

**A practical sketch of the Asiatic cholera of 1848 : its rationale and (presumed) pathology / by S.F. Statham.**

**Contributors**

Statham, S. F.  
Royal College of Physicians of Edinburgh

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*With the Author's Compliments,*

A PRACTICAL SKETCH  
OF THE  
ASIATIC CHOLERA OF 1848,  
IN AN ENGLISH VILLAGE.

*[Price Two Shillings.]*

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A  
PRACTICAL SKETCH  
OF THE  
ASIATIC CHOLERA OF 1848,

ITS RATIONALE AND (PRESUMED) PATHOLOGY.

SUPPLEMENTARY REMARKS TO PAMPHLET ON LOW INFLAMMATIONS.

BY S. F. STATHAM,

ASSISTANT-SURGEON, UNIVERSITY COLLEGE HOSPITAL, LONDON.

"AIDE TOI, ET DIEU T'AJDERA."

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## PREFACE.

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INFLAMMATIONS form, perhaps, half the amount of all diseases. In a late pamphlet I called attention to that *Low type* of Inflammation which is possibly nearly as common as the simple form, and which by its severity is far more dangerous. Though exceedingly frequent of occurrence, yet from their not having been generally recognised as a distinct class, these affections have been too often overlooked, and that to such an extent that, though of not much experience in the science of medicine, nevertheless, I feel sure that by the due rank of Low Inflammations being acknowledged, a new field will be opened in medicine, or, at the least, a fresh aspect, and that of a most practical nature both in the pathology and treatment of disease. I can no better express my estimate of the present want of *appreciation* of low inflammatory diseases as compared with those due to sthenic inflammation, than by likening our present neglect to that of sailors, who should take account only of those rocks appearing above the surface, unless when by low tide of the ocean (collapse of the vital powers) those dangers—far more to be dreaded on account of their want of prominence—become only too evident. Sailors, however, are wiser; *we* too often know and name these diseases by the constitutional or local *effects* they induce.

The poisons from the laboratory *may* act dynamically, or otherwise directly, on the system; on the other hand, *as a rule*,

it may be said, that poisons occurring free and active in nature, whether animal or vegetable, or from decomposition, act mediately; in several instances this mediate action *may be* by a change induced in the blood; in several instances this mediate action *is* by exciting a low inflammation; now, as Low Inflammations have not been properly studied, may it not be fairly expected that their elucidation will tend to the comprehension of the action of many of those poisons on the system, which cause diseases acknowledged to be due to poisons taking their origin from natural causes, and which diseases present sufficient analogy to the low inflammations from similar causes met with in daily life? I refer to Epidemic diseases, and hope that their special pathology may receive some light from this opposite and different-coloured side of the shield.

The following remarks on Asiatic Cholera are of the composition of several years ago; they are made public now in the hope that they will prove of use, although a well-known journalist having at the time declined them, it seems bold to expect that four years later a different opinion should be entertained; now, however, claiming the rank of a Type or Class for Low Inflammations, these papers seem to me much more valuable.

Should any one consider that from the above intimation of the opinion here entertained as plausible, of the nature of Cholera, it is useless to read further; it might be fairly put to him whether he would deny the death of a patient having been caused by Erysipelas because after death no inflammatory nor other change of the integument were visible to his senses; or, again, would he deny the death of a patient having been caused by Low Peritonitis when the only pathological change present is some amount of purulent or sero-purulent fluid in the cavity of the peritoneum; or, again, would he in the last and similar cases suppose that there was no local affection, but that the *local* inflammatory product had been secreted from the *general* mass of the blood, yet only in this one spot?

That I could presume to give a different opinion from one whom I am now proud to consider myself a colleague of,—Dr. Parkes, is an evidence of the greatest temerity; it may, however, be said, that he saw the disease in India under very different opportunities from those to be found in a solitary English village. Here nature had established quarantine, each individual was known to the medical man, and various excellent advantages were at hand for investigating the peculiarities of the disease.

Having been otherwise occupied of late years, there cannot be here added any description of the Cholera fungoid-sporules, or investigation of some other points so intimately connected with the *History* of Asiatic Cholera.

With hope, therefore, that Low Inflammations may explain other "Epidemic" maladies, possibly giving some light as regards the pathological cause of Plague, La Grippe, Trousseau-Galante, or other pests,—I will only premise that the poison of these inflammations may be of a more intense form, allowing of its propagation by infection, or less intense and requiring contact, or even inoculation. In certain cases the poison of such a disease may occur in both forms, or in other diseases only in one, and that in either of them.

That poisons from natural causes should not act at once dynamically on the system as the poisons of the laboratory may do, is one of those principles whose practical fulfilment is necessary for the continuance of life on the earth.

That their influence on the system *may be* due to locally excited morbid action is evident; that it *is* so in Asiatic Cholera seems to me the only probable view, and one based on sound pathology.

On entering upon this subject in the autumn of 1848 having no opinion on Cholera, I pretend to say that the conclusions here arrived at are unprejudiced.



In investigating the pathology of Cholera, and some other diseases, it seems to me that writers, frightened at the materialism of Broussais, have fallen into the other extreme, and would yet consider themselves the truer philosophers.

A very great error is certainly committed by not taking *every* circumstance into account in Post-Mortem Examinations, and in neglecting various *niceties* of proceeding, such as examination of the body as soon after death as possible, examination of the intestines before washing them, the doing *everything* one's-self, and for this no slight practice or opportunities are requisite; that the comparison of cases may be made as strictly as possible, there is subjoined here those cases of intestinal disease previously examined by me under the supervision of the physicians; for the description I am answerable.

It were utterly unjust, if *any* merit is to be found in the following pages, but that a large share should be referred to the philosophical and practical mind of John B. Hodgson; to whom I would specially refer as being a strong advocate of the "Propagation by Infection" of Cholera.

It has been said to me by several friends that the former pamphlet on "Low Inflammation" did not contain much novelty; I shall esteem it a great favour to be informed where those descriptions can anywhere be found in print? That the diseases are unknown I do not assert; that they are appreciated is another question.

Oct. 19, 1852.

CERTAIN CASES OF ASIATIC CHOLERA AT CHESHAM,  
IN THE LAST QUARTER OF 1848,

WITH

SOME REMARKS ON THE NATURE OF THAT DISEASE.

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THE data referred to in the following account are; the Parish Medical Book; my own Notes of twenty *Cholera* cases, of which twelve of the fatal ones are given at length, where post-mortem examinations were allowed; the local Registry of Deaths, &c.; the Registry of the Weather of the Rev. Mr. King Latimer's Observatory, with the freest use of which he most handsomely furnished me; and much information furnished by Messrs. Faithorn, Hodgson, & Kitelee, the medical men of the place, most especially by Mr. Hodgson, the parish officer, to whom I was assistant for two months and a half, during which cholera was prevalent.

Chesham, Buckinghamshire, is a small town of 6000 inhabitants, situated at the head of a valley among the Chiltern range of (chalk) hills, to the north-west of, and 28 miles distant from, London. In the character of the town there are a few circumstances of much importance as bearing upon the causation of disease, which may here be noticed.

Many small streams, coursing through the town, unite at its outskirts, and the resulting watercourse being dammed up to supply mills situated upon it, and beds for watercress, becomes broad, always full, but never motionless; hence there are no intermittent diseases. A number of houses of the lower class are situated on its banks, frequently within a few feet of the water, sometimes below its level; many of the better class are built over one of the streams. All the drains of the town open into this stream, at least where they have any

outlet, for in many instances cesspools are made use of and emptied at intervals.

The houses are often badly situated and worse built, and here a great difference is observable. Out of a number for which the same or nearly the same rent is paid, some are airy, dry and clean; these are generally modern houses. Others are below the level of the water, half-buried, choked by the houses around; nearly air-tight, so as to prevent all effluvia from escaping; the walls and floors permeable to, and stained by, the excrements from the neighbouring privies, or the rooms small, or filled with dusty furniture instead of fresh air; the trades (shoe-making, turning, plating) carried on in the same room that they live in, or no trade at all followed, and the house a complete specimen of the results of idleness. Fifty houses in Chesham could at once be named, which on one or other account are not suitable to live in.

Some years since the inhabitants were in a very prosperous condition. This has undergone much change; railroads, by increasing the importance of neighbouring towns, are lessening the trade of this one; the change of fashion and admission of Tuscan straw plat at a lower rate of duty has caused a lucrative employment to become a mere apology for starvation; no provision was made for future adversity, and the consequent poorer nourishment and greater exposure to vicissitudes has had the usual effect in rendering them less able to withstand the attacks of disease. A moderate-sized silk-mill however has been built of late years.

Chesham is generally considered a healthy town; nevertheless its mortality is higher than that of the county, being 2.21 per cent., while that of Buckinghamshire is 2.13 (Mr. Grainger).

Epidemic diseases are not severe; small-pox occasionally, as this year, proves fatal, owing to the neglect of vaccination, and the mortality is certainly increased by scarlatina and measles. Three deaths are referred to typhus fever every year; these were as high as 24 in the years 1844-5. Twenty-three deaths annually are accounted for by strumous diseases out of an average yearly mortality of 136, of which 50 are children dying in infancy.

During the last *quarter* of 1848, 44 persons died of Asiatic cholera, and 2 more in the ensuing month of 1849. Fever, instead of 3, gave 7 deaths; low inflammations, as typhoid peritonitis, &c., 4, where usually 2, and in January 1849 alone, 6; (several of the last-named number being doubtless due to infection). Causes of death annually referred to old age, (fewer by many since the introduction of medical certificates,) to acute inflammations, chronic diseases, accidents and disordered bowels, are all insignificant or uninteresting. (See Appendix.)

It cannot be doubted that many of these deaths annually might be averted by proper precaution. Of seven individuals who had small-pox lately, four had been vaccinated and suffered it in a modified form; of three others unvaccinated, who were all moving in a higher rank of life, the disorder was severe, and in one case fatal. Many of the deaths from fever, strumous diseases, and of infancy, are at once due to the absence of proper sanitary measures. To this is to be added cholera, which, in Chesham at least, has in all (?) instances pointed out those neighbourhoods or houses, those habits or individuals, which actively give rise or passively yield to diseases referable to the want of hygienic measures.

These were entitled in the Parish Medical Book as epidemic cholera, 81 cases. Of these many appear, on after consideration, to have been of a mixed character, and by their recovering rapidly have shown that either the poison was too weak, or the subject too strong, or perhaps the remedies were very successful. On this plea I would only consider 56 of these cases as being sufficiently well marked to allow any medical man, who had seen Asiatic cholera before, to recognise the disease in each of them without reference to other cases. Of these cases, 35 or  $62\frac{1}{2}$  per cent. proved fatal; [if I may compare the systemic affection in cholera to that in low inflammations, the proportion of deaths to the cases of "excitement" would be about 7 per cent., to that of "implication" about 44 per cent., to that of "invasion" 62 per cent.; either one might be given to make out a wished for result. (See remarks at the end.) Compared with mortality elsewhere I expect about 50 per cent. is right.] These 56 cases alone will be referred to or quoted as cases of cholera, and, as might be expected, they include all the fatal cases. (See Appendix.)

Of 56 cases, 26 occurred in the same house, where there had been a case previously; 7 in persons, or in two of the cases in the sons of persons, who were at the time most zealous in their attendance upon cholera patients; 4 cases among those living in the same bad neighbourhood as the cholera patients, and shortly after the occurrence of cholera in that spot. The above seems the simplest way of accounting for 37 out of 56 individuals being attacked; further on, almost all are shown to have had unhealthy habitations, and on looking over their names I find *all* the 37 are predisposed to disease, either by accident of position or by their own state of health, age, or habits. Thus of 56, 37 have sufficient predisposing causes, and to all appearance also, infection was the direct cause of their succumbing.

Of the 19 primary cases, 7 were weakened by age; one, the first case, aged sixty-three, will be referred to again. At this time doubtless

the poison was most powerful; three of seventy years or above, three of fifty-seven, of whom two were of dissolute habits, died. Of the 19 there remains 12; of these, 6 were weak through youth or infancy, and were either in a bad condition from want of necessaries, or had been subject to diarrhœa. Of the remaining 6, two were young girls eight years old, and had been delicately brought up. One, a young woman in poor circumstances, aged twenty-three, had been suckling seventeen months, and lived a few feet from the water, and from infected houses. The fourth, a lad eight years old, I did not see; he was one of the first, and was at school where the mistress had lost two of her own children from cholera. The fifth, also an early case, lived below the level of the water, in a confined house, and was hawking, the day before he was taken ill, among the cholera houses. The sixth, the last of the parish cases, was a lad nine years old; the day before falling ill, he had been on a long walk into the country: his was a well-marked case; he recovered. No other fell ill in the house, and but one case occurred afterwards in the person of an aged and worn-out woman.

From the above it would appear that almost all those who were attacked were, either on one account or another, predisposed to be so, and when these circumstances are specified, the case becomes very strong, that these circumstances alone have much to do with the predisposition to cholera. Probably infection was the exciting cause.

Of the 56 patients, 47 were in unhealthy habitations, of which 26 were in foul situations, 11 confined or damp, 11 close to the water, varying from one to half-a-dozen feet; of the remaining 9, one woman of seventy-three was worn out; a boy of three years was close to a foul house, where the cholera then prevailed, and where typhoid fever and erysipelas had each in previous years carried off their victims; two lads of thirteen and eight were weakly and subject to diarrhœa; in the same houses as the last there had been a case of choleroïd diarrhœa. Another, a man aged fifty-nine, had been a drunkard, and when taken with diarrhœa used for several successive nights to go to the privy, out of doors, in his night-shirt and slippers. Another, a child of eighteen months, was strumous throughout, as seen on P.M.E, especially in the mesenteric glands, and had ulcerated bowels. The eighth, his brother, aged four, fell ill a few days after; he also gives signs of struma; he recovered. The ninth, aged nine, the last which occurred, was not inquired into.

To the above statement it may be added that half of those attacked (indiscriminately old and young) were in want of the common necessaries of life, either through poverty or depravity.

On viewing the subject with reference to the fatal cases occurring

in all Chesham, one only appeared an exception to the remark that cholera is confined to the lower ranks of life (when as now the poison is weak), and that exceptional case can be fully accounted for by other circumstances predisposing the individual—debility after child-birth, and habitation over water.

With reference to the 56 parish cases, I conclude that in 55 of them a predisposition to disease was evident from known causes that depress the health.

It is, however, an easy matter to denounce all the *houses* or neighbourhoods, where cholera prevailed, as foul, or the *persons* as unhealthy; but, if possible, a statement should be given of how many of such houses or persons escaped.

This cannot be done thoroughly; the following is an attempt, and is only an approximation, for much must depend upon the sick houses being more open to observation and more observed. I arranged the 56 cholera cases in a table, marking those whom I consider to have been more likely to succumb to disease than their neighbours, with reference to age, habits (including *wearisome* nursing), habitation, nearness to water, poverty and previous health. (Neighbours here signifies either the family, or neighbours of the patient, or the inhabitants of the town, according as the cholera occurred in an individual, a household, or a cluster of houses.)

Of the 56 cases, I consider 42 to have been, without doubt, more prone to disease than their neighbours under two or more of the heads mentioned above, 12 under one of the heads, and 2 not more prone. Of these one was a woman aged fifty-six, the other the boy aged nine mentioned before, both occurring near the end of the invasion. It may be noticed the great bulk of these numbers are furnished under the heads of unhealthy habitations and habits.

From the above summaries I think I may conclude that at Chesham the occurrence of the cholera in individuals was explicable by ordinary morbid causes affecting their health; so that in these cases there is no necessity for supposing a peculiar liability to the attack of cholera.

It thus appears that certain known causes will in the present instance account for certain individuals being attacked; then the question arises, why were any attacked? The presence of a peculiar poison will be at once acknowledged, and that this was not endemic.

As regards its being epidemic; in 1832, there were no cases of cholera at Chesham; no record was kept, but the fact is acknowledged. At this period it was severe at Aylesbury, thirteen miles distant; in 1848 the disease was severe at Chesham, and at no other place near until full time had elapsed for propagation by infection. The nearest towns are Berkhamstead, Missenden, and Amersham, each about

four miles from Chesham, and equally distant from one another. Mr. Leat, a practitioner at Berkhamstead, was frequently at Chesham during the cholera, and stated each time that Berkhamstead was free from attack. Mr. Smeathman, of Missenden, informs me that, in the last quarter of 1847, 5 cases of looseness of the bowels came under his notice; in 1848, at the same time, 14; Mr. Talent, of Amersham, by his assistant Mr. Jenkins, that in 1846, 13; in 1847, 11; in 1848, 30 cases of looseness of the bowels occurred in the last quarters of each year. Since each of these gentlemen has the care of a parish practice, their returns will bear comparison with that from Chesham, always remembering that the last-named parish is a much larger one. Consequently in 1847, Oct., Nov., and Dec., we find 19 cases; in the corresponding period of 1848, 366; the book being mislaid, I am unable to give the number of cases for 1846, but that that for 1848 is really excessive, is evident by comparing the preceding quarters of the year with the last one; for these the numbers are respectively 16, 29, 49, and 366. No case of cholera occurred in the hamlets (about a dozen in number) around Chesham, and to the best of my recollection, no extraordinary number of cases of diarrhœa, until weeks after the prevalence of these disorders in Chesham. There was no cholera at Wendover, or Aylesbury at the same period. Mr. Jenkins remarks of the cases at Amersham, that many doubtless were trivial, and noticed from fear. Numbers were of a similar character at Chesham, but many such were classed under the head of colic, &c., of which I have not taken account here, and which amounted, in the last quarter of 1848, to 62; in that of 1847, to 5. Real cholera cases occurred later (by about six weeks) in at least four of the surrounding hamlets, and had not occurred earlier.

Hence we conclude, that at the time cholera and diarrhœa were prevalent in Chesham, the surrounding places in every direction were little, if at all, affected; and that therefore the evidence in this instance is against its purely epidemic character, in fact that this was of trivial importance in the propagation of the disease to Chesham.

This will apply, of course, to the weather also, which, although of little consequence in introducing the disorders in question, doubtless encouraged occasional exacerbations or the reverse, and this we should expect to see in the present instance; the less severity of the disease allowing us to estimate the importance of secondary agents.

Comparing October (see tables in Appendix) and November, 1848, with the corresponding months of 1847, we find that, in 1848, there was more moisture in the air, a lower thermometer and barometer; in October a warmer, in November a colder wind, and in November more sudden changes of temperature. This fairly accounts for an

increased number of internal diseases in November; and from the presence of the cholera poison, these were determined to the intestinal canal as diarrhœa: correspondingly, cases called "fever" in dispensary practice (being generally a chill or reaction from it) are much increased in November, and both but slightly so in October; there were, however, many cases of diarrhœa in October, 1848, probably partly accounted for by the air being often fully saturated with moisture, more than in 1847, much more than in September, 1848. In December, there was rather more moisture in the air, the barometer and thermometer rather higher and a southerly wind, the sudden changes rather more often than the year before, so that hardly any excess of diarrhœa, pulmonary complaints, or of "fever," over the former year could be expected; but from the much greater saturation of the air with moisture, and the numerous sudden changes of temperature, in spite of a southerly wind in *both* years, numerous cases were to be expected in *both*.

Thus in December, 1847, many cases of pulmonary complaint and of "fever" occurred. In December, 1848, however, a new agent was present, and this determined the action of the weather to the intestinal canal; hence pulmonary complaints and "fever" were not half the amount of the former year.

In January, 1849, there was about the same large amount of moisture in the air, the barometer rather lower, the thermometer higher; in both years cold winds, especially in 1848; many sudden changes of temperature in 1849. We should expect, therefore, numerous diseases both years, which, in 1848, occurred, as usual, as pulmonary complaints, while in 1849, the cholera was not forgotten; and though numerous pulmonary complaints regained their pre-eminence, with many of "fever," still a large number (but much less than in the two previous months) of cases of diarrhœa applied for relief.

It may fairly be concluded from this, that the weather alone is answerable for a larger number of cases of internal affections in the last quarter of 1848, as compared with 1847, thereby accounting for the somewhat increased number of cases of diarrhœa in the adjoining parishes, Amersham and Missenden, and doing away with all necessity of supposing any choleraic poison to have existed in the atmosphere at that time; indeed, as the "onus probandi" rests with those many and talented writers who assert the poison to be propagated through the atmosphere essentially, as long as we can account in another way for the facts seeming to support the opinion, that assertion must yet remain a mere opinion, at least as regards the propagation of Asiatic cholera in England. During the prevalence of the cholera,



diseases of a low inflammatory type were very prevalent: this will be referred to again.

It now remains to be shown how it was that the cholera spread. I am not able to *prove* its propagation by infection, but yet, having given negative proofs, hope to make out a strong case on the positive side of the question.

I was not at Chesham at the first invasion of cholera, and no notes were then taken of the cases; the first two or three cases which occurred were not, as I hear (from the patient or the medical attendant) of that marked character to enable any medical man to recognise the disease in them; these are not, therefore, included in the list; one of them was, nevertheless, *yeasty* diarrhœa, severe colic and cramp, prostration, but no sickness; the other showed the severe symptoms for too short a time; the one had not left his house, was very subject to constipation, had had "hepatitis" in summer, and lived in a close, choked, and dirty house, and the other had been travelling through a place where cholera prevailed.

The next (first?) case occurred in a filthy spot, and a crowded dissolute household, in an old woman of sixty-three, a widow, who used to travel (hawking) on foot with her married sister, and had been out much both on Tuesday and Wednesday, Thursday evening was taken ill, and died Saturday morning. Her sister was taken ill on the Sunday. The widow used not to go into houses when out hawking; this was done by her sister. They did not, to their knowledge, meet with any infected person or place. After this case, other members of the household rapidly sickened, and then the nurses and neighbours, until, in the course of eight days, five of the more predisposed houses were affected; at the same time, detached cases, apparently by infection, occurred and proved fresh centres in similar unhealthy spots; then a lull occurred, and except numerous cases of diarrhœa, the poison appeared ineffectual; suddenly, however, it broke forth in a fresh dirty cluster of houses, or, should the house have been alone in its insalubrity, it was in several instances confined to this one. The first person attacked was very generally a young or delicate child (the two girls attacked were the most delicate members of the family), and after these the unsound or aged inmates were taken ill.

There are great difficulties in the investigation of this subject: if, as in 1832, the cholera poison is powerful, its action is so rapid as to give no opportunity for observing its propagation; an individual might then infect numbers and be scathless himself; then the poison might be called epidemic. On the other hand, supposing the poison to be weak, as this year, its action is confined to those already

enfeebled, or diseased, or naturally feeble, and numbers escape who, with a sound constitution, may have been much more exposed to it.

Of two nurses whom I attended, who died of cholera, one was an old man of probably seventy, he said sixty; the other, a woman who had but one ordinary-sized effectual kidney, the other was of little use from former (nephritis?). Several other nurses had temporary ailments, but recovered rapidly. One other case is worth particularising; it was the third, a half-starved woman of forty-nine, living in a very confined house, and very poor. She was sent for on Saturday to lay out the first woman who died; she had not been to that part of the town before, but had had diarrhoea for a day or two; on Sunday she stayed in and had medicine for her diarrhoea; was better on Monday and went out in a cold wind, but not near the part affected; Tuesday, was taken with severe algide cholera, and died in twenty-four hours.

Including this one, twelve out of the fifteen first cases were relations or nurses of those first attacked; another, the boy at school near, where the mistress had lost two of her own children; another, the hawker among the infected houses; the last, a boy living a very few doors distant.

After this more scattered cases occurred, either in nurses as secondary cases, or in those more predisposed than their neighbours.

Here I may mention, that the bodies were buried the same day, and generally ventilation, &c., was employed. As regarding incubation of the poison, four of the secondary cases occurred in the same houses, *after* the death of the primary cases; the intervals between the death of the first and sickening of the second were respectively five, one, five and four days, giving presumptive evidence that the period of incubation may be as long as this. The other inhabitants of Chesham avoided the water-side, the part attacked; but the people living there did not avoid mixing, and suffered accordingly.

I have attempted to consider another point, doubtless of much interest, as showing how far the propagation of the disease is favoured by the state of the weather; and have attempted to show this in the Appendix: the saturation of the air with moisture, and great changes of temperature, seem to have the most effect; the proof of its negative influence, by means of the fact that a frost frequently checks the cholera, as on December 20th, was also exemplified; hence we may conclude, that of two parties equally exposed to the disease and equally prone to take it, the opposite states of weather for the time being might render the one obnoxious to attack, and equally protect the other from succumbing.

Supposing the cholera is infectious, whether the dead body can propagate the disease is still *sub judice*; in opening thirteen bodies, and spending two to three hours on each, no ill effects were experienced, nor in any other way; but here there was no predisposition. As regards the number of post-mortem examinations followed by fresh cases, few examples can be quoted; but the proportion was not more than in cases where no examination was made. Nevertheless, if believing cholera to be infectious, we must consider it may be propagated by the dead body, as by any other physical substance, which propagation may or may not be affected by the body being opened.

Many objections to the doctrine of infection may thus be answered and inconsistencies explained, while its sudden cessation, likewise urged against its being infectious, is exactly similar to that observed occasionally in small pox and some other acknowledged infectious diseases.

[As regards the propagation of low inflammatory diseases, it may be said, *infection* does not readily occur from a given case after death, though readily during life. *Propagation by inoculation* readily occurs after death in cases of low inflammation, but not in cases of erysipelatous inflammations, and equally so in life, for it appears to be dependent on the existence or not of low purulent fluid. According to this we should expect Asiatic cholera to be infectious during life, and after death the body to be non-infectious, unless in proportion to its capacity for retaining infection as any other physical substance, in which it would be far excelled by clothes, &c.;—this we also find agrees with experience.]

*Cases of Cholera, of which Notes were taken at the time (abridged).*

I.—A. R., æt. 23, labourer's wife. Often subject to privation. House damp and bleak. Is good-looking and plump in health; of ordinary figure. Has been suckling a child for seventeen months, it is her own; she has been married seven weeks.

*Nov. 25th, 4 A.M.*—Watery purging; 10 A.M., has had five motions; very sick, cramps in the legs, no pains in the belly since being relaxed. Now has constant watery dejection; the motions are water with brownish flakes. No urine since 5 A.M. Countenance is sunk, pale, somewhat livid; the hands pale, shrunk. Pulse scarcely felt. Respirations about 18. Heart sounds quick, soft, natural. The toes are at times black and contracted, but she is feeling better.—Has been taking Mist. Stimulans, and Pil. Cayenne et Hydrarg. Chlorid. every quarter hour between one another. Turpentine epithem and mustard poultices to the abdomen and feet. Was better the same evening.

*Nov. 26th, 5 A.M.*—Has not slept; dozing. At times slight cramp occurred in the abdomen; she then felt cold and looked pale; this was followed by heat and sweating. Surface is warm, she perspires much, face flushed, talks much better. Pulse intermittent, soft. Breath warm. Retches; once sick in the night. Slight cramps in the legs during the night. Bowels constantly, but less open; dejections white, watery. Has made a spoonful of urine.

*3 P.M.*—Gums sore. Pulse 120, regular, soft. Abdomen tender. Otherwise the same, but the symptoms less pronounced.—Has had a quarter of a pint of brandy; beef-tea and gruel; three pills and the draught every half hour.

*27th.*—Perspires. Pasty fur on the tongue. Is sick, and this was bitter last night. Pains in the bowels at times, relieved by warm tile; motions brown, watery, (bloody?). No urine.—Continue medicine, but less often.

*28th.*—On being moved, fainted. Is warm and perspiring. Face looks better, very pale; eyes not so sunk. Gums continue sore. Is as thirsty, somewhat hungry. Passes urine copiously. To-day only two liquid, brown, uniform motions. No cramps, nor pain in head or belly. Pulse soft, full 117. Respiration free. Left breast swollen and hard.

*30th.*—Had yesterday Dec. Cinchon. and Sodæ Carb. Tongue thick, white fur. Is very thirsty. Bowels open four times with pain. Pulse regular, full 108. Sleeps well. Urine copious, high-coloured. Takes arrow-root and brandy. From this time she progressed favourably; occasional diarrhœa, and much weakness and craving for food remaining for some weeks.

*Dec. 7th.*—Began to get up.

This, a decided and threatening case, recovered with very slight consecutive fever.

II.—J. Y., 69, a labourer. Robust-looking, hale old man; grey-headed. Health generally good. Poor. Cottage confined and damp. Is the second attack, the first was last Monday, 9 A.M.; the purging said to have been bilious, except on Wednesday.

*Nov. 26th, Sunday.*—Yesterday, for first day, went out to privy in the garden (weather was wet and cold), had frequent watery purging; to bed at 6½ P.M.; sick at 8½, and continued retching and purging all night; cramps began this morning; the vomiting is yellowish, the stools white, watery.

*9½ A.M.*—Is warmer and skin moist after brandy. Voice weak, stifled; breath warmish; lips rather dark and cold; face shrunk; eyes sunk, and have a dark rim; fingers contracted, tongue pale. Pulse

pretty good. Great tenderness of the epigastrium, especially to the left.—Pil. Hydrarg. Chlorid. et Cayenne, et Haust.  $\frac{1}{4}$  part. horæ, &c.

3 P.M.—Voice slightly improved. Fæces run constantly on retching. Is thirsty and hungry. Breathes as a person in sleep; is sleepy; could not sleep last night. Eyes ghastly, not so dark. Pulse about 60, small. Gums sore.—One pill and three draughts in an hour.

28th.—Much better. Face fuller and flushed. Tongue clean. Had dark motions for first time to-day. Copious urine since 2 A.M. Much pain about descending colon.

29th.—Got up against injunction, was fatigued, purged, and then relapsed.

30th.—Countenance not much altered. No urine, constant watery purging. Retches. No cramps nor cold. Is thirsty. Voice hoarse, husky. No sleep. Pulse jerking, regular.—Acetate of Lead and Opium. Stimulant Mixture. Brandy and sago.

Dec. 1st.—Gradually failing, dark purging.

2nd.—Pallid, cold, drowsy, but can be roused. Eyes sunk; gasping; empty pulse. Dark purging, not sick. Passes some urine. Died 4 P.M.

3rd.—P.M.E., twenty hours after death:—Body wasted, great rigidity.

Peritoneum healthy; omentum, veins injected; mesenteric glands enlarged and white. Small intestines, outer surface congested at free border. Mesenteric veins full. Stomach, contents green, viscous; some punctiform and stellate injection. Duodenum generally brown. Jejunum pale with yellowish mucus. Ilium, brown fæcal matter, lower portion congested, no glands visible. Colon, spots of redness surrounding some glands; fæces thin, brown.

Liver partly adherent to diaphragm; large vessels full of blood. Gall-bladder extends an inch below the liver, and contains dark green bile. Right kidney contains two large cysts, nothing remarkable. Spleen small, flabby, not softened. Pancreas natural. Bladder contracted, inner surface pale.

Pleuræ healthy. Lungs slightly emphysematous, not collapsed, are congested posteriorly; contain frothy serum.

Pericardium healthy. Heart, right side natural, much dark blood and some soft pale clots; left side firmly contracted, some black blood.

This case relapsed twice, and died at last of consequent exhaustion; the symptoms of cholera were disappearing fast, and we should expect the morbid anatomy to be similarly imperfect.

It was the first P.M.E., and a hurried one, although much aided by Dr. Marshall.

III.—J. D., 19, labourer. Stout, hearty, healthy-looking; not very strong.

*Nov. 27th.*—Bowels open in the morning, relaxed and copious; desire to defæcate, without doing it all day long. Poorly in the evening. Had tea and toast and brandy for supper. Slept well from eight to one. Had slept well night previously.

*28th, 1 A.M.*—Taken ill, broke wind frequently upwards. Purging. Gaped and felt sick; on irritating his fauces he began, and then continued vomiting; the first was bitter. Cramps in hands and legs, no numbness nor cold.—Mist. Stimul. Hydrarg. Chlorid. et Cayenne. Mustard poultices and warmth.

*2. P.M.*—Has continued ill. Face sunk and livid, flushes at times; very hollow and dark round the eyes, pupils dilated. Hands shrunk and somewhat cold. Cramps occasionally in the legs; they were dark, but are now better. Has just vomited a half-pint of water; is very thirsty. No appetite. Tongue clean. Fæces frequently pass; defæcation is felt, but there is no power to hinder it; is like rice-water. No urine. Breath warmish, no tightness at the chest; he noticed this last night. Has no pain except under the left ribs; since the vomiting, none under the right. The liver does not extend below the ribs. Heart about 100; none, or but slight radial pulse. No pain in the head, is heavy, and he feels sleepy. Talks pretty well, but thick and somewhat wandering.

*29th.*—Sleepy all day, pallid, bowels open once or twice, dark. No cramps. No urine.

*30th.*—Body warm; no cramps; hands less shrunk. Face not dark, is flushed; eyes less sunk, are dim, conjunctivæ injected, radiating and slight purulent discharge. Pulse 102, soft. Breath warm; slight cough. Gums turgid, sore. Tongue foul, whitish. Little appetite. Not thirsty. Is sick at times; much eructation. Bowels open twice, brown, turbid. No urine. Belly sore on taking food.—Haust. Stimulans omni horâ. Food.

*Dec. 1st.*—Head heavy, very drowsy. No purging, sickness, or vomiting.—Mist. Salinæ.

*2nd, 9 A.M.*—Very sleepy, head hot, eyes injected. No motion, but has made water to a pint. Pulse weak, but regular. Tongue dry and red.—Cold to head. Mustard poultice to the feet. (Ought now to have been bled.)

*3rd.*—Appeared better yesterday evening. Is now stupid. Face flushed, swollen. Pulse quick, jerking. Gums sore. Tongue white, clean. Perspires, no cramps, &c.

*4th.*—Pupils contracted, obstreperous at times. Micturates freely. Abdomen very tender. Teeth clean, dry. Tongue white, furred. Otherwise the same.—Head to be shaved and kept cool.

*5th.*—Eyes injected, commencing abrasion of the corneæ. Pulse

108, weak. Abdomen sunk, very tender on deep pressure. Motions loose and black. Makes water, is cold from struggling, and frequently screaming from pain.

6th.—The same, becoming comatose; urine free and motions more yellow.—Warmth; broth. Dec. Cinchonæ.

7th.—Wandering and obstinate.

8th.—Weaker, otherwise the same. *Vesp.*—Mouth open, gasping, cannot swallow.

11 P.M.—Died.

9th.—P.M.E., eleven hours after death. Weather warm, moist:—Body very rigid. No discoloration. Face pallid, not much sunk.

Head: scalp pale, interior of skull-cap of a uniform lead colour. Dura and pia mater congested. Arachnoid bulging from serum beneath, and of a milky opacity over the whole vertex. Brain congested, much serum at base and in ventricles; substance somewhat less firm than natural.

Chest: both pleuræ have extensive close, delicate, old adhesions. Both lungs much congested with numerous ecchymotic spots, some lobules much firmer and darker than others, but float. Consistence of posterior parts diminished. Pericardium and valves healthy. Right side, much dark grumous blood, and a firmish, fibrinous clot; left side, a little dark blood.

Abdomen: no fluid nor altered condition of peritoneum except close adhesions of right lobe of the liver. Mesenteric vessels much engorged, their peritoneal covering of a milky opacity and thickened for lower two-thirds; the glands enlarged, some few are much so, are pale red or partially whitened; the outer surface of the intestine is congested in portions as the lower third, which was in the pelvis. Thus a few inches of the gut appear dark at intervals, and to these portions the larger mesenteric glands appear to correspond. Large intestine of moderate size. Stomach contracted moderately; it shows some stellate injection, appears roughened, and contains much viscid mucus.

Duodenum, yellowish mucus. Brunner's glands distinct; much ramiform and some stellate injection.

Jejunum chiefly pale, with pale mucus. Parts (hypostatic) have ramiform injection and the mucus reddish; no solitary glands or broken surface.

Ilium appears healthy as far as Peyer's patches, the mucus pale, while the thinner fluid becomes gradually brown; lower the mucus becomes darker, and there is a round worm. The first Peyer's patch is large, and exhibits pits (in place of glands?) and is pale; for the rest of the ilium, the patches are distinct and have numerous

pits surrounded by delicate, uniform, bright injection. The redness is well marked from the larger vessels and their branches to the terminal redness around the glands where the vessels are not distinguishable. In one case it is continued over a gland. The solitary glands are not visible. Quite at lower end is a large patch of Peyer, pale with pale mucus, somewhat raised with numerous glands, each with a dark central spot.

Large intestine is throughout studded with small patches of redness transverse to the axis of the canal, often on the summit of the valvulæ conniventes, and in almost all cases contain one or more white (often bulging) spots, (solitary glands), which appear by comparison to have formed the central spots whence the redness spread. The vascularity is dark, very superficial, of a very fine character, some often appearing in parallel rows to the naked eye. Contained matter appears fæcal, darkish yellow; no true odour, that in the bed is almost black. Mucus is thin and pale.

Liver is of large size, appears loose and pulpy, contains much blood, substance softened. Various roundish deposits (of fat?) appear outside the capsule. Gall-bladder distended with dark green bile. Spleen dark, somewhat congested, but firm. Pancreas firm, white.

Kidneys somewhat coarse and soft in texture, dark edge at junction of cortical with tubular substance quarter of an inch wide. Bladder contains about two ounces clear urine. Mucous membrane pale.

This boy's mother was nurse in a family which had a kind of spurious cholera, rapidly becoming severe typhoid fever, and was likewise much among the cholera patients. During his illness his father and then his mother sickened and died; his death left therefore an empty house. The house was within two feet, and only a few inches above the water. He died of the consecutive fever, and had commencing pneumonia, with congested brain and the viscera softening.

IV.—E. D., 50, father of the last. A healthy, stout labourer. Had diarrhœa five or six days ago; was arrested, but returned yesterday.

*Dec. 1st, 4½ A.M.*—Three hours ago felt poorly, is now purging.

*7 A.M.*—Violent purging and sickness simultaneously, followed immediately by cramps in the legs.—Prescribed for as usual.

*10½ A.M.*—Purging continues; is, and has been rice-water. Severe cramps. Voice not much affected. Eyes much sunk. Face still florid. Respiration coldish, free.

*1 P.M.*—Much altered. Lividity of nose, round mouth and eyes, face much shrunk, hardly any colour, dusky. Voice choleroïd.



Cramps continue, motions passed in bed. Is sick. Pulse scarcely perceptible.

9 P.M.—Clammy, half-cold sweat. Countenance fallen, not so livid. Breath colder. No urine, nor cramps. Otherwise as before.

2nd, 9 A.M.—Cold sweat. Breathing slow, interrupted. Pulse very weak, but regular. No urine, cramps, nor motions. Eyes injected, head oppressed, some cough and expectoration.

9 P.M.—The same, but greater oppression.—Ammonia, &c.

3rd, 7 A.M.—Thought to be dying in the night. Now drowsy. Respiration regular, somewhat laboured. Voice improved. Pulse small, compressible, regular. Tongue whitish, furred. Gums turgid. Copious, very dark, liquid motions. No cramps. Is warm, no pain. Lower half of eye-ball injected, and some discharge.—Stimulants.

1 P.M.—Pallid and livid. Cold. Face collapsed. Eyes much injected. No radial pulse. Warm breath. Gasping with throwing back the head. No purging nor vomiting. Motions are black.

3 P.M.—Died.

P.M.E., twenty-four hours after death. Weather cold:—Body very rigid, pallid. Abdomen green. Pleuræ unaffected.

Lungs distended, and whole posterior portion, four-fifths of each lung, are much congested, exuding on section copious frothy serum; is somewhat friable.

Pericardium unaffected. Heart large, somewhat flabby, left side harder; both sides contain dark semi-fluid blood and soft fibrinous clots. Valves, &c., healthy.

Peritoneum unaffected. Stomach large; contains green, thick, pulpy fluid. Some stellate and punctiform injection, and some mammelation towards pylorus.

Intestines contain a large quantity of more or less thick matter, yellowish in duodenum and jejunum, then orange, becoming shortly of dark brownish green. The inner surface appears at first uniformly red, can be wiped off; the surface is unbroken and pale, with some large vessels loaded with blood. Peyer's patches are not visible. There are some solitary glands, and around a few of these is uniform injection. Colon contains much thick fluid, and has some solitary glands visible. Mesenteric veins are congested, the glands enlarged and pale red.

Liver large, flabby, contains much blood. Biliary matter easily seen in the vessels throughout its substance. Gall-bladder contains about three ounces of dark green, thick bile.

Kidneys appear large. The capsule being removed leaves the surface coarsely granular. The external cortical substance is not more than one-third of an inch thick, but the internal is copious, and the whole coarsely granular and pale.

Bladder contains four ounces of high-coloured clear urine. Pancreas natural, darkish. Spleen flabby, soft, pulpy, bluish pink.

This case of imperfect reaction was probably cut short by great impediment to respiration, in the loaded state of the lungs, which could not fail to produce a similar congested state of the head, as in the last case. Very probably venesection might have prolonged, if not saved his life.

V.—F. D., wife of the last. Tall, apparently healthy, well-proportioned. Often suffered from menorrhagia, from difficulty of passing water, and from great pain in the loins; of the last she had a distinct attack in harvest last. Often subject to constipation, not to diarrhœa. Had no fear of cholera, and had been freely into those already attacked, and had been nursing them until her own boy fell ill. Her house is close to water, not considered damp.

*Dec. 1st.*—Is nursing her husband and son day and night. Felt faint and giddy, and had two sudden motions without pain; feels unwell, looks natural but tired.—Pil Calom. Opii et Cayenne. Mist. Stimulans. To go to bed and get warm.

*2nd.*—Better.

*3rd.*—Weak, sick three times, not purged nor cramped.—To go to bed and take Acetate of Lead and Stimulant Mixture.

*4th.*—Headache, countenance somewhat collapsed and pale. Constant brown purging, has been sick, slight cramps.

*5th.*—Worse last night. Face much collapsed. Eyes hollow and dark. Radial pulse not perceptible. Breath warmish. Sickness frequent and yellowish. Motions like rice-water. No urine.

*6th, 9 A.M.*—Has passed some urine. Is quite sensible; easily roused, but is drowsy. Face much shrunk and pallid, except the forehead, which is blue. Eyes sunk and dark, pupils contracted. Limbs cold, shrunk. Abdomen sunk and tender. Tongue dirty brown and thick fur; is very thirsty, and drinks much brandy, also water. Motions are frequent and serous. Micturates.—Continue taking Lead and Opium.

Continued in this state, for some time struggled involuntarily before death, and an hour previously the catamenia appeared, which she had been expecting at this time; often complained of pain in the abdomen, which appeared in a ball and hard.

*6 P.M.*—Died.

*7th.*—P.M.E., seventeen hours after death. Weather warm and moist:—Body pallid, rigid. Integument much shrunk, superficial veins contain much blood.

Chest: pericardium contains a little serum and slight old lymph.

Structure of heart normal. Right side full of soft, dark, blood; none, or very slight, fibrinous clots. Left side fairly contracted; it, as well as the aorta, contains a little dark blood. Pleuræ, old adhesions.

Lungs are distended and œdematous; posteriorly are much congested, and towards the inferior portions have (in both lungs) hard masses, dark red, exuding on pressure pus-looking fluid, in many different spots, with some little air. The masses are from one to two inches in diameter.

Abdomen: peritoneum has viscous aspect. Mesenteric glands enlarged and reddish. Uterus retroflexed. Liver not below the ribs. Lower two-thirds of stomach contracted. The inner surface is congested, and there is some uniform patchy injection; in parts there are black masses adhering, under which the mucous membrane is destroyed, the edge of the surrounding membrane being of a bright red. Contains green viscous fluid, in which white masses are floating.

Duodenum: Brunner's glands distinct. No solitary glands visible. The mucous membrane has scattered, ill-defined, small spots of redness with ecchymosis in the centre of each. Contains thick white fluid, tinged yellow in the first part.

In the jejunum the same ecchymotic spots occur, gradually ceasing, and none appear in the last part. The solitary glands are very distinctly enlarged throughout; the contained fluid is becoming brown.

Ilium is at first pale for a few feet. It soon becomes dark externally, and for the last four feet is still more so, with a few patches quite black corresponding to Peyer's patches internally.

Internally there are an immense number of enlarged solitary glands, and some papular elevations of whitish pulp; in other places similar pulp is evidently effused in the course of the blood-vessels; it is easily removed by scraping. Some of the solitary glands are surrounded by uniform redness.

Peyer's patches are few in number, are raised and have a flocculent aspect; there is around them redness of a fine uniform character, not on the surface. The glands of the patches have disappeared, their spaces being marked out by the fine injection around them. Towards the lower end the patches are more raised, the spaces for the glands deeper, the redness darker, and a blackish colour seems due to such a colouring matter between the mucous membrane and peritoneal coat. There are several small ulcerations just above the ilio-cæcal valve. The matter adhering to the surface appears thick and whitish; the contained fluid is more or less thick, dark brown or blackish, with white or blackish shreds.

Cæcum: mucous membrane pale; quite in the centre are several small eroded portions, as those in the stomach, but smaller.

Colon generally pale, some slight uniform redness in long patches about the sigmoid flexure. Contents are dark and thin fluid.

Liver: hepatic venous congestion, yet pale; bile-vessels readily visible through the substance. Gall-bladder two-thirds full of dark green bile. Pancreas firm, rather dark. Spleen, firm. Kidneys, right healthy, full size; left, cortical substance of irregular thickness, generally contracted and pale. At its junction with pyramidal portion, are a great number of roundish bodies, half size of peas, firm, gelatinous, as lens of the eye; appear to be contained in the substance of the kidney, with no evident cyst. Bladder contracted.

Uterus retroflexed, old adhesions to the ovaries, which contain numerous cysts. Uterus is larger than usual, the os a wide slit, congested, and slight bloody fluid oozing. It is lined by a thick reddish layer, as of coagulated blood.

This woman died in the cold stage, not succumbing without many efforts for life. Acetate of lead and opium certainly did not stop the diarrhœa, though given freely and regularly. The acetate of lead probably accounts for the corrosions in the stomach and cæcum, and the dark staining of the intestine. The ecchymotic spots are accounted for by the large quantity of brandy which she continued drinking to the very last.

The state of the left kidney is probably accounted for by old nephritis. The retroflexed uterus might have been suspected during life. Circumscribed patches of pneumonia were evident in both lungs.

VI.—H. D., 55, widow. Always dull, stupid-looking, loose-living, improvident woman, generally ailing. Catamenia not appeared for five months.

Her daughter, and now a lodger in the house, have suffered from severe Asiatic cholera, and are now ill.

*Dec. 3rd.*—Was ill all yesterday, after washing up linen through the night previously.

3 A.M.—Three painless, rapid, and copious stools; became cold and experienced cramps.

7 A.M.—Features pallid, sunk, dark, and hollow round the eyes. Breath cold to my cold hand. Hardly any pulse at the wrist; soft and regular at the heart. Is warm throughout (from attention), skin dryish. Cramps in the legs, is not at the present time purged or sick.—Has had counter-irritation. Calomel, Opium, and Cayenne

Pills, and Stimulant Mixture. To continue with Acetate of Lead and Opium, and mustard to soles of feet.

1 P.M.—Blue, dusky. Passes watery fæces, not sick, no pulse, heart's action feeble, breath and body warm; can speak well when roused.—To have brandy, and continue Acetate of Lead and Opium.

3 P.M.—Collapsed and cold. Is hardly conscious, roused with difficulty. No pulse, cannot swallow.

4th, 3 A.M.—Died quietly.

P.M.E., ten hours after death. Weather damp and cold:—Rigidity pretty firm. Is not fairly cold. Some hypostatic congestion. Superficial veins full. Face collapsed, shrunk.

Chest: pericardium contains some serum. Structure of heart normal; left side somewhat contracted; right side contains much dark blood, and some fibrinous clots. Pleuræ, slight old adhesion at right apex. Lungs distended, pale, pits slightly. No tubercle, slight posterior congestion.

Peritoneum unaffected, except short and wide old adhesions of right lobe of the liver. Liver, transverse-colon, and stomach are directed obliquely downwards to the left. Gall-bladder distended an inch below edge of the liver. Mesenteric veins are congested, the glands enlarged.

Intestines: stomach; surface unbroken, there is some congestion, and several dark greenish-brown spots; contains dark greenish flakes floating in about four ounces of fluid. Of the small intestine the upper third appears pale, the remainder congested. On opening the duodenum the surface is pale; Brunner's glands unaffected and thick mucus adhering; similar characters are found in the upper third of the small intestine. For the whole lower two-thirds corresponding to the dark discoloration outside, there is greater distension of vessels and much bright uniform injection not removed by washing, with a few specks of ecchymosis. This injection is partial, especially round the patches of Peyer, affording here a most beautiful object; Peyer's patches being very well developed for the last two or three yards, and their component glands appeared as round white beads surrounded by the delicate bright red injection; around this there is some bright punctiform redness, none stellate. The solitary glands are similar. Towards the termination of the ilium the patches and injection remain, but their glands are wanting, white spaces alone remaining. To the surface of the gut adhere minute white flakes, size of pin-points, far more scattered than villi, and more irregular in shape and direction. These various appearances positively do not exist in the first portion of the gut.

Large intestines: solitary glands are enlarged, and there is some

injection and congestion. The fluid contained in the intestine is thick and viscous, pale towards the lower end, more yellow towards the stomach.

Liver flabby, pale, not friable. Large veins full of dark blood. Gall-bladder contains much bile, and two large gall-stones of cholesterine. Pancreas rather dark. Spleen five inches long, firm. Kidneys normal. Bladder contracted, slight clear urine. Uterus small, firm, no bloody fluid inside. Ovaries small.

This case died in the collapsed or algide stage; she had not suffered from previous diarrhœa, at least to any extent.

VII.—W. S., 2, a fine-looking boy. Had generally good health, but always relaxed bowels, the motions of a light yellow colour; had suffered occasionally from pain in his belly, especially at night. Has had no purging for a fortnight.

The cholera has been within a few houses during the last day or so only.

*Dec. 11th, 3 A.M.*—Awoke with sickness and purging. Is said to have had purging yesterday.

*5½ A.M.*—Is pale, eyes sunk and hollow, not dark. Extremities somewhat cold. Skin of abdomen shrunk rather. Tongue whitish. Retches occasionally. Has frequent watery brown motions, with white shreds floating.—Had the usual remedies.

*9 A.M.*—Died five minutes previously, had sat up, screamed, and been slightly convulsed, the eyes starting.

*11th.—P. M. E.*, six hours after death:—Expression of pain, hands contracted, eyelids will not remain shut. Bones of head of a uniform livid hue, some serum effused at the base of the brain, which is apparently normal.

Chest: pleuræ healthy. Lungs collapse on opening the chest, are pale pink, and have very slight posterior congestion.

Pericardium contains some serum. Heart appears normal, much fluid, black blood in the right side, which coagulates after removal.

Abdomen: peritoneum normal. Liver; acini very distinct, has hepatic venous congestion. Gall-bladder half full. Kidneys, pancreas and spleen firm, and of usual colours. Bladder contains a little clear urine.

Intestines pale, pinkish hue, not congested. Mesenteric glands greatly enlarged, flattened and fatty-looking. Stomach pale, hardly any points of injection; contains thick viscid fluid. Brunner's glands transparent, circumference opaque, quite in the centre a dark spot.

Small intestines: solitary glands throughout are much enlarged. They are raised from the surface by thickening, and in Peyer's

patches the separate glands are prominent, in all the central part depressed. These hollows are not worm-eaten, but have distinctly raised rims; around some of the glands at the lower end of the ilium is slight uniform injection. The surface is however in general pale, the fluid contained whitish and thick, from numerous flakes.

Colon, &c.:—solitary glands are distinctly enlarged and prominent. The surface pale. The fluid like thin gruel, and not at all fæcal.

This boy died in a fit during the collapsed stage. The post-mortem appearance caused by chronic diarrhœa existed also in his brother, who died the next day, and had likewise been subject to this complaint.

VIII.—E. T., 76, is a healthy old woman living in a house close to the water. Had diarrhœa for three months about a year ago, has not had purging lately.

*Dec. 14th.*—Her grand-child died of cholera this morning, and when laying him out at 6 A.M., she spoke of its smelling very bad.

2 P.M.—Frequent bilious purging, not complained of; had been well previously. She went to the closet out of doors; the weather was cold.

5 P.M.—Purging more frequent and copious. Frequent sickness, some cramps; she still went out of doors.—To have Calomel and Opium, Mist. Stimulans, and counter-irritation.

11 P.M.—Much worse, purging continues, but less often.—Has had Calomel 30grs., Opium 5grs. Is to have Acetate of Lead and Opium, &c.

*15th.*—Countenance shrunk; hands shrunk, cold, and dark red; is thirsty; tongue whitish; breath just warm; sickness nearly ceased. Motions now and then watery; white motion with heavy close flakes. Little or no urine. Some cramps in the abdomen.

No reaction occurred, and she died Dec. 16, at 10 A.M.

P.M.E., two hours and a half after death. Weather wet and mild:—Body warm, rigid. Features shrunk, pallid, somewhat lemon-yellow. Some venous discoloration in legs. Head not examined.

Heart; right side loaded with dark blood, nearly fluid, and one pulpy white clot; is not contracted. Left side contracted, contains a little black blood and small pulpy white coagula. Structure of heart healthy.

Aorta contains a good deal of dark blood. Pleuræ and pericardium healthy. Lungs distended and pale, collapse on opening. Chest slightly congested posteriorly.

Abdomen: peritoneum pale, viscous, some parts on ilium seem to be roughened. Mesentery loaded with fat, its vessels are congested,

and its glands often visible and reddish. Calcareous masses in the liver and between the stomach and spleen.

Stomach: all but cardiac end much contracted; contains half a pint of serous fluid with white shreds; thick mucous matter adheres to its inner coat, this is somewhat mammellated. About the œsophagus is punctiform vascularity.

Small intestines have a reddened aspect, and lie chiefly in the pelvis, except the last six inches, and this portion is not red. Colon distended.

Duodenum: Brunner's glands distinct. No solitary glands. Mucous membrane unbroken, and covered with an immense number of fine ecchymotic spots. Contains a large quantity of thick, pus-like fluid.

Jejunum contains a round worm. Ecchymotic spots continue, some large, and whole mucous membrane seems more vascular. Some enlarged solitary glands are just visible. Contents below are thinner, more watery, and larger flakes.

Ilium: the mucous membrane gradually reaches a state of such complete vascularity, as for the last three feet to present an almost uniform state of redness. The surface is unbroken, there is none or very little ecchymosis, the colour is rather dark, and the injection so complete, that no larger vascularity is apparent. The redness is due to an immense number of red points, visible to the naked eye, distinct, and of uniform size. Amidst this are numerous white and enlarged solitary glands, and a few of Peyer's patches, the glands in a similar state surrounded separately by uniform injection. Contained fluid is serous with white flakes; there is some mucus adhering.

Large intestine: cæcum: some dark spots, as if caused by foreign matter adhering.

Colon: various small patches of dark uniform redness, not well marked nor numerous, and apparently round solitary glands, which, however, cannot be themselves recognised. These spots are chiefly in the ascending and descending colon. Contained fluid is serous with white flakes, and commencing to have a blackish tint. Has a slightly fæcal odour. No fæces.

Liver full of dark blood, yet shrunk, as if it previously had been fuller. Hepatic venous congestion. Substance fragile and dark. Gall-bladder full of dark green thick bile, and a few soft and dark gall-stones. Pancreas rather dark; its duct full of limpid fluid. Spleen dark, firm.

Kidneys below full size. Capsule takes up some of the substance; cortical substance not more than one-eighth of an inch thick, is somewhat granular. One or two cysts on the surface.



Bladder contains about two ounces of clear urine, which is albuminous.

Uterus flaccid. Os small and clear mucus exuding. Its inner surface has numerous ecchymotic spots. Ovaries quite withered.

The bed is soaked with fluid, which is colourless, and has an urinous odour.

This, as the next, was another case dying without passing the collapsed stage. Her age, diseased kidneys, poor circumstances, habitation close to the water, and exposure to the disease, rendered it almost impossible for her to recover.

IX.—W. H., 65. Healthy-looking, grey-headed, aged man. Acting as nurse for some weeks, and is sleeping in the same house as another cholera patient. Often has had pain in his right side. Said to have had fits when young.

*Dec. 20th.*—7 A.M. Bowels open suddenly; yet looked well.

3 P.M.—Was purged again; in an hour had sickness, the looseness continued, and 7 P.M. accompanied with cramps.

The motions continued serous, dribbling in bed, some retching, but no sickness after 9 P.M. Had the usual remedies, but continued cold, collapsed, was just sensible, and complaining much of his side and back; spoke no more after

*Dec. 21st,* 6 A.M.,—but felt the turpentine.

2 P.M.—Died quietly.

P.M.E., twenty-one hours after death. Hard frost:—Body emaciated and rigid, not discoloured. Eyes very hollow, lower half of sclerotic exhibits radiating net-work of large vessels. Cornea unaffected. Scalp and skull-cap pale. Brain appears normal, with slight congestion and serum in the ventricles and at the base.

Chest: lungs distended and do not collapse. Pleuræ; slight old adhesion, no serum.

Lungs: posterior portion of lower lobes congested, all parts crepitant, many pit, none sink. Large vessels gorged. Bronchi are of dull red, and contain much thick mucus.

Pericardium unaffected, except one white patch. Heart; structure healthy, except osseous deposit in upper part of septum ventriculorum, and foramen ovale has a small opening, size of a probe. Right side full of dark blood with a slight fibrinous clot. Left side contracted with a little dark blood; of this the aorta contains a good deal.

Peritoneum normal.

Intestines generally of a brighter red than usual. Stomach moderately contracted. Duodenum, instead of its usual turn, makes a complete sigmoid flexure, and to its lower edge is connected the

transverse colon which follows a similar course. That part of intestine in the pelvis does not appear darker. Mesenteric vessels congested, the glands just perceptible in some parts, are either pink or white.

Stomach contains much gas and some dirty white fluid. Interior pale, except around the œsophagus, where are numerous vascular points and some minute stellate injection. Œsophagus pale.

Small intestines pale, and contain dirty and flocculent fluid, of yellowish cast in the duodenum. Brunner's glands distinct. Solitary glands are few in number. Peyer's very numerous and of good size, are raised from the surface, and have a kind of flocculent aspect; the component glands are not recognised.

Large intestine pale; contains turbid dirty-white fluid with flakes. The solitary glands are hardly visible. There is bright uniform vascularity in one part of the sigmoid flexure for about one inch and a half.

Liver is somewhat pale and rough, as if deposit and partial contraction had occurred; substance firm. Bile and blood-vessels full. Gall-bladder nearly full of thick dark-green bile. Pancreas firm, pale. Spleen firm and not dark.

Kidneys of good size; the cortical substance a quarter of an inch thick.

Bladder contracted; contains a little turbid urine.

In this case the pain in the side was very probably connected with the unusual disposition of the intestine. Every care was taken of this man, but from the first the collapse was profound, and no hope entertained.

X.—J. W., 38, sawyer. Married; stout and strong, pale from drinking, is in tolerable circumstances, but lives in a close house. Last year was subject to fits (epileptic?), and since then has often felt faint, and perspired profusely when at hard work. His daughter was taken ill of cholera thirteen days ago, and died in seven hours and a half; just before this he had diarrhœa for a short time. He is not known to be subject to diarrhœa. Two days ago his bowels were opened three times rapidly; he went to work nevertheless, but feeling faint and giddy was obliged to return.—He had Acetate of Lead and Opium Pills, and became better. Yesterday felt pretty well.

*Nov. 30th, 2 A.M.*—His son being very ill with cholera, he got out of bed two or three times to help him. Bowels open twice; brown, fœcal, and very watery. He soon became hoarse.

*5½ A.M.*—Rapid serous motion, lost voice, sunk countenance, anxious and despairing.

8 A.M.—Sickness, followed after some time by cramps. The eyes became injected. Much pain in the back and belly. The cramps and serous purging continued; the sickness ceased and sinking followed with death at 4 P.M.

At 2 A.M. his motions were just such as two days before, so that with rest and warmth he took two grains of Acetate of Lead, and a quarter of a grain of Opium every hour. If treating him again, I would apply turpentine, and mercurialise him as quickly as possible; this was done at 5½ A.M., but then at least without effect.

P.M.E., twenty hours after death. Weather warm, moist:—Integument shrunk and pale, hands and feet livid. Eyes half opened, and the exposed portion of the sclerotic of a blackish colour, the cornea slightly flaccid in the corresponding portions.

Head and chest not examined.

Abdomen: peritoneum unaffected. Intestines of a fresh colour. Colon contracted, except at its commencement. Mesentery loaded with fat, its vessels congested, the glands readily felt and occasionally seen of a larger size than ordinary. Stomach stained brown, slight coarse injection of same colour; is distended with air and brown fluid.

Duodenum: surface pale, unbroken. Brunner's glands not enlarged. Viscid mucus adhering to the coats; contains thin white fluid.

Ilium, last six feet, (the rest not examined): mucous membrane pale, no uniform injection; beneath are many large vessels anastomosing. Numerous solitary glands, white and acuminate, stand prominent from the surface. Peyer's glands are prominent, and present a regularly uneven surface, as if caused by the enlargement and mutual compression of the glands composing them; the surfaces of these separate glands appear granular, as if covered by very minute beads, and are clear white. Peyer's patches are numerous. Much thick mucus adheres to the surface. The contained fluid is thin, serous, with pulpy flakes.

Large intestines: cæcum presents slight uniform injection, size of half-a-crown apparently, around several central points. The mucous membrane of the colon is in general pale, but there are numerous little spots, transverse to the intestine, of partial injection, having in their centres white spots, some few projecting, but the most part level with the intestine. The contained fluid is white, opaque and thickish, with many white flakes. (A portion of gut, which had been tied, showed, on the inner surface corresponding to the ligature, a number of specks, just such as were called ecchymotic spots in a previous post-mortem.)

Liver pale and doughy. Gall-bladder full of bile. Kidneys full size; cortical substance coarse. Bladder contracted.

Other parts not examined.

This post-mortem was conducted in a great hurry, in the coffin.

I may here compare the state of the intestines in two patients examined the same day, February 6, in the University College Hospital. Both about the same age, both men. The one under Dr. Williams had died of cholera without reaction, having lingered four or five days. The other, under Dr. Walshe, had been subject to delirium tremens, and now died in the collapse after acute head-symptoms.

In the first the other organs were in the usual state, as observed in cholera; the intestines pale, the stomach containing greenish fluid, the small intestines lined with mucus becoming green, and then faecal in the ilium. In the ilium the solitary glands enlarged, white; Peyer's patches raised, opalescent and flocculent; the separate glands hardly distinguishable. Colon contains copious, viscid, brown faeces.

In the other there were some signs of inflammatory action in the brain. Less bile in the gall-bladder, a large quantity of urine. Brunner's glands more developed than usual; in various parts of the small intestine is racemose becoming uniform injection. Peyer's patches well marked, not raised, but they are well seen, being dark, especially in the central spots, and the intervals light; are not flocculent, their component glands are not visible. The solitary glands cannot be seen. In the large intestine the solitary glands are much enlarged, and there are some patches of redness.

The above is principally useful for showing the different state of Peyer's patches; in the one new matter has been added, in the other there is no departure from the normal state.

XI.—A. B., a lad. After three days of algide cholera, lingered for nine days in consecutive fever, and then sank. Was a strumous child, but not subject to diarrhoea.

*Dec. 5th.*—P.M.E., twenty hours after death. Weather, fine, dry:—Head not examined.

Circumscribed pneumonia in the lungs, on the right oozing pus on section. Bronchial tubes full of opaque mucus. Gall-bladder full. So also the urinary bladder. Other organs healthy.

Intestines are not in their usual direction. The mesenteric glands much enlarged, are pale red; there are six or seven intussusceptions in the small intestine, chiefly at its commencement; one of them includes eight inches of gut. For last three feet of the ilium the solitary glands and those of Peyer's patches are hollowed and injected around. Much thick mucus adheres to the coats.

Large intestines are full of fæces, and show numerous solitary glands, generally with a dark central spot.

XII.—E. T., 1½ year. A healthy-looking child, living in a confined house near where the cholera is prevalent. Has been weaned four months; not subject to diarrhœa. Had mucous diarrhœa for ten days, which was stopped by Dover's powder four days ago.

*Dec. 4th.*—Taken ill with algide cholera; continued till the 7th, when purging stopped, and shortly after she had a bilious motion.

*11th.*—Died exhausted, 2 A.M.

P.M.E., eleven hours after death:—Body rigid, shrunk and pallid. Chest normal. Peritoneum healthy. Solid organs of abdomen normal. Gall-bladder half full. Urinary bladder full of clear urine. Mesenteric glands enlarged, red, and firm; the mesenteric vessels congested.

Small intestines: Brunner's glands are similar to enlarged Peyer's glands with central dark part. Solitary and Peyer's patches are few in number and enlarged; those in Peyer's patches are hollow, with undermined and irregular edges. The surface pale, with viscid mucus adhering.

Great intestines: cæcum and colon exhibit various patches, of all sizes up to that of half-a-crown, of bright uniform injection; and almost all these patches contain in their centre one or more solitary glands. Corresponding with this the patches are often elongated, and are transverse to the direction of the intestine. There is no arborescent or dark injection. The glands are very numerous, a few prominent, far the greater number hollow, and the ulcerative process appears, as it were, in every stage; the raised edges of the gland become absent gradually after a central hollow has appeared; first a simple hollow, then the edges irregular, as if worm-eaten; and in the sigmoid flexure the ulcers have run together, and in many little patches the mucous membrane is entirely destroyed; this, surrounded by vivid injection, is very striking. Contents are of fæcal colour and consistence, the outer portion being much softer, as if mixed with much mucus or other liquid.

Many of these cases were neglected in their attendance, principally by food not being properly administered; again, no house was provided for the reception of cholera patients, for fear lest it should be avoided by tenants in future.

All the Post-Mortem Examinations and the Notes of Cases must not be expected to be complete; as, although spending two to three

hours in making the post-mortem, being by myself, I was obliged to write from memory in the evening, aided by such specimens as were carried home. The great increase of parish practice at the same time is a still better excuse.

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## PATHOLOGY, &c.

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THERE now remains to be considered the pathology of the disease, whether it is referable to mysterious operations before unknown, introducing changes in the animal fluids of an extraordinary character, or whether it can be classified with diseases concerning which much progress has been made in ascertaining their true nature.

Many of the symptoms of the disease are but accidental phænomena, and such as may and do occur in the progress of other maladies, and are merely necessary results of the implication or impairment of one or more organs or systems. Thus the sickness and cramps from intestinal irritation, transferred by reflex action from one to the other system of nerves, are nothing but the common occurrences of every-day life; so also the sunk visage and chilled body. But, if we inquire further, we find there is one symptom pathognomonic of Asiatic cholera, which, by its constancy, is proved to be essential, and by the date of its occurrence appears to lead us the nearest to recognising the cause of the disease. This symptom is the escape of a flocculent or shreddy matter into, and generally from the intestinal canal, and always accompanied by a greater or less quantity of colourless alkaline fluid. A similar discharge never occurs in adults in any other disease, and probably this applies also to children.

As the cause of such a flux, inflammatory action affords at once a plausible explanation, and one which requires no far-fetched similes or forced hypotheses.

We are already acquainted with inflammatory actions in the body, of an analogous rapidity of course, and depressing effect on the system, such as erysipelas, cellulitis, or diffuse cellular infiltration, pyæmia, phlebitis, inflammation of the lymphatics, and some other, of which the most remarkable is purulent peritonitis; this class of diseases, of which the difficulty of recognition during life can only be equalled by that of treating them effectually, are liable in many instances to be overlooked even after death; indeed the only sign of their having existed is (as a general rule) a small quantity of purulent

fluid, and, should this have escaped from the body, there would, in most cases, be no positive sign remaining of their having existed.

These typhoid inflammations are doubtless due to some peculiar poison, whether self-generating under certain circumstances is uncertain, but is, without doubt, infectious. Authors do not refer to this poison as the cause of the symptoms, and fairly not so, for the poison acts mediately. Thus, I consider that the symptoms in Asiatic cholera should not be referred to the poison, but to that state which there is sufficient evidence to show it induces in the body, and which is essential to the production of the symptoms.

There is one other class of maladies with which it should be compared, that is, where a cognizable poison acts dynamically on the frame, such as prussic acid and many others. That the poison of cholera does not act thus is disproved by the constant occurrence of the characteristic protein and serous product, and the connection of the symptoms with this effusion; (cases, indeed, are mentioned by authors where there was no purging, but in all such cases the flaky fluid was found in the intestines after death).

Seeing, therefore, that the protein effusion occurs in every case, the next point for consideration is at what time it occurs. We cannot suppose the purging to be synchronous with the excretion of the fluid into the intestinal canal; but since in the first instance it is always in large quantity, we are convinced it is the result of an action already existing for a longer or shorter period, and then when the quantity is too large to be retained, it is discharged. Again, the first motions are very generally watery, and, though bilious, are in all probability mixed with the same fluid as occurs separately later in the disease. I am not able to give the result of special observation on the time of occurrence of watery diarrhœa, with reference to the stage of collapse, but am convinced, as far as my memory will serve, that watery diarrhœa was always a previous symptom, and in most cases was present to a large extent, just before the invasion of algide collapse. That it occurs so short a time previously is an additional and valuable argument for its necessary connection with the symptoms, although—from the purging, a mere accident, being confounded with the effusion into the canal of the fluid, and the colourless fluid alone being considered choleraic—the time of effusion, which must have been previous to these, is overlooked, and the collapse and effusion are both referred to the same cholera poison, while in fact the effusion was prior to, and the cause of the symptoms of collapse.

I must, however, qualify this last statement, as we must believe there are at least four agents concerned in the production of the algide depression. First, the cholera poison itself, whose influence

is demonstrated by other diseases simultaneously taking a lower type: this direct effect would at once render any action set up in the body less sthenic. Secondly, the state of the patients, who, at least at Chesham, were more inclined to succumb than their neighbours. Thirdly, the loss of so much fluid from the blood, which may be compared to venesection in a weakly subject. And lastly, as this is probably due to inflammatory action of a low form, we should expect by analogy that the specific action, especially in such a part of the body and to such an extent, would itself greatly depress the vital powers.

If the cholera poison may be strong so as to become epidemic, and to attack those apparently healthy, it is possible a peculiar predisposition may come into play in rendering certain individuals prone to the disease; this would increase the depression.

It will be well now to notice how far the symptoms are explained by the theory of inflammation, accompanied with the loss of so much fluid. Why so much fluid should escape will be considered hereafter; the fact merely is appealed to now.

Dr. Parkes was able, in one instance, to measure the whole quantity of fluid which was passed; it amounted to eighty-eight ounces, or four and a-half pints: this, probably, does not include the fluid in the prior watery bilious stools; there were also no more than seven stools, some very small in quantity. I find that an ordinary chamber pot contains five pints: in the intestines, after death, there is generally about a pint at the very least. I should suppose by rough observation that six pints of fluid at the least are effused into the intestinal canal of every adult, when ill of cholera, and that in the first hour or two of the algide collapse, probably about two pints in every case, at the commencement of, and just prior to the first signs of cholera (such as the sunk eye). Now let us compare such a loss, with venesection or hæmorrhage, to only one pint in a weakly subject, remembering that in the choleraic patient other depressing agents are acting simultaneously, and we shall not fail to see good reason for the collapsed, pale and cold surface, the sunken eye, and failure of strength. The suddenness with which the severe symptoms set in favours the doctrine of an inflammation which, latent for a while, suddenly spreads, and being on a highly vascular mucous surface, constantly and copiously supplied with blood, and not being exposed to the cooling external air, but, as it were, fomented by the liquid effused or by the neighbouring parts of the canal, evidences its presence by a large quantity of fluid immediately derived from the blood: the fluid which flows thus freely is a means of relieving the vessels, and hence pain or tenderness is not complained of; but the inflammatory action is the cause of cramps, both general and intestinal, hence defæcation and sickness, by the irritation propagated



through the nervous centres. By this time another cause of symptoms is fully operating.

Though the choleraic fluid escapes gradually, it is almost always observed that a large quantity is lost at the onset, and this may fairly be considered, as in most cases, from two to four pints within the first quarter or half hour. Now it must be remembered that this is not a portion of the blood, but merely its fluid. The quantity of blood in an ordinary individual is twenty-eight pounds or fifteen pints, so that here is a loss at once of  $\frac{1}{8}$  to  $\frac{1}{4}$  of the bulk of the blood, a consequent contraction of the vessels, and greater spissitude of the blood with consequent far greater difficulty of circulation: this state of the blood will at once induce absorption from all the surrounding textures; hence they shrink, but the blood is not restored to its former state, for the diseased action is continuing. The blood being now much thicker than previously, and less saline, loses a great part of its powers of oxygenating; the air cannot act upon it as previously, from the blood corpuscles being so much closer together, and consequently not so well exposed: there is less water to dissolve the air, and the absence of saline matter materially retards oxygenation; hence the blood is more venous, as in asphyxia, and as in asphyxia, partial hindrance results from spasmodic action of the capillaries of the lungs or other causes. As in asphyxia, still some circulation, and this of partially black blood, takes place; now circulation, respiration, and consequently the production of animal heat, and all the cerebral and animal functions are impaired, not abolished, for circulation of arterial blood is replaced by that of a more venous character.

This alteration of the blood may possibly at once account for the change observed in it, without requiring the action of any other than ordinary morbid causes. The sodden hands and feet is due to these parts being more congested from the cramped muscles and shrunk skin hindering the return of blood. The injected conjunctiva was, in many cases, I found, merely that portion which was left exposed by the lower eyelid remaining constantly unraised, as if, as in the case of the voice and subsequently of deglutition, the venous circulation had so far impaired the voluntary and reflex actions, that extra-stimulus was wanting, and perhaps the muscles could not act readily, from not being sufficiently moist. Consequently, the conjunctivæ, being exposed, become dry, and the vessels, after contracting, relax and remain dilated; the circulation is impaired, and the portion of the cornea drawing its nourishment from them sloughs; on reaction, inflammation is as likely to be set up, as in the lungs, intestines, and brain.

Venous circulation cannot support human life. This, therefore,

fails; and since partial circulation is continued, life fails gradually, unless, as in children, convulsions may finish the scene (as in two at Chesham): this is readily explained by cerebral congestion, which is found after death.

The suppression of the secretions is a natural consequence of the blood being no longer able to afford the necessary materials. The altered respiration from cramped muscles and altered cerebral functions, pain and oppression at the epigastrium from cramps of the diaphragm, and perhaps from violent action of the stomach in vomiting. The thirst we might expect; the constant desire for *cold* drinks is an additional argument for inflammation.

The secondary fever we might expect from the altered state of the blood and the various obstructive maladies which must now be so common.

Other symptoms are explicable in the same manner.

*Post-Mortem Appearances.*—On referring to the Post-Mortem Appearances we find increased action, and this in all probability inflammatory, of the intestinal mucous membrane, a corresponding fluid in the canal, as might be expected an altered state of the blood, with venous congestion; consequently thereupon checked secretions, but otherwise a normal state of the body. Hence the integuments are shrunk, and, in particular parts, congested and sodden; rigidity of the body is great and long-enduring from the comparative health of the deceased and rapid course of the disease (thus, in bringing together the integuments after examining the body, I frequently found it necessary to divide the pectoral muscles to allow apposition); there is congestion of the brain, as evidenced by the loaded veins; serous effusion, and occasionally stained bones of the skull, which, as the loaded state of the roots of the lungs and of the right side of the heart, is due to the difficulty of viscid and dark blood passing through the capillaries of the lungs; hence, there is frequently œdema of the lungs, congestion in the large vessels, and some hypostatic congestion; the lungs collapse, showing there is no obstruction to the entrance of air; the bronchi are frequently reddened, and contain some mucus, from the congestion and want of volition to cough up the resulting accumulation of mucus.

The left side of the heart is contracted, agreeing with the state of the muscular system, and both it and the aorta constantly contain a portion of dark blood, showing the circulation of venous blood in the arteries.

The liver is congested from the pulmonary obstruction, and its biliary vessels loaded, showing that the secretion of bile was not stopped, although it is probable that, as in the kidney, its diluent

fluid is wanting; the gall-bladder is always full from secretion continuing, and is not empty probably from want of ordinary stimulus to excite its contraction; the minute structure of the liver is not congested, it is pale, it may be soft from serous effusion, but its capillaries are not loaded—hence, showing its state is that of congestion, not of determination; the size of the liver is not increased, showing that the state of the liver is merely due to *passive* accumulation of blood from venous obstruction beyond it, and that partial and temporary, and is not due to any increased or exaggerated *action* in the intimate structure of the liver.

The pancreas is firm, perhaps darker from congestion, and its duct (sometimes) full. The spleen firm and small, showing there is merely some passive venous obstruction; it is never pulpy or softened in the algide state, as in cases of fever, &c., where the blood is diseased ultimately, if not primarily; but, as we should expect, the spleen is thus altered after reaction sets in.

The kidneys are merely congested in their large vessels, as the liver, with perhaps some serous oozing, and, it is said, frequently contain thick fluid in their pelves, as if secretion failed from want of material, not from want of power. The bladder is empty, contracting with the same stimulus that cramps the muscles and empties the rectum; the gall-bladder is, we know, not under the dominion of the will, but its action, there is every reason to suppose, is closely connected with the presence of food; hence now it remains full. The peritoneum is merely a little dry, from the absence of fluid in the blood. The mesenteric veins, as the hepatic in the centre of the lobules of the liver, are passively filled with blood; the mesenteric glands are always more or less enlarged, not white, as in chronic cases, not dark, as in congestion, sufficiently extensive or continued, but pink and firm, the result of increased capillary action.

The stomach generally contains some fluid which, still regurgitating, the dying person had not strength to vomit; its coats are not altered.

(Here I must mention that, having no experience in trying the usual or altered strength of the mucous membranes of the intestines, I did not attempt to employ this test.) The small intestines contain the fluid, tinged with bile in the duodenum; it appears to be the water and salts of the blood, and its deposit to be albumen of the serum with fibrin, and some mucus. If opened sufficiently early after death, I believe uniform bright injection will always be found, a proof of inflammation. The glands, Peyer's and the solitary, are always enlarged, proving previous increased capillary action; this could not result from congestion; if life is protracted, the enlarged glands burst—and if solitary, disappear; but if aggregated, a simul-

taneous deposit (of fibrin?) takes place between the glands (in the cellular tissue of Peyer's patches); hence the patches become raised, opalescent (and flocculent on the surface), often with bright injection, and later with pits in place of glands; further than this, changes do not occur, for by now, death, secondary fever, or recovery is induced, or result on subsidence of the diseased state of the intestines.

The state of the great intestine is similar; its solitary glands are found like *those of the small intestines, as self-registering thermometers, evidences of the increased action which has been going on.* \*

I may mention here that these changes, though trifling, acquire importance from their constancy, and from not occurring in health; of twenty-five cases, in former post-mortems in which I have examined the intestines, the solitary glands of the small intestine have been enlarged in but three cases, and in all of these diarrhoea had been present during life; there was redness around the spaces for the glands in the large intestine in three or four cases, but in all of these constipation existed to a great extent. I have never seen a similar uniform injection around the glands of Peyer, or so extensive in the mucous membrane. In those cases under Dr. Williams, at University College Hospital, there has been frequently some redness where there had been diarrhoea previously, and this at very variable times after death, from fifteen to thirty hours; but in all of these it was very slight, and in several at the same time after death where it would have been expected there was none. In three cases of erysipelas the redness had entirely disappeared in four, twelve, and fourteen hours; similarly, I believe that the intestinal redness in cholera disappears after death, unless it had previously disappeared by the cessation of inflammation.

Of the Algide Cases.—In the first case (J. Y.), examined twenty hours after death, both causes came into play; and, as most of the solitary glands had probably burst, and the examination was hastily made, but little evidence was found; in two other men, examined twenty-one hours after death, there was no redness, but the other signs of inflammatory action; while in three examined at seventeen, ten, and two-and-a-half hours after death, there was more or less extensive injection about in proportion to the time which had elapsed; all these six dying in the collapsed stage. (See Appendix.)

The redness is probably more permanent than in erysipelas, on account of the increased spissitude of the blood, the portal circulation, and the venous congestion from difficulty of passing through the lungs; the lessened bulk of the blood would not give less redness from the great quantity of it present in internal organs. That the disappearance of the capillary redness is so complete is probably due

to death occurring with the play of the various functions of the system suddenly stopped from want of arterial blood, not stopped by wearing out or destruction. Similarly the rigidity of the muscular system is extensive, and the arterial contraction also being probably more active, renders the disappearance of inflammatory redness more complete. Hence life is long maintained by the circulation, not of polluted, but merely of less suitable blood. In one case (J. D.), an aged woman in which previous pulmonary disease had habituated her to a less complete oxygenation of blood, and in whom I am convinced there was a discharge of upwards of ten pints of choleraic stools alone, life was carried on for six hours; she was during this time comatose, *lying on the side*, unable to swallow, the face livid and shrunk, the hands cold, clammy, and purple, the *exposed* portion of the conjunctivæ dry, shrunk, and black, the corresponding portion of the corneæ shrunk and opalescent. This state was certainly not produced by opium. The respiration was 16 in a minute, and harsh, and the ribs moved to the bottom of the chest. Some other similar cases occurred at the end of the epidemic. In this woman there was a remarkable return of temperature before death. Her state reminded one much of that of a cold-blooded animal.

That cholera is an asphyxia pestilenta, I can see no proof of; the slight disturbance of the system, as faintness, uneasiness at the epigastrium, &c., which are commonly supposed to indicate the first reception of the poison, is very probably correct; but certainly in all the cases of cholera at Chesham, all the symptoms appeared to depend upon the intestinal disturbance, and those of the pulmonary organs among the rest.

That it is an altered state of the blood seems nearer the truth, but here the effect is considered the cause. Experiments of injection of saline fluids proves that many (I would say, all) the pressing symptoms are due to an already cognizable altered state of the blood, a want of material, and as readily capable of relief as common asphyxia by the introduction of air; but the hurtful state is as soon reinduced, just as that in recovered asphyxia, by the fresh withdrawal of the nutrient fluid.

One other point should be noticed, that when the discharge from the intestines is small, it must be remembered that there must have been a previous considerable escape of fluid to wash away all fæcal matter and odour, that a large quantity of fluid is found in the intestines, and that by the violent action of the poison and the seat of the inflammation, the failure of nervous energy—by the withdrawal of a large quantity of blood to the intestines, the collapse of the surface—is accounted for.

That it cannot be an inflammation of the intestines, because inflammation of these parts occurs without giving rise to the same symptoms (*Library of Pract. Med.*), would apply equally to all specific inflammations; that the appearances after death do not accord with the ordinary results of inflammation (as stated by the same author, Dr. George Budd) is the fact. But the disease is not simply inflammation of the coats of the intestinal canal, but one of a specific and peculiarly rapid nature, having great analogy in its course to purulent peritonitis. In cholera, the great vascularity and disposition on a free surface causes no pain, a copious supply of serum, and does not afford time for the formation of pus; in purulent peritonitis, the scanty vascularity insures a fully developed inflammation when it does occur, hence fibrin or pus is at once effused; the concurring circumstances, as hospital air, insure the effused fluid being pus, the want of space rendering the agony severe, and the amount of effusion small.

That the poison of cholera is analogous to that of low typhoid inflammations, is favoured by the fact, that at Chesham the deaths from typhoid inflammations, and from typhoid fever, were twice as many in 1848, and in the first month of 1849 much more numerous from typhoid inflammation, the large number being probably due to infection; also, diseases of a similar nature in the parish practice being more numerous, such as erysipelas, diffuse cellular infiltration, suppuration, low diphtheritic inflammation, and low peritonitis. Suppuration occurred in many cases without any known cause, as in 3 cases under the pectoral in women, and there were many more cases than usual of inflamed hand, &c., as found numerically by reference to the parish book.

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[*Treatment.*—Autumn, 1849. I attended a Syrian friend in Paris for an attack of Asiatic cholera. The algide symptoms well-marked. He was much benefitted by *Ice internally*, *Turpentine fomentations*, and leeches to the anus. He recovered after consecutive fever, with chest, head, and abdominal complications.

As regards the treatment at Chesham, the direction was complete, and I believe honestly carried out, by stimulants and counter-irritation, salivation, or astringents, and nourishment. (See the Cases.)

Tonic treatment (stimulants?) is absolutely necessary to sustain life; for the preceding diarrhoea, Acetate of Lead and Opium seem most valuable. Venesection for consecutive pneumonia may be indicated.

That rapid salivation is proper, is possible, but the question remains open as to the proper treatment; if, as for a low inflammation, local depletion and general tonic treatment is proper, it is here especially difficult to give tonics and not increase the local affection;

the manner of abstracting blood from the affected surfaces is nearly as difficult as in low peritonitis.]

*Microscopic Examinations.*—Ann Richardson.—Algide; recovered. Nov. 25, Watery fluid, white shreds in flocculi at the bottom.

Under microscope. Shreds are composed of amorpho-granular matter cohering together; among them some is darker in spots. No oil-globules, and only one or two columnar epithelia. Intermixed are some vegetable tubular and polygonal cells in masses.

Is alkaline, Nov. 30.

John Young, Nov. 26.—First motion of second attack, died in third relapse.

Under microscope. Contains several pieces of striped muscular fibre showing transverse lines, in some cases replaced on altering light by longitudinal lines or short longitudinal cracks, or a uniform granular appearance.

Very few if any epithelia.

Numerous round granular corpuscles, size of blood or  $\frac{1}{3}$  more.

The granular appearance seems to be often partly due to minute spherules on their surface. These on adding acetic acid gradually become clearer and smaller; but do not show any nucleus or distinct cell-wall.

There is also much granular amorphous matter, and irregular pieces (of foreign matter?), some tubular (vegetable) cells.

Tra. iodine and then water, has no blue effect; it tinges some yellow.

In both are numerous vibrios, dead. Nov. 30, and before.

Is slightly acid, Nov. 30.

Ann Finch, Nov. 27.—Algide; recovered.

Examined, 30th.

Muscular fibres, amorphous matter, and some few corpuscles or collections of granular matter. Also a few potato-starch granules and vegetable cells, known by shape and concentric lines. Iodine only tinges them (and other things) yellow. Also cells of various kinds of food. Dead vibrios.

Is acid.

William Nash, Nov. 29.—

Nov. 30. Is alkaline, of dirty white colour, very flocculent.

Granular amorphous matter, some colouring, no epithelium, mucus or pus, and streaked convoluted bands (viscid mucus?) contorted, apparently leaving cellular interspaces.

To these observations, being so few, I gave no importance; however, they appear to agree fully with the observations of Dr. Parkes, as to the absence of epithelium.—*London Journal of Medicine.*

## APPENDIX.

## PART I.—LIST OF CHOLERA CASES AT CHESHAM, 1848-9.

IN ORDER OF OCCURRENCE, AS TAKEN FROM PARISH BOOK. †

† Date when entered.	† Name.	† Age.	† Occupation.	† Date of death.	Time after Alg. coll.	Nature of disease, &c.	No. of case.
1848.					dys. hrs.		
Oct. 26	William H.....	50	Shoemaker .....	...	...	Yeasty Diarrhoea.	
28	Mr. C.						
Nov. 9	Hannah B. ....	63	*Widow .....	Nov. 11	...	Algide .....	1
12	Jane S.....	53	Widow .....	...	...	Algide. Secondary fev.	2
	Louisa P.....	49	*Labourer's wife .....	15	24	.....	3
13	Henry B.....	4	Shoemaker's child...	...	...	Algide. Secondary fev. ver long .....	4
	Henry S.....	16	Works at Mill .....	...	...	Algide .....	5
	Charles S. ....	13	*Works at Mill .....	18	36	.....	6
14	Sarah L. ....	29	*Platter .....	16	60	.....	7
16	William B.....	24	Closer .....	...	...	Algide .....	8
	Martha D. ....	55	*Widow .....	15	12	.....	9
	Adam B. ....	8	*Platter .....	18	38	.....	10
17	Sarah A. ....	14	Platter .....	...	...	Secondary fever, eyes bad .....	11
	Elizabeth W....		*Charwoman.....	18	19	.....	12
	Sarah H. ....	18	Platter .....	...	...	Secondary fever, eyes red .....	
18	Mary A. ....	17	Works at Mill .....	...	...	Mild.	
19	Jonathan B. ...	50	*Labourer .....	19	16	.....	13
	Elizabeth A. ...	48	*Widow .....	20	26	.....	14
20	John Y. ....	39	*Watercress .....	...	...	Fever type.	
	William G.....	62	Sawyer .....	...	...	Fever type.	
	Fanny B.....	28	*Platter .....	21	15	.....	15
	John Y. ....	70	*Labourer .....	Dec. 2	12	.....	16
	Ann A.....	16	*Platter .....	Nov. 21	32	.....	17
	Joseph D. ....	19	*Labourer .....	Dec. 8	18	Secondary fever, bad eyes.....	18
21	Sarah P. ....	47	Shoemaker's wife.	...	...	Severe Secondary fevr. eyes red.	
	William H.....	9	Labourer's son .....	...	...	Algide. Secondary fevr. Abscess underright pectoral .....	19
23	Sarah B. ....	45	Blacksmith's wife...	...	...	Algide. Little fever...	20
25	Ann R.....	23	Labourer's wife .....	...	...	Neglect.....	21
	Elizabeth A. ...	5	*Platter's child .....	Nov. 29	4	Secondary fever.....	22
26	Alfred B.....	10	*Labourer's son .....	Dec. 4	...	Slow sinking .....	23
	Ann F.....	60	*Labourer's wife .....	3	6	Severe Secondary fev.	24
29	William N.....	40	Labourer .....	...	...	Secondary fever.....	25
Dec. 2	Edward D.....	43	*Labourer .....	3	2	Choleroïd Diarrhoea. Low fever.	
	Sophia S.....	28	Silk Mill .....	...	...	Algide .....	26
	Hannah D. ....	55	*Widow .....	3	20	Child-bed. Algide.	
	Mrs. B.....		.....	...	...	Algide .....	27
3	Fanny D.....	40	*Labourer's wife .....	6	4&2	Slight Secondary fevr.	28
4	Sarah W.....	34	.....	...	...	Bad attendance .....	29
5	Ellen T. ....	1½	*Labourer's daughter	10	10	Slight Secondary fevr.	30
6	Charlotte T. ...	30	Labourer's wife .....	...	...	Choleroïd Diarrhoea.	
7	Samuel F. ....	15	Labourer's son .....	...	...	Ditto and Hysteria.	
	Charlotte Y. ...	30	Silk Mill .....	...	...		



† Date when entered.	† Name.	† Age.	† Occupation.	† Date of death.	Time after Alg. coll.	Nature of disease, &c.	No. of case.
1848.					dys. hrs.		
Dec. 10	Sarah B. ....	2	*Labourer's child.....	15	4½	Bad attendance .....	31
11	Walter S. ....	2	* ditto .....	11	6	Algide, convulsions ...	32
	Joseph J. ....	4	* ditto .....	14	4	Slow sinking .....	33
	Nimrod S. ....	3	* ditto .....	12	12	Algide .....	34
14	William B. ....	6	* ditto .....	19	5	Secondary fever.....	35
	James B. ....	38	Labourer .....	...	...	No collapse; mild.	
	Elizabeth T. ...	76	*Widow .....	16	46	Algide .....	36
15	Mary J. ....	37	Shovel-maker's wife	...	...	Mild .....	37
16	Ann H. ....	...	Labourer's wife .....	...	...	Choleroïd Diarrhœa.	
17	Robert H. ....	14	Silk Mill .....	...	...	Ditto.	
	Mary Ann W. ...	9	*Sawyer's daughter...	17	7½	Algide, and at last convulsions .....	38
18	Matilda A. ....	28	Labourer's wife .....	...	...	Mild. No fever. Just after child-bed.	
	Rebecca M. ....	8	Platter .....	...	...	Severe. Secondary fever. Bad eyes...	39
	Mary Ann S. ...	10	Silk Mill .....	...	...	Mild. No fever.	
	Elizabeth D. ...	73	*Widow .....	20	3	Algide .....	40
	George S. ....	3	Widow's child .....	...	...	Algide. Secondary fvr.	41
19	William H. ....	60	*Labourer .....	21	22	Algide .....	42
	Charlotte N. ...	33	Labourer's wife .....	...	...	Mild. No Secondary fever.	
21	Hannah D. ....	60	Widow .....	...	...	Choleroïd Diarrhœa.	
22	Charles W. ....	11	Sawyer's child .....	...	...	Algide. Secondary fvr.	43
	Jemima D. ....	70	*Labourer's wife .....	25	42	Algide. Red eyes ...	44
23	Francis P. ....	47	Shoemaker .....	...	...	Choleroïd Diarrhœa.	
24	Charles W. ....	13	Butcher's son.....	...	...	Algide. Slight Second- ary fever .....	45
25	Leah M. ....	52	*Labourer's wife .....	25	8	Algide. Asthmatic of years .....	46
	Job M. ....	55	Labourer .....	...	...	Algide, mild; long con- valescence. Head weak from previous fever .....	47
	Charles N. ....	8	Silk Mill .....	...	...	Algide. Fever. Left Hemiplegia .....	48
	Alfred P. ....	1½	*Widow's child .....	29	4	Struma and Algide ...	49
26	Maria T. ....	55	Widow .....	...	...	Algide. No fvr.; weak	50
28	Ann A. ....	56	*Labourer's wife .....	28	11	Algide; broken down.	51
29	Henry H. ....	1	Carpenter's child ...	...	...	Algide. No true col- lapse. Erythema.	
30	Joseph W. ....	38	*Sawyer .....	30	11	Algide .....	52
	Christopher W.	6	Sawyer's child .....	...	...	Algide. Slight Se- condary fever .....	53
	Isabella H. ....	10	Shoemaker's child...	...	...	Choleroïd Diarrhœa. Vomiting & purg.	
	Thomas N. ....	59	*Labourer .....	Jan. 2	28	Algide. Red eyes.....	54
1849.							
Jan. 3	James C. ....	57	Labourer .....	...	...	Choleroïd Diarrhœa.	
2	William P. ....	4	Widow's child .....	...	...	Algide. No Secondary fever .....	55
9	Henry P. ....	9	Labourer's son .....	...	...	Algide. Slight Se- condary fever .....	56

## PART II.—CONTEMPORANEOUS AFFECTIONS IN CATTLE.

*Feb. 7th.*—Mr. Allan, veterinary surgeon, has had three cases of apparently diffuse cellular infiltration in calves' legs about three weeks ago.

To-day he showed me part of the intestines of a sheep which, being taken with a now prevalent and fatal disease, "dropping," had been killed. The fæces had been squeezed out. The intestines still contained much opaque viscid mucus, and in parts greenish-yellow speckled semifluid fæces. No usual fæculent pellets. The surface is pale, now and then slight redness of very various type and very partial. The intestinal grouped glands appear raised, thickened; in a few there are central depressions; the solitary glands not visible. Was compared with another portion of gut from a healthy sheep, killed two days, and not known to be a corresponding portion. Here the solitary glands alone appear as prominent vesicles on the mucous surface, very few in number; no quantity of mucus. The fæces had been removed.

"Scouring," or diarrhœa, is common in sheep spring and autumn. "Dropping," according to Mr. Allan, is unusually fatal and epidemic just now.

The animal, previously healthy, appears to smell about, sometimes to keep jerking its head to one side, and its bowels are relaxed. This may prove fatal in an hour or so. Motion on the ground may look like a cow's, but of a slate colour; this is merely superficial, the true colour being yellowish-green.

In the pen appear motions of all kinds of consistence, from hard pellets to this relaxed state. It is stated not to be preceded by diarrhœa; that if a sheep is purged freely it recovers, the motions then being less green.

Upwards of thirty sheep, distributed among three owners, have died of this during the last month, or have been killed when taken ill. Twelve out of a flock of 130, nearly one-tenth. In this flock no cause is apparent. A few, but not many, have occurred since then.

*Feb. 23rd.*—Oldfield, corn-chandler, says, in autumn five of eleven bullocks died very suddenly; disease not known. In summer many sheep had scouring, and some died or were killed.

Chesham escaped the Asiatic cholera in 1832, but no account can be obtained of local health at that period, as Registries were not kept until 1837, and the New Poor Law, with its Medical Statistics, till after that time.

The following is a table of the causes of death in the seven years ending December, 1848 :—

PART III.—DEATHS IN CHESHAM (NOT CHESHAM BOIS) FROM JANUARY 1, 1842, TO JANUARY 1, 1849, AND IN THE FIRST MONTH OF 1849, WHICH WAS IMMEDIATELY CONSEQUENT TO THE PERIOD WHEN ASIATIC CHOLERA WAS SO FATAL. (MY OWN DIVISION.)

DISEASES	1842	1843	1844	1845	1846	1847	1848	Jan. 1849
of INFANCY, or results of; generally before two years old.....	53	24	56*	51†	56	45‡	51	6
OLD AGE, or results of	16	12	14	3	6	8	7	1
Acute Inflammations	7	3	7	7	6	12	8	0
Typhoid Inflammations ... }	4	4	0	2	1	2	4	6
... Fever.....	2	3	18	6	3	3	7+	0
DISORDERED BOWELS: Inflammations....	1	1+	2	0	0	1	0	0
Diarrhœa .....	2†	1+	†6†	†7†	3	3†+	1	0
Cholera .....	...	...	...	...	...	...	††4††+	2
CHRONIC DISEASES: Strumous .....	25	18	19	18	21	26	19	1
Non-strumous (& Apoplexy) }	34	27	24	15	13	36	23	3
Accidents (Visitation of God)..... }	5	3	6	4	2	5	2	0
Small Pox .....	...	...	...	...	...	...	1	1
Causes unknown .....	...	...	...	9	...	...	...	...
TOTAL .....	149	96	152	132	111	141	167	20
* 2 Measles. † Ditto Fatal. ‡ Scarlatina Fatal.								

*Infancy* (premature birth, debility, hooping-cough, pneumonia, bronchitis, some mesenteric disease).

*Typhoid Inflammation* (abscess, fever after parturition, diffuse cellular infiltration, low peritonitis, &c.)

+ Under two years; a + for each death.

*Acute Inflammations* (several are bronchitis, and many pneumonia).

*Old Age* (this head much fewer since deaths certified medically in July, 1845).

*Diarrhœa* (includes one or two of dysentery).

PART IV.—INFLUENCE OF THE CHANGES IN THE WEATHER AS CAUSING OR PREDISPOSING TO DISEASE DURING THE TWO YEARS 1847-8 FOR THE THREE LAST MONTHS, AND THE FIRST OF EACH SUCCEEDING YEAR. (FROM PARISH MEDICAL BOOK: MY OWN DIVISION.)

Year.	Month.	Mean Thermometer, 9 a.m.*				Wind; mean †		Days on which rain fell.	Variations daily of thermometer, at 9 a.m. ‡	Diarrhoea.	Dysentery.	Cholera.	Colic, &c.	Worms.	Typhoid Inflammations.	Erysipelas.	Inflamed Hand, &c.	Fever.	Scarlatina. Rubecola.	Acute Inflammations.	Chronic.		Disordered health.	Total.						
		Dry.	Wet.	Difference.	Maximum.	Minimum.	Difference.														Direction.	Force.			Strumous.	Non-strumous.				
1847.	Oct.	29.618	52.9	50.4	2.5	59.2	43.5	15.7	E. by S.	.3	2.9	15	Several rises and falls of 4, 5, & 6°.....	6	1	2	3	T. 10	S. V. ...	7	11	13	22	24						
	Nov.	29.657	45.6	51.0	37.3	13.7	1.8	20	S. S. W.	.4	1.8	20		Fall of 7; two rises of 5°.....	2	...	8	9	6	7	2	9	21		21					
	Dec.	29.504	40.5	39.6	0.9	43.8	33.7	10.1	S.	1.9	4.1	18		Rise of 7, & 12; fall of 9, 9, & 10°.....	2	...	1	6	61	6.1	26	6	4		19	10				
1848.	Jan.	29.602	33.7	32.6	1.1	36.3	27.4	8.9	N. E.	1	1.6	10	Fall of 10°; rise of 13°.....	7	1	1	15	5	4.1	54	3	7	16	15						
	Oct.	29.431	51.7	50.2	1.5	57.5	42.0	15.5	S.	1.6	5.1	25	Fall of 12, & 9; rise of 12°.	23	14	4	14	12	...	4	5	11	26	20						
	Nov.	29.584	42.8	41.3	1.5	48.1	34.3	13.8	N. & W.	1.0	1.5	18	Rise of 10, 11, 10, & 20°; fall of 8, 8, 9 & 10°.....	79	9	1	2	32	32	2	4	5	16	5	34	1	6	5	20	26
49	Dec.	29.590	41.7	41.0	0.7	46.0	35.3	10.7	S.	1.2	3.5	17	Rise of 14, 7, 7, & 12°; fall of 13, 7, & 6°.....	153	3	6	42	1	21	10	9	3	19	23						
	Jan.	29.551	39	37.8	1.2	37.8	26.0	11.2	N., Var.	2.60	19	19	Rise of 7, 18, 9, & 11°; fall of 7, 9, 16, 12, & 9°; rn.	47	2	3	20	4	18	2	33	5	10	25	30					

\* Dry and wet for 1847, for Aylesbury. † Wind, 1847, for Aylesbury. ‡ For 1847, from mean of whole day. § Influenza. ¶ Bronchitis, &c. in children.

PARISH CASES DURIN THE SAME YEAR. EXTRACTED FROM PARISH MEDICAL BOOK. (OWN DIVISION OF DISEASES.)																											
Month.	Mean Thermometer, 9 a.m.						Wind; mean.		Variations daily of Thermometer, $\bar{t}$ at 9 a.m. (dry).	Diarrhoea.	Diarrhoea under 2 years.	Dysentery.	Cholera.	Colic, &c.	Worms.	Typhoid Inflammations.	Erysipelas.	Inflamed Hand, &c.	Fever.	Scarlatina.	Varicella. Rubella.	Acute Inflammations.	Chronic.		Caused by "weather" &c.	Disordered health.	Total
	Dry.	Wet.	Difference.	Maximum.	Minimum.	Difference.	Direction.	Force.															Rain, inches.	Rainy days; thunder & rain.			
Jan.	29-626	33.7	32.6	1.1	36.3	27.4	8.9	N.E.	.95	1.55	10	23. 1 sudden fall of 10°; 1 rise of 13°; sleet	7	1	3	...	1	15	5	4	V.	54	3	7	16	15	
Feb.	29-332	43.2	41.6	1.6	48.1	35.1	13.0	W. & S.W.	1.19	4.48	20	20. Rise of 11°; fall of 8°; 1 thunder, rain	1	2	1	...	...	7	11	2	R.	12	5	11	26	19	
Mar.	29-261	44.1	41.9	2.2	49.3	33.1	16.2	N.W. to S.	1.26	3.92	22	21. Rise of 13°; 2 of 6°; 1 thunder, rain, hail	5	...	2	...	1	12	19	0	0	11	15	8	20	26	
April	29-339	50.5	46.6	3.9	57.	35.2	21.8	Var. then N.	1.55	3.23	20	14. Fall of 13°; rise and fall of 8°; rain	6	...	3	1	11	12	2	R.	6	6	6	24	26		
May	29-720	65.9	57.	8.9	73.9	39.	34.9	E. very Var.	1.24	.33	4	8. Thermom. high throughout; light rain	13	2	5	...	1	16	10	3	R.	21	8	6	29	35	
June	29-422	61.8	57.	4.8	69.3	45.9	23.4	S. & S.W.	1.73	4.35	22	21. Thermom. high throughout; windy showers	7	1	4	2	10	16	...	...	...	5	5	7	22	18	
July	29-601	65.6	59.7	5.9	71.4	46.6	24.8	{ N.&NW. } then { S.&SW. }	1.66	4.01	16	23. Rise of 10 & 8°; severe thunder-storms, rain	9	...	4	6	1	9	8	...	...	7	7	12	16	39	
Aug.	29-488	61.4	56.6	4.8	67.4	44.4	23.0	W. very Var.	1.36	2.96	27	20. 4 falls of 7°; 4 thunder-stms, rainy	6	5	1	2	9	9	7	...	...	9	5	10	17	13	
Sept.	29-616	59.9	55.9	4.0	64.2	41.7	22.5	{ N.W. } to { S.E. }	1.15	3.52	9	14. Fall of 10°; 2 thunder-stms, constant rain 3 days	12	10	1	5	1	8	7	...	...	4	5	6	20	17	
Oct.	29-431	51.7	50.2	1.5	54.5	39.	15.5	{ S. } about { the 15th. }	1.59	5.07	25	22. Fall of 12 & 9°; rise of 12°; rain and sleet	23	14	...	4	4	14	12	...	...	4	5	11	26	20	
Nov.	29-584	42.8	41.3	1.5	45.1	31.3	13.8	N. & W.	1.05	1.51	18	18. Rise of 10, 11, 10, & 30°; fall of 8, 9, 9, & 10°; rain	79	9	1	2	16	34	5	T.	1	6	20	26	24		
Dec.	29-590	41.7	41.	.7	43.	32.3	10.7	{ S. } at { end. }	1.21	3.48	17	19. Rise of 14, 7, 7, & 12°; fall of 13, 7, & 6°; light rain	153	3	6	2	4	11	21	1	...	10	9	3	19	23	
Jan.	29-551	39.	37.8	1.2	...	...	...	N., Var.	2.60	19	19	Fall of 7, 9, 16, 12, & 9°; rise of 7, 18, 9, & 11°; rain	47	2	4	2	14	18	...	V.	2	33	5	10	25	30	

All the statements of the weather are taken from those of the Rev. Mr. King, of Latimers, which are regularly published with those from Greenwich, &c., in the Registrar General's Report. Mr. King lives at Latimers, lower down in the same valley as is Chesham, but half-way up the side of the one range of hills, while Chesham is in the valley: they are distant two miles. Low inflammations, but not cholera, were present at Latimers during the Cholera invasion at Chesham. Latimers lies out of the general road for traffic near it, but otherwise is nearest to the "waterside" of Chesham, which the cholera especially affected.

*Diarrhœa* includes that with Colic or with Sickness. Under the head of *Diarrhœa* are included (during the Cholera period):—

Of Serous <i>Diarrhœa</i> . . . . .	14
Of Colic and <i>Diarrhœa</i> . . . . .	17
Of Sickness and <i>Diarrhœa</i> . . . . .	9

*Dysentery*, slimy, often bloody stools and pain with motions.

*Cholera* includes several of Choleroïd *Diarrhœa*.

*Colic*, also Constipation and Bilious attacks during summer.

*Inflamed Hand*, &c., Quinsy and Ophthalmia when not strumous.

*Fever*, Feverish attacks. T (typhoid).

*Acute Inflammation*, chiefly Bronchitis and Pneumonia often in children, also Hooping Cough; except of lungs very rare.

"*Weather*," &c.—Rheumatism, Pleurodynia, Syphilis, Catarrh, Accidents.

*Disordered Health*.—Vertigo, Dyspepsia, Amenorrhœa, &c., Eruptions.

*Looseness of the Bowels*:—

Quarter 1st, 1848 . . . . .	16
"    2nd " . . . . .	29
"    3rd " . . . . .	49
"    4th " . . . . .	366

*Cases of Suppuration* without known exciting cause:—

Mary G. . . . .	Age about 5.	Under Scapula.
Sarah S. . . . .	" 70.	Under Pectoral.
Sarah D. . . . .	" 60.	" "
Sarah P. . . . .	" 45.	" "
William B. . . . .	" 35.	Bursa of Knee after Cholera.

PART VI.—STATE OF THE WEATHER, (REV. MR. KING, LATIMERS).—OCTOBER, 1848.

Date	Thermometer, 9 a.m.					Wind, 9 a.m.		Rain for 24 hours.	WEATHER.	Cases of Diarrhea, Colic, &c., and Cholera. Messrs. H. P. & K.	Remarks on the Weather Tables generally, pp. 51 to 57 inclusive.	
	Barometer, 9 a.m.	Dry.	Wet.	Difference.	Maximum.	Minimum.	Difference.					Direction adopted.
Oct. 1	29.427	52	51.8	.2	60	35	25	S.	0	0	0	<p>These Tables, from pages 51 to 57 inclusive, show that the state of the weather aided the development of Influenza, Diarrhoea, Cholera, &amp;c., although certainly not causing these affections.</p> <p>The points to be chiefly considered are the difference between the wet and dry thermometer, which equals the degree of dryness of the air, also the many sudden changes of temperature, and occasionally the wind, degree of warmth, and quantity of rain.</p> <p>Page 51.—October and November, 1847, we should <i>a priori</i> consider healthy; December not so, in the large quantity of rain, large quantity of moisture in the air, and many extensive sudden changes favouring inflammatory internal affections, as seen in the Table of Disease under the heads Influenza and Fever. In the following month, the low temperature and cold wind continues internal affections, determined to the</p>
M 2	29.404	50	50	0	62	46	16	S.	1	1	3	
T 3	29.470	56	53	3	62	47	15	S.	1	1	3	
W 4	29.564	60.5	60	.5	60	55	5	S.	2	2	2	
Th 5	29.874	62.8	61.1	1.7	64.5	47.5	20	S.	1	1	2	
F 6	29.908	60.1	59.9	.1	69	49	27	S.	1	1	2	
S 7	29.888	62	60	2	69.5	50	5	S.	0	0	2	
S 8	29.774	61.1	59.7	1.4	67	45	22	W.	0	0	2	
M 9	29.730	55	54	1	56	42.5	13.5	S.E.	1	1	1	
T 10	29.404	53	50	3	57	38.5	18.5	N.W.	4	4	2	
W 11	29.647	50.5	48.8	1.7	57	37	20	N.	1	1	3	
Th 12	29.692	48.1	46.7	1.4	54.5	38.5	16	N.	0	0	3	
F 13	29.773	50.9	48.9	2	54.5	41.5	13	N.E.	3	3	1	
S 14	29.800	50	49.8	.2	52	37	15	N.E.	2	2	3	
S 15	29.636	48	47.5	.5	49	38	11	N.E.	1	1	2	
M 16	29.437	47.2	47	.2	55.5	40.5	15	N.	1	1	3	
T 17	29.557	46	45	1	45	29	16	N.	3	3	5	
W 18	29.333	34	32	2	37	31	6	N.	3	3	2	
Th 19	29.342	40	38.9	1.1	45.5	36	9.5	N.	4	4	3	
F 20	29.554	45.8	44.9	.9	46	37	9	N.	4	4	2	
S 21	29.512	43	43	0	46.5	28	18.5	N.W.	1	1	2	
S 22	29.428	49.8	49.5	.3	50.5	36	14.5	S.	3	3	2	
M 23	29.350	48	48	0	50	40	10	S.	0	0	0	
T 24	29.262	49.5	48	1.5	56	42	14	S.W.	1	1	5	
W 25	29.050	45.2	43.9	1.3	53.5	29	14.5	S.W.	2	2	6	
Th 26	29.596	40	40	0	55	38	17	S.	0	0	1	
F 27	29.193	52	52	0	51	37.5	13.5	S.	2	2	5	
S 28	29.170	49	48	1	49	37	12	S.	2	2	2	
S 29	29.220	47.5	47	.5	53	34.5	18.5	S.	1	1	6	
M 30	29.215	48	48	0	51	30	21	S.	1	1	3	
T 31	29.276	39	39	0	52	37	15	S.E.	1	1	3	

STATE OF THE WEATHER, (REV. MR. KING, LATIMERS).—NOVEMBER, 1848.

Date.	Thermometer, 9 a.m.					Wind, 9 a.m.		Rain for 24 hours.	WEATHER.	Cases of Diarrhoea, Messrs H. P. & K.	Remarks on the Weather Tables generally, pp. 51 to 57 inclusive.
	Barometer, 9 a.m.	Dry.	Wet.	Difference.	Maximum.	Minimum.	Difference.				
Nov. 1	29.237	45	45	0	47	32	15	N.W.	1	2	chest by the poison of Influenza; and probably accounts for common inflammations, as Quinsy, of wounds, &c. In October, 1848, compared with October, 1847, the large quantity of rain, the number of days on which it fell, the moisture in the air, and south wind, is remarkable. The internal disease is now Diarrhoea, due to the influence of the Choleraic poison. In November, 1848, the sudden changes of temperature; in December, the same, added to a similar state as in December, 1847, now favours Cholera (in 1847 Influenza). At the end of December, the Cholera subsiding, chest affections appear; and in January, 1849, are very prevalent, together with "Fever," &c., and still many Diarrhoeas, from the many sudden changes of weather and quantity of rain. The increase of Low Inflammations during and after the Cholera is noticeable.
Th 2	29.446	44.2	43.8	.4	50	33	17	W.	0	5	
F 3	29.310	43	43	0	47	30	17	W.	0	4	
S 4	29.223	34	33	1	33	22	11	N.W.	2	1	
S 5	29.484	34	32	2	42	33	9	N.W.	0	3	
M 6	29.368	45.4	43	2.4	47.5	33	14.5	N.W.	1	3	
L 7	29.285	40	37	3	43	25.5	17.5	N.W.	2	2	
W 8	29.700	34.8	33	1.8	43	24.5	18.5	N.	0	1	
Th 9	29.308	34.5	32	2.5	40	26.5	13.5	N.	2	3	
F 10	30.056	35.7	34	1.7	41	29.5	11.5	N.	1	1	
S 11	30.050	41	40.8	.2	41	36	5	N.	2	5	
S 12	30.100	41	40.3	.7	41	34	7	N.	2	6	
M 13	30.162	40.6	39.9	.7	44	32	8	N.W.	0	10	
T 14	30.020	42.8	41.9	1.1	47	25.5	21.5	N.	1	8	
W 15	30.134	34.5	32	2.5	40	20	20	N.	1	14	
Th 16	29.984	35	32	3	44	31.5	12.5	N.W.	0	13	
F 17	29.734	45	43	2	45	36	9	W.	2	8	
S 18	29.400	44	41.2	2.8	46	31	15	N.W.	1	4	
S 19	29.542	39	37.9	1.1	45	26	9	N.W.	1	24	
M 20	29.555	49	48.8	.2	49	35.5	13.5	W.	3	20	
T 21	29.400	42.5	42	.5	49	31	18	S.	1	13	
W 22	28.992	47	36	11	49	41	8	S.E.	3	14	
Th 23	28.884	45.7	45.2	.5	48.5	35	13.5	S.E.	1	12	
F 24	29.392	39	37.9	1.1	41.5	22	19.5	N.	1	9	
S 25	29.760	29.6	29	.6	...	...	...	S.	1	18	
S 26	29.594	49	49	0	52.5	40.5	12	W.	1	6	
M 27	29.536	44	43.5	.5	49	37	12	W.	0	12	
T 28	29.784	44	43.7	.3	49.5	41	8.5	W.	0	9	
W 29	29.680	52.2	51.9	.3	51	35.5	15.5	W.	1	1	
Th 30	29.592	43	41	2	43	29	14	S.W.	1	12	



TATE OF THE WEATHER, (REV. MR. KING, LATIMERS).—DECEMBER, 1849.

Date.	Thermometer, 9 a.m.				Wind, 9 a.m.		Rain for 24 hours.	WEATHER.	Cases of Diarrhoea, Colic, &c. H. P. & K.	Remarks on the Weather Tables generally, pp. 51 to 57 inclusive.
	Dry.	Wet.	Difference.	Maximum.	Minimum.	Difference.				
Dec. 1	38	48	0	42	30	12	.39	Overst. & drizig.; aft. clr.; nt. rn. & auro.	5	<p>Page 52—January, from north wind and cold, continues the Influenza of December, 1847. October, November, December, and January, 1849, are to be expected <i>à priori</i> to be unhealthy, from the low temperature and moisture in the air, and rain, or sudden changes, hence aiding the Choleraic poison, or causing chest affections. April, on the change to warm weather; May and September, from the fineness of the weather, doubtless led to carelessness; and in the two last, the fresh vegetable food determined Diarrhoea.</p> <p>Acute Inflammations in the Table are almost always of the lungs, and seem to depend pretty much on the height of the thermometer.</p> <p>Of course, the figures in the Tables must only be considered comparatively.</p> <p>Page 54 and on—In October, the causes of English Cholera in the beginning of the month, and those of Asiatic Cholera at the end, are</p>
2	38	37	1	38.5	28	10.5	.01	Overst. generally, and showers; nt. fine	8	
3	33	32	1	45	30	15	.09	Overcast and rainy	16	
4	47.5	47.2	.3	46.5	34	12.5	.19	Drizzling rain; evening, fine	17	
5	41	39	2	44	33	11	.05	Fine and overcast; showers	5	
6	40.2	40	.2	46	35.5	11.5	.24	Clear and overcast; night, showers	21	
7	47	47	0	51	43.5	8.5	.49	Rain	23	
8	54	53.3	.7	52	44	8	.10	Overcast; light showers	12	
9	49	48	1	53	34.5	18.5	...	Partially overcast; generally clear	10	
10	44	44	0	54	34	20	...	Partially overcast	7	
11	43	43	0	52	40	12	...	Partially overcast	21	
12	50	50	0	50	37	13	...	Generally overcast	9	
13	48.1	46	2.1	52	36	16	...	Partially overcast	7	
14	43	43	0	45.5	36	9.5	.11	Generally overcast; drizzling rain	10	
15	47	46.9	.1	50	39	11	.24	Drizzling rain	9	
16	42.5	42	.5	39	31	8	.74	Rainy	11	
17	37	34.3	2.7	40	31	9	.05	Clear; rainy	14	
18	40	40	0	44.5	37	7.5	.04	Overcast; light rain	34	
19	44	44	0	43	31	12	.06	Drizzling rain	8	
20	39	38.1	.9	35	21	14	...	Overcast; frosty	19	
21	26	25	1	29.5	22.5	7	...	Frosty	10	
22	31	30	1	32	23	9	...	Frosty	18	
23	30	29.5	.5	29	19	10	...	Overcast; night, clear	12	
24	28.5	27.9	.6	36	25	11	.13	Clear; thaw in night	10	
25	40	40	0	42	35	7	.02	Overcast; drizzling rain	15	
26	45	45	0	46	34	12	.53	Drizzling rain; night, clear	7	
27	39	39	0	43	34	9	...	Clear; night, heavy rain	7	
28	37	37	0	39	34	5	...	Partially overcast	10	
29	38.8	38	.8	40	30	10	...	Overcast; but fine	13	
30	35.3	35	.3	36	30.5	5.5	...	Overcast; but fine	12	
31	34.1	34	.1	36	26	10	...	Overcast; but fine	3	

STATE OF THE WEATHER, (REV. MR. KING, LATIMERS).—JANUARY, 1849.

Date.	Barometer, 9 a.m.	Thermometer, 9 a.m.				Wind, 9 a.m.		Rain for 24 hours.	Mildte Collapse.	Cases of Diarrhoea, Colic, &c., and Cholera, Messrs. H. P. & K.
		Dry.	Wet.	Difference.	Maximum.	Minimum.	Difference.			
Jan. 1	29.872	29.5	28	1.5				1	10	
1	29.786	22	21	1					13	
2	29.400	22	21.3	.7					4	
3	29.472	29	28.8	.2					7	
4	29.472	29	28.8	.2					2	
5	29.464	32	32	0					3	
6	29.696	27	27	0					3	
7	29.730	30	29.8	.2					3	
8	29.250	35.5	35.3	.2					4	
9	29.222	37.4	37	.4				1	10	
10	28.762	43.5	43	.5					2	
11	28.918	37	36	1					3	
12	29.820	28.5	28	.5					1	
13	29.450	47	47	0					1	
14	29.170	51.5	51	.5					...	
15	29.730	35.2	35	.2					5	
16	29.632	38	38	0					2	
17	29.416	49	49	0					1	
18	29.706	41.2	41.2	0					2	
19	29.642	47	47	0					3	
20	29.518	46	46	0					1	
21	29.924	44	43.6	.4					1	
22	29.635	35	35	0					1	
23	30.028	44	43	1					1	
24	30.074	46	43.7	2.3					1	
25	29.784	47.3	45	2.3					...	
26	29.528	43	42	1					1	
27	29.620	30.8	30.5	.3					2	
28	29.060	35.8	34	1.8					5	
29	29.292	36	34.8	1.2					3	
30	29.810	31	31	0					2	
31	29.825	36.5	35	1.5					...	

Remarks on the Weather Tables generally, pp. 51 to 57 inclusive.

aided by the weather; and in the following months of these Tables, the same is evident. Thus considering the state of moisture of the air for example; arranging those days in two separate divisions, according as the degree of moisture is either above or below the unit:—

Dryness* Days Above		Rain		Cases		Dryness Days Below		Rain		Cases	
Oct. 15	25.1	1.61	30	16	3.4	3.48	44				
Nov. 16	41	.19	111	14	4.8	1.24	136				
Dec. 8	11.8	.2	88	23	5	3.45	296				
Jan. 10	14.6		34†	21	4.1		53				

On referring to other points, as the extensive changes of temperature, as wind, &c., the weather must be considered as answerable for exerting much influence. The sources of fallacy are, of course, most numerous; hence the deductions from large numbers can alone prove true.

\* The figures representing moisture in the air are inverse, hence must be called here "dryness."  
 † This contradiction is accidental; sudden lowering of temperature increased the number by half.

PART VII.—CASES OF POST-MORTEM EXAMINATION BY SELF AT CHESHAM.—MR. HODGSON'S PRACTICE.

No. of Case.	Name.	Nature of Disease.	Hours after death.	Examined carefully or not.	Alterations in Intestines and	Elsewhere.
1	John Y .....	Cholera (algida at death.) .....	20	N, C.	A few glands in large intestines; no Peyer; mesenteric glands enlarged, white; some mucus; feces	No cause of death in other parts.
2	Jos. D. ....	Cholera and fever; pneumonia.....	13	C.	Uniform injection around glands of Peyer; mucus and thin fluid; mesenteric enlarged; no solitary glands; injection in large intestine.....	Pneumonia and congestd. brain.
3	Edward D. ....	Cholera and fever; morbus Brightii .....	24	C.	Some solitary glands; no injection; effused blood; mesenteric glands enlarged; mucus.....	Kidneys diseased; lungs congested.
4	Fanny D.....	Cholera algida; old nephritis, and } retroflexed uterus .....	17	C.	Mucous membrane largely injected; solitary enlarged; Peyer's raised, flocculent, injected, and spaces hollow; hæmorrhagic spots; mesenteric glands enlarged; brownish fluid, mucus, and fibrin.....	Pneumonia; uterus abnormal; one kidney diseased; ulcerations from acetate of lead.
5	Hannah D. ....	Cholera algida .....	10	C.	Solitary and Peyer's glands enlarged or burst, and injected; mucous membrane injected largely; shreddy fluid and mucus; mesenteric glands enlarged .....	No other cause of death; organs choleraic.
6	Alfred B.....	Cholera and fever; pneumonia; enteritis .....	20	C.	Solitary and Peyer's glands enlarged, hollow and sometimes injected; 7 intussusceptions; enlarged mesenteric glands; mucus and feces .....	Pnmmia.; kidneys very white.
7	Ellen T. ....	Diarrhœa; cholera; enteritis .....	11	C.	Solitary of small intestines enlarged or absent; of large intestines and Peyer's ulcerated, and the first injected; mucus and fecal; mesenteric enlarged .....	No cause of death in other parts.
8	Walter S..... Nimrod S (N.C.)	Cholera; fit; old diarrhœa.....	9	C.	Solitary glands enlarged of old by deposit, pale; thin fluid and mucus; no feces; mesenteric enlarged of old .....	No cause of death in other parts; congested brain.
9	Elizabeth T. ....	Cholera algida .....	24	C.	Solitary and Peyer's glands enlarged; mucous membrane completely injected; mesenteric enlarged; choleraic fluid; no feces .....	Morbus Brightii; no cause of death in other organs.
10	William H. ....	Cholera algida .....	21	C.	Solitary enlarged; Peyer's raised, flocculent, and pale; mesenteric glands enlarged; mucus and cholera fluid .....	No cause of death in other organs; brain congested.
11	Elizabeth W....	Meningitis .....	20?	C.	Glands not enlarged; some thick glairy mucus in colon; mesenteric not enlarged; feces .....	Fibrin and serum, and great vascularity of the pia-mater.
12	Alfred P.....	Struma; cholera; exhaustion .....	36	C.	Peyer's thickened, injected; below fibrin effused; mesenteric enlarged; bilious mucus; no feces.....	Struma of brain, lungs, and bowels.
13	Joseph W. ....	Cholera algida .....	21	C.	Solitary and arminated glands enlarged; those of large intestines likewise injected; mesenteric enlarged; mucus; cholera fluid; no feces .....	No other cause of death.
14	Sarah S. ....	Abscess under pectoral; uterus retroflexed; diphtheritis of mouth, &c.; } pneumonia .....	60	C.	Solitary not visible; Peyer's healthy; of large intestines injected around spaces for glands; mesenteric not enlarged; copious feces .....	No other cause of death than abscess; diphtheritis of œsophagus, and pneumonia.

CASES OF POST-MORTEM EXAMINATION AS REGARDS PATHOLOGY OF INTESTINAL DISEASE.

DR. WILLIAMS'S—MALES.

No. of Case.	Name.	Nature of Disease.	Hours after death.	Examined carefully or not.	Alterations in Intestines and	Elsewhere.
15	W—r.	Myelitis; erysipelas	4	C.	Intestines not examined	{ Erysipelatous redness has disappeared. Softening of medulla.
16	T—d.	Pneumonia (fever?)	22	C.	Peyer's patches not visible. Glands of large intestines visible.	{ Ulcers of trachea and increased redness; 3rd stage of phthisis.
17	B—k.	Phthisis; third stage	15	C.	Peyer's and solitary glands ulcerated; redness not mentioned.	{ Thick mucus, and bright redness in bronchi; 3rd stage of phthisis.
18	M—n.	Phthisis; third stage	14	C.	Ulceration in large and small intestines; no redness mentioned.	{ Inflammatory effusion in brain.
19	W—t.	Cerebritis chronica; constipatio	30	C.	Some solitary glands enlarged; no redness mentioned.	{ Clot in brain. Bright's disease.
20	I—n.	Hemorrhagic apoplexy; diarrhoea	30	C.	Intestines, enlarged glands and fine injection around.	{ Scirrhus stricture, &c.
21	F—l.	Carcinoma recti, &c.	20	C.	Large intestines ulcerated, and some bright injection	{ Peritoneum, recent lymph, no injection. Diseased heart and kidneys.
22	N—m.	Morbus cordis, &c.; peritonitis purulenta.	26	C.	Ulceration, and some redness in small intestines.	

DR. WILLIAMS'S—FEMALES.

23	P—r.	Ascites (from chronic peritonitis); pleurisy	12	C.	Mucous membrane unaffected, except some injection in rectum (from use of bougie?)	{ Effusion and thickening of peritoneum
24	J—s.	Chronic enteritis; purulent peritonitis	14	C.	Intestines, ulcers, slight redness.	{ Omentum injected. Fluid purulent in peritoneum
25	S—n.	Cerebritis; erysipelas	14	C.	Peyer's glands blackish points; some solitary glands visible.	{ Erysipelatous redness disappeared. Bright injection of pons Varolii; pneumonia.
26	H—s.	Morbus cordis, &c.; lobular pneumonia.	32	C.	Intestines healthy.	{ Diseased heart, &c.
27	M—y.	Peritonitis purulenta	9	C.	Intestines unaffected?	{ Fluid, and some vascularity in peritoneum.
28	A—s.	Idiocy; fever.	17	C.	Ilium healthy; some mucus; patch of follicles in caecum injected	{ Bright's disease. Want of symmetry in head.
29	A—n.	Tumor uteri; stricture of rectum; consequent enteritis and resulting peritonitis	13	C.	Ulceration; some capillary injection.	{ Tumor fibrous of uterus, and stricture of rectum with perforation.
30	S—l.	Erysipelas	12	C.	Dirty adhering mucus.	{ Erysipelatous redness disappeared.

CASES OF POST-MORTEM EXAMINATION AS REGARDS INTESTINES.

DR. THOMSON'S—MALES AND FEMALES.

No. of Case.	Name.	Nature of Disease.	Hours after death.	Examined carefully or not.	Alterations in Intestines and	Elsewhere.
31	H—n.....	Chronic phthisis; cirrhoted, fatty liver...	18	C.	Intestines, old ulcerations, no redness noticed.....	{ Bright injection and mucus in } /bronchi.
32	O—n.....	Chronic pleurisy, &c. ....	26	C.	Intestines, no glands nor other visible.....	
33	D—s.....	Carcinoma et stomachi et hepatis; he- patitis .....	18	N. C.	Intestines unaffected.....	{ Partial injection in surface of } liver.
34	N—n.....	Continued fever; pneumonia; typhlo- enteritis .....	18	N. C.	Intestines, glands thickened, ulcerated, injected..	
35	M—l.....	Morbus cordis, &c.; paracentesis; peri- tonitis; pyæmia .....	21	C.	Intestines, glands hardly visible; no redness nor ulceration; intestinal thick mucus contains epithelium .....	{ Peritonæum pale; fluid copious } and purulent.
36	R—e..... From former } notes.....	Morbus Brightii; poisoning by mor- phia; diarrhoea .....	15	C.	Intestines, solitary glands enlarged; slight red- ness in large intestine; thick mucus.....	
37	W—s.....	Myelitis .....	10	N. C.	Intestines healthy.....	{ Spinal cord, where inflamed, is } capillary injection.
38	H—t.....	Abscessus cerebri; cerebritis .....	8	C.	Intestines, glands hardly visible; thick mucus (copious epithelium).....	
39	Y—g.....	Peritonitis purulenta .....	26	N. C.	Intestines not examined.....	{ Peritonæum pale; pus con- } tained.

39 CASES. INTESTINES EXAMINED, 37.

INTESTINES NON-CHOLERAIC, 25.

3 erysipelas; redness disappeared in 4, 12, and 14 hours.  
Diarrhoea or enteritis; slight redness; no redness.  
(No. of hours) . . 18 14 13 20 26 30; 15 30 14 15

Changes similar to cholera, in solitary glands, 3.  
In Peyer's, none.  
In complete injection or effused fluid, none.

## CONCLUDING REMARKS, 1852.

FROM the foregoing data, the following conclusions seem to me to be justifiable; that the Asiatic Cholera of 1848, in Chesham, was neither epidemic nor endemic; was therefore—and was with moral certainty, judging from the circumstances of its diffusion,—infectious; that natural depressing agents alone accounted for those individuals being attacked, who were so; that the disease corresponds in every particular to an erysipelatous inflammation of the intestinal mucous membrane, due to a peculiar poison, which may be considered to be of the class of low inflammatory poisons, and, by its intensity, analogous to and ranking with the separate sub-class or division of erysipelatous inflammations, perhaps able—or by its degeneration able—to set up the ordinary forms of these inflammations, but requiring its own unaltered poison for the production of itself.

Its course and severity corresponds to that of purulent peritonitis, so also its effused fluid modified by the surface yielding the same. Comparison of the effusion with that of the effusion of tracheitis; of diphtheritic inflammations; in the blebs of erysipelas; from blisters; in water-brash; with hydragogue purgatives; in simple diarrhœa, &c., &c., fully accounts for the peculiarities of the rice-water stools. It must be remembered that all loss of the thinner portion of the blood in cholera, by rendering the circulation through the capillaries of the liver and lungs more difficult, reacts upon itself, and by increasing the congestion of the portal system materially aids the inflammatory effusion from the mucous surface.

Believing that it is unphilosophical to seek for far-fetched explanations of simple facts, the above proofs towards Cholera being an erysipelatous entero-colitis are given, not as being similar to the cutting of the Gordian knot, but merely as assisting to untie it, by using the proper means to loosen the right nooses; it were possible to draw the folds tighter by employing any other than the only right method of investigation.

It seems to me that in all probability the Cholera poison likewise was the cause of the many diarrhœas in man and beast; probably by exciting local changes, or possibly by inducing changes in the blood generally. Now seeing that the mere symptoms in the *course* of

Cholera alter the blood so materially, if Cholera *be due* to an altered state of the blood, it seems to me that the way to discover these changes would be by examining the blood of patients attacked with diarrhœa, faintness, colic, &c., &c., *during* the Cholera time, and in an infected place.

I cannot very well conceive the blood *filtering through* the mucous membrane in only one portion of its extent, and believe that in all diarrhœas either determination, congestion, or inflammation of the mucous membrane actually occurs, and that this change of the blood-vessels is the cause of the symptoms. I should expect that in *diarrhœas* during the Cholera, the pathological changes may be those of mere reaction to the poison; perhaps in choleroïd diarrhœa there is commencing inflammation, and in algide cholera extended inflammation, affecting perhaps two-thirds or more of the whole intestinal track. (See P.M.E.'s. of Elizabeth T., Hannah D., and Fanny D.)

It seems probable that the cases entitled in the parish book as Cholera, in which algide symptoms were not marked, were cases where the peculiar action causing algide depression had been present, but had quickly ceased, and those symptoms due to the altered state of the blood supervened.

THE END.

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