approximately 50 cases. In his own series there had been 2 cases where, despite an apparently completely successful surgical result, the vision had gradually deteriorated from 6/6 or 6/9 to 6/24 or lower with no obvious cause. In others there had been various complications including prolonged uveitis and late secondary glaucoma, necessitating removal of the lens. He felt that the indications and contraindications were by no means fully established and would like to hear more about them.

The question of complications was important. Was it always justifiable to hazard a good reserve eye, after some traumatic lesion, by undertaking additional surgery purely for the purpose of regaining binocular function, which in many patients was not appreciated? Apart from this there were the social and economic factors of multiple and delayed operative procedures to be taken into account. Where the possibility of amblyopia supervening arose, this became of overriding importance.

In one of his own cases monocularity over ten years had resulted in no particular disability. An acrylic lens was inserted; after an initial satisfactory result with 6/6 vision, maintained for about three months, secondary glaucoma set in. Two procedures directed to this were without avail and eventually the lens had to be removed. The eye, which had seen 6/6 with aphakic correction, now saw only 6/12. At one time sympathetic ophthalmia had seemed likely to ensue.

Mr T Keith Lyle (*London*) said he had visited Mr Choyce in Southend two years ago and had seen him operate and had learned many practical points about the technique. He felt that considerable experience was needed in work of this kind and that it was best done by those who were prepared to give the matter considerable time and thought.

Professor Norman Ashton (*London*) said that he had now examined pathologically four eyes (two from post-mortems) in which anterior implants had been inserted, and had found a marked degree of atrophy and distortion in the parts of the iris upon which the acrylic implant rested. In one case this had developed in only six weeks. On this evidence it appeared uncertain whether the iris bed would continue to support the implant in position with the passage of years. Until this question had been resolved by long-term observations on existing cases, he felt that the exact value of this operation must remain in doubt.

Mr Choyce, in reply to Mr Roper-Hall, said that the axis of astigmatism was related to the position of the corneal wound and not to the axis of the implant.

With regard to possible complications, Mr Choyce referred Mr Lister to his other publications (in particular to Choyce 1960*a*, p 430), which described complications occurring within five months of implant insertion. He added that so far he had not seen delayed (up to five years) complications in cases which at five months had appeared satisfactory in every respect. He was sure that all implant cases should be kept under regular observation, at least once every twelve months, so that the surgeon could remove an implant if he was not satisfied with it and felt that its continued presence in the eye might result in irreversible complications.

Mr Choyce was sorry to hear that Mr Gilkes and his colleagues had encountered a 20–25% incidence of complications, but felt he could not comment further without knowing how many cases were involved, the pre-operative state of the eyes and other data. Mr Choyce had had his worries with his first few cases, but had found that with increasing experience in the selection of cases, the surgical technique and the post-operative care, complications had markedly declined and results correspondingly improved. The incidence of serious complications had been 22% in his first 100 cases and had declined to 3% in the third 100 cases.

Anticoagulants in the Treatment of Retinal Vein Thrombosis

by B Thorne Thorne MB MRCP DO (*Brighton*)

Thrombosis of the central vein of the retina is familiar to all of us and it is evident, from the amount of congestion and hæmorrhage caused by the obstruction to the venous return, that a considerable degree of damage is going to be caused to the retinal tissues. Also, the condition is sufficiently common and the prognosis for vision is usually so poor, that if there is a method of treatment which can in some cases improve the poor prognosis, or can prevent the full picture of thrombosis occurring, that treatment will obviously be of some value.

Anticoagulants have been used in the treatment of this condition for a number of years, but until fairly recently they have not been in very general use, partly because of the difficulties in controlling the effects of the drug, and partly because the results of treatment have not been particularly encouraging.

In the last few years there have been reports of the value of the newer types of anticoagulant drugs in the treatment of retinal vein occlusion but, in my experience, this form of treatment is the exception rather than the rule. This may be due to the fact that the progress of the condition is too variable, making assessment of the value of any form of treatment too difficult, or because the giving of anticoagulants creates problems of its own, both for the patient and the doctor (though these problems are sometimes exaggerated). Or is it that this form of treatment does no good?

Lister & Zwink (1953) concluded, from their series of 13 cases, that the final visual acuity of an eye affected by venous occlusion was approximately the same as the visual acuity at the outset. They considered that anticoagulant therapy as then available was not justified; one reason being that there is considerable difference of opinion whether the obstruction of the vein is in fact due to thrombosis or to endothelial or intimal

thickening. This problem has still not been resolved.

Gayer Morgan (1955) stressed the value of the use of anticoagulants in the prophylaxis of retinal vein thrombosis, illustrating this point by reference to cases in which dilated or tortuous retinal veins were noted in the good eye, the other eye having been affected by a full-blown thrombosis. Stephenson (1956) reported some success in treating cases of partial thrombosis of a retinal vein, but considered that anticoagulants were of no value in cases of complete thrombosis.

Vannas & Orma (1957), reporting on patients with central vein occlusion and those with main branch occlusion, stated that those treated with anticoagulants, mostly heparin and what they term antisclerosis therapy, did much better (59% in the treated) than the control group (14%) both from improved vision and field. Also fewer in the treated group developed secondary glaucoma. My cases certainly do not attain the high percentage of success which these workers obtained.

The association of retinal vein occlusion and primary glaucoma has been recognized for many years. Redmond Smith (1955) drew attention to the occurrence of central retinal vein occlusion, as a complication of a pre-existing glaucoma, and Dobree (1957), in a series of 200 cases of primary glaucoma, found obstruction to the venous return at the disc in 15·5%. More recently Vannas & Tarkkanen (1960), using tonography and provocative tests in a series of 71 patients with central vein occlusion, found 42% to have primary glaucoma in both eyes, and that those patients treated with long-term anticoagulant therapy had a better final visual result than those untreated.

About two years ago, I was stimulated to see if it were possible to tell, in my hands, if anticoagulant therapy was of any use. At the outset I must say that there is nothing particularly new in this short paper. It describes the results of treatment with anticoagulants of a series of cases of retinal vein occlusion, and compares these results with a control series treated without anticoagulants. I had intended to treat alternate patients but in practice this was not possible owing partly to the difficulty of arranging for the necessary prothrombin level tests to be done, because of the distance of the patients' homes from the laboratory (and I was anxious to treat all cases as out-patients) and partly to the wish of some of the patients' doctors that this form of treatment should not be given. So the control cases and treated cases were selected more by chance, but in effect I do not think that this has weighted the evidence for or against this form of treatment, except that it has resulted in only a few of the cases with branch vein occlusion being in the treated group.

Method of Treatment

The drugs used are heparin, phenindione (Dindevan) and warfarin (Marevan). Heparin is used only where the thrombosis is probably of very recent origin, i.e. under twenty-four hours. It is used in doses of 7,500–10,000 i.u. intravenously, six-hourly for one to two days.

The same day or next day, the patient is started on Dindevan, initial dose of 150 mg on the first day, 100 mg on the second day and a maintenance dose of 50–150 mg a day. Dindevan acts fairly quickly in lengthening the prothrombin time (36 hours). The aim is to maintain a level of two-and-a-half times the normal, i.e. a prothrombin time of 30 seconds.

The following factors must be taken into account in assessing the dosage: (1) Weight – generally the heavier the patient the larger the dosage. (2) Ill patients require smaller dosage. (3) The older the patient the smaller the dose necessary. (4) Deficient renal function, leading to delayed excretion of the drug, indicates smaller dosage.

Patients are warned not to take salicylates or aspirin while on anticoagulants. Liver disease is an absolute contraindication. I have left the regulation of the dosage entirely to the pathologist, and I am much indebted to Dr J Horley for his help. Patients who show intolerance to Dindevan in the form of diarrhœa or dermatitis are changed to Marevan 3–5 mg b.d.

The treatment is continued for many weeks, even up to a year or more. In this series I have found little further improvement after six months, but in a number of cases I have continued with treatment after this, with the idea of preventing a thrombosis occurring in the fellow eye, where there has been well-marked sclerosis of the retinal vessels and some evidence of venous congestion.

For the first few weeks it is necessary to estimate the prothrombin time of the patient's blood once or twice weekly, and as the treatment becomes stabilized, the interval between blood tests can be lengthened to a month.

Assessment of Results

The assessment of the results of treatment in this condition is difficult, owing to the variability in degree of recovery of vision, but the following criteria seem to be the most useful for this purpose: (1) Visual acuity, distance, and if possible, reading. (2) Fields and Amsler charts. (3) Absorption of hæmorrhages. (4) Development of thrombotic glaucoma.

For the purposes of this paper and for reasons of brevity I have condensed all these factors together in estimating whether a case has improved, has shown no change or has become worse.

Material

The series consists of 50 consecutive cases of retinal vein occlusion seen and treated by me between July 1959 and September 1960. This number shows that the disease is fairly common in our practice. 17 (34%) were male and 33 (66%) female. Duke-Elder (1940), however, gives the incidence between the sexes as equal.

The age groups are shown in Table 1, and they conform to the usual pattern.

Table 1
Age and Sex

	40–49	50–59	60–69	70–79	80+	Total
Male	4	1	5	6	1	17
Female	3	4	10	13	3	33
Total	7	5	15	19	4	50

Twenty-eight (56%) of the patients had a diastolic blood pressure of 100 mm Hg or over; a few more had systolic pressures over 200 mm Hg, but diastolic readings below 100 mm Hg. Only 4 (8%) of the cases had undoubted primary glaucoma in the fellow eye, but this figure is probably too low, as tonography and detailed provocative tests were not carried out in the majority of cases.

Results (Tables 2, 3 and 4)

Table 2
All cases

	Anticoagulants (22)		*Controls* (28)	
	Cases	%	*Cases*	%
Improved	8	36	8	28·5
No change	11	50	12	43
Worse	3	14	8	28·5

Table 3
Central Vein (29 Cases)

	Anticoagulants (17)		*Controls* (12)	
	Cases	%	*Cases*	%
Improved	6	35	3	25
No change	9	53	6	50
Worse	2	12	3	25

Table 4
Branch Vein (21 Cases)

	Anticoagulants (5)		*Controls* (16)	
	Cases	%	*Cases*	%
Improved	2	40	5	31
No change	2	40	6	38
Worse	1	20	5	31

Three cases out of the 12 in the central vein control series developed thrombotic glaucoma, whilst none of the 17 treated cases did so.

Complications arising possibly directly from the anticoagulant therapy were:

(1) *Hæmorrhage:* Hæmaturia, 3 cases. Epistaxis (slight), 1 case. Skin bruising, 2 cases, 1 of which also had hæmaturia and the treatment had to be stopped after six weeks. Vitreous hæmorrhage, 1 case; possibly due to the thrombosis itself; it cleared within three months without the dosage being reduced.

(2) *Diarrhœa*, severe in 1 case, slight in 3; all stopped on changing to Warfarin.

(3) *Indigestion* occurred in a few cases but in none was it severe.

(4) *Dermatitis*, slight in 2 cases.

Recently, a few patients have volunteered the information that they have a dry mouth and nasty taste in their mouth when on Dindevan, but not on Marevan.

Conclusions

The numbers in this series may not be sufficient to form a very firm opinion as to the value of anticoagulants in retinal vein thrombosis, but I think the results do show that those patients having the treatment are more likely to improve than those without it, and are also less likely to suffer from thrombotic glaucoma. Only one patient in the whole series actually developed venous occlusion in the second eye, and although treated with anticoagulants she has done badly. It is not possible, on the data available here, to say whether or not this treatment can prevent a partial or suspected thrombosis from becoming a complete occlusion, but I maintain that the treatment of such cases is worth doing, and will not do harm.

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Mr E S Perkins (*London*) agreed that it was important to decide on the value of anticoagulant therapy. He had recently treated a case of partial central vein thrombosis with anticoagulants for one month, the vision in the affected eye remaining at 6/18. Two weeks after stopping treatment – owing to difficulty in controlling the prothrombin level – a further thrombotic episode occurred, reducing vision to 6/60. It seemed likely that had the treatment been continued for a longer period this relapse would have been prevented.

Mr Perkins asked Dr Thorne Thorne for what length of time anticoagulant therapy should be given in these cases.

Mr E F King (*London*) asked if Dr Thorne Thorne had at any time noticed, in a retinal venous thrombosis, an increase of hæmorrhages following anticoagulant therapy.

Mr Redmond Smith (*London*) pointed out that it was vital to know whether the apparent better results in the treated groups had been subjected to any statistical tests. Although the numbers were small, it was quite possible that the differences might be significant.

Mr J Primrose (*London*) stated that one of the first principles of medicine was that the treatment should not do the patient harm. Mr King had already mentioned the risk of further hæmorrhages resulting from anticoagulant treatment of central retinal vein thrombosis. Mr Primrose reported a case he had under such treatment who developed further hæmorrhages and a fulminating glaucoma, from which the patient lost his eye.

Mr T Keith Lyle (*London*) thought one of the greatest values of the treatment described by Dr Thorne Thorne was the prevention of thrombotic glaucoma.

He asked Dr Thorne Thorne if, in cases of tributary thrombosis, he had differentiated between those involving the nasal tributaries and those of the temporal tributaries. Where the macular region was spared, as in the case of thrombosis of either of the nasal tributaries, the central visual acuity was obviously likely to be unaffected.

Mr John A Chivers (*Orpington*) stated that he had at present a patient under treatment with a partial thrombosis of the central retinal vein in an aphakic eye, in which he had noticed that the hæmorrhages had increased as soon as the anticoagulation treatment with heparin and Dindevan was begun, and that in this case he had reduced the Dindevan so that coagulation of the blood was not reduced fully, but only to about 17% (Owren's technique – normal therapeutic range 10% to 25%). After this no new hæmorrhages appeared and after a fortnight's treatment the corrected vision improved to 6/36.

Dr Thorne Thorne, in replying to Mr King, stated that in at least one patient the hæmorrhages increased after starting anticoagulants; but increase of hæmorrhages had occurred more frequently in cases not having anticoagulants, and he thought that this sequel was due to the disease rather than to the treatment. This also answered Mr Primrose's point.

In answering Mr Redmond Smith's question, Dr Thorne Thorne stated that a statistical analysis had been obtained; the result of this had shown that there was little difference in the outcome of the treated and the control groups. However, the statistician had commented that the conclusions drawn were cautious and not open to criticism from the statistical aspect.

In reply to Mr Keith Lyle, Dr Thorne Thorne said that a review of the cases had shown that the majority of branch vein thromboses had been of the superior or inferior branches, thus involving the temporal and nasal tributaries; only two of the cases were of the nasal branch alone.

The Clinical Value of Ophthalmodynamometry

by Vernon H Smith FRCS (*Manchester*)

Recent experience has suggested that ophthalmodynamometry has a useful part to play in the diagnosis of cerebrovascular and retinal vascular disease (Liversedge & Smith 1961). The present report is based on experience in the dynamometry clinic at the Manchester Royal Eye Hospital during the past year. During this period 273 patients have had dynamometry performed on them, of whom 145 were referred from the Manchester Royal Infirmary, and 128 from the Manchester Royal Eye Hospital. Among them there were 39 with carotid occlusion and 58 with central retinal artery occlusion.

Theoretical Considerations

Ophthalmodynamometry provides a measurement of the pressure in the ophthalmic artery in terms of the grams of extraocular pressure that are required to produce a maximal pulsation at the disc for the diastolic pressure, and the moment that this disappears for the systolic pressure. These figures are not to be regarded as a true measurement of the pressure in the ophthalmic artery, but are only of value for comparison between the two eyes of one patient. A difference in the ophthalmic artery pressures as shown by dynamometry may be expected in the following conditions:

(1) *Carotid occlusion:* A fall in pressure in the artery distal to the site of occlusion might be expected. This would be followed by a corresponding fall in the ipsilateral ophthalmic artery. Dynamometry would detect this, but would have no localizing value for the actual site of the obstruction. Furthermore, if the collateral circulation was well developed – as in a longstanding case, then it is possible that this may have completely compensated for the occlusion and dynamometry would be normal.

Case 1 Mr H E, aged 60

In 1955 this patient was diagnosed as suffering from stenosis of the left internal carotid artery, and at that time a carotid angiogram (Fig 1) showed a stenosis of the carotid just above the bifurcation with good flow up the external carotid and a rich anastomosis between the branches of this artery and those of the ipsilateral ophthalmic artery. Ophthalmodynamometry performed almost five years later gave exactly equal results on both sides.

Dynamometry would also be negative in cases where the obstruction to the flow was as yet not sufficient to cause a drop in pressure.