some writers lead us to expect. For instance, Lop⁷ had in 12 cases of pneumotomy seven recoveries: three were relieved and two died. In 31 cases which he collected, 17 were cured. The indications which this author gives for operation in gangrene are:

(1) Two weeks of medical treatment including guaiacol injections, etc., without relief.

(2) Where auscultation shows a definite cavity.

(3) Where the gangrenous focus shows evidence of being covered by pleural adhesions and its seat is such as to be accessible.

Now it is just the difficulty of demonstrating that the pleura is adherent around the point of the intended opening which constitutes the source of doubt and danger.

Krause⁸ advises, in case the pleura is found not to be adherent, a double operation: (1) resection of the necessary ribs and the establishment of adhesions either by stitching the pleura, packing with iodoform gauze, the use of the Paquelin cautery or other means; (2) after waiting five or more days till the pleural cavity is shut off, the gangrenous cavity is opened and drained. The difficulty with this deliberate procedure is that it presupposes that the patient's condition is not one of great urgency. Unfortunately, before five days there is quite a chance that the patient will be forever free of any need of further operation.

I close with what seems to me a needed word of caution as to the use of the aspirating needle in this class of cases. The comparative safety with which exploratory punctures may be made in the various cavities of the body may lead us to forget what may happen if the needle is plunged into a gangrenous or suppurating cavity of the lung, the pleura being sound and not adherent. Is not the withdrawal of the needle, charged with putrid matter, likely to inoculate the pleural cavity as it passes through it, and so produce that much to be dreaded complication, a general fetid empyema?

A STUDY OF THIRTY-SEVEN FATAL CASES OF CIRRHOSIS OF THE LIVER.¹

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CONSIDERABLE confusion exists not only pathologically and etiologically but also clinically as to the classification of cirrhosis of the liver. The cases may, in a general way, be divided into two main classes, according as the size of the liver is diminished or increased. The first is usually spoken of as atrophic; the second as cirrhosis with enlargement or hypertrophic. Both are the result of an abnormal development of connective tissue; in one case contraction being a prominent feature, in the other not. In both also various changes of greater or less extent, probably secondary in origin, occur in the liver cells. The former conception of atrophic cirrhosis with its attendant symptoms as a result of the overgrowth of the connective tissue of the portal system only, and of hypertrophic as the result of that of the biliary system only, cannot now be accepted but in a greatly modified

form, as the process is always a more complex one. The division into interlobular and intralobular forms is also indefinite as both conditions invariably occur together.

The great majority of the contracted livers are probably due to alcohol, although a certain number are the result of the same agents which produce the large livers or of other influences at present unknown. Hanot's classification of cirrhosis with enlargement from the standpoint of etiology is perhaps as satisfactory as any: malarial, syphilitic, tuberculous, biliary from retention, alcoholic, fatty alcoholic, and finally, perhaps belonging in a separate class, the hypertrophic cirrhosis with chronic icterus.

While there is a pretty general agreement among medical writers as to the symptomatology of the ordinary forms of cirrhosis with contraction and cirrhosis with enlargement there seems to be considerable confusion regarding that of the form described by Hanot as hypertrophic cirrhosis with chronic icterus. In fact, many writers doubt even the existence of such a form. Hence, it may be well to give briefly Hanot's own description. It usually occurs in young and feeble men, beginning with an attack of pain in the right hypochondrium, associated with symptoms of disturbed digestion. This is soon followed by jaundice, which is persistent and gradually increases. Crises of pain and indigestion occur at intervals, but at other times there are no symptoms. Enlargement of both liver and spleen begins early, and is progressive, both becoming enormous. There is no ascites or venous enlargement, and the feces remain of normal color. The nutrition is maintained unimpaired for a long time, cachexia rarely appearing under four years. The course is from two to twelve years, more often the longer period. The cause is obscure, although alcohol, syphilis, malaria and lithiasis have seemed of etiological importance in some cases.

It was with the hope that something of interest might be learned concerning the etiology and symptomatology of these somewhat obscure conditions that this study was undertaken, and the medical records of the Boston City Hospital for the seventeen years ending with 1896 investigated. During this period 50,347 cases, exclusive of diphtheria and scarlet fever, were admitted to the medical wards. In 333 of these, or .66 per cent., the diagnosis of cirrhosis of the liver, either as the principal or as a complicating condition, was made. In the nearly 2,000 autopsies made on medical cases, exclusive of diphtheria and scarlet fever, during this same period, the clinical diagnosis of cirrhosis of the liver was confirmed in thirty-seven. On account of the difficulty in the diagnosis of this condition, and of the uncertainty inherent in all diagnoses unconfirmed by post-mortem examination, it seemed best to study only the autopsied cases.

For convenience of comparison they have been divided into three classes, according to the size of the liver as determined at autopsy. The first comprises the livers larger than normal, the second those of a size within normal limits, and the third those smaller than normal. It is possible that those of normal size should be considered with the small ones rather than in a class by themselves. It may be of interest to note that clinically the increased size of the liver was always recognized, and that its irregularity was occasionally noted. The livers of normal size, however, were usually considered large, while the diminution in

¹ Reprinted from Medical and Surgical Reports of the Boston City Hospital, 1897.

⁷ Loc. cit.

⁸ Berliner klin. Woch., April 22, 1895.

size was recognized in about half the cases. The pathological data are, unfortunately, nearly all microscopic and of little use except in establishing the diagnosis of an increase of connective tissue with greater or less destruction of liver tissue and increase of fat. Chronic perihepatitis was noted in one of the large livers, in four of those of normal size, and in three of the small. The following table shows some interesting general points: It appears from this table that cirrhosis with enlargement, without change in size and with diminution in size, are equally frequent, and that the size of the liver is increased in a third of the cases. The male sex is more frequently affected. Cirrhosis with enlargement is more common in younger people, and cirrhosis with atrophy in old. The average duration of symptoms is longer in the atrophic cases. The duration of symptoms, however, varies within wide limits in all varieties. Hemorrhage is a not infrequent cause of death in all forms, and a fatal hemorrhage may be the first symptom even in the hypertrophic form. An alcoholic history was obtained in every case in which the subject was investigated. A history of previous malaria, syphilis or gall-stones was occasionally obtained, but in none did it seem of etiologic importance.

hepatic enlargement as in the others, in which it was sometimes the first symptom noted. In all forms, however, it was, as a rule, rather a late symptom. Jaundice occurred, sooner or later, in more than half of all the cases; more often, however, in the cases with hypertrophy, in which it also, as a rule, occurred earlier and lasted longer. In the hypertrophic form it often occurred in cases in which ascites never developed, rarely without ascites in the others. It sometimes preceded ascites, especially in the cases with enlargement, but more often followed it. The abdominal veins were dilated in somewhat less than half of all the cases; proportionately more in the hypertrophic form. Hemorrhages from the alimentary tract occurred in about one-quarter of the cases, but were less common in the cases with enlargement. Indigestion was more or less troublesome in all forms, being one of the earliest and most persistent symptoms. The nutrition was retained practically unimpaired in all the cases with hepatic enlargement and in about three-quarters of the others.

The inconstancy of the symptoms and the irregularity of their development, both absolutely and relatively, is most striking. In comparison with the others the cases with hepatic enlargement maintained their

	LARGE LIVERS.	NORMAL-SIZED LIVERS.	SMALL LIVERS.
Number of cases	13	11	13
Average age at death	40	52	49
Sex	Males 9 Females 4	Males 6 Females 5	Males 8 Females 5
Average duration from first symptom in cases in which death not due to complication ¹	In eight cases . . 14 mos. Shortest 8 " Longest 30 "	In six cases . . . 9 mos. Shortest 3 " Longest 21 "	In ten cases . . . 25 mos. Shortest 3 " Longest 72 "
Cause of death	Stress of disease . . . 10 Hemorrhage 1 Complications 2	Stress of disease . . . 8 Hemorrhage 2 Edema of brain . . . 1	Stress of disease . . . 9 Hemorrhage 2 Complications 2
Cause of disease	Alcoholic history . . . 13 No data as to alcohol . 0 History of syphilis in 1 and gall-stones in 2. (Gall-stones not of importance as ducts free.	Alcoholic history . . . 6 No data as to alcohol . 5 History of syphilis . . 1 " " malaria . . . 1 (Non-alcoholic cases.)	Alcoholic history . . . 11 No data as to alcohol . 2 History of malaria . . 2 (Both alcoholic cases.)

¹ In one each of the large and normal classes a fatal hemorrhage was the first symptom.

In the following table the individual symptoms and their relative frequency in the different classes are considered. As complete data were often wanting in the records it seemed best to also compare percentages made up only from the cases in which the data were complete.

Among other symptoms pain in the region of the liver was mentioned two or three times in each class, but tenderness was never noted. Swelling of the feet and dyspnea were very common early symptoms in all forms, often preceding any others by months. Late in the disease anasarca and accumulations of fluid in the various serous cavities often developed. Delirium or coma, and in one case convulsions, often preceded for a few weeks the fatal termination. The brain was examined in several of these cases, and nothing abnormal found except in one case where there was edema. The brain in the case of convulsions was normal. There seemed, however, to be rather a definite relation between the nervous symptoms and jaundice.

An analysis of this table shows the following facts: Ascites was about half as frequent in the cases with

nutrition better, had jaundice more frequently and earlier, developed ascites less often and later, and were less subject to hemorrhage. It is to be noted that none of the cases resembled in their symptomatology the hypertrophic cirrhosis with chronic icterus of Hanot.

The size and condition of the spleen seems worthy of special attention. It was of normal size in one and enlarged in twelve of the cases with hepatic enlargement; normal in two, and enlarged in nine of the normal-sized livers; normal in three, and enlarged in ten of the atrophic form. The maximum weights were respectively 1,200, 600 and 1,200 grammes. No relation was evident in any form between the size of the spleen and the duration of the symptoms or the amount or duration of the ascites. Three of the cases with hepatic atrophy with marked ascites showed no splenic enlargement. On the other hand, some of the largest spleens were found in cases in which there was no ascites. Moreover, the spleen was enlarged in most and of increased density in all of the cases in which death occurred early from complications. Unfortu-

nately, no microscopic examinations of the spleens were made. The condition was often described as passive congestion, and sometimes as a hyperplasia of the pulp. Chronic perisplenitis was present in four of the first, three of the second and six of the third class.

The early development of splenic enlargement and the apparent independence of ascites and splenic enlargement of each other seem to suggest some other or additional cause for the enlargement than portal congestion. This discrepancy has already been noted by Oestreich who, after a careful study of the clinical histories, autopsies and microscopical examinations of the livers and spleens of early and late cases of cirrhosis, comes to the conclusion that "the existence of the splenic tumor in cirrhosis of the liver is independent of the liver and due to irritative processes." The irritant is the same as that which causes the diseased process in the liver.

SYMPTOMS.	LARGE—13		NORMAL—11		SMALL—13		TOTAL—37	
	Absolute Number.	Percentage.	Absolute Number.	Percentage.	Absolute Number.	Percentage.	Absolute Number.	Percentage.
Ascites —								
Present in	5	39	8	73	10	77	23	62
Av'ge duration	5 mos	..	6 mos	..	4 mos	..	5 mos	..
Shortest "	1 mo	..	5 wks	..	3 wks	..	..	..
Longest "	1 yr	..	2 yrs	..	1 yr	..	..	..
First symptom	0	..	4	36	1	8	5	14
Jaundice ¹ —								
Present in	9	69	5	45	8 ²	61	22	59
Av'ge duration	5 mos	..	2 mos	..	3 mos ³	..	..	..
First symptom	1	8	0	..	0	..	1	3
Without ascites	4	31	0	..	2	15	6	16
Preceded "	2	15	0	..	1	8	3	8
Followed "	3	23	5	45	5	39	13	35
Large veins —								
Present in	3	60	1	17	4	31	8	40
Abdominal	3	..	1	..	3	..	..	..
Hemorrhoidal	1	..	1	..	1	..	..	..
Hemorrhage ⁴	2	15	3	27	3	23	8	22
Vomitus	2	..	3	..	2	..	7	..
Stools	1	..	3	..	3	..	7	..
Indigestion	6	75	7	100	7	89	20	87
Slight	2	..	3	..	2	..	..	..
Moderate	2	..	1	..	3	..	..	..
Marked	2	..	2	..	1	..	..	..
Av'ge duration	7 mos	..	5 mos	..	6 mos	..	..	..
First symptom	3	37	1	14	1	13	5	25
Nutrition — Good	12	100	7	70	9	75	28	82
Poor	0	..	3	30	3	25	6	18

¹ Clay-colored stools in two of large and one of normal class.

² In four of these cases it was noted that the bile-ducts were patent.

³ In several others at intervals for two to six years.

⁴ Source of hemorrhage was esophageal veins in two; duodenal in one, ileal in one, and hemorrhoidal in one.

The associated chronic complications in the other organs were as follows:

Complications.	Large. 13	Normal. 11	Small. 13	Total. 37
Heart — Hypertrophy left ventricle.	3	3	3	9
Fatty degeneration.	2	3	..	5
Fibrous myocarditis.	1	..	..	1
Chronic fibrous pericarditis.	1	1	1	3
Arteries — Chronic endoarteritis.	3	4	4	11
Lungs — Tuberculosis, active.	..	..	2	2
Healed.	..	2	1	3
Chronic bronchitis.	..	2	1	3
Pleura — Acute fibrinous pleurisy.	..	..	1	1
Chronic adhesive pleurisy.	2	2	4	8
Gastro-intestinal canal — Chronic passive congestion.	2	1	..	3
Chronic gastric catarrh.	1	1	1	3
Dilation of esophageal veins.	1	..	1	2
Dilatation of duodenal veins.	1	..	..	1
Peritoneum — Chronic peritonitis.	..	2	1	3
Pancreas — Chronic interstitial pancreatitis.	..	..	1	1
Kidneys — Passive congestion.	3	1	3	7
Chronic diffuse nephritis.	3	2	1	6
Chronic interstitial nephritis.	2	4	3	9

These figures agree in a general way with those of Lange, based on a considerably larger number of cases. They show, as do his, a marked tendency to interstitial changes in the serous membranes, kidneys and general arterial system. They are presumably due to the same cause as those in the liver and spleen. The pathological changes in the gastro-enteric tract are surprisingly small for a disease in which portal congestion is supposed to play so large a part.

The preceding statistics justify to a certain extent the following conclusions: Cirrhosis of the liver is a comparatively rare disease. It is the result, in the vast majority of cases, of the abuse of alcohol. Alcohol may cause either an increase or a diminution in the size of the liver. Both are due to the abnormal development of connective tissue. Why hypertrophy results in one case and atrophy in another is at present unknown. All forms have the same symptoms. There is no regularity in the relative development of these symptoms. The sequence, however, varies to a certain extent in the different forms. Portal congestion plays a less important part in the development of these symptoms than is generally supposed. Many of the associated lesions are of the same nature as those in the liver, and are due to the same cause.

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THE FORCIBLE CORRECTION OF THE DEFORMITY IN POTT'S DISEASE.

A REVIEW OF THE RECENT LITERATURE.

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THE forcible rectification of the deformity of the spine in Pott's disease is a subject which in France and Germany is awakening the widest interest. It has hardly found its way into American or English literature yet to any extent, partly because it is all so recent and partly because each one hesitates to approach the subject in a critical way on the facts so far presented. The method is essentially one which requires the lapse of a considerable amount of time for the estimation of its real value.

The method proposed is this: the patient is anesthetized, and lying on his face is forcibly pulled upon by one assistant on each arm and leg while the surgeon leaning over him presses down upon the deformity until it yields or he sees fit to desist. The patient is then laid on his back on a padded board or enveloped in plaster from his head to his pelvis, for some months. It is claimed by some writers that permanent improvement and almost cure of the deformity may be obtained in this way.

We are taught, and analogy would lead us to believe, that traumatism to bones or joints affected by tuberculous disease renders the tissues more vulnerable to the inroads of the bacillus and the general system more likely to be affected by generalization of the tuberculosis. The forcible tearing of tuberculous bone tissue has been again and again demonstrated to be risky and attended often by meningitis or general tuberculosis. For example, in 27 cases of the forcible rectification of the deformity in hip disease recently reported by Raymond Sainton,¹ five developed fatal meningitis within two or three months of operation.

Objections on practical and theoretical grounds instantly occur:

(1) Pott's disease is a tuberculosis of bone, of the bodies of the vertebræ, and the deformity is secondary and is an incident. It has been regarded as in a measure conservative and as Nature's preliminary to arresting the disease. The essential is vertebral tuberculosis, not deformity of the spine. Were the treatment proposed the eradication of the disease, it would place it on a different plane; but the measure deals only with a symptom and a result which is of itself distressing, and sometimes disabling but which is on the whole fairly well controlled by early and efficient treatment. Other things being equal it is, of course, most desirable to improve or obliterate the deformity; but the essential of any rational treatment is first to cure the disease.

(2) The question of the occurrence of bony repair to fill in the gap caused by the tearing apart of the bodies of the vertebræ is, of course, an essential one. If there is to be no bony repair the operation is useless. It is asserted (Ménière)² that in Pott's disease bony repair occurs, not in the bodies of the vertebral

column, but at the sides and back. But Regnault³ quotes a specimen in the Musée Dupuytren, where a gap exists in a diseased column, but the column is stable. One must remember, as shown by experiments on the cadaver,² after rectification, that the gap to be filled may measure two to six centimetres or more. No pathological evidence beyond the specimen of Regnault has been adduced to show that bony repair of the vertebral column of a character likely to support weight is likely to occur. Chipault, the modern originator of the method of forcible correction, and the predecessor of Calot, who seems to have reaped the glory, lays much stress upon ligature of the spinous processes in Pott's disease; not only does he ligature them after forcible correction, but as a routine method of treatment of Pott's disease in children even before the occurrence of deformity he advises "ligature by a silver wire of the spinous apophyses corresponding to the diseased region, a ligature followed by immobilization."⁴ A stout silver wire is twisted from one spinous process to another, so as to embrace and steady the whole area affected. After forcible correction Chipault has either wired the spinous processes together or has clamped the laminae together by an appliance somewhat like Malgaigne's patella hooks. A column supported in this way has, he says, double the strength of the intact column. Upon this ligature Chipault insists as one of the essentials, not only of forcible correction but of all treatment of Pott's disease in the active stage.⁵ Relapse, without this is, he says, most likely to occur.

Calot claims that bony tissue fills up the gap caused by the separation of the vertebral surfaces on the ground that radiographs show it, instancing one where there were seen two beams of bone stretched across the gap after an interval of three and a half months, eight to ten millimetres thick and two centimetres long. Again, he says that where the operation is done in two stages, weeks or months apart, distinct snaps of bony tissue are felt at the second redressement.

Ducroquet has brought forward radiographs which are said to show bony repair of the gap in the vertebral bodies.⁶

The reproductions of these radiographs in his article⁷ is far from satisfying one that these statements are warranted by them. The general opinion is that the question of bony repair of the gap in the vertebræ must be regarded for the present as wholly *sub judice*. Such a process is not in line with the general behavior of bone when affected by tuberculosis and disturbed by traumatism. It cannot be dismissed as impossible, nor can it yet be accepted as proved to have occurred after the forcible correction in Pott's disease. If bony repair of the gap does not occur the operation is of questionable ultimate utility unless the spinous processes are wired, as advised by Chipault. Among those who have reported relapses are Pean, Phocas, Tausch and Lorenz⁸ and Vincent.⁹

(3) One would suppose that such operations must be attended by risk to life, but the reported casualties are surprisingly few. The cord and membranes in ex-

¹ Comptes rend. de Soc. de Biol., 1897, 10, s. iv, 793.

² Chipault: Du Mal. de Pott, Paris, 1897, p. 2.

³ Chipault: Gaz. d' Hôp., 1897, 21, 197; Presse Méd. de Paris, 1897, 240; Gaz. d' Hôp., 1897, lxx, 900.

⁴ Ducroquet: Cont. à l'étude de la consolidation du radius, etc. Communication to Twelfth International Congress, Section of Surgery.

⁵ Calot: Arch. Prov. de Chir., 1897, vi, 65; Rev. de Thor. Méd. Chir., 1897, lxiiv, 673.

⁶ Deutscho med. Woch., xxv., 556.

⁷ Lyon Méd., 27, 323.

¹ Revue d'Orthopédie, July, 1897.

² Gaz. Med. de Paris, 1897, 10, s. i, 231.