rural areas and have a poor nutritional status(1,5) as seen in our case. In children, aural myiasis is more common with 75% cases being less than 10 years and one third even less than 2 years(4). However, myiasis involving the eye is generally secondary to nasal myiasis. Maggots from the nose erode the mucous membrane and bones of the nasal cavity and spread to the eye lid and cheeks(6). However, nose in the present case was normal. The infected wound on the eye lid with purulent discharge attracted the flies which laid their eggs and subsequently developed maggots. Treatment described includes getting the maggots to the surface after pouring water locally and then manually removing them(3). Others have used 10% cocaine to paralyse the maggots and then removed them manually(7). Simple manual removal is as effective and our patient was made maggot free in 3 sittings. Antilarval measures like ether(4), terpentine oil(1) and mixture of chloroform and terpentine oil in the ratio of 1 : 4(5) were purposely avoided in this child to prevent spill over and possible deleterious effects on the eye. Interestingly, in one instance the larvae of the fly *Dermatobia hominis* was excised because it resembled and was mistaken for a large chalazion(7).

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Japanese Encephalitis– An Encephalomyelitis

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Over the last 2 decades Japanese encephalitis (JE) has assumed great importance as a preventable killer disease in the Indian subcontinent(1). The disease is endemic in the Lucknow region where cases are seen all round the year with a peak in the late monsoon and early winter(2,3). The purpose of this communication is to present 3 patients with unusual findings suggesting that the agent may occasionally

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produce encephalomyelitis rather than the usual picture of 'encephalitis'.

The diagnosis of JE in the 3 patients described here is based on the conventional hemagglutination inhibition (HI) antibody titres done in the acute and convalescent phases followed by detection of JE specific IgM. These methods are widely accepted for the diagnosis. Viral isolation from CSF was also attempted in all 3 patients but was negative.

Case Report

Case 1: A 12-year-old girl was admitted with complaints of abrupt onset of fever, vomiting, convulsions and coma 10 days back. She had previously been admitted in a district hospital where the convulsions were controlled and consciousness had improved. On examination here she was drowsy. Power in both upper limbs was 2/5 and in both lower limbs it was 0/5. Deep tendon reflexes were not elicitable in the lower limbs and no sensory loss was detectable. Cerebrospinal fluid (CSF) examination showed clear CSF with 10 cells/cu mm and protein of 40 mg/dl. Nerve conduction velocity was within normal limits. She was treated with non-specific measures as for viral encephalitis. She made slight improvement in hospital and was discharged on request due to personal reasons. Virological investigations confirmed the diagnosis of JE.

On follow up after 1 year her intelligence and memory were as before the illness. She could not stand or walk. Power was normal in both upper limbs, 3/5 in the left leg and 2/5 in the right leg. Tone was slightly increased in the upper limbs but markedly reduced in the lower limbs. Deep tendon reflexes were exaggerated in the upper limbs. Both ankle jerks were elicitable but the knee jerk was absent on the left side. Other pyramidal signs in the form of ankle clonus and upgoing plantar were not elicitable. Myelogram was done and was within normal limits.

Case 2: A 2-year-old boy was admitted with complaints of fever, vomiting, convulsions and coma of 4 days duration. On examination at admission he was in coma but responding to pain. Muscle tone and reflexes were diminished all over. CSF examination showed clear CSF with 15 cells/cu mm and protein of 60 mg/dl. In hospital his consciousness improved gradually and he was discharged after 12 days. Virological investigation later confirmed the diagnosis of JE and he was followed up. Examination after 6 months revealed that speech and mentation were normal but there was difficulty in walking due to weakness in both legs especially the left. Muscle mass was decreased in the left leg. The right leg showed exaggerated tendon reflexes while on the left side the knee jerk was normal and the ankle jerk was absent. Ankle clonus and upgoing plantar were present on the right side but no pyramidal signs were present on the left side.

Case 3: This 2-year-old boy was admitted with fever, convulsions and coma for 3 days. On admission he was comatose but responding to pain. Left facial palsy was present. Tone was reduced in the left arm but normal in other limbs. CSF was clear but contained 350 cells/cu mm, mostly polymorphs. Protein was 40 mg/dl and sugar 80 mg/dl. As consciousness improved and he was able to feed orally, he was discharged. Results of virologial investigations later confirmed the diagnosis of JE. Follow up after 1 year revealed that neck holding was deficient. Left sided facial palsy was still persisting. Power was decreased in both upper and lower limbs on the left side. Tone was decreased in the

left arm. Tendon reflexes were brisk in the left leg but absent in the left arm.

The clinical findings in the 3 patients are summarized in the *Table*.

Discussion

The neurological findings in these 3 patients are unusual because they are suggestive of a mixed upper and lower neurone damage. Due to the presence of lower motion neurone signs the possibility of poliomyelitis and even transverse myelitis was entertained initially when all the signs were not so clear cut. However,

TABLE—*Clinical Summary of the Three Patients*

	Case I	Case II	Case III
Age/Sex	12 years F	2 years M	1 year 6 months M
Initial illness	Fever, vomiting convulsions, coma power 0/5 in lower limbs & 2/5 upper limbs.	Fever, vomiting convulsion, coma ↓ muscle tone and reflexes all over	Fever, vomiting convulsion, coma Lt. facial palsy ↓ tone in Lt. arm
Period of follow up	6 months	6 months	1 year
Higher function	N	N	N
Mucle mass	↓ in both legs	↓ Lt. leg	N
			Lt, facial palsy
Power			
Rt. arm : Lt. arm	N : N	N : N	N : 3/5
Rt. leg : Lt. leg	2/5 : 3/5	4/5 : 3/5	N : 3/5
Tone	Slight ↑ in upper limbs ↓ ↓ in lower limbs	↓ in Lt. leg	↓ in Lt. arm
Deep tendon reflexes	Rt. : Lt.	Rt. : Lt.	Rt. : Lt.
—Biceps	++ : ++	+ : +	+ : −
—Triceps	++ : ++	+ : +	+ : −
—Supinator	+ : +	+ : +	+ : −
—Knee	+ : −	++ : +	+ : ++
—Ankle clonus	+ : +	++ : −	+ : ++
Ankle	Absent	Present on Rt. side	Absent
Planter response	Rt. ↓ Lt. ↓	Rt. ↑ Lt. ↓	Rt. ↓ Lt. ↑

Abbreviations used : N—Normal, Rt.—Right, Lt.—Left.

the typical presentation with fever, coma and convulsions is not described in these disorders. All these patients had upper motor neurone sign also, which are not seen in polioymyelitis.

Japanese encephalitis has 3 stages–a prodromal stage with fever, vomiting and headache, an encephalitic stage with coma, convulsion and neurological signs and a recovery or convalescent stage. In the acute stage some workers have reported "bizarre" neurological findings with variable or unpredictable deep tendon reflexes and/or plantar responses(4,5). Webb and Perriera(5) described focal neurological signs initially and hypotonia, which rapidly gave way to spasticity, hyperreflexia and clonus. Our observation was generally also that the tone and reflexes may be diminished in the deeply comatose patient and returned to normal or exaggerated stage once coma lightened. The persistence of lower motor neurone signs in these patients was unusual.

Many investigators(5,6) have described neurological sequelae in JE in the form of upper motor neurone paralysis with pyramidal signs. Others have described sequelae such as paralysis or hemiplegia without going into the nature of the deficits(7). The 3 patients described here are laboratory confirmed cases who were carefully followed up and showed mixed upper and lower neurone signs. Pathologic findings in JE show areas of neurolysis scattered over the grey matter of the brain(8). Scattered or patchy necrotic lesions in the spinal cord could explain the findings observed by us.

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