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DIAGNOSTIC APPROACH IN PATIENTS WITH LIVER DISEASES IN RURAL TANZANIA

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Von der medizinischen Fakultät der Universität Basel
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SUMMARY

The study represents an analysis of 80 patients admitted for liver disorders to a rural Tanzanian hospital in 1983. 51 males and 29 females were admitted to the study and were examined by clinical examination, liver function tests, serology, laparoscopy and histology. Their ages varied from 15 to 70 years with an average age of 40.

52 out of 80 patients had a complete step by step diagnostic approach of their liver problem. It was possible to establish a final diagnosis by clinical findings in 46 %, by additional laparoscopy in 25% and by histology in the remaining 29% of cases. Laparoscopy together with clinical examination and some laboratory tests, but without histology (not always possible in rural, tropical countries) provided important diagnostic informations in 9 cases out of 10. Clinical examination, laparoscopy and histology are accurate and have a low complication rate.

By this type of approach, it is possible to rapidly establish the diagnosis of liver diseases occuring in a rural tropical area. Hence, our findings serve as a basis to guidelines on diagnosis and management of hepatic disorders in rural hospitals and dispensaries.

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I. INTRODUCTION

Liver diseases, and especially those related to hepatitis B virus (HBV) infections and its consequences are highly prevalent in Africa (1,2) and many surveys in different countries have shown a high local incidence of HBV infections (3,4,5,6,7) and of hepatocellular carcinoma (6,7,8,9).

In addition, people returning from tropical countries to Europe present a prevalence of hepatitis greater than European residents (10).

There are, of course, many other types of hepatopathies associated with life in tropical countries, amongst which liver schistosomiasis, amoebic liver abscess, Tropical Splenomegaly Syndrome and Bantu siderosis are the most important (11).

In 1982, the first study of a project on hepatopathies demonstrated the importance of liver disorders at the St Francis Hospital in Ifakara (12). Within 3 months, 92 patients with clinical evidence of liver diseases were detected among the 1796 admissions to the hospital (5,1 %) during this time period.

92 patients with liver problems and 202 patients without evidence of liver diseases were tested for hepatitis markers. The exposure rate to hepatitis A virus (HAV) in the control group was 96 % (193/202) and was already 85 % in the first age group (0 - 15 years old). This means that the children are infected by HAV soon after birth without symptomatic disease in most instances (13).

HBV infection is highly endemic as well with an

exposure rate of 79 % (159/201) in the control group, and 81 % (77/92) in the patients with evidence of liver disease. Both groups had an exposure rate of 65 % in the first age group (0 - 15 years old), which is compatible with an infection early in life (14,15) possibly by vertical transmission from mother to child at the time of birth.

Beside these serological findings, a vague diagnosis was possible in only about 60 % of the patients with suspected liver diseases, using the results of clinical examination, liver function tests and serological viral markers. This fact led to the decision to try to improve diagnosis by using laparoscopy with guided needle liver biopsy in a second study group.

The aims of the study were :

a) to test the feasibility of laparoscopy in a rural tropical area

b) to evaluate the systematic use of laparoscopy in the diagnostic work-up of hepatopathies in a rural district hospital of Tanzania.

c) to validate the different diagnostic steps of increasing sophistication, leading finally to the diagnosis in patients with liver disease.

II. PATIENTS AND METHODS

II. 1. Geographic and demographic background

Ifakara is a rural township of about 35.000 inhabitants. It is situated at 8°9'S and 36° 40'E in the open plain of the Kilombero Valley, 4 miles north of the river at an altitude of 700 - 800 feet (230 - 260 m) (16,17) and

approximately 450 km from Dar es Salaam, the capital on the coast of the Indian Ocean.

Rainfall measurement shows an average of 1286 mm per year (observed from 1931 - 1958) (17) and was found to be nearly the same in 1982 - 1983 (18,19). The annual mean temperature is about 26 °C and the mean relative air humidity is 72%, varying according to the rainy season (March - May) and the "short rains" (December - January) (17).

Seven different tribes are encountered in the Kilombero Valley (Ndamba, Pogoro, Bena, Ngindo, Ngoni, Mbunga, Hehe) (20). Due partly to governement policy and partly to the search for work (the Kilombero Sugar Estate Co. is the biggest employer of the region) (20), the tribal mixing is higher than in the past. This latter fact explains the high number of different tribes encountered in our study.

People are mainly peasants, but as Ifakara is the chief-town of the Kilombero District, there are also highly educated people present who travel frequently on administrative tasks.

The Kilombero District population has 141'400 inhabitants whereas the Ulanga District has 118'850 inhabitants (Data for 1980 based on the Tanzanian Population Census 1978, but extrapolated to 1980 on the basis of a 2,7 % annual growth rate).

As in many developing countries, young people (0 - 29 years old) form the largest group of the population (21). Life expectancy has increased from 37 years (in 1964) to 51 years in 1980 (22).

Malaria is holoendemic in the Kilombero Valley (16) and climatic conditions are present for the growth of Aspergillus flavus and the production of aflatoxines in badly stored foodstuffs (23,24).

II. 2. Site for the study

St Francis Hospital is a 345 beds Designated District Hospital situated in the town of Ifakara. Its role is more pronounced, due to its important infrastructure. It has both preventive and curative medical services; it is a Teaching Hospital for the Medical Assistants Training Centre and it supervises the dispensaries of the Diocese of Mahenge which are spread over the Kilombero and Ulanga Districts (25).

In 1983, 8'867 in-patients were admitted and 1'008 out-patients were treated daily (25) at the St Francis Hospital.

II. 3. Admission to the study

From mid June 1983 to beginning of January 1984, 80 patients were selected for this study according to criteria specified below. All were in-patients from different wards of the St Francis Hospital (mainly medical wards : 72/80 patients). Admission to the hospital resulted from different complaints, some of them being directly related to liver problems.

Most of the patients came spontaneously; some were referral cases from Rural Health Centres of the District area or from Dispensaries.

The selection criteria were the same as in 1982,except that, for technical reasons, we chose only patients (males and females) over 15 years of age, and patients without a previous median upper abdominal laparotomy.

Each patient presenting one or more of the following criteria was reviewed by the same 2 physicians who decided upon his admission to the study. All patients

with one major criteria were included, as well as patients with any minor criteria if it was not obviously due to extrahepatic disorders.

Major Criteria :
- Scleral icterus.
- Hepatomegaly (liver size $\geq$12 cm).
- Hard consistency of the liver.
- Irregular surface of the liver.
- Liver tenderness.
- Right upper quadrant abdominal tenderness.
- Abdominal vein distension.
- Ascites.

Minor Criteria :
- Gynaecomastia.
- Melaena and/or Haematemesis.
- Splenomegaly (according to Hackett's classification, fig. 1).
- Axillar and pubic hair loss.
- Cachexia and peripheral oedema(fig.2).

The data were compiled for clinical examination, laparascopy, histology (see appendix).

A provisional conclusion was made after each diagnostic step.After evaluation of all steps, a final diagnosis was discussed and decided upon.

II. 4. Clinical diagnosis (Table 1)

The patient was examined by an ordinary physical examination to assess the clinical diagnosis. When it was not possible to come to a definite clinical diagnosis, we stated instead the summary of our findings.

II. 5. Laboratory

The following laboratory tests were performed in the Swiss Tropical Institute Field Laboratory (STIFL) : Total White Blood Count; Haemoglobin; Sickle Cell Test; Blood Group and Rhesus factor; Thrombocytes Count; Erythrocyte Sedimentation Rate; Serum glutamic-pyruvic transaminase (SGPT), Gamma-glutamyl transpeptidase (GGT), Alkaline Phosphatase, Prothrombine Time (Quick's method), Bilirubin (direct and indirect). Serum proteins and electrophoresis were performed in the Swiss Tropical Institute in Basle (Dr phil. B. Betschart) (Table 2).

In 1982, we verified in 202 patients without hepatopathies that the "normal" levels for the liver function tests were also accurate for the patients in Ifakara (12).

The laboratory results were correlated with the clinical findings for the diagnosis, according to basic literature (26,27). The reference values are given in table 3.

II. 6. Serology

Freezed sera were sent to the Swiss Tropical Institute, Basle, Department of Serology (PD Dr phil. N. Weiss; Dr phil. F. Speiser) for determination of :

- Hepatitis B markers

HB_sAG	by AUSZYME II	(Abbott Diagnostics Div., North Chicago, Ill., USA)
Anti HB_s	by AUSAB II	(Abbott Diagnostics Div., North Chicago, Ill., USA)
Anti HB_c	by CORZYME	(Abbott Diagnostics Div., North Chicago, Ill., USA)

- Parasites

Enzyme -linked Immuno-Sorbent Assay (ELISA) was

performed as a quicktest according to F. Speiser (29).

Entamoeba histolytica (HK9Antigen from ICN Ltd) and Schistosoma mansoni (total worm extract as antigen

Sera were tested at a dilution of 1:160 in PBS-7,2 containing 0,05 % Tween-20 for 15 minutes at 37°C. The conjugate was a Peroxide labelled anti-human IgG (H + L) (IgG fraction is produced in goat; Miles Nr. 61-230) and was also incubated 15 minutes at 37 ° C.

Each serum was tested for both parasites. The diagnostic limits with the antigen E. histolytica was E 492 nm = 0,50, and with the antigen S. mansoni E 492nm=0,30. Extinction values higher than these limits were considered serodiagnostically positive.

For malaria, the Indirect Fluorescent Antibody Test (IFAT) was performed according to A.J. Sulzer et al and N. Weiss & A. Degrémont (30,31). Antigens were used from washed cells from in vitro Plasmodium falciparum culture (kindly donated by Mrs. L. Reber, Hoffmann La Roche, Basle). Serum titre of 80 or above was considered serodiagnostically positive. The test was performed in patients who presented an intrasinusoidal lymphocytosis at microscopic examination of the liver searching for possible Tropical Splenomegaly Syndrome (32).

- Alpha-Fetoprotein (AFP)

by Alpha-Fetoprotein RIA Kit (Amersham, UK). AFP was determined for the diagnosis of primary hepatocellular carcinoma. We considered the upper normal level at 40 ng/ml (33) for any patient, except for a pregnant woman for whom we considered levels up to 80 ng/ml normal (34), according

to the age of gestation.

- IgM total

was measured by radiodiffusion (Nor-Partigen IgM, Behring Diagnostika, Frankfurt, West Germany). This determination was useful for the diagnosis of Tropical Splenomegaly Syndrome (TSS) (32).

To determine the usual level for IgM in the Ifakara area, a further Control group of 26 patients was taken out of the Control group of 202 persons without clinical liver disorders (study of 1982, Ifakara). They were chosen from among those without HB_sAg pos. and also without abnormal liver function tests and divided into two age groups (15 - 30 years old and 31 years old and over) because in the literature there are great differences according to age, country, altitude, malaria endemicity (27 , 33 , 35 , 36). IgM normal range among our control group (at 99 % confidence level) was 2,0 - 3,3 g/L (average 2,5 g/L).

II. 7. Laparoscopy

Material (see fig. 3,4 and Table 4)

Laparoscopy was not possible in all patients. It could be performed only with the consent of the patient, acceptable general condition, a Prothrombine Time above 40 % and Thrombocytes Count above 40'000/mm3.

Proceeding

The evening before the laparoscopy, the procedure was explained to the patient. Next morning, the patient obtained Diazepam 10 mg and Atropine 0,5 mg by intramuscular injection as praemedication, 30 to 45 minutes before the intervention. Blood pressure and pulse

rate were recorded. An intravenous drip with normo-saline (NaCl 0,9 %) was inserted.

After a check of the patient's condition and of the material by the physicians, local disinfection by iodine solution was applied to the left lower abdominal quadrant. After local anaesthesia with Lidocain 1 % the Veres' cannula was introduced (see fig. 5). 1.5 - 2.0 litres of N_2O were insufflated. After completing the disinfection of the whole abdomen and new local anaesthesia, the trocar was introduced, according to Beck and Dagnini (37, 38) (see fig. 6 and 7).

If any abnormal conditions on the left side of the abdomen (such as organomegaly or tumor) impaired the above-described procedure, we introduced the Veres' cannula and the trocar on the right side of the abdomen.

If the patient complained of pain during the laparoscopy, we added Pentazocin 15 mg IV. up to 30 mg IV.

Ascites was taken for analysis whenever it was possible. "Plasma Protein Lösung Human 3,5 %" (Immuno, Wien, Austria) was given when ascites amounting to over 2 litres was removed (2 cases).

For guided transdermal needle biopsy, we used a Menghini needle. The second physician did a local anaesthesia with Lidocain 1 % on the site chosen by the one performing the laparoscopy. After introduction, the Menghini needle was guided to the site for biopsy (see fig. 8). Sometimes, depending on the local conditions, biopsy was directly performed through the operating laparoscope by a long liver biopsy cannula. Rarely, we used a biopsy forceps to take peritoneal nodules or biopsies in the neighbour-

hood of an abscess. Liver specimens were put into Formaline 4 % solution. The patient was then placed either back in his ward or in a special monitoring room, according to his condition and the workload in the ward for the staff. Usual post-operative records were done and the patient could eat after 24 hours. If otherwise possible, he was discharged from the hospital on the third day after laparoscopy.

A detailed laparoscopy report was written and a diagnosis was formulated according to the macroscopic findings (see appendix).

After laparoscopy, the disposable instruments such as blades, syringes, needles were discarded. The other instruments were washed and brushed with water, then put into a disinfection solution (Cidex^{R} by Johnson and Johnson AG) for 30 minutes. They were then again brushed and finally rinsed with water. After drying, they were placed into a container together with Formalin tablets for at least 24 hours. Laparoscopies were performed every Wednesday and Friday, so that the instruments were, in fact, in longer contact with Formalin "vapors". Scalpel holders, hooks, Kocher's forceps and Menghini's needles were sterilized by heat and high pressure.

II. 8. Histology

Liver biopsy specimens were all sent to the Institute of Pathology, University of Basle, Switzerland (Prof. Dr med P. Heitz). They were all examined by Prof. Dr med L. Bianchi.

Beside the usual type of histological examination, special attention was paid to iron content and

to the presence of intracytoplasmic HB_sAg by special stains.

Occasionally postmortem biopsies were performed with the family's authorization in patients where laparoscopy had not been possible.

. 9. Final Diagnosis

The final diagnosis was discussed and decided upon by the medical team involved in the study after reviewing the findings of the different diagnostic steps.

10. Evaluation

To standardize the study population, we excluded all the patients who did not take part in all diagnostic steps. We only analysed those who had a full clinical, laboratory, serological, laparoscopic and histological work-up.

Some of the cases had a different diagnosis at each step. We therefore compared the diagnosis of each step with the final diagnosis in the following way :

1) is there absolute identity between an intermediate diagnostic step and the final one;
2) is there similarity, i.e. is the intermediate diagnostic step very close to the final one.

Furthermore,it was evaluated what can be achieved by laparoscopy in a rural hospital such as St Francis Hospital. Here, we considered three aspects :

1) absolute identity. The laparoscopic diagnosis is the same as the final one;
2) similarity. The laparoscopic diagnosis being almost the same as the final diagnosis;
3) positive management attitude of the liver problem brought by laparoscopy; i.e. assessment of the error into which we were falling with the purely clinical approach. It does not mean that laparoscopy provided the final diagnosis, but laparoscopy was, in fact, a useful aid in the establishment of the final diagnosis. Most of the time, the final diagnosis was provided by histology.

II. 11. Control Group

In our study, we defined it as a group of 80 hospitalized patients without clinical liver disorders and with the same distribution regarding age, sex and origin as our test group.

During the period of time of our study, we analysed sera from this control group for determination of serum glutamic-pyruvic transaminase (SGPT). This decision was taken in order to eliminate any local and sudden outbreak of liver disorder in the area, principally hepatitis.

III. CASE REPORTS

Only some of our eighty patients have their case reported here. They were chosen for demonstration of their diagnostic evolution.

Case 1 (report n° 7)

P.K., 31 years old, male, is a Veterinary Officer. History reveals a moderate alcohol intake and a recent weight loss. On examination, we find a body weight of 39 kg and a height of 158 cm, an enlarged, hard, nodular and tender liver (20 cm in the mid-clavicular line), but no ascites. Clinical diagnosis :
Malignant liver tumour.
Laboratory results : ESR 51 mm. SGPT 48 U/L. GGT 540 U/L. Alkaline Phosphatase 396 U/L. Prothrombine Time 44 %. Albumin is decreased at 1,37 g% and Albumin/Globulins ratio is 0,39. Diagnosis does not change.

Serology : HBs Ag pos.; AntiHBs neg.; Anti HBc pos. AFP : $1,8 \times 10^6$ ng/ml. Diagnosis : Probable primary hepatocellular carcinoma. On laparoscopy, we see an enlargement of the right liver lobe due to multifocal white-yellowish tumours of 1 - 5 cm in diameter. The same type of nodules is seen in the left lobe as well as a mediumsized nodular liver cirrhosis without signs of portal hypertension. Some bloody ascites is present. Diagnosis : Liver cirrhosis and malignant multifocal liver tumour. Histology confirms the primary hepatocellular carcinoma but not the liver cirrhosis. HBsAg is not found in the cytoplasm of the liver cells. Final diagnosis : Primary hepatocellular carcinoma and liver cirrhosis (HBsAg +).

Case 2 (report n° 16)

M.K., 24 years old, male, is a clerk coming from the North of Tanzania, but living in Ifakara. He is admitted because of bad general condition and enlarged lymphnodes since 3 months. The examination reveals enlarged lymphnodes and hepatosplenomegaly (spleen Hackett 2). No fever. No cachexia. Clinical diagnosis :Systemic reticulo-endothelial disease. Laboratory does not show much except :

ESR 95 mm. Prothrombine Time 50 %. Thrombocytes Count 70'000/mm^3. Diagnosis is unchanged. Serology : HBsAg neg.; AntiHBs neg.; AntiHBc pos. Titre for malaria is considered positive. IgM 2,3 g/l . At laparoscopy, we see an enlarged liver and spleen with some perisplenitis. Histology shows a non specific reactive hepatitis with intrasinusoidal lymphocytosis. Diffuse cellular siderosis is seen. Our final diagnosis remains the same as the clinical one : Systemic reticulo-endothelial disease with non specific reactive hepatitis. Later on, we obtained the result of a lymphnode biopsy : Kaposi's sarcoma.

Case 3 (report n° 43)

M.M., 40 years old, male, a well-known alcoholic peasant, is admitted for decompensated liver cirrhosis and cachexia. Laboratory results : ESR 105 mm. HB 8,1 g%. SGPT 35 U/L. GGT 81 U/L. Alkaline Phosphatase 198 U/L. Prothrombine Time 55 %. Total Bilirubin 22 mcmol/L (mainly direct fraction). Albumin is decreased and Albumin/Globulins ratio is 0,42. Serology : HBsAg pos.; AntiHBs neg.; AntiHBc pos. AFP : 26 x 10^3 ng/ml. Diagnosis : Probable primary hepatocellular carcinoma. The general condition suddenly becomes worse and we give up laparoscopy. With a protein diet and vitamins, the condition does not improve and he dies. Post mortem biopsy : bloody ascites and histology showing hepatocellular carcinoma (sinusoido-trabecular type). Presence of HBsAg in the cytoplasma of the liver cells and of the malignant cells. Final diagnosis : Primary hepatocellular carcinoma (HBsAg pos.).

Case 4 (report n° 47)

Z.S., 25 years old, female, is admitted for fever and liver tenderness. She gets a treatment with Metronidazol for a probable amoebic liver abscess. She leaves the hospital but comes again 60 days later complaining of

persistant abdominal pain. On examination, the right abdominal upper quadrant is tender indicating some liver tenderness and we diagnose a tender liver. Laboratory and serology results are normal. No findings for amoebiasis. Laparoscopy shows fine adhesions between the peritoneum, the right lobe of the liver and the omentum. At histology, we have a non specific reactive hepatitis. The final diagnosis is adhesions resulting from a probable pyogenic liver abscess, which was cured by Metronidazol.

Case 5 (report n° 74)

A.K., 56 years old, male, comes to the hospital because of a huge, painful abdomen. On examination, we find ascites and small, hard, multiple and painful nodules around the umbilicus. An intraabdominal tumour is not palpable nor the liver. We assume probable peritoneal metastases. Laboratory results do not show anything very specific : ESR 101 mm. GGT 54 U/L. Prothrombine Time 65 %. Laparascopy reveals peritoneal nodules, bloody ascites and an enormous, multinodular hard and haemorrhagic tumour occupying the whole left abdomen like a block. Liver is normal; no metastases are seen. Laparoscopic diagnosis : Intraabdominal malignant tumour with peritoneal metastases. Histology : Carcinoma adenomatosum. Final diagnosis : Intraabdominal malignant tumour with peritoneal metastases of an adenocarcinoma. This case was admitted to our study because of ascites, which,in fact, was not due to a liver problem.

IV. R E S U L T S

IV. 1. Control group

The control group consisted of 80 patients (56

males = 70 % and 24 females = 30 %). Data are missing for 3 patients (n = 77). Their ages varied from 15 to 70 years, the same as the test group. The average was 37,6 years. Most of the patients came from the Kilombero (excluding the Ifakara area) and Ulanga districts (40/77 = 51,95 %) and from the Ifakara area (32/77 = 41,55 %). The remaining patients (5/77 = 6,5 %) came from outside the above-mentioned regions. 62/77 patients belonged to tribes of the Kilombero Valley.

The patients were admitted to the surgical, medical and gynaecological wards according to their presenting symptoms and were without clinical signs of a liver disorder.

SGPT determinations varied from 4 to 64 U/L, with a mean $\pm$ 1 SD of 15,12 $\pm$ 8,35 U/L. Only 5 patients (6,25 %) had values above 30 U/L (males) and 25 U/L (females).

In 1982, out of 194 patients without clinical liver disorders 7 patients (3,6 %)had SGPT levels about these upper limit values.

IV. 2. Test Group

The test group consisted of 80 patients : 51 males (64 %) and 29 females (36 %). In 4 patients, data were incomplete. Their ages varied from 15 to 70 years; the average age was 39,9 years.

Age groups, sex distribution and origin are given in table 5 and fig. 9. They came mainly from the Kilombero and Ulanga districts. 85 % of them were peasants. 42 % did not drink alcohol and 17 % were heavy alcohol consumers (one person's data was missing). The past history revealed that 8 patients

out of 80 (10 %) suffered from a pulmonary tuberculosis and 7/80 (8,7 %) presented an icterus.

Clinical aspect

In 10 cases, it was impossible to determine the characteristics of the liver, and sometimes of the spleen, because of huge ascites. For further details, refer to table 6. The clinical findings were rather similar to those found in 1982 (see table 7).

According to definitions given in table 1 we had : 4 hepatitis, 3 icterus accompanying pneumonia among 7 cases of icterus, 4 cases of liver abscess, 4 congestive liver diseases, 25 liver cirrhoses, 5 malignant liver tumours, 1 metastatic liver disease, 2 cases of systemic reticuloendothelial disease, the other clinical diagnoses being just the summary of our findings. Refer to table 18 for more details.

The final diagnosis was made possible after the clinical examination in 20 cases out of 80 (25 %).

Laboratory

Laboratory tests proved to be relatively unreliable means regarding diagnosis, since they were found to be normal for some cases of Tropical Splenomegaly Syndrome, liver cirrhosis, chronic active hepatitis or metastatic liver disease (see table 19). Except for 11 patients, the laboratory results did not change the clinical diagnosis.

Serology

A marker for hepatitis B was found in 100 % (n = 80) as exposure rate. Out of them, 26,3 %

were HBs-Ag + ;58,8 % Anti-HBs pos. and 98,8 % Anti-HBc pos.

4 patients out of 80 (5 %) were positive for E. histolytica, one of them being strongly positive (>1,00).

28 patients out of 80 (35 %) were positive for S. mansoni; 16 patients out of them being moderately positive with titres from 0,30 to 0,40.

All the patients by whom malaria serology was performed (n = 18) were positive.

In our study, we had 3 pregnancies of different ages where the AFP level varied from 4 to 90 ng/ml. Apart from these cases, we had 7/77 (9,1 %) patients with an AFP level higher than 40 ng/ml (range : 160 to 1,8 x 10^6 ng/ml) (see table 8). Among these 7 patients with high AFP levels, we had 3 histologically proven primary hepatocellular carcinomas and 1 liver cell dysplasia, which proved to be malignant in the further course of disease. Among these 4 cases, 3 had a laparoscopic diagnosis of malignant tumour and, in 1 case, we got histology by a post-mortem biopsy. One case (male, 32 years of age; n° 29) presented a multifocal tumour of the liver at laparoscopy and a histology of primary hepatocellular carcinoma and chronic persistent hepatitis. No AFP was detected!

47 patients (58,7 %) out of our test group had an IgM level above the upper limit (up to more than 25 g/L). IgM determination helped us mainly in diagnosing the Tropical Splenomegaly Syndrome (TSS) among other criteria. 3 patients were diagnosed having a TSS.

The serological findings are summarized in table 9.

Serology changed the previous diagnosis in 7 cases (8,75 %) (see table 18).

Laparoscopy

Laparoscopy could be performed in 52 patients (32 males and 20 females) out of 80 and was not possible in 28 patients because of contraindications (14 patients), spontaneous recovery or improvement after therapy (4 patients). 5 patients refused laparoscopy, 4 patients died and one was discharged from the hospital before laparoscopy.

The age distribution in patients with laparoscopy is given in fig. 10.

No complications due to laparoscopy was registered in 47 patients out of 52 (90%). One cirrhotic patient got fever within 12 hours of the laparoscopy. She improved quickly after Cotrimoxazol therapy. Four patients died after laparoscopy, one of coma probably due to hepatic encephalopathy; one of cardiac failure most likely due to overinfusion; one due to a late stage of an hepatoma and one after a non haemorragic shock of unknown reason.

Laparoscopy changed in 46 % the previous diagnosis.

Liver biopsy

In our test group (n = 80), we got 54 liver biopsies (67,5 %): 48 during the 52 laparoscopies and 6 post mortem biopsies. A biopsy was not possible during laparoscopy in 4 patients due to long-lasting cholestasis (1 case), abscess (1 case) and adhesions (2 cases) (fig. 11).

In 26 cases, the previous diagnosis was modified

after histology.

Final diagnosis

All the results of the diagnostic steps are reported in table 18. A report on the different diagnostic entities is given in tables 10 to 15.

Out of 80 patients presenting with a liver disease, the final diagnosis was made possible after clinical examination alone in 20/80 (25 %); after laboratory alone in 3/80 (4 %); after serology alone in 3/80 (4 %); after laparoscopy alone in 11/80 (14 %) and histology alone in 36/80 (45 %). The 7 remaining final diagnoses were issued from many steps together. The effectiveness of these investigatory methods is given in table 16.

Evaluation of the diagnostic approach

We decided to compare only those cases where all the diagnostic steps were performed. Therefore, we eliminated 28 cases where laparoscopy was not done, 1 case where the liver was not observed and 3 cases where a liver biopsy could not be performed during the laparoscopy. So, 48 patients remained for a proper evaluation. Out of them, 18 patients (37,5 %) had a similar diagnosis after each step up to the final diagnosis. The remaining 30 patients (62,5 %) had their final diagnosis based mainly on laparoscopic diagnosis in 12 cases; on clinical diagnosis in 4 cases and on histology in 14 cases. Histology was always the base for such a comparative evaluation. A summary is given in table 17.

Evaluation of laparoscopy in a rural hospital

As assumed, laparoscopy could be easily performed in a rural hospital such as St Francis Hospital. This was not the case for histology, which required a special infrastructure and medical specialists. Such conditions were found only in one place in Tanzania. Because of lack of transport and because of the small number of pathologists, biopsies had to be sent to the Institute of Pathology in Basle, Switzerland.

For that reason, we tried to evaluate how laparoscopy alone could help towards diagnosis in such conditions. Thus, in the 48 cases compared, 15 laparoscopic diagnoses were the final diagnoses; in 16 cases laparoscopy helped in a decisive manner to establish the final diagnosis or brought a positive element for the elaboration of the final diagnosis in 12 cases. Only in 3 cases, laparoscopy gave a diagnosis which was completely different and wrong in comparison with the final diagnosis and it did not help at all in 2 cases. Therefore, with regard to the final diagnosis, laparoscopy was of no help in 5 cases out of 48 (10 %).

V. DISCUSSION

The sample of patients was representative for the population distribution of the region, only 11,4 % coming from outside the Kilombero and Ulanga Districts. 85 % of the patients were peasants and 44 % were alcohol consumers, mainly males over 35 years of age.

The epidemiological data of the present study and control population were similar to those of the previous study in 1982 with regard to age, sex and geographical aspects (39) (see tables 5 ,7).

No outbreak of hepatitis occured in the two districts served by the St Francis Hospital during the period of this study.

V. 1. Feasibility of laparoscopy

We did not encounter major difficulties during our study.

Four patients refused a laparoscopy and one absconded from the hospital. 52 out of 80 patients had a laparoscopy and in 48 patients a liver biopsy was taken during the examination. There were no complications related to laparoscopy except that in one patient, praemedication could possibly have induced hepatic encephalopathy. Three patients died during the days after laparoscopy because of their advanced liver disease or because of other reasons not related to laparoscopy and liver biopsy.

We had no problems in performing the laparoscopy procedure and the material was always kept in good condition by the staff.

V. 2. Evaluation of the systematic use of laparoscopy

In 1982, a proper final diagnosis could not be assigned to 60 % of the patients presenting with liver disease based on clinical examination, liver function tests (LFT's) and serology (39).

In 52 out of 80 patients studied at present, clinical, laboratory and serological examinations and laparoscopy with histology were performed leading to a final accurate diagnosis in 48 patients (liver not seen in one patient; no liver biopsies possible in 3 patients). A post-mortem biopsy was performed in 6 patients where a laparoscopy could not be done. 2 patients presenting with hepatosplenomegaly and enlarged lymphnodes had a lymphnode biopsy (one of them together with a liver biopsy). Therefore, we had an histological result in 55 patients.

The resulting list of diagnoses shows that liver cirrhosis due to hepatitis or alcohol is frequent (15/55 = 27 %) mainly in men. Schistosomiasis was the possible cause of liver cirrhosis in only one case (case n° 69) (40, 41). Chronic active hepatitis was only found together with liver cirrhosis and these cases were included in the "cirrhosis group" (see table 10).
Malignant liver disease was found in 13 cases out of 55 patients (24 %) namely :

- 5 primary hepatocellular carcinomas of which 4 had elevated AFP levels. All were males. During 1972-1976, 256 (27 %) primary hepatocellular carcinomas were found out of 939 clinically diagnos malignancies in the Dodoma Region (Tanzania) (42 documenting the high incidence of this tumour.
- 2 patients with secondary involvement of the liv by a malignant tumour.

- 2 men with systemic reticulo-endothelial disease, later diagnosed as Kaposi's sarcoma (lymphnode biopsy). In 1964-1965, Kaposi's sarcoma represented 4 % of all malignancies in Tanzania (43).
- 3 women with chronic lymphatic leukaemia.
- 1 undifferentiated malignant liver tumour found in a post-mortem liver biopsy (see table 11).

Inflammatory liver disease was observed in 14 patients (25,5 %): liver abscess in 4 patients and hepatitis or perihepatitis in 10 (see table 12). This relatively high incidence of inflammatory liver diseases is characteristic for tropical countries(11).

Alcoholic liver disease was found in 6 patients (11 %) corresponding to the fact that alcoholism is a very important problem in many parts of Africa (11) (see table 13).

The Tropical Splenomegaly Syndrome was present in 3 cases (4 %). In one of them, it was associated with liver cirrhosis secondary to chronic active hepatitis (see table 14).

In the remaining 3 cases, we had one patient with malaria presenting with liver siderosis and intrasinusoidal lymphocytosis and one patient presenting with tuberculosis. He was already under specific therapy for pulmonary tuberculosis. Laparoscopy showed a tuberculous peritonitis and histology alcoholic liver disease together with INH liver damage and a non-specific granuloma. The last patient was an HBs-carrier and a biopsy performed after death showed shock necrosis of the liver (see table 15).

One patient presenting with ascites had, in fact, peritoneal metastases of an intra-abdominal adenocarcinoma. The liver had no lesion (case n° 74).

The final diagnosis was made by laparoscopy in 12 out of 48 cases (25 %) and by histology in another 14 patients (29 %), where both investigations were performed.

V. 3. Evaluation of each diagnostic step

Out of 80 patients admitted to the study, only 48 patients (60 %) with a full investigation could be used for the evaluation of the diagnostic steps.

16 patients out of 48 had the clinical diagnosis confirmed by each following step. This means that 33 % of the final diagnoses were correctly made by the clinical examination of the patient at entry to the hospital.

Additional laboratory investigations (as ESR, total WBC, LFT's) modified the previous clinical diagnosis in 5 cases and the final diagnosis was correctly obtained by the laboratory examination in 3 cases out of the 48 patients (6 %) where a full investigation was performed.

70/79 patients (89 %) had one or more pathological LFT's (SGPT; GGT; Alk. Phosphatase; Bilirubin; Prothrombine Time). In 1982, Stahel et al (12) found one or more pathological LFT in 63 out of 92 patients (69 %) presenting with liver disorder and in 17/196 controls (9 %) without obvious liver disease. Fakunle observed in 96 % of 75 patients with clinical signs of liver disease at least one abnormal LFT (44). LFT's therefore add little to diagnosis and differential diagnosis of liver disease and should be reserved for the follow-up of liver patients and not routinely used for

initial diagnosis (44). Leucocyte count was useful in chronic lymphatic leukaemia (3 cases) and amoebic liver abscess (1 case). For more details, see table 19.

Serology was decisive for the final diagnosis in 3 cases only out of 48 (6 %). We therefore cannot recommend serology as a routine test for hepatopathies under rural, tropical conditions.

Serology confirmed the high prevalence of hepatitis B in the region (12). It helped us once to determine the amoebic nature of a liver abscess. 35 % (28/80) of the patients were positive for S. mansoni. Probably, most of them have had a previous infection by this parasite not related to the actual liver disease. In one case, a schistosomiasis granuloma was observed in the biopsy. In the 3 cases of Tropical Splenomegaly Syndrome, high titres of malaria led us to the diagnosis along with other criteria (gross splenomegaly, high IgM levels, hepatic sinusoidal lymphocytosis, hypersplenism). IgM levels were higher than those encountered in Europe and North America (0,4 - 1,5g/L) (27, 33 , 45). The high number of cases where IgM level was markedly elevated is difficult to explain. Numerous inflammatory diseases and cirrhosis could explain this finding (46), but not in all cases. The most useful laboratory test was AFP determination (34 , 46 , 47 , 48 , 49 , 50). It can raise during pregnancy (48 , 51) as we have seen in 2 out of 3 pregnant women in our study.AFP was very high in 5 and negative in one patient presenting with hepatoma.

In 26 patients out of 48 (54 %), the proper final diagnosis could not be reached by clinical

examination, laboratory tests and serology without laparoscopy and histology.

The correlation between laparoscopy (macroscopic view) and histology (microscopic view) was excellent, and both informations were important for the final diagnosis. This was true especially for cirrhosis (52 , 53 , 54) where histology may not give the diagnosis which is evident through laparoscopy (4 cases out of 15 liver cirrhoses in our study). In some cases, laparoscopy could replace a diagnostic laparotomy and sometimes could be used to drain a liver abscess (1 case in our study).

The value of laparoscopy in developing countries is high (55 ,56 ,57) since it is cost-effective and has a high diagnostic yield (56). It was not possible, however, in our study, to evaluate the cost-effectiveness. The benefit of laparoscopy was to lead to an accurate diagnosis in a short time, without biopsy in 25 % and with biopsy in 54 % of the patients presenting with liver disease in whom the diagnosis could not have been achieved otherwise. We found that laparoscopy was the most important tool to improve the clinical diagnosis in patients with suspected liver disease and ascites (cirrhosis, primary or secondary cancer, tuberculosis). Laparoscopy alone gave false or incorrect answers in 10 % of the cases or, conversely, it helped to make a correct diagnosis partially or definitely in 90 %.

In conclusion, laparoscopy is an efficient, complementary diagnostic tool in a rural, tropical country, even if processed by non-specialist medical officers. Histology is important in confirming the

laparoscopic findings and in increasing the diagnostic yield. Laboratory tests and serology are of limited value and should therefore be reluctantly used.

VI. C O N C L U S I O N S

Sex and age distribution as well as clinical findings were similar to those patients with hepatopathies reported in 1982 (39).

The study was not distorted by an outbreak of hepatitis in the area, as documented by our control group.

Liver cirrhosis (27 %), liver malignancies (24 %), alcohol damages (11 %) and inflammatory liver diseases (25,5 %) were the main disorders encountered.

Clinical findings are always fundamental, cheap and effective for diagnosis. The yield can be improved by the use of laparoscopy.

Laboratory and serology are not efficient enough to be performed routinely. It can not be decided from the study which test is most decisive, cheap and effective.

Laparoscopy is a valuable tool to improve diagnosis. It should be considered not only as a complementary diagnostic method, but also as a teaching aid. Its value is especially high in tropical areas where histology is not always available. The complication rate is low (58, 59, 60, 61, 62, 63). However, it must remain in properly trained hands, with irreprochable technique (64).

The best way, in rural, tropical conditions, would be to determine first, after clinical examination , Haemoglobin, Prothrombine Time and then to perform a laparoscopy. To this could be added a guided needle biopsy, if required. We observed in our study that such an arran gement can produce a positive and correct result in 9 cases out of 10.

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Table 1 : Definitions of clinical diagnosis (26 , 27)

Hepatitis:	Scleral icterus.Hepatomegaly.Liver tenderness.
Icterus accompanying pneumonia:	Same as above.Liver tenderness (not always present) with a concomittant pneumonia (11 ,28).
Liver abscess:	Fever.Liver pain and tenderness. Painful tumour.
Congestive liver disease:	History and signs of cardiac failure.Tender hepatomegaly.Hepatojugular vein reflux.Feet oedema.
Liver cirrhosis:	Hepatomegaly of hard consistency, irregular surface, with or without scleral icterus; with or without cachexia.Gynaecomastia may be present.
Decompensated liver cirrhosis:	The above-mentionned signs with ascites, peripheral oedema and splenomegaly. Signs of encephalopathy may be present.
Malignant liver tumour:	Grossly irregular, hard and tender hepatomegaly associated with severe cachexia, with or without ascites.
Metastatic liver disease:	The same features as above with evident history or proof of a primary cancer.
Portal hypertension:	Abdominal vein distension, ascites, haematemesis and/or melaena.
Systemic reticulo-endothelial disease:	Pronounced hepato-splenomegaly with enlarged lymphnodes.

Table 2 : Reagents for laboratory tests

Tests	through reagents from
SGPT	Hofmann La Roche, Basle (Switzerland)
Gamma GT	Boehringer, Mannheim (West Germany)
Alkaline Phos-phatase	Boehringer, Mannheim (West Germany)
Bilirubin	Boehringer, Mannheim (West Germany)
Prothrombine Time	Thromboplastin C, Dade Diagnostics Inc., Aguada (Puerto Rico)
Proteins and Electrophoresis	Helena Laboratories, Beaumon, Texas (USA)

Table 3 : Reference values for haematology and blood chemistry

Blood sedimentation rate (ESR)	Male (M) Female (F)	Age + 2 (Age + 10) + 2
Leucocytes Count		$4,3 - 10.0 \times 10^9/L$
Haemoglobin (HB)	M F	13,0 ± 2,4 g% 11,3 ± 2,4 g%
Thrombocytes Count		$140 - 440 \times 10^9/L$
SGPT	M F	up to 22 U/L up to 17 U/L
GGT	M F	6 - 28 U/L 4 - 18 U/L
Alkaline Phosphatase		60 - 170 U/L
Prothrombine Time (Quick)		70 - 100%
Bilirubin		Total up to 17 mcmol/L Direct up to 4,3 mcmol/L Indirect up to 12,7 mcmol/L
Albumin		2,1 - 3,9 g%
Globulins		2,4 - 4,2 g%
Albumin/Globulins Ratio (A/G)		0,58 - 1,26

Source:

1. A. Miller et al : Simple Rule for Calculating Normal Erythrocyte Sedimentation Rate, Br. Med. J. 286:266 (1983)
2. M.M. Wintrobe : Clinical Hematology. 8th edition. (Lea & Febiger, Philadelphia 1981)
3. Control group, phase 1; Ifakara (Tanzania 1982)
4. Boehringer, Mannheim (West Germany)
5. Control Group, Quick; Ifakara (Tanzania 1984)
6. Control Group, proteins, Kikwawila (Tanzania 1983)

Table 4 : Material for laparoscopy

Jacobs-Palmer's Operating Laparoscope	(Wolf GmbH., Knittlingen, West Germany)
Trocar sleeve with piston valve	
Light Projector	(Wolf GmbH. 4015, Knittlingen, West Germany)
Siede's Insufflator N2O-Pneu Automat	(Wolf GmbH. 2145, Knittlingen, West Germany)
Veres' Cannula	
Sterile Stethoscope	
Scalpel and Blades	
Menghini needles 1,2 and 1,4 mm	
Liver biopsy cannula (440 mm long) with Menghini's syringe for the endoscope	
Clips	

Table 5 : General comparison between the three groups tested (two from the present study and one from the first study in 1982).

	Control group 1983	Test group 1983	Test group 1982
N	80	80	92
Males	70%	63,8%	68,5%
Females	30%	36,2%	31,5%
Mean Age	37,6 years	39,9 years	35,9 years
Living in:			
Ifakara	41,6%	34,2%	
Kilombero & Ulanga	52 %	54,4%	
Outside	6,5%	11,4%	
Tribe:			
Kilombero V.	80,5%	76,3%	
Outside	19,5%	23,7%	

Table 6 : Clinical Data

Scleral icterus	23/80	(28,8%)
Liver size > 12 cm	39/70	(55,7%)
Hard liver	36/70	(51,4%)
Irregular surface	26/70	(37,1%)
Tender liver	36/78	(46,2%)
Right upper quadr.tenderness	23/78	(29,5%)
Abdominal vein distension	10/70	(12,5%)
Gynaecomastia (males only)	2/51	(3,9%)
Melaena and/or haematemesis	never objectivated	
Splenomegaly (Hackett 2 and over)	28/75	(37,3%)
Axillar and pubic hair loss	16/78	(20,5%)
Ascites	25/79	(31,6%)
Peripheral oedema	19/80	(23,8%)
Cachexia	19/79	(24,1%)

N.B.:The denominator indicates the number of registered data.
Axillar and pubic hair loss were difficult to assess because people are usually shaving themselves.

Table 7 : Comparison between the two test groups for some physical signs

Signs	Test group (N=92) 1982	Test group (N=80 1983
Icterus	17,4%	28,8%
Liver size > 12 cm	77,1%	55,7%
Right upper quadrant tenderness	33,7%	29,5%
Abdominal vein distension	9,8%	12,5%
Gynaecomastia	3,2%	3,9%
Spleen > Hackett 2	20,6%	37,3%
Ascites	10,9%	31,6%
Peripheral oedema	11,9%	23,8%

Table 8 : High AFP levels and diagnosis

Case No.	AFP level	Histology	Sex	Final diagnosis
7	$1,8 \times 10^6$ ng/ml	Primary hepato-cellular carcinoma	M	Primary hepato-cellular carcinoma Liver cirrhosis
8	$3,2 \times 10^3$ ng/ml	None	M	Clinical & sero-logical primary hepatocellular carcinoma
43	26×10^3 ng/ml	Primary hepato-cellular carcinoma	M	Primary hepato-cellular carcinoma
49	160 ng/ml	None	M	Clinical liver cirrhosis
56	300 ng/ml	Liver cell dys-plasia	M	Primary hepato-cellular carcinoma Liver cirrhosis
73	$0,9 \times 10^6$ ng/ml	Primary hepato-cellular carcinoma	M	Primary hepato-cellular carcinoma Liver cirrhosis
76	160 ng/ml	Liver cirrhosis Chronic active hepatitis	F	Liver cirrhosis in Chronic active hepatitis (HB_sAG +)

Table 9 : Serology data compared to those found in 1982.

Serology	Number of cases	Frequency	Frequency in 1982 (N=92)
HBsAg +	21/80	26,3%	14,1%
AntiHBs +	47/80	58,8%	62,0%
AntiHBc +	79/80	98,8%	78,3%
HBV exposure rate	80/80	100,0%	83,7%
Amoeba ↑	4/80	5,0%	19,6%
Schistosomiasis ↑	28/80	35,0%	46,8%
Malaria ↑	18/18	100,0%	
AFP ↑	7/77	9,1%	
IgM total ↑	47/80	58,7%	

NB: Malaria serology was performed only in the patients where a Tropical Splenomegaly Syndrome could be suspected (N=18).

Table 10 : Liver cirrhosis

⊛ = fully investigated cases PM= post-mortem biopsy

Diagnosis	Total	MALES 15-24	25-34	35-44	45-54	55-70	FEMALES 15-24	25-34	35-44	45-54	55-70
Clinical liver cirrhosis / HB_sAg +	5		**	**		*					
HB_sAg -	1					*					
Liver cirrhosis in CAH / HB_sAg +	3	⊛	⊛						⊛		
HB_sAg -	1				⊛						
Same + Alcoholic liver disease	1		⊛								
Liver cirrhosis / HB_sAg +	0										
HB_sAg -	3		⊛			⊛			⊛		
Liver cirrhosis + NRH	1							⊛			
Alcoholic liver cirrhosis	2				⊛					⊛ PM	
Alcoholic liver cirrhosis + siderosis	3			⊛		⊛				⊛	
Clinical liver cirrhosis + alcoholism	2	*			*						
Liver cirrhosis HB_sAg - and liver schistosomiasis	1								⊛		
	23	2	5	3	3	4	0	1	3	2	0

Diagnosis	Total	MALES 15–24	25–34	35–44	45–54	55–70	FEMALES 15–24	25–34	35–44	45–54	55–70
PHC AFP + HB_sAg +	1			⊛ PM							
AFP + HB_sAg −	0										
AFP = 0 + HB_sAg +	0										
AFP = 0 + HB_sAg −	0										
Clin., serol. PHC	1				*						
PHC + liver cirrhosis HB_sAg +	2		⊛		⊛						
HB_sAg −	1			⊛							
PHC and chronic persistent hepatitis	1		⊛								
Metastatic liver disease	2		⊛								⊛
Clinical metastatic liver disease	1									*	
Peritoneal metastasis of an adenocarcinoma	1					⊛					
RES disease	2	⊛ LN		⊛ LN							
Tumoral ascites	1					*					
CLL	2								⊛	⊛	
CLL + HB_s carrier	1									⊛	
Indifferentiated malignant liver tumour	1							⊛ PM			
Malignant liver tumour	1		*								
	18	1	4	3	2	2	0	1	1	3	1

Table 12 : Inflammatory liver disease

⊛ = fully investigated cases

Diagnosis	Total	MALES					FEMALES				
		15–24	25–34	35–44	45–54	55–70	15–24	25–34	35–44	45–54	55–70
Clinical liver abscess	1								*		
Old amoebic abscess	1					⊛					
Old liver abscess	2				⊛			⊛			
Amoebic liver abscess	1			*							
Pyogenic liver abscess	1		⊛								
Granulomatous hepatitis	1				⊛ PM						
NRH + fibrosis	1				⊛						
NRH (HB_sAg –)	3	⊛		⊛			⊛				
NRH + liver congestion	1			⊛ PM							
NRH + ALD	1					⊛					
Acute hepatitis	1								⊛		
Perihepatitis											
" + HSM	1						⊛				
" + NRH	1								⊛		
	16	1	1	3	3	2	2	1	3	0	0

Table 13 : Alcoholic liver disease (ALD)

⊛ = fully investigated cases

Diagnosis	Total	MALES					FEMALES				
		15–24	25–34	35–44	45–54	55–70	15–24	25–34	35–44	45–54	55–70
ALD alone	2					⊛				⊛	
ALD + HB_s carrier	1				⊛						
ALD + NRH and siderosis	1			⊛							
ALD + NRH and fibrosis	1					⊛					
ALD + Granulomatous hepatitis	1							⊛			
	6	0	0	1	1	2	0	1	0	1	0

Table 14 : Tropical splenomegaly syndrome (TSS)

⊛ = fully investigated cases

Diagnosis	Total	MALES					FEMALES				
		15–24	25–34	35–44	45–54	55–70	15–24	25–34	35–44	45–54	55–70
TSS	2			⊛							⊛
TSS + liver cirrhosis in CAH / HB_sAg +	1						⊛				
	3	0	0	1	0	0	1	0	0	0	1

Table 15 : Miscellaneous

⊛ = fully investigated cases

Diagnosis	Total	MALES 15–24	MALES 25–34	MALES 35–44	MALES 45–54	MALES 55–70	FEMALES 15–24	FEMALES 25–34	FEMALES 35–44	FEMALES 45–54	FEMALES 55–70
Congestive liver disease (CLD)	2				**						
CLD + HB_S carrier	1							*			
Malaria	1							⊛			
Hepatosplenomegaly of unknown origin	1						*				
Haemolytic anaemia and pregnancy (before or just after delivery)	2						**				
Icterus in pneumonia	1				*						
Extrahepatic cholostasis	1										*
Right upper quadrant tenderness and splenomegaly	1			*							
Shock necrosis of the liver + $HB_S Ag$ carrier	1							⊛ PM			
Infectious disease (bacterial, tuberculous)	2	*	⊛								
Upper abdominal adhesions	1					*					
	14	1	1	1	3	1	3	3	0	0	1

Table 16 : Effectiveness of investigation group for determining the final diagnosis.

Clinic	20/80	(25,00%)
Laboratory & Serology	7/80	(8,75%)
Laboratory & Histology	1/80	(1,25%)
Serology & Histology	2/80	(2,50%)
Laparoscopy & Histology	49/80	(61,25%)
Laparoscopy & Serology	1/80	(1,25%)
	80/80	(100%)

Table 17: Number of the final diagnoses given by each investigatory method separetely.

Clinic	Laparoscopy	Histology
22/48	12/48	14/48
22/48	26/48	

Table 18 :

Step by step diagnoses and special attention to those which can be done at Ifakara without external support

Abbreviations:

F	female
M	male
LAB	laboratory
SEROL	serological
LAP	laparoscopic
TSS	Tropical Splenomegaly Syndrome
CAH	Chronic active hepatitis
NRH	Non-specific reactive hepatitis
AFP	Alpha fetoprotein

Case No.	Sex	Age	Clinical Diagnosis	Clin. + Lab. Diagnosis	Clin. + Lab. + Serol. Diagnosis	Laparoscopic Diagnosis	Histology	Final Diagnosis	IFAKARA	
									Clin.+ Lab. Diagnosis	Clin.+ Lab.+ Lap. Diagnosis
1	M	30	Clinical liver cirrhosis	same	Clinical liver cirrhosis (HB_sAg +)	——	——	Clinical liver cirrhosis (HB_sAg +)	Clinical liver cirrhosis	
2	M	60	Liver tumour	same	same	Upper abdominal adhesions	——	Upper abdominal adhesions	Liver tumour tumour	Upper abdominal adhesions
3	M	30	Indurated liver	Clinical liver cirrhosis	Clinical liver cirrhosis (HB_sAg +)	Chronic hepatitis	- Liver cirrhosis - Chronic active hepatitis (intracytoplasmic HB_sAg -)	Liver cirrhosis by chronic active hepatitis HB_sAg +	Clinical liver cirrhosis	Chronic hepatitis
4	M	55	Clinical liver cirrhosis	same	Clinical liver cirrhosis (HB_sAg +)	——	——	Clinical liver cirrhosis (HB_sAg +)	Clinical liver cirrhosis	
5	M	49	Clinical liver cirrhosis	same	Clinical liver cirrhosis (HB_sAg +)	Left lobe tumour	- Liver siderosis - Non-specific reactive hepatitis - Perivenular fibrosis	- Alcoholic liver disease - HB_sAg carrier	Clinical liver cirrhosis	Liver tumour
6	M	30	Clinical liver cirrhosis	same	same (HB_sAg -)	- Right lobe tumour - Peritoneal metastasis	Carcinoma adenomatosum peritoneal metastasis	Adenocarcinoma of unknown origin metastatic to the liver and peritoneum	Clinical liver cirrhosis	Malignant liver tumour and peritoneal metastasis
7	M	31	Malignant liver tumour	same	Primary hepatocellular carcinoma (HB_sAg +)	- Malignant liver tumours - Liver cirrhosis	- Sinusoidal trabecular primary hepatocellular carcinoma (intracytoplasmic HB_sAg -) - Liver siderosis	Primary hepatocellular carcinoma Liver cirrhosis	Malignant liver tumour	Malignant liver tumour Liver cirrhosis
8	M	52	Congestive liver disease	same	Primary hepatocellular carcinoma (HB_sAg +)	——	——	Clinical, serological primary hepatocellular carcinoma	Congestive liver disease	

Case No.	Sex	Age	Clinical Diagnosis	Clin. + Lab. Diagnosis	Clin. + Lab. + Serol. Diagnosis	Laparoscopic Diagnosis	Histology	Final Diagnosis	I P A K A R A	
									Clin.+ Lab. Diagnosis	Clin.+ Lab.+ Lap. Diagnosis
9	M	36	Reticulo-endothelial system disease	same	same	——	—— Lymphnode biopsy: Kaposi's sarcoma	Reticulo-endothelial system disease	Reticulo-endothelial system disease	
12	M	43	Icterus and pneumonia	Cholestasis and bacterial pneumonia	same	Normal liver	- Non-specific reactive hepatitis - Liver siderosis	Alcoholic liver disease with non-specific reactive hepatitis and liver siderosis	Cholestasis and bacterial pneumonia	same
13	F	48	Metastatic liver disease	same	same	——	——	Metastatic liver disease	Metastatic liver disease	
14	F	45	Clinical liver cirrhosis	same	same (HB_sAg -)	——	- Alcoholic cirrhosis - Alcoholic hepatitis	Alcoholic liver cirrhosis	Clinical liver cirrhosis	
15	F	27	Congestive liver disease	same	same (HB_sAg -)	——	——	Congestive liver disease HB_sAg carrier	Congestive liver disease	
16	M	24	Reticulo-endothelial system disease	same	same	Perisplenitis	- Non-specific reactive hepatitis - Siderosis - Intrasinusoidal lymphocytosis Lymphnode biopsy: Kaposi's sarcoma	Reticulo-endothelial system disease	Reticulo-endothelial system disease	same
17	M	50	Congestive liver disease	same	same	——	——	Congestive liver disease	Congestive liver disease	

Case No.	Sex	Age	Clinical Diagnosis	Clin. + Lab. Diagnosis	Clin. + Lab. + Serol. Diagnosis	Laparoscopic Diagnosis	Histology	Final Diagnosis	I F A K A R A	
									Clin.+ Lab. Diagnosis	Clin.+ Lab.+ Lap. Diagnosis
18	F	20	Indurated hepato-splenomegaly	same	same (HB_sAg +)	- Micronodular liver cirrhosis - Splenomegaly	Chronic active hepatitis Posthepatitic cirrhosis (intra-cytoplasmic HB_sAg -) Intrasinusoidal lymphocytosis	- TSS - Liver cirrhosis by CAH (HB_sAg +)	Indurated hepato-splenomegaly	- Liver cirrhosis - Splenomegaly
19	F	55	Hepatosplenomegaly	Liver cirrhosis	same (HB_sAg -)	Liver fibrosis Splenomegaly	- Fibrosis - Intrasinusoidal lymphocytosis	TSS	- Clinical liver cirrhosis - Splenomegaly	Liver fibrosis Splenomegaly
20	F	30	Clinical liver cirrhosis	same	same (HB_sAg +)	Medionodular liver cirrhosis Portal hypertension	Liver cirrhosis (intracytoplasmic HB_sAg -) Non-specific reactive hepatitis	Liver cirrhosis (HB_sAg +) Non-specific reactive hepatitis	Clinical liver cirrhosis	Liver cirrhosis
22	F	22	Icterus	Haemolytic anaemia	same	—	—	Haemolytic anaemia	Haemolytic anaemia	
23	F	35	Liver abscess	same	same	—	—	Clinical liver abscess	Clinical liver abscess	
25	M	50	Icterus and peritonitis	Cholangitis	same	—	Granulomatous hepatitis	Granulomatous hepatitis	Cholangitis	
26	F	18	Icterus and pregnancy	Haemolytic anaemia	same	—	—	Haemolytic anaemia in pregnancy	Haemolytic anaemia in pregnancy	
27	M	45	Icterus and pneumonia	same	same	Normal liver	Fibrosis NRH	NRH with fibrosis (lober pneumonia)	Icterus and pneumonia	same
28	M	52	Indurated hepato-splenomegaly	same	same (HB_sAg -)	Medionodular liver cirrhosis	Alcoholic liver disease	Alcoholic liver cirrhosis	Indurated hepato-splenomegaly	Liver cirrhosis

Case No.	Sex	Age	Clinical Diagnosis	Clin. + Lab. Diagnosis	Clin. + Lab. + Serol. Diagnosis	Laparoscopic Diagnosis	Histology	Final Diagnosis	I F A K A R A	
									Clin.+ Lab. Diagnosis	Clin.+ Lab.+ Lap. Diagnosis
29	M	32	Clinical liver cirrhosis	same	same (HB_sAg -) (AFP = 0)	Liver tumours	- Chronic persistent hepatitis - Primary hepatocellular carcinoma	- Primary hepatocellular carcinoma - Chronic persistent hepatitis	Clinical liver cirrhosis	Malignant liver tumours
30	M	38	Hepatosplenomegaly	same	same	same	Intrasinusoidal lymphocytosis	TSS	Hepatosplenomegaly	same
31	M	28	Clinical liver cirrhosis	same	same (HB_sAg +)	Macronodular liver cirrhosis Portal hypertension	- Liver cirrhosis - CAH - Alcoholic liver disease	Liver cirrhosis by CAH (HB_sAg +) and alcoholic liver disease	Clinical liver cirrhosis	Liver cirrhosis
33	M	56	Liver tumour	same	same	Old liver abscess	Siderosis Abscess	Old amoebic abscess	Liver tumour	Old liver abscess
35	M	53	Congestive liver disease	same	same	——	——	Congestive liver disease	Congestive liver disease	
36	M	50	- Liver tumour - Splenomegaly	same	same	Old liver abscess Liver steatosis	Siderosis Intrasinusoidal lymphocytosis	Old liver abscess	Liver tumour	Old liver abscess
38	M	38	Liver tumours	same	same	Medionodular liver cirrhosis Portal hypertension	Liver cirrhosis and siderosis	Alcoholic liver cirrhosis with siderosis	Liver tumours	Liver cirrhosis
39	M	62	Clinical liver cirrhosis	same	same (HB_sAg -)	Medionodular liver cirrhosis	Liver cirrhosis and siderosis	Alcoholic liver cirrhosis with siderosis	Clinical liver cirrhosis	Liver cirrhosis

Case No.	Sex	Age	Clinical Diagnosis	Clin. + Lab. Diagnosis	Clin. + Lab. + Serol. Diagnosis	Laparoscopic Diagnosis	Histology	Final Diagnosis	I F A K A R A	
									Clin.+ Lab. Diagnosis	Clin.+ Lab.+ Lap. Diagnosis
40	M	24	- Liver cirrhosis with proved oesophageal varices - Alcoholism	same	same (HB_gAg -)	——	——	- Clinical liver cirrhosis - Alcoholism	- Clinical liver cirrhosis - Alcoholism	
41	M	35	Right upper quadrant tenderness Splenomegaly	same	same	——	——	Right upper quadrant tenderness Splenomegaly	- Right upper quadrant tenderness - Splenomegaly	
42	M	52	Clinical liver cirrhosis Alcoholism	same	same (HB_gAg -)	——	——	Clinical liver cirrhosis Alcoholism	- Clinical liver cirrhosis - Alcoholism	
43	M	40	Clinical liver cirrhosis Alcoholism	same	Primary hepato-cellular carcinoma (HB_gAg +)	——	Sinusoidal, trabecular primary hepatocellular carcinoma (intracytoplasmic HB_gAg +)	Primary hepato-cellular carcinoma	- Clinical liver cirrhosis - Alcoholism	
44	F	60	Liver tumour	same	same	Liver abscess	Carcinoma adenomatosum metastasis	Metastatic liver disease	Liver tumour	Liver abscess
45	M	60	Liver tenderness	same	same	Liver fibrosis	Alcoholic liver disease	Alcoholic liver disease	Liver tenderness	Liver fibrosis
46	M	65	Clinical liver cirrhosis	Ascites of unknown origin	same	- Old peri-hepatitis - Tumoral ascites	——	Tumoral ascites	Ascites of unknown origin	Tumoral ascites Old perihepatitis
47	F	25	Old liver abscess	same	same	Right upper quadrant adhesions	NRH	Old probable pyogenic liver abscess	Old liver abscess	same

Case No.	Sex	Age	Clinical Diagnosis	Clin. + Lab. Diagnosis	Clin. + Lab. + Serol. Diagnosis	Laparoscopic Diagnosis	Histology	Final Diagnosis	I F A K A R A	
									Clin.+ Lab. Diagnosis	Clin.+ Lab.+ Lap. Diagnosis
48	M	37	Liver abscess	same	Amoebic liver abscess	Liver abscess with peri-hepatitis	——	Amoebic liver abscess	Liver abscess	same
49	M	34	Clinical liver cirrhosis	same	Clinical liver cirrhosis (HB_sAg +)	——	——	Clinical liver cirrhosis (HB_sAg +)	Clinical liver cirrhosis	
50	M	60	Clinical liver cirrhosis	same	same (HB_sAg -)	Micronodular liver cirrhosis with portal hypertension and perihepatitis	NRH Intrasinusoidal lymphocytosis	Liver cirrhosis (HB_sAg -)	Clinical liver cirrhosis	Liver cirrhosis
51	M	30	Ascites Pulmonary tuberculosis	same	same	Tuberculous peritonitis	- Alcoholic liver disease - INH liver alteration - Granuloma	Tuberculosis	Ascites and pulmonary tuberculosis	Tuberculosis
52	F	33	Hepatosplenomegaly	Haemolytic anaemia	Malaria	Hepatomegaly	Liver siderosis Intrasinusoidal lymphocytosis	Malaria	Haemolytic anaemia	same
53	M	20	Acute hepatitis	Infectious disease	same	——	——	Infectious disease	Infectious disease	
54	F	35	Acute hepatitis	same	Acute hepatitis (HB_sAg - Anti HB_c +)	Acute hepatitis	Acute hepatitis	Acute hepatitis	Acute hepatitis	same
55	M	70	Clinical liver cirrhosis	same	same (HB_sAg -)	——	——	Clinical liver cirrhosis (HB_sAg -)	Clinical liver cirrhosis	
56	M	50	Malignant liver tumour	same	Primary hepato-cellular carcinoma (HB_sAg +)	Liver tumour Liver cirrhosis Portal hyper-tension	Liver fibrosis with eosinophiles Liver cells dysplasia Liver cirrhosis	Primary hepato-cellular carcinoma Liver cirrhosis (HB_sAg +)	Malignant liver tumour	- Malignant liver tumour - Liver cirrhosis

Case No.	Sex	Age	Clinical Diagnosis	Clin. + Lab. Diagnosis	Clin. + Lab. + Serol. Diagnosis	Laparoscopic Diagnosis	Histology	Final Diagnosis	I P A K A R A	
									Clin.+ Lab. Diagnosis	Clin.+ Lab.+ Lap. Diagnosis
57	F	21	Hepatosplenomegaly	same	same	——	——	Hepatosplenomegaly	Hepatosplenomegaly	
58	M	65	Clinical liver cirrhosis	same	same	Liver steatosis	Alcoholic liver disease Intrasinusoidal lymphocytosis	Alcoholic liver disease	Clinical liver cirrhosis	Liver steatosis
59	M	29	Liver abscess	same	same	Left lobe deep tumour	Space occupying lesion	Pyogenic liver abscess	Liver abscess	Deep liver tumour
60	M	35	Clinical liver cirrhosis	same	same (HB_sAg +)	——	——	Clinical liver cirrhosis (HB_sAg +)	Clinical liver cirrhosis	
61	F	51	Indurated hepatomegaly and splenomegaly	Chronic lymphocytic leukaemia	same	Hepatosplenomegaly	Chronic lymphocytic leukaemia	Chronic lymphocytic leukaemia	Chronic lymphocytic leukaemia	same
62	M	50	Icterus and pneumonia	same	same	——	——	Icterus and pneumonia	Icterus and pneumonia	
63	F	18	Indurated hepatomegaly and splenomegaly	Septic hepatosplenomegaly	same	Perihepatitis Hepatosplenomegaly	Mild portal fibrosis	Perihepatitis Hepatosplenomegaly	Septic hepatosplenomegaly	Perihepatitis Hepatosplenomegaly
64	M	40	Clinical liver cirrhosis	same	same (HB_sAg +)	——	——	Clinical liver cirrhosis (HB_sAg +)	Clinical liver cirrhosis	
65	F	45	Indurated hepatomegaly and splenomegaly	Chronic lymphocytic leukaemia	same (HB_sAg +)	Hepatosplenomegaly	Granulomatous hepatitis	- Chronic lymphocytic leukaemia - HB_sAg carrier	Chronic lymphocytic leukaemia	same
66	F	60	Cholestatic icterus	same	same	Extrahepatic cholestasis	——	Extrahepatic cholestasis	Cholestatic icterus	Extrahepatic cholestasis

Case No.	Sex	Age	Clinical Diagnosis	Clin. + Lab. Diagnosis	Clin. + Lab. + Serol. Diagnosis	Laparoscopic Diagnosis	Histology	Final Diagnosis	IFAKARA	
									Clin.+ Lab. Diagnosis	Clin.+ Lab.+ Lap. Diagnosis
67	F	40	- Clinical liver cirrhosis - Haematemesis	same	same (HB_sAg -)	Macronodular liver cirrhosis Portal hypertension	Liver steatosis Liver fibrosis	Liver cirrhosis (HB_sAg -)	- Clinical liver cirrhosis - Haematemesis	Liver cirrhosis Portal hypertension
68	M	28	Clinical liver cirrhosis	same	same (HB_sAg -)	- Macronodular liver cirrhosis - Portal hypertension	NRH Liver siderosis	Liver cirrhosis (HB_sAg -)	- Clinical liver cirrhosis	Liver cirrhosis
69	F	35	Right upper quadrant tenderness	same	same (HB_sAg -)	- Macronodular liver cirrhosis - Portal hypertension - Enlarged left lobe	Schistosomiasis Granuloma	Liver cirrhosis (HB_sAg -) Liver schistosomiasis	Right upper quadrant tenderness	Liver cirrhosis
70	F	53	Indurated liver Splenomegaly	same	same	Perihepatitis Splenomegaly	NRH Alcoholic liver disease	Alcoholic liver disease	Indurated liver Splenomegaly	Perihepatitis Splenomegaly
71	M	45	Indurated hepatomegaly	same	same (H_sAg -)	Micronodular liver cirrhosis Chronic active hepatitis (intracyto-plasmic HB_sAg -)	Liver siderosis Liver cirrhosis	Liver cirrhosis by CAH (HB_sAg -)	Indurated hepatomegaly	Liver cirrhosis
72	M	15	Chronic hepatitis	same	same (HB_sAg -)	Normal liver	NRH	Non-specific reactive hepatitis (HB_sAg -)	Chronic hepatitis	Normal liver
73	M	37	Malignant liver tumour	same	Primary hepato-cellular carcinoma (HB_sAg -)	Liver tumour Medionodular liver cirrhosis	- Sinusoidal, trabecular primary hepatocellular carcinoma - Liver cirrhosis (intracytoplasmic HB_sAg -)	Primary hepato-cellular carcinoma Liver cirrhosis (HB_sAg -)	Malignant liver tumour	- Malignant liver tumour - Liver cirrhosis

Case No.	Sex	Age	Clinical Diagnosis	Clin. + Lab. Diagnosis	Clin. + Lab. + Serol. Diagnosis	Laparoscopic Diagnosis	Histology	Final Diagnosis	I F A K A R A	
									Clin.+ Lab. Diagnosis	Clin.+ Lab.+ Lap. Diagnosis
74	M	56	Carcinomatous peritonitis	same	same	Intraabdominal malignant tumour and peritoneal metastases	Carcinoma adenomatosum	Peritoneal metastasis of an adeno-carcinoma	Carcinomatous peritonitis	Peritoneal metastases of an intraabdominal malignant tumour
75	F	40	Clinical liver cirrhosis	same	same (HB_sAg -)	Hepatomegaly	Chronic lymphocytic leukaemia	Chronic lympho-cytic leukaemia	Clinical liver cirrhosis	Hepatomegaly
76	F	35	Clinical liver cirrhosis	same	same (HB_sAg +)	Micronodular liver cirrhosis	- Chronic active hepatitis	Liver cirrhosis by CAH (HB_sAg +)	Clinical liver cirrhosis	Liver cirrhosis
77	F	23	Acute hepatitis	same	same (HB_sAg -)	Hepatitis	NRH	NRH (HB_sAg -)	Acute hepatitis	Hepatitis
78	F	35	Right upper quadrant tenderness	same	same	Perihepatitis	NRH	Perihepatitis NRH	Right upper quadrant tenderness	Perihepatitis
79	M	36	Right upper quadrant tenderness	same	same	Hepatospleno-megaly	NRH Intrasinusoidal lymphocytosis	Non-specific reactive hepatitis (HB_sAg -)	Right upper quadrant tenderness	Hepatospleno-megaly
80	M	40	Clinical liver cirrhosis	same	same (HB_sAg -)	——	NRH Liver congestion	Liver congestion NRH	Liver cirrhosis Alcoholism	
81	F	28	Malignant liver tumours	same	same	——	Indifferentiated malignant tumour	Indifferentiated malignant liver tumour	Malignant liver tumours	

Case No.	Sex	Age	Clinical Diagnosis	Clin. + Lab. Diagnosis	Clin. + Lab. + Serol. Diagnosis	Laparoscopic Diagnosis	Histology	Final Diagnosis	I P A K A R A	
									Clin.+ Lab. Diagnosis	Clin.+ Lab.+ Lap. Diagnosis
82	M	17	Clinical liver cirrhosis	same	same (HB_sAg +)	Macronodular liver cirrhosis	- Chronic active hepatitis - Posthepatitic liver cirrhosis (intracytoplasmic HB_sAg +)	Liver cirrhosis by CAH (HB_sAg +)	Clinical liver cirrhosis	same
83	F	30	Indurated hepatomegaly and splenomegaly	same	same	- Liver steatosis - Pericholecystitis - Splenomegaly	Alcoholic liver disease Liver steatosis Tuberculoid granuloma (fresh)	- Alcoholic liver disease - Granulomatous hepatitis (tuberculosis ?)	Indurated hepatomegaly and splenomegaly	- Liver steatosis - Pericholecystitis - Splenomegaly
84	F	50	Clinical liver cirrhosis Alcoholism	same	same (HB_sAg -)	Micronodular liver cirrhosis Portal hypertension	Alcoholic liver cirrhosis Liver siderosis	Alcoholic liver cirrhosis with siderosis	- Clinical liver cirrhosis - Alcoholism	Liver cirrhosis Portal hypertension
85	M	28	Malignant liver tumour	same	same	——	——	Malignant liver tumour	Malignant liver tumour	
86	M	62	Right upper quadrant tenderness	same	same	Right upper quadrant adhesions	NRH Alcoholic liver disease	- Non-specific reactive hepatitis - Alcoholic liver disease	Right upper quadrant tenderness	Right upper quadrant adhesions
87	F	25	Hepatosplenomegaly	same	same (HB_sAg +)	——	Liver shock necrosis NRH	Shock necrosis of the liver HB_sAg carrier	Hepatosplenomegaly	

Table 19 : Laboratory results in regard to final diagnosis

Abbreviations

BSR	Blood sedimentation rate
HB	Haemoglobin
PT	Prothrombine Time
A/G	Albumin/Globulins ratio
CAH	Chronic active hepatitis
PHC	Primary hepatocellular carcinoma
RES	Reticulo-endothelial system
NRH	Non specific reactive hepatitis
ALD	Alcoholic liver disease
CLL	Chronic lymphocytic leukaemia
RUQ	Right upper quadrant

Comments

BSR ↑	above normal value, below 100 mm 1st hour
BSR ↑↑	above 100 mm 1st hour
Leuco ↑	above 10'000/mm^3
Leuco ↑↑	above 15'000/mm^3
Leuco ↑↑↑	above 20'000/mm^3
HB ↓↓	below 7g%
SGPT ↑↑	above 100 U/L
GGT ↑↑	above 1'000 U/L
Alkaline Phosphatase ↑↑	above 1'000 U/L
Total Bilirubin ↑↑	above 100 mcmol/L
Indirect Bilirubin ↑↑	Indirect > Direct
Direct Bilirubin ↑↑	Direct > Indirect
Prothrombine Time ↓	below 70%
Prothrombine Time ↓↓	below or equal to 40%

Case No.	Sex	Age	Final Diagnosis	BSR	Leuco	HB	Thromb	SGPT	GOT	Alk. Ph.	PT	Bili Total	Bili Dir.	Bili Ind.	ALB	GLOB	A/G
1	M	30	Clinical liver cirrhosis (HB_sAg +)	↑	N	↓	↓	N	N	N	↓↓	↑	↑↑	↑	↓	↑	0,29
2	M	60	Upper abdominal adhesions	↑	N	N	N	N	N	↑	N	N	N	N	N	N	1,17
3	M	30	Liver cirrhosis by CAH (HB_sAg +)	↑↑	N	N	N	↑	↑	N	↓	↑	↑	N	↓	↑	0,41
4	M	55	Clinical liver cirrhosis (HB_sAg +)	↑	N	N	N	↑↑	↑	↑	↓	↑↑	↑↑	↑	N	N	0,85
5	M	49	Alcoholic liver disease HB_sAg carrier	↑↑	N	↓↓	↓	N	N	N	↓	N	N	N	N	↓	0,82
6	M	30	Adenocarcinoma of u.o. metastatic to liver and peritoneum	↑	N	↓	↑	N	N	N	N	N	N	N	N	N	0,74
7	M	31	PHC. Liver cirrhosis	↑	N	N	N	↑	↑	↑	↓↓	N	N	N	↓	↑	0,39
8	M	52	Clinical serological PHC	N	N	↓	(↑)	N	↑	↑	N	N	N	N	N	↑	0,62
9	M	36	RES disease	↑↑	N	↓	N	↑	↑	↑↑	↓	N	N	N	↓	↑	0,35
12	M	43	Alcoholic liver disease with NRH and siderosis	↑↑	N	↓	(↑)	N	N	N	N	↑	↑↑	↑	↓	N	0,56
13	F	48	Metastatic liver disease	↑↑	↑	↓	(↓)	N	↑	↑↑	↓↓	↑	↑↑	N	↓	N	0,64
14	F	45	Alcoholic liver cirrhosis	↑↑	N	↓	N	N	N	N	↓	N	N	N	↓	↑	0,33
15	F	27	Congestive liver disease HB_sAg carrier	↑	N	N	↑	N	N	N	N	N	N	N	N	N	0,88
16	M	24	RES disease	↑	N	↓	↓	N	N	N	↓	N	N	N	N	N	0,70
17	M	50	Congestive liver disease	N	N	N	N	N	↑	N	N	N	N	N	N	N	0,73
18	F	20	TSS. Liver cirrhosis by CAH (HB_sAg +)	↑	N	↓↓	↓	N	N	N	N	N	N	N	N	N	0,69
19	F	55	TSS	↑↑	N	↓	↓	N	N	↑	↓	N	N	N	↓	↑	0,36
20	F	30	Liver cirrhosis (HB_sAg +) NRH	↑	N	↓	↓	↑	N	↑	↓	↑	↑↑	↑	↓	↑	0,42
22	F	22	Haemolytic anaemia	↑	N	↓	↓	N	N	N	N	N	N	N	N	N	0,63
23	F	35	Clinical liver abscess	↑	↓	↓	↓	N	N	N	–	N	N	N	N	N	0,87
25	M	50	Granulomatous hepatitis	↑↑	↑↑	↓↓	N	↑	N	↑	↓↓	↑↑	↑↑	↑	↓	↓	0,87
26	F	18	Haemolytic anaemia in pregnancy	↑↑	↑	↓↓	N	N	N	↑	N	↑	↑↑	↑	↓	N	0,71
27	M	45	NRH with fibrosis (lobar pneumonia)	–	↑	N	↑	N	↑	N	N	↑	↑↑	↑	N	N	0,71
28	M	52	Alcoholic liver cirrhosis	–	N	↓↓	N	N	N	N	↓	↑	↑	↑↑	N	N	1,20
29	M	32	PHC Chronic persistent hepatitis	↑↑	↑	↓↓	↑	↑	↑	↑	↓	N	N	N	↓↓	N	0,53
30	M	38	TSS	↑	N	↓	↓	N	↑	↑	N	N	N	N	N	N	1,14

#	Sex	Age	Final Diagnosis	BSR	Leuco	HB	Thromb	SGPT	GGT	Alk. Ph.	PT	Bili Total	Bili Dir.	Bili Ind.	ALB	GLOB	A/G
1	M	28	Liver cirrhosis by CAH (HB_sAg +) + ALD	↑	N	↓	↓	↑	↑	N	↓	N	N	N	↓↓	N	0,54
3	M	56	Old amoebic abscess	↑	↓	N	N	N	↑	N	N	N	N	N	N	N	0,91
5	M	53	Congestive liver disease	↑	↓	N	↓	N	↑	N	↓	↑	↑	↑↑	N	↑	0,81
6	M	50	Old liver abscess	↑↑	N	N	N	N	↑	N	↓	N	N	N	N	↑	0,52
8	M	38	Alcoholic liver cirrhosis + siderosis	↑	N	↓	↓	↑	↑	↑	↓	N	N	N	↓	N	0,73
9	M	62	Alcoholic liver cirrhosis + siderosis	N	N	N	↓	↑	↑	↑	N	N	N	N	N	↑	0,66
0	M	24	Clinical liver cirrhosis Alcoholism	N	N	N	N	N	↑	N	↓↓	N	N	N	N	N	0,81
1	M	35	RUQ tenderness Splenomegaly	N	↓	N	↓	N	N	N	N	N	N	N	N	N	1,14
2	M	52	Clinical liver cirrhosis Alcoholism	↑	↓	↓	↓	N	↑	↑	↓↓	↑	↑	↑↑	N	↑	0,47
3	M	40	PHC	↑↑	N	↓	↑	↑	↑	N	↓	↑	↑	N	↓	↑	0,42
4	F	60	Metastatic liver disease	↑	N	↓	↑	N	N	N	N	N	N	N	N	↓	1,04
5	M	60	ALD	↑	N	↓	N	N	N	N	↓	N	N	N	N	N	1,03
6	M	65	Tumoral ascites	N	N	N	N	N	N	N	N	N	N	N	N	N	0,76
7	F	25	Old probable pyogenic liver abscess	N	N	N	↑	N	N	N	↓	N	N	N	N	N	1,16
8	M	37	Amoebic liver abscess	↑↑	↑↑	↓	↑	N	↑	↑	↓	N	N	N	↓	↑	0,47
9	M	34	Clinical liver cirrhosis (HB_sAg +)	↑	N	N	↓	↑	N	N	↓↓	↑	↑↑	↑	↓	↑	0,34
0	M	60	Liver cirrhosis (HB_sAg -)	↑↑	N	↓↓	N	↑	↑	↑	N	N	N	N	↓	↑	0,31
1	M	30	Tuberculosis	↑	N	↓	↓	N	↑	↑	N	↑	↑	↑↑	N	↑	0,54
2	F	33	Malaria	N	↑	↓↓	N	↑	N	↑	N	↑	↑	↑↑	N	N	0,85
3	M	20	Infectious disease	↑↑	N	↓	↓	↑	↑	↑	↓	↑↑	↑↑	↑	↓	↑	0,38
4	F	35	Acute hepatitis	↑	N	N	N	↑↑	↑	↑	N	↑↑	↑↑	N	N	N	0,90
5	M	70	Clinical liver cirrhosis (HB_sAg -)	N	N	↓	↓	N	N	N	↓↓	↑	↑↑	↑	↓	N	0,59
6	M	50	PHC. Liver cirrhosis (HB_sAg +)	N	↓	N	↓	↑	↑	↑	↓	↑	(↑)	↑↑	N	N	0,93
7	F	21	Hepatosplenomegaly	↑↑	↓	↓↓	↓	N	N	↑	N	↑	↑↑	↑	↓	N	0,55
8	M	65	Alcoholic liver disease	↑	N	N	↓	N	N	N	↓	N	N	N	N	N	0,76
9	M	29	Pyogenic liver abscess	↑	N	N	N	↑	↑	↑	↓↓	N	N	N	↓	N	0,69
0	M	35	Clinical liver cirrhosis (HB_sAg +)	-	N	N	↓	N	N	↑	↓↓	↑	N	↑↑	↓	↑	0,32

Case No.	Sex	Age	Final Diagnosis	BSR	Leuco	HB	Thromb	SGPT	GGT	Alk. Ph.	PT	Bili Total	Bili Dir.	Bili Ind.	ALB	GLOB	A/G
1	F	51	CLL	↑	↑↑↑	↓	↓	N	N	↑	↓	N	N	N	N	N	1,02
2	M	50	Icterus and pneumonia	↑↑	N	↓	↑	N	N	N	↓	N	N	N	N	N	0,64
3	F	18	Perihepatitis Hepatosplenomegaly	↑↑	↑	N	N	N	N	N	↓	N	N	N	N	↑↑	0,51
4	M	40	Clinical liver cirrhosis (HB_sAg +)	↑	N	N	↓	N	↑	↑	↓↓	↑	↑↑	↑	↓	↑	0,36
5	F	45	CLL HB_sAg carrier	↑↑	↑↑↑	↓↓	N	N	N	N	↓	↑	↑	↑↑	N	↑	0,67
6	F	60	Extrahepatic cholestasis	↑	N	N	N	↑	↑↑	↑↑	N	↑↑	↑↑	↑	N	N	0,87
7	F	40	Liver cirrhosis (HB_sAg -)	N	↑↑↑	↓↓	↓	N	N	N	↓	N	N	N	N	N	0,99
8	M	28	Liver cirrhosis (HB_sAg -)	↑↑	↓	↓	N	↑	↑	↑	↓	↑	↑↑	↑	N	N	0,58
9	F	35	Liver cirrhosis (HB_sAg -) Liver schistosomiasis	↑	N	↓	N	↑	↑	↑	↓	N	N	N	N	↑	0,58
10	F	53	Alcoholic liver disease	N	↓	↓	N	N	N	↑	↓	(↑)	↑	N	N	N	0,88
11	M	45	Liver cirrhosis by CAH (HB_sAg -)	↑	N	N	N	↑	↑	↑	↓	N	N	N	N	N	0,75
12	M	15	NRH (HB_sAg -)	↑	↓	N	N	N	↑	↑	N	N	N	N	N	N	0,90
13	M	37	PHC Liver cirrhosis (HB_sAg -)	↑	↑	↓	↓	↑	↑	↑↑	N	↑	↑↑	↑	-	-	-
14	M	56	Peritoneal metastasis of an adenocarcinoma	↑↑	N	N	N	N	↑	N	↓	N	N	N	N	N	0,66
15	F	40	CLL	N	↑↑	↓	↓	N	↑	↑	N	N	N	N	N	N	0,75
16	F	35	Liver cirrhosis by CAH (HB_sAg +)	↑	N	N	N	N	↑	N	↓	N	N	N	↓	↑	0,27
17	F	23	NRH (HB_sAg -)	↑	↑	N	N	↑	↑	↑	N	↑	↑	N	N	↓	1,07
18	F	35	Perihepatitis NRH	N	N	N	N	N	N	N	N	N	N	N	N	N	0,84
19	M	36	NRH	↑	N	↓	N	N	N	N	N	N	N	N	N	N	0,79
20	M	40	NRH Liver congestion	N	N	N	N	N	↑	N	↓	↑	↑↑	↑	N	N	1,08
21	F	28	Undifferentiated malignant liver tumour	↑	↑↑	N	N	N	↑	N	↓↓	↑↑	↑↑	N	N	N	0,61
22	M	17	Liver cirrhosis by CAH (HB_sAg +)	↑	↓	N	N	N	(↑)	N	↓↓	↑	↑	(↑)	N	↑↑	0,40
23	F	30	ALD Granulomatous hepatitis (tuberculosis?)	↑	↓	↓	N	↑	↑	↑	↓	N	N	N	↓	N	0,47
24	F	50	Alcoholic liver cirrhosis with liver siderosis	↑	N	N	N	N	↑	N	↓	N	N	N	↓↓	N	0,41
25	M	28	Malignant liver tumour	↑↑	N	↓	N	↑	↑	↑	N	↑↑	↑↑	↑	N	↑	0,52
26	M	62	NRH Alcoholic liver disease	N	↓	N	N	N	↑	N	N	N	N	N	N	N	0,90
27	F	25	Shock necrosis of the liver HB_sAg carrier	↑	↓	↓	↓	N	↑	N	N	↑	↑↑	↑	↓↓	N	0,52

Fig. 1 : Five stages according to spleen size (Hackett, 1944)

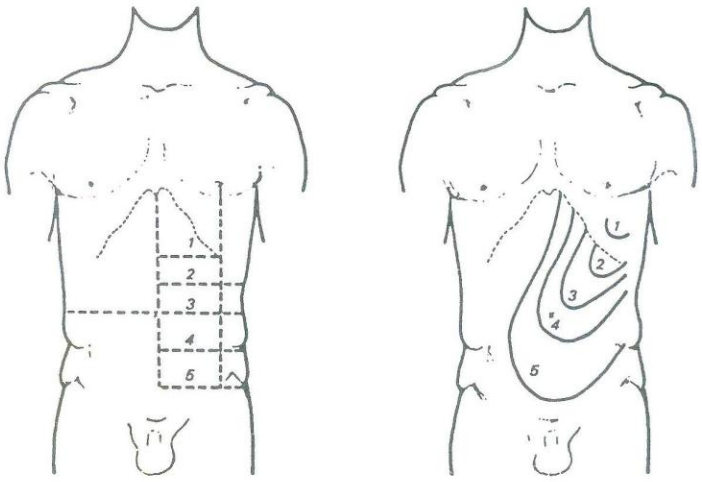


Fig. 2 : Presence of three criteria
(hepatomegaly, ascites, cachexia)

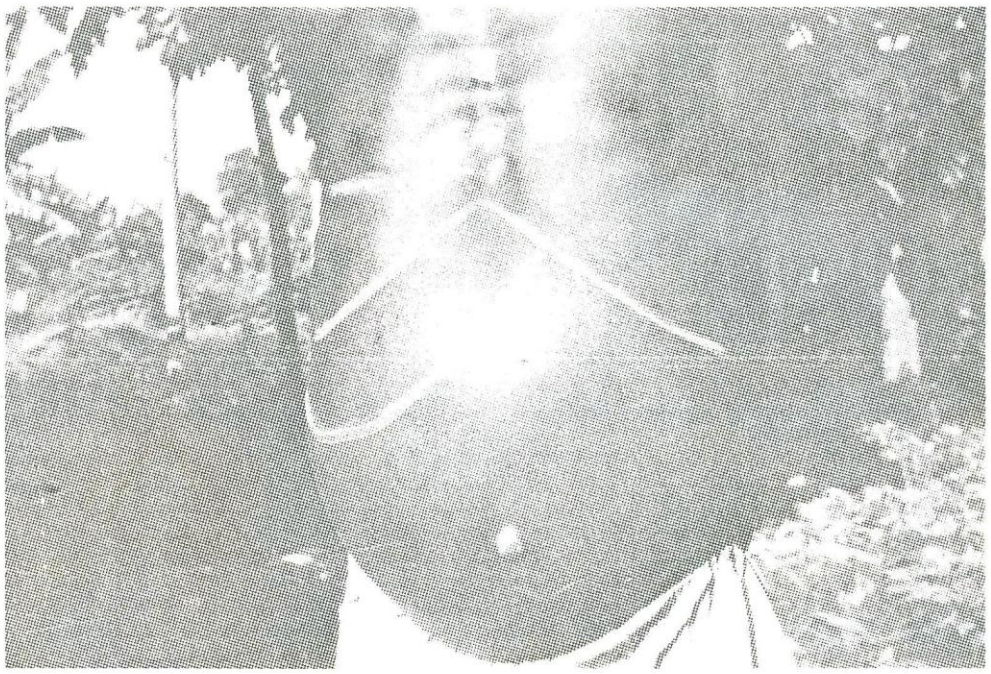


Fig. 3 : Jacobs-Palmer's Operating Laparoscope
(by kind permission of Mr. Treier, Beromünster)

Jacobs-Palmer

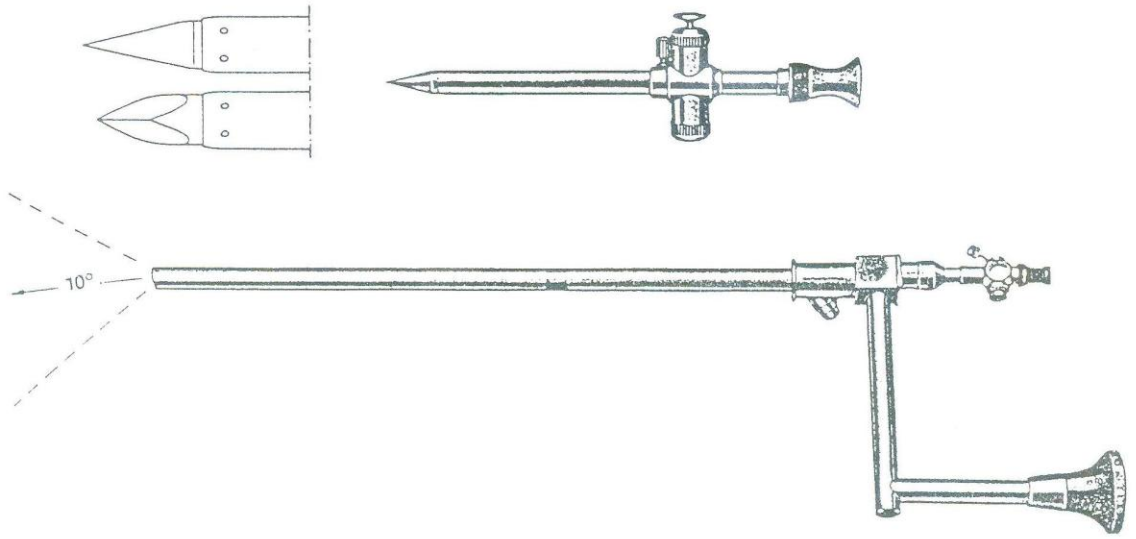


Fig. 4 : Instrumentation set for laparoscopy

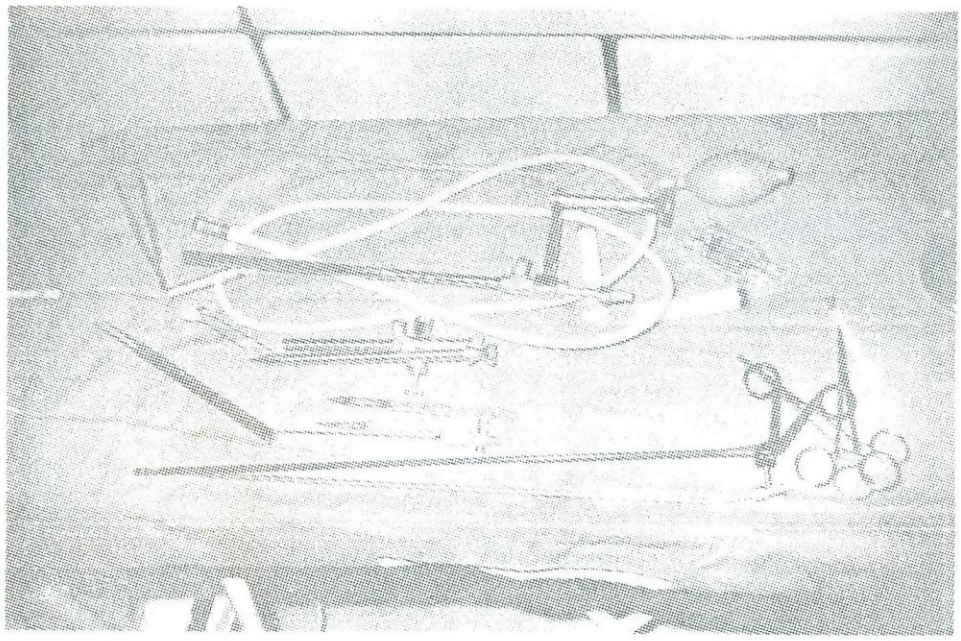


Fig. 5 : Insufflation through the Veres' cannula

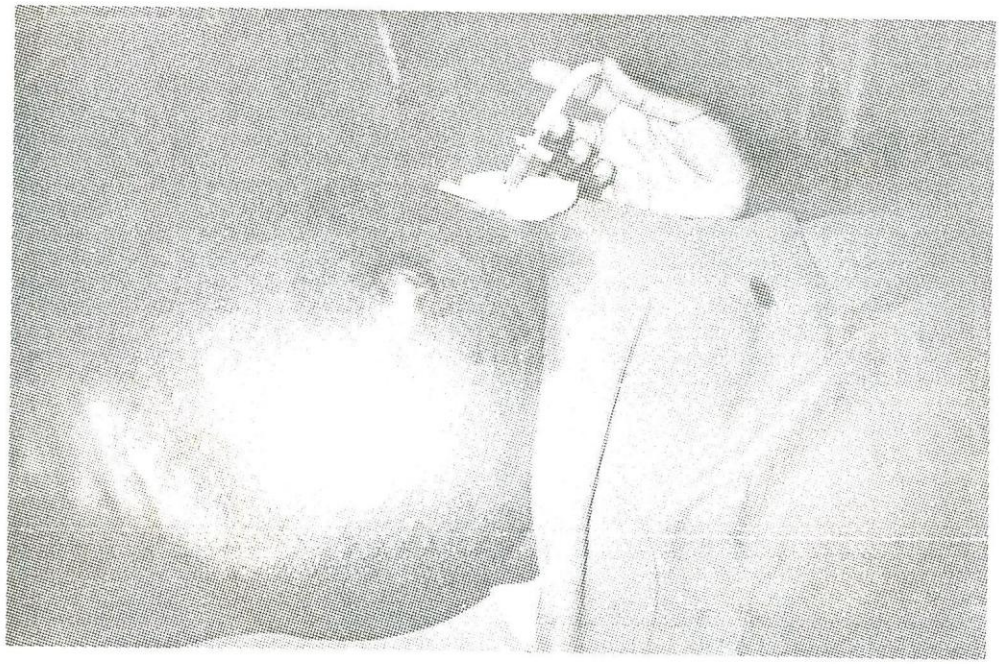


Fig. 6 : Sites for insufflation (A) and trocar introduction (B)

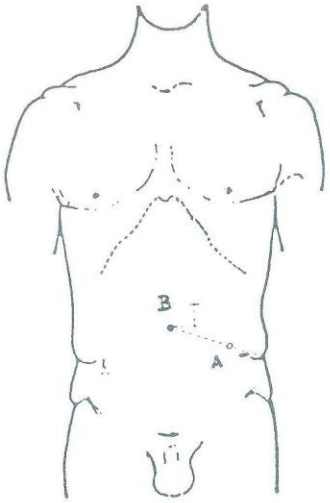


Fig. 7 : View of the operating instruments
(by kind permission of Mr. Treier, Beromünster)

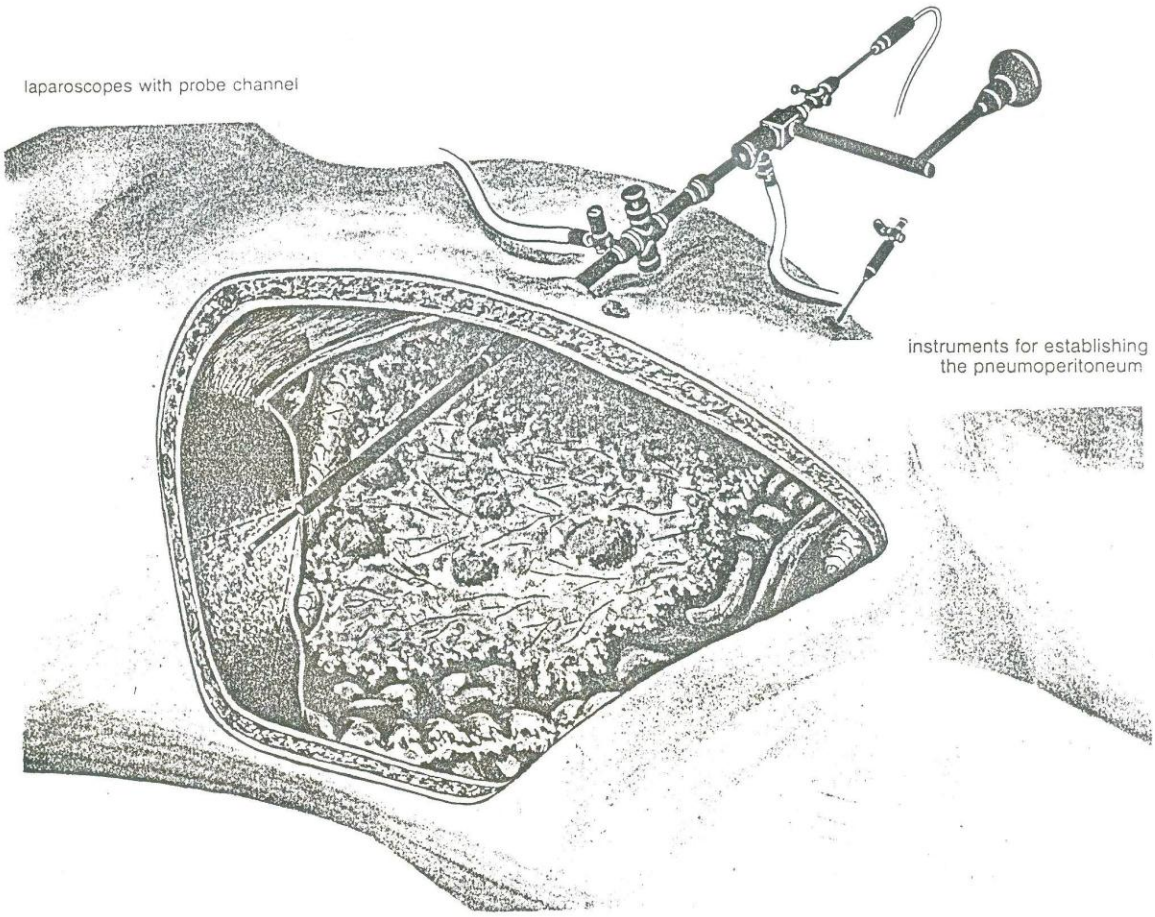


Fig. 8 : Guided needle biopsy

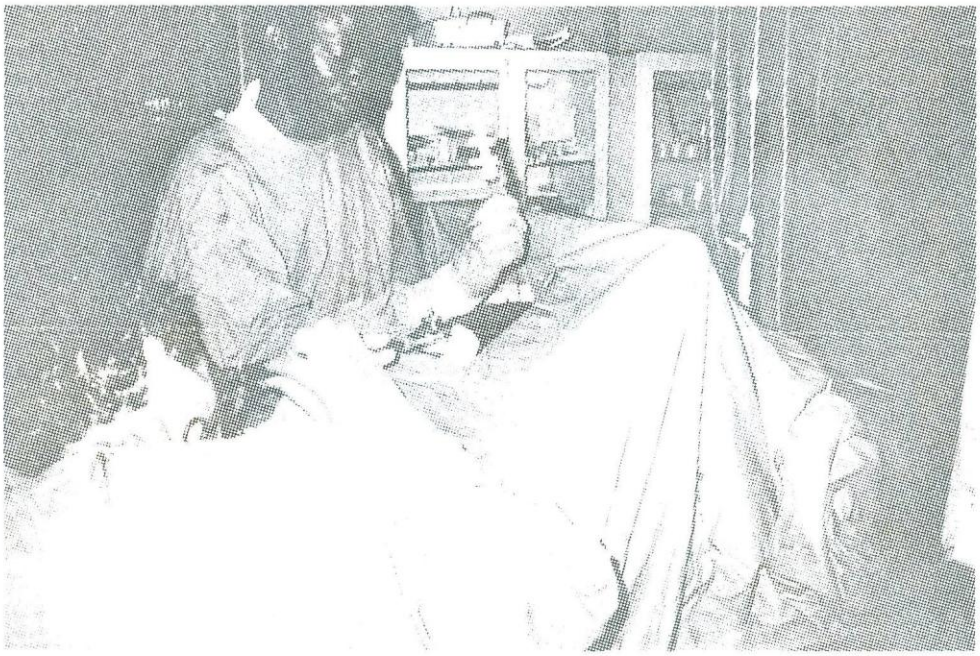


Fig. 9 : Age and sex distribution in the test group.

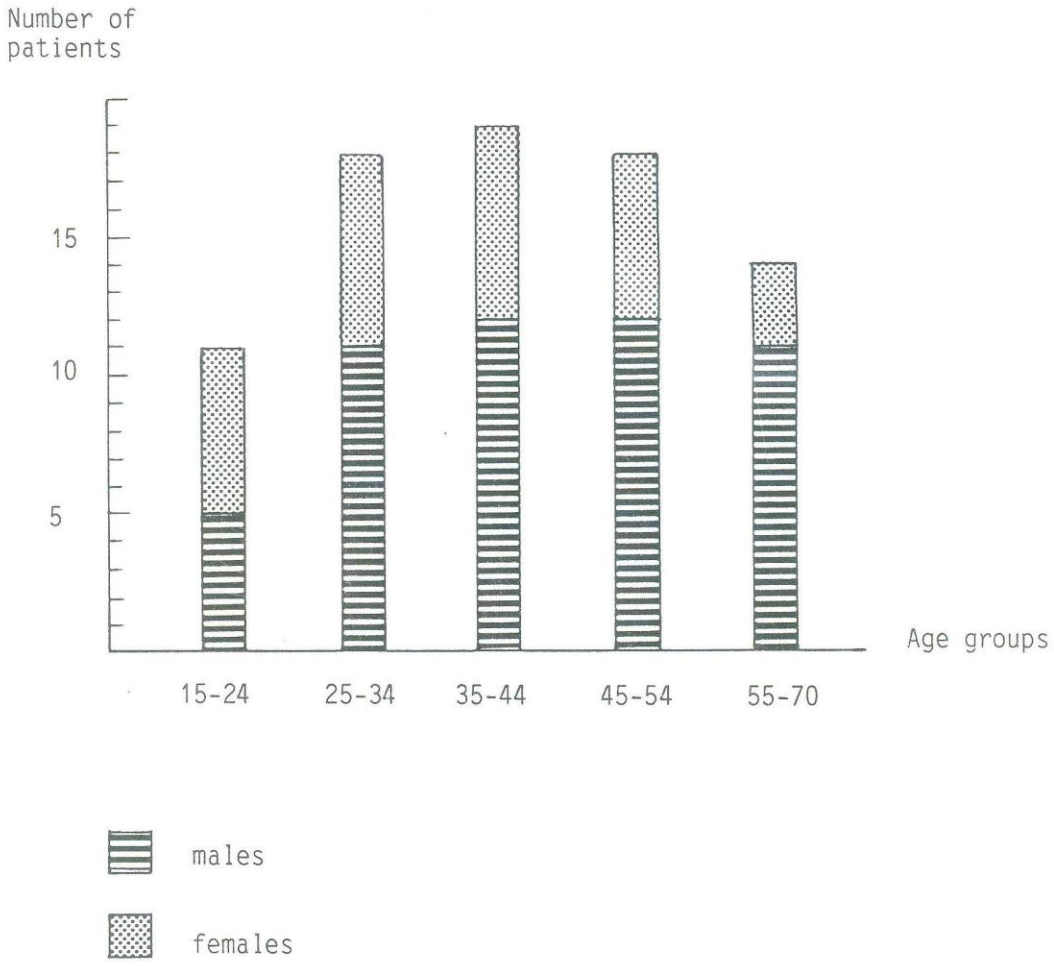


Fig. 10 : Age distribution among patients who underwent laparoscopy.

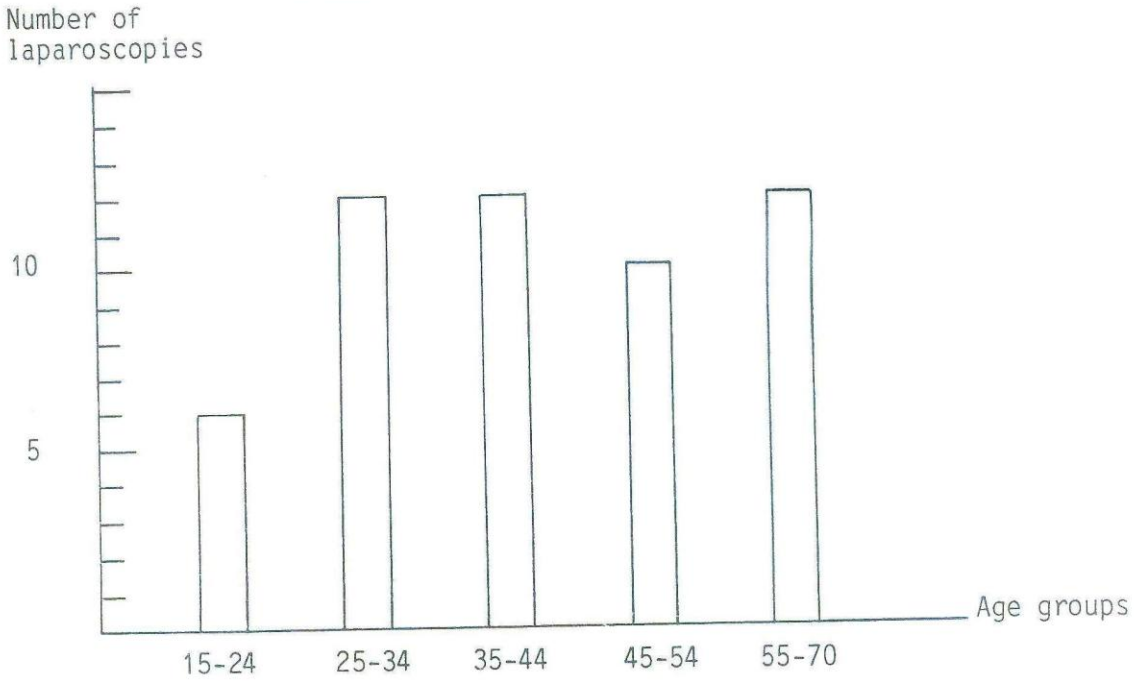


Fig.11: Biopsy distribution in the test group.

