# COMMON PAEDIATRIC EMERGENCIES AT THE UNIVERSITY COLLEGE HOSPITAL

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*In the developing world, emergency situations are common in paediatric wards due to the fact that:*

*The guardians and parents bring minor illness of children to the hospital very late.*
*Children often cannot complain*
*Financial constraints and ignorance on the part of the parents make them seek alternative help first.*
*Diseases progress very rapidly in children.*
*Children are very curious and active and are therefore prone to accidents and poisoning.*

In our environment, top on the list of common emergencies are infectious and preventable diseases.

This paper highlights the commonest diagnoses made at the Otunba Tunwase Children's Emergency Ward (OTCHEW) in U.C.H., Ibadan; their presentation, commonest causes and management undertaken in these cases.

In U.C.H., Bamgboye and Familusi reported on the commonest diagnoses at the Children Emergency Room (CHER) in 1978, 1981-1986.

In order to detect any change in the trends of prevalent diseases in OTCHEW, we carried out a review of admissions into the ward over a 1 year period (April 1999-March 2000). The results are compared below in the table. The results are similar and a reflection of observations reported from other Nigerian Teaching Hospitals[3].

| Rank | Clinical diagnoses | 1999/2000 Frequency | % | 1978/1981-6 Frequency | % |
|---|---|---|---|---|---|
| 1 | anaemia | 278 | 12.92 | 959 | 5.63 |
| 2 | malaria | 272 | 12.64 | 313 | 1.84 |
| 3 | bronchopneumonia | 254 | 11.80 | 1,231 | 7.23 |
| 4 | gastroenteritis | 129 | 5.99 | 1,606 | 8.60 |
| 5 | febrile convulsion | 123 | 5.72 | 687 | 4.03 |
| 6 | sickles cells disease | 117 | 5.44 | 259 | 1.52 |
| 7 | neonatal tetanus | 115 | 5.34 | 261 | 1.53 |
| 8 | neonatal sepsis | 95 | 4.41 | 866 | 5.08 |
| 9 | neonatal jaundice | 74 | 3.44 | 1,827 | 10.73 |
| 10 | measles | 70 | 3.25 | 1,078 | 6.33 |
| 11 | meaningitis | 56 | 2.60 | 748 | 4.39 |
| 12 | septicaemia | 52 | 2.42 | 866 | 5.08 |
| 13 | acute respiratory infection | 52 | 2.42 | 150 | 0.88 |
| 14 | asphyxia | 48 | 2.23 | N.A | N.A |
| 15 | low birth weight | 42 | 1.95 | N.A | N.A |
| 16 | intestinal obstruction | 35 | 1.63 | 192 | 1.13 |
| 17 | lobar pneumonia | 31 | 1.44 | 169 | 0.99 |
| 18 | malnutrition | 28 | 1.30 | 476 | 3.15 |
| 19 | seizure disorders | 25 | 1.16 | | |
| 20 | bronchial asthma | 21 | 0.98 | N.A | N.A |
| 21 | heart failure | 17 | 0.79 | 349 | 2.05 |
| 22 | tuberculosis | 16 | 0.74 | N.A | N.A |
| 23 | failure to thrive | 16 | 0.74 | N.A | N.A |
| 24 | neuphrotic syndrome | 14 | 0.65 | N.A | N.A |
| 25 | pyrexia of unknown origin | 10 | 0.46 | 114 | 0.67 |
| | TOTAL | 1995 | 92.46 | | |

Findings are similar to those in other Nigerian Teaching hospitals[2]. Some of the commonest childhood emergencies seen in the University College Hospital and their common presentations, aetiology and management are highlighted.

## 1. NEONATAL JAUNDICE

Neonatal jaundice can be pathological or physiological. It is pathological if it appears within the first 48 hours of life; if serum bilirubin is greater than 12mg/dl and it lasts longer than 10-14 days. The rate of rise of serum bilirubin greater than 5mg/dl/24hours also connotes a pathological process.

Common causes here include:

* G6PD deficiency
* Sepsis
* Blood group incompatibility especially ABO incompatibility
* Prematurity

### *Management*

1. For physiological jaundice - the mother is reassured and bilirubin level is checked if necessary the following day.
2. Photo theraphy is instituted when serum bilirubin is greater than or equal to 15mg/dl in a healthy term neonate.
3. Exchange blood transfusion (EBT) is carried out when serum bilirubin is greater than or equal to 20mg/dl in full term neonate or 10mg/dl/kg for preterm infants. EBT is performed at lower bilirubin levels in infants with sepsis, hypoglycaemia or history of birth asphyxia. Especially in the case of phototheraphy, adequate fluid intake must be carried out.

## 2. MALARIA

Malaria, especially that caused by *P. falciparum* remains a major health problem in the developing world.

### *Presentation*

In this environment, malaria mainly presents in childhood with severe manifestations and complications which include: Cerebral malaria, severe anaemia (the commonest here), jaundice, acute renal failure, hyperpyrexia-temperature of greater than 29°C, haemoglobinuria, circulatory collapse, pulmonary oedema and hypoglycaemia (blood glucose<40mg/100ml).

In general, fever is the commonest symptom in malaria. However intermittent periodicity of the fever is usually absent at the beginning of illness. Other features include: headache, malaise, fatigue, muscular pains, diarrhoe, vomiting, febrile convulsions which are frequent in children with temperatures above 39.4°C. The patient may be pale and liver and spleen may be enlarged.

***Manifestations:*** **Cerebral malaria.**

This is a state of altered consciousness in a patient who has asexual P. falciparum in the blood and in whom no other cause of altered consciousness can be found.

The degree of impairment of consciousness (from confused to frank coma) and there may or may not be focal or generalized seizure.

***Diagnoses***

A definite diagnosis is made on the finding of parasites in the peripheral blood film

***Chemotherapy***

A. Acute uncomplicated malaria
   1. Chloroquine-25mg (base)/kg over 3days. Oral (preferred route) 10mg/kg stat on days 1 and 2 then 5mg/kg on day 3.
   2. Sulfadoxine + pyrimethamine

Ancillary treatment:
   1. Tepid sponging/fanning/antipyretics
   2. Anticonvulsants where necessary - paraldehyde
   3. Correct severe anaemia (pcv<16%) is correct with packed red cells transfusion given at 10-15ml/kg. I.V Frusemide 1 mg/kg is often given to prevent or treat circulatory overload.
   4. Haematinics

B. For cerebral malaria, quinine is the drug of choice I.V 20mg/kg in 10ml/kg 5% Dextrose (loading dose). Then I.V 10mg/kg 5% dextrose over 2 hours is given 8 hourly till arousal, then orally 10mg/kg 8 hourly to complete 7days treatment. Total dose is 21 doses.

## 3. ANAEMIA

Anaemia is among the ten top causes of death in childdhood.It is one of the most common conditions affecting children not only in the tropics but also in temperate zones.

Anaemia is defined as a condition in which there is a decrease in haemoglobin concentration or red cell count below the normal for age and sex. See table 3) *OR* haematocrit of less than 30% after the age of one month.

***Normal Values***

The normal full term baby is born with a high haemoglobin of about 18-20g/dl, which is necessary for the foetus while in utero. however after birth, the haemoglobin levels falls to about 11g/dl by the age of 4 months. From then onwards the level rises gradually. (Table 3)

**TABLE 3**

Normal mean and lower limits: haematological vaale in children

| | Haemoglobin g/dl | | Haematocrit % | |
|---|---|---|---|---|
| | *Mean* | *lower limit (2s.d)* | *Mean* | *Lower limit (2s.d)* |
| *New born* | *16.8* | *13.5* | *55* | *45* |
| 1wk-6mths | 13.0 | 11.0 | 36 | 31 |
| 6mths-2yr | 12.5 | 11.0 | 37 | 33 |
| 2-4years | 12.5 | 11.0 | 38 | 34 |
| 4-8 years | 13.0 | 11.5 | 39 | 35 |
| 8-11years | 13.5 | 12.0 | 40 | 36 |
| 11-14yrs (f) | 13.5 | 12.0 | 41 | 36 |
| (m) | 14.0 | 12.5 | 43` | 37 |
| 14-18 yrs (f) | 14.0 | 12.0 | 41 | 36 |
| (m) | 16.0 | 14.0 | 46 | 38 |

Classification of anaemia in childhood: common cause

A. Decreased red cell production
   1. Depletion of iron. folate, protein, vit $B_{12}$ etc
   2. Bone marrow failure - primary marrow aplasia or hypoplasi, secondary marrow aplasia due to toxins and chronic infection.
   3. Lack of erythropoeitin e.g. chronic renal failure

B. Blood loss
   1. Acute haemorrhage - placental, cord, visceral, haemorrhagic disease of the newborn, accidents
   2. Chronic haemorrhage - hookworm, amoebiasis, GIT disease.

C. Excessive Red Cell Destruction
   1. Infection - malaria, sepsis, kala azar
   2. Hereditary - sickle cell disease, thalasaemia, G6PD with sensitivity to drugs (sulphonamide, fava beans)

***Clinical features***

* Pallor which can be judged best from appearance of the lips, fingernails, toenails, the palm of the hands, tongue and the conjunctiva
* Fatigue
* Anorexia
* Irritability
* Listlessness
* Severe anaemia-heart failure
* Jaundice, hepatosplenomegaly, peripheral lymphadenopathy are often present in haemolytic anaemia.
* Pica, koilonychia-in iron deficiency
* Reduced I.Q

***Investigations***

i. FBC- red cell morphology, abnormal shape (spherocytes, sickled cells, target cells (thalassaemia, HbSS)
   - Rise in wbc is often indicative of infection
   - Fall in wbc in malignancies especially with bone marrow replacement.
   - Fall in platelet count in aplastic anaemia, leukemia, severe infections.

ii. Blood film for malaria parasite

iii. Haemoglobin electrophoresis

i-iii must be carried out in any child with anaemia. Other ancillary investigations that may be done include:

a. reticulocyte count (normal is 0.5-1.5%). This is increased in haemolytic anaemia.

b. Coomb's test is positive in autoimmune haemolytic anaemia

c. Enzyme assays e.g. G6PD, pyruvate kinase

iv. Stool examination-occult blood in stool is positive in GIT bleeding and hookworm infestation. Ova of hookworm are also seen in the latter condition.

***Treatment***

This depends on aetiology, degree of anaemia, and rate of development of anaemia.

***Outline of Management***

* Treatment of aetiologic factors
* Provision of deficient nutrition factors iron, folic acid, vit B 12.

* Corticosteroids used in the treatment of autoimmune haemolytic anaemia
* Androgenic steroids-aplastic anaemia
* Splenectomy-hereditary spherocytosis
* Bone marrow transplant useful in aplastic anaemia, SCD.
* Blood transfusion. This is given to treat severe anaemia especially when it is acute. It is often not given until pcv is less than 17%. Packed red cell transfusion is preferred. It is given at 10-15 mls/kg over 1.5-2 hours. Whole blood transfusion (15-20 mls/kg) is given to treat acute blood loss or to patients with protein-energy-malnutrition. I.V frusemide 1-2mg/kg is often given to prevent or treat circulatory overload.

**Note:** Children with chronic anaemia can withstand low haemoglobin concentration than normal so do not rush to transfuse.

## 4. PNEUMONIA

Pneumonia is defined as acute inflammation of the lung parenchyma due to micro-organisms. About 5 million deaths that occur in under-five is due to acute respiratory tract tnfection and about 75% of these are due to pneumonia associated with measles.

### *Risk factors for childhood pneumonia*

* Malnutrition
* Indoor air pollution
* Low birth weight
* Non breast feeding

Others include: Social demographic factors, previous hospitalization for pneumonia, and previous of wheezing.

### *Aetiology*

The possible micro-organism that could cause pneumonia in childhood include:

1. Bacteria (constitute 71%) e.g. *Streptococcus pneumonia, H. influenza* especially b sero-type.
2. Viral: measles, respiratory syncytial virus, adenovirus, influenza virus.
3. Non viral, non-bacterial: *Mycoplasma pneumonia*, chlamydia
4. Protozoan: *Pneumocystitis carinii*
5. Fungi; canada, aspergillus, histoplasma.

### *Clinical Features*

This depends on the following factors:

1. Bacteria pathogen i.e. the infecting organism
2. Age of the patient
3. Immunological status of the patient
4. Presence or absence of underlying disease.

General: fever, chill, headache, irritability, vomiting.

Specific

* pulmonary - cough (usually absent in neomates)
  - difficulty in breathing
  - in drawing
  - crackle
* Pleural chest pain
* Extra pulmonary-skin abscess, otitis media
* General-pyrexia (temperature may be as high as 45°C but not always)
* Pulmonary signs: nasal flaring, tachypnoea, in drawing (suprasternal, intercoastal, subcoastal), alteration of percusission notes (this is of no diagnostic value in patchy pneumonia, but in lobar pneumonia it may be diagnostic).
* Breath sounds may be reduced and crackles may be present

### *Investigations*

3 basic clinical investigations are required.

1. Chest X-ray. patchy or lobar consolidation is suggestive though not pathognomonic. Demonstration of hyperinflation is most consistent with viral infection.
2. Full blood count: increase in total WBC<ESR<C-reactive protein
3. Blood culture: positive in 10%-30% of cases.

Other investigations not often done include:

* Rapid agglutination test for detection of antigen.
* Lung aspiration for m/c/s, ZN stain. Indication for lung aspiration include:
  a) Child that is severely ill so that appropriate antibiotics can boost up the child on time.
  b) Underlying immunodeficiency
  c) Research purposes
* Nasopharyngeal aspiration for;
  a) Viral identification
  b) Mycoplsma and ureaplasma
  c) Bordetella species.

### *Treatment*

The treatment modalities of pneumonia are in 3 phases.

a) Definitive-use of antibiotics: this depends on the severity of the illness. if not severe, trimethoprim sulfamethaxazole or procaine penicilline is given for 5 days. If severe but well nourished give chloramphenicol and erythromycin if there has been no previous antibiotics use. With previous antibiotics use give crystalline penicillin. If severe and malnuourished give cloxacilling and gentamicin (also for Staph aureus) for 5 days.

b) Supportive treatment.
   * Give oxygen if necessary
   * Calorie intake.
   * If in respiratory distress-mechanical ventilation is used.

c) Treatment of complications
   H - Heart failure
   E - Empyema
   A - Atelectasis
   R - Acute respiratory failure
   4 Ps - pneumatocoele, pneumothorax, pyopneumothorax, pleural effusion, septicaemia.

## 5. FERBRILE CONVULSION

A febrile convulsion is defined as a seizure occuring in a child between 6 months and 5 years, associated with fever (temperature >38°C) but without any evidence of intracranial infection or pre-existing neurological anomaly.

### *Incidence*

* Peak age is 1-3 years but can occur in as low as 3 months and as high as 6 years.
* Females are more affected than males. Peak incidence is 13-15

months in girls and 15-18 months in boys. There is an increased incidence in some families

***Causes***

In this environment, febrile convulsion is caused mainly by malaria, upper respiratory tract infections (URTI) and urinary tract infection (UTI). In the western world it is caused by URTI, UTI and pneumonia. Other causes include gatroenteritis (shigella). Febrile convulsion can result from any disorder, which cause a rise in temperature in susceptible children.

***Clinical Features***

Based on the clinical features, convulsions can be classified into:

1. Simple (benign) febrile convulsions: Seizures that are less than 15 minutes, with no associated focal features. If they do occur in series, have a total duration of less than 30 minutes.
2. Complex febrile convulsions: Seizures last longer than 15 minutes, there is associated focal features or post-ictal paresis and occur in series with a total duration greater than 30 minutes or multiple in a 24 hour period.

***Signs and Symptoms***

* Significantly elevated body temperature (38-39$^0$C)
* Majority of febrile convulsions are seen on the first day of illness.
* Seizures are usually generalized tonic clonic but without aura. Focal seizures occur in less than 5%
* Stupor is characteristic though short-lived.
* Incontinence of urine may occur.

***Management***

* Terminate seizure with diazepam.
* Find the cause of the fever and treat.
* Anticonvulsants usually not necessary.

Pyrexia should be reduced by tepid sponging, fanning, maintaining fluid intake and giving antipyretics such as paracetamol.

## 6. NEONATAL TETANUS

Neonatal tetanus (NNT) is an avoidable or preventable cause of neonatal morbidity and mortality. It has been estimated that between 5-60 per 1000 live births (23 - 27%) of all neonatal deaths is due to neonatal tetanus. The incidence, however, varies widely throughout the world; developed countries; 0-1 per 100,000 persons. Developing countries: 10-50 per 100,000 persons.

NNT is substantially underreported in developing countries and it has been said to be prevalent in rural that urban areas. The prevalence of NNT may serve as an index of quality and extent of utilization of maternal health services and impact of immunization programmes.

***Pathogenesis***

Causative organism - *Clostridium tetani* (a gram-positive rod with resistant spores).

Portal of entry:

* Contaminated umbilicus-this is because the cord has been cut with a dirty instrument or as a result of the application of dirty dressing on the cord as practiced in some areas. may also occur due to the use of contaminated methylated spirit in cleaning the cord.
* Ear piercing performed in unhygienic conditions

Following entrance in the body, *Clostridium tetani* produces two toxins-tetanospasmin and tetanolysin. Majority of the clinical features are due to the release of tetanospasmin into the body.

***Clinical Features***

1. Incubation period usually between 3-14 days. it may be as short as 1 day or as long as several months. The shorter the incubation the worse the prognosis.
2. Onset is usually gradual
3. Typically muscle rigidity and spasm are seen.
   * Trismus: occurs first and also in 50% of all cases. patient presents with difficulty in feeding
   * Neck stiffness.
   * Risus Sardonicus
   * Opisthotonus
   * Laryngeal muscle spasm-asphyxia
   * Pulmonary muscle spasm-cyanosis
   * Abdominar muscle spasm
   * Constipation
4. Noise and unexpected light provoke the spasm.
5. There may be fever.

***Management***

* Adequate bed rest.
* Antitoxins. Therapeutic and not prophylactic dose should be given
* Antibacteria - i.m. procaine penicillin.
  - i.v. cloxacillin
  - i.m. gentamicin
* Feeding - nasogastric tube feeding should be emplyed and sterilized expressed breast milk used.
* Sedation-immediate control of spasm with paraldehyde (i.m. 30mg/kg) or diazepam (1-2mg/kg i.m or i.v)
  - Continuous control of sedation with phenobarbitone, chlorpromazine, diazepam.
* Nurshing care-aim is to maintain a clear airway by regular suctioning of accumulated secretions to prevent aspiration pneumonia.

***Prevention***

1. Improved care of the umbical cord
2. Raise immunity of the neonate in highly endemic areas by educating the traditional birth attendants, actively immunizing all pregnant women-at least 2 doses of tetanus toxoid are given at 28 weeks, 32 weeks and 36 weeks, passive immunization of neonates at risk.

## 7. MENINGITIS

This is defined as inflammation of the meninges. It could be of viral, bacterial, fungal or protozoan origin. Bacterial meningitis is the most common life threatening acute infectious disease of the C.N.S in children. It occurs mainly in infants and toddlers, although children and adults of all ages can be affected.

***Aetiology***

* Neonates: Group B Streptococcus, Gram negative bacilli, *Staph. Aureus, Listeria monocytogenes.*
* Children aged 7 months to 12 years - *S. pneumonia, N. menin-*

*gitides, H. influenza*
* Children aged > 12 years - *S. pneumoniae, N. meningitides*

It is important to note that *H. influenza* is the pathogen for about two thirds of infantile and childhood meaningitis.
Sex incidence: male>female

*Clinical Features*
Usually there is an antecedent history of URTI or GTI.
* Acute onset of a febrile illness that can be accompanied by altered consciousness.
* Headache with or without stiff neck (not common in infants)
* Nausea and vomitting
* Anorexia
* Seizures
* Febrile
* Infants have bulging and tense fontanelles.
* Altered level of counsciousness.
* Hyper/hypoactive deep tendon reflexes.
* Older children usually exhibit signs of meningeal irritation: nuchal rigidity, positive Kernig's and Brudzinski's signs.
* Papiloedema may or may not be present
* Cheyne-Stokes breathing
* Signs of circulatory failure-poor pulses, oliguria e.t.c

Focal neurologocal deficits if present are ominous signs.

*Investigations*
1 Lunbar puncture: C.S.F is examined for cell count, protein, sugar and culture.
2. Full blood count
3. Electrolytes and urea
4. blood culture (positive in 80-90%)

*Treatment*
* Admit into the hospital
* I.V. antibiotics to cover all major organism-crystalline penicillin (300,000 i.u/kg/day) in 4 divided doses and Chloramphenicol 100mg/kg/day in 4 divided doses. I.V. third generation cephalosporins e.g. cefotaxime are used in the alternative.
* Intravenous fluids
* N/G tube feeding

Treat for 5 days after defervescence or at least 10 days.

## 8. POISONING

Ingestion of household products by a child is a common medical emergency in a paediatric practise. Incidence is 0.6 - 1.3% in Nigeria while the peak age incidence is 1-5 years. Male to female ratio is 2:1 Predisposing factors:
* Curiosity
* Poor Supervision
* Psychological disturbance especially in children older than 5 years

*Agents*
These depend on the environment as well as socio-economic status of the parents. Common agents include: Kerosene, drugs, cleansing agents, insecticides.

*Management*
Circumstances that increase the risk of ingestion include storing dangerous chemical e.g cleansing agents in driking glasses, soda bottles or unlabelled open containers.

*Examination*
Child may be behaving strategely or unresponsive. take a careful history. Level of consciousness should be monitored frequently.

*Principles of Therapy*

A. Removal of Gastric Contents:
1. Emesis: Emesis should not be induced in management of volatile hydrocarbon ingestion. Emesis may be induced by ingestion of syrup of ipecac, a mixture of plant alkaloids. Children between the ages of 6-12 months should be administered 10 mls; children older than 1 year - 15 mls. followed by fluids for either dose.

Contra Indications:
* Children less than 6 months
* Depressed level of counsciousness
* Corrosives will cause an increased risk of oesophageal burns if vomited.

2. Gastric Lavage: reserved for children who cannot take ipecac.

B. Absorbtion of Poison

C. Intestinal Cleansing: Carthatics are routinely recommended following gastric emptying and absorbtion. Magnesium sulphate administered at 250mg/kg. sodium sulphate at 250mg/kg and magnesium citrate administered at 4 ml/kg are most frequently used.

D. On examination, inspect the mouth for signs of burns, discolouration or particles of the agent as well as the odour of the patient. Note the respiratory pattern watching out for hypopnea (aspirin poisoning) or respiratory depression. The pupilary size can give insight to the type of poison involved.

E. Continued absorbtion of the drug should be prevented by the following measures:
* Removal from the stomach either by induction of emesis or via a gastric large.
* Use of activated charcoal powder.

F. Drug elimination can be further enhanced by use of alkaline diuresis, dialysis or by exchange blood transfussion.

G. Certain antidotes are known to be useful in the reversal of the harmful effects of poisnos. These include:
* Atropine-organophosphates
* Desferioxamine-iron
* Dimercaprol-mercury
* Protamine sulphate-heparin

*Contraindication to the induction of emesis*[5]
1. Ingestion of a causatic or corrosive
2. Loss of protective airways refexes such as in coma or convulsions
3. Ingestion of substances that are likely to produce rapid depression of consciousness e.g ethanol, tricyclic antidepressants, short acting barbiturates.
4. Ingestion of substances that are likely to produce an early onset of seizures e.g camphor, isoniazid, strychnine, tricyclic anti-depressants.
5 Ingestion of petroleum distilates.

6. Prior significant vomiting or haematemesis.
7. Age under 6 months because of possible immature protective airway reflex and the lack of data to establsih the safe and effective dose.
8. Ingestion of foreign bodies, emesis is ineffective and there is risk of aspiration and obstruction of the airway.
9. Neurologically imparaired individuals with possible impaired airway reflexes.
10. Absence of bowel sounds. Gastric lavage is preferred.

***Contra-indications to Gastic Lavage***

1. Caustic or corrosive ingestions because of the risk of perforations.
2. Uncontrolled convulsions because of the danger of aspiratioon or injury.
3. Comatose patients require endotracheal intubation to protect against aspiration.
4. Cardiac dysrhythmias: these must be controlled first because insertion of the tube may cause a vagal response and cause a life-threatening dysrhythmia.

In general, it is important to note that prevention is better in the management of poisoning. This can be done primarily by the effective supervision of children by parents to safeguard their wards from danger. In addition adequate information about poisons in general as well as the establishment of poison information centre would go a long way in reducing the scourge.

## CONCLUSION

In conclusion, certain paediatric emergencies are known to be common in this environment and it is important for any medical practitioner to know about their management. Simple preventive measures can be taken by parents in preventing these diseases. Early presentation at the emergency wards would go a long in reducing mortality associated with these illnesses.

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## ACKNOWLEDGEMENT

*We would like to appreciate the help of Dr. O.J. Fatunde of the Department of Paediatrics who helped in reviewing our work.*