Effect on the intraocular pressure of hypotensive anaesthesia with intravenous trimetaphan

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SUMMARY The intraocular pressure was measured at 5-minute intervals with a Perkins applanation tonometer in 5 patients undergoing routine dacryocystorhinostomy under low-tension anaesthesia with a continuous intravenous infusion of trimetaphan (Arfonad), the systolic pressure being maintained at 60 mmHg. Twelve patients undergoing cataract surgery under the same premedication and anaesthesia but in whom the systemic blood pressure was maintained at normal levels served as controls. A sudden and dramatic lowering of the intraocular pressure to very low levels was noted when the systolic blood pressure was reduced to 60 mmHg. It seems likely that a collapse of the choroidal circulation rather than a decreased aqueous formation due to low perfusion pressure is the most likely cause of the sudden lowering of intraocular pressure.

Transient changes in intraocular pressure related to abrupt changes in systemic blood pressure possibly arising from changes in ocular blood volume have been described.¹ Variations in blood pressure produced alterations in the ocular blood flow, and Bill² and Cohan and Cohan³ showed that the blood flow through the uveal tract varied directly with the level of systemic arterial pressure. Adler⁴ showed that artificially induced changes in systemic blood pressure in animals were accompanied by parallel changes in the intraocular pressure, which were exaggerated if the cervical sympathetic trunk was divided. There is little doubt that ocular blood flow is influenced mainly by the systemic arterial pressure, lowering of which is bound to produce a fall in intraocular pressure.

Anaesthetic procedures are also known to cause alterations in intraocular pressure. Laryngoscopy and intubation caused a rise in intraocular pressure,⁵ while intravenous induction agents caused a fall in intraocular pressure,⁶ with the exception of ketamine, which had the opposite effect.⁷ Intravenous suxamethonium caused a rise in intraocular pressure, while thiopentone sodium increased facility of outflow and so lowered the intraocular pressure.⁸ Thus the intraocular pressure at the time of anaesthetic induction is subject to much variation.

Ganglion blocking agents like hexamethonium bromide (Vegolysen) given by intravenous infusion

to lower systemic blood pressure to 90 mmHg were found to lower the raised intraocular pressure in patients with acute glaucoma to normal levels.^{9,10} In hypertensive subjects pentolinium or hexamethonium bromide produced average falls of 9 mmHg in the intraocular pressure.¹¹ Niesel¹² found that hexamethonium given intravenously to rabbits always reduced the systemic blood pressure, intraocular pressure, and choroidal blood flow. Thus ganglion blocking agents which cause a fall in blood pressure also cause a fall in intraocular pressure, but with some drugs like intravenous tetraethyl ammonium bromide the fall in intraocular pressure was of a greater degree than could be explained by a mere fall in blood pressure,¹³ and this suggests that, though the fall in intraocular pressure may be secondary to the systemic changes in the circulation, the ganglionic connections of the nervous pathways to the eye concerned with the regulation of aqueous formation or in the control of its outflow may also be involved.

The effect of hypotensive anaesthesia with hexamethonium in arresting bleeding into the anterior chamber associated with hyphaema has been observed.^{10,14} Trimetaphan (Arfonad) is a short-acting ganglion blocking agent administered by continuous intravenous infusion. No studies have been reported on the effects of trimetaphan-induced low-tension anaesthesia on the intraocular pressure. It was therefore decided to study these effects in 5 patients undergoing routine dacryocystorhinostomy.

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Fig. 1 Variation of intraocular pressure with systemic blood pressure.

Materials and methods

The intraocular pressure was measured at 5-minute intervals with a Perkins hand-held applanation tonometer¹⁵ in 5 patients under low-tension anaesthesia in whom the systolic blood pressure was reduced to and maintained at 60 mmHg. Twelve patients undergoing cataract surgery under the same premedication and general anaesthetic but in whom the systemic blood pressure was maintained at normal levels served as controls. The intraocular pressure was measured in the unoperated eye.

Premedication in all patients was pethidine 50 mg and promethazine 25 mg given intramuscularly one hour before operation. General anaesthesia was induced with thiopentone sodium and suxamethonium, an endotracheal tube was passed, and the patient was maintained with nitrous oxide, oxygen, and halothane. Trimetaphan was infused intravenously in a continuous drip, the rate of the drip being controlled to maintain systolic blood pressure at 60 mmHg. The purpose of using this hypotensive agent was to secure a bloodless operation field. Brachial artery blood pressure and intraocular pressure were recorded the day before the operation, after administration of the premedication, and at 5-minute intervals after the administration of the general anaesthetic.

Results

In the 12 patients undergoing cataract surgery in whom the systemic blood pressure was maintained

constant there was no significant change in the intraocular pressure. The maximal lowering of the intraocular pressure recorded in this group was a reduction of 3 mmHg occurring soon after induction of general anaesthesia and was probably due to the induction procedure.

In all 5 patients undergoing dacryocystorhinostomy a lowering of the systolic blood pressure to 60 mmHg produced a dramatic fall in the intraocular pressure to between 3–4 mmHg from the preoperative level of between 13–17 mmHg (Fig. 1). The intraocular pressure was observed to remain steady at this low level throughout the entire period during which the patient was under hypotensive anaesthesia. The maximal lowering of intraocular pressure was observed when the systolic blood pressure fell to 60 mmHg, and a rise of systolic blood pressure above 60 mmHg was associated with a corresponding rise in the intraocular pressure (Table 1). At no stage was the systolic blood pressure allowed to fall below 60 mmHg.

Discussion

Anatomically the eye has 2 separate systems of blood vessels—the retinal and uveal. The uveal vessels are peculiar in that they possess no precapillary sphincter, which is responsible for opening and closing the capillaries, with the result that the flow of blood in the uveal vessels is steady and not alternating¹⁶ and there is a very high rate of blood flow through the uveal vessels.

In contrast to the kidney and brain, where owing to

Table 1 Blood pressure and intraocular pressure recordings at 5-minute intervals in 5 patients undergoing hypotensive anaesthesia

		<i>Pre op.</i>		<i>With hypotension</i>															
1.	BP	140	*	70	65	60	60	60	60	60	60	60	60	+	70	75	80		
	(mmHg)																		
	IOP	13		8	7	3	3	4	3	3	3	4		3	5	8	12		
2.	BP	140	*	90	80	80	65	60	60	62	60	60	+	70	80	85	90		
	(mmHg)																		
	IOP	15		12	9	9	5	3	3	3	3	3		7	7	8	8		
3.	BP	130	*	100	80	60	60	60	60	63	60	60	+	65	80	85			
	(mmHg)																		
	IOP	14		11	8	3	3	4	3	3	3	3		7	11	11			
4.	BP	145	*	100	80	60	60	60	62	60	60	65		60	60	+	70	80	90
	(mmHg)																		
	IOP	17		10	9	5	3	3	3	4	3	3		3	3	7	9	14	
5.	BP	160	*	90	75	60	60	60	60	64	60	60		60	+	70	85	90	
	(mmHg)																		
	IOP	15		12	8	4	3	3	3	3	3	3		3	7	10	12		

Points at which the intravenous trimetaphan drip was started* and stopped†.

autoregulation a fall in perfusion pressure produces vasodilatation, thus maintaining a steady flow, there is no compensatory mechanism in the choroid, and the blood flow falls in proportion to the fall in perfusion pressure.¹⁷ Though there is no reflex which significantly stabilises the intraocular pressure when the arterial blood pressure drops, a reflex mechanism does exist to protect the eye against a marked rise in pressure with systemic hypertension.⁴

A transverse section of the choroid shows it to be made up almost entirely of a series of blood vessels, and because of this 'erectile' potential of its vascular channels the choroid undoubtedly plays a role in the regulation of intraocular pressure. As the volume of the vascular bed of the choroidal circulation is several times that of the retinal circulation, the former makes a greater contribution to the maintenance of intraocular volume and intraocular pressure. Since it is mainly the degree of filling of the choriocapillaris and other blood vessels of the choroid which determines the volume of blood in the interior of the eye and hence the intraocular pressure, a systemic hypotension which reduces the blood flow in the choroid produces a sudden fall in intraocular pressure. This was clearly demonstrated in the rabbit, where the effect of carotid clamping was an immediate fall in intraocular pressure in the ipsilateral eye and a dramatic reduction in the blood flow through the choroid.¹⁸ Stimulation of the peripheral end of the cut cervical sympathetic trunk caused a constriction of the uveal vessels, producing a reduced uveal blood flow and a marked fall in intraocular pressure. If the

eyeball was enucleated or if the circulation of blood to the eye was interrupted, the intraocular pressure fell immediately. A rhythmic variation of the intraocular pressure occurring in phase with the cardiac cycle due to alterations in the volume of the vascular bed of the eye has also been demonstrated.¹⁹ It is thus apparent that any factor which changes the filling of the choroidal reservoir must affect the intraocular pressure immediately.

The two fluids in the eye—blood and aqueous humour—contribute to the volume of the intraocular contents, and variations in the amount of either on account of a systemic hypotension will alter the intraocular pressure considerably. Formation of aqueous is partly due to secretion (75%) and partly due to ultrafiltration (25%). Ultrafiltration is the result of the hydrostatic pressure within the capillaries of the ciliary processes and is dependent on the ciliary artery blood pressure. If the systemic blood pressure falls, ultrafiltration is reduced as the blood supply to the ciliary body is reduced,²⁰ but secretion is not affected until the blood pressure falls to very low levels. It has been shown that the formation of aqueous persists in monkeys even at very low arterial pressure.²¹ Hence, unless there is extreme systemic hypotension, formation of aqueous continues by the process of secretion.

It is known that the latent period associated with the rise in intraocular pressure which follows a rise in blood pressure is 0.1 s, and no mechanism other than the distension of the choriocapillaris could adapt so rapidly. It is unlikely that the rise in intraocular pressure is due to increased aqueous formation, in which

case the latent period is bound to be longer. The rapid influx of blood into the choriocapillaris produced an immediated steep but transient elevation of intraocular pressure.²² Our results show that a similar process could account for the fall in intraocular pressure which follows systemic hypotension, because a fall in intraocular pressure was observed with each stage of lowering of systemic blood pressure, the maximum fall in intraocular pressure occurring when the systolic blood pressure was reduced to 60 mmHg. Changes in intraocular pressure were almost exactly parallel to those in brachial artery blood pressure.

Thus it is more likely that the immediate and marked fall in intraocular pressure associated with systemic hypotension is due to a reduction in the volume of the vascular bed, and the persistence of the low intraocular pressure is due to the decreased formation of aqueous consequent upon the reduced flow of blood through the ciliary processes.

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