A DISSERTATION OF TEN SURGICAL CONDITIONS

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This is to certify that the surgical conditions presented in this book were managed by Doctor Raymond K.M. Mwanga during his postgraduate training in general surgery under my direct or delegated supervision.

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ABSTRACT

This dissertation consist of ten surgical conditions studied entirely of my own choice, i.e. Four condition of general surgery, three conditions of paediatric surgery, one condition of urology and two of orthopaedics and trauma.

In every condition a case is presented followed by a brief analysis, literature review and discussion.

R.K.M.M

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CHAPTER ONE

BENIGN GASTRIC OUTLET OBSTRUCTION DUE TO PEPTIC ULCER DISEASE

ABSTRACT:

An account of 18 patients who underwent surgery due to peptic ulcer disease in surgical firms I and II between October 1980 and August 1991 is reviewed. The criteria used for selection of the patients included all those patients operated upon due to peptic ulcer disease. The source of information was the operation registry. Additional information was retrieved from their files. The age of patients ranged between 25–82 years with a mean of 51.9 years. Gastric outlet obstruction was the most common complication of disease (50%) followed respectively by perforation (33.3%), intractable pain (11.1%) and haematamesis (5.6%). There were 16 male and 2 female patients. Surgical management of gastric obstruction consisted mainly of truncal vagotomy and gastrojejunostomy. Two patients had truncal vagotomy and antrectomy. One patient, who had recurrent ulcer following a previous vagotomy and pyloroplasty, died due to postoperative sepsis after antrectomy.

INTRODUCTION:

Recent alternative terminology for pyloric stenosis is benign gastric outlet obstruction (B G00), the most common cause of which is peptic ulceration. In 75% of patients the ulcer is in the duodenum, and rarely in the pyloric or prepyloric region(s). Other uncommon conditions are lymphoma, ectopic pancreatic tissue and prepyloric mucosal diaphragm. In the Western series gastric outlet obstruction due to duodenal ulcer, is a common cause for surgery.[1] The introduction of H_2 antagonists has altered the role of surgery in

peptic ulcer disease.[2] It is possible that medical therapy only postpones the need for surgery in certain patients but the natural process of the disease remains unaltered.[2] Presumably, these patients are still prone to ulcers complications. The complications associated with duodenal ulcer and the frequency of elective versus emergency surgery, vary depending on the population studied.

Peptic ulcer disease is quite common in Africa. However, it is suggested that the low incidence reported is probably due to failure of diagnosis rather than actual incidence.[3, 4]

CASE REPORT:

S.A. a 58 years old male was referred from Medical Gastro Enterology to the Surgical Department. The main problems with the pateint had been epigastric pain of gradual onset followed by vomiting for five years. These attacks of pain usually lasted between 2–4 weeks with intervals of freedom of 3–6 months. The pain was described as burning and relieved by drinking milk about 2 hour after taking meals. His appetite had gradually deteriorated on account of feeling of fullness after meals. He thus experienced nausea and vomiting one to six hours after meals. The vomitus usually afforded a lot of relief and contained undigested food he had previously ingested. It contained no blood. He had lost weight and his bowel habit had changed in that it was less regular. Hitherto he had never been admitted to hospital but had attended peripheral hospital for which he was given tablets for the pain. He admitted to be smoking about 20 cigarette a day since at the age of 17 years but did not take alcohol. He was married with four children.

On physical examination, he was not wasted, not pale, jaundiced or dehydrated. The pulse rate was 68 per minute and BP was 130/90 mm Hg. The abdomen was not distended and there was no visible peristalsis though succussion splash was present. The rest of the abdominal examination and the other systems were normal.

He had an endoscopy done at the Medical Department which showed the stomach to be full of undigested food and a prepyloric ulcer with edema and scarry at the pyloric entrance and the duodenum could not be entered. A diagnosis of juxta–pyloric ulcer was made with gastric outlet obstruction. A biopsy had been taken and histopathology revealed a benign lesion.

Other investigations included a hemogram: HB 10.7g mll, WBC 5500 with normal peripheral film; serum creatinine 105mmo/l; Sodium 151 mmol/l and Potassium 4.8 mmol/l. Urinalysis and the chest X–ray were normal.

The patient was planned for surgery and two units of blood were grouped and cross–matched. One day prior to surgery a wide bore nasogastric tube was inserted for gastric lavage. A drip of Hartman's solution alternating with 5% dextrose at 8 hourly was instituted.

In the theatre, with the patient in supine position and under general anaesthesia, the site of the operation was prepared with savlon and iodine and draped. The abdomen was entered through an upper midline incision. The stomach was found to be grossly dilated and decompression was affected by active suction. The liver and intestine were retracted. The distal end of the oesophagus was located by palpating for the nasogastric tube. The overlying peritoneum was incised and a ribbon tap applied around the distal end of the esophagus. By gentle dissection over its anterior aspect with the assistant

maintaining a steady traction, the anterior vagal trunk was identified and divided between clamps. The posterior trunk was also identified and similarly divided. The stumps were ligated with catgut 2/0. The omentum and the transverse colon were held up by an assistant, while the most proximal part of the jejunum was located as it emerged from the fixed point of the duodenal – jejunal junction, and a segment of jejunum was marked with a pair of tissue forceps. A window of 8 to 10 cm. was made at the transverse–meso–colon through a relative avascular area, thus exposing the posterior wall and the most dependant part of the stomach. This was marked by a pair of tissue forceps. The corresponding portion on the jejunum was placed next to the stomach for anastomosis. A side–side anastomosis was then made in two layers using continuous catgut for through and through and interrupted silk for sero–muscular layer. The opening in the mesocolon was closed with sutures onto the stomach wall. The abdomen was closed in layers using continuous monofilament nylon No.1 for the rectus sheath and interrupted silk for skin.

Post operatively, the patient made an uneventful recovery. By the third day the nasogastric aspiration had decreased considerably and the nasogaastric and I.V fluid were therefore discontinued and he was allowed light meals. Stitches were removed on the 8th day and the patient discharged. Unfortunately, he was lost for follow-up.

DISCUSSION:

AGE AND INCIDENCE:

The actual incidence of peptic ulcer disease is difficult to assess due to lack of a widely accepted criteria for determing whether or not a subject has an ulcer.[5]

As mentioned earlier, the incidence of peptic ulcer diseases in Sub-saharan African though low, is probably due to failure of accurate diagnosis of the disease. In the West, special access to endoscopy service has indicated an incidence of 10% in the general population while 3–4% will have active peptic ulcer. Potentially serious complications occur in 20–40% of duodenal ulcer patients; 15–20% will bleed, 6% will peforate and obstruction will occur in another 2–4%.[6]

Gastric outlet obstruction in Western series is the second most common cause for surgery in duodenal ulcer.[1] In this series of 18 patients the commonest indication for surgery in peptic ulcer disease was gastric outlet obstruction. The reported reason for the frequency of ulcer complication being constant, despite the decrease in incidence of ulcer disease in general, is the increased proportion of ulcer patients who, because of the reduction of elective surgery, are on medical therapy. Although the ulcer diathesis may temporarily be suppressed in these patients, their ulcer disease has not been eradicated. It is therefore concluded that these group of active (but suppressed) ulcers are still prone to ulcer complications.[2]

Pathogeneses of duodenal ulcer and gastric outlet obstruction:

Peptic ulcer disease basically occurs due to an imbalance between luminal aggressive factors and mucosal defence mechanisms. The critical aggressive factor is the load at the mucosal surface which is the function of the hydrogen ion concentration (pH) and the duration of exposure. Acid is the most ulcerogenic factor in the pathogenesis of duodenal ulcer: thus, no acid no ulcer. Acid is produced by parietal cells under the influence of different

mediators which interact with unique receptor membrane system. Cholinergic agents stimulate acid through the M_2-type muscarinic receptors. Histamine binds to H_2-receptors and stimulates adenyl cyclase. Gastrin receptor has been described in few species. Unlike other mediators PGE 2 inhibits histamine stimulated acid production.

Acid regulation is a complex mechanism. It includes vagal activity, food intake, and gastrin produced by G-cells located in the antrum. Gastrin is released into circulation and acts indirectly on parietal cells via histamine containing cells. Gastrin production is under the influence of vagus and gastric pH. A rise in pH stimulates gastrin secretion.

At cellular level the membrane system has access into the cell through an intracellular secretory channels to the cell base. The canalicular membrane has the proton pump (enzyme H+/K-AtPase). When fully stimulated the pump produces isotonic hydrochloric acid (150mm) with a pH below 1.

Adequate blood flow is a major precondition for all the other protective events. Prostaglandin$_{I2}$ and E_2 present in the mucosae enhance blood flow and modulate protection on the inner side of the mucosae. The mucus barrier acts as a protective shield against mechanical and chemical influences. The bicarbonate secreted by mucosa surface cells builds up a protective pH gradient at the mucoal surface. The surfactant layer covering the mucosa becomes the hydrophobic barrier. The rapid epithelial regeneration protects the mucosae from erosion and injury. The counter protective factors include bile acid, lysolecithin from refluxing bile, strong alcohol, non-steroidal antinflamatory drugs and H. pylori whose role is still unresolved. Cigarette smoking is associated with impaired healing and high relapse rate and increased risk of complication.[7]

Over a period of 10–15 year with repeated attacks of peptic ulceration, there is healing and fibrosis. The pyloric channel or duodenal bulb becomes scarred and stenotic.[1] In active disease the obstruction may results from spasm or edema. Persistent gastric stasis of gastric contents stimulates gastrin secretion through antral mechanism which in turn increases secretion causing further irritation of the duodenum. A vicious cycle is created leading to continued gastric secretion and distension.

Vomiting is a late feature, but may become protracted leading to dehydration and metabolic disturbance. Metabolic alkalosis develops because of the continous loss of hydrochloric acid: The kidney responds by excreting excess bicarbonate to maintain the blood pH. Repeated loss of sodium in the vomitus coupled with loss of sodium, potassium and bicarbonate in urine leads to dehydration. The kidney attempts to correct the metabolic alkalosis by eliminating excess bicarbonate. Loss of potassium in the vomitus and urine leads to hypokalaemia. The initial alkaline urine becomes acid after a period of time owing to the hydrogen excretion (paradox aciduria). Signs of severe alkalosis and hypokalaenia are so interwoven that their clinical separation is well–nigh impossible. Severe form of alkalosis results in Cheyne–Stokes respiration with period of apnoea. Tetany may occur and the patient may become confused, irritable and lethargic.

CLINICAL FEATURES:

Vomiting is a common feature in patients with gastric outlet obstruction, occurring 30–60 minutes after eating. The vomitus is copious with recognisable partially or undigested food. Vomiting relieves pain and

fullness. The constant feeling of fullness and bloating causes loss of appetite. A visible peristalistic wave from left to right may be observed while a succussion splash is a characteristic finding.

Diagnosis is made by measuring the rate of gastric emptying, barium meal and or upper GI endoscopy. A withdrawal of more than 200 ml of gastric content four hours after a meal is evidence of delayed emptying. A saline loading test provides a simple measure of gastric emptying. Through a nasogastric tube 750 ml of 0.9% normal saline is instilled into the stomach and withdrawn thirty minutes later. An aspirate of more than 300 ml is suggestive of retention.

The saline load test (SLD) and standard barium meal, measure emptying of liquids only and may be normal. Barium burger test may demonstrate impairment of solid food emptying.[8]

Endoscopy allows quite an accurate assessment of the anatomical state of the gastric outlet and taking specimen for histological examination. Isotope scanning for visualization of gastric emptying shows considerable promise but has not been generally adopted.[9] Measurement of serum electrolytes, blood gases pH and are essential following protracted vomiting. In this review endoscopy and barium meal study were commonly used singly or as complementary investigation.

TREATMENT:

Specific guidelines regarding the management of gastric obstruction due to peptic ulcer disease are lacking due to the dearth of information concerning the natural history of disease.[8,10] The varying mechanisms causing

obstruction i.e. spasm, edema or fibrosis, make a statement about prognosis and recurrence rate difficult in patients treated either surgically or medically. There are series which predict recurrence rate of between 10–15% in 8 years after medical treatment.[11,12] Yet in another series about 90% of patients medically treated required surgery.[9]

CONSERVATIVE MANAGEMENT:

Medical treatment consists of giving adequate and appropriate fluids and electrolytes. Parenteral H2–blocker is administered. Gastric aspiration and decompression resolves the inflammatory edema within 72 hours. Parenteral nutrition is supportive in patients with chronic cases of obstruction and significant weight loss. Antispasmodic or anticholinergic agents are contra–indicated in patients with obstruction. Metabolic alkalosis without hypokalaemia rarely requires direct treatment. High urinary output is encouraged by good hydration with isotonic saline. Hypokalaemic alkalosis is usually corrected by adding an ampoule of 20 mmol of potassium chloride to each litre of fluid. This should not exceed 40mmol/hour unless EGG monitoring is available. Potassium should notbe given to oliguric patients.

H_2 – antagonists administered in a variety of regimens yeild an overal healing rate of 75% at four weeks and 85% to 95% at 8 weeks.[12] Risk factors like cigarette smoking and NSAID (non–steriodal anti inflamatory drugs) should be avoided.

SURGICAL MANAGEMENT:

The decision whether to operate or not for gastric outlet obstruction caused by peptic ulcer diseases should be based upon the patient's previous history, previous response to initial conservative management and estimated surgical risk. No standard protocol can be applied to patients. Weiland reported 98% of patients with gastric outlet obstruction due to chronic ulcer disease and 68% of those with acute gastric outlet obstruction required surgery.[9] Jaffin and Kaay in reviewing 55 patients noted a similar finding in that 90% of patients with chronic disease and 67% of those with acute gastric obstruction required surgical intervention.[13]

Suffice it to say that all patients treated medically, re–evaluated 72 hours later with a saline load test and found positive have a fixed outlet obstruction that is unlikely to respond to anything less than surgical correction.[14] Gastroduodenoscopy should be performed to confirm the mechanical nature of the obstruction.

The aim of surgery should be to relieve the obstruction and treat the underlying ulcer diathesis with the lowest possible mortality rate, recurrent ulcer rate and post operative gastrointestinal and metabolic side effects. To date there is no single operation which achieves such goals.[14]

Obstruction due to pyloric or prepyloric ulcers are best treated by truncal or selective vagotomy and antrectomy or vagotomy and a drainage procedure.[1,14, 15] Gastroenterostomy as a drainage procedure to accompany truncal vagotomy has been downgraded in America but is still held highly in the British Isles.[14]

Various authors consider it a drainage procedure of choice because it is associated with lower recurrent ulcer rate compared to pyloroplasty and that it maybe dismantled in patient with significant dumping syndrome.[15] In active inflammation or dense scaring gastrojejunostomy maybe the only alternative. Scaring may make pyloroplasty difficult.[1] Advocates of vagotomy and pyloroplasty are of the opinion that the lower mortality associated with this procedure compensate for the higher ulcer recurrence rate. Vagotomy and antrectomy is effective in ulcer healing but carries a slightly higher mortality rate of 2.3%. It is probably best suited for a strong young man. It is less suitable in an emergency situation. The table below compares results of operation commonly used in treatment of duodenal ulcer.[14]

	Mortality Rate %	**Recurrent Ulcer Rate** %	**Side Effects** %
Truncal vagotomy and antrectomy	0.6 – 1.8	1 – 2	13 – 29
Truncal vagotomy and drainage	0.5 – 1.4	5 – 15	11 – 26
Proximal gastric vagotomy	0.1 – 0.3	5 – 16	3 – 8

✓ Complications associated with truncal vagotomy and drainage procedure include obstruction of pyloroplasty or gastroenterostomy leading to gastric stasis and ulcer, loss of gastric motility and delayed emptying. Stomal ulcers and leaking anastomosis may lead to stomal obstruction, fistula, peritonitis and death. Loss of pyloric sphincter mechanism may cause duodenogastric reflux leading to bilious vomiting and chronic gastritis. Selective vagotomy used in combination with a drainage procedure was introduced in the belief that it would reduce some of these undescribe effects. It has a low mortality but clinical experience with selective vagotomy has not demonstrated it to be superior to truncal vagotomy in treating duodenal ulcer.[14]

CONCLUSION:

The incidence of benign gastric outlet obstruction due to peptic ulcer disease is a common indication of gastric surgery in this hospital. The advent of H_2 blockers and advances in the diagnosis of peptic ulcer disease has radically changed the management ofpeptic ulcer disease. The impact of it in our situation has yet to be determined. Perhaps more interesting is the role of surgery in this disease.

The incidence of ulcer perforation is epidemiologically taken to reflect the incidence of ulcer complications.[2] Yet in this study the author noted a slight increase in the incidence of obstruction compared to perforations. An analytical conclusion is difficult because of the small size of the study population. But it would be interesting to study the incidence of complications during this era – of H_2 receptor antagonist. Recommended areas of study would be to see what percentage of patients actually have access to H_2 receptor blockers. This drug is prohibitively expensive for an average Tanzanian. In this series of 9 patients only 4 patients had access of H_2 – blockers.

There were 16 males and 2 females (8:1). Male patient were almost uniformly affected and all groups were represented except those at 60–69 years of age group.

Table I **Age, Sex distribution of Patient**

No	Age	Male	Female	Total	%
1.	20 – 29	3	–	3	16.7
2.	30 – 39	2	–	2	11.1
3.	40 – 49	4	1	5	27.8
4.	50 – 59	2	–	2	11.1
5.	60 – 69	–	0	0	0
6.	70 – 79	1	–	1	5.56
7.	80+	1	–	1	5.56
	TOTAL	16(88.9%)	2(11.1%)	18	100

Table II **Frequency of Presentation**

	Frequency	
Indication for operation	No.	%
Intractable pain	2	11.1
Haematamesis	1	5.6
Perforation	6	33.3
Obstruction	9	50
TOTAL		

Two patients with obstruction to gastric tumours are excluded. Therefore gastric outlet obstruction was the commonest indication for operation for Peptic Ulcer disease followed by pefocation, intractable pain and vomiting of

blood, in that order.

TableIII TYPE OF SURGERY AND INDICATION

Indication	Single closure	TV + pyloroplasty	TV+ Gastrojejunoctomy	TV+ Antrectomy	Gastrec + tomy Drainage	%
Intractable pain	–	1	1	–	–	10
Perforations	3	1	–	–	–	20
Haematemesis	–	1	–	–	–	5
Obstruction due to actic PU	–	–	7	2	–	45
Obstruction due to tumour	–	–	–	–	2	10
	3(15%)	3(15%)	8(40%)	2(10%)	2(10%)	100

The most common type of operation in these series was truncal vagotomy and gastrojejunostomy. The main indication was obstruction due to Duodenal ulcer.

References

1. Stanton P.D : Results of surgery for duodenal ulcer Br. J. Surg. 1991:78;815

2. Paul H.: Surgery for peptic ulcer : Curr. Pro. Surg. Problem 1991; xxviii:308

3. Davey's A.D.: Companion to surgery in Africa 2ED.

4. Editorial : the declining role of the surgeon in the treatment of Acid – peptic Disease : Arch. Surg. 1980;115:134.

5. Kurata J.H., Haile B.M.: Epidermology of peptic ulcer disease clinic in Gastroenterology 1984;13:2

6. Mitchell M.: Conservative surgery in Duodenal Ulcer : a series of vagotomy and drainage procedure reported from Dar es Salaam: E. Afr. Med. J. 1966:43(3)78

7. Prof. Dr. Med. Schwenk. Pathophysiology of condition related to hyperacidity: Research and clinical forum vol. 10. No. 2

8. Rinthol R.F: farquharson's text book of operative surg. 6ED Churchil Livingstone.

9. Bell CJ Stiel D.: Long term management of Peptic Ulcer. Medicine Digest. 1991:17.

10. Howard M.S.: Clinical Gastroenlorology 2ED 1977 Bailliere Tindall. and J.: Helicobacler pylori : Med Digest. 1990:4

11. Boyls J.D.: Management of pyloric obstruction Med. clinic Nort. America 1966:52.

12. Goldstain H.: Gastric retention associated with gastroduodenal disease: Am J. digest. Dises 1966;11:897

13. Bruce E.S: Current surgical management of duodenal ulcers : Surg. Clinic of North America 1992 72(2):351–352.

14. Jordan H.: Surgery for peptic ulcer disease Curr. Probl. Surg. April 1991.

15. De Mattes: Vagotomy and Drainage for obstructing duodenal ulcer : Am J. Surg. 1974:127

CHAPTER TWO

RECTAL CARCINOMA

ABSTRACT:

A review of nine patients with rectal carcinoma seen between October 1989 and July 1992 is presented. The ages ranged between 34 – 67 years with a mean age of 50 years. There were 5 males and 4 females, presenting a ratio of 1.3:1. Most patients presented between 3 months to 24 months from onset of symptoms and at histopathology the majority of tumours were adenocarcinoma. The main modality of treatment were surgery and chemotherapy. Radiotherapy was given to those with advanced disease. There was no mortality.

INTRODUCTION:

Colorectal cancer is a global disease with a variable geographical, social and ethnic pattern. It is the most common malignancy of the gastro–intestinal tract in the western countries. The disease is usually diagnosed after the fourth decade with males being slightly more affected than females. The disease is prevalent in developed countries in those in the higher socioeconomic group. The varying incidence suggests a strong environmental influence.

Pre–malignant conditions include adenomatous polyps, ulcerative colitis and Crohn's disease. The disease is also related to genetically determined conditions like Gardner's syndrome and familial polyposis. The degree of differentiation and extent of spread have a prognostic implication and influence the choice of treatment. The primary modality of treatment is

surgery with an aim to provide cure while minimising disability. In most patients this should now be possible following a better understanding of the pathology and refinement technique.

The following is an account of nine patients with rectal cancer. One case is presented in detail while the rest are reviewed briefly. A review of literature and discussion of relevant aspects of the subject presented.

CASE REPORT:

KM a 59 years male patient IP 612178 presented at the outpatient clinic with chief complaints of pain in left lower abdomen for a duration of 2 years. The pain was constant and got worse a few moments before defecation. Flatulence was a constant feature. His bowel habit had gradually changed from normal, to a passage of loose mucoid stool and at times mixed with fresh blood. At one of the peripheral hospitals he had been found to have amoebiasis and was treated with a course of metronidazole but without response. His appetite was normal and he had not lost weight. Micturition was normal. He was a retired civil servant, married with eight children. He took alcohol in moderation but did not smoke. His diet constituted of local staple.

On examination he was not wasted, pale or jaundiced. The pulse was 78 per minute and a blood pressure of 140/80 mm Hg. There was no ankle oedema and no lymphadenopathy. The abdomen was soft with nonpalpable mass. The liver, spleen and kidney were not palpable. Rectal examination revealed a normal rectum with no ulcer or mass and no hemorrhoids. There was however blood on the examining finger. The other system were normal.

A provisional diagnosis of recurrent amoebic dysentery was made. A fresh stool examination showed Entamoeba hystolytica cysts. He was put on a course of tinidazole and diloxamide furoate. At follow–up the stool revealed no further trophozoite. However, this time hookworm ova were seen and a course of mebendazole was given. The patient had but just a brief improvement with recurrence of lower abdomen pain and passage of mucoid and blood stained stool. A proctosigmoidoscopy was therefore performed and this revealed an exophytic mass 15 cm. from the anal verge. The mass had a broad base and was one quarter circumferential. A biopsy was done and reported as a well differentiated adenocarcinoma. Other investigation included Hb 14.5 gm./dl : WBC 6600 with a normal peripheral film.

Serum Creatinine was 92/mol/l and liver function tests were normal. Chest x–ray revealed a left ventricular enlargement with no lung metastasis, while the ECG showed left ventricular hypertrophy. Utrasound was not possible due to technical problems in the Centre.

A decision was made to operate and an anterior resection was planned. This was communicated to the patient who agreed to have surgery. Two units of blood were cross–matched and reserved. The patient was reviewed by an anaestheologist and considered fit for surgery. Barium enema and ultrasound before surgery were planned but were not done due to technical problems existing at the time.

Preoperative bowel preparation was started three days prior to planned surgery with a low residual diet, laxatives and colonic washouts. Oral metronidazole one day prior to surgery and parenteral gentamicin on the day of surgery were prescribed. The patient was catheterised on the day of the operation.

Under general anaesthesia and the patient in Trendelburg position, the skin was routinely cleaned with iodine and spirit and draped. The abdomen was opened through a left lower paramedian incision extending slightly above the umbilicus. There was no ascities, and examination of the liver and the rest of the colon or pelvic organs showed no evidence of metastasis. At the level of the upper rectum there was a tumour which was mobile, involving the full thickness of the wall. A few lymph nodes were detected along the mesentery. The loops of the intestines were gently retracted and the sigmoid colon mobilised by dissecting the peritoneum along the lateral boarder of the colon. The left ureter was identified and safeguarded. The inferior mesenteric artery was identified and ligated between clamps and severed. Intestinal clamps were placed 10cm proximal to the tumour 5cm distally. Resection was done between clamps. An end–end anastomosis was carried in a single layer fashion using interrupted silk No. 2/0. The pelvic peritoneum was repaired with chromic catgut 2/0 and the abdomen closed in layers with catgut 2/0 for peritoneum nylon, rectus sheath and nylon 3/0 for the skin.

Post operatively he was prescribed pethidine 100mg 6 hourly for pain. dextrose/saline 3 litre 8 hourly and parenteral gentamicin and metronidazole for 72 hours were also prescribed. His vital signs were monitored hourly for the first 12 hours than 6 hourly for the next 48 hours.

The patient made an excellent recovery and on the 8th day all sutures were removed. He made three visits at the follow–up clinic in a span of 3 months. The resected specimen was reported as a well–differentiated adenocarcinoma of the rectum, Duke stage B with no lymph node involvement.

Ten months later he revisited at the outpatient clinic with attacks of constipation, abdominal cramps and passage of mucoid stool. A barium enema showed irregular narrowing at of the anastomotic site. The contrast however passed freely beyond the stricture. Proximaly the colon was normal. Sigmoidoscopy was performed but the mucosae appeared normal and a biopsy report showed no evidence of recurrence of any tumour. He was seen 6 week later in good health with no significant change in bowel habits but was then lost for follow–up.

DISCUSSION:

Colorectal cancer is the most common cancer of the gastrointestinal tract in the West. The average annual age standardised incidence rate. (ASR) is 30 per 100,000. It is 15/1,000,000 in the USA with 14.3 (Blacks) and 15.4/1,000,000 (white),England and Wales 13.7, Australia 20.7, Japan 7.7, India (Bombay) 7.7, China (Shanghai) 9.4. The incidence of colorectal cancer increases with age. The disease is predominantly diagnosed after the 4th decade with peak incidence in the 5th decade. Only 4–8 percent of these malignancy are diagnosed below the age of fourty.[1] This review correlates well with findings elsewhere.[2, 3, 4] The commonly affected group was in the 4th and decade the male to female ratio was 1.25:1, (refer to table I below).

MEDICAL UNIVERSITY LIBRARY

TABLE I SEX and AGE DISTRIBUTION

Age	Male	Female	%
30 – 40	2	–	22.2%
41 – 50	–	3	33.3%
51 – 60	2	1	33.3%
61 – 70	1	–	22.2%
TOTAL	5	4	

AETIOLOGY:

The higher incidence of cancer of the large bowel in the USA and Western Europe compared to Japan and Eastern countries, has led to speculation that the aetiology is environmental.[2,5,6] With the notable exception of Japan, the disease is prevalent in the developed countries. Even in the less developed countries the disease is predominant in the higher socioeconomic groups.[6] Studies of migrant groups have revealed that the risk of colorectal cancer is similar to that in the land of their adoption. Japanese immigrants to Hawaii and California, Puerto–Rican and black immigrants to USA all have a higher incidence of cancer than their counterpart in their place of origin. However, the actual environmental influences remain obscure. Dietary factors seems to be accepted as having a significant role in the aetiology and a number of controversial hypothesis have been postulated. Oettle (1960) and later Burkitt(s) have proposed the dietary fibre hypothesis. The incidence of large bowel cancer in Africa is lower compared to the West. While at the same time the stools are bulkier. It was therefore suggested that high fibre diet reduces intestinal transit time and logically reduces contact of the large bowel to potential carcinogens.[7] Wynder, on the other hand, hypothesised that consumption of diet high in animal fat as responsible for colonic cancer. A diet rich in fat is accompanied by an increase of secretion of bile acid, neutral steriods and bacteria in the faeces which metabolise the fecal substrate to Methylcholantherene a potentially carcinogenic substance.[3] However, these theories associating diet as a causative factor have not been conclusively validated.[6]

Ulcerative colities, Crohn's, disease and adenomatous polyps are pre-malignant conditions.[8,9] They have a familial component and, in part, are genetically determined. Gardner's syndrome and familial polyposis are autosomal dominant conditions. Familial polyposis is associated with a 100 percent risk of cancer by the age of 40 years unless prophylactically treated by surgery.[10] Adenomatous polyps are associated with a risk of 4–5 percent of malignant change. The actual risk of malignancy is related to size so that the risk is 1 percent in a polyp of less than 1cm while the risk increases to 35 percent in those greater than 1cm. Villous adenomas greater than 2cm in size have a risk of malignant change of 53 percent.[9] The overall incidence of cancer in ulcerative colits has variously been reported to be between 2–5 percent. The cumulative risk increases with the duration of the colitis.[10]

In the author's series, three patients had pre-existing malignant conditions; condyloma acuminata in one and villus adenoma in two.

PATHOLOGY:

Macroscopically rectal tumours can be polypoid, ulcerative, infiltrative or annular. Histologically the majority of rectal carcinoma are adenocarcinoma but may differ considerably in their cellular differentiation.

TABLE II HISTOLOGICAL GRADING

Grading	No. of patients
Well differentiated Adenocarcinoma	4 (44.4%)
Moderately differentiated Adenocarcimona	1 (11.1%)
Well differentiated mucinous Adenocarcinoma	2 (22.2%)
Others: Squamous cells carcinoma	2 (22.2%)
TOTAL	9 (100%)

In this series (table II), well differentiated adenocarcinoma constituted four patients (44.4 percent) which was the leading histological type followed by well differentiated mucinous adenocarcarcunona two patients (22.2 percent). Moderately differentiated adenocarcinoma 11.1 percent (1 patient) and squamous cells carcinoma one patient (11.1 percent). Broder's system of histological grading are used to predict the biological behaviour of these tumour.[9,11,12]

The spread of rectal tumour may occur by direct extension to adjacent structures or lymphatic spread via intramural lymphatic plexus in the submucosa and subserosa. These in turn drain into extramural groups of lymph nodes, the epicolic which lie along the colon, pericolic along the marginal artery, intermediate along the main vessel and the principle group along the inferior mesenteric arteries. The importance of local invasion and it's prognostic implication was postulated by Duke in 1930. The modified clinicopathological classification proposed by Astler – Coller, is the one commonly used in this Centre (Appendix).[9,12]

Cancer spread distally is rarely more than 2–3cm, except in the anaplastic variety.[13] This fact bears surgical significance. The proximal lymphatic spread follows the superior hemorrhoidal vessels, the middle rectum drains upwards and downwards along the middle inferior vessel while the lower rectum above the peritoneal reflection can be considered as downwards.

CLINICAL FEATURES:

Colorectal cancer presents usually as an alteration of bowel habit in the form of constipation, diarrhea or a combination of the two. Most often patients put little emphasis on this symptom until profound alteration occurs. Bleeding per rectum is a common symptom and occurs in 35–40 percent of cases. There is mucus discharge often mixed with stool. Rectal pain is late feature. Constitutional features include weight loss, anaemia and feature associated with involvement of adjacent structures or distant metastasis. Physical examination may reveal evidence of weight loss and anaemia. The liver should be examined for metastasis. In all but the occasional case the general and abdominal examination may fail to reveal any significant abnormality. Digital examination, rectosigmoidoscopy and barium enema are complementary examinations. About 75 percent of rectal cancer or 35 percent of cancer of large colon lie within the reach of examining finger.[9] Protosigmodoscopy examination is indispensable. A biopsy is taken for histology.[11] Barium enema is useful in excluding a synchronous colonic lesion. Pre–malignant lesion are excluded by colonoscopy or double contrast barium enema. Feacal occult blood has a limited value in making a diagnosis because of the unacceptable false positive. Its role as a screening test for a population risk is undisputed. A combination of double contrast, and rectosigmoidoscopy in primary assessment of patients with positive hemoccult test is reported to have sensitivity of 94 percent and specificity of 99

percent.[14] Carcinoembryonic antigen is a tumour marker although its value is limited except as an indicator of prognosis. A subsequent elevation (of CEA) after a return to normal following an excision of rectal cancer may signify recurrence. Intermediate result albeit elevated levels indicate incomplete excision. The antigen is detected in patients with cancer of the breast, stomach and lungs.

Endoluminal ultrasound is a promising modality for pre–operative staging. It can determine the depth of invasion of the tumour and involvement of peri–rectal nodes. The correlation of endosonogram and histological assessment of depth of invasion into rectal wall is about 90 percent.[15,16] It can also be useful for follow up of local recurrence.

TREATMENT:

Surgical resection remains the primary mode of treatment. The aim of treatment is to provide and prolong cure while minimising the immediate complication and long term disability. Recent advances include new developments and refinement in sphincter–saving procedures.

SURGERY:

The choice of operation depends on the level of the lesion from the anal verge, and the histological differentiation and grade.

The rectum is arbitrarily divided into surgical thirds from the anal verge.

TABLE III SITE OF TUMOUR

Level	No of patient
Lower third	7 (77.8 %)
Middle third	1 (11.1 %)
Upper third	1 (11.1 %)
TOTAL	9

The lower third is 3.5–7.5cm, mid–third 7.5–12cm and proximal third 12–16cm.[11] For tumours located in the lower third, the anal sphincter cannot be preserved and an abdomino–perineal resection is considered the procedure of choice.[11] Proximal third tumour are generally amenable to anterior resection. The surgery for tumours located in the middle third is surrounded by controversy. Too often surgeons in a zealous effort to avoid a colostomy compromise the margin of resection with a tragic result of a recurrence of disease, anastomotic leak and obstruction. Abdomino–perineal resection is no panacea. The final decision eventually cannot be made until on the operating table, and especially if the rectum cannot be completely mobilised from its attachment with adequate normal rectal margin distally. In well differentiated tumour distal spread is rarely more than 2cm unless the lesion is obstructing.[11] The microscopic characteristic does determine the biological behaviour of tumour and thus the type of operation. The more anaplastic the tumour the more aggressive and hence the greater resection margin. Generally the distal margin of resection should not be less that 5cm distal to the tumour margin.[17] This recommendation was based on pathological studies which demonstrated that in only a few patients will rectal cancer extend distally for a distance of 5cm or more.

Anterior resection is a sphincter sparing procedure. It is suited to carcinoma located in the upper third of the rectum. The approach is through a left paramedian incision. The tumour is resected 5cm distal to the lower margin of the tumour and proximally near the junction of the descending colon and sigmoid. The ligation of venus and lymphatic vessel prior to tumour mobilisation associated with the use of cytotoxic solution is believed to minimise the hazards of malignant cell embolisation.

Low anterior resection is recommended as an alternative to abdomino perineal resection for low or mid rectum tumour up to 5–12cm to the anal–verge. The operation involves complete mobilisation of the rectum, division of the lateral ligament and middle hemorrhoid arteries both of which are considered to contain local metastasis. Anastomosis is affected in the rectum distal to the visceral peritoneum. It is technically difficult and associated with a risk of anastomotic leak. Other complication includes obstruction at the anastomosis, pelvic abscess, faecal fistula, and anastomotic bleeding. Circular stapling device is advantageous in low anterior resection as low as 5cm from the anal verge.[13] This device is not available in MMC. Late complication of low anterior resection include stricture secondary to anastomotic breakdown and fibrosis, incontinence and irregular bowel movements of urgency and frequency.[9] As long as the anal canal and sphincter muscles have been reserved the symptoms will resolve with time.[9]

Abdominal–perineal exision of the rectum (APER) is the procedure for tumours located in the distal third of the rectum. The operation involves an abdominal resection through a left paramedian incision and the mobilisation of the rectum through an elliptical incision around the anus. These two procedures maybe done synchronously. The distal colon and the rectum are removed. The colon proximaly is mobilised and brought out as a terminal

colostomy. The operation entails removal of rectum, sufficient mesorectum and lateral ligament pelvic musculature, ischio-rectal fat, external anal sphincter and perineal skin. Failure to do so is a factor of prime importance in local recurrence. Mortality following abdominoperineal resection is between 1–6.5 percent and the incidence of complication 61 percent.[9,18] Urological complication like ureteric and bladder injury being the most common. The uncorrected five year survival is 90 percent in patient with Dukes A, 65 percent in Dukes B, and 33 percent to Duke C.[9]

Pull through procedure is performed for tumours located between 5–10cm. The tumour is resected and by stretching the anal sphincter the ano–rectal stump is drawn outside. The proximal colon is pulled through and anastomosed to the everted ano–rectal stump. The anastomosis retracts spontaneously in the pelvic cavity with 10–14 days. Cure rates and problems of recurrence are comparable to abdomino perinial resection.

Abdomino–sacral resection is an alternative procedure for middle third rectal tumour of low grade malignancy. The approach is through resecting posteriorly the coccyx and distal portion of sacrum. A major complication is associated with posterior fistula formation and partial dehiscence.

There are few reports in which a sphincter saving resection (SSR) has been compared with Abdominal–perineal exision of the rectum (APER). In a review of several series there was no significant difference noted in the overall survival rates between the two operations.[13] Studies which attempt to examine recurrence rates are fraught with difficulties. Patient may have metastasis which cannot be detected by clinical or conventional or radiological methods, the role clearing of the mesorectum hence becomes significant. Few studies available report local recurrence of cancer in the

lower third of rectum from nil to 32 percent.[13] Patients who do not have tumour invasion through the full thickness are ideal candidates for curative sphincter saving (SSR) surgery having a five–year survival of about 80 percent. Patient with advanced disease (Dukes stage B, or above) have five–year survival ranging between 20–50 percent.[19] These patients are ideal candidates for adjuvant therapy. Tumours within 15 cm. of anal verge that are stage Dukes B_2 or above may benefit from post operative adjuvant radiation therapy.[18,19] The role of preoperative radiation therapy has not proven beneficial.[18] There is no effective adjuvant chemotherapy program that is currently recommended 5–FU has been used in combination with other modalities of treatment.[18,19] Advanced or metastatic rectal cancer are best treated with palliative surgery or radiation. Chemotherapy may be an effective radiation sensitizer.[18] When the tumour has involved contagious organs or is fixed surgery is associated with a higher–operative morbidity and mortality.[9] Hartman's operation is an excellent alternative in the very sick or old patients who cannot withstand the trauma of major surgery, in an obstructing rectal tumour.

TABLE IV TYPE OF OPERATION

TYPE OF OPERATION	No of patients	%
Loop colostomy alone	–	-
Anterior Resection	1	(11.1%)
Abdomino perineal		
resection alone	5	(55.6%)
Chemotherapy + AP	1	(11.1%)
Palliative Radiotherapy with loop colostomy	3	(33.3%)
TOTAL	9	100%

CONCLUSION:

Colo–rectal carcinoma is a common condition as seen at the Muhimbili Medical Centre. Adenocarcinoma is the most common histopathological type. In this series the main mode of treatment was surgical, depending on the stage and site of lesion.

References:

1. John BS. : An. J. Surg. Dec. 1988 : 156 : 460.

2. Whelan S. L. : Pattern of Cancer in five continents: IA RC, Scientific Publication No. 102 1990 Lyon.

3. Wynder E. F. The epidemiology of large bowel cancer: Cancer Research : 1975 : 35 : 3388.

4. Lyon 1987 : Cancer incidence in Five continent in International agency for research of Cancer (IARC) volume V WHO Publication.

5. Hughes RG. : The prognosis of Ca of colon and rectum complicating ulcerative colilies : Surg Gynaecol. obstet:1978;146:46.

6. Stubbs RS : Ecology of colorectal cancer Br. J. Surg: 1983 : 70(6) : 313.

7. Burkit D P.: Epidermology of Cancer of the colon and rectum : Cancer : 1971 : 28 : 3–13.

8. van Herden JA: Carcinoma of the colon and rectum complicating chronic ulcerative colities : Disease. colon and rect.1980:23(3):155.

9. Corman ML.: Textbook of colon and rectal Surgery 1984 : JB Lippincott Ccompany Philadelphia.

10. Warren H. : Cancer of colon and rectum Surg. clinic. Nort. AM.Aug. 1972:52:(4):871

11. Golgher JC. : Dukes A. B. C. categorisation of the extent of spread of Ca of rectum. Surg. Gynaecol. Obstet : 1976 : 143 : 793 (B) Text Surgery of Anus and rectum and colon, 1975, 3ED Bailliere Tindall.

12. Philip R : Clinical Onclolgy : 5th ED, 1978 Chapt X page 85 by American

cancer society.

13. Williams N.S. : The rationale for preservation of anal sphincter in patient with low rectal cancer : Br. J. Surg. 1984 71(8) : 575 – 581.

14. Johnson J. : Double contrast barium enema and flexible rectosigmoidoscopy. Br. J. Surg. 1990 : 77(3) : 270.

15. Failel T : Assessment of depth of invasion in rectal endosonography : Endoscope 1987 19:64 – 67.

16. Thoeni RF. : CT evaluation of carcinoma of rectum and colon : Radio Clin. Nort. Am. 1989 : 27 : 721.

17. Current Problems in surgery May 1990 : 27 : 5.

18. Beard. RW. Colon, Rectum and Anus : Cancer 1990, 65(3) 684.

19. Gastrointestinal tumour study group : Prolongation of the disease – free interval in surgical treated rectal carcinoma. New Eng. J. Med : 1985;312(23):1465

APPENDIX I

Modified Clinicopathological classification

(Astller – (coller) [9]

Stage	Classification	TNM
A	lesson limited to mucosae	NO
B_1	lesion limited to muscularis propria	
B_2	lesion penetrating muscularis propria. no nodal involvement	
C_1	lesion limited to wall. positive nodal involvement	
C_2	lesion extending to all layers. positive nodes	
D	Distant metastasis	

CHAPTER THREE

PATENT DUCTUS ARTERIOSUS

ABSTRACT:

Four patients with Patent ductus arteriosus (PDA) attended the Thoracic Surgical Unit at the Muhimbili Medical Centre from June to August 1991 . All patients except one was below 10 years of age. Females formed the majority with the ratio of 3:1. The main presenting features were recurrent upper and lower respiratory infections and failure to thrive. All patients had PDA closure through left thoracotomy. The period of hospital stay ranged between 5–11 days. There was no mortality. Diagnosis was confirmed by echocardiogram.

INTRODUCTION:

PDA: PATENT DUCTUS ARTERIOSUS

The era of surgical intervention for correction of congenital heart diseases began about 50 years ago. The first successful closure of PDA was performed in a seven years old girl in 1938,[1,2,3] thus introducing the use of surgical treatment for congenital heart disease. Treatment of PDA is representative of the rapid advances made in thoracic surgery.

The incidence and mortality attributed to congenital heart diseases varies among different populations depending on the diagnostic method employed. Over the last two decades there has been a growing awareness and recognition of heart diseases in Africa and in particular congenital heart defect.[4] Congenital heart defects have been recognised as an important cause

of morbidity and mortality among our children. The need for early surgery is reinforced by the finding that PDA is not a wholly benign condition in childhood.[5] Many authors consider the optimum period to carry out elective PDA closure to be between 2–5 years of age,[1,2,6,7] when operative mortality experienced is less than 0.5 percent provided associated cardiac anomalies have been corrected.

The following is a review of four patients treated at the Thoracic Unit. One patient is presented in detail, backed with a review of the relevant literature.

CASE REPORT:

A female child age seven years had presented at the paediatric cardiac clinic due to recurrent respiratory attacks and failure to thrive since the age of four months. The mother had noted an abnormal pulsation on the child's chest associated with retardation in physical growth.

The child was born after a full term gestation. During the antenatal period the mother had not suffered from any abnormal rash or undue fevers. The child was delivered in hospital as a breach presentation. She experienced a delay in crying at birth. She was discharged home after three days in apparent good health and she was weaned at four months of age. She received primary immunization. The child is a second born in a family of two. The first born is alive and well. The father is a peasant while the mother is a housewife.

On examination the child was underweight. She had a radial pulse of 100 beats per minute which was regular and collapsing. She was not cyanosed or dyspnoeic. There was no toe or finger clubbing. The precordium was not bulging.

The apex beat was located in the sixth intercostal and slightly lateral to the midclavicular line. There was an apical heave but no parasternal heave. The first and second heart sounds were heard and normal. The was a systolic thrill on left sternal border on the 2nd interspace with a continuous machinery murmur maximally heard at the 2nd right sternal boarder. The liver and spleen were not palpable. Examination of other systems were essentially normal. A diagnosis of PDA was made and confirmed by Echocardiogram.

INVESTIGATION:

Chest X–ray:	Showed features of cardiomegaly and plethoric lung fields with a cardiothoracic ratio of 62 percent.
Electrocardiogram:	Heart rate was 167 beats/minute with sinus rhythm, normal axis and slight left ventricular hypertrophy.
Serum electrolyte:	Sodium 140 mmol/l Potassium 4.3 mmol/l
Creatinine:	88 mmol/l
Full blood picture:	HB 10.3g/dl and a normal peripheral film
Stool/Urinalysis:	No abnormality was detected

The patient was reviewed by the anaestheologist and blood was grouped and cross matched. Consent for operation was obtained from the parents.

At the operating table the operation site was cleaned and draped. Through a left thoracotomy in the fourth interspace incision the chest wall and the pleura was opened. The lung was retracted medially with a malleable retractor. The vagus and recurrent laryngeal nerve were identified and preserved. By tracing the recurrent branch of the vagus, the ductus was readily seen over the aorta.

The adventitia was gently released. A thrill was present on palpating the ductus. The ductus was doubly ligated with number 1 linen suture commencing on the aortic aspect and divided. A chest tube was inserted. The pleura was re–approximated over the area of dissection with a running catgut 2/0 suture. The ribs was approximated with nylon No.0. The muscle and skin were later closed. The chest tube was thereafter removed at full expansion of the lung.

Postoperative injection of pethidine (25mg, 6 hourly) was given for pain. Intravenous fortum 1gm 12 hourly for 48 hours and 5 percent dextrose was run at 50ml/hr for 24 hours. The patient was admitted into the Intensive Care Unit (ICU) for monitoring.

After 24 hours the child was transferred to the Paediatric Surgical Ward. The general condition was good. On the fourth day she developed a fever. A thick blood smear indicated malaria and she respond to antimalarials. On the fifth day all stitches were removed and the patient referred back to the medical paediatric cardiac unit.

Sex and Age distribution of the Case Studies

No.	Age.	Sex	Age at onset of symptoms
1.	6 years	F	3 months
2.	7 years	F	4 months
3.	5 years	F	5 years
4.	7 years	M	2 years

RESULTS AND COMMENTS:

These four patients were referred from the Paediatric Cardiac Clinic to the Thoracic Surgical Clinic during my rotation between August 1992 and October 1992. As represented in Table I, ages ranged between 5–7 years with females forming the majority of patients.

The main symptomatological feature were recurrent upper and lower respiratory infections. All patients clinically presented with a collapsing radial pulse, and continuous machinery murmur. All patient were investigated with ECG and echocardiogram for definite diagnosis and exclusion of other cardiac anomalies.

Post operative hospital stay ranged between 5 and 11 days. There were no post–operative complications or wound sepsis. There was no mortality either. Except for some who developed postoperative fever all patients had an uneventful recovery.

DISCUSSION:

The era of surgical intervention for correction of congenital heart defects began when Gross performed the first closure of PDA in 1938 on a seven year old girl.[1,2,3]

Over the last 50 years enormous experience in the management of PDA has accumulated making PDA closure a safe procedure with extremely low mortality.

AETIOLOGY AND INCIDENCE:

The ductus is an embryological remnant of the sixth arch. The ductus is a large vessel connecting the pulmonary artery to the ventrolateral aspect of the aorta just distal to the left subclavian artery. Before birth the blood is directed from the pulmonary artery through the ductus into the aorta because of the high pulmonary vascular resistance. Soon after birth the alteration in pressure ratio between systemic and pulmonary system is reversed resulting in a left to right shunt.

The overall incidence of PDA is in the range of 10–15 percent.[2,8,9] Over the last two decades there has been growing awareness and recognition of heart diseases in Africa and in particular congenital heart defects. The four major defects seen in African series are ventricular septal defects, atrial septal defects, PDA and tetralogy of Fallot in that order.[4] The pattern of cardiac diseases in East Africa is said to be the same as the technically developed countries.[4] This review of four patients is too small a sample to make any valid conclusions on the incidence. However, female sex predominance compares favourably well with other studies.

Infection with rubella is associated with PDA. High altitudes 3000 feet above seal level has been suggested to influence persistence of the ductus arteriosus. This has been attributed to chronic hypoxia due to decreased partial pressure of oxygen. This relation has not been validated in the Caucasian population in USA.[10] More significant is the incidence of congenital heart diseases which varies among studies depending on the population studied and diagnostic methods employed.

DIAGNOSIS:

It is a recognised fact that a history of congenital rubella is frequently associated with PDA. Premature infants have a high risk of the disease especially if associated with birth asphyxia or respiratory distress syndrome. Detailed history about mode of delivery and other congenital anomalies are helpful facts. Failure to thrive and frequent pulmonary infections are common features. The classical diagnostic finding is a systolic thrill in the second (right) intercostal space or suprasternal notch associated with a machinery murmur found in the second or third intercostal space. The murmur is characteristic and allows a diagnosis in about 95 percent of cases. The peripheral pulse is strong and collapsing in nature. The heart size may be normal or enlarged. X–ray films of the chest will show increased pulmonary vascular marking. The ECG will reveal a left ventricular hypertrophy with normal or increased voltage. The echocardiogram is the main diagnostic modality. The left atrium aortic ratio is increased in an infant with a large patent ductus.

TREATMENT:

Surgery is the main modality of therapy. There is no special problem associated with the decision to operate in patients with isolated PDA, apart from age. Infants under 6 months of age carry a greater surgical risk than older babies.[1,2,6] Keith Rowe refers to a study where 60 babies under 6 months (with PDA) were operated and the mortality was 8.3 percent whereas none of the 148 babies operated between 6 month and 2 years of age died.[2] Many authors consider an optimum period to do elective surgery as 2–5 year for symptomatic cases.[1,2,6,8] In this review age at operation was between 5–13 years, probably due to delay in making diagnosis. There was no death and hospital stay was between 5–10 days. Conservative treatment with indomethacin and/or protaglandin synthetase inhibitor can be considered in infants who are premature, especially those with respiratory distress syndrome or cardiac failure.

According to Campbell,[11] adults with PDA who are alive after 17 years of age have their life span halved compared to the normal population where 34 percent will die by the age of 40 years and 60 percent by the age of 60 years. Complication like congestive cardiac failure, Eisenmenger syndrome, aneurysm of the ductus, calcification, and infective endocarditis increase with age making repair technically difficult.

The ductus is surgical exposed from an antero —— posterolateral incision through the 3rd or 4th interspace. The closure of the ductus has undergone great evolution and modification since Gross and Hubbard reported their first ligation. The original technique of simple ligation has been replaced with ligation and division because of recannalisation in some cases.[1,12] Some surgeon still prefer simple ligation in young infants, using a wider ligature with sufficient tension so as not to cut through the ductus.

Simple ligation is a method of obliteration of ductus with less than 7mm diameter and transfixation suture may be used.[13] Division with suture is the method of choice where the ductus is very short, or wider than 8m and also in the older children.[2,7,13]

CONCLUSION:

Elective closure of PDA should carry virtually no risk other than that of anaesthesia. Mortality in experienced hands is less than 0.5 percent.[1] However additional heart anomalies may complicate the procedure.

References:

1. Panagopoulas PG. : PDA in Infants and children: Review of 936 operation: Thorax 1971;26:137

2. Keith Rowe – Heart disease in infancy and childhood 3rd ED Macmillan. Publishing Co. Inc. 1967.

3. Surg. Clinic of North America Latest Advances in cardiac surgery 1985;65:(3)

4. Gikonyo – Congenital heart disease in E.A. (Editorial) EAMJ. OCT. 1991:749

5. Davies : Surgical treatment of PDA at University College Hospital Ibadan: EAMJ 1989;66

6. Ekstorm G.: the sugrical treatment of patient with ductus arteriosus : Acta. Chir. Scand. 1952:169

7. Amato JJ.: Closure of Patent Ductus arteriosus : Master of Surgery an educational service provided by Glaxo : 1985 : by Cine–Med. Inc.

8. Edwards JE. Surgery of operative congenital heart disease: Am. J. Pathol 1976;82:408

9. Storstein O. Congenital heart disease in Clinical Material : Acta Med. Scand 1964;176:195

10. Alzamora E. Influence of great altitudes on determination of certain cardiac Anomalies: Paediatric 1953;12:259

11. Campbell N: Natural history of persistent ductus Arteriosus. Br. Heart J. 1968:30:4

12. Somers K. Loefler I Patent Ductus Arteriosus at Mulago Hospital Kampala: EMJ. 1968;45:7:

13. Bickford B. J: Surgical aspect of PDA: Arch. dis. childhood 1960;35:92

CHAPTER FOUR

GALLSTONES

ABSTRACT:

Ten patients are reviewed who had cholecystectomy done for cholelithiasis in the two surgical firms of the department of surgery, Muhimbili Medical Centre during a one and a half year period between October 1989 – April 1991. The mean age was 44 years with a range of 24 to 62 years. Females formed the majority with a female to male ratio of 9:1. Ultrasound and oral cholangiocystogram were the common investigations used to assist in the diagnosis. Cholecystectomy was performed in all patients while one patient had additional common bile duct exploration done due to obstructive jaundice. Only one patient had an emergency cholecystectomy. Two patients had additional surgery consisting of vagotomy and drainage due to a co–existing peptic ulcer disease. Duration of hospitalisation ranged between 5 to 19 days with a mean of 8.7 days. There was only one post–operation wound sepsis and no mortality.

INTRODUCTION:

Cholithiasis is a disease that appears to affect mainly the Western communities. It is infrequent in most of Africa and the Indian subcontinent compared to North America, Europe and Australia.[1] Adaption of the Western life style and diet may increase incidence of cholesterol stones.[2,3,4,5] The standard management of symptomatic gallstones remains operative cholecystectomy.[6,7] Recent advanced in hepatobiliary surgery, coupled with advances of fibroptic technology has however revolutionised the diagnosis

and management of biliary tract diseases. Thus, there is an increasing demand by patients for non–operative treatment for gall stones, and laparascopic cholecystectomy is fast becoming the surgical procedure of choice.[6,7,8,9]

Ten patients are reviewed in this series with one patient being discussed in detail. The final part of this paper discuss the various relevant aspect of the subject and treatment.

CASE REPORT:

MK, a 30 years old male had presented at the Surgical Firm I Outpatient Department with main complaint of a dull right upper quadrant abdominal pain which had been there for the past thirteen months. He experienced episodes of colic, on taking fatty meals or alcohol which radiated to the back. This pain was relieved on lying down. His bowel habit had been normal and regular, but he complained of flatulence. He had no history of yellow discoloration of the eyes and had not noticed undue body itching. Incidently his previous attending doctor had advised him to reduce his body weight. He had never been hospitised before. He was a businessman and was married with three children. He took alcohol in moderate amounts but did not smoke.

On examination he was obese, afebrile not jaundiced and had no lymphadenopathy. His blood pressure was 150/80mmHg and the pulse rate was 98 per minute and regular. The abdomen was soft and moved with respiration. There was no hepatomegaly and the gallbladder was not palpable. Murphy's sign was negative. Rectal examination revealed normal findings and the rest of the systems was normal. A provisional diagnosis of cholelithiasis was made.

The following investigation were done. Plain abdominal X–ray showed no radiopaque stone. The ultrasound revealed multiple gallstones with normal hepatobiliary system. Oral cholecystogram reported multiple gall stones.

Haemogram:	HB 17 4gm/dL
	WBC 3.500 total
	ESR – 1
	the peripheral film was normal
LFTs:	ASAT 63 IU/L
	ALAT 77 IU/L
	ALP 76 IU/L
	Total protein 70.9g/L
Bleeding Indices	
	PT = 12.4" = 92 percent
	Patients controll 12" = 100 percent
	PPT = 38"
Serum electrolytes:	Sodium 138 mmol/L
	Potassium 5.1 mmol/L
	Creatine:92 mmol/L
	Cholesterol: 5.1 mmol/L
Chest X-ray:	Normal
ECG:	Normal

The patient was prepared for surgery after an informed consent. Two units of blood were cross–matched and reserved. Ampicillin 500mg was given one hour before the operation. A nasogastric tube was inserted on the day of operation.

In the operating room and under general anaesthesia the abdomen was routinely cleaned and draped. A Kochers' incision was made along the right costal margin and the abdomen was entered by using electrical diathermy. The transverse colon, stomach and duodenum were packed away and the liver retracted upwards to expose a distended gallbladder. The junction between the cystic duct and common hepatic duct and common bile duct was identified. By gentle traction and dissection, the neck of the gallbladder was delivered forward on the right aspect displaying the cystic duct and the common bile duct. The overlying peritoneum was cleared off, exposing the cystic artery which was ligated and severed between ties. Using a sterile needle and syringe a sample of bile was drawn for culture and sensitivity tests. The cystic duct was thereafter identified, clamped and doubly ligated with chromic catgut no 2/0 and severed. By a combination of sharp and blunt dissection, beginning at the neck and working forward, the gallbladder was dissected off the gallbladder bed. The bleeding raw surface of the gallbladder bed was controlled by cautery. The peritoneal reflection covering the gallbladder was stitched with catgut 3/0. A fine rubber drainage was leftfor drainage and was brought out through a separate stab wound. The rest of the abdomen was examined and no abnormality detected. The abdomen was therefore closed in layers.

The gallbladder was opened displaying a thickened wall and an edematous mucosae and within the lumen were about 15–20 multifaced yellow stones. Postoperatively, the patient received injection of pethidine 100mg 6 hourly

for pain and ampicillin was continued 6 hourly for 72 hours. Dextrose–saline one litre 8 hourly was prescribed and vital signs were recorded 4 hourly for the first 24 hours.

Forty eight hours post–operation, the patient developed a spike of fever with a temperature of 38°C. Physical examination was normal. A thick blood smear revealed malaria parasites. He respond to a standard dose of chloroquine. The nasogastric tube was removed and the patient was started on oral sips. He was ambulated and allowed meals on the third day. All stitches were removed on the seventh day and the patient discharged. Two weeks later he was reviewed at the post–operative follow–up clinic. The wound had healed well and he had no pain. At six weeks he had recovered completely and had started working.

RESULTS AND COMMENT:

Table 1 **Age and sex distribution**

Age		Sex	
	M	F	Percent
20 - 29	-	1	10
30 - 39	1	3	40
40 - 49	-	2	20
50 - 59	-	1	10
60 - 69	-	2	20
Total	2	9	100

Most patient were in the range of 30–39 years with a mean age of 44 years. Female:Male Ratio was 9:1

Operation:

All patients had cholecystectomy. The patient with jaundice underwent a bile duct exploration. The two patient with peptic ulcer disease in addition had truncal vagotomy and drainage procedure. Duration of hospital stay ranged between 5–19 days with a mean of 8.7 day. Only one patient develop post–op wound sepsis.

Table II **CLINICAL FEATURES**

Symtomatology	Frequency	Percent
Right upper quadrant pain	9	52.9
Nausea and vomiting	3	17.6
Jaundice	1	5.9
Recurrent fevers	2	11.8
Itching	1	5.9
TOTAL		100

Pain in the right upper quadrant, recurrent fevers, nausea and vomiting were the predominant clinical presentations. Two patients had associated peptic ulcer disease. Except for one patient who presented with acute features of acute cholecystitis the remaining presented with chronic and insidious features. The one patient with jaundice and itching had a gallstone situated at the junction of cystic duct and common hepatic duct. The histology result later showed chronic cholycystitis with multiple cholesterol stones. Bile culture revealed no growth of organisms.

DISCUSSION:

INCIDENCE:

Cholelithiasis is a disease that is commoner in the Western civilisation. The problem is global but the prevalence varies geographically.[1,2,3] Most of the prevalence rates are based on autopsy or surgical reports. In the USA, autopsy report reveal stones in 20 percent of females and 8 percent of males.[10] In Africa prevalence from autopsy reports is 0.87 percent.[2] Females are affected about four times more commonly than males. The four 'fs': female, fat, fertile and fourty characterise the group with the highest risk.

Cholesterol containing stone are common in the West and are related to certain other diseases such as ischaemic heart diseases, diabetis mellitus and colorectal cancer. Pigment stones are commonly encounted in the Asiatics and are associated with bacterial or parasitic infection.[2] The influence of Western life styles in Africans and Asians may increase the frequency of cholesterol stone disease.[3,4,5]

In the author's series, female comprised 90 percent of cases. The most affected group were in their 3rd and 4th decade. Despite the small sample, it compares well with other studies elsewhere.

AETIOLOGY AND PATHOGENESIS:

The major constituents of gallstone are cholesterol, bile pigments, and calcium. Other constituents include iron, phosphorous, carbonate, protein, carbohydrates, mucus and cellular debris.[11] Regardless of their nature, three

factors are important in stone formation: abnormalities in bile composition, stasis and infection.[11,12]

Bile is composed of 97 percent water, 1–2 percent bile salts, 1 percent pigment and small amounts of cholesterol and fatty acids. The daily output is 500–1200ml/day or 40ml/m. About 95 percent of the total bile output is returned into the liver via the enterohepatic circulation. Bile secretion occur in the microvillus membrane of the bile canaliculi of the hepatocytes. Hepatocytes actively secrete bile salts and sodium. Water follows passively.

Active secretion of bile is associated with a passive increase in lecithin, cholesterol, bilirubin and other organic substances. At a low level of bile secretion however more cholesterol than lecithin is secreted.[2,10] Bile becomes saturated with cholesterol. The solubility of cholesterol depends on the relative concentrations of conjugated bile salts, phospholipids (mainly in the form of lecithin) and cholesterol in bile. Lecithin is insoluble in aqeous solutions but is made soluble by bile salts in micelles. The addition of lecithin to bile acid micelle increases the ability to incorperate cholesterol as lecithin bile salt micelle. The amount of cholesterol that can be solubilised in this cholesterol – lecithin bile salt complex is limited and is dependant on the total lipid concentration and relative molar percentage of bile acid and lecithin. Lithogenic index is a numerical factor which defines the degree of cholesterol saturation.[10,11] The precise cause of predominantly cholesterol stone is not known.[13] Patients or individual who are prone to cholesterol gallstone formation, elaborate excessive synthesis and secretion into bile.

Cholesterol in bile is synthesised de-novo by hepatic microsomes from acetate, the enzyme which limits the rate of cholesterogenesis being 3 – hydroxy – 3 methyl glutaryl–coA reductase (HMGCoAR). Although it is

known that high caloric intake may predispose to stone formation by increasing synthesis of endogenous cholesterol, the effect of ingestion of excessive cholesterol is less clear. In few individuals, pure supersaturation results from relative pure deficiency in the secretion of bile salts, for instance by interruption of the enterohepatic circulation as a result the bile salt pool and the rate of secretion of bile salts progressively decrease and bile becomes supersaturated with cholesterol. The risk of developing cholesterol stone is doubled in women taking oral contraceptives[10] and obese people suffer the same risk.[13] Pigment stones are more common in the Oriental then Western countries. These stones consist of insoluble black pigment polymer of calcium bilirubinate, carbonate and phosphate. Isolation of organism such as E.coli, bacterium typhosum from gallbladder wall in patient with cholelithiasis suggest infection could be a causative factor.[11] Pigment stones are also encountered in haemolylic conditions. This could suggest an increase in unconjugated bilirubin in bile. Other predisposing factors include hereditary, liver cirrhosis, ileal diseases, peptic ulcer surgery and diabetic mellitus.

Stones confined to the gallbladder rarely evoke symptoms of biliary colicky unless associated with obstruction.[14] Obstruction is associated with an increase in intraluminal pressure, distension and stretching leading to visceral pain. Obstruction of the cystic duct results into hydrops or empyema. Obstructive jaundice develops on blockage of the common bile duct. Calculus obstruction predisposes to bacterial infection. Stasis and distension of biliary system impair lymphatic and vascular drainage. Infection may spread as ascending cholangitis or cholangiocystitis to involve the hepatic duct and portal triad leading to obstructive or infectious biliary cirrhosis. An exceeding large stone may pass unnoticed through a cholecyto-intestinal fistula eventually to be impacted at any point of intestinal narrowing (gallstone

ileus). Pancreatitis as a consequence of stone impaction distally in the region of ampulla of vater is probably due to duodenal reflex into pancreatic duct.

The natural history of gallbladder stones in different series indicated that 40–60 percent of persons with stones are asymptomatic[14,15,16] and this group have a 50 percent chance of developing symptoms.[2,15]

Fatty food intolerance, vague pain in the right outer upper quadrant of the abdomen are present in equal frequency in patients with or without gallstone.[10] In a study of 2464 patients designed to establish the prevalence rates and frequency of abdominal symptoms, no differences in the frequency of distribution of symptoms could be established.[14] In this series (table II) which is hospital based, all patients came with symptoms. Pain in the right upper quadrant constituted the majority (59.9 percent), while 17.6 percent had nausea and vomiting, and 11.8 percent experienced recurrent febrile attacks. Acute choleycystitis is suspected when colicky pain and fever persists for more than 5–6 hours with a positive Murphy's sign. Only one patient in this series presented with acute cholycystitis in association with gallstone. Features of biliary colicky or acute cholycystitis may mimick other serious conditions like: acute pancreatitis, peptic ulcer disease, renal colicky, angina or basal lobar pneumonia. A positive Boas' sign is useful in distinguishing acute choleycystitis from other conditions. Should a gallstone pass from gallbladder to be impacted in the common bile duct jaundice will develop. The absence of bile in the intestine will impair fat and fat soluble vitamin digestion and absorption. Steatorrhea and pale stool may then occurs. Abnormal bleeding tendency occurs due to impaired synthesis of vit–K–dependants factors: prothrombin, factor X, IX. Serum bilirubin will also be elevated.

Gall stones and complication associated with stones are evaluated by various methods. Ultrasound is a valuable procedure of choice. Its usefulness lies in being a well tolerated and non-aggressive modality. It is of particular value in patients who are jaundiced, pregnant, allergic to contrast, vomiting and acutely ill patients, conditions which preclude conventional studies. It has a high specificity of 97 percent and sensitivity of 88 percent.[17] False negatives may occur in a solitary stone impacted in the cystic duct.[11,17] It is also unable to identify a gallbladder packed with stones. Plain abdominal X–ray reveal 10–20 percent of all gallbladder stones. Emphysematous choleycystitis and porclein gallbladder are also seen.

Oral cholecystography is valuable. Telepaque is the contrast media employed. This dye is absorbed from the intestine and taken up by the hepatocytes via the intestinal – portal venous system. The hepatocytes excrete the contrast into the biliary system which is concentrated in the gallbladder. Therefore a normal functioning gallbladder can be visualised by X–rays. Stones will be seen as filling defects against a uniformly opaque background. Non visualisation indicates a non–functioning gallbladder or blockage of the cystic duct. Extra–biliary factors include impaired liver function (bilirubin 50mmol/), failure of patient to take the contrast, malabsorption, diarrhea or faulty X–ray. Intravenous cholangiocystography is inferior to cholecystography.[18] Among its drawbacks are anaphylactic reaction to the contrast when administered parenterally to patient allergic to iodine. It is contraindicated in patients with impaired liver functions, acute choleycystitis and impaired renal functions.

Endoscopic Retrograde Cholangio – Pancreatography (ERCP) can selectively cannulate the common bile duct with the aid of a side viewing fibroptic duodenoscope via the papilla of Vater. A contrast media is introduced slowly

under fluoroscopy control. This procedure may differentiate 'medical' jaundice from 'surgical' jaundice especially if due to a stone in this context. This procedure also enables specimens to be taken for cytology and bacterial examination. A common complication is pancreatitis and cholangitis.[10,19]

Percutaneous Transhepatic Cholangiography (PTC) is an invasive procedure whereby a flexible thin needle (Chiba or Skinny) underflouroscopy is advanced through the 8th, or 9th intercostal space in the midaxillary line to about 2cm short of the right margin of the vertebral column. During withdrawal of the needle a contrast is injected to visualised the biliary anatomy. This procedure is of considerable use in the diagnosis of cholestatic jaundice and visualisation of the proximal biliary tree. The main limitation are presence of cholangitis and uncorrected coagulopathies. Bleeding and septiceamia or bile peritonitis may complicate the procedure.[10,19]

Per–operative cholangiography is an intra–operative procedure. An X–ray image intensifier with a T.V display are essential. A casset tunnel is placed in such a position as to include the whole biliary anatomy. The cystic duct is cannulated through which a contrast media is injected with the table tilted at 10^{o}. Respiration is obstructed to obviate movements which would blur the pictures. Failure of the contrast to enter the duodenum may indicate the sphincter of Oddi or the common bile duct is obstructed (stone in this context). Per operative and post exploratory cholangiography are done after choledochostomy by making sure that all stones have been removed. Post–operative cholangiography is performed 10–14 days after choledochostomy via a T–tube which is used to drain the common bile duct. The absence of stone and a normal flow of bile into duodenum indicates that the T–tube can be removed.

A choledochoscope may be passed into the biliary tree to determine the absence or presence of stone, stricture or stenosis. Computed tomography is yet another possibility, though an expensive procedure. It provides information similar to the echogram. Radio Isotope scanning is a diagnostic tool for defining acute cholecystitis secondary to cystic duct obstruction.[11]

Auxillary non-specific tests are those which are based on biochemical reactions. They measure different aspects of hepatic function and patients welfare. They include haemograms, transaminases, bilirubin levels, alkaline phosphatese serum amylase and electrolytes, urine bilirubin and bleeding indices.

TREATMENT:

The gall bladder often already affected by chronic cholecystitis may be acutely inflamed. In 95 percent of cases, a gall stone is found obstructing the cystic duct. In most cases common organism i.e. E.coli, Klebsiella and Strep faecalis can be cultured. In more than 90 percent of cases symptoms of acute cholecystitis are managed by non-operative treatment which include, nasogastric aspiration and intravenous fluid, analgesics and appropriate antibiotics. Usually within 3 days of appropriate treatment the temperature, pulse and physical signs will show resolution of the inflammation. Cholecystectomy can be done after conservative treatment three weeks later. Conservative management is not advised when there is uncertainty about the diagnosis e.g. perforated duodenal ulcer or retrocecal appendicitis and should be abandoned if the pain and tenderness spread across the abdomen and the pulse rises. In this case cholecystectomy is done as an emergency. If a stone is present in the common bile duct its removal should have priority over cholecystectomy.

The exact timing for the definitive treatment for acute cholecystitis is cholecystectomy is debatable. Proponents of early operation maintain that it minimises complications which increase with time. Advocates of delayed surgery assert that acute cholecystitis can be resolved with antibiotic and supportive therapy, thereby avoiding surgery on acute high surgical risk patients.

However, studies have reported that mortality rates for early and delayed surgery are equal with no significant difference in frequency or severity of post–operative complications.[20] Twenty five percent of patients for whom delayed operation is contemplated have recurrence of acute cholecystitis.[21]

Cholecystectomy has been performed over several years and is considered the standard treatment for gall stones with proven safety and efficacy.[7,15] As knowledge of hepatobilary surgery continues to develop, there has been an increasing demand by patients for non–operative treatment.[8] Science is taking a back seat to the demand of the consumers. The fibro–optic technology has revolutionized the diagnosis and management of biliary tract disorders. The most recent development has been laparascopic cholecystectomy. [6,7,9]

Several techniques for performing cholecystectomy have been described, they differ primarily in the order in which the essential steps are conducted.[7] The most crucial dissection however is conducted near the insertion of the cystic duct into the common bile duct.[7,11] The essential factor is that all steps are conducted under direct vision.

Vertical incision is appropriate in patients with acute costochondral angle. Conversly, exploration in obese patients with wide costochondral angle is best achieved through a transverse or right subcostal incision. All the abdominal organs as well as the gall bladder must be examined. The area is isolated with packs and dissection around the Carlot's triangle will expose the cystic artery which is clamped and ligated early. This step enables the field to be less bloody and distorted. To prevent a calculus from migrating into the common bile duct a silk loop is placed around the cystic duct. The gall bladder is dissected free from the liver bed lining, attached only by cystic duct with sufficient length for operative cholangiography. The length retained is inconsequential as long as it does not contain a stone.[7] A dissection from the fundus towards the neck of the gall bladder has a theoretical disadvantage of milking the calculus from the gall bladder to the common bile duct. However many a surgeon discount this as a mere theoretical risk.[7] This procedure is considered more bloody, however, and entails a risk of injury to the adjacent common duct, right hepatic duct and/or artery.[7,11,15]

Newer techniques for cholecystectomy require greater technical skills (laparascopic cholecystectomy, minicholecystectomy) but have the advantage of short convalescence, greater cosmetic acceptability to patients than standard surgery.[6,8,9]

Injury of common bile duct is considered and recognised as a common complication in laparascopic techniques.[7,9] In modern minicholecystectomy the operation is performed through a small incision (4–6cm), the operation may be performed without assistance and the cosmetic results are excellent.[8] Percutaneous puncture of the gall bladder is done under radiological control or laparascopic vision i.e. percutaneous cholecystoctomy, percutaneous cholecystolithotomy, minicholecystectomy are variant modalities of

treatments with high success rates of which 78 percent of patients produce minimal scaring and experiencing morbidity between 2–3 weeks.[8]

Surgical exploration of the common bile duct is usually indicated by one or more of the following conditions; a recent history of jaundice or evidence of obstruction from recent investigation (i.e. ERCP) showing a stone in the duct. A dilated duct with obvious palpable stone at operation and positive operative cholangiogram. Relative indications include: cholangitis, pancreatitis, multiple stones within gall bladder, an inflamed gall bladder which is empty in patients with biliary tract symptoms. (Hospital Update 1990 January).

Cholecystotomy is advised in patients who are deemed too ill to withstand standard cholecystectomy. These patients require decompression and drainage of the distended gallbladder.[7,11,19] This operation is also performed in situation with marked inflammation of the gallbladder obscuring the anatomical relation of critical structure. When the gallbladder is obviously necrotic or perforated an attempt to secure tube with sutures may not be possible and therefore tube cholecystotomy may be dangerous. Thus, it may be better to perform partial cholecystectomy.[7,19]

There are two completely non–invasive gall bladder preserving treatments for gall stones: oral dissolution therapy and extracoporeal shockwave lithotripsy. These method require special selection of patients. They are suitable only for 10–30 percent of patients with pure cholesterol stones.[8] These treatments are costly in terms of convenience, duration of treatment and equipment. The main disadvantage is stone recurrence due to residual stones or fragmentation and denovo formation.

Patients who have radiolucent stones measuring less than 15mm diameter and a functioning gallbladder are ideal patients for oral dissolution therapy. Patients with larger stones are less likely to respond to this therapy because of greater stone burden and reduced surface: volume ratio.[8] The overall efficacy of dissolution therapy is about 38 percent and treatment prolonged for between 6–24 months. Stone recurrence occurs in approximately 50 percent of patient within 5 years.[8]

Extracorporal shockwave lithotripsy is suitable in radiolucent stone of less than 3 cm diameter and with a number of stone less than three. The gallbladder must be functioning to allow spontaneous passage of stone fragments.[8] Provided patients are well selected, fragmentation rate of 75–90 percent can be achieved followed by complete stone clearance in about 90 percent. Best results are achieved in solitary stones. Multiple tiny residual stone fragments may provide nidus for stone recurrence. Complications of lithotripsy include transient biliary pain (35 percent), cystic duct obstruction (5 percent), pancreatitis (2 percent) and hepatitis (3 percent).[8]

Contact dissolution therapy (percutaneous) by instillation of methyl–tert–butyl ether (MTBE) as with bile acid therapy is ineffective against pigmented or calcified stone but can dissolve the cholesterol in mixed stone. In the Mayo clinic[2] with the largest published experience with MTBE, treatment was succesful in 72 out 75 patients (96 percent). About 6 percent of the sample developed stone recurrence within 6–16 months. The side–effects with this treatment include severe burning pain, transient sedation due to systemic absorption, intravascular hemolysis, nausea and vomiting, liver and renal failure. Moreover, the treatment is labour intensive.

Laparascopic cholecystectomy is an alternative to standard cholecystectomy. The procedure is performed under general anaesthesia and in experienced hands can be performed within 40–90 minutes. The standard laparoscope is introduced through a 10mm umbilical incision. This procedure is technically unsafe in patients with adhesions, severely inflamed gallbladder or in the presence of portal hypertension. Feared complications include a hemorrhage and damage to common bile duct because of limited access.

CONCLUSION:

Cholelithiasis is a common condition as seen at the Muhimbili Medical Center and this probably applies to the whole country. The availability of less expensive diagnostic facilities may enable more "asymptomatic" gall-stones to be detected. Greater awareness of the general public and public health workers is needed in case finding. New trends in the management of gallstones seems destined to replace open cholecystectomy. However non-surgical treatment modalities are yet to be proved superior.

References:

1. East African Med. J. 1964: 41: 251-53

2. Fletcher PR: Gallstone in the tropics: Med. digest.1982:8 (9):5

3. Parekh D: gallstone disease among black South African: a review of Baragwanath Hospital experience: South Africa. Med. J. 1987. 72(10):19

4. Walker Prevalence of Gall stone in elderly black women in Sowetto. Johanesburg as assessed by ultrasound: AM.J. of Gastroenterology. 1989:84(11):1383 (Abstract).

5. Burkitt DP : Gallstones: Geographical andChronological features: J. Trop. Med. Hyg. 1975:78:140

6. Terblauche J: cholecystectomy: laparascopic or open operation: American College J. of Surg. bulletin: 1990:72(10):19

7. William J. Schirmez : common operative problems in Hepatobilliary surgery: Surg. clinic. North An. 1991:71 1363

8. Chersly – Curtis S: New trends in Gallstone Management: Br. J. Surg. 1991:78(2):143

9. Peterson – Brown: laparascopic cholecystectomy: Br. J. Surg. 1991:78(2):13

10. Isselbacher : Harisons' principles of internal Medicine 9ED: McGraw –Hill International Book Company:1989

11. Schwatz: Principles of Surgery: 5ED: McGrw – Hill Book Company.

12. Robins – Cotran: Pathological basis of disease 2ED WB Saunder Company.

13. Coyne MJ : Gallstone disease: Post grad. Med. 1975:57:153–9

14. Glambek 1: Correlation between Gallstone and abdominal symptoms in random population: Scand. J. Gastroententrolog: 1989:24(3):277

15. McSherry CK: Cholecystectomy: the Golden Standard Am. J. Surg. 1989: 158 (3): 174–8, 171–73

16. Martin MA: Biliary stone and bacterial in bile study in 211 consecutive cases: (Abstract) To Kushinia J. of Experimental medicine 1989 36(1–2):11–9

17. Rahmat. Maraheri Suf. Routine operartive Cholangiography: a critical appraisal Am. J. Surg. 1977 : 134 : 566.

18. Das. S: A manual on clinical surgery: 3ED Published by DR. Das. Calcutta India.

19. Harding Rains : Bailey and Loves (ed) short practice of surgery (revised 10 ED):ELBS:. H. Klewis and Company Ltd.

20. Nahrwold D.L. Chronic choleycystitis and cholelithiasis Textbook for surgery – sabistone (ed) page 1147,1986.

21. Norrby S. Herlin P. Early or delayed cholecystectomy in acute cholecystitis? Br. J. Surg. 1983 70 : 168

CHAPTER FIVE

INTUSSUSCEPTION

SUMMARY:

Five patients with acute intestinal obstruction due to intussusception were treated in the Paediatric Surgical Unit (PSU) between June 1991 and March 1992. Their ages ranged from 3 months to 9 years. Most patients presented before one year of age and only one presented at the age of 9 years. At laparotomy the ileo-colic type of intussusception was seen in 3 patients. The other two had the ileo-ceacal variety. In 4 patients treatment was achieved by manipulative reduction intraoperatively and only one patient underwent resection and end-to-end bowel anastomosis. No "lead point" could be established in this review.

Introduction:

Intussusception is a common abdominal emergency in infancy and childhood. The peak incidence for intussusception in infancy is at around six months of age and at this age the condition is idiopathic.[1] In most published series it is believed that localised lymph node hyperplasia in the wall of the intestine (Peyer's patches) act as the lead point of intussusception. A viral infection such as adenovirus or rotavirus can be invoked in approximately one half of the infants presenting with "idiopathic intussusception". In an older child an anatomical lesion can often be identified as the lead point. The classical features of abdominal pain, abdominal mass and rectal bleeding is present in only 10-17 percent of cases therefore posing a diagnostic dilemma.

The principle cause of morbidity and mortality is diagnostic delay or late arrival to hospital. Commonly, idiopathic intussusception in infancy typically follows an episode of gastroenteritis or URTI which causes Peyer's

patches to hypertrophy. Adenovirus is usually cultured from stools of half of these patients. The fact that all patients in this series were initially seen at the Paediatric Medical Department with a diagnosis of gastroentertis emphasises the point.

The aim of this section is to discuss some of the salient features of intussusception in childhood amid the background of a relevant literature review.

CASE REPORT: IP 707674. Informant: mother

An eleven month male baby had been referred from the General Paediatric Ward to the Paediatric Surgical Unit on account of diarrhoea with blood accompanied by projectile vomiting for 24 hours. Before admission, the child had on and off diarrhoea with vomiting for which at a peripheral dispensary was given an antibiotic, promethazine and oral rehydration solutions. However, there was no improvement in his condition. When his condition deteriorated he was referred to the Muhimbili Medical Centre. The child had started having attacks of screaming with occasional episodes of calmness indicative of abdominal colics. The stool had changed from watery diarrhoea to a blood stain jelly-like substance. The vomitus had become bile stained.

On examination the child was ill-looking, afebrile, pale but not jaundiced. He was dehydrated and had a pulse of 110 per minute, thready volume. The abdomen was distended with no visible peristalsis. A palpable non-tender mass was felt extending from the left iliac fossae to the supra umbilical region. The bowel sounds were increased. Rectal examination showed an empty rectum with a mass felt at the tip of the examining finger. Frank blood was noticed on the gloved examining finger.

Plain abdominal X-ray (erect) showed distended loops of small intestine with

multiple air fluid levels. In view of the clinical presentation and the clinical findings a diagnosis of intestinal obstruction most probably due to intussusception was made. Barium enema could not be done due to technical problems.

An intra-venous line of normal saline was set and a nasogastric tube inserted. He was cross-matched and 300cc of blood reserved. Injections of metronidazole and ampicillin were given. After about an hour of fluid resuscitation the child was wheeled to the operating room.

Under general anaesthesia, the abdomen was entered through a supra-umbilical transverse incision. On opening the peritoneum an ileo-colic type of intussusception became evident with the apex at the descending colon. Manipulative reduction was achieved using warm saline compression. Except for ecchymosis and edema of the terminal ileum, the intestine and mesentry were viable. The abdomen was closed in layers.

Post-operatively he continued with Hartmans solution, ampicillin and metronidazole for 72 hours. He developed a temperature of 40°C on the same day which subsided after 24 hours of tepid sponging. An urgent blood slide was taken and it showed no malaria parasites. On the second day the child had started passing loose stools. The abdomen was soft with no distension. The nasogastric tube was hence removed and the child allowed oral feeding. He was discharged on the fourth day in good general condition. Follow up at one, and six weeks later, the child was enjoying normal health. The wound had healed well.

RESULTS AND COMMENTS:

Case	Age	Sex	Procedure	Intussusception
1	6 months	male	Colic resection and anastomosis	Manipulative reduction
2	6 months	male	Ileo-colic	Manipulative reduction
3	9years	female	Ileo-caecal	Manipulative reduction
4	3 months	female	Ileo-caecal	Manipulative reduction
5	11 months	male	Ileo-colic	Manipulative reduction

COMMENTS:

There were 3 males and two female patients. Their ages ranged between 3 month to 9 years. The mean age was 26.8 months. Infancy type of intussusception occurred in four patients and only one occurred at childhood.

Three patients had a an ileo-colic type of intussusception and two had the ileo-caecal variety. All patients in this series presented with features of intestinal obstruction. At operation only one patient had gangrene of the bowel and presented 48 hours from the onset of clinical presentation to the time of surgery. The rest of the patients underwent laparotomy and manipulative reduction. No leading point could be established in all five patients. The aetiology was thus invariably idiopathic.

DISCUSSION:

EPIDEMIOLOGY:

The incidence of intussusception varies widely not only throughout the world but even within the same national boundaries.[2,8] Characteristically, it is a disease of infancy presenting in the later half of the first year (infancy type) or early part of the second year (childhood type).[3] Boys are affected more often than girls.[1] In Tanzania the disease is reported to be more common in

Kilimanjaro region.[1]

The aetiology of the disease is obscure in infancy type of intussusception. In 91 percent of intussusception the cause is idiopathic.[4] However several theories have been proposed to postulate the cause.

Bunts and Clark suggested that spasm or paralysis of the intrinsic bowel musculature might promote intussusception.[1] The stimulus for these bowel changes have been ascribed to dietary changes during weaning, ileal inflammation and intestinal allergy.[5] Perrin and Lindsay noted that the ileo-cecal valve and the terminal six inches of ileum were studded with aggregates of hyperplastic submucosal lymphoid tissue (Peyers patches) during the first year of life which thereafter gradually and progressively diminished in size with age.[5] They therefore suggested that lymphoid patches might initiate invagination of the bowel accounting for the peak incidence in that age group and the propensity of the lesion at the ileo-cecal valve. The seasonal peaks which coincide with enteric and respiratory infections are invariably associated with mesenteric lymphadenopathy and noted at laparotomy.[1,5] Implicitly, viruses are among one of the causes.[1]

In the childhood type of intussusception, anatomical abnormalities, such as Meckel's diverticulum, intestinal duplication or enteric cyspts and other conditions such as polyps, lymphosarcomas and Henoch Schonlen pupura have been noted as leading points in cases of intussusception.[1,3,5,6] In an analysis of 10 cases Elechi noted that 60 percent had a floating caecum.

PATHOLOGY:

Intussusception is composed of a segment of intestine telescoping into an adjacent intestine. Basically there are three layers. The inner or entering

layer becomes continuous at the apex with the middle layer which in turn becomes continuous with the outer layer. The inner and returning layers form the intussusceptum. The outer layer is the intussuscipiens. The distal part which advances in is the apex. The blood supply of the inner layer may be compromised depending on the tightness of the invagination.

Gross who studied 707 cases[7] classified intussusception as;

(a) ileo-colic (the commonest)	(77.0 percent),
(b) ileo-ceacal	(12.0 percent),
(c) ileo ileal	(5.0 percent),
(d) colo-colic	(2.0 percent),
(e) multiple	(1.0 percent),
(f) retrograde	(0.2 percent),
(g) others	(2.8 percent).

This classification describes the area at which invagination starts and the area of bowel receiving the invagination.

In a review of other series including this series, the ileo-colic is the commonest type.[4,10,7] The terminal ileum is invaginated through the ileo-cecal valve and advances inside the colon.

The ileo-ileal has atypical clinical presentation leading to a delay in diagnosis with resultant irreducibility and gangrene. The colo-colic type which is seen in older children is considered less dangerous and reduction is easy.

The bowel trapped in intussusception initially causes partial obstruction but subsequently leads to complete bowel obstruction. The constriction of mesenteric blood vessels and lymphatics leads to congestion of the tissue, edema and secretion of mucous and diapedesis of red blood cells into the intestinal lumen. This is the source of "red-currant" jelly. Gangrene sets in once the intraluminal pressure exceeds the arterial pressure.

CLINICAL FEATURES:

The clinical features are abdominal pain (coliky), vomiting, rectal bleeding and a palpable sausage shaped mass may be felt on abdominal examination. Atypical presentation may lead to delay in diagnosis. In one series[7] of 288 consecutive patients with intussusception only 10 percent developed classical symptoms (blood stools, intermittent abominal pain, and vomiting) and these were predominantly found in infants with a long history. About 42 percent had none or only one of the nonspecific symptoms of vomiting and intermittent pain. A palpable abdominal mass was found in 24 percent.[7] In another series of 139 children observed only 17 percent had classical features at presentation.[8] Rectal bleeding classically termed "red-current jelly stool" is reported in 50-60 percent and often occurs at a late stage.[1] In the absence of classical features, barium enema is a useful investigation. Intussusception may also be diagnosed with sonography.[9]

TREATMENT:

Management of acute intussusception is still controversial.[5] Some authors from Australia, North America and Scandinavia advocate hydrostatic reduction while others consider surgery as a treatment of choice.[5,10] Recent literature suggests that wherever possible hydrostatic barium reduction or pneumatic reduction should be attempted first. Preliminary treatment in all cases will include nasogastric suction, restoration of fluid electrolyte balance and blood loss regardless of the definitive therapy chosen.

HYDROSTATIC REDUCTION:

This technique was first introduced by Hirschsprung in 1905 and advocated by Ravitch.[3,10] The procedure is performed in the presence of a paediatric

radiologist and a surgeon. The patient is prepared for surgery should the need arise.

The procedure is indicated when the intussusception is suspected and within 24 hours. In practice, hydrostatic reduction is less effective after 24 hours and considered unsuccessful after 48 hours.[1,4,10] The procedure is contraindicated in cases of peritonitis, perforation, profound hypovolemic shock or toxaemia. Surgery is also advised in patients over 2 years and under 2 month of age. The incidence of anatomical abnormalities is also considered higher in this group.[3]

The children are usually sedated and the enema is introduced through a balloon or Foley's catheter, often done in the X-ray department. The enema reservoir is placed 1 metre above the infant. Under radiological control the enema is allowed to flow until it outlines the intussusception giving a characteristic onion ring appearance. Complete reduction is confirmed when contrast is seen to flow freely in the loops of ileum, return of barium with faeces and flatus and disappearance of the mass if present. If reduction fails, the procedure can be repeated up to three times pausing for 5-10 minutes in between each attempt before deciding on surgical intervention. If successful, the baby is kept on nil by mouth, given intra-venous fluids and observed in hospital overnight.

The success rates of this technique differ considerably from centre to centre. Gierup et. al. experienced a success rate of 81 percent in 308 patients.[7] In a review of eleven published reports totalling 1,317 patients the success rate was 62 percent.[10] The diagnosis of non-idiopathic cases may be missed, thus increasing the risk of recurrence.[5] The incidence of recurrence is no greater in those treated by Barium reduction than in those treated surgically. Perforation is the major risk of the procedure.[9] However, this procedure is

not carried out at the MMC due to the lack of adequate facilities.

SURGERY:

Surgery is considered after unsuccessful attempts at hydrostatic reduction. Other indications include evidence of bowel obstruction with moderate of marked abdominal destension or multiple fluid levels revealed on a plain X-ray, perforation, peritonitis, severe shock or toxaemia or a history of more than 48 hours and recurrence.

A nasogastric tube is passed for decompression. Fluid requirement for the age is calculated to correct electrolyte imbalance. Normal saline or Hartman's solution is the fluid of choice. Blood is cross-matched and a broad spectrum antibiotic is given preoperative.

The abdomen can be entered through a supraumbilical transverse incision.[3,11] The intussusception is examined. By gentle pressure and squeezing applied proximally (taxis) most intussusceptions can be reduced. Traction is mentioned to be condemned. If reduction is not achieved by this method the entire mass can be delivered out of the wound and compression with warm saline packs exerted evenly over the whole tumour mass and this will generally achieve complete reduction. After reduction the terminal portion of the small intestine, is examined for tears and bowel viability. A search is also made for a possible lead point.

Resection and anastomosis is indicated in irreducible or gangrenous intussusception. The previous methods of exteriorising the colon by various manoeuvres is rarely practiced.[9] With an exact method of electrolyte balance and fluid replacement, resection with end-to-end is recommended.[3,10,11] The principle source of mortality and morbidity (1-2 percent) is diagnostic delay.[1]

At the Muhimbili Medical Centre (MMC) all children in this series were

surgically managed. There was no mortality and no recurrence in this series in follow up of at one year.

CONCLUSION:

Intussusception is a common infancy and childhood emergency. All patients in this series were referred from the Medical Paediatric Ward and in particular from the Acute Diarrhoea Unit. This emphasises the point that intussusception is not a rare occurrence and must be considered in the differential diagnosis of acute diarrhoeal conditions in our environment even in the absence of the classical features of the disease. Indeed the cardinal features occur in only 10-17 percent of cases. [7,8]

References:

1. Editorial : acute intussusception in childhood. In *Lancet* II, August 1955 : 250.

2. Prof. J. K. Shija. *Paediatrical Surgical Care for developing countries.* Heinemann Kenya, Nairobi. Published in 1989.

3. Lela Kapila - *Intussusception in children Surgery.* International Edition. 1987.

5. Robert T. Soper : Recurrent Acute Intussusception in children. *Arch. Surg.* 1964 : 89 : 188

7. Jan Gierup : Management of intussusception in infants and children: A survey based on 288 consecutus cases. *Paed.*(1972) 50 : (4) : 535

8. Sigmund. H. E : Leading points in childhood intussusception : In *J. of Paed. Surg.* 1971 6 : (1) 6 : page 16

9. Pollet J. E. Intussusception : a study of it's surgical management. *Br. J. Surgery* 1980, 67:213

10. Peter J. Raudkivi H. : Intussusception: analysis of 98 cases. - *Br. J. Surgery*. Sept. 1981 vol 68.

CHAPTER SIX

CRYPTORCHISM

SUMMARY:

Ten patients with cryptorchism in which the author personally participated in the management is presented following 3 months of attachment in the Paediatric Surgical Unit during the year 1992. Their ages ranged from 18 months to 6 years. Six patients had left cryptorchism, three on the right and one Bilateral. All patients were found to have a congenital hernia and herniotomy was done. There was no other associated congenital anomalies recognized in this series. Orchidopexy was done in all patients. Hormonal therapy was not used.

INTRODUCTION:

A clear picture of the best therapy is clouded by the varied definition and insuffient long term study.[1] The incidence of undescended testis ranges between 0.7 – 21 percent depending on the age of the patients.[2] Unilateral absence of testes is rare with an incidence of 3 percent.[3] Unilateral undescended testes is 3–5 times more common than bilateral undescended testes.[3] The causes of maldescent of testis is unclear. However, several theories have been postulated. Deficiency of gonadotrophic hormonal stimulation appears to be an attractive theory.[3] The medical significance of undescended testis is that it is prone to trauma and torsion with an unquestionable risk of infertility and development of malignant disease. The timing of operation depends on three factors: Psychological consideration, technical factors and the risk of not operating early on these patients.

Predisposition to malignant tumours and infertility are the most worrisome complications.

The incidence of developing malignant tumour vary from country to country with highest rates of 4–5 per 100,000 reported in Northern Europe and in Maori's in New Zealand.[4] The risk of a malignant tumour developing in a given adult with cryptorchism is 1,500 per year but viewed as a cumulative risk over a life time, this represents an incidence of between one in 30 or one in 66.[3] Early operations are important to prevent infertility and avoid the risk of malignancy, torsion, hernia and trauma. After puberty a unilateral underscended testis should probably be excised.[5]

CASE REPORT: Rs. I 693352.

A two year old boy had been referred from Zanzibar with a history of an empty scrotum. His mother had noticed this abnormality incidently at 3 months of age and had reported this to the hospital were he was prescribed a trial of gonadotrophin hormone twice weekly for 8 weeks with no apparent improvement and hence reason for referral. The boy was born at home. There was no similar problem in the other siblings. During antenatal period there were no drugs given other than routine anti-malarials and haematinics.

On examination he was healthy and in good nutritional status with body weight of 12.5 kg. He was not pale or febrile. The externalgenitalia was of normal appearance but the scrotum was slightly atrophic. Both scrotum were empty. No testis could be palpated in the pubic, perineal, inguinal or femoral area. The cardiovascular and respiratory system were normal. A diagnosis of

bilateral cryptorchism was reached. Surgery was recommended. His Hb was 10.gm/dl and sickling test was negative.

In the operating room and under general anaesthesia a right inguinal crease incision was made. Dissection was deepened down to the aponeurosis of external oblique. The testis was found just beneath the external ring within the tunica vaginalis. Mobilisation of the testis was done from it's distal attachment (Gubenaculum freed) and extending spermatic cord freed by the dissection further proximaly to the internal ring. The hernia sac was dissected away from the cord, safe-guarding the vascular pedicle and the vas deferens. The neck of the hernia sac was transfixed with chronic catgut, No 3/0 and herniotomy done. The spermatic cord and testis was further mobilised to sufficient length to enable it reach the bottom of the scrotum without undue tension. The testis was then enclosed at it's lower pole with vicryl 4/0 to a created subdartos pouch. The left testis was dealt with in a similar manner. Reconstruction of the anterior inguinal wall and abdomen was done with vicryl 4/0. The skin was opposed by subcuticle continuous 4–0 vicryl. Post-operatively, injection pethidine was prescribed for pain. The patient was discharged on the 2nd day. He had a uneventful recovery. He was referred back to Zanzibar. Unfortunately he was lost to follow–up.

RESULTS AND COMMENTS:

There were 10 patient in this series. The age ranged from 18 months – 6 years. The majority had left cryptorchidism. Only one had right and another bilateral cryptorchism. All patients had congenital hernia discovered at operation and herniotony was performed. In one patient with unilateral undescended testis the latter could not be seen. The vascular pedicle and the vas deferens ended blindly.

DISCUSSION:

Incidence:

Nondescended testis is one of the commonest congenital defect in boys, yet there are no absolute figures acceptable to all due to lack of uniform agreeable definition in many studies.[1] Score studied 3612 infants and found an incidence of 21 percent in premature infants, 2.7 percent in the full term and only 0.2 percent at one year of age.[6] These figures are in close agreement with some other series.[1,3] The adult incidence of undescended testis in 12,535,824 military recruits was found to be only 0.28 percent.[1] There is a general disagreement and misconception of the accepted time of spontaneous descent of the testis before it can be considered pathological. Robert Whitaker[5] believes that if no descent is observed after the age of one year there is no point in waiting up to the age of 5 years. The incidence of undecended testis at one year is 0.7 percent.[5] Congenital anorchism occur ata rate of 3 percent.[2,3] Unilateral undescended testis is said to be 3–5 times more common then bilateral undescent.[3] Concomitant abnormalities associated with undescended testis include hypospadius and certain syndromes like Klinefelter syndrome.

The true cause of cryptorchism remain unclear but there are two theories that have enjoyed popularity: hormonal and mechanical theory. Hormonal theory is based on experiment in rats. Shapiro quoting Rajler and Walsh found that premature descent would be induced by gonadotrophins.[3] The rationale of this is based on the fact that testicular descent is assumed to occur a time when endogenous gonadotrophins are rising, under the influence of

pituitary gonadotrophines hormone. This is supported by the fact that testicular descent can be delayed or inhibited by estradiol[1,2,7] the impact of this though has not been fully established in human subjects, furthermore, it is dihydrotestosterone and not testosterone that induces premature testicle descent in rats.

In the mechanical theory, the abnormality is considered to be associated with the gubernaculum. Bachouse has suggested a fibrous encroachment upon the gubernacula mesenchyma as preventing the normal growth of the processus vaginalis.[1,3] Fibrous septam are commonly encountered at the scrotal neck.[3]

Mechanisms of descent[1,3,8]:

The testis develops in the posterior abdominal wall from an outgrowth of urogenital ridge at five weeks of uterine life. By the 8th week of uterine life the testis is situated at the inguinal region. The transabdominal descent is probably produced by the differential growth of the fetus rather than migration of the testis. Distally the testis is attached to a mass of mesoderm (the gubernaculum) which is connected to the inguinal region, and by passing through the already developed inguinal cannal it is attached into the scrotum. Meanwhile the processus vaginalis which is an outpouching of the peritoaneum epithelium develops around the gubernaculum, eventually growing downwards with it.

During this time the epididymis and the vas develop rapidly and proceed to the testis into the scrotum. There is no further testicular descent until 7 months of gestation at which time the gubernaculum mesenchyme swells to dilate the developing scrotal wall. By the end of 8 months the testis reaches the scrotum. Testicular descent is probably achieved by the relative lack of

growth of the gubernaculum compared to the rest of the body.

The testis that is not in the scrotum may fall in any of the three categoris: retractile, ectopic or true undescended testis. Retractile testis are by far the commonest, and is considered a variant of the normal testis. Clinically this variant passes through the external inguinal ring and can be coaxed into and to the bottom of the scrotum. However under the influence of strong cremasteric muscle reflex, it readily retracts (up) again. Retractile testis is associated with a well developed scrotum and represents the majority that descends under the influence of gonadotrophin therapy or at puberty.[1]

Maldescended, obstructive or ectopic testes are those that have been deflected away from their normal pathways. It accounts to between 30–65 percent and are normally developed.[1] Further descent is said to be blocked by fascial obstruction at the entrance of the neck of the scrotum. These testes may be located at the femoral, perineal, or pubopenile position.

A true undescended testis (arrested) is one that is arrested along its normal line of descent. The position is variable. It may be intra–abdominal where it is impalpable or contained in a hernia sac which may be a presenting feature. An emergent testis is situated at the external ring and can be retracted down to the inguinal cannal or a high scrotal one that cannot be drawned to the bottom of the scrotum. The latter are difficult to differentiate from the ectopic variety. Commonly the testes and epididymis are associated with developmental abnormalities.

Complications associated with cryptorchism are enormous. A testis situated in the inguinal canal or in close proximity to the pubic bone is vulnerable to trauma and prone to post–traumatic atrophy. The laxity of the cord in a testis

in a hernia sac predisposes to torsion, the diagnosis of which is likely to be delayed and thetestis undergoes atrophy. Congenital hernia, usually indirectly, is an associated finding. Although not an indication for operation it accounts for 90–95 percent of cases and rarely the presenting symptom.[5] Undescended testis can be both familial and/or inherited.[3]

There is an unquestionable risk of development of malignant lexini in undescended testis.[1,4,9] The percent of malignant tumour of several series in cryptorchism is 9.8 percent.[1] All types of germ cell tumour may occur but seminoma is the commonest. The next frequent one is teratocarcinoma. The risk is 48 times greater than in normally descended testis.[4] The risk of malignant change in intra–abdominal testis may be as high as 20–40 times, whereas inguinal testis may be 20 times.[3,4,9] This risk is not reduced by orchidopexy.[4] It is known that 20 percent of tumours arising in association with maldescent occur in the normally descended testis.[3,5] This would suggest an inherent development in germinal cells underlying a bilateral pathological process. Testicular dysgenesis is suggested as an aetiological factor.[1]

Sterility is a complication of cryptorchism. Failure of the testis to descend into the scrotum before puberty is associated with impaired spermatogenesis and progresses with increasing age.[10,11] It occurs as early as two years.

TREATMENT:

The treatment of cryptorchism is surgery though controversy surrounds hormonal therapy. Hormonal therapy has a disadvantage of subjecting the child to multiple injection of an expensive drug that may well have little

beneficial effects.[1,7] Moreover, its contraindicated in the presence of hernia, ectopic agenesis and in post–pubertal patients.[1] The recommended dose is 500 IU of chorionic ganodotrophin twice a week for eight weeks. Hormonal therapy is likely to produce sexual precosity, atrophy of the retained testis and of scrotal testis. It is also known to produce psychic disturbance.[3] Success rates vary from 20–55 percent.[7]

The optimum recommended age for surgical correction is up to 2 years of age. Early surgery at this age improves spermatogenesis and reduces the risk of malignancy.[2,8,10] Furthermore, the normally descended testis may become damaged by continuing absence of the undescended testis, the mechanism of which is unknown.[8]

Orchidopexy is the surgical correction of maldescended testis. The aim of orchidopexy is to improve testicular function, prevent testicular trauma, torsion and to remove the hernia sac and to avoid psychological trauma.[2]

The principle of operative treatment is to find and mobilise the testis without injuring the vessels or vas deferens. The aim is to obtain sufficient length such that the testis may be placed in a satisfactory position in the scrotum. The most suitable method offixation is in the subdartos pouch. The approach is usually through an inguinal crease incision. In intra-abdominal and high inquiral testis, sufficient length often cannot be achieved to mobilise the testis into the scrotum and here a two stage orchidopexy at 6 months interval is recommended. For high bilateral cryptorchism a transabdominal exploration may be carried out through a Pfannenstiel incision.

Pfannenstiel

CONCLUSIONS:

Cryptorchism is a common problem in our community. Early detection and community awareness is important so that surgical treatment may be commenced earlier, preferably before the age of 2 years. Community based studies are essential to determine the incidence of the disease and associated complications in Tanzania.

References:

1. Whitaker RH.: Management of undescended testis. Br. J. Hospt. Med. July 1970 : 25.

2. Atwell. JD.: Orchidopexy: Hospital update 1985:761

3. Shapiro R.: Current concepts of undescended testis: Surgery, gynaecology & obstetric 1978;147:617

4. Barret A.: Testicular tumours : Hospital update – 1984:355–369.

5. Whitaker RH.: Underscended testis : Update J. of Postgraduate General practice 1981;22:(12)

6. Scorer C. G.: Arch. Dis. Childh. 1964;39:605–1967.

7. Christiansen P : Hormonal treatment of crytorchism – Act. Paed. 1992;81:605–8

8. Howard ER.: Diagnosis and sugical treatment of mealdescended testis BJHM. 1986;35:260

9. Oliver RTD.: Testis Cancer. British J. of Hosp. Med. Jan 1984:23–35

10. Lany. I. lipshuliz : Testicular function after orchiopexy for unilateral UDT : New Eng. J. of Medicine 1976:295(1).

11. Mengel H. A. : Studies on Cryptorchism : A comparison of Histological findings in Geminatuve epithelium before and after second year of life : J of Paed Surg. 1974;9(4):445

CHAPTER SEVEN

HIRSCHSPUNG'S DISEASE AS SEEN IN MUHIMBILI MEDICAL CENTRE

ABSTRACT:

A review of 15 patients with Hirschspung disease seen at Paediatric Surgical Department of Muhimbili Medical Centre during May 1991 – May 1992. Males accounted for 12 patients to 3 females. The age ranged between 3 months to 10 years of age. Two patient presented as acute intestinal obstruction. Eight patients presented at infancy, five as neonates and two at childhood. Two patients underwent emergency laparotomy and right transverse colostomy, two had Duhamel operation done on them, one patient had elective right transverse colostomy and one sphincterectomy. The remaining two patients who presented with acute intestinal obstruction died within 48 hours of operation.

INTRODUCTION:

Hirschsprung's disease (HD) is a congenital aganglionic megacolon, usually herald by delayed passage of meconium or neonatal large bowel obstruction.[1] The earliest description of a case of Hirchspung's Disease is an autopsy report of a 5 year–old girl by Ruysch in 1691,[1] but perhaps best known is Harald Hirschsprung for his convincing report of the disease to which the disease now bears his name (1886).[2]

Although in most cases aganglionosis involves the sigmoid colon and rectum, the extent of intestines involvement in Hirschsprung's disease varies. The cause of Hirschspung's disease is obscure, but it is thought about

Hirschsp

3–5 percent of case are genetic or familial. Hirschspung's disease results from the darrest of cranial to caudal migration of neuroblast into alimentary tract.

While in Europe and North America the commonest forms of megacolon is Hirschspung's Disease, there appears to be some significant differences between the clinical features of Hirschspung's Disease seen in Caucasian and those seen in Africans.[1]

The author reviews 15 patients who attended at Muhimbili Medical Centre. A review of literature is done and the findings discussed against a background of relevant literature.

CASE REPORT: 672492.

A seven years old female presented at the paediatric surgical outpatient department with a history of constipation and abdominal distension lasting over 4 years. She was a full term baby and had passed meconium at birth. At about 3 years of age her mother had noticed undue abdominal distension associated with intermittent constipation, getting little and hard stool approximately every once a week. There was no abdominal pain or vomiting. At a peripheral hospital she had received stool softeners and antihelminthics but without noticeable improvement. She was the second born in a family of four. There was no similar history in the family.

On examination she weighed twenty kilograms. Not pale, afebrile and in good nutritional status. The abdomen was gaseous distended, there was no visible peristalsis, non tender nor palpable organ and no shifting dullness. Bowel sound was normal. No abnormality was observed on the genitalia, or

rectal examination.

A barium enema was done which showed an elongated dilated left – sided sigmoid colon with an identifiable transition zone suggestive of Hirschsprung's Disease. Rectal biopsy showed smooth muscles of intestine in which the ganglia was absent. There was hypertrophy and disorganisation of non–myelinated nerve fibres. No abnormality was detected in the stool and urine examination. She had an Hb of 10.0gm/dl with a normal peripheral film, creatinine was 86 mol/l.

The patient was started on a daily colonic washout. A right transverse colostomy was performed in the theatre as a preliminary decompression procedure. The patient was discharged 8 days later and re-admitted 5 months later for a definitive surgical operation. She had gained 2 kgs in body weight. The abdomen was remarkably well decompressed. She had an Hb of 12.4gm/dl. Blood grouping and cross–matching was done and a unit of blood reserved. She was again put on daily saline enemas. Three days prior to surgery she was put on a light diet and started on flagyl 150mg and capsule ampicillin 250mg tds. On the morning day of the operation a nasogastric tube was inserted.

Under general anasethesia the patient was placed in a lithotomy and a urinary catheter was inserted. The abdomen and later theperineum was aseptically cleaned and routinely draped with sterile linen.

Through a Pfannestial incision the abdomen was opened in layers. The sigmoid and proximal colon were found dilated. The narrow site of transition of ganglionic to aganglionic intestine identified. The sigmoid colon was dissected down to the rectum and divided below the peritoneal

reflection. The rectal stump was closed with silk No. 3/0. A retro rectal turnel was created close to the posterior wall of the rectum. An incision was made on the posterior circumference of the rectal wall and the proximal intestinal stump brought through this incision. The colon was anastomosed in a single layer with catgut 2/0 to the posterior half of the distal rectal stump. The septum separating the anterior wall of the pulled through the colon and the posterior wall of the rectum was apposed with a pair of Kocher's forceps to crush the septum. A gauze was temporary left in the rectum. The abdomen was then closed in layers.

Post operatively the patient received 500ml of dextrose/saline 8 hourly for 24 hours. An injection of gentamycin 3mg 12 hourly, ampicillin 250mg 6 hourly for 48 hours then orally for five days and tablets metronidazole 100mg tid for 5 days. 1.M pethidine as 25mg 6 hourly was prescribed for pain.

Summary of postoperative events:

8 hours post operation the child was awake, vital signs stable.

4th day :	colostomy stool and masogastric tube was removed.
9th day :	developed abdominal distension but no vomiting. Then nasogastric tube was re–inserted and intravenous fluid re–instituted.
10th day:	clamps dropped off, abdominal distension subsided, bowel sound returns, nasogastric tube removed and IV fluid stopped.

12th day: developed fever, but responded to chloroquine.

19th day: discharged in good general condition

26th day: reviewed at outpatient department.

Good general health. Scheduled for closure of colostomy.

Results and Comments:

Table I **AGE AT ONSET OF PRESENTATION**

	Frequency
Neonatal	5
Infancy	8
Childhood	2

Table II

MODE OF PRESENTATION

Mode of Presentation	Frequency
Acute obstruction	2
Chronic construction	13

Table III

MODE OF TREATMENT

Type of operation given	Frequency
1. Emergency laparotomy & colostomy	2
2. Elective transversecolostomy (prelimenary)	3
3. Duhamel	8
4. Sphincterectomy	2

Two patients presented as acute intestinal obstruction. One patient died 2 days later, the other 18 hours post-operatively. Duhamel procedure was performed in 8 patients, sphincterectomy in two patients. Three patient had elective right transverse colostomy while awaiting for definitive surgery. The main diagnostic parameter was based on clinical, radiological and histological findings. For patients who featured acute presentation and suspected to have Hirschspung's disease at operation, a right transverse colostomy or ileostomy was effective in relieving the obstruction. Biopsies of the rectum and colon, including colostomy or ileostomy site was taken for histological diagnosis. In patients who had chronic presentation, a barium enema was done and a full thickness rectal biopsy taken for histological diagnosis. The patient wascommenced on bowel washouts until clear and a right transverse colostomy was performed as a preliminary procedure. A definitive surgery was then planned usually after 3 – 6 months when the general condition was satisfactory and the bowel adequately prepared.

DISCUSSIONS:

Megacolon is a pathological enlargement of the colon due to functional, organic or other causes. It may be congenital or acquired.[3] Hirschsprungs disease is a true congenital megacolon associated with absence of intramural ganglion cells in the distal alimentary system.[2] Harald Hirschsprung is best known for his convincing account of dilation of the colon in 1886 in Paediatric congress in Berlin, for which now the disease bear's his name.[2]

EPIDEMIOLOGY:

The incidence of Hirschsprung's disease is 1:5000 live birth in the United States. Familial occurrence has been documented in about 3–5 percent of cases.[2] The disease is more common in males. Although the disease is present in Africans, there appears to be a distinct clinical difference of the disease in the Caucasians to those seen in Africans.[4] A study of 34 Zimbabweans paediatric patients at Parrienyatwa Teaching Hospital, for the management of megacolon over 3 years revealed that 50 percent had Hirschspung's disease, 24 percent had clinical and radiological features of Hirschspung's disease but had entirely ganglionic bowel on histologicalexamination and 12 percent had secondary megacolon related to anorectal malformations.[6] Five percent could not be categorised.

PATHOLOGY:

Hirschsprungs disease results from arrest of cranial to caudal migration of neuroblast into the alimentary tract.[2,5] The basic pathology in Hirschsprung disease is the absences of intramural ganglion cells in the distal intestinal wall involving both the submucosal and intramusculure nerve plexus. This results in the disruption of the normal mechanism of colonic mobility and defeacation. In 75 percent of patients aganglionosis is restricted to the rectosigmoid, in 17 percent this extends to include the sigmoid, splenic flexure or transverse colon, in 8 percent to the distal ileum and in about 5 percent there is total colonic aganglionosis.[3,6] Absence of ganglionic innervation throughout the entire gastrointestinal tract represents the most extreme form.[7]

The essential gross feature is a dilated proximal intestine with gradual or abrupt transition to distal intestine. The transitional zone is typically funnel-like or cone shaped. The proximal intestinal wall is hypertrophied and the mucosae maybe ulcerated.

Histologically, there is the absence of postganglionic parasympathetic ganglia and intrinsic neuron with hypertrophy of cholonergic nerve fibre and an increase is acetylcholinesterase activity. The transition to normally innervated intestine may be gradual with along segment of intervening hypoganglionic intestine, but often is short and abrupt.[8] The existence of an aganglionic band with intervening intestinal segment of normal myenteric plexus has been reported.[9]

The cause of Hirschspung's disease is obscure but statistics favour the hypothesis of a multifactorial genetic system. The risk of disease developing

in sibblings of patients varies from 0.6 percent to 0.8 percent. Familial occurrance has been documented in 3.5 percent of cases.[2]

CLINICAL PRESENTATION:

In Hirschspung's disease, symptoms appear at birth and are herald by failure to pass meconium.[3,10] In African populations, the presentation is more insidious and occur at a later age[6] usually associated with complications.[3,11]

Hirschspung's disease may present as acute large bowel obstruction or gradually and progressively with disturbance of bowel habits at infancy or childhood leading to chronic constipation and general failure to thrive. Abdominal distension is a remarkable feature.

In chronic states the stools are usually toothpaste like and inadequate in amount.[10] The patient strains ineffectively but defeacation seems painless.[2] The child is content and on rectal examination there's no perianal soiling or excoriation. The rectum is empty and grips the examing finger.[10]

DIAGNOSIS:

History, physical examination and a set of specific investigations are essential for diagnosis.

Radiological examination is useful especially when suspecting intestinal obstruction. Distension of the colon with absence of air in the rectum is suggestive of Hirschspung's disease, although this should be interpreted with

caution in the first few days of life. Barium enema may identify the area of transition to dilated proximal bowel. A dilated left–sided sigmoid colon in a plain film or contrast study strongly suggests Hirschspung's disease.

Diagnosis is confirmed histologically by taking a full thickness rectal biopsy and demonstration of absence of ganglia. In acute presentation at laparotomy, several biopsy from rectum, colon, including the colostomy and ileostomy are taken. Frozen section biopsy is taken when facilities are available.

Pre-operative intravenous urogram is taken to exclude urological abnormalies. Additional diagnostic technologies include histochemistry, anorectal manometry, and electromyography mono–clonol antibodies. These facilities are present in specialised institutions but not at the MMC.

Tobin and Schuster, in comparing the diagnostic techniques in 100 patients with megacolon noted a 95 percent diagnostic accuracy with rectal biopsy, 80 percent with barium enema, and 100 percent in manometry in Hirschspung's disease.[8]

TREATMENT:

The aim of treatment of aganglionic megacolon is the operative relief of the symptoms by removing the abnormal lession. The only non–operative treatment is decompression by enemas.

Patients who have Hirschspung's disease presenting with acute intestinal obstruction should be prepared for emergency laparotomy and have a right transverse colostomy done. Ileostomy is performed in those with total

colonic aganglionosis.[3] A biopsy is taken to confirm the diagnosis. In this review, two patient presented with acute intestinal obstruction.

In chronic presentation, preoperative bowel washout is regularly performed before a right transverse colostomy is done. This is a preliminary decompression before definite surgical treatment is contemplated. Definite surgery is performed at the age of 1–2 year or when the infants weighs 9–10kg.[3]

Preoperative care include daily regular colonic cleansing using warm isotonic saline introduced either rectally or in through colostomy. In principle, it is done repeatedly until the colon is clear of fecal matter. Antibiotics (metronidazole, ampicillin) are given 3 days prior to surgery. There are centres which do not give antibiotic for fear of pseudomembrenous colities.[12]

Controversy looms as to which is the best definitive surgical procedure for aganglionic megacolon.[13] After Swenson's excellent results in 34 patients, morbidity and mortality in the hands of other surgeons has lead to the development of other alternative operative procedures.[2,14] The author had a priviledged opportunity in participating only in the Duhamel operative procedure. There are many other operative procedures, such as State–Rehbein anterior resection, Duhamel retrorectal trans–anal-pull through. The choice of definitive surgery is often according to the Surgeon's own preference.[3] Apparent complications include retraction or necrosis of pulled through intestine, leaking anastomoses, intestinal fistulae, pelvic abscess formation, intestinal obstruction, post-operative enterocolities and post-operative incontinence.[14] Specific complication related to Duhamel procedure is a feacoloma if the rectal spur is not completely crushed.

In a review of surveys conducted by the American Academy of Paediatrics of Hirschsprung's diseases (Klein-house 1979), anastomitic leaks were common following Swenson's procedure with an incidence of 11.2 percent compared to 5.8 percent in Boley's primary endorectal pull-thorugh, 2.4 percent Duhamel and 11.1 percent Soave. Individual American authors report anastomotic leaks following Swenson's procedures to range between 6 – 18 percent, in Duhamel 0 – 20 percent and endorectal pull – through between 15 – 30 percent.[14] Soave has summarized long term results of the surgical treatment of Hirschspung's Disease by various operations from information provided by many surgeons by questionnaire achieved for 8 years or more: Swenson 78.7 percent, Duhamel 81.45 percent,Rehbein 82.09 percent. Soave 82.61 percent. In general these good long term results clearly showed that no procedure was superior to the other.[2]

CONCLUSIONS:

Hirschspung's disease is common in Tanzania. However the incidence is unknown.[11] Most patients present with insidious onset and late and sometimes with severe complications. Clearly it is important to improve the public awarness of the disease.

References:

1. Leender's E. and Seiber WK : Congenital megacolon observation by Fredruck Ruysch – 1691 : J. of Paed Surg : 1970:5:1

2. Sieber WK.: Hirschspung's Disease – Current problem's in surgery (Ed) 1978:15(6)

3. Shija JK.: Paed. Surg case for developing countries (ED) Published by Heinemann Nairobi Kenya 1989.

4. Shija JK.: The Enigma of Hirschsprung's disease an African experience: E.Afr. Med. J. July 1988:65(7):438

5. Robin – Contran : Pathological Basis of disease 2ED W.B Saunder Company Philadelphia pg : 977

6. Donna. A. at el: Total intestinal aganglionosis: J. Paed Surg 1985;20(4):456–460

7. Shandling B.: Total colon aganglionosis – a new operation: J of Paed Surg 1984:19(5)

8. Schuster TF.: Megacolon : special diagnostic and therapeutic features, John Hopkin's Med J. 1974 135:91

9. Chadarevian JP, Slim M. : Double zonal aganglionosis in long segment hirschsprungs disease with a"skip area" in transverse colon. J. Paed Surg. April 1982 : 17(2) : 195

10. Harding Rains and Charless Mann. Short practice of surgery : 20 ed H. K. Lewis and Co. Ltd London 1990 PAGE 1028

11. Carneiro PMR, and Komissarov. IA : Management of Hirschspung's disease in Dar es Salaam : Tanzania Med. J. 1990;5(1):11

12. Ray Postuma : Whole bowel irrigation in Paed patients J. of Paed Surg. 1982 17(4) : 350

13. Cauly TG.: Modified Duhamel procedure for treatment of Hirschspung's Disease in infancy and childhood J of Paed. Surg. 1982;17(6):773

14. Valcek FT Klotz DH : operature failure and secondary repair in Hirschspung's Disease disease:J. Paed Surg 1982;17:(6):779

CHAPTER EIGHT

UROLITHIASIS

ABSTRACT:

During the surgical rotation in the Urology Unit the author participated in the management of five patients. However data of fifteen more patients was reviewed from a special registry of urinary stone in the department. One patient is presented in detail while the rest are discussed and reviewed briefly against the background of the relevant literature.

The age of the patients ranged from 21 to 71 years with a mean of 40 years. Males were predominant with a ratio of 3:1. Calcium oxalate stones were the commonest of the 20 patient studied. Stones in the renal pelvis constituted 42.86 percent, ureter 23.8 percent, kidney19.1 percent and the urinary bladder 9.5 percent .

Eight patients had pyelolethotomy, 4 ureterolithotomy, 3 cystolithotomy and 2 were managed conservatively. Four patients were scheduled for surgery but were not operated on because they were lost for follow–up.

INTRODUCTION:

During the last 25 years there has been great progress in the management of patients with urinary calculi. In most countries information available on renal stone epidermiology is hospital based, which may be different from the actual number of cases present in the community.[1] It is generally believed that this disorder is relatively uncommon among the sub–Saharan African

population and black race in general but, commoner in the developed world afflicting 3–4 percent of population.[2,3]

Studies conducted in Dar es Salaam indicate that the prevalence of urinary stone could be higher than previously reported, especially with improvement in living standards, urbanisation and change in lifestyle.[3,4,5] The higher incidence of upper urinary tract stone correlates with the more sedentary life in technologically developed countries.[2]

CASE REPORT:

M.K.N. A 23 year old lady was seen at the Urology Outpatient Clinic due to a one year history of pain in the left flank. The pain was of gradual onset and was intermittent and dull in nature. The pain was exacerbated by exercise. She had no chills, fever or vomiting attacks.

Micturition had been normal. Neither had she had an episode of yellow colouration of the eyes. Her appetite had been normal and she had never been on anti–acid treatment. She was a secondary school girl (Form IV). She denied any use of alcohol or smoking of cigarettes. On examination she was a young lady, afebrile, not pale with a pulse rate of 68 beats/min and a blood pressure 130/70 mm Hg. The abdomen was soft and not distended. The renal angles were not tender and the kidneys not palpable. The rest of the abdomen was normal. Other systems showed no significant abnormality. A clinical impression of urinary calculi of the left kidney was made.

Summary of Investigation:

KUB:	showed left opacification – suggesting a renal stone
IVU:	a 2 cm diameter left renal pelvis stone, hydronephrotic kidney but normal right kidney.
Ultrasound:	hydronephrotic left kidney with a calculus in the renal pelvis.

Serum Calcium	: 2.26 mmol/l
Uric acid	: 0.27 mmol/l
Phosphates	: 0.75 mmol/l
Creatinine	: 105 mmol/l
Urine culture	: no significant bacterial growth
Haemogram	: Hb 13.7gm dl.
WBC & Platelates	normal peripheral film and distribution

The patient was thus scheduled for left pyelolithotomy and blood donors were recruited and blood reserved at the blood bank.

Operation:

Under general anaesthesia the patient was placed in the right lateral position with a sand bag on the right loin to achieve a lateral flexion. The skin was cleaned and draped. A lumber incision was made along the 12th rib obliquely. With a diathermy the wound was deepened and cut muscles were retracted exposing the loose fat of the retro–peritoneal space. While protecting the peritoneum, the renal facia was identified and incised between

forceps and extended enough to expose the kidney. By blunt dissection the kidney was gently mobilised and delivered through the wound. The ureter was isolated and followed in ascending fashion up to the pelvis. Between two stay sutures, the pelvis was opened over an area where the stone was most accessible. A stone which was white and with a smooth surface was easily extracted. The pelvis was thoroughly irrigated with normal saline. The renal pelvis was closed with 3/0 chromic catgut and a tube drain left in the perinephric space. The perinephric fat, and the wound were closed in layers.

Post-operation follow-up (summary)

Immediately post operatively:

1. Pethidine 50mg in 8 hourly x 3 doses for pain
2. Dextrose saline IV 3 hourly/24 hours
3. Pulse BP hourly for 12 hours, then 4 hourly

6 hours post operative:	The patient was awake, but noted a large leak through the drainage.
1st post operative day:	The patient was awake started on oral fluids. Urine output 600cc over 20 hours and IV fluids stopped.
6th day:	Leakage had decreased and the patient was ambulated
9th day:	No urine leakage, all sutures removed and tube removed

11th day: The patient was discharged home.

4th week: She was seen at follow–up clinic. The wound had healed and she was allowed to return to school.

RESULTS:

Table I **SEX, AGE DISTRIBUTION**

Age	Male	Female	#	%
20 - 30	1	3	4	20%
31 - 40	6	2	8	40%
41 - 50	3	-	3	15%
51 - 60	3	-	3	15%
60+	2	-	2	10%
	15 (75%)	5 (25%)	20	100%

Mean age was 40.1 years. Age range 20 – 73 year and sex ratio M:F was 3:1. The disease affected mainly the age group 30 – 40 years.

Presenting Features:

Table II

Features	Frequency	%
Loin pain	17	48.6%
Frequency	7	20%
Bilharzia	4	11.4%
Renal tenderness	3	8.6%
Hematuria	2	5.7%
Previous stone	2	5.7%

As seen from table II (above),

Loin pain was the commonest feature.

SITE OF CALCULI:

TableIII

	Total #	%
Renal calcyces	4	19.1
Renal pelvis	9	42.9
Ureter	5	23.8
Urinary bladder	2	9.5

Table III reveals that renal pelvic stone was the commonest.

Table IV TYPE OF TREATMENT

	Treatment	
	No.	%
Pylolithotomy	8	40%
Ureterolithotomy	4	20%
Cystolithotomy	2	10%
Conservative	2	10%
Others	4	20%

Seventy percent of patients underwent open–surgery and pyelolithotomy constituted the majority. The group on "others" were those patient who were not operated because they were lost from follow–up.

DISCUSSION:

EPIDEMIOLOGY:

There can be few branches of medicine in which improvement in the method of treatment are occurring with such rapidity as in urolithiasis.[6] Perhaps more surprising is that despite the sophisticated modern technology, basic information about the pattern of disease is remarkably little understood. The data analysed in most cases is hospital based.[1]

The disease has a geographical variation. Bladder stones are said to be common in underdeveloped countries whereas stones in the upper urinary tract are said to be commoner in the richer industrialised countries which is thought to correlate with sedentary life in technologically developed countries.[2,6,7,8]

In Western countries, the overall prevalence is 3–12 percent.[1,6,8] Urinary stones are said to be uncommon in black people, this includes Bantu Africans and blacks in the USA.[9] However, one must be cautious in accepting of prevalence of urolithiasis amongst Africans since this could be based on inadequate information.[3,5,10]

AETIOLOGY:

The cause of renal stone is multifactorial. Most studies are based on hospital statistics and are concerned more with the relationship between, diet, race, climate and other geographical factors.[1,6] Metabolic disorders, urinary tract infection, and urological abnormalities are important factors to be considered.

Arbitrarily, causes of urinary calculi can be considered as idiopathic, hereditary, metabolic or due to infection.

Metabolic conditions like hyperparathyrodism and Cushing Syndrome are associated with calcium stone formation. This accounts for 5 – 10 percent of all stones.[1,11] Conditions associated with hyperuricemia favour uric acid stone. This includes primary gout, myeloproliferative disease, high dietary purine intake, and dehydration. In this study no patient had history of gout. All patients had normal calcium levels.

Urinary tract abnormalities cause inadequate urinary drainage giving rise to stasis and infection. Colonising bacteria split urea to form ammonia and carbon dioxide. Stones formed secondary to urinary tract infection are composed of magnesium ammonium phosphate with variable amounts of calcium. Higher urinary pH and ammonia concentration are risk factors. Hereditary hyperoxaluria and cystine stone disease are rare hereditary conditions which present themselves in early life. No patient in this study had a similar history. Idiopathic calcium stones account for about 87 percent of all calcium stones.[6,8]

Type of stones:

Oxalate calculi (calcium oxalate): These account for 80–85 percent of all stones. They are usually single and covered with sharp projections which inflict injury causing bleeding and hence giving symptoms when comparatively small. The altered blood is precipitated on the surface of the stone. Radiologically they are opaque. They were the most common type in this study.

Phosphate calculi account for 10 percent and usually occur as calcium phosphate or a combination with ammonium and magnesium phosphate. They are dirty white and smooth. They often enlarge rapidly in higher urinary pH, taking the shape of renal calcyces (staghorn calculus). These stones give few early symptoms by virtue of their smoothness until they attain enormous size.

Uric acid and urate stones are typically faceted, hard, smooth and multiple in number. Pure uric acid stone are rare. They are radiolucent unless mixed with calcium.

Cystine stones are rare. Their crystals are hexagonal. The stones are white translucent and multiple. Xanthin calculi are also rare. They are smooth, round and brick red, showing lamellar structure.

PATHOGENESES:

Urolithiasis is a result of multiple and complex interrelated phenomena. The matrix and crystals are invariably fundamental components of each stone morphorpogicaly.[12] The matrix is a mucoprotein. Immunological technique have demonstrated it as of renal origin.[2] Crystals of organic and inorganic composition on a weight to weight basis form the major portion of urinary calculi and cover or fill the gel like matrix in variable degrees.[12] Several factors lead to crystal formation : (i) Increased urinary excretion of crystalloid, variation in urine volume and urinary pH which result in diminished crystalloid solubility; (ii) diminished activity of urinary inhibitors which normally helps to solubilise the crystalloid. Obstruction of urine outflow with staseis favours infection with urea splitting bacteria. Triple phosphate

stones are associated with infection.

CLINICAL FEATURES:

The male to female ratio is 4:3 and peak age incidence is 20 – 50 years.[11,13] In this study the mean age was 40 years with a range of 20–73 years and male to female ratio was 3:1. Peak age incidence was between 30–40 years.

Stones composed of phosphate may remain asymptomatic for a long time, until obstruction and infection supervenes.[9] Abdominal pain or flank pain and renal colic with or without passage of stones are common features. Fixed renal pain may be located in the renalangle posteriorly or in the hypochondria anteriorly or both. Pain is aggravated by movement. Ureteric colic is agonisimg, usually radiates caudally to the suprapubic, perineum or to the base of the penis in the male or vulva in the female or inner thigh. Hematuria may be micro or macroscopic. Urosepsis as pyuria may occur due to infection.

In this study, 48.6 percent of subjects complained of pain and 20 percent of increased frequency. Only 5.7 percent had non–bilharzia haematuria. No patient presented with pyuria. Renal tenderness could be detected in 8.6 percent of the patients.

INVESTIGATION:

The main objectives are to classify the type of stone, to define the site and urinary risk factor giving rise to stone. In addition it seeks to diagnose the pre–urinary factor or disease causing abnormalities in the urine.

A morning specimen of urine analysis for macro and microscopy and culture is essential whenever a renal stone is found. Urinary pH lower than 5.5 is suggestive of uric acid stone whereas pH above 7 suggest renal tubular acidosis or infection with urea splitting organism. A cystine screening test (cyanide nitroprusside) is essential for cystine stone. Serum electrolytes and especially if calcium is elevated, parathyroid hormone assay should be performed. Elevated serum uric acid would suggest uric stone. The renal function will be reflected by the level of urea and creatinine clearance. Renal stones are often seen on plain abdominal X–ray. IVU can establish the precise location of stone and the associated abnormality or complication. Non–radio opaque stone (uric acid) are diagnosed as filling defects. Rectograde pyelography is usefull in ultrasound, displaying presence of a ureteric calcutus non–opaque to X–ray.

TREATMENT:

Generalisation on the management of urinary calcium is difficult. The decision rests upon the symptoms, patients age, size, numbers and location of stones, presence of infection and the degree of functional renal impairment. However, suffice it to mention that the management can arbitrarily be divided into treatment of pain, infection, removal of stone and the prevention of recurrence.

Pain if severe is relieved by strong analgesia. Contrary to former teaching there's no evidence that morphia analogues cause ureteric spasms.[6] Infection should be treated with an appropriate antibiotic. Stone removal can be conservative or surgical. The recent introduction of endoscopic approach to renal surgery and extracoporeal shock wave lithotripsy has revolutionised the treatment of urinary calculi.

CONSERVATIVE MANAGEMENT:

This is considered in a few selected patients with sterile urine. Small stones with diametre of 5mm or less may pass in urinespontaneously. Food in the diet that may promote stone formation is avoided and patients should take plenty of fluids to promote diuresis.

Drugs like penicillamine are recommended for cystine stones. Magnesium and the thiazides are sometimes used in treating patient with urolithiasis.[14,15]

SURGERY:

Only 5 percent of all stones require open surgery. Conventional open surgery is now reserved for those conditions associated with urodynamic disorders which have to be corrected at the same sitting as when the stone is being removed or a staghorn calculus with pyeloureteral junction stenosis;[7] there include repeated attacks of stones, evidence of increase in size of a stone, evidence of infection and or stone considered too large to pass. In this study open surgery was the procedure of choice for all patients.

All operations have a certain feature in common, the kidney is exposed via a lumbar incision and is mobilised and delivered. Complex renal surgery can be performed after clamping the renal artery. Renal Ischaemia is prevented by cooling the kidney to 20^{o}C for 2–3 hours. Stone to be removed should coincide with the X–ray findings.

Approach to ureteric stone located in the upper third needs a similar lumbar incision. In uteric stones located in the middle or lowerthird the incision is

above the anterior superior uliac spine and the ureters exposed extra peritoneally. For stones in the juxtavesical or intramural portion of ureters a midline incision subumbilical approach is employed.[11]

Pyelolithotomy was performed in 8 percent of patients in this study. In the literature this procedure is indicated in extra–renal stones situated in the pelvis. When the stone has been removed a sound is passed through the pelvis–ureteric junction to exclude distal obstruction. Nephrostomy is performed if the kidney is grossly infected. Only 20 percent had ureterolithotomy in this study. Cystolitholomy was done in only two patients who had vesical stone that were removed during an elective prostatectomy. Stones in the ureters can also be removed by an antegrade approach by the use of urethoscope and basket or disentegrated with laser or ultrasonic rays. Bladder stones can also be removed by a lithotrite punch (Litholapaxy) or electrohydraulic lithotripsy. Stones in the urethra are endoscopically removed.

Percuteneous Renal Surgery:

The patient is in prone position. Under fluoroscopic guide, a needle is passed posterolateral between the iliac crest and the subcostal margin, then transparenchymally into the lower calyx. The needle tract is progressively dilated and a plastic sheath (Amplatz) is passed over the largest dilator. The plastic sheath provides a conduit for the stone to be removed. Large stones are disintegrated with an electrohydraulic discharge or ultrasonically andresultant particles removed by suction or passed in urine. A nephrostomy tube is left in for 24–48 hours. This procedures is successful in 97 percent of patients,[9] post operative mobility is minimal and hospital stay short. However, facilities for this procedure are not available in this centre.

Extra corporeal shock wave lithotripsy:

The patient is anaesthesed or sedated and placed in a water bath. The renal stone is disintegrated by generating an ultra–short high tension discharge within an ellipse focused in the stone until the stone is fragmented. The sandy fragments are then micturated. The passage of these fragment may cause ureteric coliky. Stone of almost any size, composition and situation are suitable for this treatment.[2,6,16]

Concluding Remarks:

Many available studies indicate Bantu Africans and black race in USA are exempted from stone disease.[12] Most published information is based on hospital data. The prevalence in our community could be higher than expected, therefore, a community based study is recommended to assess the depth and bredth of stone disease here.[5,6]

References:

1. Scott R.: Epidermology of stone disease : Br. J. of Urology 1985;57:491–497.

2. Reza S. Malek : Renal lithiasis : A practical approach : J. of Urology : Dec. 1977 : 118 : 893.

3. Mkony CA.: Urinary stones in Dar es Salaam. 1991 EAMJ:68(6)

4. Mkony CA. : Percutaneous upper urinary stone surgery. E.A. Medical Journal 1989:21–25.

5. Kirei. B. R. : Renal stones disease in African in Dar es Salaam : MEDICOM 1983: 5 : 77.

6. Hugh N. W. : Urinary Calculi : Surgery : 7 : 1713

7. Damiano Turini : Staghorn renal stones: virtues of bench surgery and autotransplantation Journal of Urol 1977:118.

8. Munro Peacock : Renal Calculi : Medicine 1980 : 27 : 1372

9. Brannen GE.: Kidney stone removal percutaneous Vs Surgical lithotomy. Journal of Urology: 1985;133:6

10. Elem B. : Urinary Calculus in Zambia: British J. Urology 1984 56:44–47.

11. Harding R. : Short practice of surgery 20th ED 1990 : H. K. Lewi & Co. Ltd. London page 1222.

12. Malek. R. S. : observation in ultrastructure and genesis of urinary calculi : J. Uro. 1977 : 117 : 336.

13. Fetter T. R. : statiscal analysis of patients with ureteral calculi:JAMA 1963:186:21.

14. Diatrich B. L. : A classification of urinary calculi with respect to their composition and micromorphology: Scan J. Urology nephrol 1991:25:141:150.

15. Edreund R. Y. : Renal Calculi: Medicine : Dec. 1982 1:(24)

16. Smith L. H. : The diagnosis and treatment of metabolic stone disease : Med. Clin. N. Amer 1972 : 56 : 977.

which patients to admit and which to transfer to special neurological unit also has a medico–legal implication.[1] In developing countries neurosurgical services are adversely affected by lack of investigative facilities such as CT–Scan, MRI which are essential in the modern management of head injured patients.

CASE REPORT:

A male aged 11 years was crossing a road oblivious of on coming vehicle and was knocked down and fell on the roadside and lost consciousness. He was brought to the casualty one and a half hours later in the company of his father and a policeman. He had lost consciousness for 20 minutes. He remembered events before the accident. He had sustained a laceration on the scalp and bruises on the right arm. He was bleeding from a cut wound on the mouth. He denied any history of medication. He was not epileptic and there was no epilepsy in his family.

On examination he was a young boy not dyspnoiec or pale. He could open eyes spontaneously on command and responded appropriately to questions. His motor responses were normal. At the time of admission he scored 14 points (Glasgow Coma Scale). His pulse rate was 90 beats per minute and blood pressure 110/70 mm Hg. The pupils were equal, and reacting to light. Further examination revealed a minor cut wound in the oral cavity, a scalp lacerationand bruises on the right arm with no evidence of fracture. He had no neurological deficit. The abdomen, chest and spine revealed normal findings. A diagnosis of brain concussion, with soft tissue injury was made, x–ray films of the skull and left arm revealed no evidence of fracture.

Treatment consisted of a dose of injection T.T. 0.5ml stat and intravenous dextrose/saline litre for 24 hours. A head injury charting card was started. Wounds on the scalp were cleaned with normal saline and antiseptic then covered with sterile dressing. After 24 hours of observation he was discharged in apparent good condition and advised to return in case he experienced instances of persistent headache and vomiting, loss of consciousness or weakness of limbs. He never returned.

RESULTS AND DISCUSSION:

AGE AND SEX DISTRIBUTION:

Table below summarises the age and sex distribution of 59 patients studied.

TABLE I DISTRIBUTION OF AGE AND SEX

Age (years)	Males	Females	Percent
10-19	4	1	8.5
20-29	17	4	35.6
30-39	12	3	25.4
40-49	7	1	13.6
50-59	5	1	10.2
60-69	3	0	5.1
70+	1	0	1.7
Total	**49 (83.1%)**	**10 (16.9%)**	**100**

In this series of 59 patients 83 percent were males and 16.9 percent females making a ratio of 5:1. The age ranged from 10–71 years. The commonly affected group were in the second and third decades making a total of 61

percent probably because these are active ages. Males were more affected than females. This is probably related to their activities.

CAUSE OF INJURY:

Road traffic accidents accounted for 57.3 percent of all causes of head injury in this study, while 37.3 percent were due to assaults.

TABLE II CAUSE OF HEAD INJURY

cause	Frequency	Percent
Road traffic accidents	34	57.6
Falls from a height	3	5.1
Assault	22	37.5
Total	**59**	**100**

Kirambe reviewed 23,460 patients in Tanga in the northern part of Tanzania 10 years ago.[2] He observed 91 cases of head injuries. 42 percent were caused by assaults and that road traffic accidents accounted for 12.9 percent of trauma. It's interesting to note that alcohol was the major contributing factors. In this study about 60 percent of patients especially males smelled of alcohol making intoxication a contributing factor for injury.

Pathophysiology of cranio–cerebral injuries:

The close proximity of scalp to the skull allows laceration to occur easily with blunt injury.[1,2] The unyielding character of the scalp and it's unique anatomical structure predisposes it to a tendency of considerable blood loss if not controlled. Fracture of the skull may occur in any area of the skull and

has a tendency to radiate from the point of contact into weaker area of the skull e.g. basilar or temporal.[3]

The immediate damage that results from head trauma is dependant upon the energy absorbed by brain tissue, the damage can be diffused or focal.[3] This damage may occur by direct disruption of brain tissue by a penetrating object or indirectly by distortion of brain tissue from a rapid deceleration of brain against the rigid skull. At the moment of impact diffuse neuronal lession is responsible for the primary clinical features. The majority of severe cranio cerebral injuries due to road motor accidents are the result of rapid deceleration of the brain when the moving head strikes a stationary object. In assault the stationary skull is struck by moving object. These factors produce the features of distortion aggravated by brain mobility which may induce transitory damage or persisting damage to the brain stem and reticular formation.

Brain tissue responds to trauma by altering it's intra–cellular and extra-cellular fluid content. Tear of cerebral vessel cause hemorrhage and alteration of cerebral blood flow. Edema and hemorrhage cause brain "swelling" producing neurological disfunction due to increased intro–cranial pressure and transtentorial herniation. In clinical practice, patients who have intra–cranial pressure (ICP) values greater than 40 mm Hg. have a significant diminished cerebral blood flow. Brain death occurs when this pressure exceeds or equals systemic arterial pressure. The functional relation of increased intracranial pressure (ICP) , systemic arterial pressure (SAP) and cerebral blood flow (CBF) can be demonstrated as follows:

$$CBF = \frac{(SAP - ICP) = K}{VR}$$

K = constant
VR = vascular resistance

Cerebral edema, enlarging hematoma or cerebrovascular engorgement acts on the supratentorial mass resulting not only in increasing intra-cranial pressure but also distortion and herniation of brain tissue through the tentorial notch or through the foramen magnum. Transtentorial herniation is the commonest and results in distortion of midbrain, 3rd cranial nerve and posterior cerebral artery.

Hypoxemia may be caused by central damage to brain stem with loss of adequate respiratory drive and pulmonary edema. Hypotension is rarely a result of intracranial trauma. It's usually a result of multiple injuries with oligemic shock.[4]

MANAGEMENT OF HEAD INJURED PATIENTS:

RESUSCITATION:

Assessment and stabilisation of the cardiorespiratory system is important because a combination of hypoxia, hypercapnia and hypotension are potent factors which aggravate severe brain edema and venous congestion. It is therefore essential that the airway should be cleared to ensure adequate breathing and ventilation. In severely head injured patients an endotracheal tube provides protection against hypoxia and hypercapnia. Replacement of fluid and blood loss should be determined by normal requirement and renal flow. Control of severe bleeding and resuscitation should take priority. Thorough evaluation is important to detect spinal injury and concealed bleeding in the abdomen and chest.

EVALUATION:

Following the stabilisation of respiratory and cardiovascular systems, attention should be directed to assessment of the neurological system. A thorough head to toe physical examination is done to exclude associated injuries. Table III in this study indicates some of the associated injuries, fracture of the ribs and humerus was commonest.

TABLE III ASSOCIATED INJURIES

Site	Frequency
Fracture Ribs	4
Fracture Humerus	3
Fracture tibia/Fibular	2
Fracture femur	2
Pneumothorax	2
Fracture Clavicle	1
Fracture Mandibular	1
Ankle injuries	1
Total	16

Duration of the loss of consciousness is regarded as a good indicator of the seriousness of head trauma.[1] The level of consciousness is usually assessed by a scale designed to assess the depth of impaired consciousness. There are authors who advocate comprehensive level of consciousness level (CLOCS).[5] However the author is familiar with the Glasgow Coma Scale (GCS) which is easy and reproducible. It is the one commonly used in he department of trauma/orthopaedics. The GCS is not diagnostic but defines the state of altered consciousness in terms of motor responses, verbal response/performance and eye opening, refer to Appendix I. The scale

correlates with mortality rate. Patients with score less than 5 points have a morality rate of less than 50 percent. In this study six patients had points less the 5 and all died within 48 hours of admission.

Invariably all patients in this series had X-ray of the skull taken at the emergency department. Only 15.8 percent showed a fracture, raising the controversial question, particularly in developing countries, of the rationale of skull X-ray in all head injured patients. Levy, in studying 100 patients who had head trauma found only 17 percent with skull fracture and yet only 7 percent didaffect treatment.[6] Levy concludes that in Africa a prospective study is needed to look for either high yield finding in physical examination, which would help to reduce cost. CT-scan are the most accurate guide to intra-cranial injury but there is no such facility in this centre. The cardiovascular and respiratory systems are monitored with ECG, blood gases analysis, serum electrolytes, arterial pressure and pulse.

TREATMENT:

SCALP LACERATION:

Scalp laceration affected 12.7 percent of patients in this series. The scalp with its major blood vessel lying between the galea and the dermis has a tendency to hold blood vessel open following laceration with considerable blood loss. Hemostasis can be achieved by compression of the scalp against the skull in circumferential fashion or by direct pressure. Infection of scalp may spread into epidural space through emissary veins in the diploic space of the skull and into the vein into the brain tissue. Therefore scalp wound should be thoroughly cleaned, debrided and all devitalized tissue and foreign body removed.

SKULL FRACTURE:

There were 15.8 percent of patients with skull fracture. They were either simple or compound involving the base or vault of the skull. These fractures included linear or communuted with or withoutdepression. Simple linear fractures unless involving special site (pterion) were managed conservatively. In compound fractures the wound was managed as above mentioned and deep aspect explored for bleeding and foreign bodies. Depressed fractures of 0.5cm of the inner table were elevated except those crossing sagittal or transverse sinus. Complications of depressed fracture are dura tear, pressure upon the cerebral cortex or/and hemorrhage. Depressed fracture may obstruct the superior sagital or lateral sinuses leading to increased intra–cranial pressure. It should be emphasised that there is a risk of severe hemorrhage if depressed fracture over a sinus is elevated. Patients who presented with CSF rhinorrhea or otorrhea were nursed in elevated position of the head given a combination of chloramphenicol and penicillin for 10 days.

BRAIN CONCUSSION:

These were patients who had lost consciousness of up to half an hour followed by complete recovery. They constituted the majority 42.9 percent in this study. In principle they were observed for 24 hours and discharged if their clinical condition remained stable on instructions of re–admission if the condition changed.

TABLE IV TYPE OF HEAD INJURIES

	Frequency	%
Brain concussion	27	45.8
Brain contusion	15	25.4
Skull Fracture	9	15.3
Scalp wound	8	13.6

CEREBRAL CONTUSION:

These patients have a loss of consciousness for more than half an hour with amnesia or/and deterioration of level of consciousness. This group constituted 28.8 percent. In cerebral contusion there is organic structural damage to the brain tissue which may be diffused or localised. Hemorrhage may occur within brain tissue. A combination of brain edema and haematoma contributes to brain swelling and features of raised intracranial pressure which include deterioration in the level of consciousness (GCS) slowing pulse, slowing respiration and rise in blood pressure. Treatment is directed to reducing brain edema which is the most frequent cause of deterioration rather than compression intra cerebral haematonia.[6] Mannitol (20 percent) usually given at dose of 1–1.5 mg/kg. acts as an osmotic diuretic. Fursemide at dose of 40–80 mg is an alternative. The basis of this treatment assumes that by raising the plasma osmolarity, fluid from brain tissue will be drawn thereby reducing the brain edema and swelling. Dexamethasone at dose of 10mg stat then half the dose repeated 6 hourly is advocated although in head injury it is of doubtful value.[4,6] The patient is given restricted amount of fluid 1ml/kg body wt/hour in a form of dextrose saline. The airway was cleared to ensure good lung ventilation. All patients were

monitored with Glasgow Coma Scale (GCS).

Progressive loss of consciousness following head injury should always be assumed to be due to intracranial hemorrhage until provedotherwise.[3,6] The pattern of neurological change vary with type the of hemorrhage these include extradural, subdural or intracranial hemorrhage.

EXTRADURAL HEMORRHAGE:

This commonly results from a tear of the anterior and posterior branches of the middle meningeal artery by a fracture fragment. Classically there is a short period of loss of consciousness followed by a lucid interval and deterioration of the conscious level. There is pupillary dilation on the side of the lesion. Displacement of the temporal lobe by the haematoma cause it's medial aspect to press against the 3rd cranial nerve above the edge of the tentorium. A faunal flap must be turned and the haematoma evacuated.

SUBDURAL HEMORRHAGE:

Subdural hemorrhage is caused by either rupture of veins traversing the subdural space or laceration of brain that has torn the overlying piarachnoid. True lucid interval is unusual. The haematoma is usually extensive. Special investigations like MRI, CT–Scan or arteriogram may influence the outcome of surgery. Exploratory bore holes are usually carried out. Special investigations like MRI and CT–Scan are not available in this centre. There is a possibility that such patients are missed resulting in preventable deaths.

INTRACEREBRAL HEMORRHAGES:

Intracerebral hemorrhage is said to be rare and usually due to a rapture of a major cerebral vessel or burst temporal/frontal poles. Preoperative diagnosis with the aid of CT–scan and arteriogram is essential. Treatment is to arrest the bleeding.

CONCLUSIONS:

Head injury is one of the commonest problem in the department of orthopaedic and trauma. Road traffic accidents and assault are the major cause of head injuries. The inter–relationship between driver, the vehicle, the road and the pedestrian is very important determining factor. Early assessment of head injured patient should be done to save life and protect the brain from further damage. The emergency department staff and the neurosurgical unit should be strengthened in terms of personnel and essential equipment. It would be interesting to study the number of patients with head injuries who die before reaching the neurosurgical unit.

APPENDIX I : GLASSGOW COMA SCALE (9)

Criteria	Response	Score
Motor response (M)	to verbal command	6
	to painfull stimulus	
	localizing	5
	flexor withdrawal	4
	abnormal flexion	3
	extensor response	2
	nil	1
Eye opening (E)	spontaneous	4
	to speech	3
	to pain	2
	nil	1
Verbal response(V)	oriented	5
	confused conversation	4
	inappropriate word	3
	incomprehensible sounds	2
	nil	1

References:

1. Allan H. Friendmain : Craniocerebral injuries in text of surgery sabistone (ed) 1986.

2. Harding Rains : Cranio – cerebral injuries : Barley and Love (Ed) 20th Edition 1990. page 437.

3. Denis D. Spencer, Neurologic Surgery. In Principles of surgery Schwartz (ed) 1989, page 1835.

4. Kirambe M. Med. Proceeding of Ass. of Surg. of EA. 1984.

5. Jenneth. Bryan : Severe head injuries : ethical aspects of management : British J. Hospt. Medicine March 1992, 47(5).

6. Levy L.F. Are skull Radiographs necessary in head injury : Proceed of Ass. of Surg. of EA. 1984

BIBLIOGRAPHY

1. Ahlamit DA.: Haemoccult a new quantitative assay of faecal haemoglobin Ann. Int. med. 1984;101:297.

2. Allison JE.: Hemocault screening in detecting colorectal neoplasm : sensitivity. Specificity and predictive value Ann. Int. med 1990:112(5):328.

3. Arnaud JP: CEA in dragnosis and prognosis of colorectal cancer 1983:23:(3):141.

4. Baralett J.G: Campylobactor pylori : Fact or Fancy Gastroenlerol. 1988;94:229 – 38

5. Barroso F.L.: Duodenoplasty and Proximal Gastric vagotomy in peptic stenosis, Arch. Surg. 1986;121:9

6. Bates D. Predicting recovery from coma Br. J. Med. 1983 30:27–82

7. Benion : Risk factors for development of cholilithiases in man: N. Eng. J. Med. 1978:299:1221

8. Bornman P.C: Simple closure of perforated duodenal ulcer. Br. J. Surg. 1991;77:73

9. Breynon J. Ann. Royal College of Surgeon of England 1989:71:131.

10. Bruce E.: Duodenal ulcer : disease Evolution Curr. Prob. Surg: Jan 1984:xxi:1:50 – 52

11. Calon CA : Prognostic correlation of operable cancer of the rectum : Dis. of col and rect. 1983 : 26(2):98

12. Carthy JSM.: Age – dependant closure of PDA by indomethacine: Paediatric 1978;62:(2)706

13. Choichi Sugawa: Endoscopic retrograde cholangiopancreatography after cholecystectomy: Surg. Gynaecol obslet. 1983:157 (3):247

14. Chritensen BD : whole gut irrigation Vs enema in elective colorectal surgery : Disease of colon and rect : 1981 : 24 :(8) : 592.

15. Clatworthy: the optimum age for surgical closure of patent Ductus Arteriosus:JAMA 1958;167:444

16. Cocani F. Olley: the Response of the ductus arteriosus to prostaglandins: Cand. J. Physiology Pharmacol 1973;51:220

17. Collin – F. W. Duncan. Head Injury. In principles of Surgery Schwartz (ed) 1985 4th ED.

18. Daniel F. Stauczak Assessment of level of consciousness following severe head injury: J. Neurosurgery 1984 : 60 : 955 – 960.

19. Dial E.J.: Surface hydrophodicity of Gastric mucosac in developing rates Gastroenterology 1988;94:57–61

20. Disease of colon and Rectum: Local recurrence following curatine resection of carcinoma of the rectum.1983 : 26(2) : 98.

21. Disease. of colon and rectum 1979 : Radiotherapy for rectal cancer 1979: 22(7) : 455

22. Dolman's WMV : Diagnostic yield of endoscope upper Gastrointestinal bleeding Trop. Geog. Med(2) 1983:173–78.

23. Dolman's WMV : Evaluation of 140 proximal GIT endoscope : East. Afr. Med. J. 1979;56:671.

24. Dooleny C.P., Cohen H.: clinical significance of Campylobacter pylori: Ann. Int. Med. 1988:108

25. Elechi - ED. : Intussusception : Is floating caecum a causative factor? *EAM* Nov. 1990 67 : (11).

26. Elliot R. Starling M. B.: the effect of PGF in the closure of ductus Arteriosus : Prostaglandins 19722:399

27. Eshleman L. Caneiro P. Paediatric Urologic Survey in Tanzania TMJ Sept. 1990.

28. Franken EA. The use of glucagon in hydrostatic reduction of intussusception : a double study of 30pt. *Radiology* 1983:146 : 687 - 89.

29. Gleen S.: A Pilot study of sphincter sparing management of adenocarcinoma of the rectum : Arch. Surgery 1991 : 121: 696.

30. Glen. M. The current role of radical treatment of nephrolithiasis : J of Urology July 1985 134 : 6

31. Haenszel W. : Cancer of the colon and rectum and adenomatous polyps : a review of epidermological findings : cancer 1971 : 28 : 14.

32. Herman R.E: Obstructing Duodenal ulcer Surg. Cl. North America 1971;56:1403.

33. Heyman MA. : Closure of Ductus arteriosus in Premature infants by Inhibition of PG Synthatase N. Eng J. Med 1976;295:530.

34. Howard R. Champion : The major trauma outcome study: Establishing the national works for trauma care: J. Trauma 1990 30:11:1356

35. Hulme Moit I : Intussusception in Moshi (Tanzania): *Proceedings of Ass. of Surgeon of EA.* 1979, 2:114.

36. Jaffin BW & Kay M.D.: The prognosis of Gastric outlet obstruction Annals of surgery 1985;201(2): 176.

37. Jaiyesinu F.: Clinical diagnosis of congenital Heart Disease Medicine digest 1984;10:2

38. Karlstrom, : Percutenious Pin fixation of open tibia fracture : J. Bone and Joint Surg. 57–A:915–924

39. Kefene H. : Gallstone disease in an Ethipian referral hospital (Published errutum in Ethiop. Med. J. 199028(1):47 Ethiop. Med. J. 1980, 27(4): 209

40. Killermasn JA. Patient ductus Arteriosus – Current Clinical status:Arch. Disease childhood1980:55:106-9

41. Lahlinen J. Alhava EM : Acute cholycystitis treated by early and delayed surgery : Scan J. Gastroent 1978 : 13

42. Mbaga J.M : clinical and radiological evaluation of peptic ulcer. Dar. Med. J. 1982:(1)

43. Mbaruku CMD: Motor traffic accident in Dar. their effect and mortality. Dar. Med. J: 1980, 8 (1): 16–70

44. Mendelow. A. David. Management of head injury : 1990 March Hospital update.

45. Michael. D. Klein, Arnold. G. Coran : Hirschspung's Disease in new born : J of Paed Surg. May 1984 19:(4)

46. Misiewicz J.J. : The peptic ulcer dilema Med. Diges. 1989:15

47. Mwakyusa D.H: Modern treatment of peptic ulcer and role of surgery : Tanz. Med. J. 5:(2)

48. Pahlman L. : Pre – Vs – post operative radiotherapy in rectal cancer:1985;72:961.

49. Pamela H.: Peptic ulcer during H_2–receptor antagonist era. Br. J. Surg. 1991:78(1)

50. Papillon J.: New concepts in conservation treatment of rectal carcinoma : Dises of rect. colon 1984;27: 695.

51. Park. D.N.: Duodenal ulcer stenosis with Gastric dilation: Am J. Surg. 1971:121

52. Pathological Bases of disease : Robbins – 2ED 1979 by W.B. Saunder's Company 1979:677–78.

53. Petterson B. : Evaluation of a syslen for classification of stone and their site in kidneys treated with ESWL : Scand J. Urol. Nephrol. 1990 : 24 : 293 :– 299.

54. Prof. J. K. Shija: Some observation on paediatric surgical problems in E. Africa - leading Review article. *MEDICOM* Jan.-Feb. 1982 4(1):5.

55. Raffernsperger. J.G. : Swenson's paediatric surgery Appleton century – Croft's NY 1980.

56. Raskin H.F. : Barium Burger Roentgen study for Unrecognized clinical significant Gastric retention : South Med. J. 1971:64.

57. Ree GH: Coma : Med. Digest. July 1983 Vol 9:5

58. Richard D. Hayward : Acute head injury. Surgery Dec. 1985:1:27

59. Robert J. Candle : Severe open fracture of tibia: JBJS 1987: 69 – A:6: page 801

60. Sarungi P.M. Trauma in Tanzania. An address to 12th annual scientific conference of society of anesthesiology of E.A. 1984.

61. Singh P.: The modern management of peptic ulceration. British J. of Hospit. Med.1992 45(1) 44

62. Soave F : Megacolon : long term result's of surgical treatment, Prog. Paediatr. Surg. 1977 10:141

63. Steans MWE : Pre operative roentgen therapy for cancer of the rectum and rectosigmoid : Surg. Gynaecol and obset:1974;138:584.

64. Stones ED : Peptic Ulceration in Northern Tanzania J. Roy. College Surg. Endin. 1981:26(1)362.

65. Tangedahl ER : who get gallstones and why? : Postgrad. Med. 1979:66:175.

66. Wallace A.L. the effect of devascularisation upon early bone healing in dynamic Ext. JBJS: 1991:73–B (5:819).

67. Waller H. Morril: Surgical Approach to congental heart disease – Current prob. in Surg: 1985;XX11(9)

68. Walter BAY, Robert R. : Relapse of antibiotic related colitis : GUT 1983 : 24 : 206

69. Weiland D : Gastric outlet obstruction in peptic ulcer disease : A.M. J. Surg. 1982;143:90–93

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