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Volume XVII No. 1 March 1982

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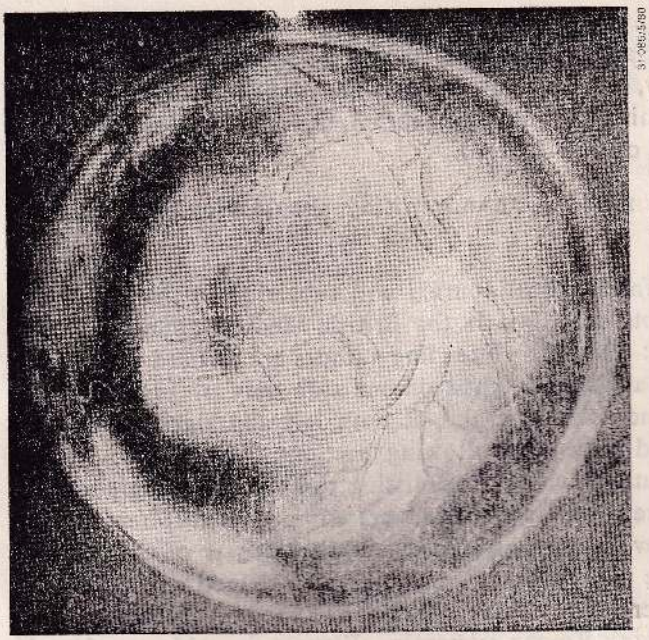

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Communications regarding business matters and advertising should also be addressed to the Editor.

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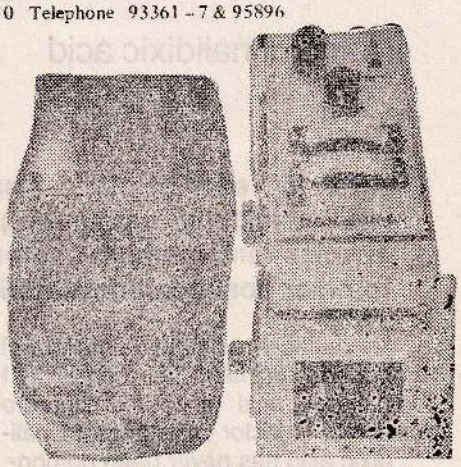

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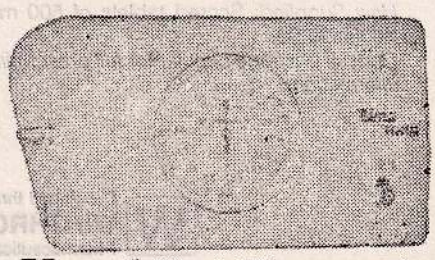

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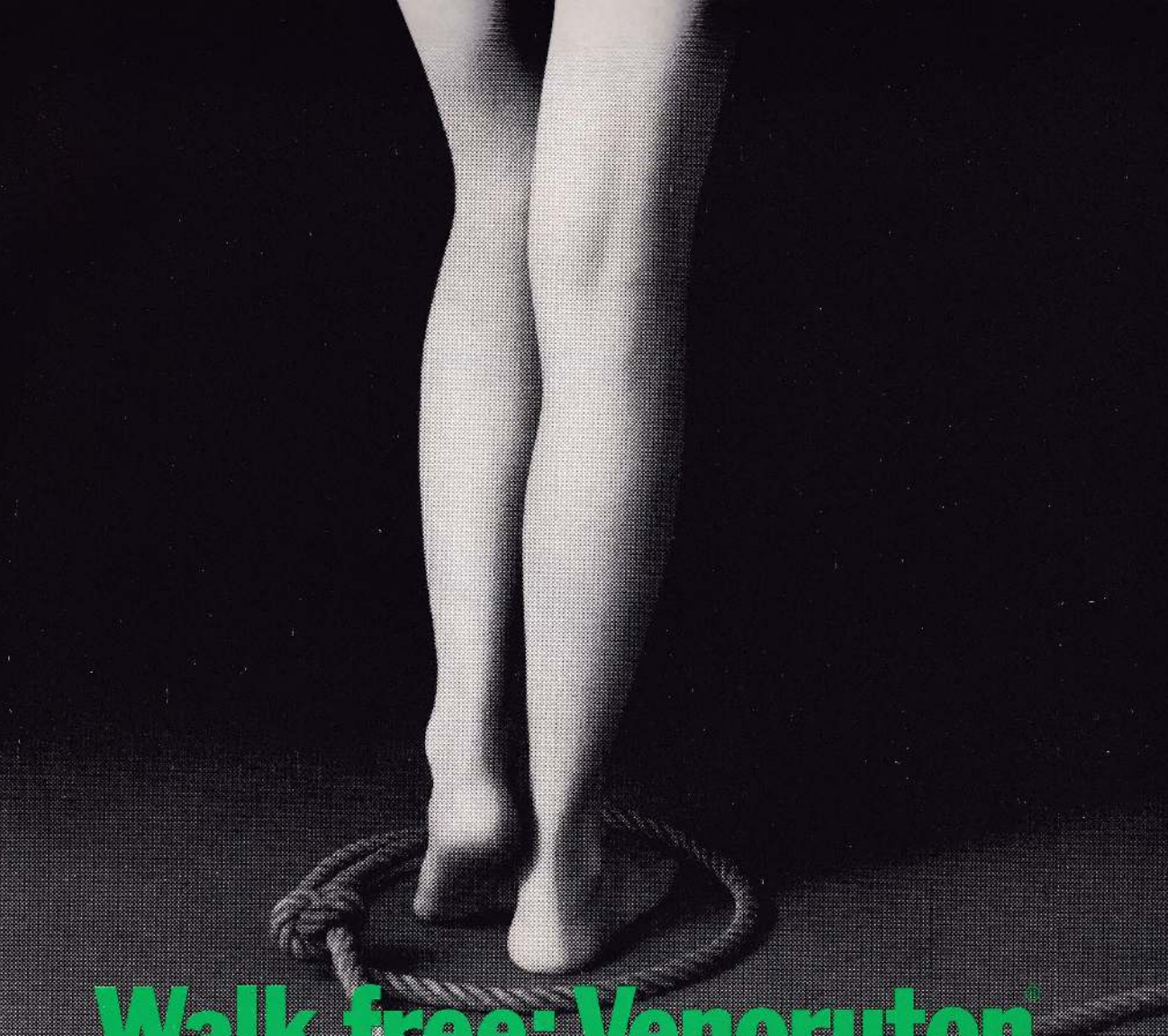

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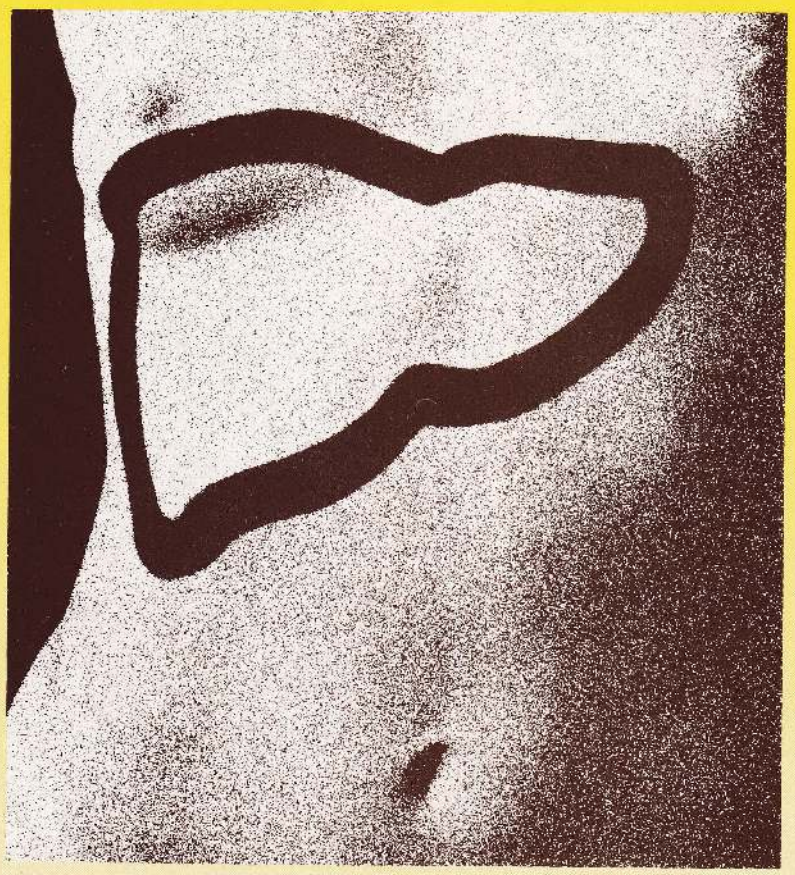

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Editorial

New Hospital for Jaffna.

Medical Education is no joke. It is the sacred responsibility of all concerned, to ensure that the medical undergraduate is given a sound and thorough training, that befits the noble art of healing. In this context the state of affairs at the Jaffna (teaching) hospital, is appalling.

The Jaffna (teaching) hospital, (except for two recent blocks) is a century old or more, with the buildings haphazardly arranged, difficult to maintain and with inadequate space for the routine treatment of patients, let alone teaching. Situated in the heart of the town, subject to the incessant noise around, there is no room for expansion too. The less fortunates, in this part of the world, cannot hope to dream of any space for an Accident Unit, a Burns Unit, a Cardiology Unit an adequate Neuro - Surgical Unit (let alone Neuro - Surgical Empires), all so vital, in this modern age of medicine, for the proper treatment of patients - nay even the proper training of undergraduates and postgraduates.

Before the first batch of students were to start their clinical training, it was pointed out that the Jaffna hospital was totally inadequate for teaching and a New (Teaching) Hospital should be built. That the New (teaching) hospital, should be sited close to the Medical Faculty (at Tirunelvely) was common sense and all were agreed on this question. At a conference held with the Government Agent and the Urban Development Authority Expert, it was decided to build a New (teaching) hospital close to the Medical Faculty, (at Tirunelvely,) as did obtain at Colombo, Peradeniya and Galle. (It is pertinent to record that the **limited** beds teaching hospital at Peradeniya, is proving to be a failure, training having to take place at two institutions, with inadequate interdepartmental collaboration and integrated teaching.) University land adjacent to the Faculty is ideal. The land on which, the Farm school stands is a less suitable alternative. Only a four month course is conducted and an Agriculature Faculty at Kilinochchi is due to be established. Owing to the position of the National Exchequer,it was clear that Foreign Aid had to be sought.

When the Hon. Minister of Health, visited Jaffna, he was appraised of the facts. Having seen for himself, the parlous state of affairs he very gallantly. promised to build a New (teaching) hospital. What has happened thereafter?.

There is no sign of any moves, either to obtain the site or to seek Foreign Aid. A plan to build a new block in the existing hospital (by the D. D. C. not the central authority), to ease the problem till a new hospital is built, has not got off the ground yet. In fact, it is no secret that the plan will not materialise for yet another five years.

Meanwhile, the first batch of students, frustrated, with their voices in the wilderness are struggling along, The second batch has joined the struggle and the third is due to join soon.

It is time that the Hon. Minister steps in and sees that treatment of patients and medical education in Jaffna is more meaningful. A New (teaching) hospital of at least thousand beds close to the Faculty should become a reality soon.

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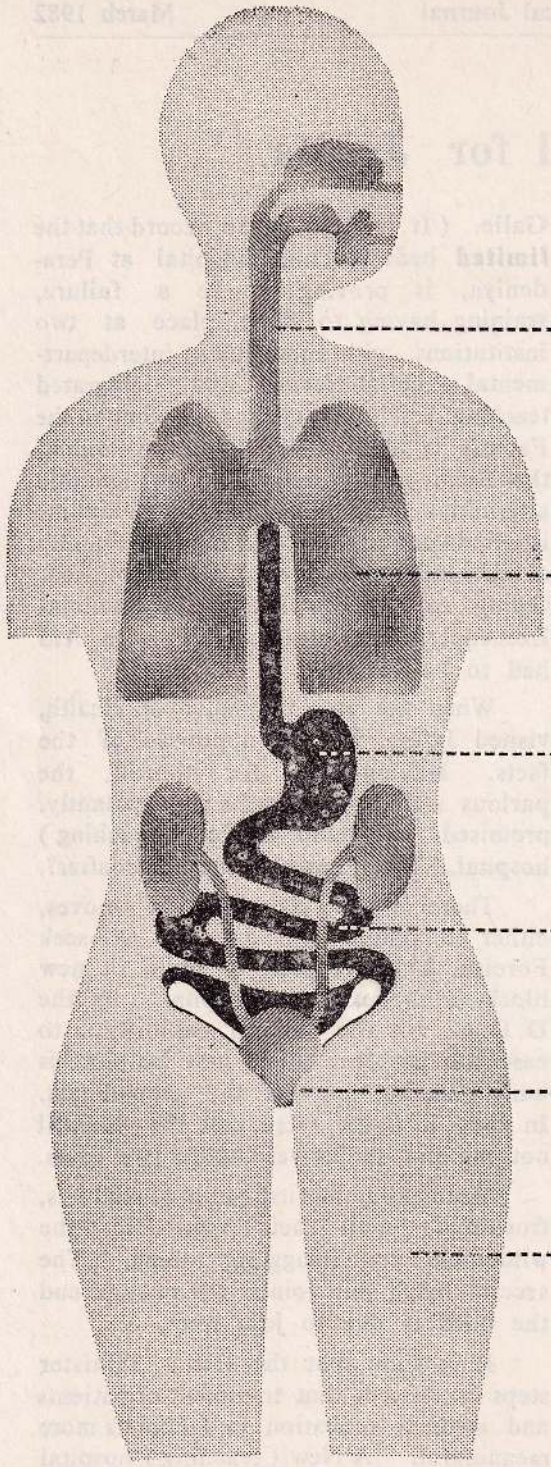

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Nasopharynx - Is it an anatomical entity?

Prof. R. Kanagasuntheram,* M. B. B. S, Ph. D (Camb), F. I. Biol (Lond).

Introduction:

It was Galen (A. D. 130 - 210) who first introduced the term 'pharynx' to denote that portion of the digestive tube connecting the mouth to the stomach. Versalius (1543)[1] introduced the term oesophagus for Galen's 'pharynx'. The subdivision of this tube into an upper part called the pharynx and a lower part, the oesophagus is a change that took place during the eighteenth century. Currently, the pharynx is further subdivided craniocaudally into nasopharynx, oropharynx and laryngopharynx. This sub-division has received acceptance by almost all modern textbooks of anatomy. At present, the nasopharynx is defined as that portion of the pharynx lying above the soft palate, communicating anteriorly with the nasal cavity while it communicates inferiorly with the oropharynx.

The inclusion of the nasopharynx as a part of the pharynx had been criticized by Wood Jones (1940)[2] who regarded it as a posterior continuation of the nasal cavity, a view, earlier expressed by Cunningham (1902)[3] and Negus (1929)[4]. The term postnasal space was introduced by Lambert (1960) since the nasopharynx is, in fact, situated posterior to the nasal cavity proper. Cave (1960, 1967)[5], [6] preferred the term 'epipharynx' as he thought that the nasopharynx belongs neither to the food tube nor the nasal cavity (nasal fossa). He regarded the nasopharynx as a preliminary collecting space for inspired air which is subjected to a cleaning up process by the collection of lymphoid tissue in this region.

Our investigations on the comparative anatomy of the nasopharynx in primates[7] as well as our observations on the embryology[8] and pattern of innervation of this region in the human[9],[10] have convinced us that the rerm 'nasopharynx' is appropriate only if it is regarded as having a respiratory component anteriorly and a true pharyngial component posteriorly. Moreover, there is a general belief that the nasopharynx is a rigid and immobile chamber. On the contrary, it will be shown that only the anterior portion is rigid while the posterior part is quite mobile.

Comparative anatomy:

The nasopharynx of different primates, ie. tree shrew, slow loris, monkey, gibbon and man was sectioned in both coronal and sagittal planes. Moreover, the histology of this region in different primates was also examined.

In all primates, the anterior portion of the nasopharynx proximal to the orifices of the auditory tubes is formed almost entirely by **bony elements** while in the part behind the tubal orifices, the bony component is replaced by **muscular tissue.** Moreover, the anterior bony part is relatively much longer in lower primates than in higher primates including man. Furthermore, the position of the epiglottis occupies a relatively lower position in man whereas it projects above the soft palate in lower primates and thus the epiglottis occupies an intranarial position in these animals. This intranarial position of the epiglottis is an obvious device to

* *Professor of Anatomy and Dean, Faculty of Medicine, University of Jaffna.*

prevent the rapid exit of air from the nasal cavity into the respiratory passages. Thus the air inside the nasal fossa is forced to recirculate among the complex system of turbinate bones where the olfactory receptors are situated. Thus the elaborate conchal (turbinate) system as well as an intranarial epiglottis are found in lower primates as in macrosmatic animals which make the maximal use of the olfactory system. With the increase in visual acuity in higher primates, the conchae become less complex and the epiglottis takes up a lower position and is situated well below the soft palate as in the gibbon and man.

The epithelial changes in the nasopharyngeal region is equally interesting. In front of the tubal orifices, the epithelium is invariably of the respiratory type, ie. columnar ciliated, while behind, the epithelium is generally stratified squamous. However, it is not uncommon to find patches of transitional, stratified squamous or even columnar ciliated epithelium especially in the junctional region. Thus there is no clear or sharp line of demarcation of the two types of epithelia particularly in the human nasopharynx.

Embryology

In human embryos up to the **eighth week,** the epithelium of the primitive nasopharynx consists of a single layer of cuboidal cells. However, the number of layers increases towards the true pharyngeal region. By early **third** month, the cells close to the openings of the auditory tubes show transition into the ciliated type. The process extends into the entire nasopharyngeal region by 50 mm stage. However, at this stage the ciliated epithelium is of the stratified type and the ciliated cells are cuboidal rather than columnar. By the latter part of the third month the entire pharyngeal region is lined by ciliated cells. Soon afterwards, there is a reversal in the trend towards the differentation of the ciliated epithelium in the true pharynx although the nasopharynx still has ciliated epithelium except for occasional patches of transitional epithelium. By the fifth month the ciliated cells have a columnar appearance in the anterior part of the nasopharynx while the epithelium over the lower reaches of the nasopharynx is still of the transitional type with occasional patches of stratified squamous epithelium. In the full term foetus the roof of the nasopharynx is lined by stratified columnar ciliated epithelium while the posterior wall has varying types of epithelia ranging from columnar ciliated to stratified squamous type.

A noteworthy feature during the fifth month of development is the differentiation of lymphoid tissue beneath the epithelium. This epithelium overlying the lymphoid tissue is often nonciliated and is infiltrated with lymphocytes.

The above findings indicate that during transformation of the embryonic (stratified) ciliated epithelium of the caudal part of the nasopharynx into the stratified squamous type, the basal cells of the epithelium serve as a germinal layer for the squamous change during which the superficial ciliated cells are shed into the lumen of the pharynx. This transformation can occur in patches as is revealed by the histology of the full term foetus. It is possible that further transformation into squamous epithelium may take place postnatally in those nonsquamous patches. Nevertheless, it must be emphasized that transformation into stratified squamous epithelium does take place during foetal

life and is therefore not a postnatal phenomenon as is usually claimed. The reason for the proposed postnatal squamous change is said to be due to apposition of the soft palate towards the posterior nasopharyngeal wall during swallowing. Such a theory does not go against the prenatal transformation to squamous change since swallowing movements do indeed occur during foetal life. The junctional zone where stratified squamous and ciliated epithelia meet is always an area where metaplastic and neoplastic changes could occur owing to constant shedding and regeneration of the nasopharyngeal epithelium. Furthermore, the presence of lymphocytes within the foetal epithelium indicates that their presence is not necessarily due to infection as there is usually no such source present in the foetus.

Innervation:

Embryonic and foetal material consisting of 18mm, 31mm, 45mm, 90mm, 140mm and 190mm CR length as well as specimens of later stages including two full term foetuses were available for study. In addition five adult nasopharynges were also examined. Both foetal and adult material were stained by the AChE (Acetylcholinesterase) technique for identifying the parasympathetic neurones while silver staining methods were used to trace the course of the main nerves, the nerve plexuses arising from the main nerves and the nerve terminals.

Extrinsic innervation

Except fot a small area of the roof in front of the tubal orifices the rest of the naspoharyngeal region is supplied by the glossopharyngeal nerve. The fibres of the ninth cranial nerve supplying the roof are conveyed by two routes: (1) via the so-called communicating syampathetic branches which join the greater superficial petrosal nerve and (2) via a communicating twig from the tympanic branch of the glossopharyngeal nerve which joins the greater superficial petrosal nerve. In addition, the glossopharyngeal nerve supplies the posterior part of the soft palate as well.

Ganglia in the nasopharynx

Only the large nerve trunks are seen in the 18 mm embryo while in the 45mm foetus, both the submucous nerve plexuses as well as the nerve terminals are seen. Only simple nerve endings are present at the 90 mm stage. In the adult too, simple nerve endings predominate while there are also a few organised but unencapsulated endings especially in the caudal part of the nasopharyangeal mucosa.

The submucous, intramuscular and perimuscular ganglia make their appearance by the 45 mm stage. In the 31 mm and 45 mm specimens, nerve cells which are the precursors of the ganglion cells are seen along the course of the branches of the maxillary and glossoparyngeal nerves. Some AChE positive (parasympathetic) neurones are present in the 90 mm foetus indicating an early functional activity of these neurones. In the adult, AChE neurons predominate while there are also some weakly positive and AChE negative neurones.

These observations show that secretory function of glands in the nasopharynx commences with the appearances of AChE positive parsympathetic neurones in the fourth month. These neurones provide nerve supply to the glands of the region. Some weakly positive and AChE negative

neurones may subserve sympathetic or sensory functions; it is possible that the sensory neurones may take part in mediating local reflex activity as occurring elsewhere in the gut.

Contrast radiography and cinefluorography

Contrast radiography and cinefluorographic studies[11] reveal that there is vigorous contraction of the pharynx in the region of the pharyngeal recess (fossa of Rosenmuller) which is situated behind the tubal elevation. Moreover, endoscopic sxamination by us has shown that there is contraction of the tubal region during swallowing. Indeed, Adams (1958)[12] has observed that there is a considerable degree of contraction or the lateral walls of the posterior nasopharynx when the patients are in light anaesthesia and on one occasion, when the patient was retching, the examining finger was tightly gripped between the auditory tubes. It is therefore not surprising that tubes contain large amount of elastic tissue to permit the stretching of the tubes during contraction of the posterior part of the nasopharynx. Thus it is clear that the posterior region is contractile while the bony another part of the nasopharynx in front of the tubal orifices is rigid owing to the bony composition of its walls.

Conclusions

The above observations indicate that there are two fairly distinct antomical components of the nasopharynx, the respiratory part in front and the pharyngeal portion behind. This view is supported by comparative anatomical, embryological, neurological and endoscopic studies as well as by contrast radiology. In accordance with this concept, the nasal cavity would terminate in front of the tubal orifices while the **pharynx proper** would commence from the region of the tubal orifices.

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Bacterial flora and wound infection in a surgical unit of the General Hospital, Jaffna.

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T. Vinayagamoorthy,+ B. Sc. (Hons.), M. Sc. (Cey.), Dip. Chem. (Uppsala).

Summary

High carriage rate of organisms was found among hospital staff. A high percentage of Klebsiella species and Proteus was isolated from doctors and patients. Operation theatres, in which casualty operations take place and the wards were heavily laden with organisms. Wound infection rate in clean operations was high. Of 6 wound infections, in clean operations, 3 appear to have occured in the ward, and 3 in the theatre, The use of metronidazole in appendicectomy (and other intestinal operations) resulted in low wound infection rate. K. oxytoca was more frequently isolated, from and around the appendix.

Introduction

Hospital infection results in increased morbidity and mortality, prolonged hospital stay and increased costs. Wound infection, one aspect of hospital infection, is a very distressing complication of surgery. Bacterial flora and their sensitivity to antibiotics vary among the population and between institutions[1]. Some idea of the bacterial flora and attempts at elucidating causes of wound infections, in our institution, was considered desirable. This study was undertaken in one of the Surgical Units of the General Hospital, Jaffna between 1st September 1980 and mid - January 1981.

Materials and Methods

A. Swabs were taken from :

1. Throat and nose of Ward staff
2. Throat and nose of Theatre staff
3. Throat and Nose of Patients, area of skin immeadiately surrounding field of operation, depth of wound (muscle layer or subcutaneous tissue), surface of wound after skin closure and where relevant from peritoneal cavity and/ or viscus.
4. Wound, 5 to 6 days after operation
5. Throat and nose of apparently normal persons in Jaffna town.
6. Septic lesions in patients in Ward.
7. Various articles, floor and wall of one Theatre and Ward

B. Settle Plates were exposed in Theatres and Ward.

Records were maintained of (A) Nature of operation, theatre in which undertaken and names of personnel involved. (B) State of wound, constitutional symptoms and use of drains.

Wounds were considered infected if there was collection of pus or a serous discharge with positive culture. Mere signs of inflammation were not considered, as these could be due to other reasons viz., surgical trauma, suture material.

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Sterile cotton wool swabs were used to collect samples. Soon after swabbing, they were dipped in sterile peptone broth. The swabs were kept in the refrigerator (for 4 to 20 Hrs.), before they were transferred to the laboratory (2 miles away). The swabs were incubated at 37°C, for 5h, before plating out on MacConkey agar and Blood agar media. These plates were then incubated at 37°C for 18 h. For samples collected from wounds, peritoneal cavity and viscera, a duplicate set of Blood agar plates were incubated at 37°C for 96h, under anaerobic conditions. All colonies appearing on these plates were identified by routine analysis [2, 3, 4].

Results

The carriage of **Staph. aureus. in the** nares, varied in the groups examined; 42.8% of ward staff, 30.6% of theatre personnel, 40% of doctors and 12.3% of patients (Tables I, II, III) Appreciable percentage, was found in throat as well, in ward staff (21.4%) and doctors (25%). The carriage of **Strep. pyogenes,** in throat varied from 14.3% to 25% (Tables I, II, III). An interesting feature is the high percentage of **Klebsiella** species isolated from the throat of doctors (15%) and throat and nose of patients (30.9% and 17.3%) respectively. **Proteus** species was also isolated in significant numbers, from nose of doctors (10%), and throat and nose of patients (16% and 14.8%) respectively.

Alkaligenes faecalis (14.8%), **Staph. aureus** (11.1%), **Klebsiella** Species (9.8%), **Proteus** Species (9.8%) and **paracolon** (8.6%) were the main organisms isolated from the skin of the area immediately surrounding operation field (Table III). Table IV shows the organisms isolated from the throat and nose of an apparently normal population.

Tables V and VI show that (a) Theatres B and C were contaminated by mainly **Staphylococcus** Species, **Sarcina,** Sporebearers and Fungi whereas theatre A, which is not used for casualty operations by **Al. faecalis,** only. (b) The ward was contaminated by most of the pathogens- **Staph. aureus, Al. faecalis, Klebsiella species, Streptococcus, Psedomonas,** and **Proteus**. Table VII shows organisms isolated from septic lesions (presumably contaminating the ward). From

Table I

ORGANISMS ISOLATED FROM WARD STAFF (excluding doctors)

Site	Organism	No. of persons	Percentage
Throat			
	Staph. aureus	3	21·4
	Strept. pyogenes	2	14·3
	K. aerogenes	2	14·3
Nose			
	Staph aureus	6	42·8

No. of persons swabbed. — 14
Nose and Throat : 2 persons. Free : 3 persons.

Table II

ORGANISMS ISOLATED FROM THEATRE STAFF.

Site	Organism	Nurse, Attendants, Labourers (36) No. of persons	Percentage	Doctors (20) No. of persons	Percentage
Throat					
	Staph. aureus	3	8·4	5	25
	Strept. pyogenes	9	25	3	15
	Strept. faecalis	0	—	1	5
	K. aerogenes	1	2·8	2	10
	K. oxytoca	0	—	1	5
	E. coli	1	2·8	0	—
Nose					
	Staph. aureus	11	30 6	8	40
	Strept. pyogenes	1	2·8	0	—
	K. ae ogenes	0	—	1	5
	Pr. mirabilis	0	—	1	5
	Pr. morganii	0	—	1	5
	Parcolon	0	—	1	5
Nose & Throat, 7 persons. Free-17.				Nose & Throat; 2 persons Free; 3.	

22 lesions, **Pseudomonas aeruginosa** was isolated in 11, **Proteus mirabilis** in 5, **Al. faecalis** in 2, **Staph. aureus** in 2, and **K. aerogenes** in 1.

The operations performed are listed in Table VIII. They were not consecutive but performed as and when transport medium was ready. Two patients who expired and one discharged early have been excluded, giving a total of 78 operations for analysis. Of the 78 operations, 47 were clean, and 31 "nonsterile", the latter being, any operation with overt sepsis or potentially septic, Viz, opening gastrointestinal tract. 15 patients had wound infection, giving an overall wound infection rate of 19.2%. 6 occured in clean cases and 9 in nonsterile cases, giving a rate of 12.8% and 29.0%, respectively. Organisms were isolated from postoperative wound swabs in 31 cases (inclusive of 15 cases of wound infection)- Table IX. **K. aerogenes** was isolated from 8 patients, **K. oxytoca** from 3, **E. coli** from 5, **Ps. aeruginosa** from 4, **Proteus** spp from 4, **Staph. aureus** from 3, **Al. faecalis** from 2, **Paracolon** from 2, **Staph. epidermides** from 2, **Bacteroides fragilis** from 1 and fungus from 1. The following were isolated from the cases of wound infection; **Ps. aeruginosa** from 4 patients, **K. aerogenes** from 2, **K. oxytoca** from 2, **Proteus.** spp from 4, **paracolon** from 1, **B. fragilis** from 1 and fungus from 1.

Table III

ORGANISMS ISOLATED FROM PATIENTS (81)

Site	Organism	No. of patients	Percentage
(a) Throat			
	K. aerogenes	18	22·3
	K. oxytoca	7	8·6
	Strept. pyogenes	13	16
	Staph. aureus	7	8·6
	Pr. rettgeri	8	9·9
	Pr. vulgaris	1	1·2
	Pr. morganii	4	4·9
	Paracolon	2	2·4
	E. coli	1	1·2
	Ps. aeruginosa	1	1·2
(b) Nose			
	Staph. aureus	10	12·3
	K. aerogenes	9	11·1
	K. oxytoca	5	6·2
	Pr. rettgeri	6	7·4
	Pr. morganii	4	4·9
	Pr. mirabilis	1	1·2
	Pr vulgaris	1	1·2
	Al. faecalis	4	4·9
	Strept. pyogenes	3	3·7
	Paracolon	5	6·2
	Ps. aeruginosa	3	3·7
	Fungus	3	3·7
(c) Skin of area surrounding operation site			
	Al. faecalis	12	14·8
	Staph. aureus	9	11·1
	K. aerogenes	6	7·4
	K. oxytoca	2	2·4
	Paracolon	7	8·6
	Pr. rettgeri	7	8·6
	Pr. morgnnii	1	1·2
	Ps. aeruginosa	2	2·4
	Sarcinia	2	2·4
	Strept. pyogenes	1	1·2
	Strept. viridans	1	1·2
	E. coli	1	1·2

Table IV

ORGANISMS ISOLATED FROM NOSE AND THROAT OF HEALTHY SUBJECTS.

	Strains	Nose	%	Throat	%
1.	**Staph. aureus**	19	35·2	Nil	—
2.	**Strept. pyogenes**	Nil	—	18	33·3
3.	**K. aerogenes**	4	7·4	6	11·1
4.	**K. oxytoca**	Nil	—	2	3·7
5.	**Prot. morganii**	3	5·5	3	5·5
6.	**Prot. rettgeri**	3	5·5	Nil	—
7.	**Escherichia**	3	5·5	Nil	—
8.	**Paracolon**	1	1·9	2	3·7
9.	**Al. faecalis**	1	1·9	2	3·7

No. of subjects tested. 54

Table V

ORGANISMS ISOLATED FROM THEATRE AND WARD

Theatre A - 2 occasions

(1) **Staph. aureus** from wall

(2) **Staph. epidermidis** from switch

Ward

Floor	—	**K. aerogenes**
Wall	—	**K. aerogenes, Staph. aureus, Al. faecalis**
Fan	—	**K aerogenes, Af. faecalis**
Tap	—	**Ps. aeruginosa**
Bed	—	**Ps. aeruginosa**
Trolley	—	Nil
Sucker	—	Nil
Cabinet	—	Nil

Table VI

ORGANISMS ISOLATED FROM SETTLE — PLATES EXPOSED IN THEATRES AND WARD

1. Theatre A — Overnight
   Alk. faecalis
2. Theatre B — Overnight — (2 plates at different sites)
   (a) **Staph. epidermidis, Staph. citreus, Sarcina**. Sporebearers, Fungi
   (b) **Staph aureus, Staph. epidermidis, Staph. citreus, Sarcina,** Sporebearers, Fungi
3. Theatre C — 5h during day
   (a) **Staph. aureus, Staph. epidermidis, Staph. citreus,** Sporbearers, **Sarcina,** Fungi
   (b) **Staph. epidermidis, Staph. citreus, Sarcina,** Sporbearers. Fungi
4. Ward — 5h during day
   Proteus, Pseudomonas, Staph. aureus, Streptococcus

Table VII

ORGANISMS ISOLATED FROM SEPTIC LESIONS IN PATIENTS IN WARD

Organism	No. of Patients
Ps. aeruginosa	11
Pr. mirabilis	5
Staph. aureus	2
Al. faecalis	2
K. aerogenes	1

No. of patients swabbed 22

Table VIII

OPERATIONS PERFORMED

Herniotomy - Noncomplicated	5
Herniorraphy - Noncomplicated	15
Herniorraphy - Obstructed	2
Herniorraphy - Strangulated	1
Herniorraphy & resection of S. I. - Strangulated	1
Herniorraphy - Incisional	1
Laparotomy	12
Appendicectomy	9
Excision - fibroadenoma	4
Prostatectomy	4
Hemithyroidectomy	2
Stripping - Varicose Veins	2
Vesicolithotomy	2
Repair of cleft lip	2
Excision Neurofibroma and skin grafting	1
Excision Neurofibroma	1
Excision biopsy breast lump	1
Z plasty - contracture	1
Excision biopsy Neck lump	1
Insertion Kuntscher Nail	1
Amputation of toe (melanoma)	1
Modified radical mastectomy	1
Orchiopexy	1

Table VIII (Contd.)

Circumcision	1
Testicular Biopsy	1
Ovarian Cystectomy	1
Partial Gastrectomy	1
Cholecystectomy	1
Ramstedt operation	1
Drainage of Subphrenic abscess	1
Ligature spermatic vein	1
Excision liposarcoma, bladder	1
Hydrocele of cord excision	1
	81

Table IX

ORGANISMS ISOLATED FROM POST OPERATIVE WOUNDS (31)

Organism	Frequency
K. aerogenes	8
K. oxytoca	3
E. coli	5
Ps. aeruginosa	4
Proteus. spp	4
Staph. aureus	3
Al. faecalis	2
Paracolon	2
Staph. epidermidis	2
B. fragilis	1
Fungus	1

No. of patients swabbed 81

(An attempt was made, to identify the possible source of wound contamination, by comparing the organisms isolated from post operative wound swabs, with those isolated from patient's throat and nostrils, skin surrounding operation site. depth of wound, surface of wound, peritoneal cavity, viscus, and staff attending on the particular operation. Table X show organisms, in only cases where some correlation was present. The common sources were, Anaesthetist, viscus, Attendant,

Surgeon and Skin surrounding operation field. **Ps aeruginosa** appears to have colonised from the ward.)

Discussion

Bacterial Flora

The carriage rate of organisms varies· Ayliffe[5], reported that 20% of normal people are nasal carriers of Styaphylococcus. Cruickshank et al[3] mentioned that Staphylococcus occurs harmlessly as a commensal parasite in the anterior nares and moist areas of skin in 20% to 30% of healthy people. Mendis et al[6], reported a nasal carrier rate, among the staff of 26.4% and among patients of 23.5%. In our study, higher percentages were observed in nares, in hospital staff (30·6% to 42·8%) and lesser in patients (12·3%), with an appreciable percentage on throat of hospital staff (Tables I, II, III). The carriage rate in an apparently normal population was (35·2%) — Table IV.

Carriage rates of **Streptococci** of 4% to 25% to as much as 40% to 60% in school children with cumulative percentages as high as 75% to 90% have been reported (8 quoted in 7). Mendis et al[6], reported a carriage rate of **Streptococci** of 29·2% among staff and 1·5% among patients. In our study the carriage rate among staff varied from 14·3% to 25% and among patients was 16% whereas the carriage rate in an apparently normal population was rather high (33·3%) Table IV.

Klebsiella was isolatad in 15% of doctors, from the throat and in 30% and 17%, from the throat and nose respectively of patients. **Proteus** was isolated from the nose in 15% of doctors and from the throat and nose, in 16% and 14.8% of patients respectively. Though an incidence of 5% is reported for **K. pneumoniae** in the upper respiratory tract[9], the rate for all the species of **Klebsiella** and for **Proteus** colonising the respiratory tract, was not discerned from the literature. The carriage rate of **Klebsiella** (and **Proteus**) appear to be high and further colonisation appears to take place in hospital. In an apparently normal population too, **Klebsiella** was isolated in 14.8% from throat and 7.4% from nose, the corresponding figures for **Proteus** being 11% and 5.5% (Table IV). **Klebsiella** species constitute 62% of organism isolated from water sources in Jaffna district[10]. This factor together with hygienic habits may be responsible for high carriage rate. It is interesting to note that 25.5% of tooth infections in Jaffna district is caused by **Klebsiella**[11].

Wound Infection

Rates of wound infection vary, depending on, definition, period of assessment and measures adopted in its control. In 449 patients who underwent, abdominal operations, the wound infection rates were, 3.8% in clean operations; and 11.4%, 15.3%, and 42.3%, in potentially, lightly and highly contaminated operations[12], respectively. In this series, peroperative dose of Cephaloridine was given and late wound infection was included. In one series with all types of, clean operations, a rate of 1.9% was reported[13]. Nobecutane was sprayed both before and after operation. An overall wound infection rate of 16.4% was reported, in another series[14]. A rate of 40.9% was reported from General Hospital, Colombo 6. However in this series, signs of inflammation was also included in the criteria. In our study, the overall rate of 19.2%, compares favourably, but the rate for clean operations (12.8%) is high. Of the six wound infections

among clean operations, 3 appear to have developed infection in the ward (late wound infection). In one of these the possibility of origin in the theatre cannot be discounted as one of the organisms isolated from wound was harboured by patient as well as surgeon. In one, a case of malignancy, organisms isolated were the same as that harboured by anaesthetist indicating origin in the theatre. It must be mentioned that phage typing was not done nor was repeated sampling of personnel. Two others, one of whom was a malignant case, had early wound infection, indicating origin in the theatre. Prophylactic antibiotics were not givin, for clean operations, except in two, inadvertently. However 10 cases received, post operative antibiotics, as and when pulmonary or gastrointestinal infections occured. Treatment of carriers in the theatre is considered unnecessary unless the strain causes autogenous infection or infection in patients[5]. The surgeon and one anaesthetist were probably responsible for infection in two patients and treatment must be considered.

It is generally accepted that, in nonsterile cases wound infection is mostly endogenous. However correlation between organisms, cultured from septic focus or viscus and those cultured from infected wound is not always present. Pollock et al[15] stated that in 34 (77·3%) of 44 patients. cultures from pus had at least one species in common with those from the incised organ or the subcutaneous tissue, whereas in the other 10 patients, pus cultures yielded organisms different from those found during operation Richards et al[16] also commented on the lack of correlation between the organisms from the intra-operation swab cultures and those isolated from postoperative wound infection. In our study, correlation was present in only 3 out of 9 wound infections in nonsterile cases and 2 out of 6 wound infections in clean cases (Table X)

Many methods have been tried, during operation, to reduce wound infection, Viz the use of plastic adherent drapes, ring drape wound protectors, peritoneal toilet with antiseptic solutions, wound irrigation with saline, wound spraying with antiseptic, topical instillation of antibiotic, wound drainage, wound sealing with plastic-antiseptic spray, peroperative single dose antibiotic, etc, The general consensus of opinion favours the use of peroperative single dose antibiotic as the most effective means of reducing wound infection rate[12]. The choice of antibiotic is important. In clean prosthetic surgery, cloxacillin; in upper gastrointestinal and biliary surgery, a cephalosporin, and in colonic surgery combination of an agent effective against anaerobes (metronidazole is less toxic than lincomycin or clindamycin) with an aminoglycoside or a cephalosporin, have been recommended[12]. In our study, of the nonsterile cases only some received peroperative antibiotic, some postoperative antibiotic, but almost all were given metronidazole. The wound infection rate among nonsterile cases, appears satisfactory comparatively.

Only one out of 12 appendicectomies (8·3%) ended in wound sepsis and that too in the case of a gangrenous appendix. Wound infection rate of 30%, has been reported in perforated appendicitis[17]. An overall wound infection rate of 19% is also reported[18]. Our numbers are small but the probale cause of low rate is the use of metronidazole on every case. In guinea pigs, it was demonstrated, that pathogenic synergy existed between **E. coli**

and **B. fragilis** and wound infection occured at certain concentrations of the organisms[19]. Metronidazole, active against anaerobic **Bacteroides** and relatively non - toxic, should be used, in abdominal sepsis, particularly colonic and appendicular.

One interesting feature, in our study is the high percentage (36·4%) of **K. oxytoca**, isolated from the serosal aspect of or around the appendix (the other organisms being **Staph. aureus**, **E coli** and **Proteus** Spp.). **Bacteroides** (78%) and other Gram negative bacilli, **Klebsiella** (29%), **Ecoli** (27%) and Gram positive cocci less frequently were identified in one series[18]. We have been unsuccessful in the isolation of **Bacteroides** (the only instance being from extra-abdominal source). Immeadiate or early plating was not possible owing to distance of the laboratory from hospital and the non - availability of personnel to transport the media early.

The relationship of wound infection to the particular theatre cannot be determined owing to insufficient numbers. 16 of the clean operations took place in Theatre A. 3 in B and 26 in C (not documented in 2). 2 of the operations in A, 1 in B and 2 in C resulted in wound infection. Theatres B and C have been heavily laden with organisms and it is probaly wiser to set apart one of them only, for casualty operations. Fumigation should be more frequent.

The growth of various organisms, from septic lesions and about the same variety frow the settle plates exposed in the ward, clearly shows the need for isolation of patients with septic lesions in a separate "septic ward". Wound infection and air hygiene studies suggest that highly compartmentalised (race track) unit is more salubrious than the open (Nightingale) ward[20], but this is a diff. cult proposition in our country with a shortage of nursing staff. Not the least important in prevention of cross infection is the washing of hands by the staff after handling each patient. The visible dust is much less efficient as a vehicle of infection than the Nurses' or doctors' hands that go directly and quickly from patient to patient[21].

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Table X Surgeon

CORRELATION OF SOURCE AND WOUND INFECTION

PT	PN	SS	D	S	P/V	WS	Ass. Dr	Ass. Nur.	Anaesthestist	Att
K. ae	Paracol.	K. ae	Paracol.	—	—	E. coli & Paracol.	St. au St. pyo	St. au	St. au St. pyo K. ae, P. m	St St
—	St. pyo	Ps. ae	St. au	—	—	K.ae, P.m	—	—	St. au St pyo K. ae, P. m	St
—	—	St. pyo	—	—	—	K. ae	—	—	K. ae, P. m	St E.
Pr. ret	Pr. ret.	K. ae	K. ae	K. ae	—	K. ae	—	—	St. au	St St
—	—	E. coli	K. ae	K. ae	K. ae	E. coll	St. pyo	—	St. au St. pyo K. ae, P. m	St E.
K. ae	—	—	—	—	—	Paracol.	—	—	St. au St. pyo K. ae P. m	St E.
—	St. pyo	Sarcinia	—	—	—	K. ae	St. pyo	St. pyo	St. au K. ae	St E.
K. ae	Pr. morg	—	—	—	—	St. au	—	St. pyo	—	St St
K. ae	K. oxy	—	—	—	—	K. ae	—	St. pyo	St. au St. pyo	
K. oxy.	K. ae	K. oxy	—	—	Pr. vul	Pr. vul	St. pyo	St. pyo	St. au St. pyo	
—	St. au	Sarcinia	K. ae Ps. ae	K. ae Ps. ae	K. ae	K. ae K. oxy	K. oxy	—	St. au St. pyo	St
K. ae	K. ae	Al. fae	—	—	—	St au	—	—	St. au St. pyo	St
—	—	Pr. ret	—	St. epi	—	St. au	St. pyo	—	K. ae, P. m	St
K. ae	K. ae	St. au	K. oxy	—	K. oxy	K. oxy	—	—	Pr. morg	St St
Pr. ret	Al. fae	St. au	E. coli	E. coli	E. coli	E. coli	St. pyo	St. au	St. au St. pyo	

KEY

PT — Patient's Throat
PN — Patient's Nostrils
SS — Surrounding Skin
D — Depth of Wound
S — Surface of Wound
P/V — Peritoneal Cavity or Viscus
WS — Wound Swab

Table X Surgeon paracolon

CORRELATION OF SOURCE AND WOUND INFECTION

D	S	P/V	WS	Ass. Dr	Ass. Nur.	Anaesthestist	Attendants	Wound Inifect-ion	Operation
Paracol.	—	—	E. coli & Paracol.	St. au St. pyo	St. au	St. au St. pyo K. ae, P. m	St. au St. pyo	+	Release of contracture & skin graft
St. au	—	—	K.ae, P.m	—	—	St. au St pyo K. ae, P. m	St. pyo	+	Excision biopsy breastlump
—	—	—	K. ae	—	—	K. ae, P. m	St. au E. coli	—	Hernia
K. ae	K. ae	—	K. ae	—	—	St. au	St. au St. pyo	—	Testicular biopsy
K. ae	K. ae	K. ae	E. coll	St. pyo	—	St. au St. pyo K. ae, P. m	St. au E. coli	—	Laparotomy
—	—	—	Paracol.	—	—	St. au St. pyo K. ae P. m	St. au E. coli	—	Appendicectomy
—	—	—	K. ae	St. pyo	St. pyo	St. au K. ae	St. au E. coli	—	Mastectomy
—	—	—	St. au	—	St. pyo	—	St. au St. pyo	—	Hernia
—	—	—	K. ae	—	St. pyo	St. au St. pyo	—	—	Hernia
—	—	Pr. vul	Pr. vul	St. pyo	St. pyo	St. au St. pyo	—	+	Vesicolithotomy
K. ae Ps. ae	K. ae Ps. ae	K. ae	K. ae K. oxy	K. oxy	—	St. au St. pyo	St. au	+	Prostatectomy
—	—	—	St au	—	—	St. au St. pyo	St. pyo	—	Obt. Hernia
—	St. epi	—	St. au	St. pyo	—	K. ae, P. m	St. au	—	Partial Gastrectomy
K. oxy	—	K. oxy	K. oxy	—	—	Pr. morg	St. au St. pyo	—	Laparotomy
E. coli	E. coli	E. coli	E. coli	St. pyo	St. au	St. au St. pyo	—	+	Appendicectomy

S — Surface of Wound
P/V — Peritoneal Cavity or Viscus
WS — Wound Swab

Ass. Dr — Assisting Doctor
Ass. Nur. — Assisting Nurse
P. m — Proteus .mirabilis

Road Traffic Accidents in the North

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Summary

An accident rate of 0·061 per 100 population for Jaffna and Vavuniya divisions and 3·3 per 100 vehicles for Jaffna division was observed. Most victims (63·6%), fell within the 10-40 age group. The motor cycle was the most common "offending" vehicle, with the private car, the next common, while the pedal cycle was involved in about half the accidents and pedestrians to a fair extent. Most number of accidents occurred in the evening, during the rush hours of 4 p. m. to 6 p. m. Speeding or failure to slow, wrong overtaking, cyclist "cutting" across and pedestrian running across were common causative factors. Soft tissue injuries were common with fractures occurring in about 20%. Death rate was 0·0058 per 100 population. Cranio-cerebral and Chest injuries were mostly responsible for the deaths.

Introduction

That road traffic accidents take a heavy toll of life is only too well known. In fact it is the commonest cause of death in the affluent countries under 50 age group and the second commonest cause in Sri Lanka. Disability caused to those surviving must be considerable and total economic loss severe. In Sri Lanka, the free import policy of the present government, led to an unprecedented inflow of motor vehicles, the road mileage remaining the same. An increasing number of vehicles, particularly, motorcycles were noted in Jaffna too. Some idea of the damage caused and causative factors was considered worthwhile. The Study commenced on 1st November 1980 and terminated in May 1981. All admissions to one Surgical Unit (122) and deaths (12) during the quoted period, coming up for post-mortem by J. M. O. Jaffna, were analysed. (Information regarding some factors was not documented in every case and totals do not aggregate to 122 for every factor)

Age and Sex incidence

Most victims (63·6%), fell within the 10-40 age group, with a decline at each extreme. — (Table I). 116 were males and only 6 were females.

Vehicles involved

Of what was considered the "offending" vehicle, the motor cycle (or scooter), 25%, the private car, 18·7% and the pedal

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Table I
Age Incidence

Age in Yars	Number of Victims
0 — 5	2
6 — 9	7
10 — 19	28
20 — 29	28
30 — 39	21
40 — 49	13
50 — 59	9
60 — 69	9
> 0	4

cycle, 17% were the most frequently involved, with the minibus or van to some extent, (Table II). It is remarkable that the taxi, (excluding private hiring car) was almost a "non-offender". Of the second vehicles involved, pedal cycles rank the highest. Taking into account, the number involved, as the "offending" vehicle also, the pedal cycle is seen to be the most commonly involved vehicle in road accidents in Jaffna District. Pedestrians were involved in 36 accidents (28%), and animals in 3 (2·4%).

Table II
Type of Vehicles Involved

Type	Number of "offending" vehicle	Number of second Vehicle
Motor Cycle, Scooter	30	7
Private Car	23	2
Pedal cycle	21	35
Mini Bus, Van	13	6
Tractor	9	0
Lorry	8	6
Bus	6	8
Cart	4	1
Jeep	4	0
Taxi	1	1
Other Vehicles	— 34	
Pedestrians	— 36	
Animals	— 3	

Time of accident

Seven accidents per hour took place between 6 a. m. and 12 Noon and also between 12 Noon and 4 p. m. whereas eleven per hour took place between 4 p. m. and 6 p. m. and 2 per hour between 6 p. m. and 6 a. m. — (Table III.)

Table III
Time of Occurence

Time	Number
6 a. m. to 12 Noon	41
12 Noon to 4 p. m.	29
4 p. m. to 6 p. m	22
6 p. m. to 6 a. m.	24
	116

Road Surface

This was wet only in 4 instances.

Causation

Excessive speeding or failure to slow was responsible in 26 (21·3%) accidents cyclist "cutting" across or the pedestrian running across in 24 (19·5%), and wrong overtaking or driving on wrong side in 23 (18·7%). Alcohol consumption was responsible in 8% — (Table IV.)

Pattern of Injuries

Lacerated wounds, Contusions and Abrasions, the last very often multiple were most frequently seen. — Table V. Fractures (27 Simple and 9 compound) were the next common. Pneumothorax in 2 patients, haemothorax in 1, rupture of terminal ileum in 1 and injury to cervical

Table IV

Causes of Accidents

Cause	Number
Failure to slow, Excess Speed	26
Wrong overtaking, driving wrong side	23
Pedestrian running across	15
Cyclist "cutting" across	9
Consumption of alcohol	10
Mechanical failure	7
Failure to signal	5
Skid	6
Animal running across	2
Engaged in conversation	2
Not keeping left at junction	1
No lights	1
Other	15
	122

Table V

Pattern of Injuries

Injuries	Number of Patients
No External Injuries	9
Abrasions	44
Contusions	25
Lacerated Wounds	47
Fractures - Simple	27
Compound Fractures	9
Dislocations	2
Pneumothorax	2
Haemothorax	1
Rupture of Ileum	1
Cervical Spine? dislocation	1

spine with quadriparesis were the other lesions noted. The site of fractures is shown in Table VI. The limbs were mostly involved with few other sites sparingly.

Table VI

Site of Fractures

Site	Number-Simple	Number-Compound
Skull	0	3
Zygoma	1	0
Mandible	2	0
Spine	2	0
Clavicle	3	1
Humerus	1	0
Olecranon	1	0
Ulna	2	0
Colles	5	1
Phalanges	2	1
Femur	4	1
Tibia and fibula	2	2
Ribs	3	0

Deaths

The injuries in those who died, have been analysed seperately and is shown in Table VII. Multiple injuries was the rule with most of them suffering cranio- cerebral damage. Injuries to thorax was next common with Liver and Spleen involvment in some.

Discussion

Magnitude of the problem

Silva. J. F[1], reported that in Malaysia, in 1975, the population was 10,438,137, with non-fatal injuries suffered by 17,106 and deaths amounting to 2324 (0·022 per 100 population), while in the United States, for the same year, the population was 213,540,000, non - fatal injuries, 1,800,000 and deaths 4,600 (0·021 per 100 population). Fernando, W. D. L.[2]; reported that in Sri Lanka, in 1965, the population was 11,232,000, the number of accidents, 13,317, (0·118 per 100 popul-

Table VII

Type of Accident and Cause of death

Motor Cycle / Pedestrian	Subarachnoid Hge, Haemothorax, Fracture Ribs
Motor Cycle / Pedestrian	Fracture Skull, Extradural Hge, Brain Stem Lesion
Motor Cycle / Cycle	Subendocardial Hge, Haemo thorax, Pneumo thorax, Rupture Spleen, Fracture ribs, tibia
Fall, Moving Van	Subdural Hge, Fracture Skull including Base
Mini Bus / Pedestrian	Bilat Haemothorax, Laceration lungs, liver
Motor Cycle / Cart	Rupture Ventricle, Haemopericardium, Laceration lung. liver
Private Car / Pedestrian	Extensive fracture Skull including Base, Contusion lung
Scooter / Pedestrian	Extensive fracture Skull, Extradural Hge
Car / Pedestrian	Subdural Hge, Brain Stem Lesion, Fracture Tibia, Fibula, Femur
Car / overturned	Extensive fracture Skull including Base
?	Extensive fracture Skull, Subarachnoid Hge, Brain Stem Lesion, Laceration liver, fracture fibula
Fall, Tractor	Contusion brain, Brain Stem Lesion. Contusion thalamus

Table VIII

Accident Statistics

	Island	Jaffna Division		Vavuniya Division	
	1980	1980	1981	1980	1981
Number of Road Accidents	23,711	546	520	196	204
Number of Injured	14,672	237	323	112	96
Number of fatal accidents	1,038	44	43	24	20
Number of deaths	1,106	44	43	24	21

ation) number of persons seriously injured, 8,076 and deaths 469 (0·0042 per 100 population). The number of vehicles then was 148,660 with an accident rate of 9 per 100 vehicles.

The accident statistics for the whole Island[3] for 1980, and for the Jaffna and Vavuniya divisions[4], is given in table VIII. The accident rate for the Island for 1980, was 0·1690 per 100 population and the death rate 0·0078 per 100 population. The population figures for Jaffna and Vavuniya Divisions, are given in Table IX[4]. The type and number of vehicles registered in Jaffna division, are indicated in Table X[4]. 724 accidents with an accident rate of 0·061 per 100 population for Jaffna and Vavuniya divisions and 3·3 per 100

vehicles for Jaffna division is observed. The death rate approximates to 0·0058 per 100 population for both divisions.

Table IX.

Population figures — 1981

District	Population
Jaffna	831,112
Mannar	106.940
Vavuniya	95,904
Mullaitivu	77,512
Total	1,111,468

The number of victims, as given in Table VIII[3], for Jaffna and Vavuniya divisions, appears to be low, perhaps due to non-reporting. 122 cases were admitted to one unit during a period of seven months. Assuming an equal frequency of admissions, to the other surgical units, the number of injured approximates to 366 for seven months or 624 for an year.

Table X

Type and Number of Vehicles - Jaffna Division

Type	Number
Cars — Private	5248
— Hiring	90
Motor Cycles	5252
Lorries	2493
Coaches (Minibuses)	125
Tractors & other types	2799
	16,007

This number does not include those (with minor injuries), who received treatment at the O. P. D. and at other hospitals; so that the actual number should be even more.

In Western Malaysia, the incidence showed a steady increase between 5 and 10 years to the age of 21 years[1]. In our study, most of the victims, were between 10 to 40 years, with a higher and equal distribution between 10 to 19 yrs and 20 to 29 yrs. Silva F.[1], found that the private car was the most commonly involved vehicle with motor cycles the next common, while the common victims were the motor cyclists and then the pedestrians. Drivers of motor vehicles were relatively immune, due perhaps to use of seat belts. In our study the motor cycle was the most common "offending" vehicle, while next common were the private car and the pedal cycle. However, taking into account, the second vehicle, the pedal cycle was the most commonly involved vehicle. Pedestrians too were involved in a large number of accidents. Education directed towards the public, in particular the pedal cyclist seems essential. This could start in schools and be organised by the Police jointly with school authorities, medical profession and voluntary organisations. Speed, wrong overtaking, cyclist "cutting" across, pedestrian running across and alcohol were found to be common causes. These could be stressed in such an educational programme. The people should be made conscious of the need to avoid harry during rush hours. The damage caused is obvious — Tables V and VII. The education of the public in First Aid measures may seem formidable but attention to some measures like keeping airway clear, arrest of bleeding and proper transport could be focussed in schools. Cranio cerebral and chest injuries, have been found to be the commonest causes of death. This was noted by Fernando[2] too. The use of helmet and seat belt though protecting the driver to some extent, does not protect the most vulnerable

pedestrian. Perhaps the most effective measure, is the inculcation in the minds of the road user a "Respect for Life".

Acknowledgement

We thank the Medical Superintendent, General Hospital, Jaffna for permission to publish, Mr. A. Navaratnam, D. I. G., Northern Range Mr. C. Sivapatham, A.S.P., Colombo, and Mr. M. Kulanayagam, Additional G. A., Jaffna, for valuable information supplied.

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Correction: In, Tuberculosis of the Skin over the Sternum, September 1981, Vyravanathan and Nadarajah 27, 23 - 6, legend of Figure 3 is for Figure 4 and vice versa.

Editor.

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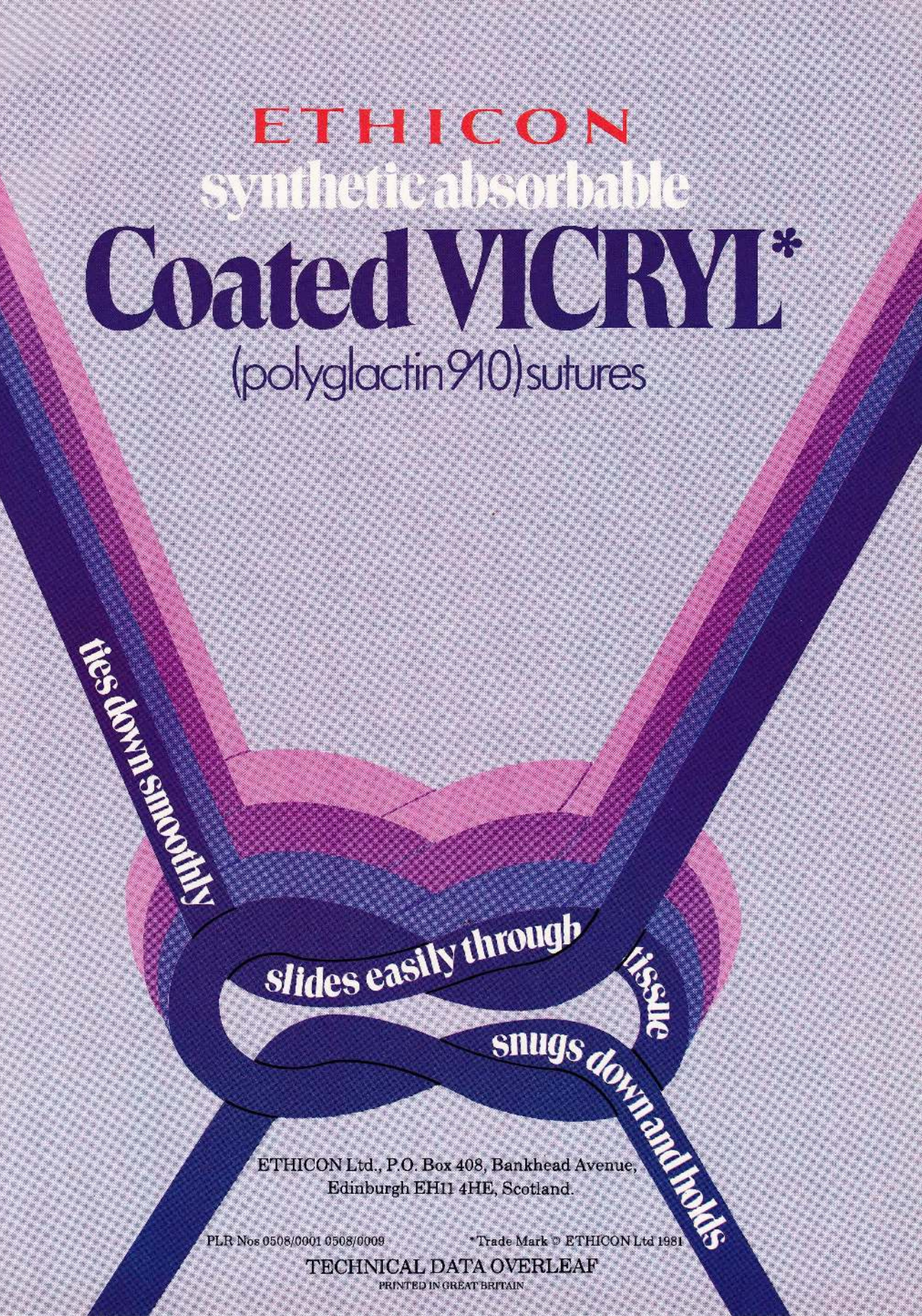

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Acute Encephalitis in Jaffna — an analysis of 21 cases.

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Summary :

21 children admitted with acute encephalitis to the University Paediatric unit over a period of 16 months are analysed with respect to clinical presentation, investigation, management & sequelae. The principles of management were medical decompression of the brain and general nursing care. 9 children out of the 21 died in the acute phase of illness. 4 children out of the surviving 12, had neurological deficits and 3 on follow up had regained muscle power. Long term follow up of the cases is being done and the results will be reported later.

Introduction :

Encephalitis is a disease of world-wide distribution. There may be sporadic or endemic cases; the latter situation occurs in some countries. In Jaffna too the disease is prevalent to some extent. Encephalitis in Sri Lanka and particularly Jaffna, does not appear to have been documented sufficiently. We report here 21 cases of encephalitis admitted to the University Paediatric unit, General Hospital, Jaffna, over a period of 16 months - October 1980 to January 1982. Diagnosis in all the cases was clinical and in a few of them supported by virological and electroencephalographic studies.

Clinical presentation :

The commonest symptoms were fever, convulsions, vomiting and altered sensorium. Headache and neurological deficits were noted in a few cases. The frequency of the presenting features is given in Table I. The distribution by age is given in Table II. The distribution according to the months of admission is given in Table III.

Table I

Frequency of the presenting features.

Symptoms	No. of cases
Fever	17
Convulsion	16
Vomiting	11
Altered Sensorium	11

Table II

Distribution by age and mortality.

Age in years	No. of cases	No. of deaths
0 — 2 yrs.	10	5
2 — 4 yrs.	1	1
4 — 6 yrs.	4	1
6 — 8 yrs.	2	1
8 — 10 yrs.	2	1
10 — 12 yrs.	2	0

Department of Paediatrics, University of Jaffna.

1. *Professor* 2. *Lecturer* 3. *Registrar*

Table III

Distribution according to the Months of admission.

Year	Month	No. of cases
1980	— October	0
	— November	1
	— December	1
1981	— January	1
	— February	1
	— March	0
	— April	0
	— May	0
	— June	4
	— July	4
	— August	0
	— September	1
	— October	2
	— November	3
	December	2
1982	— January	1

On examination, fever and altered state of sensorium were the marked features; the latter ranging from drowsiness to coma. The younger children were quite often, very ill. Nuchal regidity was noted in one child while neurological deficits were noted in 5 children.

Investigation :

White cell count and cerebrospinal fluid were studied in all cases in our laboratory. Blood and cerebrospinal fluid were sent to the Medical Research Institute, Colombo in 3 cases, for the rising arbor virus antibody titres. Electroencephalograms were done in 3 cases.

Management :

All the children were managed symptomatically. The general nursing care and attention to nutrition and fluid/ electrolyte balance was the mainstay of treatment. The principle of medical decompression of the brain was used in all cases, dexamethazone being given to all. In addition, 5 of them had intravenous 50% Dextrose and 2 of them mannitol. Penicillin and chloramphenicol were the antibiotics used in all cases. In 3 children admitted from known malarial areas a therapeutic trial of parenteral chloroquin was given.

Mortality :

The mortality with particular reference to age distribution is indicated in Table II.

Discussion :

21 cases of acute encephalitis were admitted to one unit within a period of 16 months. The total number of patients admitted to the unit during the same period was little over 2000. It is not unreasonable to assume, some cases of acute encephalitis to have been admitted and treated in other paediatric units in Jaffna. The incidence of encephalitis in Jaffna, would therefore appear to be high

Acute viral encephalitis is one of the illnesses where the diagnosis is a problem without specific tests and often creates a difficult situation for management. The diagnosis is based on clinical grounds in all cases, the presentation with fever, fits and altered sensorium being a common triad in all except one case. Such a triad of presentation may well be due to other causes. In a series of 120 patients suspected of having viral encephalitis in Glasgow over a 4 year period nearly half proved to have disease other than encephalitis[1]. In the differential diagnosis of our cases the following were taken into special consideration - meni -

ngitis including tuberculosis, brain abscess, cerebral malaria, tumours, drug overdose and pesticide exposure.

The confirmatory diagnosis of acute encephalitis is by brain biopsy and identification of the virus[2]. Brain biopsy is an investigation which is not easily undertaken. Further nonspecific investigations like air encephalography and cerebralangiography are difficult to perform in our hospital. In 3 cases we were able to perform electroencephalography, an investigation that is useful in distinguishing Herps simplex encephalitis and in the elimination of tumour or abscess[3]. The white blood count reports have been unequivocal in most instances. The cerebrospinal fluid was normal for protein, sugar and chloride in all cases. The presence of occasional lymphocytes (up to 10 per c. mm) was seen in 2 cases. In the studies for the rising antibody titre for arbor viruses in the 3 cases, 2 showed no evidence for viral aetiology while the third showed a rising titre for arbor virus antibodies (Japanese encephalitis). In the management, the principle of medical decompression of the brain was adopted in all cases. All the patients were given dexamethazone[4]. Illis & Merry claimed good results with steroids[5], with a drop in mortality from 70% to 44% in all recorded cases of Herps simplex encephalitis. Mannitol was given to 3 patients, 2 of whom survived. 5 patients had 50% Dextrose intravenously and 3 of them survived. No specific antiviral agent was used in any of the patients.

Of the 21 children admitted, 9 died giving a mortality of 42·8%. 5 were under 2 years of age and none died in the over 10 year group. Of the 9 deaths 7 were females and 2 males. Neurological deficits were noted in 5 patients of whom one died. The neurological lesion was spastic paralysis of the limbs, of varying degrees, in all the five cases. On subsequent follow up of the remaining 4, muscle power had returned to normal in 3 cases. One child in whom the rising titre of Japanese encephalitis virus antibody was demonstrated had persisting spastic paralysis of the limbs and dysphasia too.

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Management of common problems in infancy*

Dr. A. Sivapathasundaram, M. R. C. P. (U. K.) D. C. H. (Lond. & Cey.)+

It is not uncommon to find some junior medical practitioners being nervous in handling common medical problems in infancy. Erroneous diagnosis and management may lead to death in seriously ill infants or to prolonging the illness. On the other hand overcautiousness and exaggerated diagnosis may lead to unnecessary hospital admissions and unnecessary medication very often in the form of antibiotic injections thus leading to a waste of drugs and causing unnecessary pain to infants and adding on to the work of nursing staff. In this article I have endeavoured to outline the diagnosis and management of common problems in infants.

1. Noisy breathing and difficulty to breathe.
2. Vomiting.
3. "Abnormal" bowel habits.
4. Feeding problems.
5. Skin infection.
6. Convulsions.
7. Dysuria.
8. Jaundice.
9. Breath-holding attacks.
10. Failure to thrive in infancy.

1. Noisy breathing and difficulty to breathe.

This is a common presenting complaint in a large number of infants who are brought to hospitals and to general practitioners. Some of these infants may be having serious illnesses like bronchopneumonia or bronchiolitis. On the other hand the others may be having relatively benign conditions like nasal obstruction or congenital laryngeal stridor. In obtaining the history from the parents it is very important to find out the duration of the symptoms and whether the symptoms are accompanied by fever and cough.

(a) Nasal obstruction
&
(b) Congenital laryngeal stridor.

In these two conditions very often the symptoms have been present from just a few days after birth and very often the infant would have been treated for "phlegm". However the condition would not have disturbed the child's well being. On examination of the chest, air entry is good and equal on both sides of the chest and there are no adventitious sounds. Sometimes even in these conditions conducted sounds may be heard over the chest and may be thought of as rhonchi or crepitations by the inexperienced. The presence of nasal obstruction may be confirmed by using a wisp of cotton wool. Each nostril should be tested separately and in turn, while the infant's mouth is kept closed. If the wisp of cotton wool does not move with the baby's respiration then the corresponding nostril is blocked. The next step is to pass a thin but firm feeding tube through the nostril to decide whether the obstruc-

* *Based on a lecture given to the Jaffna Medical Association.*

+ *Consultant Paediatrician, Base Hospital, Point Pedro.*

tion is permanent as in choanal atresia or is transient due to mucosal oedema. If the wisp of cotton wool does not move but the feeding tube can be passed easily through the nostrils then the nasal obstruction is due to mucosal oedema and very often responds to the use of 1% ephedrine in normal saline nasal drops. This is very helpful especially if instilled a few minutes before the feeds, as the infant is then able to feed without any difficulty. Bilateral choanal atresia will have to be managed with appropriate surgery.

Congenital laryngeal stridor improves on its own when the child grows to about one to two years. However respiratory infections in infants with congenital laryngeal stridor have to be treated with respect, as these can aggravate the stridor and may cause respiratory difficulty.

(c) Bronchopneumonia

(d) Bronchiolitis

&

(e) Acute bronchitis.

In these conditions, the symptoms are very often of short duration, the difficulty to breathe is often accompanied by fever and cough, the air entry may be diminished and very often there are adventitious sounds. It is important to differentiate between stridor and wheezing. Respiratory infections are often viral and two investigations that are helpful in deciding whether to administer antibiotics are:

1. White blood cell count and differential count.
2. X'ray of chest.

In bronchopneumonia the infant is ill with fever, cough and dyspnoea and has bilateral crepitations and the white blood cell count is increased with polymorphonuclear lucocytosis and there are patchy opacities in both lung fields in the chest X'ray. In this condition there is definite indication for the use of broad spectrum antibiotics.

In bronchiolitis the infant is very ill, often cyanosed and there is leucopenia with relative lymphocytosis and the X'ray of chest shows increased lung markings and over inflation of chest with increased translucency of the lungs. Usually the air entry is diminished, and there are fine crepitations and rhonchi. In more developed countries, the causative organism namely Respiratory Syncytial Virus can be identified easily in the nasopharyngeal secretions by immuno-fluorescent studies. Antibiotics are ineffective in bronchiolitis. Management of bronchiolitis consists essentially of:

I. i. administration of oxygen

ii. maintaining the nutrition of the infant by naso-gastric or gavage feeds or if necessary by intravenous feeding.

iii. watching for cardiac failure and treating it if and when it arises.

iv. increasing the humidity of the infant's atmosphere seems to relieve some of the respiratory distress. Since we have no means of administering cold humidity we use steam inhalation for these infants.

If the signs are not prominent enough for one to differentiate between bronchopneumonia and bronchiolitis it is advisable to err on the side of using antibiotics.

Aucte bronchitis presents with some of the symptoms and signs of bronchial asthma and bronchiolitis. In infants there is hardly any smooth muscle in the bronchial walls The wheezing that may occur in acute bronchitis in infants is really due to mucosal oedema rather than bronchial spasm. Hence bronchodilators are inffective in infants. Management of acute bronchitis is in the same lines as bronchiolitis, but acute bronchitis is a relatively milder illness. Antibiotics may be useful if there is evidence of bacterial infection as shown in the white blood cell count and the differential count.

(f) Diphtheria

(g) Viral croup

or

(h) Epiglotitis.

Infants presenting with inspiratory stridor of short duration may be having Diphtheria, Viral croup or epiglotitis.

An infant who had not been given trlple or double vaccine and presenting with stridor and a membrane in the throat has diphtheria and should be managed with antidiphtheritic serum, penicillin or erythromycin, oxygen inhalation and humidity. Increasing stridor is an indication for tracheostomy.

If the Infant had been immunised against diphtheria and if there is no membrane then the condition is more likely to be viral croup or epiglotitis due to Haemophilus influenzae infection. However repeated films of throat swab smear for diphtheria bacillus and throat swab culture for diphtheria bacillus are essential to rule out diphtheria.

Occassionally stridor in a toddler may be due to an inhaled foreign body, and this should always be kept in mind.

(i) Cardiac failure.

An infant presenting with dyspnoea and crepitations may be having cardiac failure complicating congenital heart disease. The presence of cardiac murmurs, absent or feeble femoral pulses (as occurs in coarctation of aorta) are suggestive of congenital heart disease. An increasing liver size confirms cardiac failure. Jugular venous pressure is diffieult to estimate in infants. Oedema may be due to malnutrition, nephrotic syndrome or cardiac failure. Diuretics, and if necessary digoxin are used in the management of cardiac failure complicating congenital heart disease. Once the cardiac failure is controlled the infant is best referred to a unit where cardiac investigations and if necessary cardiac surgery to correct the defect can be carried out.

2. Vomiting

(a) Wrong feeding.

The eommonest cause of vomiting in infants is wrong feeding techniques and very often failure to burp the infant after feeds. This improves when the mother adopts correct feeding techniques and burps the infant well after feeds.

(b) Gastro-oesophageal reflux,

This improves when the infant is propped up after feeds, ideally in a feeding chair. Stubborn cases respond to thickening the feeds.

(c) Congenital hypertrophic pyloric stenosis.

This is an important cause of vomiting in infants. Typically the infant

presents with vomiting starting at second to third week after birth and rapidly becoming projectile. Males are affected four times more commonly than females. First born baby is affected more frequently. If a test feed is done the triad of

1. Visible gastric peristalsis
2. Palpable pyloric mass

and

3. Projectile vomiting during or after the feed confirms the diagnosis. The pyloric tumour is best felt after the vomiting.

Management is essentially surgical. Pyloromyotomy (Ramstedt's operation) is effective in relieving the condition. The infant who is very often ill due to dehydration and hypochloraemic alkalosis should be prepared by intravenous administration of normal saline before surgery.

(d) Duodenal and Intestinal atresias.

These conditions often present during the neonatal period. But partial atresias or partial diaphragms may present with vomiting in later infancy. Presence of bile in the vomitus should always arouse the suspicion of intestinal obstruction.

(e) Intussusception.

Intussusception is another cause of intestinal obstruction in infants. The common age group is 3—9 months. In this age group the finding of a local cause for intussusception is an exception. In some infants there is evidence of causal relationship with adenovirus or Herpes simplex virus infection. It is not always that infants are brought with a typical history of attacks of screaming with abdominal colic. The last two children with intussusception in our unit had been admitted to the diarrhoea ward with a history of vomiting and diarrhoea with blood and mucus. But in both these infants examination of the abdomen revealed a sausage shaped mass along the line of the colon. Per rectal examination may reveal a lump. There is almost always blood and mucus on the examining finger. When the examining finger is removed from the rectum, blood and mucus often oozes out of the rectum. This is described in text books as the "red currant jelly stools". Barium enema examination is useful and may result in reduction of the intussusception. Surgery is usually the treatment of choice. Hydrostatic reduction of intussusception is also carried out in some centres and has its limitations.

(f) Acute Appendicitis.

Acute appendicitis can occur in infants too and should be remembered as a possible cause of fever, vomiting and abdominal colic in infants.

3. "Abnormal" Bowel habits.

Very often infants are brought with a history of, "constipation" or "diarrhoea". It is common for some infants to pass stools once in two to three days. On the other hand some other infants pass five to six stools per day. When they have five to six stools per day the motions are often during or just after the feeds. These are both normal. Attention should be paid to the consistency of the stools in deciding whether the infant is having an illness. If the stools are very hard so that it cannot be passed with ease then that may be regarded as constipation. But this is rare. This could be relieved by mild laxatives like a teaspoon of milk of magnesia. In the bigger infant addi-

tion of well ripe sour plantains or papaw to the food very often helps in relieving the constipation.

Stubborn constipation

(a) Hirschsprung's Disease.

(b) Hypothyroidism.

In Hirschsprung's disease there is stubborn constipation and severe abdominal distension. This is due to congenital absence of the ganglion cells of the myenteric (parasympathetic) plexus of Auerbach from a segment of the colon extending to a variable distance from the internal anal sphincter. In most infants the aganglionic segment involves only the rectum and the lower part of the sigmoid colon and there is hypertrophy and distension of the remaining length of colon. In some infants the entire colon may be devoid of ganglion cells. Per rectal examination in most cases reveals an empty rectum. Barium enema and rectal biopsy are helpful in confirming the diagnosis. Sometimes confusion may arise due to an "Ulcerating" colitis causing diarrhoea. But the gross and chronic abdominal distension should suggest the diagnosis of Hirschsprung's disease. Treatment is by surgery.

(d) Hypothyroidism.

Constipation in a lethargic infant should suggest the possibility of hypothyroidism and one should look for other features of cretinism. X'rays for bone age and estimation of Thyroid Stimulating Hormone, Serum thyroxine, Serum Triiodothyronine and Free thyroxine index are helpful in confirming the diagnosis. Treatment consists of administration of appropriate dose of Thyroxine.

Acute diarrhoeas.

If the stools are loose or watery with blood and mucus it is suggestive of acute gastroenteritis. Large number of cases of acute diarrhoeas are due to viral infections and are best managed by administration of fluid and electrolytes alone, to replace lost fluid and electrolytes. The exceptions are infants with Bacillary dysentery and infants with septicaemia who need antibiotics for management.

Chronic diarrhoeas.

Chronic diarrhoeas following acute gastroenteritis are a relatively common problem. In our society this may be due to one of three causes. They are

(a) Malnutrition

(b) Transient lactose intolerance and

(c) Overgrowth of anaerobic bacteria secondary to the use of antibioties.

In malntrition and transient lactose intolerance the diarrhoea is due to partial villous atrophy and management consists of building up the nutrition of the infant with foods like thriposha, eggs etc. and withholding milk for some time if nccessary. Prosobee is a lactose free milk, that is available here and is useful but expensive.

Excessive use of antibiotics in children with acute gastroenteritis may lead to overgrowth of anaerobic bacteria. This in turn can cause diarrhoea. The more antibiotics are used the more the condition will be perpetuated. Clostridium difficile has been incriminated in this condition. Overgrowth of anaerobic bacteria can be controlled by using metronidazole.

4. Feeding problems.

Very often infants are brought with a history of not eating or not drinking enough milk. These infants are usually normal and the complaint is very often a reflection of the parents anxiety. If the infant's weight is adequate and if the infant continues to gain weight, the parents must be reassured and advised not to force food on children for fear that they develop a negativistic behaviour. Offering a variety of foods prepared in different ways may make the infants eat more. Often in practice when the infant for some unknown reason refuses a feed, the mother goes on presisting with that feed till it is time for the next feed. Obviously the infant is not in a mood for the feed. Further, the author finds that if the mothers start giving solids to the infants at about 4 to 5 months of age, very often these infants have no feeding problems. When solids are withheld till the infant is about 10—12 months of age this often crops up. Giving too much of sweets and chocolates too interferes with normal feeding habits of infants. In infants brought with a history of feeding difficulty, it is always good to weigh them periodically to ensure that there is a reasonable weight gain. If there is reasonable weight gain and if this can be demonsirated to the parents this will help in allaying their anxiety a lot.

5. Skin infections.

Infants are brought with recurrent skin sepsis like boils and ulcers. This is very often due to staphylococcal infection. Apart from general cleanliness like cutting the finger nails shorta local application of 1 percent Gentian violet or an antibiotic skin ointment like Polymyxin Bacitracin skin ointment will be adequate if there are a few localised lesions. If the lesions are widespread and in large numbers, in addition to the local application, a systemic antibiotic like erythromycin is very useful. Cleaning the skin with phisohex (hexachlorophane) lotion is also useful in stubborn cases.

6. Convulsions.

This is another common problem in infancy and childhood. A large number of infants whom we see with convulsions, have febrile convulsions. However meningitis as a cause of convulsions should be considered. Usually the first attack of febrile convulsions occurs between 6 months and 6 years of age. Outside this age group, one should always look for other causes for the convulsions. Some would advocate lumbar punctures in all cases of first fit in infancy. The author does lumbar punctures, if antibiotics have to be used to treat the child with the first attack of febrile fits. If the infant remains well inspite of having had fits and gets better without antibiotics, and if the fontanelle is not bulging then one may or may not perform a lumbar puncture.

The infant with fits should be nursed in the coma position and all secretions sucked out. Oxygen may be useful and is necessary if the infant is cyanosed. Intravenous Diazepam is useful in controlling the convulsions. Overdosage or rapid administration of intravenous Diazepam can cause respiratory arrest. The high temperature could be controlled by nursing the infant under a fan or by tepid sponging with tap water. Nothing should be given by mouth when the infant is fitting and unconscious.

If the febrile fits recur or if the first attack of fits was prolonged, lasting for more than three to four minutes, long

term anticonvulsants are used to prevent further convulsions. Phenobarbitone may be used in the appropriate dose for a period of about 3 years.

Infantile spasms.

Infantile spasms is a special type of convulsions that may be seen in infants. This consists of sudden and short lived abnormal movements of the trunk and limbs of the infant and several attacks may occur within 24 hours. Electroencephalogram is typical and is termed "hypsarrythmia". A. C. T. H. or prednisolone is used in the management of these infants.

7. Dysuria

Infants are sometimes brought with a history of crying before micturition. The parents get more disturbed when they see a white patch on the spot where the infant's urine has dried up. Very often the crying and micturition appear to be a coincidence. However in these circumstances it is always wise to do a full examination of the urine for any evidence of infection. This will also help to allay the anxiety of the parents.

In male children the stream of urine should be observed. If the stream of urine is good and there is no ballooning of the prepuce during micturition and urine full report is normal the parents should be reassured. It is normal for the prepuce to be adherent to the glans penis in infants. Hence the prepuce is not very often retractable. On the other hand a poor stream of urine, ballooning of prepuce during micturition and recurrent balanitis are indications for further investigations and, or surgical management.

8. Jaundice

Jaundice is a problem during the neonatal period. However if jaundice which started during the neonatal period persists in the post neonatal period three important conditions have to be remembered and appropriate investigations carried out.

(a) Hypothyroidism :- Investigations as mentioned earlier.

(b) Galactosaemia : Urine should be tested for reducing substances. If it is positive then identification of reducing substance, is proceeded with.

(c) Extrahepatic biliary atresia - The following investigations are necessary; Urine for bile & urobilin, Stools - inspection, Serum bilirubin-Direct & Indirect, Liver function tests, and Cholangiogram. Laparotomy may be necessary for the definitive and early diagnosis. Ideally the diagnosis should be established before the completion of three months of age

9. Breath - holding attacks

These are not uncommon in infants and are usually precipitated and perpetuated by pain or frustration. Shortly after a bout of crying the infant suddenly stops breathing in expiration and becomes cyanosed. The infant may lose consciousness and become rigid. This may even lead to a few convulsive twitches. But respiration always starts with rapid recovery and there is no danger to life. The susceptibility to breathholding attacks is usually short lived and they cease spontaneously as the infant matures. If there is any diagnostic difficulty electro-

encephalography (E. E. G.) may be helpful. E. E. G. is normal in breathholding attacks.

10. Failure to thrive in infancy

The height and weight of the infant brought with a history of failure to thrive should be checked. The observations should be repeated at intervals to confirm whether the infant is failing to thrive. Good dietetic history and advice on diet are very important in these infants. Other important causes of failure to thrive in infancy and appropriate investigations are given below.

Eventhough hookworm infestation is rare in the infants we have occassionally seen it in the under one year old. Coeliac disease and Fibrocystic disease of pancreas appear to be rare or absent in our society but we should be on the look out for these illnesses and appropriate studies should be carried out before we can comment on the prevalence of these conditions in our society.

Investigations

(a) Urinary tract infection.	Midstream urine for full report & culture.
(b) Congenital renal abnormalities.	Intravenous Urogram and, or Micturating cystourethrogram
(c) Congenital heart disease	Teleradiogram of chest Electrocardiography
(d) Hypothyreidism	As mentioned earlier
(e) Hook worm infestation	Stools for A. O. C.

Leprosy in the Village of Kattupulam in Jaffna

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Dr. N. Sivarajah, M.B.B.S. (Cey). D.T.P.H. (Lond)+

Summary

A new pocket of leprosy cases was discovered in the Jaffna District at Kattupulam. In this village with a total population of 514, there were 57 cases of leprosy -5 lepromatous and 52 tuberculoid. These cases were all confined to 29 families out of a total of 104. The commonest clinical manifestation was either single or multiple hypopigmented macular lesions with a variable loss of touch, pain and temperature sensation in the limbs.

Introduction

It is well known that cases of leprosy tend to occur in certain areas in clusters as pockets of infection. We are already aware of such pockets of infection in Jaffna, at Pungudutivu, Valvettithurai and Atchuveli. Few years ago we noticed a number of new cases of leprosy arriving from Kattupulam a small hamlet at Thiruvadinilai 12 miles from Jaffna. These cases were referred to the clinic by patients or volunteer workers. A survey was organised in the village itself in February 81 to detect new cases and to study the clinical features. Suspected cases were further investigated at the leprosy clinic in General Hospital Jaffna. Slit skin smears were done in all cases. The cases already registered from the area are also included in this study.

Results

A total of 57 cases of leprosy were diagnosed from this area which has a population of 514 with 253 males and 261 females. Of these 27 were males and 30 females. The age distribution is given in Table I and familial incidence in 11 families with more than one case is given in figure 1. Of the total 104 families all the cases were confined to 29 families.

Table I

Cases of leprosy by age distribution

Age	No. Cases
0 — 10 Years	9
11 — 20	13
21 — 30	15
31 — 40	4
41 — 50	4
51 — 60	7
Over 61	5

Of the 57 cases 5 were lepromatous and 52 tuberculoid. The lepromatous cases were all males and included a father and son. Father was diagnosed in 1970 and son in 1979. Other cases were diagnosed in 1975, 1980, 1981. The tuberculoid cases number 52 and consisted of 22 males and 30 females.

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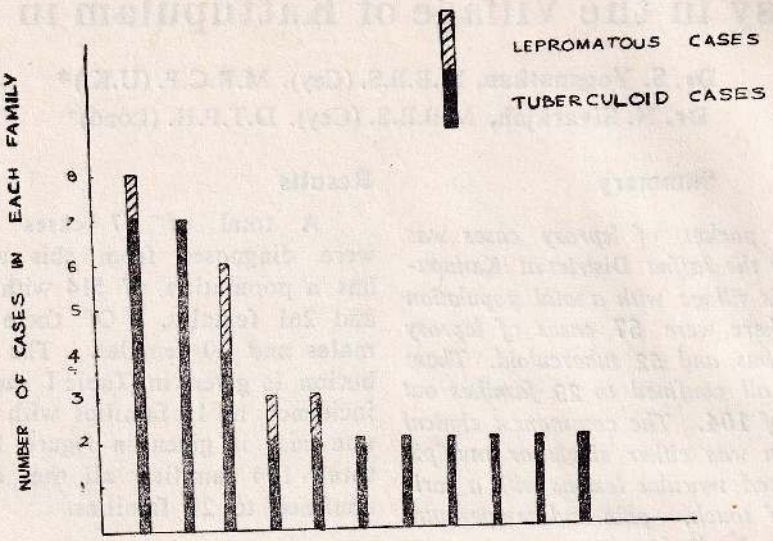

FIGURE 1 DISTRIBUTION OF CASES IN EACH OF THE 11 FAMILIES WITH MULTIPLE CASES

Commonest clinical presentation of the lepromatous cases was generalised nodular infiltrates with thickened ear lobes. The ulnar and greater auricular nerves were also thickened. All these cases had their slit skin smears positive for leprosy bacillus. These cases were already diagnosed and attending the leprosy clinic for treatment although not regularly.

The tuberculoid cases had single or multiple hypopigmented macular lesions with variable loss of touch, pain and temperature senses. Some of the lesions had a raised margin with central flat area. Few others had an erythematous appearance. The lesion was single in 25 cases. Of these 19 were found on the limbs, 4 on trunk and 2 on face. Other 27 had multiple lesions on trunk and limbs, The size of the lesion was variable from a few millimeters to several centimeters. The shape of the lesion was oval or rounded. In 24 cases the nerves were thickened and palpable. Only ulnar nerve was thickened in 14 cases, greater auricular and ulnar nerves in 8 and lateral popliteal in 2. One case presented without any skin lesions with ulnar nerve thicking and palsy.

Duration of symptoms and signs at the time of diagnosis was under 6 months for 25 cases and between 6-12 months for 26 cases and 2 years for one case.

None of the cases had any permanent deformities.

Discussion

The number of cases of leprosy in the world is estimated to be around 15 million of which about 3 million are found in the Indian Subcontinent. Africa

has the greatest prevalence rate of 20-50 per thousand.[1] Prevalence rate for Sri Lanka is 9·74 per thousand[2] and that of Jaffna 0·57 per thousand.[3] For Kattupulam the rate is 110 per thousand. Table II[4] gives a few other comparable rates. These rates although not strictly comparable are very high. It is known that in certain villages in endemic areas virtually all the inhabitants contract leprosy sooner or later.[5] This situation may occur at Kattupulam if active surveillance and treatment are not carried out.

Table II

Prevalence rates of leprosy in certain selected countries

Country	Prevalence rate per 1000 population
Argentina (Chaco)	5·6
Thailand (Khonkaen)	12·4
Brazil (Candeias)	10·6
Cameroon	25·8
Northern Nigeria (Katsina)	28·8
Burma (Shewebo)	32·6
Burma (Myingyan)	44·4

Leprosy can occur at any age. In our series the youngest was 3 years old and eldest 76. 47% of cases were clustered in the 11-30 age group. This tendency is seen in other series too.[6] The possible reason for this is their greater exposure and greater susceptibility.

In this series all the lepromatous cases were males while in the tuberculoid cases 22 were males and 30 females. Leprosy in adults is more prevalent in males than in females. (1·6 : 1). Lepromatous rate is significantly higher in males than in females. However in children there is no significant difference between sexes.[7] The overall figures in our series are 27 males and 30 females.

21 cases (36·8%) were found in 3 families and the rest in 26 families. The current view explains this by a dual mechanism of heredity and contagious transmission. The disease is believed to appear in persons hereditarily susceptible when exposed to infection. This may have happened at Kattupulam. The two lepromatous cases discovered in 1971 and 1975 may have provided the source of infection for all these cases.

In this study 29 patients (50·9%) had skin lesions and nerve enlargement. 27 patients (47 4%) had only skin lesions and one (1·7%) had only nerve enlargement. In the WHO leprosy BCG study in Burma[8] the corresponding figures were 9·8% : 79·5% : 10·6% respectively. The possible reason for the variance may be because most of the patients in WHO study were children. However the total number of patients with skin lesions was 98% compared to 90% in the WHO study.

Skin lesions may be single or multiple. In this study 25 patients (43·8%) had single lesions and of these in 19 (76%), the lesions were found on the limbs and in 4 (16%) on trunk and 2 (8%) on face. The comparable figures in the WHO trial, are given in Table III.

Table III

Distribution of single lesion compared with leprosy B. C. G. trial in Burma

Area Involved	Kattupulam	Burma
Limbs	76%	84 6%
Trunk	16%	12·6%
Face	8%	2·5%

These figures clearly show the ease with which these cases can be spotted. It is essential for medical officers to look for and test for hypopigmented anaesthetic lesions. If this is done routinely we could detect many more cases of leprosy early.

One consoling feature was that none of these patients had any permanent deformities. The possible reason for this is the fact that these patients had contracted illness only within the last few years. Good results can be achevied if these cases are treated rugularly.

Acknowledgement

We would like to thank Mr. V, Wijeyaratnam, Public Health Inspector, Anti-Leprosy Campaign who has been of immense help in preparing this paper. We would also like to thank SHS Jaffna and the Health Educator for organising the survey.

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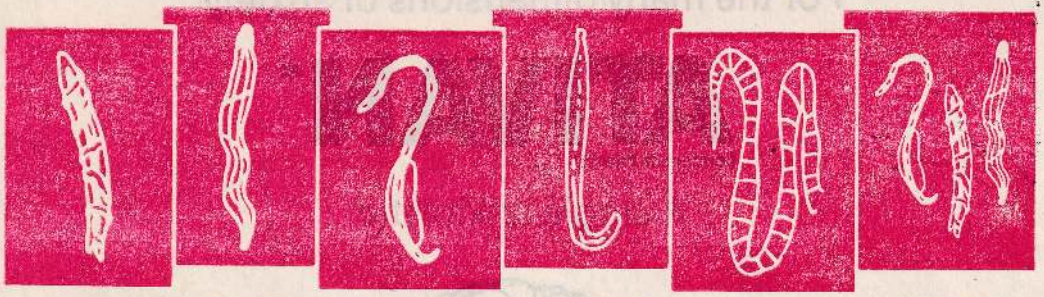

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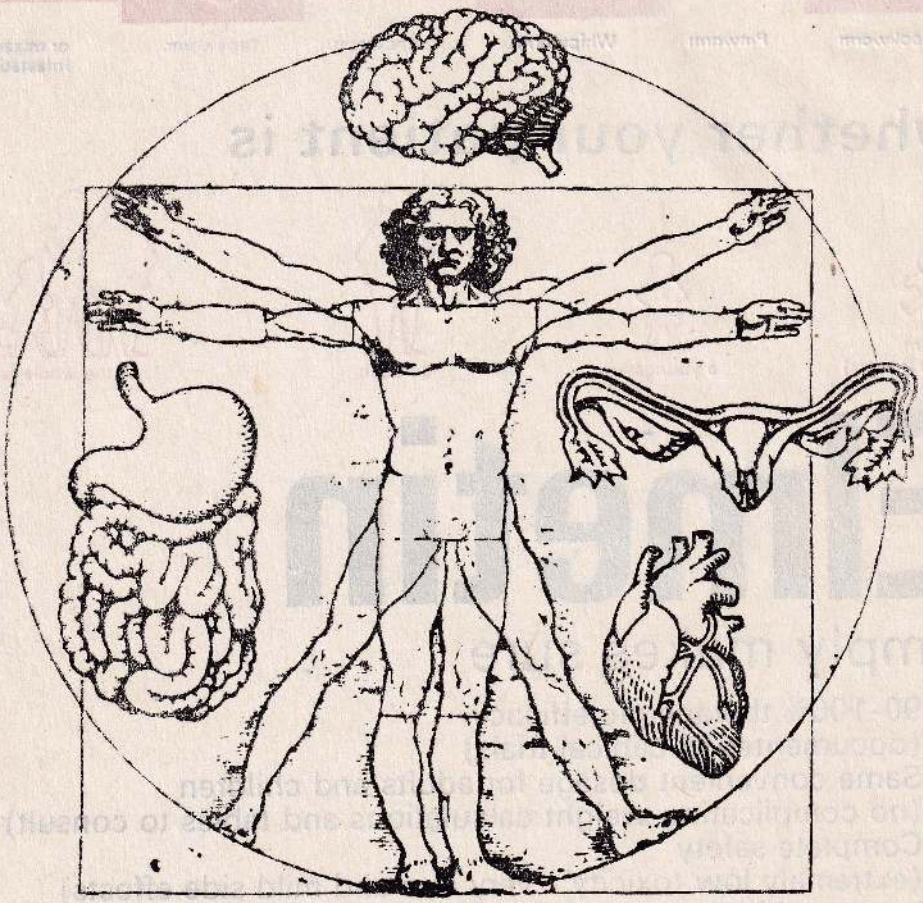

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Hereditary Spherocytosis

— Case Report —

S. Yoganathan, M. B. B. S. (Cey.); M. R. C. P. (U.K.)[1]
Miss. R. Manickam, M. B. B. S. (Cey.)[2]
Mrs. V. Velummylum, M. B. B. S. (Cey.)[3]

Introduction.

Hereditary Spherocytosis is a haemolytic disorder in which the fundamental abnormality is an intrinsic defect of the red cells, which results in the cell being spheroidal in shape. Though a few families have been diagnosed to have this disorder in Sri Lanka, there are no published reports in this country. This disease occurs most frequently in persons of British Northern European stock, in whom it is the commonest form of a hereditary haemolytic disease. Its incidence in those countries is approximately 1 : 4500. It is a disease of autosomal dominant inheritance. Though there are no unusual features in this case, we report it for the following reasons:-

(1) To make the doctors aware that this disorder is prevalent in the North, as this can be treated very effectively. Wrong treatment with iron can be harmful to the patient.

(2) There was a strong family history, with all the living members of this family being affected.

Case Report.

Miss. V. S. (figs 1 a & b) — 24 years old, was admitted to Base Hospital, Point Pedro on 15th of August, 1981 with a history of dyspnoea on exertion of five years duration. She also complained of yellowish discolcuration of her eyes and this had been coming on and off for the previous five years. The colour of her stools and urine had been normal. There was no history of fever, colicky abdominal pain or pruritus. Before the onset of this illness, she had been quite well. There was no family history of pallor or jaundice. She had oligomencrrhoea for the previous three years. On examination she looked ill. She was very anaemic and mildly jaundiced. There was no lymph node enlargement. She had mild ankle oedema. On examination of the abdomen, liver was enlarged to 3 cms below the costal margin and the spleen, to 2 cms below the costal margin (fig. 2) On examination of the cardio vascular system there was sinus tashycardia, blood pressure of 110/70, mild cardiae enlargement, triple rhythm and an ejection systolic murmur in all areas. Other systems were clinically normal.

Investigations at this stage gave the following results :

Haemoglobin — 5 G %

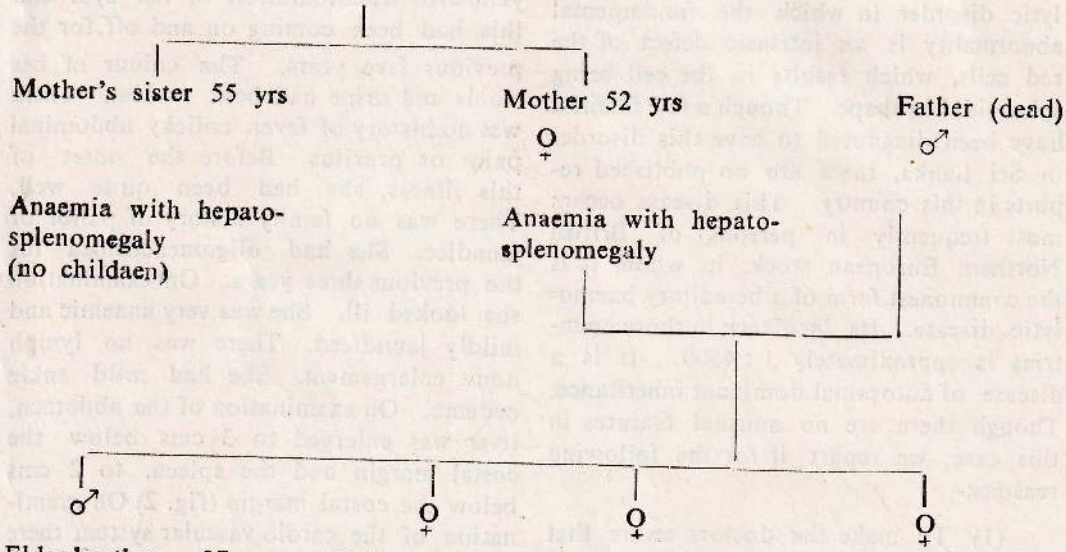
Blood Picture — large number of spherocytes with polychromasia and macrocytes.

---

1. *Consultant Physician, Base Hospital, Point Pedro.*
2. *House Officer, Base Hospital, Point Pedro.*
3. *House Officer, Base Hospital, Point Pedro.*

Reticulocyte count — 5 %
W B. C. — 3,200 / Cmm P-46 L-45 E-09
Serum Bilirubin — 2.4 mg %
Alkaline Phosphatase — 5.5 K. A. units
Urine — Bile – Nil
Urobilin — Normal
Platelet Count — 200,000 / Cmm
Antinuclear factor — Negative
Coomb's Test — Negative
Haemoglobin electrophoresis — No abnormal haemoglobin
Red cell fragility — Increased
X'ray's of skull and hands — Normal

As the investigation at this stage was suggestive of a haemolytic anaemia, all the members of the family (fig. 3) were examined. Clinical findings are shown in the family tree given below



The blood films of all the living membrs in the family showed hereditary spherocytosis. They all had negative Coomb's test and showed no abnormal haemoglobins on electrophoresis.

Our patient was transfused with 2 units of blood and splenectomy was done on 28th of August, 1981. A congested spleen weighing 400 G was removed. There were no palpable stones in the gall bladder. On 1st of October, 1981 when the patient was reviewed, she was very well and the repeat investigations showed the following results.

Haemoglobin — 12·5 G%
W. B. C. — 11,000 per Cmm. P—76 L — 14 E — 10
Platelet count — 500,000 per Cmm
Serum Bilirubin — 0.8 mg%

Fig. 1 a

Fig. 1 b

The Patient

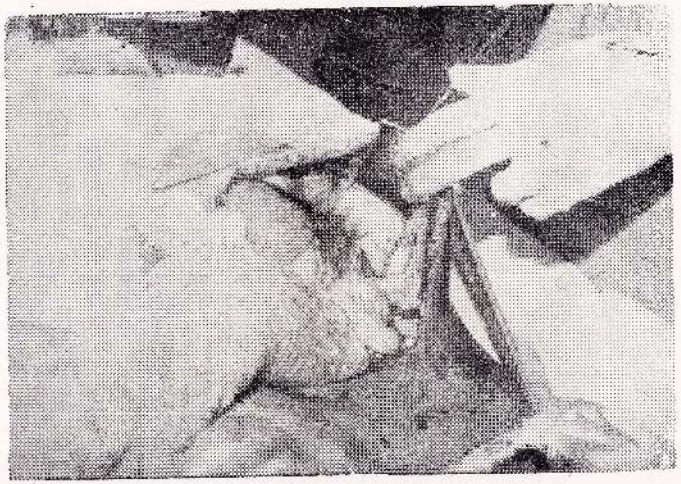


Fig. 2 Hepato-splenomegaly seen at the operation

Fig. 3 Other members of the family

Discussion.

The characteristic red cell abnormality in this disorder is the spherocytosis. The red cells are spherical in shape, rigid and have increased permeability to sodium. Compensation for this increased permeability is achieved by an increased rate of glucose metabolism to generate more ATP with which the red blood cells extrude this excess of sodium. Spheroidal contour and rigid structure of the red blood cells impede their passage through the spleen. There the red blood cells are exposed to an environment in which their increased metabolic rate cannot be sustained. The first injury imposed upon them by the spleen is a further loss of surface membrane ("conditioning") which produces a sub-population of hyperspheroidal red blood cells in the peripheral blood These are subsequently destroyed in the spleen.

Unlike our patient, most patients with this disorder, present within first ten years of life. Occasionally the disorder is obvious shortly after birth. Like the relatives of our patient, some many have no symptoms and the disorder is discovered on a routine examination when they are found to have an enlarged spleen. Anaemia, splenomegaly and jaundice are the major clinical features of this disease. Though the jaundice is usually persistent, it can be intermittent and is less pronounced in early child-hood. Because of the increased bile pigment metabolism, gall stones of pigment type are common, even in childhood. Because the bone marrow's capacity to increase erythropoiesis by six-to-ten-fold exceeds the usual rate of haemolysis in this disease, anaemia is only mild or moderate and often absent in otherwise healthy individual. This is exemplified by the other members of this family. Compensation may be temporarily interrupted by episodes of erythroid hypoplasia (aplastic crisis) or by increased hamemolysis during systemic infections (haemolytic crisis). Splenomegaly is a constant feature of hereditary spherocytosis. Chronic leg ulcers may occur. The blood picture typically shows anaemia with spherocytes, and increased erythrocyte osmotic fragility, a raised reticulocyte count, increased serum bilirubin level and negative Coomb's test. The diagnosis is based on clinical features, haematological features and family history, Hereditary sphrocytosis must be distinguished from the spheerocytic anaemia associated with red cell anti-bodies. The Coomb's test will help to differentiate the latter. Splenectomy reliably corrects anaemia although the red cell defect persists. The operative risk is low. Rare relapses have been reported and are probably attributed to postoperative growth of splenic autotransplants or to hyperplasia of accessory spleens which were overlooked at the operation. Splenectomy is indicated in all patients except those who are symptom free and well compensated. Even in these patients, splenectomy should be considered because of the risk of gall stone formation. When diagnosis is made in child hood, splenectomy is better postponed until the age of 7 years, because the splenectomised children become susceptible to infection. A cholecystogram should be performed before operation and if stones are found a cholecystectomy will have to be done at some stage. Due to lack of facilities,

we could not do a cholecystogram on our patient, but there were no palpable gall stones in the gall bladder at the operation. Because of the increased requirement of folic acid in patients with haemolysis, a deficiency of this vitamin may exist and therapy with folic acid may result in an increased haemoglobin level.

Acknowledgements :

We wish to thank the D. M. O. and Staff of Base Hospital, Point Pedro for their encouragement, Prof. C. C. Balasubramaniam F. R. C. (Path), Department of Pathology, University of Jaffna for his assistance and Mr. K. Sabaratnam for typing the script.

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A case of acute abdomen due to torsion of left fallopian tube.

Dr. M. Vetpillai. M B.B.S (Cey), F.R.C.S (Lon), F R.C.S. (Edin).*

Summary.

A case of torsion of fallopian tube, in a fourteen year old school girl presenting as an acute abdomen is reported. The gangrenous fallopian tube was excised following whieh the patient had an uneventful recovery.

Introduction.

Torsion of the fallopian tube is rare. The first case was published in 1890 by Bland-Sutton and since then approximately 350 cases have appeared in the literature.[1] This case is noteworthy in view of very few cases having been reported in children.

Case report.

Miss. Y. A , a fourteen year old school girl, who had not attained menarche, was admitted to the surgical unit as an emergency on IIth September 1981. She complained of severe lower abdominal pain on the left side, vomiting and high fever for the two days prior to admission. On further questioning, she admitted that the abdominal pain started about twenty days prior to admission, was colicky in nature lasting for few minutes and recurred once in two or three days. The severity of the pain increased to a greater extent for the last two days prior to admission, during which period the pain became continuous. There was no alteration of bowel habits or micturition. She had no history suggesting any pelvic infection in the past.

On examination, the child looked ill and the temperature was 103° F. She was mildly dehydrated with a pulse rate of 110 per minute. The blood pressure was 90/50. Her abdomen was distended and was very tender all over. The peristalsis was sluggish. With this clinical picture of acute abdomen, after resuscitating the patient with intravenons fluids, laparotomy was performed through a lower midline incision. There was about two hundred mls. of blood stained fluid in the peritoneal cavity. The left fallopian tube had undergone torsion and was distended resembling a loop of small intestine. The loop was gangrenous Fig 1. The opposite fallopian tube and both the ovaries looked normal. The rest of the

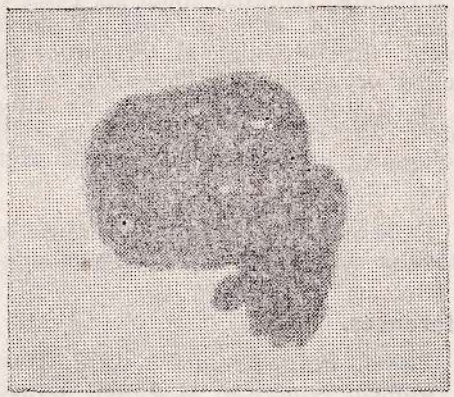

Figure 1.
The gangrenous fallopian tube.

* *Consultant Surgeon, Base Hospital, Point Pedro.*

abdominal viscera too was normal. The gangrenous fallopian tube was excised and routine closure of the abdomen done. The postoperative recovery was uneventful.

Discussion.

The fallopian tube is an elongated structure lying horizontally along the upper border of the broad ligament with the proximal and the distal ends wide apart. This makes the torsion in a fallopian tube a rare event unlike pelvic colon[2], where there is a loop of bowel with the proximal and the distal ends closer making torsion, occur commonly. Hence, when torsion occurs in the fallopian tube, there is usually some antecedent pathology or abnormal anatomy present.[3] Regad, in 1933, reviewing 201 cases of torsion. found that in 24%, the tube had previously apparently been healthy; in 18% there was hydrosalpinx; in 14% it was part of a hernia; in 13·5% there was infection, tumour, or ectopic pregnancy; and in 12% there was intrauterine pregnancy. Among these cases, 80% occured in the age range of 13 to 49 years while the remaining 20% of the affected were before puberty. In 1972, Powell etal,[1] reviewing the literature, reported about six cases of torsion of the tube in postmenopausal women. Rarely[4], bilateral torsion of the tube has occured. In Srilanka,[5] a torsion of the fallopian tube following tubal ligation was reported. In this case the development of hydrosalpinx was the cause of the torsion.

There are several[6] theories of causation of torsion of the tube: (I). The anatomical theory is that malformations of the mesosalpinx or the tube, such as excessive length, tortuosity, long mesosalpinx, and hydatids of Morgagni, favour torsion. (2) the physiological theory is that disturbances in the normal peristaltic movements of the tube, either due to autonomic dysfunction or drugs, may result in some spasm and lead to torsion, (3) the haemodynamic theory is that the veins of the mesosalpinx are longer and more flexible than the arteries and, in the event of venous congestion, they may assume a spiral course which favours torsion.

Torsion of the tube is liable to occur in pregnancy or the puerperium due to the rising or falling uterus which acts as a positive or negative rotating force. The fact that the torsion of the tube occurs rarely in postmenopausal women may be due to the postmenopausal atrophy of the tube and its vascular supply.

In this child, there was no evidence of any abnormal anatomy or any adhesions to suggest any previous pelvic infection. The history of colicky abdominal pain on and off, becoming severe and continuous is typical in a case of volvulus of bowel and a similar history was present in this case. The intermittent pain was probably due to the partial twist that untwisted itself. It is difficult to make a diagnosis of this condition preoperatively and a review of the literature reveals that no one has claimed to have made the diagnosis preoperatively. Early intervention and untwisting of the tube, may save the tube.

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4. Lewis, A. C., 1971, Bilateral torsion of Fallopian tube, J. Obstet and Gyn. Br. Commonwealth, **78**, 93-94.
5. Panchalingam, 1967 Torsion of Hydrosalpinx - a complication of tubal ligation, Ceylon Medical Journal, **12**, 176-8.
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News and Notes

A Medical Exhibition, organised by the Jaffna Medical Association, will be held from 21st April 1982 to 25th April, between 10 A. M. and 8 P. M. at St. Patrick's College, Jaffna. It is intended to serve the dual purpose of fuud raising for a Building (Office & Library) for the J. M. A. and public education. Contributions. for the Building Fund are welcome and should be sent to the Treasurer, Jaffna Medical Association, General Hospital, Jaffna.

* * *

The V. T. Pasupathy (Inaugural) Memorial Lecture will be delivered by Dr. P. Sivasubramaniam, F. R. C. S. Ophthalmic Surgeon, Colombo,on 9th May 1982 at 6 P. M. Please contact, President, J. M. A, for venue.

* * *

An Emergency Unit sited in the O. P. D., General Hospital, Jaffna has started functioning.

* * *

The site has been cleared and work has just started on an Intensive Care Unit, near the Operation Theatre. The building is estimated to cost 4 lakhs of Rupees and the equipment much more. It is being funded by the public. Contributions may be sent to the Treasurer, Jaffna General Hospital, Development Association, General Hospital, Jaffna. Donations of equipment are also welcome.

* * *

Dr. V. Kunananthan, F. R. C. S, has been appointed, Neuro-Surgeon General Hospital, Jaffna, and is functioning in a "Mini - Hut" of 12 beds.

* * *

The post of E. N. T. Surgeon, was advertised but not filled. We appeal to E. N. T. Surgeons, here and abroad, to apply, in the interests of the Medical Students and the patients.

* * *

It is proposed to compile an Index to the Volumes of the Jaffna Medical Journal. Those in possession of volumes published between 1953 and 1965, (both years inclusive), are requested to contact the Editor.

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