SUMMARY AND CONCLUSION
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Fifty eight patients with chronic diarrhoea and associated nutritional deficiencies were investigated for evidence of absorptive defects.

Fourteen of them had fat malabsorption. Twelve had detectable cause with tubercular enteritis (35.7%), ulcerative colitis, giardiasis (14.3%), obstructed jaundice (14.3%), cirrhosis of liver (7.1%), and chronic pancreatitis (14.3%). Remaining two had no detectable primary cause for diarrhoea and fat malabsorption. But they responded to antibiotic course.

The study included:

(i) Clinical assessment based upon history and physical examination.

(ii) Haematological studies.

(iii) Determination of daily fecal fat output.

(iv) D-xylose absorption test.

(v) Roentgenological studies of small intestine with barium meal.

The study revealed that the clinically suspected cases of malabsorption chemical analysis of faeces for daily total fat was not so sensitive (24.13% of 58 cases) for the detection of malabsorption though invariably always. It fail to indicate the underlying cause. In comparison to occurrence of subnormal result in other
test was D-xylose absorption test (12.06% in 58 cases) Roentgenologic study (57.1±28.6%) in malabsorptive patients.

In fat malabsorptive patients, according to statistical significant test both test fecal fat estimation and D-xylose test are insignificant at p = 0.05. In patients with steatorrhoea D-xylose absorption test was positive in 50%, and barium meal study result positive in 57.1% cases and 28.6% picture was equivocal.

D-xylose test did not increase the incidence of correct diagnosis. It was further observed that in patients of malabsorption syndrome the radiological examination of the small intestine gave abnormal results with one or more tests in 50% cases.

No constant correlation could be demonstrated between the severity of steatorrhoea and the results of D-xylose test and roentgenologic study. We had not used any test in differentiating pancreatogenous from intestinal malabsorption in a case of chronic pancreatitis.

In conclusion the result of this study indicate that though chemical determination of fecal fat is a "Gold standard" test, for malabsorption syndrome, yet the study of malabsorption does not correlate well with clinical features. We recommend, before doing quantitative test, other valuable screening procedure for malabsorption should be done like microscopical examination of the stool for steatorrhoea, which is a
valuable screening procedure in detection of malabsorption syndrome. It showed an error 11 to 14 percent (Weijers and Van de Kamer, 1953). The use of continuous marker provides a method for assessing the degree of steatorrhoea on a single stool sample without the disadvantages of the conventional method of fecal fat analysis.

If screening results are positive, a quantitative determination of fecal fat should always be done to objectively confirm that malabsorption exists. If the D-xylene test and small bowel follow through are abnormal, diseases of the intestine or presence of bacterial overgrowth should be considered. The small bowel biopsy established the cause of the intestinal lesion and fluid aspirate for cultures, plus the Schilling test may suggest bacterial overgrowth. If the serum trypsinogen is abnormal and pancreatic calcification are present on an abdominal plain film the diagnosis of chronic pancreatitis may be made. If the calcification are absent, the bentiramide or secretin cholecystokinin tests should be performed.

Study also indicate that tuberculosis is definitely closely related to malabsorption syndrome.