SERIES 5.—Imperfect Cases in which it was found impossible to remove the whole Disease at the time of Operation.

No.	Age.	Duration of life.	No.	Age.	Duration of life.
1	52	6 months.	5	51	1 year 6 months.
2	36	7 „	6	49	4 months.
3	51	7 „	7	54	2 years.
4	42	1 year 1 month.	—	—	—

SERIES 6. Cases done under one year and about which nothing can as yet be said.—16 cases have been operated upon during the past twelve months successfully.

SERIES 7. Cases which recovered from operation but cannot be traced.—These numbered 15, of whom one was alive after two years 10 months, another after two years six months, and a third after eight months.

SERIES 8.—Cases Operated upon during 1896, 1897, 1898, 1899, and Three Months of 1900.

No.	—	No.	—	No.	—
	1896.		1897.		1899.
1	March.	21	January.	42	December.
2	January.	22	February.	43	April.
3	November.	23	November.	44	March.
4	October.	24	September.	45	October.
5	November.	25	November.	46	March.
6	August.	26	January.	47	January.
7	April.	27	May.	48	October.
8	February.	28	March.	49	January.
9	October.	29	June.	50	November.
10	August.	30	November.	51	August.
11	October.		1898.	52	April.
12	December.	31	June.	53	April.
13	March.	32	November.	54	June.
14	December.	33	November.	55	July.
		34	June.	56	June.
	1897.	35	June.	57	April.
15	April.	36	March.		1900.
16	December.	37	April.	58	March.
17	March.	38	September.	59	March.
18	April.	39	October.	60	January.
19	May.	40	December.		
20	October.	41	March.		

Among these 60 cases there was no fatal one.

The above table of statistics speaks for itself. I am sorry that during the removal from our old Liverpool Infirmary to the present one the histories of many cases were lost. But what I have laid before you have been collected with as much care and accuracy as I could give to the matter. Mistakes there may be, but I am certain that they cannot be sufficiently numerous to invalidate in any way any general conclusions that may be drawn from the table. The cases number 213, of which 175 are available for statistical comparison. In Series 1 it is noteworthy that of the 67 persons who died from local recurrence 17 lived for periods between three and 13 years. Series 2 shows 38 persons alive and well between three and 21 years after operation. To Series 3 it may be objected that I cannot prove that some of the patients did not die from internal recurrence. It is a fair argument and all I can reply is that I have taken all the pains I could to make sure of the causes of death and have put any doubtful ones into Series 4. But, after all, if a cancer of the breast seems a fit subject for removal we do not speculate on internal recurrence, for our business as surgeons is to find out whether we can eradicate the disease locally so that we may be able to give patients who apply early for relief a good prospect of future immunity. Now, the first four series comprise 175 patients and of these 108 have remained free from local recurrence. Of the 108 there have lived 73 over three years as follows: cases that lived between three and six years after operation, 40; cases that lived between seven and 14 years after operation, 28; and cases that lived between 16 and 21 years after operation,

5. Take from these 73 cases half a dozen in case of errors, and still it follows that for every one of my 67 failures I can pit against it a case where the patient remained free from three to 21 years. With regard to tables of statistics in general the plan of taking percentages of small numbers of cases ought to be abolished. The figures in a surgical article nowadays make it look like an extract from an accountant's report on a bankruptcy case. As for the Three Years' Cure System it, too, must be abolished if people are to be instructed fairly. Let no woman be reckoned cured of cancer of the breast till she is dead.

And now, gentlemen, I have only to thank you for the patient courtesy with which you have listened to my poor attempts in a line of surgery to which I have devoted the best efforts of my life during the past quarter of a century.

EXPLANATION OF THE ILLUSTRATIONS.—PARASITES IN MAMMARY CARCINOMA.

FIG. 1.—Parasites in a mammary carcinoma; two in centre well defined. $\times 375$. Reichert $\frac{1}{2}$ oil immersion. Dr. R. J. M. Buchanan.

FIG. 2.—Parasites in an axillary gland secondary to mammary carcinoma. $\times 375$. Reichert $\frac{1}{2}$ oil immersion. Dr. R. J. M. Buchanan.

FIG. 3.—A single well-defined parasite from infiltrated skin over a mammary carcinoma. $\times 375$. Reichert $\frac{1}{2}$ oil immersion. Dr. R. J. M. Buchanan.

FIG. 4.—Group of parasites in a mammary carcinoma. $\times 350$. Mr. Keith W. Monsarrat.

FIG. 5.—Coverslip preparation of parasites cultivated on artificial media from a mammary carcinoma. $\times 75$. Low power. Mr. Keith W. Monsarrat.

FIG. 6.—Lung of guinea-pig showing parasites the result of the inoculation of cultivation, Fig. 5. $\times 350$. Mr. Keith W. Monsarrat.

The specimens were prepared by Dr. R. J. M. Buchanan and Mr. K. W. Monsarrat.

REMARKS ON THE PHYSICAL SIGNS OF PULMONARY DISEASE.¹

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THE great step made by Laennec in scientific medicine was not the invention of an instrument, like the ophthalmoscope or the laryngoscope, for we can do without the stethoscope in auscultation; and Auenbrugger's discovery of percussion, made 50 years before, had been almost forgotten until Corvisart translated it in 1808. Mediate auscultation was the great discovery it was because Laennec was a morbid anatomist as well as a clinical physician and was thus able to compare intelligently his observations at the bedside with the anatomical facts of the dead-house. The same is true of Bright and albuminous nephritis and of the application of electricity to diagnosis by Duchenne and Erb. If diagnosis is to advance it must still go hand-in-hand with post-mortem inspections.

In forming a diagnosis we depend partly on symptoms and partly on physical signs. The distinction is neither logical nor important. We call dropsy a symptom of heart disease, but why not a sign? and why is ascites a physical sign rather than a symptom of cirrhosis of the liver? The real distinction would seem to be rather between symptoms which are subjective conditions of the patient's consciousness, such as pain of all kinds and degree, pruritus, inability to move, nausea, dyspnoea, amaurosis, diplopia, anorexia, and symptoms which are objective, recognised by the physician independently of the patient's consciousness, such as pallor, cyanosis, convulsions, mydriasis, albuminuria, and hæmoptysis. The former set can be simulated, the latter cannot.

Among objective symptoms the so-called physical signs form a separate group. The most important thing to notice of this group is that physical signs tell us only of physical conditions. The Argyll-Robertson pupil, the absence of a knee-jerk, the presence of Jacksonian convulsions, icterus, or pyrexia are all objective symptoms; but they are physiological conditions and point to physiological causes; whereas dulness of the chest on percussion means solid lung, fluctuation of the abdomen means water in the peritoneum, a cardiac murmur infers the presence of a fluid vein, and a rale or rattle the formation of bubbles. Unlike other objective symptoms physical signs tell us nothing of

¹ Part of this paper was read before the Medical Society of Torquay in October, 1899, and part before the South London Branch of the British Medical Association in February, 1900.

the physiology of disease, the perversion of functions; they do not even tell us the nature of the solidification of a pulmonary lobe or that of the liquid which causes bubbles in a bronchus. The former may be due to hepatisation, compression by pleural effusion, or infiltration by cancer; the latter to the mixture of moving air with mucus or blood or pus, or beef-tea. Physical signs point only to physical conditions, not to pathological processes—hence it is undesirable to speak of “pneumonic” crepitation, of “bronchitic” râles, or “cavernous” breathing. This may be thought hypercritical, but I feel sure that I am right in deprecating the loose descriptive way of reporting the physical sounds of the chest by which each writer makes some new comparison or coins some new term. Nothing is more repellent to students and nothing makes reports of cases of pulmonary disease so tedious to read and so difficult to understand.

The essentials of auscultation of the lungs are, I venture to think, neither very numerous nor very difficult, and they admit of being stated in precise, definite, and recognised language. For instance, the difference between dulness and resonance is easy for even an untrained ear to appreciate, though the slight degrees of dulness need attention and experience to recognise. But a “boxy” note, “skodaic” resonance, “tympanitic dulness,” a “flat” or “empty” (*leer* as Skoda called it) percussion sound—these terms are either indefinable or contradictory or tautological. A resonant sound is louder by amplitude of vibrations, longer by duration of vibrations, lower pitched by frequency of vibrations, and it possesses more tone—i.e., a greater proportion of regular overtones. A dull note is much less sonorous, much shorter in duration, higher in pitch, and above all is a mere toneless noise without any musical quality. Between the absolute dulness on percussion of a chest full of fluid or of the muscles of the thigh and the resonance of healthy lung there is doubtless every gradation; as, again, there is between healthy pulmonary resonance and the over-resonant note given out by the larynx on percussion, the “amphoric” note of pneumothorax, and the drum-like note of tympanites. But the whole difference is of degree, and there is no special “quality” of tone or *timbre*, as Piorry imagined, peculiar to the liver, the spleen, the muscles, the heart, or the solidified lung. One only of his delusive distinctions must, I think, be admitted—the kind of resonance which is produced on percussing a subcutaneous osteal surface, a solid but very elastic tissue. When three of the four characters of dulness above given are present, shortness, weakness, and high pitch, combined with one element of resonance—namely, the presence of “tone”—there results the so-called “tympanitic dulness” which may be easily imitated by tightening up a drumhead. The sound is then high-pitched, faint, and short, but it still has the musical quality due to regular overtones, and we instinctively infer that air is present in a tight cavity.

The practice of auscultation of the breathing has suffered even more than has that of percussion from needless refinements, inaccurate language, and vaguely descriptive or fancifully metaphoric terms. The healthy pulmonary murmur (wrongly called “vesicular,” for whatever its mechanism its seat is certainly not in the alveoli) is, I am convinced, liable to considerable variation in healthy persons. It is loud when the chest is thin and faint when the walls are well clothed with fat and muscle. It is loud in children, and this “puerile” character is often mistaken for a morbid sign. Much of what is called “harsh” breathing is nothing but loud breathing, for a mere noise like the pulmonary murmur of health is apt to become harsh—i.e., unpleasant to the ear—when its loudness is much increased; and I would fain exclude the former epithet altogether. The distinction between a pulmonary breath-sound, however loud or harsh or “coarse” (another undesirable adjective) it may be, and bronchial, tubular, or tracheal breathing is one apparently difficult to catch. At least, most learners call puerile, exaggerated, or supplementary breathing “bronchial” and true bronchial breathing “amphoric.”

I do not deny that there are gradations between vesicular and tubular which may if we please be recognised as “broncho-vesicular,” “obscured,” “veiled,” or “transitional,” but I am sure that they are rarely met with and that the less often such ambiguous terms are employed the better. Bronchial or tubular breath-sounds are almost always marked by prolonged expiration, but their distinctive character is what is called “blowing” or “puffing” or “whiffing”—i.e., the inspiratory and expiratory murmurs are more continuous,

more equal in character as well as in duration, and have more tone than have those of vesicular breathing. Though often loud, tubular breath-sounds are often very weak. In fact, the loudness of the sounds heard over the larynx or trachea makes them somewhat misleading as an example of tubular breathing. In teaching this part of the subject I have always regarded the healthy “vesicular murmur” as the laryngeal murmur due to a fluid vein in the glottis modified by spongy lung, and not tubular respiration as the sound in the bronchial tubes transmitted by solid lung; and the same theory applies to bronchophony and vocal resonance, and also to tactile fremitus.

The adventitious pulmonary sounds were all described by Laennec as *râles* (in English, rattles) and the term is still used in this comprehensive sense by, I believe, all French, most German, and some American writers. But in England we have for half a century been accustomed to confine the word “râles” to “moist sounds” like the *râle muqueux*, and to use the term “rhonchus” for all “dry sounds,” including the *râle sonore* or *rhonchus* of Laennec and his *râle sibilant*. “Moist” and “dry” are not happy descriptions of the *râle* and the rhonchus, for the mucous membrane of the trachea and bronchi are never dry, and it seems absurd to associate “dry” sounds with free expectoration. “Râle” and “rhonchus” as we use them are arbitrarily assigned, and the original meaning of the latter—“a snore”—would exclude sibilant rhonchus. Still the difference between the two kinds of adventitious breath-sounds is important and admits of exact definition, both as an auditory perception and as a physical phenomenon. Râles or moist sounds are interrupted, rhonchi or dry sounds are continuous; râles are caused by successive explosions of bubbles, rhonchi by a fluid vein. The loudness and the low pitch of a râle or a rhonchus are indicative of the larger size of tube, the trachea, or larger bronchi; faintness and shriller pitch lead one to refer them to the smaller bronchi. Moreover, in the case of râles a very few explosions give the impression of large bubbles in a large tube like the trachea, while a great number of them suggest to the listening ear small bubbles in a small bronchus. A much more important character and one which has been misapprehended by many writers is that between consonating and non-consonating râles. The former have a musical quality or at least some degree of tone, the latter are mere noises. The distinction is that felt by Laennec when he distinguished *râles crépitants* from *râles à grosses bulles*. The late Dr. George Barlow called the former “crackles” and the latter “rattles.” Most writers speak of the former as “bright,” “clear,” “musical,” or “metallic” râles, and the latter as “bubbling” râles. But all râles are bubbling, and the word “crepitant” does not in itself denote anything more definite than Laennec’s comparison with the sound made by burning salt. No one who has auscultated with care can overlook the difference referred to, but it does not seem to be generally recognised that the difference between a “clear” or crackling râle and a bubbling râle is really that between more or less musical sounds with regular overtones, and mere noises without “tone.” Moreover, the distinction is of great practical importance, which has not, I think, been clearly recognised by most writers on auscultation. The first kind, “non-consonating” or toneless râles, always imply a spongy condition of the lung, whereas râles with tone (consonating or crepitant) always point to solidification of the lung; the only exception being when they are rendered musical by the bubbles bursting in a large cavity filled with air, as in cases of a very large vomica or of pneumothorax. The peculiar and characteristic distinctions of true pneumonic crepitation are the large number—i.e., small size—of the detonations which compose it, and their eminently consonating and therefore wonderfully clear and distinct character.

Is a râle a sign of pulmonary oedema? I think not. Oedema of the lung is effusion of serum into the interstitial tissue between the air-passages. Edematous lung does not sink in water and therefore it still contains air, and it does not cause dulness during life. The râles sometimes heard in cases of oedema from Bright’s disease are, I believe, due to inflammatory effusion of mucus into the bronchioles. They are small but toneless. When I have heard consonating râles during life I have found lobular pneumonia after death.

What is the nature of so-called capillary bronchitis in the aged? The term “capillary” dates from a period when the anatomy of the lung was not understood, for there are no

capillary bronchioles or air-passages; but is there a form of bronchitis which spreads as far as the pulmonary lobules and no farther? I doubt it, and if I am right in the doubt does it not follow that capillary bronchitis of the aged is anatomically identical with lobular or catarrhal pneumonia (pulmonary catarrh) of children? The opportunities of seeing the lungs in the former case are much fewer than in the latter, but where I have had an opportunity I have seen some of the lobules hepatised in cases of suffocative bronchitis in old people. It is exceedingly rare for death to occur from bronchitis in early and middle adult life—twice only have I met with it. May we not say that (with similar rare exceptions) fatal acute bronchitis in children is lobular pneumonia with more or less collapse; that in aged patients also it is lobular pneumonia; and that in the periods between it is a secondary complication of acute miliary tuberculosis, of lobar pneumonia, or of phthisis? In cases of life assurance I have scarcely ever known a death reported as due to bronchitis between puberty and 60 years of age which did not on inquiry turn out to be phthisis or pneumonia, often disguised under the meaningless term of "congestion of the lungs."

With regard to the physical signs of phthisis, are we not too apt to look for the striking and interesting sounds which belong to its later stages, such as absolute subclavicular dulness, amphoric breathing, consonating râles, and the percussion note of the cracked pot? In early cases of the disease, when we are most concerned to detect and to treat it, do we often find decided dulness? I think not, and if we check our auscultation by our knowledge of anatomy we cannot expect it; for at this early period the tubercles and pneumonic patches affect only separate lobules or alveoli or inter-alveolar passages, and such an affected area (only observed after death when there are more advanced changes in other parts) does not sink in water. Moreover, even in chronic cases we often find so much vesicular emphysema at the necropsy that it is no wonder that small patches of consolidation fail to deaden the percussion note. For resonance spreads, but dulness does not. From observation on the lobular pneumonia (pulmonary catarrh) of children I am convinced that isolated lobules in a hepatised state do not appreciably affect the percussion note of the chest; it is only when three or four contiguous lobules are solidified that we recognise the dulness which they produce. Even in the later stages of phthisis I am convinced from post-mortem experience that the subclavicular dulness is often due to pleuritic thickening and adhesions—no doubt tuberculous in origin—and not to extensive consolidation of the apex. This would explain why we not infrequently find a flattening under the clavicle with deficient resonance, and yet the breath-sounds are not bronchial in character nor is the vocal resonance altered. Another cause of complete dulness at one apex is the presence of a large cavity. This never, I believe, gives a resonant note. Such large cavities are always surrounded by a dense thick capsule of fibrous tissue and are adherent to the chest wall. They can only form slowly and therefore they belong to the most chronic cases—i.e., to the most resistant and the most hopeful; whereas when there are very slight dulness and no other striking signs, only non-consonating râles and prolonged expiration, if there are fever, wasting, and diarrhoea, we have to deal with a rapid and almost hopeless form of the disease.

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THE SURGICAL TREATMENT OF DILATATION OF THE STOMACH.¹

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THE following are the principal causes of dilatation of the stomach: (1) cicatricial contraction of the pylorus, due usually to old gastric ulcer; (2) malignant disease of the pylorus; (3) adhesions outside the pylorus; (4) gastroparesis; (5) spasm of the pylorus from ulcer or from excess of free hydrochloric acid; (6) atony (?); (7) various neuroses; and

(8) floating kidney on the right side. It is only to the first three of these causes that I can refer in this paper.

With regard to diagnosis, the following are the principal methods of examining the stomach: (1) inspection; (2) palpation; (3) percussion; (4) auscultation; (5) intubation; (6) artificial distension of the stomach with gas; and (7) the chemical examination of the stomach contents. It would take too long to refer to these methods in detail, but I would point out that on inspection of a case of pyloric stenosis one often sees, not only a rounded tumour caused by the dilated stomach, but waves of peristalsis passing across the abdomen from left to right. The methods of palpation and percussion are so well known that nothing need be said on them. I might, however, mention that by auscultation we may hear splashing sounds within the stomach and bubbling of gas in the region of the pylorus. Intubation is a most valuable method, and its use should never be omitted in the examination of a case of dilated stomach, except where active ulceration is strongly suspected. A soft rubber tube is swallowed by the patient with practically no discomfort, the stomach contents can then be evacuated by expression of the stomach or by the attachment of a syphon tube. It is usual, half an hour before using the tube, to give Ewald's test breakfast, which consists of a plain roll and half a pint of warm water or weak tea; the fluid obtained after extraction is available for the application of chemical tests. To cause artificial distension of the stomach the two constituent parts of a seidlitz powder may be given separately in a little water. The carbonic acid gas thus generated causes dilatation of the stomach and enables one to define its limits with precision. This method, however, causes considerable discomfort to the patient, since carbonic acid gas produces spasm of the pylorus; less discomfort is experienced if the stomach be distended by air pumped into the stomach by means of the stomach tube. The chemical examination of the stomach contents is undertaken to determine whether there is an excess or absence of free hydrochloric acid and of lactic acid or acid lactates. The simplest test for hydrochloric acid is a solution of phloroglucin and vanillin in absolute alcohol. A few drops of this are placed in a china plate and a drop of the stomach filtrate is placed close to it, so that the edges of the two drops coalesce. The plate is then warmed, and if any free hydrochloric acid is present a red tinge appears; the amount can be estimated by making a control test with a 1 per cent. solution of the acid. The easiest test for lactic acid or acid lactates is to place a few drops of neutral ferric chloride solution in three drachms of 1 in 20 carbolic acid, and then add water until an amethyst-blue colour results; a few drops of the stomach filtrate added to this will produce a yellow colouration if lactic acid or acid lactates be present.

These tests are of importance in distinguishing between the various forms of dilated stomach; thus in malignant disease we usually get an excess of lactic acid and an entire absence of hydrochloric, and in gastric ulcer or cicatricial contraction due to this we find an excess of free hydrochloric acid, and in dilatation due to gastroparesis or to neuroses we find the amount of free hydrochloric acid fairly normal. The foregoing, however, is not absolutely correct, and I have obtained false information in two of my series of cases. In No. 13, which was advanced malignant disease, an excess of free hydrochloric acid was demonstrated, and in No. 12 there was an entire absence of free hydrochloric acid, although the dilatation was due to cicatricial contraction. The presence or absence of a tumour is not of much value in the diagnosis of a case, although in some cases one can be felt in the region of the pylorus; and when the stomach has been much dilated this tumour may be freely moveable and may be found even as low as the iliac fossa. As a general rule, however, in malignant disease, if a tumour can be felt, the case will prove to be too far advanced for radical treatment, and its absence is certainly no evidence against the existence of this disease; on the other hand, a distinct nodule can sometimes be felt in cicatricial stenosis, and a small moveable tumour was felt before operation in No. 10 and No. 11. Another well-recognised symptom of malignant disease—viz., coffee-ground vomit—is often not present till late in the case, and so its absence is of no clinical importance.

Briefly, then, the symptoms of malignant disease of the pylorus which would indicate surgical interference are: (1) pain, usually localised to the pyloric region and felt in

¹ A paper read before the West London Medico-Chirurgical Society on Jan. 5th, 1900.