A dissertation of Ten surgical conditions submitted as part of the requirements for the award of the degree of Master of Medicine (Surgery) of the University of Dar es Salaam - 1986.

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by

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DECLARATION

I declare that this dissertation is on patients in whose management I have participated and that this dissertation has not been submitted for the award of a degree or any other academic award of any other University.

B. Mhina.

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ABSTRACT

Surgery is a very wide field and the emphasis of various surgical conditions varies from place to place depending on the magnitude of the prevalent conditions.

During my three years training at the Faculty of Medicine, University of Dar es Salaam, situated at the National Consultant Hospital - Muhimbili, much emphasis has been on conditions that are more prevalent in our environment.

The post graduate is extensively exposed theoretically and practically to the field of surgery so that he gains confidence and learns the art of surgery of the commonest procedures under the supervision of his senior colleagues and consultants. This aims at producing a competent surgeon who can work independently upcountry and thereby limiting the unnecessary referrals to consultant hospitals.

This dissertation consists of 10 different common surgical conditions fully managed by the author and studied in detail to warrant presentation. It is presented in three sections. Section one consists of five common general surgical conditions, Section two consists of three common urological conditions and Section three consists of two common orthopaedic and trauma conditions.

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PERFORATED PEPTIC ULCER

SUMMARY

Eight patients presenting with perforated peptic ulcers were seen during the duration of two years (July, 1983 to June, 1985) in firm III surgical Dept. Muhimbili Medical Centre.

Six of these were duodenal ulcers and two were gastric ulcers making a duodenal:gastric ratio of 3:1. There were seven males and one female.

The clinical features i.e. symptoms and signs are analysed. Radiology demonstrated pneumoperitoneum in five patients in whom an erect chest film was taken.

Three patients had a history of dyspepsia of three months and more, while 4 patients had history of dyspepsia of between 2 weeks to two months. No history of dyspepsia was obtained in one patient.

All the patients were treated by simple plication with an omental patch. Two patients died postoperatively.

One typical case is presented in detail followed by a discussion on the subject.

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INTRODUCTION

Perforation of a peptic ulcer is one of the complications of peptic ulcer disease and brings about a remarkable series of dramatic changes in the body. It remains a common and serious problem and occurs in 10-15% of chronic peptic ulcers (Webster & Weir 1958, Kay, Moore & Clark 1978, Wilson, Hondry, David and James, 1984).

Acute perforation of a peptic ulcer may be life-threatening abdominal catastrophe that in usual circumstances can be easily diagnosed and treated (Steiger & Cooperman, 1976). 30 to 50% of patients with peptic ulcers have no history of duodenal ulcer disease or ingestion of alcohol or salicylates.

Dark and MacArthur (1983) reviewing 288 patients presenting with perforated peptic ulcer in South West Scotland between 1966 to 1980 found that the incidence of perforated peptic ulcer had fallen from 21.6 per 100,000 in the period of 1946 to 1965 to 14.6 per 100,000.

Stone (1981) working in Northern Tanzania, operated on 42 patients with true established peptic ulceration, among these 3 presented with perforation and one with bleeding. Hiza (1974) reported 40 cases of peptic ulcers among 1871 general surgical admissions to Muhimbili hospital in 1971, 6 of these cases presented with perforations.

In rural Tanzania peptic ulcer disease is a significant cause of morbidity and probably mortality. The African is by nature an introvert and his constant subjection

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to poverty, disease, bad housing and poor food ensures that his life is by no means free of stress (Stone 1981).

Perforations are more common in males than in females and there are more duodenal ulcer perforations than gastric ulcer perforations (Mackay 1966, Booth, Alexander and Williams 1971, Willson, Hendry, David & James 1984).

Perforations of a peptic ulcer is mostly a disease of the elderly and carries a high rate of morbidity and mortality in this age group. (Webster, Weir 1958, Mackay, 1966, Wangensteen et al 1972, Willson, Hendry, David & James, 1984).

CASE PRESENTATION

J. S. a 56 year old male Msukuma by tribe was referred from Kisarawe District Hospital on 3rd January, 1985 with a history of abdominal pain for 2 days, vomiting for one day, absolute constipation for 2 days and a fever of one day.

The patient gave a history of having experienced a sudden onset of abdominal pain the day before at around late afternoon, the pain was epigastric in location to start with but later involved the right upper quadrant and became generalised as time went. The pain has been described as burning in nature.

As the patient used to have such pains in the past one year, he took some two tablets which gave him temporary relief and later the pain recurred and was unresponsive to treatment.

He also gives a history of having vomited once projectile in nature and produced only food previously eaten.
He gave a history of absolute constipation since the day before the symptoms started.

The patient has been a regular attendant to hospital due to Epigastric pains and was being treated with antiacids for the previous one year.

On examination the patient was found to be in pain lying supine with his legs flexed, and sweating. He was not dyspnoeic with a temperature of 38.4°C. Blood pressure of 110/60 mm Hg and Pulse of 92/beats per minute.

Abdominal Examination revealed an abdomen which was slightly distended, not moving with respiration, marked guarding of the abdominal musculature which was generalised especially on the right side (board like). The abdomen was quite tender with rebound tenderness positive. The abdomen was tympanic to percussion at the middle. The liver dullness was obliterated. No bowel sounds could be heard. Respiration and Cardiovascular systems were essentially normal.

Rectal examination revealed only scanty soft stools and no masses were felt.

A provisional diagnosis of peritonitis secondary to a perforated viscus, most probably peptic ulcer was entertained and the patient prepared immediately for an exploratory laparotomy.

An Erect Chest X-Ray and Plain Abdominal X-Ray in the erect and supine position were ordered. The Chest X-Ray showed significant accummulation of gas under the diaphragm.

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The patient was immediately started on intravenous fluids and received 2.5 litres of Normal Saline before operation. A Ryles tube was inserted and active gastric decompression started.

A blood sample for Blood Grouping and Cross matching was taken and two units of blood ordered. Another sample was taken for electrolyte estimation.

Parenteral Antibiotics Ampicillin 500 mg 6 hourly intravenous together with Chloramphenicol 500 mg. intravenously were started preoperatively. A consent for operation was obtained from the patient and the patient shaved ready for operation.

After one hour the patient was sent to the theatre for operation. General anaesthesia was used and the patient positioned supine.

Through a supraumbilical midline incision which was extended to just below the umbilicus the peritoneal cavity was opened. A gush of air was heard when opening the peritoneum. Yellowish green fluid and in some areas appearing like pus, not foul smelling was found free in the peritoneal cavity. The whole of the intestines were covered by fibrinous exudate.

A perforation about 0.5 cm was found on the greater curvature of the stomach with very much scarred indurated margins.

Biopsy from two areas of the edges of the perforation were taken for histopathology. The defect was later closed by simple plication reinforced with an omental patch using silk 2/0 interrupted. The duodenum was inspected and found to be not oedematous.

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Peritoneal toilet using warm saline was done and wound closed in layers, prolene 1/0 to linea alba and peritoneum single layer interrupted followed by silk 2/0 to skin. A corrugated rubber drain was left to drain the morrison's pouch.

Post operatively the patient continued with the antibiotics started preoperatively for 5 days, he was transfused with one unit blood available.

Intravenous fluids 2 litres of Normal Saline and one litre 5% Dextrose were given in the first 24 hours. Active gastroduodenal suction through the naso-gastric tube continued and the amount obtained charted. Pethidine 100 mg. 6 hourly intramuscularly was given as an analgesic during the first 24 hours and the vital signs charted 1 hourly until they became stable.

The ryles tube was removed on the third day when it was found to drain little and by then the patient had opened his bowels.

He started on oral sips and advised to sit up on bed. The intravenous fluids were also stopped. On the fourth day the patient developed fever, vomiting and coughing. A blood slide for malaria parasites was taken and the patient started on antimalarial therapy. On the 6th post operative day the wound was found to discharge pus. Alternate stitches were removed to facilitate drainage and a pus swab taken for culture and sensitivity. From then the post operative period was uneventiful and the patient was discharged on the 18th post operative day.

After two months the patient was seen at the outpatient clinic for evaluation. He was in quite good general

condition but complained of having epigastric pains and abdominal fullness. He was kept on antiacids and asked to report after one month for evaluation.

On the second visit the symptoms of dyspepsia were still present and the patient claimed that they were getting worse. So he was booked for upper Gastrointestinal Endoscopy.

On 29/5/85 the patient was subjected to endoscopy, the aesophagus was found to be normal. An ulcerating polypoid tumour was seen on the body of the stomach. The antrum, fundus, and duodenum were normal. Biopsies were taken from five different sites on the tumour. The examining physician entertained the features to be consistent with carcinoma of the stomach.

The histological results taken at operation showed a benign chronic peptic ulcer of the stomach, and those taken at endoscopy showed atrophic gastitis with chronic ulcer. No malignancy.

On 30/8/85 the patient was readmitted for partial gastrectomy. He was fully prepared within three days, the haemoglobin was 13.2 gm/dl, blood crossmatched and a consent obtained. A ryles tube and an intravenous line of normal saline were given a day before at midnight.

On 2/9/85 the patient was taken to theatre for operation. Under general anaesthesia the abdomen was cleaned and drapped by sterile towels. The abdomen was opened through a supraumbilical midline incision. The stomach was found to be indurated in the pyloric antrum with areas of hyperemia. There were also areas

of fibrous reaction around the area. The regional lymphnodes were not enlarged except one in the subpyloric region which was removed and sent for histology.

Mobilisation of the stomach was started by resecting the greater omentum from the greater curvature as close as possible and ligating with chronic cutgat 2/0. The dissection was carried out using haemostats and ligature past the pylorus and as far as the first part of the duodenum. The lesser omentum was next resected. The right gastric artery was identified just about the upper border of the first part of the duodenum and ligated after division. The division of the lesser omentum was then continued towards the left parallel with the lesser curvature to just before the left gastric artery is reached. The stomach was then lifted upwards and the posterior surface of the first part of the duodenum separated from the pancreas by blunt dissection.

The duodenum was then divided in between two straight clamps placed side by side. The proximal cut end held in a clamp was covered with a gauze and the stomach turned to the left. The duodenal stump was then closed in two layers all mucous membranes invaginated and later covered by a patch of omentum. About two thirds of the stomach was then resected in between two straight bowel clamps.

A loop of the proximal jejunum was then brought to the gastric stump through a hole in the transverse mesocolon and a retrocolic anastomosis with the whole surface of the cut end of the stomach done in two layers.

The defect in the mesocolon was then obliterated using interrupted 2/0 silk. Peritoneal lavage with normal

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saline was done, a drain left in situ and the abdominal wound closed.

The post operative measures were the same as in the first operation and the patient made an uneventful post operative course. The stitches were removed on the tenth day and discharged from hospital to attend as an out-patient.

He was seen in the surgical outpatient twice at an interval of two weeks and then after one month. In all these visits there were no symptoms detected and the patient had started putting on weight. From that time the patient was never seen for follow up again.

ANALYSIS OF THE 8 PATIENTS PRESENTING WITH PERFORATED PETRIC ULCER.

1. Sex distribution : Male:Female - 7:1
2. Duodenal Ulcer : Gastric Ulcer ratio - 3:1

TABLE I : The age distribution of 8 patients presenting with perforated peptic ulcer during July, 1983 - June, 1985.

Age Group (years	No. of patients
10 - 19	1
20 - 29	1
30 - 39	1
40 - 49	2
50 - 59	3
>60	0
TOTAL	8

Most of the patients are between 50-59 years old.

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TABLE II (a): The presenting symptoms of the 8 patients presenting with perforated peptic ulcer during July 1983 - June, 1985.

Symptom	No. of Patients
Sudden onset of epigastric pain	6
Sudden onset of (R) hypochondriac pain	2
Absolute constipation	5
Vomiting	4
Fever	3

Pain is the commonest symptom seen in all patients.

TABLE II(b) : The signs presented by the 8 patients with perforated peptic ulcer during July, 1983 - June, 1985.

Sign	No. of pts.
Right sided tenderness with guarding	2
Generalized abdominal tenderness with guarding	6
Absent bowel sounds	4
Obliteration of liver dullness	4
Positive pneumoperitoneum on Chest Rays	5

NB: Tenderness was present in all patients.

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TABLE III : Previous history of dyspepsia in the 8 patients presenting with perforated peptic ulcer July 1983 - June, 1985.

Previous history	No. of Patients
Previous history of dyspepsia of 3 months or more	3
Previous history of dyspepsia of 2 weeks to 2 months	4
No history of dyspepsia	1
T O T A L	8

NB: Seven patients had a previous history of dyspepsia.
Mortality - 2 patients.

D I S C U S S I O N

Pathophysiology

Immediately prior to perforation the patient may feel entirely well, within a few minutes be in great pains and within an hour be desperately ill.

Perforations occur when an anteriorly or laterally placed ulcer erodes through the full thickness of the wall of the duodenum into the free peritoneal cavity in case of duodenal ulcer and also anteriorly or laterally placed in the stomach.

Gastric and duodenal contents, that is gastric juice, peptic juice, bile and pancreatic juice are thus spilled in the peritoneal cavity. These chemically

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active ferments cause havoc, the resultant chemical injury has been aptly compared to a burn of the peritoneum. Within a short time massive amounts of extracellular fluid may be sequestered in the area of peritoneal injury, and this loss of fluid may bring about hypovolaemic shock. This stage of reaction lasts for 3 to 6 hours and is followed by bacterial peritonitis.

Advanced peritonitis may be complicated by shock toxaemia and death.

Clinical features and Diagnosis

The patient usually gives a typical history of sudden development of severe epigastric and later generalised abdominal pain. Often the exact time of perforation can be recalled by the patient (Sabiston, Steiger and Cooperman 1976). In this study all the 8 patients presented with abdominal pain, 6 of them was epigastric and 2 was in the Right-hypochondriac region (Table IIa).

Movements are painful and the patients usually tend to limit any type of movements. There may be vomiting but is not profuse. Fever may be present when peritonitis due to bacterial contamination has occured. This may also give features of paralytic ileus, that is absolute constipation with a distended abdomen. Some of the patients may present with shock-due to massive sequestretion of fluid into the peritoneal cavity.

A considerable number of patients may give a history of previous dyspepsia or a diagnosed peptic ulcer before the perforation (See Table III). Some of the patients may have no history of dyspepsia. It is estimated that

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about 75% of acute free perforations occur in patients who give no history suggestive of previous duodenal ulceration (Hallenbeck 1976).

Examination reveals a patient who is very ill, lying supine still with legs flexed. His breathing is quick and might be sweating and pale. The temperature may be sub-normal but the pulse rate is usually raised above 90 per minute. The physical findings depends on the time of presentation after perforation. The abdomen moves little with respiration, is rigid (board like) and is tender to touch with rebound tenderness positive. Enough gas may accumulate under the diaphragm to reduce the area of liver dullness. Pelvic tenderness may be elicited by rectal examination.

The definitive diagnosis is made by the presence of pneumoperitoneum. Three roentgenograms may be helpful, a left lateral decubitus film, an erect chest film and a supine view of the abdomen.

Using these views pneumoperitoneum will be detected in 60-80% of patients (Steiger and Cooperman 1976). The left lateral decubitus view may demonstrate as little as 1 ml of free air. The erect chest film must be taken after the patient has been in the upright position for 10-20 minutes to allow the intraperitoneal air to be detected under the diaphragm. The plain abdominal film may show air outlining the bowel wall.

If roentgenographic diagnosis is uncertain, air or gastrografin may be injected through the nasogastric tube to increase the amount of intraperitoneal air or to identify the site of perforation. In our study positive pneumoperitoneum was seen in five patients

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(Table IIb). Laboratory tests do not help confirm the diagnosis and sometimes treatment may be delayed because laboratory findings suggest other possibilities. For example an increase in serum amylase may lead one to suspect acute pancreatitis. The leucocyte count is of little help and in one series, it was normal in 80% of patients (Steiger and Cooperman 1976).

Differential diagnosis

Atypical conditions do occur. In elderly patients, those who are seriously ill with other diseases, or patients with spinal cord injuries, pain may not be a major feature.

In other patients if the gastric contents have flowed into the right lower quadrant through the right paracolic gutter, the patient may have signs and symptoms of peritonitis in the right lower quadrant which may simulate acute appendicitis. Other conditions to be considered in the differential diagnosis include cholecystitis, pancreatitis, perforated colonic diverticular, mesenteric vascular occlusion, rupture of an abdominal aortic aneurysm, spontaneous rupture of the spleen and ruptured tubal pregnancy.

T R E A T M E N T

PERFORATED DUODENAL ULCER

In all patients the initial treatment consists of intravenous fluids, Nasogastric decompression and correction of electrolyte and fluid abnormalities. The third space fluid sequestration should be considered and this should be corrected prior to surgery.

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There are at present three modalities of treating a perforated duodenal ulcer namely:-

(i) Non operative treatment.

(ii) Operative treatment - Simple plication

- Definitive ulcer surgery.

Non operative treatment.

This consists of Nasogastric intubation, intravenous fluids, antibiotics and close observation (Taylor 1957, Steiger and Cooperman 1976, Webster & Weir 1958).

Although formerly a popular method, not more than 10% of perforations can be treated this way today. This method is presently used in two situations, a perforated ulcer that leaked into the peritoneal cavity but has been sealed by omentum or surrounding tissues, and in patients (minority) who are unfit for operation because of advanced age, chronic cardio-respiratory insufficiency or shock due to long delay between perforation and the inception of treatment. Conservative treatment may also be indicated when surgical or anaesthsetic services are not available; on board a ship for example or in remote parts of the world (Taylor 1957, Elliot and Lome 1959, Steiger and Cooperman 1976, Johnston 1978).

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In patients with acute medical illness where the operative risk is great, as in acute myocardial infarction, continued close monitoring of patients may be done and if peritoneal signs progress, laparotomy must be undertaken despite the high risk (Steiger & Cooperman 1976).

Although satisfactory results have been reported using this treatment today, most surgeons have reserved it for the high risk moribund patients, for perforations discovered late or for sealed perforations. The mortality rate for this treatment ranges from 50-100%.

In our study no patient was treated as such (Table IV). However it doesn't mean that we do not have patients treated as such in our community. Some patients might have presented atypically and misdiagnosed while at the same time are treated conservatively without our knowledge.

OPERATIVE TREATMENT

This is generally regarded as the best treatment for most patients with a perforated peptic ulcer and is indicated for about 95% of patients (Sabiston, Steiger & Cooperman 1976, Kay, Moore and Clark 1978, Johnston 1978).

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Most patients in Britain as reported by Johnston 1978, arrive in hospital within a few hours of perforating and need little preoperative treatment apart from the administration of morphine and aspiration of the gastric contents. Others are shocked and require the intravenous infusion of several litres of water, electrolytes and either plasma or dextran. This is the case with most of our patients in our situation. They usually report to hospital late after a trial of local herbs or are delayed in the peripheral hospital because of either misdiagnosis or problems of transport to a well equipped hospital.

The operative dilemma exists when deciding which operation should be done, that is simple suture plication (Graham's technique) or definitive ulcer operation. Simple closure was the treatment performed in all our patients as shown in table iv.

Many reports plication being the procedure of choice because it is accomplished quickly and with minimal operating time. The perforation may be sutured directly, sealed with omentum or both (Steiger & Cooperman 1976, Mackay 1966, Dark, MacArthur, 1983, Johnston 1978).

Simple suture of the perforation should be used in patients who have no previous history of epigastric

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pain, or else only a brief history of pain. It may also be used in very ill, elderly patients, but such patients probably do better with non-operative treatment. (Johnston 1978, Kay 1968). Simple suture should also be used if the surgeon is inexperienced.

Plication used as a primary treatment, the mortality rates reported in most series is less than 10%. Jordan et al noted that the mortality rate in an otherwise healthy patient, younger than 50 years, was less than 1% with all types of surgical therapy. McEvans and Milligan noted 3 factors increased the operative morbidity and mortality after plication of perforated peptic ulcer. These include; patients over 50 years of age, associated medical disease, and a delay of more than 6 hours between the time of perforation and the time of surgery.

Simple surgical closure as a treatment for perforated duodenal ulcer leaves much to be desired because it has no definitive effect on the basic process (Hinshaw, 1968). Follow up studies show that a high percentage of patients receiving simple closure have sufficient subsequent difficulty so that definitive ulcer surgery is required.

In general, after simple plication one third of the

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patients will require additional surgery, one third can be managed on medical therapy and about one third will be asymptomatic. That means, once free perforation of a duodenal ulcer has occured and been closed surgically about 70% of patients have further trouble with ulcer symptoms later (Booth 1971, Steiger & Cooperman 1976, Hallenbeck 1976).

McDonough and Foster (1972) noted that 75% of their patients needing additional surgery after simple closure had the operation within the first two years after perforation.

Regarding the importance of a history of Ulcer disease at the time of perforation Dark and McArthur (1983) found that patients with chronic ulcers (with preceding symptoms for more than 3 months) accounted for 64% and stood a much higher chance of requiring subsequent surgery after simple plication. Very few of those with acute ulcers required subsequent operation.

In contrast, Wangensteen et al (1972) reported no statistical difference in the incidence of patients requiring subsequent surgery. When comparing patients with symptoms less than three months duration with those of more than 3 months duration.

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Therefore although it is difficult to predict with certainty the subgroups that will require further surgery, a definitive operation should be considered for middle aged men with a history of ulcer symptoms who have had perforation less than 12 hours before operation, who have no evidence of major intra-peritoneal sepsis and who can tolerate the additional operative time (Steiger and Cooperman 1976).

Definitive treatment in the form of Highly Selective Vagotomy, Truncal vagotomy and pyloroplasty, or Truncal vagotomy and Gastrojejunostomy should be used if the patient has a long history of ulcer or if he has previously perforated or bled from his ulcer, because if the perforation is merely sutured he is likely to develop recurrent trouble (Johnston 1978).

With proper selection of cases, the operative mortality of Vagotomy and pyloroplasty or Vagotomy and Gastro-jejunostomy for perforation should be less than 1%. (Hinshaw 1968, Johnston 1978).

In summary, there is no one single correct method for the treatment of a patient with perforated duodenal ulcer. The method which is chosen will depend upon the patient (how old? how fit, how fat?, how long perforated) the ulcer (how chronic) and not least, the surgeon (how experienced) (Johnston 1978).

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PERFORATED GASTRIC ULCER

The overall mortality of perforated gastric ulcer ranges from 20% to 40% (Taylor, 1957, Gall & Talbot, 1964, Rees & Thorbjarnason 1973) as compared with 3 to 10% for Duodenal ulcer.

The high mortality is due to a variety of factors such as the advanced age and poor general condition of many of the patients, the large size of the perforation, gross contamination of the peritoneal cavity with food and bacteria, haemorrhage from the ulcer and carcinoma in a few of the ulcers. (Johnston 1978). The results of conservative management are poor (Gall & Talbot, 1964) and operative treatment is advisable.

The surgical management of chronic benign gastric ulcer has met with a remarkable success. (Landor, Manouchear & Clark, 1966). Simple suture of the perforation is indicated if the ulcer is acute, rather than chronic or if the patient is desperately ill. Definitive treatment, usually in the form of partial gastrectomy, is indicated for perforation accompanied by haemorrhage, for large perforated ulcers and for perforated ulcer-cancer (Johnston 1978).

It has been shown that some patients with benign

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gastric ulcers are associated with duodenal ulcers, the proportion varying between 7.9% and 35.6% in different series (Landor, Manouchear and Clark 1966). These patients as opposed to those with gastric ulcer alone, are more likely to have a severe and complicated course and, therefore might account for a higher proportion of surgically rather than medically treated patients. In any event, it appears that at least one fourth of all patients with benign gastric ulcers will be found to have a duodenal ulcer.

Patients with Gastric ulcer and hypersecretion need an operation such as high subtotal gastrectomy or vagotomy and hemigastrectomy, to lower the fasting level of gastric acidity and to reduce the risk of development of stomal ulceration.

The knowledge that 25-50% of patients with gastric ulcer can be expected to have hyperacidity deserves emphasis. A definitive procedure carried out under these circumstances, must be expected to cure the peptic ulcer diathesis whether the patient has hypersecretion or not. It is believed that additional vagotomy to a 50% distal gastrectomy in all such cases will accomplish this purpose with little increase in operating time and with little or no increase in morbidity and mortality (Landor, Manouchear, and Clark 1966, Johnston 1978).

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In our series only two patients presented with perforated gastric ulcer and were treated by simple plication. However one patient who has been presented by the author continued to have ulcer symptoms post-operatively for a period of more than three months. His biopsy results shows a benign gastric ulcer and underwent partial gastrectomy and polya anastomosis.

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SIGMOID VOLVULUS

SUMMARY

Thirteen patients with Volvulus of the sigmoid colon were attended by Firm III, one of the Surgical firms in the department of Surgery, Muhimbili Medical Centre from July, 1983 to June, 1985. All the patients were males, six patients were in the fourth decade and this comprises the majority of cases. Only one patient was above 50 years of age and he was 65 years.

Eight patients presented to hospital within the first four days of their symptoms, one patient within six days and four after seven days. Abdominal pain, distension and constipation was found in all patients. Ten patients also presented with vomiting but this was a late feature. Three patients gave a previous history of a similar attack which resolved spontaneously.

All the patients were operated upon on the same day after an initial resuscitation. Seven patients underwent sigmoidectomy and double barrell colostomy, five patients underwent Hartmann's operation and one patient had primary resection and anastomosis.

All the thirteen patients were found to have a simple

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volvulus at operation of which six were gangreneous while seven were viable. The most significant macroscopic findings were a thickened mesentery in 13 patients and loss of haustration in 4 patients.

Four patients died and all these had gangrene of the sigmoid colon at operation. One typical case managed by the author is presented and a review of the literature relevant to the case outlined.

INTRODUCTION

Volvulus of the colon results from rotation of a segment of the bowel about its mesenteric axis sufficient to produce partial or complete obstruction. Volvulus of the sigmoid colon was first described in 1836 by Von Rokitansky and much of the volumnious literature around the end of the 19th century was concerned with its aetiology.

In United States, sigmoid volvulus is a relatively infrequent but serious cause of intestinal obstruction comprising about 1% of all intestinal obstruction (Abrahams 1963). It is also a rare surgical emergency in other Western countries. For example the incidence of sigmoid volvulus in England was 0.8%, Germany 4.0% and Sweden 7.5% (Shepherd 1969). In Eastern Europe and Russia sigmoid volvulus accounts for 30% to 50%

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of all instances of intestinal obstruction (Forward 1966).

A thorough survey of the literature from Africa reveals that there is much variation in this continent as in Europe. In parts of West Africa it is even less common than in Britain (Shepherd 1969). Only in parts of East and Central Africa has a high incidence comparable to that of Eastern Europe been reported (Shepherd 1968).

In Uganda, sigmoid volvulus as seen at Mulago Hospital is about ten times as frequent as volvulus of the small bowel. It is the second commonest cause of intestinal obstruction and accounted for 10% to 20% of all such cases between 1949 and 1965 (Shepherd 1969). In Kenya, sigmoid volvulus is the second commonest cause of intestinal obstruction after adhesions and bands (Wambwa 1974, Ojara 1982). The incidence of sigmoid volvulus is low in Tanzania (Shija 1975) whereas it is the second cause of intestinal obstruction in some African countries (Mac-Adam 1961, Wambwa 1974).

CASE PRESENTATION

A.M. a 65 year old male Mnyagatwa was referred from Mafia Hospital on the 19.6.84 with the history of abdominal pain for thirteen days, abdominal distension

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of ten days, and absolute constipation of twelve days. The pain was colicky in nature, dull and situated on the left part of the abdomen, and as days went it became generalised. This was later followed by progressive distension of the abdomen to attain quite an enormous size making the patient short of breath. After three days the patient was unable to pass flatus or faeces until admission at our hospital. There was no vomiting since the problem started, though he experienced abdominal fullness whenever he tried to feed. He had no urinary symptoms and there was no significant symptoms on reviewing the rest of the systems. He had been experiencing fever for two days before admission.

On examination, the patient was found to be sick, very much dehydrated, and breathless especially on lying flat. He was not pale or cyanotic. The blood pressure was 110/70 mm Hg lying down and he had a pulse rate of 82 beats per minute of normal volume and character.

Abdominal examination revealed a uniformly distended abdomen more in the flanks, slightly moving with respiration and there were no visible peristaltic waves seen. It was tense on palpation with moderate guarding of the abdominal musculature throughout the abdomen. It was extremely tender with rebound tenderness.

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On percussion it was found to be tympanitic and no bowel sounds could be heard on auscultation. A rectal examination revealed only mild tenderness anteriorly. The rectum was empty and the examining glove was not blood stained. The other systems were normal.

A provisional diagnosis of intestinal obstruction with peritonitis secondary to a gangrenous sigmoid volvulus was made. Urgent plain X-ray of the abdomen in erect and lateral decubitous positions were ordered and immediately an intravenous line using a cannula was set in the forearm and intravenous Ringers lactate solution started. Three litres were given within one and half hours. Before the infusion a blood sample for grouping and cross-matching was taken and three units of blood ordered, another sample for urgent serum electrolytes was ordered. Gentamicin injection 80 mg intravenously was started to be given every eight hours.

The patient was then prepared for an emergency laparotomy. A wide bore nasogastric tube was inserted for gastro-duodenal decompression from which about 250 mls. of greenish yellow fluid was aspirated and charted. A consent for operation was obtained and the patient shaved ready for the operation.

The supine X-ray showed distension of the whole colon,

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a marked loop of bowel was seen arising from the pelvis with two fluid levels. After about one and half hours of initial resuscitation the theatre and blood were ready and the patient was taken to theatre for operation.

The patient was given ketalar 75 mgs. and scoline 100mgs. intravenously as induction, intubated and maintained on oxygen and nitrous oxide. The abdomen was then cleaned with Savlon followed by weak iodine painting and drapped by sterile towels. Through a subumbilical midline incision which was extended upwards the peritoneal cavity was opened. Foul smelling straw coloured fluid and loops of viable small intestines started escaping through the wound. A massively distended gangrenous loop of sigmoid colon extending up to the diaphragm was seen and it was impossible to deliver it through the wound. It was twisted 360 degrees and there was marked scarring at the base of the mesentery which was also very oedematous. The rest of the colon was distended but viable. The small intestines, and the other viscera were all normal.

The gangrenous loop was deflated using a needle connected to a suction machine, delivered through the wound and untwisted. Resection of the gangrenous bowel was then done and because the distal stump was very short, it was closed in two layers with chromic cutgat 2/0 and silk 2/0 and anchored on the left pelvic

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wall with number 1/0 silk. The proximal stump was then brought out as a terminal colostomy through a separate abdominal wound midway between the umbilicus and the left superior anterior iliac spine. The lateral space was then obliterated using chromic cutgat 2/0. The peritoneal cavity was then lavaged using warm saline, a corrugated rubber drain left to drain the pelvis and the wound closed in layers. The patient received two units of blood intraoperatively.

Postoperatively the patient received one unit of blood and later continued with Ringer's lactate alternating with 5% Dextrose at the rate of three litres per 24 hours. The ryles tube was also left in place for suction and gaseous decompression until active bowel sounds resumed. The gentamicin previously started was continued for five days and this was combined with flagyl 400 mg intravenously every six hours for 48 hours then orally for another 72 hours.

The blood pressure and pulse rate were monitored every hour during the first 48 hours and pethidine 100 mg intravenously every 6 hours was given for analgesia. Strict input and output chart was kept by the bedside.

The postoperative course was quite uneventful. The ryles tube and peritoneal drain were removed on the second day post operatively when they were found not

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to drain. On the fourth day the patient had passed flatus and faeces through the colostomy, bowel sounds were heard on auscultation and the drip was discontinued and oral feeding reintroduced starting with black tea and soft diet and advised to be ambulant.

The patient was then taught how to care for his colostomy while in the ward. The stitches were removed on the eighth postoperative day and he was discharged back to Mafia with stomahesive colostomy bags for use at home. He was advised to come back for colostomy closure after three months.

On 14.11.84 the same patient was readmitted in our ward for preparation of closure of colostomy. He was prepared as follows for four days before the operation date. The patient was kept on low residual diet for three days. He was given liquid paraffin 15 mls three times daily for four days. Two days before the operation the patient was given a simple enema to the proximal stump and rectal washings twice daily until the morning before the operation. One day prior to operation the patient was given magnesium sulphate 60 mls every two hours until he started purging, then it was stopped. Flagyl tablets 400 mg were also given three times daily for three days before the operation. On the morning of the operation, apart from

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the usual simple enema to the proximal stump and rectal washings from below, he was started on chloramphenicol 500 mg. intravenously and Gentamicin 80 mg at 7.00 in the morning. A ryles tube was inserted together with an intravenous line ready for the operation. Blood was also already cross-matched and two units of blood available in the blood bank.

On the 19.11.84 the colostomy was closed. Under general anaesthesia and routine cleaning of the abdomen and drapping the abdomen was opened through the old incision removing the previous scar subumbilically in the midline. A lot of adhesions were found and separated. The distal stump was explored and identified in between adhesions and mobilised fully. The proximal stump was detached from the anterior abdominal wall and mobilised. Both stumps were freshened at their tips and approximated. End to end anastomosis in layers with chromic cutgat 1/0 and silk 2/0 was done without tension. A corrugated rubber drain was left at the site of anastomosis and brought out through the freshened old colostomy stoma after approximating the two edges. The abdominal wound was then closed in layers.

Post operatively the patient received normal saline alternating with 5% Dextrose 3 litres for 24 hours. He also continued with Gentamicin and Chloramphenicol intramuscularly as previous indicated for 72 hours,

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then stopped. Pethidine was given for analgesia 100mg every 6 hours in the first 24 hours. The usual monitoring of vital signs was done as in the previous operation.

The postoperative period was uneventiful and the stitches were removed on the nineth day and he was discharged. The patient was followed up in the out-patient clinic twice at an interval of one month and later discharged from clinic back to Mafia in good health and normal bowel habits.

ANALYSIS OF THE THIRTEEN PATIENTS (ADMITTED AND OPERATED FOR SIGMOID VOLVULUS FROM JULY, 1983 TO JUNE, 1985)

TABLE 1 : The age distribution of the 13 patients (admitted and operated for sigmoid volvulus from July, 1983 to June, 1985).

Age (years	No. of Patients
Less than 21	0
21 - 30	4
31 - 40	6
41 - 50	2
Above 50	1
T o t a l	13

The majority of patients are in the third and fourth decade - 4 and 6 patients respectively.

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Table II : The duration of symptoms in days of the 13 patients (admitted and operated for sigmoid volvulus from July, 1983 to June, 1985).

Duration of symptoms (Days)	No. of patients
1	1
2	2
3	3
4	2
5	0
6	1
More than 7	4
T o t a l	13

NB: 4 patients presented to hospital after seven days.

Table III: The clinical features of the 13 patients (admitted and operated for sigmoid volvulus July 1983 - June, 1985).

Symptom	No. of Patients
. Abdominal pain distension & constipation	13
. Vomiting	10
. Dehydration	8
. Past episode of such attack	3

NB: Abdominal pain, distension and constipation is the triad of symptomatology in all our patients.

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Table IV: The mode of treatment of the 13 patients (admitted and operated for sigmoid volvulus from July, 1983 to June, 1985).

Mode of Treatment	No. of Pts.
Resection and anastomosis (primary)	1
Sigmoidectomy and double barrell colostomy.	7
Hartmann's operation	5
Total	13

The majority of patients (7 patients) underwent sigmoidectomy and double barrell colostomy. These had viable colon. Hartmann's was done in patients with gangrenous bowel - (5 patients)

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Table V : The operative findings of the 13 patients (admitted and operated for sigmoid volvulus from July 1983 to June, 1985).

Operative findings	No.of patients
Simple volvulus	13
Compound volvulus	-
Loss of haustrations	4
Thickened mesentery	13
Gangrenous bowel	6
Viable bowel	7

NB: All patients had simple volvulus.
Mortality - 4 patients
All had gangrene at operation.

D I S C U S S I O N

AETIOLOGY

There is a general agreement that the essential anatomical features predisposing to sigmoid volvulus are: long and freely mobile sigmoid colon, a long and freely mobile mesosigmoid and a short mesenteric attachment of the proximal and distal mesocolic limbs forming a narrow inverted "V".

Given these predisposing factors, the colon is thought to rotate intermittently about its narrow attachment

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either clockwise or anticlockwise between 180 degrees and 360 degrees (Smith 1969). In addition, the bowel twists along its own long axis to a variable degree.

Either congenital or acquired factors may lead to volvulus. Among the congenital factors predisposing to sigmoid volvulus is a congenitally redundant sigmoid colon. This is usually a result of failure of peritoneal fixation of the colon, or colon elongation as in congenital megacolon (Hirschsprung disease) or malrotation of intestine (Wuepper 1966, Sutcliffe 1968, Kerry and Ramsom 1969, Schwartz 1979). Among the acquired factors is any condition bringing about distension of the sigmoid colon. This condition is quite commonly encountered in megacolon of various causes.

Many cases have been reported as being secondary to the megacolon of Chaga's disease (Shepherd 1969, Sinha 1969), functional megacolon which is related to poor bowel habits (Wuepper 1966), and acquired megacolon from any other cause.

How far the dietary conditions are responsible for producing a long redundant loop of pelvic colon is a matter for further observation (Sinha 1969). Various workers have attributed the production of volvulus to

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overloaded bowel as the exciting factor. Coarse grain, poor in quality and bulky in quantity has been incriminated (Wuepper 1966, Sinha 1969, Kerry and Ramson, 1969).

Sutcliffee (1968) explains that the geographical distribution of the condition appears to be explained by dietary factors and that where volvulus is/was commonest, people habitually eat large amounts of cereals and sometimes as a single large meal.

A history of severe chronic constipation can be elicited in nearly all patients with sigmoid volvulus in the United States and Western Europe and thus appears to be a predisposing factor. It is therefore common to find volvulus associated with conditions leading to constipation such as Parkinsonism, senility, other central nervous diseases (Drapanas and Steward 1961), feeble mindedness and psychiatric diseases (Sutcliffe 1968).

Mesosigmoiditis has been seen being the main aetiological factor in 182 laparotomies (Sinha 1969) leading to a long, but narrow-based pelvic mesocolon. This resulted in a tough, thickened and fibrosed axis around which the pelvic colon twisted.

Purgation colitis and sudden movement or strain have

been implicated. Many cases have also been in association with pregnancy, labour and the post-partum period.

In summary it would appear that the major factors are distension of the sigmoid colon and a congenitally long sigmoid loop. Excess bowel mobility and straining appear to be associated, possibily precipitating an attack.

PATHOPHYSIOLOGY

Once volvulus has occurred the twist is either in a clockwise or counterclockwise manner resulting in a twist of 180 degrees or more.

Because of this continuing influx of arterial blood, some route for escape is necessary. This is accomplished by engorgement of vessels and frank haemorrhage in the bowel lumen, and wall (Abrahams 1963, Neely 1970, Schwartz 1979). At the same time there is degeneration of the wall as a result of anoxia and proteolytic activity of the bacteria and their toxins.

It has been reported that embarrassment of the circulation leading to gangrene can occur if the volvulus has been present for more than 6 hours (Neely 1970). Shepherd (1969) found that in East

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a simple obstruction of the large bowel to the volvulus. If the ileocaecal valve remains competent, then a double closed loop obstruction occurs. (Abrahams 1963, Kerry and Ramson 1969).

CLINICAL MANIFESTATIONS AND DIAGNOSIS.

Sigmoid volvulus is reported as having a heavy predominance of males with figures of 90% in Peru (Shepherd 1969), 85% in Kenya (Wambwa 1974, McAdams 1961), and is commonly regarded as a disorder of old age. Wuepper and other (1966) in America recorded a mean age of 67 years. Makwani (1984) reviewing cases of sigmoid colon at Bugando Hospital had no female in his series and most patients, 13 out of 20 cases (65%) were between 35 years and 50 years old. In our series the figures match with Makwani's series in that all our patients were males and most patients (8 out of 13 cases) are between 31 years and 50 years of age. The age group differs with most series from the West probably because ours is a younger population. So the clinical presentation is usually a male, middle aged, who presents with a triad of abdominal pain, distension and constipation, occasionally vomiting but this is a late symptom. (Abraham 1963, Kerry & Ramson 1969, Schwartz 1979, Ajao 1980). These symptoms are not always present. Abraham (1963) had abdominal pain in only 50% of his cases, distension in 75% and vomiting in 25%. Ojara (1982) in Kenya

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found in his series that abdominal pain was present in 61.2% of his patients, abdominal distension in 89.6% constipation in 88.1% and vomiting in 53.7%.

The first evidence of constipation is difficulty in passing flatus, then over a few days there is increasing abdominal distension which may become gross enough to make respiration difficult or prevent clothes fitting. The abdominal pain is usually intermittent, felt in the lower or central abdomen, occuring at intervals of upto half and one hour and lasting for up to one minute. (Sutcliffee 1968). Diarrhoea has been reported in 13% of Sutcliffee (1968) series. The duration of the attack varies from a few hours to many days or even months.

The most common physical findings are abdominal distension usually marked, uniform and tympanitic and a palpable mass. There is usually dehydration from hypovolaemia resulting from fluid sequestration in the peritoneal cavity or obstructed loop. Some patients may present with shock (Kerry and Ramson 1969).

Majority of patients have had a similar episode in the past that terminated by passage of large amounts of flatus and stool with relief of the pain and

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distension (Sutcliffe 1968, Schwartz 1969). Any additional feature of peritonitis such as muscle guarding, abdominal tenderness with rebound tenderness, absent bowel sounds and an elevation of temperature and pulse indicates strangulation obstruction with gangrene.

In addition to careful history and examination the most useful aids to diagnosis are plain radiographs of the abdomen taken in the upright and lateral decubitus position (Abrahams 1963, Sutcliffee 1968, Schwartz 1969, Kerry and Ramson 1969, Ojara 1982). A large loop of sigmoid colon arising out of the pelvis with the bent inner tube effect may be seen. One may also see that the walls of the loop are often relatively thick without haustrations (Anderson 1968).

Fluid levels may be seen, one in each limb of the loop, and generally are small and short compared to the overlying and accummulated gas (Abrahams 1963, Ojara 1982). The remainder of the large bowel is dilated but not to such a gross degree.

Barium enema is time consuming and may further complicate the disease by causing necrosis or perforation (Kerry & Ransom 1969). If properly done it will reveal obstruction to barium at the twist

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called "bird's beak" or "ace of spades" deformity.

TREATMENT

The treatment of sigmoid volvulus is controversial. The type of treatment advocated often depends on the experience of the Surgeon and whether gangrene is present or not.

The general principles of treatment are fluid and electrolyte therapy, decompression of the bowel and timed surgical intervention.

Since fluid and electrolyte abnormalities progress slowly in colon obstruction, a brief period of hydration while laboratory studies are being made is all that is necessary before operation. This is not true in our circumstances because most patients present to hospital very late and probably with peritonitis secondary to gangrene of the obstructed loop. In such circumstances fluid and electrolyte therapy should continue until the patient is fit to stand anaesthesia and the operation trauma.

Two specific problems arise in sigmoid volvulus, management of the acute episode and definitive management of the megacolon.

The treatment schedule recommended by many authors is as follows:-

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A: <u>When the bowel is viable</u>

When the bowel is viable, there are two alternatives which can be followed:-

(i) Non-operative detorsion:- This method has been championed by Bruusgaard (1947) who reported 136 successful treatments with four deaths.

This procedure is done with the patient in a knee-chest position. This position alone has led to spontaneous detorsion in some cases. A lubricated sigmoidoscope is gently passed into the rectum and the mucosa inspected for frank mucosal ulceration, or slough or dark blood. If these are seen strangulation of the bowel is probable and the procedure is abandoned, later to be followed by operative intervention.

If these features are not present the scope is advanced until the obstruction site is reached. A well lubricated rectal tube is then slowly introduced and advanced further. Dramatic deflation usually results by passage of a lot of flatus and faeces. The tube is then secured in place by silk stitches for at

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least 48 to 72 hours to avoid recurrence.

Because of the high recurrence rate, sigmoidoscopic decompression should be considered as an initial stage of treatment, which aids in the evacuation and preparation of the colon for elective resection. (Wueper 1966). Sigmoid resection and anastomosis should then be performed preferrably within the same week of admission after proper preparation of the bowel.

After decompression, close attention must be paid to rectal losses of fluids and electrolytes. Careful follow up of the patient is essential and frequent determinations of haemoglobin, serum electrolytes and protein levels are necessary (Forward 1966).

(ii) Operative treatment

(a) Detorsion with or without fixation

In this procedure the abdomen is opened and the bowel is decompressed possibly by passing a well lubricated rectal tube through the rectum by an assistant. Puncturing of the bowel should be avoided lest peritoneal soilage occurs. The volvulus is

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then derotated and fixed on the lateral pelvic wall or left alone.

This procedure has been used by some authors after failure of nonoperative detorsion. Ojara 1982 working at Kenyatta National Hospital found a very high mortality (50% of all deaths), and attributed it to be due to the practice of deflation of the distended bowel with needles at various sites, which often leads to contamination and peritonitis. This procedure is also associated with a high recurrence rate and should be followed if possible by sigmoid colectomy and anastomosis after bowel preparation when the patient is fit to stand operation.

No case in our series underwent this type of operation.

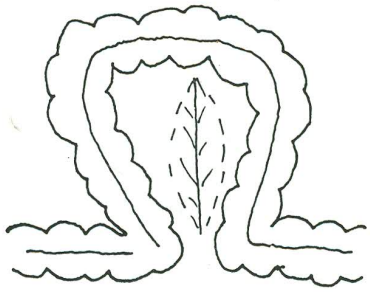
(b) Mesocoloplasty:

The technique of mesocoloplasty for the prevention of recurrence of sigmoid volvulus is simple, and does not open the gut (Tiwary, 1976). It can be

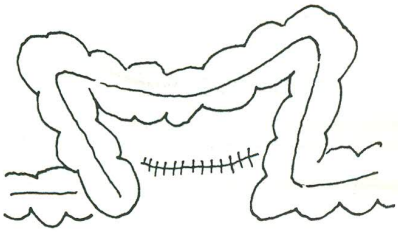
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performed confidently by junior doctors and appears to be an eminently safe technique as a routine procedure in any emergency service.

An elliptical incision is made along the longitudinal axis of the mesocolon running from the base of the redundant sigmoid loop to the tip through the fibrous band. The mesocolic fibrous band is removed and the incision is stitched in the transverse direction by interrupted sutures. At the end of the operation the redundant sigmoid colon with a long mesocolon and narrow base initially appears to have a broadened base and short mesocolon.



Initially



Final appearance

(c) Resection and primary anastomosis

The only satisfactory way to prevent recurrence is to resect the affected

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loop at the initial presentation if practicable and safe (Sutcliffe 1968).

In fact, resection and primary anastomosis are not difficult, the dilatation helping rather than hindering, once the contents of the loop have been allowed to escape through a rectal tube partly inserted preoperatively, and later manipulated into the sigmoid colon.

Grounds for supporting this course of action include no loss of potassium (Forward 1966) and fluid into the bowel wall and lumen, and the prevention of early recurrent attacks. It is also suggested that removal of the large and easily distensible sigmoid loop help to prevent post operative abdominal distension as compared with operative reduction, and so aids respiratory function (Sutcliffee1968). One patient in our series underwent this procedure.

B: <u>When the bowel is Gangreneous</u>.

When the bowel is gangrenous there is no safe procedure apart from immediate operative intervention and resection of the non viable bowel.

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(i) Resection and primary anastomosis.

Occasionally in certain selected cases it is possible to resect the gangrenous bowel and do a primary end to end anastomosis. The condition of the patient and the amount of distension and oedema of the bowel all have to be taken into consideration.

Most of the patients with a gangrenous loop are poor surgical risks and complications at the anastomosis are likely to occur when the colon is unprepared and dilated (Wuepper and Others 1966). In view of an existing mortality of more than 70% in the presence of gangrene, the point of resection and primary anastomosis seems difficult to justify (Sutcliffee 1968). None of our patients with gangrene was treated by this method. The anastomotic site should be stented by a rectal tube or a proximal defunctioning colostomy established.

(ii) RESECTION AND DOUBLE BARREL COLOSTOMY

This operation is recommended but subsequent gangrene of the distal stump has been reported. (Sutcliffee 1968). After resecting the gangrenous bowel the proximal and distal stumps are brought together, the antimesenteric borders sutured together for about

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ten centimeters, and brought out through a separate incision.

If the distal limit of the twisted loop is too deep in the pelvis to permit easy exteriorization, it must be fully mobilised if tension with resultant embarrassment of the circulation is to be avoided.

The advantage of this operation is that the colostomy can be easily closed extraperitoneally or by using an enterotome without opening the peritoneal cavity.

This has been the commonest method employed in our series and it was performed in 7 patients.

(iii) HARTMANN OPERATION

In this operation the distal stump is closed and the proximal stump brought out as a terminal colostomy. It is the operation of choice when gangrene has extended down to the upper segment of the rectum, but later reanastomosis is difficult because the distal stump is usually buried in a lot of fibrous tissues necessitating extensive dissection and mobilisation. Furthermore it needs a second laparotomy

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(Neely 1970).

Five patients were treated by this method in our series.

THE TREATMENT PROTOCOL FOLLOWED IN OUR FIRM DURING THE PERIOD OF STUDY.

As previously outlined, that treatment by sigmoidoscopy and intubation is a better initial treatment and allows later resection and anastomosis of a prepared colon. This procedure is rarely used by us because it has been observed that after deflation and relief of the acute condition, patients do not come back for a planned colectomy, but only return when obstruction has reoccured. We are unable to perform sigmoid colectomy and anastomosis within the same week after deflation because our wards are usually congested, full of patients waiting for other operations. The author has operated on one case who had laparotomy and detorsion who came back a year later with a recurrent extensive strangulated sigmoid volvulus. A hartmann's operation was performed but the patient died of septicaemia two days later.

All patients presenting with viable bowel, sigmoid colectomy is done and the two stumps double barrelled and brought out as a colostomy. If the distal stump was too short as in those cases with gangrene

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extending down to the upper part of the rectum a hartmann's operation is preferred or the distal stump is brought out through the lower end of the main abdominal incision as a mucus fistula. All patients with a colostomy usually return for closure at a later date and so did our patient.

This is the same situation as reported by Ojara (1982) at Kenyatta National Hospital in Kenya when reporting his 67 cases diagnosed and treated during the period of 1976 to 1980. He recommended initial non-operative management in an attempt to reduce morbidity and mortality which is a rational view.

MORTALITY

The overall mortality in our series was 4 patients, and occurred in patients who had gangrenous bowel. This is comparable with figures from Western Europe and the United States who report an overall mortality rate of 20% to 50% per attack (Sutcliffee 1968). The figure at Mulago, Kampala is a bit high and the mortality rate is 50% (Shepherd 1969). This is the same as that reported from India (Shepherd 1969).

The overall mortality at Kenyatta National Hospital is 26.9% (Ojara 1982) mainly due to the presence of gangrene, a situation in which a mortality of 75% following resection has been reported (Kerry and

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Ramson 1969). Makwani (1984) reported an overall mortality rate of 20% at Bugando Hospital in Tanzania which is lower compared to our series.

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ACUTE APPENDICITIS

SUMMARY

A total of 36 patients were seen and operated for acute appendicitis or its complications during the period starting from July, 1983 to June, 1985 by firm III one of the Surgical firms in the department of Surgery at the Muhimbili Medical Centre, Dar es Salaam.

There were 26 males and 10 females (male:female ratio 2.6:1). 26 patients (71.9%) were in the age group of 20 years to 39 years. The range was 16 to 48 years. No patient was above 50 years of age.

Abdominal pain was the commonest presenting symptom and was present in all our patients. It was localised in the right lower quadrant in 28 patients (77.8%). Nausea was present in 10 patients (27.8%) and vomiting was present in 25 patients (69.4%). The most important sign elicited was localised muscle guarding and tenderness in the right lower quadrant which was present in 20 patients (55.6%) and 24 patients (66.7%) respectively.

21 patients (58.3%) were found at operation to have a simple non perforated appendix and all presented to hospital within two days of their symptoms, while 15 patients (41.7%) presented with complications namely

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perforation in 11 patients (30.6%) and appendiceal abscess in 4 patients (11.1%) respectively. All these patients presented to hospital after 24 hours of their symptoms.

The mortality rate was 13.9% (5 patients) all of whom died of complications of acute appendicitis except one.

All the patients were operated upon and in 30 patients (83.3%) appendicectomy was done while in 6 patients (16.7%) only drainage of the appendix abscess was done followed by interval appendicectomy. The most common complication encountered in our series was wound sepsis.

INTRODUCTION

Acute appendicitis is still the most frequent reason for an emergency surgical operation throughout the world (Schwartz 1979, Sabiston 1981, Peltokalio et al 1981, Cooperman 1983). The recognition of appendicitis as a clinical and pathologic entity for which surgical therapy is essential dates back from 1886 when Reginald Fitz, Professor of pathologic anatomy at Havard, gave his paper giving the direct relationship between inflammation of the vermiform appendix and the late stages of peritonitis or iliac fossa abscess. His word appendicitis took the place of the old indefinite terms typhlitis, perityphlitis or paratyphlitis.

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Soon after, McBurney described the clinical manifestations of early acute appendicitis prior to rupture, including the point of maximal abdominal tenderness, and an incision made in the abdominal wall in cases of appendicitis.

The disease occurs at all ages but it is most frequent in the second and third decades of life. It is quite rare in the very young, probably because of the configuration of the appendix at this age makes obstruction of the lumen unlikely. Acute appendicitis in the young and elderly group is attended by a high rate of mortality and morbidity and this is because of its more rapid progression to perforation in this age group especially in the elderly group together with the concomitant carciovascular and respiratory diseases and the inability of the young to articulate symptoms and poor development of the omentum (Fitz 1886, Hobston and Rosenman 1964, Marchildon & Dudgeon 1977, Lewis et al 1975, Peltokallio & Tykka 1981, Cooperman 1983). The disease has a male preponderance.

One case managed by the author is presented in details and a discussion relevant to the subject outlined.

CASE PRESENTATION

T.M. a thirty year old female Mzaramo from Tandale in the outskirts of Dar es Salaam, was referred from Magomeni Hospital, one of the peripheral hospitals in

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Dar es Salaam on 3.4.85 with a one day history of abdominal pain. The pain was in the right iliac fossa, burning in nature but occasionally colicky, which was continuous with no alleviating factors but was aggravated by movements. The pain was strictly localised, and did not radiate to any of the abdominal quadrants.

The patient gave a history of nausea prior to the pain, but had not vomited since the problem started. She was feeling a bit of fever, but no history of any urinary symptoms or change in bowel habits. She was on lactational amenorrhoea of six months and there was no similar episode in the past.

On examination, the patient was found to be sick, obese and not pale clinically. She was febrile with a temperature of 38°C but the hydration status was fair. The blood pressure was 110/60 mm of mercury, with a rapid pulse of full volume of 120 per minute and a respiration rate of 18 breaths per minute.

Abdominal examination revealed an obese abdomen, moving with respiration. There was local guarding of the abdominal musculature on the right iliac fossa with marked tenderness and rebound tenderness which was more marked at the McBurney's point. No mass was palpable and the bowel sounds were normal. Rovsing

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sign and psoas signs were all positive. Vaginal examination was normal with the exception of tenderness in the right adnexal region. Rectal examination revealed tenderness anteriorly and to the right side and no masses were felt. The rest of the systems were normal.

A tentative diagnosis of an acute appendicitis was made and the patient prepared for an emergency operation. Pre-operatively the patient was given intravenous fluids of normal saline 2 litres, ryles tube was inserted and a blood sample taken for grouping and crossmatching. The patient was started on a combination of Crystalline penicillin 500 mg intramuscularly every six hours and chloramphenicol 500 mg intramuscularly every six hours. The patient was shaved, and a consent obtained ready for operation. After one hour the theatre was ready and the patient was taken for operation.

The patient was given general anaesthesia and after thorough cleaning of the abdomen with antiseptics and drapping the peritoneal cavity was opened through a grid-iron muscle splitting incision. Fibrinous exudate was seen oozing out of the wound and the greater omentum was seen covering a pelvic inflammed appendix about 8 cm long amidst matted coils of small intestines. The appendix was found to be perforated in the middle.

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The surrounding edges of the wound were covered by sterile gauze and the appendix together with a portion of the caecum delivered through the wound while held by a babcock forceps at the tip of the mesoappendix. The appendicular vessels were held between haemostats within the mesoappendix, divided and tied using silk 3/0.

A crushing artery forcep was applied at the base of the inflammed appendix and then moved distally. The crushed area was later tied by a chromic catgut 2/0 and the appendix amputated distally near the artery forceps over a free sterile gauze. The appendix, knife, artery forceps were then removed from the operating field.

The raw surface of the amputated appendix was cauterised using weak iodine, no inversion was done because the wall of the caecum was so oedematous. After making sure that haemostasis was secured, the area was mopped dry and the caecum was returned into the peritoneal cavity. A corrugated rubber drain was left to drain the site. The wound was closed in layers using chromic catgut 2/0 to peritoneum, the muscles loosely apposed and the skin loosely stitched using silk 2/0.

Post operatively the patient was given intravenous fluids of normal saline to alternate with 5% Dextrose

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3 litres in 24 hours. Pethidine 100 mg intramuscular 6 hourly was also given as an analgesics for 24 hours.

Ampicillin injection intravenously 6 hourly started pre-operatively was continued for 72 hours post-operatively and later changed to oral for two more days. Chloramphenicol injection 500 mg intravenously 6 hourly was also added for 72 hours and later changed to oral for 2 more days.

The vital signs, blood pressure, pulse were monitored hourly within the first 24 hours and later 4 hourly for the next 24 hours. The temperature was recorded twice daily. Strict input/output chart was kept at the bedside.

The post operative course was uneventful. The Ryles tube was removed after 48 hours when it was found not to drain much, and the bowel sounds present. The intravenous fluids were also stopped the same day and the patient allowed to be ambulant and start on oral sips. The patient was on normal diet on the third day.

The temperature was back to normal after 24 hours. The patient was discharged on the fifth day and returned on the eighth day when the stitches were all removed after the wound was found to have healed nicely.

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The patient was seen at the surgical outpatient clinic after one month. She had no complaints and was discharged from the clinic.

ANALYSIS OF THE 36 PATIENTS OPERATED FOR ACUTE APPENDICITIS FROM JULY, 1983 - JUNE, 1985.

RESULTS:

TABLE 1 : The age distribution of the 36 patients operated for acute appendicitis from July, 1983 - June, 1985.

Age (years)	Number of patients	Percentage
10 - 19	5	13.9
20 - 29	14	38.9
30 - 39	12	33.3
40 - 49	5	13.9
Over 50	0	0
TOTAL	36	100.0%

NB: Most patients are in the age group of 20 - 29.

TABLE II: The sex distribution of the 36 patients operated for acute appendicitis from July, 1983 to June, 1985.

Sex	No.of Patients	Percentage
Males	26	72.2
Females	10	27.8
TOTAL	36	100.0

Note: Male:female (13:5)

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TABLE III: The presenting symptoms and signs of the 36 patients operated for acute appendicitis from July 1983 - June, 1985.

Symptom/sign	No. of Patients	Percentage
(1) Abdominal pain		
. Localised RIF	28	77.8
. Periumbilical and/or right hypochondriac	2	5.6
. Generalised	6	16.7
(2) Vomiting	25	69.4
(3) Nausea	10	27.8
(4) Fever	20	55.6
(5) Frequency of micturation	2	5.6
(6) Constipation	7	19.4
(7) Diarrhoea	1	2.8
(8) Muscle guarding		
. Localised RIF	20	55.6
. Generalised	6	16.7
(9) Abdominal tenderness		
. Localised RIF	24	66.7
. Generalised	6	16.7
(10) Abdominal distension	9	25.0
(11) Mass in RIF	4	11.1
(12) Silent abdomen	5	13.9

NB: Pain is the chief symptom in all the patients and tenderness the only sign in 83.4% of patients.

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TABLE IV : Duration of symptoms before admission in relation to pathology at operation of the 36 patients operated for acute appendicitis from July, 1983 - June, 1985.

Days	Simple appendi-	Perforated appendicitis	Appendi- ceal abscess	Total	%
1	11	-	-	11	10.6
2	10	2	-	12	33.3
3	-	5	-	5	13.9
4	-	4	1	5	13.9
5	-	-	1	1	2.8
6	-	-	-	-	-
Over 7	-	-	2	2	5.5
TOTAL	21	11	4	36	100.0

NB: Patients with complications present more than 24 hours of their symptoms.

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TABLE V : The hospital stay of the 31 patients operated for acute appendicitis from July 1983 - June, 1985.

Days	Simple appendicitis	Perforated appendicitis	Appendiceal Abscess	Total
1-7	19	5	-	24
8-14	1	4	-	5
15-21	-	1	-	1
Over 21	-	-	1	1
Total	20	10	1	31

NB: Most patients were discharged within one week.

TABLE VI : The post operative complications noted in the 36 patients operated for acute appendicitis from July, 1983 to June, 1985.

Complication	No. of Patients	Percentage
Wound sepsis	5	13.9
Septicaemia/peritonitis	3	8.3
Endotoxic shock	2	5.6
Wound dehiscence	1	2.8
Hypstatic pneumonia	1	2.8

NB: Wound sepsis ranks in the post operative complications.

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TABLE VII : Analysis of the 5 deaths from acute appendicitis and its sequelae from July, 1983 - June, 1985.

Age (Years)	Operative findings	Sex	Duration of symptoms	Cause of death	Operation offered	Post operative death in days
28	Gangrenous & perforated appendix	M	4	Septicaemia	Appendicectomy	1
45	Appendix abscess	F	7	Septicaemia Hypostatic pneumonia.	Drainage alone	24
18	Acute appendicitis (simple)	M	1	Acute renal shut down	Appendicectomy.	1
30	Appendix abscess	M	4	Endotoxic shock	Drainage	4
227	Appendix abscess	M	14	Septicaemia Multiple organ failure	Drainage	6

NB: Septicaemia was the commonest cause of death in almost all patients except one.

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D I S C U S S I O N

AETIOLOGY AND PATHOGENESIS.

Obstruction of the lumen of the appendix is the dominant factor in the production of acute appendicitis. Faecaliths are the usual cause of appendiceal obstruction (Schwartz 1979, Fitz, 1886). Less common are hypertrophy of lymphoid tissue, variety of seeds, hair, worms and their eggs, pins, gall stones and inspissated barium from previous X-Ray studies.

In about two thirds of all cases of perforating inflammation of the appendix either dried faeces or foreign bodies were present in the tube (Schwartz 1979). External trauma is occasionally recorded as an immediate precursor of the attack.

Digestive disturbances are of obvious importance in the aetiology of inflammation of the appendix, since this organ is part of the alimentary tract. Attacks of diarrhoea and vomiting are usually a result of indiscretion in diet, but they were sometimes occasioned by the use of domestic remedies which were administered for the relief of constipation or other disturbances attributed to a sluggish action of the stomach or bowels.

Not withstanding the frequency of typhoid fever, and of intestinal tuberculosis, in which affections of the

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mucous membrane of the appendix is often diseased, a resulting perforation seems to have been relatively infrequent (Fitz 1886).

It is apparent that perforating appendicitis is a disease most occuring among healthy youths and young adults especially males. Further, that attacks of indigestion and acts of violence, particularly from lifting, jumping and falling, are exciting causes in one fifth of cases (Fitz 1886). In our series it is seen that above 72.2% of our patients are young adults falling in the age group of 20-39 years (table 1).

The retention of faeces may be promoted by a constipated habit, but congenital or acquired irregularities in the position and attachments of the appendix frequently act as favouring causes.

The frequency of obstruction also rises with the severity of the inflammatory process. Faecaliths are found in approximately 40% in simple acute appendicitis, in about 62% of gangrenous appendicitis without rupture, and in 90% of gangrenous appendicitis with rupture (Schwartz 1979).

The sequence of events following occlusion of the lumen is probably as follows:- A closed loop obstruction is produced by the proximal block, and continuing normal secretion of the appendiceal mucosa very rapidly produces

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distension. Distension stimulates nerve endings of Visceral afferent painfibres, producing vague, dull, diffuse pain in the mid abdomen or lower epigastrium. Peristalsis is also stimulated by the rather sudden distension, so that some cramping may be superimposed on the visceral pain early in the course of appendicitis.

Distension continues not only from continued mucosal secretion but also from rapid multiplication of the resident bacteria of the appendix. As pressure in the organ increases, venous pressure is exceeded. Capillaries and venules are accluded, but arteriolar inflow continue, resulting in engorgement and vascular congestion. Distension of this magnitude usually causes reflex nausea and vomiting, and a diffuse visceral pain becomes more severe.

The inflammatory process soon involves the serosa of the appendix and in turn parietal peritoneum in the region producing the characteristic shift in pain in the right lower quadrant of the abdomen. Fever tachycardia, and leukocytosis develop as a consequency of absorption of dead tissue products and bacterial toxins.

As progressive distension encroaches on the arterial pressure the area with the poorest blood supply suffers most. Ellipsoidal infarcts in the antime-

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senteric border develops which are the areas later to perforate.

The sequence is not inevitable, some episodes of acute appendicitis subside spontaneously. In those cases where the appendicular peritonitis represents the extension of an inflammation through the wall of the appendix without perforation, permanent adhesions of the appendix to the neighbouring parts remain as evidence of the process. When it is associated with necrosis of the wall, the inflammation of the peritoneal coat tends to become diffused and productive of serous and cellular exudations. The adherence of coils of intestines to each other and to the abdominal wall favours the accumulation of the exudation in a limited space, and thus the formation of an appendicular tumour.

This is the same phlegmon which occurs after perforation. It is the nature's walling-off process and the greater omentum (the abdominal policeman) is also involved and usually this process is able to quarantine the inflammation in about 95% of patients and confine the spill to a periappendiceal area (Fitz 1886, Schwartz 1979).

This process may slowly resolve spontaneously, or may be hastened by timely surgical intervention. In some patients, however a progressive suppurative process

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produces an expanding collection of pus contained by the walling-off process, a periappendiceal abscess.

If the walling-off process is not completed by the time appendiceal rupture occurs, contamination spreads beyond the right lower quadrant either to the pelvic cul de sac via gravity drainage or the right sub-hepatic space via the right paracolic gutter. Depending on the virulence of the organism, bacteria thus seeded may initiate spreading diffuse peritonitis with its attendant complications.

Ascending septic thrombophlebitis of the portal venous system, pylethrombophlebitis, is a very grave but fortunately rare complication of gangrenous appendicitis.

CLINICAL PRESENTATION

Abdominal pain is the prime symptom of acute appendicitis in all age groups (Fitz 1886, Owens & Hamit 1978, Janik & Firar 1979, Gomez & Wood 1979, Schwartz 1979). Classically the pain initially is diffusely centered in the lower epigastrium or umbilical area, is moderately severe and is steady. Sometimes with intermittent cramping superimposed.

After a period varying from one to twelve hours, usually within 4-6 hours the pain localises in the right lower quadrant. This classic pain sequence,

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though usual, is not invariable. In some patients the pain of appendicitis begins in the right lower quadrant and remains there. Variations in the anatomic location of the appendix account for many of the variations in the principal locus of the somatic phase of the pain.

Anorexia nearly always accompanies appendicitis. It is so constant that the diagnosis should be questioned if the patient is not anorexic. Vomiting occurs in about 75% of patients but is not prominent or prolonged, most patients vomiting only once or twice.

In our series all our patients presented with abdominal pain which was localised in the right lower quadrant in 77.8%. Nausea and vomiting are also conspicuous symptoms observed (Table III).

Most patients give a history of constipation from before the onset of abdominal pain: Diarrhoea occurs in some patients particularly children. In our series children are not included as they are usually admitted in the Paediatric surgery unit.

The sequence of symptom appearance has great differential diagnostic significance. In over 95% of patients with acute appendicitis, anorexia is the first symptom, followed by abdominal pain, which is

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followed in turn by vomiting (if occurs). If vomiting precedes the onset of pain, the diagnosis should be questioned (Schwartz, 1979).

Fever is another constant symptom. It is usually low grade and the constitutional disturbances usually associated with an elevated temperature are frequently slight, if not absent. If voilent or extreme changes take place, a complication may be expected, as an abscess of the liver, perforation or a diffusing peritonitis from an extension of the local inflammatory process.

During the first 3 days following the onset of pain, micturation is occasionally disturbed, perhaps unusually frequent on the first day, it is likely to be difficult on or after the third day. The right testicle may be retracted and swollen, in which case the course of the pain is apt to be towards this gland.

A circumscribed swelling in the right iliac fossa may occur. This symptom, when present, is evidently of the utmost value in diagnosis as its appropriate treatment most favourably modifies the prognosis. The swelling represents the accumulation of pus, the increased exudation at the outset and of the product of peritonitis and lies beneath the adherent coils of intestine which later become attached to the abdominal walls. Four patients in our series had a

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mass in the right iliac fossa (Table III).

The signs are determined by the anatomic position of the inflammed appendix as well as whether the organ has already ruptured when the patient is first seen.

Vital signs are not changed very much by uncomplicated appendicitis. Temperature elevation is rarely more than 1°C; the pulse rate is normal or slightly higher. Changes of greater magnitude usually means that a complication has occured or that another diagnosis should be considered.

Patients with appendicitis usually prefer to lie supine with thighs, particularly the right, drawn up, because any motion increases pain.

The classic right lower quadrant physical signs are present when the inflammed appendix lies in the anterior position. Tenderness is often maximal at or near the McBurney's point. Direct rebound tenderness, usually referred or indirect rebound tenderness frequently, are present and are also felt maximally in the right lower quadrant indicating peritoneal irritation. Lewis, Holcroft and Boey et al (1975) when critically reviewing 1000 cases with appendicitis found that direct tenderness parallels the incidence of pain and was present in 99% of their cases. They also found that in patients with non-perforated appendicitis or appendicitis with local peritonitis, tenderness was

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localised in the right lower quadrant in 91%. In patients with generalised peritonitis tenderness was localised in 58% and was diffuse in 41%.

In our series, overall tenderness was localised in the right lower quadrant in 66.7% and generalised in 16.7% (Table III).

Rovsing's sign, pain in the right lower quadrant when palpatory pressure is exerted in the left lower quadrant also indicates the site of peritoneal irritation. Cutaneous hyperesthesia in the area supplied by the spinal nerves on the right at T10, T11 and T12 is a frequent but not a constant accompaniment of acute appendicitis.

Muscular resistance to palpation of the abdominal wall parallels the severity of the inflammatory process. Early in the disease it consists mainly of voluntary guarding, but as the parietal peritoneum irritation progresses, muscle spasm increases and becomes largely involuntary.

Variations in the position of the appendix usually produces variations from the usual physical findings. For example, with an inflammed retrocecal appendicitis the anterior abdominal findings are less striking, and tenderness may be more marked in the flank. With a pelvic appendix, abdominal findings may be absent and the diagnosis missed unless the rectum is examined.

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The psoas and obturator signs may be positive if the irritative focus is in the proximity of that muscle.

When the patient presents with rupture at the time of examination, the patient is usually quite ill, prostrated, toxic, and distended. Right lower quadrant pain increases in severity and spreads over a somewhat larger area. The physical findings are more definite after rupture with a periappendiceal phlegmon or abscess, a tender boggy mass with ill defined margins can usually be felt. Tenderness which was finger point with simple appendicitis, now encompasses the whole right lower quadrant. Rebound tenderness with muscular rigidity are usually marked and corresponds in extent to the local peritonitis.

The clinical characteristics of the tumour and its composition are made evident by modified resonance on percussion, circumscribed resistance on palpation, and a sense of fluctuation. Fluctuation, as a rule appears late in the course of the disease. (After the second week), so it is of little diagnosic value. An exploratory puncture with the needle of the aspirator is frequently recommended to determine the nature of the tumour.

The presence of the abscess may be expected as early as the third day. It may be large enough to contain some three pints of fluid on the fifth day.

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The chief danger of appendicular peritonitis is that it becomes general. General abdominal pain is regarded as suggestive evidence of the beginning of a general peritonitis. Tympanitic distension and generalised abdominal tenderness and guarding are signs of general peritonitis.

Majority of cases the resulting general peritonitis begin on the second, third and fourth day after the inflammation of the appendix is established (Fitz 1886).

DIFFERENTIAL DIAGNOSIS

Essentially this is the diagnosis of the acute abdomen. This is because clinical manifestations are not specific for a given disease but are specific for disturbance of physiologic function or functions.

Thus an essentially identical clinical entity can result from a wide variety of acute processes within or near the peritoneal cavity that produce the same alterations of functions as acute appendicitis. Accuracy of preoperative diagnosis should be about 80%-85%. If it is consistently below 75%, some unnecessary operations are probably being done, and a more rigorous preoperative differential diagnosis is in order (Schwartz 1979). On the other hand, an accuracy consistently more than 90% should cause concern since this usually means that

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some patients with atypical but bona fide acute appendicitis are being observed, when they should have prompt surgical intervention.

There are however a few conditions in which operations is contraindicated but in general the disease process that are confused with appendicitis are also surgical problems or, if not are not made worse by operation.

The differential diagnosis of acute appendicitis depends upon the anatomic location of the inflammed appendix, the stage of the process, the age and sex of the patient.

Diseases to be considered in children include primary acute lymphadenitis, acute gastroenteritis, acute pyelonephritis, Meckel's diverticulitis, intussusception, regional enteritis, Yersiniosis and Henoch-Schonlein purpura. Gynaecological disorders in the female such as pelvic inflammatory diseases, ruptured graafian follicle, twisted ovarian cyst or tumour, and ruptured ectopic gestation should never be forgotten in the differential diagnosis. Acute epididymitis and torsion of the testis are conditions which should be considered in the male.

Other diseases which should be considered regardless of the sex are perforated peptic ulcer, diverticulitis coli, perforating carcinoma of colon (especially caecal),

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foreign body perforation of the bowel, closed loop intestinal obstruction, mesenteric vascular occlusion, pleuritis of right lower chest, acute cholecystitis, acute pancreatitis, right ureteric calculus, infarcted epiploic appendage and haematoma of the abdominal wall.

ACUTE APPENDICITIS IN THE YOUNG AND ELDERLY AND DURING PREGNANCY.

Appendicitis, in its classic form is easily diagnosed, but in the very young and elderly it is commonly atypical and presents a diagnostic problem with a reported perforation rate as high as 59 percent. (Lewis, Holcroft & Boey et al 1975).

Appendicitis in the elderly is generally considered to be difficult to diagnose even though the main symptoms are similar to those of younger people. The most significant difference, as noted by Peltokallio & Tykka (1981), is the faster progression of the disease to the perforation stage. This may be because the arteries in the older person's appendix are sclerotic, the mucous membranes atrophied, and the lymphatic tissue is scanty; also, the lumen may be partially obstructed or the walls weakened by previous inflammations. All these factors enhance the spread of inflammation and progress to perforation.

The elderly group of patients have also a significant

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incidence of concomitant major illnesses complicating appendicitis. Slowness in seeking medical attention by the elderly and the surgeon's attempt of a more definite diagnosis because of the greater operative risk further delays treatment and increase the risk of perforation (Owens and Hamit 1979, Pettokallio & Tykka 1981, Cooperman 1983). In our study none of our patients were over 50 years of age and it has been difficult to compare the presentation of the elderly patients. However, it has been noted (Table IV) that most patients presenting with perforation and its sequelae attended to hospital with symptoms more than 24 hours. This is a real delay in seeking medical advice.

Infants also have a high rate of perforation owing to their inability to articulate symptoms and the difficulty encountered in performing physical examination (Cooperman 1983). Savrin (1979) reported a 42% incidence of perforation in infants.

Diagnosis of acute appendicitis is often difficult in the pregnant patient as the symptoms may be attributed to the pregnancy (Gomez and Wood, 1979). Physical findings may be obscured or altered by the cephalad displacement of the appendix as pregnancy progresses.

Basically it is the same disease process as in the

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non-pregnant woman. It is most common in the 20-25 year age group and most frequently seen in women having two or more pregnancies (Gomez & Wood 1979).

Gomez & Wood (1979) also found that it was less common in the last trimester and approximately half of their patients were in the second trimester.

DIAGNOSTIC AIDS

Moderate leukocytosis, ranging from approximately 10,000 to 18,000 per cubic millilitre and associated with polymorphonuclear predominance, is the rule in acute uncomplicated appendicitis (Lewis Holcroft & Boey et al 1975, Schwartz 1979).

If the White Blood cell count is greater than 18,000 per cu.mm. or if the shift to the left is extreme, perforated appendicitis or an acute inflammatory disease of greater magnitude than simple appendicitis is probable.

Urinalysis except for the increased specific gravity of dehydration, is normal unless the inflammed appendix lies near the ureter or bladder in which case white blood cells and occasionally red blood cells may be seen. Lewis, Holcroft and Boey (1975) showed that 20% of all their patients had five or more white blood cells in the urine sediment per high power field, irrespective of the diagnosis.

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X-rays are rarely used and in most of our patients in this series it was not used as a diagnostic aid. X-rays of the abdomen are not useful in diagnosing appendicitis and even when abnormal, the findings are non-specific. Its main value is to exclude other diagnoses in the difficult case (Lewis, Holcroft & Boey 1979).

Of the features which may be seen on plain films of the abdomen in acute appendicitis are: Local distended loop or two of small intestines in the right lower quadrant, less often a distended caecum. A gas filled appendix may be visualised, but not invariably indicate acute appendicitis with proximal appendiceal obstruction and a radiopaque fecalith may be seen, and when present in the right lower quadrant is nearly always associated with gangrenous appendicitis.

Barium enema examination may be helpful in selected patients particularly children in whom the diagnosis is in doubt and operation is thought to be hazardous. Complete filling of the appendix and absence of mucosal changes in both appendix and ileocecal region rules out acute appendicitis.

Chest films are sometimes necessary to rule out diseases of the right lower lung field, since lesions that irritate nerves at T10, T11 and T12 may simulate appendicitis.

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TREATMENT

Every surgeon has faced the dilemma of diagnosing acute appendicitis for the surgeon a diagnosis should be more a decision than a description.

A decision for appendicitis is a decision for treatment and not for further investigation. An important consideration in that decision is the consequence of its error. A graver consideration is the consequence of erroneously delaying the decision. Plainly it means that a delay in recognising that a patient has appendicitis may allow time for perforation of the appendix and the sequence of abscesses, peritonitis, intestinal obstruction, draining wounds, hernias and other evils to go with them leading to death.

Immediate appendicectomy is the treatment of choice in acute appendicitis without rupture just as soon as the minimal work up compatible with good medical practice is completed.

There is no need of peritoneal drainage or antibiotics and the wound should be closed by primary closure (Gomez & Wood 1979).

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Ruptured appendicitis with local peritonitis or phlegmon formation should be operated up early in the hospital course.

A brief period of preparation is advisable during which a nasogastric suction is instituted and sufficient intravenous fluids, usually Ringer's lactate solution and 5% Dextrose in water are given to correct systemic fluid and electrolyte deficits. Systemic antibiotics in large doses are administered since bacteroides organisms play a major role in appendiceal infections, clindamycin should be one of the antibiotics used pending culture and sensitivity data.

Patients with free perforation producing diffuse peritonitis should also be treated by adequate fluid replacement, administration of appropriate antibiotics and appendectomy. Preparation may take somewhat longer than with localised peritonitis because of the greater magnitude of the physiologic derangements caused by more aggressive process but rarely requires more than 3-4 hours. It is essential to remove the necrotic appendix to prevent continued contamination of the peritoneum.

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There is a difference of opinion, concerning the optimal timing of the operation for ruptured appendicitis with frank appendiceal abscess formation.

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Paramedian and midline incisions are most commonly

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used when diffuse peritonitis from appendiceal perforation is present, and the diagnosis is not apparent preoperatively, because they can readily be extended to deal with another problem encountered (Lewis, Holcroft & Boey 1975).

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incidence of concomitant major illnesses complicating appendicitis. Slowness in seeking medical attention by the elderly and the surgeon's attempt of a more definite diagnosis because of the greater operative risk further delays treatment and increase the risk of perforation (Owens and Hamit 1979, Pettokallio & Tykka 1981, Cooperman 1983). In our study none of our patients were over 50 years of age and it has been difficult to compare the presentation of the elderly patients. However, it has been noted (Table IV) that most patients presenting with perforation and its sequelae attended to hospital with symptoms more than 24 hours. This is a real delay in seeking medical advice.

Infants also have a high rate of perforation owing to their inability to articulate symptoms and the difficulty encountered in performing physical examination (Cooperman 1983). Savrin (1979) reported a 42% incidence of perforation in infants.

Diagnosis of acute appendicitis is often difficult in the pregnant patient as the symptoms may be attributed to the pregnancy (Gomez and Wood, 1979). Physical findings may be obscured or altered by the cephalad displacement of the appendix as pregnancy progresses.

Basically it is the same disease process as in the

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non-pregnant woman. It is most common in the 20-25 year age group and most frequently seen in women having two or more pregnancies (Gomez & Wood 1979).

Gomez & Wood (1979) also found that it was less common in the last trimester and approximately half of their patients were in the second trimester.

DIAGNOSTIC AIDS

Moderate leukocytosis, ranging from approximately 10,000 to 18,000 per cubic millilitre and associated with polymorphonuclear predominance, is the rule in acute uncomplicated appendicitis (Lewis Holcroft & Boey et al 1975, Schwartz 1979).

If the White Blood cell count is greater than 18,000 per cu.mm. or if the shift to the left is extreme, perforated appendicitis or an acute inflammatory disease of greater magnitude than simple appendicitis is probable.

Urinalysis except for the increased specific gravity of dehydration, is normal unless the inflammed appendix lies near the ureter or bladder in which case white blood cells and occasionally red blood cells may be seen. Lewis, Holcroft and Boey (1975) showed that 20% of all their patients had five or more white blood cells in the urine sediment per high power field, irrespective of the diagnosis.

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X-rays are rarely used and in most of our patients in this series it was not used as a diagnostic aid. X-rays of the abdomen are not useful in diagnosing appendicitis and even when abnormal, the findings are non-specific. Its main value is to exclude other diagnoses in the difficult case (Lewis, Holcroft & Boey 1979).

Of the features which may be seen on plain films of the abdomen in acute appendicitis are: Local distended loop or two of small intestines in the right lower quadrant, less often a distended caecum. A gas filled appendix may be visualised, but not invariably indicate acute appendicitis with proximal appendiceal obstruction and a radiopaque fecalith may be seen, and when present in the right lower quadrant is nearly always associated with gangrenous appendicitis.

Barium enema examination may be helpful in selected patients particularly children in whom the diagnosis is in doubt and operation is thought to be hazardous. Complete filling of the appendix and absence of mucosal changes in both appendix and ileocecal region rules out acute appendicitis.

Chest films are sometimes necessary to rule out diseases of the right lower lung field, since lesions that irritate nerves at T10, T11 and T12 may simulate appendicitis.

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TREATMENT

Every surgeon has faced the dilemma of diagnosing acute appendicitis for the surgeon a diagnosis should be more a decision than a description.

A decision for appendicitis is a decision for treatment and not for further investigation. An important consideration in that decision is the consequence of its error. A graver consideration is the consequence of erroneously delaying the decision. Plainly it means that a delay in recognising that a patient has appendicitis may allow time for perforation of the appendix and the sequence of abscesses, peritonitis, intestinal obstruction, draining wounds, hernias and other evils to go with them leading to death.

Immediate appendicectomy is the treatment of choice in acute appendicitis without rupture just as soon as the minimal work up compatible with good medical practice is completed.

There is no need of peritoneal drainage or antibiotics and the wound should be closed by primary closure (Gomez & Wood 1979).

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Ruptured appendicitis with local peritonitis or phlegmon formation should be operated up early in the hospital course.

A brief period of preparation is advisable during which a nasogastric suction is instituted and sufficient intravenous fluids, usually Ringer's lactate solution and 5% Dextrose in water are given to correct systemic fluid and electrolyte deficits. Systemic antibiotics in large doses are administered since bacteroides organisms play a major role in appendiceal infections, clindamycin should be one of the antibiotics used pending culture and sensitivity data.

Patients with free perforation producing diffuse peritonitis should also be treated by adequate fluid replacement, administration of appropriate antibiotics and appendectomy. Preparation may take somewhat longer than with localised peritonitis because of the greater magnitude of the physiologic derangements caused by more aggressive process but rarely requires more than 3-4 hours. It is essential to remove the necrotic appendix to prevent continued contamination of the peritoneum.

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Kingsley (1969) found on reviewing over 1000 cases of appendicitis, that in general there were fewer complications when invagination was omitted.

Getzen (1968) when reviewing 403 cases of appendicectomies for a duration of five years found that post appendectomy wound infection appear to be correlated more with the degree of inflammation within the appendix, with the thckness of the subcutaneous fat, or both, than with destruction or inversion of the mucosa of the appendiceal stump.

Sinha (1977) concludes from his series that "leaving the stump free in the peritoneal cavity does not increase the incidence of adhesions".

Where inversion of the stump is difficult to perform for any reason, it is quite safe to leave the stump unburied as it is to invert it, and in cases where the caecum is oedematous and friable and inversion difficult, it would seem preferrable to use the technique of simple ligation as an equally safe procedure (Kingsley 1969).

So it appears that the technique of simple ligation only and ligation with invagination have no advantage over the other as far as post operative complications are concerned.

In our series both techniques have been used depending

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on the surgeons preference or the condition of the appendix whether perforated or not and the caecal wall whether oedematous or not.

Most authors think that the omission of invagination of the crushed and ligated appendix stump after appendicectomy simplifies and shortens the operation. No added risk of complication has been found and indeed there is a lower risk of wound infection and a slightly shorter postoperative stay when simple ligation of the appendix stump is practiced (Getzen 1968, Kingsley 1969, Sinha 1977).

When the base of the appendix is so necrotic that closure of the appendix is impossible, a caecostomy tube should be placed through the base of the appendix and brought out through the lateral abdominal wall. Ten to fourteen days postoperatively, the caecostomy tube may be with-drawn after an adequate tract has been allowed to develop. The ensuing faecal fistula should quickly close spontaneously (Cooperman 1983).

Whether or not to utilize peritoneal drainage in patients with diffuse appendiceal peritonitis has been a subject of controversy for years.

Halsted in 1898 made the comment that "No drainage at all is better than the ignorant employment of it". This statement was in contrast to Tait's dictum of 1887 "When

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in doubt drain". Unfortunately no definitive data has yet been presented to settle this dilemma.

A majority of surgeons avoid drains in adults with generalised peritonitis. There is a general feeling that it is physically impossible to drain the entire peritoneal cavity, and that placement of a drain in these circumstances may be harmful because it is a foreign body which is rapidly walled off and may enhance the formation of adhesion around the drain and subsequently lead to intestinal obstruction (Getzen 1968, Haller, Shaker & Donahoo 1973, Marchildon & Dudgeon 1977, Cooperman 1983).

Other surgeons feel that a peritoneal drain may also act as a nidus for infection (Haller, Shaker & Donahoo 1973, Cooperman 1983). However, if perforation has occured and an appendiceal abscess is present, soft rubber penrose drains should be placed in the abscess and brought out through a separate stabwound in the abdominal wall (Cooperman, 1983). Many surgeons also favour the use of drains when the intra-peritoneal contamination is localised to the area of the appendix (Marchildon and Dudgeon 1977).

Most children's surgeons have been insistent on the use of drainage, especially in infants and young children in whom there may be less ability to localise infection and in whom drainage may be more effective

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because of their small peritoneal cavities (Haller, Shaker & Donahoo 1973).

In our present series most of the drainage were used to drain appendiceal abscesses (Table VI). Though most of the patients with perforated appendices were also drained locally at the site of appendix, we did not compare the complications, which might have occurred with the non drained group. Those patients with simple appendicitis were not drained at all.

The skin and subcutaneous tissues should be routinely closed primarily unless the appendix is perforated. (Cooperman 1983). If perforation has occured, the skin and subcutaneous tissues should be left open and packed with Betadine-soaked gauze. If the wound remains clean (usually after 72 hours), delayed primary closure may be safely accomplished. If not the wound is allowed to granulate secondarily.

In our series this was not seriously taken into account and most of the wounds closed primarily. Only in the appendiceal abscess cases were the wounds left to granulate secondarily. This could explain the high incidence of wound sepsis in our series (Table V).

ANTIBIOTIC INFLUENCE AND COMPLICATIONS

The high morbidity rate of perforated appendicitis in most clinical reviews has lead to controversy over

appropriate therapy including the use and timing of appropriate antibiotics.

The use of systemic broadspectrum antibiotics as soon as the diagnosis is clinically suspected or beginning antibiotics intraoperatively if an unsuspected perforation is found is favoured by most surgeons (Marchildon and Dudgeon 1977). There is strong evidence that preoperative antibiotics reduce the incidence of post operative intra-abdominal abscesses.

While antibiotics are useful in treating systemic sepsis, they have no noticeable effect on local wound sepsis if given intraoperatively or later (Lewis, Holcroft & Boey, 1975).

In our series antibiotics were started immediately the diagnosis was suspected.

PROGNOSIS.

The morbidity rates parallels mortality rates, being precipitously increased by rupture of the appendix and to a lesser extent by old age. Most of the serious early complications are septic and include abscess and wound infection.

Wound infection is the common complication following appendectomy occuring in upto 30% of patients (Gilmore

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and Martin 1974). Indeed in our series wound sepsis was the commonest post appendectomy complications and accounted for 13.9% of patients (Table V).

It is most common following appendectomy for perforated appendices and least common after removal of a normal appendix (Cooperman 1983). Wound infection is common but is nearly always confined to the subcutaneous tissues and promptly responds to wound drainage, which is accomplished by reopening of the skin incision.

Abscesses may be pelvic, may occur from a buried stump, or can be hepatic. The commonest of all is pelvic abscess which can easily be drained either rectally or trans-abdominal. Appendiceal stump abscess may be intramural or may burst giving rise to spreading peritonitis. Hepatic abscess is usually a sequelae of generalised septicaemia. Abscesses elsewhere can develop such as subphrenic and in the subhepatic area.

Other complications include faecal fistula which is an annoying but not particularly dangerous complication of appendectomy. It may be produced by sloughing of that portion of the caecum inside a constricting purse-string suture, by the ligature slipping off a tied but not inverted appendiceal stump, by necrosis of an abscess encroaching on the caecum, or by a cecostomy produced by removing a cigarette drain that

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is adherent to the caecum (Schwartz 1979). Significant bleeding is rare but may occur either intra-peritoneally or into the caecum from the inverted appendix.

Intestinal obstruction, initially paralytic but sometimes progressing to mechanical, may occur with slowly resolving peritonitis, with loculated abscesses and exuberant adhesion formation. Intussusception of the appendiceal stump though a rare complication has also been reported to occur (Cooperman 1983).

Late complications are quite uncommon, adhesive bands intestinal obstruction rarely occurs after appendectomy. Incisional hernia is anologous to wound dehiscence, infection predisposes to it, and it rarely occurs in a McBurney's incision but is not uncommon in a lower right paramedian incision. Sterility due to presence of pelvic adhesions in females and right inguinal hernia have also been reported as rare complications of appendicectomy.

Mortality:

There has been worldwide striking improvement in the mortality from appendicitis. The mortality from unperforated cases has remained admirably low, less than 1% and usually less than 0.2% (Getzen 1968).

Perforation of the appendix is the most important influencing morbidity and mortality. In one series, the overall mortality rate was 1.3%, but in those with

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perforation it was 8.3% (Hanswald, Bivin & Meeker et al 1976).

Death is usually attributable to uncontrolled sepsis, peritonitis, intraabdominal abscesses, or gram negative septicaemia. Sepsis may impose metabolic demands of such magnitude on the cardiovascular or respiratory systems, that they can not be met, in which case cardiac or respiratory insufficiency is the direct cause of death.

Among the factors responsible for the significant reduction in mortality especially in the United States are better diagnosis and treatment attributable to the now available antibiotics, intravenous fluids, blood and plasma, but particularly to the greatly improved training of most surgeons; and a higher percentage of patients receiving definitive treatment before rupture.

In our series the mortality rate is 13.9% and all this occured in patients with perforated appendices together with its sequelae of abscesses except one (Table VI). This is higher than expected compared to other series. This can be attributed mostly by a delay in seeking medical advice by most of our patients (see table IV) with its attendant outcome of perforation.

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BURN INJURY IN ADULTS

SUMMARY

During the period of two years starting from July, 1983 to June, 1985, Surgical Firm III one of the Surgical firms at the Muhimbili Medical Centre, attended nine patients.

Seven of these patients were males and two were females (M:F - 3.3:1). The age range was from 16 years to 45 years. The third decade predominates with five patients followed by the second decade which had 2 patients.

The commonest cause of burn in our patients was open flame from petrol and kerosene which had seven victims followed by scalds in the remaining two patients. There was no electrical of chemical burn injuries in our patients.

Four patients died and all of them attended to hospital within 24 hours post burn injury. All the dead patients had burn involving more than 35% of total body surface.

One case fully managed by the author is presented and the literature relevant to the topic discussed.

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INTRODUCTION.

Burn injury constitutes a major national health problem. More than 2,000,000 persons suffer thermal injury annually in U.S. of whom 70,000 must be hospitalised (Schwartz, 1979). The skin being the largest organ in the body is the most frequent site of burn injury.

Burn injury to the skin can produce a multitude of complex systemic changes which if unrecognised and appropriate steps are not taken to circumvert them are detrimental to life.

Pulmonary damage due to smoke inhalation and carbon monoxide intoxication is thought to be the leading cause of death in fire victims. Of 12,000 annual deaths in the United States due to fire, 50 to 60% are secondary to inhalation injury (Donald, Trunkey 1978). Severe inhalation injury in humans is not that common in the absence of thermal injury to the skin, and if such a situation exists then the prognosis becomes very poor. However, it is well documented that thermal injury alone, particularly when combined with shock, will cause severe pulmonary sequelae (Donald D. Trunkey 1978, Schwartz 1979).

The complex pathophysiologic alterations which accompany major thermal injury present the surgeon with an extraordinary therapeutic challenge.

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As in the other types of trauma thermal injury afflicts children and young adults. Prolonged morbidity, as well as temporary or permanent disability, associated with thermal injury results in a staggering economic drain on social resources.

Financial support is often required to defray expenses associated with prolonged hospitalisation, loss of family income sources, and replacement of lost manpower within the working force.

CASE PRESENTATION

A.K. a 26 year old male, residing at Magomeni was admitted to our ward on 15.9.84 at around 4.30 p.m. after having sustained burn from an open petrol flame two hours before admission.

He was working in a garage and unfortunately a colleague threw a burning cigarette near a petrol tank which immediately caught fire and exploded. The fire caught the patient's shirt and trousers which were made of nylon and burned him on the back, legs and arms before he could successfully undress them. The patient was immediately taken to Magomeni dispensary where nothing was done as first aid except setting up an intravenous drip of normal saline before being referred to Muhimbili.

Examination revealed a healthy, conscious young male,

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in great pain, irritable, not dyspnoeic, afebrile and not pale. Vital signs:- blood pressure was 110/70 mm Hg, pulse of 64 beats per minute of good volume and regular. The respiratory rate was 20 breaths per minute. His heart sounds were normal and there was nothing remarkable on abdominal examination.

He had burns involving the whole of the back extending from the nape of the neck down to the buttocks, and also involving the posterior aspect of the lower limbs and upper limbs bilaterally. There were scattered blisters and peeling of skin through most of the skin lesions. The colour of the burn of the back was whitish and it was insensitive to pin prick. The remaining lesions had scattered areas which were pinkish in colour and sensitive to pain.

He was provisionally diagnosed as having sustained burn of approximately 38 percent of the total body surface, full thickness on the back and partial thickness on the remaining areas with impending shock.

A blood sample was immediately taken for the estimation of PCV, serum electrolytes and a drip of Ringer's lactate solution started. Pethidine 100mg was given intramuscularly to be repeated every six hours for the first 48 hours. The blood sample

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results were reported back the same day by telephone as:- serum sodium - 122 mmol/L, Serum Potassium - 2.4 mmol/L., Serum Chloride - 102 mmol/l., PCV - 54.

The patient was considered to be at risk of developing hypovolaemic shock and thus fluid and electrolyte therapy was begun early.

A cutdown using the long saphenous vein on the right leg was done after failing to get a vein in the upper limbs. The previous one had run out of the vein.

The fluid requirements were calculated using the Parklands formula and a volume of about 9 litres obtained to be given within the first 24 hours. Half of this was given within the first 8 hours and the remaining half divided equally in the next two periods of 8 hours. The type of fluid administered was Ringer's lactate solution. The patient was kept on pethidine 100 mg. intramuscularly every six hours in the first 48 hours. A course of Procaine Penicillin Fortified 1.2 mega units was started daily for 5 days and a booster dose of tetanus toxoid 0.5 mls. was given by a deep intramuscular injection.

The wound was then cleaned with antiseptic, all the peeled off skin removed and the blisters opened and the patient nursed under a mosquito net (exposure method).

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Clean linen was used and advice was given to change them whenever they got soaked.

Emphasis was made to monitor the pulse rate, respiratory rate, temperature and urine output through an in-dwelling catheter. It was impossible to record hourly blood pressure because all the limps were burned and swollen. By the end of the first 24 hours the patient's vital signs monitored above were within normal limits.

During the second 24 hours the patient received two units (1000 mls) of plasma from the blood bank and about 3000 mls of 5% Dextrose. Oral fluids were encouraged. A second specimen of serum electrolytes was taken and the results were:- Serum Sodium - 136 mmol/L, Serum Potassium - 5.7 mmol/L, and Serum Chloride - 98 mmol/L. High protein diet was also encouraged together with fluids provided there was no vomiting or nausea.

From the second day onwards the general condition of the patient tremendously improved and all the vital signs remained stable. The wounds on the arms and legs started drying up and were being cleaned twice daily with antiseptics and left open.

On the 13th day post burn the general condition of the patient started deteriorating. He started running

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temperatures and becoming confused. The burn wound on the back was found to be septic accumulating pus below the eschar which had formed. A pus swab was taken for culture and sensitivity which yielded Pseudomonas aeruginosa sensitive only to Gentamicin.

Gentamicin was started empirically before the results of culture and sensitivity at a dose of 80 mg. 8 hourly for 7 days, the wound irrigated with Eusol before dressing twice daily and any loose eschar removed. He was planned for excision of the eschar under general anaesthesia the next operation day.

A blood culture for aerobic and anaerobic organisms was taken but unfortunately the patient died on the seventeenth day post burn without the results of the blood culture. The cause of death was thought to be Septicaemia.

ANALYSIS OF THE PATIENTS STUDIED

1. Sex distribution M:F - 3.3:1

TABLE I : Age distribution of the 9 patients admitted with burns from July 1983 to June, 1985.

Age Group (years)	No. of Patients
11 - 20	2
21 - 30	5
31 - 40	1
41 - 50	1
over 50	0
TOTAL	9

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Most patients were in the third decade.

TABLE II: The Aetiology of burns in the 9 patients admitted from July 1983 - June, 1985.

Cause	No. of Patients
Scalds	2
Open flame	7
Electricity	-
Chemical	-
T o t a l	9

Open flame was the commonest cause of burn in 7 patients.

Mortality : 4 patients.

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TABLE III : GENERAL ANALYSIS OF ALL 9 PATIENTS WITH BURNS JULY, 1983 - JUNE, 1985

	Sex	Age (yrs.)	%Burn	Cause of burn	Area of body burned	Duration of burn before arrival at hospital(hrs)	Comment	Cause of death
1	F	45	35%	Open flame (kerosene)	Both lower limbs, buttocks, perineum, upper parts of back, some areas of upper limbs.	19 hours	Died on same day	Hypovolaemic shock.
2	M	38	15%	Petrol flame	face, hands and back of right thigh.	9 hours	alive	-
3	M	16	12%	flame	Right leg (lower part ant. and post) Posterior aspect $\frac{1}{3}$ right thigh.	3 hours	alive	-
4	M	26	38%	Petrol flame	Whole back & buttocks lower limbs ant.& post. patch areas upper limbs.	2 hours	Died on seventeenth day	Septicaemia
5	M	22	4%	Scalds	Right arm & left flank	4 hours	Alive	
6	F	30	5%	Scalds	Abdomen & left thigh (infected)	68 hours	Alive	
7	M	22	15%	Petrol flame	Half of the back, part of arm & half lower limb.	4 hours	Alive	
8	M	24	40%	Petrol flame	Whole back and one third abdomen, Hands and arms, part of neck, buttocks, legs & Dorsum of feet.	2 hours	Died after 2 days	Hypovolaemic shock.
9	M	24	45%	Kerosene	Trunk anteriorly, face & left arm, left leg, right leg, anterior right thigh and below knee right leg.	5 hours	Died after 7 days	. Endotoxic shock . Anaemia

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DISCUSSION

Aetiology of Burns.

Burns are caused by the application of heat to the body. This can either be directly by contact or indirectly by radiant heat. The depth of the resulting burn injury depends on the intensity and duration of heat application and the conductivity of the tissues involved (Schwartz 1979).

The common heat sources are an open flame and hot liquids. This is also true in our environment where we find that all the patients sustained burn from these two sources (Table 3). In addition, thermal injury can also be caused by direct contact with toxic chemicals, high voltage electric current, actinic rays, irradiation and friction.

Damage as a result of heat rarely occurs below 45°C. Between 45°C and 50°C gradation of cell injury may occur; and above 50°C denaturation of protein elements become apparent (Moncrief 1974, Baxter 1977).

Laboratory accidents, civilian assaults, industrial mishaps, and inexpert application of agents used for medical purposes account for most of the chemical burns in the civilian population.

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PATHOPHYSIOLOGY

Coagulation necrosis due to thermal energy causes tissue death and injury, resulting in a loss of fluid from the intravascular compartment which necessitates administration of intravenous replacement of fluids to prevent shock and maintain organ function.

The magnitude of this intercompartmental fluid shift is in direct proportion to the extent of the burn, with the vascular leak due to this pathologic change producing oedema in the area of the burn.

In patients with burns of more than 25% of the total body surface, there is increased capillary permeability and oedema formation in unburned tissues as well (Pruitt 1978). The loss of extracellular fluid from the tissues is seen as exudate from the surface of burn filling blisters and soaking sheets, and later oedema under the burn (Bayley 1978).

Several vasoactive substances have been proposed as mediators of the early post burn increase in capillary permeability including histamine, serotonins, the kinins and most recently the prostaglandins.

Whatever the cause of oedema, fluid appears to accummulate progressively during the first 24 to 36 hours post burn with the intravascular fluid

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loss rate greatest in the first 6 to 8 hours following injury (Baxter 1978, Pruitt 1978).

Oedema accumulation thereafter decreases as post burn time increases with the rate of fluid loss from the vascular compartment influenced by vasoconstriction, haemoconcentration, and red cell and platelet aggregation, all of which act to reduce the capillary surface through which fluid is lost, and by hydrostatic effect of the increase in tissue pressure resulting from oedema formation per se.

The most measurable and probably the most important organ system affected directly and indirectly by serious burn injury is the heart and circulation.

The rapid increase in extravascular volume in the burned tissue is associated with a prompt decrease in plasma volume and in cardiac output (Rudowski 1979). The cardiac output may fall precipitously to between 30% and 60% of predicted normal values (Baxter 1978).

Although fluid resuscitation results in return of urine flow (20-50 mls/hour) within 2-3 hours after initiation of therapy, the cardiac output will only gradually improve, approaching 3.1 ml/m^2 between 6 and 18 hours post injury. Further increases are unusual

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until plasma is administered in the fourth eight hour period.

Pulmonary wedge pressures are low throughout even in the resuscitation period unless high output cardiac failure occurs. Systemic blood pressure (intra-arterially) is normal both prior to and after initiating resuscitation.

Parabiotic experiments have shown that in severe third degree burns, the serum contains a substance able to greatly diminish contractions of the heart muscle (Baxter 1976).

Excessive cardiac work is caused by the added insults of progressive anaemia, fluid mobilisation, inadequate nutrition and electrolyte imbalance or infection.

The most common fluid volume problem encountered in adults is continued plasma loss during the first five days post burn injury (Pruitt 1978, Baxter 1978, Schwartz 1979, Cuono 1980).

In a small number of patients, particularly those with burns of more than 50% of total body surface area, a significant quantity of plasma is required between 30 and 38 hours post burn to prevent the development of hypovolaemic shock.

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Several problems are commonly encountered in plasma electrolyte concentrations. Most frequently changes in sodium concentrations and hypokalaemia, and hypernatraemia results from the accelerated insensible water losses through burn skin. The amount of evaporative water loss in burned patients ranges from 4 to 14 times the normal insensible loss of 10 mls/kg/day (Pruitt 1978, Cuono, 1980). The variability of water losses is not only different depending upon the depth of the burn and the post burn day, but is profoundly influenced by the ambient temperature and air velocity of the environment (Baxter 1978). In patients with burns of between 40% and 70% of total body surface area, an intake of between 3 and 5 litres is a mean average replacement of the evaporative losses. Underestimation rapidly results in hypernatraemia.

Hyponatraemia is the most common electrolyte abnormality encountered between the third and seventh post-burn day. This is usually a result of overestimation of water requirements. The sodium ion is the one which determines the osmolarity of blood. In hyponatraemia the plasma osmolarity falls and a concentration gradient begins to favour shift of water into cells. If the extracellular fluid loss is then replaced by solutions which are hypotonic with respect to sodium, for example,

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by allowing the patient to drink tap water or infusing 5% Dextrose in water intravenously, then further dilution of plasma increases the probability of intracellular overhydration leading to the syndrome of water introxication (Bayley 1978).

The circulating levels of Antidiuretic hormone (ADH) remains very high for the first five to six days post injury, promoting maximum water retention. In patients with persistently low plasma volume, the hyponatraemia is often produced by secondary aldosteronism.

Potassium is usually excreted in large amounts, usually beginning 36 to 48 hours post burn in the presence of good renal function. If replacement is not affected hypokalaemia results.

Low serum calcium concentrations routinely accompany burn injury but, usually do not require therapy since calcium, both ionised and total, return to normal by the fourth and fifth day.

Significant changes in all formed elements of the blood and in the clotting mechanisms are found routinely in major thermal trauma. The most important and treatable hematologic abnormality is the progressive anaemia of the early post burn period. This is a

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progressive ongoing hemolytic state resulting in destruction of between 4 and 14% of the red cell mass per day requiring repeated red cell transfusion (Baxter 1978).

The haematocrit is elevated mainly due to haemoconcentration in the early post burn period. If blood transfusion is affected at this time, it tends to aggravate the viscosity further leading to sludging and diminishing further the already impaired tissue perfusion.

Respiratory failure may be caused by inhalation of smoke, carbon monoxide and other incomplete products of combustion with damage both to larynx, proximal and distal airways and to the lung parenchyma.

The most important factor is failure of the pulmonary parenchyma with alveolar collapse, reduction of compliance and trans-pulmonary shunting (Rudowski 1979).

In contrast to both thermal and chemical burns, electrical injury usually results in minimal destruction of skin. The magnitude of the injury is directly related to the amount of current passing through tissue between the point of contact with the electric source and the exit site at which the patient

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is grounded. Nerve, blood and muscle offer the least resistance to electric current and thus suffer the maximum amount of tissue damage.

Thermal injury to musculature is frequently associated with release of hemachromagens into the blood stream which are subsequently excreted via the urinary tract. Thus "port-wine" coloured urine containing myoglobin is not unusual following major electrical injury.

MANAGEMENT:

The initial therapy of the patient with a major burn should be directed towards restoration of normal physiologic parameters and prevention of life threatening complications.

With the exception of chemical burns, in which the toxic agents must be diluted with water and physically removed as rapidly as possible to prevent further destruction, the burn wound is of secondary importance during the first few hours after the injury.

Immediate pulmonary complications may become manifest in the thermally injured patient. Excessive exposure to smoke may result in carbon monoxide poisoning which necessitates the immediate administration of 100% oxygen with ventillatory support if necessary

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in order to displace the tightly bound carbon-monoxide from the haemoglobin molecule.

Upper airway obstruction in patients with burns of the head and neck may occur during the first 48 hours and the obstruction is usually related to soft tissue oedema of the oral pharynx and vocal cords following inhalation of hot gases. In such cases immediate intubation using an endotracheal tube should be done and the tube should not be removed until the third post burn day. Other authors have advocated tracheostomy as a means of relieving the upper airway obstruction, but it is felt better to intubate rather than do tracheostomy (Worsonu 1978).

Direct thermal injury of the lower respiratory tract is noted in patients injured by superheated steam and may lead to tracheitis, pulmonary oedema and bronchopneumonia.

The rapid shift of fluid and the accompanying pronounced changes in the size of the fluid compartments in the early post-burn period is minimized and corrected by resuscitation fluids. Otherwise hypovolaemic shock with its attendant sequelae supervenes (Pruitt 1978).

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A child with a burn of 10% or more of the body surface and an adult of more than 15% will develop shock. This can be anticipated and prevented by immediate intravenous fluid therapy (Bayley 1978).

Assessment

Assessment starts with the estimation of the surface area involved. This is commonly estimated by Wallaces Rule of 'NINE' which divides the body into the following: involvement of head and neck - 9%, upper extremities each carries 9%, lower extremities each carries 18%, anterior and posterior parts of the trunk each carries 18% and the perineum 1%.

In the patchy scattered burns the estimation can be accomplished by assuming that the patient's palm represents approximately 1% of total surface area.

The next thing to be estimated is the depth of the burn wound. The depth of the wound is classified into three groups. First degree burn is that which involves the superficial layers of the epidermis. There is simple erythema of the skin, with only microscopic destruction of superficial layers of the epidermis. This is of little clinical significance, since the water barrier of the skin is not disturbed. Systemic cardiovascular disturbances are rarely seen. It rapidly heals if the patient avoids further exposure to heat source with minimal or no scarring.

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Second and third degree burns are of equal physiologic significance and may be summated in the estimation of total body surface. Second degree burn extends through the epidermis to the dermis. Viable epithelial elements from which epithelial regeneration can occur are usually retained, thus this type is often described as partial thickness burn. Third degree burns entail involvement of the whole skin thickness without residual dermal appendages or epithelial elements. Spontaneous regeneration of the epithelium is not possible, and the burns are described as full thickness. This type of burns requires skin grafting if extensive scarring is to be avoided.

Fluid Replacement

If shock has set in or is expected, fluid therapy should be instituted immediately to avoid the possible sequelae of hypovolaemia.

Since 1947 a variety of formulae have been advanced for use in estimating the fluid needs of burned patients during the first 48 hours post burn (Pruitt 1978). These formulas have been developed as guides to initial resuscitation in hypovolaemic shock following thermal injury.

Most utilise various combinations of crystalloid and colloid solutions but differ widely in the ratio of

colloid crystalloid as well as the rate of fluid administration.

It is of relatively little consequence which formula is utilised to begin such therapy, as long as this is modified according to the patients changing requirements.

Most formulas recommends that one half of the total volume of fluids estimated for the first 24 hours following injury be given in the initial eight hours post burn when the rate of fluid loss from the vascular compartment is at its greatest. Moreover, the timely institution of fluid infusion and the administration of adequate volume have essentially eliminated acute renal failure as an early complication of burn injury.

The formula below has been popularised by Baxter and is known as the Parkland formula. This formula recommends the infusion of 4 mls of electrolyte solution (Ringer's lactate) per kilogram of body weight per percentage of burn in the first 24 hours. This is necessary to support cardiac output, maintain an adequate hourly urine volume and restore more rapid cell function as indexed by membrane electro-potential difference. Half of this amount is given in the first 8 hours, a quarter in the second 8 hours and a quarter in the third 8 hours. During the second 24 hours glucose in water is given to replace

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evaporative water loss, and maintenance of sodium concentration of about 140 mEg/l.

Approximately 4 to 5 litres are needed in a 70 kg patient with 50% burn or more. Colloid solutions (plasma) are also given to maintain plasma volume in patients with more than 40% second and third degree burns. Approximately 250 mls for each 10% total body surface area is needed in burns of more than 20% (Pruitt 1978).

Another formula in use is the Brookes formula and the modified Brookes formula. Brookes formula has been popularly used by us to manage most of our patients in our firm.

Brookes formula aims at giving crystalloid solution containing water and electrolytes and colloids in the following way. Crystalloids (Ringer's lactate or Normal Saline) 1.5 mls/kg. per percentage burn and colloids 0.5 mls/kg per percentage burn in the first 24 hours. To this about 2 litres of Dextrose 5% in water are added to replace the insensible water loss. The administration rate is the same as in the Parkland formula. In the next 24 hours you give half the calculated crystalloids and colloids in the first 24 hours plus the same 2 litres of 5% Dextrose in water to replace the insensible water loss.

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In the modified Brookes formula 2 mls are given per kilogram body weight per percentage burn, all of which is either Ringer's lactate or normal saline. No colloid or glucose is given in the first 24 hours. In the second 24 hours half of the Ringers lactate or normal saline is given plus 0.5 mls/kg/per percentage of burn of colloid and 2 litres of glucose. Glucose is given to supply the metabolic needs, to replace evaporated water and to facilitate sodium load excretion.

Other formulas in use are Evans formula, Moore's budget formula and Massachuset General Hospital formulas. During the first 24 hours post burn, studies indicate that colloid containing fluid was retained within the circulation to no greater extent than an equal volume of colloid-free electrolyte containing fluid. (Pruitt 1978, Bayley 1978). In the second 24 hours data indicate that colloid-containing fluids were retained within the circulation to a greater extent than were colloid-free electrolyte containing fluids. Subsequent studies by many authors have confirmed the increased plasma volume augmentation of colloid-containing fluids during the second post burn day. This finding speaks for the use of colloid-containing fluids in the second 24 hours post burn to reduce fluid loading in patients with extensive burns. (Rudowski 1979). Plasma and dextran are not

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fully available and they are expensive. Plasma is also potentially dangerous when blood is unscreened for Hepatitis B in a continent where 5% of all donors carry the virus.

Recent reports have suggested that the infusion of hypertonic saline will effect resuscitation and avoid fluid overload (Pruitt 1978, Bayley 1978, Cuono 1980). Monafo (1973) must be credited for having performed the first convincing clinical trials that demonstrated not only the feasibility of using hypertonic sodium solutions but their superiority to either isotonic or hypotonic regimens. Hypertonic lactated saline is most easily prepared by adding two ampoules of sodium lactate (50 mEq each) to one litre of normal saline solution (with a sodium concentration of 154 mEq/l).

Patients resuscitated with hypertonic lactated saline, however receive only about two thirds of the water load given in hypotonic resuscitation. No additional free water is given during the first 24 hours. The infusion rates of hypertonic lactated saline during this period average 2 ml/kg/% of body surface burned.

Calculated insensible water requirements are administered during the second 24 hours in conjunction with a proportionate decrease in the rate of

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administration of hypertonic lactated saline. The total volume load administered during the second 24 hours averages 1.5 ml/kg/% of body surface area burned in adults. (Monafo 1973).

As with any resuscitative regimen, recommended rates of administration of fluid derived from formulae are purely guidelines for initial therapy which must be monitored continuously and modified as the general condition of the patient dictates. In all cases, hypertonic resuscitation is terminated at 48 hours post burn.

Moderate hypernatraemia is the rule during resuscitation with hypertonic lactated saline and it is generally well tolerated. However Shimazaki and Co-workers (1977) have observed that when the serum concentration exceeds 165 mEq/l in patients receiving hypertonic lactated saline, urine output falls sharply to unacceptable levels. In such circumstances appropriate parenteral and/or oral free water supplementation is instituted in conjunction with a temporary reduction in the rate of administration of hypertonic lactated saline.

Fluids by mouth should also be started if there is no nausea or vomiting. These can supplement intravenous infusion and obviate the need for a drip in burns just larger than the critical size (up to

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15% in a child and 20% in an adult). Oral fluids should be isotonic and a satisfactory and palatable solution is made up by dissolving one teaspoon of salt and one teaspoon of sodium bicarbonate in one litre of water (Bayley 1978).

MONITORING OF RESUSCITATION

The adequacy of resuscitation, is most readily judged by frequent assessment of the patients general condition, hourly or more frequent measurement of vital signs and hourly measurement of urinary output following placement of an indwelling catheter.

Well resuscitated patients with major thermal injury rarely display hysteria, acute anxiety, or hostility. If these symptoms occur it indicates hypoxia most probably secondary to hypovolaemia and necessitates the increase in the rate of fluid-administration.

The reliability of the blood pressure monitoring by means of sphigmomanometer is compromised by peripheral Vasoconstriction in the immediate post-burn period and by oedema (Pruitt 1978). If because of other injuries blood pressure measurements is deemed essential, an intra-arterial cannula should be placed for that purpose. In our situation the hourly recording of the Blood pressure and the pulse rate

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recordings have been extensively used to determine the adequacy of fluid replacements in conjuction with the hourly urine output.

The hourly urinary output does not precisely reflect the status of the circulatory system, but is sufficiently reliable in the vast majority of burned patients to justify the reliance placed upon it as a guide in managing resuscitation (Moncrief 1971). In the adult one should aim to administer fluids sufficient to obtain about 30 to 50 mls of urine per hour. Oliguria in the first 48 hours is seldom due to acute renal failure and usually responds to an increased rate of fluid administration.

During the period of increased capillary permeability, the haematocrit as noted by Moyer (1965) is unresponsive to fluid administration and is therefore not a useful guide for resuscitation.

The serum electrolyte estimation will guide as to how should replacement be done. In cases where potassium is excreted in large amounts by the kidney, especially beginning around 36 to 48 hours post burn, from 80 to 200 mEq of potassium per day may often be required to maintain a normal serum potassium concentration. Hyponatraemia and hypernatraemia are mainly regulated and corrected by a good

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estimation of fluid replacement. Underestimation rapidly results in hypernatraemia (Baxter 1978).

In the first day patients with thermal injury tend to have high temperatures due to tissue destruction. From the third day onwards high temperatures will reflect infection.

GENERAL THERAPEUTIC MEASURES

An analgesic and/or a sedative should be given either intravenously or intramuscularly but not subcutaneously. If there is full thickness burn, the intrinsic sensory nerve endings have also been destroyed and the wound itself is painless. In contrast the second degree burn can be quite painful initially. Therefore the requirement for sedation is inversely proportional to the depth of the initial thermal injury. Sedation should be kept at a minimum to prevent depression of cardiopulmonary function and to allow evaluation of the sensorium.

All burn injuries must be considered contaminated and tetanus prophylaxis is mandatory except in those patients actively immunized within the preceding twelve months. If a booster was received within the preceding 10 years, the administration of 0.5 mls of Tetanus toxoid will usually provide adequate prophylaxis (Sinha 1978). In the absence of active

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immunization within 10 years, tetanus immunoglobulin (Human) should be simultaneously given at another site. This is the regimen used in our patients.

Systemic antibiotics in the burned patient are indicated for the treatment of systemic infection and as an adjunctive therapy in the treatment of uncontrolled septic process. (Haburchall, Pruitt 1978, Monafo 1978). Uncontrolled infection may be recognised first as local disease such as burn wound infection, pneumonia, urinary tract infection of suppurative thrombophlebitis. If treated aggressively and early with appropriate medical and surgical intervention, such local sources may be eradicated before the development of the clinical picture of sepsis.

When sepsis is diagnosed, an intensive search should be made to identify the source. Auscultation of the chest and radiologic examination of the chest, urinalysis, sputum, urine and paired blood cultures obtained. Also rule out suppurative thrombophlebitis. Only after these data have been collected should the patient be given systemic antibiotics.

Prophylactic administration of antibiotics, especially in the early post burn period to prevent haemolytic streptococcal infection, which may be fulminating

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has been recommended by some authors (Monafo 1978, Schwartz 1979). B haemolytic streptococcal wound colonization is commonest in the first 3 to 4 days post burn. The prophylactic administration of penicillin systematically has been found to be highly effective in preventing this infection. All of our patients were given prophylactic penicillin for this purpose in the first week post burn.

Some authors no longer practice routine prophylactic administration of penicillin in the fear that it is associated with more rapid emergence of resistant gram negative organisms (Haburchall, Pruitt 1978).

There are two clinical situations in which short courses of prophylactic systemic antibiotics are probably indicated in the burned patient; during autografting and at the time of wound excision (Haburchal, Pruitt 1978, Monafo 1978).

The choice of antibiotic depends upon the suspected organism causing infection, its likely resistance pattern, and its site of localisation. As results of cultures are often not available for up to 48 hours, the decision as to which antibiotic to give must often be made on the basis of gram stain or epidemiologic consideration.

Early after burn injury, the burn wound is colonised

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with gram positive respiratory and dermal flora such as streptococcus and Staphylococcus. Later gram negative enterics especially Pseudomonas may become predominant.

Blood transfusion in burned patients should be avoided in the first 48 hours. The haematocrit is usually elevated in the first 48 hours, and since the viscocity of whole blood is directly related to the haematocrit, the administration of blood will further accentuate the impairement of tissue blood flow. Furthermore, redblood cell destruction is higher in the first 24 hours post burn and stored blood is rich in potassium. Blood is given during the first 48 hours only if the patient has profuse blood loss or in patients with pre-existing anaemia. (Pruitt 1978).

Burned patients having infection need more blood as do patients who undergo frequent wound excision and grafting procedures. Patients with deep burn of more than 25% of body surface area, blood transfusion is mandatory.

NUTRITIONAL SUPPORT

Patients with major thermal injury exhibit hyper-metabolism as a result of neurohormonal alterations (Curreri, Luterman 1978, Rudowski 1979). Burn injury is associated with a resetting of the central nervous

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system temperature control centres. As a result the critical temperature is slightly increased and the patient attempts to maintain an elevated core temperature. Increased sympathetic discharge is accompanied by elevated levels of plasma catecholamines that stimulate heat production and substrate mobilisation. Simultaneously severe catabolism is observed during the immediate post-burn period. (Curreri, Luterman 1978).

The clinical implication of the hypermetabolic and catabolic response is striking. Energy requirements of up to 5000 k cal per day are often observed. Failure to supply enough exogenous calorie intake to meet these needs results in rapid consumption of endogenous fuel stores and eventual morbid consequences such as wasting, delayed wound healing, decreased host resistance, and cellular dysfunction (Curreri, Lutterman 1978, Rudowski 1979).

In severe burns it is essential to supply between 6000 and 8000 calories and from 200 to 300 grams of proteins per day with careful attention to electrolyte balance and to the addition of vitamins (Rudowski 1979).

Although the cornerstone of nutritional management is the provision of adequate calories from an exogenous source, much can be done to minimize energy demands. Every effort should be done to minimize pain,

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anxiety, fear and cold stress which stimulate the release of catechlamine. Appropriate use of narcotics and tranquilizers to provide comfort as well as the provision of external heat are effective in this regard. (Curreri, Lutterman 1978).

Another stimulus to higher metabolic expenditure is infection which can be avoided by early burn wound closure.

The daily caloric requirements of adult with burns of more than 20% of body surface area may be estimated by the formula; 26 Kcal per kg of body weight plus 40 Kcal per % burn (Curreri, Lutterman, 1978). After caloric requirements are calculated, dietary programs should be designed to produce enough nitrogen to ensure a calorie:nitrogen ratio of approximately 150:1.

Whenever possible the enteral route should be utilised for dietary supplementation. In patients with burns of greater than 30% total body surface the enteral route alone will not achieve the maintenance of good nutritional state. The use of small silastic naso-gastric feeding tubes, through which complete liquid diets may be delivered is recommended as soon as gastrointestinal function has been restored.

In patients with burns of greater than 40% total body

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surface, complete diets of high caloric density are preferred in order to limit the volume of free water administration. One commercially prepared diet (Ensureplus) contains 1.5 k cal per ml. allowing delivery of 4500 k cal per day when given at a rate of 125 ml/hour (Curreri, Luterman 1978).

When caloric requirements cannot be met entirely by enteral feeding, dietary supplementation may be accomplished by the peripheral administration of Intravenous fat emulsion (intralipid). After testing for anaphylaxis, 0.8 gm/kg/day may be administered and this dose gradually increased to 2gm/kg/day which may provide slightly more than 1200 k cal to a 70 kg. adult.

Unfortunately nothing much can be achieved in our environment as far as the commercially available diets and intravenous fat emulsion are concerned. These are not readily available and are expensive. Dependence has been placed on the oral feeding of locally available high protein diets such as soup, meat, eggs, milk to those who can tolerate oral feeding.

BURN WOUND CARE

The main objectives of burn wound management are the prevention of infection, removal of dead tissue and resurfacing of the wound with durable epithelium

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(Sharma 1978).

In superficial burns prevention of local sepsis alone results in spontaneous healing of the wound by regeneration of epithelium with little or no disability. In full thickness burns prevention of infection gives rise to dry eschar formation. This is removed at the earliest opportunity and the raw area skin grafted (Shuck 1978, Sharma 1978). Failure to prevent secondary infection of deep second degree burn wounds may result in conversion of partial thickness injury to a full thickness injury.

Numerous treatment policies of burn wound care have been advocated by various workers all over the world. Acceptable methods include exposure, "occlussive" dressing, topical antimicrobial agents, primary excision and skin grafting, delayed excision and skin grafting and conservative removal of slough by wet dressing.

Exposure method

The aim of exposure method of burn wound management is to encourage dry crust formation which is protective and an unrewarding site for bacterial proliferation. (Sharma 1978).

In superficial burns epithelial regeneration takes

place within 10 to 21 days and the crust separates spontaneously leaving an unscarred and well healed surface. This is recommended for facial injuries, because of the good blood supply and difficulty of dressing. No topical agents are needed for the face since infection in this area is rare. Any dressing near the face and neck runs the risk of maceration. The method is simple, inexpensive and allows daily inspection of the wound. Most surgeons treat most burns by the exposure technique. However circumferential burns require sophisticated equipment to expose the whole area at one time.

Open method that employes topical creams cannot be utilised for a child who must attend school or for an adult who needs to run the home or go to work. So in these situations occlussive dressings are used. Open method is messy and soils the bed clothing and furniture. Open method is defeated when clothing is placed over the wound. It is clear that burns of the buttocks, trunk and thighs are excluded from this type of management, particularly if the patient must leave the home.

In our circumstances most of our patients with superficial burns have been treated by this method under a bed craddle or mosquito net. This is because it is very difficult to isolate our patients in a room where the environment is controlled.

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Occlusive dressing

At present this method is used only for minor burns in the outpatient department and in circumferential burns. In this method the dressing should be bulky, dry to allow any drainage to come into the dressing but not through it and so eliminating dead space and also it gives vascular support and produces a splinting effect. It should be comfortably affixed to the extremity or trunk and the first layer should be non adherent (Shuck 1978, Sharma, 1978).

If a dressing is utilised on the hand it should be lightly applied so that finger and wrist motion can be continued.

Although the method is cumbersome and expensive it protects the area from bacterial invasion and is liked by the patient and his relatives.

TOPICAL THERAPY

Burns are prone to infection. The larger the injury, the greater is the risk. It has been recognised for a long time that 75% of all severely burned patients die several weeks after their injury from infection (Rudowski 1979). Age and general debility both probably add significantly to the propensity to infection and protein calorie malnutrition, which adversely affects the already impaired immunocompetence

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of the burned patient.

Antibacterial drugs administered systemically have no access to necrotic tissue and influence neither the proliferation of micro-organism nor the production of toxins. The only way to prevent burn wound sepsis is by the use of local antimicrobial drugs (Rudowski 1979). Many have been developed and are very effective.

Moyer (1965) and Moncrief (1969) introduced 0.5% Silver nitrate solution. It is effective against most gram positive organisms and most strains of pseudomonas, although it has limited effectiveness against other gram negative bacteria such as Enterobacter and Klebsiella. Properly used it is an effective prophylactic agent although its efficacy decreases when wound size exceeds 50% to 60% of body surface (Monafo 1978). Fungal superinfections are rare unless concomittant administration of systemic antibiotics is intense and prolonged.

It is readily available but its major disadvantage is severe electrolyte depletion primarily sodium and chloride, necessitating frequent monitoring of serum electrolytes. It needs continuous bulky wet dressing which appreciably increase the cost of the method and also it inhibits early active movements of the extremities.

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Silver Sulfadiazine (Silvadene) was developed by Fox in 1968. The drug has proved effective against a wide range of gram positive, and gram negative organisms as well as most anaerobes. Treatment failure is frequent, however when the wound size exceeds 60% of body surface area. Silver sulfadiazine cream should be liberally applied to the wound, which can either be left open or covered with a dressing. Application is soothing and not painful and keeps the eschar soft. There is no inhibition to carbonic anhydrase. (Monafo 1978).

There is recent evidence that the modification of silver sulfadiazine by the incorporation into it of cerium nitrate strikingly enhances its clinical efficacy.

Mendelssohn and Lindsay (1962) used a 10% water soluble cream of Mafenide acetate (Sulfamylon). It has essentially the same bacterial spectrum as silver sulfadiazine. This cream once applied it allows open treatment of burn wounds. It is a potent carbonic anhydrase inhibitor and may induce acid-base derrangement (Monafo 1978). Mafenide causes pain on application and occasional hypersensitivity reactions occur in 5 to 7% of cases. It produces delayed eschar separation and the emergence of opportunistic infections. Despite its numerous and significant disadvantages, Mafenide can be used successfully to control invasive

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burn wound infections especially when they are relatively localised.

Geogiade et al (1972) introduced Betadine (Povidone-iodine). It is a water soluble topical antiseptic complex of polyvinyl-pyrrolidone iodine with a long record of clinical efficacy as an antiseptic for use on normal skin and mucous membranes. It is manufactured as an ointment or aerosol cream. It is effective against a wide range of gram positive and gram negative organisms as well as some fungi. It readily diffuses through eschar, absorbed and rapidly excreted. Although it has been recommended for use in moderate and major burns, clinical data documenting its efficacy in suppressing the wound flora in such patients are scanty (Monafo 1978).

Gentamycin sulfate is available for topical use as a 0.1% cream or ointment. It is applied topically to open wounds and is easily absorbed. Ototoxicity or nephrotoxicity can result. It may be useful for brief periods, applied to the smallest possible areas of infection, in which case blood levels of the drug should be carefully monitored. Superinfection with resistant bacterial strains will quickly occur with its protracted topical use.

Topical antibacterial efficacy can best be monitored by quantitative and qualitative bacteriological analysis

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of the burn wound. Periodic bacterial cultures should be done. Culture results properly interpreted may be helpful in predicting treatment failure and a change in topical agent done (Rudowski 1979).

EXCISIONAL THERAPY AND WOUND CLOSURE

Clinical and experimental experience concerning the treatment of burned patients has demonstrated that mortality is not related to the toxic biologic effect of thermally killed skin, but to the metabolic and bacterial consequences of a large open wound, depletion of patients host resistance and extensive malnutrition (Burke 1978). Antibiotics and topical therapy act only as adjuncts to more definitive therapy; they do not solve the problem presented by large open wounds and related protein and calorie deprivation.

A more rational approach to the problem of extensive skin destruction secondary to burns appears to be rapid excision of devitalised tissue and immediate closure of the wound following excision. In extensive burns, the problems of wound closure become increasingly complex due to lack of available autograft and the rejection, in a short period of time, of temporary allograft. A key addition to treatment of these patients is the use of immunosuppression, which extends the period before rejection takes place and makes it possible to proceed with prompt excision of dead tissue and immediate wound closure. Furthermore it gives time for the areas

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previously harvested for reharvesting (Burke 1978, Rudowski 1979).

Primary wound excision of deep dermal and full thickness burns utilises two surgical techniques namely fascial excision and sequential eschar excision. (Rudowiski 1979). Facial excision involves excision of the entire thickness of the destroyed skin and subcutaneous tissue down to the level of the deep fascia. If deep thermal injury extends into the area below the deep fascia, debridement must remove all the devitalised tissue including muscle. This is followed by autografting during the same operative procedure.

Sequential eschar excision is employed when the burn injury is a combination of deep dermal and full thickness injury without extensive involvement of the subcutaneous tissue. The burn eschar is excised using the blair knife or an electric dermatome in sequential layers until healthy tissue is reached as evidenced by free bleeding. Haemostasis is maintained followed by immediate autografting.

The grafting procedure employed is mainly autografting using split thickness skin grafting or meshed split skin autografts which allows coverage of a large area by a given amount of skin. Following excision of large deep burns (35% to 65% of body surface) it is not possible to close the wound entirely with autografts

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even if meshed. In such circumstances viable cadava allografts are used to supplement autograft and provide closure. By the time the allografts begin to be rejected the previous donor sites are ready for reharvesting.

Skin grafts from cadavers involves many legal and practical difficulties. These problems have now been overcome through the commercial production of porcine cutaneous Zenograft which is widely used todate and is produced by Ethicone by the name of Corenthium (Rudowski, 1979, Capperauld 1978). In our circumstances the regime of primary excision and wound closure is not practiced because of technical difficulties. What we do is after treating the burn wound by exposure or occlussive dressings, early physiotherapy is encouraged especially when the burn is across a joint and temporary splinting. The eschar which remains tightly adherent to the underlying subcutaneous tissue is dressed with eusol or saline soaks so as to soften it and facilitate easy removal. Sometimes the eschar is excised under anaesthesia but not grafted immediately. When the wound is clean and covered with healthy granulation tissue, it is then autografted at a single sitting or as a staged procedure if the burn wound is so extensive. Allografting or the use of xenografts is not practiced.

PROGNOSIS

The prognosis of a patient with a burn depends most

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on the extent of the surface area involved. Most patients if unattended die within the first 48 hours of hypovolaemic shock and its sequelae. If the patient survives the shock period then the next danger is infection which also increases the nutritional demands resulting in acute starvation.

Four patients in our series died of hypovolaemic shock and infection. (Table III). All of these patients had burn of more than 35% of total body surface area. Most of our patients reported to hospital within the first 24 hours post burn injury. This is encouraging in the sense that fluid replacement therapy can be instituted immediately before shock has set in. This minimises the sequelae of shock.

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RUPTURE OF THE SPLEEN

SUMMARY

During the period of two years starting from July, 1983 to June, 1985, Surgical Firm III one of the three surgical firms in the Muhimbili Medical Centre attended ten patients with rupture of the spleen.

Six of these patients were males and the remaining four were females. The majority of patients fell in the age group of 21 to 30 years. The cause of the rupture in all these patients was trauma, 5 patients being due to motor accidents, 3 patients due to falls of various kinds and 2 patients due to penetrating abdominal injuries.

The commonest symptoms presented by all these patients was abdominal pain which was presented by 9 patients, four patients complained of pain in their left shoulder (Kehr's sign) apart from the abdominal pain. The signs elicited in most of these patients were abdominal distension in 8 patients, pallor in seven patients and evidence of free fluid in the peritoneal cavity in six patients as exemplified by a positive shifting dullness. 8 patients presented with features of peritonitis of which 3 patients was generalised and 5 patients was localised mostly on the left upper quadrant. Abdominal paracentesis was positive in 9 patients.

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Splenectomy was the surgical treatment of choice in 9 patients and one underwent surgical conservation of the spleen (splenorrhaphy) after it was found to have been stabbed anteriorly only.

At operation seven patients were found to have parenchymatous tear, fragmentation of the spleen in two patients, avulsion of the splenic pedicle in one patient and a subcapsular haematoma which subsequently gave way in one patient.

Autotransfusion was performed in seven patients. One patient died and this one had multiple injuries which included an extensive hepatic tear and left sided pneumothorax.

One typical case managed by the author is presented followed by a discussion relevant to the subject.

INTRODUCTION

Abdominal trauma continues to account for a large number of trauma related injuries and deaths. Motor vehicle accidents and urban voilence, respectively are the leading causes of blunt abdominal trauma and penetrating trauma to the abdomen in our environment.

Adekunle and Ajayi (1977) reviewing 75 cases of abdominal injuries treated at the University College Hospital, Ibadan, Nigeria also found that approximately 60% of

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the cases between 1967 to 1975 were due to road traffic accidents.

Of all the abdominal viscera, the spleen is undoubtedly the most vulnerable, particularly following non-penetrating injuries. In patients who have sustained visceral injuries following such trauma, the spleen may be damaged in as many as 47.6% of cases (Wright & Prigot 1939).

Blunt abdominal injuries accounted for 1% of all trauma cases admitted at Muhimbili Hospital in 1971. Of the blunt injuries 3 had ruptured spleen and one a ruptured liver (Hiza, P.R. 1974).

Many preventable deaths due to trauma are a result of inadequately treated abdominal injuries. Advances in resuscitation, evaluation and surgical technique have increased the ability to salvage people with major abdominal injuries and minimize preventable deaths. (Anthony & Richard 1982).

CASE PRESENTATION

K.M. a 16 year old female, Mngindo by tribe residing at Mlandizi village in Coast Region was referred from Kibaha District Hospital on 11:10/84 at around 5.15pm. after having fallen down from a cashewnut tree at noon the same day. She fell on her abdomen and chest.

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From that time she experienced severe abdominal pains and chest pains which were slowly increasing in intensity as time went. She also experienced severe pains in her left shoulder which radiated to the medial aspect of the forearm worse on lying flat. She gave no history of loss of consciousness but gave a history of feeling dizziness and general body weakness.

She denied history of nausea or vomiting but volunteered a history of having opened her bowels that same morning. She gave no history of any urinary symptoms. Regarding her obstetric and gynaecological history, she had amenorrhoea of three months and has never been pregnant before.

On examination she was found to be in severe pains, conscious, very pale, sweating, not cyanotic and not dyspnoeic. He blood pressure was 70/40 mm of mercury supine position. Pulse rate of 120/per minute and thready, Respiratory rate of 26 per minute and a temperature of 37.4^{o}C axillary.

The abdomen was found to be slightly distended but moving with respiration, and no visible peristalsis could be seen. There was generalised guarding of the abdominal musculature more on the left upper quadrant and generalised tenderness throughout the abdomen more on the left upper quadrant with rebound tenderness.

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The abdomen was dull to percussion especially in the flanks with shifting dullness positive. Balances sign was not elicited. The bowel sounds were absent. The other systems were essentially normal. Vaginal and rectal examination did not reveal any significant findings.

A diagnosis of traumatic haemoperitoneum secondary to a ruptured viscus was thus entertained and emergency exploratory laparotomy was planned.

A blood sample for grouping and crossmatching was immediately taken, three units of whole blood requested and an infusion of normal saline immediately started at the rate of two litres per hour. A nasogastric tube and a urethral catheter were inserted, the patient cleaned, shaved and a consent for operation obtained.

The blood pressure was recorded every hour to monitor the progress and stability of the cardiovascular system. After one hour the patient was reassessed and found fit for operation and by this time the operating theatre was ready and we also had two units of whole blood at hand from the blood bank.

The patient was induced with thiopentone 250 mg., Atropine 1mg and flaxedil as a muscle relaxant 120 mg followed by intubation using a cuffed endotracheal tube. Later the

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patient was maintained using Oxygen, Nitrous Oxide and Halothane.

The abdomen was then thoroughly cleaned with Savlon, painted with weak iodine followed by spirit and then draped using sterile towels. The abdomen was then opened using a supraumbilical midline incision.

There was a massive haemoperitoneum, the blood appeared fresh and there were some clots in the left side covering the spleen and the greater omentum adhering to the spleen. Blood was then collected in sterile A.C.D. bottles and about three units collected for autotransfusion. The clots were removed to aid visualisation of the spleen which was found to be fragmented at the apex.

The left hand was passed into the abdomen and the splenic pedicle identified and grasped between the fingers and pressure applied so as to reduce the continuing haemorrhage. The right hand was then passed over the lateral surface of the spleen, between it and the diaphragm and the lieno-renal, phrenicolienal and the splenocolic ligaments divided and the spleen delivered through the wound.

The gastrosplenic ligament was then divided while the stomach was drawn medially by the assistants hand and the short gastric vessels divided between haemostats

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and ligated with silk 2/0. The tail of the pancreas was then mobilised away from the hilum of the pancreas and the hilar vessels, the artery and vein divided between haemostats and separately doubly ligated with silk 2/0 and the spleen removed.

The splenic fossa was then packed with warm saline packs and later inspected for any extrableeders which were coagulated with diarthermy. The remaining abdominal viscera were inspected for possible injury and found none. The uterus was found to be gravid about 12 weeks and the fallopian tubes and ovaries found normal.

The peritoneum was mopped dry and the wound closed in layers, prolene 2/0 interrupted to rectus sheath (linea alba) and peritoneum in one layer followed by silk 2/0 to skin. No drain was left to drain the splenic fossa.

Post operatively the patient was transfused the blood collected during the operation which started intra-operatively after ligating the hilar vessels and continued postoperatively together with the two units obtained from the blood bank. This was followed by intravenous fluids Normal Saline alternating with 5% Dextrose 3 litres in the first 24 hours. Pethidine 100 mg i/m 8 hourly was given for analgesia for the first 24 hours.

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Free nasogastric drainage with intermittent active aspiration was carried out and the amount collected charted. An antibiotic cover Procaine Penicillin Fortified 1.2 mega units was given daily for five days and an input/output chart kept.

The vital signs, blood pressure, pulse rate and respiration rate were recorded every hour until stable for the first 24 hours and thereafter were recorded every 4 hours. The temperature was recorded twice daily.

On the second postoperative day the patient developed vaginal bleeding which was painful and full of clots and ultimately aborted with retention of the placenta. The gynaecologists were consulted and evacuation performed on the same day.

The nasogastric tube was removed on the third day when it was found to drain very little and there was no abdominal distension. The bowel sounds were also present on the third day and the patient had passed flatus and so the patient was allowed to take fluids and light diet. The drip was also discontinued on the same day and the patient allowed to be ambulant.

She was able to take normal diet on the fourth day, stitches removed on the 8th day and was discharged home on the 9th day to be followed up in the outpatient

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clinic after two weeks.

At two weeks the patient was seen at the surgical outpatient clinic with no complaints and the wound had healed well. She was later seen after one month and discharged from the clinic.

ANALYSIS OF THE TEN PATIENTS MANAGED BY THE AUTHOR:

Table I : The sex distribution of 10 patients with ruptured spleen.

Sex	No. of Patients
Males	6
Females	4
Total	10

Male : Female ratio - 3:2

Table II: The age distribution of 10 patients with Ruptured spleen during July, 1983 - June, 1985.

Age group (years)	No. of Patients
11 - 20	3
21 - 30	6
31 - 40	1
41 - 50	0
Over 50	0
Total	10

NB: The highest incidence was between 21 - 30 years.

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Table III : The cause of injury in 10 patients with Ruptured spleen during July 1983 to June, 1985.

Cause of Injury	No. of Patients
Trauma - Motor accident - Assault (stabwounds) - Falls - Sports	5 2 3 0
Spontaneous	0
T O T A L	10

Motor accident was the cause of injury in 5 patients.

Table IV(a) : The presenting symptoms in the 10 patients with Rupture of the spleen during July 1983 - June, 1985.

Symptom	No. of Patients
Abdominal pain : - Generalised - Localised left upper side abdomen	 5 4
Chest pain	1
Left shoulder pain	4
Dizziness	3
Shock	3
General body weakness	1
Breathlessness	3
Convulsions	1

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NB: Abdominal pain is the commonest symptom in 9 patients.

Table IV(b) : The presenting signs in the 10 patients with Rupture of spleen during July 1983 to June, 1985.

Sign	No. of Patients
Pallor	7
Abdominal distension	8
Tenderness - Diffise	3
- Localised left side	5
Shifting dullness positive	6
Positive Kehr's sign	4

The chief symptoms were abdominal distension and pallor.

Table V: Operative findings in the 10 patients with Ruptured spleen - June 1983 - July, 1985.

Type of injury	No. of Patients
Parenchymatous tear	7
Fragmentation	1
Pedical ovulsion	2
Subcapsular Haematoma	1

Note : (1) One patient had more than one type of injury.

(2) Parenchymatous tear is the commonest finding.

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Table VI : Associated injuries in the 10 patients with Ruptured spleen June 1983 - July 1985.

Associated Injury	No. of Patients
Fractured ribs	1
Liver tear	1
Torn mesentery / intestinal tear	2
Fracture of pelvis	1
Pneumothorax	1

Torn mesentery and intestine was seen in two patients.

Table VII: Post operative complications in the 10 patients with Rupture of the Spleen July 1983 - June 1985.

Complication	No. of Patients
Wound sepsis	2
Fever	1
Prolonged palalytic ileus	2
Peritonitis	1

Wound sepsis and prolonged paralytic ileus are the commonest post operative complications.

D I S C U S S I O N

AETIOLOGY OF SPLENIC RUPTURE

The aetiology of rupture of the spleen falls under four major headings which include the following:-

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Blunt abdominal trauma (non penetrating trauma), Transabdominal or transthoracic penetrating trauma, operative trauma (Iatrogenic) and Spontaneous rupture.

The spleen alone or in combination with other viscera is the most frequently injured organ following blunt trauma to the abdomen or lower cage (Anthony & Richard 1982), Foster R.P. (1970) Wright, L.T. & Prigot, A. 1939.)

In this study it is seen that five patients sustained blunt abdominal trauma from road traffic accidents. This corresponds to the study done by Adekunle and Ajayi in Ibadan where they found that more than 50% of the cases of rupture of the spleen were due to blunt abdominal trauma.

The commonest cause of blunt abdominal trauma from this study is Road Traffic Accidents followed by falls of various kinds. Lieberman and Stuart (1968) also found out that 80% of the 72 patients studied with rupture of the spleen sustained blunt abdominal trauma resulting from automobile accidents.

Splenic rupture which follows blunt abdominal trauma is an isolated event in only 30% of patients (Schwartz 1974). The other organs which may be injured in decreasing order of incidence include the chest (rib fractured), kidney, spinal cord fractures, liver, lung,

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craniocerebral structures, small intestine, large intestine, the pancreas and stomach. Associated injuries in our study can be seen in table vi.

Transabdominal or transthoracic penetrating trauma was seen in only 2 patients in this study and mostly resulted from assaults (table III). The most common inflicting agent is a sharp object such as a knife (Ekwueme, O. 1974), Anthony and Richard, (1982). Stabwounds to the abdomen have a 30-40 percentage incidence of visceral injury and gunshot wounds have an 80-90 percentage chance of such injury (Anthony & Richard, 1982).

Operative trauma to the spleen most commonly occurs during operations on adjacent viscera. In a recent review, Brown and his associates have noted that the spleen was injured in about 2% of patients whose operations involved viscera of the left upper quadrant such as hiatal herniorrhaphy, gastric resection and vagotomy and pyloroplasty. In our study no case of rupture of the spleen was secondary to operative trauma.

Although spontaneous rupture is very rare, it is a more common event when the spleen is involved with a haematological disorder. It is likely that the majority of patients classified as experiencing spontaneous rupture had trauma which was not appreciated (Schwartz 1974, Adekunle & Ajayi 1977).

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Spontaneous rupture or rupture associated with minor trauma is the most common cause of death in patients with infectious mononucleosis, which is second to malaria as a cause of splenic rupture.

Splenic rupture has also been reported in patients with sarcoidosis, acute and chronic leukaemia, haemolytic anaemia, congestive splenomegally and polycythemia vera.

None of the patients in this study had spontaneous rupture of the spleen though it has been documented in this area.

PATHOLOGY

Splenic rupture may be acute, delayed or ocult. Acute rupture is attended by immediate intraperitoneal bleeding and occurs in 90% of the cases of blunt trauma to the spleen.

Delayed haemorrhage from the spleen is not an uncommon syndrome and it may well be encountered more frequently in the future (Foster R.P. - 1970, Olsen W.R. 1977). It is either a subcapsular haemorrhage or a renewed haemorrhage from a laceration after initial tamponade by the omentum or surrounding structures. It gives rise to signs and symptoms after an arbitrarily selected period of 48 hours to several weeks and is reported in about 10% of cases sustaining blunt trauma, (Schwartz 1974, Foster R.P. 1970 and Willox G.L. 1965).

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Delayed haemorrhage has a higher mortality than simple uncomplicated rupture of the spleen and is due to difficulty and subsequent delay in making a diagnosis. The quiesent period is called latent period of Baudet and persists for less than 7 days in half the patients and less than 2 weeks in three quarters of the patients and this is probably related to a temporary temponade of a minor laceration or the presence of a slowly enlarging subcapsular haematoma which eventually ruptures. The Latent period of Baudet occurs in 15%-20% of splenic injuries.

Balance in 1898 discussed the pathogenesis of blunt spenic injury and the reason that some patients do not bleed rapidly. He states that the bleeding at any rate will be arrested by the formation of a clot in the ruptures and around the organ.

The latent period attributed to Baudet was expressed by Balance as to be followed by secondary haemorrhage rather than delayed rupture. Confusion and debate over delayed rupture versus secondary haemorrhage has continued since that time.

In 1932 McIndoe collected 45 cases from the literature, added one of his own, and published his classic study on delayed splenic haemorrhage. He found that one half of the patients with late haemorrhage had micro-scopic evidence that intrasplenic bleeding

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had occurred before capsular rupture. The remainder had frank capsular rupture from the onset, with a contained perisplenic haematoma.

In either case, he stated that the latent period was due to cessation of bleeding and was terminated by secondary haemorrhage. This has also been documented by Zabinski and Harkins in 1943. Clark et al have recently reported a well documented case that ruptured 5 years after injury.

A small number of patients survive major rupture of the spleen which is undiagnosed and untreated, an entity which has recently been recognised as Occult rupture of the spleen (Drapanas, Yates, Brickman & Wholey 1969, Schwartz, 1974). This term is applied to traumatic pseudocysts of the spleen and appear in less than 1% of patients sustaining trauma and are generally caused by organisation of an intrasplenic or parasplenic haematomas.

Clinical presentation and diagnosis

Signs and symptoms vary according to the severity and rapidity of intraabdominal haemorrhage, the presence of other organ injuries and the interval between injury and examination.

There is always a history of trauma to the abdomen or chest though sometimes it could be so trivial that the

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patient could not remember. In this study all of our patients gave history of trauma (Table III).

Majority of patients present with some degree of hypovolaemia and tachycardia is always present. A slight reduction in the blood pressure is also characteristic. Some of the patients with head injury may present to hospital unconscious making the diagnosis a difficulty.

Campbell and Whitfield (1956) stated that "as an absolute rule the presence of shock following head injury is indicative of injury elsewhere until it is proved that it is not". In our series 3 patients presented in a state of shock but none had head injury (Table IV(a).

Patients usually complain of generalized upper abdominal pain which in one third is localised in the left upper quadrant (Schwartz 1974). In our series 9 patients with abdominal pain of which in five patients it was generalised and four patients localised in the left upper quadrant.(Table IVa).

Pain at the tip of the shoulder (Kehr's sign) is evidence of diaphragmatic irritation but occurs in less than 50% of patients. It occurred in four patients in our study (Table IVb).

Pallor is a very frequent sign in any type of profuse haemorrhage and was detected in 7 patients in our study

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(Table IVb). Tenderness of the left upper quadrant or left flank is a frequent sign and was elicited in 5 patients (Table IVb).

A mass or a percussible area or fixed dullness in the left upper quadrant (Ballance's sign) secondary to subcapsular haematoma or omentum surrounding an extracapsular haematoma is rarely detected.

In a number of cases the diagnosis of rupture of spleen with its attendant massive haemorrhage is quite straight forward from the history of trauma followed by the important symptoms and signs.

Controversy exists regarding the difficulties presented in diagnosis of an intraabdominal injury by the many associated injuries only if blunt and open trauma are discussed together (Williams and Yurko 1966). Injuries to the head, chest and extremities often cause delay in diagnosis of an intraabdominal disorder and may thereby increase mortality.

The peripheral venous haematocrit is valuable in evaluating patients with intraabdominal haemorrhage. Serial documentation of a falling haematocrit after injury will point to active intraabdominal bleeding in the absence of other demonstrable sources of haemorrhage. The haemoglobin also tends to fall.

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The white blood cell count has been reported to be of value in the diagnosis of intraabdominal injury, especially in injuries of the spleen and liver. A few reports seriously challenge this concept of leucocyte count as a diagnostic aid (Shaftan G.W. 1960), Kleinst, Romeo 1961). No standard white blood cell count has been set which, when exceeded suggest abdominal visceral injury. If the leucocyte count is over 20,000 per cu mm it is assumed to imply intraabdominal bleeding. A higher percentage of patients with splenic and mesenteric injuries would have these values. Since a considerable number of patients with other visceral injuries have a leucocyte count of over 20,000 per cubic mm, the diagnostic value of leucocytosis becomes non specific.

The leucocyte count may reflect the degree of general trauma but otherwise has no specific diagnostic value. (Williams and Yurko 1960).

The need for a radiologic examination in acute abdominal injuries must always be measured against the general condition of the patient. If the patient is critically ill, Xray examination may be luxurious, which cannot be afforded without seriously compromising the patient's wellbeing (Kurkzman R. - 1979). It is often wiser to perform immediate surgery on a patient in shock resulting from a ruptured spleen or similar injury than to delay for the purpose of obtaining aesthetically

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pleasing but irrelevant arteriograms, but should be resuscitated first.

Plain films and regular contrast examinations can be of great value in detecting splenic injuries. The presence of any of the following should always raise the suspicion that the spleen is damaged. Fractures of the lower left ribs (especially 9th, 10th and 11th) posteriorly, elevation of the left leaf of the diaphragm, left pleural effusion, a large splenic shadow or a mass in the left upper quadrant displacing the stomach medially or the splenic flexure of the colon downwards, an enlarging splenic shadow, sentinel loops of distended bowel in the left upper quadrant (due to the irritating effect of the blood in that area), and Sawtoothed or irregular gastric rugae, particularly along the greater curvature of the stomach.

Nuclear imaging with 99m TC colloid can show an altered or irregular image or defect in the parenchyma. (Kurkzman R. 1979, Nebedar R.A. et al 1974). Ultrasound studies, though promising, are not reliable in most hands.

Angiography is probably the most sensitive means of detecting splenic injury and may demonstrate a space occupying mass, defects at the periphery, or actual leakage of angiographic contrast (Foster R.P. 1970, Nebedar R.A. et al 1974, Schwartz 1974, Kurkzman 1979, Love, Greenfield &Brayn 1968). Indeed arteriography may

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too sensitive, revealing small subcapsular haematoma the spleen which may lead to splenectomy in patients who would recover without incident if left alone.

In our situation most of these sophisticated radiographic techniques were not used with the exception of plain films.

Abdominal paracentesis was performed in all patients and was positive in 8 patients. None of our patients underwent peritoneal lavage.

Abdominal paracentesis has been a common practice, but its 80% or less accuracy and potential for visceral injury has led to its decreasing use. A positive tap is when there is a return of blood which does not clot in 50% of cases. A negative tap should be regarded as meaning nothing. Needle abdominal paracentesis is attended by a high number of false negative results. The introduction of four quadrant tapping reduced the frequency of false negatives but did not eliminate them (Gill, Champion Long & Jamaris 1975, Schwartz 1974, Olsen W.R. 1977, William & Yurko 1966).

Peritoneal lavage described by Root et al in 1965 is a simple procedure which could be performed in twenty minutes. It is a very sensitive procedure that even small amounts of haemoperitoneum could regularly be detectable (Olsen W.R. et al 1971, Gill, Champion,

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Long & Jamaris 1975). If anything (peritoneal lavage) its use in the management of trauma is more essential because of the detrimental effect of delay in treatment. A negative lavage indicates no significant haemoperitoneum.

TREATMENT.

Nine of the patients in our series underwent total splenectomy while one underwent surgical conservation of the spleen (splenorrhaphy).

In abdominal injuries, the concept of 'wait and see' has given way to the concept of 'open and look' because of the inevitable rise in morbidity and mortality with each passing hour (Adekunle and Ajayi 1977).

Before performing splenectomy as already described in the case report the patient should be resuscitated and evaluated as previously indicated.

Usually resuscitation and evaluation of traumatised patients should begin simultaneously. The extent and speed used to resuscitate a patient are individualised, depending upon the mechanism of injury, haemodynamic stability, neurologic status, associated injuries, and suspected potential for rapid deterioration (Anthony & Richard 1982). One large bore intravenous line and frequent physical examination and assessment of vital signs constitute the minimal treatment for any victim

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of trauma who has potential of having abdominal trauma.

Splenorrhaphy was done in one patient in our study and in this patient it was due to a penetrating knife injury which involved only the anterior aspect of the spleen.

Partial resection, non operative management and the preservation of accessory spleens are other alternatives to total splenectomy. All these procedures were not done in our patients.

Routine drainage of the splenic bed appears to increase the likelihood of subphrenic infection, drain tract infection, wound infection and fever of unknown origin presumed to be due to subphrenic inflammation (Cohn L.H. 1965, Robert, J.F. 1977).

The reasons cited for drainage of the splenic fossa are to prevent collection of pancreatic fluid when there is possible injury to the tail of the pancreas with resultant pancreatitis and abscess formation, and to prevent formation of haematoma especially following the removal of large spleens with vascularised adhesions, thus reducing the possibility of subphrenic abscess.

Even in the face of faecal contamination of the splenic bed, the benefit of drainage is uncertain, and it appears likely that meticulous haemostasis and thorough

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lavage is more effective in reducing the incidence of subphrenic infection (Robert J.F. 1977).

COMPLICATIONS

Very few patients in our series developed postoperative complications as seen in Table VII. Wound sepsis occurred in 2 patients, fever in 1 patient and prolonged paralytic ileus in 2 patients and this was in the patients who had associated injuries to intestine and mesentery. Peritonitis developed in the patient with torn intestine.

The question of whether there is an increased incidence of infection following splenectomy was first raised by Morris and Bullock in 1919, but the problem was not adequately addressed until King and Shumaker reported the development of meningitis and sepsis after splenectomy in 5 patients in 1952 (Trankle E.L. and Neu H.C. 1981).

Splenectomy done at any age and for any reason increases the risk for death due to overwhelming bacterial infection. The immune aberration following splenectomy includes decreased serum Igm levels, altered serum opsonic function and failure to respond to intravenous particulate antigen challenge (Kivit and Others 1979).

Constantoponlos and other workers (1973) found out

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that the activity of phagocytosis stimulating peptide, tuftsin, was decreased in the serum of 18 of 23 splenectomized patients. The peptide tuftsin is an intergral part of a specific cytophilic and globulin (leucokinin). Leucokinin coats the circulating polymorphonuclear cell and enables it to maintain a high level of phagocytosis. The mechanism of action of leucokinin on the polymorphonuclear cell rests fully on the presence of this peptide tuftsin.

Studies indicate that this peptide is synthesized in the spleen and that tuftsin activity is diminished or absent in splenectomized individuals who suffer overwhelming post splenectomy infections (Constantopoŭlos 1970, Kivit 1979). It is the current impression that the spleen plays an important role in combating infections.

The infecting organism most often recovered from patients with overwhelming post splenectomy infections is S. pneumoniae. In decreasing frequency, N. meningitidis, E. Coli, H.influenza and S.aureus.

In the prevention of overwhelming postsplenectomy infections should be techniques of splenic repair in cases of trauma, awareness of overwhelming postsplenectomy infection and the need of penicillin prophylaxis and pneumococcal vaccination. (Kivit 1979). Unfortunately this is not the practice in our situation because this

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syndrome has not received much attention.

Other complications following splenectomy reported in literature are persistent or recurrent haemorrhage, subphrenic abscess, left lower lobe atelectasis with a small pleural effusion and is quite a common occurance following splenectomy and accounts for early post-operative fever. (Goddon et al 1977).

Phlebothrombosis or thrombophlebitis can occur as a thrombotic complication. Splenic vein thrombosis is rare and usually occurs in the second post operative week.

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CARCINOMA OF THE URINARY BLADDER

SUMMARY

Sixteen patients with carcinoma of the urinary bladder were diagnosed and treated by firm III, one of the surgical firms at the Muhimbili Medical Centre from July, 1983 to June, 1985.

There were 12 males and 4 females giving a male/female ratio of 3:1. Fifteen patients were above 40 years of age with the highest number (six patients) in the fifth decade. Twelve patients presented with haematuria as the commonest symptom followed by hypogastric pain and frequency of micturation which was present in seven patients.

Six patients the tumour was located in the lateral or anterior wall of the bladder at cystoscopy, and this was followed by the posterior wall in three patients. 12 patients had T_3 and T_4 tumours at the time of presentation and the commonest histological type of the tumour was squamous cell in nine patients.

Ten patients were treated by palliative radiotherapy and four patients no treatment was offered because three died before treatment was begun and one was discharged on relatives request.

A typical case fully managed by the author is presented and the literature relevant to the subject discussed.

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INTRODUCTION

Carcinoma of the urinary bladder is a world wide disease. It is not uncommon in Africa where it is among the top three causes of malignancy.

The causal relationship between industrial carcinogens and transitional cell carcinoma was established by Rhenn in 1894 and a similar association between Schistosomiasis and Squamous Cell carcinoma has been postulated (Ferguson 1911). In our study nine patients had squamous cell carcinoma. Most of the patients in other studies (Eshleman 1966, Bowry 1974) show that there is a high prevalence of the disease in areas which are hyperendemic for schistosoma haematobium than in non-endemic areas supporting the causal association of Schistosoma haematobium and Carcinoma of the urinary bladder.

Haematuria however slight it may be is a symptom which should never be neglected even if Schistosoma ova is seen in urine. This is the common cause of late presentation in areas endemic for Schistosomiasis because most patients are treated as having Schistosomiasis. It is only when they fail to respond that they are referred for cystoscopy.

The treatment of carcinoma of the bladder takes into consideration the degree of tumour infiltration and the degree of tumour differentiation. Various methods are

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available but the problem lies in the selection of the best method to be given for a given case of bladder carcinoma (Jewet 1970).

For those countries, such as Tanzania and Egypt where Schistosoma haematobium is endemic, vigorous measures aimed at controlling and eventually eradicating schistosomiasis should materially reduce the incidence of bladder cancer (Kirei 1980).

CASE PRESENTATION

M.S. a 45 years Mshirazi male was admitted in our ward on 13/8/83 as a referral case from Morogoro with a history of lower abdominal pain and passage of blood in urine for the past ten months. The pain was dull in nature, localised and at times could feel it radiating to the back and right flank. There was no aggraviating or alleviating factors. At the same time he had been noting that his urine has been having blood especially at the end of micturation and about one week back he had noticed blood in the whole stream for one week and then stopped.

Associated with these symptoms was frequency in micturation more at night but later progressed to day time. There was also a history of dysuria of one week duration. The patient denied a history of having seen necrotic tissue in his urine and had never had any difficulty in micturation or total inability to void.

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He gave a history of feeling general body weakness, easy fatigability and dizziness for one month, no history of cough and has never been treated or suffered bilharziasis in childhood. With these problems he attended Morogoro Regional Hospital from where he was referred here.

On examination the patient was conscious, well oriented, not pale, wasted, and moderately dehydrated. He was not breathless or icteric. His blood pressure was 110/70 mm Hg, Pulse rate of 84 beats per minute and a respiration rate of 15 per minute. His temperature was 36.8°C.

The most significant findings were in the abdomen where there was a visible suprapubic mass of about 15 cm in diameter and intra-abdominal. It was not moving with respiration and the skin over it was normal. It was hard to touch, slightly mobile, and non tender. There was no evidence of free fluid in the abdomen, the renal angles were free and bowel sounds present.

A rectal examination revealed a hard non tender mass anteriorly and extending more to the right. The rest of the systems were normal.

A diagnosis of carcinoma of the urinary bladder was made and the patient investigated on those lines. A mid-morning urine sample was taken for ova, pus cells, red blood cells, specific gravity and casts. Another

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specimen was sent for culture and sensitivity and another for cystology. Blood samples were taken for a full blood picture, blood urea, serum creatinine and serum electrolytes and a chest Xray ordered. The patient was later booked for an intravenous urography after the above results were out and later was followed by cystoscopy.

Urinalysis showed proteinuria, many leucocytes and red blood cells. The culture was sterile and the cystology did not show any malignant cells. The blood picture showed anaemia of 8.2 gm/dl, and a lymphocytosis of 45%. The erythrocyte sedimentation rate was 52 mm/hour. Blood urea was 6.8 mmol/l, serum creatinine 62 umo/l. The serum electrolytes were normal. The chest Xray was normal.

The intravenous urogram showed moderate hydroureters and hydronephorosis of the right side with a large filling defect on the right side of the bladder. The left kidney and ureter were normal. There was no evidence of bladder calcification.

Cystoscopy was done on 24.8.83 and showed a large solid tumour occupying the right lateral side of the bladder and extending to the anterior aspect. It was surrounded by a zone of hyperemia which was easily bleeding. Two punch biosies were taken at the base of the tumour for histopathology. The bladder was

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emptied and palpated bimanually. The supra-pubic mass did not disappear with bladder emptying. Bimanually a hard mass was felt, it was extensive and was fixed to the surrounding structures but the rectum did seem to be infiltrated because the mucosa was tethered and moving slightly with the mass. This tumour was staged as T_4 N_oM_o. The histological results showed a well differentiated squamous cell carcinoma.

The patient was booked for tumour board discussion where it was advised that he be given a dose of palliative radiotherapy. A total of 4000 rads were given in 20 fractions in an overall time of 30 days. All treatments were given on cobalt machine and were well tolerated by the patient.

Check cystoscopy showed the tumour had shunk a bit but as far as the symptoms were concerned they were well palliated. The patient was discharged back to Morogoro though he still had slight suprapubic pains but no haematuria and the mass was only palpable on deep palpation. He was scheduled to come for follow up after three months but did not turn up.

ANALYSIS OF THE 16 PATIENTS DIAGNOSED WITH CARCINOMA OF THE URINARY BLADDER.

1. MALE : FEMALE RATIO - 3:1

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Table I : The age distribution of the 16 patients with Carcinoma of the Urinary Bladder.

Age (years)	No. of Patients
Below 30	-
30 - 39	1
40 - 49	6
50 - 59	4
60 - 69	4
Over 70	1
T o t a l	16

The 5th decade had the highest number of patients.

Table II : The Symptoms/signs of the 16 patients with Carcinoma of the Urinary bladder.

Symptom/sign	No. of Pts.
Haematuria	12
Hypogastric pain	7
Frequency of micturation	7
Debris in urine	4
Dysuria	6
Hypogastric mass	5
History of bilharzia in past	2

The commonest symptom was Haematuria.

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Table III : The location of the tumour in the 16 patients with Carcinoma of the bladder by cystoscopy.

Location	No. of Patients
Whole bladder	2
Posterior wall	3
Base	1
Lateral/anterior wall	6
Trigone	1
Not known	3
T o t a l	16

The lateral and anterior wall the commonest site of tumour.

Table IV : The stage of Tumour in the 16 patients with Carcinoma of the bladder.

Stage	No. of Patients
T_{1s}	-
T_1	-
T_2	1
T_3	3
T_4	9
Not staged	3
T o t a l	16

12 patients had T_3 and T_4 Tumour.

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Table V : The histological type of the bladder tumour in the 16 patients.

Histology	No.of Pts.
Squamous cell	9
Transitional cell	2
Adenocarcinoma	1
Anaplastic	1
No result	3
T o t a l	16

NB: Squamous cell the commonest histological type.

Table VI : The type of treatment offered to the 16 patients with CARCINOMA of the bladder.

Type of treatment	No. of Patients
Curative Radiotherapy	-
Radiotherapy and partial cystectomy	1
Palliative radiotherapy	10
Chemotherapy and Radiotherapy	1
No treatment	4
T o t a l	16

Palliative radiotherapy was the mode of treatment in 10 patients.

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DISCUSSION

INCIDENCE AND DISTRIBUTION.

Neoplasms of the urinary bladder is a common problem in Africa and throughout the world. In Zambia it is the third commonest malignant tumour and comprises 9.7% of all cases registered in 1971 (Currethers 1976, Cairns 1979). In Kenya carcinoma of the urinary bladder is rare, unlike Malawi where it is the second commonest malignancy in women and the third in frequency for men (Lucas 1982). In Tanzania the incidence is not well documented but Eshleman working in Northern Tanzania reported that the disease constituted 5% of all malignant tumours in Shirati (Eshleman 1966). In Sudan the incidence constitutes 3.1% of all malignancy (Ibrahim 1977).

A causal relationship between Schistosoma haematobium and cancer of the urinary bladder has been suspected since the turn of the century (Ferguson 1911), but a true aetiological relationship remains unproven. Despite the high incidence of the squamous carcinoma in a relatively younger age group in areas of endemic schistosomiasis and the known tendency for schistosomiasis to induce squamous metaplasia of the bladder mucosa, sceptics hold that the association is possibly coincidental. No epidemiological proof of a causal link has yet emerged (Eshleman 1966, Kirei 1980, Elem 1983, Awori, 1983 and Phillip 1969).

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In Shirati, Eshleman (1966) found that these patients usually come from lowlands where approximately 60-80% of the population is infested by Schistosoma haematobium. And so one might expect to see more cases of bladder cancer tumours if schistosomiasis is a direct cause. In Kenya the distribution of Carcinoma of the bladder parallels with the areas which are hyperendemic for Schistosoma haematobium infection. The prevalence of the disease is high along the East Coast, which is a hyperendemic region for Schistosomiasis and lower in the non-endemic areas of Kenya, supporting the causal relationship of Schistosoma haematobium and carcinoma of the bladder. (Bowry 1974).

In Egypt the incidence of Carcinoma of the bladder is very high in areas endemic for Schistosomiasis where 20-25% of all malignant tumours are bladder cancer.

Squamous cell carcinoma is the commonest type associated with urinary schistosomiasis, but transitional and anaplastic types can also occur frequently (Bowry 1974). In the schistosomal areas it has been found that bladder cancer occurs a decade earlier than in non-schistosomal areas and the incidence increases with increase in infestation.

The commonest victims for bladder cancer are males. In our series the male:female ratio was 3:1. This is

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comparable with 3:1 (Kirei 1980) in Tanzania, 2.9:1 (Awori 1983) in Kenya, and 2.6:1 (Currenthers 1976) in Zambia. This ratio tend to vary a lot in different series up to 8:1. This could be explained as being an occupational hazard because men are more exposed in the fields than females.

The age group commonly affected is the fifth decade and above. The average age in Egypt is 46.3 years and in Iraq 73% of patients are between 40 - 60 years of age. In USA 80% of cases are above 50 years of age. Kirei (1980) found that most patients were between 45 and 55 years at diagnosis. In our series six patients were between 40 - 49 years and this comprised the majority. More than 90% of our patients were above 40 years of age. (Table I). This correlates with the other series mentioned above.

AETIOLOGY

The causal relationship between industrial carcinogens and transitional cell carcinoma of the bladder was established by Rhenn in 1894. He noted a high incidence of bladder cancer developing in patients working in Aniline dye industries. It turned out that it was not aniline which was responsible, but an intermediary product, 2-naphthylamine. Later another group of compounds of similar benzene - ring structures were indicated and these included 1-naphthylamine, benzidine and 4-aminophenol. 2-naphthylamine is metabolised in

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the liver into a potent carcinogen (Aminophenol) which is then conjugated by glucuronic acid and transported and excreted in urine. In the urinary bladder it is deconjugated and the carcinogen is released. (Blandy 1977).

The same compounds were used in chemical industries, dyeing, rubber moulding and rubber cable coverings. Up to 26% of exposed persons may develop cancer and in Britain this is a compensatable disease.

Tryptophan metabolites have also been implicated as possible carcinogens. Abul-Fadl and Khalafallah (1961) showed an increase in tryptophan metabolites in the urine of Egyptian patients with bladder cancer. For instance, 3-hydroxy-anthranilic acid was increased to twice the normal level in patients with schistosomiasis, but there was a four fold increase in those with bladder cancer. Similarly serotonin was increased three-fold and ten-fold respectively but other workers (Trout et al 1962) have failed to confirm this.

The association between carcinoma of the bladder and chronic retention due to urethral stricture is well recognised (Owor 1975). Weston (1961) in Jamaica showed that approximately 24% of patients carcinoma of the bladder had gonococcal stricture. Dodge (1962) in Uganda recovered 76 cases of carcinoma of the bladder

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and of these 38% had associated urethral strictures. It is interesting that this association is not found in retention due to benign prostatic enlargement.

Vesical irritation or urinary stasis as seen in Schistosomiasis or calculosis have been found to play a role in the aetiology of bladder cancer. Smoking and food additives such as nitrosamines have also been implicated. (Campbel and Harrison 1970).

PATHOLOGY

In industrialised countries about 60-70% of the tumours are of the transitional cell type while in the tropics and subtropics squamous cell carcinoma predominates. In our series, in 9 out of 16 patients the tumours were of the squamous celltype (Table V). This slightly correllates with other African series such as 64% (Kirei 1980) in Tanzania, 69% (Currenthers 1976) in Zambia, 66% (Ibrahim 1977) in Sudan. Other series from Zambia report a very high incidence of Squamous cell carcinoma such as 72% as reported by Elem (1983) and 75% by Cairns 1979. Awori (1983) reports the opposite in which 50% of the cases were transitional type as compared to 29% squamous cell type. Adenocarcinomas and Anaplastic types can also occur in bladder but are rare.

The sites of tumours in order of frequency are the lateral walls, base of the bladder anteriorly and

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posteriorly, the fundus and bladder neck. Bilharzial bladder cancer rarely arise from the trigone which is the commonest site of non-bilharzial tumours. In our series 6 out of 16 of all tumours were located on the lateral and anterior walls - (Table III).

Squamous cell tumours typically appear as solid, fungating or ulcerative and are broad based. 70% of transitional cell tumours appear as papillary though about 20% may be solid in appearance.

Any tumour in the bladder which is surrounded by an area of inflammation should be considered as malignant unless proved otherwise.

Tumours of the urinary bladder are graded according to the degree of anaplasia and this is done at histology (Wallace et al 1975). Grade 1 (G1) - these are well differentiated tumours and are said to have a good prognosis because they are least malignant. Grade II (G2) these are moderately well differentiated and are of medium grade malignancy. Grade III (G.3) these are poorly differentiated and are said to be high grade malignant tumours.

Staging of the tumours enables one to determine the treatment and prognosis of the patients. Staging is done at cystoscopy and bimanual examination under anaesthesia. Staging is done using the TNM

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classification of the UICC - 1974 (Wallace et al 1974) and is summarised as follows:-

TNM	BIOPSY	BIMANUAL PALPATION
Tis	Preinvasive Carcinoma (Carcinoma in situ)	Normal bladder.
TX	The minimum requirements to assess fully the extent of the primary tumour cannot be met.	
T_1	Tumour does not extend beyond lamina propria.	Small, mobile bladder mass. Not felt after resection of lesion.
T_2	Invasion into superficial muscle.	Mobile indurated bladder wall.
T_{3a}	Invasion of deep muscle only (full thickness involvement.)	Nodular indurated mobile mass in bladder bladder wall.
T_{3b}	Extension through bladder wall (Paravesical tissue involvement).	
T_{4a}	Invasion of the prostate, uterus, vagina or rectum.	Tumour fixed and invading the neighbouring structures.
T_{4b}	Fixation to the pelvic wall and/or inflitrating the abdominal wall.	

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The lymphnode involvement is classified as follows (according to Regional and Juxta-regional lymphnode involvement.)

Nx - minimum requirements to assess the lymphnodes cannot be met.

No - No lymphnode involved.

N1 - Single homolateral regional lymphnode involvement.

N2 - Contralateral or bilateral or multiple regional lymphnode involvement.

N3 - Fixed mass on the pelvic wall separate from the bladder.

N4 - Involvement of Juxta-regional nodes (iliac and paraaortic.)

The metastatic status is classified as follows:-

Mx - minimum requirement to assess the presence of distant metastasis cannot be met.

Mo - No evidence of metastasis.

M1 - Distant metastases present.

Mia - Occult metastases

Mib - Single metastases in a single organ.

Mic - Multiple metastases in a single organ.

Mid - Metastases in multiple organ sites.

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CLINICAL FEATURES AND DIAGNOSIS

Haematuria and Suprapubic pain are the most common symptoms. Haematuria is present in 80% of patients with bladder cancer (Blandy 1977). Most series report that haematuria is the cardinal feature such as Kirei (1980) and Cairns (1979). In our series it was the commonest symptom in 12 out of 16 patients. It is usually terminal but later becomes total in some patients. Patients from areas endemic for schistosomiasis report for treatment late because haematuria is taken for granted and are treated for schistosomiasis. It is only when they fail to respond that, with a bit of luck, patients are sent to a centre for cystoscopy (Kirei 1980).

Other symptoms are frequency and pain on voiding. It is very easy to jump to a conclusion that these patients merely have cystitis. Some patients present with pyuria and if it is sterile this ought to ring a warning bell.

Some patients might have noticed the passage of necrotic tissues in their urine. Incontinence, retention of urine and wasting are accompanied features.

Loin pains may be present when complications of urinary obstruction supervenes and in some they may be in a state of uraemia.

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Physical signs are very seldom helpful. Occasionally one will encounter an advanced case where there is a large fixed indurated mass to be felt either suprapubically, or on rectal examination, but this is in the exception. Ibrahim (1977) found out that 50% of his cases had a suprapubic mass at presentation. In our series only 5 out of 16 of our patients had a mass palpable in the suprapubic area. Diagnosis therefore depends on intelligent investigation of the subject.

Sterile pyuria, or a positive Papanicolau stain in the urine deposite should call for confirmatory cystoscopy (Blandy 1977). The urine may show haematuria (microscopic) or show infection. It has been suggested that better yields of urine cystology is by bladder washings rather than normal urine. Schistosoma ova when seen in urine, should be regarded as one differential of haematuria and further investigations instituted. The blood urea may rise if obstruction has supervened.

Intravenous urography may show a filling defect in the bladder, or it may have grown into the wall of the ureter and causing obstruction to the upper tract. In some instances a non functioning kidney may be demonstrated on the side of obstruction.

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Cystourethroscopy is the mainstay in the diagnosis of bladder carcinoma. At cystoscopy the surgeons discover the site, the number, the size and the appearance of the tumours. A biopsy, usually multiple should be taken from the base of the tumour and sent for histopathology. Bimanual examination when the bladder is empty helps in the determination of the depth to which the tumour has invaded.

A chest Xray is important in determining lung metastasis. Lymphography, liver scan and bone scan may be used to determine metastases in these areas but these investigations are expensive and not available in most underdeveloped hospitals. Computed Tomography and Ultrasonography determine the degree of muscle involvement.

T R E A T M E N T.

The management of patients with carcinoma of the urinary bladder depends on the stage and the degree of differentiation of the tumour. One of the most perplexing problems in urology is the selection of the best method of treatment for a given case of carcinoma of the bladder (Campbell and Harrison 1970). This is because these tumours vary so widely in potential curability that a workable, meaningful classification until recent years has remained elusive.

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Naturally, the best results follow early diagnosis and prompt adequate treatment, but in this early stage many tumours escape detection because of absent, few, or minimal symptoms (Hugh J. Jewet 1970).

Delay in diagnosis is probably the most important single factor that stands in the way of cure, inspite of the development of remarkable irradiation techniques and ultraradial surgery. This delay may be unavoidable, or the fault of the patient or the urologist especially when symptoms of haematuria or frequency occur late in the course of the disease. In our series 12 out of 16 of the patients presented late with T_3 and T_4 tumours (Table IV). All these patients were considered as having advanced disease and were treated by palliative radiotherapy.

For the purpose of simplicity treatment of Carcinoma of the bladder can be considered in two main groups.

(i) The treatment of non-invasive Carcinoma, invading no further than the lamina propria (T_{is} and T_1). This should also consider two groups according to the degree of anaplasia of the tumour - that is well differentiated and moderately differentiated on one side and the poorly differentiated on the other side.

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(ii) Invasive Carcinoma - (T_3 - T_4).

Non Invasive Carcinoma

T_{is}, T_1, G_1, G_2.

For non invasive carcinomas which are well differentiated or moderately differentiated, a variety of modes of treatments are available. Most of these tumours can be treated by cystodiathermy or transurethral resection of the tumours. These methods can be used on tumours even of considerable size. When bleeding renders further treatment hazardous, the operation can be stopped and a second attempt made ten days later. After this the patients should be followed up closely. If at three months the tumour has recurred then the procedure is repeated. If there is no recurrence the patients are seen again after three months.

Multiple and large tumours can be treated by using the Helmstein baloon distended inside the bladder to a pressure greater than the diastolic pressure, and for periods of time up to six hours. This balloon causes pressure necrosis of the tumours which can subsequently be removed by electrodiathermy or excision.

Intravesical chemotherapy has been reported to produce better results in some patients. Intraversical

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chemotherapy was first used in 1961 by Jones and Swinney in an attempt to reduce the recurrence rate in patients with multiple tumours and also as prophylactic therapy (MacFarlane et al 1985). Thiotepa has been used but later was abandoned because it was too toxic. Other drugs currently in use are Epodyl and Adriamycin. Recently, McFarlane (1985) has used Mitomycin C in patients with primary or recurrent multifocal transitional cell carcinoma of bladder which could no longer be readily controlled endoscopically. His response rate was 70% in all cases.

Total cystectomy is reserved only as a last option when all the above measures have failed.

T_{is} T_1 G_3

In non invasive and poorly differentiated tumours of the bladder the treatment follows the same lines as previously described for the well differentiated and moderately differentiated. These patients are then closely followed up and if the tumours keep on recurring then total cystectomy is advised. It has been noted that the initial presence of multiple tumours or if they keep on recurring is associated with a significantly greater tendency to progress (Heney et al 1982).

If the patient refuses cystectomy then cystodiarthemy

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and radiotherapy should be alternatives. External radiation can cause regression, and at the same time side effects are minimal.

INVASIVE CARCINOMA (T_2 - T_4)

Invasive bladder cancers pose a very big challenge as far as treatment is concerned. Generally speaking the results of any modality of treatment is poor. Radiotherapy and cystectomy (radical) remains to be the two main treatment modalities at present. The overall five year survival rates for preoperative radiotherapy followed by cystectomy was 38% and that for radical radiotherapy was 29% (Bloom et al 1982). These two modalities of treatment are mainly used in T_2 and T_3 cases.

The aim of preoperative irradiation is to reduce tumour bulk and tumour cell viability by limited irradiation prior to surgery, thereby hoping to decrease the risk of local tumour spill and general dissemination at operation. The purpose of elective radical cystectomy is to remove residual and radioresistant tumour at the primary site and in regional lymphnodes. The reported five year survival rates following the combined approach has been encouraging 44% as compared with radiation (radical) followed by salvage cystectomy or alone in which it was 31%. (Bloom et at 1982). The combined treatment produces better results in younger

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patients, especially those aged less than 60 years. Partial fixation of the tumour to the pelvis is not considered a definite contraindication to cystectomy as provided there is a line of cleavage between the growth and pelvis removal of the bladder is possible (Currenthers 1976).

Recent evidence suggests that in the management of invasive bladder cancer, radical radiotherapy may be as effective as radiotherapy and cystectomy, thus avoiding the trauma risks and detriment to quality of life which follow cystectomy (Blandy 1980).

In most series it seems that the preoperative use of radiotherapy by fractional doses (2500-4000 rads) followed by radical cystectomy offers the best results (Whitmore 1978).

T_4 tumours are considered as very much advanced disease and are inoperable. All one can attempt here is to make the patient comfortable. This can be done by giving the patient a course of palliative radiotherapy. Radiotherapy can get rid of the local tumour and all its distressing symptoms for several years, and should be tried first. But in time it may be necessary to consider urinary diversion as a form of palliation (Kirk et al 1982). Kirk et al (1982) in his study of 19 patients who underwent palliative diversion without cystectomy for intractable symptoms concluded that there is a small

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number of patients in whom cystectomy is inappropriate, but who may benefit from diversion alone.

Combination chemotherapy used alone or in combination with Radiotherapy has also been used in the treatment of advanced carcinoma of the bladder with good results. Reported response rates using single drug or combination chemotherapy in advanced bladder tumours vary greatly (McGeorge et al 1982). A response rate of 50% using doxorubicin and cyclophosphamide has been recorded by Merrin et al (1975). A 56% response rate using methotrexate has also been reported by Turner et al (1977). Other drugs which are being used are Bleomycin, Fluorouracil and mitromycin, c. The drugs are used mainly as adjuvant chemotherapy to suppress micro-metastasis present at the time of primary local treatment (Hall et al 1982).

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URETHRAL STRICTURES

SUMMARY

A total of 26 patients with urethral strictures were seen and managed by firm III, Department of General Surgery at the Muhimbili Medical Centre from July, 1983 to June, 1985.

All of our patients were males, majority of them being in the third and fourth decades accounting for 50% of all patients. Acute retention of urine was the commonest mode of presentation in 16 patients (61.5%) and 22 patients (84.6%) were initially relieved of their obstruction by suprapubic catheterisation after failure of urethral catheterisation.

The commonest cause of strictures in our patients was post inflammatory occuring in 15 patients (57.7%) followed by trauma in 8 patients (30.8%). The commonest site of the stricture was the bulbar urethra in 10 patients (38.5%) followed by the membranous urethra in 8 patients (30.8%).

14 patients (53.8%) were managed by urethroplasty most often after failure of bouginage while the remaining strictures were amenable to intermittent dilatation at regular intervals.

One typical case which underwent a single stage

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urethroplasty is presented and a detailed account of the literature relevant to the subject discussed.

INTRODUCTION

Stricture of the urethra is one of the oldest problems in Urology and the earliest medical records abound with descriptions of its management by Catheters and dilators.

Urethral stricture is the result of cicatrization of the fibrous submucosal layer of the urethra and adjacent corpus spongiosum. The elastic fibres in these layers are replaced with fibrous tissue, which causes progressive concentric, eccentric, and longitudinal narrowing of the urethral lumen.

Post inflammatory strictures are usually always due to previous gonococcal urethritis, but owing to improved methods of treating the primary infection they are much less frequent than was the case three or four decades ago. (Singh & Blandy 1976). This is also a common problem in our environment where most of the causes of urethral stricture are post inflammatory infective in nature. Trauma to the perineum by direct voilence or from pelvic fractures is another major cause of strictures. With the increase in motor

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accidents the incidence of traumatic stricture is certainly expected to be high.

A question to be answered when considering the management of urethral stricture is when should we use a bougie and when ought we to consider urethroplasty? (Blandy 1976). It is only proper to start with less invasive procedures in straight forward cases and reserve invasive procedures to the difficult and complicated cases (Blandy 1976).

CASE PRESENTATION

A.A. a 24 years old Mrufiji was admitted in our ward on 16.9.84 at 5.00 p.m. with inability to pass urine for one day. This condition was associated with severe pains in the suprapubic region and distension. He could only pass a few drops of urine on straining. The problem started about three months back when he noticed that his stream was becoming weaker and had to strain in order to improve it. This was followed by frequency of micturation particularly during the night when he could go out for about five times to void and later experienced the same thing occuring during the day. Whenever he went to void he had to strain and the stream ends by dribbling. There was no history of dysuria, urgency or hesitancy. Furthermore, he had never seen blood in his stream of urine.

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He gives a history of having suffered urethritis about four years back which was successfully treated at the Agakhan Hospital in Dar es Salaam. There was no history of chronic illness in the family such as tuberculosis.

On examination he was found to be in pain, not pale clinically, not dyspnoeic and afebrile. His blood pressure, pulse rate, respiratory rate were all normal.

On examination of the genitalia, there were no perineal fistulas, no discharge through the urethra with a normal male external genitalia. Multiple indurations firm in consistency and non tender could be felt in the anterior urethra especially the distal half.

Abdominal examination revealed a suprapubic distended and tender bladder as the only positive finding. The rest of the systems were normal. A diagnosis of Urethral Strictures possibly inflammatory was made.

Attempts to relieve the obstruction by urethral catheterisation failed and a suprapubic cystostomy was done. Investigations done included a full blood picture, urine for routine examination, culture and sensitivity, blood urea and creatinine, an intravenous urogramm and a urethrogram. All the investigations were

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normal with the exception of the urethrogram which showed an extensive long stricture in the bulbous urethra starting from the external urethral meatus to about the midline. The patient was then scheduled for Orandi Urethroplasty.

On 5/11/84 the patient underwent a one stage Orandi Urethroplasty. With a probe in the urethra, the latter was laid open, cutting through the thick fibrous tissue. Haemostasis of the cut edges was achieved by cautery and by continuous suture of the corpus spongiosum with 3/0 chromic catgut.

An incision was made on one side of the cut edge parallel to it and mobilised. Using 4/0 dexon the lateral edge was stitched to the opposite edge of the corpus spongiosum. Finally the foreskin edges were approximated thus closing the wound using dexon 4/0. A nelaton catheter was left in situ to drain the bladder together with a suprapubic catheter. Post operatively the patient was given gentamicin 80 mg every eight hours for five days intramuscularly together with 500 mg. of chloramphenicol intramuscularly every 8 hours for five days. Pethidine 100 mg. intramuscularly every 8 hours was given for analgesia in the first 24 hours.

The post operative course was uneventful, except

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for slight sepsis of the wound which gradually cleared by continued sitz bath. The urethral catheter fell on its own accidentally on the fourth day postoperatively and was not reinserted. The suprapubic catheter was removed on the twelveth day, stitches removed on fifteenth day and the patient discharged after it was seen that he could void normally as before.

The patient was seen again at the Urology Clinic after one month and was voiding well and thus declared cured. He was never seen at the Urology clinic again though he was scheduled for follow up after three months.

ANALYSIS OF THE 26 PATIENTS WITH URETHRAL STRICTURE ATTENDED FROM JULY 1983 TO JUNE, 1985.

TABLE 1 : The age distribution of 26 patients with Urethral stricture:-

Age(yrs)	No. of Patients	Percentage
11 - 20	3	11.5
21 - 30	6	23.1
31 - 40	7	26.9
41 - 50	4	15.4
51 - 60	5	19.2
Over 60	1	3.9
Total	26	100.0

NB: Most of the patients (50%) are in the 3rd and 4th decades.

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TABLE II : The mode of presentation of 26 patients with Urethral Stricture.

Mode of Presentation	No. of Patients	%
Acute retention of urine	16	61.6
Chronic retention of urine	3	11.5
Straining at micturation	3	11.5
Perineal urinary fistula	4	15.4
T O T A L	26	100.0

NB: Commonest presentation in 61.6% of patients was acute retention of urine.

TABLE III : The initial method of relief of retention in the 26 patients.

Method of relief	No. of Patients	%
Urethral Catheterization	-	-
Suprapubic cystostomy	22	84.6
None	4	15.4
T O T A L	26	100.0

Most patients, 84.6% were initially relieved of their obstruction by suprapubic cystostomy.

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TABLE IV : The cause of stricture in the 26 patients with urethral stricture.

Cause of stricture	No. of Patients	%
Post inflammatory	15	57.7
Traumatic	8	30.8
Not known	3	11.5
T O T A L	26	100.0

Most of the Strictures 58.7% are inflammatory in origin.

TABLE V : The site of stricture by urethrogram of 26 patients.

Site of Stricture	No. of Patients	%
Penile	2	7.7
Bulbar	10	38.5
Membranous	8	30.8
Multiple	6	23.0
T O T A L	26	100.0

The commonest site was the bulbar urethra.

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TABLE VI : The mode of treatment of the 26 patients with urethral stricture.

Mode of treatment	No. of Patients	%
Bouginage only	12	46.2
Urethroplasty		
- First & Second stage	11	42.3
- One stage	3	11.5
T O T A L	26	100.0

NB: Most of the patients underwent bouginage only (46.2%) while those who underwent urethroplasty had a two stage procedure (42.3%).

DISCUSSION

AETIOLOGY:

The aetiology of strictures in the male can be grouped into congenital strictures, traumatic strictures, inflammatory strictures and malignant strictures.

CONGENITAL STRICTURES

The commonest causes of congenital strictures are congenital posterior urethral valves, congenital anterior valves and meatal stenosis. These cases were not met in our study because these patients usually

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under 10 years old are admitted in the paediatric surgical wards.

TRAUMATIC STRICTURES

Iatrogenic trauma heads the list and the largest proportion follows the use of an endoscope or a catheter (Blandy 1980). The introduction of an endoscope for cystoscopy or urethroscopy can cause lacerations which later leads to stricture formation. Catheters lying too long in the urethra tend to cause bed sores in the urethra in the congenitally narrowed areas such as at the external sphincter, the penoscrotal junction, and just inside the external meatus. The importance of only using narrow catheters cannot be overstressed (Blandy 1980).

The passage of urethral calculus, or perhaps the instrumental manouvre needed to remove it damages the lumen of the urethra with subsequent development of a stricture. Endoscopic diathermy for small tumours in the urethra will be followed by coagulative necrosis and scarring. Chemical inflammation, from formaldehyde used to sterilise catheters and bougies or injected into the urethra to treat gonorrhoea can cause the same sequency of events as endoscopic diathermy. Fortunately this is rare nowadays because of the improved methods of sterilising catheters and bougies

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together with the use of antibiotics in the treatment of gonorrhoea.

Apart from the iatrogenic causes of strictures, external trauma plays a very important role in the causation of urethral strictures. The commonest causes are direct voilence to the bulb from a blow on the perineum and injury to the membranous part from fractures of the pelvis. Other less common causes include penetrating and gun shot wounds and post prostatectomy trauma.

Nearly 80% of bulbous injuries develop strictures, compared with 65% if the injury was membranous (Harbison 1976). With complete rupture, all the bulbous injuries develop a stricture, against 70% if the site was membranous (Harbison, 1976, Janknegt 1975). Pelvic fractures are very notorious causes of urethral injury especially in the membranous portion. The urethra is damaged only when the pelvic ring is broken in such a way that the proximal fragment can carry with it the prostate while the distal fragment retains the bulbar urethra (Blandy 1980). The stricture usually arises between the gap of the disrupted ends of the urethra. If this gap is left to heal with fibrous tissue reaction it ultimately results in stricture formation (Lipsky & Hubner 1977).

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It is the initial management of all types of traumatic urethral injuries that determines the development and the severity of the stricture that follows. Controversy arises as to the correct management of these injuries in the early stages. Some surgeons advocate that when a urethral rupture is suspected, no attempt should be done to pass a catheter for fear it may make an incomplete laceration complete, and a simple fracture compound (McIlroy 1976). A suprapubic urinary drainage should be performed and allow definitive operation to be performed upon the urethra at a time when the local tissue haematoma and oedema has subsided. At this time the general condition of the patient is optimal for prolonged general anaesthesia.

This is the line of management followed by our firm in cases suspected as having urethral injury from trauma.

On the contrary, many other surgeons believe that bony deformities will necessitate a gap in the membranous urethra, but they feel that every effort should be exerted to make this gap as short and as well alligned as possible (Blandy 1980). Glass et al (1978) had 4 cases in which a stricture was entirely prevented and in 17 cases an easy urethroplasty done after the two ends of the urethra were approximated soon after injury.

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Most authors agree, that the best prevention of a stricture should be direct approximation of all layers of the ruptured ends, but primary anastomosis retro-pubic or perineal is, however, technically difficult or impossible (Janknegt, 1975).

INFLAMMATORY STRICTURES

There are two kinds of inflammatory stenoses, those consisting of a cross adhesion between the mucosal surfaces, and those which are made up of full thickness scar of the corpus spongiosum. The inflammation is obviously more often caused by gonococcal or non-specific urethritis than any other agents (Blandy 1980). In our series post inflammatory strictures accounted for 57.7% of the cases (Table IV). Bhandari et al (1983) found out that 95.3% of his cases were inflammatory in nature. The sites of predilecton for post-gonococcal stricture exactly reflect the richness with which the urethra is supplied with para urethral glands (Singh & Blandy 1976).

No glands are to be discovered in the membranous urethra, but the proximal bulbar urethra has a rich distribution of glands extending deeply into the corpus spongiosm, and are distributed circumferentially all round the urethra. The clinical features of the stricture may be delayed for many years, usually 3 to 10 years after the first attack of gonorrhoea.

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In elderly patients, these features of urinary obstruction caused by a stricture cannot be distinguished from those of prostatic enlargement. Often it is the complications of urethral stricture which bring the patient to hospital. Extravasation of urine and the development of perineal abscess is one very severe complication. This may end up by opening with multiple fistulas through which urine passes giving rise to the watering can perineum.

Other complications bringing the patient to hospital may be urinary fistulas which fail to heal because there is an untreated stricture downstream. Others are pocket lined with epithelium often containing calculi. The most dangerous complication is septicaemia especially in long standing hospitalised patients treated with broad spectrum antibiotics (Blandy 1980).

In strictures of longstanding there is shortening of the corpus spongiosum, which gives rise to a chronic chordee. A few patients have upper urinary tract damage so severe that they are uraemic, and some are hypertensive.

The most important signs are periurethral induration at the site of the stricture, a tender mass may be present if periurethral abscess has developed. A visible or palpable bladder may be found if urinary

retention has supervened (Smith 1978).

Clinical observation of the patient passing his water gives the observer more information than can be obtained from any electronic machine. However, the invaluable objective evidence of the flow rate provides a way of measuring and documenting the severity of the outflow obstruction which avoids bias and subjective error (Blandy 1980).

The traditional way of evaluating the calibre of the urethra with the bougie is still part of everyday urological practice. Using curved bougies of more traditional shape one can both judge the calibre of the stricture and dilate it. This can as well be done by passing a well lubricated urethral catheter which will be held up at the site of stricture or pass with some difficulty.

Laboratory studies are very helpful in detecting complications which are secondary to obstruction. If urinary infection is present, the white blood count may be elevated and bacteria will be found in urine and its sensitivity pattern known. The blood urea and serum creatinine may be raised if there is renal damage by backpressure (Smith 1978, Blandy 1976). Radiographic examination are also important to detect complications and to confirm the diagnosis. The urethrogram and

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voiding cystourethrogram are the most important diagnostic studies (Blandy 1976, Smith, 1978). ___ will reveal the site and degree of the stricture. Fistulas may be demonstrated together with false passages.

Viscous contrast medium should not be used for urethrography within 10 to 14 days of recent difficult bouginage, or the irritant contrast medium may be forced into the periurethral tissues and cause an inflammatory phlegmon. If a urethrogram is absolutely necessary, it should be made with a water soluble contrast.

The investigation of a patient with a stricture is incomplete without an intravenous urogram. Often coincident infection is accompanied by stenoses, diverticular or calculi in the upper tract. Hydroureters and hydronephrosis may be demonstrated.(Blandy 1980).

Urethroscopy is necessary to look into the urethra as far as it can go. If cancer is suspected it will permit a biopsy to be taken.

TREATMENT

Most urologists were brought to believe on the precept "Once a stricture always a stricture". Gentle dilatation of the urethra was the first practical lesson any

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urologist had to learn, but over the last decade techniques of urethroplasty have been devised so diverse and so adaptable that almost any stricture can now be offered a permanent cure (Blandy 1976).

The question which remains to be answered is when should one use a bougie and when one ought to consider urethroplasty?. The answer to this question is that, "It is only right and proper to start by being somewhat cautious in offering a new and untried operation to a patient who could be managed by regular and gentle dilatation, and offer urethroplasty in those patients in whom dilatation had to be done very frequently, or was followed by complications such as bacteremia and bleeding or was exceeding difficult to do. (Blandy 1976 a, b).

The treatment of urethral strictures mainly falls under three main headings namely dilatation, internal urethrotomy and urethroplasty.

DILATATION (Intermittent)

This was usually the first mode of treatment in our series and failure of this, the patient was offered urethroplasty either single stage or staged urethroplasty. 12 patients (46.2%) in our series were managed in this way without much difficulty (Table VI).

No matter how excellent are the results of urethroplasty

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the majority of strictures will best be managed by gentle and repeated bouginage (Blandy 1976). Down the centuries bougies have been made of every conceivable materials. Today the urological unit requires five sets of bougies, a complete set of curved polished steel bougies, a set of flexible bougies, made of plastic or gum-elastic, flexible filiform bougies with a screw fitting, into which can be attached curved steel followers and flexible plastic followers.

Bouginage can be done under local anaesthesia or general anaesthesia. Using metal bougies number 7 or 8 which are well lubricated try to guide the bougie down the urethra usually aided by gravity.

No attempt should be done to force it down. Smaller bougies should not be used for their tips may perforate the friable urethra at the site of the stricture (Smith 1978).

If the bougie passes through the stricture then dilate up to size 10. Avoid causing bleeding because this will predispose to a much stronger stricture.

In difficult strictures one can use filliform bougies, many at one time. Usually one will find its way through the stricture. This is then attached to a follower

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and the stricture gradually dilated. Alternatively one can use the dog-leg bougie to negotiate a difficult or tortuous stricture.

In straight forward dilatation a repeat dilatation should be performed every three months for one year, then every year throughout life. In difficult strictures this can be done at less frequent intervals such as weekly for one month, and then monthly for three months and then every three months for one year, and later every year throughout life. The frequency of dilatation is best judged by the Urologist himself performing dilatation depending on the difficulties encountered.

The complications associated with bouginage are bleeding, false passages, bacteremia, and some patients develop upper tract deterioration.

INTERNAL URETHROTOMY

Optical urethrotomy has shown to have made a significant contribution to the management of urethral strictures in Europe and North America where approximately 80% of patients are helped (Harrison 1984).

The cutting of strictures by internal urethrotomy dates from the early 19th century and the urethrotome

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designed by Otis in 1872 is still in use today virtually unchanged. Urethrotomy was seen then not as a means of curing a stricture, but enabling bouginage.

The incision is usually done using the cold knife at around 6 o'clock throughout the whole thickness of the stricture and extending proximally and distally in the healthy mucosa. The essential problem is how to stop the raw edges from sticking again. To prevent this some have left catheters for more or less prolonged periods, some have instilled steroids in the hope of preventing fibrosis and others have enjoyed a regime of autodilation on the patient (Blandy 1980, Lipsk et al 1977).

Follow up is done thereafter so that further treatment can be given if needed. Most recurrences occur within the first three to six months. A repeat urethrotomy done at an early stage is usually an easier procedure and should be performed at least two or three times before the method is abandoned for other treatment (Harrison 1984). Other authors among them Patterson et al (1978) have tried to diminish scar tissue formation after urethrotomy by primary skin grafting. They treated four patients and all grafts taken.

URETHROPLASTY

The most radical method in the treatment of urethral

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strictures is, of course, to seek a lasting result by reconstruction of the strictured portions of the urethra. (Johanson 1953). Many different ways of achieving this result have been tried.

The most common indications of urethroplasty are for socioeconomic reasons, difficult strictures, complicated strictures, in young men and in special strictures such as meatal stenosis.

Some strictures do not yield easily to gouginage and instead they seem to split and later heals as tight as before within 10 to 14 days. This is the rule in post amputation strictures, those following balanitis xerotica obliteranis and radiotherapy for carcinoma of the penis. The alternative is to try to split up the meatus (meatotomy). After meatotomy bouginage should be carried out for the first three weeks to make sure that the edges do not stick together.

Meatoplasty is done when meatotomy fails and the narrowing quickly returns and the diseased zone extends a centimeter or so down the urethra. Meatoplasty gives a more permanent cure (Blandy 1980).

Excision and End to End Anastomosis can be used for strictures in the penile urethra and bulbar and membranous strictures. It should be used for very

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short strictures not more than 1.5 cm. and should not be inflammatory in nature because the inflammatory process goes even in the pink area.

If the mobilised ends of the urethra are spatulated, and each end rotated through a right angle, it is possible to produce a long elliptical suture line with minimal risk of restenosis (Blandy 1976).

Lead Better and Lead Better (1962) referred to the idea of island skin implantation, but it was left to Orandi (1968) to publicise it.

Urethral strictures are commonly repaired by a two stage operations with an interval of three or four months between stages. Recent progress in urethral surgery has tended towards a one stage repair using either pedicle flaps or skin grafts. The use of a full thickness free skin graft for a single stage urethroplasty in stricture disease has been popularised by Devine and Horton (1976). This operation is indicated for strictures which were short, accessible and uncomplicated by false passage or fistulae.

If the plane of cleavage between dartos and skin is opened, a pedicle can be fashioned for a patch of scrotal skin which can be adapted to almost any

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urethral stricture and so permit a scrotal inlay urethroplasty to be performed in a single stage (Leadbetter and Leadbetter 1962, Orandi 1968, Blandy and Singh 1975). It is possible to make use of more than one patch, if the stricture seems to require this, and on occasion the patch could be rolled up into a complete tube and used to replace a length of urethra resected for tumour.

The objections to this procedure is the ingrowth of hair in the urethra since the scrotal skin is hair bearing. So hair balls may occur and have occured. This evil consequence has been reported as occuring in 4 of the 72 patients (5.5%) in Gardiners et al (1978) series. These hairballs are easily removed endoscopically. Restenosis is another problem but can be solved by revision of the procedure or undertaking a two stage procedure.

The single stage urethroplasty has its merits technically and financially as well as in an aesthetic sense. Since only one operation is necessary the patient is hospitalised only once. The interim, often long term care needed to prepare and maintain a first stage result for second stage closure is eliminated and perhaps most important from a psychological stand point, the patient maintains a normal posture when voiding after a single stage repair (Betz et al 1978).

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In our series this was carried upon on 3 patients (11.5%) (See table VI).

Multistaged procedures require much more total hospitalization time, repetitive anaesthetic administration, increased loss of time from employment and a waiting period between stages of 8 weeks or more (OLSSON & Krane 1978). It is mainly used in patients with long strictures, strictures with induration or severe scarring in the region of the perineum or urethra, or in patients with multiple fistulas or sinuses. It will be found that such patients might have undergone several trials of other procedures such as repeated dilatation or a one stage procedure which has failed.

In strictures of the anterior urethra the two-stage Swinney urethroplasty gives best results and is the operation of choice in complicated cases. The first stage consists of laying open the entire stricture with or without excision of the scarred spongiosum. When it has healed, a buried strip of skin is used to reconstitute the urethra as in the Denis Browne technique of hypospadias repair (Blandy 1976). An alternative to the Swinney second stage is to construct a tube for the urethra of full thickness skin. This leaves a larger defect, to be overcome by more generous dorsal slit relaxing incision, or by swinging up a flap of secrotal skin. This avoids the development of small

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fistulae along the suture line which tend to follow a skin strip reconstruction.

The scrotal flap urethroplasty is best used in strictures of the posterior urethra. The first of these was Johanson's technique (1953). Based on Swinney's idea, he folded back a funnel shaped flap of scrotal skin to enlarge the divided urethra. This was followed by a modification by Turner-Warwick's push-back operation. Blandy (1968) modified the operation which was also a modification of the Leadbetter (1960) technique.

The operation is started with a ∩ -shaped flap in the first place. This lead to the development of the Scrotal flap.operation in which the limbs of the ∩ inverted incision points to the Ischial tuberosity. The main advantage of the posteriorly based, scrotal flap is the good exposure it allows of the posterior urethra and verumontanum and the ease with which sutures may be put (Blandy 1976, Materu & Phillip 1980). A trans-sphincteric inlay is facilitated by a fibre-lit posterior speculum and a posterior urethral needle. In the absence of these instruments the proximal mucosa could be brought into view by introducing a large bougie from above through the suprapubic cystostomy. Stay sutures could then be

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placed to hold the mucosa while suturing the skin flap to the posterior urethra (Materu & Phillip 1980). The second stage is the same as Denis Browne's technique of hypospadia repair. It is important to examine the patient regularly before the second stage is done. If narrowing of the orifices occur, they should be repeated or adjusted.

Most of the patients in our series (42.3%) underwent a two staged urethroplasty though in most instances the first stage failed and had to be readjusted or dilated frequently. The final results in our series have been impressive.

The Badenoch's pull-through operation is used only when there is a very short posterior stricture, as it occurs after a partial dislocation of the prostate. It is possible to mobilise the posterior urethra, and attaching it to a catheter, to pull it up into the bladder. The Catheter and mobilised urethra are left in position for two weeks or so, until the catheter works loose, by which time the urethra has become healed in its new position.

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BENIGN PROSTATIC HYPERPLASIA

SUMMARY

Thirty five patients with benign prostatic hyperplasia were diagnosed and operated upon by firm III, one of the surgical firms at the Muhimbili Medical Centre from July, 1983 to June, 1985.

Twenty one patients (60%) were in the age group of 60 to 79 years which forms the majority. One patient (29%) was above 80 years. No patient was below 40 years of age. 23 patients (65.7%) presented with acute retention of urine and this was the commonest mode of presentation. Clinically 34 patients (97.1%) the prostate was smooth, firm and not nodular. 20 patients (57.1%) were initially relieved of their obstruction by urethral catheterisation, 7 patients (20.0%) by suprapubic catheterisation and 8 patients (22.9%) had no catheter insertion.

Transvesical prostatectomy was performed in 19 patients (54.2%) and the Millins prostatectomy in 16 patients (45.8%). Majority of the patients (28.6%) who underwent Millins prostatectomy had their catheters removed within the first week while 7 patients (20%) in the transvesical prostatectomy had their catheters removed within one week. The commonest post operative

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complications seen were wound infection and vesical cutaneous fistula which were seen in 8 patients (22.8%) who underwent Millins prostatectomy and in 11 patients who underwent Transvesical prostatectomy. Clot retention was only seen in 4 patients (11.4%) in the Transvesical prostatectomy group.

INTRODUCTION

The prostate was long recognised as a cause of outflow obstruction to the bladder, and a big prostate proved to be a common hazard in operations for stones in the bladder (Blandy 1976). Virtually every man over the age of 40 has some degree of benign nodular hyperplasia of the prostate, but only one in ten will get obstruction from it. (Blandy 1977). The size of the nodular enlargement has nothing to do with the degree of obstruction, for some of the smallest prostates are accompanied by some of the worst outflow obstruction, and many huge glands are unaccompanied by any obstruction at all.

With the progressive enlargement of the prostate, fairly discrete nodules that involve the middle and lateral lobes of the prostate progressively encroach upon the urethra to cause partial to sometimes virtually complete obstruction of urinary flow. (Robbin 1967).

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Suprapubic removal of the odenoma is most easy when the odenoma is large. When a gland is small and fibrous, and the bulk of the obstructing tissue is small, suprapubic enucleation is unsatisfactory.

Transvesical prostatectomy was first described by McGill of Leeds in 1887. Later efforts were developed to make the operation standard and safe (Freyer 1900). Later various authors developed the retropubic protatectomy (Millins 1947) and recommended it as a safe, aseptic, and rational operation. Transurethral resection was developed and became the operation of choice for all but the very largest prostates (Barnes 1943). Young (1903) devised his safe perineal enucleative operation.

Today, there are various selection of operations from which the surgeon may choose the one best adapted to the needs of the situation of the patient.

CASE PRESENTATION

M.F. a 50 years old Mpogoro was seen at the Surgical Outpatient Clinic on 18.7.84 as a referred case from Kisarawe district hospital. He had experienced inability to void urine for one day three weeks back, and had attended Kisarawe hospital where he was relieved of his problem by urethral catheterisation. The catheter was then left in situ and spigoted.

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The patient was well until two years ago when he started having frequency of micturation of more than ten times at night but each time he went to void the quantity was small. He gave a history of his stream being weak and had to wait for sometime before micturation starts. He had no pain during micturation, no blood or pus in urine but had urgency. Whenever he strains in an effort to improve his urinary stream the opposite occurs and sometime the stream stops.

Three weeks ago he had the first bout of failure to pass urine and was attended by urethral catheterisation. There was no history of cough, appetite was normal, bowel habits normal but gave a history of having lost some weight.

He was an old man in good general condition, not pale or cyanotic and there was no lymphadenopathy. The abdomen was normal. He had a urethral catheter in situ, no pus leaking around the catheter but had a right sided hydrocele.

Rectal examination revealed a moderately enlarged firm prostate, could reach above it, non nodular, the median groove was not clearly felt but the rectal mucosa was freely mobile over the prostate. There was no tenderness elicited and the examining glove was not blood stained. The rest of the systems were normal.

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The patient was diagnosed as having a benign prostatic hyperplasia and was immediately worked out for operation as an outpatient. The investigations requested included a full blood picture and erythronyte sedimentation rate, urine analysis, blood urea and creatinine, acid phosphatase and alkaline phosphatase and a chest Xray.

The results were as follows:- Haemoglobin 12g/dl, with 70% segmented neutrophils, 28% lymphocytes, 2% monocytes and an ESR of 10 mm per hour. The peripheral blood smear showed normochromic, normocytic red blood cells. The blood urea was 5.2 mmol/l, creatinine 54 umol/l, sodium 135 mmol/l, Potassium 3.9 mmol/l, chloride 98 mmol/l, Alkaline phosphatase 104 Iu/l and acid phosphatase 9 IU/l. The chest XRay was normal. With these results the patient was considered fit for operation.

With three units of Group A positive cross-matched blood the patient was taken to theatre on 3.8.84 for retropubic prostatectomy. The patient was given general anaesthesia, his abdomen cleaned with cetrimide and weak iodine and drapped with sterile towels.

The prostate was approached through an extraperitoneal subumbilical midline incision and the retropubic space opened and cleared of fat to expose the prostatic bed. All obvious bleeders encountered were arrested by coagulation. A self retaining retractor was then inserted. Two gauze packs were inserted one on each

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prostatic fossa. Two rows of stay sutures were applied transversely on the false capsule to act as haemostats to the prostatic venous plexus. Using a diarthermy the false capsule was opened in between the two rows of cutgat 1/0 stay sutures until the adenomatous tissue was reached.

A point of cleavage between the false capsule was identified and the self retaining retractor removed. Using the index finger the adenomatous tissue was carefully enucleated starting on the lateral aspects and the apex. The urethra was then divided at the apex using scissor and the prostate easily enucleated and removed from the prostatic bed and out of the field.

The self retaining retractor was reinserted, the raw prostatic bed packed with a hot rolled pack and left in place for five minutes. After five minutes the pack was removed, the prostatic bed retracted and the remaining obvious bleeders coagulated under vision.

A three way number 24 folleys catheter was inserted through the urethra, passed through the prostatic bed into the bladder and the baloon inflated by 30 mls of normal saline. Slight traction was applied on the catheter, and the prostatic incision closed using chromic cutgat 1/0 interrupted. Continuous bladder irrigation using normal saline was then started.

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The two gauze packs in the lateral prostatic fossa were removed, the self retaining catheter withdrawn and a corrugated rubber drain was left to drain the retropubic space and brought out through a separate stab wound. The wound was then closed in layers, prolene 2/0 to rectus sheath interrupted and silk 2/0 to skin. The patient received one unit of blood intraoperatively.

Post operatively the patient was given pethidine 100 mg intramuscularly every 6 hours for 24 hours. Intravenous fluids of normal saline and 5% Dextrose at the rate of three litres in 24 hours. Procaine Penicillin Fortified 1.2 ml intramuscularly was given daily for five days and this was combined with oral chloramphenicol 500 mg every 6 hours for 5 days. Continuous bladder irrigation with normal saline continued post operatively. The vital signs;blood pressure and pulse were monitored every hour in the first 24 hours.

The post operative course was uneventful, the irrigation fluid was clear on the second day and irrigation was stopped but continuous bladder drainage continued. The corrugated rubber drain was also removed, the intravenous drip stopped and the patient allowed to sit and start on oral sips. The urethral catheter was removed on the sixth post operative day and the stitches on the eighth day. His urinary stream

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was strong and was discharged on the same day and advised to attend surgical outpatient clinic after one month.

The patient was seen twice at the surgical clinic and in all the visits he had no urinary complaints and reported that his urinary stream was strong. He was thereafter discharged from the clinic.

ANALYSIS OF THE 35 PATIENTS DIAGNOSED AND OPERATED FOR BENIGN PROSTATIC HYPERPLASIA.

TABLE I : The age distribution of the 35 patients diagnosed and operated for Benign prostatic hyperplasia.

Age (yrs)	No. of patients	Percentage
Below 40	0	0
41 - 60	13	37.1
61 - 79	21	60.0
Over 80	1	2.9
T O T A L	35	100.0

Highest incidence in the age group 61-79 years.

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TABLE II : The mode of presentation in the 35 patients diagnosed and operated for Benign prostatic hyperplasia.

Mode of presentation	No. of pts.	%
Acute retention of urine	23	65.7
Chronic retention of urine	5	14.3
Symtoms of prostatism	7	20.0
T O T A L	35	100.0

Acute retention of urine was the commonest mode of presentation in 65.7% of cases.

TABLE III: The digital rectal findings in the 35 patients diagnosed and operated for benign prostatic hyperplasia.

Digital findings	No. of Patients	%
Smooth, firm, non nodular	34	97.1
Firm and nodular	1	2.9
Hard and nodular	0	0
T O T A L	35	100.0

97.1% of the prostate was smooth, firm and not nodular on rectal examination.

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TABLE IV : The initial mode of relief of urinary obstruction in the 35 patients with prostatic hyperplasia.

Mode of relief	No. of pts	%
Suprapubic cystostomy	7	20.0
Urethral catheterisation	20	57.1
No Catheter	8	22.9
Once only urethral catheterisation.	0	0
T O T A L	35	100.0

In most cases it was possible to pass a urethral catheter without difficulty.

TABLE V: The method of treatment in the 35 patients with benign prostatic hyperplasia.

Mode of treatment	No. of pts.	%
Millins prostatectomy	16	45.8
Transvesical prostatectomy	19	54.2
T O T A L	35	100.0

54.2% of the patients underwent Transvesical prostatectomy.

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TABLE VI : The post operative urethral catheterisation in the 35 patients operated for benign prostatic hyperplasia.

Days of urethral Catheterisation	No. of Patients			
	Millins	%	Trans vesical	%
1 - 7	10	28.6	7	20.0
8 - 14	5	14.3	8	22.8
Over 14	1	2.9	4	11.4
Total	16	45.8	19	54.2

10 patients (28.6%) who underwent Millins prostatectomy ahd 7 patients (20%) who underwent transvesica prostatectomy had their urethral catheter removed within one week.

TABLE VIII : The post operative complication of the 35 patients operated for benign prostatic hyperplasia.

Complication	No. of Patients		Total	%
	Millins	Trans-Vesical		
Haemorrhage (reactionary	-	2	2	5.7
Wound infection	5	6	11	28.6
Urinary retention (clot)	0	4	4	11.4
Vesicocutaneous fistula	3	5	8	22.8
Secondary haemorrhage	0	1	1	2.9
Pneumonia	1	0	1	2.9
Orchitis	0	2	2	5.7
Urethral Stricture	1	0	1	2.9
Persistent incontinence	1	1	2	5.7

The commonest complication in each group and overall are wound infection and vesicocutaneous fistula.

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DISCUSSION

INCIDENCE

The incidence of pathological benign prostatic hyperplasia is only 8% at the fourth decade; however, 50% of the male population has pathological benign prostatic hyperplasia when they are 51 - 60 years old (Berry et al 1984).

Benign prostatic hyperplasia has been recorded from all over the world though there are slight racial differences in incidence. In contrast to the prevalence of prostatic hyperplasia among Europeans and American Male populations is its rarity among Chinese, in whom Chang and Char observed an incidence of about 14% that of white male subjects (Mostofi 1970). Koreans, Japanese, Asiatic Indians, and Bantu also show fewer cases of prostatic hyperplasia. Comparing Asiatics and Africans, Movsas (1966) reported that prostatic hyperplasia was twice as common in Asiatics. Among eastern Mediterranean and North African population the incidence is high.

The American Negro is not only as prone to this as in the Caucasian but is said by Kahle and Beacham (1936) to develop it about a decade earlier. The difference in the incidence of hyperplasia of the prostate if it is actual, may be in part due to the fact that the Asiatic and African prostates are smaller

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and more firmly bound to the false capsule than the European.

Studying prostatic obstruction in Sudan, Kambal (1977) found that the disease to be commonest in Northern Sudan where most of its inhabitants are half castes of Arab African origin as compared to Southern Sudan where its inhabitants are purely Africans. Kambal (1977) attributed this difference as not being due to genetic factors but rather being to factors relating to diet and sexual habits.

The previous assumption that senile hyperplasia of the prostate is uncommon among Africans need certain qualifications because several authors have found it to be quite common among the African population (Kambal 1977, Materu, 1983, Malya, 1979).
In our series it is seen that the disease is not uncommon in our environment and the age distribution is the same as in other reported series (Table I).

AETIOLOGY:

The cause of nodular hyperplasia of the prostate is still unknown. Earlier opinion put forward in the nineteenth century include perverted sexual acts and imperfectly or neglected simple or bacterial posterior urethral inflammation as the principal causes. Abnormal

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functional activity of the testes and obstruction in urethral canal were also incriminated as possible principal causes. Of the many theories postulated for the aetiology of prostatic nodular hyperplasia, none has been accepted in the sense that generally recognized therapeutic or prophylactic methods have not been based upon them (Mostofi 1970).

Several theories todate have been put forward to try and explain the aetiology of nodular hyperplasia of the prostate. Among these theories are the following:-

(i) Theories based on constitutional, racial and social factors.

The prevalence of nodular hyperplasia among Europeans in contrast to its rareity in Chinese is a factor in support of this theory. Negroids were thought to have a low incidence but Kambal (1977) desputed this idea.

(ii) Neoplastic theory:

The question here basically evolves definition of neoplasia versus hyperplasia. Virchow (1870) thought that the disease was a true neoplasm and applied the term myoma to the stromal variety and adenoma to that of predominantly grandular structures. Other workers later found that

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the disease is more of a hyperplastic than neoplastic change.

(iii) Arteriosclerotic theory: This theory was formulated by Guyon in 1888 and has been defended since then. It was thought that the inner group of prostatic glands undergoes compensatory hypertrophy as a result of atrophy of the outer glands induced by arteriosclerosis (Moore 1943). Moore (1943) could find no histologic correlation between the degree of vascular sclerosis and the character of the ischemic changes in any part of the gland. In addition, Moore was unable to observe any correlation of prostatic atrophy or hyperplasia with the degree of arteriolar disease in other organs or with clinical hypertension.

(iv) Inflammatory theory: Chronic inflammation as a cause of nodular hyperplasia was first emphasized by Gechanowiski in 1901. Because there are almost always foci of lymphoid infiltration in hyperplastic glands and because the gland enlarges in acute and subacute inflammation, a cause and effect relationship was suggested between hyperplasia and chronic inflammation (Mostofi 1970).

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(v) Metabolic and Nutrition factors

Disturbances of general metabolism may possibly play a role in the development of prostatic hyperplasia but there is as yet no definite evidence to this effect. (Mostofi 1970). Vitamin A deficiency, hyperglycaemia, hypercholesterolaemia and low protein diet are some of several metabolic disorders associated with either a high or low incidence of benign prostatic hyperplasia.

(vi) Hormone theory

John Hunter in 1786 observed that the prostate, seminal vesicles, penis and other accessory organs depended for their existence on the testes, and that the prostate became atrophic after bilateral castration, but if one testis was intact, no atrophy resulted (Mostofi 1970). Because of the progressive increase in incidence of this disease with age, it is presumed that decreased testicular function may underlie nodular hyperplasia (Robbins 1976).

In senility, with progressive drop in androgen formation, there is a consequent relative hyperestrinism. Nodular hyperplasia may then be due to an absolute or relative hyperestrinism. It is believed today that nodular hyperplasia

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is a result of androgen/oestrogen ratio changes with oestrogen dominance.

PATHOLOGY

Benign prostatic hyperplasia affects the grandular and connective tissue in variable degree. The innermost and the periurethral group of glands are the sites of hyperplasia. The lateral and middle lobes are the commonest sites while the posterior lobe which is the site of carcinomatous change is not affected.

With the development of benign prostatic hyperplasia, there is a nodular proliferation within the tissues that lies proximal to the verumontanum and in close proximity to the cylindrical urinary sphincter in the region. (Berry et al 1984). The hyperplasia occurs in the stromal and epithelial tissue, and may occur alone or together.

As these nodules grow larger, they bulge inwards, compress the prostatic urethra from side to side into a sagittal cleft, and may bulge upwards through the ring of muscle at the neck of the bladder, which moulds them into the form of a rounded lobe (Blandy 1977). Surgeons by tradition talk of this as the middle lobe of the prostate, and the lateral bulges as "lateral lobes". They are however not real anatomical entities,

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but the distorted forms which anatomical limitations have forced the adenomas to adopt (Blandy 1977).

Characteristically, the hyperplasia is nodular in appearance and has been classified morphologically into five forms; stomal nodule, fibromuscular nodule, muscular nodule (uncommon), fibromuscular nodule and fibromyoadenomatous nodule (Berry et al 1984). Of the five types of nodules, the fibromyoadenomatous nodule is believed to be the most common type of large nodule removed surgically.

As the adenoma becomes larger it compresses the outer prostatic tissue into a false capsule. During the procedure of prostatectomy it is only the adenomatous tissue which is enucleated leaving behind the false capsule. This explains why prostatic cancer, new nodule formation, cyst formation, fibrosis and several varieties of hyperplàsia may be seen after prostatectomy (Blandy 1976).

CLINICAL FEATURES AND DIAGNOSIS

The clinical features in a patient with nodular hyperplasia are directly related to the pathological consequences of outflow obstruction.

Because of the outflow obstruction there is a poor stream. The patient may take a long time to pass urine. Most patients take long waiting before micturation

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starts. Straining in order to improve the stream usually results in further dimunition of the stream. As the obstruction gets worse the patient may go into acute retention of urine. Acute retention is a very dangerous complication, yet it still accounts for a high proportion of patients admitted with prostatic disease even today (Blandy 1976). In our series this was the commonest mode of presentation in 65.7% of patients (Table II).

Frequency of micturation is perhaps the most common presenting symptom of prostatic enlargement. This may be due to the presence of residual urine or irritation from infection or calculosis. This is often most prominent at night. When the bladder becomes really enormous, there is overflow incontinence, wet trousers, and lower abdominal distension.

Haematuria is a common symptom and always demands a full investigation to rule out a vesical neoplasm. Infection may result from urinary stasis and may present with severe cystitis, rigors, renal pain and tenderness or epididymitis. If there is uraemia and hydronephrosis there may be confusion, loss of appetite and loss of weight. Anaemia occurs from lack of erythropoietin production by the kidneys and failure of water and salt conservation leads to haemoconcentration, dehydration and acidosis.

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There are usually very few signs in a non-complicated case. There may be a distended bladder when acute or chronic retention has occurred. It is tender in the acute and non-tender in the chronic case. The renal angles may be tender when hydronephrosis with infection has supervened. A rectal examination will usually demonstrate an enlarged prostate. The enlarged prostate may be impossible to distinguish on rectal examination, before the patient is deeply anaesthetized, from a thick walled bladder containing some residual urine (Blandy 1977).

The aim of investigations in a patient with benign prostatic hyperplasia is to confirm the diagnosis, detection of complications of outflow obstruction and evaluation of the patients fitness for surgery.

Urine examination may be normal in asymptomatic cases of prostatism or may show the results of infection, such as alkaline reaction or the presence of red and white cells, albumin, or bacteria. Examination of the blood, both chemically and cellular is extremely valuable in these cases. Elevation of blood urea nitrogen and creatinine are indicative of renal damage, as a result of back pressure effects (Flocks 1971). Electrolyte changes in blood may be seen in these situations. The haemoglobin and red cell count may be low and may need correction before surgery.

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Intravenous urography and excretory cystography are very important examination in a patient suspected as having prostatic hyperplasia. This will reveal diverticula, give a rough but accurate measure of the volume of residual urine, detect stones in the bladder and often reveal a coexisting carcinoma, all of which may call for modification of the plan of operation (Blandy 1976). Hydroureters and hydronephrosis can also be detected.

It has been very difficult to subject all of our patients to this investigation in our circumstances because of the cost of the procedure and the increase in workload. Only specific cases where there was deterioration of the renal function as shown by the biochemical studies were subjected to this investigation.

Cystourethroscopic examination is also very helpful in accurately defining the pathological process that may be present. The extent of bladder trabeculation, cystitis, stone formation, diverticular and other lesions, such as bladder cancer, can be seen and properly evaluated (Flocks 1970). This procedure is also important when the method of prostatectomy is not transvesical.

TREATMENT

The treatment of a patient with nodular hyperplasia

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of the prostate causing obstruction to the urinary flow in our study was mainly surgical. If a patient presents with acute retention of urine an attempt is made to relieve the obstruction by urethral catheterisation using a small folleys cather number 14, 16 or 18 F.G. under aseptic conditions. Usually there is no difficulty in introducing the catheter if it is well lubricated in a patient with benign hyperplasia. If under all these circumstances introduction of the catheter fails it is better done when the patient is relaxed under general anaesthesia. Failure to pass a catheter will necessitate suprapubic caterisation. The catheter is left in situ and the patient prepared for operative treatment.

In our study 20 patients (57.1%) were initially relieved of their obstructive symptoms by urethral catheterisation and 7 patients (20%) by suprapubic catheterisation and in 8 patients (22.9%) no catheter was needed (Table IV).

Patients with chronic retention of urine also need bladder drainage so as to improve the renal function which is compromised by the effects of backpressure.

Ideally once the patient has been decompressed by catheterisation and the catheter left in situ, he should be prepared for operative treatment as soon as possible

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otherwise the complications of long standing urethral catheterization such as infection, calculous formation and the subsequent development of stricture may develop. Furthermore, an indwelling urethral catheter is a great psychological problem in most patients.

Because of an increased work load in our circumstances it is impossible to operate on all patients with benign prostatic hyperplasia early, and there are patients who have stayed with urethral catheters for more than six months and sometimes a year. In such patients it is advised that their catheters should be changed at least every month and urinary antiseptics given whenever there are signs of infection. The urine of a patient with a urethral catheter is never sterile. (Blandy 1976).

During this preparatory phase the patients should be fully investigated as outlined and the haematological and biomedical status brought to normal. This is achieved by the correction of anaemia, improving the renal function by catheterisation and treatment of any urinary infection. The patient is then screened for any other medical illnesses such as diabetes, asthma and other pulmonary infections, hypertension and other cardiovascular diseases. These should be controlled or treated accordingly. Most aged patients are usually found to be having an increased incidence of cardio-

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pulmonary diseases which affects very much the selection and outcome of any operative management (Mostofi 1976).

Definitive treatment of a patient with prostatic hyperplasia causing obstruction has been divided into three groups: permanent urinary diversion, medical treatment and surgical removal of the enlarged prostate.

Permanent urinary diversion is indicated in those patients with permanent severe irreversible renal damage (Beck 1970) in whom any other treatment is not possible and in patients with coexisting diseases which preclude surgical treatment such as severe cardiovascular insufficiency, respiratory failure, cerebral failure and locomotor failure (Miller 1965). No patient was offered this type of treatment in our series but it may explain the majority of old patients who are on follow up in the medical wards with medical diseases and those who have absconded treatment after the initial relief of the obstruction. Again most of these patients with severe medical illness were excluded from this study.

Various methods have been used medically in attempts to shrink down the prostate and relieve the obstruction. Various chemotherapeutic agents have been tried among them are hormones, sclerosing agents and neuroactive agents (Geller 1969, Caine 1976). None of our patients were treated in this way.

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The surgical removal of the enlarged prostate is the commonest current method of dealing with nodular hyperplasia of the prostate causing obstruction to the urinary flow. Ideally a cystoscopy performed immediately before the operation is a useful guide to the surgeon in the choice of operation to be done. Cystoscopy will enable the surgeon to make sure that there are no tumours, diverticular or stones in the bladder. The size of the prostate can also be ascertained by doing a bimanual examination (Blandy 1976). In general very large glands are better dealt with by an open enucleative procedure and small ones transurethrally.

Several approaches are available to the surgeons and these include Transversical, retropubic, transurethral and perineal prostatectomy.

In our series only Transvesical and retropubic approaches were done but the tendency in our firm which admits all the urological cases in the hospital is towards retropubic prostatectomy whenever feasible. (Table V). Prostatectomy by any route should accomplish the same aims, first to remove all the adenomatous nodules from the inner zone, and leave the surgical capsule stripped bare, and secondly, to divide and clear away the obstructing, hypertrophied and sclerotic fibromuscular tissue from the bladder neck.

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Transvesical Prostatectomy (Freyer's operation)

Among the oldest prostatectomies employed, this operative approach continues to be widely practiced. In its early history, mortality rates as high as 25 to 50% were recorded (Flanagan 1974). However, with better control of urinary infection, haemorrhage, and renal function, the operative mortality at present is acceptable.

The chief indication is extensive prostatic hyperplasia, presence of stones or tumours which need removal and the presence of a previous cystostomy. The chief advantage is that in properly selected cases it produces excellent long term post-operative results in voiding and urinary control and disturbs sexual capacity minimally. The chief disadvantage is the inability to control haemorrhage effectively. In addition to excessive blood loss, the presence of blood clots in the bladder and prostatic fossa in the early post operative period gives rise to severe pain and bladder spasm. Post-operatively continuous bladder irrigation is done using normal saline using an inflated three way folleys catheter, or by using two catheters one urethral and another suprapubically until the urine is clear.

Materu(1983)and Malya(1979) used this method in all their patients with good results. Kambal (1977) used this method in 403 patients and claimed good results. However, other authors think that transvesical prosta-tectomy is a hazardous operation which should not be

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decided upon lightly. Whilst the ultimate outcome is usually satisfactory it is a procedure which requires courage, vigilance and perseverance (Nevil and Loefler 1983).

In our study this operation was done on 19 patients (54.2%) and the post operative catheter insertion was not more than seven days in 7 out of 19 patients (20%) (Table V & VI).

RETROPUBIC PROSTATECTOMY

The operative approach to the prostate by the anterior exposure of the prostate located inferior and posterior to the symphysis is the most recently developed. The main advantage of this approach is the ideal exposure of the prostatic bed and vesical neck, which permits complete removal of obstructive tissue with precise control of bleeding and plastic treatment of the vesical neck (Blandy 1976,,Flanagan 1974). The major drawback of this approach is that it is accomplished through an extremely vascular field, it is difficult to execute on obese patients and in patients with deep narrow pelvis and it is occasionally complicated by postoperative infection in the prevesical space with possible osteitis pubis.

This operation was done in 16 of our patients (45.8%) and 10 patients had their urethral catheters removed within one week as compared to transvesical prostatectomy.

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We have found this procedure to be quite safe in our circumstances in properly selected patients because bleeding is less severe, the bladder irrigation being stopped within 3 to 5 days in most cases and the post operative complications have been very low (Table VI and VII).

There are many variations on the original technique described by Millin in 1948, among them is the technique of combined prostatectomy which was described by Council and Bourque (Firfer and others 1984). The term combined was first introduced by Leadbetter and associates in 1959 to suggest that the procedure was a modification of the suprapubic and retropubic approaches to prostatectomy.

In this technique the incision is longitudinal over the prostate and bladder so that the bladder can be easily inspected and any pathology in it dealt with accordingly. Haemostasis is easily established, and corrective procedures at the vesical neck are accomplished easily, additional suprapubic transversical drainage is unnecessary and closure of the prostatic urethra is simplified. Post operative urethral irrigation is short and sometimes for only 24 to 48 hours. (Firfer and Associates 1984).

Transurethral prostatectomy

This method is suitable for small and moderately enlarged

prostates, and has the advantages of its ability to be used in patients of poor operative status. It is widely practiced todate but it is one of the most difficult of all operations to master (Flanagan 1974).

Post operative urethral stricture is still a much too common complication of transurethral resection. Normally the length of hospital stay is generally shorter than with open method of prostatectomy, a factor to consider in these days of rising hospital costs and shortage of hospital beds. (Chitton 1978). None of our patients underwent this operation.

Perineal Prostatectomy

Although the oldest type of prostatectomy still in use, perineal prostatectomy and the perineal approach to the prostate has never gained wide acceptance because of such complications as post operative urinary incontinence and sexual impotence. The advantages are the direct vision excecution of the operation and the dependent post-operative drainage from the prostatic operative bed.

Loefler (1983) advocated the Gadhvi's prostatectomy because it was easy to perform, minimal blood loss and is suitable to high risk patients.

Complications:

All methods of prostatectomy share more or less the same types of complications, though these vary a little in

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severity and incidence from method to method.

In our study it was found that the transvesical approach of prostatectomy was attended by a high frequency of complications than the retropubic approach (Table VII). Wound infection and vesicocutaneous fistulas were among the leading complications in either approach.

The complications of prostatectomy could be grouped as early and late complications. Among the early complications are haemorrhage with or without clot retention, and wound infections, and late complications are urethral stricture, pulmonary embolism, fistulas, incontinence, infertility, recurrent adenoma, orchitis to mention a few.

Materu (1983) found that epididymoorchitis and clot retention were the commonest complications encountered in her series, while Mallya(1979)had wound infection as the commonest complication in his series. Nicoll (1978) found that epididymitis was not a common complication and recommended that routine vasectomy at prostatectomy was unnecessary as previously advocated by other authors (Nicoll 1978).

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CHRONIC OSTEOMYELITIS

SUMMARY

A total of eleven cases with chronic osteomyelitis were seen in the department of Orthopaedic/trauma at the Muhimbili Medical Centre during a period of three months between December, 1985 and February, 1986.

There were seven males and four females. The majority of patients (5 patients) were in the second decade of life. The tibia and femur were the commonest bones involved in which it was the site of infection in five and four patients respectively.

Ten patients presented with discharging sinuses and nine patients presented with swelling of the affected bone. Fever was not a constant feature and was only recorded in one patient.

The commonest source of infection was endogenous in origin in 8 patients followed by trauma in three patients. Staphylococcal aureus was the commonest organism isolated in 4 patients, while mixed infection was seen in three patients and these had the infection following compound fractures. Saucerisation was done in 9 patients and sequestrectomy in two patients.

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One typical case fully managed by the author is presented and the literature relevant to the subject discussed.

INTRODUCTION

Infection of osseous tissue by pyogenic organisms is termed pyogenic osteomyelitis. It occurs in acute and chronic forms, but subacute forms of osteomyelitis have been reported. Osteomyelitis constitutes an autonomous, pathological and clinically well defined entity. The pathophysiology and anatomical properties of the osseous tissue are of decisive importance for the occurance and the course of infections (Walenkamp 1983). These qualities also have important implications for the treatment of osteomyelitis and for the evaluation of their treatment.

Inspite of growing knowledge of their pathogenesis and treatment, skeletal infections continue to cause patients to suffer severely, treating surgeons to experience repeated frustrations and the cost of health care to rise substantially.

Notwithstanding the therapeutic possibilities offered by antibiotics, 15 to 30% of all cases of acute haematogenous Osteomyelitis turn into chronic osteomyelitis (Waldvogel 1970). Most skeletal infections necessitate several operations and as a rule they heal after many years of treatment. It is

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every surgeon's duty to do all he can to prevent infection, and it is a challenge to keep the proportion of postoperative infections at a minimum.

Chronic osteomyelitis in our environment seems to be a rampant problem though little work has been done to evaluate the magnitude of this problem in our country. The challenge offered in the treatment of this condition in our environment are great and the results are so disappointing.

CASE PRESENTATION

L.M. a 22 year old school boy was referred from Mwadui hospital in Shinyanga region on 3.12.85 with a long standing history of pain in his left shoulder and upper aspect of his arm. The pain was persistent and burning in nature and tended to spread throughout the entire left arm. This was later followed by occasional episodes of swelling of the upper part of the arm which usually responded to the administration of antibiotics. At one time the swelling was incised and a lot of pus was drained but the wound took time to heal and in the end a discharging sinus appeared which keeps on recurring from time to time. There was no history of trauma and there were no constitutional disturbances. The remaining systems were found to be normal.

When examined he was found to be a healthy young adult,

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not in pain, afebrile and not pale. His vital signs were normal. Examination of the left upper arm showed a swollen proximal third of the arm with a depressed scar on the lateral aspect presumably a closed up previous sinus. The skin of the arm was normal. Palpation revealed deep seated tenderness on a hard indurated bone and the affected area was warm. Active and passive movements at the shoulder and elbow joint were normal and non tender. The rest of the systems did not reveal any significant findings.

A diagnosis of chronic osteomyelitis was made and an anteroposterior and lateral view of humeral Xrays were ordered. This showed extensive rarefaction and expansion of the proximal third of the humerus. There was also a suspicious cavity which had a fluid level. A brodie abscess was suspected and the patient worked up for operation.

A sample of blood was taken for full blood picture and sugar and urine taken for routine analysis. On 6.1.86 the patient was taken for operation.

Under general anaesthesia the site was prepared by cleaning it with cetrimide followed by weak iodine and drapped using sterile towels. Through a lateral longitudinal incision the muscles were separated to expose the bone. A lot of pus was drained from the soft tissues and bone. A longitudinal window was made on the bone and a cavity

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filled with pus was encountered. A pus swab was taken for culture and sensitivity. The cavity was thoroughly curettaged and irrigated with normal saline. The wound was not closed and was left to drain and allowed to heal by second intension.

Post operatively the patient was kept on Cloxacillin 500 mg intravenously every 6 hours for five days. Pethidine 100 mg was given every 6 hours intramuscularly for the first 24 hours. The vital signs were monitored every hour in the first 24 hours.

The postoperative period was uneventful and the discharge of pus was diminishing and the wound closing up. The results of culture and sensitivity revealed Staphylococcus aureus sensitive to Cloxacillin and erythromycin and resistant to Tetracycline.

Since the young man is a student he was discharged back to Mwadui after five days and advised to be followed up at his nearest hospital.

ANALYSIS OF THE 11 PATIENTS WITH CHRONIC OSTEOMYELITIS

TABLE 1 : The age distribution of the 11 patients with Chronic Osteomyelitis.

Age (yrs)	No. of Patients
11 - 20	5
21 - 30	2
31 - 40	2
41 - 50	1
Over 50	1
Total	11

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Majority of the patients were in the age group of 11 - 20.

TABLE II : The site of infection in the 11 patients with chronic osteomyelitis.

Bone involved	No.of pts.
Femur	4
Tibia	5
Humerus	1
Phalanx	1
Total	11

The femur and tibia were the commonest bones involved.

TABLE III : The symptomatology of the 11 patients with chronic osteomyelitis.

Symptoms	No.of pts
Swelling	9
Pains	7
Sinus	10
Fever	1
Flexion deformity	4

Sinus and swelling were the commonest symptoms.

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TABLE IV : The aetiology of chronic osteomyelitis in the 11 patients.

Aetiology	No.of pts
Traumatic	3
Endogenous	8
T o t a l	11

The commonest source of infection was endogenous in nature.

TABLE V : The bacteriology of chronic osteomyelitis in the 11 patients.

Organism isolated	No.of pts.
Staphyloccocus aureus	4
Streptococcus pyogenes	2
Mixed growth (E.coli, and Proteus)	
(Staph. and Strep.)	3
Proteus	1
Pseudomonas	1
T o t a l	11

The commonest organism isolated is Staphylococcus aureus.

TABLE VI : The mode of treatment offered in the 11 patients with chronic osteomyelitis.

Mode of treatment	No. of patients
Saucerisation	9
Sequestrectomy	2
T o t a l	11

Most of our patients (9 patients) underwent saucerisation.

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D I S C U S S I O N

AETIOLOGY

Chronic osteomyelitis may occur secondary to inappropriate or inadequate therapy of the acute stage of the disease and in most cases it is a result of haematogenous spread from the onset of the disease. It can also occur secondary to trauma as in cases of compound fractures or after bone operations or any penetrating trauma. For this reason chronic osteomyelitis has been termed as being exogenous or endogenous in nature according to the way it is produced (Bonakdar pour 1984). Trivial trauma has been reported in up to 30% of patients with osteomyelitis (Waldvogel 1970).

An increased incidence of Osteomyelitis occurs in systemic diseases including diabetes and sickle cell disease. The prolonged use of broad spectrum antibiotic therapy or the presence of a compromised immune system may predispose the patient to Osteomyelitis caused by unusual organisms (Bonakdar-pour 1984). In our series Endogenous form of osteomyelitis was the commonest form of chronic osteomyelitis. This reflects the inadequacy in the treatment of acute osteomyelitis which has thus progressed to the chronic form. Most of these patients would have been saved of this problem if diagnosis was made earlier and aggressive antibiotic therapy begun (Table IV).

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Haematogenous (or Endogenous) chronic osteomyelitis is usually caused by gram positive organisms, the most common being Staphylococcus aureus. Other organisms include Pseudomonas aeruginosa, Klebsiella pneumoniae Escherichia coli and Streptococcus. Opportunistic organisms such as Candida albicans have been isolated in drug addicts, patients with diabetes mellitus, immunosuppressed patients on long term antibiotic therapy and premature infants.

There is an increased incidence of Salmonella Osteomyelitis in patients with sickle cell disease. In our series Staphylococcus aureus was the commonest infecting organism in four out of eleven patients. This figure is lower than that reported by Weiland and others (1984) which was 76%. The frequency of Staphylococcus aureus as a causative agent varies from 35 to 90% in various series reported in the literature (Walenkamp 1984). So our figure correlates with these reported series. (See table V).

Mixed bacterial flora is a feature most commonly found in exogenous osteomyelitis particularly those secondary to compound fractures. Exogenous Osteomyelitis has also got a high frequency of being involved by Anaerobic organisms (Walenkamp 1984). In our series no anaerobic organisms were isolated and this was because it was not requested or the specimen was not sent in a proper transport medium. It is not surprising to find that

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anaerobic organisms play an important role in Osteomyelitis, and that specimens of tissue from all patients with osteomyelitis should be cultured anaerobically (Hall and Associates 1983).

PATHOLOGY

Osteomyelitis is most common in the long bones, particularly in the lower extremities (Waldvogel 1970). Verterbral involvement is another major site in adults. The tibia and femur accounted for 9 out of 11 cases in our series. This finding is the same as that reported by other series. Gilmour (1962) found out that the lower limb is affected six times more than the upper limb.

The medullary canal of the metaphysis is the most common site for haematogenous osteomyelitis. The inflammatory process and pus so formed within the metaphysis may obstruct the vascular channels and extend into the haversion systems and Volkmanns canals. Subperiosteally, pus tracks both longitudinally and circumferentially around the bone, stripping the periosteum and interrupting the periosteal vessels. Thus progressive areas of the cortex become infarcted and involved in the inflammatory process.

The infection enters the chronic phase as the periosteal new bone proliferates to become the involucrum, which eventually surround the shaft. Holes or skip areas

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within the involucrum are called cloacae. These represent areas of necrosis where the pus extends into the surrounding soft tissue (Bonakdarpour 1984). This may lead to the formation of abscess in the soft tissues. The firm fixation of the periosteum to the bone at the level of the epiphyseal disc prevents subperiosteal spread in the direction of the joint. Where the metaphysis extends to inside the articulation, as is the case in the hip and the shoulder, its intra-articular position after penetration of pus through the thin periosteum may lead to rapid development of arthritis (Walenkamp 1984).

Thrombosis of the nutrient artery on one hand and separation of the periosteum on the other deprive the cortex of, respectively, its endosteal and periosteal blood supply resulting in large cortical sequestra. A sequestrum can be surrounded by pus or may be adjacent to viable cortex. The sequestra and involucra usually take at least three weeks to appear. Bacteria in the necrotic bone from time to time cause the inflammation to flare up, more bone is devitalised and a vicious cycle may develop, not rarely with gradual spread of the infection.

CLINICAL FEATURES AND DIAGNOSIS

Chronic osteomyelitis is characterised by its protracted course and by the rarity of spontaneous recovery. Chronic osteomyelitis may remain quiescent for months

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or years, but from time to time acute or subacute exacerbations occur. An exacerbation is ushered in with constitutional upset and local evidence of inflammation which may culminate in a discharge of pus, often from a pre-existing sinus (Bailley and Love 1975).

It is rare for spontaneous expulsion or reabsorption of a sequestrum to lead to spontaneous cure. Treatments are characterised by a usually high recurrence rate. Recurrence after periods of up to 80 years duration have been reported in the literature (DODI 1962). The average duration of the disease is several decades, and often, numerous surgical interventions and long courses of antibiotic treatment are necessary.

Most patients with chronic osteomyelitis feel hardly any pain. However, early closure of a sinus resulting in congestion of pus causes pain as the pressure in the sinus rises and in addition frequent general inflammatory symptoms. When pain develops in a still discharging sinus, the possibility of malignant degeneration of the epithelium of the fistulous duct should be seriously considered. (Walenkamp 1984). The constitutional disturbance in exogenous chronic osteomyelitis are less severe as the causative wound provides some measure of drainage.

The extremity has a characteristic appearance with several, often deeply retracted scars and one or several sinuses. The soft parts around the osteomyelitic

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focus are greatly scarified and the skin is often dystrophic. Pus is secreted in small quantities and most patients may claim to have seen small bone chips from the sinus. The presence of the sinus, however, frequently constitutes a severe psychic stress. The bone may be locally sensitive to tapping.

A brodie's abscess may cause intermittent pain near the end of the involved bone and occasionally transitory effusions in the adjacent joint during an exacerbation. Some patients may present with pathological fractures or flexion deformities of joints as long term complication of the disease.

In our series the presence of a discharging sinus and swelling of the affected bone were the commonest symptoms in over 80% of the patients. Most of these patients are usually referred from other centres because of an abscess which was drained and now has not closed for a long time, or due to a complication of compound fractures. This explains the inadequacy of aggressive treatment of most acute osteomyelitis in our environment.

As the main symptom, pain might be a parameter, this holds true only of acute osteomyelitis and an exacerbation of chronic osteomyelitis. Fever is also present only in acute or exacerbating chronic osteomyelitis.

Apart from the symptomatology laboratory aids may give a

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help. Leucocytosis with a left shift occurs exclusively in the acute stage or exarcebartion of chronic osteomyelitis. The ESR, however, is an excellent parameter to follow the evolution of osteomyelitis and its treatment. The ESR is of little help in the diagnosis because it shows little or no rise and does not exclude osteomyelitis.

Roentgenological examination is of great importance in all skeletal diseases. The radiologic examination of patients with osteomyelitis has become complex because of the effects of antimicrobial therapy (Bonakdar-pour 1984). The important radiological features are rarefaction of the bone, osteoporosis with periosteal reaction.

These are areas of bone destruction with loss of structure of bone. A sequestrum can be seen separated from the bone and surrounded by a sclerotic area of new bone formation the involucrum. These features can be localised but sometimes may involve a large portion of bone. Radiological changes are rare in cases of acute osteomyelitis.

Sinograms made with aqueous radiographic media may aid in localisation of sequestra or points of persistent infection and will demonstrate the course of the sinus tract. C.T. scan may be helpful in identifying deep areas of bone destruction. Occasionally, bone scanning

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with radioisotopes will localise otherwise occult infection. Tomography may also be helpful in localising the extent of a sequestrum.

Bacteriological examination is an important part of the diagnosis. Specimen collected for cultivation may yield direct information when examined microscopically after staining. This combined with clinical symptoms, often permit a tentative conclusion concerning the identity of the causative agent and a choice of antibiotic can be made while waiting for the results of the culture (Bonakdar-pour 1984, Walenkamp 1984).

The culture is the key element in the diagnosis and treatment of osteomyelitis. Numerous organisms are described in literature as possible causative agents of osteomyelitis. A number of authors have pointed out that as a rule the bacteria present in sinuses are not the same as those in the more deepseated focus of infection. In the sinuses, contamination often leads to a mixed flora whereas in the deeper layers a mono-infection often persists. Mixed bacterial flora is also a feature of exogenous osteomyelitis especially secondary to compound fractures.

Staphylococcus aureus is the commonest aerobic micro-organism isolated. It is the most common organism isolated in our series in four patients. Anaerobic organisms, fungi and viruses are also known to be

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causative agents. Anaerobes are frequent in non-haematogenous infection (Lewis et al 1978). In patients with a very ill smelling wound with much necrosis, the possibility of an anaerobic infection should be seriously considered.

TREATMENT

Modern medical and surgical therapy has proved to be effective in most patients afflicted with chronic osteomyelitis (Ruttle and Others 1984). However, in 20 to 30% of patients treated with surgical debridement and specific parenteral antimicrobial therapy, recurrent sepsis has developed within two years. Retained sequestra, a poor blood supply, residual dead space, and resistant microorganisms have been associated with recurrent sepsis.

The control of osteomyelitis wound demands a three pronged attack, (i) adequate debridement to remove devitalised bone, scar and granulation tissue, (ii) Control of infection by local wound care and systemic administration of antibiotics and (iii) good soft tissue coverage and obliteration of dead space (Irons et al 1984, Ruttle 1984, Weiland 1984).

Efforts to improve the treatment of chronic osteomyelitis has proceeded in two directions, the use of specific parenteral antimicrobial therapy for long periods of time and aggressive surgical treatment.

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Specific antimicrobial therapy cannot be successful due to failure to achieve adequate bacteriocidal levels at the foci of infection and to the development of resistant organisms. (Weiland 1984, Ruttle 1984).

After debridement and preparation of a clean wound direct closure frequently is not possible because of loss of non-pliability of surrounding tissue. Even in those cases in which closure is possible there usually remains dead space, which fills with blood clot and provides a site for recurrent infection.

Therefore, both bone coverage and dead space obliteration must be achieved for the best result. In addition, because poor vascularity is an underlying problem in the osteomyelitic wound, the tissue used for coverage, should be as well vascularised as possible (Irons 1984). Generally, when choosing tissue for coverage, one should choose the simplest, safest procedure that will provide a satisfactory result. A local flap should be considered first (Ruttle 1984). If a local flap is not adequate, the next consideration is a distant flap in the form of either a pedicle flap or a free flap. (Irons 1984, Weiland 1984). Later if all the above are inadequate then the use of Vascular bone graft should be considered (Wood 1984).

Papineau (1973) popularised the technique of obliterating

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the dead space with cancellous bone graft. This is an excellent technique when the mechanical integrity of the underlying osseous structure has been undermined by the infectious process. This procedure is not superior to the local muscle flap. The local muscle flap has its limitations in that the defect to be obliterated must be in an anatomic location that permits rotation of the local muscle without disruption of its neurovascular supply and the mechanical integrity of the osseous structure should be intact.

Free tissue transfer can be in various forms, skin, muscle, skin and muscle, bone with muscle, bone with skin or bone with skin and muscle. Muscles are the best tissues for coverage in this technique because it provides bulk for filling cavities, it is well vascularised, and it can carry skin with it. Weiland and Associates (1984) used the latissimus dorsi in all their 24 patients. Other donor areas are the groin, gracilis muscle and others have reported using omentum (Irons 1984). In Weiland's series 79% of the transfers survived. May and Associates (1982) reported on 22 patients with a success rate of 100%.

Free soft tissue transfer may be useful for obliteration of dead space and direct wound coverage, whereas free microvascular bone transfers may be useful for osseous reconstruction following extensive resections. In this

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later technique it involves the isolation and transfer of a bony segment with or without an attached muscle or skin flap on a nutrient vascular pedicle. Therefore reconstruction of a bony defect may be accomplished by a mode of healing similar to that of a segmental fracture rather than the lengthy process of creeping substitution graft incorporation (Wood 1984). The donor sites include the fibular, rib, lateral scapular, metatarsals and the lateral portion of the radius.

The principles of treating chronic osteomyelitis in our circumstances are mainly centered on the eradication of the infection by specific antibiotics and adequate debridement of the infected bone by sequestrectomy, curettage of the cavity and filling the cavity with cancellous bone chips or by saucerisation.
Saucerisation is another procedure done so as to prevent cavitation. The cavity after it has been opened and curettaged the overhanging walls are smoothened, changing the cavity into a kind of saucer. This also facilitates easy drainage and the wound is left to heal by second intension. This method has the drawback that often much sound bone has to be sacrificed, so that it should not be used close to articulations. Also the resulting weakening of the bone sometimes lead to pathological fractures. The affected limb should therefore rest in a plaster cast to avoid this complication as well as to avoid contractures. In our series saucerisation was performed in 9 patients and sequestrectomy performed

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patients. Obliteration of the cavities is
ely practiced here as is the case in other
ed countries and most of the wounds are left
ulate slowly and later closed by secondary
g or skin grafting.

tics are very important in the treatment of
elitis and should be present in the infected
at sufficiently high levels to be able to
or destroy the infecting microorganism. The
tics should be started preoperatively during
cerbation period and continued postoperatively
eral weeks until cure is obtained (Walenkamp

ice of antibiotic depends on the local known
vity pattern of the suspected pathogen and
e modified when the culture results are out.
y, the antibiotic concentration attained in
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use of high doses of antibiotics.

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ND TROCHANTERIC REGION

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response are Cloxacillin, erythromycin, flucloxacillin, lincomycin and clindamycin. Others are tetracycline cephalosporin, chloramphenicol and Vancomycin. In our environment the commonly used antibiotics are cloxacillin, erythromycin and tetracycline.

Several authors have recommended the use of local application of antibiotics topically or by continuous irrigation of the wound with good results (Law 1975). Recently the use of Gentamycin chains placed in the cavity after removal of all necrotic tissue have gained popularity and the results are very impressive.

Sometimes amputation is indicated for chronic osteomyelitis under the following circumstances:- When the infection is so extensive that it cannot be cured by antibiotics and surgery; when resecting the diseased bone is possible but so disabling that function would be better after a satisfactory amputation and prosthesis, and lastly amputation is indicated before the patient is endangered by the infection (Campbel 1963).

CONCLUSION

Osteomyelitis is an important disease in our environment and throughout the world because of the long man hours lost in treating the suffering patients for long periods.

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In this small series it is seen that the disease mainly affects the young, who are thought to be the productive members of our society. The causative organisms compares well with other series. To avoid chronicity, medical personnel as well as patients should be able to recognize the importance of vigorous initial treatment of the acute stage.

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FRACTURES OF THE FEMORAL NECK AND TROCHANTERIC REGION

SUMMARY

Fifteen patients with traumatic fractures of the femoral neck and trochanteric region were managed by the orthopaedic and trauma department at the Muhimbili Medical Centre between December, 1985 and February, 1986.

There were twelve males and three females giving a male:female ratio of 4:1. Most of the patients were above 50 years of age in which there were 6 patients. The commonest type of fracture was pertrochanteric found present in eight patients followed by basal cervical in three patients.

Eleven patients were treated conservatively by skeletal traction in abduction while four patients underwent operative fixation usually after failure of the conservative treatment. Malunion with varus deformity was the commonest complication observed in three patients.

INTRODUCTION

Fractures of the neck of femur are very common in persons over the age of 60, in whom there is a tendency for the bone to become increasingly fragile in consequence of generalised osteoporosis (Solomon

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1973). They are also among the most challenging injuries to treat; as the complication rate in most published series is 60% (Tachdjian 1972).

Trochanteric fractures are more benign injuries than are fractures of the neck of femur, because they unite readily no matter how they are treated, and they are relatively immune from the serious complications from which femoral neck fractures are notorious (Adams 1978). They are common in the old especially in females who form the bulk of the aged population in developed countries. They are very often seen in persons of 75 to 85 years old. In the elderly and senile patients these fractures are difficult to treat because of the degree of osteoporosis in their hips and the high incidence of posterior and medial comminution that occurs in the fractures of these patients (Dorr 1981).

Subtrochanteric fractures of the femur again are predominant in elderly females and differ from fractures through the neck or intertrochanteric area in aspects of anatomy, biomechanics, treatment and results (Fielding 1973). These fractures are the least common of the three types of hip fractures consisting 7% of a series studied at Saint Luke's Hospital (1966). Subtrochanteric fractures are associated with varying degrees of comminution, and are much more difficult to reduce and maintain in reduction resulting in more complications.

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The treatment of hip fractures has been directed towards decreasing the morbidity and mortality of patients who suffer this injury. In young people, the restoration of anatomical structure and biomechanical function is of high priority. In elderly patients, prevention of death and medical complication is of first concern. In senile patients, concern has existed as to whether these patients should be treated with bed rest or Surgery (Dorr 1981).

In our environment hip fractures are not that much uncommon and it has been found from our study that the majority of patients (6 out of 15 patients) were above 50 years of age. This corresponds with other studies reported in the literature that this is a fracture of the elderly. (See Table I). From our series it is seen that the majority of our patients are males and this could be explained by the fact that old males in our community are still ambulant trying to keep their families.

CASE PRESENTATION:

A.P. a 21 year old male from Kawe was referred from Lugalo Military Hospital on 11.12.85 with a painful right lower limb after having fallen into a ditch. It is alleged that he was running and fell into a ditch with his right lower limb hitting the ground first and later fell on his right hip. He experienced severe pains in his right hip joint and was unable to stand up

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or move the affected leg. There was no bruising of the leg but the patient claims that after some time he noticed that there was slight swelling of his right upper thigh. There were no other associated injuries.

Examination revealed a young man in good nutritional state who was in pain, afebrile, not pale with his right lower limb splinted on a Thomas Splint. The vital signs, blood pressure, pulse rate and respiratory rate were all normal.

Local examination of the right lower limb showed that the leg was on a Thomas Splint and in extreme lateral rotation and a shortening of about two centimeters. There were no bruises or echymoses but the upper part of the thigh was slightly swollen. There was extreme tenderness at the right hip joint and no movements were possible at this joint because of pain. He could passively move the digits. The distal pulses were intact and there was no area of loss of sensation.

The rest of the systems were essentially normal. A diagnosis of fracture of the right femoral neck was made and a pelvic Xray which included the two proximal thirds of the femur was ordered. This showed a transverse baso-cervical fracture with cephalad displacement of the distal fragment. The patient was

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kept on skeletal weight traction in abduction using 7 kilogram weight. Check Xrays at four weeks showed that the fracture had healed with a varus deformity and it was thought impossible to correct the deformity by conservative means at this time and that the patient should be prepared for open reduction and internal fixation.

A full blood picture blood sample was taken, urine taken for routine analysis and the site of operation cleaned daily with brush and soap. Blood for grouping and crossmatching was taken a day before operation and two units of blood requested for the operation.

On 30.1.86 the patient was taken to theatre for operation. Under general anaesthesia, thorough cleaning of the site with antiseptics and drapping was done. A hockey stick incision was made on the hip joint. The hip joint was opened after deviding the iliotibial tract and vastus lateralis and later opening the capsule.

The femoral neck fracture was found to have scanty callus bridging the fracture site in a varus deformity. The callus was removed using periosteal elevator and the two fragments cleared free until fresh bleeding was oozing. The two fragments were reduced and aligned and later fixed together using two spongiosa screws on the lateral side of the trochanteric region. The fracture

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site was irrigated with normal saline, haemostasis maintained, a tube drain was inserted and the wound closed, after repairing the divided muscles using vicryl 2/0 and silk 2/0 to skin.

Post operatively the patient was given intravenous fluids of normal saline and 5% Dextrose at the rate of 3 litres in 24 hours. Pethidine 100 mg. intramuscularly was given for analgesia every 6 hours for the first 24 hours. Penicillin (PPF) 1.2 mu intramuscularly daily was given for five days and this was combined with oral chloramphenicol 500 mg. every 6 hours for five days. The blood pressure and pulse rate was monitored hourly for 24 hours.

The post operative course was uneventful, the drain was removed on the second day and the stitches removed on 8.2.86 and the patient discharged home with crutches. He was advised not to bear weight on that affected limb until advised later. He was seen at the clinic after two weeks and was in good condition but still on crutches.

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ANALYSIS OF THE 15 PATIENTS WITH FRACTURE OF FEMORAL NECK AND TROCHANTERIC REGION.

TABLE I : The age distribution of the 15 patients with femoral neck and trochanteric region fractures:-

Age (yrs.)	No. of pts.
11 - 20	2
21 - 30	2
31 - 40	1
41 - 50	4
Over 50	6
T o t a l	15

Most of the patients are above 50 years of age.

TABLE II: The site of fracture in the 15 patients.

Site of fracture	No. of pts
Neck of femur (intracapsular)	
. Subcapital	1
. Transcervical	1
. Basalcervical	3
Trochanteric region	
. pertrochanteric	8
. Subtrochanteric	2
T o t a l	15

Most of the fractures occured at the intertrochanteric region.

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TABLE III : The mode of treatment in the 15 patients.

Mode of treatment	No. of pts.
Conservative:	
- Skeletal traction in abduction.	11
- Hip spica	0
Operative	4
T o t a l	15

Skeletal traction in abduction was the commonest mode of treatment which was applied in 11 patients.

TABLE IV : The complications encountered in the 15 patients.

Complication	No.of pts.
Malunion-Varus deformity	3
Non Union	1
Avascular Necrosis	-
Distraction of fracture fragments	1

Varus deformity was the commonest complication.

DISCUSSION

Classification of Fractures of Proximal Femur

The classification of fractures of the proximal femur can be broadly put into fractures of femoral neck, fractures of the trochanteric region and fractures of the subtrochanteric region. They could also be

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classified as intracapsular fractures and extracapsular fractures. Intracapsular fractures are those which occur within the capsule of the hip joint and are mainly femoral neck fractures, and extracapsular fractures are those which occur outside the capsule such as in the trochanteric and subtrochanteric region. Basalcervical fractures of the femoral neck could also be extracapsular.

Fractures of the femoral neck can further be classified anatomically or pathologically. The anatomical classification considers fracture of femoral neck as subcapital, transcervical and Basocervical fractures. The subcapital and transcervical fractures are very notorious in producing avascular necrosis as they inevitably interfere with the blood supply to the femoral head through the retinacular vessels (Watson-Jones 1955).

Pathologically femoral neck fractures are classified using the Garden (1961) system of classification as follows:-

Garden I - This is an incomplete fracture and is commonly known as the abducted or impacted fracture. This may be treated non operatively.

Garden II - This is a complete fracture but without displacement. This fracture may displace further and operative fixation is indicated.

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Garden III - This is a complete fracture with partial displacement but the fractured surfaces are still in apposition postero-inferiorly and the fragments have rotated in opposite directions. These fractures need operative reduction and fixation.

Garden IV - There is complete fracture with full displacement. Contact between the fractured surfaces has been lost. This indicates that the soft tissue attachments between the head and neck are completely torn, allowing the head to rotate back to its normal position in the acetabulum. The rate of avascular necrosis in this type of fracture has been reported as 21% (Barr 1973).

Trochanteric fractures can be classified clinically as stable fractures when the medial cortex is in contact with the distal medial cortex, and unstable fractures when there is displacement, the proximal medial cortex is not in contact with the distal medial cortex. Anatomically a fracture which occurs through the intertrochanteric line is called an intertrochanteric fracture and this can be either stable or unstable. This classification is important in determining the mode of treatment and prognosis. Boyd and Giffin classified trochanteric fracture into four types. Type I and 2 were purely intertrochanteric, and Type 3 were essentially subtrochanteric and Type 4 comprises

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comminuted fractures extending through the trochanteric area into the sub-trochanteric area and usually on down the shaft.

Watson, Campbell and Wade (1964) noted that classification of trochanteric fractures was extremely difficult, finding that many of their fractures did not fit into Type 3 and 4 of Boyd and Griffin. They reclassified fractures utilising a'reference point' or an indication of where the fracture line began, in relation to the 'notch' on the superior neck; they also used the fracture length and finally, a 'comminution digit' denoting the number of fragments. Therefore, a classification of 4-2-2- would mean that the fracture line began 4 cm below the notch, was 4 cm in length and had 2 fragments. In our series we only classified trochanteric fractures as stable or unstable and did not use the other modes of classifications because they are more complex. However the Watson, Campbell and Wade's classification gives more information pertaining to the exact site of the fracture.

The subtrochanteric fractures of the femur have never been adequately described, a situation which makes attempts at classification confusing (Fielding 1973). For classification, subtrochanteric segment has been divided into three regions, corresponding to the types of fractures observed, because the clinical problems encountered, as well as the prognosis, differ at each

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level. In general, the higher the fracture, the better the prognosis (Fielding 1973).

Type I fractures occur at the level of the lesser trochanter, Type 2 fractures occur between 1 and 2 inches below the upper border of the lesser trochanter and Type 3 fractures occur between 2 and 3 inches below the upper border of the lesser trochanter.

The most common types of fractures encountered in our series were pertrochanteric fractures in 8 patients followed by femoral neck fractures in 5 patients.

MECHANISM OF INJURY

Severe trauma is the commonest cause in 80% of cases in the young age group (Belkin 1980). Automobile accidents and falls from heights are among the leading causes. In about 30% of such patients they usually have serious associated injuries. From the above causes it is seen that the impact of injury could either be a direct voilence or transmitted as from a fall from a height on the feet.

In femoral neck fractures which usually occur in elderly people with osteoporotic bones the causative injury is often slight, usually a fall or a stumble. (Adams 1978). Fatigue or stress fracture of the femoral neck is a well recognised entity, though uncommon. The

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fracture usually develops insidiously. It is common in young adults exposed to unusual physical activities such as route marches with a full millitary pack.

Sideway falls producing a blow over the greater trochanter are commonest causes of pertrochanteric fractures, and subtrochanteric fracture. In sub-trochanteric fractures the history is of severe trauma in a healthy adult. The subtrochanteric region is also a favourite site for pathological fracture in paget's disease of bone. (Henry 1974).

CLINICAL MANIFESTATIONS AND DIAGNOSIS

In femoral neck fractures which are displaced a typical history is that the patient, usually an elderly woman, tripped and fell, and was unable to get up again unaided. She was subsequently unable to take weight on the injured limb. The most striking feature on examination is the marked lateral rotation of the limb which is slightly flexed at the hip and knee. The rotation can be as much as 90 degrees, so that the patella and the foot points laterally. The limb is shortened and any movement of the hip causes severe pain.

In the exceptional case in which the fracture is impacted the history and signs are different. The patient may have been able to pick herself up after falling, and

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she may even have walked a distance afterwards. Some patients may have proceeded with their duties and have not sought medical advice for several days. On examination there is usually no detectable shortening and no rotational deformity. The patient can move the hip through a moderate range without severe pain.

In trochanteric and subtrochanteric fracture the history and finding are almost the same as in femoral neck fracture, but the voilence causing the fracture is usually severe. The lateral rotation is not so severe as in femoral neck fracture but pain is most marked over the trochanteric region. Blood loss into the tissues may be considerable and can cause shock, and after a day or two a visible ecchymosis often appears at the back of the thigh. This feature is not seen in cases of femoral neck fracture because extravasated blood is retained within the joint capsule (Henry 1974, Adams 1978).

Radiographic examination in patients suspected as having any fracture is the single important investigation The aim of taking Xrays are:- to confirm the presence of a fracture, to know the pattern and alignment of the fracture, and to determine the degree of displacement of the fracture. All these parameters are important in determining the mode of treatment,

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to foretell the possible complications anticipated and to determine the prognosis. They are also important in the follow up of the patient postoperatively or during conservative skeletal traction treatment. The Xrays should always be taken in two views anteroposterior and lateral. A pelvic Xray may be important for comparison.

In an ordinary case with displacement of the fragments, the fractures are usually obvious and cannot be mistaken, but in cases of impaction of femoral neck fractures and pertrochanteric fractures without displacement, the radiographic changes are slight and the fracture may be overlooked.

In fractures of the subtrochanteric areas if complete there is usually severe overiding displacement due to the action of the strong hip flexors and abductors. (Fielding 1973).

TREATMENT

FRACTURES OF FEMORAL NECK

In the treatment of fractures of the femoral neck, displaced fracture and impacted abduction fractures must be considered separately. The age of the patient is another factor which should be taken in consideration when planning the treatment.

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An impacted abduction fracture will usually unite readily without operation, and conservative treatment should therefore be advised (Adams 1978, Massie 1973). This mostly applies to Garden type I and 2. The patient should rest in bed for three weeks but should be encouraged to practice hip and knee flexion exercises under supervision. Check radiographs are obtained weekly at first to make sure that the original impacted position has been maintained.

After three to six weeks the patient is allowed to start walking with crutches without weight bearing. Full weight bearing is avoided until about the eighth week.

Most surgeons distrust this conservative approach to impacted abduction fractures, fearing that the fragments may become separated and displaced (Dorr 1981).especially in Garden 2.

For quite a long time operative treatment has been the acceptable method of dealing with displaced fractures of the femoral neck, but there is no agreement as to what metallic devices produces the best result (Barr 1973). Failure rates of up to 50% are common in the literature and dissatisfaction with these results led to a number of investigators to develop mechanically better fixation devices. The tri-flanged nail of Smith-Petersen has been followed by a variety of sliding nails and multiple

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pin devices in recent years (Barr 1973, Massie 1973, Stromqvist 1984, Svenningsen 1984).

The requirements for stable fixation of femoral neck fractures are:- (a) Proximal fragment fixation - the device must penetrate well into the femoral head, preferrably within 6 mm of the joint surface, in order to achieve secure purchase. During recent years, various hip compression screw systems have been developed for this purpose, (b) lateral shaft fixation - this is essential to prevent the nail from backing out and allowing displacement of the fracture, (c) Impaction of the fracture surfaces - this can be achieved by hip compression screws. The aim of this type of fixation is to obtain compression of the fracture at surgery, and the sliding mechanism is supposed to secure interfragmentary contact even if resoption of the fracture ends should occur (Svenningsen et al 1984). Frandsen and other workers (1984) argue that the sliding screw plate is best in treating trochanteric fractures, and although it is used in applying compression across femoral neck fractures as recommended by several workers, its value has not been demonstrated.

In Massie's series (1973) using a triflanged sliding nail in displaced femoral neck fractures had an 87% success rate if one disregards the technical failures. He concludes that with adequate reduction and proper technique the results of sliding nail fixation are

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highly acceptable. Advanced age or displacement of the fracture do not contraindicate nailing.

Dissatisfaction with the results of operative fixation of displaced fractures of the neck of femur resulted in the widespread use of prosthetic replacement as primary treatment (Evarts 1973). Indications for use of endoprosthesis for femoral fractures include; high subcapital or comminuted fractures, fracture dislocations, irreducible fractures in which adequate fixation cannot be obtained, pathological fractures, mental illness, Parkinson's disease, hemiplegia or blindness, elderly and debilitated patients, secondary avascular necrosis and non-union of the femoral neck.

Boyd commended that the sacrifice of the head and neck of the femur and replacement by a metallic foreign subject is not the answer for the majority of patients. In over half, the best available material is in the acetabulum, and its indiscriminate removal should be avoided. This comment should be considered when planning the use of primary endoprosthesis. The child or adolescent is not a candidate for primary prosthetic replacement (Evarts 1973). The use of an endoprosthesis in the treatment of a general neck fracture has

