalities were observed in the eyes which were subjected to a standard posterior penetrating injury plus an intravitreal injection of BSS. A rim of pigment epithelial atrophy and a minimal amount of fibrous proliferation developed around the site of the penetrating injury.

GROUP III: COMBINED STANDARD POSTERIOR CONTUSION AND PENETRATING INJURY

Anterior segment. The appearance of the anterior segment after injury was identical to that described in the eyes in group I.

Vitreous. A small vitreous haemorrhage developed immediately posterior to the contusion site. After the penetrating injury but before the injection of blood this haemorrhage increased considerably and became more diffuse.

The 3 control eyes which had an injection of physiological solution (BSS) at the time of injury showed the same initial anterior retinal changes. However, posterior retinal detachments did not occur, and posterior vitreous separation and the transvitreal bands were not observed.

After the injection of blood the view of the fundus was obscured for various periods of time. At first the blood clot was situated in the middle of the vitreous cavity, but during the first week after injury the blood began to diffuse through the vitreous gel, and at times the clot, which condensed anteriorly, could be seen to maintain its attachment to the disc.

By B-scan ultrasound examination the posterior vitreous appeared to separate from the retinal surface at about 11 days after the injury. However, between the third and the fourth weeks, when the vitreous became more clear, vitreous strands were seen connecting the penetrating wound site and the retina.
increased in intensity and extent following the penetrating component of the injury, often reaching the posterior pole. Concurrently, circumferential folds developed in the equatorial and anterior retina associated with subretinal haemorrhage. These localised subretinal haemorrhages persisted until the seventeenth day after injury (Fig. 1).

In eyes enucleated at 8 days after injury full-thickness retinal folds appeared at the posterior pole. These were arranged in a radial fashion, and vitreous strands connected the tips of the folds to the site of the penetrating wound (Fig. 2). The posterior radial folds persisted throughout the period of the follow-up, even though the transvitreal strands became less prominent. In the late stages of follow-up small peripheral retinal detachments occurred in the form of a forward roll of the ora serrata onto the pars plana 180° from the injury site (Table 2). Extensive retinal detachment did not develop, though subtotal detachment did occur in one eye.

**Discussion**

In this study we attempted to investigate the contusive component of a severe penetrating eye injury. Others have documented the effects of a contusion injury alone, but our aim was to examine how the combination of contusion and penetration relate to the development of intraocular haemorrhage.

In clinical practice the overall picture after ocular contusion is that of retinal opacification, retinal break formation, and haemorrhage from choroidal vessels. Blood accumulates in the suprachoroidal space as well as under the retina and may gain access to the vitreous cavity. Haemorrhage may result from direct rupture of choroidal vessels or may follow capillary dilatation secondary to tissue anoxia caused by initial vasoconstriction. In this study the standard contusive injury was designed to produce choroidal vascular changes and haemorrhage without producing a retinal break, and, despite the relatively low kinetic energy which we used, subretinal and vitreous haemorrhage was consistently present in the area of the injury.

The vitreoretinal changes following our standard contusion injury were similar to those usually seen clinically and to those described in previous experiments. Retinal whitening and wrinkling adjacent to the site of the impact were a constant feature. Interestingly, the retinal opacification appeared more pronounced and extensive after the combined injury (group III) than after contusion alone (group I); this may have been due to the sudden lowering of intraocular pressure or to a delay in the observation time or both.

The circumferential retinal folding posterior to the

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**Fig. 1** Gross section of an eye 6 days after the combined contusion and penetrating injury. A dense blood clot is situated posterior to the penetrating wound. Note anterior circumferential retinal fold (black arrows) and early anteroposterior bands (empty arrows). The posterior retina is attached.

**Fig. 2** Gross section of an eye 4 weeks after a combined contusion and penetrating injury. High posterior retinal folds are drawn towards the penetrating wound by transvitreal traction bands. The anterior circumferential retinal folds are no longer present.
Combined posterior contusion and penetrating injury in the pig eye. I. A natural history study

797

contusion site resembled that observed previously by Cox in association with anterior retinal dissolution,24 even though in our model fragmentation of the retina did not occur. Although no large haemorrhagic choroidal detachments developed, the presence of subretinal haemorrhage beneath the anterior circumferential retinal folds indicated bleeding from the choroid. Similarly, since no breaks in the retinal vessels were evident, the vitreous haemorrhage adjacent to the impact site was assumed to have originated from the uveal tract. As in the clinical setting, the vitreous haemorrhage gradually became absorbed and the localised retinal detachment settled; this was accompanied by localised proliferation of the retinal pigment epithelium in the areas of contusion.

In the pig eye, unlike the monkey eye, total retinal detachment did not develop after a standard posterior penetrating injury with blood injection. The retinal folds in the pig eye were frequently extensive but remained localised to the posterior pole, although accompanied by signs of anteroposterior vitreous traction; such a situation is unusual in man and was not observed in the rhesus monkey eye.19 However, anteroposterior vitreous traction and localised detachment of the posterior retina have been previously described in the rabbit eye after an experimental injury29 as well as following injection of retinal pigment epithelial cells into the vitreous.27 Significantly, posterior vitreous detachment does not occur in the rabbit eye after a posterior penetrating injury.28 The apparent lack of posterior vitreous separation in the pig eye was subsequently confirmed on histological examination.28 In pig eyes with the combined contusion and penetrating injury, without intravitreal blood injection, vitreoretinal traction or traction retinal detachment did not occur.

Changes secondary to contusion were obvious immediately. These changes persisted for less than 2 weeks, whereas the tractional complications of the penetrating injury did not become clinically apparent until the second week after the injury. Indeed, there appeared to be a period of transition between resolution of the effects of contusion and the onset of complications of the penetrating injury. The former was shown by the disappearance of retinal opacification and the spontaneous reattachment of localised anterior retinal detachment; the latter was characterised by the development of transvitreal strands, localised posterior retinal detachment, and, in the late stages, anterior roll of the ora serrata located 180° opposite the penetrating injury site. Secondary haemorrhage did not occur in any of the animals in this part of the study.

The results of this study support clinical observations on the haemorrhagic effects of the contusive component of a severe penetrating injury. It is recognised that intraretinal haemorrhage due to contusion occurs at the time of injury but may also occur during a very early vitrectomy. From this study it would seem that by the second week after injury haemorrhage due to contusion and the accompanying choroidal changes begins to abate. This may be an interval of time during which the effects of contusion subside and the tractional complications of the penetrating component have not yet become irreversible. It is therefore possible that in this model, if vitrectomy is performed during this period, traction retinal detachment may still be prevented and yet the potentially disastrous operative haemorrhage may be avoided.

As a result of this experiment we obtained a useful model with clinical evidence of vitreoretinal traction which will be used for a controlled trial of vitrectomy in the treatment of experimental combined contusive and penetrating posterior eye injury.

We thank Kate Borkowski and her colleagues for technical assistance and Sue Gerton for secretarial help.

This study was supported in part by NIH grants EY 02061 and EY 03040 (Dr Ryan).

References


