British Journal of Ophthalmology, 1986, **70**, 918–924

Estimation of visual function after optic neuritis: a comparison of clinical tests

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SUMMARY A group of 53 patients who had suffered an attack of unilateral (n=45) or bilateral (n=8) optic neuritis more than six months before were subjected to a battery of tests to determine their spatial contrast sensitivity, visual field, and colour vision. The 106 eyes investigated were classified according to their clinical status and visual acuity at the time of the study into unaffected (n=45), recovered (n=33), and non-recovered (n=28). At least one of the three tests gave an abnormal result in 67%, 88%, 100% of the three groups respectively. The results obtained with these three tests showed a significant statistical association.

Patients with optic neuritis (ON) generally have a good short-term prognosis as regards visual acuity as tested by Snellen types.[1 2] Normal vision is recovered in 50 to 78% of the patients within six months. However, in some cases ON produces long-lasting objective and/or subjective visual dysfunction.[3 4] There are strong indications not only clinically but also electrophysiologically that ON causes impairment of the optic nerve which may persist for a long time after the initial attack.[5–10]

Several authors claim that Snellen's test and similar tests of visual acuity measure only a limited aspect of visual perception,[11–13] since it has been found that the visibility of objects depends not only on their sizes at high contrast but also on their luminance relative to the background.

Optic neuritis and multiple sclerosis (MS) patients have been found to have abnormal contrast sensitivity in combination with normal visual acuity.[14–17] This suggests that the Snellen test of visual acuity may be an inadequate measure of visual function in investigations of the degree of recovery after ON.

In the present study clinical tests of visual function (including determination of spatial contrast sensitivity, colour vision, and visual field) were applied to a group of 53 persons, who had an attack of ON from six months to 3·5 years before the start of the study. The main aim was to determine which combination of these tests gives the best measure of visual function after ON, especially in those cases where the Snellen visual acuity is $\geq 1{\cdot}0$, suggesting (nearly) complete recovery of visual function. Since there is disagreement about the presumed higher risk of MS after bilateral optic nerve involvement,[3 18] we performed the above set of tests on both eyes of all test subjects, to see whether these tests offered a sensitive means of detecting asymptomatic optic nerve involvement.

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Patients and methods

PATIENTS

Fifty-three patients (40 female, 13 male) treated for acute ON at the Eye Clinic of Erasmus University, Rotterdam, between 1980 and 1983 were re-examined at the same clinic in 1984 for the present study. The diagnosis of ON was mainly based on the presence of features as summarised by Glaser.[19] A pattern visual evoked response (PVER) was recorded in order to support the diagnosis electrophysiologically. The patient's age at the time of the study ranged from 17 to 54 years (mean 32 years).

Eleven patients had had a relapse of ON within the period from 1980 to 1984. In eight of them the recurrence affected the contralateral, initially unaffected eye, while in the other three cases the relapse was on the ipsilateral side. At the time of this investigation 29 of the 53 patients had clinical signs and symptoms of mild central nervous system (CNS) involvement affecting structures other than the optic

Table 1 *Classification of eyes*

Classification	*Clinical signs and symptoms in this eye during ON attack*	*Snellen visual acuity during present study*
Unaffected (n=45)	No	>1·0
Recovered (n=33)	Yes	>1·0
Non-recovered (n=28)	Yes	<1·0

nerve, which was regarded as justifying the diagnosis of clinically probable or possible MS as defined by the criteria of McAlpine *et al.*[20]

The eyes investigated were classified on the basis of clinical status and Snellen visual acuity as unaffected, recovered, and non-recovered on the basis of the criteria shown in Table 1.

METHODS

Contrast sensitivity was determined with equipment based on the modified Von Bekesy tracking method.[21] Vertical sinusoidal gratings presented on a television monitor at a mean luminance of 5 cd/m^2 were used as stimuli. The contrast sensitivity was determined in the spatial frequency range from 0·1 to 25·6 cycles per degree (c/deg). The stimulus was 8° wide for the range 0·8 to 25·6 c/deg and 32° wide for the range 0·1 to 0·4 c/deg to prevent the contrast sensitivity from being influenced by two small a number of cycles at low spatial frequencies.[22-24] To prevent afterimages the grating was counterphased every 0·8 s. Further details have been described previously.[25]

The abnormal range of contrast sensitivities was determined on the basis of measurements on both eyes of 26 controls—that is, persons without known subjective or objective ophthalmological complaints. The control group was age and sex matched with the ON group. A contrast sensitivity more than 2 standard deviations below the normal value in the low (0·1 to 0·4 c/deg), medium (0·8 to 3·2 c/deg), or high (6·4 to 25·6 c/deg) spatial frequency range was considered as abnormal.

Visual acuity was determined with a Snellen chart and expressed as the reciprocal of the mean angle of resolution in minutes of arc. A visual acuity of 1·0 or more was considered normal.

Colour vision was examined under standard conditions by the Hardy Rand Rittler (HRR) and desaturated panel D-15 tests. Normal values were obtained in 25 control subjects according to the code of Verriest.[26] The results of the HRR and panel D-15 tests showed a very strong statistical association ($p<0·001$). We therefore decided to pool the results of these two tests for the purposes of comparison with the other measures of visual function considered in this study.

Table 2 *Incidence of abnormal contrast sensitivity function in all eyes investigated*

Spatial frequency range	*Incidence of significantly reduced contrast sensitivity in:* *Unaffected eyes*		*Recovered eyes*		*Non-recovered eyes*	
	No.	*%*	*No.*	*%*	*No.*	*%*
Low	2	4	2	6	0	0
Medium	0	0	0	0	1	4
High	6	13	3	9	2	7
Low+medium	0	0	1	3	1	4
Low+high	0	0	1	3	2	7
Medium+high	0	0	1	3	1	4
Low+medium+high	4	9	5	15	19	68
Total	12	27	13	39	26	93

Visual fields were determined with the 'computerised octopus display'.[27] Visual field defects were classified as either central or paracentral.

Clinical ophthalmological and neurological examinations were performed on all 53 patients. During these examinations the patients were asked whether they had only subjective visual complaints.

Statistical analysis was performed with the SPSS computer program. Differences between sets of experimental data were tested for significance by the Wilcoxon, χ^2, and Kendall's t-B and t-C or by regression analysis.

Results

CONTRAST SENSITIVITY

The mean contrast sensitivity curves for all three groups of eyes (unaffected, recovered, and non-recovered) were lower than normal, that for the non-recovered eyes being lowest (Fig. 1). The highest incidence of contrast sensitivity abnormalities (93%) was found in the impaired eyes, the corresponding figures for the recovered and unaffected eyes being much lower (39% and 27% respectively; Table 2). The abnormality most frequently found in the overall group of eyes with decreased contrast sensitivity (n=51) was a reduction in contrast sensitivity for all spatial frequency ranges investigated (n=28; Table 2).

VISUAL ACUITY AND CONTRAST SENSITIVITY

As may be seen from Fig. 2, a significant correlation was found between visual acuity and contrast sensitivity in each of the three frequency ranges employed, the correlation coefficients for the high, medium, and low ranges being 0·59, 0·65, and 0·71 respectively. A significant correlation (r=0·68) was also found between the visual acuity and the grating acuity (that is, the spatial frequency at which the

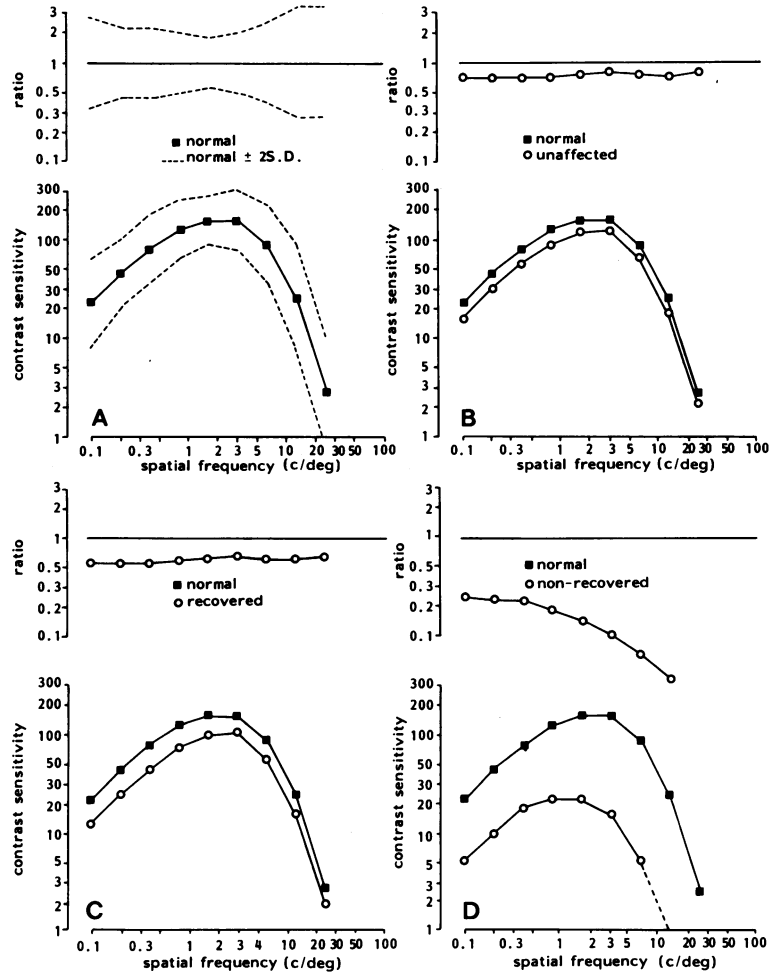

Fig. 1 *Contrast sensitivity as a function of spatial frequency for (A) normal eyes, (B) unaffected eyes, (C) recovered eyes, and (D) non-recovered eyes. The curve for normal eyes is repeated for the sake of comparison in each of Figs. 1B, C, and D, and the ratio of the contrast sensitivity for the group of eyes in question to that for normal eyes is plotted against the spatial frequency at the top of each graph. In Fig. 1A the broken lines represent the confidence limits (mean value ± twice the standard deviation); the ratio of these limits to the mean value is also plotted at the top of this graph.*

extrapolated contrast sensitivity equals 1·0). This is also shown in Fig. 2.

COLOUR VISION AND VISUAL FIELD DEFECTS IN RELATION TO CONTRAST SENSITIVITY

Significant associations were found between reduced contrast sensitivity in the low, medium, and high spatial frequency ranges and defects of colour vision and visual field (Table 3). Moreover, central and paracentral visual field defects also showed an association with reduced contrast sensitivity when considered separately. This is probably due to the fact that these two types of visual field defects were likewise statistically associated ($p<0·001$). Furthermore, colour vision disorders showed a significant association with visual field defects ($p<0·001$).

ASYMPTOMATIC OPTIC NERVE INVOLVEMENT

Table 4 surveys the results of all clinical and paraclinical examinations of the unaffected, recovered, and non-recovered eyes. All non-recovered eyes (n=28) and 88% of the recovered eyes (n=33) had at least one defect. Even 30 (67%) of the eyes that had never been attacked by ON at a clinical level showed one or more visual abnormalities. This asymptomatic optic nerve involvement could perhaps be explained by demyelination in the contralateral optic nerve. In other words there is reason to believe that many of our patients were actually suffering from bilateral optic nerve involvement.

Since bilateral ON is thought to be associated with a high risk of subsequent development of MS,[3] it is instructive to see how many of our patients did actually have signs and symptoms of MS at the time of the present study. In fact 29 of our 53 patients showed signs and symptoms of mild CNS involvement affecting structures other than the optic nerve (limb paraesthesiae 38%, limb paresis 18%, diplopia 12%,

cerebellar ataxia 9%, bladder dysfunction 9%, Lhermitte's sign 6%, facial paresis 3%). Asymptomatic optic nerve involvement tended to be found more often in the patients who had signs and symptoms of MS (18 out of 29). However, this tendency is not statistically significant (χ^2 test yielded $p>0{\cdot}05$ for all combinations; not shown in the tables).

SUBJECTIVE VISUAL SYMPTOMS AND CONTRAST SENSITIVITY

Subjective complaints were mentioned by 38 patients and attributed to 42 out of 106 eyes. They were of blurred or misty vision in 32, colours which seemed paler than normal in 20, and silhouette vision in 11. Contrast sensitivity was decreased in 30 of these 42

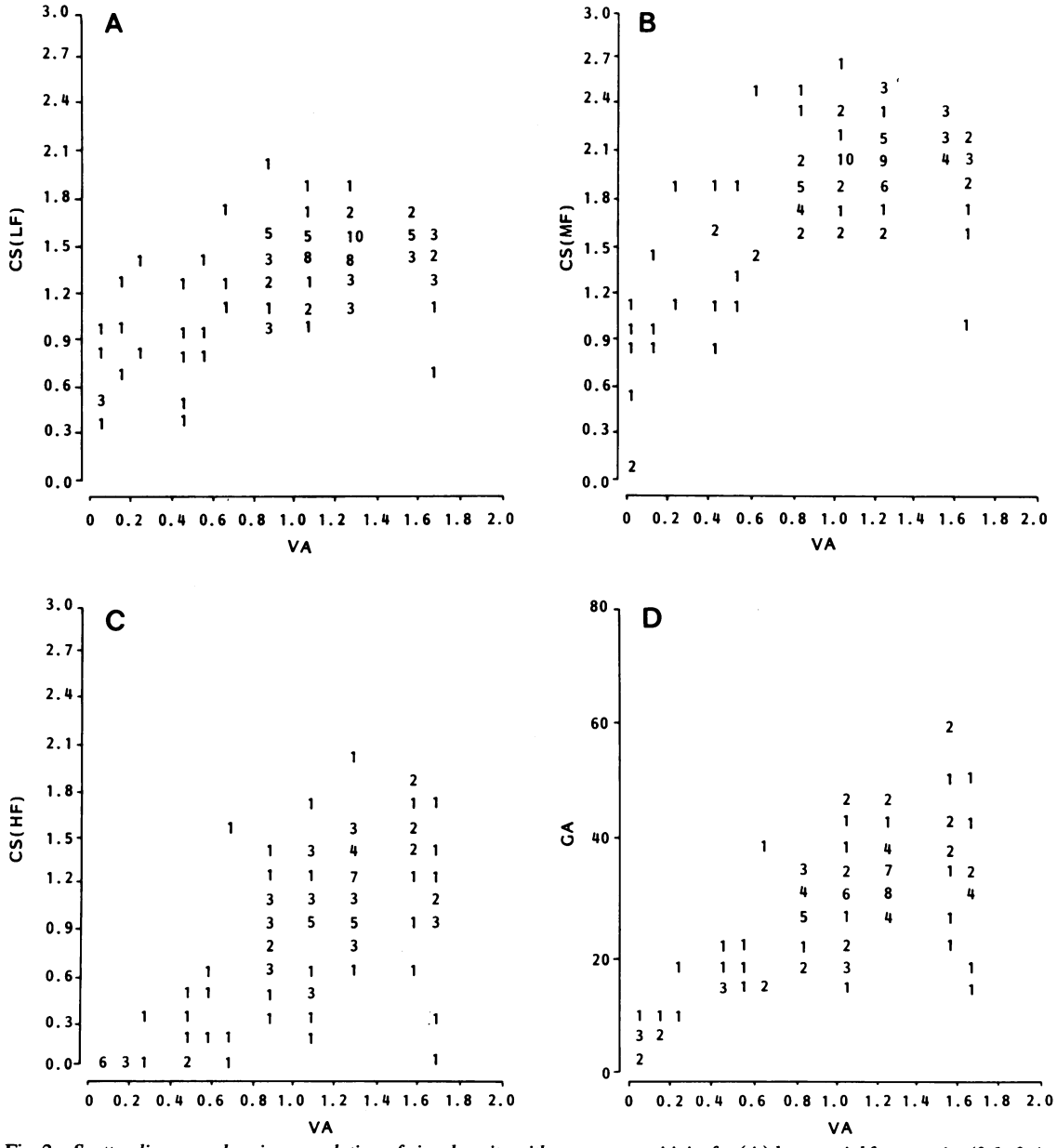

Fig. 2 *Scatter diagrams showing correlation of visual acuity with contrast sensitivity for* (A) *low spatial frequencies (0·1–0·4 c/deg),* (B) *medium spatial frequencies (0·8–3·2 c/deg), and* (C) *high spatial frequencies (6·4–25·6 c/deg).* D: *correlation of visual acuity with the 'grating acuity'—that is, the spatial frequency at which the extrapolated contrast sensitivity equals 1·0.*

Table 3 *Association between the various visual tests and abnormal contrast sensitivity*

Number of eyes with			*Incidence (%) of significantly reduced contrast sensitivity, in the*		
			Low spatial frequency range	*Medium*	*High*
Defective colour vision:	Yes	33	61	61	64
	No	73	23	18	32*
Reduced visual field:	Yes	31	65	65	71
	No	71	21	13	28
Subjective symptoms:	Yes	42	55	60	64
	No	64	22*	13	27

*In these cases the association between reduced contrast sensitivity and the clinical symptom in question is significant at the $p<0.01$ level; in all other cases shown in this table this association is significant at the $p<0.001$ level.

(71%). The other tests yielded lower percentages of abnormalities in the eyes associated with subjective complaints: colour vision was affected in 62% and the visual field in 56%. Diminished contrast sensitivity and defects of colour vision and of the visual field showed a statistically significant association with subjective complaints ($p<0.001$; not shown in the tables).

COMBINATION OF TESTS

As may be seen from Table 5, more defects of visual function can be detected if two or more tests were combined. In the non-recovered group 19 of the 27 eyes (70%) gave abnormal results in all three tests, while all eyes in this group gave abnormal results in one or more of the three tests. At the other extreme, in the 'unaffected' group none of the eyes gave abnormal results in all three tests, and only 19 out of 44 (43%) showed signs of a visual defect in at least one test.

Table 4 *Incidence of abnormal visual tests in unaffected, recovered, and non-recovered eyes*

Function tested	*Incidence of abnormalities found in:*		
	Unaffected eyes (n=45), %	*Recovered eyes (n=33), %*	*Non-recovered eyes (n=28), %*
Clinical			
Pallor of disc	9	76	93
Visual field			
Central scotoma	4	15	33
Paracentral scotoma	2	12	71
Colour vision			
HRR	7	18	71
Panel D-15	13	12	68
Subjective complaints			
Fogginess	7	30	68
Pale colours	0	15	54
Other	0	0	29
Reduced contrast sensitivity	27	39	93
Total*	67	88	100

*This total is not equal to the sum of the figures given above, as many eyes showed more than one abnormality.

Discussion

INCIDENCE OF TEST ABNORMALITIES IN THE THREE GROUPS OF EYES

The first question we set out to answer was whether these tests, alone or in combination, provide us with more information than Snellen's test of visual acuity. The short answer is Yes. As may be seen from Table 5, 100% of the non-recovered eyes (visual acuity <1·0) showed abnormalities in one or more of the three tests. Our data for these non-recovered eyes are comparable with published findings on ON in the acute stage: disturbed contrast sensitivity in 63 of 100%,[15 28] visual field defects in 61 to 92%,[29] colour vision defects in 63 to 99%.[30 31]

The overall incidence of test abnormalities (55%) in the recovered eyes (visual acuity >1·0) is naturally lower than in the non-recovered eyes. The only comparable findings in the literature are those of Kirkham and Coupland,[32] who found 53% colour vision abnormalities in 93 patients. However, these authors gave no information about visual acuity in

Table 5 *Cumulative incidence of the combination of test abnormalities in unaffected, recovered, and non-recovered eyes*

Function tested	*Incidence of abnormalities found in:*		
	Unaffected eyes, %	*Recovered eyes, %*	*Non-recovered eyes, %*
Colour vision	14	19	74
Visual field	5	19	85
Contrast sensitivity	27	39	93
Colour vision *and* visual field	0	6	70
Colour vision *and* contrast sensitivity	2	10	74
Visual field *and* contrast sensitivity	—	10	78
Colour vision *and* visual field *and* contrast sensitivity	—	3	70
Colour vision *or* visual field	18	32	89
Colour vision *or* contrast sensitivity	39	48	93
Visual field *or* contrast sensitivity	32	48	100
Colour vision *or* visual field *or* contrast sensitivity	43	55	100

their patients and did not test the visual field or the contrast sensitivity. Some other authors[16 29 33 34] had also reported that suitable tests could detect visual defects in eyes that showed clinical recovery from ON; however, these studies concerned smaller numbers of patients.

A more surprising result was obtained in the clinically unaffected eyes in our study. No fewer than 19 of these 45 eyes (43%) showed at least one abnormal result in our three visual function tests (Table 5). The contrast sensitivity yielded the highest frequency of abnormalities in all groups and the highest association with subjective complaints.

ASYMPTOMATIC OPTIC NERVE INVOLVEMENT

The finding of 'subclinical' abnormalities in 43% of the 'unaffected' eyes leads us to a second question: is bilateral optic nerve involvement more indicative of MS than unilateral involvement? On the basis of the evidence gathered during the present study the answer to this question must be No. Five of the eight patients who had suffered clinical attacks on both eyes (see under 'Patients' above) belonged to the group of 29 patients with mild CNS involvement, while the other three patients with bilateral attacks showed other signs, attributable to multiple CNS lesions. A similar picture was obtained in the 30 'unaffected' eyes showing one or more abnormalities in one of our three visual function tests or on clinical examination or associated with subjective complaints (Table 4); only 18 from this group were associated with any signs of other CNS structures. These data stress the evidence that bilateral optic nerve involvement, either simultaneous or sequential, does not include a higher risk of development of MS. These data only partly support the report of Parkin *et al.*, [18] who found a higher risk after bilateral simultaneous ON but not after bilateral sequential ON.

ASSOCIATIONS BETWEEN THE TESTS

Contrast sensitivity abnormalities were found in 39% of the recovered eyes in our study and in 27% of the unaffected eyes. This confirms earlier findings that normal visual acuity on Snellen testing (1·0 or more) need not always coincide with normal contrast sensitivity.[14-16] However, none of the previous authors subjected their results to statistical analysis in view of the relatively low numbers of patients involved. As shown in Fig. 2, we found a statistically significant correlation between visual acuity and contrast sensitivity in all three spatial frequency ranges. This finding seems at first sight paradoxical in view of our observation, mentioned above, that an appreciable proportion of persons with normal visual acuity (>1·0) nevertheless show a marked drop in contrast sensitivity.

However, the following consideration may help to reconcile these two sets of observations. The results shown in Fig. 2 seem to establish that there is a general tendency for a higher visual acuity to be associated with a higher contrast sensitivity. But there is a very appreciable variation about this trend, so that if we consider only good eyes, or consider only bad ones, the trend is not persistent in relation to all eyes investigated and results plotted in one curve (Fig. 2). It is only when we consider a population with a sufficiently wide range of visual function that the statistical significance of the trend becomes clear. It now becomes understandable that, in view of the above mentioned spread in the results, many eyes may have a significantly low contrast sensitivity.

The presence of the spread in the correlation curves of Fig. 2 allows a further conclusion to be drawn, namely, that persons with a given visual acuity as measured by the Snellen chart may have an appreciably worse (or appreciably better) contrast sensitivity than might be expected. This would seem to indicate that visual skills other than the perception of contrast play an appreciable part in the correct perception of the letters on the Snellen chart.

This conclusion is in line with the claim by Campbell[11 12] and Wollner and Diamond,[13] mentioned above; Snellen's test and similar tests of visual acuity measure only a limited aspect of visual perception.

The significant association we observed between reduced contrast sensitivity and defective colour vision and visual field (Table 3) is hard to understand, since each of these various types of defect may be expected to arise by a different physiological mechanism. The common factor in all these cases might possibly be a demyelinative attack on the optic nerve during the ON episodes.

The authors are indebted to Dr R H Bathgate (Eindhoven) for critically reviewing this paper and Mrs Ch J Th Sanders-Bozon for preparing the manuscript.

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Accepted for publication 21 February 1986.