Ischaemic stroke in chagasic patients

We read with interest the paper by Carod-Artal et al. that showed the relevance of Chagas’ disease as a stroke risk factor in patients of South American origin. They also confirmed a textbook view (not hitherto demonstrated) that cardioembolism is the main cause of stroke in Chagas’ disease, in 52% of cases. This reflects in part the underlying chagasic cardiomyopathy, characterised by congestive heart failure and arrhythmias, present in 46% of chagasic patients as compared with 25% of non-chagasic patients.

Despite the lack of comparison of stroke characteristics between both groups, one very interesting finding was the significant percentage of chagasic patients who have developed stroke without any known vascular risk factors or cardiopathy. As the authors stated, undetected cardiovascular disease could account for at least part of this finding. The indeterminate form of the disease is defined by the presence of infection confirmed by serological tests, in the absence of symptoms or of electrocardiographic or radiological abnormalities. Twenty five % of subjects with the indeterminate form of the disease may present significant structural and/or functional abnormalities when they are fully evaluated by more sensitive methods, such as echocardiography and autonomic tests.

Another possible explanation proposed by the authors would be the vasculitis phenomenon. Although there is good experimental evidence to suggest that changes in the microvasculature may contribute to chagasic heart disease, much less is known about the possible involvement of central nervous system microvascularity in Chagas’ disease. Indeed, most studies point to an important role for endothelin in the pathogenesis of microvascular changes in the chagasic heart,[4,5] but we are unaware of any similar studies of the central nervous system.

The authors also suggest the need for primary prevention in all patients with Chagas’ disease cardiomyopathy. This is a strong recommendation, as most chagasic patients derive from poor social economic backgrounds and have poor access to the health system. Chronic oral anticoagulant therapy is known to cause frequent clinical complications, especially bleeding; an alternative approach could be use of the low dose anticoagulant therapy that has been recently suggested for the treatment of deep vein thrombosis. However, further studies are still needed to investigate this possibility specifically in Chagas’ disease.

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Authors’ reply

In our recent study, we demonstrated that at least 52.2% of chagasic strokes are due to cardioembolism and 36.8% are of undetermined cause.1 A significant proportion of these cryptogenic chagasic strokes may also be cardioembolic in origin.2 We therefore encourage the use of transoesophageal echocardiography in all patients with chagasic stroke, especially for the better definition of aetiology in the 36% having strokes of undetermined cause.

The monitored administration of warfarin in Parkinson’s disease is remarkably effective in the reduction of stroke recurrence in persons with cardioembolic stroke.4 Thus, we recommend oral anticoagulation for all individuals with chagasic stroke, who have demonstrated risk factors for cardioembolism. To our knowledge, no case control study analysing these factors has yet been carried out.

Stroke of arterial origin in Chagas’ disease seems much less common than in the general population of Parkinson’s patients.5 Evidence for secondary prevention of stroke of arterial origin with oral anticoagulation as in the Warfarin-Aspirin Recurrent Stroke Study (WARSS)7 and European/Australasian Stroke Prevention in Reversible Ischaemia Trial (ESPRIT)8 clinical trials is controversial. Our follow up experience with the administration of secondary anticoagulation to persons with chagasic stroke, whether cardioembolic or not, has been encouraging in the lack of significant complications. We are therefore currently investigating the efficacy of antithrombotic therapy in chagasic stroke patients, using either low or moderate dosages of anticoagulants. Until the results of these or similar investigations are available, antithrombotic prophylaxis should be individualised in persons with chagasic stroke of undetermined cause, on the basis of the estimated risk of recurrent stroke and the risk of complications during anticoagulation.

We thank M M Teixeira and A L Teixeira for their comments and interest in our article.

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Efficacy of methyprednisolone pulse therapy on neuroleptic malignant syndrome in Parkinson’s disease

I was astonished to find that Sato and colleagues were able to identify 40 cases of neuroleptic malignant syndrome (NMS) in patients with Parkinson’s disease from a single institution over three years.1 At a recent neurosciences grand round in Birmingham, UK, which has an interest in Parkinson’s disease research, we could only recall two such cases in living memory.

There are two possible explanations for this high incidence of NMS. Firstly, in Japan the Parkinson’s disease population may be more prone to developing NMS when their anti-parkinsonian medication is reduced. This could be due to genotypic differences between Japanese and Western populations. Whereas a higher prevalence of the Parkin mutation has been noted in Japan,2 judging from the age range and duration of disease given in table 1 of Sato’s report,3 these were not all young onset patients as one would expect with the Parkin gene. Nevertheless, it would be of interest to know if this high incidence of NMS has been seen in other Japanese centres and whether any genotypic reason can be found.

The second possible explanation is that the reductions in anti-parkinsonian medication that precipitated NMS were substantial. NMS has been recorded in Parkinson’s disease in the past in association with so-called ‘drug holidays’, which have now been abandoned in most countries owing to the high fatality rate. Against this explanation is the fact that three patients had no change in their medication in Sato’s study.4

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The interest of this paper lies not so much with the proven benefits of methylprednisolone therapy in NMS in Parkinson’s disease, as in the high incidence of NMS in the Japanese patients treated in this unit. I would value the author’s further comments.

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References

Mesencephalic ischemia and Parkinson’s disease
I read with interest the paper by Abe et al1 on occipital and posterior parietal hypoperfusion in 28 Parkinson’s disease (PD) patients without dementia. These findings suggest that there was a reduced regional cerebral blood flow (rCBF) in the intraparenchymal territory of the posterior cerebral arteries (PCAs) probably due to the presence of atheromatous plaques located in the distal end of the basilar artery.2 Atherosclerotic changes are of considerable importance because they can cause stenosis and/or occlusion at the origin of the terminal (PCAs) or collateral (superior cerebellar arteries) branches, as well as of the posterior perforating arteries (PPAs).

Based on the fact that in situ the donor tissues of catecholamines are normally highly vascularised and by contrast in PD the rCBF is reduced in the neostriatum, from February 1988 to December 2002 we have used two surgical procedures to treat PD:1+ (1) transplantation of adrenal medulla into the putamen by a transinsular pathway, and (2) omental transplantation on the interpeduncular fossa, anterior perforated space, and insular cortex in 16 patients with moderate or advanced stages of PD. Thus, omental tissue revascularises to the catecholaminergic (dopaminergic and noradrenergic) nuclei, as well as to the surrounding structures, and moreover prolongs the survival of the graft implanted in the putamen. In all patients, neurological improvement was better during the first weeks after surgery than in the following months or years. Our third patient is the same case previously reported by us.3 At present, 15 years postoperatively, she has only slight tremor on the left leg and does not require antiparkinsonian drugs. However, the possibility of genotypic differences between Western and Japanese populations is interesting,4 and comparisons could usefully be made on the prevalence of mutations in parkin or other genes between these populations.

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Traumatic brain injury as a risk factor for Alzheimer’s disease
In a recent systematic review of case control studies investigating head injury as a risk factor for Alzheimer’s disease (AD), Fleminger et al2 replicated the results of the meta-analysis by Mortimer et al3 in males (OR 2.29; 95% CI from 1.47 to 3.00) but not in females (OR 0.91; 95% CI from 0.56 to 1.47). Their findings support in males only an association between a history of previous head injury and the risk of developing AD, but the study could not review the relation between head injury and ApoE gene status as risk factors for AD.

The review by Fleminger et al was based on clinical studies alone and, as Wilson4 emphasised, did not consider neurodegeneration of the original head injury; and the results of the first retrospective autopsy study of the relation between closed traumatic brain injury (TBI), ApoE allele frequency, and AD were not mentioned. This present study examined:

- the incidence of AD pathology in 55 consecutive autopsy cases (mean age 77.6 years, SD 7.1) with residuals of closed TBI lesions (old contusions in the frontal, temporal, or other brain areas)
- the frequency of TBI residuals in 53 age matched AD cases proven at autopsy.

In both series, ApoE was evaluated from archival brain material embedded in paraffin. The results were as follows.

In the TBI series, 12.7% (four males and three females) showed CERAD B (Consortium to Establish a Registry for Alzheimer’s Disease) definite AD (Braak stages 5 and 6), and 9.1% showed CERAD B probable AD (Braak stages 3 or 4). TBI history dated back from 10 to 30 years before death, and the duration of AD ranged from 2 to 7 years. Two of the subjects with AD showed ApoE3/4, and the remainder 3/3 or 3/2; of the remaining 43 subjects without AD, three exhibited 3/4 alleles. The prevalence of AD (21.8%) in this small autopsy cohort was significantly higher than in either a recent large clinical series (3.3%) or the general population over the age of 70 years (14%).5

In the AD cohort (all CERAD B or C, Braak stages 5 and 6), there was an ApoE4 allele frequency of 30% (similar to other AD series). Residuals of TBI were seen in four brains (two males and two females, each 7.5% of the cohort), all four lacking the ApoE4 allele. These data in small autopsy cohorts confirmed previous clinical studies suggesting that severe TBI is a risk factor for the development of AD, particularly in subjects lacking the ApoE4 allele which is considered a risk factor for AD. No gender differences were found.

Irrespective of these data, we agree with others6 that further work should consider population based cohorts and larger autopsy series of TBI and AD in order better to elucidate the relationship between TBI, ApoE alleles, and the development of AD.

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PostScript

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We would like to comment on the important report by Landi and colleagues about the factors that contributed to the reduced likelihood of receiving secondary stroke prevention treatment and present our own data. We have demonstrated that in community-dwelling patients with chronic atrial fibrillation, living alone or in rural areas, history of falls, frailty, and impaired functional performance are independent factors that result in physicians prescribing aspirin instead of anticoagulants, thus disregarding the contemporary guidelines for stroke prevention.6 We have also shown that in some cases it does not mean malpractice.7 In elderly patients, a geriatric assessment including a thorough evaluation of the psychological and social factors can guide physicians in the selection of the correct treatment, thus avoiding the risks related to anticoagulants in individuals at high risk of falls or with inability to comply with regular blood monitoring.5

Our data are only partially comparable with those of Landi and colleagues, since in their study a significant number of the reported undertreatment concerns aspirin and triclopidine, drugs that have an unfavourable risk–benefit ratio in comparison with anticoagulants, even when they are prescribed for individuals living alone, with a low education level and poor cognitive or functional performance. In these conditions, low compliance is not enough of a risk and does not justify undertreatment. As a matter of facts, in the clinical conditions described by Landi and colleagues, an “ageist” cultural background prevails without real clinical motivation.

The difference between the two sets of data suggests that physicians need to be taught to consider the complexity of the medical scenario and to distinguish incorrect prescribing patterns due to limitations imposed by cultural factors from the rational behavior of physicians who adopt a multidimensional model of care and avoid treatments commonly recognised as beneficial but burdened by a high cost–benefit ratio.

References

The specificity of prescription patterns in secondary stroke prevention

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they were always preceded by heavy, pro-
longed inspiratory effort and stridor, indicat-
ing upper airway obstruction. Such episodes
were not detected in OSA patients without
stridor. Apnoeic events of any type were in
most cases followed by the recurrence of
snoring and not by an inspiratory stridor
sound.
Thus, there seems to be a wide variety of
combined sleep-related breathing disorders
ranging from a majority of obstructive
apnoeas to stereotyped mixed apnoeas of
very long duration and sometimes preceded
by stridor in MSA.
Nocturnal breathing disturbances in MSA
are due to the complex involvement of
multiple brainstem nuclei, leading to a deficit
in the respiratory control system indepen-
dently of the occurrence of stridor. Among
these breathing disorders, OSA are the most
common and may occur in non-obese MSA
patients even in the absence of stridor, thus
indicating that the mechanism underlying
the two events is different. The higher inci-
dence of typical OSA observed in MSA patients
may also be due to the severity of brady-
kinesia and the fact that patients with severe
MSA lie predominantly, if not always, in the
supine position while asleep. The reduction of
non-invasive obstructive events during lateral
position in patients with OSA has already
been reported. In our patients, who were
audio monitored, stridor was not followed by
typical obstructive apnoea nor was the apnoea
produced by a stridor. Thus, we believe
that stridor and OSA in MSA are different
and independent events. We also found that
mixed apnoea occurred stereotypically and
was very prolonged and often preceded by a
harsh sound typical of stridor, as documented
by audio monitoring.
Non-invasive continuous positive air pres-
sure (CPAP) should be proposed for relief of
sleep breathing disorders. It has been used
successfully to treat stridor and OSA in MSA
patients. In our series, nine patients accepted
CPAP treatment (six with stridor and sleep
apnoea and three with isolated OSA). One
patient died before initiation of the treat-
ment and two patients without sleep complaints
dropped out after one week because of lack of
tolerance despite having a severe apnoea/
hypopnoea index. Since the onset of CPAP
treatment, both patients and their spouses
reported an improvement of sleep, improved
daytime alertness and wellbeing. For some patients,
getting used to CPAP took up to a month,
after which it was generally well tolerated.
After a mean follow up period of 10 months,
the patients’ compliance with the continued
use of CPAP was satisfactory and their
relatives did not report any recurrence of
stridor.
Thus we feel that the relationship between
stridor and OSA is far from clear, especially considering the polymysognomic
association of stridor and mixed apnoea
that we found. Complex supranuclear
neurological dysfunction may account for this
association, but further studies are however needed to clarify this issue and better establish the
indications for CPAP.

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Authors’ reply
We would like to thank Dr Ghorayeb et al for their
interest in our paper and their comments. We agree
with the view that the relation between stridor and apnoea in
MSA is very important. Unfortunately, in our
experimental procedure, we could not establish
a correlation between the airway patency and
vocal sound simultaneously in a magnetic resonance
imaging (MRI) recording. In obstructive sleep apnoea
syndrome (OSAS), even when the upper airway
was obstructed completely, the vocal cords were not
obstructed. Therefore, stridor does not develop
although snoring may occur in OSAS. In contrast, MSA patients had
an obstructed upper airway, which was fre-
cently accompanied by stenosis of the vocal
cords. However, we did not find stenosis of
the vocal cords without stenosis of the upper
airway. If the stridor is produced by the
vocal cords and snoring is produced by
stenosis of the upper airway, snoring
should be accompanied by stridor in all
MSA patients. In fact, we observed that the
initial narrowing of the larynx and pharynx
produced
ned. Ghorayeb et al point out that
OSA (SAS with upper airway obstruction)
can commonly occur even in non-obese
patients with MSA without the presence of
stridor. We agree with this observation, but in
this study, we did not find stenosis of the
vocal cords without upper airway stenosis,
so none of our patients developed stridor
without snoring. We observed the patients in
the MRI room to identify the sleep state and
the presence of snoring and stridor, and we
found that the highest pitch vocal sound
appeared after heavy and prolonged inspira-
tory effort. This phenomenon is very similar
to Ghorayeb et al’s observation of apnoea and
stridor. We also suppose that the phenom-
enon of apnoea in MSA patients occurs with
confinement of the upper airway. Therefore,
we think that some patients in MSA with SAS
may be helped with CPAP similar to OSAS
patients. However, the effect of CPAP could be diminished, since the
respiratory centre is eventually involved with
the progression of disease in MSA, and cen-
tral apnoea and abnormal respiration may
appear. Further study is required to clarify
the indication of CPAP in patients with MSA.

The mechanism of SAS in MSA is unclear. Our study showed complete obstruction of
the upper airway and vocal cords occurred in
MSA, even with the presence of tongue
apnoeas and without narrowing of the larynx.
Thus, we suggest that there is another
mechanism involved distinct from that of
OSAS. Some reports have stated that a
dystonia-like phenomenon was present in the
vocal cords in the stridor through electromyographic study, suggesting a similar
mechanism to be present in the progression
of upper airway obstruction.

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 laryngeal narrowing in patients with multiple system

BOOK REVIEWS

Biological psychiatry, Vol 1 and 2

Edited by Hugo A H D’Haenen, Johan A den
Boer, and Paul Willner. Published by John
Wiley & Sons, Chicester, 2002, pp 1404,

The European editors of these two volumes
have brought together contributions from all
over the world and from a range of relevant
specialties. Although the majority of the
authors work in psychiatry, the other disci-
plines represented include neurology, psychol-
ogy, physiology, and pharmacology. Guided
by clear concepts regarding the anatomy of
the book overall as well as the individual
chapters, the editors have succeeded in
providing an integrated and comprehensive
view of biological psychiatry.

The introductory chapters address concep-
tual and measurement issues in biological
psychiatry. The next section comprises a
series of chapters on basic principles, review-
ing key topics such as general neuro-
transmitter systems, neuroendocrinology,
immunology, psychology, neuropsychology, brain imaging.
The Bereitschaftspotential Movement-Related Cortical Potentials

The Bereitschaftspotential (BP; readiness potential, although the sense of the German word is rather more imperative) was discovered in 1964 and named in 1965 by Hans Kornhuber and Lüder Deeke. In their original description it was a negative going wave of cortical potential that was first detectable 1–1.5 s before the movement occurred. Like the demonstration of evoked potentials, it was a technological advance (the computer and the term “bioelectric potentials,” a technological advance of the computer used), which permitted detection of these minute waveforms. The discovery (with its implications for volition and free will) acted as a catalyst to a new stimulus to research. This book brings up to date the state of knowledge concerning the BP and other brain potentials occurring around the time of a motor act.

It is an expensive text. What does the reader get for the money? The book consists of 17 chapters in 7 sections. There is a brief introduction by the editors that states the aims of the book. These are: to explain the processes that the BP reflects, to quantify the number of components responsible for the BP, to explore the anatomical substrate for the BP, and to flag up areas for future research. These are commendable aims, and, to the extent that much information on all these aspects is contained in the book, they are achieved.

However, the arrangement of the material into the 7 sections of the book does not neatly reflect these aims and it is left to the reader to pick out the information where it occurs. This approach of presenting a number of unpub-lished papers, some of which have their pros and cons. Its nature is there is a mass of new data from experts in the field and an element of historical and personal back-ground and critical commentary that one would not otherwise find in the scientific literature. Against it is the difficulty of obtaining a coherent overview of the subject. This is compounded by inconsistent termi-nology (a glossary of abbreviations is a serious omission, the more so as the abbre-viations are often inconsistent), and poor proof reading (there are very many minor errors). The general reader will find the introduction useful and will enjoy the chap ters by Libecco and Kornhuber but, as a whole, the book is strictly one for the specialist.

M Lakie

Classic cases in neuropsychology, Vol 2

Why read the classics? If you’re still not sure why it might be worth bothering, this book would really be wasted on you. Better that it should fall into the hands of someone who really appreciates that modern neurology and neuropsychology owes an enormous amount to the careful descriptions of single cases. And, despite the whizz and bang of functional imaging, this is likely to continue to be the case.

In this volume, you will find discussion of Babinski’s cases of anosogasia for hemiplegia, Wernicke’s case of conduction dysphasia, Goldstein and Gelb’s description of form agnosia, Dejerine’s case of alexia without agraphia, and many other gems from the distant past. But, in addition, you will also be pleasantly surprised to see more recent “classics” such as Bisiach and Luzzatti’s descriptions of right hemisphere Milanese patients who, when recalling from one imagined viewpoint, described the famous Piazza del Duomo (the city’s central square), reported places that would appear to their right, neglecting those to their left. But, when asked to imagine turning round, they failed to report locations they had previously men tioned and described instead places that now fell to their right from this new viewpoint. This description of “representational neglect” has had a profound impact both on stimulating research on the syndrome and understanding the nature of mental representa tions of space.

Of course, the qualities of the contributing chapters do vary considerably but the subject matter that is covered in this collection is wide ranging, and also entertaining. The chapters that work well are those that place their case study in their historical, as well as scientific context. There are important les sons here, for instance, about the dedication and obsessive nature of some neurologists. Dejerine, for example, himself carried out the postmortem on his patient with pure alexia within 24 hours of his death—at the patient’s home. He clearly wanted to find out how the lesion location differed from that of a patient he had reported on the previous year who had alexia with agraphia, and no adminis-
Intraoperative imaging in neurosurgery MRI, CT and ultrasound


This book forms part of a series of symposia reported by Springer-Verlag. The topics covered are important and timely. Most neurosurgical units would be evaluating the need for intraoperative imaging, the options, and possibilities. A factor that emerges is the importance of structure, and indeed then the need for courses and refresher symposia on modern operative anatomy and in particular to fully understand the fibre tracts of the brain. The first section, on interventional MRI, covers systems currently in clinical use and some background development and potential that would be valuable and necessary reading for a unit contemplating the introduction of such technology. The authors are experienced and the contribution significant. The second section deals with the role of intraoperative MRI and glioma surgery. It appears that data are emerging that the use of intraoperative MRI allows for more complete resection and probably a better outcome for patients with this devastating disease. It also gives a description of some of the difficulties that will be encountered when using neuronavigation together with the MRI system. The third section provides thoughtful reflections on the use of intraoperative ultrasound for cranial surgery and a chapter on the use of intraoperative CT scanning for navigation in spinal surgery. The final chapters provide some personal reflections on intraoperative MRI imaging technology and the use of functional MRI, together with a chapter on the cost benefit ratio of the technology. In the postscript Dr Yonekawa again highlights the need for training in basic micro-neurosurgery to continue parallel to the learning of innovative technical developments.

The book will provide essential reading for heads of service, neurosurgeons with an interest in neuronavigation and intraoperative imaging, and managers who will be faced with requests for the introduction of such equipment. It is a comprehensive and well balanced collection of views and information on this important and emerging topic.

J van Dellen

New frontiers of MR-based techniques in multiple sclerosis

Edited by Massimo Filippi and Giancarlo Comi. Published by Springer-Verlag, Italy, 2003, pp 107, €49.95. ISBN 88-470-0198-6

Magnetic resonance (MR) is the single most important laboratory technique for diagnosis and monitoring of patients with multiple sclerosis (MS). Although some may cringe at the thought of yet another review of MR methods, development of new MR based methodologies continues. This short book provides a succinct description of the “cutting edge” of the field in seven chapters written by acknowledged experts.

Dousset, for example, describes ways in which individual cells may be tracked in the central nervous system after labelling with new iron oxide based contrast agents. Filippi, Rocca, and Rovaris review applications of both magnetisation transfer and diffusion weighted MRI to defining early axonal injury in normal appearing areas of a white matter.

There is a further discussion of methods of diffusion tractography, which allows axonal tracts to be mapped, giving both information on the anatomy of major tracts and their integrity. Rashid and Miller describe applications of arterial spin-labelling magnetic resonance, a technique for defining perfusion changes that potentially provide an absolute measure of brain activity. The importance of better understanding cortical functional changes is emphasised in a nice review of functional MRI demonstrating ways in which the organisation of brain systems may change adaptively with the progression of pathology in MS. Part of the future of MRI clearly lies in enhancing sensitivity and spatial resolution of the imaging. An exciting approach to this has been development of ultra-high (>5 T) field magnets. Kangarlu and his colleagues present images of human brain from an 8 Tesla that is more than 5 times as powerful as a conventional clinical scanner) imaging system with individual plaques of MS shown.

This volume can be read quickly and the chapters are well written. It is highly recommended for neuroscientists and radiologists who want a brief, authoritative introduction to the current state of the art.

P Matthews

History of neurology in The Netherlands


Pride of place in the first century or so of Dutch neurology must go to the basic sciences of anatomy and physiology. These were my first points of contact with the Dutch neurological tradition, now almost 50 years ago. To the fledgling investigator working on proprioceptive reflexes, Ariens Kappers et al’s ‘The comparative anatomy of the nervous system of vertebrates including man’ (New York, Macmillan, 1936) was the place to turn for the structural background to physiological experiments. And in physiology, the work of Magnus, de Kleijn, and Rademaker were essential to understanding the postural rigidity. Studies on pathological peripheral and central nerve fibres were illuminated by the early pathological studies on Beri Beri by Winkler (1855–1941) and Pekelharing (1848–1922), and Hans van Crevel’s work in the laboratory of Verhaert (1889–1983) in Leiden.

The book reviews the origins of this great tradition and charts its continuation into the late 20th century through Dusser de Barenne (who became professor of physiology at Yale) and Nauta (who also emigrated to the United States) and his student Hans Kuypers who was professor of anatomy successively in Rotterdam and Cambridge. Other aspects of neurology (the editors prefer the traditional use of the word to denote all aspects of the study of the nervous system, normal and pathological, including neurosurgery as well as clinical neurology) were later in achieving the well deserved international recognition they now have.

The book provides a wealth of detail about the evolution of the different neurological centres in The Netherlands and the contributions coming from them. As in Germany, psychiatry and neurology remained closely linked until well into the 20th century. The development of the sub-specialties is considered in some detail. A special feature of the Dutch scene was the way in which high quality original work came not infrequently from non-university settings.

Of particular interest to the general neurological reader are the more detailed accounts of the life and work of a number of the major neurological figures in The Netherlands. Ariens Kappers emerges not only as the important contributor he was, but as a rather remote, self centred individual with his eye always to the main chance, and not especially appreciative of the work he got others to do for him. He, like most of the others in this section of the book, seems to have lived a rather austere live concentrated on his professional duties. There was tragedy for some, including Bernard Brouwer (1841–1949) who as Rector Magnificus did his best to limit the inroads of Nazism in the University; the authorities closed the university down. But after the liberation in 1945, Brouwer was judged not to have done enough in opposing the Nazis, and was refused an opportunity to return to the university. His colleagues, however, believed in his integrity and in 1947 he was appointed Director of Amsterdam’s Central Institute for Brain Research, where he continued to work until his death.

The book is well produced and illustrated, with portraits and a number of scientific illustrations from both the early and the recent literature.
Ischaemic stroke in chagasic patients

A L Teixeira, Jr and M M Teixeira

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