DISCUSSION

Malabsorption and maldigestion are significant causes of morbidity and mortality throughout the world. Malabsorption can result from a large number of diseases. Incidence and prevalence may vary according to geographically variation.

In third world countries infections of the gastroenteral tract are common causes of malabsorption syndrome.

A number of disorders result in malabsorption of one or more of the essential nutrients electrolytes, minerals or vitamins. Some or all of the following features may ensue, - diarrhoea, abdominal pain and distension, loss of weight, anaemia or other evidence of specific deficiency. However, some patients complained only of vague ill-health and the diagnosis may not be made for many years.

Malabsorptive disorders can be classified according to whether the primary disturbance is within the intestinal lumen due to insufficiency of digestive enzymes or bile acids, or within the intestinal mucosa. In disorder of intraluminal digestion which include pancreatic insufficiency (chronic pancreatitis, cystic fibrosis, carcinoma of pancreas). Deficiency of bile acids (interuption of the enterohepatic circulation of
bile acids due to resection or disease of the terminal ileum, colonisation of small intestine with bacteria which deconjugate bile acid which reduces their efficiency, stagnant loop syndrome) uncoordinated gastric emptying which delivers gastric chyme too quickly to the intestine (gastroenterostomy, partial gastrectomy). Disorder of transport in the intestinal mucosa in which include, generalised mucosal abnormalities, the mucosa is abnormal histologically (Coeliac disease, tropical sprue, lymphoma, Whipple's disease, radiation enteritis). Malabsorption of specific substances in which mucosa is normal histologically (lactose deficiency).

A number of signs indicates possible malabsorption:

1. Unexplained weight loss or malnutrition without obvious systemic illness (disease such as malignancy and thyrotoxicosis excluded).

2. Unexplained anaemia especially if two or more of the following are abnormal like iron, $B_{12}$, folate albumin.

3. Diarrhoea, with normal stool investigation, normal sigmoidoscopy and normal barium enema.

4. Osteomalacia without a dietary or renal cause.

Most of the test useful in the diagnosis of malabsorption indicate the presence of abnormal absorption or digestive function and only a few test may suggest
a specific diagnosis. Accordingly, it is frequently necessary to employ a combination of tests to establish a diagnosis.

In our study, out of 14 proved cases of malabsorption, there were 5(35.7%) cases of tuberculosis, 2(14.3%) cases of obstructed jaundice, two (14.3%) cases of chronic pancreatitis, 1(7.1%) case of cirrhosis of liver, 1(7.1%) case of ulcerative colitis and other one ulcerative colitis with giardiasis (7.1%), 2 cases remained undetermined (14.3%), but responded to antibiotic, most probably they would be cases of bacterial over growth. 14 proved cases of malabsorption mean age was 35.7±6.2 years, Range was 14-60 years and male : female ratio was 9 : 5. (Table 3)

While some relevant incidental observations were made during the study and are discussed in brief in the following paragraphs.

AGE AND SEX INCIDENCE

Average age of control group (20 cases) was 33.1±10.3 years with range of 16-50 years. Male : female ratio was 3 : 2. (Table 2).

Mean age of patients (58 cases) was 33.1±10.4 years with range of 14-60 years and male to female ratio was 33 : 25 years (Table 1).
Mean age of steatorrhoeic patients was 33.6±14.5 years with range of 14-60 years. Male : female ratio was 4 : 3 (Table 9). Comparative findings were reported by Baker (1962) from a study of 60 cases of chronic unexplained diarrhoea with secondary nutrition deficiencies. The mean age of the patients with secondary malabsorption syndrome was 34.5% years. No significant sex difference was observed and male : female ratio was being almost equal to in number 9:8.

**SOCIAL STATUS**

All patients came from neighbouring villages of Bundelkhand region, Jhansi, Central India. All of them belonged to low to lower middle income group and were on a poor diet.

**ALTERED BOWEL HABIT**

For three months or longer, the symptoms of anaemia were common to all patients. Ranging from mild to severe (Table 4).

**ABDOMINAL PAIN**

Mild to moderate abdominal pain was present in 50% of cases. 3 patients had moderate to severe pain.

Irregular fever anusea, vomiting and loss of appetite were vaguely present in 50-60% cases. Infact severe abdominal pain associated with these symptoms always suggested an organic gastrointestinal disorders.
GLOSSITIS AND STOMATITIS

Glossitis and stomatitis were observed in 30% cases. Green and Willaeger (1960) and Cook et al (1953) recorded glossitis in 86% and 90% of their cases of sprue and idiopathic steatorrhea respectively.

OEDEMA

It was observed in 21% cases. This incidence was less than that reported in the series of Green and Willaeger (1960) and Cook et al (1953).

PARASTHESIAS

With signs of peripheral neuritis parasthesias were observed in 1 case only. Pigmentation over dorsum of hand were seen in 20% cases.

HAEMATOLOGICAL STUDIES (Table 6)

All the patients were anaemic. Morphologically speaking from absolute values only 3 patients had mean corpuscular volume more than 94 cubic micron. But macrocytosis as observed from the peripheral blood film was present in 8 (57.1%) cases of malabsorption. Cook et al (1953) observed macrocytosis in two thirds of their 100 cases of idiopathic steatorrhea. Perez Santiago and Butterworth (1957) have also reported high incidences of macrocytosis in their cases of tropical sprue. Although over all patients had 18(31%) cases macrocytosis.
Measuring faecal fat, the qualitative examination of the stool, for undigested muscles fibres neutral fat, and split fat is a simple and reliable screening test for steatorrhoea. Stool appearance (bulky, floating malodorous stools) and volume may helpful (a daily stool output less than 150 gm effectively rules out malabsorption). The finding of an increased number of muscle fibres indicates impaired intraluminal digestion, properly performed the qualitative microscopic examination of a stool specimen with the sudan III stain is of value and correlate well with the quantitative determination of fecal fat by the Vande Kamer method. This microscopic technic appears to be very adequate for demonstrating varying degree of steatorrhoea and is a good screening procedure (Drummey et al, 1961). It is also reliable in excluding steatorrhoea in children over the age of 3 months (Ghosh et al, 1977). Stool microscopy using oil Red O to stain fat globules had a sensitivity of 72.2% and specificity of 95.4% (Teh lipbin et al, 1983).

The definitive "Gold standard" test with which to establish the diagnosis of malabsorption is the quantitative determination of fecal fat in stool described by Vande Kamer et al (1949), has been shown to be as accurate as gas liquid chromatography in quantifying fecal fat (Finley and Davidson, 1980).
Fecal fat excretion is increased under several physiological conditions like when the diet is high in fibre (7100 gm/day) (Levine and Silvis, 1979). When dietary fat is ingested in solid form such as whole peanuts (Levine and Silvis, 1980). In the neonatal period when intraluminal levels of pancreatic lipase and bile salts are low (Finley and Davidson, 1980).

The fecal fat concentration = fecal fat(gm/day) divided by stool wet weight (gm/day) multiplied by 100%, may provide a clue to the cause of the steatorrhoea. Steatorrhoea secondary to pancreatic insufficiency or hepatobiliary fecal fat concentration (79.5%, normal = 27% on the average than patients with other conditions (Bo Linn and Fordtran, 1984; Robert et al, 1986).

Once the presence of steatorrhoea is confirmed, the next step is to determine the cause by disease of the intestinal mucosa or by abnormalities of intraluminal digestion. To distinguish between these possibilities several other tests are helpful. But these other tests were not feasible in our institution.

**Fecal Fat Determination**

In our study, daily fecal fat values determined in 20 control subjects over a dietary intake of 75 gm fat per day, ranged between 3.00-4.90 gm/day with a mean ±SD of 4.0±0.6 gms. Age range was 16-60 years and mean ±SD was 33.1±10.4 years (Table 7).
Frazer (1955) used a similar method of fecal analysis stated that in normal persons on a diet containing 50-150 gms of fat per day. Fecal fat should be 5 gms or less. Pimplarker et al (1961) also used a similar technique reported the normal as being 7 gms or less of fat per day.

Backer (1962) recorded that fecal fat excretion in 15 normal subjects on a daily fat intake of 50 gms ranged between 1 to \( \leq 5 \) gms.

In our study, clinically suspected malabsorption patients (58 cases) on whom daily fecal fat determined were made. 14(24.13%) patients excreted more than 5 gms of fat per day. The mean excretion of fat per day in patients with steatorrhoea as 8.76±5.67 gm/day with range of 5.0-26.0 gms/day age range: 14-60 years with mean age of 33.6±14.5 years (Table 8 and 9).

Comparing patient to patient there was little correlation between the degree of steatorrhoea and clinical state of the patient or number of stools passed per day.

Perez Santiago and Butterworth (1957) used the technique of Va de Kamer and reported fat excretion of over 5.5 gm/day. But rarely above 20 gms in many patients with sprue, but not all. In our study there was no any patient in which fecal fat was normal but D-xylose was abnormal. But a study in Delhi (1965) by Mangia who showed that 2 patients had daily fecal fat less than 5 gm
with definite impairment of a D-xylose absorption and equivocal radiological signs of sprue. pattern, whether or not these patients showed be included in malabsorption syndrome was difficult to decide. Similarly it was difficult to explain abnormal D-xylose absorption in absence of steatorrhoea. The consideration that chronic anaemia (both patients were anaemic) can impaired the cellular metabolism of intestinal mucosa and gave rise structural and functional defects was one of the possibilities. Unfortunately, intestinal biopsies were not done in those patients.

In our study, one female, 22 years (case No. 7) had more than 20 gm of fecal fat/day. She was diagnosed ulcerative colitis. But this to much steatorrhoea (26 gm/day) was not explained. Unfortunately jejunal biopsy facilities are not available in our institution. So it was not possible. But she responded well to anti ulcerative colitis treatment.

In our study over all fecal fat mean ± S.D. was 4.4±3.7 with range of 1.2 to 26 gm/day (Table 8).

**D-XYLOSE TEST**

Many workers have stressed that D-xylose test is more specific of intestinal carbohydrate metabolism malabsorption than the traditional glucose tolerance test (Gardner and Perez Santiago, 1956, Benson et al, 1957).
It was abnormal in over 90% of patients with steatorrhoea of intestinal origin particularly the primary malabsorption (Fourman, 1948). reported 3 cases each of idiopathic steatorrhoea and tropical sprue in all of which D-xylose excretion was decreased.

D-xylose test is one of the most useful way to examined the absorption integrity of the intestinal mucosa. D-xylose is a pantose sugar that requires on intraluminal digestion and is absorbed from the small bowel via the same transport mechanism as glucose. In addition, it is not appreciably metabolised once absorp- tion occur and is excreted unchanged in the urine. The usual dose is 25 gm administered orally. 5 hours urine xylose excretion of 4 gm or greater is considered normal (Benson et al, 1957; Shamaa, Ghazanfar, 1960). Low values may be obtained in patients with ascites, intestinal overgrowth, or renal insufficiency, after administration of certain drugs (asprin indomethacin)and most commonly if the urine collection is incomplete. To prevent difficulties in interpreting the test, it is advisable to determine the blood xylose level 2 hours after ingestion to xylose. A blood xylose level of 30 mg/dl or greater indicates normal absorption of D-xylose. An abnormal D-xylose absorption test is found most frequently in disorders affecting the mucosa of the proxymal small intestine such as non tropical and tropical sprue.
The D-xylose absorption test is a basic simple test used during the last 40 years for the evaluation of small intestinal absorption (Fourman, 1948).

The pediatric patients, volume urine collection is problematic xylose absorption was monitored using the 1 hour serum concentration (Hawkin, 1970; Buts et al, 1978).

Another modification involved using 5 gm instead of 25 gm xylose (Santini et al, 1961). Craig and coworkers introduced a new approach to the evaluation of the D-xylose test by infusing 10 gm of xylose, a week apart from the 25 gm oral test. Multiple blood samples were taken through out both stages (Craig et al, 1983 and Atkinson, 1988).

They showed that total amount of xylose absorbed by related to 5 hour urine xylose with a high correlation coefficient. In addition they showed that the 1 hour serum test correlated highly with the rate constant for intestinal absorption. On these basis of these kinetic studies the authors suggested that the 1 hour serum might be used clinically to assess small intestinal malabsorption where as 5 hour urine test would be abnormal not only under these circumstances but in situation where there would be increased non absorptive loss of xylose. Kwatt and Beeton (1975) and Bode and Gudmand Hoger (1987) recommended abandoning the xylose absorption test
on the grounds of it being nonspecific. Slade and Kumar suggested omitting the test in cases where jejunal biopsy is feasible (Sladen & Kumar, 1973). As yet there is no complete agreement on the most accurate way to perform the xylose absorption test in adult patients (Zarling, 1988). However the test is still considered by most investigators as an important and reasonably sensitive one. A study by Peled et al. (1991) D-xylose absorption test urine or blood they suggested that in adults the 5 hour urine collection more accurately reflects intestinal absorption in comparison with one hour blood value. In a study D-xylose test in coeliac disease abnormal levels were found in 98% of total or subtotal villous atrophy. It was suggested to apply this test for screening in severe cases (Hedvig et al., 1983).

Since with increasing age the absorption of xylose improves this to be considered when evaluating the test. D-xylose test serves as an indication for small bowel biopsy. An abnormal D-xylose test after introduction of the gluten free diet points to its deficiency.

In another study from Vellore, South India Rolston and Mathew (1989) suggested that jejunal D-xylose absorption at concentration used clinically, is by passive diffusion, which process completely over rides a minor D-xylose cotransport component. The D-xylose tolerance test, therefore, reflects jejunal mjcosal
surface area and mucosal permeability to D-xylose and not nutrient carbohydrate absorption.

A study anti-gliadin antibody pannel and xylose absorption test in screening for celiac disease. Edward and Dennis (1990) recommended screening with the AGA panel with obtaining a xylose test if only the IgG is abnormal biopsy should be performed in cases with high IgA. IGA: or with abnormal IgG, AgA and xylose values. This approach is clinically preferable, does not add cost, and spares children from unnecessary small bowel biopsies. Screening test for carbohydrate malabsorption three test have been recommended (1) postprandial breath hydrogen production, fecal reducing substance, and fecal pH. Although all three test have limitations, the hydrogen breath test is the most useful and versatile in evaluating various types of carbohydrate malabsorption. This test is relatively simple, sensitive specific non-invasive and applicable to any age group (Newcomer, 1984).

Another study Rice Flour breath hydrogen and malabsorption by Paul et al (1984) proposed if indeed rice flour is well absorbed. It may provide a simple safe and noninvasive method to investigate carbohydrate absorption in a variety of diseases commonly associated with malabsorption.

In our study in which 58 suspected malabsorption clinically D-xylose test was abnormal in only 7(12.06%) cases. These all were steatorrheic patients. In other
there was no case who has negative fecal fat test but positive D-xylose absorption test. We can say that in steatorrhea patient, 50% patients have positive D-xylose test (Table 8). Included in those with impaired D-xylose absorption were patients with Koch's lung with Koch's abdomen or tubercular enteritis, ulcerative colitis with giardiasis, obstructed jaundice, chronic pancreatitis, Koch's abdomen.

In one (case No. 44) was diagnosed a chronic pancreatitis has marginal value that is 22 mg%. In other steatorrheic patients D-xylose test was negative.

In infiltrating condition of small intestine like tuberculosis, an abnormal D-xylose test would suggest mucosal damage rather lymphatic obstruction. In lymphatic obstruction only, as in intestinal lymphangiectasia. Waldmann et al (1961) have reported normal D-xylose absorption.

Impaired D-xylose absorption in the patient with cirrhosis and ascites is explained due to sequestration of a proportion of D-xylose in the ascitic fluid (Prokipchuk et al, 1961).

Normal or marginal result are however, obtained in pancreatic insufficiency (as in case No. 44) and is of value in differentiating pancreatogenous from intestinal steatorrhea.

In ulcerative colitis with giardiasis an abnormal D-xylose test was not explainable.
In controlled group (20 cases), age ranged 16-50 years with mean \( \pm \text{SD} \) 33.1 \( \pm \) 10.3 years. D-xylose value range from 28-110 mg\% with mean \( \pm \text{SD} \) 46.5 \( \pm \) 19.0 (Table 7).

In a one female age 16 years D-xylose value was quite high that is 110 mg\% we have no any explanation for that.

In patients group over all patients considering D-xylose test range was 10-118 mg\% with mean \( \pm \text{S.D.} \) 44.67 \( \pm \) 22.7 mg\%.

In patients group one male age 16 years has quite high value of D-xylose test, we were not able to explain the cause.

In the light of above results, D-xylose test cannot be considered as diagnostic of malabsorption syndrome.

**RECENTGENOLOGICAL STUDY**

All patients with malabsorption should have radiographic examinations of the small intestine and, in many cases of the esophagus, stomach and colon as well occasionally the latter two examinations may provide important clues to the presence of such disorders as gastroileostomy, scleroderma, Zollinger-Ellison syndrome ulcerative colitis, and intestinal fistulas. Traditional radiographic findings suggesting a diagnosis of
malabsorption include floculation of barium within fluid filled loops, causing fragmentation and segmentation of barium column. However, these patterns are no longer demonstrated reliably in small bowel series because of wide spread use of barium products, that contain a non-floculating suspension of micropuluerized barium sulfate. In non tropical sprue, the most consistent abnormalities are thickened and nodular duodenal folds and dilatation of the small bowel. However, these findings are nonspecific and may be found in several of the disorders.

Radiologic manifestations of malabsorption:
A nonspecific finding, study by Weizman et al (1984). Children demonstrating a radiologic malabsorption pattern of small bowel follow through study performed for other reasons are frequently subjected to intestine gastrointestinal investigations, even in the absence of clinical manifestation of malabsorption. Thirteen patients fulfilled the criteria for radiological malabsorption patterns, but six(4.6%) had no clinical evidence of malabsorption, according to 3-5 days fecal fat analysis.

In addition, five of these patients had normal mucosal histological findings on duodenal biopsy. It was concluded that radiologic malabsorption pattern is a nonspecific finding, and in the absence of other clinical features suggestive of malabsorption or growth failure further investigations may not be justified.
In our study, the changes in barium meal study of small intestine included dilatation, segmentation, fragmentation thickening of mucosal folds. The commonest were segmentation and fragmentation but dilatation of jejunal loops and coarsening of mucosal folds were also frequently seen.

Abnormalities were observed in steatorrhoeic patients. 8 patients had positive changes (57.1%). In four patients changes were equivocal (28.6%). Two patients had no change (14.3%) (Table 10).

These radiological barium meal follow through were done only in steatorrhoeic patients only, not in other patients nor in control subjects.

A good correlation was observed between the degree of roentgenological change and the clinical condition of the patient. Correlation was not so well marked with the intensity of steatorrhoea.

For the purpose of comparing the results of various diagnostic technics in relation to chemical fat determinations. It would seem that D-xylose and radiologic study of small intestine constitute the most useful screening procedures. Fecal fat determinations, however, would be required for confirmation and quantitation of steatorrhoea.
Radiological study of small intestine in majority of cases showed changes when abnormal. However, these tests were of diagnostic assistance in chronic pancreatitis, localisation of lesion in enteritis, and in demonstrating intestinal stasis due to strictures.