

MARCH 1934.

THESIS FOR THE DEGREE OF  
DOCTOR OF MEDICINE.

TITLES:

- (1). Occult and Latent Tuberculous Infection of Mesenteric Lymph Glands.
  
- (2). Description of two Stretcher Splints for the non-operative treatment of bone or joint Tuberculosis with notes on cases treated, photographs and sketches.

Presented by:

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LATENT AND OCCULT  
TUBERCULOUS INFECTION OF MESENTERIC GLANDS.

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TUBERCULOUS INFECTION OF MESENTERIC GLANDS.

INTRODUCTION.

Opie,<sup>(1)</sup> writing in the British Medical Journal says:

"I am inclined to believe that more knowledge of the pathology of tuberculosis can be obtained by careful examination of the tuberculous lesions of those who die from causes other than tuberculosis than from fatal instances of the disease.

The frequency of concealed tuberculous infection is thoroughly recognised, in great part as the result of almost innumerable studies made with tuberculin tests, but the variety and severity of the latent tuberculous lesions is not fully understood".

Shortly after graduation I became Resident Medical Officer at Grassington Sanatorium under W.M.Cumming, Esq., M.D., Ph.D. D.P.H. who suggested that I should carry out a bacteriological and histological investigation on mesenteric glands obtained post mortem from patients dying of diseases other than tuberculosis, and who showed no naked eye evidence of tuberculosis beyond slight apical scars.

This Sanatorium is situated some 70 miles from Leeds, the nearest teaching School, and although it had a well equipped laboratory for bacteriological work, it contained no histological equipment beyond a microscope.

The first step, therefore, was to arrange for the supply of post-mortem material - revise my histological technique, and obtain a microtome - embedding oven - hardening chemicals - wax - stains etc., Prof.M.J.Stewart, Pathologist, Leeds University, readily agreed to supply the post mortem material and post specimens to me twice a week in sterile containers. He also permitted me to spend a good deal of time in his laboratory revising my technique in section cutting, mounting, staining etc., To these two gentlemen I should like to acknowledge my indebtedness.

Latent and occult tuberculosis of tracheo-bronchial glands had already been investigated and my search of the literature has revealed a good deal of work done on latent and occult tuberculosis of the upper digestive tract but so far, I have been unable to discover any prior work on the lower digestive tract on the lines suggested above.

Infection by the respiratory route had dominated the field for many years until about 1900 when various workers demonstrated latent lesions in the tonsils.



The demonstration of latent and occult tuberculosis in mesenteric glands in patients dying of diseases other than tuberculosis, and who showed no post mortem macroscopic evidence of tuberculosis elsewhere seemed well worth investigating.

If positive results histologically, bacteriologically or by animal inoculation were obtained, it would give further proof, if such were necessary, that the tubercle bacilli can pass through the intestinal mucosa and reach the glands of the mesentery without leaving any trace of their pathway.

Koch and Banningarten both thought it difficult to admit the infection of these glands without the presence of a previous lesion in the intestinal walls.

#### REVIEW OF LITERATURE.

I have made a search of the complete set of the quarterly Cumulative Index Medicus in the B.M.A. library and can find no direct reference to any previous work on this specific subject but much literature exists on analogous lines.

Latham<sup>(2)</sup> points out the striking fact "that the curve of tuberculosis in children commences at the age of 6 months, reaches maximum about 2 years, and undergoes a remarkable fall between the third and fourth year; that is to say, tuberculosis is

*This passage has already been inserted in the Dr. Colwell's*

most marked at an age when infected milk would have most play and becomes less frequent as the children leave their milk years".

Latham goes on to say:-

"My impression is that both in adult and child the primary infection is by means of the lymphatic system; and up to  $3\frac{1}{2}$  years is often dependent upon the tubercle bacilli of an infected milk supply passing through either the upper or lower portion of the alimentary tract and eventually setting up changes in bronchial glands".

"We know further, that in very young children, on account of the frequency with which they are fed, milk is practically always in contact with the upper part of alimentary tract and that it is uncommon to perform post mortems on young children without finding semi-digested milk in stomach or intestines, so that, although the number of bacilli may be small, yet such bacilli as there are, have every opportunity of passing through the alimentary tract".

Again on p.1786:-

"With regard to infection from alimentary canal, we know that tubercle bacilli can pass through the intestinal mucous membrane without leaving any trace of their entrance and yet cause

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tuberculosis in more distant parts. Much has been made of the absence of ulceration of the intestine, except when the tuberculosis of the mesenteric glands is extreme".

"My impression is that when the tuberculosis of the mesenteric glands - which appear to be less susceptible to tubercle in the human subject than the bronchial glands - is extreme, it is due in many to secondary infection back against the lymph stream. Further it is a noticeable fact that tuberculous ulceration of the bowel nearly always has a vascular rather than a lymphatic distribution".

Bollinger (3) showed that on injecting dilute solutions of phthisical sputum into the peritoneum and intestine, the peritoneum remained sound in two-thirds of the cases, although the lungs became tuberculous.

Latham (2) in forty-five cases of tonsils removed post mortem or at operation, showed tubercle in seven cases by inoculation experiments. Case ages - three months to thirteen years.

Shennan and Still (4) in investigations on the proportion of ingestion and inhalation infections from post mortems conducted in Edinburgh and London hospitals for sick children, present results strikingly similar.

Shennan, in 28.1% of 331 cases in which he could trace the primary focus, the alimentary route was found, the bacilli had entered by some part of alimentary tract including pharynx and tonsils.

Still's figures were 29.1%.

Carmichael (5):- "Tubercle of the tonsils and glands".

"In 50 cases, 7 or 14% of Tonsils were shown to be tuberculous by microscopic section".

"One or two sections only of the tonsils were examined. If serial sectioning had been done no doubt a larger percentage would have been found".

Woodhead (6) has traced tuberculous infection by way of mesenteric, retroperitoneal, and bronchial glands and lymphatics to the lung and stresses ingestion route of infection via tonsils.

Mitchell (7) in his report to National Medical Research Committee on "Primary tuberculosis of faucial tonsils in children" states that:-

"The primary localisations of tuberculosis is still a much vexed question, especially in regard to the relative frequency in which infection takes place through the alimentary and respiratory tracts and also in what way the primary focus becomes the source of subsequent secondary and more generalised infection".



He found that culture from guinea pigs tissue which had been inoculated and found positive was difficult. This I also found difficult, contaminated tubes being an outstanding feature.

Mitchell thought that histological examination of entire tonsil was a reliable method, but inoculated guinea pigs to meet criticism and to determine the type of bacilli.

In his cultures he used alcoholic basic fuchsin to aid detection of scanty colonies as advocated by Cruickshanks (8). (B.M.J. 1912 - vol 11. P.1298).

Mitchell's Results:-

Group A. - Tonsils removed from cases with Tub.

Cerv.glands:

In 106 cases 41 cases were positive = 38%.

Group B. - Tonsils removed for hypertrophy only.

In 100 cases 9 cases were positive = 9%.

He points out "that in no case could tuberculosis of tonsil be diagnosed clinically nor could tuberculosis be diagnosed macroscopically after tonsils were enucleated".

"Inoculation experiments lagged very much behind histological examination due to large amount of contamination of culture tubes and deaths in guinea pigs due to septic infection".



Mitchell also points out that "in 406 samples of mixed milk he found 20% positive for tubercle bacilli; and that it requires a much larger dose of tubercle bacilli to infect a patient by the alimentary route than by respiratory route although milk from a single tuberculous cow may have more than sufficient (or a frankly tuberculous herd)."

The factors of virulence, quantity, and resistance of the body, apply equally to both routes.

Kingsford (9) held that tonsillar infection was blood spread.

Recklinghausen (10) held that tonsillar infection was a backward spread from lymph nodes of neck.

Dieulafoy (11), Orth and Baumgarten believe from results of experiments that the tonsils, as a result of ingestion infection, are a frequent primary source of infection.

Mitchell concludes that:-

- (1). "Tubercle of upper cervical glands develops from a primary focus in faucial tonsils much more frequently than is generally supposed.
- (2). That it can only be diagnosed by aid of a microscope or inoculation experiments.
- (3). Hypertrophied faucial tonsils may be the seat of primary tubercle, though rarely, compared with tonsils from cases of tuberculous cervical adenitis.

(4). That primary tuberculosis of faucial tonsils in children must be attributed to drinking of milk from tuberculous cows rather than from inhalation of human tubercle bacilli from droplet or dried sputum from consumptive patients".

Webster<sup>(12)</sup> repeated Mitchell's work on "Occult tuberculosis of tonsils in relation to tuberculous cervical adenitis" in Australia. His results are:-

Group A.

In 70 cases 31 tonsils were positive or 44.2% by histological examination

In 16 cases 9 tonsils were positive either histologically or by inoculation experiment  
= 56.2%.

Macroscopically - none were positive even after cross section, hence all were truly occult.

In eight cases positive by animal inoculation, four were bovine, three were human, one undetermined.

The four bovine cases had no family history of tuberculosis. Of the three human cases, two had positive family history.

The histological examination was not exhaustive, only one section through the centre of each tonsil being taken.

Group B.

In 46 cases of tonsils removed for simple hypertrophy (repeated tonsillitis or glandular swelling which subsided after tonsillectomy, and were regarded as non-tubercular), histological examination proved entirely negative.

Serial sectioning may have shown a small percentage positive. No animal tests were done in this Group.

Grouping these results:

Table 1.

Primary Tuberculosis of faucial tonsils in subjects with Tuberculous Cervical Adenitis.

Name	No. of Cases	Tonsils Tuberculous	Percentage
Carmichael 1910	50	7	14%
Hard & Wright 1909	12	9	-
Matthews 1910	8	5	-
Mitchell 1917			
(a) Histological	106	41	38%
(b) Animal Inoc.	92	20	21%
Webster 1927- 1929			
(a) Histological	86	37	43%
(b) Animal Inoc.	16	8	-

Table 2.

Results of investigation regarding Tuberculosis of Tonsils in persons presenting no clinical evidence of Tuberculosis.

Name	Date	No. of Cases	Tubercular Tonsils.
Kingsford (9)	1904	20	0
Latham (2)	1900	45	7
Dieulafoy (11)	1895	61 (Inoc).	8 (inoc).
Mitchell (7)	1917	100	9 (Hist.) 9 (Inoc.)
Mitchell (13)	1914	90	6 (Hist.) 9 (inoc.)
Diggle (14)	1924	75	5 (Hist.)
Webster (12)	1932	46	0 (Hist.)

The inferences to be drawn from the foregoing work are:-

- (1). Tuberculosis of the tonsils as a primary focus to cervical adenitis is much more common than was thought possible a short time ago.
- (2). That ingestion of tubercle bacilli in infected milk or food can and does infect the upper alimentary tract and plays a more important role than was hitherto given to it.
- (3). That it is not possible to diagnose tuberculosis of the tonsils clinically or macroscopically, even in those cases with gross cervical adenitis.
- (4). Where tuberculous cervical adenitis is present we can say that in 40% of cases the tonsils are the seat of primary focus.

(5). In simple hypertrophy of tonsils a small percentage may be tubercular.

Leonard (15) :-

In 161 complete autopsies in children between ages of 1 day to 14 years dying of all causes, 50 showed anatomic evidence of tuberculosis in one or more tissues of the body.

In 18 of those 50 cases he found the mesenteric nodes were the only sites of tuberculosis in the body. No other tissue was found which was the sole site of tuberculosis.

Hop (16) :-

In 14,000 cases found tuberculosis of mesenteric glands as the only demonstrable evidence of tuberculosis infection in the body -

in 1.4% of children  
in 0.8% of adults at autopsy.

Woodhead (17) :-

found tuberculous mesenteric lymphadenitis in 78.7% of autopsies on tuberculous children; and in 11% it was the only lesion present.

Other workers have shown from 1% to 20%.

Opie (18) :-

Estimates the incidence of latent mesenteric tuberculosis in U.S.A as 5%.



Stewart (19):-

made a study of 1000 consecutive autopsies for tuberculous lesions and in a detailed analysis of 20 cases of intestinal tuberculosis from the point of view of the primary lesion and the proximate cause of death, found 14 cases in which the primary focus was in the mesenteric glands. A primary pulmonary lesion was found in 5 cases and one case of primary hyperplastic tuberculosis of intestine. He concludes that these twenty cases of intestinal tuberculosis were extensions from the mesenteric glands to the gut.

Macgregor (20):-

points out that tuberculosis is pre-eminently a focal infection, the bacilli being established in the body in certain localised areas, most commonly in one or more group of lymphatic glands, or in the lungs, bones or other sites. From the primary focus, infection spreads at a subsequent date, by various channels, and manifests itself in new situations by the formation of fresh foci. The survival and multiplication of the bacilli in the body are apparently dependent upon their ability to establish themselves in foci, otherwise they perish or are excreted. It is a well known fact that in children the lymph glands are

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peculiarly prone to form a nidus for the development and a centre for the dissemination of tuberculous infection.

In 149 post mortems Macgregor found 42 cases of tuberculosis, and in every case infection in one or other of the three principal groups of glands, thoracic, cervical, or mesenteric, which were probably the primary, certainly the most advanced lesions in the body.

In these 42 cases, the distribution of advanced lesions in the three groups were as follows:-

Cervical glands	2 cases	= 5%
Thoracic glands	21 cases	= 50%
Mesenteric glands	22 cases	= 52%

In 3 cases in more than one group advanced caseous lesions were found.

In 33 fatal cases the distribution of advanced lesions were:-

Cervical glands	1 case	= 3%
Thoracic glands	19 cases	= 58%
Mesenteric glands	16 cases	= 49%

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OBJECTS OF THE PRESENT INVESTIGATION.

OBJECTS OF THE PRESENT INVESTIGATION.

- (1). To determine the percentage of cases in which histological evidence of tuberculosis could be demonstrated in mesenteric glands obtained from post mortems on patients dying of diseases other than tuberculosis, and in whom no macroscopic evidence of tuberculosis could be discovered.
- (2). If the glands were histologically positive, could tubercle bacilli be demonstrated by staining the sections by Ziehl-Nielsen method?
- (3). The percentage of mesenteric glands from which virulent tubercle bacilli could be recovered:-
  - (a). By direct culture
  - (b). By inoculation into the guinea pig
  - (c). By indirect culture through the guinea pig.
- (4). To determine the type, bovine or human, of the isolated strains, if any.
- (5). Whether there was histological evidence of tuberculous infection in glands giving negative inoculation or cultural results and vice versa.

PROCEDURE AND TECHNIQUE.



PROCEDURE AND TECHNIQUE.

Two or three mesenteric glands complete in their mesentery were removed from selected cases, in which systematic search failed to reveal macroscopic evidence of tuberculosis at post-mortem, beyond apical scars of doubtful origin. Such search naturally limited the number of cases available and only 32 cases were found suitable in the 8 months which I had to devote to the work, apart from routine hospital work and other experimental work on splints which I was carrying on at the same time.

A further difficulty was the fact that the glands had to be posted to me from Leeds in sterile glass containers and took 10 hours to arrive and in a few instances the glands were delayed in the post for some 15 to 20 hours. Spore bearing organisms multiply at an enormous rate and no doubt this accounted for the many failures in culture.

On arrival, the glands were washed in running water two or three times and then dissected out of their mesentery with sterile scissors and forceps. Two to four glands from each case were taken, cut in halves and examined for macroscopic evidence of tuberculosis. One half of each gland was put into a sterile mortar, cut up into small pieces and then ground up into a homogeneous mass, sufficient normal sodium hydroxide

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being added to make the mass fluid and to kill off the contaminating organisms.

If the glands were fibrous, sterile sand was added to assist grinding. Two layers of sterile muslin were used to cover the mortar while grinding.

If the glands had been delayed in the post, the fluid mass was pipetted into a sterile centrifuge tube and incubated for  $\frac{1}{2}$  hour at  $37^{\circ}\text{C}$ . in order to bring out spore bearing organisms which were responsible for heavy contamination of culture tubes.

The fluid mass was in each case pipetted off into a sterile centrifuge tube and centrifuged for 15 minutes at 3000 r.p.m.

The supernatant fluid was pipetted off with sterile pipette allowing the surface scum and sediment to come together at the bottom of the tube.

Sufficient normal hydrochloric acid was added to neutralise the alkaline mixture.

This formed the inoculum and the following steps were taken.

#### BACTERIOLOGY.

- (1). With a sterile platinum loop a smear film of the inoculum was made and stained ziehl-Neilsen.
- (2). Two tubes non-glycerinated egg medium) Lowe<sup>N</sup>stein's  
Two tubes glycerinated egg medium )medium  
were inoculated, sealed with waxed wool plugs )modified  
and put into the incubator at  $37^{\circ}\text{C}$ .

(3). The remaining inoculum was then drawn into a sterile 1 c.c. glass syringe and injected into the flank of a guinea pig.

The guinea pig was in each case injected with 0.9 c.c. anti-gas gangrene serum (B.&W.) equal to 500 units. This was done on the basis of the work carried out by Cumming, Hartfall and Thomson (21) who had a large mortality in guinea pigs when inoculating with tracheo-bronchial gland material.

#### HISTOLOGY.

The other half of the glands from each case was hardened in formol for the requisite period, taken up through methylated spirit, alcohols, to xylol and embedded in wax ready for sectioning.

From each case serial sections were taken, averaging every 10th to 20th section, depending on the size of the gland, two adjacent sections being taken at each point. These were mounted and stained Haemalum and Eosin, the second sections at each point being retained for staining by Ziehl-Neilsen if the corresponding sections stained H. & E. were found histologically positive. In this way some 20 to 30 sections were examined for each case.

ANIMAL INOCULATION.

All guinea pigs found dead or still alive after two months - in which case they were killed - were examined post mortem with precautions to ensure sterility, for evidence of tuberculosis. Spleen smears, enlarged glands, or any evidence of exudate at the site of injection were inoculated on to culture tubes or injected into a fresh guinea pig for investigation.

Owing to lack of funds only one guinea could be spared for initial injection in each case. Dr. Cumming was at the same time carrying on his well known investigations on bovine tuberculosis and using a good many animals.

Pigs

RESULTS OF THE INVESTIGATION.



RESULTS OF THE INVESTIGATION.

Thirty-two cases in all were examined and ten of those cases call for comment as being positive either histologically or bacteriologically.

Charts 4 and 4A give the details of each of the 32 cases.

Table No.5 is an abstract giving details of the ten positive cases.

The following table gives these results in a simplified form.

Table No.3.

Case No.	P.M.No.	Age	Bact.	Hist.
1	593.32	20	+	-
3	612.32	70	+	+
4	613.32	55	-	+
7	625.32	68	+	+
15	96.33	62	+	-
22	217.33	67	-	+
27	245.33	64	+	-
29	247.33	69	-	+
30	250.33	57	-	+
31	279.33	38	-	+
Totals			5	7

From the above table it will be seen that:

Two cases were positive both histologically and bacteriologically.

Seven cases were positive histologically.

Five cases were positive bacteriologically for acid-fast bacilli.

## COMPLETE CHART OF CASES WITH RESULTS

CASE NO.	P.M. NO	AGE	SEX	CAUSE OF DEATH	NAKED-EYE APPEARANCE OF GLANDS	HISTOLOGY	
						SERIAL SECTIONS STAINED H & EOSIN	Z-N
1	593.32	20	M	Cerebral Haemorrhage Cerebral Haemangioma	Normal	Negative	
2	601.32	2 $\frac{10}{12}$	F	Meningococcal Meningitis	Normal	Negative	
3	612.32	70	F	C.I.N. Gall Stones Post operative death	Anthracotic	Positive	Negative
4	613.32	55	M	Stricture of Urethra Extravasation of Urine c̄ infection.	Anthracotic	Positive	Negative
5	615.32	9 $\frac{9}{12}$	F	Meningococcal Meningitis	Normal	Fibrosis No Tub.	Negative
6	621.32	51	M	Bronchial Carcinoma Infiltration & Secs.	Anthracotic	Negative	
7	625.32	68	M	Carc. of Rectum. Cholelithiasis. G.B. Carcinoma?	Anthracotic	One slide min. amt. caseation. Fibrosis, Endarteritis	Negative
8	636.32	11 $\frac{11}{52}$	M	Meningococcal Meningitis	Normal	Negative	
9	641.32	68	F	Bronchiectasis & Fibrosis of lungs.	Anthracotic	Negative	
10	644.32	37	M	Saddle Ulcer of Stomach. Basal Broncho-Pneumonia.	Normal	Negative	
11	674.32	48	M	Scirrhus Carc. of Pylorus. Congestion and Oedema of Lungs.	Normal	Negative	
12	75.33	14	F	Broncho-Pneumonia	Normal	Negative	
13	77.33	43	M	Carc. of Stomach. Broncho-pneumonia - Peritonitis.	Normal	Negative	
14	85.33	51	F	Aplastic Anaemia	Normal	Negative	
15	96.33	62	M	Cerebral Haemorrhage Silicosis & Anthracosis.	Marked Anthracosis.	Negative	
16	104.33	6 $\frac{6}{12}$	F	Broncho-Pneumonia	Enlarged Normal.	Negative	
17	105.33	53	M	Atheroma of Aorta & Cerebral vessels Hypertrophy L.V. c̄ Sclerosis.	Anthracotic	Negative	
18	106.33	60	M	Heart failure. Chronic Emphysema Sub-acute Nephritis.	Anthracotic	Negative	
19	117.33	69	F	Duod <sup>1</sup> . Stenosis. Chronic Nephritis Terminal pneumonia.P.O.	Anthracotic	Negative	
20	122.33	2 $\frac{10}{12}$	M	Mongol child. Acute Bronchitis. Diverticulum	Enlarged Normal	Negative	
21	182.33	39	M	Purulent B. Pneumonia Lung Abscess.	Anthracotic	Negative	
22	217.33	67	M	Atrophic Cirrhosis of Liver Prim. small cell Carcinoma.	Anthracotic	Positive Fibrotic	Negative
23	230.33	1 $\frac{10}{12}$	F	Aleukaemic leukaemia Partial collapse posterior half L.Lobe.	Normal	Negative	
24	231.33	5 $\frac{11}{12}$	M	Broncho-pneumonia.L.L.Lobe. Emphysema Appendectomy	Normal	Negative	
25	243.33	1	F	Enteritis	Normal	Negative	
26	244.33	5 $\frac{5}{12}$	M	Sinus Thrombosis Hydrocephalus. Mal-dev. of brain.	Normal	Negative	
27	245.33	64	M	P.M.A. c̄ Resp. failure. Ch. Bronchitis. Emphysema.	Anthracotic	Negative	
28	246.33	7	F	Diabetes. Pyæmic abscess R. lung.	Normal	Negative	
29	247.33	69	M	Carcinoma Oesophagus Broncho-pneumonia	Anthracotic	Positive Fibrotic	Negative
30	250.33	57	M	Carcinoma of Oesophagus Hypostatic Pneumonia	Anthracotic	Atrophic Fibrotic	
31	279.33	38	F	Neuro-myelitis optica. Septic Meningitis & Cystitis.	Normal	Positive	Negative
32	280.33	28	M	Lobar Pneumonia Fibro-purulent pleurisy	Normal	Negative	



## COMPLETE CHART OF CASES WITH RESULTS.

CASE NO.	P.M. NO.	AGE	SEX	FILM SMEAR of INOCULUM Z-N.	DIRECT CULTURE FROM INOCULUM	SUB CULTURE	GUINEA PIG INOCULATION					REMARKS
							DATE INOC.	DATE OF DEATH	CAUSE OF DEATH	P.M. EVID. TUB.	CULTURE RESULT	
1	593.32	20	M	-	+ A.F.B.	-	G.P.3 15.11.32	29.11.32	?	No.Tub. Haem.Exud. Site of Inoc.	Negative	Negative Culture from exudate at site of injection
2	601.32	2 $\frac{10}{12}$	F	-	-	-	G.P.4 18.11.32	27.11.32	Broncho-Pneu.	Tub ? exudate	Pneu. G.Gang.	G.Pig.5 inoc. exudate = neg. Spleen smear neg.
3	612.32	70	F	-	+ A.F.B.	+A.F.B	G.P.6 29.11.32	Jan 1933	?	?	?	Died when I was away and P.M. was not done.
4	613.32	55	M	-	-	-	G.P.7 29.11.32	28.12.32	Cannabal-ism.	Negative	Negative	G.Pig perfectly normal.
5	615.32	$\frac{9}{12}$	F	-	-	-	G.P.8 30.11.32	21.1.33	Broncho-Pneu.	Negative		
6	621.32	51	M	-	-	-	G.P.9 3.12.32	10.12.32	Septic sore at site of Inoc.	Negative	Negative	Site of inoc. exudate used into G.P.12. Killed 12.3.33. Healthy. Spleen & liver smears negative.
7	625.32	68	M	-	+ A.F.B.	-	G.P.10. 6.12.32	6.1.33	Broncho-Pneu.	Negative		
8	636.32	$\frac{11}{12}$	M	-	-	-	G.P.11. 10.12.32	Killed 12.3.33	Killed	Healthy	Negative	Spleen and liver smears all negative.
9	641.32	68	F	-	-	-	G.P.13 14.12.32	9.1.33	Pneu.	Negative	-	
10	644.32	37	M	-	-	-	G.P.14 17.12.32	29.3.33	Enteritis	Negative		
11	674.32	48	M	-	-	-	G.P.1/33	13.2.33	Enteritis	Negative		
12	75.33	14	F	-	-	-	G.P.2/33 3.2.33	14.3.33	Enteritis	Negative		
13	77.33	43	M	-	-	-	G.P.3/33 7.2.33	20.3.33	Enteritis	Negative		
14	85.33	51	F	-	-	-	G.P.4/33 13.2.33	25.2.33	Septic-aemia	Negative		Glands were two days in post.
15	96.33	62	M	-	+ A.F.B.	-	G.P.5/33 16.2.33	13.3.33	Exudate Pneu.	Negative		Exudate smear Film neg.
16	104.33	$\frac{6}{12}$	F	-	-	-	G.P.6/33 24.2.33	29.3.33	Enteritis	Negative		
17	105.33	53	M	-	-	-	G.P.7/33 24.2.33	28.3.33	Strept. Perit <sup>s</sup> .	Negative		
18	106.33	60	M	-	-	-	G.P.8/33 28.2.33	16.3.33	Enteritis	Negative		
19	117.33	69	F	-	-	-	G.P.9/33 4.3.33	21.3.33	Pneum <sup>l</sup> Exudate	Negative		
20	122.33	2 $\frac{10}{12}$	M	-	-	-	G.P.10/33 G.P.11/33 6.3.33	4.3.33 25.3.33	Enteritis Enteritis	Negative		2 days in post. Inoc. in G.P.11 given $\frac{1}{2}$ hr. incub. before injection.
21	182.33	39	M	-	-	-	G.P.12/33 12.4.33	26.4.33	Sepsis	Negative		
22	217.33	67	M	+	-	-	G.P.13/33 5.5.33	15.6.33	Enteritis	Negative		
23	230.33	1 $\frac{10}{12}$	F	-	-	-	G.P.14/33 12.5.33	12.7.33	Killed	Healthy.		
24	231.33	5 $\frac{11}{12}$	M	-	-	-	G.P.15/33 13.5.33	13.7.33	Killed	Healthy.		
25	243.33	1	F	-	-	-	G.P.16/33 20.5.33	16.7.33	Enteritis	Negative		
26	244.33	$\frac{5}{12}$	M	-	-	-	G.P.17/33 20.5.33	14.8.33	Killed	Negative		
27	245.33	64	M	-	+ A.F.B.	-	G.P.18/33 23.5.33	27.6.33	Septic-aemia	Negative		Smears from site of inoc. enlarged Glands also neg.
28	264.33	7	F	-	-	-	G.P.19/33 23.5.33	20.8.33	Killed	Negative		
29	247.33	69	M	-	-	-	G.P.20/33 23.5.33	20.8.33	Killed	Negative		
30	250.33	57	M	-	-	-	G.P.21/33 23.5.33	20.8.33	Killed	Negative		
31	279.33	38	F	-	-	-	G.P.22/33 14.6.33	4.8.33	Enteritis	Negative		
32	280.33	28	M	-	-	-	G.P.23/33 14.6.33	15.8.33	Enteritis	Negative		

KEY: A.F.B. = ACID-FAST BACILLI.



TABLE NO: 5.

## ABSTRACT OF POSITIVE CASES.

CASE NO.	P.M. NO.	AGE	SEX	HISTOLOGY	FILM SMEAR INOCULUM	DIRECT CULTURE FOUR TUBES	SUB-CULTURE	GUINEA PIG
1	593/32	20	M	Negative	Negative	A.F.B. in chinese figures in smear films from surface of two tubes.	Negative	Negative
3	612/32	70	F	Small tubercle showing caseation running through all sections serially.	Negative	A.F.B. in smear films from surface of three of the four tubes. Evidence of pin point colonies and commencing growth.	Small pin point colonies in one tube. Smear film showed A.F.B.	Died after two months while I was away on business. P.M. not carried out.
4	613/32	55	M	Tubercle running through sections and showing caseation. Epitheloid cells and one giant cell.	Negative	Negative	Negative	Died cannibalism one month.
7	625/32	68	M	Fibrotic tubercle with minimum amount of caseation and G.cell of poor shape - well capsulated old tubercle.	Negative	A.F.B. in smear film one tube. Very small and shrivelled. No evidence of growth on tube.	Negative	Negative.
15	96/33	62	M	Negative	Negative	Two tubes were scraped and smear film showed A.F.B. which were scanty and poor staining.	Negative	Negative.
22	217/33	67	M	Fibrotic tubercle running through sections.	Positive	Negative	Negative	Negative.
27	245/33	64	M	Negative	Negative	One tube scraped and smear film showed a few A.F.B. No growth.	Negative	Negative.
29	247/33	69	M	Fibrotic nodule through sections.	Negative	Negative	Negative	Negative.
30	250/33	57	M	Glands atrophic Fibrotic Tubercle.	Negative	Negative	Negative	Negative.
31	279/33	38	F	Small tubercle under capsule showing caseation and running through sections.	Negative	Negative	Negative	Negative.

KEY: A.F.B. = ACID-FAST BACILLI.

DISCUSSION AND SUMMARY.

DISCUSSION AND SUMMARY.

Thirty-two cases were examined histologically and bacteriologically for evidence of occult or latent tuberculosis in mesenteric glands. In each case the glands were obtained at post-mortem from patients dying of disease other than tuberculosis, and who showed no naked eye evidence of tuberculosis after careful search by the pathologist.

The post-mortem reports of the positive cases are appended.

Guinea Pig Inoculation proved negative in all cases and it is difficult to account for this in those cases which were positive histologically. Sufficient protection against gas-gangrene infection was given by the injection of 500 units of anti-gas gangrene serum, as in no case was death due to this cause. Sixty per-cent of the guinea pigs were alive forty days after inoculation. Broncho-pneumonia, enteritis or other intercurrent disease accounted for most of the deaths, while three or four died as a result of cannibalism.

Case 3, P.M.612, G.P.6. may have been positive, for it died three months after inoculation and had been showing signs of ill health.

It died while I had to be away on business and a post-mortem was, unfortunately, not carried out.



Guinea pig death rate.

Four died between	8 to 14	days after inoculation			
Nine "	"	14 to 28	"	"	"
Twelve"	"	40 to 60	"	"	"
Seven were killed	60 to 90	"	"	"	"

Although the Sanatorium where the work was carried out is very exposed to the elements and the work was carried out in the winter, the fact that 60% lived forty days after inoculation excluded criticism of these conditions as having a bearing on the results.

The preparation of the inoculum was always carefully done and the method used was that adopted by Cumming, Hartfall and Thomson <sup>(21)</sup> in their work on Occult Tuberculosis of Bronchial Glands.

When a guinea pig died or was killed after two months, culture tubes and a further guinea pig were inoculated with any exudate at the site of inoculation, macerated enlarged glands nearest the site, or spleen smear if there was any evidence that it might possibly be tubercular in origin. Guinea pigs and culture tubes thus treated were all negative.

The only reasonable conclusion is that in this series, the number of bacilli was too small or the virulence too attenuated to overcome the resistance of the guinea pigs.

Age incidence had also a bearing on these results since eight of the ten positive cases were over 55 years of age; the remaining two were 20 years and 38 years respectively. Since there was no active tuberculosis in any of these ten cases the patient's resistance was great, and it may be confidently assumed that the lesions were of very old standing and the virulence of the infecting organisms very low.

#### CULTURAL EXPERIMENTS.

The results of cultural experiments were likewise very disappointing.

In only one case was the direct smear film from the inoculum positive for acid-fast bacilli, and culture from this inoculum proved negative.

In one case only, P.M.612/32, was there any real evidence of growth of tubercle bacilli. Small pin point colonies could be made out at one period and it was possible in this case to sub-culture those colonies for a little time but growth soon stopped and contamination set in.

It was only after two months work and becoming somewhat despondent with the results, that I decided to scrape the surface growth on a few culture tubes, make films, and examine them microscopically for acid-fast bacilli. Finding one case P.M.593/32 to be positive, I decided to test all tubes in this manner.

Had this not been done, a completely negative result would have had to be recorded. In making these smear films, the most likely looking growths were picked off and then the growth was smeared over the surface of the tube in the hope that spreading would encourage the growth.

In five cases out of the thirty-two, acid-fast bacilli were demonstrated in smear films (stained Z.N.) from the surface growth on culture tubes.

In one of these five cases I was able to transfer the acid-fast bacilli on to sub-culture tubes, and ten days afterwards was able to demonstrate by smear films that they still existed although they showed no real evidence of colony growth.

The five positive cases are as follows:-

Case No.1 P.M.593/32.

Smear film showed groups of A.F.B. forming typical chinese-like figures from surface of one tube and scanty A.F.B. from surface of a second tube.

Sub-culture - negative.

Case No.3 P.M.612/32.

Smear film showed small groups of A.F.B. from three out of four tubes.

Sub-culture - growth suggestive, and smear film showed A.F.B. Contamination set in and growth failed to go beyond suggestive stage.

Case No.7. P.M.625/32.

Smear film showed A.F.B. but rather shrivelled in appearance.

Sub-culture - negative.

Case No.15.P.M.93/33.

Smear film from two tubes showed scanty A.F.B.

Sub-culture - negative.

Case No.27. P.M.245/33.

Smear film from one tube showed scanty A.F.B.

Sub-culture - negative.

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Case 22 P.M.217/33 was the only case in which a smear film of the original inoculum showed A.F.B. when stained Z.N. but culture smears were negative.

The term-acid-fast bacilli-has been purposely used, for there is no positive evidence that they were Koch's Bacilli since cultural characteristics and animal inoculation could not be tested. In two cases, Nos 2 and 7, however, histological evidence was positive so that the assumption that they were tubercle bacilli would be strongly supported.

In case No.1 the A.F.B. arranged themselves in chinese-like figures and this is again strongly suggestive.

In looking for an explanation of these results, one naturally looks for weaknesses in technique. The method of preparing the inoculum /



was, as already stated, that used by Cumming etc., in tracheo-bronchial glands and one saw no reason for changing it.

The use of normal sodium hydroxide for killing off the contaminating organism in the preparation of the inoculum, may have been too strong for the tubercle bacilli in their attenuated form, and one would have liked to experiment with weaker solutions and other methods of preparation, had time and material been available. It is only by reviewing and analysing the results of one series of experiments, or drawing from the experience of other workers, that one can improve on one's technique.

The fact that a large percentage of tubes showed no growth whatever over long periods, strongly supports the view that the Na O.H. may have been too strong and in addition to accounting for the contaminating organisms, may have dissolved the lipid sheath of the attenuated tubercle bacilli and caused their disintegration.

The medium used was Lowenstein's Egg Medium. This medium was the standard in use on other experimental work at the sanatorium and gave good results with growth from sputa. Even better results are said to be obtained on media of more recent origin and it is possible that its use may have influenced the results in this series. Concurrent inoculation of

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different media from the same inoculum would have been most interesting. The failure to isolate and type the acid-fast bacilli was a source of disappointment. The results, however, are recorded for future reference and for the benefit of those who may care to undertake similar experiments.

SUMMARY OF CULTURAL EXPERIMENTS.

- (1). In five out of thirty two cases A.F. Bacilli were demonstrated in smear films from the surface growth on culture tubes inoculated with macerated mesenteric glands from patients dying of diseases other than tuberculosis and who showed no evidence of tuberculosis at post mortem.
- (2). No naked eye growth typical of tuberculosis could be detected in any tube although in one case the growth was suggestive and it was possible to carry that growth on to sub-culture, though only for a short time.
- (3). The term acid-fast bacilli has been purposely used as there is no positive proof that they were tubercle bacilli since it was not possible to get a pure culture, or a sufficiently pure culture, for injection purposes, the growths being extremely scanty and the bacilli so attenuated.

DISCUSSION AND SUMMARY CONTINUED.

HISTOLOGY.

Seven cases out of thirty two, were positive histologically.

In four of the seven cases the glands were caseous, the remaining three were purely fibrotic.

In all seven cases healing was by fibrosis, in no case was there any evidence of calcification.

The seven cases are as follows:-

Case No.3 P.M. 612/32.

Macroscopic - glands normal.

Microscopic - 30 serial sections,

representing every 20th section were cut, stained H.E and examined.

A small tubercle showing caseation ran through the whole of the serial sections.

The tubercle had a well formed capsule.

Corresponding sections were stained Z.N. but no tubercle bacilli were observed.

Case No.4 P.M. 613/32.

Macroscopic - glands normal.

Microscopic - 24 serial sections

representing every 20th section were examined. A distinct area of caseation with scanty epithelioid cells around it could be seen running through all sections. The capsule in this case was not well formed. A giant cell was seen in two sections.

Corresponding sections stained Z.N. were negative for tubercle bacilli.

Case No.7. P.M. 625/32.

Macroscopic - glands were firm but normal.

Microscopic - 30 serial sections were taken from four half glands in this case and examined.

Sections corresponding to three half glands showed increased fibrosis, endarteritis, and disintegration of Malpighian corpuscles. One showed a completely fibrosed nodule.

Sections from the fourth half gland showed an old standing fibrotic nodule with a minimum of caseation, disintegration of Malpighian corpuscles, one or two giant cells and a ring of epithelioid cells.

The tubercle was well capsulated and lay just under the capsule of the gland.

Corresponding sections stained Z.N. were negative for tubercle bacilli.

Case No.22 P.M.217/33.

Macroscopic - glands firm but normal looking.

Microscopic - 24 serial sections were cut and examined. An old fibrotic tubercle ran through all sections.

Corresponding sections stained Z.N. were negative for tubercle bacilli.

Case No.29 P.M.247/33.

Macroscopic - small glands, normal but firm.

Microscopic - 20 serial sections examined. An old fibrotic tubercle ran through all sections.

Corresponding sections stained Z.N. were negative.

Case No.30. P.M. 250/33.

Macroscopic - glands atrophic and fibrotic.

Microscopic - 20 serial sections examined.

All sections showed a healed fibrotic tubercle.

Corresponding sections stained Z.N. were negative.

Case No.31. P.M. 279/33.

Macroscopic - glands normal

Microscopic - 24 serial sections stained H.E. and examined. A small caseous tubercle ran through all sections just under the capsule.

Corresponding sections stained Z.N. negative.

A few other cases showed fibrotic changes in the glands microscopically but none was included in which any doubt existed as to the cause being tuberculosis.

Illingworth and Dick in their recent book on pathology say: "Tuberculous mesenteric glands tend to heal spontaneously by fibrosis and ultimate calcification is not common."

This was borne out by the cases reviewed above, no case showing calcification.

Age incidence.

The ages of the seven histologically positive cases, varied from 38 years to 70 years, six of them being over 55 years of age. In the five cases which were positive bacteriologically for acid-fast bacilli, one was aged 20 years and four were over 60 years of age.

Of much more interest would have been a series of cases in which the ages fell within the first decade of life but the difficulty is in getting the material.

The type of lesion found in the above series namely - much fibrosis and a minimum of caseation - was to be expected considering the ages of the patients, and lends confirmation to the view that tubercle bacilli may pass through the first line of defence, namely the mucous membrane, and be arrested in the second line, namely the glands, and there lie latent for a long period ready to be reactivated either by a fresh infection or by a breakdown of the patient's resistance.



In the above series we must assume that the lesions were of such old standing, and the bacilli so attenuated, that the breaking down of the patient's resistance by other disease had no influence on the tuberculous lesions. The fact that tubercle bacilli were unable to be cultured would point to their being non-viable.

How long the bacilli can remain viable in non-active lesions such as were found in the present series, is difficult to say. A series of such cases embracing different age periods would be required and also the date upon which the patient first became tubercularised.

There can be no doubt that the pathway to the mesenteric glands in these cases was via the alimentary canal, and although the number of the cases is too small to give percentage results, we may, taking histological results only, say that 7 cases in 32 or 22% showed latent tuberculosis in mesenteric glands.

Wang (22) in 10 mesenteric calcareous or caseous glands found at necropsy in cases showing no other lesions of a possible tuberculous nature, found one which yielded material capable of transmitting tuberculosis to the guinea pig and again in 5 mesenteric glands from children, two were found to transmit bovine tuberculosis to guinea pigs.

Shennan & Still's figures were 28.1% for alimentary route of infection in children.

Mitchell found the tonsils, which are to the upper digestive tract what the mesenteric glands are to the lower digestive tract, were the site of the primary lesion in 38% of cases of tuberculous cervical adenitis, and in 9% the tonsils were the site of latent tuberculosis in cases where simple hypertrophy of the tonsils was the only clinical finding.

Opie estimates the incidence of latent mesenteric tuberculosis at 5% in U.S.A.

Macgregor found the mesenteric glands to be the site of the most advanced lesion in 22 out of 42 cases of tuberculosis and in 16 out of 33 fatal cases.

Stewart, in a detailed analysis of 20 cases of intestinal tuberculosis found the mesenteric glands in 14 cases to be the site of primary focus and concludes that the 20 cases of intestinal tuberculosis were extensions from the glands.

Whilst none of the previous work mentioned is quite comparable with that under review, the general indications all tend to the same view.

On page 15 are set out five specific objects of this investigation.

(1). The first was:-

To determine the percentage of cases in which histological evidence of tuberculosis could be demonstrated in mesenteric glands obtained from post mortems on patients dying of diseases other than tuberculosis, and in whom no macroscopic evidence of tuberculosis could be discovered.

The answer to this question is that in seven cases out of 32, or 22%, there was histological evidence of healed or healing tuberculosis.

(2). The second object was:-

To determine if tubercle bacilli could be demonstrated in the histologically positive sections.

In none of the histologically positive cases could this be demonstrated.

(3). The third object was:-

To determine the percentage of mesenteric glands from which virulent tubercle bacilli could be recovered (a). by direct culture  
(b). by inoculation into guinea pigs  
(c). by indirect culture through guinea pigs.

The answer to object 3:-

(a). that in none of the 32 cases could

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tubercle bacilli be cultured, but that in five cases the presence of acid-fast bacilli very clearly resembling tubercle, could be demonstrated in smear films from the surface of the culture tubes when stained Z.N.method; that there is little doubt that they were tubercle bacilli, but in the absence of pure growth, one is not justified in saying so definitely.

The answers to (b) and (c) are in the negative.

(4). The fourth object was:-

to determine the type, bovine or human of the isolated strains.

As in no case was there colony growth, this object was unattainable.

(5). The fifth object was:-

to determine whether there was histological evidence of tuberculosis in glands giving negative inoculation or cultural results or vice versa.

The results in this series show that culture, and inoculation into guinea pigs, lag behind histological evidence. This is in agreement with Mitchell's work on Tonsillar Tuberculosis. It is, of course, more likely that such would be the case in occult or latent tuberculosis than in active tuberculosis.

Conclusion:

On histological evidence alone then, this series goes to prove that, as in the upper digestive tract so also in the lower, tubercle bacilli may pass through intact mucous membrane without leaving a trace of their pathway, be arrested in the nearest lymph glands, and there lie latent and occult for a long period awaiting the opportunity for further progression.



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POST MORTEM REPORTS ON THE TEN CASES

UNDER REVIEW.

ABSTRACTS.

CASE 1. P.M. 593/32 M. Aet. 20 yrs.

Anatomical Diagnosis: Haemangeomatous tumour springing from dura and penetrating top of right occipital lobe. Gross rupture with fatal subdural haemorrhage.

Clinical Diagnosis: Cerebral aneurysm.

Subject: Well nourished and well developed.

Brain & Membranes: Large clot underneath dura over right cerebral hemisphere. Vascular tumour - haemangioma. Softening of brain.

Supra-renals: Normal.

Kidneys: Congested.

Stomach & Duodenum: Slight congestion.

Spleen: Dense adhesions to neighbouring structures.

Pleurae: Normal

Lungs: Slight thickening of pleura over right apex, congested and slightly oedematous.

Bronchial Glands, Little anthracosis.

Intestine: One or two polypi, otherwise normal.

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CASE 3. P.M. 612/32 F. Aet. 70 yrs.

Anatomical Diagnosis.	Chronic interstitial Nephritis, Gall stones. Duodenal scar. (post-operative death).
Clinical Diagnosis:	Uraemia - ileus.
Subject:	Well developed - emaciated elderly woman.
Peritoneum:	No peritonitis.
Pleural Sac:	Slight adhesions left apex, otherwise healthy.
Lungs:	Congested and Oedematous - emphysema of borders.
Spleen:	Slightly fibrous - congested - otherwise normal.
Kidneys:	Reduced in size, granular surface, renal vessels thickened.
Mesenteric lymph nodes:	A few calcified, others apparently normal.
Duodenum:	Old scar.
Small Intestine:	Slightly dilated upper part - contracted below. Mucous surface shows slight irregular congestion, especially Jejunum.
Lower Ileum:	Perfectly healthy.
Colon:	Patchy congestion.

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CASE 4. P.M. 613/32 M. Aet. 55.

Anatomical Diagnosis: Stricture of Urethra.  
Extravasation of Urine.  
Infection of abdominal wall -  
Surgical treatment.

Oesophagus: Contains pus, otherwise normal.

Tracheo-Bronchial  
Glands: Anthracotic.

Pleurae: A few small subpleural nodules  
over which pleura is slightly  
scarred, particularly right  
side.  
Adhesions on left side at middle  
of anterior surface of upper  
lobe.

Lungs: Right lobe is adherent to  
middle of anterior one-third  
of fissure.  
Lungs are enlarged, oedematous  
and heavily anthracotic.

Spleen: Enlarged, twice normal, soft  
toxic looking.

Intestines: Normal.

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CASE 7. P.M. 625/32 M. Aet.68.

Anatomical Diagnosis:	Carcinoma of Rectum - colotomy. Cholelithiasis. Carc.of gall bladder?
Oesophagus:	Normal.
Tracheo-Bronchial Glands:	Anthracotic.
Lungs and Pleurae:	Adhesions over whole of right lung of considerable standing. Left pleura is free. Lungs - normally divided lobes. Slight collapse of left base.
Large intestine:	Diffuse carcinoma.

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CASE 15. P.M.96/33. M.Aet.62.

Anatomical Diagnosis: 1. Right sided cerebral haemorrhage.  
2. Silicosis of lungs.  
3. Anthracosis of abdominal glands.

Oesophageal Glands: Tough  $\bar{c}$  carbon

Tracheo-Bronchial Glands: Intensely anthracotic, hard, enlarged. Some are silicotic.

Pleurae: Some haemorrhages under pleurae which give it a "Shotty" feel on posterior surface of left lung. Numerous small white spots scattered under pleura and in some areas small pleural scars with one larger one on posterior aspect of left lung. Adhesions and scars both apices.

Lungs: Some nodules diffusely scattered through parenchyma, one especially under pleura. This is particularly marked at apices but there is no evidence of tuberculosis. Lungs are somewhat congested. The nodules have typical appearance of silicosis.

Peritoneum: Normal.

Spleen: Capsule thickened, parenchyma soft.

Stomach: Glands lesser curvature contain carbon. Stomach normal.

Intestines: Normal.

Condition of lung is entirely independent of cerebral haemorrhage.

Attention drawn to anthracosis of abdominal glands.

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CASE 22. P.M.217/33 M. Aet. 67 years.

Anatomical Diagnosis: Atrophic Cirrhosis of liver & small primary carcinoma.

Clinical Diagnosis: Abdominal Carcinomatosis.

Lungs: Large, irregular, emphysematous and deeply anthracotic. Irregular pleural thickening at both apices with adhesions at bases.

Tracheo-Bronchial Lymph Nodes: Not much enlarged; contain a large amount of coal dust.

Larynx: Ossified.

Abdo. Lymph Nodes: Coeliac and even mesenteric lymph nodes show anthracotic pigmentations.

Spleen: Shows several irregular flat fibromata on lateral surface.

Intestines: Normal.

Liver: Nodular, rough irregular & fibrous bands separating nodes. 6-8 m.m. dia. & soft faintly lobulated tumour tissue greyish-yellow, haemorrhagic areas, bile stained.

Subject: Well developed but emaciated & distended abdomen.

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CASE 27. P.M. 245/33 M. Aet.64.

Clinical Diagnosis: Prog.Muscular Atrophy.

Anatomical Diagnosis: P.M.A.̄ respiratory failure.  
Chronic bronchitis, emphysema,  
old apical tubercle.

Oesophagus: Normal.

Tracheo-bronchial Glands: Small, anthracotic.

Pleurae: Old dense adhesions both apices.

Lungs: Typical apical scarring both sides. Puckered mass on right side of old tubercle. Marked emphysema and chronic bronchitis.

Intestines: Normal, except for small transverse ulcer.

Spleen: Normal.

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CASE 29. P.M.247/33 M. Aet. 69.

Anatomical Diagnosis: Carc.of Oesophagus. Extensive broncho-pneumonia both lower lobes. Old scarring at apex in right middle lobe. Diverticulosis of pelvic colon.

Lt.Pleural Sac: Very firm adhesions over wide area at apex. No adhesions elsewhere. 3-4 oz.turbid serous fluid & flakes of fibrin in sac. Fibrino-purulent exudate over posterior border lower half lung at base.

Rt. Pleural Sac: Dense adhesions at apex extending down posterior border at base. Adhesions very oedematous but no free fluid adherent to diaphragm.

Tracheo-bronchial glands. Enlarged oedematous deeply pigmented & carbon - show no evidence of secondary growth.

Para.Aortic Glands: Enlarged and malignant.

Oesophagus: Carcinoma

Lymph nodes: Malignant deposits.

Lungs: Widespread broncho-pneumonia in lower lobes, etc.,

Abdo: Aorta: Atheroma.

Spleen: Pale - fibrous.

Large Intestine: Caecum and ascending colon show trace of melanosis. Splenic flexure large diverticulum present. Pelvic colon is seat of marked diverticulosis.

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CASE 30. P.M.250/33 M.Aet. 57 years.

Anatomical Diagnosis: Carcinoma of Oesophagus lower third. Early hypostatic broncho-pneumonia. Diverticulosis of pelvic colon.

Oesophagus: Carcinoma.

Lungs: Hypostatic pneumonia and oedema.

Pleurae: Extensive pleural adhesions on right side, none on left side.

Mediastinal Lymph Nodes: Deeply anthracotic, small. No secondary deposits.

Spleen: Small - fibrous - haemorrhagic.

Intestines: Marked P.M. changes. Several diverticuli - congested.

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CASE 31. P.M.279/33. F. Aet. 38.

Anatomical Diagnosis: Neuro-myelitis optica.  
Terminal lepto-meningitis  
mainly spinal from deep  
bedsore. Cystitis and  
pyelonephritis.

Oesophagus: Healthy. Larynx healthy.  
Complete ossification of  
thyroid cartilage.

Thyroid: Colloid adenomatous nodules.

Lungs: Apart from simple catarrh,  
healthy.

Pleurae: Healthy.

Spleen: Normal.

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DESCRIPTION OF TWO STRETCHER SPLINTS  
FOR THE NON-OPERATIVE TREATMENT OF  
BONE OR JOINT TUBERCULOSIS  
WITH  
NOTES ON CASES TREATED, PHOTOGRAPHS  
AND SKETCHES.

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The principles of local non-operative treatment of tuberculosis of the hip, spine or knee are well known and need no elaboration. They are essentially immobilisation, abduction, extension or hyperextension, with or without traction.

Some form of mechanical splintage is necessary and there are many forms in use at the present time, particularly for children, e.g. the plaster bed, plaster spica, the Pugh frame and carriage, the Gauvain box, the Pyrford frame, the Jones abduction frame etc.,

The best results are obtainable in large orthopaedic hospitals, where the Superintendent is a specialist, has a large number of cases, and is fully conversant with the advantages and disadvantages of the particular splint he uses. Such a hospital has generally a good engineer and carpenter who can alter the splints to suit individual cases.





There are, however, a great many hospitals and sanatoria throughout the country treating pulmonary cases essentially, but receive from time to time, cases of spinal, hip, knee or bone tuberculosis. Such sanatoria are not equipped for these cases and the problem of supplying a suitable apparatus often taxes the ingenuity of the Superintendent if he wishes to avoid an accumulation of appliances and increased costs.

It is only in specialised orthoepedic hospitals that some form of standardisation has been possible with consequent reduction in costs.

Standardisation in medical treatment is not desirable, but standardisation in the application of fundamental principles of treatment, with facilities for simple improvisations for individual cases, seemed to the Writer to be worthy of some thought.

Shortly after graduation, I became assistant M.O. at Grassington Sanatorium, Yorkshire (Bradford City). Although essentially a Sanatorium for pulmonary cases, we had when I arrived, about ten cases of spine, hip, knee or bone tuberculosis, all of which were being treated on ordinary hospital beds with the usual totally inadequate extension apparatus attached to the foot of the bed, in cases needing extension. All these cases had been sent

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to us from the general hospitals where they had received their preliminary treatment.

Not having had previous experience, but realising that the treatment available was very inadequate, I set myself the following problem.

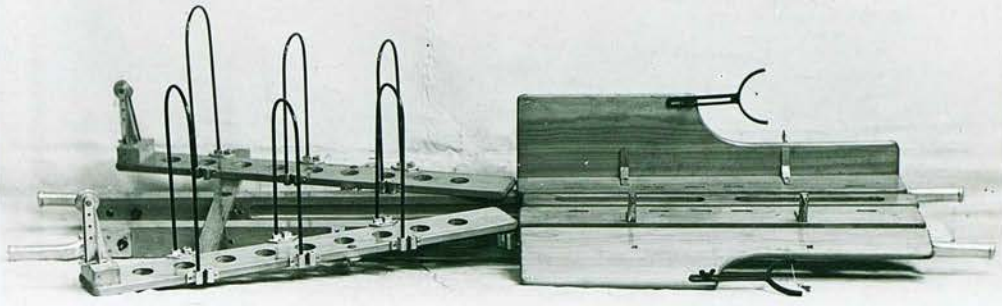
- (1). Could I design a single splint with a very wide range of application suitable for spine, hip and knee cases which would give the fundamental principles of treatment, namely immobilisation, abduction, extension, hyperextension, traction, either fixed or movable, with or without body-weight countertraction, as desired.
- (2). The splint must be capable of improvisations for individual cases - e.g. flexed hip cases, genu valgum etc.,
- (3). Facilities for nursing attention and bed pan service without disturbing the patient's position essential to local treatment.
- (4). Patient must be easily exposed for the application of general treatment, namely:-  
Heliotherapy, hygiene, radiology, dressing of wounds and sinuses, massage etc.,
- (5). The splint must be simple and easily understood by nurses having no special experience, so that the position once set, may be retained.

The splint about to be described is the result of much experimental work carried out at Grassington Sanatorium upon an idea given to me by Dr. J.J.Galbraith, M.O.H. Ross & Cromarty and Superintendent Invergordon Hospital, to whom the Writer is greatly indebted for this and many other practical ideas in treatment.

The Writer is also greatly indebted to Dr. W.M.Cumming, Superintendent, Grassington Sanatorium, for his encouragement, for facilities granted to carry out the experimental work, and his permission to publish here particulars of one or two cases treated.

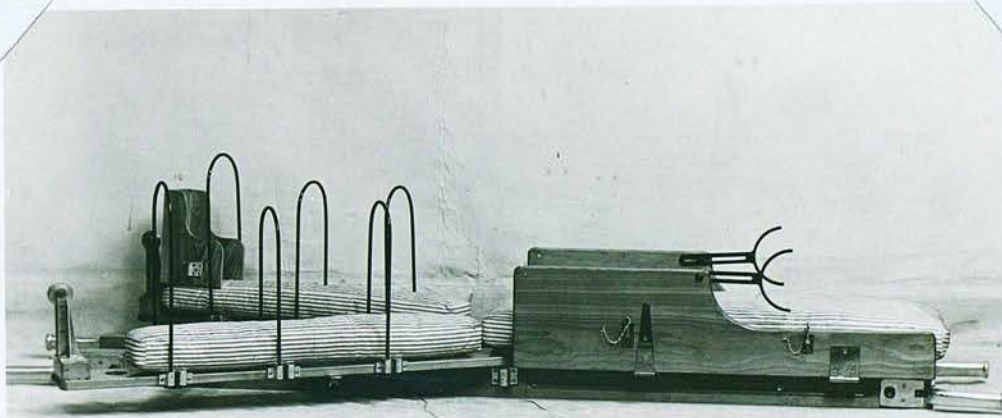
From the photograph (fig 1) it will be seen that the splint is mounted on two longitudinal bearers which are capable of adjustment in width to suit stout or thin patients. The patient lies in the recumbent position on floor boards to which sides are hinged. A loose board is fitted between the two floor boards and a firm body mattress is placed between the boards and patient. The sides are let down by removal of two cotter pins which are chained to the side boards and cannot be lost. Buckles and straps can be fitted at any position on the side boards to ensure that the patient keeps a flat spine. With children, simple reins round the shoulders and attached below to the splint ensure perfect immobilisation.

Fig.2. shows the mattresses.



*Super Spring*  
MATTRESS MAKING MACHINE  
PAT. 1912

Fig. 1.



*Super Spring*  
MATTRESS MAKING MACHINE  
PAT. 1912

Fig. 2.  
showing mattresses.



Being enclosed between the side boards, the body mattress retains its original firmness essential to the body curves. The limb boards are hinged underneath to the side of the bearers and hence are adjustable in width with the bearers and body boards.

The pelvis is immobilised by fitting snugly between the side boards and by double abduction of the limbs.

The buttocks lie on a small square mattress which is retained in position between the side boards and by dropping one side board, this mattress is withdrawn and bed pan substituted. Again, the rapid dropping of the sides allows of nursing, dressing, sponging etc., to be carried out quickly and efficiently, and full or partial exposure for heliotherapy or local heat treatment is obtained with the minimum of disturbance to the patient's position.

#### SPINAL TUBERCULOSIS.

For spinal cases, the splint bearers form a convenient base for attachment of a pulley for head or shoulder traction when necessary.

The rigid floor boards form the flat base for spine and by inserting wood blocks at any desired position under the mattress hyperextension of the spine becomes an easy matter. When the kyphotic lesion is

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fixed, then the same treatment can be adopted for correction of curves before ambulatory jacket is fitted, the patient being placed in recumbent or prone position with hyperextension of spine at desired position.

### HIP-JOINT TUBERCULOSIS.

Immobilisation or fixation, abduction and traction, are the fundamental principles which must be obtained in any splint. The limb boards in this splint hinge to the side of bearers and are supported at the further end by a cross bar, an aluminium quadrant and thumb screw fixing the abduction at any desired angle. Traction is by extension strapping in the usual way, the cords being taken over an aluminium pulley fitted to a bracket on the end of the limb board. The height of the pulley is adjustable in the bracket.

Three pairs of aluminium sockets are fixed to the sides of the limb boards which accommodate "croquet" hoops to support bed clothes. Leg mattresses are fitted along the limb boards, and by inserting a wooden block for which a series of holes are provided, under the mattress opposite the knee-joint, the knee can be flexed as it lies upon the board at the desired angle. The quadrant allows  $33^{\circ}$  abduction either side.

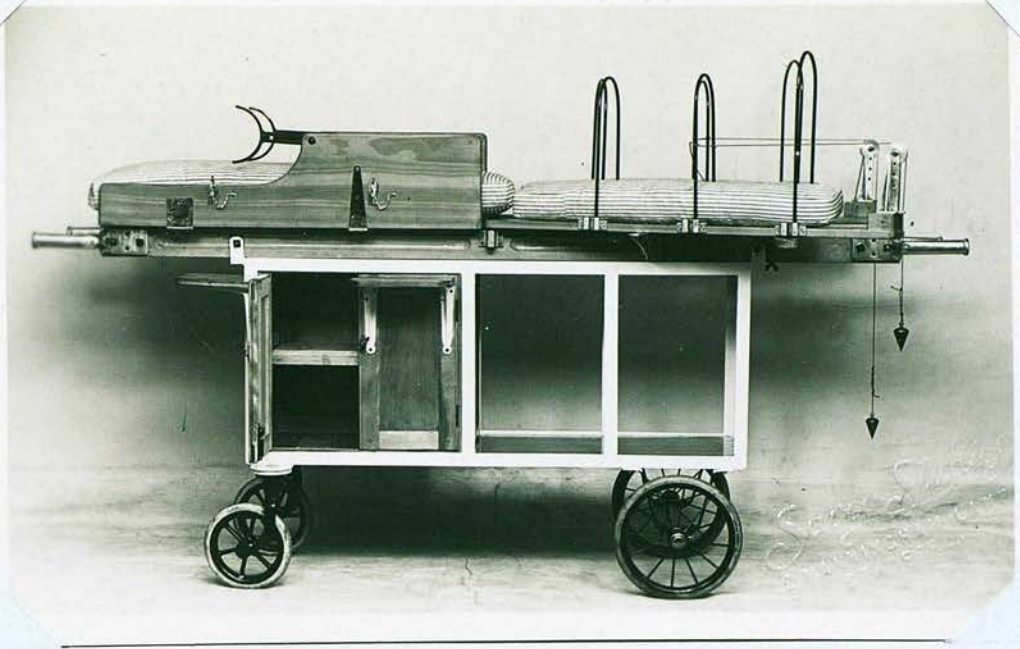


Fig.3.  
showing splint mounted on trolley

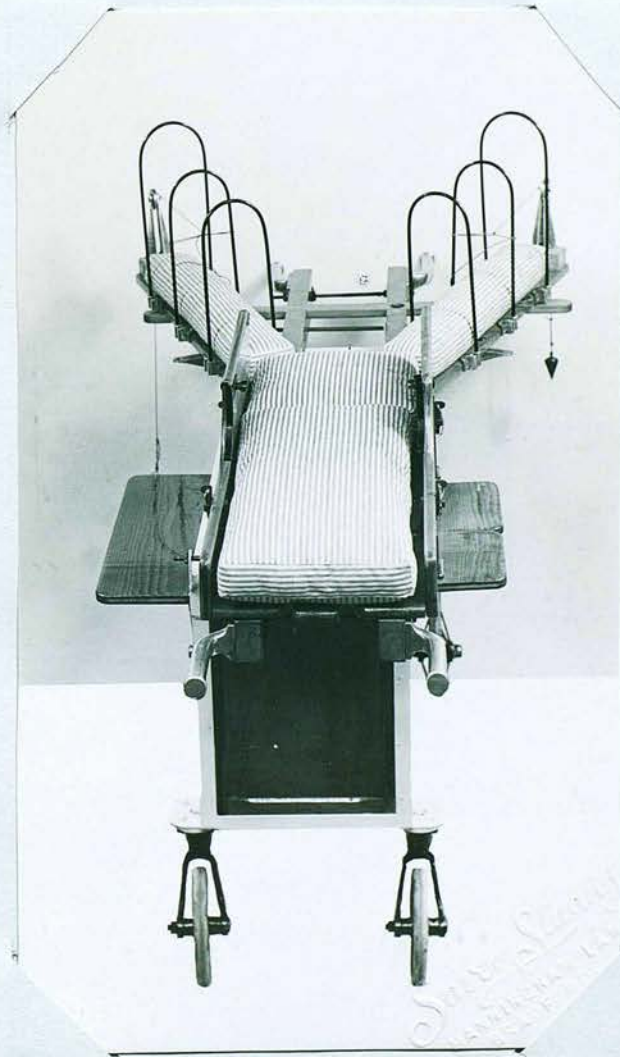


Fig.4.  
showing limbs abducted and arm rests.



The bearers terminate in aluminium handles making the splint portable, so that the patient can be lifted about from place to place. The splint can be used to replace the old type stretcher and the patient transported by ambulance from hospital to sanatorium while still retaining his or her immobile and abducted position.

For sanatorium or hospital use, a special trolley (fig 3) complete with locker and side trays or arm rests has been designed, but the splint can be placed on top of an ordinary ward bed or wood underframe if desired. The trolley can be wheeled about from place to place and replaces the standard bed and large mattress, it occupies less room and the patient is at a convenient height for nursing purposes. The splint has been fully tried out in Grassington Sanatorium for a number of months now and the results have been most gratifying, the patients without exception, feeling very comfortable in the splint after a few days' trial.

#### KNEE-JOINT TUBERCULOSIS.

The problem of treating a knee-joint lesion with a flexed thigh has always presented a great difficulty to the physician or surgeon-in-charge



because it is necessary to get a pull on the thigh as well as the leg at two different angles and at the same time the limb must be abducted. On this splint, by superimposing an adjustable inclined plane with jockey pulleys, to a limb board, a pull on the thigh can be obtained at the proper angle and a second pull when necessary on the leg (see fig 5). Both pulls are thus diverted on to the standard pulley bracket, in which are two pulleys of different diameter, so that two independent pulls can be obtained while yet the bottom board gives the necessary abduction on the quadrant. This special thigh and knee extension apparatus is made up as an extra to the splint and is substituted for the ordinary leg board, the one special board being suitable for either right or left leg by the simple fixation of the hinge pin underneath to one side or the other.

It will be seen that the splint can be adapted for:-

- (1). Simple traction with recumbency.
  - (2). Weight extension with body weight counter-traction.
  - (3). Fixed traction and body-weight counter-traction,
- whichever method one prefers and that the limb boards are convenient for obtaining side traction to control genu valgum etc.,



Fig.5.

Splint fitted with inclined plane  
for flexed thighs.

THE PROBLEM OF DROP FOOT.

Another useful addition to the splint was an improved foot box. The leg mattresses were cut short to below the knee and a three ply wood tray fitted snugly between the "croquet" hook socket castings. The usual three sided foot box was used but the sides were extended to make up for the thickness of the mattress. Two rollers were fitted between the sides and ran smoothly in the wooden tray thus reducing friction and keeping the foot in the correct position. A padded webbing ankle strap supported the foot, the heel itself being free.

White print No.11 illustrated this appliance and Fig.6. shows it in actual use.

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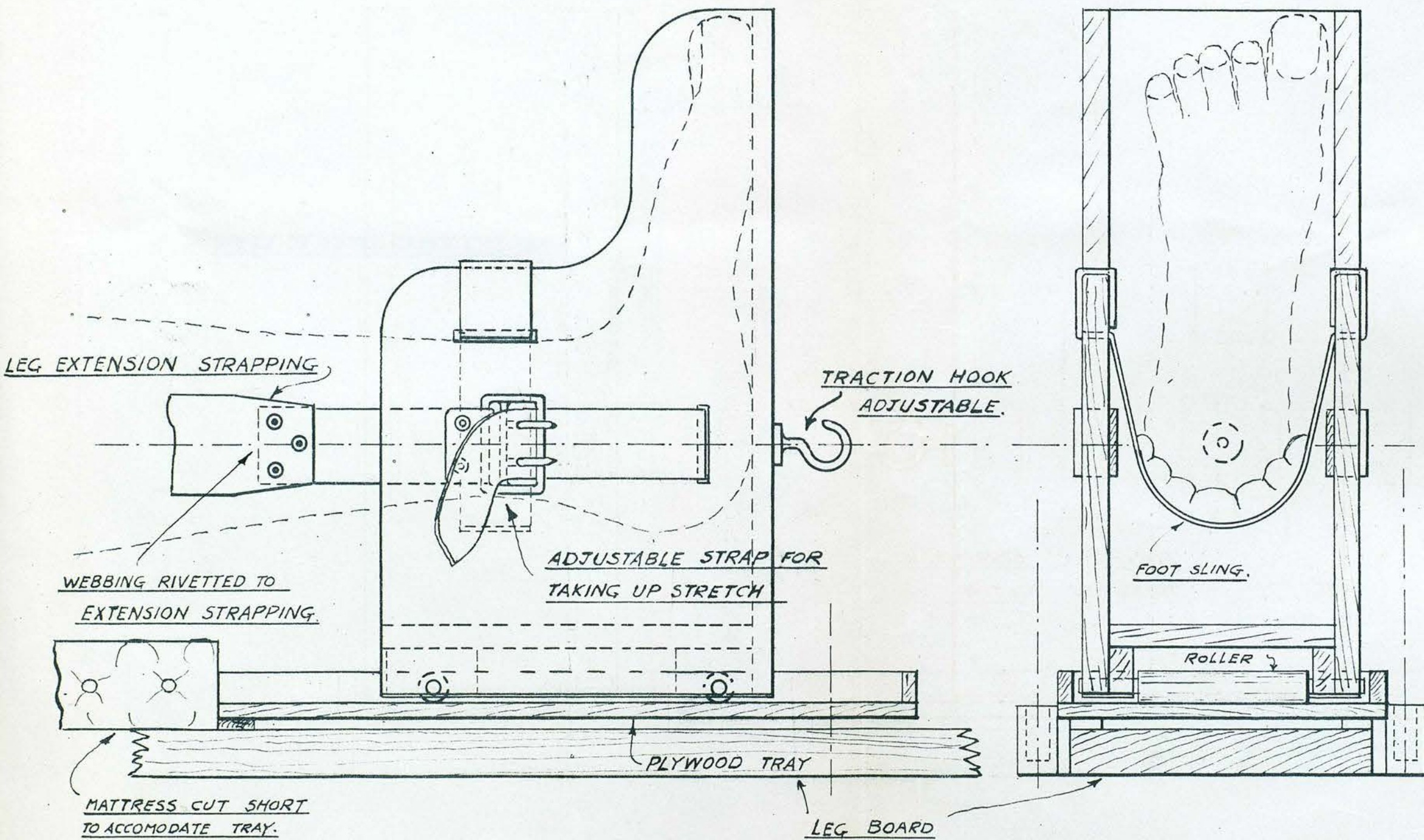




Fig.6.

illustrating foot box in use.





SPECIAL FOOT BOX FOR SPLINT  
scale half size

N° 11

NOTES ON THREE CASES TREATED.



CASE No.1. EDGAR ODDY. AGE 18 YEARS.

Diagnosis - Tuberculosis of left hip.

Brief History.

Well and working until July 1929. Numb pain in left leg - sudden onset. Temperature 105° - July 3rd 1929. Taken to Bradford Infirmary July 5th. Abscess opened - left groin anteriorly - sinus formation - drainage for 8 weeks. Lay between sand bags - no extensions. Thirteen weeks in infirmary - no splintage - no extensions.

Sent to convalescent home for 14 days - wheeled about on chair with leg straight.

Home in bed until Christmas 1929. Told he could get up and walk with stick.

Limped about but got gradually worse.

Sent to St.Luke's Hospital February 11th 1930. Extension applied to leg over end of bed, leg between sand bags. Remained in St. Luke's from February to September 1930 with extensions and dry dressings to sinuses. From September to January 1931 no extension and gauze dressings only.

January 15th artificial sunlight - Psoas abscess developed - pointed backwards towards spine. Aspirated once. Sent to Grassington Sanatorium May 1931. Had been treated on ordinary bed with fracture boards - extensions to both legs over end of bed - small abduction. Abscess continued and when seen by

the Writer in October 1932, had three discharging sinuses and two others breaking down. Sequestrum extruded from one sinus. Superintendent thought patient looked very much like a case developing amyloid disease and the prognosis was bad.

In December 1933 he was put into the new splint, with gradually increasing extensions and as much abduction as was possible. Within two months this patient's general condition improved considerably. The sinuses started to dry up - no further abscesses developed and by the end of June patient became the picture of health and at the end of July, when I left the Sanatorium, only one sinus was unhealed and a dry dressing changed once daily was sufficient.

I visited the Sanatorium in January of this year and this patient was still improving. He was still in the splint and perfectly comfortable.

Fig ( 7 ) shows this patient viewed from above.

Fig.(8) shows a few patients being treated on these splints. It will be noted that there are no trolleys, the splints being merely laid on the top of ordinary beds.

We had ten splints in actual use at Grassington and they were made in the Sanatorium workshop.





Fig.7.  
Case No.1 E.Oddy.  
Tuberculosis of left hip.

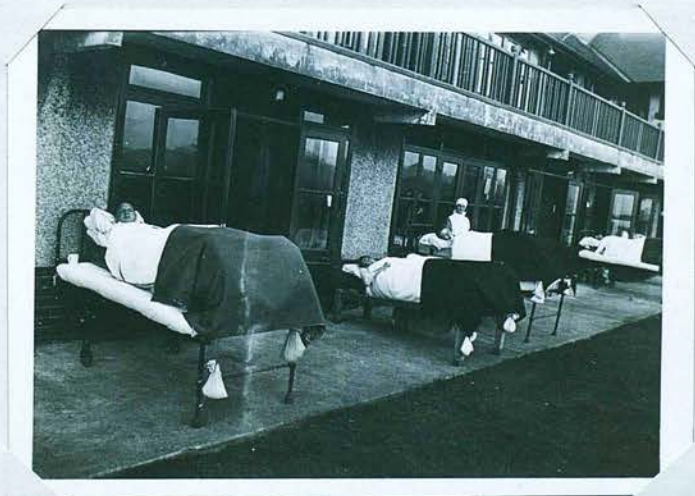


Fig.8.  
A few of the patients lying on their splints,  
the splints being mounted on top of ordinary beds.

CASE No.2. ALEX HANNEY. AGE 18 YEARS.

Diagnosis - Tuberculous Osteomyelitis Rt.Tibia.  
Tuberculous Synovitis Rt.Knee.

Brief History.

Quite well up to June 1931 and working. Developed pain in right leg - right knee swollen - no temperature. Linaments applied and sent into hospital next day.

1st operation for tibial abscess.

2nd operation three weeks later - opened further down.

3rd operation three weeks later - again further down.

Between operations wore a Thomas splint. Knee, however, became flexed and could not wear splint.

In August 1931 informed that knee would be permanently flexed. U.V.Rays given August 1931 to May 1932. Sent home under care of own Doctor. Six weeks at home - got worse. Readmitted to hospital for three weeks - taught to use crutches. Home for eighteen months using crutches. Leg flexed 47° to horizontal.

Admitted to infirmary for 7 weeks for discharging sinuses - sequestrum removed and sent home again. Readmitted to Hospital for three weeks and then transferred to Grassington Sanatorium January 1933 using crutches - thigh flexed 47° to horizontal - discharging sinuses - two in front of tibia - knee swollen and stiff - large area of skin ulceration on front of tibia.

Treatment:

Patient was put into ordinary splint (Fig 2.) and a single pull put on to lower leg. Flexion was reduced by  $10^{\circ}$  after a week or so but could not be maintained. Special knee board (Fig 5 ) fitted and two pulls exerted, one on the thigh and one on the lower leg (Fig 9. ) on April 7th 1932. In a week the flexion was reduced to  $25^{\circ}$  (Fig 10) by gradually increasing the weights to 6 lbs on each pull. To maintain the reduced flexion and prevent spasm from undoing the improved position when the weights were removed for any reason, a knee cap was fitted also shown in Fig 11. In a further two weeks the flexion was reduced to  $10^{\circ}$  (Fig 12). by increasing the pulls to 8 lbs.

In a further week, i.e. four weeks in all, the flexion was down to  $5^{\circ}$  and a Thomas walking caliper was fitted (Fig 13). The boy got up the first day and walked with two sticks. The sinuses dried up - the leg wound healed and pain was absent from the beginning of the treatment.

After two weeks patient walked without sticks and took his own discharge in July 1933. On enquiry in January 1934 the boy was very well and still at home walking about.



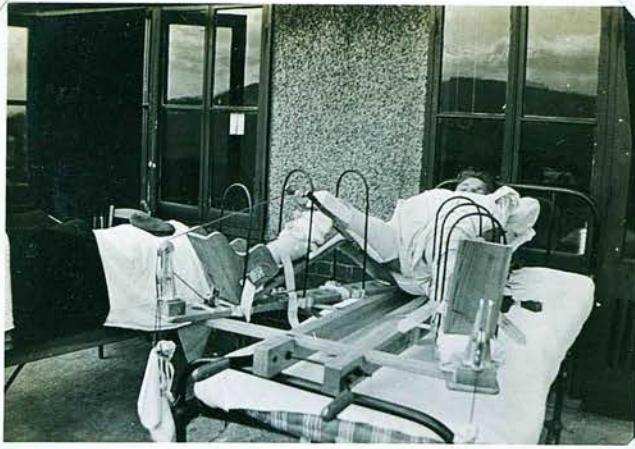


Fig.9.  
Case No.2. A.Hamey. Thigh flexed  $47^{\circ}$ . April 1932

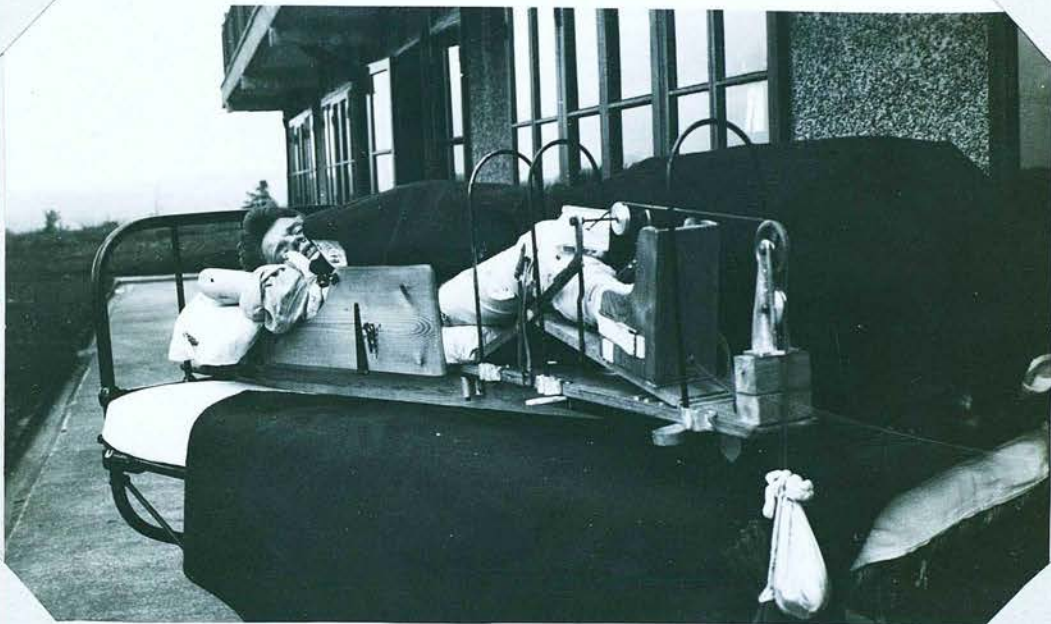


Fig.10.  
Same case. Flexion  $25^{\circ}$ . April 14th 1932

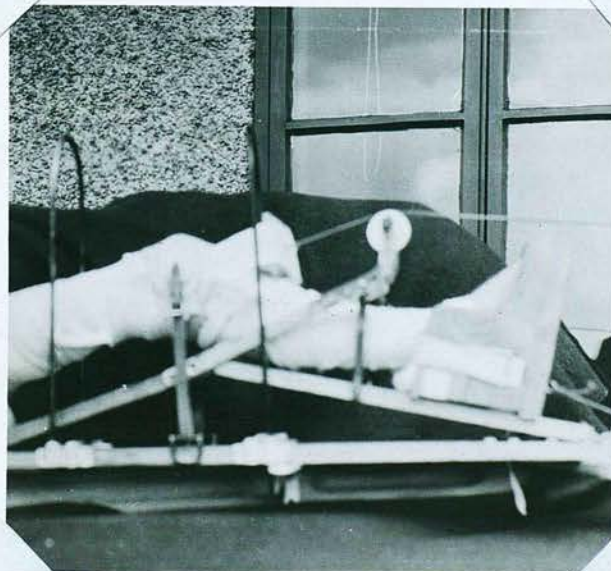


Fig.11.  
Case No.2. Showing knee cap.





Fig.12.  
Case No.2. A.Hanney. Flexion reduced to  $10^{\circ}$ . April 28th  
1932.



Fig.13.  
Case No.2. A.Hanney in Thomas walking caliper  
four weeks after commencement of treatment.

CASE No.3. MISS LUCAS AGE 26 YEARS.

Diagnosis - spinal caries dorsal region.

This was a case of spinal caries with a very acute kyphos.

The last three months of immobilisation were carried out on the splint. The kyphos was fixed and splint was then used to correct the centre of gravity of the body by compensatory curving the spine above the kyphos by means of a block under the body mattress and shoulder traction. (Figs 14 and 15 ). By hyperextending the spine the muscles of the back were strengthened preparatory to making a spinal jacket.

Figs (16) and (17) show the same case fitted with a "Certus" cement jacket. The jacket is strengthened along the spine by a duralumin plate which is rivetted to the jacket, beaten out to house the kyphos, and slotted to take webbing straps for buckling the jacket in place.

This strengthening was done because of the very acute angulation and narrow waist line.

After a few weeks wear, the greater part of the shoulder prolongations of the jacket shown in these photographs were cut away as being unnecessary.

The jacket was completed towards the end of July 1933 just before the writer left Grassington.

I saw this patient in January of this year and she was walking about quite confidently and looking very well indeed.





Fig.14.  
Case No.3. Miss Lucas. Correction of spinal curves.



Fig.15.  
Case No.3. Shoulder traction.

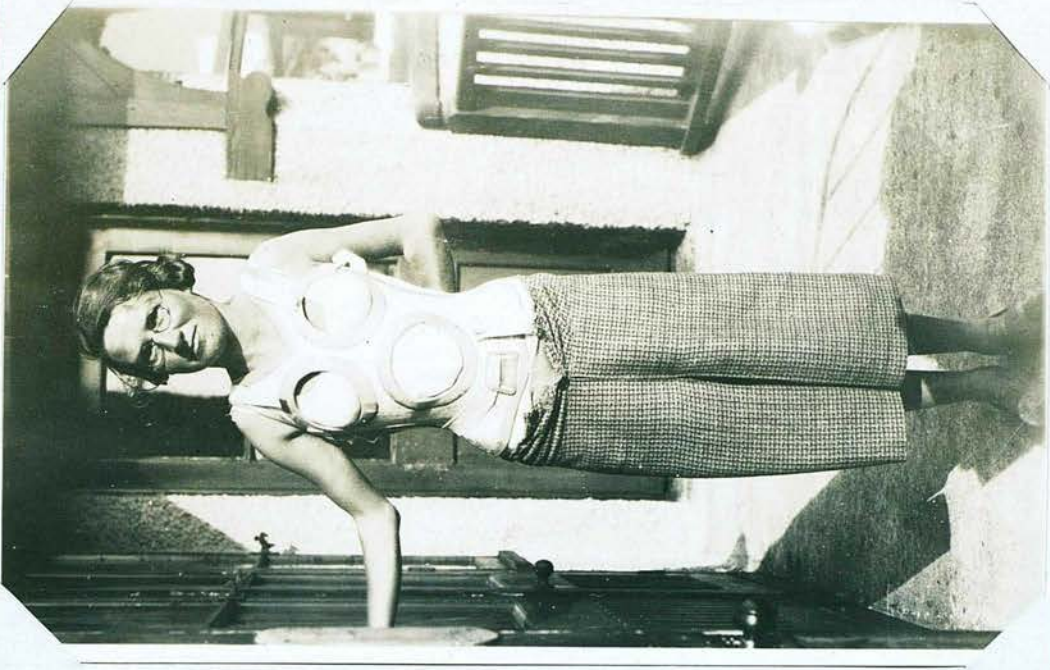


Fig.16.  
Case No.3. Miss Lucas fitted with "Certus" cement jacket.



Fig.17.  
Case No.3. Back of spinal jacket showing  
Duralumin plate.



A PORTABLE SPLINT FOR THE CORRECTION  
OF LATERAL DEVIATION OR SCOLIOSIS OF THE SPINE.

A PORTABLE SPLINT FOR THE CORRECTION  
OF LATERAL DEVIATION OR SCOLIOSIS OF THE SPINE.

White Print No.10 and photograph Fig (18) illustrate an improved type of splint of a portable nature, which the Writer designed while A.M.O. at Princess Mary's Hospital for Children, Margate, for the correction of lateral deviation of the spine or scoliosis.

Construction:

A posterior plaster bed is made in the usual way to include the head and the legs to just below the knee.

In making the plaster bed the child is laid prone on a Bradford frame, the canvas being adjusted to give the correct hyperextension of the spine, the chin resting on a small sandbag. The body is covered with a sheet of felt snipped at suitable spots so that it fits snugly and the plaster bandages are run backwards and forwards in the usual manner.

When the plaster cast is finished, it is carefully marked at the exact position where the correction to the spine is required. The openings for the buttocks, armpits etc., are trimmed away for comfort and nursing purposes.

The bed is then mounted on the underframe as illustrated, at a sufficient height to enable a bed pan to go under the buttocks.

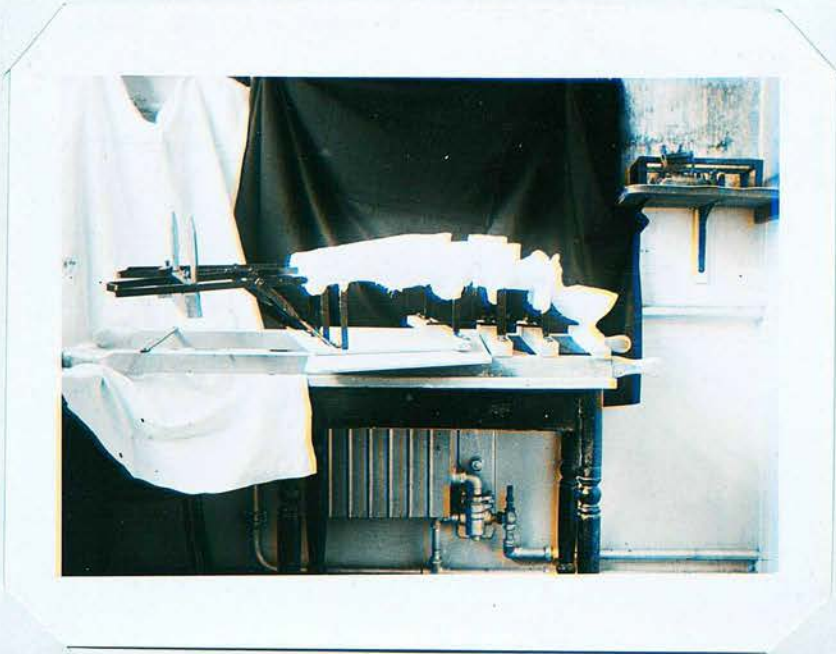


Fig.18

The underframe consists of two wood runners (E). terminating in handles, with cross pieces of wood which carry the metal supports (G) for the bed. A strong three ply board (A) is then firmly fixed to the runners, the board projecting over the edge of one runner as shown, and to that side which the lower part of the child's body has to be drawn to correct the deviation.

Upon this board lies a second board (B), to which the lower part of the bed is attached by iron supports (H) in the manner shown in sketch. The board (B) is free to slide across the board (A). A curved iron quadrant (D) provided with pin holes is secured to the board (B) and is locked by a thumb screw fitted to the cross piece supporting the upper part of the bed. This arm serves the purpose of fixing the deviation at the required angle.

Leg extension irons (C) are incorporated into the sides of the plaster legs, the end supports and leg extension irons being made in one as illustrated. The leg extension irons carry adjustable foot rests (F).

All the supports are fixed to the bed by plaster bandages and plaster cream, and when set, it makes a good rigid job.

The next step is to divide the bed into an upper and lower portion by sawing across at the mark already made. After dividing the bed in the above /



J.H.C.

TELEPHONE N° 41003.



PROFESSOR SYDNEY SMITH, M.D.D.P.H.  
DEAN OF THE FACULTY OF MEDICINE.

*The University New Buildings,*  
*Edinburgh.8.* 23rd April, 1934.

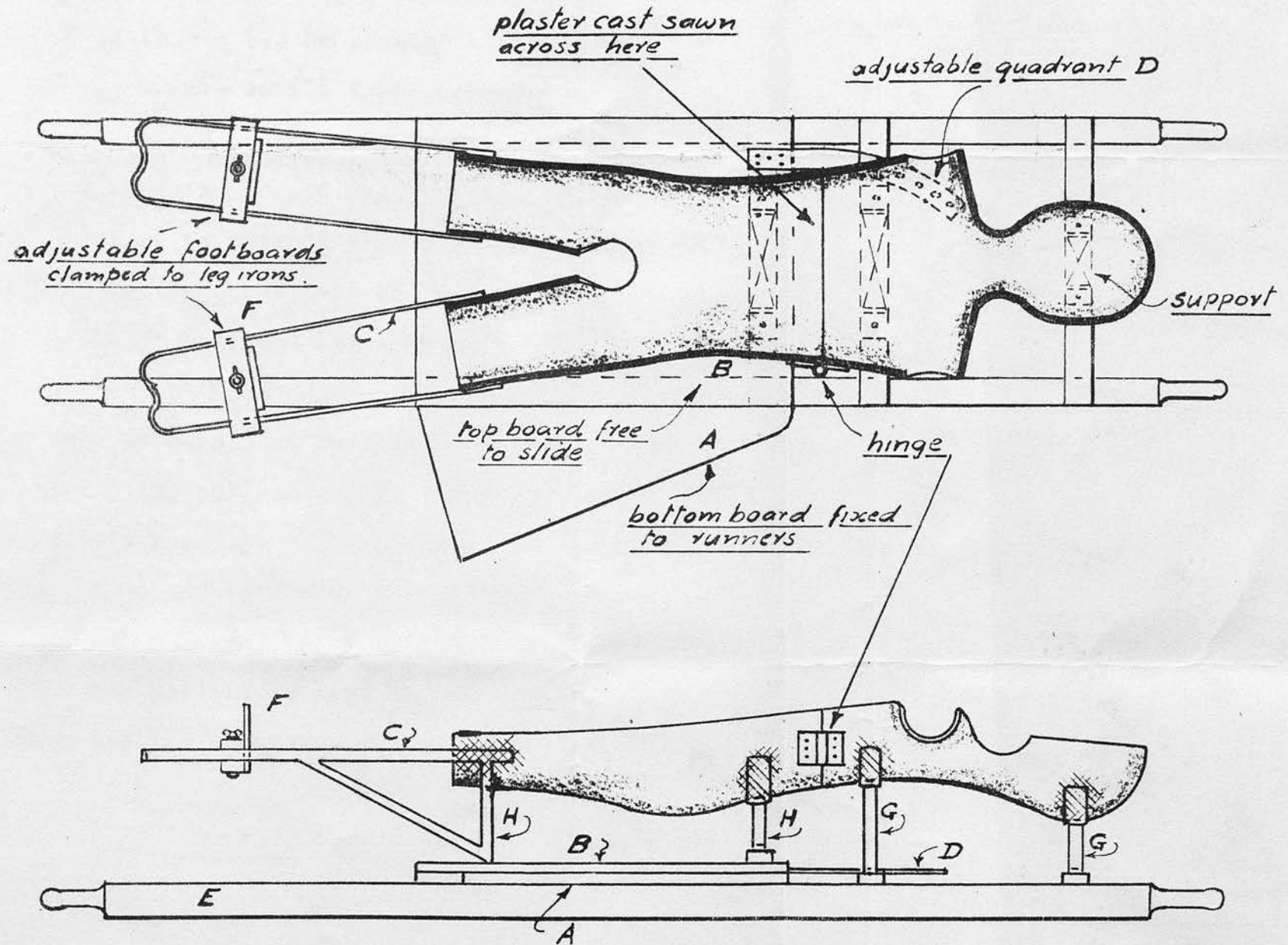
Professor John Fraser,  
20 Moray Place,  
Edinburgh.

A Thesis by Robert Porteous which has been lodged for the Degree of M.D. is sent to you herewith for examination and report.

*Sydney Smith*

Dean of the Faculty of Medicine.

PORTABLE SPLINT FOR THE TREATMENT OF  
SCOLIOSIS OR LATERAL DEVIATION OF SPINE



manner, the upper and lower portions are connected by a strong double flap hinge as shown in sketch, so that the lower portion can thus rotate in the desired direction and be clamped at the desired angle by means of the quadrant and thumb screw already described. The hinge is fixed to the bed by plaster bandages and cream in a similar manner to the iron supports.

When the lower half of the bed is rotated on its hinge a triangular shaped gap is formed between the two halves; this is covered with a stout piece of leather attached to the upper half of the bed and overlapping the lower half and lying beneath the felt bed sheet.

It will be seen that nursing is an easy matter in this splint and the patient can be transported from place to place very readily.

Figs (19,20) and (21) show a small patient, Margaret Smith, age 5 years, being treated in one of these splints. The patient is kept in position by a cuirass, body and leg straps.

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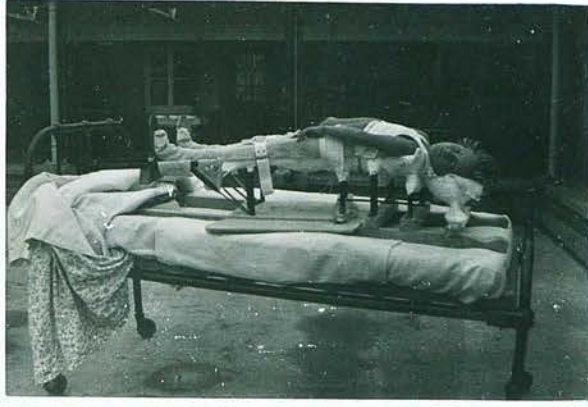


Fig.19.



Fig.20.



Fig.21.