Personal Practice

Headaches in Children and Adolescents

S. Aneja

Headaches occur commonly in children and may be due to a primary disorder such as migraine or a symptom accompanying childhood infections, systemic disease, or a neurologocial disorder. A great majority of headaches are caused by conditions that are not life threatening. However, it is incumbent on the part of the doctor to arrive at the correct etiology of headache and to alleviate pain.

Epidemiology

The data on childhood headache is sparse and most of it is focussed on migraine. In a monumental study comprising of nearly 9000 school children(l) it was observed that by the age of 7 years 2.5% of children had frequent non-migrainous headache, 1.4% had true migraine, and 35% had infrequent headaches of other varieties. An increase in incidence was seen with increasing age. In a study which took in account school absence due to all kinds of headaches, 1% of all missed school days were because of headaches and 3.7% of children missed school on one or more occasions due to headache(2).

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Pathophysiology

There are no pain receptors in the brain parenchyma. Headache is often caused by referred pain from structures of head and neck other than brain. The intracranial structures that carry pain are dura, large arteries, large veins and the venous sinuses. Pathology involving periosteum, pharynx, orbit, sinuses, muscles of neck, face and skull can cause headache. All these structures are innervated by Vth cranial nerve or upper cervical spinal cord roots (in case of posterior scalp and neck). Direct or indirect traction of dura due to increased intracranial pressure also gives rise to pain. Alteration in cerebral blood flow as in systemic arterial hypertension is also associated with headache.

Clinical Classification of Headache

The presentation of headache can be separated into 5 distinct patterns depending on the temporal profile, *i.e.,* acute, acute-recurrent, chronic progressive, chronic non-progressive and mixed(3). A pictorial depiction of temporal patterns of headaches has been aptly described by Rothner *(Fig. 1).* An acute headache is defined as a single event with no history of similar episodes in the past. A graph with spikes separated by return to baseline at periodic interval (acute recurrent) is typical of vascular headache particularly migraine. The common causes of headache are enumerated in *Table I.*

Acute Headache

Data regarding acute headache in childhood population are not available. In

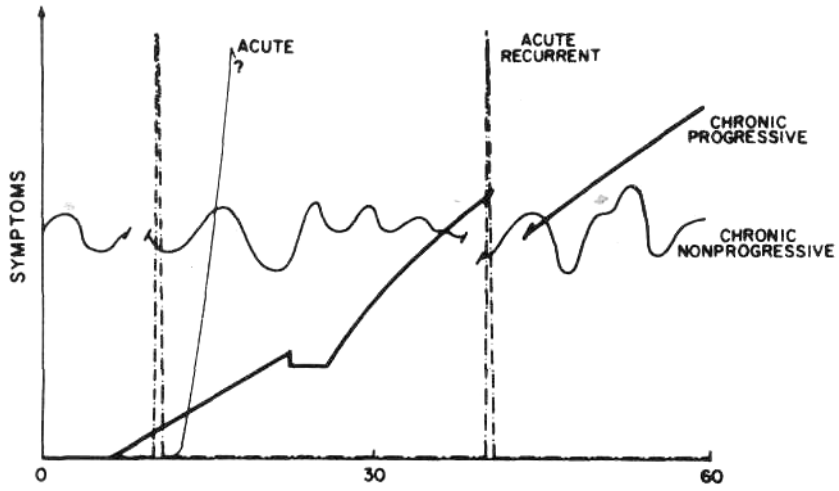

Fig. 1. The temporal pattern of headache. Reprinted with permission(4).

study, 25% of all patients visiting emergency room because of headache were younger than 17 years(5). The evaluation of acute headache is directed towards ruling out organic disease and life threatening conditions which may require urgent intervention. The measurement of temperature and blood pressure are critical. The history, general physical examination and detailed neurological examination including fundoscopy will further point to the likely cause of headache. Non-cephalic infections are frequently associated with headache(6). Differentiating a CNS infection from a non-cephalic infection is of paramount importance in a patient who presents with fever and headache. Pyrogens can cause vascular headache due to release of prostaglandins and vasoactive substances. Besides, the body's thermoregulatory response can produce changes in cerebral blood flow and thus cause headache. Patients with family history of migraine are more likely to have these headaches. Patients with preexisting migraine and tension headache also tend to get exacerbation when they have febrile illness.

Acute Recurrent Headache

Migraine

Migraine, the most common cause of acute recurrent headache syndrome, is characterized by periodic, paroxysmal episodes of headache accompanied with nausea and/or vomiting, and intense desire to sleep. The prevalence of migraine was 3.9% in a large series-(l). The incidence increases with increase in age from 2.7% at age 7 years with slight male preponderance to affect 6.4% of boys and 14.8% of girls by age 14(7).

Pathogenesis: Although the initiating event in migraine is not known, it is believed that either an ischemic or neurochemical noxious trigger stimulates the trigeminal nerve. Through antidromic action, neuropeptides are released from small diameter sensory afferents into dural and meningeal vessels initiating vasodilation and release of histamine from mast cells and serotonin from platelets. Serotonin (5-HT) is considered to be a principal mediator in the cascade of events resulting in vasodilation and exudation of plasma(8).

TABLE I-*Causes of Headache*

A. Acute
- CNS infection
- Non cephalic infection
- Sinusitis
- Trauma
- Hypertension
- Ocular disease
- Post lumbar puncture
- Subarachnoid hemorrhage

B. Acute Recurrent
- Migraine
- — Common (without aura)
- — Classic (with aura)
- — Complicated migraine
  Basilar artery migraine
  Hemiplegic migraine
  Retinal migraine
  Ophthalmoplegic migraine
- Cluster headache
- Headaches associated with seizures
  e.g., ictal or postictal
- Vascular headache due to
  AV malformation, vasculitis

C. Chronic Progressive
- AV malformation
- Hydrocephalus
- Pseudotumor cerebri
- Mass lesions, e.g., tumor, abscess or inflammatory granulomas
- Chronic subdural hematoma
- Infection *e.g.,* TB meningitis
- Vasculitis syndrome
- Chiari malformation

D. Chronic Non-Progressive
- Tension headache
- Chronic pansinusitis
- Analgesic rebound
- Psychiatric states

E. **Miscellaneous**
- Occipital neuralgia
- Indomethacin responsive headache
  —exertional headache
  —hemicrania continua
  —cyclic migraine
- Ice cream headache
- Mitochondrial encephalomyelopathy with lactic acidosis & stroke like episodes

The neurogenic inflammation and release of substance P causes distension of cranial arteries and pain.

A major advance in the understanding of migraine pathophysiology and its treatment is related to serotonin (5-HT) receptors. At least 5 serotonin receptor subtypes, *i.e.,* 5HT-IA, 5HT-ID, 5HT-IC, 5HT-2, and 5HT-3 have been identified. It has been seen that agents with agonist activity at 5HT-ID and/or 5HT-1A receptors provide relief in acute attack. On the other hand prophylaxis of migraine is dependent on antagonistic activity at 5HT-2 and/or 5HT-1C receptor sites.

Diagnostic Criteria: The gold standard for diagnosis of migraine has been the International Headache Society (IHS) Classification(9) as shown in *Table II* which has been tailored to adult migraine but is applicable to adolescents as well. However, these criteria may not be applicable to children since: *(a)* most episodes of childhood migraine are of short duration, *(b)* inability of child to provide adequate description, and *(c)* infrequency of associated symptoms in childhood migraine.

Some studies have questioned the basis on which IHS set down two hours as the minimum essential criteria in children under the age of 15 years(10,ll). A revised set of criteria reducing the minimum duration of headache to 30 minutes and including bilateral headaches as well has been proposed(12).

The age of onset of migrainous headaches can be quite early in life. Approximately 20% of the patients had their first attack by 5 years of age. Attacks can be precipitated by psychological factors, certain foods, physical exertion and by hormonal factors. Migraines are classified according to their clinical features into various types *{Table I).*

TABLE II—*The International Headache Society Classification (1988).*

1. MIGRAINE

1.1 Migraine without aura

A. At least five attacks fulfilling B to D
B. Headache attack lasting 2 to 72 hours (untreated or unsuccessfully treated). In children <15 years, attack may last 2-48 hours.
C. *At least two of the following;*
   (1) Unilateral location
   (2) Pulsating quality
   (3) Moderate to severe intensity
   (4) Aggravated by routine physical activity
D. *During headache at least one of the following:*
   (1) Nausea and/or vomiting
   (2) Photophobia and phonophobia

2.2 ***Migraine with aura***

A. At least two attacks fulfilling B
B. *At least three or more of the following:*
   (1) one or more reversible aura symptoms indicating focal cortical and/or brainstem dysfunction.
   (2) At least one aura symptom develops gradually over 4 or more minutes, or two or more symptoms occur in succession.
   (3) No aura lasts more than 60 minutes.
   (4) Headache follows aura with a free interval of <1 hour.

Typical Auras include

- Homonymous visual disturbance
- Unilateral parasthesias
- Aphasia or speech difficulty
- Unilateral weakness

2. TENSION-TYPE HEADACHE

2.2. ***Episodic tension type headache***

A. At least 10 previous headache episodes fulfilling B-D listed below. Number of days with such headaches <180/year (<15/month)
B. Headache lasting from 30 minutes to 7 days.
C. *At least 2 of the following pain characteristic:*
   (1) Pressing/tightening (non-pulsating)
   (2) Mild or moderate intensity
   (3) Bilateral location
   (4) No aggravation by routine physical activity
D. *Both of the following*
   (1) No nausea or vomiting
   (2) Photophobia and phonophobia is absent, or one but not the other is present.

2.2. ***Chronic Tension type***

Average headache frequency >15/month for >6 months fulfilling C and D listed above for episodic tension.

* In both 1 and 2, history and physical and neurological examination do not suggest a structural disorder; or it is ruled out by appropriate investigation; or if present migraine or tension type headache do not occur for the first time in close temporal relation to the disorder.

Migraine without aura is reported to be more common in children(13,14). The headache is identical in both classic and common migraine. A major feature of pain is throbbing or pulsatile nature. Other important associated symptoms are presence of nausea and/or vomiting, abdominal pain, photophobia and phonophobia, and facial pallor. Sleep is generally effective in relieving migraine headache. Pediatric migraines are often bilateral. Migraine headaches usually last for a few hours and usual frequency is 2-4 times in a month. More than 8-10 episodes in a month should arouse suspicion regarding the diagnosis. In younger children stereotyped paroxysmal pattern of vomiting, behavioral withdrawal and sleep in patients with family history of migraine may point to the diagnosis(15). Aura is an inconsistent feature in pediatric migraine. Visual aura is the commonest with scotomas and visual distortions.

Complicated migraine occurs in 5-10% of all migraines and is characterized by neurological signs that develop with headache and persist for hours or days beyond the headache. The presence of neurological signs necessitates further evaluation; therefore, complicated migraine remains a diagnosis of exclusion. Complicated migraine implies a greater risk of an organic lesion and therefore, is an indication for CT. Basilar artery migraine is the most frequent of the complicated migraines and represents 3-9% of all pediatric migraines. Dizziness, vertigo, ataxia are the usual early signs which are followed by headache phase. Unlike the typical frontal or temporal location, the headache may be occipital in nature and may not be typically pulsatile or throbbing as in case of common migraine. The differential diagnosis of basilar artery migraine includes acute labyrinthitis, Arnold Chiari malformation, head trauma, AV malformations and complex partial seizures. The evaluation of basilar artery migraine should include EEG and imaging studies to rule out such disorders.

Vascular Headache Other Than Migraine

AV malformations can present as chronic periodic headache either with the quality of common or with classic/complicated migraine(13). Similarly patients with vasculitis syndromes like SLE or polyarteritis may complain of vascular headaches.

Cluster headaches are rare in children. The typical attack occurs 2-10 times per day and lasts for 10 minutes to 3 hours (average 45 minutes). The pain may awaken the patient from sleep. The pain is severe, unilateral, localized in and about the eye and is accompanied with ipsilateral lacrimation and runny and stuffy nose.

Chronic Progressive Headache

Chronic progressive headache denotes a pathologic process within the skull *(Table I).* Rapid diagnosis and appropriate treatment is necessary in such cases. The patients with this type of headache generally have other prominent symptoms besides the headache. Symptoms of raised intracranial tension include nausea, vomiting, visual disturbances, personality changes and even seizures. Papilledema may be the first sign of raised intracranial tension. Neurological signs if present, indicate the site of involvement. CT and if necessary, MRI are diagnostic in such cases.

Headache in hydrocephalus is due to increase in ventricular volume and later increase in pressure. Signs of non-localized raised intracranial pressure, macrocephaly and cracked pot sign may be present in chronic hydrocephalus.

In brain tumors the pain is either due to traction on pain sensitive structures or due to hydrcephalus. Location of pain is of limited value although supratentorial tumors

generally cause frontal headache and posterior fossa tumors cause occipital headache. The headache gets worse on coughing, and defecation. About 70% of children with brain tumors have headache as presenting symptom. The headache often awakens the patient at night.

Benign intracranial hypertension (pseudotumor cerebri) is a syndrome of headache and papilledema with normal CSF composition in face of CSF pressure greater than 200 mm of water. The intracranial-hypertension in this entity is because of either increased brain water or reduced resorption of CSF. Headache can be constant or intermittent. Pain is usually frontal, throbbing and gets worse on leaning fowards or performing valsalva manuover. Visual changes are very common. Many disease states, particularly endocrinal disorders, and drugs (vitamin A, phenytoin and tetracycline) have been associated with benign intracranial hypertension. Papilledema and enlarged blind spot are the other findings. CT scan is diagnostic. MR venography may show lateral sinus thrombosis. In countries where neuro-cysticercosis is endemic, a picture similar to pseudotumor cerebri is seen with parenchymatous invasion of brain with cysticerci(16).

Arnold Chiari malformation may present with chronic headache often located suboccipitally. Pain in the neck, sensory symptoms, weakness of upper extremities and gait disturbances may be the other features.

Chronic Nonprogressive Headache (Tension Headache)

Tension headaches, also referred to as chronic muscle contraction headache and psychogenic headache, are the most frequent type of headaches affecting the general population. There are conflicting reports on prevalence of tension headache. According to some studies perhaps three times as many adolescents have this form of headache than have migraine(17). However, a recent study(18) found only 0.9% prevalence in children 5-15 years old. Tension headaches can be further classified depending upon the pattern into acute intermittent tension type headache and chronic tension type. The acute intermittent is the most frequent type affecting the general population although most patients may not report to a physician for this kind of headache. Adolescents may overuse analgesics for such episodic headaches often leading to rebound headaches. Chronic tension type headache occurs on an almost daily basis affecting more than 15 days in a month.

Headache is frequently described as frontal and pressing in nature, although sometimes it may be a band like pain or bioccipital. Most children with these headaches continue with their activities despite the headache and do not try to sleep, although some may shun light or noise. These headaches rarely if ever awaken the patient from sleep although they may be present in morning. There are no other symptoms suggestive of any neurological or systemic disease and examination and investigations are normal. Tenderness in the occipital and cervical region may be present. School absence is an important associated feature. In some patients stress factors may be obvious. However, in the majority of patients these factors may be found only after a detailed psychological evaluation. It has been suggested that if the patient has had constant headache for longer than 6 weeks, in the absence of symptoms of increased intracranial tension and in the presence of a normal general physical examination and detailed neurological examination, an organic etiology is unlikely.

Two otherwise benign causes of chronic daily headaches deserve mention. Chronic pansinusitis may present with chronic headache without the usual sinus symptoms and may be diagnosed only on imaging(19). Rarely, subtle ocular defects of convergence may masquerade as tension headache.

Evaluation of a Patient with Headache

The key to successful management lies in taking good history, interpretation of symptoms and a thorough physical examination. The history should include the following features of headache: temporal profile, laterality, location, duration, presence of nausea and/or vomiting, focal elementary or behavioral neurologic symptom. In addition, triggering and aggravating factors should be looked into.

Laterality: It is important to distinguish whether headache is unilateral from onset or is unilateral initially and then becomes bilateral. Muscle contraction headaches and common migraines are bilateral from onset whereas classic migraine is more often unilateral. In unilateral headaches, it is important to find if the sides alternate; alternating laterality would support the diagnosis of classic migraine whereas a headache which is consistently localized to one side should arouse the suspicion of focal space occupying lesions.

Location: Location of the headaches may also point to the diagnosis. Occipital headache point to occipital neuralgia, basilar migraine or upper neck problem. Retro-orbital pain is often seen in cluster headaches. Muscle contraction headaches are either bifrontal or bioccipital.

Description of Pain: Children may find it difficult to describe the type of pain felt. Migraine is classically described as throbbing or pulsatile whereas muscle contraction headaches are described as band like. Headache of increased intracranial pressure is made worse by recumbency, coughing and straining. History of warning symptoms is elicited in an age appropriate language. In young children, the mother may tell that the child becomes pale or lethargic before he actually complains of a headache. If the warning symptoms are focal and localized to same side a structural cause should be suspected. The severity of the headache does not necessarily correlate with severity of the disease. Mild headaches may be associated with serious medical problems.

Aggravating and Relieving Factors: Association of these factors is important for management of the patient and may also point to the diagnosis. Photophobia and phonophobia are common in migraine and sleep relieves the headache.

Duration: Duration of headache is important for distinguishing between periodic headache syndromes and headache as manifestation of seizure disorder. Whereas seizures last for minutes, the headache syndrome lasts for hours. Cluster headaches usually last 30-60 minutes. A headache that is always there is more likely to be psychogenic in origin.

The presence of neurological symptoms and/or signs in between the headaches is particularly important to differentiate progressive headaches from those that are non progressive. A patient with raised ICP or mass lesion will have symptoms in between the exacerbation of headache, *e.g.,* nausea or visual difficulty.

A family history of migraine and past history of motion sickness is highly associated with migraine. Sometimes

headache may be a learned behavior from adults suffering from headaches in the family.

Assessment

The physical examination should include measurement of weight, height, head circumference, blood pressure and careful inspection of skin for neurocutaneous stigmata. Complete neurological examination including fundoscopy should be done in all cases. The frontal and maxillary sinuses should be percussed and temporal area and orbits auscultated for the presence of bruits. The posterior cervical muscles should be palpated with the patient in a supine position. Painful parasthesias shooting down the neck upon hyperflexion of neck are suggestive of upper cervial lesion(20). It is prudent to include tests for visual acuity and latent squint, and to check for dental malocclusion.

At the end of history and physical examination, the physician should be able to decide whether the headache is acute, acute recurrent or chronic progressive/non progressive. The next step is to decide as to the cause of the headache. Specific tests and the urgency in obtaining them will obviously depend on the presumed etiology, particularly in acute and chronic progressive headaches. If the diagnosis is muscle contraction headache or migraine there is no need for further neuro-diagnostic studies.

Indication of CT/MRI

The probability of headache due to a intracranial tumor causes irrational fear in doctors and leads to unnecessary imaging. Headaches which awaken the child from sleep, get worse on coughing or straining or if headaches are increasing in severity or frequency should alert the physician. The indications of CT scan are shown in *Table III.* Magnetic resonance imaging is a better modality if AV malformations are suspected.

TABLE III- *Indications of CT/MRI in Patients with Recurrent or Chronic Headache**

1. Age less than 5 years
2. Short stature
3. Recent change in behavior
4. Symptoms and/or signs of raised intracranial pressure
5. Frequent nocturnal awakening due to headache or early morning headache especially if history is less than 6 weeks or headaches are increasing in severity.
6. Complicated migraine
7. Abnormal neurological signs including enlarged head circumference and reduced visual acuity.
8. Cough headache
9. If migraine and seizure phenomenon occur in the same episode

* Adapted from Barlow(13).

In patients with pseudotumor cerebri, MRI and MR venography are better modalities. Plain radiography or computed tomograph of the sinuses is indicated in case chronic sinusitis is suspected.

EEG

EEG is not a rewarding investigation in diagnosis of chronic headaches(21). Non-specific EEG abnormalities are common in migraine(22) but EEG is only indicated if there is a suspicion of headache as a manifestation of epilepsy. In such cases, an EEG may be useful but again it should be remembered that 10% of patients with migraine may have benign focal epileptiform discharges not related to headaches(3).

Management

General Measures

A large majority of parents seek medical attention due to anxiety regarding the cause

of headache. Therefore after history, examination and if necessary, imaging, the physician must reassure the parents and the child regarding the cause of headache. In patients in whom the diagnosis is not clear but are otherwise normal on clinical examination, a revisit following an observation period during which the patient or parent maintains a headache calendar will clarify the matter.

Patients with migraine should know that it is a periodic disorder with good spells and bad spells and often the good spells will last for years. Regulation of sleep and work and avoiding the triggers will help to reduce headache. An acute episodic tension type of headache in a child or adolescent can be treated by acetaminophen or ibuprofen and rest. However, if it becomes a daily affair, detailed psychological evaluation is required to ascertain whether the headache is associated with depression or it is an expression of some situational problem. Identifying and resolving the situation will help the patient in the latter case. In case tension headache coexists with depression, consultation with a psychiatrist should be sought.

Biofeedback, relaxation and stress management training are useful in a large percentage of patients with headaches. Skin temperature biofeedback is easily learned by children and has proved effective and without negative side effects(23). Behavior contingency management by the parents has also been shown to be an effective treatment for children with psychogenic headaches. This comprises of teaching the parents to reinforce non-pain behavior such as increase in daily activities, and to avoid attending to pain complaints.

Pharmacological Treatment of Migraine Treatment of the Acute Attack

Ergotamine compounds are traditionally used in adults to abort the attack particularly when the patient gets aura. However, these are of limited use in children due to inconsistencies of aura in children and the side effects of the drug. The mainstay of pharmacological treatment is oral analgesics. The analgesic should be taken as early in the headache as possible. Acetaminophen (10 mg/kg/dose), ibuprofen (5-10 mg/kg/dose) and naproxen sodium (5-7 mg/kg/dose) are all effective. Even though the headache does not disappear completely with these, the patient gets substantial relief. Nearly 90% of children with migraine experience some amount of nausea and/or vomiting. The accompanying gastric stasis also reduces the absorption of oral analgesics. Antiemetics are useful in such patients. Indeed antiemetics have been shown to be effective in ameliorating all symptoms including headache itself. Metoclopramide, domperidone and promethazine are all effective.

Sumatriptan, a 5HT receptor agonist, has potent activity at 5 HT-1A receptor site. The clinical efficacy of sumatriptan (oral, subcutaneous and intranasal formulations) has been documented in adults. Subcutaneous sumatriptan was found to be effective and safe in childhood migraine in an open prospective study(24). Double blind placebo controlled pilot studies of oral sumatriptan in adolescent migraine patients also showed that the drug was superior to placebo and was well tolerated(25). The cost of this drug is prohibitive at present and further trials are required to recommend it in childhood migraine.

Prophylactic Agents

Majority of children do not require prophylactic treatment. A small group of children whose lives are disrupted by severe and frequent headaches especially if complicated by neurologic symptoms may require prophylactic agents(26). There is a paucity of double blind placebo controlled trials in management of migraine

in children. Anecdotal experience of pediatric neurologists suggests that propanolol, cyproheptadine and pizotifen are the common drugs used for prophylaxis. Of these cyproheptadine (2-8 mg) at bedtime is perhaps the cheapest and often used although its use has not been supported by research. Pizotofen is well tolerated but causes drowsiness in some patients. The beta blocker propanalol has been subjected to three double blind placebo controlled trials, 2 of which showed no benefit of the drug over placebo(27). However, it is still valued as a first line drug for prophylaxis. The usual starting dose is 10 mg twice daily which can be increased depending on the efficacy and side effects. A wide variety of other drugs have been used as second line drugs for prophylaxis. Calcium channel blockers-verapramil and flunarizine, and anticonvulsants like valproic acid, phenytoin and carbamazepine are also effective prophylactic agents and may be considered in patients who do not respond to conventional therapy. Migraine in children has a high rate of remission. The prophylactic drugs can be tapered and discontinued after the child has been in remission for 3 months.

Pharmacological Treatment of Other Headaches

An episodic tension headache is amenable to common analgesics such as acetaminophen or ibuprofen. The chronic tension type of headache requires detailed psychological evaluation and judicious use of anti-depressant or anxiolytic drugs in consultation with a child psychiatrist.

Cluster headache is rare in children. Ergotamine compounds or sumatriptan can be used to relieve the acute episode. The headache syndrome classified under indomethacin responsive headaches are specifically responsive to indomethacin. However, these are very infrequent in childhood.

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NOTES AND NEWS

SECOND AIIMS WORKSHOP ON NEONATAL VENTILATION

The Division of Neonatology, Department of Pediatrics, All India Institute of Medical Sciences, New Delhi, is organizing a Workshop on Neonatal Ventilation from February 3-5, 1997. It will focus on practical aspects of assisted ventilation of newborn infants. Overseas faculty will consist of Dr. Vinod Bhutani, Dr. D. McMillan and Dr. Nalini Singhal. The format of the Workshop will be skill-oriented with emphasis on group work in tutorials and on problem-solving. If interested, please write to the undersigned along with registration fee of Rs 750/-(by cheque/DD in favor of CME in Neonatology AIIMS by 31st Dec, 1996. The number of participants will be restricted to 30, on the 'first come, first served basis'. Please contact: Dr. V.K. Paul, Additional Professor, Department of Pediatrics, AIIMS, Ansari Nagar, New Delhi-110 029. Ph.: 6594372 (O); 6868849 (R); Fax: 686 2663