On a form of bronchitis (simulating phthisis) which is peculiar to certain branches of the potting trade : a graduation thesis to which was awarded the gold medal of the University of Edinburgh, August 2, 1864 / by Charles Parsons.

ON A FORM OF BRONCHITIS

(SIMULATING PHTHISIS)

WHICH IS PECULIAR TO CERTAIN BRANCHES OF THE POTTING TRADE.

A GRADUATION THESIS

TO WHICH WAS AWARDED THE GOLD MEDAL OF THE UNIVERSITY OF EDINBURGH, AUGUST 2, 1864.

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ON A FORM OF BRONCHITIS PECULIAR TO THE POTTING TRADE.

It has been said that we acquire real wisdom from our failures only, and not from our successes in life. Perhaps in nothing is this axiom more strikingly exemplified than in the practice of medicine, inasmuch as it is governed by none of those fundamental laws which characterize the so-called "Exact Sciences:" so that, on the one hand, we cannot predict with absolute certainty, that any one disease will assuredly be attended, in every instance, by an unvarying train of physical signs and symptoms; nor, on the other hand, can we assert that these signs and symptoms are invariably and indubitably the manifestations and concomitants of one and the same disease at all times. Were it otherwise, our diagnosis would be almost infallible, and liability to error would be nearly excluded. But this very liability, which attaches to medicine, confers lasting advantages which are of peculiar worth. The lessons which an error in diagnosis teaches are sometimes painful, always humiliating, but never forgotten; they remain, as it were, indelibly stamped in the memory. The painstaking and honest investigation of one case of mistaken diagnosis, with its possibly disastrous consequences, imparts more instruction than the consideration of many conducted to a successful issue. The one gradually

but speedily fades from the recollection: the other exists, mentally photographed, so to speak, with all attendant circumstances in minutest detail. Some of the most valuable researches in Pathology and Medicine have originated in patient endeavours to comprehend the causes which have led to mistakes in diagnosis.

The signs and symptoms proper to one disease have sometimes manifested themselves in another differing entirely from it in nature and essence, so that two distinct diseases have been confounded merely from some similarity in their phases. It is only when their morbid anatomy has been carefully investigated and patiently studied, that we are able rightly to interpret their pathology, and to reconcile apparently opposite diseases with nearly identical signs and symptoms. Thus some trades and occupations are peculiarly obnoxious to a form of disease closely resembling phthisis in nearly every feature, with the exception that tubercle is generally absent. I refer particularly to the so-called "black phthisis," "cotton phthisis," "stone phthisis," and "knife-grinders' phthisis." It is to be regretted that these names have been thus applied, for phthisis conveys the idea of tubercle, and in these diseases the existence of tubercle is purely accidental and not necessary to their constitution. The lung-substance, it is true, undergoes destructive inflammation and excavation, just as in phthisis; but Pathology declares this to be simply the sequel to protracted bronchitic irritation, induced by the long-continued inhalation of irritating particles of various kinds.

It was not until the year 1860 that I became aware of the existence of a similar form of disease amongst potters; and, so far as I can ascertain, no account of the affection has ever been placed on record. I venture,

therefore, in this way to bring the subject under the notice of the profession, and to communicate such information as I have been enabled to collect, accompanied by such deductions and remarks as may, I hope, tend to elucidate the nature and cause of this distressing malady.

My attention was first drawn to this subject by an error in diagnosis so long ago as the year 1859. In the summer of that year I was appointed House Surgeon to the North Staffordshire Infirmary, in the midst of a dense population composed for the most part of potters, colliers, and iron-workers. The following winter soon indicated what diseases were peculiar to each of these classes, and clearly demonstrated that chest-affections were prevalent in the district, and especially amongst the potters. One of those admitted early with pulmonary complaint, under the care of Dr. Wood, was a potter of middle age, whose sunken emaciated appearance, and distressing cough and hot dry skin, at first sight gave one the impression that he was the victim of phthisis. Further examination after he was in bed only confirmed the unfavourable opinion that had been formed of his case. There was dulness on percussion at both apices with flattening of the chest surface, whilst immediately below, the lungs were unusually resonant, and the stethoscope conveyed a gurgling sound to the ear. There was prolonged expiration at the anterior margins of the lungs and anterior surface of the lower lobes. Some places in the middle of the right lung were dull on percussion, and here loose crepitation was audible. The patient had had several "attacks on the chest" before, but lately his cough had never left him, and his breathing had become greatly oppressed. He had gradually lost flesh, and his appetite had deserted him. He

expectorated profusely a purulent matter, occasionally darker in colour than usual, but not sufficient to attract particular notice. There was no diarrhœa. I was unable to detect any pulmonary fibres in the expectoration under the microscope, but had no hesitation in pronouncing the case to be one of tubercular phthisis that would end fatally. I was however greatly surprised, at the post-mortem examination of the body, to find not even a trace of tubercle in the lungs! Cavities indeed there were, and a puckering and thickening of pulmonary tissues such as I had never witnessed in cases of phthisis, whilst the minute bronchial tubes possessed a more than cartilaginous hardness to the touch, and the lung was infiltrated or discoloured with a black matter, somewhat like those to be described hereafter, only in a much less degree. Several other cases occurred in Dr. Wood's wards similar in character, but varying according to their stages of progress, so that it was impossible not to recognise the existence of this peculiar malady. It was not however until the following winter that my mind was specially attracted to this subject by the occurrence of one or two cases unusually well marked, whose histories I subjoin, as they are nearly typical of the disease I am describing.

It becomes me to state here, that I am indebted to the kindness of my friend, Dr. Wilson Fox, for permission to copy these cases from his Hospital Case-Book for my thesis.

Case 1.—William Bowden, æt. 35, single, potter, admitted into North Stafford Infirmary, March 22, 1861.—Is a native of Exeter; reddish hair, light brown eyes, clear complexion; finger-nails rather clubbed and incurvated. Has had "asthma" as long as he can recollect. His father had "asthma," and died at 45. He

has never, so far as he is aware, had any severe illness in the chest; nor any other illness that he is aware of, except measles. Has never had hooping-cough; has come into hospital on account of extreme difficulty of breathing, which has increased greatly during the past three weeks; has not much cough, expectoration not much to speak of. Emaciation excessive.

Examination of Chest.—Respiration in great measure diaphragmatic. Expansion of thoracic parietes on inspiration very slight. Sternum from fourth cartilage is depressed. Intercostal spaces depressed. Clavicles very prominent, left more so than right. Ensiform cartilage greatly depressed. Respiration short and difficult, 28 per minute. A deep respiration is accompanied by a wheeze in expiration audible to bystanders. *Percussion* in front of chest unduly resonant everywhere. Precordial region resonant everywhere. No superficial cardiac dulness to be made out. Heart's apex beats in normal situation. Inspiration over front of chest weak and imperfect. Expiration prolonged to twice normal length, attended everywhere with sibilant and some sonorous râles. No moist râle in front. Vocal resonance in front weak. Posteriorly, right apex is dull as low as middle third of scapula; absolutely dull as low as infra-spinous fossa. At upper part of dulness, inspiration is blowing almost tubular; expiration blowing and prolonged. In middle third, inspiration is weak but tubular; expiration tubular and prolonged. Some fine moist râle heard with forced breathing at this spot. Vocal resonance so weak as scarcely to be audible anywhere. Voice rather hoarse. Left lung posteriorly dull in spots in middle third, mingled with places where percussion resonance is unusually resonant. Expiration is greatly prolonged over whole of this lung. Some fine

moist râles heard in spots here. No cavernous breathing in either lung. Appetite bad. Tongue covered with whitish fur. Bowels regular. Sweats much. Pulse very weak, 68.

Ordered Tr. Iod. fort. to right side. Nitrated paper to be breathed.

℞ Vin. Ipecac. ♏ x., Sp. Æth. Chlor., Tr. Camph. cō. *aa.* ♏ v. Mist. Acaciæ ℥j. t. d. s.

Full diet. Wine, 4 oz.

April 9th.—Very little relief to chest. Nitrated paper relieves him a little, but only temporarily. ℞ Inf. Ros. cō. ℥j. bis die.

April 28th.—Has been gradually losing strength. Great dyspnœa came on this morning. Face is now livid. Expectoration has ceased. Breath has gangrenous odour. Some dulness is now to be found under right clavicle, and there is a large loose moist râle there. Otherwise state of chest unchanged.—℞ Sp. Æth. Sulph. Sp. Ammon. Arom. *aa.* ♏ xv. Ac. Hydrocyan. dil. ♏ iij. Dec. Senegæ ℥ſs. Mist. Camph. ℥j. 4tis. horis sum. Omitte alia.

April 30th.—Patient died yesterday.

Post-mortem.—Heart entirely covered by lungs, otherwise healthy. Right lung contains at apex a large cavity filled with thick creamy adhesive pus. Cavity is situated at posterior part of organ. This lung is highly emphysematous, but is interspersed throughout with masses of black substance which cut firmly, mingled with whitish opaque elevated spots which are very firm. The blackish masses are of irregular extent, sometimes of the size of a walnut, and sometimes of a Maltese orange. Section of the whitish masses is gritty, that of black is smooth, and not much elevated above the surrounding tissue. Whitish spots are nowhere larger than

millet-seeds. Several smaller cavities are found in the centre of middle and lower lobes, all filled with pus of the same character as that at apex. They are all bounded by a fine wall, are all simple, crossed by trabeculæ, but not communicating with others. Left lung has the same characters, but to a less extent. Near its base and root is a spot of about three inches in diameter, filled with rough granular spots like those above described, but are much more free from colouring matter. Injection does not penetrate into any of these masses.

Abdominal viscera are healthy.

Case 2.—Charles Barlow of Hanley, æt. 36, married, admitted into North Stafford Infirmary, March 5, 1861. —A pale, emaciated-looking man, dark hair, grey eyes, sallow complexion. Is a hollow-ware presser. Has always been a sober man. Has had two children ; one dead of scarlatina, the other ill from epilepsy. Has suffered from some palpitation and from winter cough for the last seven years. Has never had rheumatism. Breath has been growing shorter of late, and he has had dyspnœa on exertion. Had no expectoration till two years ago. Sputum at first frothy, became subsequently viscid, and then purulent. Has never noticed it particularly black ; has rarely seen blood in it, which has never existed in more than a few streaks.

Present attack.—Caught a severe cold in October last. Recovered slightly, but never perfectly ; and was again taken worse at Christmas. Has kept his bed for some weeks before coming here. Says that expectoration has only become purulent during the past few weeks.

Present state.—Urine acid, scanty, very high colour, containing a faint trace of albumen, and a very con-

siderable quantity of purpurine. Face has slightly livid tint, very pale and sallow. Conjunctiva not congested, nor eyes prominent. Skin rather cold, cough very frequent, expectoration thick, purulent, running together into masses, not painful. Legs swollen, pit on pressure. Abdomen slightly swollen. Pulse 98, jerking, irregular. Respiration 32. Deeply formed thorax, flattened at both sides in lower lateral regions. Elevation movements exaggerated. Only very little expansion movement even on deep inspiration. There is undue resonance in anterior mediastinum. Resonance impaired under both clavicles, more so under right than under left. Both bases have diffused dulness, left more so than right. Large and small loose râles heard with inspiration and expiration all over front of chest. Expiration on left side not prolonged, but is so on right where it has a slightly blowing character. Sibilant râles, heard with inspiration and expiration, exist in lower two-thirds anteriorly on right side. Posteriorly, fine loose râles same as in front, heard at left base. Right base but little moist râle, a good deal of sibilus heard. Vocal resonance exaggerated at left apex and left base. No bronchophony.

Cardiac superficial dulness 1½ hands'-breadth transversely, and two hands'-breadth vertically, begins mid-sternum head of fourth cartilage, and extends to three inches below nipple : does not extend to right of sternum. Apex-beat very indistinct, not to be distinctly seen or felt. Murmur with first sound loud and harsh ; heard most distinctly two inches below nipple, and half-inch outside line drawn vertically from it. It is propagated faintly towards axilla, and hardly at all towards sternum. No murmur at base. No murmur with second sound anywhere, but occasional reduplication at base. Ordered

turpentine stupes to chest. Beef tea Oj.—℞. Inf. Digital. ʒij. Mist. Expect ℥j. quater quotidie.

March 7th.—Continued to sink all yesterday. Ordered brandy, 3 oz. Now he is excessively prostrate, and face a livid pale. Expectoration greatly diminished.—℞. Sp. Æth. Sulph. ♏ xx. Vin. Ipecac. ♏ viii. Morph. Mur. gr. $\frac{1}{12}$ Dec. Senegæ ℥j. 4 quotidie. Death took place at 7 A.M. on March 8th.

March 9th.—*Post-mortem* 28 hours after death. Nothing remarkable externally. Rigor mortis still persisting. Thorax opened; only a small portion of heart uncovered by lung. In anterior margin of left lung, which overlies the heart, are several hard nodules, firm, solid, resisting the finger. Heart when removed is found much enlarged, particularly the left auricle. Left ventricle is also large, but its walls are not much thickened. Mitral opening admits tips of thumb and four fingers. Some puckering of edges of valve. Right ventricle greatly enlarged. Tricuspid valve much puckered. Right auricle enlarged. Microscopic examination shows much fat in muscular substance, both of auricles and ventricles. Aortic and pulmonary valves healthy.

Lungs.—Left lung firmly adherent throughout to pleura. Emphysematous at anterior margin, where nodules described are felt. Apex firmly adherent to pleura. When cut into, it is found to be almost entirely solidified at apex. When cut into here, the lung tissue is found to be converted into masses of firm fibrinous resisting matter, lying closely to one another, but separated by thin lines of condensed pulmonary tissue. All greatly blackened. These masses are about the size of walnuts. Interspersed among them are calcified masses, grating under the knife, scattered through the black fibrous masses, and sometimes existing in the centre of

these, giving them a mottled look. No cavity to be found, except in some places in centres of calcified masses, where cretification has not proceeded so far as in others, and substance breaks down when cut into. These masses exist scattered throughout the lung, both at anterior when cut into, and also at base. They appear to be encapsuled with firmer fibrous substance than rest of lung, but cannot be enucleated. They are intensely black in colour, and stand above level of cut surface, and are strongly resistent to the knife. Whole appearance of lung very black, even in emphysematous spots. Bronchi are highly nigrated. No ulceration anywhere, even in finest divisions. Black striæ on pulmonary pleura, wherever it is not adherent to costal pleura. Bronchial glands at root of lung filled with black matter. One which has softened in the centre is of the size of a walnut, and contains a matter not gritty, but of appearance, feel, and consistence of thick black grease. No black matter to be found in mucous membrane of bronchi even in finest divisions. Lobes of lung adherent. Right lung less retracted at side than left, but not so emphysematous at anterior margin; lobes adherent, but lung not attached to costal pleura. Exactly resembles left lung in appearance above described.

Liver normal size, presents nutmeg appearance with portal congestion. Is deeply stained with bile.

Kidneys contracted. Left contains many cysts. Under the microscope gives increase of fibrous tissue, and fatty degeneration of epithelium.

Stomach and Intestines and Brain healthy.

Case 3.—Thomas Jervis of Shelton, æt. 40, single, potter, admitted into North Stafford Infirmary on January 8, 1861. Has suffered from winter-cough for

many years, with increasing shortness of breath. Has had several bad attacks on his chest during the past four or five years. Has had a good deal of expectoration, at first frothy, but now purulent. Has never spat blood. Family healthy.

State on admission. Emaciated sunken appearance. Dark hair and grey. Grey eyes, fingers not clubbed. Respiration hurried. Frequent cough. Much purulent expectoration of grey colour, running together in masses, and very tenacious. Face rather flushed. Skin hot; not perspiring.

Physical examination of Chest. Dulness under right clavicle, but not absolute, extends as low as third rib. Absolute dulness under left clavicle as low as fourth rib. Both sides to the extreme base deficient in resonance. Under right clavicle inspiration harsh, altered towards termination, with fine subcrepitant râle. Expiration here blowing, not prolonged; not divided from inspiration, but attended also at close with fine subcrepitant râle. Spoken voice under right clavicle harsh, not bronchophonic. Whispered voice gives imperfect pectoriloquy. Under left clavicle inspiration harsh, expiration tubular, blowing, and prolonged. Neither inspiration nor expiration is attended with râle. At level of third cartilage, left side, inspiration has a distinct cavernous character. Vocal resonance bronchophonic, but no pectoriloquy on this side. Blowing character of expiration extends to base in front, left side, but loses tubular and cavernous character below fourth rib. A large loose râle, evolved in a few distinct bubbles in expiration, attends forced breathing over the whole of this side in front. No fine râle heard here.

Back right side. Imperfect dulness in right supraspinous fossa. Resonance at base moderately good.

Inspiration has the same characters as in front. Expiration more distinctly tubular and cavernous in supra-spinous fossa. The fine râle heard in front is not audible here. Vocal resonance is cavernous in infra-spinous fossa, more markedly so than in front. It becomes bronchophonic in middle third of the scapula, below this it is simply intensified. On forced breathing a medium-sized mucous râle is heard over whole of right back below infra-spinous fossa.

Back left side. Dulness absolute in supra-spinous fossa. It is less so in infra-spinous fossa. Below this it is good. Inspiration blowing, but neither tubular nor cavernous, except at one spot near spinal column at middle third of scapula, where it has the latter character. Expiration almost inaudible in supra-spinous fossa. No râle, even on forced breathing, on this side posteriorly. No pectoriloquy. Vocal resonance is somewhat exaggerated throughout. It has a somewhat hollow sound at spot where expiration is cavernous.

Heart's apex in normal site. Dulness normal. Action rather irregular. Sounds are heard most clearly at base, but are unattended with murmur.

Tongue moist and clean. Appetite pretty good. Throat exceedingly sore. Voice hoarse. Has difficulty in swallowing. Follicles of throat enlarged. Epiglottis reddened, not ulcerated.

℞. Mist. Ferri Iod. ℥j. t. d. s. Ol. Morrhuæ ʒfs. bis die.

℞. Ac. Hydrocyan. dil. ♏ iij. Vin. Ipecac. ♏ xv. Tr. Camph. cō. ♏ v. Aq. ℥j. 4tis horis. Acetum Lyttæ under both clavicles. Sol. Argent. Nit. (gr. x. ad ℥j.) to throat.

Full diet without beer.

Jan. 15*th.*—Cough better. Dulness remains the same, and also the other physical signs. Sputa the same.

Jan. 21*st.*—Left hospital at own request.

The lungs, which were injected in the case of William Bowden (No. 1), with a view to the further investigation of this disease, gradually decomposed, and were rendered useless. I hoped, however, to be able to secure another specimen to accompany this paper, but the difficulty of obtaining post-mortem examinations is so great, that it is only at long intervals that permission to examine a body is granted. Frequently it happens, that patients in a dying state insist on being removed to their homes to die, merely to avoid the possibility of a post-mortem examination without their sanction, should death occur in the Infirmary. My friend Dr. J. T. Arlidge, who succeeded Dr. Wilson Fox as Senior Physician to the North Stafford Infirmary, writes to me as follows in reply to my application for a diseased lung to illustrate this thesis :—"I am much interested in the question, but am deterred from working at the pathology by reason of the almost impossibility of getting post-mortems to ascertain the real condition in the several stages of the disease." This feeling of antagonism to post-mortem examinations is so strong in the Staffordshire potteries, that it can hardly be surpassed in any other district of England. Since my resignation in Nov. 1862 to the present time, but one post-mortem has been performed on a case of Potter's Bronchitis, of which, unfortunately, neither notes nor specimens were preserved.

The cases, whose histories I have just narrated, are only examples of the disease in its advanced stage, where the lung tissues are more or less completely disorganized ; and the admission of the patient to the hospital may be regarded as "the beginning of the end." The malady however is essentially progressive, and consists of three distinct stages. At the outset it differs neither in physical signs nor symptoms from an ordinary bronchitic

attack. There is nothing to arouse suspicion of more serious mischief. The features are well marked and characteristic, so that no doubt can be left on the mind that the first stage is one of acute, or more commonly sub-acute bronchitis. This gradually passes, after an interval varying in different cases, into the second stage, that of confirmed chronic bronchitis, with more or less emphysema. And now it is, when the patient is compelled through increasing dyspnœa and continuous cough to seek medical advice, that the real nature of the malady is suspected. Throughout the chest a general wheezing is heard, with much rhonchus and less sibilus; expiration sound greatly prolonged; bronchial breathing is usually audible about the middle of each lung (supposing the disease to be symmetrical), or rather above the middle, bronchophony being less constant; respiratory sounds generally are coarse and harsh, differing greatly from those of simple exaggerated respiration. There is dulness on percussion mostly about four fingers'-breadth beneath the clavicle; and exaggerated resonance over either lung towards the median line anteriorly, which is never absent in these cases. The other sounds are sometimes more audible behind than in front, but rarely so; and occasionally they may be heard equally well in both positions. There is some emaciation, and the patient says he has been losing flesh "a good while." The expectoration is copious and purulent in character, varied every now and then, when the attack is aggravated, with frothy liquid. The dyspnœa is persistent and does not occur in paroxysms. The respiration is peculiar, nearly asthmatical in character, and instantly arrests the attention. So striking is this symptom that I was able, after closely observing many cases, to diagnose accurately in nine cases out of ten, both the patient's particular occu-

pation, and the state of his chest, from his general appearance and this characteristic respiration. To pass from this condition into the third stage is an easy gradation, and is only a question of time. I am not prepared to say that the transition is inevitable, but I have yet to see the patient who has remained stationary in the second stage. The symptoms which obtain in the third stage have already been related in detail in the narrative of the three cases, so that nothing more need be said respecting them in this place.

Before proceeding further, I may remark, that this disease appears to be confined to certain branches of the potting trade, and not to be common to potters generally. I have never met with a patient suffering from the malady that was not either a "hollow-ware presser" or a "flat-presser," so that one is led almost irresistibly to the conviction, that beyond these departments the disease does not extend. I have looked for it most carefully in every patient engaged in other branches of potting, but without success. None of the men thus employed live long; they all suffer from chest affections, and if any escape, it must be by deserting their particular calling at an early period. But I am not prepared to state that they all are afflicted with this peculiar bronchitis, though it must be conceded, that when a number of men, engaged in the same pursuit and surrounded by the same circumstances, are liable to pulmonary disease from the nature of their occupation, it affords a strong presumption that the morbid processes would assume somewhat similar features in all, modified only by those peculiarities of constitution proper to each individual.

Now if it be true that this malady is confined to a certain class of artisans, it is clear that there must be something in the nature of the employment itself which

these men follow, or in the circumstances attending its prosecution, which renders them peculiarly obnoxious to the disease. A brief description of their daily work will render this apparent, and will greatly facilitate the future investigation of the pathology of the affection by affording some insight into the nature of the causes which lead to its development.

And first, of the "Flat-pressers." Under this name are included dish-makers, plate-makers, saucer-makers, and cup and bowl makers. They roll out a piece of prepared clay, which, when of proper thickness, they shape upon the mould. The material is used in a wet and ductile state, but bits of it get scattered over the floor, and rapidly drying, are stirred up by the feet of the boys who are continually running about the workshop. The atmosphere is thus more or less impregnated with a fine dust, clearly observable only when it lodges on a flat surface, or is seen in the sunshine during a bright day. The articles made by the flat-pressers are carried immediately into the "stove" or drying-room (to be presently described), by young boys, who are kept running to and fro all day, thereby filling the atmosphere of the shops with dust. The quantity of dust varies according to the cleanliness of the place. Some workshops are swept daily, others only once a week, and of course the operatives employed in the latter are more exposed to inhale dust than those in the former. But even in those establishments where the floors are swept daily, the dust raised in the process has not time to settle again before the people commence work.

The temperature of the workshops depends upon the heat of the "stoves," which are close at hand, and this, in its turn, is regulated by the sufficiency or deficiency of the supply of moulds. When the men are well sup-

plied with them it is not necessary to hasten the process of drying, and the "stoves" need not be so highly heated. When, on the other hand, there is a deficiency of moulds, the potters endeavour, by way of compensation, to hasten the process of drying, in order that the moulds may be again soon ready for use.

Dish-makers are less exposed to heat and dust than plate and saucer makers, the operations of the former being of slower progress. The "stoves," therefore, do not require to be so highly heated, and it is less essential to have them placed near the men.

China flat-pressers are less exposed to heat, but quite as much exposed to inhale dust as those who work in the commoner material. China articles are partially dried on a shelf before being placed in the "stove," which therefore requires to be neither so highly heated, nor to be placed so near the workmen.

Saucer-makers create much dust in giving an edge to the saucers after they have been dried in the "stove."

"Hollow-ware pressers" or "squeezers" are employed in the manufacture of jugs and other kinds of hollow ware, which are formed by pressing the clay inside the mould. Their occupation is much more laborious than that of the flat-pressers, owing to the size of the ware they manufacture, *e.g.*, ewers and soup-tureens. They do not employ any assistants, and carry their own moulds into the "stoves." They are exposed to the same influence as the "flat-pressers," and though their work proceeds more slowly, and it is not necessary to place the "stove" so close to the workman, yet this is only an apparent advantage, for the hollow-ware presser has to transfer his moulds to the stove himself, and experiences its injurious effects to the fullest extent. I have observed that pulmonary affections are more prevalent amongst the work-

men in this particular department than in any other branch of the potting trade.

The "Stove," to which I have alluded above, is a little room, or rather oven about 13 feet square, and from 8 to 12 feet high, partitioned off from the shop, closely confined except at the door, and without windows. They are fitted inside with shelves, on which the moulds with the moist ware upon them are placed, in order that the ware may be dried sufficiently to be removed. In the centre of the room is a large cast-iron stove or furnace, which I have often seen heated to redness. As these "stoves" are placed in the workshop, and frequently, especially among plate-makers, close to the operatives for the sake of convenience, the atmosphere in which they work is necessarily of an elevated temperature, and very dry. The communication between the two is uninterrupted, a doorway alone separates them. In one of these "stoves" or drying-rooms the thermometer rose to 120°, in another to 130°, and in a third to 148°.

I proceed, in the next place, to consider the nature and progress of those morbid processes, which produce such extensive alterations in the texture and such impairment of function of the pulmonary organs, as detailed in the cases given above, and which seem to terminate in the disorganization and ultimate excavation of the lung tissue itself.

It is not difficult to understand how the acute attack of bronchitis invades these operatives. There are two causes which, either singly or combined, are sufficient to explain this. The one is the sudden transition from the highly heated and very dry atmosphere of the workshop to the cold air of the streets, and the low temperature and superabundant moisture which invariably prevail during certain seasons of the year in districts with a clay

subsoil ; the other is the constant inhalation of the particles of fine dust which abound in the atmosphere of the shop. The potters, as a class, are greatly below the average in vigour and robustness of constitution ; inheriting, as they mostly do from their parents, a cachectic habit, they have the appearance of sickly plants ; their vitality is low and offers little or no resistance to the access of disease. The elevated temperature to which they are exposed may predispose them to the inroads of disease generally, *indirectly* by modifying the amount of oxygen inspired, and *directly*, by elevating the sensibility of the heated surface to impressions of cold. This probably of itself may be sufficient to give a *special* direction to the general predisposition, and to render the pulmonary organs in particular liable to morbid lesions. But when to this is superadded the presence of irritating particles of fine dust throughout the mucous tract of the bronchial ramifications, it is no longer matter of surprise that disease should assume the bronchitic form.

It may be objected that the evils arising from alternations of temperature are greatly lessened, if not altogether prevented by the influences of habit? Doubtless, "the power of accommodation in the body, depending on the generation of animal heat, and on the functions of the lungs and of the skin, provides in the healthy state against all changes which are not in excess," as Sir Henry Holland observes. But when these functions are impaired, or the body otherwise disordered, as usually obtains in potters, every such change has influence, either by disturbing the balance of circulation between the external surface and the membranes or different glandular structures within the body, or by checking or augmenting the discharge of perspirable matter. And yet the objection in some instances holds good. Every now and then we

meet with cases which have assumed a chronic character from their onset, and acute symptoms have never formed a part of their history. And this is the more remarkable, when we take into consideration the twofold nature of the combination to which these patients have been exposed. How much of this immunity is due to the influence of habit, and how much to individual idiosyncrasy, it is difficult to determine. Probably they both have weight though not equally. We certainly know that the sensibility of that most sensitive of mucous surfaces, the conjunctiva, to the presence of foreign bodies, gradually diminishes when the extraneous matter has become permanently established in its tissues. There is, so to speak, a reconciliation between them. May it not then be assumed, that in a similar manner the bronchial membrane gradually becomes accustomed to the irritating dust, and never manifests symptoms indicative of the acute form of inflammation? The two cases, pathologically considered, are not strictly parallel, and I refer to the conjunctiva as an exemplification merely of the modifying influence of habit in the development of disease.

But the question still remains, Why is it that the same causes produce in one person the acute variety, and in another the chronic form of bronchitis? The solution of this problem is a matter of infinite difficulty. We are in the habit of attributing it to idiosyncrasy. But this is equivalent to acknowledging our inability to give a satisfactory answer to the question. We cover our ignorance with this name. It is a conventional phrase, and so perhaps is convenient, but it adds nothing whatever to our previous knowledge of the subject.

I think I have observed that, as a rule, persons with a quiet, low, and rather languid pulse, whose vitality is somewhat below par, as it is called, are mostly liable to

chronic diseases; whilst, on the other hand, those in whom the vital processes are carried on energetically, and whose full bounding pulse would seem to carry health to every part of the body, have appeared to be singularly obnoxious to the attacks of acute diseases. In these latter, too, disease extends with greater rapidity, and pursues a more uniformly fatal career; as if what formerly contributed to an excess of healthy action, so to speak, now *mutatis mutandis* materially assisted in the development of a morbid action. The same may be said perhaps with equal truth of children, with whom acute diseases are notoriously fatal and rapid in their course. But amongst potters robust health can hardly be said to exist, so that in their case we have not to consider the form which the same disease will assume in a strong person and in a weak person; but rather under what aspect will it manifest itself in a weakly person, and in one more weakly? My own observation leads me to the conclusion, that in the one case the disease is developed in a sub-acute form, and in the other in a chronic shape. The conviction that the lower the state of the vital powers, the greater is the tendency of disease to chronic development, and *vice versâ*, seems to my mind to be almost irresistible.

In the cases under consideration, however, the establishment of the chronic form is simply a question of time. That the occurrence of a disease once leads to a predisposition to other attacks of the same, certain specific diseases excepted, is a fact which all must admit. And in practice it is found that bronchitis is especially apt to recur every winter in those who have once been the subjects of it. Amongst potters it is not unusual for an acute or sub-acute attack gradually to pass into the chronic form, even in summer time, and then to

remain permanent. There is always more or less secretion from the bronchial tubes, but not sufficient to attract attention or to hinder from working. It is only when the winter weather sets in, when they "catch cold," and the symptoms are exaggerated, that they are driven a second time to the Infirmary. Then we learn that ever since their first illness they have suffered from shortness of breath (or as they express it, have been "touched in their breathing"), and a short cough; that lately they have been getting weaker, and the dyspnœa and cough have increased in severity so much as to render them unable to follow their employment any longer. When this state of things once obtains, it is commonly persistent, modified only by atmospheric changes or constitutional disturbance. Meanwhile anatomical changes are taking place in the structures of the lungs, till a condition is reached such as that in the cases narrated, which is no longer consistent with the proper performance of function and the maintenance of life.

What are those changes? Independently of the thickening and hypertrophy of the walls of the bronchial tubes resulting from inflammation, the presence of fine dust acting as a foreign body creates irritation of the mucous surface, followed by exudation from the bronchial membrane in which probably the dust is imbedded. In this way the tubes become narrowed directly, and respiration is impeded; and indirectly by the pressure of infiltrated exudation-matter which sometimes extends beyond the walls of the tubes, and encroaches on the adjacent lung substance. This diminution of calibre and constriction may gradually lead to obliteration of the finer bronchi, and to collapse of the air vesicles in which they terminate. Nay more, it is not impossible

that particles of fine dust may find their way into the minute air-cells themselves, and induce changes in them similar to those which take place in the air-tubes.

But this induration-matter may impede the function of the lung-substance in another way, by obliterating the vessels distributed to it and cutting off its supply of blood, so that atrophy of the tissues follows; and by obstructing the capillary circulation local congestion is favoured, and the blood but imperfectly oxygenated. Hence arises distressing dyspnœa and a dusky hue of the face.

Under circumstances such as these it is, I apprehend, that the emphysema recorded is apt to occur. Many are the theories that have been offered in explanation of its mechanism, and none of them is entirely satisfactory. Most of them have some truth in them, but not the whole truth. Neither of them is applicable in every instance.

Dr. Williams maintains that distension is the consequence of extra work thrown upon the healthy vesicles, the air-cells communicating with the plugged bronchi (in bronchitis) escaping inflation. To this it might be objected, and very justly, that at the end of inspiration we have in the healthy chest 131 cubic inches of air, and the lungs can take up 119 inches more, if force be used, without causing emphysema; hence the lungs must be more than half useless for emphysema to follow as a consequence! Dr. Gairdner holds that when a lung is atrophied from any cause, if the chest expand normally, the residual lung must follow that the vacuum may be filled. This theory, however, seems to me to be untenable, for in phthisis we find that diminished capacity is compensated for by increased frequency of respiration. In pneumonia, too, the same phenomenon is observed;

and yet emphysema in pneumonia is indubitably a great rarity, and in phthisis the alliance is but seldom met with.

These are the views which are most popular at the present day, but they fail to elucidate the cases under consideration. I venture, therefore, to offer the following explanation, as that which coincides most readily with the anatomical characters of the disease I am describing :—

It is not improbable that the act of coughing of itself is sufficient to cause some dilatation of even healthy air-vesicles if continued for a number of years ; for the glottis being closed, and the walls of the chest, the diaphragm, etc., thrown into violent contraction, the pressure that is exercised upon the air-cells during the act must be enormous. But when to this is added the obliteration of some vesicles, the diminished expansion of others, and obstruction of the finer bronchi through the infiltration of exudation-matter, the strain that is thrown upon the patulous tubes and cells is necessarily so much the greater, whilst their elastic resistance remains the same, and dilatation is the consequence.

If, then, such change can take place in air-cells whose walls are healthy, how much more likely is the distension to occur in vesicles whose parietes are degenerated ? I have already shown in what manner the small vessels surrounding the air-cells are obliterated ; and the nutritive supply being cut off, atrophy of the walls must follow as a natural sequence. Independently of this, however, the mere act of distension alone by compressing the capillaries between the vesicles cannot but interfere with the nutrition of the texture, and initiate atrophic changes favourable to further distension. This emphysematous condition is usually observed on the surface of the lung where it is most deficient of support.

'Again, commensurate with this deficiency of nutrition in the air vesicles is the impairment of their elasticity and tonicity. They become as it were paralysed, and offer little or no resistance to the ingress of air in excess, whilst at the same time they contribute but feebly to its expulsion; a large portion of it apparently remaining stagnant, and the remainder being slowly expired. When the respiratory process is accelerated, as under unwonted exertion, inspiration and expiration become strangely commingled, and produce the extreme dyspnœa and wheezing so characteristic of emphysema; the normal exchange of oxygen and carbonic acid is interfered with, and though the oxygenizing surface of the dilated cells is increased, the blood is no longer perfectly arterialized. I shall have occasion to allude to this subject again presently.

I proceed in the next place to consider the nature of the other *post-mortem* appearances that were observed, viz., the tough fibrous character of some portions, the black nature of others, and the extensive infiltration of the whole with minute cretifications.

"Induration-matter is endowed to a remarkable degree with the property of slow contraction—a property which renders its presence most beneficial or most baneful; often becoming the seat of saline deposits."—(Adventitious Products, Todd's *Cyclop. of Anat. and Physiol.*) It is not difficult to conceive how this exudation material mixed up with dust, slowly contracting and gradually hardening, sets up irritation in the surrounding tissues, and excites a species of chronic inflammation in them. The systemic disturbance, of course, is slight or imperceptible on account of the protracted nature of the invasion. Those portions which have not submitted to the further process of softening maintain their thickened

fibrous nature, and are recorded amongst the *post-mortem* appearances.

The blood itself being but imperfectly oxygenated by reason of a defective respiratory process, ceases to afford the necessary stimulus to the capillary system, and a condition obtains highly favourable to congestion and stagnation. The exudation-matter likewise contributes no small impediment to the free circulation in the minute vessels. I cannot, therefore, but regard this stagnation as one of the sources from whence the black matter in these cases originates.

It is generally believed that there is always a certain amount of melanic pigment present in the lung tissue and bronchial glands of even healthy persons, and that this has a tendency to increase with advancing age. Amongst potters, however, advanced age is seldom attained; and besides, the quantity of black matter is too large to be accounted for in this manner. It is, I think, highly probable, that in addition to the pigment normally present, the stagnation of the blood in the capillary system, by altering the character of the hæmatin, tends greatly to its augmentation. This view receives support from the researches of Wedl (*Pathological Histology*), who says: "It may be assumed with considerable probability that the pigmented involution originates for the most part from the dissolved colouring matter of the blood, which penetrates the cell-wall, and undergoes various changes of colour within the cell, although perhaps the pigment may also arise in a kind of carbonizing process of the protein substance contained in the cell."

In the next place, as the inflammatory process pursues its course the tissues soften and gradually break down, leaving cavities in the lung-substance filled with the pigmentary material of the disintegrated textures. In

some portions this is solid, or rather negatively firm, and in others thickly fluid, like black grease or cream, depending probably on the more or less advanced stage of the softening process in each case. But another and perhaps co-extensive source of this pigment is to be found in the inhalation of CO_2 to which these potters are liable in the "stoves" or drying-rooms. The stove itself (*i.e.*, the furnace) is a very common affair, certainly not tight at the seams, and allowing the free escape of CO_2 in the process of combustion. It is supplied abundantly with coal, that a high temperature may be maintained, and CO_2 therefore is very rapidly generated. The workshop is very imperfectly ventilated, so that a considerable quantity of CO_2 expired by the operatives accumulates and is again inhaled by them. May this not be the origin of the "carbonizing process of the protein substance contained in the blood-cells" mentioned in the quotation from Wedl?

Continental writers have entertained various opinions relative to the formation of pigment in the pulmonary organs, but have failed to throw much light on the subject.

Bichat supposes it to be owing to small bronchial glands extending along the surface of the pleura. Breschet believes that it is formed by the blood exhaled into the cellular tissue, stating that its chemical composition leads him to that conclusion. Trousseau says that it is produced by a misdirection of the natural pigments of the body, resulting from age, climate, or disease. Andral says that the black appearances are the result of secretion, and that it is more manifest as the individual advances in life. Heasinger agrees with Trousseau. Laenner appears to be doubtful as to the real origin of the black matter.

So long ago as 1813, Dr. Pearson (*Philos. Trans.*) drew attention to the subject in the following words :— "I think the charcoal in the pulmonary organs is introduced with the air in breathing. In the air it is suspended in invisible small particles, derived from the burning of coal, wood, and other inflammable materials in common life. It is admitted the O. of atmospherical air passes through the pulmonary air vesicles or cells into the system of blood-vessels, and it is not improbable that through the same channel various matters contained in the air may be introduced. But it is highly reasonable to suppose that the particles of charcoal should be retained in the minutest ramifications of the air-tubes, or even in the air-vesicles under various circumstances, to produce the coloured appearances on the surface and in the substance of the lungs."

Mr. Graham (*Edin. Med. and Surg. Jour.* vol. xlii.) attributes the deposit in the case of colliers to the inhalation of the smoke from their lamps whilst at work.

Dr. Makellar, too (*Edin. Med. and Surg. Jour.* vol. for 1846), holds the same view, but includes the inhalation of CO_2 generated in ill-ventilated pits, and that expired by the miners themselves. He observes : "It is not, therefore, to be supposed improbable that a portion of the infinitely small particles" (of smoke) "thus suspended in the atmosphere should effect a settlement in the more minute air-cells, and in course of time be conveyed to the interlobular cellular tissue by the process of absorption, and thence to the bronchial glands." Again, "there is little doubt that the bronchial glands are the recipients of a portion of the impurities which have been carried into the pulmonary structure by inhalation, and also those left after the process of oxygenation of the blood ; and when it is fully ascertained, from

the character of the atmosphere, that deleterious matter in this form must be conveyed to the air-cells during respiration, there is little difficulty in coming to the conclusion that the black fluid found to such an extent in these glands is similar to, and a part of, that discovered infiltrated into the substance of the lungs. If we trace the black matter in the lymphatic vessels (which has been done) from the pulmonary organs to the bronchial, mediastinal, and thoracic glands, and from thence to the thoracic duct, we cannot but admit that it does find its way into the venous system, and thereby contaminates the vital current." In another place he remarks: "It is still my belief, that the carbon being once inhaled, there is an affinity found for that in the circulating fluid, and from its not being consumed, owing to a deficiency of O, there is a progressive increase going on."

These observations of Dr. Makellar coincide with the opinion of Wedl relative to the "carbonizing process;" and they both give confirmation to my view, that the pigment in the cavities and amid the indurations is due to stagnation of the blood in the capillary system, and ultimate softening and excavation; the blood having previously undergone the "carbonizing process" of Wedl, and the "contamination" of Dr. Makellar, in the manner already described by me.

There is much similarity in some features of Dr. Makellar's cases and mine, but with the carbonaceous deposit all resemblance ceases. (I may remark in passing that I have never met with a case of black phthisis in the colliers of North Staffordshire, probably because the mines are well ventilated, and the "Davy lamp" is in common use.)

Lastly, the cretifications remain to be considered. I

regret very much that I did not submit these to chemical analysis at the time. I hope, however, to continue my investigation of this subject so soon as I can procure another specimen.

If we regard the various fluid secretions of the body as so many simple saline solutions, whose chemical constitution is unerringly accurate, it follows that any deviation from this standard proportion of constituents will materially affect the character of the solution. If the balance between the fluid and solid elements be disturbed, so that the fluid be diminished, the solid must be precipitated; or again, if the solid be superabundant, the fluid remaining normal, the same phenomenon is observed. This deposition occurs also if the chemical relations of the materials of the secretion be destroyed by the presence of a foreign substance which interferes with the suspension of the saline elements in solution. Probably the existence of any obstacle in the excretory passages by which the exit of a fluid secretion is delayed may indirectly cause the solid constituents to be in excess, the fluid, or some portion of it, being removed by the absorbents, or even by evaporation or by exosmosis.

Instances of this calcareous deposition are furnished abundantly by the human body, and will readily recur to the mind of every one. Perhaps the most familiar of all with which we are acquainted is the occurrence of "tartar," as it is called, around the teeth. No one can doubt that this is precipitated from the saliva. It appears first as a layer of slimy mucus, which gradually hardens, and is succeeded by another layer which hardens in its turn, and so on till it accumulates in vast quantities. Calculi of the salivary glands are commonly regarded as depositions from the saliva; and though,

according to Berzelius, phosphate of lime does not exist at all in the healthy saliva, or only in small quantity according to Simon, yet these calculi are found to consist essentially of this salt; and the explanation offered by Dr. Walshe is, that "the excess of phosphate is generated through the influence of irritation of mucous membrane."

"The pulmonary parenchyma is an extremely frequent seat of concretions. The basis in which the saline material accumulates is by far the most frequently tuberculous; more rarely, the fibrinous substance of simple inflammatory exudation forms its nidus." From the analysis of healthy pulmonary mucus by Simon and Nasse, it is clear that phosphates are normally present in the secretion from the lungs. Hence, when the secretion of mucus is exaggerated, as in bronchitis, the proportion of phosphates is relatively increased. In the case of the potters we have in addition the constant irritation of an inflamed mucous surface by particles of dust, and therefore, if the hypothesis of Dr. Walshe be sound, an excessive generation of phosphates must ensue, and precipitation of them in the form of deposits take place. Again, the constriction and narrowing of the bronchial tubes, to which I have already alluded, impedes the exit of the secretion, which is thus rendered liable to a further increase of solid material by the removal of some of the fluid portion in the manner described.

This then is one way in which cretifications in the lungs may have origin. But it is too much to suppose, that in the cases under consideration they were thus formed. It is possible, but not probable. It seems to me to be more reasonable to regard these minute masses as concretions of fine dust in the ultimate vesicles. Countless particles are inhaled with each inspiration and

lodged in the air cells, inflammation is set up followed by exudation, and when contraction ensues the mass of dust is encapsuled, as it were, by the exuded material, and the vesicle itself possibly obliterated, or occupied by it. In the recorded cases they were all of small size, exceedingly numerous in every part of the lung, and similar in character and appearance, affording strong grounds for presuming that they had but one mode of origin.

Again, reasoning from analogy, these cretifications might probably be formed by the deposition of phosphate of lime from irritated mucous membrane upon the particles of dust regarded as foreign bodies, and forming, so to speak, the nuclei of the concretions. Just as happens in the urinary bladder, when substances are introduced from without they soon become coated with the salts suspended in the urine ; and as in the case of a plum-stone lodged in the trachea, which became encrusted with phosphate of lime ; so here, the dust plays the part of a nucleus around which the salts of the pulmonary secretion are gathered. Many other examples might be cited in illustration, but these sufficiently demonstrate my position. The observations of Dr. Walshe lend force to this explanation. Speaking of phosphate of lime he says : "So frequent is the occurrence of this salt in calculous masses on mucous surfaces, as to lead irresistibly to the conclusion, that mucous membrane has a specific tendency to secrete this salt, under certain conditions of local irritation."

The question next arises, Can anything be done in the way of treatment ? Probably removal from the workshop at the first onset of the disease, and abandonment of these particular branches of the potting trade, is the only method of arresting the further development of the malady. But the remedy rests chiefly with the manu-

facturers. Many of the workshops are so badly ventilated that they could not well be worse. Inprovement in this respect alone would be followed by corresponding improvement of the general health of the workmen. A sufficient supply of moulds in every manufactory would remove the temptation to hasten the drying process by raising the temperature of the drying-rooms. But the "stoves" are the crying evil. They are very primitive in construction; indeed, they are just what they always were; no advance whatever has been made in their structure for the past fifty years, notwithstanding that countless operatives have succumbed to the pernicious system. Parents are so well aware of the destructive nature of the employment, that many of them decline to apprentice their children to it. If the "stove" were built off from the shop, it would be something gained, but one that need not be entered at all would be a great boon.

Since writing the above, I learn that Messrs. Minton and Co. of Stoke-upon-Trent have erected a "stove" on this principle by way of experiment. I am sure it will effect a great saving of coal, and will recommend itself therefore. But of 168 manufacturers in the Staffordshire Potteries not all influenced by considerations for the wellbeing of their workmen, few will be inclined to adopt new-fangled notions, which are certain to involve some little outlay of capital at first. It will, I fear, be a work of time to introduce this self-acting stove into general use, and nothing but its economizing character will prevail with most employers, and lead them to adopt it. Till then, Potters' Bronchitis will continue rife in the district, and will carry its suffering victims as usual to a premature grave.

February 29, 1864.