antibodies were, however, present and a
labial salivary gland biopsy showed numer-
ous lymphoid aggregates consistent with a
diagnosis of the sicca syndrome. The patient
subsequently admitted to rather dry eyes, and
had moderate caries. A Schirmer's test
confirmed poor tear production.
This patient provides another example of
an association between the sicca syndrome
and inflammatory polyneuropathy, and
lends further support to the hypothesis that,
like the sicca syndrome and systemic lupus
erthymatosus with which they are also asso-
ciated,\(^2\) the inflammatory poly-
neuropathies have an auto-immune basis.
Our patient developed a clinical neuropathy
at a time when symptoms of the sicca syn-
drome were minimal. This finding illustrates
the value of maintaining a high index of clin-
ical suspicion, and of performing a sero-
logical auto-antibody screen in patients
suspected of having an inflammatory poly-
neuropathy, but in whom there are no overt
features of an associated disease. By this
means, associated auto-immune disorders
may be uncovered prior to the development
of serious manifestations.

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Serotal pain with testicular jerking: an
unusual manifestation of epilepsy
Sir: I was interested in Dr Bhaskar's descrip-
tion of a man presenting with focal epilepsy
in his scrotum.\(^1\) I am afraid it has been
described before, but only to the students
and junior staff of Dr Michael Kremer. Dr
Kremer is at present unable to relate the
story himself, but he described a man whom
he saw during the 1939–45 war who had a
bullet wound to the brain. Bullets in those
days created damage only between entry and
exit wounds, and he survived the experience
to present with a focal painful epilepsy
affecting one half of his scrotal sac.
We told him at the time he should have
published it.

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Thymoma without myasthenia gravis: electro-
physiological study after thymectomy
Sir: Martinez and Jimenez\(^1\) reported a
patient with thymoma without clinical evi-
dence of myasthenia gravis, in whom single-
fibre EMG (SF-EMG) studies demonstrated
abnormal neuromuscular transmission. We
had previously reported increased jitter on
SF-EMG studies in three patients with thym-
oma, none of whom had clinical myasthenia gravis.\(^2\) One patient had a malignant thymoma with red cell aplasia (table, Pt 1) but there was no weakness on
examination. Acetylcholine receptor anti-
body (AChR-Ab) levels were normal and jitter
was increased in the extensor digitorum
communis (EDC) muscle. After receiving
prednisone for treatment of anaemia, he
developed mild weakness of ocular and
shoulder muscles which improved after
dedrophonium. SF-EMG studies performed
on four occasions over a 6 week period
demonstrated persistently abnormal neu-
romuscular transmission (table). He died of
infection 2 months after his initial SF-EMG
study.

In the second patient (table, Pt 2), a medi-
astinal mass was discovered on routine chest
radiography and a benign, encapsulated
thymoma was removed. The patient had no
weakness on examination before surgery or
6 months after surgery. AChR-Ab was normal.
Jitter was increased in the EDC before surgery and miniature end-plate potential amplitude was decreased in an intercostal muscle biopsy obtained
at thymectomy. Jitter was still increased in
the EDC three weeks after surgery. Six months
after surgery there was a decremental
response to stapedial reflex fatigue testing,
and this was partially reversed after the
administration of endrophonium.\(^3\)

The third patient (table, Pt 3) was a man
who was found to have an anterior medi-
astinal mass on routine radiography at age 28
years. Retrospective review of previous
radiographs revealed that the mass had been
present for at least three years, with slowly
progressive enlargement. There was no evi-
dence of myasthenia gravis by history or on
examination and serum AChR-Ab levels
were normal. Jitter was increased in the
EDC and there was a decremental response
to stapedial reflex testing which did not
change after administration of endrophonium.

The clinical manifestations of myasthenia
gravis may be subtle and may not be
interpreted unless carefully sought. These
patients demonstrate that neuromuscular
transmission may be abnormal in patients
with thymoma even when clinical myasthe-
nia is normal. We feel that all patients
with thymoma should be suspected of hav-
ing myasthenia gravis and should be evalu-
ated for this possibility before surgery. Since
the history, physical examination, AChR-
Ab level and repetitive nerve stimulation
may all be normal in such patients, increased
jitter may be the only evidence of abnormal

Table Details of patients

<table>
<thead>
<tr>
<th>Patient Diagnosis (Normal value)</th>
<th>SF-EMG in EDC</th>
<th>Mean MCD (µSec)</th>
<th>% Fibre pairs with blocking</th>
<th>Normal jitter ( &gt; 90%)</th>
</tr>
</thead>
</table>
| 1. Malignant thymoma, red cell aplasia | 10/76 | 10/28/76 | 11/1/76 | 11/23/76 | 43 | 41 | 38 | 64 | 11% | 5% | 0 | 0 | 0
| 2. Benign thymoma | 7/13/77 | 7/21/77 | 8/7/77 | 7/13/77 | 49 | 46 | 44 | 43 | 12% | 9% | 0 | 0 | 0
| 3. Probable thymoma | 5/13/78 | 5/13/78 | 5/13/78 | 5/13/78 | 43 | 43 | 43 | 43 | 0 | 0 | 0 | 0 | 0


neuromuscular transmission. Our experience, like that of Martinez and Jimenez, indicates that these patients may never demonstrate clinical manifestations of myasthenia gravis, thus therapeutic decisions should be made based on the clinical findings, rather than EMG abnormalities.

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Stridor during dystonia phases of Parkinson’s disease

Sir: Doctors Corbin and Williams’ report of respiratory stridor co-occurring with limb and orofacial dystonia in two patients with idiopathic Parkinson’s disease is noteworthy.1 However, their statement that “…spasmodic dysphonia is now recognised as being a form of focal dystonia of the laryngeal muscles” (p. 821) needs clarification.

The cardinal signs of spasmodic (spastic) dysphonia, principally (1) intermittent or regular adductor voice arrests secondary to vocal fold/laryngeal hyperadduction, (2) moments of strained, effortful vocal quality interspersed within apparent normal phonation, and (3) intermittent or regular breathy moments secondary to abductory glottal arrests may represent psychogenic, essential tremor and, as implied by Corbin and Williams, other disorders of movement.2 8

Co-occurring impairment of one or more of the other components of motor speech, namely articulation, respiration, phonation, and prosody (speech rhythm) is consistent with dysarthria rather than a focal laryngeal disorder.2 7 9

The response of spastic dysphonia to treatment seems to depend upon aetiology, type of disorder (aductor, abductory, mixed), and mode of therapy.4 7

For those types of spasmodic dysphonia for which a recognisable aetiology cannot be identified, the term “idiopathic spastic dysphonia” has been recommended along with regular follow-up, which may eventually reveal an underlying substrate for the disorder.2 4

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References

Corbin and Williams reply:
Sir: Dr Hartman reminds us of the possible mechanisms of vocal cord dysfunction in spasmodic dysphonia; however, in our final paragraph1 we sought to emphasise the need for clinicians to consider dystonia as a cause of what may otherwise be an enigmatic disorder; we refer not only to some cases of spasmodic dysphonia but also to problems such as piano-player’s dystonia and the occupational dystonias.2 Many such cases were previously classified as hysterical. Dr Hartman’s policy of accepting that some cases of spasmodic dysphonia lack recognisable aetiology is safer in that it does not deny the possible existence of an organic cause.

References

Book reviews


This is the fifth monograph in a series on management and treatment in specialities in medicine and serves as a perfect riposte to those who would believe that the neurologist has a major role in diagnosis but only a minor role in therapy. It contains a practical approach to major neurological disorders which is spiced by the pharmacological expertise of Dr Jenner in explaining not only when, but why, a particular agent should be prescribed.

The initial chapter is a review of the actions of drugs on the nervous system and briefly considers the role of the blood brain barrier. It contains useful tables of drugs which are agonists and antagonists for the various neuro-transmitter receptors and examples of compounds which act upon specific receptor sub-types. There follow chapters on each of the common neurological disorders with the appropriate therapy and, where known, a summary of their mode of action. The authors’ own interest and expertise in the treatment of movement disorders and sleep disorders is apparent in these, the best, chapters in the book and the whole provides a useful practical guide to logical therapy in neurological disorders. Inevitably the particular biases of the authors are revealed and not everyone will agree with their suggestion that steroids have a role in ischaemic stroke. Indeed the authors seem somewhat uncertain themselves in that on one page they state “it may therefore be beneficial to treat focal oedema around an infarct using osmotic diuretics or steroids” and on the next page “post-infarction oedema does not respond to steroids”.

In the chapter on infections it would have been useful to have a suggestion as to the most reasonable combination therapy in the infant, adult or aged patient presenting with a presumed but unidentified or partially treated bacterial meningitis rather than the bald statement that therapy “depends on isolation of the causative organism”. No