tial fibre loss in the smaller range that would be expected to come from small sensory neurons.

How much of the neuronal damage was caused by cisplatin and how much by adriamycin cannot be stated, of course, but adriamycin has always been said by clinicians not to produce a sensory neuropathy; but the ability to detect clinical damage to small sensory neurons is rather limited. From what we know of the mode of action of this and similar agents it is possible that this insensitivity to adriamycin toxicity is more apparent than real. Only careful future studies will show where the truth lies.

JB CAVANAGH
Institute of Neurology,
Queen Square
London WC1N 3BG, UK

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Visual field rehabilitation in the cortically blind?

Sir: Recently, Balliet *et al*¹ reported that, in contrast to our work, they were not able to find any effect of specific treatment on visual field recovery in 12 patients suffering from

homonymous visual field loss due to unilateral ischaemic or embolic posterior artery infarctions. As it stands, the paper deserves some comment because Balliet *et al* obviously misinterpreted our data and our discussion, drawing misleading inferences about our results from their data. We called Balliet *et al*'s attention to this discrepancy in detail in a personal letter in July 1983 which Balliet *et al* ignored completely. We would like, therefore, to clarify some major points.

1 It seems as if Balliet *et al* concluded from our work that every patient suffering from homonymous field loss could be treated successfully, otherwise one cannot understand why they concluded from their data that "visual field increases are not trainable". This generalised conclusion has never been drawn by us. When we started this investigation in 1976 our main interest was to find out whether field defects can *in principle* be reduced by treatment. The question as to how frequent and to what extent training effects can be expected in a large group of patients is another aspect; while perhaps important from the clinical and rehabilitative viewpoint, it must not be confounded with the first, more fundamental one. This latter aspect has been addressed by us in two recent papers.^{2,3} We can, therefore, not see how Balliet *et al*, on the basis of their data can conclude, that visual field defects cannot be treated at all. They would have been correct in stating that in their 12 patients they were not able to find any effect. In the light of our own data this is by no means a surprise, because (as pointed out above) one cannot expect field enlargement in every patient showing homonymous field loss.

It has long been known that irreversible damage to the striate cortex and its afferents (optic tract, optic radiation) causes permanent blindness. On the other hand, observations on spontaneous recovery and recovery after systematic treatment in patients with cerebral damage suggest that in some cases the functional loss may, at least in part, be due to reversible damage. Unfortunately the nature of structural damage in terms of irreversible/reversible, at least at the moment, cannot be determined reliably. Therefore, it is impossible to predict which patient may benefit from treatment and which not. In a recent article² we reported that return of visual field portions were observed only in cases with incomplete damage, as evidenced by, for example, lowered CT density values, whereas in cases with complete damage (pseudocystic necrosis) we could not find any evidence for visual field enlargement.

2 Having failed to find any effect of their method of visual field training Balliet *et al* conclude that our results may be due to artifacts resulting from "any combination" of (a) large stimuli variability in perimetric testing, (b) changes in detection strategies with practice and (c) eccentric fixation. These assertions can easily be tested for since (a) large stimulus variability as well as changes in detection strategies should lead to inconsistent shifts of the border of the visual field (including artificial enlargement as well as shrinkage) and (b) eccentric fixation would result in an overall shift of the field border towards the affected side.

Regarding measurement variability, the range of perimetric measurement variability in our studies was $\pm 0.5^\circ$ within 15° eccentricity, and $\pm 1.75^\circ$ beyond which is in good agreement with other authors (see ref 3). Considering this range of measurement variability one is surprised to learn that Balliet *et al*, in their study, found variations up to 40° (mean: 15°) but it is even more astonishing that these authors, on the basis of their measurement variability, conclude that our data can be explained on the basis of their inaccuracy in perimetric testing. Patients with such large variations would never have been included in our series. However, even if we accept the variability Balliet *et al* found for their normals in the far periphery of the visual field (between 1° and about 5°) we are not in a position to explain the field enlargement in our studies simply as measurement variability because field enlargement was in many of our cases much larger. Eccentric fixation, which may be additive, as Balliet *et al* suggest, but did not test, thus leading to an artificial field increase in the perimeter, can also not explain our results nor can changes in head position. In both cases one would expect that visual field should increase along the whole field border or at least along each meridian where training has been performed. Our data show clearly that this is not the case. In contrast, recovery was incomplete in all cases and field enlargement was limited to particular field regions and was not observed in any field portion subjected to training which should be the case if Balliet *et al* were right. Furthermore, as Teuber *et al*³ have shown, the blind spot should also change its position in correspondence with eccentric fixation. However, we did not find any evidence for such a change.² Finally one should, in addition, also keep in mind that any combination of these factors could also lead to changes in the reverse direction, that is, to a reduction of the extent of visual field (see, for example reference 4).

In the light of this evidence Balliet *et al*'s generalised conclusion that, based on their data, visual field regions cannot in principle be recovered by systematic treatment must be regarded with caution. Furthermore, from a scientific point of view, any replication of an investigation depends critically upon the degree of correspondence with the original study. Differences regarding methods of measurement, treatment and patient variables lead, depending on the influence of these factors on the expected result, either to a failure to replicate the study in question or reduce at least the probability of obtaining similar results. Balliet *et al* report that their methods "are not an exact replication" of ours, but they fail to discuss further the consequence of essential methodological differences between their study and our investigations. In addition, they did not even mention that their failure to find any treatment effect could also be explained in terms of differences in the group of patients participating in both studies. Obviously in order to be able to replicate our findings the most crucial variable is the selection of patients belonging to the group with a good prognosis for treatment, that is, with only reversibly lost field regions. Any attempt at treatment of, for example a hemianopia resulting from irreversible damage, must fail. Lest there be any misunderstanding: we do not suppose (and our data support this assumption) that there exists "neural plasticity in the visual system" in the sense that any functional impairment caused by brain damage can be reduced irrespective of the "quality" of damage in terms of reversible/irreversible. Thus, one cannot take every patient with homonymous field loss for treatment and expect that his field defect may shrink. Regarding the significance of visual field recovery for rehabilitation we can say now that in single cases it is of potential value, but we have pointed out that for the majority of patients the appropriate method of treatment is the use of compensation strategies.⁵

In conclusion, the study reported by Balliet *et al* is by no means a replication of our studies and their data can, as discussed above, rather easily be explained simply by the fact that their patients suffered from irreversibly lost field regions which cannot be influenced by treatment. In this sense they support partially our findings in that not every patient is a "good candidate" for visual field treatment. Thus, there is no compelling need for us, as they did with our study, to have recourse to various untested sources of possible artifacts, in order to understand the outcome of their study.

J ZIHL
D VON CRAMON
Max-Planck-Institut für Psychiatrie,
Department of Neuropsychology,
Kraepelinstr. 10
D-8000 München 40, FRG

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Balliet *et al* reply:

In science, proving that something exists is often as difficult as proving that something does not exist. In either case the primary objective must be to provide proof in a manner that has no possibility of artifact. This is a primary issue in research and discussions pertaining to the question of whether or not it is possible to retrain the visual fields of hemianopic individuals.

Unfortunately, we are not privy to "exactly" how Zihl and von Cramon tested and trained their hemianopic subjects to supposedly enlarge their visual fields. The best we can do is, based on their written reports, try to replicate their results under conditions where potentially confounded cues or variables are eliminated. In our report of this work *Visual field rehabilitation in the cortically blind*¹ we concluded that "when we controlled or made allowance for these variables, no significant visual field increases were found in our cortically blind patients. Until additional studies which control for compensatory strategies are brought forth, we must support the opinion that lesions to the striate cortex in humans result in permanent blindness." Unfortunately, nothing in Zihl and von Cramon's previous or present data gives us any indication that this premise is not true today.

Zihl and von Cramon's previous letter to us in 1983 was in response to a request by us

to comment on our manuscript, since we were perplexed by the differences in our results. In their response, the evidence that they presented was based on "unpublished results"; furthermore, no request for comments was made at that time. We are certainly interested in Zihl and von Cramon's most recent publications that indicate that they are not able to train increased visual fields in most of their patients since this has not been made clear in their past papers. For example, Table 2 of their 1981 paper² and Table 3 of their 1979 paper³ report 14 and 12 patients, *all* of whom apparently demonstrated visual field increases. There is no indication that, as they presently report, some or most of these patients did not show field increases. However their present comments indicating that the "return of visual field portions were observed in cases with incomplete damage as evidenced by, lowered CT density values" say nothing about the validity of their visual field testing methods. Correlations are not causal. Accordingly, these results do not change our published criticisms of their basic visual field testing methods.

Zihl and von Cramon are correct when they say that we did not select our patients relative to measurement variability performance. We saw no need to do so since the amount of variability that our patients had seemed quite reasonable. In fact, we agree with Zihl who has said that "the problems connected with defining visual field boundaries have been discussed again and again in textbooks and articles in handbooks".⁴ Similarly, the potential for Zihl and von Cramon's results to have artifacts due to large stimuli variability in perimetric testing and/or to changes in detection strategies with practice still have not been answered.

In either case, depending upon the detection criteria of the examiner, such detection strategies may dramatically effect the border of visual field measurements. This is because attention and reaction times of patients and examiners fluctuate and are not consistent.³ These fluctuations should cause variable data with dips, peaks, ridges, and valleys. As a result, isopters should not turn out smooth, unless such fluctuations are not in agreement with the examiner's subconscious expectations. It is well known that subjectively determined perimetry thresholds obtained by well-trained human operators can omit these irregularities³ and that "the answer will be: The one which fits the expectations of the perimetrist best".⁵ In fact we are not comfortable with Zihl and von Cramon's extremely low measurement variability of $\pm 0.5^\circ$ at $\leq 15^\circ$ and