"In a form of Droochitis (simulating Phthisis) which is peculiar to certain branches of the Potting trade."

Charles Parsons.
3.4. Claims first notice of Potter's Phthisis.
5 to 15: Two fatal cases with distension, and one not fatal.
10. Three types: 1. Subacute bronchitis. 2. Emphysema and chronic bronchitis, with acute signs of emphysema in lower regions of contractions in the wall, and with material breathing. 3. Like phthisis.
15. Compared to the Potter's. A fellow-work member working in ill-aired rooms filled with dust and particles derived from dried up lots of coal unshaded in bags, then exposed to alternation of temperature. No chemical analysis of expectoration, or waste from emanations in lungs. More per defective.
On a form of Bronchitis (simulating Phthisis), which is peculiar to certain branches of the Pottery Trade.

It has been said, that we acquire real wisdom from our failures only, 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 characterise the so-called "exact sciences"; so that on the one hand we cannot predict with absolute certainty, that any one disease will always be attended, in every instance, by an unvarying train of physical signs, symptoms; nor on the other hand can we assert, that these signs, symptoms are invariably inductively the manifestations or constants of one and the same disease at all times. Were it otherwise our diagnosis would be almost infallible, the 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 pains-taking thorough investigation of one case of mistaken diagnosis, with its possibly disastrous consequences, impresses more instruction than the consideration of many conducted to a successful issue. The one gradually but steadily fades from the recollection; the other is to be 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, sequence, so that two distinct diseases have been confounded merely from some similarity in their phases. It is only when their micro-anatomy has been carefully investigated and patiently studied, that we are able rightly to interpret their pathology, to reconcile apparently opposite diseases with nearly identical signs and symptoms. Thus some trades occupations are peculiarly obnoxious to a form of disease closely resembling Phthisis in nearly every feature, with the exception that tubercle is
generally absent. In fact, particularly to the so-called "Black Phthisis," "Cotton Phthisis," "Stone Phthisis," "Knife-grinders Phthisis." It is to be regretted, that these names have been thus affixed, for Phthisis conveys the idea of tubercle, and in these diseases the existence of tubercle is purely accidental, not necessary to their constitution. The lung-substance it is true undergoes destructive inflammation, excavation, just as in Phthisis, but Pathology declares this to be simply the sequel to long-continued bronchitic inflammation 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 Potter's; as far as I can ascertain, no account of the affection has ever been placed on record. In virtue, therefore, in this way to bring the subject under the notice of the Profession, to communicate such information as I have been enabled to collect, accompanied by such deductions, remarks, as may, I hope, tend to elucidate the aetiology, nature, cause of this distemper 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 Potter's, Cotton, Iron-workers. The following winter both indicated what diseases were peculiar to each
of these classes, clearly demonstrated that chest affection were prevalent in the district, especially amongst the Potter. One of those admitted early with pulmonary complication, was a Potter of middle age, whose features evinced appearance, disturbing cough, hot, dry skin at first sight gave me 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 sides with flattening of the chest surface, whilst immediately below the lungs were unusually resonant, 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 or percussion, there 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 his affluence 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 diarrhoea. I was unable to detect any pulmonary fibres adherent 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!
Caries indeed there were, & a 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, the lung was infiltrated or discoloured \\nwith a black matter somewhat like those to be described hereafter, only in a much less degree. Several other cases occurred 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 a 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. My own notes having been packed in cases with my books for removal, when I ceased to be H.S. in 1862, they remain as yet in Staffs., in state quo.

Case 1. William Bowler, 25 yrs., single, admitted into N. Stafford Infirmary March 22, 1861. A native of Epper, Holbeck Lane. Light brown eyes, clear complexion. Finger-nails rather clubbed, thickened. Has had "asthma" as long as he can recollect. His father had "asthma" died at 45. He has never, so far as he is aware, had any severe illness
in the chest; no 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 3 weeks. Has not much cough, expectoration not much to speak of.

Diagnosis: Chronic Bronchitis.

Examination of Chest: Respiration in great measure diaphragmatic. Expansion of thoracic cavity on inspiration very slight. Stenosis of 4th cartilage is suspected. Intercostal spaces palpable. Clavicles very prominent, left more so than right. Sternal cartilage greatly depressed. Respiration short and difficult, 28 per minute. A deep inspiration is accompanied by a wheeze, in expiration audible to bystanders. Percussion in front of chest unyielding resonant everywhere. Percussion in side 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. Inspiration prolonged to twice normal length, attended everywhere with vibratory some resonant rales. No moist rale in post. Local resonance in front weak. Posteriorly, right apex is dull as low as middle third of scapula; absolutely dull as low as infraspinatus fossa. At upper part of dulness inspiration is hollow, almost tubular; expiration blowing. Prolonged. In middle third, inspiration is weak but tubular; expiration tubular prolonged. Fine fine moist rale heard with forced breathing at this spot. Local resonance so weak as scarcely to be audible anywhere. Voice rather hoarse, left lung
Posteriorly dull in spots in middle third. Matted with places of respiration, over areas is a dull resonant, rales or rales, present. The rales are quite prolonged over a large portion of this lung. Some fine moist rales heard in spots here. No cavernous breathing in either lung


Ordered: 1. Cod fort. to right side. Nitrated paper to be breathed


April 9. Very little relief to chest. Nitrated paper relieves him a little but only temporarily. Gg. Iod. Ipecac. F. t. d. s. 60s die.

April 28. Has been gradually losing strength. Great dyspnea came in this morning. Face is now livid. Expectoration has ceased. Breath has a pungent odor. Some dulness is now to be found under right clavicle. There is a large loose moist rale there. Otherwise state of chest unchanged. Gg. Oth. Sulph. Gt. Ammon. Am. tann. ac. Tinct. cur. q.d. 7:00. Sir. Scragg F. G. Phis. C. a. F. t. d. s. 60s die.

H. 30. 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 apex. 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 half a peanut.
of a walnut, sometimes of a Maltese orange. Section of the
white flesh is gritty, that of black is smooth, not
much elevated above the surrounding surface. White spots
are nowhere larger than millet seeds. Several smaller
cavities are found in the centre of middle three lobes, all
filled with pieces of the same character as that at apex. They
are all bounded by a fine wall, are all simple, crossed by
radicles, but not communicating with others. Left lung
has the same characters but to a slight extent. Bean its base
root is a spot of about 3½ inch diameter filled with rough
granular spots like those above described, but are much
free from colouring matter. Injection does not penetrate into
any of these masses.
Abdominal viscera are healthy.

Case II. Charles Barlow of Hanley, at 36, married, admitted into
North Stafford Infirmary March 5, 1861. A pale, emaciated
looking man, dark hair, grey eyes, hollow countenance. Is a Hollander
from Belgium. Has always been a sober man. Has had 2 children
1 dead of scarlatina, the other ill from typhoid.
Has suffered from some palpitation and from winter cough for
the last 7 years. Has never had Rheumatism. Health has
been growing shorter of late, the last year dyspepsia or tertian
fever has been expecting till 2 years ago. Spitting at first poorly,
became subsequently vivid, rather prevalent. Has never noticed
it particularly black, has rarely seen blood in it, which
has been excited in more than a few streaks.
Present attack. Caught a severe cold in October last. Recovered slightly, but never perfectly; worse again. Taken worse at Christmas. Has kept his bed for some weeks before coming here. Says that expectoration has only become prominent during the past few weeks.

Present state. Urine acid, scanty, very high color, containing a faint trace of albumen, in a very considerable quantity of pus. Face has slightly bluish tinge, very pale, strung. Conjunctiva not congested, no eyes prominent. Skin rather cold, cough very frequent. Expectoration thick, prominent, running together into masses, not putrid. Lungs swollen, pit of stomach.

Abdomen slightly swollen. Pulse 98, irregular, irregular. Respiration deeply forced, clear, flattened at both sides in lower lateral region. Elevation movements exaggerated. Only very little expansion movement seen on deep inspiration. There is under 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 smell loose rales heard with inspiration. Respiration all over front of chest. Respiration on left side not prolonged, but is so on right where it has a slightly blowing character. Sibilant rales heard with inspiration. Respiration, quiet in lower two thirds anteriorly on right side. Posteriorly, fine loose rales same as in front, heard at left base. Right base but little moist rale, a good deal of fibrillary heard. Vocal resonance exaggerated at left after left base. No bronchopty.
Cardiac superficial dulness ½ hands breadth horizontally, 2 hands breadth vertically, begins mid sternum head of 4th. cartilage, extends to 3 inches belowipple. Does not extend to right of sternum. Affer that very indistinct, not to be distinctly seen or felt. Murmurs with first sound and 1st part, and most distinctly 2 inches below nipple, ½ inch outside line drawn vertically from it. It is propagated faintly towards axilla, hardly at all towards sternum. No murmur at base. No murmur with second sound anywhere, but occasional auscultation at base. Ordered: Perservative syrup to chest. Beef tea ½ p. Syr. Digital. Fj. 4. Pect. Fj. 4. Emer. Fj. 2. quart. quotidie.


March 9. Post. Notes. 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. Initial opening admits tips of thumb & 4 fingers. Some puckering of ves of valve. Right
ventricle greatly enlarged. Tricuspid valve much puckered.
Right auricle enlarged. Microscopic examination shows
much fat in muscular substance both of auricles &
ventricles. Heart & Pulmonary valves healthy.

Lungs: left lung finely adherent throughout to pleura.
Emphysema at anterior margin, where nodules described
are felt. Also finely 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, fibrous resisting matter lying closely to one another
but separated by their lines of containing pleural tissue.
All partly blackened. These masses are about the size of
walnut. Intermixed among these are calcified masses,
placing under the knife scattered through the black fibrous
masses, 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 cavitation has
not preceded so far as in others. Substance breaks down when
cut into. These masses exist scattered throughout the lung both
at anterior when cut into, also at base. They appear to be
encapsulated with firm fibrous substance than rest of lung,
but cannot be incised. They are intensely black in
colour, stand above level of cut surface, are strongly
resistant to the knife. Whole appearance of lung very black
even in physiometric spots. Bronchi are highly congested.
No ulceration anywhere, even in finest divisions. Black
strica or pulmonary plaque whenever 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, contains a matter not quite hot of appearance, feel, 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 left retracted at side than left, but not so hypersthenous 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, fatty degeneration of epithelium.

Stomach & intestines healthy.

Case III. Thomas Brown of Shelton, 26, 40, single, Potter, admitted into North Stafford Infirmary on Jan. 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 4 or 5 years. Has had a good deal of dyspnoea, at first firstly but now persistent. Has never spat blood. Family healthy.

Frequent cough. Much perspirable expectoration of frothy colour, running together in masses, very tenacious. Face rather flushed. Skin hot, not perspiring.

Physical examination of chest. Sibilts under right clavicle but not absolute, extends as low as third rib. Absolute sibilts 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 five subaperturate ridge. Inspiration here blowing, not protended, not divided from expiration but attended also at close with five subaperturate ridge.

Spoken voice under right clavicle harsh not bronchophonic. Whispered voice gives imperfect pectoriology. Under left clavicle inspiration harsh, expiration tubular, blowing, not protended. Neither inspiration nor expiration is attended with rale. At level of third cartilage left side, inspiration has a distinct cavernous character. Pectoral resonance bronchophonic, but no pectoriology on this side. Blowing character of expiration extends to base in front, left side, but lower tubular cavernous character below fourth rib. A large coarse rale evoked in a few distinct bubbles in expiration attends forced breathing over the whole of this side in front. No fine rale heard here.

Back right side. Imperfect sibilts in right supraclavicular fossa. Resonance at base moderately good. Inspiration has the same character as in front.Expiration more
Distinctly tubular resonances in supra spinae fossa. The fine rales heard in front is not audible here. Vocal resonance is cavernous in infra spinae fossa, more marked here than in front. It becomes bronchophonic in middle third of the scapula. Below this it is simply intensified. On forced breathing a medium sized resonant rale is heard over whole of right back below infra spinae fossa. Back left side:—Soldrps absolute in supra spinae fossa. It is also so in infra spinae 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. Inspiration almost inaudible in infra spinae fossa. No rale, even on forced breathing, on this side posteriorly. Inpectoscopy. Vocal resonance is somewhat exaggerated throughout. It has a somewhat hollow sound at spot where expiration is cavernous. Apex deep in normal site. Badly normal. Action rather irregular. Sounds are heard most clearly at base, but are unattended with murmur.


P. Rist. Fini d.s. F. E. d.s. H. Rosthewz. Fis. bis die.

Jan. 15. Cough better. stools remains the same, also the other physical signs. tonsils the same.

Jan. 21. left Hospital at our request.

The lungs, which were injected in the case of William Board (3rd.) with a view to the further investigation of this disease, gradually decomposed in some vessels. It proved, 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. Aldridge, who succeeded Dr. Wilson Fox as Senior Physician to the N. 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 suspected 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 Bronchitis, of which, unfortunately, neither notes nor specimens were preserved. The cases, whose histories have just narrated, are only examples of the disease in its advanced stage, when the lung tissues are more or less completely disorganized; the admission of the patient to the hospital may be regarded as the beginning of the end. The malady, however, is essentially progressive, consists of three distinct stages. At the start it differs little in physical signs or symptoms from an ordinary Bronchitis 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 progresses, 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 dyspnoea 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 thick mucous and sputum. Inspiration 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 thraw, differing greatly from those of simple exaggerated respiration. There is dulness on percussion mostly about 4 fingers' breadth beneath the clavicle; 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; occasionally they may be heard equally well in both positions. There is some eparaxiation, the patient says he has been losing flesh "a good while." The expectoration is copious and purulent in character, varied every day or often, when the attack is aggravated, frothy liquid. The dyspnea is persistent, colors not occur in jaundices. The respiration is peculiar, nearly costal, and in character, frequently arrests the attention. So striking is this symptom that I was able, after closely observing many cases, to diagnose accurately in 9 cases out of 10, both the patient's particular occupation and the state of his chest from his general appearance, this characteristic respiration. To pass from this stage into the third stage is an easy process, being only a question of time, I am not prepared to say, that the transition is inevitable, but 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 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, that is to commerce to Potters generally. I have never met with a patient suffering from this malady, that was not either a "Hollow Ware" or a "Flat Ware" so that one is led almost irresistibly to the conclusion, 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 these men, although employed long, they all suffer from chest affecting if any escape, it must be by clearing their particular calling at an early period. But I am not prepared to state that they are all afflicted with this peculiar Bronchitis; though it must be conceded that when a number of men, engaged in the same pursuit, surrounded by the same circumstances, are liable to pulmonary disease from the nature of their occupation, it affords a strong presumption, that the mobb'd 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 are subject to in the circumstances attending its
prosecution, which renders their peculiarly donations to the disease. A brief description of their daily work will render this apparent, 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 “Hatmakers.” Under this name are included Bonnetmakers, Plate-makers, Sareamakers, and Bowl-makers. They will out a piece of prepared clay, which, when of proper thickness, they shape upon the wheel. The material is used in a sort pliable state, but bits of it get scattered over the floor, rapidly drying and 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 Hatmakers are carried immediately into the “store,” 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 operations 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 process commences.

The temperature of the workhouse depends upon the heat of the "stoves," which are close at hand; this, in its turn, is regulated by the sufficiency or deficiency of the supply of moulds. When the men are well supplied with them it is not necessary to hasten the process of drying, the "stoves" need not be so highly heated. When, on the other hand, there is a deficiency of moulds, the potter endeavors by way of compensation to hasten the process of drying, in order that the moulds may be again soon ready for use.

Dishmakers are also exposed to heat and dust, that Plate and Saucer makers, the operation of the former being of slower progress. The "stoves" therefore do not require to be so highly heated, it is less essential to have them placed near the workmen.

China Hatrefers 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 Wine Prefers," or "Squeezers," are employed in the manufacture of Jugs to give kinds of hollow ware, which are formed by pressing the clay inside the mould. Their
occupation is much more laborious than that of the Platepresse, owing to the size of the ware they manufacture, e.g. iron strap turrets. They do not employ any assistant to carry their own moulds into the "stores". They are exposed to the same influence as the Platepresse, though their work proceeds more slowly, it is not necessary to place the "store" so close to the workman, yet this is only an apparent advantage, for the hollow ware presse has to transfer his moulds to the store himself, experiences its injurious effects to the fullest extent. I have observed, that pulmonary affections are more prevalent amongst the workmen in this particular department, than in any other branch of the Potting trade.

The "store", to which I have alluded above, is a little room or cellar, about 13 feet square, from 8 to 12 feet high, partitioned off from the shop, closely confined except at the door, without windows. They are fitted inside with shelves on which the moulds with the moist core upon them are placed, in order that the core may be dried sufficiently to be removed. In the centre of the room is a large cast-iron stove or furnace, which has often been heated to redness. As these "stores" are placed in the workshop, frequently, especially among platemakers, close to the operatives for the sake of convenience, the atmosphere in which they work is necessarily of an elevated temperature, often 85°. The communication between the two is uninterrupted, a
dooryard alone separates them. In one of these "rooms" of a housetop, the thermometer rose to 120°, in another to 130°, for a third to 148°.

Proceed in the next place to consider the nature of the processes of these nasal passages, which produce such extensive alterations in the texture, such impairment of function of the pulmonary organs, as detailed in the cases given above; or which seem to terminate in the disorganisation and ultimate excavation of the lung tissue itself.

It is not difficult to understand how the acute attacks of Bronchitis invade 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 tory dry atmosphere of the workshop to the cold air of the streets, the low temperature, insufficient 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 failing below the average in vigour and strength of constitution; inventing, as they mostly do from their parents, a decalectic habit, they have the appearance of sickly plants; their vitality is low, offers little or no resistance to the attacks 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
injured, 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, to render the pulmonary organs in particular liable to local lesions. But when this is superimposed on the presence of irritating particles of fine dust throughout the mucous coat of the bronchial ramifications, it is no longer a matter for surprise that disease should assume the Bronchitis form.

It may be objected that the risks 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, for the functions of the lungs and 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 Pottery, every such change has influence, either by disturbing the balance of circulation between the external surface, the membranes, or different glandular structures within the body, or by checking or augmenting the discharge of fermentable 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, whose symptoms have never formed a part of their history. And this is the more remarkable, when
we take into consideration the two-fold nature of the
condition to which these patients have been exposed.
How much of this immunity is due to the influence of
habit, how much to individual idiosyncrasy it is
difficult to determine. Probably they both have acted 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
fissures. There is so to speak a reconciliacion between them.
They it not then be affirmed, that in a similar manner
the bronchial membrane gradually becomes accustomed to
the irritating dust, hence manifests symptoms indicative
of the acute form of inflammation? The two cases, pathologically
considered, are not strictly parallel. 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, 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 earn our ignorance with this base.
It is a conventional phrase too perhaps is convenient,
but it adds nothing whatever to our previous knowledge.
of the subject. It thus appears that, as a rule, persons with a quiet, low, rather languid pulse, whose vitality is somewhat below
par, as it is called, are most liable to chronic diseases; whilst
on the other hand, those in whom the vital processes are carried
on energetically, though full, bounding pulse would seem to
carry health to every part of the body have appeared to be
singularly resistant to the attacks of acute diseases. In these
latter, too, disease extends with greater rapidity, 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
malignant action. The same may be said perhaps with equal
truth of children, with whom acute diseases are notoriously
fatal, 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, in a weak person; but
rather under what aspect will it manifest itself in a
weakly person, in no more weakly? My own observation tends
me to the conclusion, that in the one case the disease is
developed in a sub-acute form, in the other in a chronic
shape. The conviction, that the lower the state of the vital
forces, the greater is the tendency of disease to chronic
development, vice versa, seems to my mind to be almost
inviolable.
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 Potter's it is not usual for an acute or sub-acute attack gradually to pass into the chronic form, even in summer time, 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," 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 (so as they expect, have been "touched in their breathing") by short cough; that lately they have been feeling weaker, the dyspnoea 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 these changes? Independently of the thickening and hypertrophy of the walls of the bronchial tubes arising 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 tubi become narrowed directly respiration is impeded; indirectly by the pressure of infiltrated exudation of matter which sometimes extends beyond the walls of the tubes for reaches on the adjacent lung substance. This diminution of calibre, obstruction may gradually lead to obliteration of the fine bronchi, the collapse of the air vesicles in which they terminate. May more, it is not impossible, that particles of fine dust may find their way into the minute air-cells themselves, induce changes in them similar to those which take place in the air-tubes.

But this inculcation of matter may impede the function of the lung substance in another way; by obliterating the vessels distributed to it, scarring off its supply of blood so that atrophy of the tissues follows; by obstructing the capillary circulation, local congestion is favoured, the blood is imperfectly oxygenated, hence arises distressing dyspnoea, a dusty hue of the face.

Under circumstances such as these it is, I suspect, that the simple emaciation so often occurs. Many are the theories that have been offered in explanation of its mechanism, none of them are 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 vessels, the air-cells communicating with the plugged bronchi (in Bronchitis) during inflation. To this it might be objected, very justly, that at the end of inspiration we have in the healthy chest 131 cubic inches of air, the lungs can take up 11 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!

St. Vincent holds, that when a lung is atrophied from any cause, if the chest expand normally, the residual lung must fill up that the vacuum may be filled. This theory, however, seems to me to be untenable, for in Pneumonia we find that diminished capacity is compensated for by increased frequency of inspiration. In Pneumonia the same phenomenon is observed; yet emphysema in Pneumonia is indubitably a great evil; in Pneumonia 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 both healthy
air resembles if continued for a number of years, for the glottis being closed, the walls of the chest, the diaphragm &c. 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 vessels, the diminished expansion of others, obstruction of the finer branches through the infiltration ofEdition, matter, the strain that is thrown upon the tubular tubes is necessarily so much the greater, whilst the elastic resistance remains the same, obliteration 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 vessels whose parietes are degenerated? Have already shown in what manner the small vessels surrounding the air cells are obliterated; 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 vessels cannot but interfere with the nutrition of the tissue, vitiate 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, concomitantly with this deficiency of nutrition in the air vesicles is the impairment of their elasticity. They become as it were paralysed, offer little or no resistance to the influx of air in excess, whilst at the same time they
contribute but feebly to its expulsion; a large portion of it apparently remaining stagnant, the remainder being slowly expired. When the respiratory process is accelerated, as under unwanted exertion, inspiration and expiration become strangely convulsed, producing the extreme dyspnoea and heaving so characteristic of emphysema: the normal exchange of oxygen and carbonic acid is interfered with, though the oxygenising surface of the diluted cells is increased, the blood is no longer perfectly arterialised. 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, the extensive infiltration of the whole with minute crystallisations.

"Induration matter is endowed to a remarkable degree with the property of slow contraction, a property which renders its presence most beneficial or most offensive: often becoming the seat of saline deposits." (Anatomical Products, Food's Cyclop. of Human Physiology.) It is not difficult to conceive how this exudation material mixed up with dust, slowly contracting gradually hardening, sets up irritation in the surrounding tissues, excites a species of chronic inflammation in them. The systemic disturbance of course is slight or imperceptible on account of the restricted nature of the incision. Those portions which have not submitted
to the further process of softening. Maintain their thickened fibrous nature, are reckoned 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, a condition obtains highly favourable to congestion stagnation. The exudation—matter likewise contributes no small impediment to the free circulation of 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 melanin pigment present in the being fívere striated muscles of even healthy persons, that this has a tendency to increase with advancing age. Amongst the latter, however, advanced age is seldom attained; 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 haematin tends partly to its augmentation. This view receives support from the researches of Mell (Pathological Histology) who says: "It may be presumed, with considerable probability, that the pigment in red blood matter of the blood, which penetrates the cell-wall undergoes various
changes of color within the cell, although, perhaps the
pigment may also arise in a kind of carbonising process
of the protein substance contained in the cell.
In the next place, as the inflammatory process progresses in
Course, the tissues often gradually break down, leaving cavities
in the lung substance filled with the pigmenetary material
of the disintegrated tissues. In some portions this is solid,
or rather negatively firm, in others thickly fluid like black
pease or cream, depending probably on the more or less
advanced stage of the softening process in each case. But
another, perhaps co-extensive source of this pigment is to
be found in the inhalation of CO₂ to which these Potter are
liable in the "stoves" or dining-rooms. The stove itself (i.e.
the furnace) is a very common affair, certainly not tight at
the seams, allowing the free escape of CO₂ in the process of
Combustion. It is supplied abundantly with coal, that in
high temperature may be maintained, CO₂ therefore is
very rapidly generated. The workshops is very imperfectly
ventilated, so that a considerable quantity of CO₂ expired
by the operatives accumulates to again inhaled by them.
May this not be the origin of the "carbonising process of the
protein substance contained in the blood-cells" mentioned
in the quotation from Mill?—
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 connective glands extending along the surface of the pleura. Forchet believes that it is formed by the blood exhaled into the cellular tissue, stating that its chemical composition leads him to that conclusion. Pringsheim says, that it is produced by a mis-direction of the natural pigments of the body, resulting from age, climate, or disease. Andral says, that the black appearances are the result of secretion, that it is more manifest as the individual advances in life. Haeckel agrees with Pringsheim. Lacroix appears to be doubtful as to the real origin of the black matter.

Long ago at 1813 Dr. Pearson (Philos. Trans.) drew attention to the subject in the following words: "It is thought 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, or other inflammable materials in common life. It is admitted the 0. of atmospheric air passes through the pulmonary air vessels or cells into the system of blood vessels, it is not improbable, that through the same channel carbonaceous matter 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 vessels under various circumstances to produce the coloured appearances on the surface of the lungs."
Mr. Graham (Edin. Med. Surg. Journ. Vol. 42.) 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. Surg. Journ. Vol. 62 1846) holds the same view, but includes the inhalation of CO₂ generated in ill-ventilated pits, that expired by the miners themselves. He also 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, in course of time, be conveyed to the interlobular cellular tissue by the process of absorption, 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, also those left after the process of oxygenation of the blood; - 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, or part of, that discem'd infiltrated into the substance of the lungs. If we have the black matter in the lymphatic vessels (which has been done) from the pulmonary organs to the bronchial, mediastinal, thoracic glands, from thence to the thoracic duct, we cannot but admit, that it does
find its way into the venous system, thereby contaminating 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, & 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 Bell relative to the "carbonising process"; they both give confirmation to my view, that the pigment in the canals around the incisions is due to stagnation of the blood in the capillary system, ultimate septicity & decarboxination; the blood having previously undergone the "carbonising process" of Bell, the "contamination" of Dr. Makellar, in the manner already described by us. There is much similarity in some features of Dr. Makellar's case twice, but with the carbonaceous deposits all obstacles cease. (I may remark in passing, that I have never met with a case of Black Pox, as in the counties of N. Staffordshire, probably because the piles are well-ventilated, & the "grey coat" is in common use.)

Lastly, the excretions remain to be considered. I regret very much, that I did not submit these to chemical analysis at the time. Hope, however, to continue my investigation of this subject as 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 unwary by 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 excratory passages by which the exit of a fluid secretion is delayed may indirectly cause the solid constituents to be reduced, the fluid, a certain portion of it being removed by the absorption, or even by evaporation, or by resorption.

Instances of this calcareous deposition are furnished abundantly by the human body, it will readily occur 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 shiny mucous, which gradually hardens, is succeeded by another layer which hardens in its turn, and so on till it accumulates in vast quantities. Calcoli of
the salivary glands are commonly regarded as deposits from the saliva, though according to Breschlin, 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. The explanation offered by St. Weiske 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 salivary material accumulates is by far the most frequently tuberculous, more rarely the fibrous substance of simple inflammatory exudation forms its nucleus. From the analysis of healthy pulmonary mucus by Simon Nafe 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 Polsters we have in addition the constant irritation of an inflamed mucous surface by particles of dust, if therefore, if the hypothesis of St. Weiske be sound, an excessive generation of phosphates must ensue, precipitation of them in the form of deposits take place. Again, the contraction of narrowing of the bronchial tube 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 these concretions in the lungs may have arisen. 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. Lodged in the air cells, inflammation is set up, followed by degeneration, then contraction, where the mass of dust is encapsulated, as it were, by the fibrous material, the vesicle itself is finally obliterated, or occupied by it. In the recorded cases they were all of small size, exceedingly numerous in every part of the lung, similar in character to appearance, offering strong grounds for presuming that they had but one mode of origin.

Again, reasoning from analogy, these concretions might probably be formed by the deposition of phosphate of lime from inhaled mucous membrane upon the particles of dust regarded as foreign bodies, 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; so in the case of a slim stone lodged in the trachea, which became saturated 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. Walsh are quite to this explanation.
Speaking of Phosphate of Lime he says, "So frequent is the occurrence of this salt in calculus masses or mucous surfaces, as to lead inevitably 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 any thing be done in the way of treatment? Probably removal from the workshop, abandonment of the first act of the disease, abandonment of their particular branches of the Potting trade is the only method of averting the further development of the malady. But the remedy rests chiefly with the manufacturers. Many of the Workshops are so badly ventilated that they could not well be worse. Improvement in this respect alone would be followed by corresponding improvement of the general health of the workers. A sufficient supply of moulds in every manufactory would remove the temptation to hasten the dying process by raising the temperature of the dying-rooms. Not the "storos" are the dying-rooms. 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 50 years, notwithstanding thatcountless 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 "storos" 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. Shobart & 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, but I'll recommend itself otherwise. But of 168 
manufacturers in the Staffordshire Potteries not all influenced 
by considerations for the well being 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 often be a work of time to introduce this self-acting 
stove into general use; nothing but its economising, 
character will prevail with most employers, lead them to 
adopt it. Till then Potter's Bronchitis will continue to 
kill in the district, till carry its suffering victims to usual 
to a premature grave.

Feb. 29. 1864.  

Charles Parson.