Communications

Ophthalmic arterial and venous pressures

Effects of acute intracranial hypertension

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Duke-Elder (1926) rightly pointed out that "There is no question in the whole of ophthalmology more important and fundamental than the vascular pressures of the eye". The ophthalmic artery pressure (OAP) has been measured almost invariably by ophthalmodynamometry or other methods based on the same principle, and the results obtained have shown very wide variation (Duke-Elder, 1926; Koch, 1945). Because this has led to much confusion on the subject, and more reliable estimates of the OAP are desirable, direct cannulation of the ophthalmic artery has been used to measure the OAP in rhesus monkeys. The OAP was correlated with the systemic arterial blood pressure (BP) recorded from the aorta, in order to determine whether a significantly close relationship existed between the two arterial pressures, and whether the OAP could be deduced from the BP. In fact, a very close correlation was found, indicating that it is possible to deduce the OAP from the BP by the formula computed in the present study.

The effects of intracranial hypertension on the ophthalmic arterial and venous pressures, and their significance, have been the subject of considerable controversy, especially the role played by raised ophthalmic venous pressure in the pathogenesis of oedema of the optic disc and various types of haemorrhage (pre-retinal, retinal, from the optic nerve and its sheath, and orbital).

The aim of the present study was to investigate the following:

(*i*) The normal ophthalmic arterial and venous pressures.

(*ii*) The correlation of the OAP with the BP, and of the ophthalmic vein pressure (OVP) with the pressure in the intracranial venous sinuses.

(*iii*) The effects of acute intracranial hypertension on these pressures.

(*iv*) In intracranial hypertension, the correlation of change in the ophthalmic arterial and venous pressures with changes in the systemic arterial and venous pressures and the pressure in the intracranial venous sinuses.

(*v*) The effects of acute intracranial hypertension on the fundus of the eye.

In this study, the various pressures were recorded in rhesus monkeys after direct cannulation of the ophthalmic artery, superior ophthalmic vein, abdominal aorta (*via* the femoral artery), internal jugular vein, and superior sagittal sinus. This investigation formed a part of studies on the pathogenesis of oedema of the optic disc in cases of raised intracranial pressure (Hayreh, 1964a, 1965a, 1968).

Material and methods

This study was carried out in 27 healthy adult rhesus monkeys, weighing between 4 and 8 kg.

They were anaesthetized with intraperitoneal nembutal, 40 mg./kg. body weight, and placed on an electrically warmed animal operating table. Atropine 1 per cent. and phenylephrine 10 per cent. were instilled into each eye to dilate the pupil and the lids were then sutured together to preserve corneal transparency, so that the fundus could be examined at the end of the experiment for any changes.

With the aid of a Zeiss operating microscope the ophthalmic artery and vein were exposed for cannulation at the right supraorbital margin. The superior sagittal sinus was exposed at the vertex, the right internal jugular vein in the neck at about the level of the thyroid cartilage, and the femoral artery and vein just below the inguinal ligament. The vessels were then cannulated in the following order:

(1) *Femoral vein*

This was cannulated in order to inject 5000 i.u. heparin to minimize clotting of the cannulae, and subsequently to administer maintenance doses of nembutal.

(2) *Femoral artery*

1 mm. nylon tubing was inserted as far as the abdominal aorta.

(3) *Internal jugular vein*

A glass T-cannula was used. Each arm of the T carried a soft PVC tube about 2 cm. long and with a 1·5 mm. bore, to prevent the collapse of the wall of the vein where it had been exposed and had lost the support of the surrounding tissue and to maintain the flow of blood to the heart. The vein on the right side was used because it is in direct line with the superior vena cava and right atrium.

(4) *Ophthalmic vein*

0·5 mm. nylon tubing was pushed in for about 2 cm. down the intraorbital section of the vessel.

(5) *Ophthalmic artery*

0·2 mm. nylon tubing was inserted for 0·5 to 1 cm. into the intraorbital section.

(6) *Superior sagittal sinus*

1 mm. nylon tubing was introduced through a small incision in the superficial wall, and pushed in for 4 to 5 cm.

(7) The animal was turned over to lie on its left side and a short-bevel No. II serum needle was introduced into the *cisterna magna (cerebello-medullary cistern)* to enable the cerebrospinal fluid pressure (CSFP) to be recorded and elevated.

The cannulae were filled with normal saline and connected to six pressure transducers, the amplified outputs of which were fed to six mirror galvanometers in an ultra-violet recorder, using 12 in. paper.

The pulse rate was measured in seventeen animals by recording the aortic pulse at a high paper speed on another recorder at frequent intervals throughout the experiment.

The initial pressures were recorded first, and a tap connecting a saline-filled reservoir to the CSFP transducer was then opened so that the pressure could be elevated to any desired level by raising the reservoir. The pressure was raised initially to 10 mm.Hg and subsequently increased in steps of 5 mm.Hg about every 5 minutes. The maximum elevation depended upon the response of the animal, so that in some experiments the CSFP did not reach the planned level of 50 mm.Hg or more because the monkey showed signs of distress or collapsed. In fifteen experiments the pressure was raised to 50 mm.Hg, in four to 45, in three to 40, in one to 35, in one to 55, in two to 60, and in one to 70 mm.Hg.

In 22 animals the CSFP was raised above normal for periods varying betewen 45 and 92 minutes, while in the remaining five experiments the time varied between 36 and 120 minutes. The total duration of the elevation of pressure in any particular experiment generally depended on how long the maximum pressure could be tolerated by the animal.

After this, the reservoir was lowered rapidly to zero to record the effect of sudden decompression after an acute intracranial hypertension. Most of the monkeys collapsed immediately or shortly after this procedure. The fundus was examined at the end of the experiment for evidence of any changes.

Results

The responses of the rhesus monkeys to the acutely raised CSFP varied widely. A majority, however, showed a significant degree of similarity. The arterial responses tended to fall into three phases:

(1) *Initial* The CSFP was raised from normal to 15 mm.Hg.

(2) *Middle* The CSFP was raised from 15 to 30–40 mm.Hg.

(3) *Final* The CSFP was raised from 30–40 mm.Hg up to a level which depended on the animal's tolerance of the high pressure.

The effects of the sudden fall in the CSFP are presented separately.

A detailed account of the effects on the systemic arterial blood pressure (BP), pulse rate, superior sagittal sinus pressure (SSP), and internal jugular vein pressure (JVP) are reported elsewhere (Hayreh and Edwards, 1971).

From the continuous recordings in the various animals, the levels of the various pressures and the pulse rate were estimated at the normal CSFP and at each step just before the pressure was raised to the next level, *i.e.* 10, 15, 20 mm.Hg and so on. The data thus collected have been subjected to detailed statistical analysis. Before tests for significance of the differences between the various pressures at the various levels of the CSFP were done, the Friedman two-way analysis of variance was carried out to determine the overall significance of the data.

EFFECTS OF ACUTE INTRACRANIAL HYPERTENSION

Systemic arterial BP

These results have been reported in detail elsewhere (Hayreh and Edwards, 1971). The normal systolic and diastolic BP are given in Table I.

Table I *Normal pressures in Rhesus monkeys*

Pressure (mm.Hg)		*Mean and standard deviation*
Systemic blood	Systolic	132·2 ± 13·4
	Diastolic	71·3 ± 14·1
Ophthalmic artery	Systolic	93·0 ± 15·0
	Diastolic	70·8 ± 10·7
Ophthalmic vein		3·94 ± 1·35
Superior sagittal sinus		3·93 ± 1·53

The variations in BP caused by the rise in CSFP are summarized in Table II.   Fig. 1 shows the mean systolic and diastolic blood pressures with standard deviations at different levels of the raised CSFP.

Table II   *Variations in systolic and diastolic pressure caused by raised cerebrospinal fluid pressure*

Phase of CSFP		Initial		Middle		Final	
BP		*Systolic*	*Diastolic*	*Systolic*	*Diastolic*	*Systolic*	*Diastolic*
Variation in BP (mm.Hg)	Mean	−7·31	+1·12	+43·43	+18·91	−26·47	−28·94
	SD	37·22	27·20	63·05	30·02	59·39	26·54

+ = rise in blood pressure; − = fall in blood pressure; SD = standard deviation
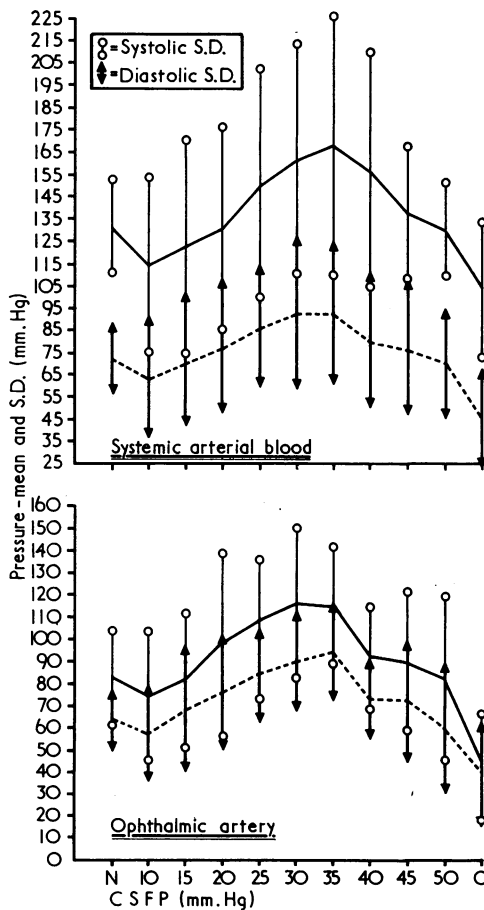

FIG. 1   *Mean systolic and diastolic systemic arterial blood pressures with standard deviations at different levels of raised cerebrospinal fluid pressure*

FIG. 2   *Mean systolic and diastolic ophthalmic artery pressures with standard deviations at different levels of raised cerebrospinal fluid pressure*

Ophthalmic artery pressure

The normal OAP was 93/71 mm.Hg (Table I).   Fig. 2 shows the mean systolic and diastolic OAP with standard deviations at different levels of the raised CSFP.

The correlation coefficient of the OAP (systolic and diastolic) with BP was calculated in the eight animals in which a satisfactory systolic and diastolic OAP was recorded (Figs 3, 4, 5), and the results (Table III) show a significant correlation (at the 0·01 level).

Table III *Spearman's rank correlation coefficient (r) between ophthalmic artery pressure and blood pressure and their significances*

Phase of CSFP	Normal		Initial		Middle		Final	
BP	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
r	0·99	0·98	0·99	0·99	0·82	0·98	0·81	0·99
Significance	0·01	0·01	0·01	0·01	0·05	0·01	0·05	0·01
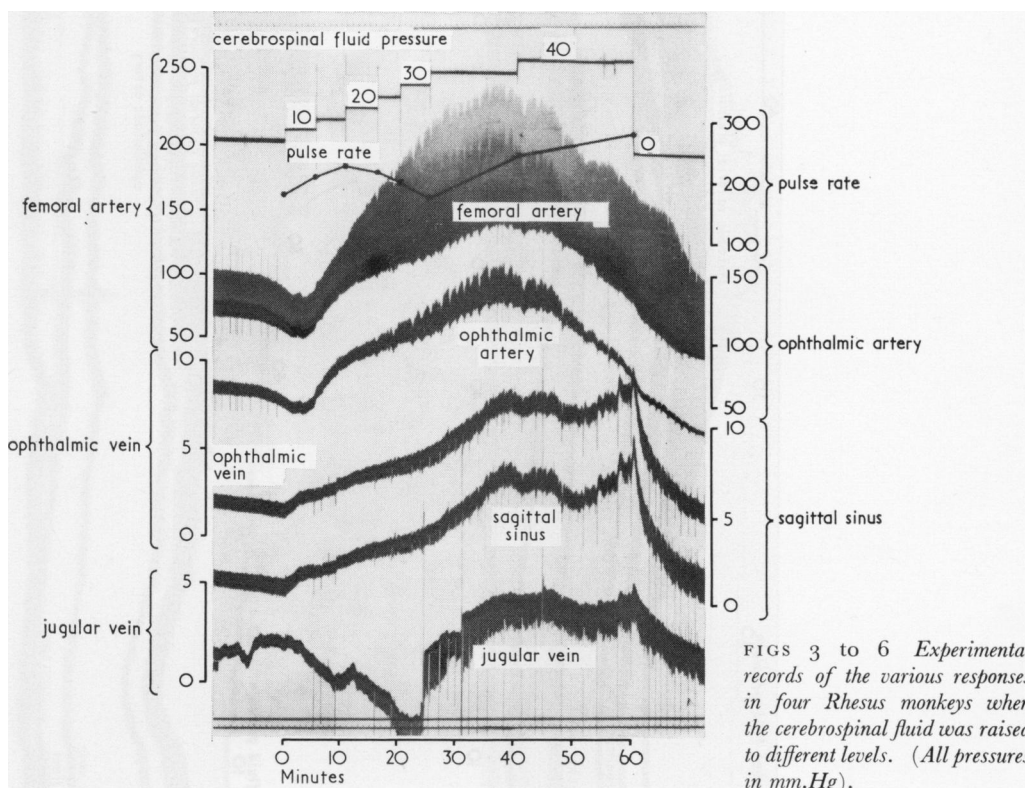
FIGS 3 to 6 *Experimental records of the various responses in four Rhesus monkeys when the cerebrospinal fluid was raised to different levels. (All pressures in mm.Hg).*

Regression equations were computed to calculate the OAP from the BP in normal animals and during the various phases of the rise in CSFP.

(a)   *Normal animals*

Systolic OAP = 0·80 × systolic BP − 8·63 mm.Hg

Diastolic OAP = 0·80 × diastolic BP +6·95 mm.Hg

The standard error of the estimate (corrected for small sample) for systolic and diastolic OAP was 3·8 and 3·4 mm.Hg respectively.
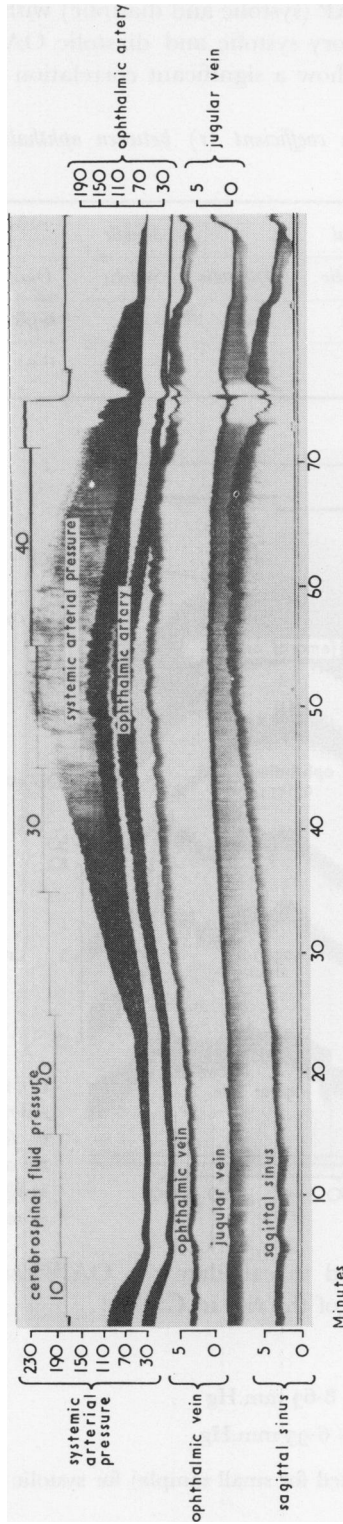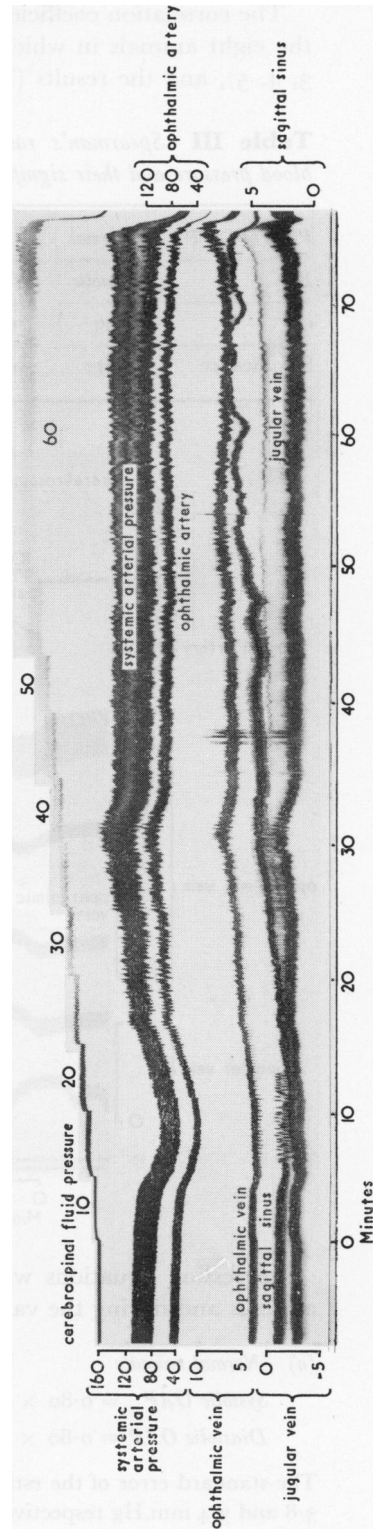
FIG 4

FIG 5

(b)   *Initial phase of raised CSFP:*

Systolic OAP = 0·75 × systolic BP −3·6 mm.Hg
Diastolic OAP = 0·86 × diastolic BP +2·94 mm.Hg

(c)   *Middle phase of raised CSFP:*

Systolic OAP   = 0·43 × systolic BP +41·6 mm.Hg
Diastolic OAP = 0·80 × diastolic BP +12·5 mm.Hg

(d)   *Final phase of raised CSFP:*

Systolic OAP   = 0·88 × systolic BP −40·6 mm.Hg
Diastolic OAP = 0·86 × diastolic BP +5·5 mm.Hg

A calculation of the ratio between the OAP and BP from these formulae reveals that during the final phase, the ratio between the two systolic pressures fell to about 47 per cent. from the 70 per cent. that obtained during the earlier phases and in normal animals.   Thus the systolic OAP is relatively much lower than the systolic BP during the final phase, but the diastolic OAP and BP show no such disparity.

In eleven other animals, the amplitude of pulsation of the OAP on the record was too small to enable the systolic and diastolic values to be measured separately (Fig. 6).   In these cases the pressure recorded was considered to be the mean of the systolic and diastolic OAP. The responses of the OAP in this group were similar to those of the diastolic OAP in the eight animals with satisfactory systolic and diastolic recordings.
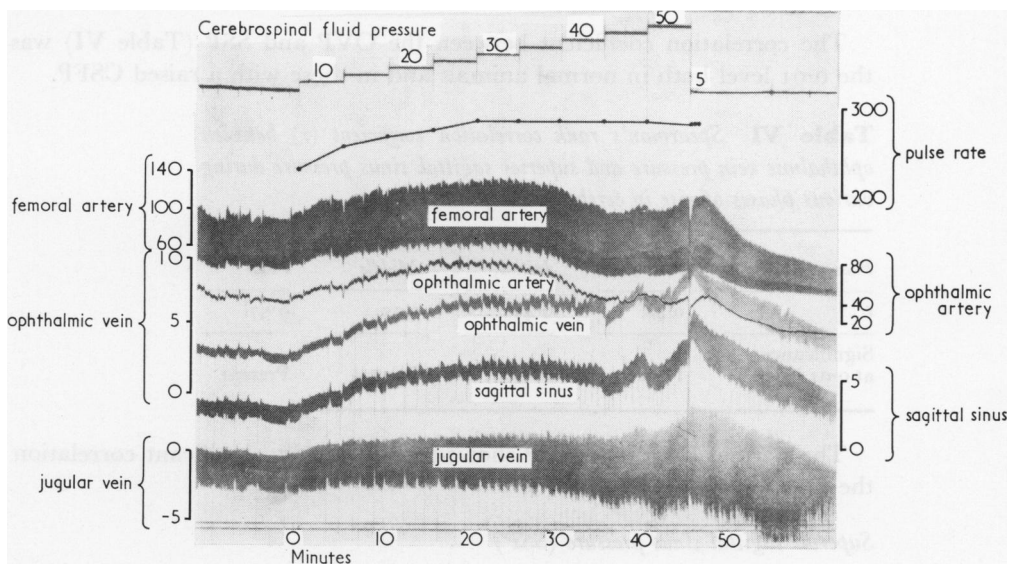

FIG 6

Ophthalmic vein pressure (OVP)

The normal OVP was 3·94 ± 1·35 mm.Hg (Table I).   The mean OVP with standard deviations at various levels of CSFP is shown in Fig. 7 (overleaf).   There was a significant difference between the spread of results at the beginning and at the end of the raised CSFP.

The correlation coefficient (r) between the OVP and the CSFP mostly varied between 0·80 and 1 (Table IV, overleaf) which is significant at the 0·01 level.

The correlation coefficient between the rise in OVP and the time taken by the CSFP to reach its highest value was equal to 0·21, which is statistically not significant.

Table IV *Spearman's rank correlation coefficient (r) between ophthalmic vein pressure and cerebro-spinal fluid pressure*

r	1·0	0·99	0·98	0·97	0·96	0·94	0·93	0·92	0·90	0·89	0·87	0·80	0·60	0·46
No. of animals	1	7	2	2	4	3	1	1	1	1	1	2	1	1
Significance	Present at 0·01 level												None	

The correlation between the OAP (both systolic and diastolic) and OVP is shown in Table V. Although the correlations are positive and substantial, we failed to detect a significant association between the OVP and OAP.

Table V *Spearman's rank correlation coefficient (r) between ophthalmic vein pressure and ophthalmic artery pressure during various phases of rise in cerebrospinal fluid pressure*

Phases of CSFP	Normal		Initial		Middle		Final	
OAP	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
r	0·58	0·60	0·58	0·55	0·69	0·67	0·35	0·21
Significance	None	None	None	None	None	None	None	None

The correlation coefficient between the OVP and SSP (Table VI) was significant at the 0·01 level both in normal animals and in those with a raised CSFP.

Table VI *Spearman's rank correlation coefficient (r) between ophthalmic vein pressure and superior sagittal sinus pressure during various phases of rise in cerebrospinal fluid pressure*

Phases of CSFP	Normal	Initial	Middle	Final
r	0·48	0·47	0·59	0·57
Significance at 0·01 level	Present	Present	Present	Present

The normal OVP and JVP showed a statistically significant correlation (r = 0·46) at the 0·01 level.

Superior sagittal sinus pressure (SSP)

The normal SSP was 3·93 ± 1·53 mm.Hg (Table I). Fig. 8 (opposite) shows the mean and standard deviations of the SSP at different levels of the CSFP.

The correlation coefficient between the SSP and the CSFP was significant at the 0·01 level (Hayreh and Edwards, 1971).

The correlation coefficient between the SSP and the JVP with normal and raised CSFP was significant at the 0·05 level.

EFFECTS OF SUDDENLY LOWERING THE CSFP AFTER ACUTE INTRACRANIAL HYPERTENSION

Table VII (opposite) shows the responses of the BP, OAP, OVP, SSP, and JVP to the

sudden lowering of the CSFP after acute intracranial hypertension. The correlation coefficient between the OVP and SSP changes was o·82, which is significant at o·o1 level.

A correlated "*t*"-test of the difference between the normal OVP level and that after lowering the CSFP was significant at the o·o1 level.
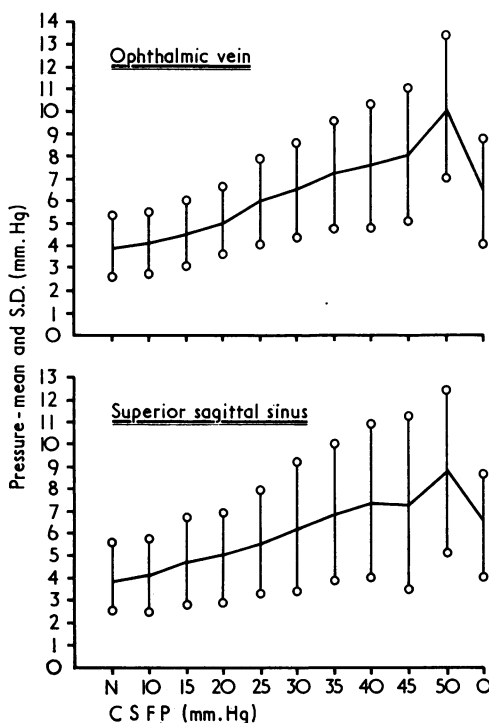

FIG. 7 *Mean ophthalmic vein pressures with standard deviations at different levels of raised cerebrospinal fluid pressure*

FIG. 8 *Mean superior sagittal sinus pressures with standard deviations at different levels of raised cerebrospinal fluid pressure*

Table VII *Effects of suddenly lowering cerebrospinal fluid pressure after acute intracranial hypertension*

Pressure (mm.Hg)	BP		OAP		OVP	SSP	JVP
	Systolic	Diastolic	Systolic	Diastolic			
I. Post-lowering level	95·8±32·2	39·1±19·9	42·5±23·0	40·0±25·7	6·4±2·4	6·3±2·3	2·1±2·3
II. Difference in levels between pre- and post-lowering	−36·9±42·7	−21·2±22·2	−23·8±10·4	−16·6±11·0	−3·2±4·5	−2·6±4·1	−0·7±2·4
III. Correlated 't' test for significance of values in Line II	Significant at o·o1 level	Significant at o·o1 level	Significant at o·o1 level	Significant at o·o1 level	Significant at o·o1 level	Significant at o·o5 level	Not Significant

Minus signs in Line II indicate a fall in pressure

It was discovered that if the OVP was less than 7·5 mm.Hg before the CSFP was lowered to zero, it always rose, but if the OVP was 7·5 mm.Hg or more it always fell. A χ^2 test of these changes (with Yates correction for continuity) established that they were significant at the o·o1 level. A significance at the o·o2 level was discovered in similar changes in the SSP.

Discussion

Ophthalmic artery pressure (OAP)

A very close correlation was found in the normal animals between the systemic BP and the OAP. The systolic and diastolic OAP can be estimated from the BP by the formulae given above. This indicates that the systolic OAP is about 71 per cent. of the systolic BP and the diastolic OAP about 87 per cent. of the diastolic BP. These findings are in sharp contrast to those obtained by ophthalmodynamometry (Duke-Elder, 1926; Koch, 1945). Borras and Cabot (1963) recorded the OAP in human beings by cannulation of the supraorbital artery, and in one of their patients, where they had the exact values of the various pressures recorded, the systemic BP was 120/75 mm.Hg and the OAP was 83/65 mm.Hg. The ratios for the systolic and diastolic pressures were 69 and 87 per cent. respectively. These compare very well with those recorded in our series in the normal rhesus monkey.

Duke-Elder (1932) pointed out that, when the column of blood contained in a vessel was partially or completely immobilized, the pressure registered was the lateral pressure at the most proximal arterial branching. The ophthalmic artery is the first major branch of the internal carotid artery. Thus the OAP reflects the internal carotid artery pressure. In rhesus monkeys, the ophthalmic artery gives off large branches during its course through the orbit, the last being the medial palpebral artery (Hayreh, 1964b). We cannulated the ophthalmic artery near the supraorbital margin and the pressure recorded was that of the blood column in the ophthalmic artery distal to the branching of either the ethmoid artery or the medial palpebral artery. Thus, the OAP recorded by this procedure may be some-what lower than the internal carotid artery pressure, but for all practical purposes we can assume that the OAP is a fairly good index of the internal carotid artery pressure and the various changes in it. Hence the OAP may also be used to determine changes in the cerebral circulation (Hayreh and Edwards, 1971). Since a close correlation exists between the OAP and BP (Table III), it is assumed that the BP is normally a fair index of the ophthalmic and cerebral circulation. This, however, would not apply in cases of stenosis of the carotid system or the ophthalmic artery. There is thus an urgent need for a more accurate means of measuring the OAP than that provided by ophthalmo-dynamometry.

Effects of raised CSFP on OAP

Hedges (1963) raised the CSFP to 40 mm.Hg in one monkey and did not record any change in the OAP. He concluded that subacute experiments of any duration were not possible with this technique. We have however been able to perform similar experiments in nineteen monkeys in the present series and have found responses in the OAP in all of them.

Responses of the systemic BP to raised CSFP are discussed elsewhere (Hayreh and Edwards, 1971). During the rise in CSFP from normal to 15 mm.Hg, the systemic BP showed a slight fall, particularly in the systolic pressure; it then began to rise with further elevation of the CSFP. In the majority of the animals, it reached its highest level when the CSFP was at 30–40 mm.Hg. After that, any further rise in the CSFP usually led to a fall in the BP (Table II, Fig. 1).

On elevation of the CSFP in the present study, the correlation between the diastolic ophthalmic and systemic blood pressures was well maintained throughout the time that

the CSFP was raised (Table III). The correlation of the systolic OAP with the systolic BP was maintained up to about 35 mm.Hg CSFP, but beyond that level the ratio dropped significantly, so that during the final phase the systolic OAP was 47 per cent. of the systolic BP as compared to the normal 71 per cent. This fall in the ratio of the systolic pressures at high levels of the CSFP seems to be due to the "cuffing effect" of the raised CSFP on the intracranial portion of the ophthalmic artery. This cuffing effect would not interfere with the diastolic OAP. The formulae for calculating the OAP from the BP at various levels of the CSFP are given above.

Berens, Smith, and Cornwall (1928), using ophthalmodynamometry in patients with raised intracranial pressure, found a raised pressure in the central retinal artery which was not associated with a rise in the systemic BP. They concluded that there was a selective rise in the OAP in these cases. Bailliart (1923) said the pressure in the retinal arteries always rose in patients with brain tumours and that this was associated with a normal systemic BP (Bailliart, 1954). Huber (1961) also concluded that "in an incipient papilloedema a discordant increase in the pressure in the retinal arteries, provided there is no evidence of a systemic hypertension, may be interpreted as an indication of an increased intracranial pressure." Similarly, Magitot (1922) thought that a raised OAP: BP ratio was of significance in such cases. Streiff and Monnier (1946) and Weigelin (1954), however, showed that such an increase in the OAP: BP ratio was the exception rather than the rule.

Our studies have shown no such selective rise in the OAP in acute intracranial hypertension. The rise in the OAP simply reflects a similar rise in the systemic BP. It may be stressed that all these authors measured the central retinal artery pressure by ophthalmodynamometry; this is an unreliable and erratic means of determining the absolute central retinal artery pressure and not much significance should be attached to it.

A sudden fall in the CSFP always produced a significant fall in the OAP (both systolic and diastolic) which seemed to be secondary to similar changes in the systemic BP. No relationship was discovered between the original and final steady-state OAP, or between the level from which the CSFP fell and the amount of the change in pressure.

Ophthalmic vein pressure (OVP).

Hedges (1963), on cannulation of the superior ophthalmic vein in rhesus monkeys, found the OVP to be approximately 4 mm.Hg. In our series the normal OVP was 3.9 ± 1·3 mm.Hg.

On raising the CSFP to about 40 mm.Hg in nine experiments in rhesus monkeys, Hedges (1963) found a significant rise in the OVP in seven animals (30 mm.Hg in two, 11 in two, and 13, 16, and 24 in the remaining three). The effects of the raised CSFP on the OVP in our series (Fig. 7) are in sharp contrast to the findings of Hedges (1963).

A highly significant correlation was seen between the OVP and CSFP (Table IV): the higher the CSFP the higher the OVP. The time taken by the CSFP to reach its highest level did not significantly influence the OVP.

A correlation between the OVP and OAP (both systolic and diastolic) was looked for (Table V) but no significant relationship between the two was found. This indicates that the OAP does not influence the OVP.

A significant correlation was observed between the OVP and SSP (Table VI): the higher the OVP, the higher the SSP. This seems to us to be an important finding. It is reasonable to assume that the OVP reflects the venous pressure in the cavernous sinus, into which the ophthalmic veins drain. This is because the raised CSFP cannot

influence the OVP directly. The correlation discovered between the OVP and the internal jugular vein pressure (JVP) in normal animals does not mean that the OVP is controlled by the general venous pressure in the head and neck, but that it is an indirect reflection of the significant correlation between the SSP and JVP. This is because the superior sagittal sinus and other intracranial venous sinuses drain into the internal jugular vein and the SSP and OVP show a close correlation. The fact that no correlation was discovered between OVP and JVP on elevation of the CSFP supports the concept that OVP and JVP are independent.

The observations indicate that the venous pressures in the cavernous sinus and superior sagittal sinus are related to one another, although no direct communication was found to exist between the two sinuses in the specimens studied by Weinstein and Hedges (1962).

According to Hedges (1963), the orbital venous responses to raised CSFP could be due to:

(**1**)   Interference with venous outflow from the orbit into the cavernous sinus, produced by compression of the wall of the sinus.

(**2**)   The arterial pressor responses.

(**3**)   Possibly on auto-regulatory haemodynamic mechanism in the orbital vascular bed.

Hedges suggested that the initial increase in the OVP was due to the first factor and that the continued and prolonged elevation of the venous pressure was probably due to the third factor, while the second factor was unimportant. We find it difficult to postulate the existence of an auto-regulatory haemodynamic mechanism in the orbital vascular bed, as there is no evidence available to suggest its existence. Since no correlation was seen between the OVP and OAP, the presence of any influence by the OAP on the OVP can be excluded. It must, however, be mentioned that, during the later part of the final phase of the raised CSFP, the OAP usually fell (Fig. 2) while the OVP rose (Fig. 7); this may have been due to the change in the peripheral resistance in the orbital arterioles as part of the generalized vasomotor depression in the body, not limited to the orbital vessels. A fall in peripheral resistance may lead to a fall in the OAP and allow blood at a higher pressure to reach the veins. This may be a contributory factor to the raised OVP in the later stages of the raised CSFP only. Our conclusion is that the primary factor controlling the OVP is the pressure in the cavernous sinus, because a significant correlation exists between the SSP and OVP in the normal animals and throughout the duration of the raised CSFP. There is no reason to assume that the pressure in the various intracranial venous sinuses is not the same.

The raised pressure in the orbital veins, including the central retinal vein, in cases of intracranial hypertension has been postulated as the cause of oedema of the optic disc (papilloedema) and of haemorrhages in the sheath of the optic nerve and pre-retinal haemorrhages. Both these subjects have been discussed at length elsewhere (Hayreh, 1963, 1965a, 1968), and a brief review is given below.

Türck (1853) was the first to suggest that the retinal haemorrhages in cerebral tumours were due to stasis in the cavernous sinus caused by the raised intracranial pressure. A similar view, explaining the pathogenesis of oedema of the optic disc in intracranial tumours, was put forward by von Graefe (1860), according to whom this produced choking of the blood in the retinal veins resulting in oedema of the disc. Heymann (1864), Judeich (1900), Merz (1900), and von Krüdener (1907)

considered a rise in pressure in the cavernous sinus to be a factor responsible for oedema of the disc.

Sesemann (1869), Picqué (1888), and Schieck (1910) contradicted this hypothesis on the basis of the existence of extensive anastomoses of the ophthalmic veins with one another and also with the anterior facial vein. The latter communication of the ophthalmic vein would prevent venous stasis in the event of an impediment at the cavernous sinus. In our experiments, as in those of Hedges (1963), the cannulation of the superior ophthalmic vein blocked the communication with the anterior facial vein; thus the rise in the OVP seen in our experiments may represent a higher rise than that seen under normal circumstances. Gurwitsch (1883), after a thorough anatomical examination, concluded that the greater part of the orbital venous blood flowed into the cavernous sinus.

Weigelin (1950), in his haemodynamic theory, has tried to resuscitate the original theory of von Graefe and to rule out the objections to it of Sesemann (1869). According to Weigelin, blood flows from the ophthalmic veins towards the cavernous sinus, but a large part of the drainage of the anterior part of the orbit takes place *via* the anastomotic channels into the facial veins. Normally the difference in pressure between the cavernous sinus and the point where the anastomotic channels leave the ophthalmic vein is very small. Whenever the pressure in the cavernous sinus rises because of intracranial hypertension, blood starts to flow from the sinus towards the ophthalmic vein. The central retinal vein opens into the cavernous sinus directly or through a short branch into the ophthalmic vein. In the event of a rise in pressure in the cavernous sinus, the blood from the central retinal vein cannot flow directly into the cavernous sinus. This leads to a rise in pressure in the central retinal vein, producing oedema of the optic disc.

Kornder (1919) and Hayashi (1955), from their observations in experimental intracranial hypertension in dogs, concluded that raised intracranial pressure produced a generalized rise in venous pressure, leading to engorgement of the retinal veins.

According to Hedges (1963), there is a dramatic rise in pressure in the ophthalmic vein with acute elevation of the intracranial pressure, and the rise is associated with retinal venous engorgement and haemorrhages; the mechanism of this rise in the OVP, as described by him, is discussed above (p. 660). He also found, however, that the SSP fell when the intracranial pressure was raised.

Experimental investigations by Lamach, Claude, and Targowler (*cited by* Lauber, 1935) showed that the blood pressure in the venous sinuses was partly independent of the intracranial pressure. Ryder, Espey, Kristoff, and Evans (1951) observed a fall in pressure in the sigmoid sinus when the intracranial pressure in rhesus monkeys was raised, and they considered the sigmoid sinus pressure to be dependent upon the cardiac output.

The role of raised retinal venous pressure in the causation of oedema of the optic disc has been discussed at length elsewhere (Hayreh 1965a, 1968). It was observed that the raised retinal venous pressure was not the primary factor in the pathogenesis of disc oedema, but was in fact secondary to it (Hayreh, 1965a, 1968, 1969). In the present series of animals with acute intracranial hypertension, oedema of the optic disc was not seen, and the raised OVP in cases of raised intracranial pressure is thus irrelevant to the pathogenesis of disc oedema. The absence of disc oedema in all the monkeys in the present series also indicates that acute intracranial hypertension is not associated with disc changes, and that there is a latent period of many hours, if not days, between the rise in intracranial pressure and the production of oedema of the disc.

The exact mechanism of haemorrhages in the orbit, the optic nerve, the sheath of the optic nerve, and the retina (particularly pre-retinal haemorrhages) in cases of a sudden rise in intracranial pressure, especially in subarachnoid haemorrhages, has been the subject of considerable speculation. Liebrecht (1906), Greear (1943), Walsh and Hedges (1951), Smith, Kearns, and Sayre (1957), Hansen (1958), Ikui and Mimatsu (1961), and many others, have considered these haemorrhages to be secondary to the raised ophthalmic venous pressure produced by the sudden rise in the intracranial pressure.

In the present study, the rise in the OVP, though always seen, was never greater than 12 mm.Hg with a rise of 50 mm.Hg in the CSFP, and on average the rise was about

7 mm.Hg (Fig. 7).   The possibility of a patient having an increase in intracranial pressure of more than 50 mm.Hg and remaining alive is remote.   Therefore the findings in the present series are applicable clinically.   It is extremely unlikely that the extent of the rise in the ophthalmic venous pressure, as indicated by the present study, could produce such haemorrhages.   The blood pressure in the normal retinal veins is more than 20 mm. Hg (*i.e.* higher than the intraocular pressure).   In cases of partial or complete central retinal vein occlusion (not associated with any arterial ischaemia), no retinal haemorrhages are usually seen in spite of a very high venous pressure (Hayreh, 1965b, 1971). This was further confirmed by the fact that no pre-retinal, retinal, or optic nerve sheath haemorrhages were seen in any of the animals in the present series.   Most probably the various ocular and extraocular venous haemorrhages described in cases of subarachnoid haemorrhage are due to the accompanying convulsions.   Generalized convulsions of the body would be associated with a sudden tremendously high venous pressure in the head and neck, far greater than that seen with raised intracranial pressure.   This sudden rise in venous pressure to extremely high levels could rupture the thin-walled venules and result in widespread haemorrhages in the orbit, the optic nerve and its sheath, and the retina.   The absence of haemorrhages in the monkeys of the present series may be due to the absence of convulsions.

A peculiar response of the ophthalmic vein to a sudden drop in the CSFP was discovered, *i.e.* if the OVP was less than 7·5 mm.Hg before the CSFP was lowered to zero, the OVP always rose, but if the OVP was 7·5 or more it always fell.   It is not possible to give a satisfactory explanation for this variation.   Possibly it is due to the presence or absence of cerebral venous drainage to the cavernous sinus.   If the cavernous sinus has no cerebral tributaries, the venous return to it is mainly from the ophthalmic vein and the OVP may fall when the CSFP is lowered.   However, if the sinus has large cerebral tributaries, a rise in the CSFP could hamper venous drainage from the cerebral veins and a fall in the CSFP would lead to increased venous drainage and hence to a rise in the pressure in the sinus, reflected in the raised OVP.

Summary

In 27 rhesus monkeys, the normal systemic and ophthalmic arterial and venous pressures and superior sagittal sinus pressure were recorded after cannulation.   The effect of acute intracranial hypertension on each was studied.   The ophthalmic and systemic arterial pressures showed a close correlation and formulae were computed for deducing the former from the latter.   Both rose, with a rise in cerebrospinal fluid pressure up to 30–40 mm. Hg, but further increases in CSFP usually produced arterial decompensation.   The ophthalmic venous pressure and superior sagittal sinus pressure showed a significant correlation with each other and with the rise in the CSFP.   The role of a raised OVP resulting from a raised CSFP in the pathogenesis of oedema of the optic disc and various ocular and extraocular haemorrhages is discussed.   Sudden intracranial decompression after hypertension produced a fall in the arterial pressures and rise or fall in the OVP and SSP.   The fundus afterwards showed no abnormality.

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