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Vol. XXVII February 1961 No.

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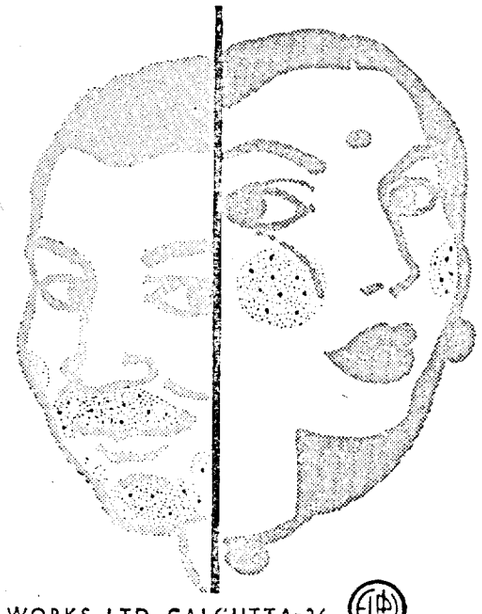

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Selections for the award of the prizes will be made by a Selection Board.

In the case of a joint authorship of a publication, the prize shall be divided between the authors in such proportion as the Selection Board may decide. The role of the person who applies for the prize should be clearly indicated so as to make it easy to determine whether the major part of the work has been done by that person.

THE AWARD of the prizes will be announced at the Meetings of the Council's Advisory Committees in November/December, 1961.

CANDIDATES, for award of prizes are required to submit, 10 REPRINTS of their papers published during 1960. These should be sent to the DIRECTOR, INDIAN COUNCIL OF MEDICAL RESEARCH, P. O. BOX 494, NEW DELHI, so as to reach him NOT LATER THAN 1st SEPTEMBER, 1961. The PAPERS should be accompanied by a short biographical sketch and two copies of passport size photographs of the candidate / candidates concerned.

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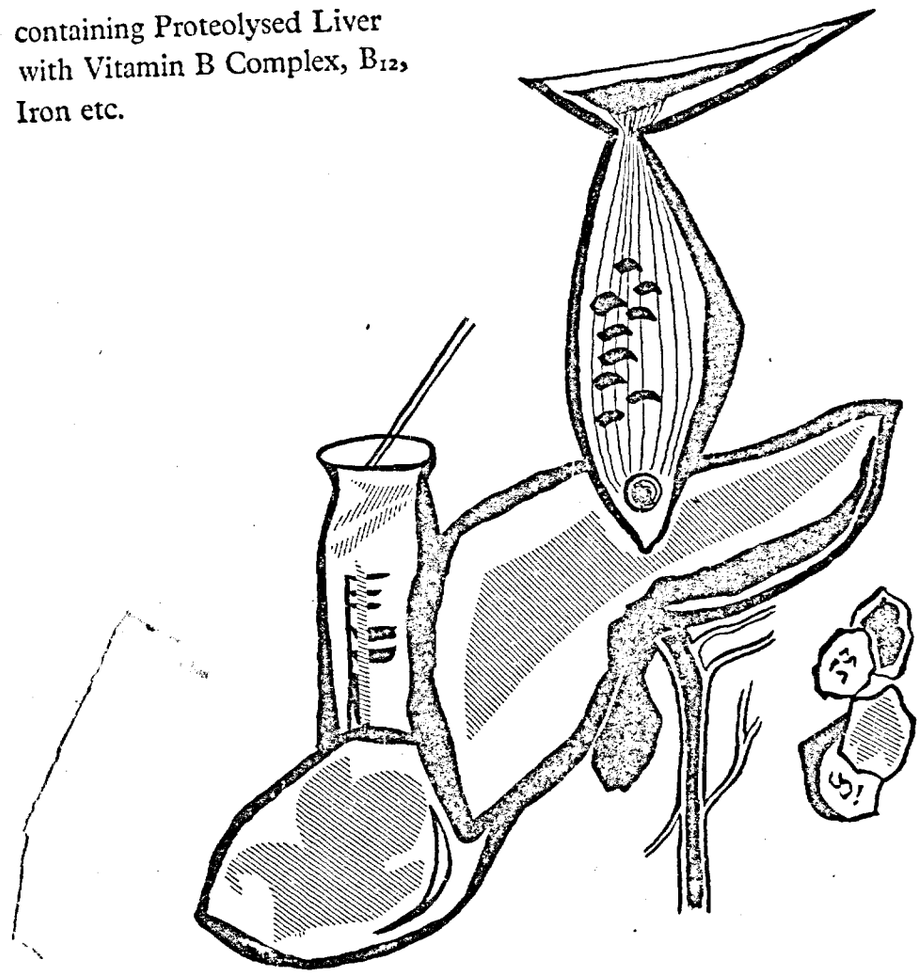

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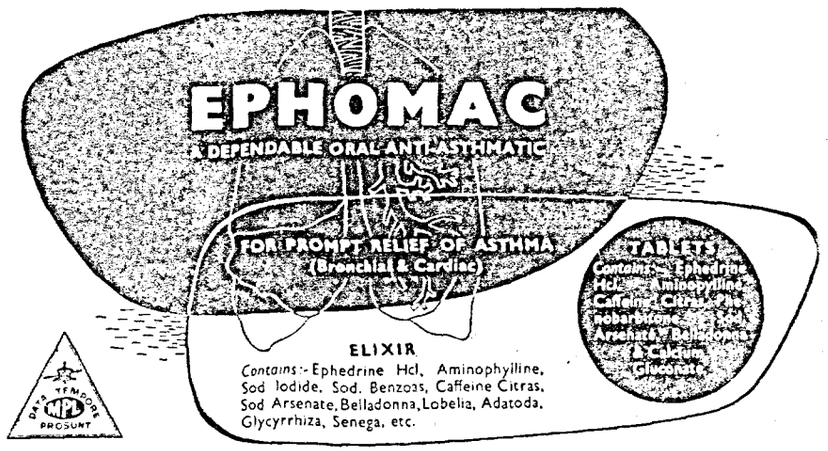

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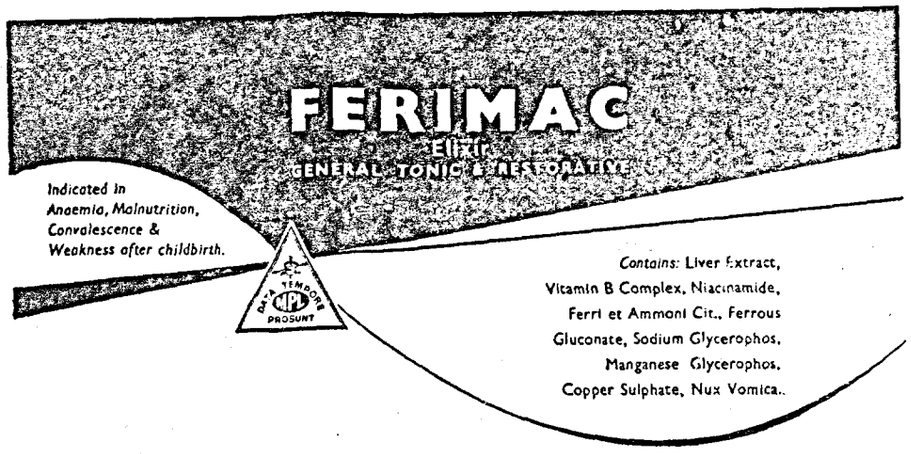

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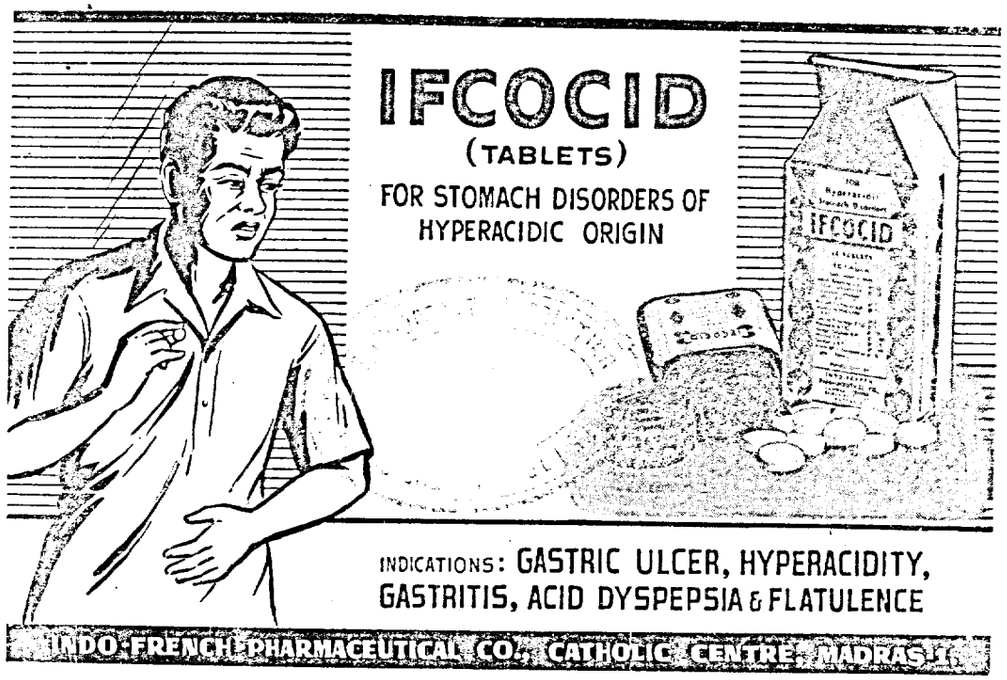

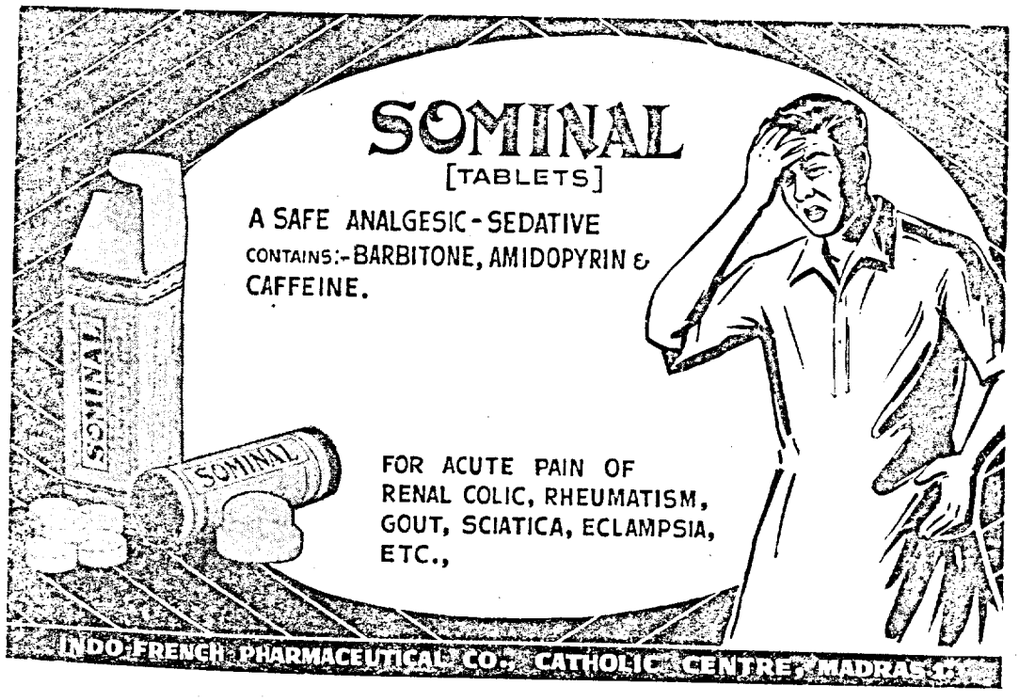

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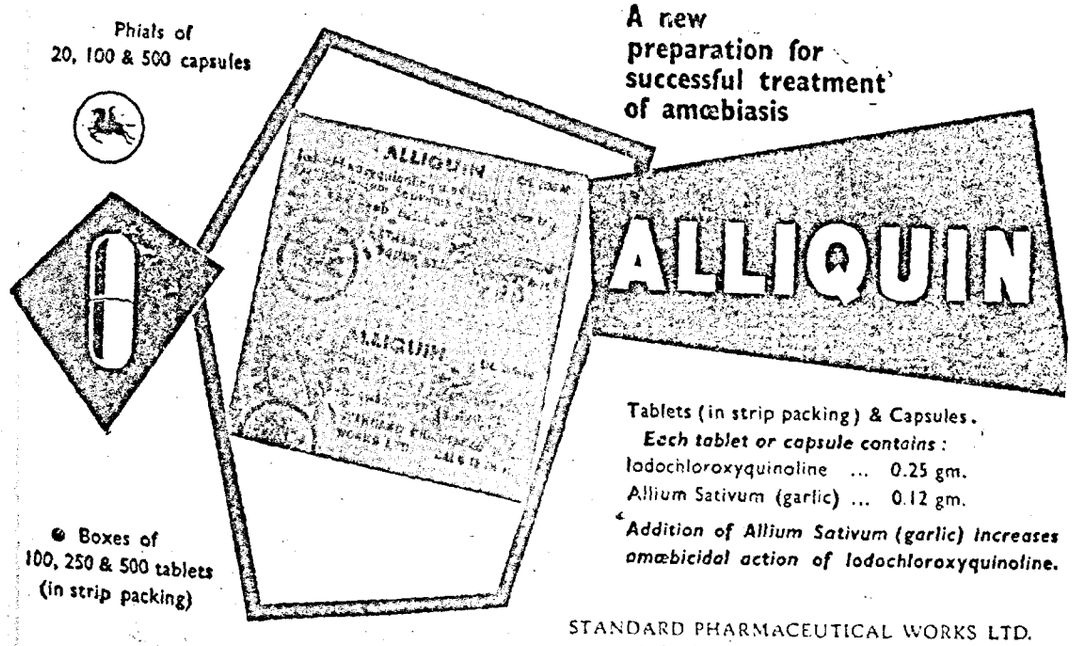

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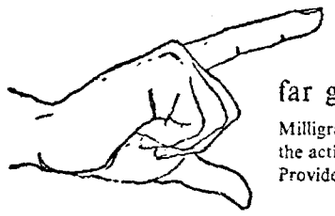

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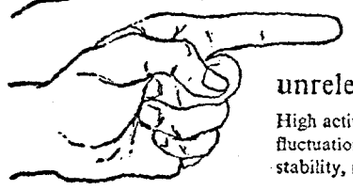

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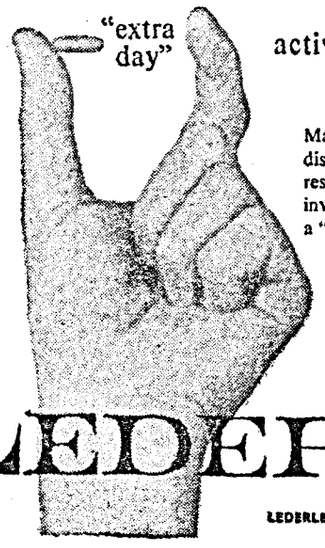

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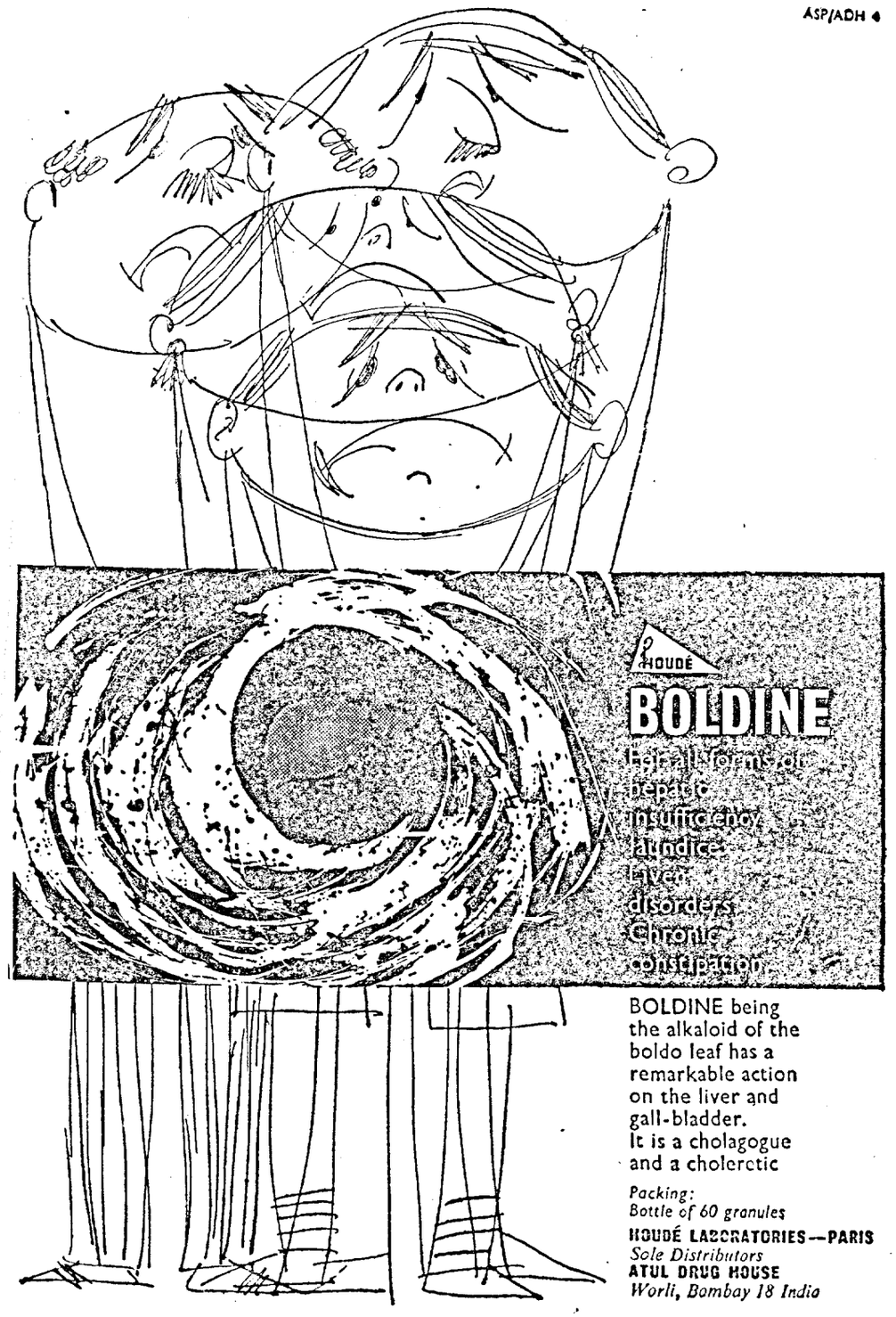

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JOURNAL OF THE MADRAS STATE BRANCH OF THE INDIAN MEDICAL ASSOCIATION
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Vol. XXVII February 1961 No. 8

CHYLOUS CYST OF THE MESENTERY

DR. V. SRIRAMULU, M. B., B. S.,
Honorary Asst. Surgeon, Govt. Head-Quarters Hospital, Coimbatore.

Among the various surgical conditions of the abdomen, tumours attract considerable interest in the undergraduate student and the senior most surgeon because of its elusiveness. The following is a report of a comparatively rare abdominal tumour — a large chylous cyst of the mesentery — diagnosed on the operation table.

Marudappa Gounder, aged 45 years, was admitted in the surgical wards on 24—10—1960 for a swelling and pain in the abdomen since three months.

Family history: Nil particular.

History of previous illness: The patient is a farm hand doing hard work and he had no complaint before the onset of the present illness. He used to have a vague discomfort in the abdomen after a meal occasionally.

History of present illness: The patient experienced dull pain in the back 3 months ago. The pain had no relation to food; he had no vomitting; he was feeling uncomfortable after a full meal; no history of disturbance in micturition; no history of diarrhoea or dysentery before; and no history of fever with rigor simulating malarial fever.

On Examination: The patient was anaemic and poorly nourished; tongue was clean and moist, but there was some amount of glossitis present but no jaundice. There were no enlarged lymph glands or oedema anywhere. Bowels regular and micturition normal.

The epigastrium was distended, but was moving freely with respiration. There were no visible peristaltic movements in the abdomen. The left loin appeared full. There were no superficial enlarged veins over the abdominal wall.

On palpation, a firm cystic smooth swelling lying obliquely in the left hypochondrium and extending beyond the midline upto the right subcostal margin, was made out. Fingers could be insinuated between the swelling and the sub-costal margin. The swelling was dull on percussion.

No "fluid thrill" could be made out. There was no band of resonance over the swelling. In the knee-elbow position, the tumour was felt prominently and movement in one oblique axis only. There was no evidence of free fluid in the peritoneal cavity. Regional lymph glands were not palpable.

Investigations :

Motion : Hook worm ova

Urine : Clear, no albumin, no sugar
Microscopic examination - Nil abnormal

B. P. : 120/80

Blood : Total leucocyte count: 10,300
Differential count: P 66, L 17 E 13%
R. B. C. count: 1·9 million
Haemoglobin %: 50% (Sahli)
E. S. R.: 6 mm. 1st hour.
Blood group: 'O'
Blood urea: 20 mgm%
Blood V. D. R. L.: Negative.

Ba meal : Not done.

Ba enema : Transverse colon and hepatic flexure displaced upwards - suggestive of extra-lumenal swelling.

I. V. Pyelography : Appearances suggestive of polycystic kidney. Both kidneys excreting the dye.

A provisional diagnosis of intra-abdominal cystic tumour was made and an exploratory laporatomy, with a view of removing the tumour, if possible, was decided upon. The patient agreed for the operation.

Pre-operative Preparation :

Hook worm treatment: Ol. chenopodium m. 10; Tetra chlorethylene m. 40 — Fiat Haustus. Liquid paraffin oz. 1. at bed time followed by a mild saline purge the next morning.

Diet rich in proteins and less in bulk.

Injection liver extract 2 cc. I. M. on alternate days.

Injection Vit. B. Complex 2 cc. on alternate days.

Oral iron tablets one t. d. s. after food.

300 cc. of 'O' group blood ten days after admission.

10—11—1960: R. B. C. count: 2·5 million. Hb. 60%

20—11—1960: 300 cc of 'O' group blood was given.

30—1—1960: R. B. C. count: 3 million. Hb. 65%

Operation notes :

2 — 12 — 1960 : Pre-medication : Carbitral capsule one at 9 a. m. Injection atropine 1/100 gr. with 1/6 gr. morphine at 10 a. m.

Anaesthesia : Under endotracheal anaesthesia of nitrous oxide, oxygen and ether, induction with 0.5 gm Pentothal I. V.

Operation : The abdomen was opened by a midline incision and the peritoneal cavity opened into. A large thin walled cyst arising from the root of the mesentery was found. Coils of intestines were twisted over the swelling but not adherent. The stomach was normal and free from the cyst wall. There was no evidence of any communication with a loop of the intestine or the transverse colon or the stomach. Liver, gall bladder and spleen were healthy.

In the manipulation of untwisting the coils of the small intestine, the cyst burst and a chylous milky liquid escaped out of the cyst. Nearly a pint of the fluid was suctioned out of the cyst and the cyst wall seperated upto the root of

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the mesentery. The cyst wall was removed and the rent in the mesentery was sutured. The kidneys appeared normal. The peritoneal cavity was drained by a rubber dam drain through a seperate stab incision in the left loin and the abdomen was closed in layers. The cyst wall was sent for histopathological report.

During the operation, 300 cc of dextraven and 300 cc of 'O' group blood were transfused to the patient.

Post-operative Management :

2—12—1960 : I. V. saline with 5% glucose 1 pint and 2 pints 5% glucose in distilled water. Injection 1/6 gr. morphine 6th hourly. Intermittent intra-gastric aspiration with an indwelling Ryle's tube.

3—12—1960 : I. V. fluids maintained. Patient had passed urine and flatus. Aspiration from the stomach had become less. Sips of glucose water allowed.

4—12—1960 : Drainage tube removed. I. V. fluids reduced to two pints a day. Half ounce oral feeds commenced. Injection Achromycin 100 mg. b. d. for the first four days. Later, injection procaine penicillin 4 lakhs with ½ gm. streptomycin I. M. daily upto the 10th day.

7—2—1960 : Biscuits, milk and soft bread allowed. Patient was allowed to walk around the bed with support. Temperature ranging from 99°F to 100·8°F for the first four days had touched normal. Dressings were changed.

11—12—1960 : Sutures removed. Wound had healed by primary intention. There was a small raw area at the site of the drainage tube.

At the time of writing this the patient is still in the hospital, walking and taking his normal food.

Histopathological Report : Report No. 2864 dated 4—1—1961 - Shows a fibro-connective tissue wall of a cyst infiltrated mostly with plasma cells and eosinophils and a lymph gland showing chronic inflammatory changes. The cyst does not show any definite lining epithelium.

Discussion : Mesenteric cyst is one of the rarest of abdominal tumours. It must be differentiated from oniental cysts and or enterogenous cysts. Mesenteric cysts are commonly found in between the two layers of of the mesentery of the small intestines (though rarely in the mesocolon). The size varies from about 1" in diameter to about 8" to 10". They are thin-walled and made up of connective tissue lined by endothelial cells. The contents may be clear, straw-coloured or milky in appearance (chylous). The origin of these cysts is not definite. Some believe they are a type of retention cysts due to obstruction to the lymphatics in the region that are present normally. Others contend that they develop in an aberrant lymphatic tissue and fluid accumulates as there are no out-flow channels.

Because of their anatomical relationship, the mesenteric cysts are easily movable, if they are nearer the gut and not so mobile, if they are nearer the root of the mesentery. In the case presented here, the swelling was not so freely movable and it was placed more laterally and posteriorly, and hence it was mistaken to be a kidney tumour; but on exploration only, it was found to be a mesenteric tumour, the tumour being incarcerated in the

right paracolic gutter. So, probably, it was not movable. The mesenteric cysts do not communicate with the intestines as an enterogenous cyst which contains usually succus entericus or faeces, if it was in communication with the colon. In the case presented here also the cyst was not communicating with the segment of the ileum to which it was densely adherent. Enterogenous and enteric cysts are reduplication of some portions of the gastro-intestinal tract and their walls contain serosal, mucosal and muscular layers. Omental cysts are similar in origin to mesenteric cysts.

Clinically, the case presents with a lump in the abdomen and vague abdominal pain and in most of the cases the lump was freely movable in the transverse axis. Sometimes the patients come with signs of chronic intestinal obstruction. In women, it has been mistaken for an ovarian cyst sometimes. These cysts occur more frequently in the mesentery of the ileum than elsewhere and the cysts contain clear, watery fluid more often than chylous fluid or solid material.

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2. Ian Aird - "A companion in surgical studies" (1957)
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SMOKING IN RELATION TO HEART DISEASE *

E. Cuyler Hammond, sc. d., f. a. p. h. a.
Director, Statistical Research Section, American Cancer Society, New York, N. Y.

The known acute effects of nicotine on the heart and circulatory system long ago led to suspicion that smoking might have serious consequences in relation to heart disease. Unfortunately, because of the risk involved, this cannot be tested by direct experimentation on human beings. However, during the last twenty years, a great deal of evidence on the subject has been obtained through epidemiological studies. I will first review the epidemiological findings and then give a brief summary of other types of evidence.

In 1938, Pearl[1] presented evidence that the death rate from all causes combined increases with amount of smoking and is very much higher among heavy smokers than among nonsmokers. Two years later, English, Willius, and Berkson[2] reported a positive association between cigarette smoking and coronary artery disease. Specifically, they found a higher percentage of cigarette smokers, particularly heavy smokers, among patients with coronary disease than persons free of this disease. In both studies, the relationship was largely confined to the middle-age groups and tended to disappear in old-age groups.

Within the last nine years, four other groups of investigators have carried out case-control studies of smoking in relation to coronary disease. There were Mills,[3] Gertler and White,[4] Hegglin and Keiser,[5] and Buechley, Drake, and Breslow[6,7]. In all these studies, a higher percentage of cigarette smokers, was found among persons with coronary disease than among persons free of this disease.

The early findings from the Framingham study[8] appeared to be contradictory. However, later findings from that study[9] are in essential agreement with the findings reported by all other investigators.

Data are now available from three large-scale prospective studies on smoking in relation to death rates from all causes. Doll and Hill,[10] by means of a mail questionnaire, obtained information on the smoking habits of a large number of British physicians and then ascertained their death rates in subsequent years. Dorn[11] is conducting a similar study of veterans holding United States government life insurance. Hammond and Horn[12] utilized the services of American Cancer Society volunteers to obtain information on the smoking habits of 187, 783 white men between the ages of 50 and 69 who were then traced for 44 months; 11,870 deaths occurring among them during that interval of time.

The results of these three prospective studies are in remarkably close agreement with each other. I will therefore not describe them all in detail. I will just give a brief summary of the findings in our study[12]. Figure 1 shows total death rates by type of smoking for each of four age groups. Note that cigarette smokers had the highest death rates and nonsmokers had the lowest death rates. The

* By Kind Courtesy of American Journal of Public Health - Volume 50 - November 3.

death rates for cigar and pipe smokers were only slightly higher than those

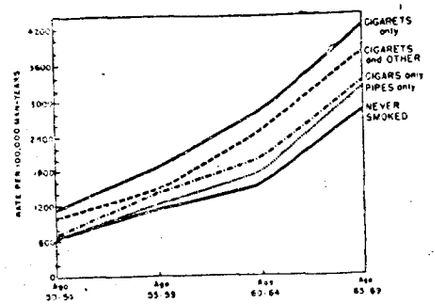


Figure 1—Total Death Rates by Type of Smoking (Life-time History) and by Age at Start of Study

for nonsmokers. Evidence from another study[13] indicates that most cigarette smokers inhale the smoke moderately or deeply, while cigar and pipe smokers generally inhale the smoke little if at all. This may perhaps account for the observed difference in death rates.

Figure 2 shows mortality ratios for total deaths in relation to amount of cigarette smoking. As a basis for comparison, the death rate of men who never smoked is taken as unity. Note that the mortality ratio increases with amount of cigarette smoking.

Altogether, 7,316 deaths occurred among men with a history of regular cigarette smoking (Figure 3). Only 4,651 deaths would have occurred in this group had the age-specific death rates of cigarette smokers been exactly the same as the age-specific death rates of men who never smoked. The difference of 2,665 deaths may be considered as the number of "excess deaths" associated with cigarette smoking. More than half of these "excess deaths" were attributed to coronary artery disease by the physicians who signed the death certificates.

Figure 4 shows mortality ratios for various circulatory diseases other than heart disease. Deaths attributed to hypertensive diseases showed no association with cigarette smoking

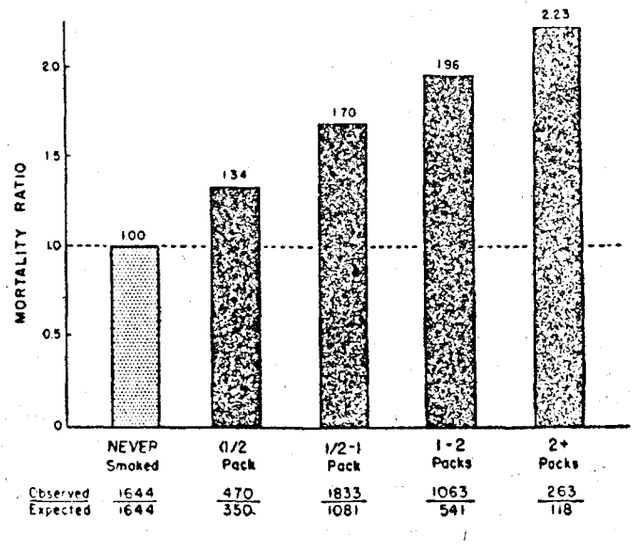


Figure 2—Mortality Ratios for Total Deaths by Current Amount of Cigarette Smoking

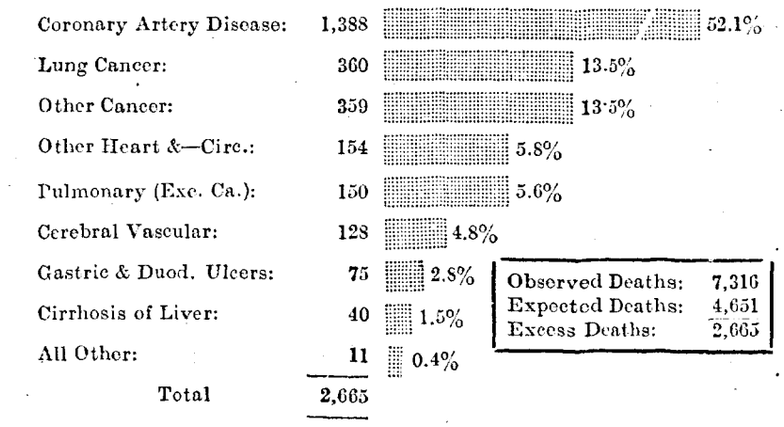


Figure 3—Excess Deaths Among Men with a History of Regular Cigarette Smoking

and deaths from cerebral vascular lesions showed only a slight relationship to cigarette smoking. Deaths from aortic aneurysm showed a pronounced relationship to smoking habits as did a small group of other circulatory diseases including Buerger's disease.

Mortality ratios for various heart diseases are shown in Figure 5. Coronary artery disease was the only type of heart disease showing a marked association with cigarette smoking.

The death rate from coronary artery disease increased rapidly with amount of cigarette smoking (Figure 6). The death rate from this disease was nearly two and a half times as high among men who smoked two packs or more of cigarettes a day as among men who never smoked.

Of particular interest is the coronary disease death rate of ex-cigarette smokers as compared with men who continued to smoke cigarettes (Figure 7). Men who stopped smoking less than a year before being enrolled in the study had higher death rates than men who were still smoking cigarettes at the time of enrollment. This is probably due to the fact that some people stop smoking following a heart attack; and the death rate is high among people who have suffered such an attack. The coronary disease death rate of men who had stopped cigarette smoking for a year or longer was lower than the rate for men who continued to smoke cigarettes.

Considering the evidence from such a large number of epidemiological studies of both the retrospective and prospective types, it can hardly be doubted that an association exists between cigarette smoking and coronary artery disease. The question is whether this association reflects a direct causal relationship; and, if so what machanisms are involved. For this reason, it is pertinent to give a brief review of other evidence concerning the apparent effects of cigarette smoking.

It has been demonstrated that smoking has the acute effects of producing constriction of the peripheral blood vessels, an increase in blood pressure and an increase in pulse rate[14]. However, it has been found that the

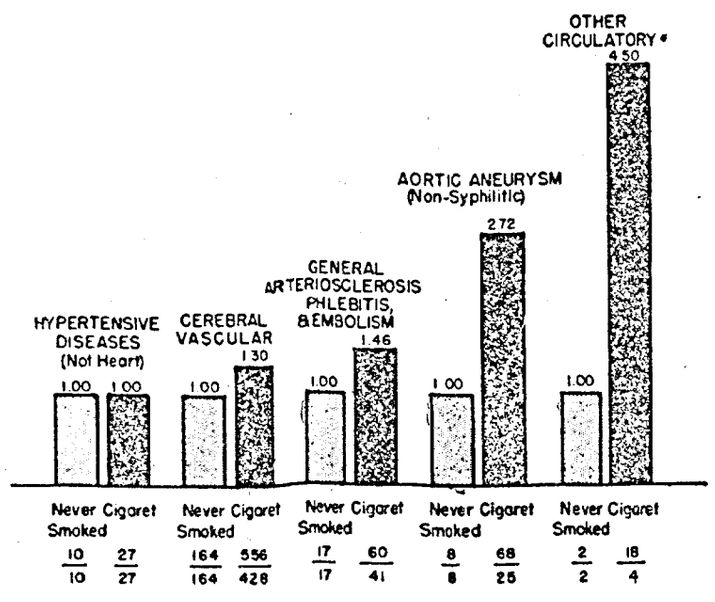
blood pressure of cigarette smokers tends to be slightly less than that of nonsmokers[15]. The chronic effects of cigarette smoking on blood pressure and pulse rate are unknown. Possibly the acute effect of an increased blood pressure produced by smoking results in a subsequent decrease in blood pressure. Alternatively, it may be that people with low blood pressure have a greater tendency to smoke than do people with normal or high blood pressure. This point needs clarification.

Carbon monoxide is present in tobacco smoke in sufficient quantities to form several per cent of carboxy-hemoglobin in the blood of the smoker[16]. Perhaps this accounts for the experimental finding that cigarette smoking produces both an immediate and a relatively long-term increase in red blood cell count and in packed cell volume[17]. Conceivably, this might have an influence on the formation of a thrombosis in a damaged blood vessel.

It has been reported that smoking produces marked changes in the electro-cardiograms[18] and ballisto-cardiograms[19] of some patients with coronary artery disease.

Smoking seems to diminish appetite and ex-smokers usually report a gain of weight.[20] This has been directly observed by Brozek and Keys.[21]

Gofman and his associates,[22] Thomas,[23] and Karvonen, Keys, and their associates[15] have reported finding higher average serum-cholesterol values among heavy smokers than among nonsmokers. The latter authors point out that this does not necessarily prove that cigarette smoking causes the serum-cholesterol to rise. It would seem most desirable to test this experimentally.



* Buerger's disease, aneurysm, varices, arteritis, etc.

Figure 4—Mortality Ratios for Various Circulatory Diseases other than Heart Disease

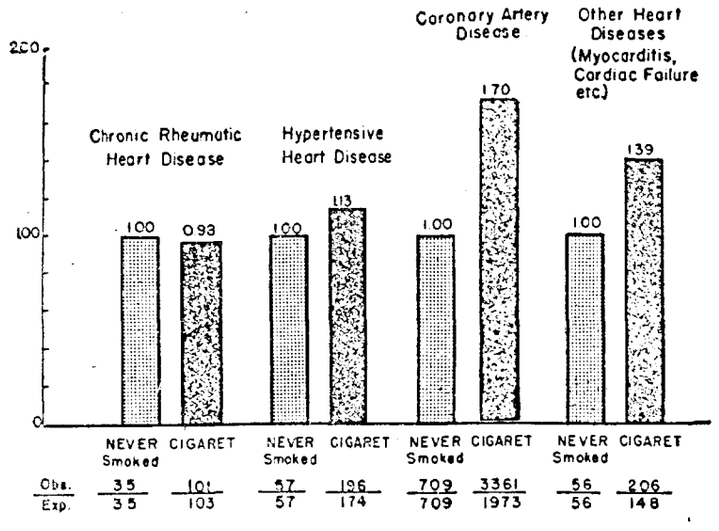


Figure 5—Mortality Ratios for Various Heart Diseases

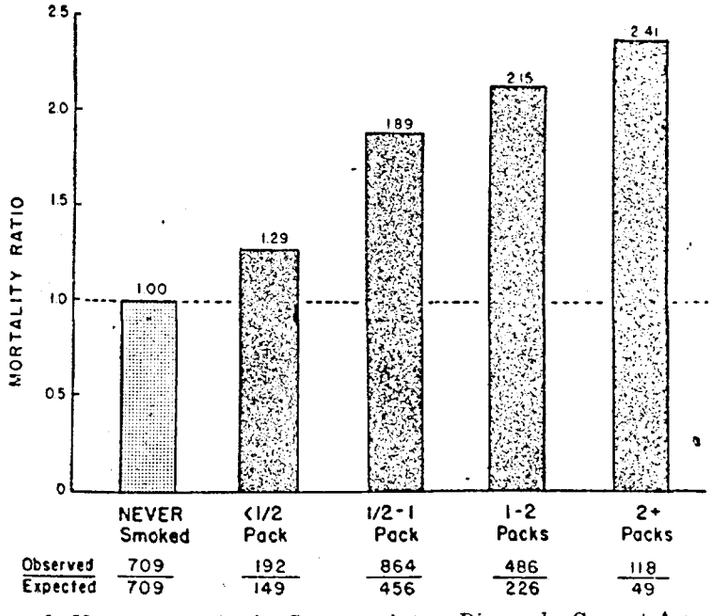


Figure 6—Mortality Ratios for Coronary Artery Disease by Current Amount of Cigarette Smoking

Preliminary findings from a study I am now conducting indicate that cigarette smokers complain of coughing and of shortness of breath far more frequently than do nonsmokers. There is a great deal of evidence that cigarette smoking has an effect on the lungs.[24] It has been suggested that lung damage from cigarette smoking puts a strain upon the heart and may perhaps increase the case fatality rate of persons suffering from coronary artery disease[25].

This in brief is the evidence. What does it mean? Certainly, the observed association between cigaratte smoking and coronary artery disease must have some rational explanation. The explanation must account for the findings that: (1) cigarette smokers as a group have higher death rates from coronary artery disease than do nonsmokers; (2) the coronary artery disease death rate increases with degree of exposure to cigarette smoke; and (3) among persons who have smoked cigarettes for many years, the death rate is lower for those who have stopped smoking for a year or longer than for those who continue to smoke cigarettes. Personally, I find it hard to escape the conclusion that cigarette smoking increases the death rate from coronary artery disease.

Two alternative hypotheses have been suggested. One is that people with a predisposition to coronary artery disease have a desire to smoke cigarettes; and that the degree of this desire is directly proportional to the degree of predisposition to the disease. Under this hypothesis it would further have to be assumed that some people with a predisposition to coronary disease lose this predisposition; and when they do so they also lose their desire to smoke cigarettes.

The other alternative hypothesis is that cigarette smokers tend to have

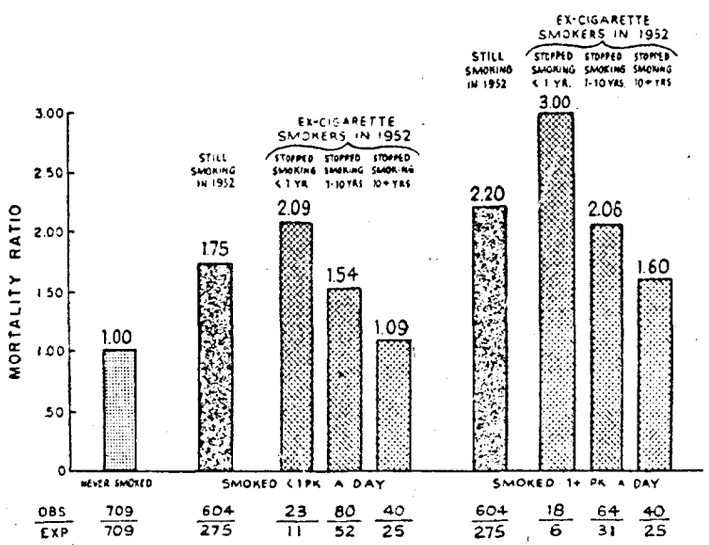


Figure 7—Mortality Ratios, Coronary Artery Disease for Ex-Cigarette Smokers Compared with Men Who Never Smoked and Men Still Smoking in 1952

certain other habits; and that it is these other habits which account for their high death rate from coronary disease. It would be rather difficult to disprove this possibility but as yet there is no evidence in support of it. Only an experiment carried out on human beings would provide absolutely conclusive proof as to which of these several hypotheses is correct. However, recent developments may provide us with a means of at least testing the hypothesis that the high nicotine content of cigarette smoke accounts for the high coronary artery disease death rate of cigarette smokers.

The epidemiological studies previously described were carried out at a time when the main stream smoke of almost all American cigarettes contained between two and three milligrams of nicotine, the most popular brands containing about 2·5 milligrams. About a year ago, some brands dropped the nicotine content to slightly over one milligram and these brands became popular. During the last few months several new brands came on the market with less than one milligram of nicotine in the main stream smoke and three of them are reported to have 0·6 milligrams or less.[26] If nicotine is the responsible agent, then heavy smokers who switch to these new brands should have a lower coronary artery disease death rate than those who continue to smoke brands with a high nicotine content. However, this might not be apparent for several years if it should happen that those who change to the new brands are mainly people who have already developed symptoms of heart disease.

I should point out that in addition to nicotine cigarette smoke contains a number of different poisons including carbon monoxide. It may be that carbon monoxide or one of the other components of cigarette smoke rather than nicotine is the responsible agent. If so, then lowering the nicotine content of the smoke would not have the desired effect (unless the responsible agent was also reduced).

Determining the coronary artery disease death rate in relation to nicotine content as well as the daily consumption of cigarettes, should provide excellent evidence on this point. Such evidence may be available in several years from now from a new prospective epidemiological study which we started in the fall of 1959.[27]

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STATE SECRETARY'S ANNOUNCEMENT:

THE 15th MADRAS STATE MEDICAL CONFERENCE

The members of the Madras State Branch of the I. M. A. are informed that for want of adequate response from the local branches in the State, the 15th Madras State Medical Conference due to be held in November 1960 could not be held in time. The local branches of I. M. A. in the State were circularised as long ago as on 1st April 1960 regarding the venue of this Conference. A circular letter has been sent round again to all the members of the Council of the Madras State Branch of I. M. A. on 22—12—1960 soliciting their kind co-operation in fixing up the venue. All efforts are being made to fix up the venue early so that the Conference and the Annual Meeting of the State Council could be held; and the venue and dates of the Conference will be announced in these columns shortly.

M. P. JESUDASEN,
Honorary State Secretary,
Madras State Branch, I. M. A.

A CASE OF MORQUIO-BRAILSFORD DISEASE

(Chondro-osteo Dystrophy)

DR. K. R. VENKATESALU, M. B., B. S.,
Honorary Assistant Surgeon, Orthopaedic Section,
Government Head-Quarters Hospital, Coimbatore.

A female child, aged four years, was admitted on 30—6—1958 in the Government Hospital, Coimbatore for kyphosis of the dorso-lumbar spine and inability to walk.

On going through the history, it was found that though the child used to stand up, it never walked like a normal child up to that time. There was nothing particular about the obstetrical history. The mother had two other children, quite normal according to her.

On examination, the child was having rather stunted growth. The skull, though looked bigger for the age, was normal in circumference. The joints looked bigger than usual, especially the wrists and knees. On standing, the kyphosis was marked at the dorso-lumbar spine and knock knees (coxa-valga) and pronation of both feet were present. No tenderness was elicited either over the spine or over the joints. The movements in the joints suggested some laxity of ligaments. The child never complained of any pain anywhere.

Routine examinations of the other systems including the central nervous system did not reveal any positive findings.

The following investigations were done:

Blood : E. S. R. 13 mm 1st hour
V. D. R, L. — Negative
Serum calcium, phosphorous and alkaline phosphatase within normal limits.

Urine : Nothing specific.

Montoux: Negative.

ROENTGENOGRAPHY:

Spine: The kyphosis of the spine was seen. The superior and inferior surfaces of the vertebral body were irregular. There was a tongue-like forward projection in the middle third of the anterior surface of the body of the vertebra. The anterior border was smaller than the posterior border resulting in a general wedge shape.

Extremites: The ossific nuclei of the upper and lower extremities were fragmented. In fact, the fragmentation of the upper femoral capital epiphyses, which was seen while looking at the spine film, first drew attention to this pathological condition. This fragmentation was well seen in the epiphyses of both humeri and femora. The capitellum also showed these changes. In the knees and ankles, the epiphyseal lines were irregular, widened and appeared eroded. In the skull and pelvis, no changes could be made out.

DIAGNOSIS:

With the above clinical, roentgen and negative laboratory findings, the condition fits in with the Chondro-osteo-dystrophy of Morquio-Brailsford. The latter demonstrated a series of similar radiographs at the Royal Society of Medicine in 1928 and the paper was published by him a year later. Morquio described

this entity in 1929 as a "form of familial osseous dystrophy" and reported four cases. Hirsch, in his classification of chondro-dystrophies, ranges this disease under the generalised form associated with irregular development of the epiphyses. He calls it the eccentro-chondroplasia which is identical with atypical achondroplasia of the peripheral type of Silverskiold.

DIFFERENTIAL DIAGNOSIS:

1. *Achondroplasia:* This type was known to the ancient. The Egyptian God, Ptah is portrayed as an achondroplastic. This well known figure among the court jesters and circus baffoons only clinically resembles the above disease in the childhood. Here the disorganisation in the cartilage plate with retardation of enchondral ossification is symmetrical and produce short extremities. The epiphyseal stippling is not present and the typical changes in the spine found in Morquio's disease are not present.

2. *Dyostos Multiplex Congenita or Gorgolysm:* Clinically and roentgenographically, this may resemble Morquio's; but the enlargement of the liver and spleen, and clouded corneas distinguish this condition. There is also mental deficiency in this condition. In the presence of generalised vascular, nervous and alimentary changes, the chandrodystrophic changes are minor and incidental.

3. *The stippled epiphyses or congenital chondrodystrophia calcificans:* This very unusual condition is characterised by discrete punctate areas of calcification in the cartilegenous epiphyses. These may coalesce. According to Steindler, it was first described by Tisdall and Erb. In the cases described, the patient had multiple flexion deformities of the joints and in some the extremities were strikingly shorter and thicker. No spinal changes are reported.

4. *Hypothyroidism:* Epiphyseal fragmentation may be present in this case. There will be also delay in the appearance of the ossific nuclei. The stature, coarse features of the skin, basal metabolic rate and response to the thyroid treatment will distinguish this entity.

5. *Rickets:* Florid rickets has to be thought of clinically. But the X-ray appearance which shows generalised osteoporosis with blurring of the ossific nuclei for the epiphyses, deformities and pseudo fractures easily distinguish that.

6. *Koch's infection of the spine:* The kyphosis suggests this condition, but once roentgen and laboratory findings are available, the diagnosis is obvious.

PROGNOSIS:

According to Fairbanks, these changes are progressive. Pathological deformities of the bones occur as the age advances.

PATHOLOGY:

D. H. Shelling disclosed that biopsy of the shaft of the long bones showed normal structure. Section of the epiphyses showed an irregular defective line of endochondral ossification with cartilagenous rests within the bony trabeculae. The metaphyseal trabeculae were porotic.

I am not aware of any autopsy specimen being reported.

TREATMENT:

As the etiology is unknown, no specific treatment is available. When the disease is progressive and deformities occur, they are corrected orthopaedically as and when necessary.

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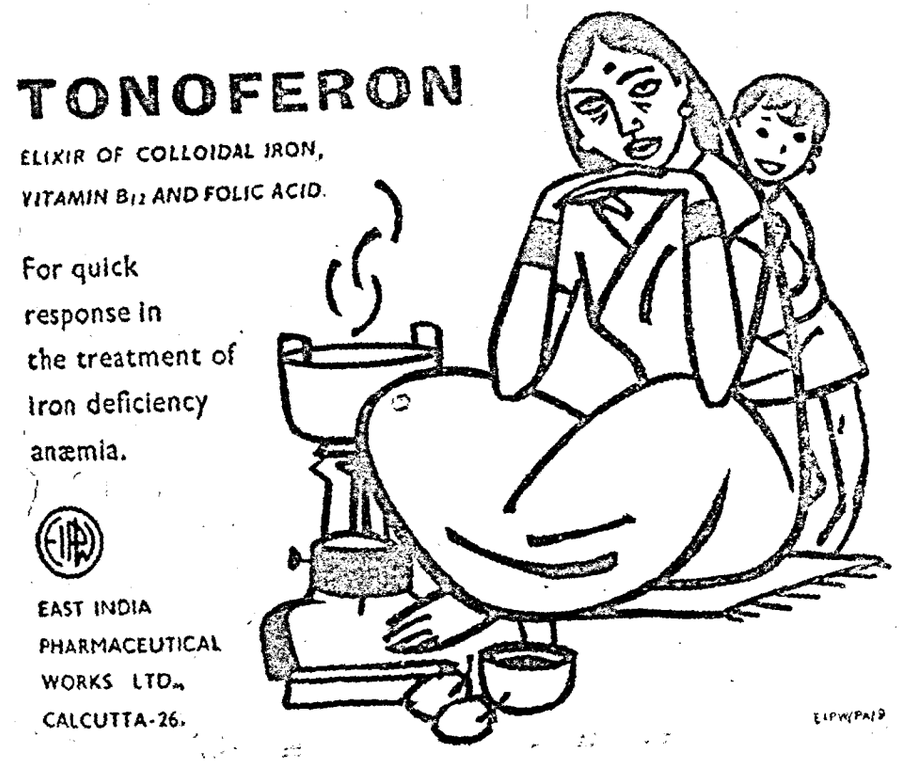
READERS! ATTENTION PLEASE:

Attention of our readers is invited to their respective addresses pasted on the wrapper. Any errors in their names or changes in their qualifications and addresses that may be noted therein may kindly be intimated to the MANAGING EDITOR for corrections in the mailing list proposed to be printed shortly.

Managing Editor.



THE MOST EXCLUSIVE HOSPITAL IN THE UNITED STATES *

VICTORIA STONE

The National Institutes of Health, near Washington, D. C., admits only those patients who will aid a particular research programme — the major fatal and crippling diseases, such as cancer, heart ills, and arthritis.

Just north of Washington, D. C., in the suburb of Bethesda is the most exclusive hospital in the United States.

There, in a series of hotel-like rooms, each patient receives about half the attention of a single physician, enjoys six hours of nursing care a day, and rests in knowing everything possible is being done for him. And there is no charge.

Every day the National Institutes of Health (U. S. Department of Health, Education, and Welfare), which runs the dream hospital, receives a dozen letters from afflicted and disabled persons in all corners of the country, begging admission to one of its 516 beds. They are all turned down.

The NIH's hardheartedness is strictly in the interest of all Americans. As the Public Health Service's centralised researchers, NIH runs a smoothly integrated war on disease at the 300-acre "campus" in Bethesda, Maryland. The Clinical Centre and the hospital at its heart are a focal point at that battle.

At the hospital, patients are helping medical research answer its most important question: What will our scientific approach mean to an afflicted man or woman? To do this, the Centre admits only those patients who will aid a particular research programme at NIH. They need not have a rare disease, for NIH is after the Nation's major killing and crippling diseases — cancer, heart ills, and arthritis among them — not the one-in-a-million case.

To become the study target of NIH scientists, a prospective patient must have his doctor submit a full medical report, which must then be weighed by researchers there against their own programme. Once admitted, the patient becomes the centre of attention. The Clinical Centre's peculiar floor plan, for one thing, guarantees it.

Along 11 of the 14-storeyed Centre's central corridors, patient and nursing rooms neatly parallel laboratory space. A patient may find himself only ten steps away from his doctor's office and laboratory.

Making an astounding ratio of one doctor for two patients, 215 physicians and 17 dentists work directly with the ward. More than 300 Public Health Service nurses function in spotless work and treatment rooms adjoining the hospital areas.

Rooms provided for walking patients are nearly indistinguishable from the best hotel rooms. Each room has an attractive southern exposure, its own bath, and even a pillow radio for bed listening.

In each storey, a solarium affords patients a place to relax and a view of the city horizon. Also for up-and-about patients there is a chapel, gymnasium, and sun deck in the fourteenth storey.

According to Clifton K. Himmelsbach, associate director of the Centre, the patient is more than a well-cared-for project.

"The tremendous kindness, the tender loving care, on the part of everyone in the operation is the most striking thing here," he said. "The spirit we started with in 1953 has lasted. There's nothing cold-blooded about the Centre."

The Centre hospital is really seven small hospitals. Each patient is under the observation of one of the seven institutes located around the base of the giant building. Men from the Institute of Allergy and Infectious Diseases may be examining a respiratory ailment in a child in one storey while Heart Institute researchers are weighing effects of a new heart depressant drug on a man in the storey below.

Other wards house patients for the Institutes of Cancer, Mental Health, Arthritis, Metabolic Diseases, Neurological Diseases, Blindness, and Dental Research.

The patient's payment for this best of hospitals takes the form of a vigorous research programme in which he undergoes a series of often discomforting tests. He knows that when the study is complete that he will be discharged, usually within four to eight months.

Nearly 1,100 "investigators" meet there from scattered campus institutes. This allows, in the associate director's words, an exchange of ideas between the patients' physicians and the basic-research men who operate "just down the hall" at the wing ends of each storey.

The "forced association" is not a new idea, said Himmelsbach, but it has certainly found an effective expression in the Clinical Centre plan.

Laboratory corridors dotted with doors marked Conference Room give evidence to the collaboration. Within, research teams map their strategy over test tubes and slides, or clinical physicians keep basic researchers posted on their human slide-rules. The NIH estimates that every seventh worker one meets there from the yard man to the senior medical officer will be a "doctor" of one kind or another.

For every independent investigator, whether physician, dentist, biochemist, or psychiatrist, there are 2½ research backstoppers. Many of these technicians and laboratory aides are graduate-degree holders themselves.

For some 60 temporary research posts, many of them at the Clinical Centre, nearly 1,500 applications are received from all over the United States.

One out of five each year's U. S. medical graduate roster, NIH estimates, wants to become a clinical resident, clinical associate, or laboratory research associate.

The Centre also draws 32 privately hired "guest workers" to aid individual scientists and several "visiting scientists" from the United States and abroad, whose special skills make them must "imports."

(From the Washington Post reprinted by permission).

* Courtesy — United States Information Service.

ARTIFICIAL KIDNEY*

A. GRIGORYEVA

"Meet our patient V."

V. was brought to the kidney centre in a dangerous state, with acute post-operational insufficiency of the kidneys. The patient's kidneys were unable to cope with their blood clearing functions and intoxication of the organism had set in. V. lay unconscious. All possible means were tried, but they could not help the kidneys to clear the patient's blood of noxious substances.

Hemodialysis was then performed with the aid of the artificial kidney.

V. lies on the operating table. The surgeon responsible for the operation opens access to the lower vena cava and a narrow polyethylene tube is introduced into it. The surgeon watches its progress through the large vessel as far as the level of the renal veins. Another narrow tube is joined to the vein in the bend of the elbow and both tubes are connected with an apparatus performing the functions of the kidneys.

DEVICE FOR FILTERING BLOOD:

It is switched in. The blood, saturated with noxious elements, leaves the body through the lower vena cava and flows a long way through the apparatus between layers of cellophane film (they are calculated to measure 18,000 square c.). The products of disintegration pass through the tiny pores of the film into a special dialysing solution. From the apparatus the clean blood returns into the organism through the vein of the arm and is then delivered back to the apparatus.

Engines whirr. The blood is filtered several times between the dialysing films. The artificial kidney works more intensely than the natural one. In 4 or 5 hours the operation is over. The subsequent analysis shows the proportion of urea in the blood to be considerably reduced and intoxication of the organism has ceased. In these favourable conditions the kidneys begin operating again and resume their normal functions.

SUCCESS OF OPERATION:

This operation, like all others in the Soviet Union, has not cost the patient anything. The staff of the "kidney centre" are very pleased with this "assistant" made of metal and plastics. "New Soviet medical technique," says doctor V. Agranenko opens ever greater prospects before physicians and scientists. Saving patients from the consequences of kidney insufficiency was until quite recently a rather difficult matter. In many cases physicians failed to achieve success. But with the "artificial kidney" we achieve extraordinary medical results. In cases not too far advanced, when hemodialysis is performed in time, it saves the patients' lives.

With the acceptance of "artificial kidney" apparatus, designed in the Research Institute of Surgical Equipment and Instruments, operations can be performed without complications.

In the surgeon's opinion, the Soviet device has several advantages in comparison with foreign models. It is much simpler to operate and requires a small amount of donor's blood in starting.

Physicians and engineers continue to improve the "artificial kidney", striving to make it more portable and simpler to operate. The use of perfect devices in medical practice provides facilities for a successful campaign for the people's health.

* Courtesy — U. S. S. R. Embassy in India.

LETTERS TO THE EDITOR:

THE MEDICAL COUNCIL OF INDIA:

The Indian Medical Council Act of 1933 ushered in by the then British Government, much against the strong protests and opposition of the entire medical profession and the two All India Medical Associations in our country, has resulted, after a lapse of 23 years in the establishment of the Indian Medical Council Act of 1956 which is neither welcome nor desirable in independent India as it does not conform to the conditions of a Medical Act and still clings to the policy of the alien administration in dividing the profession into casteism and compartments showing thereby the attitude of some of its members who are obsessed with notions not congenial to the healthy growth and progress of the profession and adopt a bureaucratic attitude of the erstwhile reactionary Government. Unless and until this undesirable complex is shed once for all, the Council wont be doing its duty to the profession and the Medical Act which does not find any parallel any where in the civilised globe. The functions of a Medical Council are threefold as stated below :—

1. It must maintain a medical register of all qualified medical practitioners.
2. It should exercise a disciplinary control over them and preserve the dignity and the ethics of the profession.
3. It should standardise the minimum qualification required for registration so that medical education in various schools and colleges in India may be maintained at a uniform level.

The Medical Council of India has so far subserved none of the above conditions beyond the fact that the Act has included in its purview a large section of statutorily registered medical practitioners in the State Medical Councils who have been left hitherto unrecognised. The anomaly of this Act is the existence of schedules of qualifications under four categories further differentiating the profession which the Act does not envisage. The only function it seems to be exercising is to bring about a rapproachment between the degrees awarded by the Indian Universities and the General Medical Council of Great Britain and get its recognition as well as inspection of Medical Colleges, their examinations and the curriculum of studies for recognition. This Council as it exists does not fulfil all the attributes of a Medical Council, because neither the Government nor the Council has any clear idea of its functions. Unless the Council whole heartedly passes what the Act stands for, the Medical Council of India may as well cease to function as an all India body and be termed as a Council for Medical Education in which case there need not be any agitation against its existence by a large body of medical men who are quite satisfied with the functions of the State Medical Councils.

Madras,
27—1—1961.

DR. D. V. VENKAPPA.

ABSTRACTS AND EXCERPTS

LIVE POLIO VACCINE:

In June, 100 scientists from 20 countries met in Washington, D.C., to report their experiences with live polio-virus vaccine in laboratory experiments, field trials, and mass campaigns. The international conference was sponsored by the WHO and the PASB.

The conference was informed of these developments, for example:

In the USSR, Sabin vaccine, Soviet-tested and mass-produced, has been administered to more than 66 million persons. (See also June, 1960, Supplement, A.J.P.H. Dr. A. A. Smorodintsev: "New Live Vaccines Against Virus Diseases.") Supplies have also gone to Czechoslovakia, Hungary, Bulgaria, mainland China, and North Vietnam.

Poland, using another vaccine of United States origin (Koprowski), has embarked on compulsory vaccination of all susceptible age groups.

In Latin America, a third United States-bred vaccine (Cox) has been used for vaccination of millions of persons under tropical and epidemic-menaced conditions.

In the United States, which for several years has been using needle-injected dead-virus vaccine (Salk), various communities (Dade County, Fla., and others) have more recently been giving live-virus vaccine by mouth.

The conference agreed that "in almost all those trials, including the massive experience in the USSR, untoward reactions were either absent or insignificant."

Among the speakers who cited advantages in the use of live virus vaccine that can be administered orally, was Dr. Oscar Vargas Mendez of Costa Rica who stressed ease of giving in countries lacking the doctors and clinical staff required for needle vaccination.

In summing up, Fred L. Soper, M.D., director-emeritus, PASB, and vice-president, American Public Health Association, stated that the challenge of the conference was no less than an attempt to rid the world of polio, with the greater part of the world unable to afford the expense or professional man-power needed for needle vaccination. Safe, live vaccine easily dispensed to hundreds of millions could be the answer, he said.

Following the conference, WHO's Expert Committee on Poliomyelitis met in Washington, June 13-16, and expressed belief that both types of vaccine had a major role to play in world control of polio. The committee observed that the information on the effectiveness of the live vaccine,

although hopeful, is not conclusive as to its degree under all circumstances. Also that the new vaccine could not be relied on to give full protection in one dose and that repeated administrations are needed.

SYPHILIS AND THE TRIPLE-TEST PLAN:

With monotonous regularity syphilis in the United States continues its upward path, a swing that began in 1956. During the fiscal year 1959-1960, there was a 52 percent increase in reported cases of primary and secondary syphilis over the previous year, and the rates for fiscal 1959 were 22 percent greater than those for 1958. Thus, in the past two years infectious syphilis has risen by 87 percent.

Approximately 60,000 new cases of syphilis occurred last year in the United states-not all of which were found or treated, let alone reported to health departments. The unfound, undiagnosed and untreated cases add each year to the infectious reservior in the community. People are infected faster than they are found and treated. As a consequence, there are now about 1,200,000 persons in the United States who have syphilis and need treatment.

The patient who has symptomatic syphilis, in either its early or its late manifestations, presents a minor problem in diagnosis and treatment. But what taxes the diagnostic acumen of the physician is the patient with a persistently positive blood test for syphilis who has neither historical, physical nor epidemiologic evidence of the disease.

Before the availability of the treponemal tests, considerable time and judgement were expended to arrive at a decision of infection with syphilis or a biologic false-positive reaction. The arsenicals, in contrast to penicillin, were not without measurable risk. Although most patients with repeatedly positive blood tests for syphilis later proved to be syphilitic-an observation that holds true at the present time-there was a reluctance, even, as today, to label a patient as syphilitic without adequate evidence.

Fortunately, today, the diagnostic problem of syphilis versus biologic false-positive reaction can be resolved with very little difficulty, thanks to the introduction of the treponemal tests in 1949.

The triple-test plan should be reserved for the few cases that cannot be diagnosed by conventional method. In either event, the diagnosis of syphilis or of a biologic false-positive reaction can usually be established within four office or clinic visits.

Although technology has accelerated the speed of diagnosis, epidemiology continues to reveal that there are at least 3 infected persons involved in every case of syphilis. Two patients naming each other as sole contacts did not contract their syphilis from each other; there must be at least 1 other infected person. Syphilis can never be controlled, let alone eradicated, until every case is found and reported, and its source ascertained, and all contacts followed up to find possible infection.

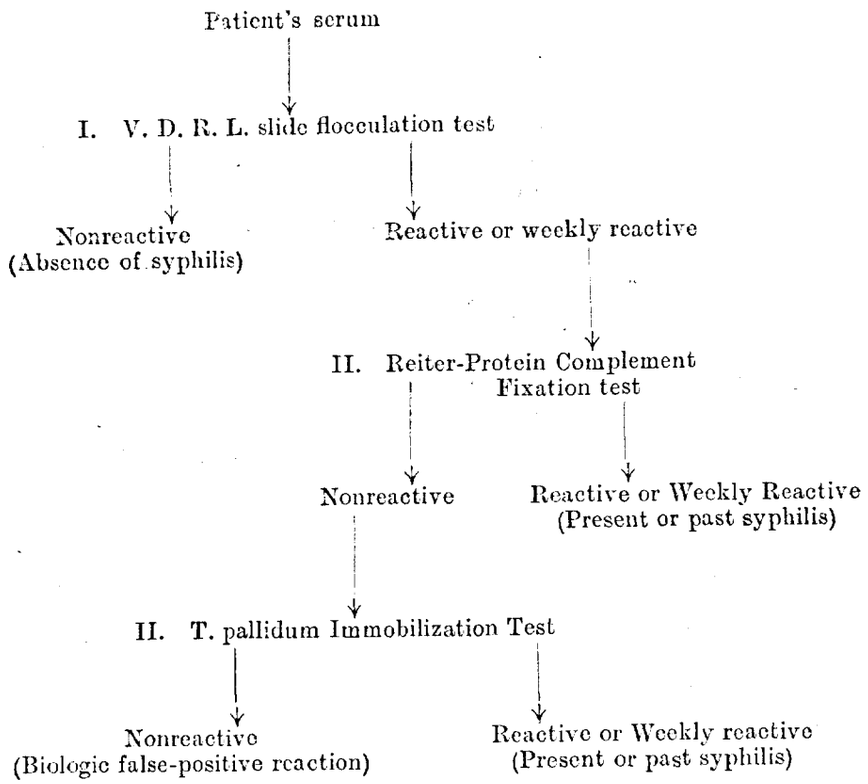


Diagram Showing the Triple-Test plan.

— New England Journal of Medicine (263, 1031—Nov. 17, 1960)—Editorial.

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Anti-tetanus serum (ATS) is an unsatisfactory prophylactic for several reasons. Tetanus may follow trivial injury for which anti-tetanus serum is not given. ATS may produce serum reactions, many mild, many severe, the rare one fatal. A patient who has had ATS may develop the ability to excrete the antibody very rapidly so that a dose given for a second injury may be ineffective. The only satisfactory prophylactic is active immunization. If a patient is given ATS after an injury, he should also be offered a course of 3 injections of tetanus toxoid. If a patient has previously had ATS, he should be given toxoid rather than ATS. All patients with a breach of the epithelium should be given a course of toxoid. All infants should have tetanus toxoid included in their immunization programme. In this way a high level of active immunization could be built up in the community and the prophylactic use of ATS would cease.

(Fulford, G. E. Lancet - 1960, May 21, 1121-3 (15 refs.)

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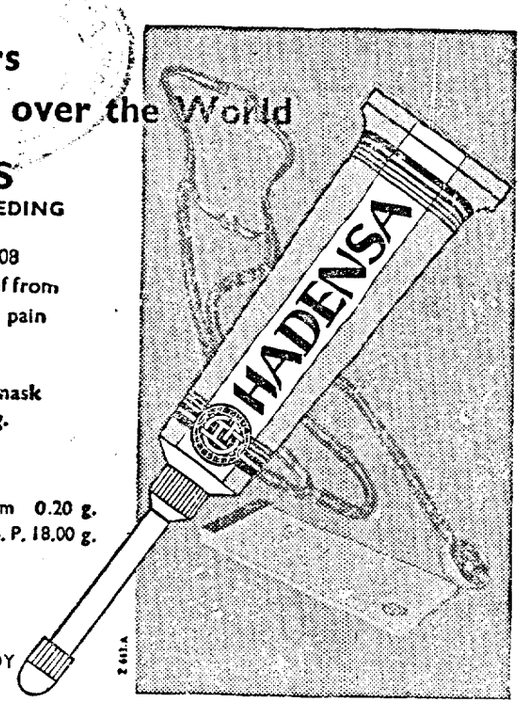

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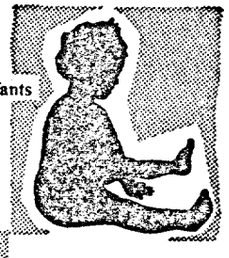

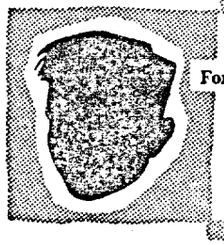

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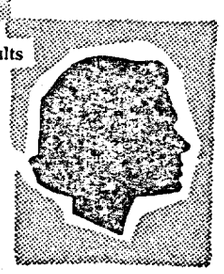

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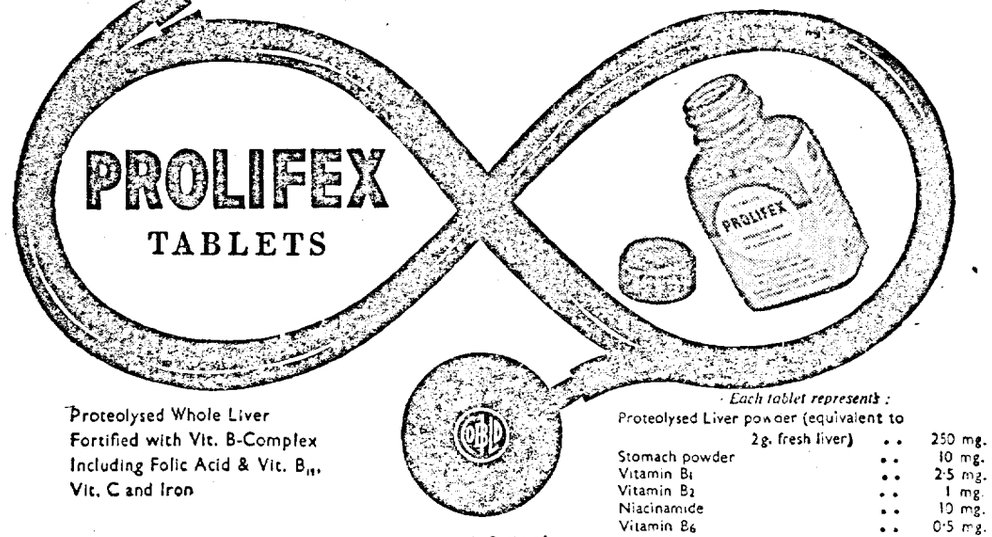

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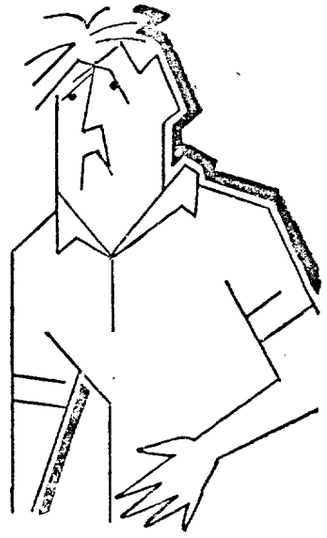

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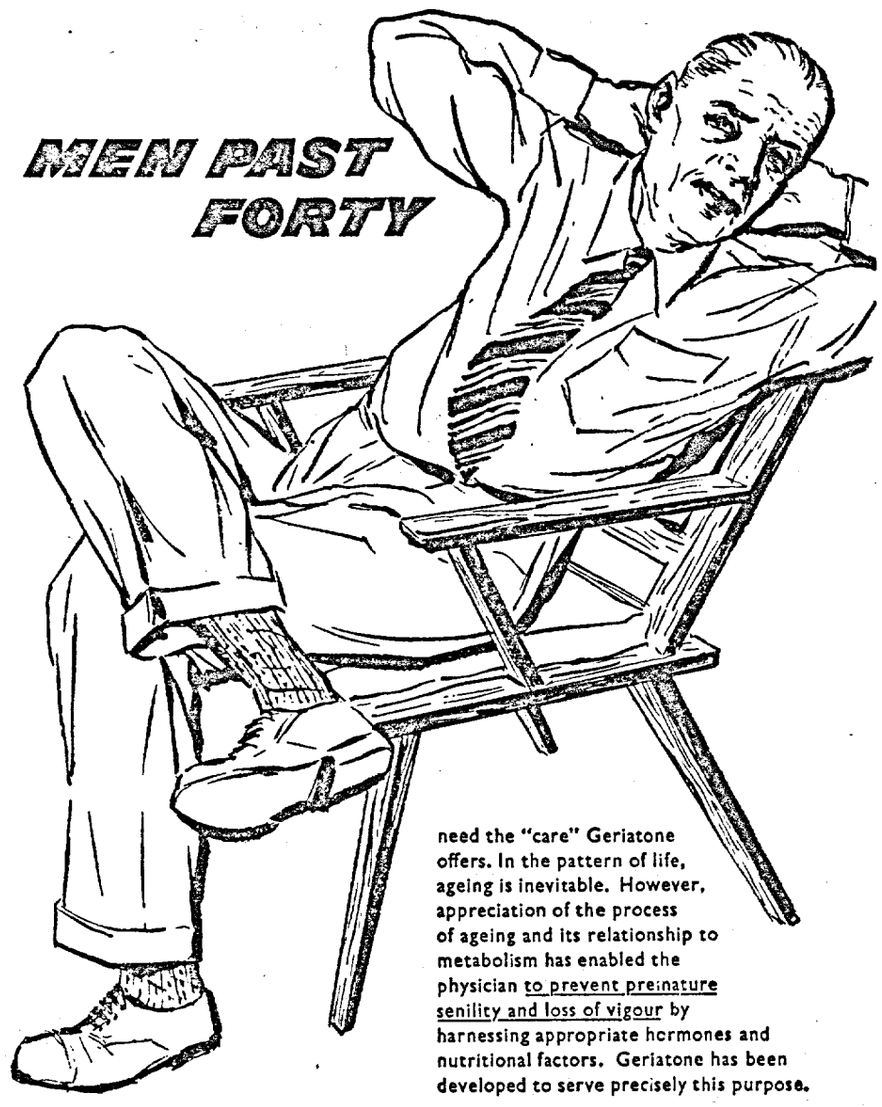

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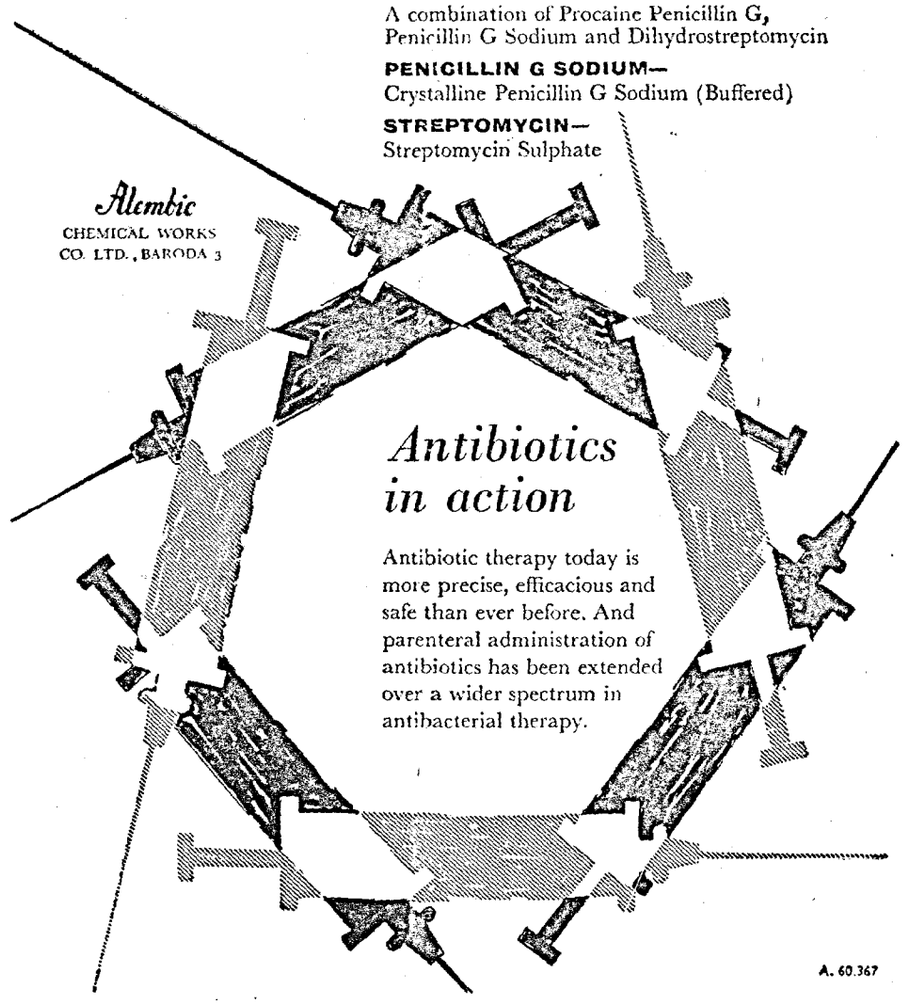

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