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(THE OFFICIAL PUBLICATION OF THE JAFFNA MEDICAL ASSOCIATION)

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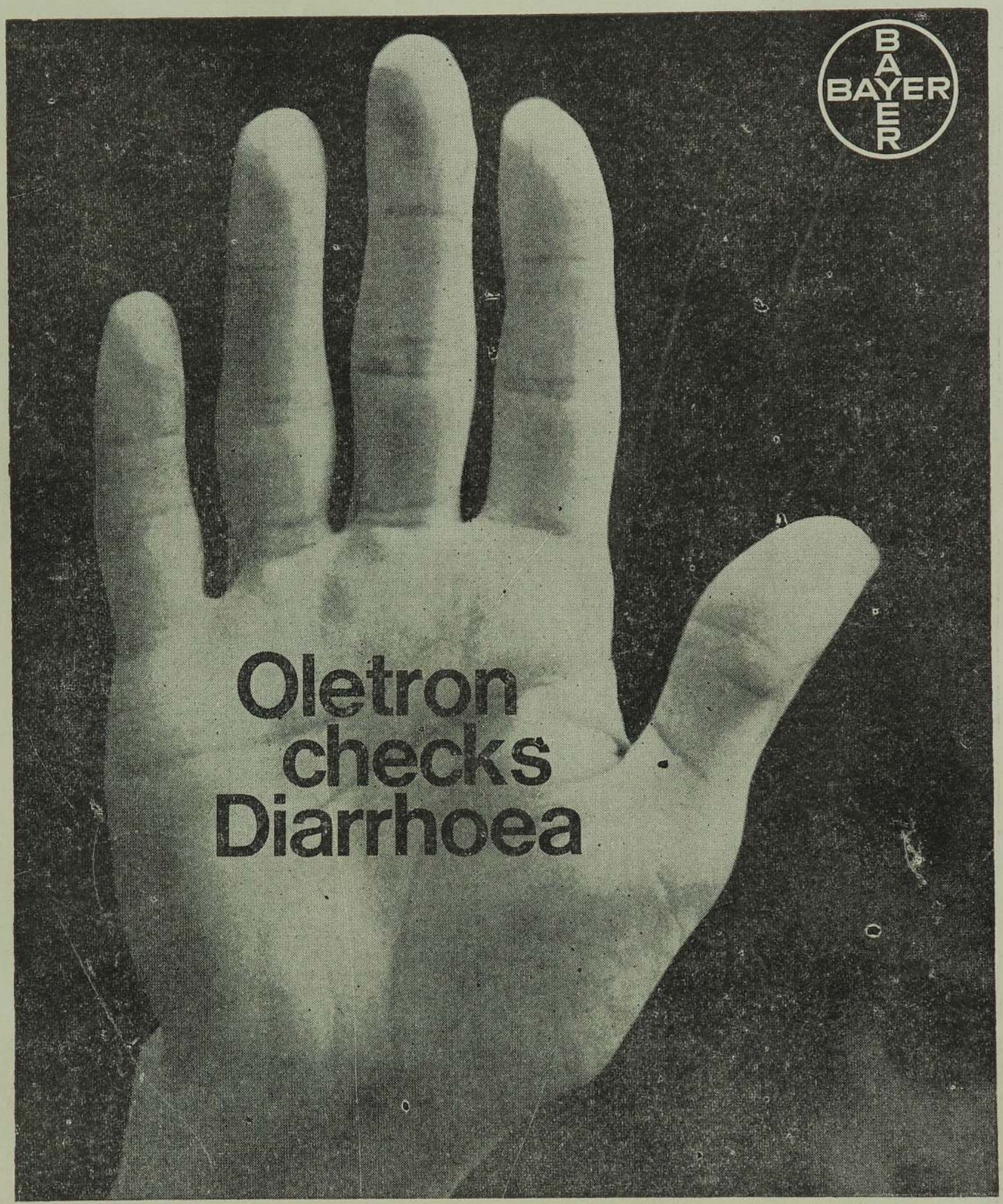

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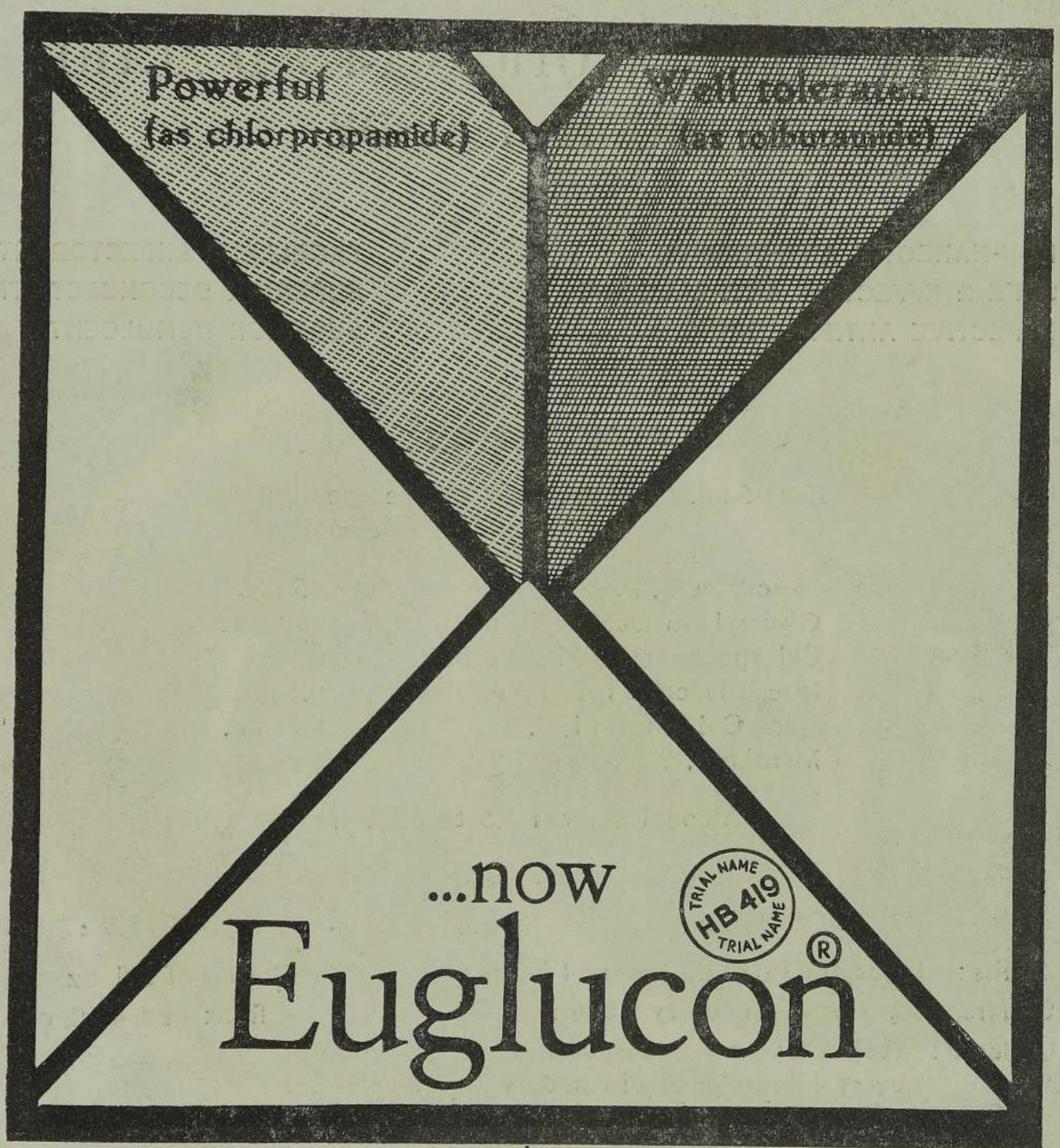

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Editorial....

Post-Graduate Medical Education

THE idea of providing post-graduate medical education in Ceylon had in the past been opposed by several persons including doctors. The lack of facilities in the form of equipment, finances and even talented personnel has been mentioned as the chief objection. If this is carefully analysed it will be seen that at the moment there is certainly no lack of talented personnel for post-graduate teaching. There however is the problem of equipping a hospital and a school for post-graduate teaching, an undoubtedly expensive project, especially if one is to keep up with the advances in medicine in other countries. This too should not be a very serious objection if the foreign exchange saved by conducting post-graduate medical education in Ceylon itself is considered. Another objection often pointed out is that the younger doctors would be denied the chance of going abroad to other countries to see recent trends in medicine. It has often been pointed out that at present doctors going abroad for post-graduate studies, spend most of their time following lectures and cramming away in the libraries preparing for their examinations. It appears to be more profitable for one to finish his post-graduate studies in Ceylon and then go abroad to the foreign centres of medicine to gain practical experience in his branch of medicine.

The present trends indicate that post-graduate medical education in Ceylon will soon be a reality and that Ceylonese post-graduate degrees will be accepted as equivalent to foreign degrees. In fact they may be the only post-graduate degrees many doctors in Ceylon would possess in the near future. Therefore, however opposed we may be to this move, it is our duty to rally round and help the future doctors in this country by ensuring that the post-graduate medical education to be imparted in Ceylon will be of a high standard, a standard that we would be proud of, a standard that will be recognised by medical bodies even outside Ceylon.

With this in view various colleges in the different branches of medicine are being formed. Some have come into existence, others are in the process of being formed. The fact that Ceylon is a small country with a proportionately small number of medical men, poses certain problems. Unlike the colleges of medicine and surgery abroad the membership of Ceylonese colleges is bound to be small. The Royal College of Surgeons of England has at present over 7500 fellows in Surgery, over 2900 fellows in Anaesthesia and over 1100 fellows in Dental Surgery. After the formation of the different colleges in Ceylon it is very unlikely that corresponding figures in Ceylon would exceed 300 in Surgery, 100 in Anaesthesia, 50 in Dental Surgery. This would mean that unlike countries abroad a smaller number of members would be available to be at the helm of the college affairs for long periods.

Similarly selection to the examining boards of the colleges would be limited. This may not be in the best interests of the individuals selected to be on the executive bodies or examination boards, as in addition to working hard they may have to face accusations (often unfair and unjust) of inflicting their individual prejudices on the conduct of the college affairs. Nor would it be in the best interests of the Colleges and the future of Post-graduate medical education. In the matter of examinations, appointment of foreign examiners to the boards eliminates such bias and helps maintain a standard comparable to similar degrees abroad.

A post-graduate medical school with an attached hospital having all modern facilities for the teaching of the various branches of medicine is being planned. While whole-heartedly welcoming this step and stressing the importance of a well equipped hospital for teaching, one should not fail to emphasise the need for providing the hospitals in the provinces with their basic requirements at least. The principal hospitals in the provinces at least should not lag very far behind in the matter of equipment. It is only then that one can practise at least in part what one has learnt in the post-graduate medical schools at home or abroad. The standard of work in the provincial hospitals will have a very great influence on the standard of post-graduate medical education in Ceylon, as all the students would be working in these institutions at some time or other before or after their post-graduate studies. Improving the provincial hospitals is therefore an important step in the conduct of post-graduate medical education in Ceylon.

Developing the provincial hospitals would help the public directly as it would ensure that a patient in what ever part of the country he is, need not be transported very far for specialised treatment and care. Patients should not feel that while one hospital is being modernised and fully equipped the rest of the country has to manage with out-dated or substandard facilities.

It must be admitted that providing provincial hospitals with even the basic facilities is a costly project, and one cannot expect the government to provide every thing the hospitals need. Most hospitals abroad have welfare associations or social service organisations to help them in various ways. The societies help the hospital by collecting funds for purchasing equipment or for repairs to existing equipment. These are steps which are normally greatly hampered by administrative red-tape. It is heartening to note that at least in one provincial hospital a Hospital Development Society has been formed. Let us hope that other areas too would follow and help the government to help the hospitals.

We hope that the departmental authorities too would help by reducing red-tape in hospitals. To cite just one example, inspite of the inadequate strength of medical and nursing staff in hospitals, at present doctors and nurses spend more time chasing after every pill and ampoule (even if it costs only a few cents) and balancing drug account books, rather than treating and nursing patietns.

CONGENITAL ABNORMALITIES OF THE ANUS AND RECTUM IN JAFFNA

BY

DR. V. A. BENJAMIN M. S. (Cey.) F. R. C. S. (Eng.) F. I. C. S.

Surgeon, General Hospital Jaffna.

President, Jaffna Medical Association

(This study was the subject of a lecture delivered on 12th November, 1970, at the Joint Scientific Sessions of the Ceylon Medical Association and the Jaffna Medical Association.)

CONGENITAL abnormalities of the anus and rectum are fortunately uncommon. Aird [1] described the incidence as 1 per 10,000 births. McPherson [2] of Bristol believed the incidence to be between 1 : 5,000 and 1 : 10,000 live births. Partridge and Gough [3] of the Hospital for Sick Children, Great Ormond Street, London were inclined to agree with Bradham [4] in giving the incidence as once in every 5,000 births. Nixon and O'Donnell [5] estimated a slightly higher incidence, of 1 in 3,000 babies. These figures were for European communities. American figures did not significantly differ from these. However, for South Africa, Louw [6] estimated a much higher incidence of 1 in 1,740 for their white population, 1 in 1,770 for the coloured community, and 1 in 2,260 for the Bantu, giving an average of 1 in 1,800 for the entire population of that country.

The study undertaken in Jaffna seems to point to a much higher incidence in Jaffna. It appears that in Jaffna, the incidence of congenital malformations of the anus and rectum is approximately between 1 in 800 and 1 in 1,000 births. The author, being fortunate in having worked for many years as surgeon in several different districts, serving approximately equal lengths of time at Trincomalee, Batticaloa, Colombo South, and Jaffna, is convinced that Jaffna is unfortunate in having a significantly higher incidence of this malformation than among residents south of Killinochchi. The only other possibility is that there has been an epidemic of this malformation among children born in Jaffna during the period under study, a possibility that cannot entirely be ruled out.

During the 34 months, from 1st January 1968 to the 31st of October, 1970, a total of 34 patients with anorectal anomalies presented under the personal care of the author, and these 34 patients were surgically operated on by him. 32 of these cases occurred at the General Hospital, Jaffna, while Green Hospital, Manipay and McLeod Hospital, Inuvil had one case each. 34 patients operated in 34 months, worked out to an average encounter of 1 case per month. The first 12 cases were met with in 1968, 16 in 1969, and the rest in the first 10 months of 1970.

It would be difficult to work out the incidence of this anomaly in the district, but it would be quite easy to arrive at the incidence of the malformation among births that took place at

the General Hospital Jaffna. In 1963, some form of anorectal malformation occurred in 8 out of 5381 total births in the hospital, giving an incidence of 1 in 672.6 births in that year. When worked out for the full period under review, the incidence appears to be in the region of approximately between 1 in 800 and 1 in 1,000 births. This is also in agreement with the impression conveyed by the Obstetric Colleagues at General Hospital Jaffna (personal communication), that each of them have one infant with anorectal malformation, born at each of their respective units, every 2 or 2½ months.

NOMENCLATURE

It is suggested that the word "Imperforate anus" be avoided when referring to anorectal malformations, because the word "imperforate anus" implies the presence of a membranous occlusion at the anus or the anal canal, a membrane that has not been perforated. Not a single case that could have come within this meaning was seen among the 34 patients reviewed. This is in accordance with reports of other observers too, who found that true imperforate anus was an extremely rare phenomenon. The importance of this fact lies in that this knowledge should deter any doctor from the temptation to perform a cruciform incision in the perineum under the mistaken belief that there is only a membrane to be ruptured. Nothing is more tragic, because children have been condemned to a lifetime of embarrassment and misery from such an error in initial management.

The words "Atresia" and "Agenesis" are often used synonymously when describing some of these malformations. It would be better to retain the word "Atresia" to denote occlusion, i.e. obliteration of a pre-existing lumen or orifice. "Agenesis" will on the other hand denote imperfect or defective development.

The term "Fistula" is retained in connection with anorectal agenesis because of its common usage, although some fistula in this connection should perhaps be more accurately regarded as 'Ectopic rectal or anal openings."

CLASSIFICATION

The current trend in classification, dividing the cases into HIGH MALFORMATIONS and LOW MALFORMATIONS was adopted. Such a classification was originally suggested by Browne [7] and had been modified differently by different authors subsequently. Basically, High malformations referred to cases where the bowel ended or became abnormal, on or above the pelvic floor. On the other hand, in low malformations, the bowel extended through the levator ani muscle and its pubo rectalis sling, before abnormality commenced.

Malformations could affect

(a) the rectum alone
(b) the rectum and the anal canal
or (c) the anal canal alone.

Such a classification would not be in conflict with the embryological origin of the rectum and anal canal, for it is believed that the rectum is derived from the hindgut, the proximal anal canal (as far as the anal valves) from the cloaca, and the distal part of the anal canal from the proctodeum. Table I records the pattern of the abnormalities noted in the 34 cases.

Type of Malformation	Males	Females	
1. *Rectal malformations* (i e. affecting the rectum alone)			
Atresia of rectum	1	—	High Malformations
2. *Anorectal malformations* (i.e. affecting rectum and anal canal)			
Anorectal agenesis without any fistula	2	—	
Anorectal agenesis with fistula			
(a) with recto-vesical fistula	3	—	
(b) with recto-urethral fistula	1	—	
(c) with recto-vaginal fistula			
(i) high fistula (i.e. opening high up in vagina)	—	1	
(ii) low fistula (i.e. opening low down in vagina)	—	1	Low Malformations
3. *Anal malformations* (i.e. affecting anal canal alone)			
Vestibular ectopic anus	—	10	
Perineal ectopic anus	2	1	
Anal agenesis	5	2	
Normally situated anus and anal canal, but with a congenital ano-vestibular fistula	—	1	
Total	14	20	

TABLE 1. 34 cases classified

SURGICAL ANATOMY AND CLINICAL DETAILS

1. RECTAL MALFORMATIONS —

ATRESIA OF THE RECTUM

(Fig. 1)

One male infant was brought on the second day after birth, because no meconium had been passed, although the infant had a normal looking perineum. The anal orifice looked somewhat narrow, but it admitted dilators quite easily. There was obstruction to the passage of dilators beyond 2 inches from the anal orifice. As the significance of the pathology was not fully appreciated at the time of the operation, a perineal operative procedure was undertaken without success. That infant died. It was subsequently realised that the infant had a blind rectal pouch ending in the pelvis, separated from the commencement of the anal canal by a gap. A colo-

stomy, or an abdomino-perineal operation might have been more appropriate.

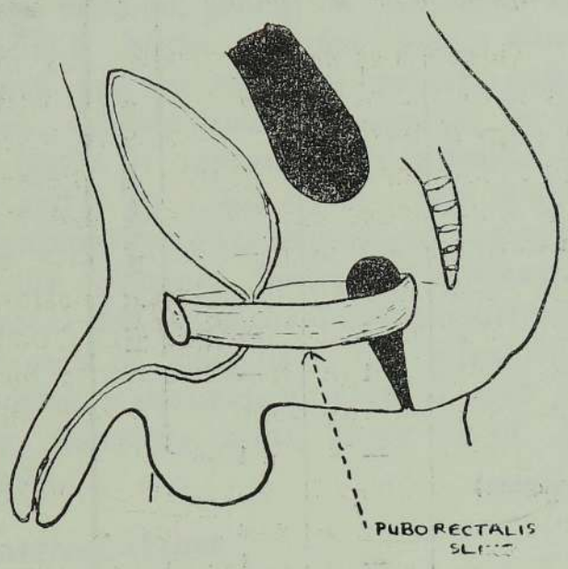

Fig. 1

Under-estimation of the pathology had been made at the time of the operation. This anomaly could have been confused with colonic atresia or Hirschsprung's disease, as the initial signs and symptoms in all three conditions would have been those of lower intestinal obstruction associated with a normal looking perineum.

2. *ANORECTAL MALFORMATIONS*

(1) *ANORECTAL AGENESIS WITHOUT ANY FISTULA. (Fig. 2)*

In this malformation, the anal canal and the lower terminal part of the rectum were absent. The rectum ended as a blind pouch above the pelvic floor. No fistulous communication existed between the blind rectal termination and the uro-genital organs or the exterior.

Six children presented with this variety of malformation. 2 were males and 4 were females. The fact that there was no anus, combined with the total obstruction to rectal contents always resulted in early

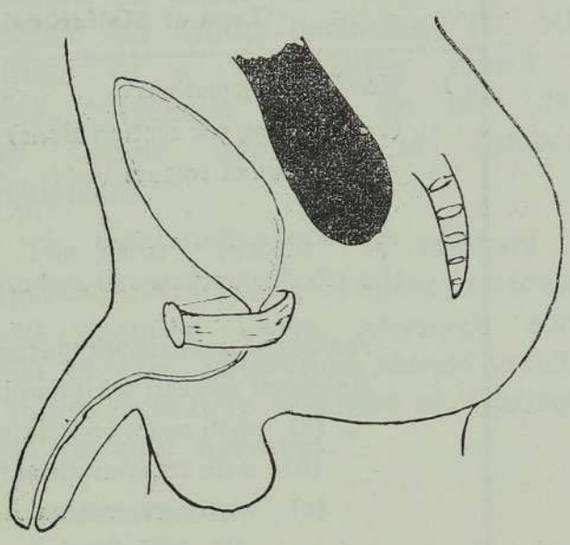

Fig. 2

recognition of the abnormality. Thus, 4 of the 6 children were brought within 24 hours of birth. One came on the second day, while the other came at the age of 3½ years, with a left iliac loop colostomy performed at birth. Perineal proctoplasty was performed on all 6 children. The meconium in 2 infants was pale in colour, and free of bile, indicating other associated malformations, probably in the nature of some alimentary atresia more proximally. Both those neonates died. The third death occured in the neonate seen on the second day after birth; this infant too had other recognizable congenital abnormalities in the form of an extra finger in one hand. 3 of the 6 children stood the perineal proctoplasty well and went home. One of those who survived surgery initially was the 3½ years old child who had a colostomy done at birth. The colostomy was closed 7 weeks after the proctoplasty. The newly constructed anal canal tended to stenose in that child, and the child required anal dilatations under anaesthesia on several occasions until death intervened little over a year later. One infant was lost for follow-up after a few months. The other surviving infant thrived well because her parents

bestowed excessive care and affection on her, but the functional result was not very satisfactory. The child was always constipated owing to colonic inertia, and required frequent dosage of "Senekot" granules. Although the newly constructed anal canal remained patent, digital examination of the rectum always revealed hard solid faecal masses in the rectum. Growth of this child, and her mental development proceeded normally.

2. *ANORECTAL AGENESIS WITH FISTULA*

(a) Anorectal agenesis with recto-vesical fistula. (Fig. 3)

The malformation in this group was essentially the same as in the previous group of anorectal agenesis, but with the exception that the rectal pouch which ended above the pelvic floor communicated with the urinary bladder through a recognizable fistulous opening. The anal canal and the lower terminal part of the rectum were absent.

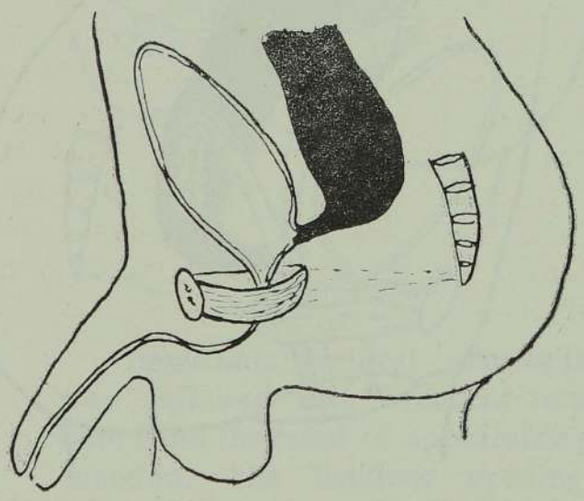

Fig. 3

This anomaly too resulted in the development of symptoms and signs of intestinal obstruction soon after birth, because the fistula seldom proved an adequate vent for the evacuation of the rectal contents although the fistula permitted the passage of some meconium per urethra.

Three males were seen with this malformation. 2 of the 3 were brought within 24 hours of birth. The third was 3 years and 10 months old at the time of presentation in this series, but the child had been taken to hospital on the day of birth and been given a left iliac loop colostomy. Perineal proctoplasty was performed on the two infants brought immediately after birth. The operation was found to be very difficult; although the fistulous tract from the rectum to the bladder was reached without much problem, surgical closure of the fistula on its bladder side proved virtually impossible through the limited access the perineal incision afforded. Urine and meconium welled out through the wound on division of the fistula, and continued to do so, obscuring the fistulous opening in the bladder and making its obliteration difficult. Both infants never passed urine through the urethra after the operation. The urine found easier exit through the newly constructed anus. One of the infants was incontinent post-operatively, and that infant died on the 8th day after operation. The other infant went home, but his parents were never satisfied with the result, as the child continued to pass all its urine through a continent anus. Although the parents tried very hard, they were unsuccessful in persuading the writer to perform any further operation on that child. The third child, who had thrived 3 years and 10 months with a colostomy was handled differently. A formal laparotomy was performed, and the proctoplasty was undertaken through an abdomino-perineal approach. This permitted better visualisation of the fistula between the rectum and the bladder, and more adequate repair of the bladder end of

the fistula after its division. Other associated intra-abdominal malformations were also observed in this child. He had a gall bladder that was deeply buried in the liver, and a well formed Meckel's diverticulum. Post-operative abdominal distension, wound infection and wound disruption occurred, and the child died on the 15th post-operative day.

(b) Anorectal agenesis with recto-urethral fistula. (Fig. 4)

One case in a male infant was encountered. The infant was 4 days old when brought to General Hospital Jaffna. Part of the delay in arrival at the hospital was due to the fact that the infant had been born at Analaitivu, (an island off Jaffna, from where communication and transport are normally difficult).

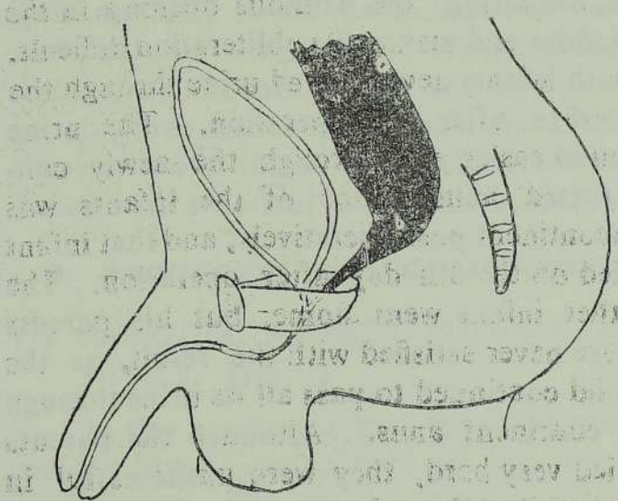

Fig. 4

The blind rectal pouch ended high, but communicated with the prostatic urethra through a narrow fistulous tract. This infant had passed meconium through the urethra, but urine had been observed for the first time only on the third day after birth. A perineal proctoplasty was done, with good results.

(c) Anorectal agenesis with recto-vaginal fistula.

Two cases were met with, one where the fistula opened very high up into the vagina, and the other where the fistulous opening was recognized quite low down in the vaginal canal.

(i) High recto-vaginal fistula. (Fig. 5)

The patient was a 2 year old girl, brought with the complaint that faeces was passed through the vagina from birth. There was no anus. Constipation had been a troublesome feature from birth. Clinical under-estimation of the malformation was followed by an attempt at a perineal repair. As failure to locate the bowel through a perineal incision occured, the abdomen was immediately opened, and an abdomino-perineal operation was performed. Other associated malformations were noticed.

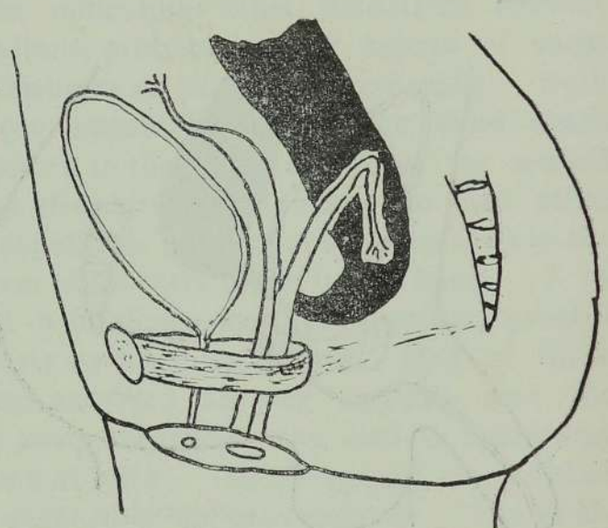

Fig. 5

The uterus was in two separate halves, and each half had its own separate Fallopian tube and ovary. The vagina was also found divided into two by a septum. The recto-vaginal

fistula opened very high up into the vagina. The malformation of the uterus and vagina was due to failure of fusion of the Mullerian ducts, and constituted the deformity called "Uterus didelphys". Post-operative abdominal distension and vomiting proved troublesome, and the child died on the 4th day after operation.

(*ii*) *Low recto-vaginal fistula.* (Fig. 6)

The infant with low recto-vaginal fistula associated with anorectal agenesis was brought on the day of birth, as meconium was appearing through the vagina, with absence of an anus.

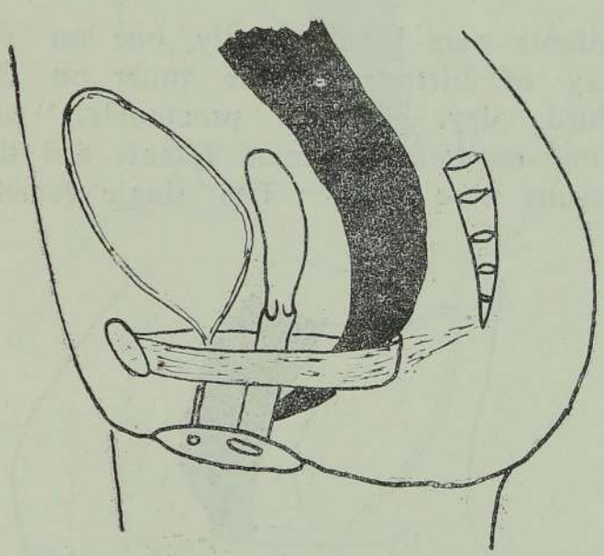

Fig. 6

Immediate perineal proctoplasty was followed with excellent results, both from the point of appearance and function. The fistulous opening in the vagina was probably an ectopic anus.

3. ANAL MALFORMATIONS

(*i*) *Vestibular ectopic anus* (Fig. 7)

This was the commonest variety of anorectal malformation met with in this study, and accounted for 10 out of the 34 cases; it occurred in exactly half the number of 20 females in the series.

With this anomaly, 3 orifices were present in the vestibule, the urethral orifice in front, the vaginal orifice in the middle, and the ectopic anal orifice just behind the vaginal orifice. All 10 children were brought with the complaint that faeces came through the vulva, and that no anus was present. It was surprising that several mothers had not noticed the existence of the abnormality in their daughters until many weeks or months after birth. The vestibular ectopic anal orifice, although of very small calibre, was adequate for evacuation of the rectal were contents, and no symptoms or signs of obstruction was noted. Further more, all these infants were fully continent.

It was not surprising therefore, that only one of the 10 children born with this malformation presented for surgery soon after birth. That solitary exception was referred for surgery on the second day after birth, because the infant had been born in the same hospital. One

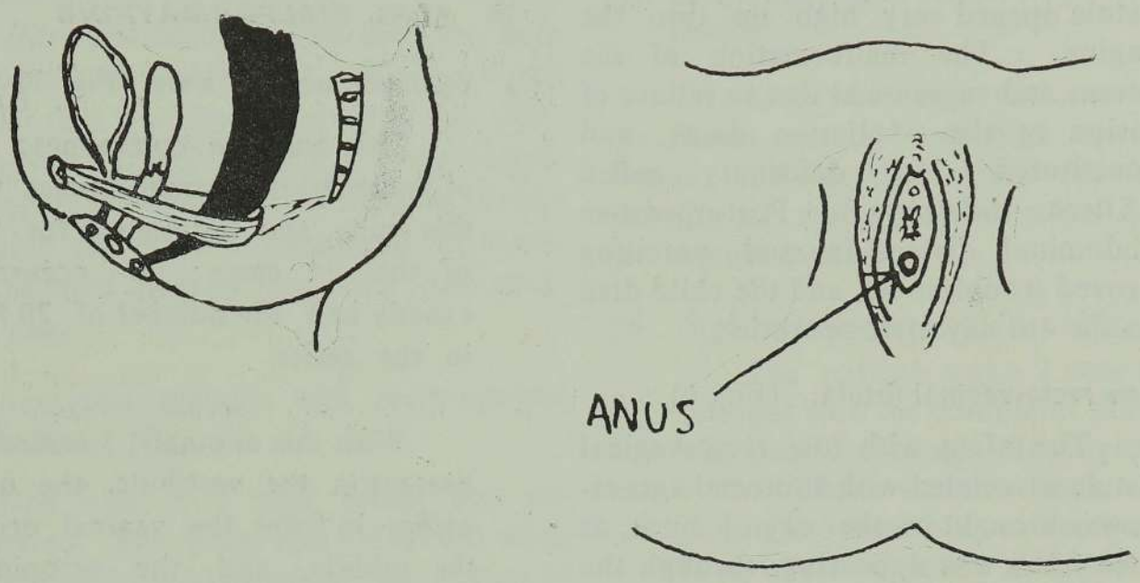

Fig. 7

infant was brought during the third month, on the very day the mother noticed the anomaly for the first time. 3 children were brought in the 5th month, 1 in the 7th month, and 2 in the 9th month. The remaining 2 children were brought when they were 4 years old. In one case, the mother noticed the anomaly for the first time only when the girl was 4 months old. Many of these children had a suggestion of a dimple at the site where the normal anus should have been found.

Perineal proctoplasty was undertaken in every case; the ectopic anus was enlarged and transplanted to the normal site. The result was good in every single case.

(2) Perineal ectopic anus.

Three infants were seen with this malformation. The ectopic anus was situated in the perineum, but at a point further forwards than where it would have been considered normal. 2 were males, and had the ectopic anal orifice just behind the scrotum. Both these infants were brouhgt early, one on the day of birth, and the other on the third day. Perineal proctoplasty was done on both the male infants and the results were good. The single female

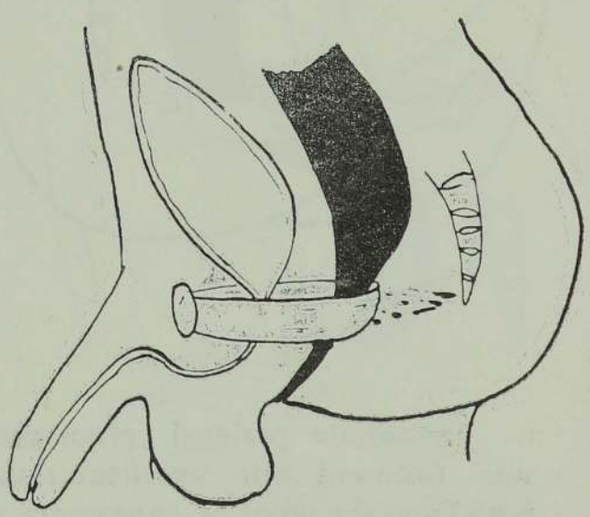

Fig. 8

infant was 2 months old when brought for surgery. She had a continent ectopic anus, but perineal proctoplasty transplanting the anus to the normal site resulted in post-operative incontinence. This child might have been better off had she been left alone, with the malformation, although

the close proximity of the ectopic anus to the vulva had been unacceptable to the parents.

(3) *Anal agenesis.*

This was the second most common malformation in this series. The rectum descended through the levator ani muscle, but ended blindly, without any ectopic opening or fistula. There was however a reasonable distance between the termination of the blind rectal pouch and the perineal skin.

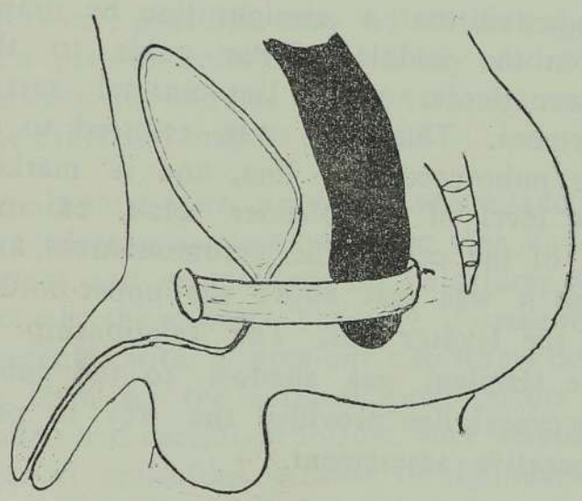

Fig. 9

The nature of the malformation was such that immediate recognition was possible. No meconium was passed, and anus was absent. Of the 7 infants with this malformation, 5 arrived for surgery within 24 hours of birth. The other 2 infants came on the 3rd day. Males accounted for 5 infants, and females for 2. One of the female infants had associated malformation recognizable as a prominently bulging, imperforate hymen, which on incision yielded creamy whitish liquid.

Perineal proctoplasty was done on all 7 infants. Both female infants died. It was presumed that one female (who had the imperforate hymen referred to) probably had other malformations also. The other infant who died was one that was brought in a poor state on the 3rd day after birth, with gross abdominal distension, dehydration, and vomiting, due to alimentary obstruction caused by the malformation. The results of the 5 infants who survived were good.

It was interesting to note that in this group, one of the males was one of twins; the other twin infant was normal.

(4) *Bizarre malformation*
(Fig. 10)

This was a freak abnormality, as the child presented with 4 orifices in the perineum. The female child had a normal urethral orifice, a normal vagina, and a normal anal orifice. The 4th orifice was the abnormal one, situated in the vestibule, just behind the vaginal opening.

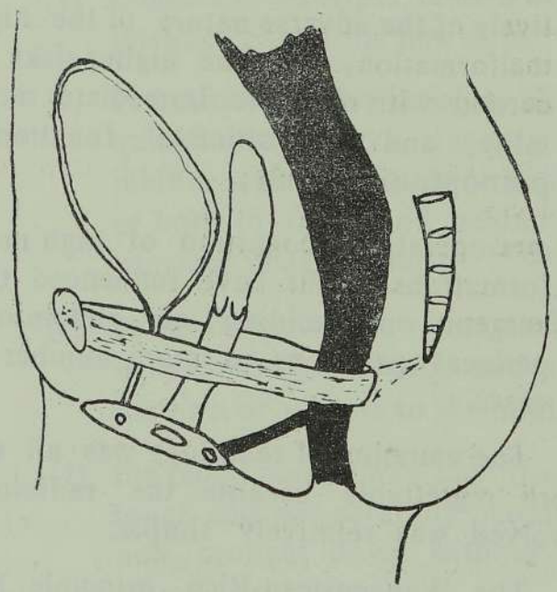

Fig. 10

It had intermittently discharged faecal matter, although the bulk of the faeces found its exit through the normal anus. The infant was 2 months old when brought for surgical consultation. At operation, on the anal side, the fistulous opening was found to be rather high. The impression was that the fistula tract passed above the anal sphincters. Formal fistulectomy in 2 stages was undertaken, and the result was good.

Later on, while this paper was in preparation, another infant with this same malformation presented and was treated, but that second case was not included in this series.

PRE-OPERATIVE RADIOLOGICAL DIAGNOSIS

In this series, no pre-operative radiological aid to diagnosis was resorted to. This omission was a handicap for two reasons, namely,

(a) radiological recognition of high malformations would have been possible; the parents of the malformed child could have been informed pre operatively of the adverse nature of the high malformation, and the higher risks it carried with regard to immediate mortality, and the ultimate functional post-operative result;

(b) pre-operative recognition of high malformations might have influenced the surgeon on deciding on abdomino-perineal operations in more number of cases.

The omission of radiology was all the more regrettable because the radiology involved was relatively simple.

The Wangensteen-Rice principle has been known for many years, and is probably routinely used in most centres. It involves taking radiographs of an inverted infant, a radio-opaque marker having been previously placed at the site where the anus should be present normally. If sufficient time had been allowed after birth of the infant, the gas filled terminal bowel outline showed up, and the distance between the bowel termination and the marker was estimated or measured. Stephens [8] suggested that true lateral radiographs be taken of the inverted infants, and the relationship of the terminal gas shadow to the bony pelvic landmarks be compared. He suggested that a straight line be drawn from the middle of the pubis to the lower border of the last normal sacral vertebra. This line was referred to as the pubo-coccygeal line, and it marked the level of the bladder neck, external os of the cervix, the verumontanum, and that it was just above the upper border of the levator ani. The relationship of the terminal gas shadow to the pubo-coccygeal line provided the key to pre-operative assessment.

TREATMENT

The aim of treatment in anorectal malformations should be to provide the child with a properly functioning anus of normal calibre, and situated at the normal site. This sometimes becomes an unattainable objective, particularly in high malformations, but no effort should be spared in trying to reach this high objective. Proper diagnosis, appropriate surgical procedure, and careful and prolonged aftercare (particularly directed towards prevention of stenosis, and towards establishment of proper bowel function) are of importance.

It would appear that the earlier the treatment is commenced, the better would

be the ultimate result. Where doubt exists, preliminary colostomy may be lifesaving, and certainly preferable to unenlightened or injudicious surgical exploration of the perineum. If exploration of the perineum is commenced, it is recommended that the bowel should be brought down and an anus fashioned at that same instance; if difficulty is encountered, one should not hesitate to achieve this objective through an abdomino-perineal approach to the problem. Extreme gentleness is of great importance, when perineal tissues are dissected or handled. Sometimes, the end result of treatment, despite every precaution and every care possible, may be nothing short of a 'perineal colostomy.' Every gradation between this extreme and a normal anus at the other end may be expected.

In this series, a primary reconstructive procedure as a single operation was undertaken, and no preliminary colostomy was done by the writer. The only 2 cases who presented with a previous colostomy done at birth by the surgical predecessors at Jaffna had special problems, and imposed special difficulties because of high malformations. They failed to impress on the author that a preliminary colostomy was an advantage in the subsequent definitive reconstructive procedure. However, they served to emphasize very clearly that colostomy can be a life saving step. Both children with preliminary colostomy had passed their 3rd birthdays when they presented for definitive surgery. Except for the malformation and the colostomy, (both of which could have been easily concealed by suitable clothes), the children had developed physically and mentally in a perfectly normal way. Following reconstructive procedure, one died soon after operation and the other died a little after a year following surgery. The writer's firm conviction is that if a colostomy is done as a preliminary procedure, the definitive reconstructive operation should not be delayed very long, whatever the problem or difficulty may be. An infant should not be permitted to grow into childhood with a colostomy, if it could be avoided.

Whether immediate reconstruction should be attempted, or whether preliminary colostomy followed at a later date by the reconstructive procedure is better, can be the subject of a debate. In fact, this has been debated in in literature, several times in the past. In practice, it might be correct to say that the decision very often depends not on validity of reasoning, but mainly on the personal preferences or prejudices of the individual surgeon handling the case. The writer has very strong prejudices against a colostomy, and this probably was the reason why he performed no colostomy in this series.

Perineal proctoplasty which was done in 32 of the 34 cases, is no new operation. The important steps were :

(1) excision of an elipse of skin at the intended site of the new anus ;

(2) extension, of this incision, if required was always done in the midline, either in front, or behind, or both in front and behind the previous anal incision ;

(3) perineal dissection was carried out with utmost gentleness, and in the midline, or as close to the midline ;

(4) the rectal pouch was identified by touch and was carefully mobilised and brought down without tension ;

(5) any fistula was divided and ligated or sutured;

(6) The rectal pouch was opened and sutured to the anal skin incision with interrupted catgut sutures;

and (7) attention to haemostasis was meticulous.

Where appropriate, modifications of surgical procedure were incorporated, depending on the particular needs of the situation met with. Post-operatively, the perineum was kept scrupulously clean. Anal dilatation with the little finger was commenced from about the 10th-14th day after operation. The infants were allowed home as soon as it was felt that the mother had learned to dilate the anal orifice.

RESULTS AFTER SURGICAL TREATMENT

1. EARLY POST-OPERATIVE DEATHS

9 of the 34 children died soon after operation. Of the 9 deaths, 7 occurred among the 12 Cases with high malformations, while only 2 deaths took place among the 22 children with low malformations. (Table 2).

Type of malformation	No. treated	Deaths
HIGH MALFORMATIONS		
Atresia of rectum	1	1
Anorectal agenesis without fistula	6	3
Anorectal agenesis with recto-vesical fistula	3	2
Anorectal agenesis with recto-urethral fistula	1	—
Anorectal agenesis with high recto-vaginal fistula	1	1
Total	12	7
LOW MALFORMATIONS		
Anorectal agenesis with low recto-vaginal fistula	1	—
Vestibular ectopic anus	10	—
Perineal ectopie anus	3	—
Anal agenesis	7	2
Bizzare malformation with ano-vestibular fistula normal anal orifice	1	—
Total	22	2

Table 2—Mortality soon after surgery

6 of the 9 children who died soon after surgery had other recognizable associated congenital malformations. In this series, no special search or investigation was done to screen the infants for the coexistence of other malformations. But in 6 instances, existence of other malformation was recognized or inferred during the course of the management. It is perhaps very significant that all 6 children on whom existence

of other malformations were easily observed or inferred, died in the early post-operative period.

It appeared therefore that

(1) high malformations definitely carried a bad prognosis

and (2) recognition of other associated abnormalities also pointed to poorer prospects of survival after surgery.

If surgery had not been offered, many of the infants who survived would also have died, although there was no doubt that at least some infants, particularly those with an ectopic anus of adequate calibre, could have lived without any surgery, at least for a number of years.

2. LATE POST-OPERATIVE DEATHS

Of the group of 6 children who came into the category of having anorectal agenesis without any fistula, 3 had died soon after operation, and 3 had gone home alive. One of the infants who went home alive was lost for follow up after a few months, and it was presumed that that infant died. Another child in the same group, the 3½ year old child who presented with a colostomy done at birth, required repeated dilatation for stenosis of the newly constructed anus. That child died little over a year after the reconstructive surgery.

Both late failures also came from the group with high malformations, noted earlier to carry a bad prognosis.

3. UNSATISFACTORY FUNCTIONAL RESULTS

Among the survivors from surgery, four varieties of unsatisfactory functional results were encountered, namely

(i) stenosis or narrowing of the newly constructed anus and perhaps the terminal segment of the bowel,

(ii) defective control of normal defaecation, and intractable constipation, associated with colonic inertia,

(iii) post-operative anal incontinence,

and (iv) persistence or recurrence of recto-vesical fistula, leading to permanent voiding of urine through the anus.

Four children developed stenosis which necessitated dilatation under anaesthesia on more than one occasion. The other 3 problems were encountered in one case each.

Although these 4 varieties of complications have been introduced as unsatisfactory functional results after surgery, there were definite structural or organic causes for each of these disappointment. These are discussed in greater detail below.

(i) STENOSIS.

It was possible to recognize three groups of predisposing causes for post-operative anal stenosis, on the basis of whether the predisposing factors were present before the operation, came into play during the operation, or influenced the result entirely by becoming significant in the post-operative stage. Sometimes, more than one cause could have been present.

(a) Adverse pre-operative factors.

The most important adverse pre-operative factor was the presence of a high malformation. Since the bowel became abnormal at a high level, reconstructive surgery involved

more perineal dissection, and more mobilisation of the terminal bowel to permit pulling down the bowel to the site of the surgically constructed anus.

Greater tension on the suture line at the new anus, greater tension distributed over the terminal bowel and its blood vessels, greater likelihood to post-operative haematoma formation, and greater extent of scar tissue formation and fibrosis, became unavoidable hazards when high malformations were dealt with.

(b) Adverse intra-operative factors.

Haste, inadequate mobilisation of the terminal bowel, lack of meticulous attention to arrest of bleeding even from small vessels, and surgical technique permitting excessive tension, on the suture line at the new anus predisposed to stenosis.

(c) Post-operative factors.

Partial, or complete breakdown of the suture line at the new anus inevitably resulted in stenosis. Excessive tension, haematoma formation, infection, and lack of proper perineal toilet were adverse post-operative factors.

To prevent stenosis, and keep the new anal canal patent, the mother was trained to pass a finger up the infant's anal canal, several times daily, after lubricating the finger. The little finger was used initially, and gradually, other fingers were brought to use as anal dilators. Mothers were advised to carry on this daily manoeuvre on the child for 6—8 months, or even longer. The importance of this unexciting routine was lost on some mothers, and default predisposed to stenosis.

Post-operative stenosis was therefore partly preventable, although sometimes one had to accept it as an inevitable hazard.

(*ii*) *DEFECTIVE CONTROL OF DEFAECATION*

Anal or rectal continence is the ability to retain or hold the bowel contents untill evacuation of the contents becomes convenient. This implies the ability to retain solid, liquid and gaseous contents in the rectum, and the differential control of their exit. The control depends on a reflex that works under cerebral cortical control. In normal circumstances, the call to evacuate can be suppressed whenever necessary, by cerebral control. The ability to differentiate between faeces and flatus is also a process which involves consciousness.

For anal continence therefore, a sensory receptor area, uninterrupted nervous pathways, and a motor component capable of controlling impulses to defaecate, are essential. In the absence of these vital components normal anal continence is impossible.

The principal sensory receptor area for defaecation reflexes lies in the terminal part of the rectum, and the upper portions of the anal canal. In anorectal agenesis, this principal sensory receptor area is congenitally absent, or maldeveloped. The higher the malformation, the greater will be the degree of maldevelopment or absence of the sensory receptor area. None of the more proximal parts of the bowel can substitute for the absent sensory receptor area.

The nervous pathways for the reflexes may also be underdeveloped, due to associated sacro-coccygeal spinal malformations, which often coexist in some degree with

anorectal anomalies. This would further impair continence or control.

The motor component for defaecation control is provided by the combination of the sphincters and the pubo-rectalis component of the levator ani muscle. These components may also be deficient or malformed in anorectal malformation.

Thus, the higher the malformation, or the greater the degree of agenesis, the greater will be the deficiency in the components of the reflex are for control of defaecation. The importance of the anorectal ring consisting of the pubo-rectalis sling and the deeper portions of the external sphincter, has been stressed by several authors, who believed that this ring was capable of maintaining continence, even if the rest of the external sphincter was divided. With high malformations, the pubo-rectalis sling surrounds the urethra or vagina, but does not encircle the rectum. In repair, if the bowel is not brought down in the correct position in relation to the limbs of the pubo-rectalis sling, the pubo-rectalis sling cannot act in contributing to continence.

Further, quite apart from serious developmental deficiencies in the components necessary for proper control of defaecation, one should also accept the possibility of injury to whatever rudimentary vestiges there may be, of these components, during the surgical dissection which goes with repair.

Following proctoplasty, therefore, defective control may take the form of either incontinence, (which is easily understandable), or constipation because the defaecation reflexes do not work owing to lack of the necessary components for control of that function. When defaecation reflexes do not work, faecal retention in the rectum and colon result. This is followed by colonic inertia, and sometimes paradoxically, by a variety of overflow incontinence.

(*iii*) *RECURRENCE OF RECTO-VESICAL FISTULA*

It is suggested that the recurrence of recto-vesical fistula in this series was due to more than one factor. Technical difficulty in closure of the bladder end of the fistula, through a perineal approach has already been admitted. The importance of drainage of the bladder after the operation was perhaps underestimated. The bladder contents at the time of operation would have been urine and meconium. The meconium, being of fairly thick consistency, would have porduced some blocking of the narrow urethra in the infants. Retention of urine probably followed. With the resultant distension of the bladder, it was not surprising that the bladder end of the fistula reopened, and thereafter served as a wider and easier exit for urine than the urethra.

On the basis of experience gained, it is suggested that where a recto-vesical fistula is encountered and divided, that post-operative decompression of the bladder be attained and maintained for at least a week through a supra-pubic catheter, at least until time is afforded for the severed recto-vesical fistula to close up by healing.

DISCUSSION

In the management of children with anorectal malformations, a particular policy of treatment which emphasized early commencement of treatment, definitive surgery in a single stage attempting at total reconstruction as a primary procedure, and a bias towards a perineal operative approach was preferred. It is not disputed that other lines of treatment involving preliminary colostomy, a two or multiple staged procedure in correction and an abdomino-

perineal approach can produce equally good or even better results. Discussion on management, although it be a contemporary problem, is also surgical history now, because most of the arguments have had their eloquent proponents in the past.

The unusually high incidence of this malformation met with in the 34 months, in a provincial general surgical practice in Ceylon is worthy of investigation. Having been engaged in a fairly comprehensive provincial general surgical practice for 12 years, in several different parts of Ceylon (owing to the policy of transfers of government medical officers), the writer is more than convinced that the experience encountered during the 34 months under review is something exceptional. The malformation was encountered with great frequency in 1969, and then with reduced frequency in 1970. At the time of preparation of this paper, the impression gained is that the frequency is further dropping, which if correct is fortunate.

Statistics on incidence of malformations in Ceylon is sadly lacking. In the absence of statistics, it would be very difficult to recognize an epidemic of congenital malformations that may suddenly occur in the community or country. It is suggested that at least statistics of visible or gross congenital malformations (like cleft lip or palate, club feet, anorectal malformations, etc.) among infants born in the larger hospitals (Hospitals for Women in Colombo, Provincial Hospitals and Base Hospitals) be maintained by a central agency like the Department of Health, so that significance of fluctuations in the incidence of congenital malformations may be observed and studied. The statistics may prove to be of epidemiological significance.

If really an epidemic of anorectal malformations did actually occur in 1968, 1969 and the early part of 1970, it would be interesting to note whether it was confined to Jaffna or the Northern Province of Ceylon alone, or whether such an unfortunate rise in incidence was shared by the rest of Ceylon too. The lesson of the Thalidomide tragedy should serve to alert any community to take serious notice of unexpected or unexplained rise in incidence of this or any other congenital defect. An epidemiological study should be undertaken if there is even a suspicion that an epidemic has occurred.

If no epidemic has occurred, then could it be that Jaffna is unfortunate in having a higher incidence of this malformation than other parts of Ceylon, and other countries? Could it be some environmental factor in Jaffna, or is it the genetic constitution of the people in Jaffna that renders them more susceptible to this abnormality? It is a well accepted practice over the generations, for the Jaffna man to seek his bride from near relatives: he seldom had to look very far in search for a partner, who came either from his relatives, or at least from the same or adjacent village. Perhaps the resultant relative inbreeding over the generations has left the Jaffna community in a more vulnerable position than people living in other parts of Ceylon.

During the period under study, apart from the two children who came with colostomy performed at birth, and a few children who had anal stenosis resulting from earlier surgery, no cases of older children or young adults who had a history of anoreetal malformation treated by surgeons who worked in Jaffna prior to the author was noted. This could mean 3 possibilities, namely,

(1) this abnormality did not occur earlier, a theoretical possibility, but certainly most unlikely,

(2) that all cases treated by earlier surgeons had excellent results, and that they were rendered virtually normal, so that they were not observed as any different from the healthy normal population,

or (3) that all patients treated earlier did not survive past early childhood.

Perhaps, the incidence was definitely lower in earlier periods, and suggestions contained in the second and third possibilities also partly took place, some children being virtually rendered perfectly normal, while the others failed to survive beyond childhood, not entirely due to surgical reasons, but due to associated malformations in cardiovascular, urinary, or nervous systems, associated anomalies that became clinically manifested at a later age group. Therefore one wonders how many of the surviving children from this series will grow to be adults.

SUMMARY

34 cases of congenital anorectal malformations personally treated in Jaffna during the 34 months period from January 1968 to the end of October 1970, are reported. It was observed that the incidence of this malformation in Jaffna during the period under study was in the region of about in 800 to 1,000 births. This was a much higher incidence than in other reported series. Current trends in classification into high malformations and low malformations was adopted. High malformations carried a bad prognosis. Presence of other associated malformations in other organs also seemed to have an adverse effect on the prognosis. This was in conformance with observations of other workers too. One of the 34 malformed children was one of twins, the other twin partner being unaffected as far as malformations were concerned. The trend in management was for early surgery, as a single procedure aimed at a primary total reconstruction of the malformation. There was a definite bias towards a perineal operation in the correction of the defects. The results of treatment are briefly discussed. Possibilities for the high incidence of this malformation in Jaffna, during the period under study, are discussed.

ACKNOWLEDGEMENTS

The opportunity afforded for this study to have been the basis of a short lecture on 12th November, 1970, at the Joint Scientific Sessions of the Ceylon Medical Association and the Jaffna Medical Association is appreciated with gratitude. Several members of the staff of General Hospital, Jaffna, Green Hospital at Manipay, and McLeod Hospital at Inuvil rendered valuable assistance in the management and care of the 34 malformed patients. The excellent paediatric anaesthesia administered skillfully by the anaesthetists at consultant and junior levels contributed in no small measure to the confidence with which major primary total reconstructive procedure was undertaken.

The debt the author owes to the 34 children he was privileged to study, (11 of whom are no longer alive now), and for the lessons he learned from their deformities, can never be adequately accounted. The author also acknowledges that he borrowed freely from the work of other authorities from different parts of the world, in the management and presentation of the complex problem he encountered in Jaffna.

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HYPEREMESIS GRAVIDARUM

BY

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History

THE association of vomiting with pregnancy was noted by man thousands of years ago and a clear description is found in a Papyrus dated about 2000 B. C. – a part of Petrie collection. [1] Hippocrates has made reference to 'Maux de coeur' in reference to early pregnancy.

Soranus an Ephesian by birth who practised medicine in Rome between 98 and 138 A. D. has described in his book on gynaecology how sickness of pregnancy sets in around the fortieth day and persists for about four months. He also mentions that some women may have excessive vomiting and how this can lead to pain in the thorax, gastric distension, fever and jaundice. It is obvious from his description, that he had recognised the occurence of jaundice in severe hyperemesis.

Even though many others described hyperemesis, it was Paul Du Bois[2] in 1852, who gave the first classical description of pernicious vomiting of pregnancy. Even in non-medical literature a classic description of hyperemesis in and the fatal termination of Charlotte Bronte can be found in Mrs. Gaskell's book[3] on Life of Charlotte Bronte published in 1858.

Definition

It is very difficult indeed to give a satisfactory definition of hyperemesis gravidarum. But it is at the same time very important from the point of view of treatment to diagnose this condition. So when does the vomiting of pregnancy become serious enough to warrant the label of hyperemesis ?

The American Council on Pharmacy and Chemistry [4] in 1956 accepted that the term hyperemesis gravidarum or pernicious vomiting of pregnancy is considered to apply only to the condition occuring in a few patients who develop intractable vomiting, disturbed nutrition such as alteration of electrolyte balance, loss of weight of 5% or more, ketosis, and acetonuria with ultimate neurological disturbance, liver damage retinal haemorrhage and renal damage.

Aetiology

It is said that ancient Greeks believed that pregnancy occured through the mouth and that vomiting is evidence of a psychological conflict—a rejection tendency or distorted attempt of the unconscious mind to get rid of the embryo. Hyperemesis seems to complicate only the pregnancies of human beings and attempts to produce similar symptoms in animals have been unsuccessful [5] Even veterinary surgeons and farmers find no comparable vomiting in pregnant animals. [6]

The exact aetiology of this condition is still unknown and it is not surprising that it is described as a disease of theories. After many years of conflict between theories, gradually they became to be classified under three main groups namely

1. Toxaemic
2. Reflex and
3. Neurotic

Even this classification, is of historical interest as there is no proof of any toxins or reflex factors causing hyperemesis. But there is little doubt that a considerable neurotic element or better termed psychosomatic element, is present as an aggravating factor in a large proportion of cases of hyperemesis.

Further certain endocrine factors are worthy of consideration. Holloday [7] suggested that the disease was related to the action of a secretion from an abnormally persistent corpus luteum. Finch [8] postulated that the nausea and vomiting accompanying pregnancy were due to an allergic sensitivity to the secretion of corpus luteum of pregnancy. He based his theory on the fact that the onset of vomiting coincided with the development of the corpus luteum in early pregnancy and that the symptoms usually disappeared at about the time of retrogression of corpus luteum. This theory has not been confirmed.

Brindeau, Hinglais [9] in 1937 reported that the titre of gonadotrophic hormone (Prolan B) in the blood of patients with hyperemesis was above normal and that the level was directly proportional to the severity of symptoms. This report gave food for thought and many others later observed a high incidence of hyperemesis in patients with hydatidiform mole and in cases of multiple pregnancies [10].

Schoeneck [11] produced evidence to show that there was increased concentration of chorionic gonadotrophic hormones in the serum and urine of pregnant women who suffered from nausea and vomiting as compared to those who were free of symptoms. He also pointed out that the greatest concentration of chorionic gonadotrophins (H. C. G.) was found between six and twelve weeks of pregnancy — the period when hyperemesis is most commonly found. Bandstrup [12] on the other hand was critical of the above observation as he in his investigations did not find any correlation between the levels of chorionic gonadotrophins and the occurrence of hyperemesis.

In the light of such conflicting evidence Fairweather and Loraine [13] undertook an extensive study to assess the part played by chorionic gonadotrophins in the causation of hyperemesis. They thought that there could be two possibilities.

1. That there could be an excessive amount of H. C. G. actually present in such patients, or
2. That these patients may be unduly sensitive to the normal levels of H. C. G. thus precipitating the symptoms.

In favour of the first was of course the observation of some, that hyperemesis was commoner in cases of hydatidiform mole and multiple pregnancies and both conditions are thought to be associated with higher than normal levels of H. C. G. But at the end of their study Fairweather and Loraine found no evidence to support the suggestions that either higher levels of H. C. G. or undue sensitivity to normal levels can in any way be incriminated as aetiological factor in hyperemesis gravidarum. In fact they found that from the seventh to the fifteenth week of pregnancy the mean chorionic gonadotrophin levels in hyperemesis patients were significantly lower than in normally pregnant women. From the fifteenth to the twentieth week of pregnancy a significant difference between the two groups was not observed.

As early as in 1932 Kemp [14] suggested that relative adrenal cortical insufficiency was an important aetiological factor in hyperemesis. Wells [15] supported this suggestion and Carreras and others [16]

used Adrenocorticotrophic hormone in the treatment of hyperemesis. They and many others appear to have been satisfied with the results of this treatment. As recently as in 1952 Jarvinen [17] and others presented some further evidence of adrenal dysfunctions in hyperemesis gravidarum, but this has not been widely accepted.

Coming back to the psychosomatic factors in hyperemesis there is little doubt that these factors must be implicated in the majority of cases. Fairweather who is considered to be an authority on this subject of hyperemesis is of opinion that the above is true in 75 to 80% of all cases of hyperemesis. As evidence in favour of this, one can confidently show the uniform success with treatment by psychotherapy mainly suggestive encouragement.

Thus it may be said that hyperemesis is not a disorder, entirely and always of emotional origin, nor is it an organic illness pure and simple. Severe and continued vomiting in early months of pregnancy is perhaps best regarded an instance of viscious spiral reaction.

Epidemiology

Influence of parity

It has been generally stated that parity has no influence on the incidence of hyperemesis. Fairweather [18] in an analysis of materials collected from various centres in England and Scotland observed that the incidence did appear to be higher in the first pregnancies in the white population but in the non-white population there was no significant difference between the first and the later pregnancies.

Multiple pregnancies and hyperemesis

It has long been suggested that there is a higher incidence of hyperemesis in multiple pregnancies. Even though Bender [19] in a review of 472 consecutive twin pregnancies stated that there was no greater incidence of hyperemesis in twin pregnancies as compared to single pregnancies, Fairweather [20] in his recent study found a highly significant relationship between twin pregnancies and hyperemesis.

Illegitimacy and prenuptial conception and hyperemesis

Judging from the fact that psychogenic factors are clearly linked with hyperemesis gravidarum one would expect that a girl with an illegitimate pregnancy would be a good candidate for hyperemesis. But available evidence shows that there is no correlation between hyperemesis and both illegitimate pregnancy and prenuptial conception Fairweather. Sinnathamby in his book on midwifery [21] published in 1969 states that there is no increased incidence of hyperemesis in unmarried mothers in Ceylon. But previous history of hyperemesis has some relationship to the condition occurring in subsequent pregnancies. Fairweather found that 26.6% of patients in the second or subsequent pregnancies admitted to hospital because of hyperemesis gave a history of hyperemesis in a previous pregnancy. Another significant finding was that in 41·5% of hyperemesis patients there was a history of previous unsuccessful pregnancy. He also found that these patients did not show any greater incidence of toxaemia developing in them.

Outcome of pregnancy in hyperemesis

It is very interesting to find that Medalie [22] observed the association of high abortion rate and absence of nausea and vomiting in the first trimester. Brandes [23] also found increased incidence of abortion, neonatal and perinatal mortality in mothers who did not have any vomiting in the first trimester. Fairweather reports

that in his series hyperemesis patients did not have any increased incidence of spontaneous abortion or still birth or neonatal death. Further there is no evidence that hyperemesis per se caused any increased risk of congenital malformations of the child or that it is likely to produce an underweight baby.

Incidence

There is no doubt that there is marked variation in the incidence of hyperemesis in different countries. Diekman [24] suggested that the incidence was related to western civilization stating that hyperemesis occurred more commonly in the west than in the African tribes or Eskimos. In British centres Fairweather found an incidence varying from 0.54/1000 to 10/1000 live births. A report from Hawaii gives the incidence to be 2.03/1000 live births and a centre in Tokyo quotes a figure of 1.36 per 1000 live births. Even though reliable statistics are not available in Ceylon the senior obstericians of this Country say that the incidence is definitely much lower now than about 15 to 20 years ago. The report for the year 1965/66 of the Department of Obstetrics & Gynaecology of the Peradeniya Medical School gives the incidence in the Central Province of Ceylon to be 6/1000 live births.

Clinical features

Hyperemesis gravidarum usually begins as ordinary morning sickness and this gradually becomes more frequent and severe. Repeated vomiting begins to interfere with the patient's normal activity and her ability to take sufficient fluid food. She may struggle along for some time in this state not ill enough to go to hospital but not really well enough to carry on her household duties, until either the vomiting gradually subsides or the condition deteriorates and she becomes really ill. Serious deterioration may occur quite suddenly.

The vomiting may be frequent and continue throughout the day and the vomitus may have a 'Coffee Grounds' appearance due to small haemorrhages that occur from the gastric mucosa. The vomiting induces changes due to dehydration, chloride, sodium and potassium loss, starvation, carbohydrate depletion together with vitamin deficiency. The patient loses weight chiefly due to dehydration and partly due to starvation in chronic cases. The appearance of emaciation with hollow cheeks, sunken eyes and coated dry tongue is due to fluid loss. The pulse rate rises to about 100 per minute and there may be fall of blood pressure The urine output falls and it contains acetone, diacetic acid and in some cases albumin and bile, but little or no chloride. Signs of circulatory failure and disorders of central nervous system should be looked for. Continuous retching may cause epigastric tenderness. In modern obstetric practice one rarely sees very severe cases who develop mental changes, nystagmus, occular palsies or retinal haemorrhages. Jaundice which was said to occur in terminal cases of hyperemesis is very rare indeed and this, if occurs may be due to other causes such as infective hepatitis of drugs.

Diagnosis

Diagnosis is fairly straight forward even though every textbook and article on this subject emphasises that this should be differentiated from associated conditions such as appendicitis, pyelonephritis, intestinal obstruction, hiatus hernia, achalasia cardia etc. It is of course worthwhile keeping in mind that hyperemesis is sometimes the most outstanding symptom in hydatidiform mole and in multiple pregnancy with acute hydramnios.

Metabolic changes.

Vomiting leads to loss of fluid, chloride and to a less extent sodium and acid. The dehydration thus produced is further aggravated by lack of intake and obligatory loss through the breath, perspiration and urine. It is very important not to forget that potassium is also lost in large amounts in gastric vomitus.[25] Starvation leads to ketosis, and breakdown of body proteins with resultant elevation of blood urea. Vitamin difficiency especially of B^1 also occurs.

Treatment

Can hyperemesis gravidarum be prevented? There is reasonably sufficient evidence that the incidence can be reduced by selecting patients from those having morning sickness and interviewing them frequently, explaining to them the physiology of pregnancy, the mechanism of labour and reassuring them thus giving them enough confidence in themselves and in the doctor. It is also worthwhile discussing with her, her problems if any personal, household or otherwise. As nobody else, the obstetrician stands in a very close relationship to his patients. In the same way the pregnant woman can reveal the body to her doctor, she ought to be able to reveal her emotions to him. And the obstetrician should be prepared to spend a lot of time with individual patients. Even though the help of a psychiatrist may be of tremendous value in a few carefully selected cases he should not be called in on the mere suspicion of 'psychic factors' as this is often misinterpreted and misunderstood by the patient.

The management of the patient with hyperemesis depends on the degree of vomiting and the resultant dehydration electrolyte imbalance and ketosis. Broadly it should be on the following lines.

1. Once hyperemesis diagnosed it is essential for the patient to be hospitalised. The change of environment thus produced has a remarkable effect on the patient's morale and attitude.
2. Reassurance by the attending doctor.
3. Correction of dehydration and electrolyte imbalance.
4. Supply of carbohydrate in an easily assimilable form such as glucose or lactose to enable the liver to regenerate quickly and to correct acidosis
5. Sedation to keep the patient calm and tranquill.
6. Combat vitamin deficiency by administration of vitamins especially B_1, parenterally.

Recording and investigations

1. Routine recording of the pulse rate, temperature and blood pressure are essential.
2. Fluid balance chart is very important.
3. Patient should be weighed on admission and then daily.
4. Estimation of serum electrolyte levels blood urea and haematocrit should be done.
5. Urine should be examined for specific gravity, presence of albumin, acetone, chlorides and bile. Microscopic and bacteriological examination of the urine also should be done.
6. Recently Nuemayer and Stark[26] made assays of Aspartate amino transferase (S. G. O. T.), Alanine Aminotransferase (S. G. P. T.) Bromsulphthalein excretion (B.S.P.), serum bilirubin, thymol turbidity, haematocrit, red cell count, pH,

buffer base, pCO_2 and also assays of urinary acetone, diacetic acid, urobilinogen etc. But they did not find any metabolic disorder nor were they able to correlate any of these with the severity of hyperemesis. This shows that such investigations are unnecessary.

Vomiting usually subsides with the above regime. If it does not, then the help of an anti-emetic drug should be sought. There is no generally accepted standard drug to combat vomiting in pregnancy. This is quite evident by the availability of a vast number of preparations produced by many pharmaceutical firms one claiming superiority over the other and by the varied types of drugs given to patients with hyperemesis in different centres. Further the modern woman is also aware of the possible dangers of drugs if adminstered in early pregnancy. Greatest discrimination should be employed in the exhibition of any drug prescribed. Every obstetrician has his or her own preference but the author finds that trifluoperazine (stelazine) gives remarkable results in cases of hyperemesis. It has a double action in that it has a powerful anti-emetic action and it produces rapid relief of anxiety and emotional tension thus restoring calm and confidence. Trifluoperazine has been in the market since 1959 and many investigators in the United Kingdom and Canada have carried out follow up surveys in women who have taken this drug in pregnancy. This clinical experience and foetal studies in animals have shown no evidence to suggest that this drug may cause congenital abnormalities in the child. [27, 28]

Throughout the period of treatment the patient should be dealt with kindly but firmly. The guide to recovery is found apart from the clinical improvement, in the patient's weight chart. The patient should be seen one to two weeks after her discharge from the hospital to assess progress and for regulation of therapy.

Termination of Pregnancy

Even though rapid deterioration of the patient's condition inspite of treatment, persistent albuminuria, persistently low blood pressure, optic neuritis, retinal haemorrhage, jaundice etc. are listed as indications for termination of pregnancy, in practice one very rarley encounters such a situation. At Queen Charlotte's Hospital and University College Hospital, London, termination has not been required for hyperemesis for the last twenty years. Discussions with senior obstetricians in Ceylon revealed that very severe cases of hyperemesis requiring termination are very rare in most centres in Ceylon too. On the other hand if termination is deemed necessary one should seriously consider abdominal hysterotomy under local anaesthesia if the pregnancy is over twelve weeks.

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PSYCHIATRY IN JAFFNA, CEYLON

BY

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(Paper read on 21-1-71 at the World Federation for Mental Health, 22nd Indian Psychiatric Conference — Workshop in Madurai, India.)

"Chairman and Friends,

I am very grateful indeed to the President, Prof. G. M. Castairs for asking me to speak a few words about my experiences in organising and looking after a Psychiatric Unit in Jaffna, Ceylon.

Ceylon, the island neighbour of South India has a population of 12 million people living in 25,000 square miles of land. Upto the middle of 1966 the entire psychiatric service in the island consisted of two large mental hospitals in Angoda, in the proximity of Colombo. Patients from all over the island, however mild, moderate or severe their affliction, had to go all the way to Angoda to get effective treatment. The Angoda Hospital is grossly over crowded. It is evident now, that the majority of patients did not go to Angoda but remained in their towns and villages availing of whatever Ayurvedic or Western medical treatment available locally or went to spiritualists and faith-healers or had no treatment at all. This led to chronic disability, loss of earning capacity and they became a burden on their families. The minority who went to Angoda from the provinces underwent great inconvenience, incurred heavy expenditure and were dislodged from their homes and social background, and were separated from their families and friends for varying periods. Even when they recovered and returned, poor reception ridicule and rejection awaited them, as the place name Angoda, over the decades, had become symbolic of and almost synonymous with lack of sanity. A Mental Health Committee,[1] under the Chairmanship of Dr. W. G. Wickremasinghe and which included Dr. J. Hoenig, W.H.O. Consultant Psychiatrist recommended to the Government, the opening of Psychiatric Units in the big general hospitals in all the nine provinces with a view to making available psychiatric treatment close to patients' homes and also to reduce the congestion in Angoda[2]. The unit in Jaffna is the second such unit to be set and was declared open in July 1966.[3]

The Jaffna Unit caters to the psychiatric needs of about 700,000 people in North Ceylon. The Unit has 66 beds in two General Hospitals in Point Pedro & Kankesanthurai.

The Staff consists of 1 Psychiatrist, 2 House Officers, 1 Psychiatric Social Worker, 9 Male Nurses, 7 Female Nurses and 11 Nurse-aides or labourers. From the very beginning there was a rush of patients seeking treatment and 9,000 new patients have sought treatment in the last 4½ years. In the first two years the average was more than 2000 year—probably we were clearing the backlog that did not go to Angoda and treating the new cases incident in the population In the latter 2½ years the average was slightly less than 2000 new patients/year. About one fourth this number have received indoor treatment and the usual stay is 2 – 4 weeks. When the wards were opened crowds began to gather and stare, probably expecting to see disorderly behaviour and drama. Within

a few weeks the crowds vanished and psychiatric patients have become accepted like other patients by the staff and by the population. Attempts were made to educate and enlighten the public about mental illnesses and mental health with a view to minimising, if not eradicating, the stigma and misconceptions about mental illness. Numerous requests were received from Schools, Rural Development Societies and Village Councils for talks on Mental Health. About 40 talks were given in 4½ years. After each talk a cluster of patients would turn up from that locality. A similar phenomenon was observed when a patient who was disabled for sometime recovered with treatment. Attempts were made to involve the friends and relations of patients in the management, as much as possible, e g. when an indoor patient required a hair cut or a shave relatives were asked to take them out to the hairdresser. When a patient wished to and was well enough to visit a temple or cinema relatives were encouraged to take them out. Visits by relatives and friends were encouraged. The hospitals supply three meals and tea to all patients free of charge. However, relatives were encouraged to bring food whenever they wished to, as so doing gives the patients the feeling that their relatives care for them and demonstrate their attachment. Manual restraint was never encouraged though resorted to in a few instances till a restles patient could be sedated. No attempts were made to demolish rapidly deep-seated beliefs prevalent in the people.[4] Quite a number of patients believed that they were charmed or drugged. They and their relatives reported that symptoms got worse during full-moon days and new-moon days. Attempts to oppose these beliefs resulted in some patients ceasing to attend our clinics and seeking non psychiatric treatment or taking recourse to magical procedures. Since we had no intention of driving patients away from our clinics, we did not persist in opposing their beliefs. After our unit had functioned for one year, a breakdown of the patients seeking treatment showed that 25% of them had treatment in Angoda, 50% of them had either Ayurvedic or Western medical treatment and 25% were attending the Psychiatric Clinics direct. The proportion of patients coming to the Psychiatric Clinics direct was steadily increasing over the years. Patients either came of their own free volition or were brought by relatives. Admission involved no legal formalities. Patients under Police or Fiscal custody were not admitted to our wards as the presence of a guard upsets the psychodynamics of a General Hospital ward set up.

Of the 9,000 patients who sought treatment about 3,600 suffered from Manic-Depressive illness, about 3,300 from Schizophrenic illness and the balance 2,400 belonged to all other diagnostic categories. This finding is compatible with an earlier survey that showed Manic Depressive illness was more common than Schizophrenia in a village in Jaffna.[5,6] Yet it is difficult to arrive at such a conclusion. It is possible that more Schizophrenics from North Ceylon have gone to and remained in Angoda whereas the Manic-depressives remained in the community and were brought to the surface by the opening of our Unit, Further, in December 1964 North Ceylon was struck by an unprecedented cyclone where in addition to destruction of homes and property about 2,000 fishermen perished at sea overnight leaving many widows, numerous orphans and many elders without their sons who supported them. To what extent these two factors contribute to the greater incidence of Manic Depressive illness in our series is not clear. Treatment in our Unit is essentially with drugs, E. C. T. Occupational therapy and group

activities. There is hardly any time for psychotherapy. I would have preferred to to keep indoor patients on drugs for a few weeks before giving E. C. T.[7] But on account of the numbers turning up from quite far off places, a quick turnover and fuller utilisation of the 66 beds became necessary. So E. C. T. is given early along with drugs to psychotics. Occupational therapy organised by the nursing staff consists of sewing, gardening and mat-making, which activities patients could continue at home after discharge. Well wishers of the Unit have organised themselves into a society, named "FRIENDS OF THE PSYCHIATRIC UNIT KANKESANTURAI", visit and help in the rehabilitation of patients. Two welfare programmes are organised every month and patients actively and enthusiastically participate in these group activities.[8] This Society also organises functions for Thai Pongal, Sinhala and Hindu New Year and Christmas, giving presents. They are very popular and much appreciated by the patients. After discharge relatives are advised and instructed to bring patients for follow-up treatment at least once a month for varying periods from 6 months to 2 years. The Psychiatric Social worker in addition to investigating the Psycho-Socio economic background of selected patients, visits homes, gives support and urges patients to attend for follow-up treatment.

We have been able to treat satisfactorily all categories of acute and chronic patients without referring them to the Mental Hospital in Angoda. It is my humble view that more and more Psychiatric Units should be opened in General Hospitals.[9] These Units could treat all patients. Chronic patients could be looked after by their relatives who could be offered financial incentives. A few chronic patients will have no homes to go to and no relatives to care for them. They could be housed in small long stay units of 100—150 beds adjoining each provincial hospital. In fact with more effective treatment methods becoming available chronicity should in course of time cease to be a problem. It will be a happy day for psychiatry when the large Mental Hospitals with the fortress-like walls and castle-like buildings wither away and cease to exist altogether leaving the patients to be cared for by the community."

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SOME ASPECTS OF THE MANAGEMENT OF TRAUMATIC INTRACRANIAL LESIONS

BY

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DO not ever think that the management of head injuries cannot be done by a general surgeon. Opening and closing a skull was regarded as a great feat in those days. Now, it is regarded as a nuissance. Many neurosurgeons will not agree with this, statement, but I am convinced of it. There are only three neurosurgical units in Ceylon, and it is humanly impossible for these three to tackle all the head injuries in Ceylon. Every general surgeon should regard himself as a part-time traumatic neurosurgeon, till Ceylon is provided with adequate neurosurgical units.

PATIENTS SEEN IN OUTPATIENT DEPARTMENTS

The cases of head injuries, seen here, can be classified into three broad groups.

1. Conscious throughout.
2. Unconscious from the time of injury.
3. Brief unconsciousness followed by full consciousness

All cases that belong to groups 2 and 3 need admission to a hospital for observation. Those cases in group 1 needing admission are :

1. Those having amnesia of the accident
2. Those with open or closed fracture of skull.
3. Infants, and
4. Drunken patients.

A full clinical examination and an X-ray of skull must be carried out. These should be recorded legibly under the following headlines:

1. Local injuries in head.
2. Any other injuries.
3. Basic physical signs (pulse, blood, pressure, respiration, temperature)
4. Special physical signs (level of consciousness, state of pupils, abnormal signs in the central nervous system)
5. Any fracture of skull (simple or compound)

INITIAL CARE IN THE WARD

Once the patient is admitted, the patient is examined again to detect any change of physical signs recorded by the admitting officer. The house officer and the nurses should be aware of the expert nursing care that a head injury patient needs. To refresh their memory, I give below the A, B, C, D, E of head-injury nursing.

A. AIRWAY care
B. BLADDER, BOWEL and BEDSORE care
C. CHART keeping, CUBICLE nursing and CHANGE OF POSITION of patient
D. DIET and DRUGS
E. EYE, EAR care and EXERCISES

If the breathing is disturbed, the airway should be kept clear by intermittent suction of the throat. If this does not help, a cuffed endotracheal tube is inserted immediately. The cuff is deflated hourly for 5 minutes to prevent trauma to trachea. If the endotracheal tube is needed for more than one day in adults, and one week in children, a tracheostomy should be done on the expiry of the above-said periods. There is no place for emergency-tracheostomy in neurosurgery unless the head injury is complicated by facial or thoracic injuries.

Bladder and bowel care arise only in the unconscious patients. A Paul's tubing in males, and an indwelling catheter in females may be needed. Wet beds give bed-sores. Bed-sores and bowels need the usual nursing care.

An ideal place to nurse a head injury patient is a noise-insulated cubicle. An unconscious patient should be turned from side to side two-hourly.

It is better not to give any oral feeds for the first 12 hours, because he may have to be given an anaesthetic if any surgery is to be done. After 24 hours, the feeding should be commenced either by mouth or via a naso-gastric tube; and an Intake–Output chart maintained.

The only drug that these patients need is tetanus toxoid. If there is a compound fracture, or bleeding per ear, nose, throat, then an antibiotic (preferably penicillin and suphadiazine) is necessary. The routine use of steroids and hypertonic solutions is of questionable value. No sedatives need he given unless the patient has convulsions.

A dry ear is a safe ear. Never plug a beeding ear! A sterile pad is to be used to cover the ear. In unconscious patients, the eyes should be kept moist with sterile paraffin. All patients, who are not ambulant need physiotherapy to chest and limbs.

The nurse is instructed to maintain a half-hourly observation chart consisting of pulse-rate, pupils whether equal, central, regular and reacting to light, blood pressure, level of consciousness, and temperature. She is also asked to watch for any fits or development of weakness of face and limbs. Any change in the pulse rate, pupillary reactions or level of consciousness should be conveyed to the house officer, and the surgical team alerted. In an ideal neurosurgical unit, carotid angiography can be done to detect where and what the lesion is. Only the general surgeons can help the patient in outstations.

WHEN NO NEUROSURGEON IS AVAILABLE

What can a general surgeon do, when he is confronted with a problem like this? Every sick patient should be regarded as having an extra-dural haematoma, unless proved otherwise by exploratory burr holes. Never do a lumbar puncture! It is unpardonable if he tries to transfer this patient to a neurosurgical unit (whether Colombo or Jaffna) This is an emergency and the operation may have to be done on the the patient's bed, with unsterile instruments. The pressure on the brain should be relieved wherever the patient is, and by whoever is available[2].

The burr holes or trephines are done at the classical sites. If an extradura haematoma is not found, the dura is

opened to let out any subdural haematoma. If neither of these are found, and the brains is tense, the lateral ventricles of the brain are tapped by any blunt needle (with stillette), and the cerebrospinal fluid replaced by 15 cc of air[3]. X-rays of skull (AP and lateral) are taken with the patient's face down. The ventricular shift detected on these films will pinpoint the site of the lesion. A burr hole is made on that point and this hole is extended by nibbling the bone around, and the clots are then evacuated. A neurosurgeon may call this woodpecker, surgery. It is better to be a woodpecker saving lifes, rather than being a transport-agent for the neurosurgical units, there by (delaying surgery and killing the patients).

If no surface clots are found, the patient may be transferred to a neurosurgical unit for further investigation and management. This transfer of the patient can be done with a clear conscience, because an easily treatable condition like an extradural haematoma, is excluded by the general surgeon.

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INTERPRETATION OF THE SEROLOGICAL TESTS FOR SYPHILIS

BY

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IN the recent past several serological tests have been developed to assist in making a diagnosis of syphilis. Assist, because no serological test so far known can be relied on to make an absolute diagnosis in all cases. However, if the significance of the tests employed is clearly appreciated, then with sound clinical judgment the physician can in nearly all cases satisfactorily resolve diagnostic problems. Here lies the immense value of the tests. Wrongly interpreted, going only on the face value of the results, they create more confusion and mislead rather than enlighten. It is therefore imperative, that all clinicians who are faced with the task of making a diagnosis have a clear understanding of the values and limitations of the various tests, before making a request for them. A reactive VDRL result for example may in certain cases prove to be due to a non syphilitic reaction, while a syphilitic, can in some circumstances yield a non reactive result. In addition to assisting in the diagnosis, some of the tests also serve to determine the efficacy of treatment. An effort is made in this short paper therefore, to discuss the serological tests in common use without going into technical details, with particular emphasis on those available in this country.

During the course of infection by the Treponema pallidum, the body responds by the formation of different antibodies to the various antigens found in the organism. The basis of the serological tests, is the detection of one or other of these antibodies, and the tests are of two main types depending on the type of antigen used:

(A) Nontreponemal tests, which employ a nontreponemal antigen, either extracts from normal tissue such as beef heart or the active principle in it, cardiolipin, and (B) Treponemal tests, which are performed with treponemes or extract of treponemes.

NONTREPONEMAL TESTS (STANDARD TESTS, REAGIN TESTS):

These detect an antibody-like substance in the serum called "reagin", which develops as a result of the interaction between the treponeme and the body tissue. Reagin appears in the serum in significant amounts, not only in syphilitic infection but in certain other conditions too. The tests are thus not absolutely specific for syphilitic antibodies. However, syphilis being the single most common cause of a reactive result, these tests are highly indicative. The added advantages of being inexpensive and easy to perform make them the most widely used serological tests for syphilis. In fact in many situations their value far outweighs that of the more specific and glamorous tests.

The reagin tests are of two basic types:

(i) Complement—fixation test:

The Kolmer test is the one in common use today and this employs a cardiolipin antigen. The original Wassermann test (1906) was also a complement—fixation test now given up owing to its poor sensitivity.

(ii) Flocculation test (Kahn, VDRL, Kline, Mazzini):

The Kahn test (1922), the first flocculation test to be developed, employs a natural tissue antigen. Most other flocculation tests use cardiolipin antigen, the active principle in the natural extract, as this has been found to improve the specificity and sensitivity of the test. Thus, more laboratories are now switching over from the Kahn to the other tests. The Venereal Disease Research Laboratory (VDRL) test (1946) is the one currently most favoured and is the reagin test available in this country.

While many VD clinics are satisfied with employing only one type of reagin test as a routine, some others, especially in U. K. prefer to do both a complement-fixation and a flocculation test in all cases, so that this could serve as a cross check and also make use of the advantage of one test being more sensitive than the other.

Under special circumstances, such as for mass screening purposes e.g. blood donors, surveys etc. where accuracy has to be sacrificed for speed, modified flocculation procedures have been developed such as the Rapid Plasma Reagin (RPR), Unheated Serum Reagin (USR), and the Plasmacrit (PCT) tests. All reactive specimens have to be further investigated with other tests.

Serological Course of Syphilis:

The reagin test becomes reactive in about 1-2 weeks after the appearance of the chancre (i.e. about 4-5 weeks after infection), the primary stage being thus divided into a seronegative and a seropositive stage. Untreated, the titre rises rapidly to a peak in the secondary stage and all cases without exception are reactive in this stage. Thereafter, the titre remains high for a varying period and then declines slowly to a low level and plateaus off. Thus, late cases of syphilis are often seen, in whom the serology may even be weakly reactive (WR). After several years one third of the individuals show spontaneous reversal of the titre to zero, while in the others this persists indefinitely, enabling serological confirmation of the disease. Spontaneous seroreversal does not necessarily mean spontaneous cure, as occasionally active late cases of syphilis are seen who are seronegative. In such cases where the routine nontreponemal tests are not sufficiently sensitive to detect the subthreshold levels of reagin, the treponemal tests (TPI, FTA) are extremely valuable in detecting most of them. It is estimated that in approximately 10% of patients with late benign syphilis (osseous, cutaneous, visceral), in approximately 20% with cardiovascular syphilis and in approximately 33% with tabes dorsalis the nontreponemal tests are non reactive (NR).[1]

False positive reactions:

Unfortunately the nontreponemal tests suffer from another drawback, in that they are not absolutely specific for syphilis. Certain other conditions, acute and chronic, also give reactive results with these tests owing to the presence of reagin, but here the treponemal tests are always negative. They are termed Biologic False Positive (BFP) reactions and are arbitrarily divided into 'acute' and 'chronic' reactions. Those conditions that give rise to the acute reactions are malaria, active pulmonary tuberculosis, vaccinia, virus pneumonia, glandular fever, immunisation against small pox, typhus, Weil's disease and filariasis. These reactions are characterised by a reactivity that is of low titre and temporary, lasting seldom over three months. But often no specific cause for a BFP can be recognised. It is extremely important to remember that the presence of infection or history of recent

immunisation in no way confirms a BFP, as syphilis is the single most common cause of a reactive test and if facilities for a treponemal test are not available to exclude a BFP, every effort must be made to look for evidence of syphilis, from a careful history, complete physical examination and epidemiological investigation, before labelling a seroreactor a BFP. Chronic false positive reactions are less frequent and may be associated with leprosy, collagen diseases including lupus erythematosus, and the 'auto immune' diseases They are often of high titre and tend to be more persistent. The incidence of BFP reactions in the U.S.A. has been found to be around 3% of all seroreactors with the modern nontreponemal tests using cardiolipin. [2]

Technical false positive reactions are also seen due to human errors in the collection and labelling of specimens and in the conduction of tests and their reporting etc. but these are detected on repeating the test with another specimen. This emphasises the importance of repeating a test with another specimen whenever a reactive result is obtained. When more than one type of test is done with the same specimen these technical errors tend to be minimised.

Reporting of Results :

(i) Qualitative test :

This is reported as Reactive (R), Weakly Reactive (WR) or Nonreactive (NR). A specimen which contains reagin in large amounts adequate to provide a definite reaction (moderate or large clumps), is reported Reactive. One that has just sufficient reagin to cause a weak reaction (small clumps) only, is reported Weakly Reactive. Such a result could well signify a syphilitic reaction, if on repeating the test it still remains weakly reactive. A specimen which contains no reagin or reagin in amounts not detectable by these tests, is reported Nonreactive.

(ii) Quantitative test :

This is done on all weakly reactive and reactive sera in the qualitative test, mainly to establish a baseline from which a change can be measured, to evaluate therapy, to detect a temporary reaginaemia in acute BFP reactions and in following up a reactive serology in the newborn to determine either an active infection or a passive transfer of reagin from the mother. The result is reported as an end point titre — the highest dilution of serum that gives a reactive result e.g. as R (1), R (2) or R (4) etc. WR (0) means a weakly reactive reaction in the undiluted serum.

It is worth repeating, that late cases of syphilis even active ones could give a non reactive (NR) result. A treponemal test is therefore very essential in a clinically suspicious case that gives a nonreactive VDRL result, before regarding it as nonsyphilitic. A weakly reactive (WR) VDRL test should always be repeated, and if still weakly reactive, could well signify a syphilitic infection. Many late latent and late cases of syphilis give only a weak reaction. A high titre does not necessarily mean syphilis but it is strong evidence for its presence. High titres are found not only in secondary syphilis but also in some cases of late cutaneous and visceral syphilis. But, since nonsyphilitic conditions such as systemic lupus erythematosus and haemolytic anaemia are also known to give high titres, a treponemal test may be necessary to diagnose cases with no evidence of syphilis.

Effect of treatment :

Repeated quantitative serological examinations following treatment, serve to

determine the success of therapy. Titres become nonreactive in 6 to 12 months in cases of primary syphilis and in 12 to 18 months in secondary syphilis following therapy. Early latent cases take a little longer or may follow the pattern of a late case. A persistently high titre or a rise in titre by a 2 tube dilution after an initial fall, suggests active infection and failure of treatment, and retreatment is indicated. It could also be due to a fresh infection following adequate treatment. In the case of late syphilis, treatment has little or no effect on the titre, and the serological response is no guide to the adequacy of treatment. The titre either tends to fall after a time and remain low or remains unchanged. Subtherapeutic doses of antibioties administered by the physician either as a prophylactic measure of for an associated ailment during the incubation period, can distort the normal serological development by either delaying the seroresponse or by producing a lower serological titre than expected (e.g. in secondary syphilis) thus making diagnosis difficult. The appearance of the chancre is also delayed or suppressed.

The value of non treponemal tests:

Despite the limitation that nontreponemal tests detect only reagin and not specific antibodies, in the following situations their value has been undisputed:

(i) For routine use when ease of performance and economy are required.

(ii) When history or clinical evidence points to a diagnosis of syphilis, a reactive test is confirmatory.

(iii) In epidemiological investigations of contacts or suspects a reactive result is highly significant.

(iv) In following up babies suspected of congenital syphilis, quantitative testing is a very valuable diagnostic aid.

(v) To determine the efficacy of treatment, the serological response serves as a valuable guide.

(B) TREPONEMAL TESTS:

These detect antibodies to the treponema itself and are therefore more specific. There are at least two separate treponemal antibodies. One reacts with the T. pallidum as well as the other treponemes including the non pathogenic Reiter treponema, while the other reacts only with T. pallidum The latter is specific for T. pallidum while the former which reacts with the shared or group antigens found in all treponemata, is specific for treponemes in general. Several treponemal tests have been described but of them only four need comment.

(i) Treponema pallidum immobilisation (TPI) test (1949):

This was the first test to be described and uses the T. pallidum as antigen. In the presence of complement, syphilitic serum immobilises the treponemes while the non-syphilitic serum fails to do so. It is highly specific but as the appearance of the immobilising antibody is delayed, the test is less sensitive in early syphilis than the nontreponemal tests, about two thirds of primary syphilis and one third of secondary syphilis being TPI negative. It is 100% positive in about 2-4 months after infection. It has considerable sensitivity in late syphilis and except in a few cases of tabes and late congenital syphilis, it is of immense value in the diagnosis of syphilis of several years duration, in which the nontreponemal tests have become nonreactive. Its high specificity enables BFP reactions to be differentiated from those due to syphilis However,

as it is very expensive to perform, technically difficult and time consuming, it is available only in a few specialised laboratories in the world. It is not performed in Ceylon.

(ii) Reiter protein complement-fixation (RPCF) test (1957)

This is a complement-fixation test employing a protein extract from the Reiter treponeme as antigen, and is inexpensive and easy to perform. It becomes positive a little after the reagin test but this has not been much of a problem. Although its specificity is good, some saprophytic and mouth treponemes sharing the common group antigens also give positive results. After several years of use, it has been found that the main drawback has been its low sensitivity in syphilis of many years duration, especially cases of cardiovascular syphilis and tabes. For these reasons the RPCF test is losing much of the promise it initially held out. The test is still available in this country at the Medical Research Institute, though with the introduction in Ceylon of the FTA test, the demand for it has declined.

(iii) Fluorescent treponemal antibody (FTA) test (1957)

The Central VD Clinic laboratory in Colombo reserves this test mainly for cases presenting diagnostic problems. The test uses the fluorescent antibody technique and is a more recent and promising one. The antigen consists of dead T. pallida. The treponemes are allowed to dry on a slide and the test serum is added to it. If the serum contains antibodies to the T. pallidum, these adhere to the organisms. To detect this, a conjugate (fluorescin tagged antibody to human globulin) is next added which reacts with the syphilitic antibody (globulin), and the organisms fluoresce when viewed under ultraviolet light. No fluorescence will be observed if the syphilitic antibody is absent in the serum. In the original test a 1 : 5 dilution of serum in saline was employed, but as the group antibodies yielded nonspecific reactions, a 1 : 200 dilution was later used to eliminate this effect, and the test was referred to as the FTA '200'. Although very specific the sensitivity was lowered by the dilution. To overcome this difficulty, the serum was initially treated with an extract of Reiter treponeme, which absorbed the group antibodies and also gave it a 1 : 5 dilution. This enabled the test to have a high specificity without reduction in sensitivity. This modified procedure is called the FTA (ABS) test (1962). The test is considered as specific as and much more sensitive than the TPI test. In early syphilis it may become positive even before the VDRL test, while in the late stages it helps to confirm cases of late symptomatic syphilis which are nonreactive with the routine reagin tests.

(iv) Treponema Pallidum haemagglutination (TPHA) test (1965):

Very recent studies indicate that this could prove to be a valuable serodiagnostic test. It has been shown to have a high specificity and sensitivity. The simplicity of the equipment and the low cost involved could even make it a routine serological test for syphilis. Formalised tanned sheep red cells are conjugated with antigens from a lysate of T. pallidum. A suspension of the sensitised erythrocytes is then used to detect antibodies in the test serum in serial dilutions. Still being evaluated, the test is not in use in most countries and is just in the experimental stage in Ceylon, at the Central VD Clinic laboratory.

Conclusion

Having dealt with the significance of the more important serological tests for syphilis, it must be stressed, that in practice certain situations could arise, in which the results of serological tests are found to be in conflict with the clinical opinion. If such problems cannot be satisfactorily resolved with a careful history, further tests and investigation of contacts, one should not hesitate to give the patient the benefit of a full course of antisyphilitic treatment, for it is far better to treat unnecessarily a pregnant mother, than face the risk of delivering an infant congenitally infected. It should also be remembered that whatever value a serological test may be endowed with, the test cannot replace a thorough clinical examination. It must be conceded however, that with the recent progress made in syphilis serology, one is able to approach a near 100% accuracy in diagnosis, which is of great consequence because of its social implications.

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AN UNUSUAL CASE OF DYSTOCIA – Case Report

BY

Dr. (Miss) H. VANNIASINGHAM M.B.B.S. (Madras.)

Tellipallai Co-operative Hospital, Tellipallai

A 19 year old female in the 26th week of her second pregnancy was seen at The Tellipallai Co-operative Hospital for the first time on 28-4-71. Her first pregnancy had ended in an abortion at The Tellipallai Co-operative Hospital in April 1970.

Her menstrual rhythm was regular with her last regular menstrual period on 2-11-70.

On examination, her general condition was good, her B.P. 120/80 mm. Hg. The cardio-vascular and respiratory systems were normal and her Hb. 50%. The urine was free of sugar and albumin.

The height of the uterine fundus was 38 weeks although the period of gestation was only 26 weeks. The foetal head was small, the foetal heart sounds normal and there was no evidence of multiple pregnancy.

On the 13th May she was seen again with a complaint of discomfort in the abdomen and slight difficulty in breathing. The abdomen was grossly distended. The uterus was large and a fluid thrill was present. The foetus was presenting by the head. The heart sounds were normal and a diagnosis of acute hydramnios was made. The patient was advised admission to hospital but she did not want to stay. On the next day, 14th May, however she was admitted with pains. The head was engaged, the foetal heart normal, but the patient did not feel the foetal movements from the time pains started.

Artifical rupture of membranes was done at 10-00 a.m. and very little amniotic fluid drained. By 2-30 p.m. as progress was poor, the scalp hair being visible at the vulva for 12 minutes a vaginal examination was done. The cervix was fully dilated and an outlet forceps under local anaesthesia was decided upon. Kiellands forceps was applied and the head delivered after an episiotomy. Traction produced no further descent. On passing one hand along the chest of the foetus the abdomen was found to be grossly distended and cystic, obstructing descent.

Under general anaesthesia, a long artery clamp was passed in, guided by the two fingers and the foetal abdomen perforated just beyond the costal margin. A large amount of clear fluid was drained. Traction now produced only a small amount of descent. Further examination revealed another cystic swelling. This too was perforated in the same manner as the first and a large amount of clear yellow fluid drained. The delivery of the body was easy after that. The blood loss was minimal and the patient's general condition good.

EXTERNAL APPEARANCES

Autopsy of the foetus was done. The head, neck and chest were normal. The abdomen was grossly distended

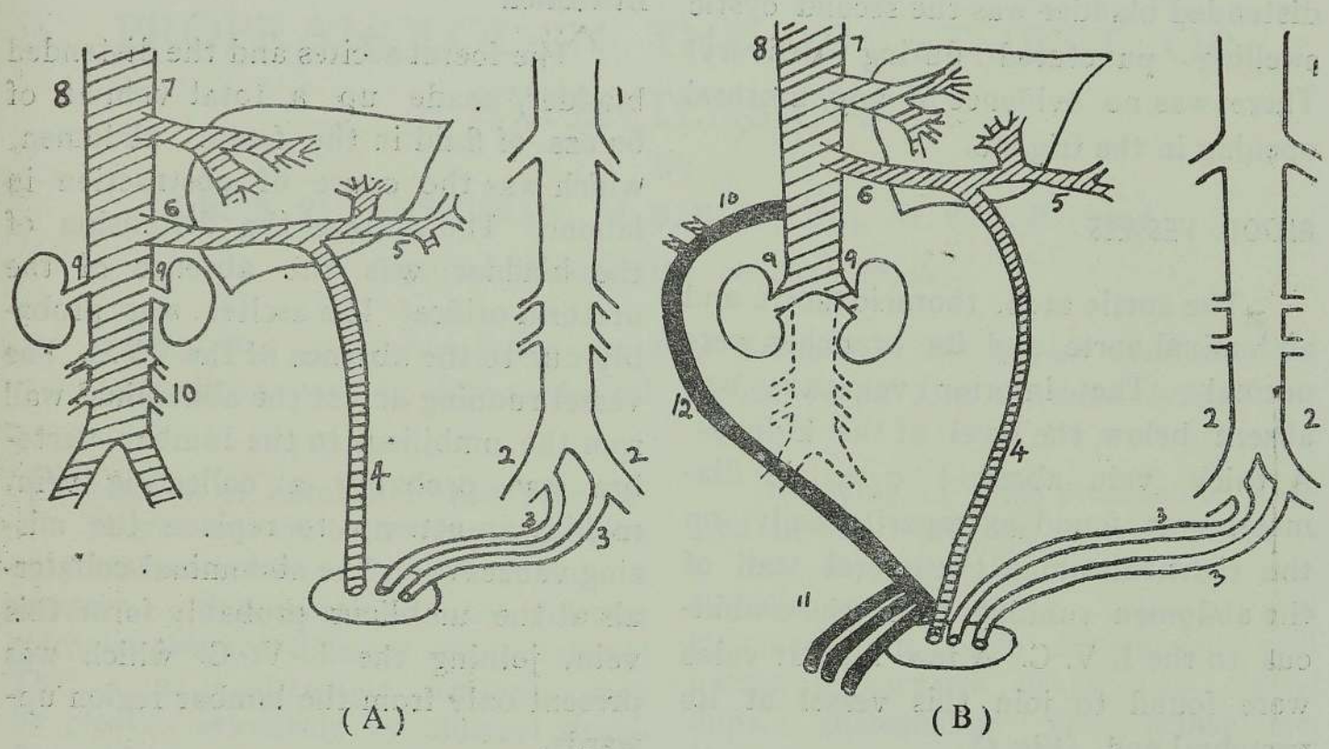

Fig. 1 Diagramatic representation of the abdominal part of the foetal circulation seen in (A) normal infant. (B) the foetus autopised.

1. Abdominal aorta
2. Common Iliac arteries
3. Umbilical arteries (later obliterated hypogastric arteries)
4. Umbilical vein.
5. Portal vein
6. Ductus venosus
7. Hepatic vein
8. InferiorVena Cava
9. Renal veins
10. Lumbar veins
11. Abnormal veins in the abdominal wall leading to the umbilicus
12. Abnormal venous channel from umbilical vein to the I. V. C.

with numerous blood vessels over the lower abdominal wall converging towards. the umbilicus. The external genitalia were not developed. There was no evidence of any urethral orifice. The anal orifice was absent only a dimple being seen.

INTERNAL APPEARANCES

The heart and lungs were normal, the lungs being solid and unexpanded. The abdominal cavity was found to be grossly distended. The intestines were very small. The rectum was loaded with meconium. The liver and spleen were normal. The right ureter was tortuous and distended. The left kidney was about 3 c.m. in length, and its ureter greatly distended and tortuous. Both ureters led into the bladder, which was hypertrophied and grossly distended with a puncture mark on the fundus (The

distended bladder was the second cystic swelling punctured during delivery) There was no evidence of any urethral opening in the trigone.

BLOOD VESSELS

The aortic arch, thoracic aorta and abdominal aorta and its branches were normal. The inferior venacava was absent below the level of the kidneys. A thick vein about ½ c. m. in diameter was found extraperitoneally on the right side along the lateral wall of the abdomen running from the umbilicus to the I. V. C. A few lumbar veins were found to join this vessel at its vertebral end. (Fig 1)

Discussion

The foetal ascites and the distended bladder, made up a total volume of 60 ozs. of fluid in the foetal abdomen, which was the cause of obstruction in labour. The cause of the distension of the bladder was the absence of the urethral orifice. The ascites was probably due to the absence of the I V.C. The vessel running across the abdominal wall rom the umbilicus to the lumbar vertebra was probably a collecting vein, making an attempt to replace the missing venacava. The abdominal collaterals at the umblicus, probably form this vein, joining the I. V. C. which was present only from the lumbar region upwards.

PROPRANOLOL IN THE TREATMENT OF HYPERTENSION

BY

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(Based on a paper read before the Joint Sessions of the Ceylon Medical Association and the Jaffna Medical Association on 13th Novembor 1970)

THE effects of adrenaline have been explained on the basis that two types of receptors exist – α receptors and – β receptors. Stimulation of the different receptors produces different effects (Table 1) The α or β adrenergic recetpors may be blocked selectively by different drugs. Propranolol is a drug blocking the β – receptors.

The hypotensive action of β – adrenergic blocking drugs was observed by Prichard during the early clinical trials of pronethalol in angina pectoris. He subsequently demonstrated this action with propranolol too.

Propranolol reduces cardiac output but probably produces hypotension by blocking the cardiac component of the pressor responses to stimuli and as a result the baro-receptors gradually regulate the blood pressure at a lower level. The place of β blockers in the treatment of hypertension is controversial.

According to some propranolol, when used in the treatment of hypertension is at least as potent as methyl dopa, guanethidine and bethanidine. Severe postural hypotension is often encountered with potent hypotensive drugs like methyl dopa, guanethidine and bethanidine. However propranolol does not produce postural or exercise hypotension and is often more tolerated by the patient. Thus propranolol appears to produce the best control of supine blood-pressure. The hypotensive effect of propranolol often takes 6 – 8 weeks to reach its maximum. It is contra-indicated in obstructive airways disease and in uncompensated heart failure.

This study deals with 10 hypertensive patients who were treated with propranolol, three of them had angina pectoris as well. Of the ten patients, four had not received any hypotensive treatment before, (Table 2) while two were on guanethidine (Table 3) and four were on methyldopa (Table 4)

α –effects	β–effects
Vasoconstriction	Vasodilation — marked in muscles
Mydriasis	Heart — increased force and rate
	increased excitability
	Bronchial relaxation

Table 1. gives the α and β effects of adrenaline.

Age	B. P. Before propranolol		B. P. After propranolol		Maximum Dose Propranolol
	Supine	Standing	Supine	Standing	
54	210/120	210/120	190/100	190/100	240 mg.
58	225/130	225/130	200/115	200/100	400 mg.
56	185/110	185/110	170/100	165/95	400 mg.
54	190/100	190/100	160/100	160/95	400 mg.

Table 2 Supine and standing blood pressures of four patients whose hypertension was treated with propranolol

Age	B. P. on Guanethidine		B. P. on Propranolol	
	Supine	Standing	Supine	Standing
54	185/100	150/90	140/90	130/90
50	195/110	170/100	160/100	150/100

Table 3 Supine and standing blood pressures of two patients who were originally on guanethidine and later transferred to propranolol.

They were all reviewed fortnightly as out-patients. Their systolic and diastolic blood pressures were taken both in the supine position (after lying down on a couch for 3 minutes) and also standing (1 minute after standing).

Propranolol was started with a dosage of 10 mg. q.d.s. and increased at each visit, the dosage of the previous drug being gradually reduced. Sensitivity to propranolol varies widely and dosage should be gradually increased. There was no correlation between the dosage of propranolol required and the dosage of guanethidine, bethanidine or methyl dopa used earlier. A low or high dosage on previous therapy did not necessarily mean that a similarly low or high dosage of propranolol was required to control blood pressure.

The greater part of the hypotensive effect is seen within the first two weeks or so after dose adjustment. The maximum action occurs about three weeks after the dose adjustment and some further fall of blood pressure may occur over the next month. A bradycardia of 55 beats per minute after rest was normally regarded as a side-effect. The other side-effects noted in these patients were tiredness in two patients, occasional dizziness in three patients, none of these being so severe as to necessitate stopping treatment. Of the six patients on guanethidine or methyl dopa, who were transferred on to propranolol four felt better and two felt no better than before.

Age	B. P. on Methyl Dopa		B. P. on Propranolol	
	Supine	Standing	Supine	Standing
51	175/120	145/115	155/90	155/90
51	170/125	155/220	155/95	145/90
55	160/85	140/80	140/85	140/75
53	155/90	140/85	150/90	140/85

Table 4. Supine and standing blood pressures of four patients who were originally on methyl dopa and later transferred to propranolol.

Discussion

The results support the view that propranolol has a significant hypotensive action. All ten patients acheived a level of standing diastolic pressure of 100 mm Hg. As discussed by Prichard about 40–60% of patients in various series treated with bethanidine, guanethidine or methyl dopa usually reach this level of control. Prichard and Gillam studied 34 patients on propranolol and found that in 33 patients the blood pressure was well controlled. In 25 of these patients propranolol was used alone. In a subsequent study in 109 hypertensive patients treated with propranolol upto 4 years, 92 acheived a supine or standing diastolic presure of 100 mm Hg. or less. In 9 the drug was withdrawn. A diastolic blood pressure of 100 mm Hg. or less was acheived in more patients with propranolol than with guanethidine bethanidine or methyl dopa.

Studies by Waal, Prichards and others showed that propranolol has only a minor effect in control of hypertension. However these authors used it either for a short period or in a lower dosage. When propranolol was used with hypotensive drugs, in many instances the hypotensive effect of propranolol appeared to be additive to diuretics, bethanidine, guanethidine, and methyl dopa etc although no formal study has been made. Propranolol has also been used with hydralazine and reserpine.

In moderate and severe hypertensives treated with oral propranolol, though there is an immediate slowing of the heart, the hypotensive effect is often delayed up to 1—2 months. Propranolol reduces cardiac output immediately, but a progressive fall in cardiac output is unlikely as there is no progressive fall of heart rate. It is possible that the hypotensive effect of propranolol is not due to its β– receptor blocking action. However other β– blocking drugs have a hypotensive effect. Waal pointed out the similarity in hypotensive action of quinidine and propranolol and sugested that the hypotensive effect of propranolol is mediated by its quinidine like action.

It is thought that the hypotensvie action of propranolol is a result of its blockade of the cardiac sympathetic nerves (Prichard & Gillam 1964). This reduces the cardiac component of the pressor stimuli and therefore the magnitude of transient rises in blood pressure in response to various stimuli, as seen for example with

the Valsava manoeuvre. To explain the delay in onset of the full hypotensive action it was suggested that the baroceptors are gradually conditioned by the reduced pressor responses, to regulate the blood pressure at a lower level, so that the mean blood pressure falls. A parallel situation probably exists when a hypertensive patient is put to bed. Bed rest results in a reduction in sensory stimuli and the baroceptors regulate blood pressure at a lower mean level.

It could be stated that propranolol is a hypotensive drug of a potency that appears comparable to that of guanethidine and methyldopa. Propranolol usually results in good control of the supine blood pressure and does not cause postural or exercise hypotension. It seems possible that it produces fewer side effects than the other potent hypotensives. It is particularly useful in patients who have a postural drop of blood pressure and other side effects without good control of the supine pressure. It is also useful when angina is associated with hypertension.

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VIBRAMYCIN

a point worth pondering

To be effective, most oral antibiotics need to be given four times daily. Admittedly, a few are dependable when given in a twice daily dosage, but only one—Vibramycin—is fully effective when administered once daily. Why is this ?

The answer lies in the deep-rooted differences between Vibramycin and the conventional tetracyclines. For instance, up to 95% Vibramycin is absorbed after oral administration, and neither the absorption nor the activity of the drug is noticeably affected by the ingestion of food or drink. With tetracycline, on the other hand absorption is not notably good, rarely exceeding 50%. Again, the half-life of Vibramycin is 15 hours after a single dose, extending to 22 hours after multiple dosing. This compares with about 8 hours for tetracycline.

But perhaps the greatest difference of all lies in the unusually high lipoid solubility of Vibramycin. It is postulated that this property has the effect of enhancing the transfer of the drug through the cell membranes, thereby easing absorption into the tissues. Certainly, the power of Vibramycin to penetrate tissue is well proven clinically and in vivo.

In lung tissue, the level of Vibramycin was twice that of oxytetracycline and over three times that of DMCT 6 hours after administration.[1]

In sputum, Vibramycin was more effective than ampicillin in eradicating the invading pathogens—particularly H. influenzae.[2]

In sinus secretions, levels of Vibramycin were ten times higher than the M.I.C. values for the bacteria found in the pus.[3]

In tonsillar tissue, the levels of Vibramycin were still therapeutically active 24 hours after the final dose.[4]

Thus the virtue of Vibramycin is not so much that it may be given once daily, but that *it is singularly effective clinically* when given once a day.

Supply: 100 mg. capsules, in packs of 5, 25 and 100.

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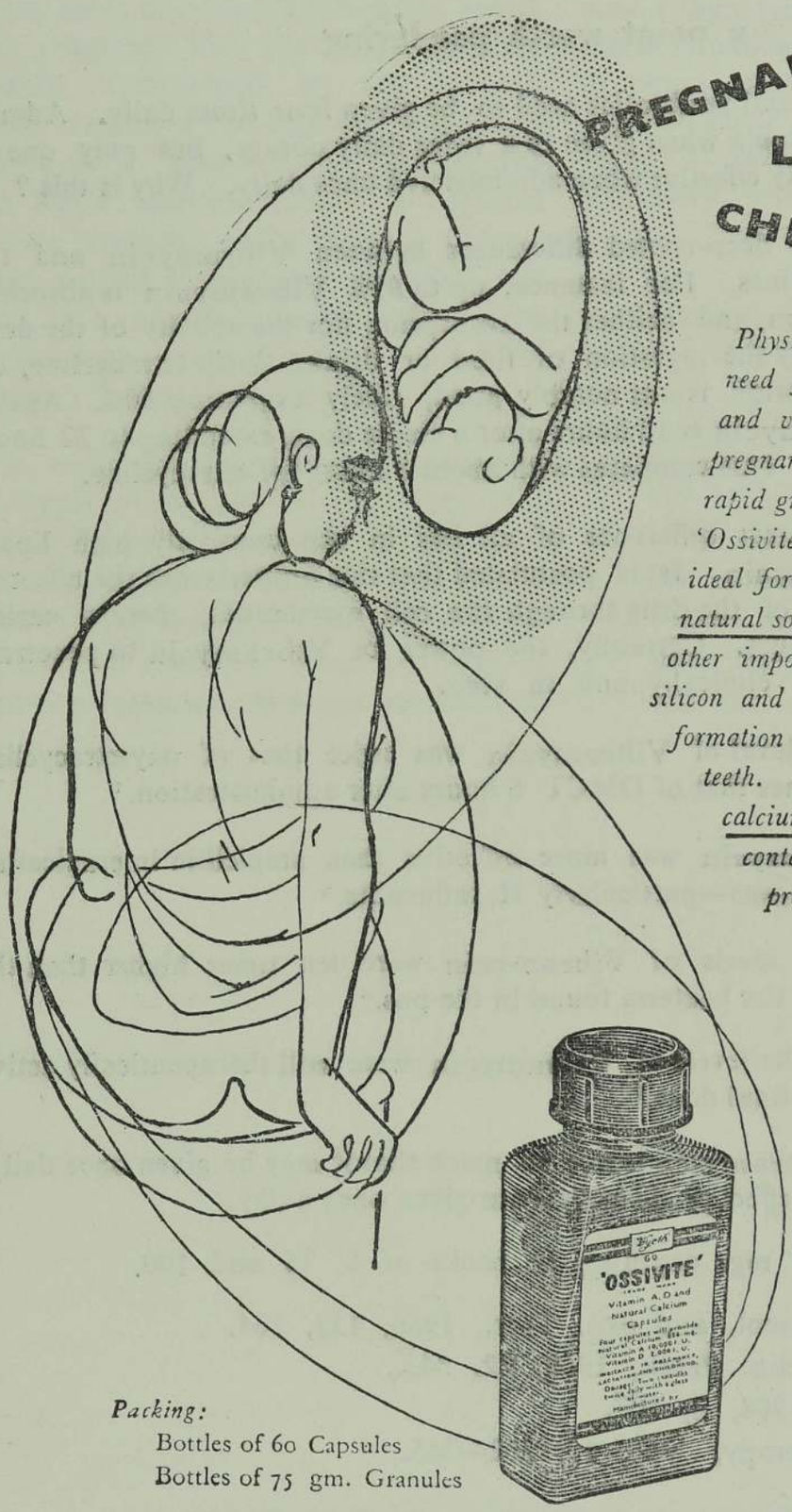

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ASSOCIATION NEWS

Report for the period October 1970 to July 1971

Due to the unsettled conditions in the country, the Annual General Meeting had to be postponed to July 1971. Other meetings too were not held for a period of 2½ months from the end of March 1971. Inspite of this, the period under review has been a very active one.

Membership :— This has been maintained at the previous level, with the addition of some out-station members.

Meetings: — 13 Scientific Meetings and a Joint-Session with the Ceylon Medical Association were held during this time. Details of these meetings are given below. One of the Scientific Meetings was held at Base Hospital, Pt. Pedro.

We thank the following guest-speakers for addressing us: Dr. S. Nusbaum, Dr. V. T. Herath-Gunaratne, Dr. R. Sri Pathmanathan and Dr. J. St. George.

Pharma Distributors and S. Appucutty and Sons of Colombo were responsible for screening a film on "Diabetes". We thank them for this film show.

All Scientific Meetings, especially the Clinical Demonstrations, were well attended.

Joint Scientific Sessions:— A very successful Joint-Meeting with the Ceylon Medical Association was held on the 12th and 13th of November 1970. Many distinguished delegates represented the Ceylon Medical Association. The detailed programme is given below. On the 12th night, a dinner was held at the Jaffna Town Hall for delegates and their guests.

We thank the Principal and Staff of the School of Nursing. Jaffna for letting us use their premises and also for all the help they gave us during these sessions.

We thank all the advertisers, who helped us publish the programme booklet for the Joint Sessions of the C. M. A. and J. M. A.

Our thanks also go out to Wyeth International Ltd., for screening 2 films of medical interest and serving cocktails at the dinner for the delegates. of C. M. A.

Social Activities:— Six dinners were organised during this period.

Epidiascope :— An epidiascope has been purchased for the association. Dr. K. E. de S. Karunaratne and Dr. S. Varatharajan helped in the purchase of this instrument and took great pains in getting it painted and renovated. It is hoped that contributors at subsequent meetings will make use of the epidiascope to project diagrams ECG records, tables etc. This will do away with the costly process of making slides to illustrate talks.

Library:— Miss P. Iyadurai has been appointed Library Assistant. Many new books were added to the library, the accent being on recognized medical monographs.

Departures :— The following members of the Council left us during the last few months: Dr. W. J. K. M. de Silya, Dr. S. Narendran, Dr. V. Sivagnanavel Dr. K. E. de. S. Karunaratne, Dr. K. Arumugam, Dr. T. Parameswaran and Dr. E. Kankesu. We thank them for all the work they have done for the association and hope that they will continue maintain their ties with this Association.

Obituary:— We wish to record with deep regret the death of Dr. S. Kumarasamy, one of our active members. Dr. Kumarasamy once worked at The Co-operative Hospital, Moolai and later at The Co-operative Hospital, Tellipalai and finally in a very busy General Practice at Sri Jothy Dispensary, Tellipalai. He was very popular wherever he worked, as evidenced by the long queues of patients waiting to see him. Inspite of this busy practice he would always find time to attend the association meetings and functions. He has made contributons to this journal in the past. A vote of condolence was passed and two minutes' silence observed as a mark of respect to the late Dr. Kumarasamy, at a meeting held on 12-7-71.

In conclusion, I wish to thank all members who helped me in my duties during the last one year. My special thanks to the President, Dr. V. A. Benjamin M. S., F. R. C. S. forwhaving taken a great load off the secretaries' duties.

N. Ganesbanandan,
Hony. Secy.

SUMMARY OF MEETINGS

Lectures

Date	Lecturer	Subject
12-10-70	Dr. A. E. D. Navaratnam M. R. C. P. (Lond.) Dermatologist, G. H. Jaffna.	Topical steroids in Dermatological Practice.
18-12-70	Dr. S. Nusbaum B. D. S., Visiting Oral Surgeon (Project Hope) Presently attached to the Peradeniya Dental School Hospital.	The Management of Facial Deformities.
26-12-70	Dr. V. T. Herath Gunaratne F.R.C.P. (Edin.) Regional Director for South East Asia— W. H. O.	The Role of the W. H. O. in the Developing countries
17- 1-71	Dr. R. Sri Pathmanathan L. R. C. P., M. R. C. S., L. D. S., F. D. S. R. C. S., D. L. O., Lecturer, The London Hospital U. K.	The Future of Medical Education in the U. K.
14- 6-71	PRESIDENTIAL ADDRESS by Dr. V. A. Benjamin M. S., (Cey.) F. R. C. S. (Eng.), F. I. C. S., Surgeon G. H. Jaffna.	Surgery of Prostatic Obstruction.

12- 7-71 Dr. J. St. George F. R. C. S. (Eng.) F. R. C. O. G. (Gt. Brit.) F. I. C. S., Senior Lecturer in Obstetrics and Gynaecology at the University of Trinidad. — Vaginal Discharges

Panel Discussions

27-10-70 Hypertension — Dr. M. Kanagarajah, Dr. B. A. Mills, Dr. S. Varatharajan, Dr. V. A. Benjamin

Medical Films

18-11-70 Diabetes — Shown through the kind courtesy of Pharma Distributors and Appukutty and Sons of Colombo

Clinical Demonstrations

5-10-70
Frontal lobe tumour
Hydronephrosis
Cases of Agitated Depression
Manic depressive illness
Multiple pulmonary emboli
Quadriplegia of obscure origin
(This meeting was held at the Base Hospital Point Pedro)

12-10-70
Multiple myeloma treated with cyclophosphamide and melpheran
Acute monocytic leukaemia presenting with hypotrophic gums.
Dysphagia due to systemic sclerosis
Achalasia cardia in a child
Serial X-rays in a case of amoebic liver abscess ruptured into the pleural cavity.
Long-term survival of patient with pulmonary secondaries

31- 1-71
Staphylocoocal panophthalmitis, meningitis, and pneumatocoele in a child.
Idiopathic diabetes insipidus
Diaphragmatic hernia
Gingivosis
Septic arthritis of hip in an infant
Ankylosing spondylitis
Spinal cord claudication treated by laminectomy
Bladder obstruction with vesical calculus
Dwarfism with hydronephrosis
Utero-vaginal prolapse with rectal prolapse.

22- 2-71
Continued fever for 14 months
Tuberculoma of the frontal lobe
Polyneuritis cranialis affecting the optic nerve
Meningioma of the spinal cord treated surgically
Prolapse of intervertebral disc
Saddle-embolus of the aorta following myocardial infarction treated surgically
Localised obstructive emphysema in an infant.

23- 3-71 Acute severe paralytic ileus following ingestion of manioc
Two cases of intra-abdominal tumours.

Joint Sessions of the Ceylon Medical Association and Jaffna Medical Association

12 11-70

Morning Sessions

1. Opening Ceremony
2. Paper—"Cardiac complications of the Hong Kong flu epidemic" by Dr. K. E. de S. Karunaratne M. D. (Cey.), M. R. C. P. (Lond.), M. R. C. P. (Edin.)
3. Paper—"Surgery in the jaundiced patient" by Dr. P. R. Anthonis F. R. C. S. (Eng)
4. Demonstration of clinical cases by Dr. K. Arumugam F. R. C. S. (Eng.)
   (a) Fibrous epulis
   (b) Pancreatic lithiasis
   (c) Secondary deposit in the umbilicus from an intra-abdominal tumour
5. Paper—"Massive dilatation of the left atrium" by
   Dr. N. Ganesharanthan M. B. B. S. (Cey.), F. R. C. S. (Eng.)
   Dr. (Miss) S. Kanthapillai M. B. B. S. (Cey.)
   Dr. R. Rajanayagam M. B. B. S. (Cey.)
6. Paper—"Inhibition of uterine activity in pregnancy and labour"
   by Dr. S. Rajanayagam F. R. C. S. (Eng.), F. R. C. O. G. (Gt. Brit.)

Afternoon Sessions

1. Paper—"Circumcision" by Dr. T. Kumarasamy M.B. B. S. (Cey.), M. R. C. P. Edin.), F. R. C. S. (Eng.), M. R. C. O. G. (Gt. Brit.)
2. Demonstration of clinical cases of Manic Depressive illness by Dr. T. Arulambalam M. B. B. S. (Cey.), D. P. M. (Lond.)
3. Paper—"Ligamentous strains" by Dr. T. Parameswaran M. B. B. S. (Cey.), M. Ch. (Orth), F. R. C. S. (Eng.), F. R. C. S. (Edin.)
4. Paper—"Our experiences in hemiplegia" by Dr. A. C. Jayasuriya M. B. B. S. (Cey.), D. Phys. M. (Lond.)

TEA

Evening Sessions

1. Paper—"Neurological conditions associated with the cervical spine" by Dr. G. S. Ratnavale M. D. (Lond.), F. R. C. P. (Lond.), L. M. S. (Cey.)
2. Paper—"Diagnosis of intra-thoracic tumours" by Dr. S. J. Stephen M. S. (Cey.), F. R. C. S. (Eng.), F. R. C. S. (Edin.)
3. Paper—"Spontaneous Intra-cranial Haemorrhage" by Dr. D. F. Weinman F. R. C. S. (Eng.), M. B. B. S. (Cey.), F. R. C. S. (Edin.)
4. Paper—"Ano-rectal abnormalities" by Dr. V. A. Benjamin M. S. (Cey.), F. R. C. S. (Eng.), F. I. C. S.
5. Clinico-Pathological Conference.
   Case presented by Dr. K. E. de S. Karunaratne M. B. B. S. (Cey.), M. R. C. P. (Edin.), M. R. C. P. (Eng.)
   Discussed by Dr. P. R. Anthonis F. R. C. S. (Eng.)

DINNER at TOWN HALL Jaffna

(Films on "Birth control" and "Open-Heart Surgery" were screened by Wyeth International (Ltd.) at the dinner.)

13-11-70

Morning Sessions

1. Paper—"Fallacies in blood sugar levels in diabetes" by Dr. J. Balachandra L. M. S. (Cey.)
2. Paper—"Hyperemesis Gravidarum" by Dr. T. Kumarasamy M. B. B. S. (Cey.), M. R. C. O. G. (Gt. Br.), M. R. C. P. (Edin.), F. R C. S. (Edin.) F. R. C. S. (Eng.)
3. Demonstration of the use of "Sonicaid" — an ultra-sonic blood flow detector in obstetrics by Dr. T. Kumarasamy.
4. Paper—"Some eye problems in childhood" by Dr. R. Pararajasegaram M. B. B. S. (Cey.), M. R. C. P. (Lond.), F. R. C. S. (Eng.), D. O. (Lond.)
5. Paper—"Vaginal Atresia" by Dr. R. Ramalingam M. B. B. S. (Cey.), F. R. C. S. (Edin.), M. R. C. O. G. (Gt. Br.)
6. Paper—"Treatment of hypertension with propranolol" by Dr. K. Puvanendran M. B. B. S. (Cey.), M. R. C. P. (Lond.)
7. Clinical case for discussion
   "Dysphagia and dilatation of loops of small bowel, due to systemic sclerosis"
   Case presented by Dr. N. Ganeshananthan M. B. B. S. (Cey.), F. R. C. S. (Eng.)
   Discussed by Prof. M. A. Paul M. R. C. P. (Lond.), F. R. C. S. (Eng.)

We regret the long delay in the publication of this number of the journal. Due to the unsettled conditions prevailing in this country a few months ago and the reluctance of some of our regular advertisers to advertise as a result, the publication had to be delayed.

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Manuscripts of articles for publication should be sent with a covering letter to the Editors, Jaffna Medical Association, General Hospital, Jaffna. Articles are accepted for publication on the understanding that they are submitted solely to this journal. They are subject to editorial revision.

Manuscripts should indicate the title of the paper, the name, qualifications, and full address of the author (or authors). The text should be in double-spaced typing on one side only with a wide margin.

Tables:

All tables should be on separate sheets and be capable of interpretation without reference to the text.

Illustrations:

Photographs submitted should be unmounted glossy prints. Drawings, charts and graphs should be drawn in black Indian ink on white paper. All illustrations should be clearly numbered with reference to the text on the back and should be accompanied by a suitable legend.

Nomenclature of drugs:

Only B. P. C. approved names should be used but in the case of new or unusual drugs the trade name should appear in parenthesis after the approved name. The initial letter of the approved name should be a simple letter and of a trade name a capital letter. All weights, volumes and doses should as far as possible be given in metric units.

References:

References should be indicated in the text by superior figures in order of appearance, e.g. "Burman and Gibson[3] investigated the E. C. G. changes .... etc."

The list of references should be set out in numerical order at the end of the article. Each reference should give in order the following: author's name and initials the year of publication (within parenthesis), the name of the journal (abreviations used to be according to the World List of Scientific Publications), the volume number (underlined with a wavy line), and the number of the first page of the article referred to;

e.g.—

3. Burman, S. O., and Gibson, T. C. (1963). Ann. Surg. 157, 134.

If reference is made to a book, the reference should give the following in order: author's name, year of publication (in parenthesis), title of book, volume, edition, page number of reference, city where published, and name of publisher;

e.g.—

Hewer, C. L. (1948), Recent Advances in Anaesthesia and Analgesia, 6th ed., p. 120. London: Churchill.

Abbreviations used in the articles should be those given below: —

ABBREVIATIONS TO BE USED

Blood Pressure	B. P.	Litre	l.
Centimetre	cm.	Metre	m.
Cubic millimetre	cu. mm.	Microgram	mcg.
Feet	ft.	Milliequivalent	mEq.
Fluid ounce	fl. oz.	Milligram	mg.
Gallon	gall.	Minute	min.
Grain	gr.	Molar	M.
Gramme	g.	Ounce	oz.
Hour	hr.	Percent	%
Incher	in.	Pint	pt.
International unit	i. u.	Pound	lb.
Intramuscular	i. m.	Yard	yd.
Intravenous	i. v.	Year	yr.
Kilogram	kg.		

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