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DE

LA CONTAGION

SEULE CAUSE DE LA PROPAGATION

DE LA LÈPRE

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PAR

LE Dr Ch. L. DROGNAT-LANDRÉ

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PRÉFACE

Quoique la plupart des États de l'Europe soient délivrés d'un fléau terrible, la lèpre, il n'en est malheureusement pas encore de même des autres parties du monde, où cette maladie sévit encore avec toute sa rigueur. Il n'est donc pas étonnant qu'on se soit occupé, surtout dans ces derniers temps, de l'étiologie de cette affection.

Voici les raisons qui m'ont conduit à écrire sur ce sujet.

Il y a deux ans que j'eus l'occasion d'observer, en Hollande, dix cas de lèpre contractée dans les colonies néerlandaises. A cette époque, l'attention du corps médical étant plus particulièrement dirigée sur les causes de cette maladie, je fus donc conduit à examiner la question et, quelque temps plus tard, à publier un mémoire sur ce sujet.

Mais sans le secours de mon père, le docteur Ch. Landré, qu'une longue pratique à Surinam, où la lèpre règne endémiquement, a rendu compétent en cette matière, mon peu d'expérience aurait enlevé toute valeur à mes conclusions.

Je suis heureux de pouvoir dire ici que dans ce travail j'ai été guidé par son intuition si prompte et si pénétrante; c'est avec une vraie satisfaction que je lui rends aujourd'hui hommage pour ses précieux renseignements.

Dans le premier chapitre de ce mémoire, j'ai traité à à dessein longuement l'histoire de la lèpre à Surinam, histoire qui, comme on le verra, est d'une haute valeur et pleine d'enseignements pour l'étude étiologique de cette affection.

Il y a deux ans, j'étais le seul défenseur de l'opinion qu'émet le titre de cette publication, et bien que depuis on se soit beaucoup prononcé contre mes conclusions, je les maintiens encore, et au lieu d'être ébranlé par les attaques de mes adversaires, je suis plus que jamais convaincu que la contagion est le seul mode de propagation de la lèpre; les anti-contagionnistes m'en ont eux-mêmes fourni les preuves.

La tâche n'était pas facile, et si la plupart des auteurs en avaient été convaincus, ils ne se seraient pas prononcés si légèrement sur la cause de cette maladie, *cause que l'on ne peut trouver qu'en traitant la question au point de vue général et comparatif*. Il ne s'agit pas ici de

conclure d'un fait particulier à la non-contagion de la maladie, comme on le fait généralement. L'humanité est trop intéressée à la solution de ce problème, pour ne pas y apporter toute l'attention, tout le sérieux et toute l'ardeur qu'exige une question de cette importance.

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CHAPITRE PREMIER

HISTOIRE DE LA LÈPRE A SURINAM

La colonie de Surinam est une partie de la contrée connue sous le nom de Guyane, sur la côte nord-est de l'Amérique méridionale ; elle est située à peu près entre le 2e et 6e degré de latitude nord, et entre le 56e et 59e degré de longitude occidentale de Paris. Elle a une étendue de 2800 milles ou lieues géographiques carrées, dont 700 sont seulement connues.

Les aborigènes de la Guyane sont des Indiens; ils sont divisés en un grand nombre de peuplades, dont les Warrauws, les Caraïbes et les Arrowaks sont les plus connues à Surinam.

Les efforts que firent les Européens pour s'établir dans ces contrées, rencontrèrent, au commencement, beaucoup d'obstacles, surtout l'insalubrité du littoral et du bord des rivières qui étaient, en ces temps-là, couverts de forêts épaisses et de marais, et les combats continuels qu'il fallait livrer aux Indiens belliqueux.

La première colonisation régulière d'Européens à Surinam eut lieu en 1630 : environ soixante Anglais, sous le capitaine Maréchal, se fixèrent au bord de la rivière Para, qui est un affluent du fleuve Surinam.

Dès l'année 1644, les Israélites portugais, qui étaient vivement persécutés au Brésil, commencèrent à s'établir dans des lieux éloignés de la mer, sur les bords du fleuve Surinam ; il paraît qu'un assez grand nombre d'esclaves, qu'ils avaient emmenés avec eux du Brésil, fut la principale richesse de cette colonisation. Quoique l'histoire ne le mentionne pas d'une manière positive, il paraît aussi que les Anglais avaient alors à leur service des esclaves (Indiens ou nègres?). On ne peut dire au juste à quelle époque l'importation directe des nègres de l'Afrique à Surinam a commencé; il est néanmoins certain qu'elle a déjà eu lieu sous le règne de Charles II, roi d'Angleterre, de sorte qu'on peut dire avec quelque certitude que, dans les plantations de cannes à sucre établies par les Anglais, dont, en 1665, le nombre avait déjà atteint quarante ou cinquante, les travaux furent faits par des nègres.

Comme les colons avaient continuellement beaucoup à endurer des attaques imprévues et irrégulières des Indiens, surtout des Caraïbes, quelques intéressés dans la colonie de Surinam adressèrent avec instances, aux États généraux de la mère patrie, une supplique dans laquelle ils demandèrent des secours contre ces importunes attaques. En 1684, les Indiens furent assaillis avec succès et repoussés jusqu'au fleuve Coppename; il en résulta que la paix fut conclue et que les trois peuplades mentionnées plus haut furent reconnues comme libres.

Chez ces indigènes de la Guyane, la lèpre était tout à fait inconnue ; or, *Schilling* (1), *Nissaeus* (2), *Bajon* (3), *Dazille*, la commission royale de Cayenne (4), *Campet* (5), *Rodschied* (6), sont unanimes pour reconnaître que la lèpre a été introduite

(1) G. G. Schillingii *De lepra commentationes*, recens. I. D. Hahn. Lugd. Batav., 1778.

(2) *Spec. de nonnulla in colon. surinamensi observ. morb.* Harderwyk, 1791.

(3) *Mémoires pour servir à l'histoire de Cayenne et de la Guyane française.* Paris, 1777.

(4) *Rapport sur le mal rouge de Cayenne.* Paris, 1786.

(5) *Traité pratique des maladies graves*, etc. Paris, 1803.

(6) *Med. und chirurgische Bemerkungen aus Rio Essequebo*, 1796.

dans la Guyane par l'importation des nègres. Si, dans la suite, quelques cas de lèpre parmi les Indiens ont été observés, la cause en doit être recherchée dans la communication habituelle avec les nègres. *Schilling*, qui a habité Surinam, où il a observé la lèpre, se prononce, à ce sujet, de la manière suivante : « Endemium Americæ morbum fuisse non puto. Nam » licet hodie ipsi aborigines eo passim laborent, sunt tamen » integræ gentes ab eo prorsus immunes; atque in illis etiam » tribubus, quas jam attigit, eos tantum infectos esse deprehendimus, qui cum Æthiopibus corpora sua miscent, alia» rumve rerum commercio junguntur. »

En 1682, les États généraux avaient favorisé la compagnie des Indes occidentales du privilége d'exporter des nègres de l'Afrique et de les vendre à Surinam, toutefois sous condition de les livrer en nombre requis, parce que, dans la colonie, « on ne connaissait ou on ne voulait autre chose que des esclaves noirs pour cultiver la terre ». (*Octrooi van den* 23 *september* 1682.)

La compagnie jouit de ce privilége, auquel cependant il fut fait mainte infraction par une importation clandestine de nègres, jusqu'en 1730, époque à laquelle l'importation des nègres devint libre; la compagnie néanmoins ne fut pas exemptée de l'obligation d'importer annuellement 2500 Africains.

Pendant les années que le gouverneur *van Sommelsdyk* eut la direction de la colonie (1683-1688), des esclaves commencèrent à s'évader des plantations et à se cacher dans les bois, et déjà au commencement du XVIII^e^ siècle, ces fugitifs se réunirent en bandes qui, de temps en temps, manifestèrent des dispositions hostiles; ils formèrent à Surinam une population séparée de la colonie. Ces nègres sont connus sous le nom de *marrons ;* nous en parlerons plus tard.

Parmi les nègres qui furent importés d'Afrique, il s'en trouvait qui étaient atteints de la lèpre, de la framboisie et d'autres maladies que l'on croyait contagieuses; mais on ne commença à y faire attention que lorsqu'il devint manifeste que le nombre de ceux qui en étaient attaqués allait en augmentant, et que l'on commença à craindre que ces mala-

dies ne se communiquassent à d'autres personnes. Le gouverneur *de Cheusses*, qui prit la direction de la colonie le 9 novembre 1728, jugea prudent d'interdire dans ce même mois, par une ordonnance, la circulation des nègres infectés dans les rues de la ville de Paramaribo et sur la voie publique. Nous lisons à la tête de cette ordonnance la phrase suivante : « L'expérience journalière (je traduis littéralement) nous a prouvé qu'il y a plusieurs esclaves, tant rouges que noirs, qui se promènent ou qui se reposent dans les rues de Paramaribo, où passent continuellement des blancs, d'où il pourrait résulter de graves inconvénients, non-seulement pour les adultes blancs, mais aussi pour des enfants blancs ignorants qui s'approcheraient de ces esclaves affectés de framboisie (yaws), de boasi (nom qu'on donne vulgairement à la lèpre dans la colonie de Surinam), d'ulcères et d'autres maladies contagieuses. »

Après ce préambule il est interdit, dans cette ordonnance, aux maitres d'esclaves infectés, de les faire circuler sur la voie publique, et il leur est enjoint de les retenir dans leurs demeures, sous peine d'amende.

Insensiblement cette défense tomba en oubli, et il y fut fait tant d'infractions, qu'en 1761, le gouverneur général d'alors, *W. Crommelin*, crut nécessaire de renouveler l'ordonnance.

Vers les années 1763 et 1764, on commença à s'apercevoir des progrès que la lèpre avait faits. Les fraudes mises en œuvre par les capitaines des négriers pour dissimuler les premiers indices de la lèpre, obligèrent le gouvernement de défendre la vente des Africains atteints de cette maladie (1). Mais l'inspection des nègres nouvellement importés n'étant pas surveillée, la cupidité de plusieurs planteurs les induisit à acheter à vil prix des esclaves infectés, pourvu qu'ils fussent en état de travailler aux champs.

Les abus qui s'étaient introduits dans l'exercice de la méde-

(1) Je n'ai pas pu me procurer les ordonnances qui ont été décrétées à ce sujet.

Déjà un article du Code Noir de 1718 autorisait les tribunaux des colonies françaises à déclarer nulle la vente d'un esclave atteint du moindre signe de la lèpre.

cine favorisaient ces menées ; le 14 mai 1763, il fut ordonné par le gouverneur que, désormais, personne ne pourrait s'établir dans la colonie comme chirurgien sans avoir subi un examen devant une commission nommée pour cela par la cour, ou bien sans avoir produit un diplôme des Pays-Bas. Peu de temps après cependant, le gouverneur jugea nécessaire de faire visiter les nègres nouvellement arrivés dans la colonie par un docteur-médecin et par le chirurgien-major des troupes.

Malgré cette surveillance plus sévère de l'importation des nègres infectés, la lèpre continua à faire des progrès sensibles dans la population, tellement qu'en 1790, le gouverneur *Wichers* jugea utile de publier une ordonnance plus rigoureuse. Comme motif de ces nouvelles dispositions, le gouverneur nous fait connaître le triste fait que, depuis quelque temps, la lèpre s'était communiquée, « par la contagion », non-seulement à un grand nombre d'esclaves et de gens de sang mêlé, mais aussi à plusieurs blancs. Dans cette ordonnance, nous voyons pour la première fois faire mention de l'isolement des malades.

Aux articles 3-5, il est ordonné que tous les nègres et les mulâtres, tant esclaves que libres, infectés de la lèpre, seront mis à l'écart dans un lieu séparé au bord de la rivière Saramacca ; tandis que l'article 6 enjoint aux blancs atteints de cette maladie de quitter la ville de Paramaribo, et de se tenir séparés de toutes les personnes qui ne seraient pas indispensables à leurs besoins. — Article 7 : Les nègres arrivés dans la colonie seront visités par le docteur et par le chirurgien-major, et s'il y en a qui soient reconnus lépreux, ils seront déposés, aux frais du capitaine, dans un endroit près de la côte destiné pour cela ; ils seront réintégrés à bord du navire qui les aura amenés lorsque celui-ci effectuera son départ de la colonie, ou bien le capitaine fournira une caution pour l'entretien des nègres qu'il ne prendrait pas avec lui.

Des ordonnances ultérieures, en date du 20 mai 1791 et du 29 février 1792, engagent aussi les capitaines à faire transpor-

ter dans la léproserie, aux bords du Saramacca, les esclaves infectés qu'on aurait trouvés à bord de leurs navires.

Cet établissement, ou léproserie, auquel on a donné le nom de *Voorzorg* (la précaution), était situé sur la rive droite du fleuve Saramacca; le nombre des lépreux qui s'y trouvaient, et qui en 1790 ne dépassait pas le chiffre 7, s'accrut bientôt tellement que déjà en 1792, le gouverneur général *de Frederici* dut faire agrandir l'établissement, et qu'en 1795 on y comptait 200 individus. En 1797, le chiffre s'élevait à 300; en 1808, à 400, et déjà, en 1812, à plus de 500. Mais il n'est pas probable que, dans ce nombre, il se soit trouvé des blancs, puisque à cette dernière époque, et même plusieurs années après, l'article 6 de l'ordonnance de 1790 était encore en vigueur et fut strictement exécuté.

Avant de continuer l'histoire de la lèpre, je dois revenir sur les nègres marrons, dont j'ai fait mention. Pendant un grand nombre d'années, ils firent contre la colonie une guerre ruineuse que l'on ne put considérer comme terminée qu'en 1776. La paix fut conclue avec eux à plusieurs reprises, et ils furent alors divisés en trois peuplades, et reconnus indépendants. Ces trois peuplades qui occupent le haut pays, le long des rivières, sont : les nègres marrons d'Auca, de Saramacca, et les nègres Becoe.

Ce n'est qu'avec de grandes difficultés qu'on peut arriver jusqu'aux habitations de ces trois peuplades, parce que les hautes rivières sont tellement encombrées de rochers, et si peu navigables, qu'eux-mêmes ne peuvent y passer que dans des pirogues, et le plus souvent au risque d'y perdre la vie. La distance de leur pays à la mer est évaluée, en droite ligne, à peu près à cinquante lieues de marche. Parmi ces trois peuplades, qui depuis un siècle ont vécu indépendantes les unes des autres, et séparées des esclaves, la lèpre règne (d'après l'opinion généralement reçue dans la colonie) avec beaucoup de vigueur. Le médecin *Hostmann* (1), qui en 1840 visita les nègres d'Auca, observa parmi eux la lèpre tuberculeuse.

(1) Dr F. W. Hostmann, *Over de beschaving der Negers in Amerika.*

Jusqu'à présent, nous avons parlé uniquement de la boasie ou lèpre (*lepra arabum*, *elephantiasis græcorum*, mal rouge de Cayenne); l'éléphantiasis des Arabes (*lepra græcorum*, mal de Barbade), qui, depuis le commencement de notre siècle, s'est étendu d'une manière si alarmante, surtout dans la ville de Paramaribo, n'était alors nullement mentionné dans les actes officiels du gouvernement. Ceci ne doit pas nous étonner, puisqu'il a été suffisamment démontré que, dans le siècle précédent, cette maladie n'a point du tout, ou peut-être rarement, été observée à Surinam et à Berbice, colonie qui, alors, appartenait aux Hollandais : elle n'y fut connue qu'après 1799, époque à laquelle la colonie de Surinam fut soumise à la domination de l'Angleterre; beaucoup d'esclaves affectés d'éléphantiasis furent alors transportés de Barbade à Surinam et à Berbice (1). Le gouvernement anglais ne mit aucun obstacle à ce transport; on sait que dans les possessions anglaises, où le législateur ne reconnaît de contagion, ni dans la lèpre, ni dans l'éléphantiasis, il n'y avait alors, d'après mes recherches, pas plus qu'à présent, d'ordonnances légales.

En 1816, Surinam fut rendu aux Pays-Bas, et ce fut le 27 février que les Anglais remirent cette colonie entre les mains du gouverneur général *van Panhuys*. Ce magistrat n'était pas étranger à la colonie; auparavant, c'est-à-dire sous la domination anglaise, il avait été conseiller de la Cour de justice, mais il avait quitté la colonie en 1813 : il avait donc eu l'occasion de connaître la triste condition dans laquelle la population se trouvait par rapport à la lèpre. Peu de temps après qu'il fut arrivé à la tête des affaires, il proposa de nouvelles mesures, que nous trouvons dans les comptes rendus des séances de la Cour de police et de justice criminelle (2), et dont nous donnons la traduction suivante :

« Malgré toutes les précautions sanitaires prises jusqu'ici contre l'infection progressive de la boasie (lèpre), cette mala-

(1) Voyez ce que mon père, le docteur Ch. Landré, a écrit sur cette matière, dans : *West-Indie, bydragen tot de kennis der Ned. West-Ind. Kolonien*, t. II.

(2) *Register der Ordinaire Notulen.*

die a pris une extension si grande, que plusieurs familles, tant parmi les mulâtres que parmi les juifs, et principalement parmi ces derniers, en ont été atteintes. Je crois qu'il est absolument nécessaire de prendre, contre cette calamité, les précautions les plus efficaces, et en particulier de fonder deux établissements pour les infectés des deux nations juives, etc. »

Quoique les mesures proposées par le gouverneur n'aient pu être exécutées, il est évident que pendant l'interrègne anglais, la lèpre s'est étendue au milieu de la population libre. Il faut cependant que je fasse remarquer qu'il ne fut ici parlé que de la boasie, mais que malgré cela on doit penser à l'éléphantiasis des Arabes, qui s'était répandu rapidement dans la ville, surtout parmi les israélites et les mulâtres.

Le gouverneur *de Veer*, voyant que l'établissement de Voorzorg ne remplissait plus le but que l'on en attendait, depuis que l'on avait découvert que plusieurs des lépreux s'étaient évadés pour se rendre dans les plantations et vers la ville de Paramaribo, résolut, en 1823, de faire transporter l'établissement au Coppename, fleuve le long duquel on ne rencontre que quelques villages indiens : au reste, cette contrée est entièrement inhabitée et séparée de toute les autres parties de la colonie, avec lesquelles la communication ne pourrait avoir lieu que par mer.

Le 6 juin 1823, quarante-trois nègres de l'établissement Voorzorg, qui étaient capables de travailler, furent envoyés au Coppename pour défricher le terrain, auquel on a donné le nom de Batavia, et le préparer pour l'établissement de la nouvelle léproserie. Le 17 décembre, la cour de police et de justice criminelle reçut communication que la plus grande partie des nègres infectés ou contagieux (noms par lesquels les lépreux sont souvent désignés dans la colonie) avait été transportée de Voorzorg à Batavia.

Peu de temps après leur transport à l'établissement Batavia, le bruit se répandit qu'il y avait parmi eux beaucoup de nègres qui n'étaient pas lépreux. Il paraît que ce fut l'envoi des quarante-trois nègres pour défricher le terrain destiné à la nouvelle léproserie qui donna lieu à ce bruit. Plusieurs de ces

nègres avaient travaillé dans différentes plantations avant d'avoir été relégués dans le lieu de séparation ; c'est pourquoi il ne faut pas s'étonner si les maîtres, s'imaginant que leurs esclaves n'étaient pas lépreux, voyaient avec regret cette perte de leurs forces actives. Ce bruit se répandant de plus en plus, donna lieu à plusieurs plaintes contre l'administration coloniale, de la part des propriétaires des esclaves, réclamant au gouvernement de la mère patrie plusieurs esclaves, qui se trouvaient à la léproserie. Pour faire cesser ces griefs, le gouvernement hollandais ordonna en 1827 une réinspection exacte de toutes les personnes que renfermait l'établissement Batavia, afin de pouvoir se convaincre si les plaintes des propriétaires étaient réellement fondées.

Le gouverneur *de Veer* chargea de cette inspection une commission spéciale, composée de quatre personnes, dont deux étaient des médecins. Quoique, parmi les deux cents individus et plus qui furent soumis à la visite, il s'en trouvât dix chez lesquels on n'observa pas le moindre symptôme de la lèpre, la commission décida de déclarer toutes les personnes qui se trouvaient à la léproserie « en état de contagion ». Par conséquent aucun des esclaves de l'établissement ne fut rendu aux propriétaires ; nous verrons cependant que déjà en 1830, sous le gouverneur suivant, il fut pris des mesures afin de prévenir dans la suite de pareilles plaintes.

Le contre-amiral *Cantzlaar*, ayant été nommé gouverneur général de la colonie, publia le 7 septembre 1830 une ordonnance « contenant de nouvelles mesures pour vaincre cette fatale maladie et en empêcher l'extension. » Cette ordonnance était la première publication officielle dans laquelle il fût fait mention de l'éléphantiasis des Arabes. A l'article premier (1) nous lisons, que tous les individus non libres « reconnus atta» qués de lèpre, vulgairement appelée à Surinam boasie, ou » d'éléphantiasis, maladie qui est de la même nature, quand

(1) Cette publication est actuellement encore en vigueur à Surinam. Cette loi a été amendée et amplifiée par les publications de 1831, n° 12, 1834, n° 7, 1845, n° 13, 1853, n° 2, et 1855, n° 8.

» elle a atteint le haut degré qui la rend contagieuse », seront transportés à l'établissement Batavia.

Par les articles 2 et 3, l'obligation est imposée aux propriétaires des esclaves, s'ils soupçonnent que l'un d'eux soit atteint de cette maladie, d'en donner avis au chef de la police, sous peine d'amende.

Aux articles 5 et 6, il est arrêté, qu'à tous les individus libres, atteints de la lèpre, sera imposée avec rigueur l'obligation de s'isoler de la société, de manière à se tenir à l'écart de tout le monde, et qu'il ne leur sera pas permis de quitter leur demeure; que dans le cas où ils seraient trouvés sur la voie publique, ils seront soumis à la visite de la commission sanitaire, et transportés à l'établissement Batavia s'ils sont reconnus lépreux.

Aussitôt après que cette loi eut été mise en vigueur, elle fut rigoureusement maintenue, et aussi soutenue par les habitants, soit par conviction de l'utilité de ces mesures, soit par crainte du châtiment. Ainsi, par exemple, nous voyons que, dans l'année 1831, 320 personnes furent présentées à la commission sanitaire, dont 194 furent reconnues lépreuses; ce chiffre ne s'est jamais élevé aussi haut dans la suite, ainsi que nous le verrons plus tard.

Plus tard cependant ces règlements ne furent pas seulement contrevenus par les habitants, mais la police, les instituteurs, les médecins et la commission sanitaire chargée de l'inspection des lépreux, s'occupèrent bientôt moins soigneusement des devoirs qui leur étaient imposés, ou même les négligèrent entièrement.

Le gouvernement colonial même ne mit jamais à exécution ce qui était prescrit aux art. 35-39, au sujet des personnes saines, ou guéries de la maladie, et des enfants sains nés de parents lépreux, que l'on pourrait trouver à l'établissement Batavia, et qui devraient être éloignés de la léproserie et renvoyés dans la société, bien que les rapports annuels présentés au gouvernement démontrassent qu'il s'y trouvait un grand nombre de personnes saines.

Vers l'année 1844, le bruit courut qu'il y avait des enfants

infectés dans les écoles de la ville de Paramaribo, que fréquentaient seulement les enfants de la classe libre; le collége médical adressa au gouverneur, par la missive du 17 février 1845, des propositions à ce sujet. Il en résulta qu'une ordonnance fut publiée le 9 avril de la même année, contenant « des » mesures de précaution pour éloigner les maladies cutanées » contagieuses des écoles » (1). Ces mesures n'étaient pas prises en vain : dans la suite, presque annuellement, plusieurs enfants, chez lesquels on découvrit les premiers symptômes de la lèpre (2), devaient être éloignés des écoles.

Le collége médical se convainquit de plus en plus que les lois en vigueur exigeaient à beaucoup d'autres égards des réformes; et il résolut de faire connaître ses réflexions au gouvernement colonial, et d'y ajouter les propositions qu'il jugerait nécessaires. Mon père, qui fut chargé de la direction de cette affaire, exposa dans un rapport détaillé les causes qui, d'après lui, avaient provoqué l'extension de la maladie (3).

Le gouverneur, pénétré de la justesse des raisonnements allégués par le collége médical, envoya ce rapport au ministre des colonies dans la mère patrie : il insistait pour qu'il fût pris des mesures plus efficaces que toutes les précédentes. Le ministre remit l'affaire entre les mains de l'Institut royal des Pays-Bas, et invita cette Société savante à lui faire part de ses considérations :

1° Sur ce qu'on pourrait faire pour guérir la lèpre et pour arrêter son extension;

2° Sur les moyens à employer contre la transmission de cette maladie en Europe.

L'Institut royal exposa dans un rapport détaillé les mesures que le gouvernement devait prendre. Pour ne pas trop étendre

(1) Cette ordonnance a été amendée par les publications de 1847, n° 7, et de 1853, n° 3.

(2) Parmi les symptômes les plus saillants au début de la maladie, il faut signaler les taches de psoriasis, de préférence dans les régions fessière et fémorale.

(3) Voyez : *Rapport van de geneeskundige commissie*, d.d. 28 jan., 1847, n° 13 (*Nederl. prakt. Tijdschrift voor Geneesk.*, 1849, bl. 554).

notre récit historique, nous renvoyons aux rapports sur la lèpre à Surinam, présentés au département des colonies, le 16 décembre 1847 et le 5 mars 1851, par la 1re classe de l'Institut royal des Pays-Bas (1).

Néanmoins, les résultats de ces travaux n'aboutirent à aucun résultat; les amendements proposés dans les rapports ne furent pas effectués : un médecin fut seulement institué pour le service spécial de la léproserie, qui jusqu'alors en avait été privée. C'est depuis cette époque qu'on a pu obtenir des statistiques plus détaillées; mais quant aux expériences thérapeutiques, dont il était question dans un des rapports, elles n'ont pas, que je sache, été faites.

Comme je l'ai déjà remarqué, la commission sanitaire spécialement chargée de la visite des personnes soupçonnées d'être atteintes de la lèpre, commençait déjà peu de temps après la publication de la loi de 1830, à négliger les devoirs qui lui étaient imposés. Par les articles 10 et 11 de cette loi il est prescrit à la commission qu'elle visitera soigneusement chaque individu qui lui sera présenté, et qu'elle exposera amplement les motifs qui l'auront déterminée à le déclarer suspect ou non, et d'en faire mention dans le registre tenu par elle. Cependant ces articles ne furent jamais observés; aucun détail sur la différente nature de la maladie, ou sur les symptômes observés chez les personnes soumises à la visite, ne fut inséré dans le registre, quoique cela eût été rigoureusement prescrit par la loi. Il en résulta qu'après un certain laps de temps on ne put jamais constater si la maladie, chez les personnes que l'on avait jugées suspectes, s'était aggravée ou améliorée, et par conséquent on se prononçait légèrement sur le bannissement des personnes de la société. Ce fut en 1856, sur les instances de mon père, lorsqu'il fut nommé médecin en chef, chargé du service de santé dans la ville de Paramaribo, qu'il fut mis fin à cette excessive négligence, et il fut bientôt prouvé combien cela était nécessaire, entre autres par le fait suivant :

Le sieur P..., âgé de cinquante ans, chez lequel des mar-

(1) *Tydschrift voor wis-en Natuurkundige Wetenschappen, uitgeg. door de eerste kl. van het Koninkl. Ned. Instituut*, dl. IV, bl. 233.

ques distinctes démontraient qu'il avait été affecté de la lèpre dans sa jeunesse (il avait déjà perdu plusieurs phalanges de la main droite), fut déclaré lépreux par la Commission en 1851. En 1856, il demanda à être visité de nouveau par la Commission, et fonda cette demande sur l'illégitimité de la décision antérieure. — Alors, après des visites répétées, on put conclure des annotations faites dans le registre, que la maladie s'était arrêtée dans sa marche; le sieur P... retourna donc en 1858, pour cause de guérison spontanée, dans la société, après en avoir été banni pendant sept années. C'était la première fois, quoique beaucoup trop tard, qu'il était question dans la Commission de guérison spontanée. Lors de l'émancipation des esclaves, plusieurs décisions semblables ont été prononcées.

Le 1er juillet 1863, l'émancipation des esclaves eut lieu, et c'est par elle qu'on a pu connaître pour la première fois le nombre total des esclaves lépreux dans la colonie. Tous les esclaves furent alors soumis à une inspection, parce qu'à l'article 9 de la loi du 8 août 1862 (Staatsblad, n° 164), il avait été arrêté « qu'aucune indemnité ne sera donnée pour les esclaves qui, pour cause d'infection, sont ou doivent être isolés de la société. » C'est à cette époque que je terminerai l'histoire de la lèpre à Surinam; mais je tâcherai de l'éclaircir par quelques données statistiques.

Le nombre exact des lépreux dans la colonie n'a jamais été connu. Les premières données à ce sujet ont été publiées par mon père, dans le rapport du collége médical du 28 janvier 1847, dont nous avons déjà fait mention, et plus tard elles ont été continuées par lui. Elles contiennent l'énumération des individus qui, depuis 1831-1859, furent présentés à la commission sanitaire (1).

Dans l'année	1831	présentés	320,	reconnus lépreux	194
—	1832	—	193,	—	53
—	1833	—	100,	—	35
—	1834	—	73,	—	32

(1) Parmi les individus présentés, sont aussi compris ceux qui ont été désignés comme suspects, et qui, pour cela, sont revenus une ou plusieurs fois devant la Commission.

Dans l'année	1835	présentés	78,	reconnus lépreux	31
—	1836	—	103,	—	41
—	1837	—	85,	—	48
—	1838	—	85,	—	52
—	1839	—	192,	—	66
—	1840	—	189,	—	63
—	1841	—	121,	—	53
—	1842	—	123,	—	51
—	1843	—	172,	—	59
—	1844	—	82,	—	27
—	1845	—	81,	—	48
—	1846	—	126,	—	57
—	1847	—	224,	—	150
—	1848	—	68,	—	29
—	1849	—	100,	—	49
—	1850	—	44,	—	26
—	1851	—	46,	—	24
—	1852	—	63,	—	37
—	1853	—	53,	—	24
—	1854	—	45,	—	18
—	1855	—	47,	—	22
—	1856	—	34,	—	12
—	1857	—	79,	—	26
—	1858	—	31,	—	6
—	1859	—	32,	—	11

Ce tableau nous montre que pendant 29 ans, de 1831-1859, annuellement, en moyenne 103 personnes furent présentées à la Commission, dont 46 furent reconnues infectées. Nous voyons en outre que le nombre des personnes présentées à la Commission a de plus en plus diminué. Mais, comme dans le registre d'où ces chiffres ont été tirés, il n'était pas fait mention des conditions dans lesquelles les malades se trouvaient, ni même s'ils étaient atteints d'*elephantiasis arabum* (dont le nombre était cependant très-petit), ou bien d'*elephantiasis græcorum* (*lepra atrabum*), ces données statistiques ne pouvaient être que très-défectueuses.

Dès l'année 1856, les symptômes que l'on observa chez les personnes présentées à la Commission furent enregistrés avec précision, de sorte que, depuis cette époque, on pourra donner un aperçu détaillé, qui ne permettra pas seulement d'apprécier le nombre des malades atteints d'*elephantiasis*

arabum et d'*elephantiasis græcorum*, mais aussi de connaître les différentes formes sous lesquelles ces maladies se présentent ordinairement à Surinam, en même temps que leurs modifications au bout d'un certain temps.

Il serait à désirer que l'on eût enregistré aussi quelques particularités concernant les conditions sociales des malades, etc., afin de pouvoir en déduire quelques conséquences sur l'étiologie de ces maladies.

Je suis aussi à même de donner l'aperçu suivant sur le rapport qui existe dans les années 1856-1859, entre le nombre des malades, qui furent reconnus atteints de l'*elephantiasis arabum* dans les hauts degrés de cette maladie, et celui des individus attaqués d'*elephantiasis græcorum*.

ANNÉES.	Nombres des personnes présentées.	RECONNUES INFECTÉES DE			DÉSIGNÉES SUSPECTES DE		
		Elephant. Græc.	Elephant. Arabum.	Total.	Elephant. Græc.	Elephant. Arabum.	Total.
1856.....	31	8	4	12	8	8	16
1857.....	79	20	6	26	31	12	43
1858.....	31	6	»	6	17	»	17
1859.. ..	32	11	»	11	9	3	12

Parmi les cas mentionnés dans cet aperçu, on en compta quatre dans lesquels l'*elephantiasis græcorum* et l'*elephantiasis arabum* furent observés simultanément chez la même personne, sans qu'une de ces maladies parût avoir eu la moindre influence sur la marche de l'autre. Le nom d'*elephantiasis paralytica*, que *Duchassaing* donne à cette complication, doit en conséquence être désapprouvé.

Il faut aussi remarquer que les malades cités dans ce tableau comme affectés d'*elephantiasis arabum*, appartenaient pour la plupart à la classe libre, et, par conséquent, n'ont pas été envoyés à la léproserie; le nombre de ceux qui étaient atteints d'*elephantiasis arabum* y a toujours été relativement très-petit.

Quant à l'établissement Batavia, je puis donner les statistiques suivantes :

En 1851, le docteur *Deutschbein* (1), de 461 individus qui se trouvaient à l'établissement, en soumit 433 à une inspection, 28 ne furent pas visités.

121 (de ces 433, ainsi, plus de la quatrième partie) étaient nés à l'établissement, soit de parents lépreux, soit de parents sains; ces derniers, cependant, moins nombreux, et avaient eu presque tous des aïeux lépreux.

Parmi 100 individus (de ces 121 qui étaient nés à l'établissement, savoir : 16 hommes, 28 femmes, 38 garçons et 39 filles), il trouva : 54 sains, 46 atteints d'autres maladies; et, parmi les 21 restants, il n'en trouva qu'un petit nombre qui fussent réellement lépreux, les autres suspects.

Le docteur *Ooykoas* (2) fournit le dénombrement suivant de l'établissement en 1853 :

Nombre total, 448 (216 hommes et 232 femmes), parmi lesquels 112 (104 sains et 8 attaqués d'autres maladies) n'étaient pas lépreux, 315 lépreux et 21 suspects. Je n'ai pas besoin de répéter que presque tous ces non-lépreux avaient eu aussi des parents ou des aïeux lépreux.

Grâce à l'obligeance du docteur *F.-A.-C. Dumontier*, je suis à même de produire la statistique (3) suivante :

En mai 1857, l'état de la population à l'établissement était le suivant :

Total des individus : 386, dont 245 étaient lépreux, 26 suspects et 115 sains.

De ces 245 individus lépreux, 24 étaient nés de parents lépreux (savoir : 7 de père et de mère, 3 seulement de père, et 14 de mère seulement lépreux), tandis qu'aucune de ces 245 personnes ne se rappelait avoir eu des aïeuls lépreux.

De ces 386 personnes, 145 étaient nées dans les établissements des lépreux. Parmi ces 145, on en constata : 12 atteintes de lèpre, 20 suspectes et 113 saines.

(1) *Weekblad voor Geneesk.*, 1852, bl. 95.

(2) *Verslagen der Koninkl. Acad. Jaargang*, 1854.

(3) Dans l'édition hollandaise se trouvent quelques inexactitudes dans les chiffres, que je m'empresse de rectifier.

Parmi les 115 personnes saines (2 étaient nées hors de l'établissement), il y en avait 78 (1) qui avaient eu un père, une mère ou des aïeuls lépreux (c'est-à-dire 30 le père et la mère, 18 seulement le père et 30 seulement la mère, tandis que 8 d'entre eux avaient eu les aïeuls lépreux du côté de la mère et 7 les aïeuls du côté du père). De ces 78 personnes, 16 avaient été, depuis plus d'un tiers de siècle, exemptes de lèpre.

Parmi les 245 lépreux, 12 étaient nés à l'établissement, 158 à Surinam, et 75 dans d'autres pays.

Les chiffres suivants donnent un aperçu des naissances, des décès et des réceptions à la léproserie, pendant les dix années suivantes.

ANNÉES.	NAISSANCES.			RÉCEPTIONS.				DÉCÈS.				POPULATION ENTIÈRE A LA FIN DE L'ANNÉE.		
	Garçons.	Filles.	Total.	Hommes.	Femmes.	Garçons.	Filles.	Total.	Hommes.	Femmes.	Garçons.	Filles.	Total.	Hommes.	Femmes.	Total.
1857	7	5	12	7	5	2	»	14	9	7	4	3	23	164	218	382
1858	2	6	8	4	3	»	»	7	8	6	1	»	15	161	221	382
1859	8	9	17	5	6	»	»	11	11	8	2	2	23	163	224	387
1860	6	8	14	9	4	»	»	13	12	13	5	3	34	162	216	378
1861	2	»	2	4	2	1	»	7	18	10	10	4	42	148	204	352
1862	7	5	12	10	2	»	»	12	11	5	»	»	16	152	206	358
1863	4	5	9	25	19	3	»	47	19	12	1	2	34	162	214	376
1864	2	4	6	10	5	»	»	15	17	6	4	5	32	(*)	»	»
1865	1	2	3	6	»	1	1	8	9	8	»	»	17	»	»	»
1866	2	4	6	9	10	1	»	20	6	8	2	1	17	»	»	»
	41	48	89	89	56	8	1	154	120	84	29	20	253			

(1) On ne savait pas exactement l'origine des autres 37 personnes saines, car il se peut qu'elles soient nées d'individus non lépreux, mais affectés d'autres maladies, qui dans le temps, par une erreur de diagnostic, avaient été envoyées à l'établissement comme lépreux ; on n'a pas pu savoir d'une manière certaine si elles avaient eu des aïeux lépreux, ce qui cependant est plus que probable pour la plupart.

(*) Dans les années 1864 et 1866, plusieurs individus, ayant été reconnus sains, sont rentrés dans la société.

Ainsi, en termes moyens, les naissances s'élevaient annuellement à 8,9; les réceptions de personnes nouvellement arrivées à la léproserie, à 15,4, les décès à 25,3. L'accroissement 24,3 (naissances et réceptions) fut donc à peu près égal au décroissement (25,3).

Les décès surpassent, en outre, de beaucoup les naissances, ce qui ne doit pas tant être attribué à la diminution de la faculté de procréation chez les lépreux, qu'à la circonstance que les lépreux ne sont envoyés à la léproserie que quand ils sont dans un stade très-avancé de la maladie, et considérés par leurs maîtres comme incapables de travailler.

Enfin, je puis donner un aperçu important et très-exact du nombre des esclaves lépreux, dans la colonie, au 1er janvier 1863.

Le nombre total des esclaves qui se trouvaient, à cette époque, dans la colonie, s'élevait à.	33 560
A la léproserie.	362
TOTAL.	33 922

De ces 362, quelque temps après, 89 ayant été reconnus sains, rentrèrent dans la société; il resta donc 273 lépreux à la léproserie.

Parmi les 33 560 esclaves, 107 furent reconnus comme lépreux, de sorte que le total des esclaves infectés (=273+107) s'élevait à 380, c'est-à-dire 1,1 pour 100.

On voit, en même temps, qu'il ne s'était pas trouvé moins de 89 individus sains à l'établissement Batavia.

Le nombre des malades qui se trouvaient parmi les 21 987 personnes libres est inconnu; mais, assurément, il n'était pas inférieur à celui des esclaves.

CHAPITRE II

CONSIDÉRATIONS SUR L'ÉTIOLOGIE DE LA LÈPRE

Les ténèbres qui toujours ont enveloppé la connaissance de la lèpre n'ont été nulle part plus profondes que sur la partie qui a rapport à son étiologie; car ce n'est pas une tâche facile que de se prononcer sur des causes qui donnent naissance à une maladie qui, depuis des temps très-reculés, a régné dans la plupart des pays, et qui, finalement, a disparu dans quelques endroits sans qu'on ait pu indiquer des faits exacts concernant sa dispersion et sa disparition. Cette tâche devient encore plus pénible quand on considère combien il est difficile de juger du développement autochthone d'une maladie, lorsque celle-ci se rencontre presque dans toutes les parties de la terre, sous les conditions les plus variées pour ce qui regarde le sol, le climat, les aliments, etc.; quand on pense combien grande est la difficulté d'obtenir quelque certitude à l'égard de la contagion, lorsque la possibilité d'un développement spontané, l'hérédité et la prédisposition individuelle rivalisent entre elles pour couvrir autant que possible d'un voile épais cette question si importante.

Si, en outre, on considère que la lèpre n'a presque jamais été observée congénitale, et qu'en conséquence la question sur l'hérédité ou la non-hérédité devient extrêmement difficile, alors il n'est pas étonnant que, aussi bien dans les temps les plus reculés qu'aujourd'hui, les notions les plus différentes et les plus étranges sur la dispersion aient vu le jour.

On doit aussi envisager comme une complication aggravante que la maladie est endémique surtout parmi les peuples

moins civilisés, et que par cela même une recherche rigoureuse est presque toujours impossible.

Dans l'examen des causes de la lèpre, il faut distinguer trois termes différents :

1° L'hérédité ;

2° L'origine autochthone ;

3° La contagion.

Je ne crois pas qu'il soit préférable de traiter ces trois questions séparément, parce que plusieurs auteurs qui ont écrit sur cette matière, ont souvent cru voir une liaison entre ces trois différentes causes ; et aussi parce qu'en les traitant séparément on tomberait dans des répétitions inutiles, et on finirait par embrouiller la question.

En premier lieu, nous jetterons un coup d'œil sur le nord de l'Europe, qui est tourmenté d'une manière bien affligeante par ce fléau, mais qui, en même temps, possède beaucoup d'excellents observateurs, remplis de zèle pour cette cause si importante, et qui, les premiers, ont répandu une clarté scientifique sur cette maladie.

DE LA LÈPRE DANS LE NORD DE L'EUROPE.

Danielssen et *Boeck* (1), desquels on peut dire qu'ils ont les premiers fait des études suivies et approfondies sur cette maladie, et ont pu acquérir une riche expérience dans d'excellents hôpitaux, déclarent l'hérédité comme une des principales causes de la dispersion de la lèpre ou spédalskhed, sans qu'une cause occasionnelle quelconque ait besoin d'y coopérer.

Ils tâchent de confirmer leur opinion par une statistique, rédigée sur 213 malades, dans l'hôpital de Saint-Georges, à Bergen. Afin de mieux pouvoir en juger, je reproduis ici cette statistique en entier :

(1) *Traité de la Spédalskhed.* Paris, 1848.

Hérédité chez les spédalsques tuberculés à l'hôpital de Saint-Georges.

NOMBRE TOTAL des spédalsques tuberculés.	DANS LA LIGNE DIRECTE ET DESCENDANTE.						DANS LA LIGNE COLLATÉRALE.					
	Nombre des spédalsques du côté paternel.	Nombre des spédalsques du côté maternel.	SÉRIE des générations de cette ligne des deux côtés.				Nombre des spédalsques du côté paternel.	Nombre des spédalsques du côté maternel.	SÉRIE des générations de cette ligne des deux côtés.			
			1re génération.	2e génération.	3e génération.	4e génération.			1re génération.	2e génération.	3e génération.	4e génération.
145	17	26	13	25	1	4	40	44	28	40	6	10

« Ainsi, sur 145 spédalsques tuberculés, 127 le sont devenus par hérédité. »

Hérédité chez les spédalsques anesthétiques à l'hôpital de Saint-Georges.

NOMBRE TOTAL des spédalsques tuberculés.	DANS LA LIGNE DIRECTE ET DESCENDANTE.						DANS LA LIGNE COLLALÉRALE.					
	Nombre des spédalsques du côté paternel.	Nombre des spédalsques du côté maternel.	SÉRIE. des générations de cette ligne des deux côtés.				Nombre des spédalsques du côté paternel.	Nombre des spédalsques du côté maternel.	SÉRIE des générations de cette ligne des deux côtés.			
			1re génération.	2e génération.	3e génération.	4e génération.			1re génération	2e génération.	3e génération.	4e génération.
68	12	14	7	15	»	4	12	20	10	18	1	

« Ainsi, sur 68 spédalsques anesthétiques, 58 le sont devenus par hérédité. »

Chez 185 spédalsques, ils croient donc que l'origine de la maladie était héréditaire, chez 28, spontanée. De plus, cette statistique nous démontre que l'hérédité serait plus fré-

quente dans la ligne collatérale que dans la ligne directe; que la maladie se déclarerait bien plus dans la deuxième et la quatrième génération que dans la première et la troisième. Ils affirment que si la maladie laissait la première génération intacte, elle attaquerait la plupart des membres de la deuxième génération, et celle-ci la propagerait de nouveau par hérédité. Souvent elle sauterait aussi la deuxième et la troisième génération, pour dominer plus tard avec plus de vigueur dans la quatrième. Ils accusent ensuite différentes influences météoriques et telluriques, et de mauvaises conditions hygiéniques, parmi lesquelles une alimentation défectueuse, comme causes occasionnelles qui favoriseraient le développement de la maladie. Qu'il me soit permis de faire quelques remarques sur leurs assertions.

Il me semble que, dans la ligne collatérale, il ne peut être d'aucune manière question d'hérédité; et que, par conséquent, 84 (57 pour 100) des lépreux tuberculeux et 32 (57 p. 100) des lépreux anesthétiques ne prouvent rien pour l'hérédité de la spédalskhed. Les cas de lèpre dans les lignes collatérales (sans parler des lignes directes), plaident au contraire en faveur de la contagion de cette maladie : nous y reviendrons plus tard en parlant de la lèpre en Amérique.

Une propagation qui saute la première ou même les troisième et quatrième générations, dans la ligne directe et descendante (en effet, chez 30 (20 pour 100) des lépreux tuberculeux et 19 (28 p. 100) anesthétiques), n'est pas non plus encore constatée avec certitude par ces auteurs. Lorsqu'ils disent : « Quand la disposition à la spédalskhed est présente, qu'elle » soit héréditaire ou acquise, il est évident que tôt ou tard elle » se convertit en maladie, sans se comporter, ni d'après le cli» mat, ni d'après d'autres relations », et qu'ils démontrent ceci par des faits nombreux, il s'ensuit très-distinctement que si, par exemple, le grand-père et le petit-fils sont lépreux, et le fils sain, ce dernier, selon ces auteurs, ne peut avoir aucune disposition à la spédalskhed; car, selon eux, cette disposition aurait dû se prononcer.

Je puis à peine m'imaginer qu'on puisse envisager l'hérédité

de cette manière; si l'on eût présumé la prédisposition chez le fils, mais pas encore tellement développée qu'elle pût se trahir par des symptômes visibles, alors on aurait pu parler avec quelque droit d'une hérédité chez le petit-fils.

Cette statistique nous démontre ensuite (la possibilité d'hérédité étant acceptée) que l'intensité avec laquelle l'hérédité se développe est très-faible, parce que dans la ligne directe descendante on remarque très-peu de lépreux dans la première génération, la plupart, au contraire, dans la deuxième.

Une hérédité dans les troisième et quatrième générations devrait bien être considérée de plus près, quand on réfléchit que les mariages avec des personnes saines par cette suite de générations, doivent entraver d'une manière évidente l'hérédité de ces mêmes conditions anormales.

Dans chaque maladie, même quand elle n'est basée que sur un développement autochthone, on obtiendra une statistique pareille, et c'est pourquoi il n'est pas permis d'en déduire la dispersion d'une maladie par une transmission héréditaire.

Il serait à désirer que *Danielssen* et *Boeck* eussent mentionné si les spédalsques, dans la ligne descendante, étaient nés avant ou après que la lèpre se fut déclarée chez leurs parents ou chez leurs ancêtres; pour ne rien dire sur les chances qu'on a de se tromper, quand des individus (surtout dans les classes inférieures de la société) doivent se rappeler si leurs bisaïeuls ou leurs trisaïeuls ont été lépreux avant ou après qu'ils ont procréé leurs descendants. J'ai cru devoir faire cette remarque, parce que la lèpre se déclare souvent en Norwége à un âge plus avancé (de la trentième à la soixantième année de la vie), et parce qu'il paraîtra plus tard des communications de *Holmsen*, qui rapportent que de douze lépreux, il n'en restait, après un examen exact, qu'un seul de qui il pût être démontré que les parents ou les ancêtres avaient été lépreux avant sa naissance.

Je crois qu'il y a une raison naturelle (pourvu qu'on accepte la contagion ou le développement spontané) pour expliquer le grand nombre des lépreux dans la deuxième génération : on n'a qu'à remarquer que, puisque chaque individu a quatre

aïeuls, et seulement deux parents, la chance de rencontrer la maladie chez les aïeuls est une fois plus grande que de la trouver chez son père ou sa mère. Le nombre des lépreux dans la troisième et la quatrième génération est si petit dans ces statistiques, qu'on peut difficilement déduire de ces chiffres un rapport mutuel.

Mais *Danielssen* et *Boeck* n'admettent point la contagion; ils disent à ce sujet : « Parmi la foule des spédalsques, que nous » avons observés par centaines, et que nous avons journelle- » ment fréquentés, il n'existe pas un seul exemple que le » mal se soit étendu par la contagion; nous connaissons beau- coup de mariés, dont l'un a été spédalsque, qui ont vécu » beaucoup d'années ensemble et conjugalement, sans que » l'autre ait été attaqué de la maladie. De même à l'hôpital » de Saint-Georges, il a vécu beaucoup d'individus sains en » compagnie de spédalsques, plus de trente ans, sans être » affectés de cette maladie. C'est aussi, en vérité, un bonheur » pour notre pays que la spédalskhed n'y soit pas contagieuse; » car s'il en eût été autrement, elle aurait fait un bien » plus grand nombre de victimes. D'après nos observations, » nous ne pouvons que nier la contagion de la spédalskhed. »

Il ne reste donc que la possibilité d'un développement spontané : ils virent plusieurs fois ce développement spontané chez des individus nés de parents sains, dans la famille desquels la lèpre n'avait jamais été observée, qui étaient nés ailleurs, mais qui avaient séjourné pendant un laps de temps plus ou moins long dans des endroits où la lèpre était endémique, et dans des conditions favorables au développement de la maladie. Si donc l'origine autochthone n'est point exclue par eux, il serait possible que chez les individus mentionnés dans leurs statistiques, la lèpre ne dût pas son origine à l'hérédité, mais à un développement spontané. En outre, ceci serait possible, parce que la plus grande partie de ces individus se trouvaient dans les mêmes conditions que celles de leurs parents ou de leurs ancêtres, et qu'il est démontré par ces mêmes tableaux, que le nombre de ces cas de développement spontané (y compris ceux de la ligne collatérale) est très-considérable, c'est-

à-dire 102 (70 pour 100) des lépreux tuberculeux, et 42 (62 pour 100) des anesthétiques.

Si, avec la contagion, ces auteurs avaient aussi exclu le développement spontané, leur assertion se serait présentée sous un tout autre aspect.

Mais même leur décision positive à l'égard de la contagion, est sujette à quelque doute. N'est-il pas possible que ces cas de développement spontané qu'ils crurent avoir observés, aient été précisément occasionnés par la contagion; — surtout quand on considère que le développement spontané eut lieu dans des endroits où la maladie était endémique,—et que pour les autres cas, dans lesquels la communication avec les lépreux n'eut aucun effet facheux, les conditions pour être infecté manquèrent?

Le gouvernement norwégien, guidé par l'avis de ces deux célèbres auteurs, et convaincu de l'hérédité de la spédalskhed, fondait là-dessus toutes ses mesures contre la dispersion de ce fléau; mais justement à cause de cela, il rencontra un adversaire redoutable dans le docteur *Hjort*. Celui-ci soutenait : que l'hérédité jouait un rôle subordonné parmi les causes qui favorisaient le développement et la dispersion de la spédalskhed, mais que, au contraire, les influences extérieures, pouvant seules occasionner cette maladie, devaient être considérées comme les causes les plus importantes de son extension, et que, en conséquence, les mesures prises par le gouvernement norwégien étaient inutiles et devaient être désapprouvées.

Le professeur *Conradi* (1) tâcha de réfuter *Hjort*, et lui signala l'observation du docteur *Hjatelin*, à l'île d'Islande, par laquelle il fut démontré que, en 1837, parmi 125 lépreux, à peine un seul pouvait être indiqué qui n'appartint pas à une famille atteinte de la lèpre; il prétendit ensuite : que la plupart des médecins suédois affirment que les cas dans lesquels la lèpre se montre indépendante, sans être la suite de l'hérédité, sont rares. Il soutint que les statistiques de *Danielssen* et

(1) *Norsk Magazin*, 1857, 4.

Boeck prouvent que, parmi 130 malades, 94 (72 pour 100) doivent être censés appartenir à des familles dans lesquelles la spédalskhed est manifeste : chez 35 (27 pour 100) de ces malades, la maladie s'était propagée dans la ligne directe; chez 59 (45 pour 100) dans la ligne collatérale, et chez 36 (28 pour 100), elle s'était développée spontanément; qu'en outre la statistique de *Danielssen* et *Boeck* (que nous avons produite à la page 21) plaide pour l'hérédité; que les tableaux de *Danielssen* et *Læberg* font mention de 114 spédalsques parmi lesquels 82 (72 pour 100) descendent de familles lépreuses (savoir, 46 (40 pour 100) en ligne directe, 36 (32 pour 100) en ligne collatérale), et que chez 32 (28 pour 100), la maladie s'est développée spontanément.

Conradi s'en rapporta ensuite : 1° à un tableau qu'il avait dressé lui-même, d'où il résultait que, de 114 cas, 62 (54 pour 100) appartenaient à des familles lépreuses (1); 2° aux tableaux de Hoëgh, qui observa 34 individus attaqués, dont 15 (44 pour 100) descendaient de lépreux, savoir : 11 (32 pour 100) en ligne directe, 4 (12 pour 100) en ligne collatérale, tandis que chez 19 (56 pour 100), un développement autochthone avait eu lieu; 3° aux chiffres de *Holmsen*, concernant 93 spédalsques, dont 55 (59 pour 100) étaient nés de lépreux [12 (13 pour 100) en ligne directe, 43 (46 pour 100) en ligne collatérale], et parmi lesquels, chez 38 (41 pour 100), la maladie s'était développée spontanément.

Il allégua de plus, comme argument en faveur de la transmission héréditaire de la lèpre, la disparition de ce fléau durant le moyen âge, par la fondation de léproseries.

La réfutation du professeur *Conradi*, à l'aide des chiffres qu'il produisit, ne peut nullement être considérée comme définitive. Je dois faire ici quelques-unes des remarques que j'ai déjà alléguées lorsque je parlais des statistiques de *Danielssen* et *Boeck*. Dans l'observation du docteur *Hjatelin*, il n'est pas dit si dans le chiffre donné sont aussi compris les lépreux de la ligne collatérale ; il est donc difficile de juger de la justesse

(1) Je ne trouve pas mentionné le nombre dans la ligne directe et collatérale.

de ses arguments. Aux chiffres de *Boeck* sont applicables les mêmes remarques que j'ai déjà faites sur la statistique de *Danielssen* et *Boeck;* elles sont de même applicables à celles de *Danielssen*, de *Læberg* et de *Hoëgh*.

Pour ce qui regarde le dernier argument de Conradi, que la lèpre diminua à mesure qu'on érigea des léproseries, il pourrait plaider également en faveur de la contagion de la spédalskhed.

Holmsen (1) révoqua fort en doute, et de plein droit, l'hérédité de cette maladie, en raison de ses statistiques de la lèpre norwégienne. Ses observations portaient, comme nous l'avons vu, sur 93 malades (43 hommes et 50 femmes). Parmi ces 93 malades, il ne s'en trouvait pas plus de 12 (13 pour 100) chez le père et la mère ou les aïeuls desquels la lèpre se fût présentée en ligne directe. Donc, pour prouver l'hérédité, dit *Holmsen*, — et je suis entièrement du même avis —, on ne peut seulement s'en rapporter qu'à ces 12 individus, qui ont eu des parents ou des aïeuls lépreux.

En continuant ces recherches, il trouva qu'il n'y avait qu'un seul individu qui eût été dans la possibilité d'hériter de la maladie de ses parents ou de ses aïeuls, attendu que les parents et les aïeux des 11 autres n'avaient été attaqués par la spédalskhed qu'après la naissance de leurs enfants. Il en tire donc la conclusion qu'on ne peut pas attacher à l'hérédité toute l'importance qu'on a généralement coutume de lui donner. *Holmsen* produit ensuite comme arguments, pour soutenir son opinion : que la lèpre acquiert quelquefois, dans certains endroits, un caractère épidémique, tandis qu'elle diminue rapidement dans d'autres endroits, la population restant la même; et qu'on rencontre si rarement la spédalskhed dans le jeune âge, c'est-à-dire ordinairement pas avant l'âge de vingt à quarante ans. Par ces raisonnements, il se convainquit à la

(1) *Norsk Magazin*, 1857, Heft 3. — N'oublions pas de dire que dans différentes citations que j'ai trouvées ailleurs, il y a eu une erreur dans les noms : les observations attribuées à *Holmsten* et *Holmeijer* sont toutes du docteur *Holmsen*, à Bergen.

fin qu'il devait exister un miasme spécifique pour la spédalskhed, qui s'exhalerait sur les côtes de la Norwége.

La statistique de Holmsen démontre évidemment que la doctrine de *Danielssen* et *Boeck* sur l'hérédité, — qui, après eux, fut considérée par tant d'autres auteurs comme un arrêt définitif, — n'est point du tout constatée par des faits.

Le docteur *Follum* exprima aussi, dans un rapport officiel de 1857, l'opinion que surtout l'influence du climat et des relations hygiéniques devait être considérée comme la cause de la spédalskhed. Il ne nie cependant pas entièrement que l'hérédité, et peut-être aussi l'infection, soient de quelque valeur; mais selon lui on exagère en y voulant voir la cause efficiente.

Kierulf, qui publia (1) un traité très-important pour la connaissance de la spédalskhed en Norwége, dans lequel il parle longuement des causes de cette maladie, considère l'hérédité comme la cause principale de la grande dispersion de ce fléau en Norwége (en 1850 : 1500; en 1862, sur une population d'à peu près 2 000 000 : 2119 lépreux). En confirmant son opinion par les statistiques de *Danielssen*, de *Boeck* et de *Conradi*, il observe en même temps que plusieurs lépreux se marient (dans un district, par exemple, de 373 lépreux, 107 étaient mariés) pour être sûrs d'être secourus plus tard en cas de besoin. Il traite ensuite des causes occasionnelles, qui font paraître la spédalskhed là où il y a une disposition héréditaire; il remarque qu'on accusait généralement comme causes du développement spontané chez les individus dont les parents étaient sains, la même influence nuisible que celle qui agit dans la disposition héréditaire. Ceci était une inconséquence. Or les habitants de la côte occidentale de la Norwége sont exposés partout aux mêmes influences pernicieuses, et, malgré cela, *le développement spontané y a toujours lieu dans des endroits où la maladie est endémique, et jamais dans des lieux où elle est inconnue*. A cause de cette absurdité, dit *Kierulf*, on a été obligé de chercher la solution de cette question dans la contagion. Aussi

(1) *Virchow's Archiv*, 1853, B. V, p. 13.

le peuple en Norwége croit-il de plus en plus à la contagion de la spédalskhed, quoique la plupart des savants soient d'une opinion contraire; le comité médical norwégien y fait cependant une exception, car ce comité ne rejette pas tout à fait la possibilité de l'existence d'un virus lépreux.

L'auteur examine ensuite les observations que *Holmsen* (1) avait faites à Fosen, desquelles il résulterait : qu'avant les cinquante dernières années, la spédalskhed était très-rare dans cette péninsule, mais qu'à présent elle sévit beaucoup sur la population de tout le district, tant sur les pêcheurs que sur les cultivateurs, tous des gens bien nourris; il en résulterait encore que la maladie s'est toujours dispersée par accumulation sur les côtes, le long des baies (fiords) et dans l'intérieur du pays.

La spédalskhed montrerait, selon *Holmsen*, quand elle est dans son état naturel et libre (?), un penchant à former des foyers séparés. Elle augmenterait dans un tel foyer par une propagation héréditaire et un développement spontané, pour se montrer brusquement, après un certain temps, dans un lieu fort éloigné du premier foyer, *cependant toujours transportée par un individu lépreux;* tandis que la maladie s'éteindrait insensiblement dans le lieu primitif. *C'est seulement dans ces foyers* que *Holmsen* observa un développement spontané (duquel nous parlerons plus tard). Jamais il n'a vu un foyer une fois éteint se former de nouveau; aussi ne croit-il pas que cela puisse avoir lieu; il remarquait toujours dans un tel foyer une augmentation ou une diminution graduelle. Quand, dans un de ces endroits, la maladie se trouvait dans la période de déclin, alors la disposition héréditaire elle-même n'était pas en état de la reproduire.

Holmsen ainsi que *Kierulf*, se fiant à ces faits, résolurent d'admettre un virus spécifique pour la spédalskhed, qui est différent de tous les autres, parce que, agissant *seul* sur l'organisme humain, il n'est pas en état de faire naître la spédalsked, mais seulement la *prédisposition* à cette maladie,

(1) *Norsk Magazin*, B. V, p. 433.

et que par conséquent il est nécessaire qu'une influence extérieure s'y joigne pour faire paraître les symptômes : cette cause occasionnelle consisterait principalement dans le refroidissement. Cette circonstance, ainsi que les relations locales (et on ne pouvait pas leur refuser toute influence, quand on voyait la spédalsked s'éteindre dans un foyer, même où il y avait disposition héréditaire), ont induit *Holmsen* à considérer la lèpre comme une maladie miasmatique, dans laquelle cependant les malades deviennent nuisibles aux personnes bien portantes (1).

Kierulf est d'avis que la spédalskhed peut se reproduire de deux manières : en premier lieu par l'hérédité, en second lieu par la contagion, toutes les deux aidées par un miasme. Cet auteur est donc, pour ce qui regarde la première proposition, à peu près du même avis que *Danielssen* et *Boeck ;* il n'en diffère que parce qu'il admet de plus une cause occasionnelle.

Je ne m'explique pas bien comment il a pu accepter la seconde proposition : c'est-à-dire qu'il ait considéré le virus seulement comme une cause prédisposante, et qu'il ait cru un miasme nécessaire comme cause occasionnelle pour pouvoir donner une explication à ses observations.

Virchow (2), invité par le gouvernement suédois, à cause de la forte opposition de quelques médecins norwégiens contre les lois proposées pour faire disparaître la lèpre, et qui toutes étaient basées sur la transmission héréditaire, se rendit en 1859 en Norwége, afin d'y observer cette maladie. Pour ce qui regarde ses recherches sur l'étiologie, il n'eut pas lieu d'en être satisfait : à la fin du chapitre dans lequel il traite cette matière, il dit, que d'après les connaissances actuelles, on ne peut pas déterminer la vraie cause de cette maladie.

(1) En parlant du docteur Holmsen, je veux en même temps lui témoigner ici ma reconnaissance pour les détails précieux, relatifs à cette maladie, que ce savant docteur a bien voulu me communiquer.

Dans sa dernière lettre, il émettait l'opinion que la spédalsked est très-probablement une maladie, *malaria sui generis*, ne pouvant pas se communiquer d'une personne malade à une personne bien portante (du moins en Norwége).

(2) *Die krankhaften Geschwülste*. Berlin, 1863, B. II.

Pourtant il est d'avis qu'un seul point est depuis longtemps hors de doute, savoir, la propagation héréditaire de la lèpre, ou pour dire plus exactement, *l'hérédité de la prédisposition*, parce que la maladie n'est presque jamais observée congénitale, mais se développe au bout d'un temps plus ou moins long. Il ajoute que cette manière de se propager est constatée par de nombreux faits, partout où la lèpre est endémique.

Mais plus tard, même lorsqu'il eut fait un appel aux médecins de différents pays, pour les inviter à faire des recherches sur la lèpre, et qu'il eut par ce moyen rassemblé beaucoup de matières à ce sujet, il avoue que ces données ne suffisent pas pour établir la cause de la lèpre.

De toutes les réponses que *Virchow* avait reçues, il parut que parmi tous les peuples et dans tous les pays, il n'y avait qu'une seule voix sur la propagation héréditaire, ou plutôt sur la fréquence de la lèpre dans quelques familles.

Des recherches récentes de *Bidenkap* ont constaté, pour la Norwége, que la prédisposition peut se transmettre par hérédité jusque dans la quatrième génération; mais en même temps il a été prouvé par les recherches de ce même auteur, et par celle de Hoëgh, que seulement pour la quatrième partie des lépreux la maladie peut être démontrée dans la ligne directe et ascendante.

Virchow, en discutant ce sujet, remarque, que c'est de la prédisposition seule que l'on peut hériter, parce que aussi *Bidenkap* ne vit jamais cette maladie se présenter, à l'exception de peu de cas, sinon à un âge plus avancé. Il croit par conséquent qu'on ne peut expliquer ce fait sans avoir recours à des causes occasionnelles spéciales : surtout parce que la lèpre chez les émigrants norwégiens dans l'Amérique du Nord, comme le docteur *J. A. Holmboe* l'a démontré (1), prend non-seulement un caractère plus bénin, mais se montre bien moins souvent chez eux, que parmi leurs compatriotes et parents qui sont restés dans leur patrie. Le grand fait histo-

(1) *Den Spedalske sygdom blandt de Norsk i Amerika*. Chikago, 1863.

rique, que la lèpre régna presque partout, mais qu'elle a disparu entièrement chez la plupart des peuples civilisés, confirme aussi *Virchow* dans son opinion.

Plus tard cependant, il dit, et avec raison, que la prédisposition héréditaire n'est pas, il s'en faut de beaucoup, la seule cause de la propagation de cette maladie, et il s'en rapporte à la statistique de *Hoëgh* et *Bidenkap*, dont nous venons de faire mention, et à celle de *van Someren*, à Madras, dans laquelle on ne rencontre parmi 31 malades, que 2 qui descendent de lépreux.

L'admission d'un virus ne peut pas être approuvée par *Virchow*, parce que dans les temps modernes on n'a pu citer un seul exemple de l'apparition de la lèpre dans les pays où cette maladie n'est pas indigène, quoique l'occasion de se trouver en contact avec des lépreux s'y présentât souvent; et qu'on n'a pas vu de cas de lèpre (sauf quelques exceptions) par suite de contagion dans les hôpitaux ou ailleurs.

Ce n'est qu'en passant que je veux remarquer ici — pour en parler plus tard d'une manière plus détaillée — que les exemples d'importation de cette maladie dans des pays auparavant exempts de lèpre ne manquent pas; et que, d'après mon opinion, *Virchow* n'attache pas assez de valeur aux observations de Holmsen dont nous venons de parler, quand il croit les pouvoir mettre de côté, en traitant l'étiologie de la lèpre en général. Ces observations, qui au premier abord paraissent de peu d'importance, pourraient précisément éclaircir la question, parce qu'elles ont été faites sur des personnes dont les relations sociales étaient connues. Il est de la plus grande importance, en traitant la question de la contagion de cette maladie, d'entrer dans des détails sociaux des plus minutieux.

Parmi les causes de la lèpre, *Virchow* paraît attacher ensuite beaucoup d'importance aux relations diététiques, et en premier lieu, malgré les objections qui furent faites de plusieurs côtés, à l'usage de poisson de mauvaise qualité. Il fait aussi mention de quelques cas de lèpre, qui se sont présentés dans les derniers temps en Angleterre et en Allemagne.

Après tout ce que nous venons de rapporter sur les différentes opinions à l'égard de la spédalskhed, il paraît évident que presque tous les médecins, dans le Nord, attachent beaucoup d'importance à la propagation héréditaire, qui seule, ou bien à l'aide d'une cause occasionnelle, serait en état de donner naissance à la maladie, et qu'ils acceptent en même temps un développement spontané, qui pourrait avoir lieu sous des différentes conditions; tandis qu'il y en a très-peu ou mieux presque pas qui ne nient point la propagation de la maladie par la contagion.

Les recensements des spédalsques, faits en Norwége, pendant les années 1862-1866, ont donné les chiffres suivants:

ANNÉE.	NOMBRE DE TOUS LES SPÉDALSQUES.	DOMICILES.		
		Dans les hospices (*)[2]	Habitants de la côte.	Habitants de l'intérieur du pays.
1862.....	2119	696	1399	24
1863.....	2155	747	1392	16
1864.....	2182	779	1385	18
1865.....	2136	770	1345	21
1866.....	2126	793	1314	19

DE LA LÈPRE AU SUD DE L'EUROPE, EN ASIE ET EN AFRIQUE.

Non-seulement dans le nord, mais aussi dans le sud de l'Europe, en Asie et en Afrique, on trouve très-peu de partisans de la contagion; les communications suivantes, faites par des médecins établis dans ces pays, le démontreront suffisamment.

Dans le Portugal, où la lèpre se présente fréquemment, on trouvait encore, en 1821, 800 lépreux, principalement parmi les classes indigentes, tant habitants des côtes que des endroits situés plus dans l'intérieur du pays (la vallée de Lafoës).

(*) Tous ces spédalsques des hospices sont arrivés des districts de la côte (spécialement des fiords), et pas un seul des districts de l'intérieur du pays.

Le docteur *B. von Kessler* (1), qui observa attentivement la lèpre dans le Portugal et dans ses colonies, rapporte que la plupart des médecins, dans la province d'Algarve, où la maladie est très-répandue, sont persuadés qu'elle n'est pas contagieuse, parce qu'ils rencontrèrent beaucoup d'hommes lépreux dont les femmes et les enfants étaient exempts de tout symptôme de la maladie. Il ajoute que d'autres lépreux vivaient dans un commerce continuel et familier avec des parents et des amis, sans qu'ils en eussent jamais aperçu des suites fâcheuses; qu'au contraire, la plupart des médecins cherchent la cause de la maladie, principalement dans des relations hygiéniques.

L'exposé sommaire des recherches des médecins qui étaient attachés à diverses époques à l'hôpital Sancto-Lazaro, à Lisbonne, est d'une plus grande importance. Le rapport le plus détaillé est du docteur Beirao (1851), qui pendant plusieurs années fut médecin-directeur de cet hôpital, et qui, après avoir examiné avec soin 43 cas de lèpre, en conclut que la lèpre n'était pas héréditaire, parce qu'à peine 5 de ces 43 lépreux étaient en état de démontrer qu'un seul membre de leurs familles eût été affecté de cette maladie; et parce que des observations ultérieures lui avaient appris qu'il n'arrivait que très-rarement qu'un lépreux pût indiquer si un de ses parents avait été atteint de la lèpre.

Beirão attache cependant beaucoup de valeur à des causes qui proviennent du climat. Et quoique, dans les contrées où la lèpre est endémique, on se nourrisse exclusivement de poisson et de crustacés de mauvaise qualité, l'auteur n'est pourtant pas disposé à considérer un aliment déterminé, par exemple celui que nous venons de nommer, comme la cause unique, et même comme une cause contribuante de la maladie, parce que :

1° Dans beaucoup de cas de lèpre, une telle alimentation n'avait pas eu lieu;

2° Dans plusieurs contrées du Portugal, beaucoup d'habitants se nourrissent presque exclusivement de poisson salé, sans qu'ils soient jamais affectés d'aucune maladie cutanée, et que même il existe en Portugal des ordres religieux, comme les Chartreux et les Carmélites, auxquels le poisson est prescrit comme seule nourriture, et qui pourtant ne sont jamais atteints de lèpre.

(1) *Virchow's Archiv*, 1865, Bd. XXXII, S. 257.

Cet auteur n'est pas non plus d'accord sur la contagion de cette maladie. Aussi, dans le Portugal, est-on, presque généralement, peu disposé en faveur de la doctrine de la contagion de la lèpre.

Un exposé très-intéressant sur la lèpre à l'île de Crète a été publié par le docteur *Smart*, médecin établi dans ce pays. L'île de Crète présente un champ étendu pour des recherches de ce genre.

En 1852, le nombre des personnes qui étaient bannies de la société ne s'élevait pas à moins de 628 (non compris plusieurs lépreux qui savaient cacher leur maladie et dont le chiffre montait au moins au tiers de tous les lépreux dans l'île), de sorte que sur une population de 250 000 âmes, à peu près 900 (savoir presque 1/250 de toute la population) étaient atteints de la lèpre.

La population de la Crète croit, ainsi que nous l'avons déjà trouvé dans le Nord, et comme il paraîtra encore bien des fois en parlant d'autres pays, que la maladie est contagieuse. *Smart* n'est pas de cet avis : il remarque que, dans les villages où se tiennent les lépreux, des femmes saines vivent avec leurs maris malades, et que malgré cela elles ne sont pas atteintes de la lèpre.

Clot-Bey assure aussi que, pendant un voyage qu'il fit dans la Crète, il n'entendit jamais dire que la maladie se fût communiquée par la contagion, quoique avec le nombre des femmes lépreuses qui se prostituent, l'occasion s'y présentât souvent.

Smart, au contraire, trouvait la transmission héréditaire plus manifeste : dans quelques familles il avait vu la lèpre se propager de cette manière dans plusieurs générations; dans quelques familles, pas un seul membre n'en était resté exempt.

La cause occasionnelle doit, selon lui, être cherchée plutôt dans les relations sociales que dans les influences du climat.

Le professeur *Rigler* (1), à Constantinople, nie aussi l'influence d'un virus lépreux, pour la plupart des cas qu'il a observés; il mentionne que les enfants de parents lépreux, quoique sains en naissant, sont attaqués le plus souvent entre leurs vingt-cinquième et quarantième années, par la maladie de leurs parents, soit par hérédité, soit par un développement spontané, tandis que les causes occasionnelles lui restèrent entièrement inconnues.

Tobler (2), à Jérusalem, et *Polak* (3), en Perse, furent de la même opinion.

Clot-Bey (4), l'ancien médecin en chef en Égypte, prétend au contraire que la lèpre n'est pas absolument héréditaire, et qu'elle ne doit pas être indispensablement transmise d'une manière ou de l'autre, et il le constate par un grand nombre d'observations qu'il a faites à cet égard. Jamais il ne vit un enfant qui fût infecté par sa nourrice; dans plusieurs ménages il vit que la maladie ne se transmit pas des personnes affectées aux personnes saines; il vit le contraire dans d'autres.

Griesinger ne put pas encore se déclarer d'une manière déterminée sur l'étiologie de la lèpre en Égypte.

Le docteur *Carl Wolf* (5) envoya à Virchow une relation détaillée concernant la propagation de la lèpre à Madère, et s'y prononça contre l'admission d'un virus, mais très-énergiquement en faveur de l'hérédité. Parmi 23 lépreux dans l'hôpital, il en trouva 3 dont les deux parents, et 9 dont le père et la mère étaient atteints de la lèpre.

La même opinon fut confirmée aux îles Canaries (6). Jusqu'à l'année 1860, la lèpre y allait en augmentant, ce qui est démontré par les chiffres suivants : dans l'année 1788 il y eut

(1) *Wiener Zeitschrift.* Febr. 1847.
(2) *Schmidt's Jahrb.*, 1857, Bd. 95, S. 253.
(3) *Schmidt's Jahrb.*, 1857, Bd. 95, S. 116.
(4) Séance de l'Académie de médecine de Paris, 20 mai 1851.
(5) *Virchow's Archiv*, 1862, Bd. XXVI, S. 44-78.
(6) *Virchow's Archiv*, 1861, Bd. XXII, S. 340.

195 lépreux; en 1831, 346; en 1857, 500; en 1860, plus de 600. Le mariage était, sur ces îles, défendu aux lépreux par des ordonnances légales, mais il y fut fait mainte infraction. Le système d'isolement, au moyen de léproseries, n'y est presque pas appliqué.

Le docteur *Domingo J. Navarro* (1), à Palmas, dans l'île Gran-Canaria, et le docteur *Bolle* (2), à Berlin, qui a séjourné pendant un long espace de temps aux îles Canaries, sont de la même opinion à l'égard de l'étiologie de la lèpre; ils virent que les riches étaient atteints de cette maladie aussi bien que les pauvres, de sorte que la mauvaise qualité des aliments ne pouvait pas en être considérée comme la seule cause.

Bazin (3), qui constate l'hérédité de la lèpre (son opinion est fondée principalement sur les statistiques de *Danielssen* et *Boeck*) et qui nie la contagion, pose la question suivante : N'y aurait-il pas une certaine liaison entre la lèpre d'un côté, et la goutte et le cancer de l'autre? Il fit cette question parce qu'il avait observé trois lépreux, qui avaient pris le germe de la maladie ailleurs, et dont les pères avaient tous souffert de la goutte et les mères du cancer.

Dans les contrées un peu plus éloignées du territoire européen, on croit davantage à la contagion, quoique presque toujours dans un sens limité; mais ceux qui maintiennent la propagation héréditaire y ont cependant toujours eu le dessus.

Dans la Chine, où la lèpre est répandue dans tout le pays (*B. Hobson*, qui y observa cette maladie, nous en donne une description détaillée (4)), existe encore aujourd'hui la même croyance sur la cause de ce fléau, qu'on trouve mentionnée dans l'Ancien Testament. Elle est en vigueur parmi le peuple,

(1) *Virchow's Archiv*, 1861, Bd. XXII, S. 362.
(2) *Virchow's Archiv*, 1861, Bd. XXII, S. 367.
(3) *Leçons théoriques et cliniques sur les affections cutanées et artificielles, et sur la lèpre, etc.* Paris, 1862.
(4) *Virchow's Archiv*, Bd. XXII, 1861, S. 326. — Ici je dois mentionner, que le docteur Dumontier, à Surinam, me communiqua qu'il avait observé la lèpre chez des émigrants chinois, importés dans cette colonie; et qu'il a pu se convaincre que les symptômes ressemblaient complétement à ceux qu'on remarque dans la lèpre à Surinam.

c'est-à-dire, qu'il croit que la maladie est contagieuse, mais qu'elle est en même temps un châtiment du ciel pour les péchés commis; à cause de cela, les Chinois fuient les lépreux. De plus, ils considèrent comme un fait constaté, que la maladie se propage par l'hérédité, que *chaque* enfant de parents lépreux reçoit le mal en partage, que cependant à la troisième génération la maladie prend un caractère plus bénin, et qu'elle s'éteint tout à fait dans la quatrième génération.

Les mariages entre les enfants de parents sains et de lépreux ne sont pas tolérés en Chine. Si la maladie se manifeste dans une famille entièrement saine, toutes les promesses et les contrats de mariage sont annulés. Seulement le contrat est valable dans le cas où les fiancés ou mariés sont dans le même degré de parenté avec leurs parents ou ancêtres lépreux, et que la maladie a atteint chez eux le même degré. Des lépreux d'une génération déterminée ne peuvent se marier qu'avec des individus de la même génération : un lépreux, par exemple, de la quatrième génération, même quand il ne porte pas le moindre indice de la maladie, ne peut épouser qu'une femme de la même génération; *les enfants issus de la quatrième génération sont considérés comme sains, et peuvent rentrer dans la société.*

Les Chinois reconnaissent, comme je l'ai déjà remarqué, la contagion de la lèpre; et la loi est basée sur ce principe.

Les lépreux pauvres sont transportés dans les léproseries; les riches sont obligés de se tenir éloignés de la société sous peine d'être conduits dans les léproseries. Mais il paraît que malgré ces mesures légales, plusieurs femmes, qui descendent de lépreux, se tiennent dans les maisons publiques à Canton, et les Chinois croient avec raison que la maladie se communique par ce moyen à d'autres personnes.

Par une connivence blâmable, le gouvernement permet que beaucoup de ces malheureux, qui sont à peine suffisamment nourris, pourvoient par la mendicité à leur entretien; il n'est donc pas nécessaire de démontrer toute l'inconséquence qu'il y a dans le système d'isolement en Chine.

Les lépreux les plus âgés du village des lépreux, près de Canton, affirment que les enfants n'héritent pas toujours de la

lèpre de leurs parents; qu'on ne rencontre point de symptômes de lèpre chez quelques femmes mariées du village; que la disparition de la maladie dans la troisième et la quatrième génération n'a pas lieu sans exceptions.

Les médecins chinois admettent la propagation héréditaire, et aussi la contagion, mais seulement par le commerce charnel.

C'est à dessein que j'ai parlé plus longuement de la lèpre en Chine, que je ne l'ai fait pour d'autres pays. Quoique j'eusse désiré que *Hobson* eût fait sa relation avec plus de clarté, il me semble qu'elle peut donner lieu à une observation très-intéressante.

J'avoue qu'on ne peut pas attacher une grande valeur à l'opinion d'un peuple peu éclairé sur l'étiologie d'une maladie, et à plus forte raison d'une maladie comme la lèpre, dont les causes sont si difficiles à reconnaître, mais on peut néanmoins, dans les pays où la lèpre se présente fréquemment, croire au suffrage du peuple, quand il s'agit de décider si quelqu'un est atteint de cette maladie, oui ou non.

Il est vrai que les Chinois ne sont nullement capables de reconnaître la maladie dans ses premiers stades, — comme on peut en juger d'après les singuliers procédés qu'ils font subir, selon les communications de *Hobson*, aux individus suspectés de lèpre —; mais on ne peut pas leur refuser quelque aptitude à distinguer la maladie dans un stade plus avancé, où elle est accompagnée de symptômes pathognomoniques bien prononcés.

Je pense donc qu'il doit y avoir un fond de vérité dans le fait qu'ils croient avoir observé, que les descendants de lépreux dans la troisième ou la quatrième génération sont presque tous sains, et peuvent rentrer dans la société.

Il y aura aussi, selon toute apparence, plusieurs personnes saines dans la première et la deuxième génération, — ce qui, selon les Chinois, ne peut pas avoir lieu —; mais les préjugés invétérés concernant l'hérédité auront fermé leurs yeux à ce sujet. Il me semble que j'ai le droit de soutenir cette assertion, parce qu'à Surinam, où régnait la même opinion qu'en

Chine, quelque chose d'analogue a eu lieu dans l'établissement Batavia, dont j'ai fait mention dans le premier chapitre. Là aussi on était tellement convaincu de l'hérédité de la maladie, qu'on n'a pas voulu permettre qu'un grand nombre d'individus, nés à cet établissement de parents ou d'aïeux lépreux, et chez lesquels on ne put trouver le moindre indice de lèpre, fussent renvoyés dans la société; on prétendait qu'ils portaient le germe de la lèpre.

Les Chinois, qui ont la lèpre en horreur, ne permettraient pas que des descendants de lépreux de la troisième et de la quatrième génération rentrassent dans la société, s'ils n'étaient pas convaincus par l'expérience de leur parfaite santé; il y a donc lieu d'admettre que leur affirmation à cet égard est basée sur de bonnes observations.

Par les conditions sociales dans lesquelles les lépreux vivent en Chine, et auxquelles ils se soumettent sans doute, ils présentent un champ d'expérience très-vaste. On doit reconnaître qu'on chercherait en vain une situation pareille dans d'autres pays.

Chaque maladie héréditaire se propagera avec d'autant plus de véhémence, que les individus affectés de cette maladie se marieront plus entre eux. Les mariages entre parents démontrent assez clairement, que dans ces cas, les conditions physiologiques et pathologiques se propagent à l'extrême, à cause d'une trop grande ressemblance des mêmes conditions chez les propagateurs. Si la lèpre était héréditaire, elle devrait s'aggraver dans la postérité ou du moins s'y reproduire avec le même caractère, en admettant que les autres conditions (comme l'alimentation, le climat, etc.), qui pourraient influencer l'origine et la marche de cette maladie, fussent restées les mêmes dans les différentes générations. C'est ce qui a eu lieu sans aucun doute dans la Chine, qui, d'habitude, ne progresse point à pas de géant.

Supposons un instant que ce soit seulement de la prédisposition que l'on hérite, et qu'il soit nécessaire qu'une cause occasionnelle survienne pour pouvoir élever la condition anomale à un assez haut degré d'intensité pour qu'on puisse lui donner

le nom de maladie, — la réflexion que nous venons de faire serait aussi applicable.

Muni de ces données, je crois pouvoir révoquer en doute l'assertion que la lèpre, en Chine, se répandrait principalement par l'hérédité.

Les médecins japonais (1) ont répondu aux questions relatives à l'étiologie, posées par *Virchow*, de la manière suivante :

a. La disposition héréditaire doit être considérée comme prouvée; quelquefois elle saute une génération;

b. L'alimentation, et surtout le poisson salé et presque décomposé, dont les Japonais se nourrissent de préférence, doivent être considérés comme favorisant le développement de la lèpre;

c. La maladie est répandue par tout le Japon, principalement dans la partie méridionale et le long des côtes, surtout dans les contrées marécageuses;

d. L'infection n'a lieu ni par le mariage, ni par le commerce charnel;

e. La maladie n'épargne aucune classe de la société, ni les riches, ni les pauvres;

f. Ils ne connaissent pas de cas de contagion directe et bien constatée;

g. La lèpre est toujours sporadique au Japon;

h. Il n'y a au Japon ni léproseries ni hôpitaux, pas plus que de prescriptions hygiéniques ou ordonnances légales relatives aux lépreux.

Le docteur *Macnamara* (2), qui a traité beaucoup de lépreux à Mozufferpore, au Bengale, nous en donne un aperçu très-intéressant. Ses observations s'étendent sur trois peuples différents : les Indiens (Hindous), les Musulmans et les Européens. Je rapporterai brièvement tout ce qui concerne le sujet que nous traitons.

Les Indiens se nourrissent presque uniquement d'aliments végétaux; les Musulmans mangent journellement de la viande,

(1) *Virchow's Archiv*, 1861, Bd. XXII, S. 336.
(2) *Virchow's Archiv*, 1861, Bd. XXII, S. 312.

et vivent de la même manière que les Européens qui séjournent au Bengale.

Des lois relatives aux lépreux n'ont jamais été publiées au Bengale; les indigènes ont la maladie tellement en horreur, qu'ils abandonnent tous les lépreux et se tiennent éloignés d'eux; et c'est à cause de cela qu'on n'a pas jugé nécessaire de faire des lois pour empêcher la propagation de la maladie.

Macnamara remarque, au sujet de cette propagation, que les Indiens, tant riches que pauvres, de même que les Musulmans, sont affectés de cette maladie, de sorte qu'il ne peut pas indiquer la moindre liaison entre la lèpre et l'alimentation ou les relations sociales; aucun des lépreux qu'il avait observés ne s'était nourri d'aliments salés.

Cet auteur remarque, en même temps, qu'il n'a vu qu'un seul Européen qui fût atteint de lèpre dans l'Inde; que pourtant les gens de sang mêlé en sont affectés aussi bien que les indigènes.

Cette communication est très-intéressante pour le but que je me suis proposé : il en résulte clairement que l'on ne peut accuser l'alimentation de contribuer à la naissance de cette maladie; que, par rapport au développement de cette dyscrasie, on ne doit attacher aucune importance à une nourriture déterminée, comme, par exemple, au poisson salé.

Dans la plupart des pays dans lesquels la civilisation est restée stationnaire, où l'agriculture n'a pas pris une grande extension, et où l'on n'élève pas des bestiaux en assez grand nombre, on se nourrit souvent de poisson salé. Ces conditions s'ajoutant à plusieurs autres circonstances nuisibles qui accompagnent l'indigence, exercent une influence désavantageuse sur l'état hygiénique en général; mais il n'en résulte pas que l'on doive considérer l'alimentation défectueuse ou l'ichthyophagie comme une des principales causes du développement de la lèpre.

Ne résulte-t-il pas de ces privations, quelquefois du plus nécessaire, d'autres inconvénients qui ne sont pas moins à redouter? Les circonstances concomitantes de la pauvreté, qu'il

ne faut pas perdre de vue, sont : la malpropreté, des habitations étroites, l'accumulation de plusieurs personnes dans le même logis, la difficulté et même l'impossibilité de s'abstenir du contact des malades; ajoutons à cela une incurie et une indifférence quelquefois révoltantes.

On peut, par ce même argument, expliquer la fréquence de la lèpre dans quelques contrées littorales de l'Europe : elle s'est réfugiée dans ces lieux comme un reste du moyen âge, et y prospère dans des conditions favorables.

La propagation de la lèpre au moyen âge, dans toute l'Europe, ou peu s'en faut, son existence encore actuelle dans les autres parties du monde, démontrent assez qu'elle peut aussi ravager l'intérieur des pays, si les conditions y sont favorables.

Ce n'est donc qu'avec beaucoup de prudence que l'on doit attribuer aux contrées littorales une influence spécifique sur le développement de la lèpre.

Je ne puis nier, que la rareté de cette maladie parmi les Européens, au Bengale, ne soit digne de remarque, d'autant plus que *Macnamara* parle de contagion et d'un croisement des races. Il faut en conclure qu'il doit y avoir un commerce assez assidu entre les Européens, les indigènes et les Musulmans; et même quand il n'en avait pas été fait mention, déjà *à priori*, on aurait dû comprendre que ce commerce est inévitable. Mais comme je ne suis pas assez au courant des relations réciproques de ces races différentes, je préfère laisser cette question indécise.

Le Bengale offre un vaste champ pour acquérir des connaissances étendues sur la lèpre, surtout à cause des trois nationalités que l'on y rencontre, et de leur différente manière de vivre.

Le docteur *P. Blecker* (1), qui observa la lèpre dans les possessions néerlandaises des Indes-Orientales, croit qu'il n'est pas constaté ni même vraisemblable que la maladie soit con-

(1) *Reis door de Minahassa en de Moluksche Archipel in het jaar* 1855. Batavia, 1856.

tagieuse ou héréditaire, parce qu'on n'a pas pu s'en convaincre dans un grand nombre de cas. Il demande pourquoi on rend le sort des lépreux, déjà si malheureux, encore plus pénible en les enfermant comme des malfaiteurs, tandis que leurs compatriotes ne craignent pas de les fréquenter, et ne tâchent pas de les éviter.

Un auteur plus récent, M. *Van Leent* (1), nous communique que la lèpre, aux Indes-Orientales,— qui, selon lui, ne diffère sous aucun rapport de ce qu'elle est aux Indes-Occidentales ou dans les pays septentrionaux de l'Europe, — se présente surtout dans l'archipel des Moluques (aux îles de Banda et de Ternate), mais qu'elle n'est pas non plus rare dans la partie occidentale de Java et à Sumatra. Il observe que les tribus arabes de l'archipel, ainsi que leurs descendants qui vivent assez isolés, et qui ne contractent pas d'unions avec les indigènes d'autres races, ne sont pas atteints de lèpre. Il en déduit que l'immunité dont jouissent ces Arabes est un argument puissant contre la contagion, ainsi que contre la théorie du développement autochthone de la lèpre; qu'elle témoigne, au contraire, en faveur de l'origine héréditaire chez les autres races.

Cet auteur remarque ensuite que, dans ces contrées, les Européens sont rarement atteints de lèpre; qu'il n'en est pas de même pour les gens de sang mêlé, qui, au contraire, en sont souvent atteints à un très-haut degré.

Si nous nous en rapportons aux militaires lépreux revenus des Indes-Orientales, que j'ai vus en Hollande, et dont j'ai déjà fait mention, je crois que les cas de lèpre parmi les Européens, aux Indes, ne sont pas si rares que le pense M. *van Leent*. Il est néanmoins évident qu'à cause de la position ordinairement plus élevée des Européens dans la société, et du peu de rapports qu'ils ont, par conséquent, avec la population indigène, les cas de lèpre parmi eux seront toujours moins fréquents; nous en exceptons cependant les militaires

(1) Une traduction de la communication de M. van Leent se trouve dans les *Archives de médecine navale*, année 1867, p. 248.

européens d'un rang inférieur, qui ont parfois de fréquentes relations avec les autres races. Nous en avons vu les conséquences!

Il en est de même de l'immunité des tribus arabes, dont cet auteur parle : le peu de contact qu'ils ont avec les indigènes, par suite de l'isolement dans lequel ils vivent, sera bien la principale raison de cette immunité apparente.

C'est à dessein que j'ai traité avec quelques détails les opinions émises par des médecins, pour montrer l'enchaînement des idées qui en général les ont conduits à se prononcer sur l'étiologie de cette maladie.

En 1867, le Collége royal de médecine de Londres (1) posait différentes questions sur la lèpre, dans toutes les colonies anglaises.

Voici le résumé des deux cent cinquante réponses, en ce qui regarde notre sujet :

La lèpre se présente partout sous la même forme. Dans les climats chauds, la maladie se trouve beaucoup plus parmi les races colorées que dans la population blanche. Chez cette dernière, elle se présente, la plupart du temps, parmi des personnes nées dans les pays ou ayant habité longtemps les contrées où cette maladie est endémique.

La grande majorité des lépreux se rencontre dans toutes les contrées, dans les classes les plus pauvres; quant aux personnes qui se trouvent dans de meilleures conditions sociales, quoique étant loin d'en être exemptes, il semble que leur susceptibilité varie surtout dans les différentes régions. Dans quelques réponses, on a remarqué que les classes élevées étaient aussi susceptibles que les classes inférieures.

La lèpre semble se rencontrer le plus souvent dans des contrées basses et marécageuses, surtout aux bords de la mer ou dans le voisinage des côtes; mais elle n'est pas du tout localisée dans ces parages, car on la rencontre souvent dans l'intérieur ou dans les pays montagneux, comme chez les Hottentots, les montagnards du Liban, du nord de la Perse et de l'Hindoustan.

(1) *Report on Leprosy by the Royal College of Physicians*, etc. London, 1867.

Les conditions hygiéniques des lépreux pauvres sont partout très-défavorables : leur nourriture est presque toujours représentée comme insuffisante dans tous les rapports. L'usage fréquent ou presque constant de poisson salé, et souvent dans un état de putréfaction, est mentionné plus qu'aucun autre élément comme cause de la maladie. Le défaut partiel ou total de viande ou légumes frais est généralement cité. La consommation en grande quantité d'huile gâtée est aussi accusée d'aggraver, sinon d'occasionner la maladie.

Il existe un accord presque unanime pour considérer la lèpre comme souvent héréditaire, mais aussi que la maladie se manifeste souvent sans qu'il puisse être question d'hérédité.

Il est constaté que la lèpre saute souvent une génération.

On a observé beaucoup de fois que seulement un membre d'une famille en est atteint.

La conviction *unanime* de tous les observateurs les plus habiles est que la lèpre n'est contagieuse, ni d'une manière médiate ni immédiate.

DE LA LÈPRE EN AMÉRIQUE; DE LA POSSIBILITÉ D'UN DÉVELOPPEMENT SPONTANÉ, DE LA CONTAGION ET DE L'HÉRÉDITÉ DE CETTE MALADIE.

Nous aborderons maintenant l'existence de la lèpre en Amérique. Je traiterai plus spécialement de la lèpre à Surinam, parce que je suis à même de juger avec pleine connaissance de cause des différentes conditions dans lesquelles cette maladie se présente dans la colonie.

Au commencement du XVII^e^ siècle, comme nous l'avons déjà remarqué dans l'histoire de la lèpre à Surinam, des Européens se joignirent aux aborigènes (les Indiens). Vers la moitié de ce siècle, on commença à importer des nègres de l'Afrique, et ainsi que nous l'avons déjà observé, avec les nègres on importa aussi la lèpre.

A en croire quelques auteurs, il paraît que ceci a eu lieu dans toute l'Amérique : « *Igitur non dubito* », dit Schilling (1), « *Quin ex Africa in novum orbem primum venerit* », et Campet (2): « Le mal rouge est endémique dans la Nigritie; c'est une maladie contagieuse qui règne dans le Nouveau monde depuis que le commerce a commencé à y faire passer des nègres. »

Nous avons vu ensuite que cette maladie était inconnue chez les indigènes, mais qu'elle se communiqua parfois à quelques-uns d'entre eux, qui avaient eu des relations avec des nègres.

Jusqu'à ce jour, la lèpre est encore inconnue à la plupart des peuplades indiennes, ce qui certes ne doit pas nous étonner, quand nous réfléchissons que les Indiens vivent dans l'intérieur du pays, entièrement isolés de la colonie proprement dite, et que le peu d'Indiens qui séjournent pendant un temps plus ou moins long dans la partie la plus habitée de la colonie, s'abstiennent de toute relation avec les nègres qu'ils ont en horreur.

Le docteur Dumontier m'a assuré que pendant les vingt années qu'il a séjourné à Surinam, il n'a vu qu'un seul Indien affecté de la lèpre. Cet Indien, qui habitait les bords de la rivière Coppename, avait beaucoup fréquenté la Léproserie, et il n'y a pas de doute que ce ne soit là qu'il ait été infecté.

La raison pour laquelle on voit si rarement des cas de lèpre parmi les Indiens qui visitent de temps en temps la Léproserie doit, d'après le docteur Dumontier (et c'est aussi mon avis), être cherchée dans des conditions qui ont rapport à la propreté du corps : l'Indien ne se sert d'aucun vêtement, il va nu et, au surplus, il se baigne et se plonge dans la rivière à tout moment; les nègres, au contraire, surtout ceux de la Léproserie, sont souvent vêtus de guenilles et vivent dans une malpropreté dégoûtante.

Nous avons donc tout droit de dire qu'un développement

(1) *Lib. l.*, p. 20.

(2) *Traité pratique des maladies graves qui règnent dans les contrées situées sous la zone torride, etc.* Paris, 1802, p. 290.

spontané de la lèpre n'a jamais eu lieu, ni parmi les Européens, ni parmi les Indiens; car avant l'importation des nègres, cette maladie n'avait jamais été observée parmi ces peuples. Les premières ordonnances des gouverneurs, qui ont eu rapport à la lèpre, démontrent assez que cette maladie se déclara insensiblement parmi les Européens; mais remarquons-le bien, assez longtemps après que des nègres lépreux avaient été importés; et déjà dans ces temps-là on était persuadé que les Africains lépreux communiquaient leur maladie aux Européens.

Au XVII^e^ siècle, la lèpre avait presque entièrement disparu dans la partie civilisée de l'Europe; ainsi on peut avec raison exclure une importation d'Européens lépreux.

Les nègres marrons, qui descendent d'Africains importés, vivent depuis à peu près un siècle dans l'intérieur du pays, et pendant tout ce temps-là se sont tenus séparés du reste de la population; ils n'ont eu même aucun rapport avec les esclaves : ils se craignent réciproquement. Ces nègres marrons ne sont pas non plus exempts de lèpre (1).

Leur manière de vivre diffère peu de celle des Indiens; ils pourvoient à leur entretien par la chasse, la pêche et la culture de quelques plantes alimentaires. Il paraît qu'eux aussi ou leurs ancêtres ont apporté la maladie de l'Afrique.

Maintenant, il nous reste à examiner de quelle manière les Européens ou ceux qui sont nés dans la colonie de parents Européens, ont été atteints de la lèpre.

Il est évident qu'ici il ne peut être question d'hérédité; il ne peut non plus être question d'un développement autochthone chez des individus qui vivent dans les meilleures conditions hygiéniques, et chez lesquels on ne peut accuser, comme nous l'avons vu, ni le climat, ni le sol, ni la nourriture. Les Indiens, qui se trouvent de même dans des conditions favorables, ne connaissent pas jusqu'à ce jour la lèpre; quoique le peu de cas de cette maladie qu'on a observés chez quelques-uns de ces Indiens, qui avaient eu des rapports avec des

(1) Docteur F. W. Hostmann, *Over de beschaving der Negers in Amerika.*

nègres, et que l'existence de la lèpre parmi les Peaux-rouges du Brésil, prouvent que cette race est susceptible de cette maladie.

La fréquentation des lépreux peut à peine être évitée dans la colonie, et quoique les individus infectés soient obligés de se tenir à l'écart, les prescriptions légales à ce sujet sont très-mal observées, et l'on est souvent exposé au contact des lépreux, surtout quand les symptômes sont encore peu prononcés. Ajoutez à cela que la plupart des Européens vivent dans un commerce familier avec les négresses.

On peut donc toujours constater le contact entre Européens et lépreux, presque jamais entre Indiens et lépreux. Il n'est pas rare du tout que de jeunes enfants de parents européens, qui n'ont jamais connu la lèpre dans leurs familles, soient atteints de cette maladie; pour tous ces enfants il peut être démontré qu'ils ont été en contact avec des lépreux, soit par négligence, soit à l'insu des parents, ou bien qu'ils ont été allaités par des nourrices lépreuses. Chez ces enfants l'existence de la lèpre ne peut être expliquée que par la contagion.

Depuis que dans mon travail antérieur sur l'étiologie de la lèpre (1) j'ai prononcé ces mots sur quelques enfants de parents européens et auxquels j'attache beaucoup de valeur, mon opinion a été contestée par le médecin de première classe de la marine hollandaise, M. *van Leent*, et par le docteur *Vinkhuyzen*. Le premier a observé la lèpre à l'archipel des Moluques, le second en Norwége; tous les deux nient formellement la contagion et sont partisans déclarés du développement autochthone et surtout de l'hérédité.

Tout en remerciant ces messieurs de l'attention particulière qu'ils ont bien voulu accorder à mon travail, et de la bienveillance qu'ils me témoignent, ainsi que des paroles pleines de considération qu'ils prononcent à l'égard de mon père, — je dois observer que je ne partage pas leurs opinions sur les arguments qu'ils ont allégués.

(1) *De besmettelijkheid der Lepra Arabum*, p. 75.

M. *van Leent* (1), en révoquant en doute la vérité de ce que j'ai dit à l'égard des enfants d'Européens, tache d'en paralyser toute la conséquence; tandis que M. *Vinkhuyzen* (2) fait une peinture si peu exacte, disons plutôt si erronée, des différentes situations dans la colonie, que les conclusions qu'il en tire sont dénuées de tout fondement. Il faudra donc, pour mieux nous entendre, que je sois plus explicite, quoique je regrette beaucoup d'être obligé d'entrer dans des détails que sans cela j'aurais passés sous silence.

Depuis 1840, et quelques années auparavant, il arriva douze fois que des enfants de parents européens furent atteints de la lèpre. Ce sont ces cas qui méritent toute notre attention. Afin d'éviter tout soupçon d'hérédité, nous ne dirons rien des cas de lèpre observés chez des enfants issus d'unions d'Européens avec des négresses, des mulâtresses, des quarteronnes et les autres degrés de mélange entre la race noire et la race blanche; nous nous abstiendrons de même de parler des Israélites arrivés à Surinam dans les derniers temps, parce que pour la plupart ils se sont alliés aux familles juives établies depuis longtemps dans la colonie, et dont nous ignorons les relations de parenté.

Dans ces douze cas il ne peut être question d'hérédité : les parents ou aïeux de ces enfants, comme nous le verrons, étaient Européens, de familles dans lesquelles on sait que la lèpre n'avait *jamais* été observée; tous ces enfants n'avaient connu que l'abondance; leurs parents appartenaient à la classe la plus élevée de la société coloniale, et étaient favorisés autant qu'on peut l'être sous le rapport du bien-être matériel. Ajoutons-y qu'aucun de ces enfants n'était maladif ou n'avait l'apparence chétive, avant et même encore après l'apparition de la lèpre; il paraît au contraire, selon la déclaration de mon père, qu'à Surinam cette maladie cherche ses victimes plutôt parmi les individus les mieux nourris et bien portants, que parmi ceux qui, comme le docteur *Vinkhuyzen* tâche de le dé-

(1) *Geneeskundig Tydschrift voor de Zeemagt*, 1868, p. 70 et 71.
(2) *De Melaatschheid's Gravenhage*, 1868, p. 133 et 134.

montrer, sont épuisés par le travail et les privations. Mon père est persuadé que tous les médecins qui ont observé la lèpre à Surinam reconnaîtront la justesse de cette assertion.

Nous considérerons séparément chacun de ces cas, qui sont notoires, et plus particulièrement connus de tous les colons qui ont vécu pendant ce temps à Surinam.

1*er cas.* — Mademoiselle S., née dans la colonie de parents hollandais (1) très-aisés, fut atteinte de la lèpre dans sa première jeunesse. La cause de la maladie était restée une énigme pour les parents, et ce ne fut que lorsque leur fille eut atteint l'âge de trente ans, qu'elle fut révélée par le triste fait suivant : Une esclave qui était mourante fit appeler madame S. auprès d'elle et lui confessa qu'elle avait mis son enfant souvent en rapport avec un lépreux.

2*e cas.* — Une petite fille d'un major en retraite allait à l'école chez une veuve européenne. Cette dame avait un fils, chez lequel on avait observé des taches sur la peau, mais que l'on croyait inoffensives; il se trouvait toujours à l'école de sa mère parmi les autres enfants. Quelque temps après, les premiers indices de la lèpre se déclarèrent chez la fille du major; mais le malheur ne s'arrêta pas là; l'enfant communiqua la maladie à sa mère, puis à son père, et enfin à une petite fille dont nous parlerons plus tard. (voyez le 4*e* cas).

Le major et sa femme étaient Hollandais.

L'enfant de la veuve W. mourut de la lèpre.

3*e cas.* — Mademoiselle K., née dans la colonie, fut atteinte de la lèpre et mourut à l'âge de vingt-trois à vingt-cinq ans.

La cause de la maladie se découvrit bien des années plus tard : M. F. se trouvant en commission avec d'autres personnes à la léproserie Batavia, vit se traîner devant lui une négresse lépreuse qui se trouvait dans la dernière phase de son existence. Cette femme, qui avait pris M. F. pour le père de la demoiselle, lui avoua qu'elle avait conduit souvent et à des-

(1) Par les mots *hollandais, allemands, anglais*, etc., dans les lignes suivantes, nous entendons : *natif de la Hollande, de l'Allemagne, de l'Angleterre.*

sein l'enfant chez un lépreux. Cette esclave mourut quelques jours plus tard.

Le père de la demoiselle était membre du conseil colonial, un homme riche, Danois de naissance; la mère était née dans la colonie, de parents hollandais.

4e *cas.* — Dans la famille d'un officier supérieur, il y avait plusieurs enfants, dont un seul, une fille, fut atteinte de la lèpre. Étant toute jeune encore, cette fille passa la plus grande partie de ses jours avec l'enfant du major dont nous avons parlé dans le 2e cas.

La jeune demoiselle mourut de la lèpre à la colonie; son père était Hollandais, sa mère était Anglaise.

5e et 6e *cas.* — Des quatre filles d'un membre du tribunal, les deux aînées furent atteintes de la lèpre, et moururent à l'âge de dix-neuf à vingt-deux ans. L'aînée avait été allaitée par une négresse, chez laquelle les symptômes de la lèpre se manifestèrent à un très-haut degré, et qui ensuite mourut de cette maladie. La seconde fille fut allaitée par sa mère. Ce ne fut que bien des années après que ces jeunes filles eurent quitté l'école, que la maladie se déclara chez elles d'une telle manière, que l'on fut obligé de les isoler immédiatement. Dès leur tendre enfance ces jeunes filles avaient eu le malheur d'être privées des soins de leur mère; il y a toute apparence que les premiers symptômes de la maladie existaient déjà depuis longtemps.

Le père de ces enfants était Hollandais; la mère et la grand'-mère étaient nées dans la colonie, cette dernière de parents européens. Tous appartenaient à des familles hollandaises très-distinguées, et avaient toujours vécu dans l'aisance.

7e et 8e *cas.* — La fille d'un avocat ayant perdu sa mère fut allaitée par une négresse. Elle eut souvent pour compagne une petite fille à peu près du même âge, enfant d'un homme riche, membre du conseil colonial. Ces enfants étaient dans leurs jeux d'enfance continuellement en contact avec un garçon nègre, qui avait des taches de psoriasis (connu dans la colonie sous le nom de *treef*), qui plus tard paraissaient être des macules lépreuses; il fut envoyé à la léproserie. Les suites

funestes de ce contact continuel ne se firent pas attendre, car à l'âge de seize ans la fille de l'avocat avait la lèpre très-prononcée. Elle mourut jeune. Mais aussi chez la compagne de sa jeunesse, les premiers symptômes se firent sentir à l'âge de onze à douze ans; elle succomba à l'âge d'environ vingt ans, victime de cette triste maladie.

Les parents de ces deux enfants étaient Hollandais.

9e *cas.* — Un médecin observa chez un de ses enfants, un garçon de deux ans, une petite tache rouge sur le côté extérieur de la cuisse, sans qu'elle eût été précédée d'une indisposition (1). Cette tache n'augmenta que lentement en circonférence, et se fit de plus en plus connaître comme une tache de psoriasis; elle fut, après un espace de temps assez long, suivie d'autres taches sur les jambes, sur le dos et au visage. Lorsque l'enfant eut atteint l'âge de six ans, la maladie n'était plus douteuse : le centre des grandes taches, qui avaient la forme du psoriasis scutellata, était blanc et anesthétique; les macules rouges, qui couvraient tout le scrotum, se boursouflaient et s'épaississaient insensiblement. Plus tard, le teint du visage devint d'un rouge plus ou moins livide; plusieurs tubercules se développèrent au menton, aux lèvres, aux ailes du nez, aux oreilles. A l'âge de douze ans, l'enfant mourut d'une dysenterie, qui régnait alors épidémiquement dans la colonie (2).

(1) Ce n'est que dans un seul cas, observé par mon père chez une femme adulte, que l'éruption de plusieurs taches rouges fut précédée d'un accès de fièvre, accompagnée de douleurs qui faisaient penser à un rhumatisme. Ordinairement les taches apparaissent sur la peau, sans qu'elles soient précédées de fièvre ou d'autres symptômes précurseurs.

(2) On observe moins souvent à Surinam, au début de la maladie, le *Morphœa nigra* (*Ophiasis umbrosa*, *Fuchs*); mais les symptômes subséquents à cette tache noire sont finalement les mêmes qu'après les macules rouges et le psoriasis. Les symptômes observés par mon père, chez un enfant mulâtre, âgé de douze ans, se présentèrent de la manière suivante : en 1853, tache noire au visage. En 1855, petites taches d'un brun foncé au cou; le dos et la région lombaire sont marbrés (exactement comme dans la planche XIII de l'atlas du *Traité de la Spédalskhed*, de *Danielssen* et *Boeck*). En 1856, tuméfaction des ailes du nez; de temps en temps de petites bulles de pemphigus aux plantes des pieds. En 1857, les taches sur la peau ont entièrement disparu; la guérison paraît prochaine; inopinément un ulcère profond se déclare sous le gros orteil,

L'enfant avait été allaité, pendant les premiers mois, par sa mère; il fut ensuite nourri de lait de vache, de biscuit, etc.; jamais on ne lui donna de la farine de bananes, à laquelle le docteur *Vinkhuyzen* attribue une influence spécifique si pernicieuse (*l. c.*, p. 135); on ne lui faisait boire que de l'eau de pluie : cette eau, dont use la classe aisée de Paramaribo, est d'une qualité supérieure, et ne contient pas tous les mauvais éléments que le docteur *Vinkhuyzen* veut y trouver (1); en somme, l'état hygiénique de l'enfant dans le sens le plus étendu, ne laissait rien à désirer.

Cependant l'enfant avait été conduit plusieurs fois à l'insu des parents chez une lépreuse.

Il ne pouvait être question d'hérédité : le père de l'enfant était Hollandais, et la mère, née dans la colonie, de parents hollandais; les ancêtres, tant du côté paternel que du côté maternel, nous sont connus en remontant jusque dans les XVI^e^ et XVII^e^ siècles. Nous ajoutons ces détails, afin de démontrer que du moins cet enfant-ci ne se trouve pas dans les mêmes circonstances que les militaires hollandais, revenus lépreux des Indes orientales, et chez lesquels M. *van Leent* (*l. c.*, p. 68) prétend que l'hérédité ne peut pas être niée avec certitude, parce que leurs généalogies ne sont pas connues!

Dans ce cas spécial, dont les moindres détails me sont connus, je ne puis admettre un développement spontané : l'enfant avait été élevé, comme nous l'avons vu, dans les meilleures conditions hygiéniques. Ainsi donc, aussi longtemps qu'on ne me prouvera, d'une manière satisfaisante et convaincante, que dans ce cas il faut accuser ou l'hérédité ou le développement spontané (dont j'ai démontré l'impossibilité, et sur lequel je reviendrai encore), je ne puis admettre qu'une infection.

le malade ne s'en était pas même aperçu. En 1858, guérison de l'ulcère; développement d'un nouvel ulcère. Le malade présentait quelques années plus tard (1864) des tubercules bien prononcés au visage, aux oreilles, etc. (*Lepra tuberculosa* des auteurs).

(1) Le docteur *Vinkhuyzen* cherche ensuite dans l'absence des sels calcaires dans l'eau potable une des causes prédisposantes de la lèpre. La ville de Paramaribo étant bâtie sur un banc de coquilles, ancien littoral, dans lequel sont creusés les puits à l'usage du peuple, l'eau potable fournie par ces puits est très-riche en sels calcaires, c'est-à-dire précisément le contraire de ce que prétend le docteur *Vinkhuyzen* (*l. c.*, p. 83).

10^e^ *cas.* — Une fille d'un capitaine de la marine coloniale, née à Surinam, fut atteinte de la lèpre, dont elle mourut quelques années après. Les détails ne me sont pas connus.

Les parents étaient Hollandais.

11^e^ et 12^e^ *cas.* — A quelques années d'intervalle, on observa les premiers symptômes de la lèpre chez deux enfants de cinq à six ans. Tous les deux étaient nés dans la colonie de parents allemands, faisant partie de la mission des frères Moraves.

La première, une jeune fille, avait une tache très-suspecte au bras. Cette enfant quitta la colonie, avec ses parents, qui retournèrent en Allemagne. Nous ne savons pas quelles en ont été les suites.

Le second est un garçon; il fut envoyé en Hollande avec des signes peu douteux de la lèpre : tuméfaction et épaississement de la peau du scrotum, analogues à celles du 7^e^ cas. Nous avons vu cet enfant à Amsterdam; on ne découvrit alors, à part les symptômes mentionnés, pas la moindre macule sur les autres parties du corps.

Ces enfants avaient été élevés dans les conditions hygiéniques les plus avantageuses; mais n'omettons pas de dire que tous les enfants de missionnaires à Surinam sont continuellement en contact avec des nègres et des mulâtres du peuple.

Ne doit-on pas être étonné, après avoir lu tout ce que je viens de dire sur ces enfants et leurs parents, de trouver dans l'ouvrage du docteur *Vinkhuyzen* le passage suivant : « Enfin » je dois y ajouter (nous traduisons) que dans la colonie de » Surinam la lèpre n'est presque pas connue parmi les Euro- » péens et les créoles aisés qui sont en état de suivre un ré- » gime européen. Le peu de cas qui sont connus se rapportent » à des personnes qui, avant d'avoir fait fortune, ont été » exposées pendant de longues années à toutes les influences » nuisibles et à la misère, et qui ont été privées du nécessaire;

» par exemple d'anciens soldats et de pauvres aventuriers à » qui la fortune se montra favorable (*l. c.*, p. 87). »

Ces mots ont été transcrits d'un rapport d'un certain médecin *Uhlig*; *Vinkhuyzen* y attache beaucoup d'importance (1). Mais des enfants *blancs* d'anciens soldats et de pauvres misérables, devenus riches, qui seraient atteints de lèpre, — ne sont pas connus à Surinam! Nous avons, par conséquent, le droit de demander quelle foi il faut ajouter aux autres récits de ce docteur *Uhlig*: par exemple à ce qu'il dit au sujet des Indiens (*l. c.*, p. 129), etc.?

Ne doit-on pas regretter de trouver dans un rapport, qui est adressé au gouvernement hollandais, une assertion si contraire à la vérité? Je regrette de même, et je me demande comment il est possible que le docteur *Vinkhuyzen* ait reproduit de telles absurdités dans un ouvrage, moitié populaire, moitié scientifique (*l. c.* préface), qu'il a écrit pour éclairer la nation et le gouvernement hollandais sur une maladie qui sévit dans ses colonies.

Vinkhuyzen, à ce qu'il paraît, entièrement étranger à la colonie de Surinam, se fiant à un rapport officiel, a bâti là-dessus tout son système. Après avoir parlé longuement sur ce sujet (*l. c.*, p. 87, 88, 124, 132-134, 167), il en déduit que les principales causes de la lèpre parmi les Européens à Surinam sont : un travail rude et assidu, une alimentation peu abondante et mauvaise, des vêtements insuffisants, des demeures malsaines et le manque d'instruction (p. 133).

Nous verrons plus loin combien cette opinion, même pour les classes inférieures de la société, est dénuée de tout fondement.

Mais, pour en revenir aux enfants des Européens dans la colonie, nous parlerons encore d'un jeune homme et d'une jeune fille, nés dans les possessions hollandaises, dans les Antilles, de parents de race blanche, et élevés dans l'aisance, qui arrivèrent à Surinam, portant avec eux les premiers in-

(1) *L. c.*, page 87.

dices de la lèpre. Le jeune homme, né à l'île de Curaçao, s'était trouvé dans la maison de son grand-père (un médecin qui ne croyait pas à la contagion de la lèpre), en contact continuel avec un garçon nègre infecté de cette maladie. Arrivé à Surinam, le malheureux jeune homme, abandonné par ses parents, se présenta, de sa propre volonté, devant la commission sanitaire, implorant, comme une faveur, d'être transporté à la léproserie. Il avait alors seize ans; les symptômes de la lèpre étaient manifestes. Quant à la jeune fille, née à l'île de Saint-Eustache, on m'a rapporté qu'étant enfant elle avait été continuellement en contact avec une de ses tantes, qui était lépreuse. Voilà un de ces cas qui, quoique, évidemment, il n'y pût être question d'hérédité, aurait été attribué ailleurs à l'hérédité dans la ligne collatérale!

Le fait que la lèpre se montre souvent dans la même famille, aussi bien dans les lignes collatérales que dans la ligne directe, ne serait-il pas une conséquence naturelle de la vie de famille, de cette vie intime, de la cohabitation continuelle? On conviendra qu'en général les occasions de se trouver en contact avec des lépreux se présenteront plus souvent aux membres d'une même famille qu'à des étrangers. Même en admettant l'hérédité dans la ligne droite, elle ne serait jamais admissible dans les lignes collatérales. La fréquence des cas de lèpre dans les lignes collatérales doit être considérée, comme je l'ai déjà fait observer, en parlant des statistiques de *Danielssen* et *Boeck*, comme un argument en faveur de la contagion.

Le nombre des Européens lépreux en général, dans la colonie, a toujours été en proportion de l'occasion qui se présente aux individus de se trouver en contact avec des lépreux, jamais en proportion de leur condition plus ou moins aisée ou élevée dans la société. Nous tâcherons d'éclaircir cette assertion.

En premier lieu, nous nous en rapportons aux militaires de rang inférieur qui, pour la plupart, ont des liaisons avec des femmes du peuple, et chez lesquels le plus grand nombre de cas de lèpre ont été observés.

Aussi, dans les classes plus élevées de la colonie, les cas de lèpre ne sont pas rares, mais principalement parmi les hommes (et parmi ceux-ci nous comptons un médecin), rarement parmi les femmes, et par la simple raison que ces femmes n'étaient presque jamais en contact avec des lépreux. Dans les familles des paysans hollandais, arrivées à Surinam en 1845, qui, pour la plupart, se sont établies sur un terrain bas et humide, près de la ville Paramaribo, et dont beaucoup se trouvent dans une indigence complète, jamais un cas de lèpre ne s'est présenté.

Toutes ces familles, même les plus favorisées du sort, vivent dans des conditions hygiéniques très-défavorables : l'anémie, suite de fièvres intermittentes (rangée par le docteur *Vinkhuyzen* parmi les causes prédisposantes de la lèpre les plus énergiques, *l. c.*, p. 85), et en général du manque de nourriture animale, est surtout évidente chez leurs enfants. Remarquons que tous ces paysans vivent dans un même quartier, qu'ils ne se marient qu'entre eux, qu'ils ne s'occupent guère de la population nègre ou mulâtre et qu'ils ont une aversion marquée pour les lépreux.

D'après le docteur Vinkhuyzen, ces paysans devraient être atteints très-souvent de la lèpre !

Le contraste avec les autres classes inférieures de la population dans la colonie est frappant; il fournira une nouvelle preuve pour la justesse de notre assertion.

La crainte de l'attouchement des lépreux, qu'on trouve chez les peuples de beaucoup d'autres pays, n'existe pas parmi les nègres et les mulâtres à Surinam. Pour s'en convaincre, on n'a qu'à se rendre dans les quartiers retirés du centre de la ville de Paramaribo; là on verra, çà et là, dans des demeures étroites et rapprochées les unes des autres, une population compacte où des lépreux vivent pêle-mêle avec des gens bien portants. L'insouciance et l'incurie, suites d'une croyance à la fatalité, sont arrivées parmi eux à un très-haut degré.

Une femme lépreuse que j'ai vue à l'hôpital d'Amsterdam, et dont M. van Leent fait mention (*l. c.*, p. 71), était, si je ne me

trompe, une mulâtresse; elle avait eu mainte occasion de se trouver en contact avec des lépreux : elle avait été mariée à un sergent-major, un quarteron, dont un frère et une sœur étaient lépreux. Ces deux derniers étaient logés dans une maisonnette située immédiatement derrière la maison où vivait leur belle-sœur, et chacun, pour peu qu'il connaisse la situation des maisons de la ville de Paramaribo, sait combien une telle séparation est tout à fait illusoire, et qu'il ne faut pas y attacher, comme M. *Van Leent* paraît le faire (*l. c.*, p. 71), la moindre importance. Ce cas nous montre au contraire tout le danger qu'il y a dans le commerce avec des lépreux; l'origine de la lèpre de cette femme n'est pas douteuse. Mais, parce qu'elle ne communiqua pas la maladie à son mari, cet auteur croit voir là un développement autochthone et cite ce cas comme un exemple qui doit plaider d'une manière très-évidente contre la contagion!

La position des Israélites pauvres ne diffère en rien de celle du peuple en général; mais pour peu qu'on s'élève dans les classes plus civilisées, on voit une différence notable. Les Européens surtout ont la lèpre en horreur, parce qu'ils croient à la contagion.

Nous ne devons pas omettre d'observer que généralement il est d'usage, chez les nègres et les mulâtres, que la grand'-mère se charge d'élever ses petits-fils, et que, par conséquent, ces enfants viennent demeurer chez elle. Ne doit-on pas par cet exemple expliquer ce fait observé quelquefois à Surinam, que la lèpre avait sauté une génération?

Surinam, où la lèpre a été importée relativement depuis peu de temps et où les trois races dont nous avons parlé se trouvent dans des relations si exceptionnelles, peut, plus que tout autre pays, répandre de la clarté sur l'existence d'un virus lépreux.

Dans les contrées où la lèpre existe depuis des temps très-reculés et d'où l'on ne peut exclure par cela même avec assez de certitude un développement spontané, occasionné par l'influence du climat ou d'une nourriture vicieuse, la possibilité d'infection ne peut être décidée que difficilement, et encore

moins la question de savoir si la maladie y a été la suite de l'hérédité ou de la contagion.

L'histoire de la lèpre à Surinam, au contraire, nous offre une base sûre, qui nous servira de point de départ pour discuter la question en général.

Pourquoi donc, malgré des faits aussi palpables, tous les savants se déclarent-ils contre l'admission d'un virus lépreux?

Je crois que la cause en est évidente : on voyait que très-souvent la fréquentation des lépreux avait lieu impunément, et l'on apercevait à peine le développement spontané *apparent*, parce qu'on était complétement pénétré de l'idée de l'hérédité; ajoutez-y qu'on ne pouvait pas exclure le développement autochthone avec assez de certitude. Presque tous les auteurs parlent d'un commerce assidu entre des lépreux et des personnes saines sans qu'il en résultât aucun inconvénient, et ils nient à cause de cela la contagion; mais ils n'ont pas réfléchi que c'étaient justement ces cas spontanés qui pouvaient être opposés à ceux qui n'eurent pas de suites fâcheuses.

En effet, l'étiologie de la lèpre n'est pas si palpable et si facile à découvrir que l'on puisse tirer si légèrement des conclusions pareilles! Je le répète encore une fois : on ne saura jamais la cause de cette maladie si on ne l'étudie pas en général et à grands traits. C'est l'examen comparé, seul, qui pourra dissiper les ténèbres qui enveloppent cette question.

Mais si la lèpre est contagieuse, me dira-t-on, comment se fait-il que le contact soit si souvent inoffensif!

J'avoue franchement que j'ignore les conditions que doivent offrir à un moment donné l'infectant et l'infecté, conditions inconnues pour la plupart des maladies unanimement considérées comme contagieuses.

De même qu'il est prouvé expérimentalement que la syphilis n'est contagieuse que dans sa forme primaire et secondaire, qu'il est d'observation que la rougeole n'est contagieuse que dans le stade des prodromes et d'éruption, il se pourrait, et tout me porte à le croire, que la lèpre, comme ces affections, ne fût contagieuse que dans un certain stade, probablement

de très-courte durée. Ne se pourrait-il pas aussi que, relativement, très-peu de personnes présentent de la susceptibilité pour cette maladie?

En tout cas, on n'est nullement autorisé à rejeter l'existence d'un virus, par la seule raison que le contact n'est que rarement funeste, car, pour la syphilis, c'est tout à fait la même chose. Combien de personnes n'ont pas été impunément en contact avec des syphilitiques?

Dans les hôpitaux des vénériens on ne voit non plus que les gardiens soient infectés : argumentation dont on se sert toujours pour démontrer que la lèpre n'est pas contagieuse.

Il est étonnant qu'on n'ait pas fait plus d'attention à la contagion dans le sens le plus étendu, lorsqu'on ne voyait jamais se produire un cas de développement spontané dans les contrées où la lèpre ne régnait pas et où toutes les conditions étaient égales à celles des pays voisins où la lèpre était endémique, et lorsque la maladie ne s'y répandait qu'après y avoir été importée! Nous le demandons avec instance, pourrait-on bien produire des preuves plus palpables en faveur de la contagion?

Il faut aussi remarquer que presque tous les peuples de la terre, tant anciens que modernes, ont toujours été enclins à croire à la contagion de la lèpre. Il semble pourtant que ce sont surtout les classes aisées qui croient le plus à la contagion. Mais ajoutons qu'en face de preuves scientifiques et bien constatées, l'opinion publique est d'une valeur subalterne.

Les aliments nuisibles, surtout le poisson, dont on se nourrit dans les contrées où la maladie est endémique, ont beaucoup contribué à augmenter la confusion et à attribuer à cette circonstance une des causes du développement et de la propagation de cette maladie; mais en examinant les faits plus attentivement, on voit qu'on ne peut attacher aucune valeur à cette influence.

La lèpre règne aussi bien et même avec rigueur dans beaucoup d'endroits où l'on ne prend pas de nourriture gâtée ou salée : comme en Norwége (les fiords), aux îles Canaries, au Bengale, en Chine, au Japon (pour les classes aisées); au

Brésil (où le porc sert de nourriture principale), à Surinam (pour les Européens et les nègres marrons), etc.; tandis que dans des contrées où l'on mange continuellement de mauvais aliments, on n'observe pas les moindres indices de la lèpre, comme dans plusieurs pays du littoral de l'Europe et ailleurs.

Au fait, tout porte à admettre, — comme je l'ai déjà démontré, — que ce n'est pas la nourriture malsaine et salée qui est la cause du développement spontané apparent; mais qu'il faut attribuer la fréquence des cas de lèpre parmi certains peuples moins civilisés à l'incurie et aux occasions multiples qui les contraignent à vivre avec des lépreux.

Le nègre, à Surinam, qui en apparence a une très-grande disposition à la lèpre, se nourrit, nous l'avouons, presque exclusivement de poisson salé (souvent en état de décomposition) et d'aliments végétaux indigestes (des bananes vertes); mais n'oublions pas que par sa manière de vivre, qui ne diffère en rien de celle des classes inférieures de la ville de Paramaribo, il n'est que trop souvent exposé au contact des lépreux.

Il faut de la même manière expliquer les autres relations hygiéniques. On ne doit pas non plus accuser les influences atmosphériques, cosmographiques et telluriques de pouvoir occasionner un développement spontané de la lèpre; on conviendra certainement que cette maladie existe dans toutes les zones et sous des conditions de natures les plus variées. Tout au plus pouvons-nous admettre que ces influences soient en état de modifier la susceptibilité d'être infecté par la maladie.

Afin de bien nous faire comprendre, disons que par le mot « susceptibilité » nous entendons ici la propriété de recevoir les impressions du virus et nullement la prédisposition qui suppose toujours un état anormal plus ou moins morbide et qui, selon moi, n'existe pas pour la lèpre.

L'assertion de quelques auteurs que les influences atmosphériques et telluriques peuvent avoir une influence sur la marche de la maladie aussi bien qu'une nourriture défectueuse, n'est peut-être pas dénuée de fondement.

Il se présente une autre question dont la solution est peut-être plus compliquée et plus difficile, savoir : si le principe de contagion, le virus, garde son caractère actif dans les zones différentes, dans les climats froids comme sous les tropiques? Nous parlerons plus tard de cette question en traitant de l'étiologie de la lèpre dans les autres parties du monde. Qu'il soit dit d'abord que je ne serai pas enclin à embrasser l'opinion de *Fuchs*, qui prétend que la lèpre est contagieuse en Asie, en Afrique et en Australie, mais qui nie cette propriété de la maladie en Europe, et qui veut que l'absence de la contagion soit la preuve la plus évidente que la lèpre n'existe plus en Europe comme autrefois. Il compare la maladie à une plante qui possède bien des feuilles et des racines, mais qui ne fleurit pas et ne produit pas de semences.

Donnons maintenant un exposé des opinions de quelques auteurs, qui ont traité de la contagion de la lèpre en Amérique.

Schilling, l'auteur le plus ancien sur la lèpre à Surinam, se prononce (1) de la manière suivante : « Superfluum videri » posset de contagio lepræ disputare, de quo nemo fere dubitat », et un peu plus loin : « Stat igitur sententia, contagio» sum esse lepræ virus, atque de parentibus in liberos, de » nutricibus in alumnos, de conjuge in conjugem transire; » quin etiam persuaseum habeo ex diuturno contubernio » absque intima illa corporum miscela per spiritum oris et » hircina ulcerum effluvia hanc luem cum aliis communicari. »

Hasselaar (2) dit que la maladie est contagieuse; cependant pas autant par un simple contact, que par le commerce charnel ou par une fréquentation trop familière, et par l'allaitement de nourrices lépreuses.

Encore aujourd'hui, la plupart des médecins dans les Indes occidentales sont entièrement de l'avis de ces deux auteurs.

Dans le Brésil, où se présentent les mêmes conditions clima-

(1) *L. c.*, p. 31.

(2) *Beschrijving der in de Kolonie Surinaame voorkomende Elephantiasis en Lepra.* Amsterdam, 1835.

tériques et où se trouvent les mêmes peuples (nègres, Indiens et Européens) qu'à Surinam, la lèpre se rencontre sur toute l'étendue du pays parmi les trois races dont nous venons de faire mention ; mais dans plusieurs contrées, comme par exemple dans la province Saint-Paulo, endroit qui du reste est très-sain, on trouve plus de lépreux que dans d'autres. Le docteur *Lallemant* (1) nous raconte combien on y craint le contact des lépreux, surtout dans les classes inférieures ; mais il remarque en même temps, qu'en général il est très-difficile de démontrer l'influence du contact dans une maladie endémique.

Le docteur *J. d'Aquino Fonseca*, au Brésil (2), ne pouvait pas encore dire avec certitude si la lèpre était contagieuse ou non.

A la Guyane française, la législation par rapport aux lépreux est fondée sur la contagion : en 1839 il fut établi une léproserie sur l'îlot dit la Mère, dans le port de Cayenne, et décrété par le gouverneur que toute personne, même libre, atteinte de lèpre, y serait mise en séquestration (3). Il paraît cependant que, par la suite, cette loi fut peu observée. *J. Laure* (4) dit à cet égard : « Depuis l'émancipation (1848), la lèpre envahit les familles blanches ; elle se propage avec une telle rapidité, qu'un dixième de la population en est infecté. »

A quoi attribuer cette extension si rapide de la maladie ? Dans ces quelques années, l'émancipation n'a pas pu changer l'état hygiénique des familles blanches d'une manière assez considérable pour faire naître la lèpre ; mais la séparation d'autrefois entre gens libres et esclaves n'existant plus, et comme cela a eu lieu dans toutes les colonies après l'émancipation, les nègres et surtout les mulâtres ont tâché de se mettre de niveau avec les blancs ; il y a eu des rapprochements et d'autres liens sociaux se sont formés.

(1) *Virchow's Archiv*, 1861, Bd. XXII, S. 341.
(2) *Virchow's Archiv*, S. 344.
(3) Décret du 13 août 1839 (voy. *Bulletin officiel de la Guyane française*).
(4) *Considérations pratiques sur les maladies de la Guyane*, Paris, 1859, p. 75.

Aux Antilles, la lèpre est endémique, — sans même en excepter l'île de Saint-Eustache, comme le fait le docteur *Vinkhuyzen* (*l. c.*, p. 69). Le docteur *Brassac* (1), qui observa la lèpre à la Désirade, croit à l'hérédité. Il se prononce fortement contre la contagion, incrimine surtout une alimentation incomplète et peu réparatrice.

Dans le nord de l'Amérique, la lèpre fut observée au Kamschatka par le docteur *Inosenz'off* (2) et au Nouveau-Brunswick (Canada) par le docteur *Skene* (3). Tous les deux sont partisans de la contagion, mais en même temps de l'hérédité.

On a prétendu que dans le nouveau Brunswick le premier cas de lèpre se serait présenté en 1817, chez un individu sain. Nous avons déjà révoqué en doute ce récit (4) ; et dernièrement je me suis aperçu que ma conjecture n'avait pas été sans fondement. En effet, le rapport anglais (5) nous relate à ce sujet : qu'une famille française, saine, originaire de Saint-Malo, en Normandie, s'arrêta pendant un long espace de temps à la Martinique, où la lèpre est endémique ; que de là elle se rendit en 1815 au Nouveau-Brunswick, où la lèpre était inconnue, et y importa cette maladie ; que la population de cette dernière contrée est composée d'Anglais, d'Écossais, d'Irlandais, d'Allemands et de Français ; que ces derniers y vivent dans les colonies françaises au bord de la rivière Miramichi, où, à cause des différents idiomes, ils n'ont pas contracté d'unions avec les autres nationalités ; que la lèpre ne règne au Nouveau-Brunswick que parmi les Français, à l'exception cependant d'un seul cas, observé chez un Écossais, nommé Stewart, et ses descendants ; que les Français s'occupent ordinairement de la pêche ; qu'ils vivent dans des demeures basses et humides, et qu'ils se nourrissent principalement de poisson et de lard de mauvaise qualité ; que la maladie règne exclusivement dans la classe pauvre.

(1) *Archives de médecine navale*, année 1866.
(2) *Schmidt's Jahrbücher*, 1844, Bd. 44, S. 194.
(3) *London med. Gazette*, Juni, 1844.
(4) *De besmettelykheid der Lepra*, etc., p. 80.
(5) *Loc. cit.*, p. 1 et 6.

De tous ces faits, Vinkhuyzen a conclu que la contagion dans de telles circonstances, doit être niée formellement, et prétend que jamais un exemple plus éclatant que celui du Nouveau-Brunswick n'a été donné pour prouver l'hérédité de cette maladie.

Je ne comprends pas comment le docteur Vinkhuyzen explique le développement de la lèpre chez cette famille française pendant son séjour à la Martinique, ni à quelle cause il attribue la maladie de l'Écossais Stewart? Certainement pas à l'hérédité! S'il attribue ce dernier cas à un développement spontané, nous ne comprenons pas comment il explique l'immunité apparente du reste de la population au Nouveau-Brunswick, qui vit dans les mêmes conditions hygiéniques désavantageuses que les Français (il l'avoue lui-même, *l. c.*, page 128) et qui certainement ne vit pas dans un état primitif, auquel il attribue un effet tout à fait exceptionnel (page 130 et 167). Le fait est vraiment très-intéressant : *avant* 1815, la lèpre était *tout à fait* inconnue, et dès qu'il se présente des lépreux venant d'ailleurs, voilà un colon qui en est atteint, quoiqu'il ne soit *nullement en parenté* avec les personnes qui ont importé la maladie.

Jamais on ne verra un exemple aussi marquant en faveur de la contagion.

Le médecin *van Leent* (*l. c.*, page 72), en parlant des peuples du Nord, dit : que ce sont surtout les pêcheurs et les bergers qui sont atteints de la lèpre ; que ceux-ci vivent dans la misère ; que pendant les longs hivers ils sont entassés dans des huttes étroites et basses, que leurs vêtements sont insuffisants; qu'ils sont *très-sales* (ces derniers mots nous les écrivons avec cet auteur, en lettres cursives); et enfin il y ajoute qu'ils mangent une nourriture peu abondante et malsaine. (*Vinkhuyzen* nous en donne une description encore bien plus triste *l. c.*, p. 108.) — Faut-il s'étonner, si cette maladie que nous croyons contagieuse, se communique facilement dans de telles circonstances?

Mais je ne suivrai pas le docteur *Vinkhuyzen* dans tous ses raisonnements et dans ses conjectures, ni le médecin *van Leent* dans ses citations et conclusions, qui s'écartent un peu de la

logique. Je me suis borné à réfuter quelques points les plus saillants de leurs écrits. En confrontant mon travail actuel avec le précédent, ils se convaincront aisément que je ne suis pas revenu de ce que j'avais avancé antérieurement ; mais qu'au contraire ils m'ont fourni de nouveaux arguments en faveur de la contagion, et contre le développement spontané, ce refuge pour la plupart des auteurs, quand il ne leur réussit pas de constater l'hérédité.

Ici nous nous arrêterons un moment pour dire quelques mots sur la possibilité de l'importation de la lèpre dans des contrées où cette maladie était auparavant inconnue.

Virchow, comme nous l'avons vu à la page 32, prétend que, dans les temps modernes, on n'a pu citer aucun exemple d'une telle importation ; ce qui, nous l'avouons, prouverait fortement contre la contagion. Ces exemples cependant ne manquent pas. Si l'on peut compter le XVI^e^, le XVII^e^ et le XVIII^e^ siècle parmi les temps modernes, nous pouvons citer l'importation de la lèpre à Madère, aux îles Canaries et aux Açores, où avant la prise de ces îles par les Portugais, cette maladie n'était pas connue chez les aborigènes (1) ; l'importation de cette maladie à l'île Maurice à la fin du siècle dernier (2), et ensuite celle que nous avons décrite dans l'histoire de cette maladie à Surinam, et qui ne laisse aucun doute.

Si l'on faisait de pareilles recherches historiques sur les autres contrées de l'Amérique, on obtiendrait, à en croire la plupart des auteurs, absolument les mêmes résultats. Mais nous n'avons pas besoin de monter si haut, l'importation au Nouveau-Brunswick, en 1815, a été démontrée par le docteur Chipman, qui a fait des recherches à ce sujet avec le plus grand soin.

Par tout ce qui précède, nous croyons être autorisés à conclure que la lèpre est contagieuse en Amérique. Mais cette maladie est-elle aussi contagieuse dans les autres parties du

(1) Vinkhuyzen, *De Melaatschheid*, p. 67.
(2) *Ibid.*, p. 209.

monde, dans des relations différentes? Nous tâcherons de résoudre cette question.

La lèpre peut se développer partout et dans les conditions les plus différentes, en conservant toujours ses mêmes caractères. Les maladies qui sont liées à une certaine température, au climat, au sol, à la nationalité, etc., perdront, dans des circonstances peu favorables à leur développement, leur caractère endémique ou épidémique. L'importation, dans quelque lieu de la terre que ce soit, ne paraît pas avoir la moindre influence sur le type de la lèpre; elle est toujours accompagnée de symptômes pathognomoniques; toujours la maladie reste contagieuse. La lèpre, dans les temps anciens, s'est fait connaître par les mêmes symptômes qu'aujourd'hui. Elle disparut entièrement dans quelques pays ; mais jamais elle n'a subi de métamorphose comme quelques auteurs l'ont soutenu. Il a été démontré que l'opinion que la syphilis serait une lèpre transformée, est dénuée de fondement.

Sans revenir à ce que j'ai déjà dit sur la lèpre en Amérique, je tâcherai de prouver par l'existence même de la maladie dans les autres parties du monde, qu'elle y est contagieuse ; mais je serai obligé de me borner, dans mon examen, aux pays où l'on a fait une étude spéciale de cette maladie ; car les rapports sur l'étiologie de cette affection dans la plupart des pays hors de l'Europe sont trop peu détaillés pour que l'on puisse en tirer une conséquence quelconque. Il me semble toutefois, que, quand la contagion de la maladie a été constatée aussi bien dans les tropiques que dans le Nord, on peut en conclure qu'il en sera de même pour les régions tempérées.

Afin de pouvoir répondre à cette question d'une manière satisfaisante, je tâcherai d'abord de démontrer que la lèpre ne se développe pas plus spontanément dans le nord de l'Europe que dans l'Amérique. Les faits suivants prouvent cette assertion :

1° Pour ce qui concerne la propagation de la lèpre, le nord de l'Europe doit être considéré comme se trouvant dans la même situation que presque tout le reste de l'Europe au

moyen âge. Dans la plus grande partie de l'Europe la lèpre disparut environ aux XVI^e^ et XVII^e^ siècles; premièrement de l'Italie, puis de la France, ensuite des Pays-Bas (XVII^e^ siècle) (1), et enfin du nord de l'Allemagne; il est à remarquer, comme l'a observé très-judicieusement le docteur Vinkhuyzen (2), que ce changement eut lieu à mesure que la civilisation se répandit en Europe.

Quelles sont les causes de cette disparition ?

a. L'exécution rigoureuse de l'isolement forcé ;

b. L'amélioration des relations sociales.

La première cause a été sans contredit plus efficace que la dernière ; c'est à elle seule que l'on doit d'avoir vu disparaître cette maladie de presque toute la superficie de l'Europe, sans qu'elle y ait laissé aucune trace. L'amélioration des relations sociales n'aurait jamais eu une influence aussi radicale, car les progrès à cet égard ne vont que très-lentement; encore aujourd'hui les conditions dans lesquelles se trouve le peuple dans plusieurs endroits, ne sont certainement pas meilleures qu'au moyen âge; quelles que soient les maladies qui en résultent, on ne les verra jamais produire la lèpre. Mais la question se présente sous un tout autre aspect, si l'on remarque que le bannissement des lépreux de la société se fit avec une très-grande énergie; et ce qui le prouve, ce sont les milliers de léproseries qui ont été élevées au moyen âge, et la persécution acharnée que l'on dirigea contre ces malheureux dans le but de les exterminer. Je le demande : contre quelle maladie a-t-on fait une guerre si terrible?

Nous rencontrons encore aujourd'hui cette maladie dans tous les lieux où l'isolement n'a pas eu lieu, ou n'a pas été exécuté d'une manière satisfaisante (la Norwége, le Portugal, les îles Canaries, la Crète, l'Égypte, la Chine, le Japon, Surinam, Cayenne, le Brésil, etc.) ; si elle n'a pas complétement disparu dans le moyen âge, — alors que l'isolement s'effec-

(1) *Israëls, Bydragen tot de geschiedenis der Lepra in de Noordelyke Nederlanden. Ned. Tydschrift voor de Geneeskunde*, 1857.

(2) *Geneeskundige opmerkingen op eene reis door het Noorden. Leiden*, 1865.

tuait avec une rigueur aussi terrible que souvent exagérée, — elle ne rétrogradera pas non plus dans les temps modernes, surtout depuis que l'on nie presque partout sa contagion, et que l'on admet le système d'isolement comme complétement inutile.

En Norwége, on n'a jamais poursuivi la lèpre, et ce n'est qu'à la fin du XVIII^e^ et au commencement du XIX^e^ siècle, que le gouvernement s'occupa de cette question si importante ; et même aujourd'hui, le système d'isolément forcé n'y est pas admis. Aussi n'est-il pas étonnant que la lèpre y règne encore avec tant d'énergie.

Si la lèpre pouvait se développer spontanément, il n'y a pas de doute qu'on verrait encore aujourd'hui des cas sporadiques de cette maladie dans les pays où depuis longtemps elle n'a plus laissé de traces, quoique les relations sociales s'y soient améliorées, et qu'en général la susceptibilité pour la maladie y soit moins prononcée.

Vidal nous démontre dans un mémoire très-intéressant sur la lèpre qui régnait encore à la fin du siècle dernier à Martigues, petite ville du département des Bouches-du-Rhône (1), que la maladie ne devait pas être attribuée à des influences pernicieuses locales. Si, dit-il, les causes assignées par *M. Raymond* (une atmosphère humide, des eaux stagnantes, une nourriture aqueuse et putride, surtout du poisson et des salaisons, avec boisson faible et mal préparée) étaient les vraies causes de la lèpre, elle aurait dû être extrêmement répandue et devrait être encore fort commune à Forz, village distant de Martigues de deux lieues, à une portée de fusil de la plage du même nom. Dans ce village, qui est exposé à l'influence de toutes les causes dont parle *M. Raymond*, et où l'on voit souvent le scorbut, il n'y a cependant *aucun* lépreux et l'on ne se souvenait pas qu'il y en ait eu autrefois.

Vidal ne croyait pas à la contagion de la lèpre ; dans un petit nombre de cas, il crut voir l'hérédité. La plupart des

(1) *Mémoire sur la lèpre à Martigues* (*Mémoires de la Société royale de médecine*, 1776, p. 167).

lépreux dont il avait entendu parler à Martigues, étaient ou marins, ou issus de gens de mer. Il y ajoute : « Si l'on excepte la propreté, qui est maintenant plus grande dans les maisons et dans les vêtements, il ne s'est fait depuis plusieurs siècles aucun changement notable dans la manière de vivre des habitants de Martigues ; on s'y nourrit aujourd'hui comme autrefois, de poissons, tantôt très-frais, tantôt salé. » — Il paraît pourtant que le nombre des lépreux y avait beaucoup diminué ; en 1776, Vidal n'y observa pas plus de trois cas de lèpre. Dans les temps actuels la maladie y est entièrement inconnue.

Je crois donc être autorisé à déclarer que la lèpre ne s'est jamais développée spontanément, ni dans les temps passés, ni de nos jours, — en reconnaissant toutefois comme non éclaircie la question de la toute première apparition de cette maladie.

En 1867, le docteur Steudener (1) décrit trois cas de lèpre d'origine autochthone, observés à Halle.

Mais disons d'abord que l'ensemble des symptômes chez les malades, dont il est question ici, ne nous présente pas l'image de la lèpre.

Ici tout bien considéré, l'anesthésie est le seul symptôme qui puisse faire penser à cette maladie. Mais l'anesthésie n'est pas un symptôme uniquement propre à la lèpre ; on l'observe aussi dans d'autres maladies ; ainsi donc par elle seule, si les autres symptômes pathognomoniques de la lèpre font défaut, on ne peut pas conclure à l'existence de cette maladie. Ajoutons que l'anesthésie n'était pas toujours bien marquée dans les trois cas.

Nous ne suivrons pas Steudener dans tous les détails qu'il nous donne ; nous nous bornerons à faire ressortir l'absence complète de symptômes qui caractérisent la lèpre.

Les changements morbides que Steudener observa dans le système osseux ne nous présentent rien de spécifique ; nous n'y voyons que la carie et la nécrose suite ordinaire de la périostite.

(1) *Beiträge zur Pathologie der Lepra mutilans*. Erlangen, 1867.

Au lieu de cette marche lente mais continue, et au lieu d'absence de douleurs, propres à la lèpre, nous trouvons dans le premier cas la nécrose à la suite d'une inflammation intense avec douleurs aiguës et térébrantes, occasionnée par une cause externe et suivie, après des interruptions de plusieurs années, d'affections analogues. Dans deux de ces cas Steudener observa la chute de l'ongle au début de l'affection du doigt. Remarquons que dans la lèpre les ongles au contraire se rétrécissent et se courbent par-dessus le bout du doigt, mais qu'ils se maintiennent même après que le doigt s'est raccourci par la carie d'une ou de deux phalanges. L'ankylose observée dans un de ces cas n'est pas propre à la lèpre.

Dans l'atrophie des muscles de la main, décrite par Steudener, nous ne retrouvons pas ces signes caractéristiques pour la lèpre, c'est-à-dire l'atrophie excessive du muscle adducteur du pouce et autres petits muscles par laquelle la surface bombée de cette partie de la main qui correspond auxdits muscles est remplacée par un creux qui se reconnait au premier abord, et l'hyperextension des premières phalanges des doigts. Dans le deuxième cas, les ulcérations et la destruction des orteils chez un enfant rachitique à l'âge de cinq à huit ans ne peuvent pas nous faire penser à la lèpre. Dans cette dernière maladie, de telles affections ne se produisent qu'à un âge bien plus avancé. Si nous ajoutons à ceci l'absence de tant d'autres symptômes, surtout des affections de la *peau*, nous avons tout lieu de croire que les cas observés par Steudener n'ont eu rien de commun avec la lèpre.

2° Les faits observés en Norwége, tant par *Holmsen* que par d'autres, prouvent que l'importation de la lèpre dans des lieux, qui auparavant étaient exempts de cette maladie, se fit toujours par l'*intermédiaire de lépreux*.

Cela a été clairement démontré par l'histoire de la propagation de la spédalskhed dans les fiords. On voit régner la lèpre dans un endroit, tandis que dans un autre on n'observe pas un *seul* cas de cette maladie, quoique dans ces deux endroits les conditions soient tout à fait les mêmes; quand cependant la maladie est *importée* dans un endroit qui en avait été

exempt, on y observe presque toujours un nombre relativement grand de personnes lépreuses, tant parmi des personnes qui sont parentes que parmi celles qui n'ont aucun rapport de parenté avec l'individu qui a introduit la maladie.

Qu'on ne me dise donc pas, comme on l'a prétendu, que ces faits plaident entièrement en faveur de l'hérédité. De pareilles objections sont dépourvues de la logique la plus simple.

Je ne suis donc pas de l'opinion de *Danielssen* et *Boeck*, qui prétendent que la spédalskhed peut se développer spontanément.

Mais dans quelle catégorie faut-il donc ranger ces cas dans lesquels les auteurs admettent un développement spontané? On ne peut que les mettre au nombre des cas occasionnés par la contagion.

Je ne puis non plus admettre, comme le docteur *Hjort*, que des influences extérieures (à l'exception du virus), puissent donner naissance à cette maladie.

J'ai déjà remarqué qu'un grand nombre de lépreux, dans les statistiques de *Conradi* (dans quelques-unes, près de la moitié des malades) descendaient de parents ou d'aïeux non lépreux; dans ces cas-là, l'influence du virus est évidente.

La statistique de *Holmsen* est une des plus importantes pour prouver la contagion de la spédalskhed. On y voit que, sur 93 lépreux, la maladie ne fut causée par l'hérédité que chez un seul. Les 92 restant ont dû, par conséquent, être infectés par des lépreux, et non pas, comme le pense cet auteur, par un miasme qui émanerait des côtes de Norwége.

Il est vrai que dans les temps modernes on n'a pas pu constater un seul cas d'infection dans les pays de l'Europe où la lèpre fut importée de temps en temps. Mais n'oublions pas que le nombre de ces cas importés est excessivement restreint, que presque tous sont des personnes âgées, ordinairement des soldats, qui, pour la plupart, vivent hors de la société; que même, dans les pays où la lèpre est endémique, l'augmentation annuelle de ses victimes est relativement très-minime, comme les statistiques nous le prouvent.

Nous ne devons pas perdre de vue que la vie moyenne des

lépreux est relativement très-longue (en Chine, la maladie n'abrégerait pas la vie; en Norwége, la durée moyenne s'élevait: de la lèpre tuberculeuse, de 9 à 10 ans; de la lèpre anesthétique, de 18 à 20), de sorte que les mêmes lépreux paraissent longtemps dans les statistiques de plusieurs années de suite.

Nous voyons aussi, dans les Indes occidentales, que, pour beaucoup dans la plupart des cas, la fréquentation des lépreux peut se faire impunément; ne nous étonnons donc pas de voir la même chose dans les pays où cette maladie est importée accidentellement par un si petit nombre de malades.

En abordant le sujet de la possibilité d'une propagation héréditaire de la lèpre dans les Indes occidentales, je tâcherai de résoudre en même temps la question pour les autres pays.

A la fin de l'histoire de la Boasie, à Surinam, j'ai communiqué quelques chiffres qui déjà, au premier abord, ne plaidaient pas en faveur de la propagation héréditaire.

Nous y avons vu que, parmi 121 individus (1) nés à l'établissement Batavia, presque tous de parents ou aïeux lépreux, il n'y en avait pas moins de 100 (82 p. 100) qui n'étaient pas lépreux; que deux ans plus tard (1853), parmi 448 individus qui se trouvaient à l'établissement, 112 n'étaient pas lépreux (tous se trouvant dans les conditions mentionnées plus haut); tandis que le docteur *Ooykaas* découvrit en même temps que, parmi le 336 lépreux restant, il n'y en avait que 33 (17 hommes et 16 femmes, savoir : 9,8 p. 100), dont on pouvait dire avec certitude qu'ils étaient nés de parents ou d'aïeux lépreux.

On peut encore y remarquer que, dans l'année 1857, de 145 individus, nés aux établissements, 12 (8 p. 100) étaient lépreux, 20 (14 p. 100) suspects et 113 (78 p. 100) sains.

Parmi 245 lépreux, 221 (90 p. 100) avaient eu des parents et aïeux sains; 24 (10 p. 100) avaient eu des parents lépreux [c'est-à-dire 7 (3 p. 100) les deux parents, 3 (1 1/4 p. 100) seulement le père et 14 (5 3/4 p. 100) seulement la mère].

(1) Malheureusement je ne suis pas à même de mentionner l'âge de ces personnes; non plus que pour les 112 individus sains mentionnés dans la statistique de 1853.

De 115 individus sains : 78 (68 p. 100) avaient des parents ou aïeux lépreux [c'est-à-dire 30 (26 p. 100), père et mère, 18 (16 p. 100) seulement le père et 30 (26 p. 100) seulement la mère]; la plupart des autres, 37 personnes, avaient eu plus que probablement des aïeux lépreux.

De ces 78 personnes, 16 (20 p. 100) étaient restées saines plus d'un tiers de siècle.

On ne peut parler du nombre des individus suspects, leur position étant loin d'être connue d'une manière certaine.

Voici une statistique que je trouve mentionnée par le docteur *Vinkhuyzen :* parmi 164 individus (savoir : 72 hommes et 92 femmes) nés à l'établissement Batavia et ayant *tous* des parents ou aïeux *lépreux*, il ne se trouve que 23 lépreux (savoir : 10 hommes et 13 femmes) ou 14 pour 100.

Mais remarquez bien qu'un chiffre pareil est mentionné en faveur de l'hérédité! Mais est-ce qu'on oublie donc que, soit parents, soit aïeux de ces 164 individus, ont été tous lépreux? Non, une maladie héréditaire, mise dans des conditions si favorables à son développement, aurait poduit chez les descendants d'autres résultats.

Je n'ai pas besoin d'ajouter d'autres explications, car la conclusion est évidente. Mais on objectera peut-être que, dans le nombre des individus sains de la statistique de 1857, on compte beaucoup d'enfants, et qu'il est possible que la disposition morbide se réalise plus tard! Nous voyons cependant que, des 78 personnes saines nées dans les établissements, 16 (20 p. 100) étaient restées saines plus d'un tiers de siècle.

Ici nous devons remarquer que, quoique la lèpre, à Surinam, puisse apparaître chez des enfants très-jeunes, il est très-rare qu'ils soient envoyés à l'établissement (nous n'y en trouvons, en 1857, aucun au-dessous de 10 ans), car leurs parents et leurs maîtres cachent la maladie aussi longtemps que possible, et cela est facile tant que les symptômes ne sont pas très-prononcés. Le petit nombre d'enfants lépreux en bas âge, à l'établissement, n'est donc pas une conséquence de l'apparition de la maladie à un âge plus avancé.

Nous avons vu ensuite que, dans l'année 1863, 89 individus,

tous nés à l'établissement, sont rentrés comme sains dans la société.

Pour avoir le rapport entre les naissances et les décès à l'établissement, je renvoie à la page 17.

A Surinam, — je m'en rapporte aux observations de mon père à cet égard, — des enfants en bas âge ne meurent pas de la lèpre (si le contraire fût arrivé, le chiffre aurait été moins favorable à la propagation héréditaire). Les enfants d'Européens, qui succombent à cette maladie plus tôt que les enfants de nègres ou de mulâtres, atteignent au moins l'âge de la puberté (ils meurent ordinairement entre 17 et 25 ans), ce qui ne doit pas nous étonner si nous considérons la marche très-lente de cette maladie; tandis qu'en général les nègres et les mulâtres s'éteignent à un âge plus avancé.

Il n'y a pas de doute que la lèpre, à Surinam, doive être considérée comme non héréditaire, et que ces individus, qui sont nés à la léproserie, et qui, plus tard, sont devenus lépreux, aient été infectés par un virus.

Les statistiques de l'établissement Batavia offrent tant d'intérêt, parce que (comme je l'ai fait remarquer en parlant de la lèpre chez les Chinois) les expériences sur la propagation héréditaire s'y font dans des conditions très-favorables pour la déduction de conséquences importantes; et parce que, dans les statistiques d'autres pays, qui démontrent que peu de lépreux sont issus de parents ou d'aïeux malades, il se pourrait que le mariage, dans ces pays-là, fût entravé par des circonstances sociales. Il ne faudrait pas, dans ces conditions, s'étonner si, parmi beaucoup de lépreux, un petit nombre seulement eussent eu des parents ou des ancêtres lépreux.

On objectera peut-être que ces statistiques témoignent tout autant contre la contagion de la lèpre! Pour répondre à ceci, je dois renvoyer le lecteur à ce que j'ai dit plus haut à cet égard.

Si nous avons démontré l'existence de la contagion et exclu la possibilité d'un développement spontané, il est évident que nous devons accepter un développement par infection chez ces quelques individus qui sont nés à l'établissement, et qui,

plus tard, sont devenus lépreux; car, avec des statistiques comme celles fournies par l'établissement Batavia, ou mieux en face de pareilles conséquences expérimentales de mariages entre lépreux, on ne peut pas songer un seul instant à l'hérédité.

Une maladie véritablement héréditaire, bien loin de se borner à un aussi petit nombre de victimes, nous en offrirait toujours au contraire de nouvelles, dans des conditions aussi favorables que celles de l'établissement. Dans d'autres maladies héréditaires, quoique un seul des parents soit malade, et malgré toutes les précautions que l'on prend (comme dans la tuberculose, la goutte, etc.), nous les voyons souvent se manifester chez les descendants; il serait donc fort étonnant que la lèpre, — en admettant qu'elle fût héréditaire, — ne se transmît pas de même avec vigueur à la postérité, lorsque toutes les circonstances favorables à son développement se trouvent réunies!

On peut établir, en thèse générale, que toute maladie dont l'hérédité est bien constatée (syphilis, tuberculose, cancer, etc.) et qui se présente chez le père et la mère, se manifestera presque infailliblement chez les enfants (toutes conditions égales d'ailleurs).

Quant à la contagion, il en est tout autrement; en effet, pour qu'elle ait lieu, il faut une coïncidence de conditions différentes chez l'infectant et chez l'infecté; chez l'infectant: un certain stade de la maladie; chez l'infecté: d'abord la susceptibilité (suite d'influences diverses, telles que l'âge, sexe, constitution, etc.), et ensuite toutes les circonstances nécessaires à l'infection médiate ou immédiate.

Après ce que je viens de dire, on comprendra facilement qu'une maladie peut être contagieuse et néanmoins faire très-peu de victimes, quoiqu'elle semble dans les conditions les plus favorables à son développement.

Les 107 esclaves lépreux (sans parler des gens libres), qui, à l'époque de l'émancipation, furent trouvés dans la société, témoignent combien le système de l'isolement fut mal exécuté à Surinam.

La plupart des auteurs qui ont écrit sur la lèpre à Surinam

(*Schilling*, *Hasselaar*, *Ter-Beek*, etc.) croient à l'existence de la propagation héréditaire; le premier, cependant, pense que le transport des enfants nés de parents lépreux, dans des climats sains et tempérés, où ils seraient soumis à une bonne alimentation, empêcherait l'apparition de la maladie (cela, cependant, n'a pas été confirmé par l'expérience). *Hasselaar* parle même de la lèpre congénitale (qui, toutefois, n'a jamais été observée à Surinam par des médecins dignes de foi). Il raconte plusieurs faits curieux au sujet de l'hérédité (1)!

Cependant *Ooykaas* fait exception parmi ces auteurs; il n'attache pas à l'hérédité l'importance qu'on lui attribue généralement.

Tâchons maintenant de démontrer aussi, pour les autres pays, que la lèpre n'est pas non plus héréditaire. Nous pourrions déjà décider cette question *à priori;* mais il se pourrait que les influences extérieures qui pouvaient transformer la prédisposition héréditaire en maladie n'existassent pas à Surinam. On comprend parfaitement que les influences extérieures doivent exister à Surinam aussi bien que dans d'autres endroits où la lèpre est endémique; par conséquent, si la propagation non héréditaire est prouvée pour Surinam, elle doit l'être aussi pour ces autres endroits.

Déjà, en parlant de la lèpre dans le Nord, j'ai remarqué que *Danielssen* et *Boeck* ont très-peu motivé leur opinion; pour ne pas revenir sur ce sujet, je renvoie à la page 22. Il en est de même pour les statistiques de Conradi (p. 26); mais je ne saurais trop appeler l'attention sur les statistiques de Holmsen (p. 27).

Dans les rapports des médecins des autres pays je n'ai pu découvrir des preuves qui témoignent en faveur de l'hérédité de la lèpre. Pour bien juger toutes ces statistiques, il ne faut pas oublier que les enfants de lépreux, qui, pour la plupart, appartiennent aux classes inférieures de la société, sont, pour des raisons que j'ai déjà développées, surtout exposés à l'infection.

Avant de quitter la question de l'hérédité, je ferai une

(1) Hasselaar, *l. c.*, p. 39.

seule remarque. Le docteur *Vinkhuyzen*, qui, dans son ouvrage, se pose comme défenseur des opprimés, propose aux gouvernements d'abolir toute contrainte contre les lépreux, qu'il met dans la même catégorie que les malades affectés de tuberculose, de carcinome, etc. Dans les pays où la lèpre est endémique, il veut faire abroger toutes les lois établies pour l'isolement et la séquestration des malades, mais en même temps il veut interdire aux lépreux et à *ceux qui descendent de parents lépreux*, toute fonction publique (*l. c.*, p. 231). Comment expliquer, dans ses principes, une telle contradiction?

Enfin, je dois dire quelques mots sur les propriétés du virus de la lèpre.

Ce virus est-il fixe ou volatil? Les auteurs ne sont pas d'accord sur ce point; quelques-uns (*Schilling*, *Arnoldus* et les médecins du moyen âge) croient que l'air atmosphérique suffit à la transmission de la contagion; *Richter*, au contraire, ne croit à la contagion que par le contact et surtout le coït; *Hasselaar* et autres sont d'avis que l'infection peut avoir lieu de la manière la plus compliquée, même par l'intermédiaire de personnes chez lesquelles la lèpre est latente. Suivant mon père, il est plus que probable que l'infection ne peut se transmettre que par le contact très-intime.

Je ne crois pas, les savants ayant fait peu d'attention à la possibilité d'une infection, que l'on connaisse jusqu'à présent des faits bien constatés qui démontrent comment l'infection peut se produire.

Cette même incertitude existe aussi quand il s'agit de savoir dans quel stade la lèpre est en état de reproduire le virus.

Schilling pense qu'une seule macule, à peine visible, suffit à communiquer la lèpre à plusieurs personnes, même à un très-haut degré; tandis que l'individu, source de tant d'infection, peut recouvrer la santé ou souffrir d'une maladie toujours stationnaire.

Hensler et la plupart des médecins sont d'avis que la lèpre doit avoir atteint un certain degré de développement pour être en état de se communiquer par le virus.

Je n'ai pas besoin de dire qu'il est très-difficile de produire

des faits positifs à cet égard, parce que, réellement, on ne sait rien de la période d'incubation.

Si l'on en juge d'après quelques cas, cette période serait très-longue : chez deux malades observés par *Danielssen* et *Boeck*, la période d'incubation aurait duré au moins 6 et 9 ans. On aurait aussi vu, dans les Pays-Bas, les premiers indices de la maladie se présenter environ 8 ans après le départ de l'individu des Indes orientales. Dans les Indes occidentales, au contraire, il n'est pas rare de voir, chez des enfants de parents européens sains, les premiers symptômes de la lèpre même en très-bas-âge (2 ans).

C'est à cause de la marche très-lente pendant plusieurs années, dans lesquelles la maladie ne se trahit souvent que par une petite tache, qu'il est très-difficile d'acquérir des données positives. On ne doit donc pas trop se fier aux récits des malades qui souvent eux-mêmes n'ont pas remarqué les premiers symptômes.

On possède naturellement encore bien moins de renseignements sur la ténacité ou la capacité du virus, et sur la manière ou la voie par lesquelles le virus est introduit.

Il est probable que l'incubation du virus lépreux, si elle pouvait avoir lieu, ferait cesser bien des doutes sur la question de la contagion. Le médecin *van Leent* propose de faire des expériences sur des cochons, chez lesquels on observe quelquefois une maladie qui paraît analogue à la lèpre. Cette idée mérite, en premier lieu, toute l'attention des médecins dans les pays où la lèpre est endémique. A Surinam, cette maladie des cochons dont parle M. *van Leent* n'est pas inconnue.

Il n'est pas constaté que la lèpre ait une prédilection marquée pour l'un ou l'autre sexe, comme le rapport anglais nous le démontre.

Le développement de la maladie n'est propre à aucun âge; le plus souvent elle se déclare depuis la puberté jusqu'à l'âge mûr.

FIN.

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THERMOMETRICAL OBSERVATIONS.

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THERMOMETRICAL OBSERVATIONS.

That the exact value of the thermometer as a guide to the diagnosis, prognosis, and treatment of disease is as yet undefined, is abundantly proved by the diversity of opinion regarding its utility which is found amongst medical men. By some it is lauded as an infallible guide to the attainment of a proper knowledge of many diseases; whilst others regard it as an unnecessary and fallacious attempt at refinement—"a mere scientific toy." Extreme views are apt to be erroneous, and probably are so in the present case. An instrument which has been found valuable in the hands of men noted for professional and scientific attainment cannot be a mere toy: at the same time, it does not become us, as honest searchers after truth, to accept the conclusions of even such men as Wunderlich, Ringer, Aitken, etc., without submitting them to the proof, and testing, by further observation, the value and accuracy of the conclusions at which they have arrived. Imbued with this impression, I began to observe for myself, and was, ere long, led to regard the thermometer as a very valuable aid in acquiring a knowledge of various diseases, but as not possessing those high virtues which some have ascribed to it.

My object in the present paper is to try to summarize the results of my own observations, and to give the practical conclusions which I have been led to draw, inserting occasionally, for the sake of illustration, the particulars of an individual case, but avoiding, as much as possible, unnecessary detail.

The instruments used were Casella's straight self-registering, as recommended by Aitken. The observations were made in the axilla, and the directions usually given were strictly attended to.[1]

In many cases the observations were made four times a day—at 10 A.M., 2 P.M., 7 P.M., and midnight. I found, latterly, that for

[1] One of Aitken's instructions, that the thermometer should be slightly warmed by holding the bulb in the hand, was at first neglected; I afterwards found that neglect of this precaution was apt to lead to an understatement of the temperature. I accordingly abandoned all my earlier observations, and now only use those which were made after the plan of holding the bulb in the hand till the mercury showed a temperature of 95° was adopted. They were made during the years 1865 and 1866 (chiefly the latter) in the Dundee Infirmary, while I was superintendent of that institution.

all practical purposes, it was sufficient to note the temperature twice a day—morning and evening—with an occasional observation at midnight.

The pulse and respirations were noted at the same time. After many careful observations, I came to the conclusion that the mere rapidity of the respirations was not to be regarded in all cases as an exact index of the patient's condition; that the number of respirations taken per minute were apt to vary, and be influenced by very slight causes, such as the swallowing of saliva, or the existence of a slight catarrh; and that in cases in which there was much affection of the nervous system (typhus, for instance), the character of the respirations was more worthy of note than the mere frequency. I accordingly, in many cases, did not note the number of the respirations with anything like regularity, but confined myself to an occasional record of their frequency, and a general description of their character—irregular, cerebral, etc.

For clearness' sake I shall take separately the diseases on which a sufficient number of observations were made to enable me to form some definite opinion regarding the utility or otherwise of the instrument, and shall further subdivide what I have to say into remarks on diagnosis, prognosis, and treatment.

Typhus Fever.—The range of temperature which I have found in this disease does not altogether accord with that which has been noted by other observers. As my object in this paper, however, is rather to give the results of my own observations than to criticise those of others, I shall simply note the fact, and remark that the difference may in part be due to the varying circumstances of different epidemics: those symptoms which usually accompany a high range of temperature may be more marked and frequent in one epidemic than in another; and as the state of the temperature is the effect, not the cause of the condition which produces these symptoms, it will naturally vary according as they are more or less marked. Such appears to me the most probable explanation of the fact, that I have never found in any of my cases such a high range as that given by Wunderlich and Aitken, and given too as the typical range. At page 387 of the last edition of his work on the Science and Practice of Medicine, Aitken gives, from Wunderlich and Traube, a table to show the typical ranges of the temperature in Typhus and Enteric fevers, in which the typhus range is shown to be above 106° on the 5th, 6th, 7th, 8th, 9th, 10th, and 11th evenings; and again, at page 434, he gives another typical case, in which the morning temperature is for nearly a week above 105°, whilst the evening temperature is, during the same time, above 106°; and on the 4th evening is as high as 107°. The highest temperature that I ever found was in a girl aged 17 years, with very bad head-symptoms, who for 36 hours before death had complete suppression of urine, who died comatose on the 14th day, and in whom, a few hours prior to the fatal termination, the thermometer indicated a temperature of 106·4°. In cases which terminated favourably, I never found the temperature higher than 105·2°, at which I have several times noted it.

Aitken states that both in mild and severe cases the temperature always rises above 104·7°; it only did so in 13·7 per cent. of my cases, and these were all severe.[1]

The following table shows the average temperature at morning and evening on each day up to the time of commencement of defervescence in 58 cases which recovered, and in the selection of which nothing was considered except that there should exist no complication; that no disturbing influences (such as those to be hereafter noted) should prematurely and abnormally lower the temperature; that each should have a definite commencement, and that no doubts should exist as to the time of onset of the disease.

			Morning.	Evening.
In	1 case the temperature was on the	1st day		100·8°
,,	3 cases the mean temperature on the	2d day	102·4°	102·5
,, 10	,, ,,	3d	102·7	103·2
,, 20	,, ,,	4th	103·1	103·5
,, 30	,, ,,	5th	103·3	104·
,, 41	,, ,,	6th	103·1	103·3
,, 47	,, ,,	7th	103·	103·5
,, 54	,, ,,	8th	102·8	103·4
,, 58	,, ,,	9th	102·8	103·1
,, 55	,, ,,	10th	102·8	103·1
,, 37	,, ,,	11th	102·4	103·4
,, 28	,, ,,	12th	102·6	103·1
,, 17	,, ,,	13th	102·7	103·
,, 13	,, ,,	14th	102·5	102·9
,, 7	,, ,,	15th	102·6	102·9

From this it appears that the morning temperature was habitually lower than the evening: the average of the former being 102·7°, that of the latter, excluding the first day's observation, 103·2°. In 26 of these cases in which the observations were also made at 2 P.M., the mean temperature at that hour was 103·1°, or very little below the evening average. In 31 the mean temperature at midnight was 102·8°, a little over the morning average.

The average maximum range was 104·3°—the highest being 105·2°, and the lowest 103°.

It would seem that the temperature gradually and steadily rose, showing little or no tendency to a morning fall, till it reached its highest point on the 5th evening, that it then fell

[1] The range which I have found accords with that given by more recent observers. See papers in St George's Hospital Reports, vol. i., by Dr Thompson; St Bartholomew's Hospital Reports, vol. ii., by Dr Warter; Dublin Quarterly Journal, vol. xlii., by Dr Compton; the same journal, vol. xliii., by Dr Grimshaw. It seems, indeed, that Dr Aitken's cases cannot be regarded as representing the typhus usually met with in this country; it is possible, as suggested by Dr Grimshaw, that the instruments used were not sufficiently accurate. As Aitken's observations are most generally known and read, it is well that their probable inaccuracy should also be known.

somewhat, and did not again reach the same height; and that on the 6th day commenced those morning falls which characterized the remaining course of the case, and which, though occasionally slight, were generally quite appreciable. The range above the average on the 6th, 7th, and 8th evenings I am inclined to attribute to the fact, which I have several times noted, that in those cases in which the appearance of the eruption is delayed for a day or two beyond the usual time, the temperature does not reach its maximum so early as in those in which it is out on the 5th day. It seems, in fact, that the febrile disturbance, as indicated by the temperature, reaches its height about the time at which the rash is fully developed.

The following table shows the rate of the pulse in the same cases from which the last table of the temperatures was compiled:—

		Morning.	Evening.
In 1 case the pulse on the 1st day			120
„ 3 cases the mean pulse on the 2d	day,	125·3	120
„ 10 „ „	3d	113	114·2
„ 20 „ „	4th	108·1	115·6
„ 30 „ „	5th	110·4	116·2
„ 41 „ „	6th	115	116·3
„ 47 „ „	7th	115	118
„ 54 „ „	8th	117·3	118·5
„ 58 „ „	9th	116·3	115·2
„ 55 „ „	10th	114·6	114·9
„ 37 „ „	11th	119·3	118·8
„ 28 „ „	12th	118	118·1
„ 17 „ „	13th	122	124·7
„ 13 „ „	14th	123·3	121·3
„ 7 „ „	15th	117·5	119·5

Those cases in which the observations were made on the 1st and 2d days were children; hence the high rate of the pulse at that early stage of the illness. Calculating only from the 3d day, the average morning pulse is found to be close on 116, and the average evening rate nearly 118°. On the 5th day there is no rise corresponding to the increased range of temperature. Before that day, contrary to what was observed in the case of the temperature, there is a greater difference between the morning and evening rate than is to be found later on in the case. The pulse remains pretty equable, varying only a few beats, on either side of 116 up to the 13th day, on which, and on the 14th, it is over 120; thus showing a tendency to rise at a time when the temperature showed a slight fall. From these tables it would seem that, though the pulse and temperature keep pretty well together, there is no fixed and definite relationship betwixt them. This fact will be further illustrated in an after part of the paper.

Diagnosis.—For the purposes of direct diagnosis in typhus fever, the thermometer is only of use in so far as it shows that the main characteristic of a febrile state, abnormal heat, exists; it conveys no

information which enables us to say "this is a case of typhus"—the information requisite for this must be got from other sources.

Prognosis.—When once a case is diagnosed as typhus, it must, to a conscientious practitioner, be one of more or less anxiety, and anything which enables us to form a more definite idea of the actual state of the patient, and adds certainty and precision to our knowledge, even in only a few cases, is to be regarded as a most valuable adjunct to the other means at our disposal. Such I believe the thermometer to be: alone, it is in many cases one of the least valuable of the means by which we judge of a patient's state; but, taken in conjunction with the general aspect of the patient, the state of the pulse, heart, nervous system, etc., it often gives to our prognosis a satisfactory precision which would otherwise be wanting.

To form a diagnosis, no single sign or symptom can be depended on; the eruption, the most valuable of all, is occasionally absent. To form a satisfactory prognosis it is still more necessary that everything should be considered and weighed, and each symptom assigned its due place and importance; but, as the varying circumstances of each case give to an individual symptom, or train of symptoms, a value and importance in one case which it does not possess in the next, and as nothing but personal experience and observation can give the tact and information requisite for the elimination and proper discrimination of those symptoms and signs which are to be regarded as the most important and noteworthy, it is difficult for me to convey to those who have not used the instrument a definite idea of the exact value which I would attach to the thermometer in a given case. Its value as a sign is very variable; in one case it may be the best guide to a knowledge of the patient's condition; in a second it may be of little or no service; whilst in a third, if trusted to alone, it may positively mislead us. The same thing may be said of most of the other means by which we form our estimate of a case. They vary in value in different patients, but not one ought to be completely ignored. I have been led to regard the thermometer as one of the ordinary means of attaining a knowledge of the patient's condition, and one which I would now no more think of discarding, than I would think of ceasing to note the state of the pupil, or to listen to the cardiac sounds; but, at the same time, one which I do not use regularly in all cases, because experience has taught me to discern those in which it is likely to be of use from those in which it is not likely to convey information sufficiently important to counterbalance the expenditure of time inseparable from its employment: just as any one who has seen much of typhus can, in many instances, form a perfectly accurate idea of a patient's state without daily listening to the cardiac sounds, or looking narrowly at the state of the pupil. Though I may not be able to point out the steps by which I have been led to the above position, I may indicate some circumstances which are apt to have such an influence on a case, and so affect its nature and

course, as to render it probable that the thermometer may, or may not, be of service.

The formation of a satisfactory prognosis presupposes a knowledge of the probable course of the case, and of the mode in which death is threatened. In uncomplicated typhus, the mode of fatal termination may be by coma or asthenia, or a mixture of both; death may commence at the brain, or at the heart. The former is more common in young people, the latter in those of mature years. When complicated with some pulmonary affection (as typhus so frequently is), asphyxia is apt to share in the production of the fatal result.

When symptoms referable to the nervous system exist only to a slight extent, when there is a good cardiac action, and when no complication exists, we have no hesitation in forming a favourable prognosis. When, however, the case is of such severity as to cause considerable anxiety as to the result, the first thing that the medical man has to do is to satisfy himself as to the mode in which death is threatened, to find out what is likely to be the chief source of danger; having satisfied himself on this point, he has next, by the help of all the means in his power, to calculate and estimate the amount of that danger; and here it is that the power of discrimination, and capacity for weighing and calculating the value of each particular symptom, to which I have referred, becomes of such service.

For the sake of illustrating what has already been said, and of conveying a better idea of the exact place and value of the thermometer, I shall give examples of three different kinds of cases: (*a*) uncomplicated typhus, in which the nervous symptoms ran high, and death was threatened by coma; (*b*) uncomplicated typhus, in which death was threatened by asthenia; and (*c*) typhus complicated with some pulmonary affection, in which death was threatened by asphyxia.[1]

1. A male, æt. 22, came under observation on the sixth day of illness, with the eruption out and other symptoms well marked; in morning the pulse was 108 and the temp. 103°; in evening they were 116 and 103·5°.

7th day.—Tongue much furred; patient is restless and has foreboding of death; pulse 108, temp. 101·8°: *vesp.* pulse 116, temp. 103·6°: *midnight*, is wakeful and restless; pulse 128, temp. 104·5°.

8th day.—Is restless and somewhat unmanageable; tongue dry in centre; pulse 132, temp. 104·5°: *vesp.* pulse 132, temp. 104·6°: *midnight*, eyes suffused; is restless, and with difficulty kept quiet in bed; pulse 148, temp. 102·5°.

[1] In giving these cases, I have noted the temperature along with the other symptoms in the usual manner, and have, for brevity's sake, omitted all unnecessary particulars regarding treatment. Any one interested in the matter can, in a few minutes, form a diagram which will, at a glance, show the range of temperature throughout the case.

9th day.—General state as last noted; tongue dry in centre; pulse 132, temp. 103·9°: *vesp.* pulse 136, temp. 103·8°: *midnight*, pulse 140, temp. 102·5°.

10th day.—Passed a restless night; bowels well moved after enema; cardiac sounds good; had an attack of convulsions about mid-day; pulse 144, temp. 103·6°; resp. 44, cerebral in character; urine contains a trace of albumen: *vesp.* pulse 120, temp. 103·6°: *midnight*, pulse 120, temp. 103·2°.

11th day.—Passed a quieter night; he had one or two slight convulsive attacks; abdomen is markedly tympanitic; urine contains rather more albumen; pulse 132, temp. 102·5°: *vesp.* 132 and 102·4°: *midnight*, 120 and 103·6°.

12th day.—Patient is much quieter; is heavy and tending to somnolency; urine contains less albumen; conjunctivæ injected, pupils rather small; cardiac sounds pretty good, impulse quite perceptible; pulse 112, temp. 102·9°: *vesp.* 108 and 103·2°: *midnight*, 116 and 103·6°.

13th day.—Sleeps a good deal; tympanitis very troublesome; bowels open; tongue dry in centre; pulse 108, temp. 102·1°: *vesp.* 100 and 101·1°: *midnight*, 96 and 100·5°.

14th day.—Tongue dry in centre; patient still heavy and somnolent; bladder distended; pulse 96, temp. 101°: *vesp.* pulse 92, temp. 100·2°.

15th day.—Tongue cleaning; general aspect improved; still requires the catheter; pulse 84, temp. 100°: *vesp.* pulse 104, temp. 100°.

16th day.—Continues to improve; urine drawn off by catheter; pulse 96, temp. 98·2°: *vesp.* pulse 88, temp. 98·4°.

Let us take the individual symptoms of this case, examine each separately, and try to give to each its proper value and significance, and so arrive at a just appreciation of the merits of each. The general aspect and manner of the patient, the injected conjunctiva, and almost wild expression of the eye were sufficient of themselves to lead one to regard the case as one in which the head-symptoms were likely to prove troublesome. The character of the respirations tended only to confirm this view. They were hurried and irregular, varying from 20 to 48 per minute; the inspirations were only such as might be looked for in a patient taking so many in that space of time. It was the expirations which gave the peculiar character to the breathing; instead of being of equal or greater length than the inspirations, they were short, hurried, and forcible, occupying only half the time taken up by the act of inspiration. This character of the breathing is always to be regarded as of grave import, more so than mere frequency, by which it is invariably accompanied; it indicates danger from the head-symptoms. The very rapid pulse showed that the case was a severe one, whilst the clearly perceptible cardiac impulse and good character of the systole showed that the danger was not from failure of the heart's

action. The presence of even a trace of albumen in the urine was a symptom of grave import, but, be it remarked, this piece of evidence was not forthcoming until two days after the other symptoms had led to the conclusion that the case was likely to be a severe one. It thus seems that, without any aid from the thermometer, a perfectly just estimate could have been formed of the nature and probable course of the case. All the other symptoms showed that it was a severe one, and that the head-affection was the source of danger. What, then, was the use of the instrument in this case, which has been instanced as one tending to prove its utility? *It was the first thing which gave certainty to the unfavourable opinion which had been formed of the nature of the case.* The rise of the temperature at midnight of the 7th day, and the continued high range on the 8th, gave a stamp of precision and certainty to the prognosis which at that stage of the illness would otherwise have been wanting. On the 9th and 10th days the temperature was from half a degree to a degree lower, but there was an absence of the morning fall—a want of difference between the morning and evening temperatures—which, when persistent, is a sign of unfavourable import. This continued on the 11th and 12th days. At midnight of the former a rise of more than a degree took place, and on the 12th there was no fall; the morning range was nearly as high as the evening, and higher than that of the previous evening, whilst the midnight range was highest of all. The general state of the patient at this time was one of extreme danger, and the worst prognosis was given; his pupils were small, he was getting heavy and somnolent; the urine was scanty and contained more albumen; the abdomen was tympanitic; and though the pulse had fallen from 132° to 112°, it was doubtful whether, with such other symptoms, this could be regarded as of favourable import. On the 13th morning the temperature fell from 103·6° at midnight of 12th to 102·1°; in the evening it was 101·1°; and at midnight 100·5°; thus showing that defervescence had fairly set in; the pulse fell at the same time, and though the general aspect of the case was only slightly if at all improved, there was no hesitation in pronouncing the greatest danger to be past. From that time improvement was progressive. In this case the thermometer gave the first certain sign of impending danger, and likewise the first indication that the danger was past. True it is that, contemporaneously with the rise of the temperature, there took place an even greater rise of the pulse, and that throughout the course of the case the pulse was, as compared with the normal standard, higher than the temperature. It is also true that, when the temperature fell at the commencement of defervescence, the pulse continued to keep it company; but, nevertheless, the state of the pulse did not convey to my mind such definite and satisfactory information as was derived from the temperature, and that for the following reason:—when a patient is restless, excitable, and frequently endeavouring to get out of bed and

perform other muscular feats (as this one was), the rapidity of the pulse and respirations is apt to be much increased by these means, and ceases to be an accurate and reliable guide to a knowledge of the patient's real state. The temperature, however, is not so affected. The mere muscular effort required to stand on the floor may send a typhus patient's pulse up 20 or 30 beats per minute, but has no corresponding effect on his temperature; hence it was that the fall of the pulse which took place on the 12th day, when the patient got heavy and somnolent, and ceased from his muscular efforts, was not (seeing that the temperature kept up) looked on as being of favourable import.

I shall give one more case of a similar character:

2. A female, aged 24, came under observation on 4th day of illness, with general febrile symptoms, but no eruption; pulse in morning 120, temperature 102·3°: *vesp.* pulse 120, temp. 104·5°.

5th day.—Distinct typhus rash; tongue dry in centre; pulse 112, temp. 103·9°: *vesp.* pulse 120, temp. 105·1°; cardiac action good.

6th day.—Passed rather a restless night; eyes suffused; has great thirst; pulse 108, resp. 24, temp. 103·3°; urine slightly albuminous: *vesp.* pulse 114, temp. 104·3°.

7th day.—General state as last noted; pulse 120, resp. 44; temp. 103·3°: *vesp.* pulse 108, temp. 104·4°.

8th day.—Very restless; eyes suffused; urine contains more albumen, and deposits a few granular casts; pulse 128, resp. 30, temp. 103·1°: *vesp.* pulse 132, temp. 104·3°

9th day.—Very restless and noisy all night; tongue dry; rash very copious; pulse 128; temp. 102·7°: *vesp.* pulse 148, resp. 56, temp. 104·5°.

10th day.—As at last note; pulse 128, temp. 102·7°, resp. 44: *vesp.* pulse 140, temp. 103·1°.

11th day.—Passed a quieter night; seems heavy and depressed; eyes much suffused; pupils rather small; cardiac systole plain and audible; pulse 144, resp. 54, temp. 103·9°: *vesp.* pulse 144, resp. 42, temp. 104·3°.

12th day.—Rather more restless; has considerable stupor; urine still albuminous, and depositing granular casts; pulse 138, resp. 48, temp. 104·4°: *vesp.* pulse 148, resp. 38, temp. 104·6°.

13th day.—Is heavy and stupid; swallows very badly; tongue very dry, pulse 140, resp. 36, temp. 103·2°: *vesp.* pulse 156, resp. 48, temp. 104°.

14th day.—Still considerable stupor, but swallows better; urine albuminous; pulse 132, resp. 30, temp. 102·4°: *vesp.* pulse 136, resp. 36, temp. 103·3°.

15th day.—General state unchanged; pulse 132, resp. 30, temp. 101·2°: *vesp.* pulse 136, resp. 40, temp. 101·9°.

16th day.—Has less stupor; general aspect somewhat improved; tongue moist at edges; urine albuminous, deposits urates and a

few granular casts; pulse 132, resp. 32, temp. 99·5°: *vesp.* pulse 132, resp. 38, temp. 101·1°.

17*th day.*—General improvement continues; pulse 124, resp. 30, temp. 98·8°: *vesp.* pulse 132, resp. 40, temp. 100·6°.

18*th day.*—Pulse 116, resp. 30, temp. 98·4°: *vesp.* pulse 120, resp. 28, temp. 99·9°.

19*th day.*—Pulse 116, resp. 24, temp. 97·3°: *vesp.* pulse 120, resp. 28, temp. 97·0°; tongue moist and cleaning; general aspect greatly improved.

The high range of the temperature early in this case led me to be on the look-out for head-symptoms, and to examine the urine, which was found as early as the 6th day to be slightly albuminous; as the case advanced, the albuminuria increased, and granular casts of the renal tubes were found in the deposit; the nervous symptoms ran high; the patient was restless; and had much delirium; the pupils were small, the conjunctivæ much injected, and towards the end of the case there was considerable stupor. It was not, however, till the eighth day that the general symptoms were such as to call for special notice. During the first week, the pulse was never above 120; on the eighth day, it rose and continued very high (from 132 to 156) throughout the remainder of the case; the respirations during the first six days were not high, from 24 to 32; on the 7th, they became hurried and irregular, and remained so throughout. The temperature was high *from the very first*, the evening range up to time of defervescence varying from 104·3° to 105·1°. There was thus, during the first week, nothing either in the general aspect and symptoms, or in the pulse and respirations, to induce me to regard the case with more than usual anxiety. The albuminuria was of course a most serious complication, and likely to be attended with bad head-symptoms; but it was the thermometer which, by showing a temperature of 105·1° on the 5th evening, gave the first warning note, and led to the belief that the nervous symptoms would run high, to the suspicion that there might be some renal complication, and to the formation, at an early stage of the illness, of a prognosis which the subsequent course of the case fully bore out, and which could not have been arrived at without its aid for some days later. It also gave the first indication of commencing defervescence. It was not till the 16th day that the tongue began to moisten at the edges, and that the improvement in the general aspect and symptoms of the case was such as to warrant the assertion, on these grounds alone, that defervescence had commenced: it was not till the 18th day that the pulse got down to 120; and on the 20th (on which day the observations ceased), it was still 112; whilst the temperature, which on the 11th and 12th days was high, and had shown little or no tendency to fall in the morning, gave, on the 13th day, decided signs of a decrease, which was even more marked on the 14th, and continued till the normal standard was reached. The thermometer

gave indications of commencing defervescence three days earlier than any other symptom, and enabled the opinion that defervescence had commenced to be pronounced at least two days sooner than could have been done without its aid; for of course the fall of the temperature on the 13th day, unaccompanied as it was by any other signs of improvement, would have been of little or no value unless continued on the 14th.

These two examples serve to show that in those cases in which there is danger from the head-symptoms, the thermometer is a most useful aid to the early attainment of a just estimate of the patient's state: they are given as instances well calculated to show the benefit to be derived from the employment of the instrument, and as types of a class of cases which are of frequent occurrence during an epidemic.

Let us now see if it is of equal value in those in which the nervous symptoms do not predominate, but in which the chief source of danger is failure of the heart's action.

3. A female, aged 42, came under observation on the 7th day, with the eruption out, and general symptoms well marked; on morning of that day, the pulse was 112, respirations 36, temperature 102·5°: *vesp.* pulse 116, respirations 32, temperature 104°.

8*th day.*—Passed a restless night; tongue dirty; eruption abundant; conjunctivæ injected; pulse 120, resp. 40, temp. 102·7°: *vesp.* pulse 116, resp. 40, temp. 103·8°.

9*th day.*—General condition unchanged; pulse 116, resp. 36, temp. 103·2°: *vesp.* pulse 108, resp. 42, temp. 102·4°.

10*th day.*—Is still restless; tongue dry in centre; cardiac impulse and systole rather feeble; pulse 116, resp. 38, temp. 102·9°: *vesp.* pulse 102, resp. 40, temp. 102·8°. Wine to 6 ounces.

11*th day.*—General state as last noted; pulse 128, resp. 36, temp. 103: *vesp.* pulse 128; resp. 34, temp. 103·8°.

12*th day.*—Pulse 124, resp. 36, temp. 103·3°: *vesp.* pulse 128, resp. 40, temp. 103·3°.

13*th day.*—Is more sunk in bed; tongue dry and brown; cardiac systole faint, impulse scarcely perceptible; pulse 132, resp. 36, temp. 102·9°: *vesp.* pulse 148, resp. 44, temp. 103·3°. Omit wine; whisky to 6 ounces.

14*th day.*—As last noted; pulse 138, resp. 42, temp. 102·2°: *vesp.* pulse 132, resp. 48, temp. 103·4°.

15*th day.*—Passed a quiet night; is sunk down in bed; cardiac systole faint; pulse 132, resp. 42, temp. 102·4°: *vesp.* pulse 140, resp. 48, temp. 102·3°; has wheezing in both fronts.

16*th day.*—Skin moist; tongue moistening; has general bronchitis, causing some embarrassment of breathing; pulse 136, resp. 38, temp. 100°: *vesp.* pulse 120, resp. 48, temp. 101·2°.

17*th day.*—General state much the same; pulse 140, resp. 48, temp. 98·8: *vesp.* pulse 136, resp. 50, temp. 101·4°.

18*th day.*—General improvement continues; pulse 124, temp. 98·5°, resp. irregular: *vesp.* pulse 120, temp. 98·4°.

The general symptoms in this case were more those of depression than of excitement; the pulse was rapid after the 10th day, and so far partook of the character of the pulse in the two previous cases; but the cardiac phenomena were different. Instead of a clearly perceptible impulse and audible systole, there was a very feeble impulse and a first sound faint and indistinct; the range of the temperature was equable, and not at all high; it was not a good guide to prognosis, but was a reliable index of the declension of the fever. The general aspect of the case, and the feeble compressible pulse indicated that the danger was from failure of the cardiac action, but it was the stethoscopic examination of that organ which gave precise knowledge of the actual state of affairs, and led to a correct estimate of the amount of the danger.

This case is given in illustration of a very numerous class in which, though symptoms referable to the nervous system exist to a greater or less extent, the chief danger is from failure of the heart's action; it serves to show that in such cases the information derived from the thermometer is of little positive value, and that for the formation of a just estimate of the patient's condition, we must rely mainly on the evidence afforded by a stethoscopic examination of the heart. For the sake of further illustrating this point, I shall instance two cases of uncomplicated typhus, in which the thermometer not only conveyed no positive information on which to found a prognosis, but in which it even tended to mislead.

4. A female, aged 18, came under observation on the seventh day, with the eruption out and general symptoms well marked.

7th day.—Pulse 150, temp. 104·6°: *vesp.* pulse 144, temp. 104°.

8th day.—Pulse 150, temp. 104°: *vesp.* pulse 144, temp. 102·3°.

9th day.—Pulse 136, temp. 102·1°: *vesp.* pulse 148, temp. 102·6°.

10th day.—Is much depressed; extremities cold; cardiac impulse scarcely perceptible, systole feeble; pulse 144, temp. 100°; to have wine to 8 ounces: *vesp.* pulse 156; temp. 102·6°.

11th day.—Has rallied considerably, but still very low; pulse 132, temp. 100·8°: *vesp.* pulse 132, temp. 101·5°.

12th day.—General state little changed; pulse 132, temp. 100°: *vesp.* pulse 144, temp. 104°; cardiac systole still very feeble.

13th day.—Pulse 120, temp. 102·2°: *vesp.* pulse 128, temp. 103·5°.

14th day.—Has improved expression; tongue dry; pulse 120, temp. 100·3°: *vesp.* pulse 112, temp. 102°.

15th day.—General condition unchanged; pulse 108, temp. 99·8°: *vesp.* pulse 108, temp. 101°.

16th day.—Tongue moist; has improved aspect; cardiac sounds better; pulse 96, temp. 99·5°: *vesp.* pulse 92, temp. 99·2°.

17th day.—Pulse 100, temp. 99°: *vesp.* pulse 96, temp. 98°.

The pulse in this patient was very rapid, ranging from 120 to 156, never getting below the former number till defervescence commenced; she had the general aspect and signs of depression, and

a cardiac systole faint and soft: the temperature, which was 104·6° on the 7th evening, fell somewhat on the next day, continued to fall on the 9th, and on the 10th morning was down to 100°. This low range, continued for two days, might, unless the other symptoms had been attended to, have been regarded as of favourable import; as it was, no signification could be attached to it, further than to regard it as the result of the great depression under which the patient was labouring, as indicated by a feeble and rapid pulse, coldness of the extremities, and a scarcely audible cardiac systole. The patient was freely stimulated, and under this treatment rallied considerably. On the 12th evening, the temperature was again up to 104°. On the 14th day defervescence began and was progressive.

5. The next case was similar in character, but even more marked. A male, aged 53, came under observation on the 6th day, with the eruption and general symptoms well marked.

7th day.—Pulse 120, temp. 103·8°: *vesp.* pulse 120, temp. 104·3°.

8th day.—Tongue moist; there is considerable delirium; has some hypostatic congestion of bases of both lungs posteriorly; pulse 124, temp. 104·2°: *vesp.* pulse 116, temp. 104·8°.

9th day.—General state unchanged; cardiac sounds fair; pulse 120, temp. 103·5°: *vesp.* pulse 108, temp. 103·4°.

10th day.—Passed a restless night; very delirious; constantly endeavouring to get out of bed; pulse 120, temp. 103·2°: *vesp.* pulse 120, temp. 103°.

11th day.—Is much sunk; pulse scarcely perceptible; eyes suffused; tongue dry in centre; is unable to swallow; apex beat not felt; first sound very feeble; pulse about 120, temp. 99·6°; to have an ounce of whisky with a pint of beef-tea as enema every hour: *vesp.* pulse 112, temp. 99·9°; can swallow; whisky to 12 ounces.

12th day.—Is considerably revived; cardiac systole still very feeble; pulse 112, temp. 99·5°: *vesp.* pulse 108, temp. 98·7°.

13th day.—Has muscular tremors; tongue dry; heart's sounds faint; pulse 112, temp. 99·2°: *vesp.* pulse 112, temp. 98·6°; continue stimulants.

14th day.—General state unchanged; pulse 108, temp. 97·2°: *vesp.* pulse 100, temp. 97°.

15th day.—Has still muscular tremors and great depression; cardiac sounds better, but still feeble; pulse 108, temp. 97·2°: *vesp.* pulse 108, temp. 96·4°.

16th day.—Passed a restless night; has still muscular twitches; pulse 112, temp. 97°: *vesp.* pulse 112, temp. 96·6°. Continue stimulants.

17th day.—Tongue dry in centre, pulse 120, temp. 98·3°: *vesp.* pulse 108, temp. 96·8°.

18th day.—Pulse 116, temp. 97·3°: *vesp.* pulse 116, temp. 98·2°.

19th day.—Tongue cleaning; cardiac sounds improved; pulse

108, temp. 98·2°: *vesp.* pulse 108, temp. 97·8°; whisky to 8 ounces.

As was to be expected in a man of his years, all the symptoms were well marked; he was restless and delirious at night; there was hypostatic congestion of bases of both lungs posteriorly; the pulse varied from 116 to 124, and the temperature during the first week was high (104·8° on 8th evening). On the 10th day, the pulse was 120, and the temperature 103·2°, the general condition being as above; on the 11th day, the temperature was, in morning 99·6°, and in evening 99·9°; on the 12th and 13th days, this fall continued, and on the 14th day, it was down to 97°, and remained below the normal standard till the 20th day. A fall of 3½° on the 11th day of illness, and a continued low range on succeeding days, would in most cases be proof of commencing convalescence; in this case, however, it was otherwise, for the patient's condition was one of the utmost danger; he was suffering from extreme depression; the pulse was small and scarcely perceptible, the eyes suffused, the tongue dry, the cardiac impulse not to be felt, and the first sound faint and distant; the patient was in imminent danger, could not swallow, and was only rallied by having an ounce of spirit, along with a pint of beef-tea, given as an injection every hour until he could take nourishment by the mouth. He gradually and slowly recovered, but it was not till the 19th day that the general state of depression, as indicated by the occurrence of muscular tremors, feeble cardiac action, etc. was such as to permit of a diminution below 12 ounces of the daily quantity of spirits; and yet, during the entire week in which the patient was in this critical state, the temperature was low, generally below the normal standard, and never reached 100°. The thermometer alone would have given a dangerously false idea of the state of affairs: the general aspect of the patient, and the condition of the heart as revealed through the stethoscope, were the proper guides to the formation of a just prognosis, and to the adoption of a proper mode of treatment. It may be said that the low range of the temperature was an index of the state of depression under which the patient laboured. Grant that it was so, the information derived from it could not, for a moment, be compared in value or precision with that which was derived from the other symptoms, and especially from the stethoscopic examination of the heart.[1]

To illustrate those cases in which there was danger from asphyxia, I shall choose two which were complicated with bad bronchitis.

6. A female, aged 19, came under observation on the 7th day; at that time the eruption was out, the tongue dirty, and there existed wheezing all over the chest; the respirations were hurried and irregular; pulse 132, temp. 102·9°: *vesp.* pulse 132, temp. 103·4°.

[1] In this case may also be noted the fact, that with a high temperature during the first week there were very prominent head-symptoms during the early part of the second.

8th day.—Passed a noisy night; conjunctivæ injected; pupils of good size; pulse 132, temp. 103·5°: *vesp.* pulse 132, temp. 103·9°; is very delirious.

9th day.—Passed a better night after having an opiate; is now quieter; pulse 132, temp. 105·1°: *vesp.* pulse 132, temp. 105°; is noisy and delirious.

10th day.—Is more restless; bronchitis very troublesome; breathing greatly embarrassed; face and lips turgid; abundant sibilation, mingled with mucous râles all over chest; pulse 144, temp. 104·4°: *vesp.* pulse 132, temp. 102·7°.

11th day.—Breathing very bad, almost gasping; lips blue and face congested; pulse 132, temp. 101·1°: *vesp.* pulse 144, temp. 104·4°.

12th day.—General state much the same; cardiac action pretty good; pulse 132, temp. 103·6°: *vesp.* pulse 132, temp. 104·4°.

13th day.—As last noted; tongue dry; rests very little at night; pulse 120, temp. 101·4°: *vesp.* pulse 132, temp. 103·2°.

14th day.—Has improved expression; tongue moistening; chest symptoms still troublesome; pulse 120, temp. 100·7°: *vesp.* pulse 130, temp. 101·8°.

15th day.—Is still restless and somewhat noisy; tongue cleaning; pulse 132, temp. 101°: *vesp.* pulse 120, temp. 101·2°.

16th day.—Eruption faded; bronchitic symptoms still very marked; expectoration abundant; patient is still noisy and unmanageable; pulse 120, temp. 99·2: *vesp.* pulse 120, temp. 101·1°.

17th day.—Shows signs of general improvement in a more marked degree; pulse 108, temp. 98·6°: *vesp.* pulse 116, temp. 99·1°.

Improvement continued, but was very gradual; the temperature was some days in reaching the normal standard, and the pulse was still later in doing so; the bronchitic symptoms did not totally disappear for some weeks, and for three or four days after defervescence had really commenced the patient was still noisy and restless at night.

The first noticeable fact in the case is the high range of the temperature (105·1° on 9th morning) in connexion with the prominence of the head-symptoms; the chief reason for instancing it, however, is to show how, at the time when the patient was in the utmost danger from the bronchitis, and when great fears were entertained as to the result, the temperature fell from 104·4° (10th mo.) to 101·2° (11th mo.), more than 3° in 24 hours; had the patient been other than young and vigorous, it is probable that she would not have rallied; as it was, the cardiac action was good, and with the help of appropriate remedies, enabled the patient to struggle against the pulmonary embarrassment; by the evening of the 11th day, she had rallied considerably, and the temperature again rose to 104·4°.

Here the thermometer, if trusted to alone, would have misled;

when taken in conjunction with the other symptoms, it suffers so by the contrast that its utility, so far as the estimation of the real danger from the bronchitis was concerned, seems almost *nil*. The cardiac action gave no indication of the danger, but was of use in showing that there was little or nothing to fear from failure in that quarter; the greatly embarrassed breathing, the blueness of the lips and face, and the stethoscopic examination of the chest, were the only criteria by which an exact knowledge of the patient's condition could be reached, and a correct estimate formed of the probability or otherwise of a fatal result.

The next case is one which proved fatal, and in which death was due mainly to the bronchitis.

7. A female, aged 31, came under notice on the 5th day, with the eruption out; pulse 116, temp. 103·3°: *vesp.* pulse 116, temp. 104·3°.

6*th day.*—Is restless; pulse 124, temp. 103·5°: *vesp.* pulse 124, temp. 103·5°.

7*th day.*—Cardiac sounds rather feeble; apex beat scarcely felt; pulse 124, temp. 102·6°: *vesp.* pulse 140, temp. 101·6°.

8*th day.*—Urine contains albumen and a few granular casts, sp. gr. 1028; pulse 112, temp. 101°, resp. 40: *vesp.* pulse 132, temp. 103·1°, resp. 48.

9*th day.*—General state unchanged; pulse 116, temp. 100·1°, resp. 40: *vesp.* pulse 124, temp. 102·3°, resp. 48.

10*th day.*—Cardiac sounds feeble, but quite audible; urine albuminous; pulse 116, temp. 101·4°, resp. 40: *vesp.* pulse 120, temp. 100·8°, resp. 40.

11*th day.*—Pulse 132, temp. 102·9°, resp. 44: *vesp.* pulse 128, temp. 103·2°, resp. 48.

12*th day.*—Passed a restless night; tongue moist; pulse 128, temp. 100·7°, resp. 44: *vesp.* pulse 122, temp. 103·6°, resp. 48.

13*th day.*—Passed a restless night; passes water and stools in bed; tongue dry in centre; rather feeble cardiac systole; has some wheezing in chest; pulse 128, temp. 101·1°, resp. 40: *vesp.* pulse 120, temp. 101·9°, resp. 42, irregular.

14*th day.*—Has general bronchitis; there is great wheezing all over chest, and considerable embarrassment of breathing; pulse 116, temp. 100·6°, respirations irregular; pulse 112, temp. 99·7°.

15*th day.*—Is greatly depressed; abundant sibilation mingled with mucous râles all over the chest; tongue dry; urine still albuminous; pulse 108, temp. 97·8°, resp. 48: *vesp.* pulse 156, temp. 100·2°, resp. 54.

16*th day.*—Moribund; pulse 120, temp. 98°, resp. 54. Died at midday.

There are several points worthy of note in this case: (*a*) The occurrence of albuminuria was a source of anxiety, and led to the expectation of marked nervous symptoms; (*b*) the temperature,

however, was not so high as to point to danger from that quarter; and the subsequent course of the case showed that the thermometer had not misled in that respect, for the head-symptoms were never such as to cause unusual anxiety; (*c*) the pulse and temperature varied much during the case; and (*d*) when bronchitis set in, the pulse and respirations kept up, whilst the temperature fell a little below the normal standard, and at time of last observation, a few hours before death, stood at 98°. Up to the time of the onset of the bronchitis, neither the pulse, temperature, nor general symptoms, indicated unusual danger; the first sound of the heart was rather feeble, but not so much so as to preclude a hopeful prognosis: with the occurrence of that complication, the general symptoms were, of course, altered by the addition of those referable to the pulmonary organs; the pulse underwent little change; the respirations became irregular and increased in rapidity; while the temperature fell, and continued to do so till it reached the normal standard. Here, as in the last case, the amount of the danger could be estimated neither by the pulse nor the temperature, but only by attention to the general symptoms caused by the pulmonary complication, and by a stethoscopic examination of the chest. In both instances, the fall of the temperature might be taken as an indication of the extent to which the pulmonary obstruction interfered with the proper oxygenation of the blood, and such, to a certain degree, it undoubtedly was, but the information so obtained was wanting in the precision and accuracy which accompanied that got from the other sources already mentioned, and would alone have been of little or no real value.

There is yet another class of cases to which I would allude, and of which I would give an instance. I refer to those not very common ones in which the eruption and general symptoms are so well marked as to leave no room for doubt as to their real nature, but in which the pulse rises little, if at all, above the normal standard.

8. A male, aged 27, came under observation on the eighth day of his illness.

8*th day.*—Abundant typhus rash; tongue dry in centre; conjunctivæ injected; pulse 68, resp. 26, temp. 103·2°: *vesp.* pulse 68, resp. 32, temp. 104·8°.

9*th day.*—Pulse 72, resp. 32, temp. 104·2°: *vesp.* pulse 64, resp. 34, temp. 104°.

10*th day.*—Eruption dingy; tongue dry in centre; passes water in bed; pulse 64, resp. 32, temp. 103·4°: *vesp.* pulse 80, resp. 32, temp. 104·4°.

11*th day.*—Passed a restless night; has considerable delirium; catarrhal wheeze in both fronts; tongue dry and brown; pulse 64, resp. 32, temp. 102·3°: *vesp.* pulse 78, resp. 30, temp. 103·3°.

12*th day.*—Eruption less dingy; otherwise as last noted; pulse 64, resp. 28, temp. 101·3°: *vesp.* pulse 56; resp. 24, temp. 102·3°.

13*th day.*—Tongue moist at edges; pulse 60, resp. 24, temp. 99·1°: *vesp.* pulse 56, resp. 20, temp. 99·3°.

14*th day.*—Pulse 40, resp. 18, temp. 97·3°: *vesp.* pulse 48, resp. 18, temp. 99·6°.

15*th day.*—Tongue moist and cleaning; has generally improved aspect; pulse 36, resp. 18, temp. 98·8°: *vesp.* pulse 48, resp. 24, temp. 97·5°.

16*th day.*—Pulse 36, resp. 16, temp. 96·2°: *vesp.* pulse 42, resp. 18, temp. 97·4°.

Here, then, was a well-marked case of typhus, with distinct nervous symptoms, and abundant dingy eruption, in which the pulse only once got up to 80, was habitually below the normal standard, and was, during the first week of convalescence, on two occasions, as low as 36, and not once above 48. The respirations were distinctly but not greatly increased in frequency. The temperature was the best, in fact the only means by which the amount of febrile disturbance could be accurately estimated.

9. A male, aged 21, came under observation after six days' illness.

7*th day.*—Skin hot; tongue furred; typhus eruption distinct; has a strong heaving cardiac impulse, and a slight diastolic blow at base; pulse 84, resp. 32, temp. 102·7°: *vesp.* pulse 88, resp. 40, temp. 103·4°.

8*th day.*—Has considerable stupor; eruption copious; pulse 88, resp. 38, temp. 102·1°: *vesp.* pulse 84, resp. 36, temp. 104·1°.

9*th day.*—Tongue dry in centre; pulse 88, resp. 32, temp. 103°.

10*th day.*—Pulse 84, resp. 28, temp. 103·2°: *vesp.* pulse 88, resp. 28, temp. 104°.

11*th day.*—General condition as last noted; pulse 84, resp. 32, temp. 102·7°: *vesp.* pulse 84, resp. 48, temp. 103·1°.

12*th day.*—Pulse 80, resp. 28, temp. 100·5°: *vesp.* pulse 80, resp. 28, temp. 102·2°.

13*th day.*—General state much the same; tongue not quite so dirty; pulse 76, resp. 32, temp. 102·3°: *vesp.* pulse 72, resp. 28, temp. 101·2°.

14*th day.*—Has improved expression; tongue cleaner; pulse 68, resp. 26, temp. 99·5°: *vesp.* pulse 72, resp. 28, temp. 99·2°.

15*th day.*—Improvement continues; pulse 64, resp. 18, temp. 97°: *vesp.* pulse 64, resp. 18, temp. 97·6°.

The remarks made regarding the last case are applicable to this one; the thermometer gave the best indication of the amount of "fever" which existed, the pulse being of no value in determining that point.

The cases already instanced (except the last two) are types of those in which there was danger of a fatal termination by coma, by asthenia, or by asphyxia. I shall now give the particulars of a well-marked, but mild case, which never presented any unfavourable symptoms, and which contrasts admirably with the one which I have instanced first, in which the nervous symptoms were high, and went on to convulsions. The cases were those of two gentlemen who acted as my assistants; they both had the same duties to

perform, lived in the same manner, took ill about the same time, and were similarly situated in all respects, so far as external circumstances were concerned; they were, moreover, of the same age.

10. A male, aged 22, had the first observation made on the fourth evening of his illness, at which time the skin was hot and had a few elevated spots on it; the tongue was slightly furred; the pulse 104, resp. 24, temp. 103·3°.

5*th day.*—Distinct typhus rash; pulse 104, resp. 24, temp. 102·7°: *vesp.* pulse 104, resp. 24, temp. 103·5°.

6*th day.*—Had a pretty good night; is quite rational; tongue slightly furred; pulse 104, resp. 24, temp. 102·8°: *vesp.* pulse 104, resp. 24, temp. 103·3°.

7*th day.*—Pulse 108, resp. 26, temp. 102·7°: *vesp.* pulse 108, resp. 28, temp. 103·1°.

8*th day.*—Pulse 108, resp. 20, temp. 102·4°: *vesp.* pulse 116, resp. 28, temp. 103°.

9*th day.*—General state much as last noted; occasionally wanders slightly; pulse 116, resp. 20, temp. 102·2°: *vesp.* pulse 104, resp. 24, temp. 102·7°.

10*th day.*—Passed a good night; skin moist; tongue dry in centre; pulse 104, resp. 24, temp. 102·8°: *vesp.* pulse 108, resp. 26, temp. 103·4°.

11*th day.*—Slept well; skin moist; tongue dry in centre; pulse 104, resp. 20, temp. 100°: *vesp.* pulse 100; resp. 20; temp. 102·4°.

12*th day.*—General state unchanged; tongue moist; pulse 92, resp. 22, temp. 100·5°: *vesp.* pulse 88, resp. 18, temp. 99·4°.

13*th day.*—Expression natural; tongue clean; skin cool; pulse 80, resp. 18, temp. 97·7°; *vesp.* pulse 76, resp. 18, temp. 98·5°.

Here, along with a mild train of general symptoms, the pulse, respirations, and temperature showed a low range and marked equability throughout the case; in both respects it contrasts strongly with the one given first.

Treatment.—In mild cases, which tend to recovery, there is no occasion for any active treatment; a sensible nurse is the chief requisite. In severe cases, tending to death, prompt and judicious treatment is of the utmost importance. I have already pointed out the kind of cases in which the thermometer is of most use in prognosis. The same circumstances which teach us, at an early stage, to be on the outlook for head-symptoms, lead us also to adopt such measures as are calculated to ward off the threatened danger, and mitigate the severity of the attack when it does occur. When a high range of temperature indicates danger from this source, every care should be taken to keep the patient as quiet as possible,—the light should not fall on his face; his bowels should be moved every day by castor-oil or an enema, if necessary; his head should be shaved, and cold kept constantly applied; sleep, if not obtained naturally, should be brought on by opium (unless specially contra-indicated), with or without antimony, according as the pupil is

small or large—a well-timed opiate is, I am confident, often the means of saving a patient's life; the bladder should be regularly examined to see that there is no retention—*the nurse's assurance that the patient has made water, should never be trusted to*, as incontinence and retention often go together. The bladder may be not only full, but running over; when such is the case, every care should be taken to promote the patient's comfort by keeping him as clean and dry as possible. Notwithstanding all these precautions, should he continue to get worse, and coma or even convulsions be apprehended, the scalp should at once be blistered, either by the ordinary emplastrum cantharidis, or (what I believe is better, as acting more speedily,) the acetum cantharidis,—of course care must be taken that the scalp is closely shaved, and that when the acetum is employed, it is of good quality, and not merely painted on, but rubbed in. As long ago remarked by Graves, the good effects of the cantharides are often brought about by applying it only for a sufficient length of time to produce a stimulant and rubeficient action, not pushing it to vesication, and repeating the application as circumstances may require; that is, I believe, the best mode of using it for the alleviation of comatose symptoms when they come on gradually and are not immediately alarming. In cases, however, in which the coma is deep or has come on rapidly, and in those in which convulsions supervene—in cases, in short, in which the head-symptoms are a source of immediate and urgent danger—it is better to push it on to vesication at once, and, for this purpose, the acetum cantharidis, employed as above indicated, is more prompt than the emplastrum. By the early adoption of the above measures, I feel confident that, in not a few cases, the nervous symptoms have been kept in check; and that in several of the cases in which they went on to coma and convulsions, they were so far mitigated as to be more easily controlled by the more active measures required for the treatment of these dangerous symptoms. Of eight cases complicated with convulsions, four recovered,—an unusually large proportion.

The great use of the thermometer, so far as treatment is concerned, is to point out, during the first week, the cases in which the head-symptoms are likely to be a source of danger, and so enable us to adopt proper precautionary measures sooner than they would be indicated by the other symptoms. It is useful in treatment in the same cases in which it is useful in prognosis. Where death is threatened by asthenia or by asphyxia, it is of little or no service; the general symptoms, and condition of the heart and lungs, being in such cases the best guides to the proper line of treatment.

Every case which I have instanced may be regarded as a well-marked type of a class; it must not be supposed, however, that all cases can be ranged under one or other of the heads indicated above. In the great majority, both the cardiac and nervous symptoms are well marked, though the nervous apparently preponderate, in consequence of their more obvious character. Every one who

has seen much of typhus, knows that it is often difficult to say whether there is greater danger from failure of the heart's action or from coma; such cases require for their treatment the utmost nicety of judgment, and at the same time a prompt decision, which nothing but experience can give. When coma and asthenia clash (if I may use the term), the case is usually well advanced, and minutes are precious; indecision on the part of the physician is apt to be attended with the most serious consequences to the patient. Given such a case, what are we do? With the patient comatose, and the pupils small, can we act on the cardiac evidence and give alcohol freely? With a heart's first sound inaudible, and an impulse not to be felt, is it safe to create a discharge by blistering the scalp?—Yes; most undoubtedly. If you don't blister, the best means of overcoming the coma and rousing the patient are neglected; if you don't stimulate freely, the heart's action will fail. Cardiac feebleness does not contra-indicate blistering, if the head-symptoms call for it; the presence of coma is no reason for not stimulating, if the heart requires it. Spirits may be given and that freely, and their employment in many such cases beneficially supplemented by camphor and ammonia, remedies which are often of great service under such circumstances; as their action is transient, too long an interval should not be allowed to elapse between the doses. Finally, in consequence of the patient's inability to swallow, it may be necessary to administer both stimulants and nourishment per rectum. I have, occasionally, seen cases apparently hopeless saved by this means. Such cases should never be given up; treatment should be persevered in to the last; the patient may be just at the turn, requiring only to be kept alive for a few hours until the disease has run its course, and defervescence commences. At this stage of such a case, the thermometer conveys no information which can guide us in treatment; indeed, the symptoms are all so fully developed that no aid is required from it. It is often useful, however, in giving an early indication of commencing improvement.

The following statement shows that the thermometer is the best index of commencing defervescence:—

In 81 cases, in which the fact was specially noted, the fall of the temperature *alone* gave the first indication of commencing improvement in 18; the temperature and pulse declined simultaneously in 32; improvement in the general aspect and symptoms occurred simultaneously with a fall of pulse and temperature in 27; improvement in the general symptoms and a fall of the temperature preceded a fall of the pulse in 4: thus a fall of the temperature was one of the earliest indications in 100 per cent. of the cases; a fall of the pulse in 72·8 per cent.; and improvement in the general symptoms in 38·2 per cent. In making this calculation, I have omitted all cases in which there was any doubt as to the time at which improvement began. In connexion with the above statement must be borne in mind the fact already noted, that a fall of the temperature may be the result of great and alarming depression.

The average time occupied by the process of defervescence, that is to say, the time which elapsed between the hour at which a progressive fall of the temperature was first noticed, and that at which it reached the normal standard, was, in these 81 cases, 41 hours; the longest was 96 hours. In 13 cases, or in 16 per cent. of the whole, the fall was sudden, by crisis, and was completed between the morning and evening, or between the evening and morning observations; whilst in the remaining 68, or 84 per cent., it was by lysis, was gradual, requiring for its completion one, two, three, or even four days. In complicated cases, the temperature was often kept up for a length of time by the secondary affection.

Whether or not the temperature is raised by the complicating malady, seems to depend on the nature of the secondary disease, and the time at which it appears; when it springs up during convalescence, it raises the temperature if its nature is inflammatory.

The pulmonary complications which usually occur during the height of the fever, even though of an inflammatory nature, do not, so far as my experience goes, necessarily have this effect; and often, when fully developed (as in the two cases already instanced), tend to lower it. I am aware that this does not quite accord with what has been found by other observers, but honesty compels me to say, that whilst an elevation in the temperature has occasionally been noted at the commencement of an attack of bronchitis or pleurisy, I have seen several cases in which pleurisy, and many in which bronchitis occurred, without finding the thermometer give any indication of their presence. The action of pneumonia in this respect I have never had the opportunity to test. The only two affections which I have found invariably raise the temperature, are parotid swelling and inflammation of the ear; and only in the latter has the increased heat ever given the *first* indication of something wrong, and led to the discovery of the secondary affection.

Hæmorrhages (epistaxis, abortion, etc.), and diarrhœa, never have an elevating, but frequently a lowering tendency; this latter is, in great part, due to the fact that they are apt to occur towards the end of the case, when they may be of a critical character, and when the temperature, independently of their occurrence, may be expected to fall.

I do not think that age has much influence in increasing the range of the temperature in typhus; any apparent action which it has in that respect, is probably due to the fact that the blood-poison is more apt to produce acute head-symptoms in the young. The pulse is undoubtedly more frequent in children during the fever as well as during health, but this difference is not apparent after puberty has been reached. I have made a calculation of all my cases which recovered, and find that in those under 15 years the mean temperature was, in morning 102·8°, in evening 103·8°, the pulse being at same time 116 and 117; in those from 15 to 30, it was 102·9° in morning, and 103·4° in evening, the pulse being 112 and

112; in those above 30 the averages were—for the temperature 102·6° and 103·1°, and for the pulse 111 and 113.

Neither does sex have any practically appreciable influence; if it has any effect at all, the preponderance is probably on the female side, and may be accounted for by the greater delicacy of the nervous organization in that sex. In my cases (which by mere chance are exactly half males and half females), the average temperature for males was, in morning 102·6°, and in evening 103·3°; for females it was, at the same times, 103·3° and 103·4°.

I have never found the temperature of the ward have any effect on that of the body during the height of the fever.

My experience quite accords with that of other observers, when they say that the temperature invariably falls below the normal standard during convalescence, and remains so for some time. My own observations lead me to regard the generally adopted standard, 98·4°, as being, as nearly as possible, correct.

The facts given above serve to illustrate those on which are founded the inferences which I have been led to draw, after a large number of observations made on all sorts of cases. I shall try to summarize them, but would premise this by the warning, that the temperature should not be looked to alone, but should be taken in conjunction with all the other symptoms, and that any apparent neglect of reference to them in my remarks is to be attributed to a desire to be as brief as possible.

1. In the formation of a diagnosis, the thermometer gives, at an early stage of the illness, no evidence sufficient to enable us to distinguish typhus from other forms of continued fever.

2. As an aid to the formation of a satisfactory prognosis, and as a guide to a proper line of treatment, its value varies in different cases.

3. It is of most value in those in which there is likely to be danger from the head-symptoms: a high temperature during the first week being the earliest indication of danger from that source.

4. A morning temperature of 104°, or an evening temperature of 105, during the first week, and a morning range of 103·8° with an evening one of 104·5°, after that time, should teach us to treat the case as one likely to be endangered by the severity of the head-affection.

5. The highest range occurs in cases which prove fatal by acute head-symptoms, and in which there is partial or complete suppression of urine.

6. Irregularities and abnormalities in the range often characterize the course of severe cases, and are of specially unfavourable import when occurring in those in which a high temperature has been noted at the end of the first week.

7. Equability of the temperature is one of the usual characteristics of a mild case.

8. A rise at midnight, and a *continued* absence of a morning fall, are (when the range is high) still further indications of dangerous

nervous symptoms, though their absence does not denote the contrary.

9. When probable failure of the heart's action is the source of anxiety, the thermometer is of no value in enabling us to estimate the exact amount of the danger; the state of the heart, as revealed by a stethoscopic examination of that organ, being the best guide to a knowledge of the patient's condition.

10. When pulmonary complications exist to so great an extent as to threaten death by asphyxia, the thermometer conveys no reliable information regarding the amount of the risk, this being best estimated by attention to the general state of the patient, and by the stethoscopic examination of the chest.

11. In cases in which (either from greatly enfeebled cardiac action, or from pulmonary obstruction causing deficient oxygenation of the blood) the patient gets into a state of great depression, the temperature may fall to, or even below, the normal standard, indicating, not a commencing defervescence, but an extreme degree of depression, the exact extent of which, and the danger to be apprehended from it, are best estimated by a reference to the causes which produced it.

12. In ordinary uncomplicated cases which go on to a favourable termination, the temperature generally attains its maximum about the time of development of the eruption.

13. In consequence of the non-liability of the range of the temperature to be affected by various causes which have a disturbing influence on the pulse and respirations, the thermometer is the most generally accurate means of estimating the amount of febrile disturbance which exists.

14. As an indication of commencing defervescence, a fall of the temperature is earlier and more reliable than any other single sign of improvement.

15. The effect on the temperature of complications springing up during the course of the fever varies according to the nature of the secondary affection, and the time at which it appears.

16. Complications coming on during convalescence seem to act, in this respect, just as if no febrile attack had preceded them.

In conclusion, I would repeat, that the chief use of the thermometer in practice is to enable us, during the first week, to recognise and pick out with greater precision those cases in which the head-symptoms are likely to run high; later on in the case, when all the symptoms are fully developed, its chief use is to give an early indication of commencing defervescence. It must also be borne in mind, that severe head-symptoms indicate a probably protracted case; and that in elderly persons, and in young people of weak constitution, there is likely, towards the end of the second week, to be superadded to the other risks the additional one of enfeebled cardiac action, requiring its own treatment, but not interfering with that called for by the head-symptoms. As compared with those last mentioned, I believe that all the other indications of the thermometer are of secondary importance.

ENTERIC FEVER.

The thermometry of this disease is most interesting. Its chief feature is the tendency which the temperature shows to fall in the morning and rise in the evening. This characteristic is more marked at some stages of the disease than at others; but, in cases tending to a favourable termination, is never altogether wanting, and at the commencement of defervescence becomes so exaggerated as to be quite a distinguishing feature of the disease.

This characteristic range, as well as its practical utility in diagnosis, prognosis, and treatment will be best shown by giving a few illustrative cases. For this purpose, I shall give instances of three classes:—(*a*) Well-marked cases tending to recovery, regarding whose nature there could be no doubt; (*b*) Cases which, in the severity of the head-symptoms, so closely resembled typhus as to render a faulty diagnosis possible; (*c*) Cases in which the symptoms were so mild as to leave room for doubts as to their real nature.[1]

I. A female, aged 16, came under observation on the 7th day of an illness which had commenced with shivering, headache, and general malaise.

7th day.—Tongue moist and slightly furred; eyes clear, pupils large; no eruption; bowels regular; pulse 104, temp. 100°: *vesp.* pulse 104, temp. 104°.

8th day.—Has had one stool of good consistence; pulse 104, temp. 100·3°: *vesp.* pulse 112, temp. 103°.

9th day.—No eruption; pulse 112, temp. 101·6°: *vesp.* pulse 112, temp. 102·4°.

10th day.—Tongue furred in centre, red at tip and edges; has several rose-coloured spots on abdomen; pulse 102, temp. 100·6°: *vesp.* pulse 104, temp. 102·6°.

11th day.—Several fresh spots; pulse 96, temp. 100°: *vesp.* pulse 112, temp. 102°; has had no stool for two days; to have one teaspoonful of castor-oil.

12th day.—Had three loose stools after oil; no fresh spots, the old ones fading; pulse 96, temp. 99·2°: *vesp.* pulse 108, temp. 102°.

13th day.—Eruption faded; pulse 100, temp. 99·5°: *vesp.* pulse 92, temp. 101·8°.

14th day.—General state unchanged; rests pretty well at night; is quite intelligent and rational; eyes clear, pupils large; pulse 108, temp. 99·6°: *vesp.* pulse 112, temp. 103°.

15th day.—No stool since 12th day; pulse 96, temp. 100·8°; to have half an ounce of castor-oil: *vesp.* pulse 120, temp. 104·5°.

16th day.—Has had two loose stools; complains of sore throat; fauces are congested; pulse 116, temp. 103°: *vesp.* pulse 116, temp. 105·1°.

17th day.—Throat as before; has slight tenderness in right ilium; pulse 100, temp. 103·2°: *vesp.* pulse 116, temp. 104·4°.

18th day.—Tongue dry in centre; has a small ulcer on each tonsil; one loose stool; pulse 112, temp. 102·2°: *vesp.* pulse 116, temp 103·5°.

19th day.—Has had two ochrey stools; rests well; intellect is quite clear; pulse 108, temp. 101·2°: *vesp.* pulse 112, temp. 104°.

20th day.—Two loose stools; tongue dry in centre; has several fresh spots on abdomen; pulse 104, temp. 101·6°: *vesp.* pulse, 108, temp. 103·2°.

21st day.—One ochrey stool; tongue moist; pulse 112, temp. 101°: *vesp.* pulse 112, temp. 102·2°.

[1] For brevity's sake all details regarding treatment, etc., have been omitted.

22*d day*.—Pulse 100, temp. 97·8°: *vesp.* pulse 112, temp. 102·1°.
23*d day*.—Has had one stool of good consistence; pulse 92, temp. 98·1°: *vesp.* pulse 116, temp. 102·2°.
24*th day*.—Pulse 96, temp. 97·5°: *vesp.* pulse 120, temp. 102·8°.
25*th day*.—Pulse 96, temp. 98·4°: *vesp.* pulse 112, temp. 102·8°.
26*th day*.—General improvement continues; pulse 96, temp. 97·4°: *vesp.* pulse 100, temp. 100·3°.
27*th day*.—Pulse 96, temp. 97·7°: *vesp.* pulse 116, temp. 99·9°.
28*th day*.—Pulse 80, temp. 98·2°: *vesp.* pulse 96, temp. 99°.

II. A female, aged 17, came under observation on the 7th day of her illness.

7*th day*.—Tongue slightly furred; eyes clear; no eruption; pulse 120.
8*th day*.—Tongue red at tip and edges; expression languid; sclerotics pearly; has several rose-coloured spots on the chest and abdomen; no stool; pulse 112, temp. 104·3°: *vesp.* pulse 120, temp. 104·5°.
9*th day*.—Several fresh spots; no stool; pulse 124, temp. 104·4°; to have ℥ij of castor-oil: *vesp.* pulse 108, temp. 104·3°.
10*th day*.—Bowels moved once after oil; pulse 120, dicrotous, temp. 102·8°: *vesp.* pulse 120, temp. 104·4°.
11*th day*.—Two ochrey stools; several fresh spots; first crop faded; pulse 132; temp. 104·2°: *vesp.* pulse 124, temp. 104·5°; wanders at night.
12*th day*.—One good stool; has circumscribed flush on each cheek; sclerotics pearly; pupils large; pulse 124, temp. 104·6°: *vesp.* pulse 124, temp. 104·7°.
13*th day*.—Several fresh spots; tongue dry in centre; one pretty good stool; pulse 120, temp. 104·6°: *vesp.* pulse 124, temp. 105·2°.
14*th day*.—Pulse 120, temp. 102·1°: *vesp.* pulse 132, temp. 105°.
15*th day*.—Is wandering, and talking nonsense; tongue dryish; two loose stools; several fresh spots on abdomen; has tenderness on pressure in right iliac region; pulse 120, temp. 102·2°: *vesp.* pulse 120, temp. 105·3°.
16*th day*.—Two copious ochrey stools; pulse 124, temp. 102·8°: *vesp.* pulse 120, temp. 103·8°.
17*th day*.—Eruption fading; tongue dry in centre; still wanders a good deal, but answers rationally enough any questions that are put to her; pulse 124, temp. 102·8°: *vesp.* pulse 120, temp. 103·3°.
18*th day*.—One stool; pulse 124, temp. 103·2°: *vesp.* pulse 124, temp. 104·2°.
19*th day*.—One ochrey stool; pulse 132, temp. 103·2°: *vesp.* pulse 132, temp. 103·2°.
20*th day*.—Two loose stools; still talking nonsense, but is quite docile and manageable; pulse 120, temp. 102·8°: *vesp.* pulse 128, temp. 103°.
21*st day*.—Two scanty stools; tongue moist; pulse 132, temp. 103°: *vesp.* pulse 132, temp. 104·3°.
22*d day*.—One stool; tongue dirty; pulse 120, temp. 102·2°: *vesp.* pulse 132, temp. 103·2°.
23*d day*.—As last noted; pulse 120, temp. 101·8°: *vesp.* 124, temp. 103·6°.
24*th day*.—Has improved expression; tongue cleaner; right parotid is swollen and tender; pulse 124, temp. 100·6°: *vesp.* pulse 124, temp. 103·3°.
25*th day*.—Sleeps a good deal; pulse 108, temp. 99·7°: *vesp.* pulse 120, temp. 103·4°.
26*th day*.—Pulse 132, temp. 100°: *vesp.* pulse 124, temp. 103·6°.
27*th day*.—Parotid considerably swollen; pulse 120, temp. 102°: *vesp.* pulse 120, temp. 103·2°.
28*th day*.—No stool for two days; pulse 132, temp. 101·1°; to have one teaspoonful of castor-oil: *vesp.* pulse 132, temp. 102·8°.
29*th day*.—Had two stools after oil; pulse 132, temp. 103·2°: *vesp.* pulse 124, temp. 103°.
30*th day*.—Passed a restless night; parotid much swollen, very hard, and tender; bowels moved twice; pulse 120, temp. 102·4°; *vesp.* pulse 144, temp. 103°.

31*st day*.—Pulse 124, temp. 102·6: *vesp.* pulse 132, temp. 102·7°.
32*d day*.—One stool; pus discharging from right ear; pulse 124, temp. 100°: *vesp.* pulse 120, temp. 101·4°.
33*d day*.—Pulse 132, temp. 102·4°: *vesp.* pulse 112, temp. 99°.
34*th day*.—Discharge continues from ear; gland less tense and swollen; pulse 108, temp. 98·7°: *vesp.* pulse 128, temp. 100·6°.
For the next twelve days improvement continued; the pulse varied from 100 to 120, and the temperature from 98° to 100° up to the 43d day, on which the normal range was persistent: it continued so till the 47th.
47*th day*.—Tongue clean and moist; pulse 120, temp. 98·4°: *vesp.* pulse 136, temp. 102·6°.
48*th day*.—Tongue slightly furred; has had no stool for four days; pulse 120, temp. 101·2°: *vesp.* pulse 140, temp. 104·4°; to have a teaspoonful of castor-oil.
49*th day*.—Bowels freely moved; complains much of thirst; chest sounds normal; pulse 136, temp. 103·2°: *vesp.* pulse 136, temp. 104·1°.
50*th day*.—No stool since last note; tongue is more furred; has been vomiting; pulse 120, temp. 102·2°: *vesp.* pulse 140, temp. 103·5°.
51*st day*.—Sickness continues; pulse 132, temp. 103°: *vesp.* pulse 120, temp. 102·8°.
52*d day*.—One good stool; pulse 132, temp. 103°: *vesp.* pulse 132, temp. 104·2°.
53*d day*.—Pulse 120, temp. 103°: *vesp.* pulse 128, temp. 103·6°.
54*th day*.—Vomiting continues; has had no stool for two days; pulse 120, temp. 102·6°; to have warm-water enema; *vesp.* bowels well moved; pulse 124, temp. 103·2°.
55*th day*.—Vomits less; pulse 120, temp. 102·4°: *vesp.* pulse 120, temp. 103·6°.
56*th day*.—Vomiting has ceased; pulse 120, temp. 102°: *vesp.* pulse 128, temp. 102·6°.
57*th day*.—Pulse 120, temp. 102·9°: *vesp.* pulse 124, temp. 103·1°.
58*th day*.—Has two rose-coloured spots on abdomen; no stool for two days; pulse 112, temp. 101·6°; repeat enema: *vesp.* pulse 112, temp. 103°.
59*th day*.—Pulse 108, temp. 101·6: *vesp.* pulse 120, temp. 103°.
60*th day*.—Pulse 116, temp. 103°: *vesp.* pulse 112, temp. 103°.
61*st day*.—Pulse 116, temp. 102·8°: *vesp.* pulse 116, temp. 102·4°.
62*d day*.—No stool for three days; pulse 108, temp. 102·8°; repeat enema: *vesp.* pulse 120, temp. 102·8°.
63*d day*.—Pulse 108, temp. 103°: *vesp.* pulse 120, temp. 103·8°.
64*th day*.—Bowels moved; pulse 100, temp. 99·9°: *vesp.* pulse 116, temp. 102·6°.
65*th day*.—Tongue clean; pulse 100, temp. 100·2°: *vesp.* pulse 112, temp. 102·6°.
66*th day*.—Pulse 96, temp. 100·2°: *vesp.* pulse 108, temp. 102°.
67*th day*.—Pulse 100, temp. 100·4°: *vesp.* pulse 104, temp. 102·5°.
68*th day*.—Pulse 92, temp. 99·8°: *vesp.* pulse 100, temp. 101·8°.
69*th day*.—Continues to improve; pulse 104, temp. 99·8°: *vesp.* pulse 112, temp. 102°.
70*th day*.—Pulse 108, temp. 99·2°: *vesp.* pulse 120, temp. 102·1°.
71*st day*.—Pulse 100, temp. 98·4°: *vesp.* pulse 104, temp. 100·2°.
72*d day*.—Pulse 96, temp. 98°: *vesp.* pulse 104, temp. 99·7°.
Continued to convalesce satisfactorily. The pulse remained high, never below 92, for ten days after the temperature in the evening had fallen to the normal standard.

III. A male, aged 15, came under observation on the 5th day.
6*th day*.—Skin hot; tongue dry in centre; complains much of pain in head; no eruption; sclerotics clear, pupils of good size; pulse 108, temp. 105°: *vesp.* pulse 120, temp. 104·6°.
7*th day*.—Passed a restless night, constantly trying to get out of bed; tongue

moist; no eruption; one stool of fair consistence; pulse 120, temp. 104°. *vesp.* pulse 108, temp. 103°.

8th day.—Had a bad night, wanders much, is heavy and listless, takes no notice of what goes on around; passes water in bed; no stool; no eruption; respirations irregular; pulse 108, temp. 103°: *vesp.* pulse 108, temp. 102·5°.

9th day.—Had a bad night; one loose stool; no eruption; sclerotics clear; pulse 132, dicrotous, temp. 104°; *vesp.* pulse 128, temp. 103°.

10th day.—Passed a very restless night, constantly trying to get out of bed, takes no notice of anything; eyes clear, pupils of natural size; no eruption; one rather loose stool; pulse 132, temp. 103°: *vesp.* pulse 132, temp. 104·3°.

11th day.—Had a better night; no stool; muscles somewhat rigid, resisting any attempt to bend the arms or legs; urine distinctly albuminous, deposits urates on standing, chlorides very deficient; no eruption; pulse 144, temp. 104·7°: *vesp.* pulse 140, temp. 104·3°.

12th day.—Seems more intelligent; answers questions, but not with facility; tongue moist and dirty; still passes water in bed; no stool; pulse 144, temp. 104°: *vesp.* pulse 150, temp. 104·5°.

13th day.—Has muscular tremors; tongue dry; pupils of good size; sclerotics clear and pearly; cardiac systole soft, but quite audible; impulse fair; one loose stool; pulse 136, temp. 103°; *vesp.* pulse 140, temp. 104·1°.

14th day.—Is much depressed, and less conscious; pupils dilated and sluggish; pulse 144, temp. 104°; heart's sounds fainter; to have a teaspoonful of whisky in water every quarter of an hour: *vesp.* pulse 132, temp. 101·7°; double the amount of whisky.

15th day.—Quite unconscious of what is said; pupils large, eyes clear; has had no stool for two days; breathing hurried and irregular; pulse 144, temp. 103·3°: *vesp.* pulse 164, temp. 103·1°; moribund.

Died during the night. Post-mortem examination not obtained.

IV. A female, aged 19, came under observation on the 4th day of illness.

4th day.—Tongue dry; skin hot; no eruption; patient wanders greatly, and takes no notice of what is going on around; eyes clear, pupils of good size; has passed one stool of fair consistence in bed; pulse 136, temp. 102·5°: *vesp.* pulse 120, temp. 101·8°.

5th day.—Passed a quiet but sleepless night; passes water in bed; notices nothing, and does not seem to understand what is said to her; pulse 116, temp. 101°: *vesp.* pulse 120, temp. 102·5°, resp. irregular.

6th day.—Passed a restless night; pupils large, sclerotics pearly; no stool; pulse feeble; cardiac systole soft, but distinct; pulse 132, temp. 104·1°; to have six ounces of wine: *vesp.* pulse 132, temp. 103·6°; to have tinc. opii. gt. xii.

7th day.—Slept well; is more sensible; protrudes her tongue when told to do so; tongue dirty and gummy; has made no water; has had one stool, for which she requested the pan; no eruption; pulse 120, temp. 102·2°: *vesp.* pulse 128, temp. 104·2°, resp. 40.

8th day.—Restless night; asks for vessel to make water in; eyes clear, pupils large, urine albuminous, sp. gr. 1026, chlorides slightly deficient; pulse 128, temp. 103·2°: *vesp.* pulse 136, temp. 104·9°.

9th day.—As last noted; no eruption; pulse 128, temp. 104·1°: *vesp.* pulse 136, temp. 105°, resp. 40, cerebral in character.

10th day.—Passed a better night; bowels rather loose; stools dark; pulse 144, temp. 103·8°: *vesp.* pulse 140, temp. 104·6°.

11th day.—Restless night; two dark stools passed in bed; no eruption; sclerotics clear, pupils of good size; cardiac systole and impulse less distinct, but still quite perceptible; tongue dry in centre; urine abundantly albuminous, sp. gr. 1030; pulse 144, temp. 104°; to have 12 ounces of wine per diem: *vesp.* pulse. 148, temp. 105·1°, resp. 44.

12th day.—Is much sunk; heart's sounds much impaired; pulse very weak;

two loose stools passed in bed; pulse 144, temp. 102·6°; sudaminal eruption over chest and abdomen; to have 6 ounces of whisky besides her wine: *vesp.* pulse 164, temp. 105·7°.

13th day.—Greatly depressed; pulse 140, scarcely perceptible, temp. 100°, resp. 30; 2 P.M., pulse 132, temp. 98°; continue stimulants: *vesp.* pulse 156, temp. 103°, resp. 52; swallows very badly; to have half an ounce of whisky with half a pint of beef tea as an injection every hour.

14th day.—Is greatly depressed; skin moist; no eruption; eyes clear, pupils large; can swallow; to have half an ounce of spirits every hour; pulse 148, resp. 50, temp. 101°; *vesp.* pulse 150, resp. 44, temp. 101·2°.

15th day.—Is much sunk; takes no notice of anything; breathing laboured and irregular; pulse 160, temp. 103·8°: *vesp.* pulse 164, temp. 104·2°.

16th day.—Moribund; pulse imperceptible; temp. 103·2°. Died at 1 P.M. comatose.

Post-mortem examination revealed extensive disease of Peyer's patches, and a considerably increased quantity of fluid in the brain.

V. A male, aged 21, was admitted into hospital on the 6th day of an illness which had commenced with general malaise, loss of appetite, and wandering pains in limbs. On admission, he complained chiefly of weakness and loss of appetite; the skin was warm, the tongue slightly furred, bowels regular, pulse 84, temp. (in evening) 103·8°. Throughout the case the general symptoms were so very slight that a daily record of them would be superfluous, suffice it to say that the expression was languid, the eyes clear, the pupils natural; the tongue was never more than very thinly coated, and was generally clean; the bowels were regular, and the stools light coloured, but never liquid; there was never the slightest tendency to delirium or other head symptoms; there was no eruption; there was nothing in fact beyond the patient's feelings, and the range of temperature, as shown below, to guide one in the formation of a diagnosis.

7th day.—Pulse 84, resp. 24, tranquil, temp. 99·7°: *vesp.* pulse 96, temp. 103°.
8th day.—Pulse 84, resp. 28, temp. 101·2°: *vesp.* pulse 96, temp. 101·2°.
9th day.—Pulse 72, temp. 100·2: *vesp.* pulse 72, temp. 101·7°.
10th day.—Pulse 60, resp. 24, temp. 99·4°: *vesp.* pulse 72, temp. 100·3°.
11th day.—Pulse 60, temp. 98°: *vesp.* pulse 72, temp. 100·8°.
12th day.—Pulse 68, resp. 24, temp. 98°: *vesp.* 72, temp. 102·4°.
13th day.—Pulse 60, temp. 97·7°: *vesp.* pulse 80, temp. 101°.
14th day.—Pulse 72, temp. 97·1°: *vesp.* pulse 72, temp. 99·8°.
15th day.—Pulse 60, resp. 20, temp. 98·2°: *vesp.* pulse 68, temp. 100·8°.
16th day.—Pulse 68, temp. 98·2°: *vesp.* pulse 72, temp. 98°.

VI. A female, aged 30, came under observation on the 11th day of an illness which had commenced very much like the last case; as in it also the general symptoms were so slight as scarcely to call for notice, and gave very little aid in the formation of a diagnosis.

11th day.—Complains of general debility and anorexia; has languid and depressed aspect; eyes clear, pupils natural, tongue pretty clean; bowels rather loose; pulse 104, resp. 20, tranquil, temp. 100·5°: *vesp.* pulse 100, temp. 103·2°.
12th day.—Had a good night; one ochrey stool; no eruption; no abdominal tenderness; pulse 92, resp. 24, temp. 99·8°: *vesp.* pulse 100, temp. 102·9°.
13th day.—No stool; tongue dry; skin perspiring; no eruption; pulse 100, temp. 101°: *vesp.* pulse 96, temp. 102°.
14th day.—Pulse 92, resp. 24, temp. 98·4°: *vesp.* pulse 96, temp. 101·6°.
15th day.—Tongue moist and pretty clean; skin moist; pulse 92, temp. 99·2°: *vesp.* pulse 92, temp. 101·3°.
16th day.—Bowels regular; pulse 92, resp. 22, temp. 98·6°: *vesp.* pulse 92, temp. 101·1°.
17th day.—Eruption of sudamina on abdomen; tongue pretty clean; pulse 88, temp. 98°: *vesp.* pulse 84, temp. 100·5°.

18*th day*.—Tongue clean; bowels regular; pulse 84, temp. 98·6: *vesp.* pulse 84, temp. 101°.
19*th day*.—Pulse 84, temp. 98°: *vesp.* pulse 84, temp. 99·5°.
20*th day*.—Pulse 84, temp. 98°: *vesp.* pulse 76, temp. 99·8°.
21*st day*.—Pulse 84, temp. 98·3°: *vesp.* pulse 88, temp. 101·8°.
22*d day*.—Pulse 84, temp. 98·4°: *vesp.* pulse 96, temp. 100·2°.
23*d day*.—Pulse 80, temp. 97·8°: *vesp.* pulse 88, temp. 97·6°.

VII. A female, aged 16, came under observation on the 9th day of an illness which had commenced with shivering and general malaise.
9*th day*.—Has several rose-coloured spots on the abdomen; tongue dry; has had one ochrey stool; pulse 96, temp. 100·9°: *vesp.* pulse 108, temp. 101·4°.
10*th day*.—Had a quiet night; one ochrey stool; no abdominal tenderness; pulse 108, temp. 100·8°: *vesp.* pulse 108, temp. 101·6°.
11*th day*.—Tongue moist at edges; has several fresh lenticular spots, some of the old ones gone; pulse 96, temp. 101°: *vesp.* pulse 120, temp. 102·6°.
12*th day*.—Pulse 96, temp. 101·4°: *vesp.* pulse 96, temp. 101·6°.
13*th day*.—Passed a good night; had rigors this morning; tongue dry in centre; no stool for three days; pulse 128, temp. 103·2°; to have warm-water enema: *vesp.* pulse 120, temp. 101·8°; no fresh spots; the old ones not so numerous.
14*th day*.—Enema did not act well; to have two teaspoonfuls of castor-oil; pulse 108, temp. 101·3°: *vesp.* pulse 108, temp. 100·5°; two ochrey stools after oil.
15*th day*.—Tongue moist and cleaner; pulse 92, temp. 98·8°: *vesp.* pulse 124, temp. 103°.
16*th day*.—Eruption gone; pulse 104, temp. 98·6°: *vesp.* pulse 116, temp. 102·4°.
17*th day*.—Pulse 96, temp. 98·2°: *vesp.* pulse 120, temp. 103·2°.
18*th day*.—Is doing well; bowels confined; pulse 84, temp. 97·4°; to have two teaspoonfuls of castor-oil: *vesp.* bowels moved; pulse 120, temp. 102·6°.
19*th day*.—Pulse 96, temp. 97·8°: *vesp.* pulse 108, temp. 100°.
20*th day*.—Pulse 90, temp. 97·5°: *vesp.* pulse 96, temp. 98·4°.
From this time convalescence was satisfactory.

Cases I. and II. serve well to illustrate the normal range of temperature in typical cases of enteric fever tending to recovery. The first was mild, but well marked; its most striking feature was the morning fall and the evening rise of the temperature. The average morning range up to the commencement of defervescence was 100·9°; that for the evening, 103·2°—a difference of 2·3°. At the close of the 2d week, this difference was more marked, being on the 13th day, 4°; on the 14th, 4·9°; and on the 15th, 3·7°. Defervescence commenced on the 22d day, on the morning of which the temperature was 97·8°. During the first four days of defervescence, the mean temperature was, in morning, 97·9°, and in evening, 102·4°—a difference of 4·5°. It will be observed also that on the 16th, 17th, and 18th days, when the patient had sore-throat, the temperature attained its maximum, and that the high range at that time was due partly to an increased rise in the evening, but chiefly to an absence of the morning fall; the morning range during these days being 2·1°, and the evening, 1·1°, above the average.

The second case was also well marked, but not so mild as the former. There was in it a good deal of quiet delirium, whilst in

the first case there was never any head-affection. Altogether, in this instance, the general symptoms were more decided and prominent, and the case was more calculated to cause uneasiness as to the result. The temperature did not show that tendency to an evening rise and a morning fall which was so marked in the other. The morning average before defervescence was 103·3°, the evening, 104·2°—a difference of only 9-10ths of a degree. It will be observed, however, that there were two days at the close of the 2d week (14th and 15th), on which the difference between the morning and evening range was very marked—3 degrees. It will also be observed that this difference was due partly to a greater rise in the evening, but mainly to an exaggerated morning fall; and that subsequently to this break in the previously equable, though high range, the temperature was persistently lower than it was before it; the difference between the morning and evening range, as shown by the general average, is in fact due almost entirely to this break on the 14th and 15th days:—

	In Morning.	In Evening.	Difference.
Before the 14th day, the mean temperature was,	104·4°	104·6°	·2°
On the 14th and 15th days, " "	102·1	105·1	3·
From the 15th to commencement of defervescence, " "	102·9	103·6	·7

During the 3d week, the average morning temperature was 1·5°, and the average evening temperature 1° lower than it was during the 2d. The characteristic nocturnal rise and morning fall of the early stage of defervescence began to show itself on the 22d day; was more marked on the four following days, but was interrupted by the occurrence of parotid swelling, which sent the temperature up, and kept it high for a week. This rise, however, was chiefly due to the absence of the morning fall; the average temperature of the five days during which the parotid swelling was at its height, was in the morning, 102·2°, and in the evening, 102·9°; that of the three preceding days was in the morning, 100·1°, and in the evening, 103·4°. An additional point of interest in this case is to be found in the fact that the first indication of the occurrence of a relapse, was the simultaneous rise of the pulse and temperature on the 47th day. In consequence, however, of the cardiac action having been persistently frequent (from 100 to 120) during the twelve days of convalescence (on which the temperature was seldom much above the normal standard), and in consequence of the rate of the pulse having shown at times considerable, and apparently causeless rises, its increased frequency at this period was not considered of so much value as the elevation of the temperature in prognosticating what was about to happen. The characteristic range of the period of defervescence began on the 64th day of illness, and the 18th of relapse.

In neither of these two cases was there any necessity for the aid of the thermometer in forming a diagnosis. They were unmistak-

able cases of enteric fever. Before proceeding to give the inferences which may be drawn from the facts to be found in them, and in other similar cases, it may be well, by way of contrast, to examine in detail the other cases which have been instanced, and note the relation existing between the severity of the symptoms and the range of the temperature.

Case III. had a high range at an early stage (105·1° on 6th morning), but showed no tendency to a morning fall; the chief feature in its thermometry was the irregularity of the range; the morning temperature was as often above as below that of the evening—the average for the former was 103·8°, for the latter 103·7°. The pulse was very frequent after the 1st week. On the 5th and 6th days, the temperature was comparatively much higher than the pulse; on the 7th and 8th, the two indicated almost the same degree of febrile disturbance; but after the 8th day, the pulse was, as compared with the normal standard, much higher than the temperature. The respirations were frequent but irregular. The general aspect and restless condition of the patient on admission led to the belief that the case would be severe; the high temperature (105° on 6th morning) strengthened this impression. On the 7th and 8th days, the nervous symptoms became so marked as to lead, at that early stage, to the formation of an unfavourable prognosis, notwithstanding a fall in the temperature: as the case advanced, the head-symptoms became more marked, the urine gave evidence of the presence of albumen, the pulse increased in frequency, and the irregularities in the range of the temperature became more marked; the abdominal symptoms were never such as to attract attention. The patient died comatose on the 15th night.

Case IV. was the sister of the girl whose case was given first, and with which it therefore contrasts well. On the first day on which she came under observation (the 4th) the cerebral symptoms were so very marked for that stage of the illness that the most gloomy prognosis was formed, and though under treatment an occasional slight improvement was noticed during the course of the case, it was only temporary; albumen appeared in the urine, and continued to increase with the increasing urgency of the general symptoms; the pulse was frequent throughout, during the last four or five days particularly so; towards the end it became very feeble, and the cardiac action was such as to call for the free administration of stimulants; the respirations were frequent and irregular; the temperature did not show the same tendency to irregularity in the range which was noted in No. III., but evidenced some of the inclination to a nocturnal exacerbation and matutinal remission which has been noticed as characterizing the normal range of mild cases of enteric fever; the average temperature up to the 13th day was in morning 103·7°, and in evening 104·9°, a difference of 2·2°; during the 1st week the range was by no means high, much below that of the pulse; during the 2d week it got up and continued to

rise till it reached its maximum (105·7°)[1] on the 12th evening; on the 13th day the patient was greatly depressed, and the temperature fell to 100° in the morning, and to 98° in the afternoon; the pulse at the same time was 132; the patient was in imminent danger, and was prevented from completely sinking by the administration every hour of half a pint of beef-tea and half an ounce of whisky; she never recovered consciousness, and died comatose on the 16th day.

Each of these cases bore so strong a resemblance to typhus that a cursory examination of the patient would most likely have led to a faulty diagnosis, especially as typhus was epidemic at the time of their occurrence. The sudden onset of the illness, the prominence of the cerebral, and the absence of abdominal symptoms were more like the general characteristics of typhus than of enteric. On the other hand, the absence of eruption, along with such marked head-affection, tended rather to oppose the view that we had to deal with typhus. In neither case was the evidence afforded by the thermometer of any use in determining the particular form of fever which we had to treat. *The main point in a diagnostic point of view, the feature which led me in both instances unhesitatingly to say that both patients were suffering, not from typhus, but from enteric fever, was to be found in the condition of the eyes;* the conjunctivæ were not injected, the sclerotics were clear and pearly, and the pupils were large. Cases of typhus, with such marked cerebral symptoms, would have had the conjunctivæ injected, the eyes suffused, and the pupils small. In all cases with marked head-symptoms in which it has seemed doubtful from which form of fever the patient was suffering, I have been in the habit of placing great reliance on this condition of the eyes as a diagnostic sign, and have never been misled by it. In the case of No. IV. the accuracy of the diagnosis was substantiated during life by the admission into hospital of the patient's sister, suffering from a well-marked attack of enteric fever, and was fully confirmed after death by the condition of the glands of the small intestine as revealed by dissection. It may be said that the morning fall and evening rise, which we have seen formed one of the features of the thermometry of this case, were of some value as a diagnostic sign; it will be observed, however, that this character did not show itself till the 7th day, by which time a diagnosis had been formed; and even when it did appear, it was not more marked than I have seen it in many cases of typhus;[2] it therefore gave no assistance in the formation of a diagnosis.

[1] This is the highest point which I ever found the temperature reach in enteric fever.

[2] In Case II., with marked head-symptoms given in a paper on the Thermometry of Typhus in the Edinburgh Medical Journal for January 1868, this tendency to a morning fall is seen in a sufficiently marked degree, though I have seen many in which it was even more marked.

In both of these cases (III. and IV.) the great risk of error was in the diagnosis—they were very apt to be mistaken for typhus; in neither did the thermometer cast any light at all calculated to help us out of the difficulty; for purposes of direct diagnosis it was valueless; it only corroborated the evidence of the pulse that a general febrile state existed; and did not at all either confirm or contradict that given by the general state of the patient (and most markedly by the state of the eyes), that enteric fever was the disease with which we had to deal.

In the formation of a prognosis it was not altogether valueless, though here also it contrasts very unfavourably with the other symptoms.

In Case III. the high range of the temperature on the 6th day led to the expectation of severe head-symptoms, which on the same night began to show themselves. After that day the range was not so high as to induce one, from a consideration of it alone, to give an unfavourable prognosis—irregularity (in itself almost as bad a sign as unusual height) was its chief feature, but the case had to be observed for some days before this could be recognised; in the meantime, the pulse got up, and the other symptoms became so prominent that the irregularity could do no more than show that the temperature differed from that of a typical case tending to recovery, just as the general symptoms differed from those usual in such a case; but as a sufficiently certain prognosis was formed before this feature in the thermometry of the case could be detected, it follows that the temperature was only of secondary importance in the formation of a correct opinion as to the probable result.

In Case IV. the patient was very early struck down by the severity of the attack, and from the 4th day (that on which she first came under notice) there was no doubt that the case was likely to have an unfavourable termination, and that the cerebral affection was the chief and most urgent source of danger. A perfectly conclusive prognosis was at this time formed from a consideration of the general symptoms alone; the rapidity of the pulse tended to corroborate the evidence so obtained; the temperature on the 4th and 5th mornings gave no corroborative testimony. On the 6th morning it was high (104·1°), and on the following day began to show a tendency to a morning fall and evening rise, which characterized the remaining course of the case; at the same time there was observed from day to day an increase in the height of the range, which tended to substantiate the evidence given by the greatly increased rapidity of the pulse and the growing urgency of the general symptoms, but which could not for a moment be contrasted with these as a prognostic sign. On the 13th day the temperature had fallen to the normal standard, and did not again reach the high range which it had attained previous to that day; this fall, if attended to alone, would have greatly misled—it was a mere accompaniment of most alarming prostration.

Cases V. and VI. are given in illustration of those in which the general symptoms were so slight as to render it difficult, if not impossible, to form a satisfactory diagnosis from them alone. In V. there was a languor of expression, a loss of appetite, and a condition of general malaise, which showed that the patient had something the matter with him; the pulse was slightly increased in frequency, but never above 96; the respirations were tranquil, regular, and very little accelerated, only once above 24: besides these indications, there was nothing to guide one, except the patient's statement of the manner in which his illness commenced, and of his feelings on admission. Of all these symptoms the expression was that which most tended to lead to the *suspicion* that the case was one of enteric fever; but a *diagnosis* could not have been formed without the aid of the thermometer. The pulse, it will be seen, reached the normal standard on the 9th day, after which it was generally lower than natural, and only once as high as 80. The temperature on admission (6th evening) was 103·8°; for the next four days the average range was in morning 100·1°, and in evening 101·6°; on the 11th day (two days after the pulse had reached the normal standard), there commenced those morning falls and evening rises of the temperature which we have seen to be characteristic of the defervescence of enteric fever, on which was founded the diagnosis in this case, and without a knowledge of which it would have been impossible to say what the real nature of the illness was. The mean morning range for the 11th and four succeeding days was 97·8°, the mean evening range 100·8°—a difference of 3 degrees. During the actual existence of the febrile state no definite opinion as to the nature of the malady was possible; it was only when the characteristic thermometric range of commencing convalescence showed itself, that the doubts were dissipated and a satisfactory diagnosis formed.

In Case VI. the general symptoms were very much the same as those noted in V.; there was no head-affection, nor were the abdominal signs such as to give a distinct clue to the nature of the case; the bowels were indeed said to be loose on admission, and on the 12th day there was one ochrey stool, but after that time there was no tendency to diarrhœa. As in the former case, the languid expression and the clear pearly sclerotic pointed to enteric fever; the dry tongue on the 13th day was also of some value. The pulse ranged from 92 to 108, and on the 17th day fell to 84, which seemed to be its normal standard. The respirations were tranquil, and varied from 20 to 24 per minute. The temperature had on the 11th, 12th, and 13th days a mean range in the morning of 100·4°, and in the evening of 102·7°; on the 14th day began the characteristic morning fall and evening rise of commencing defervescence; the average morning temperature for the 14th and four succeeding days was 98·5°, that for the evening 101·1°—a difference of 2·6 degrees. In this, as in the last case, there would

have been no certainty in the diagnosis had the range of the temperature not been noted; without the thermometer the nature of the case might have been suspected, but not diagnosed. It may, indeed, be argued that these two cases were not enteric fever at all,—that, with the absence of all the usual symptoms of the disease (delirium, diarrhœa, eruption, etc.), and with a convalescence commencing in one on the 11th, and in the other on the 14th day, the peculiar range of the temperature which has been noted is not sufficient to establish a diagnosis. I think it will be granted, however, that if, even with the absence of other marked signs and with so early a defervescence, there had been an eruption of rose-coloured lenticular spots coming out in successive crops, the diagnosis would have been established independently of the thermometer. Such a case I have instanced in No. VII.: in it there were neither head nor abdominal symptoms; the bowels were indeed so confined as to call for the administration of laxatives; the general aspect of the patient differed in no respect from that of Cases V. and VI.; but there was, on admission into hospital on the 9th day, an eruption of rose-coloured spots, disappearing on pressure; on the 11th day some of these had faded, and several fresh ones had come out; on the 13th day they had begun to disappear, and by the 16th they were all gone. During the continuance of the fever the pulse ranged from 96 to 128; with commencing defervescence it showed a tendency to fall in the morning and rise in the evening. Up to the 15th day the average temperature was in morning 101·4°, and in evening 101·5°; on that day began the usual morning fall and evening rise; the mean range for that and the four succeeding days was in morning 98·1°, and in evening 102·2°—a difference of 4·1 degrees. In this case the symptoms generally supposed to accompany enteric fever were, with the exception of the eruption, no more marked than in V. and VI.; the pulse indicated only a greater degree of febrile disturbance; the temperature on the 15th day began to show the characteristic morning fall and evening rise, which, had the eruption not appeared, would in this instance also have been the only certain guide to a satisfactory diagnosis. These are by no means isolated or exceptional examples; they represent a very numerous class of mild cases of enteric fever, in which the general symptoms and local lesions are seldom such as to attract particular attention, and never such as to cause anxiety as to the result, in which defervescence sets in generally about the end of the 2d week,—maybe even earlier,—and whose real nature can seldom be satisfactorily determined without the aid of the thermometer, and rarely even by this means until defervescence is two or three days advanced. To this class belong, I believe, the majority of the cases of nondescript forms of fever which occur in this country, and which are generally classed under the head of "common continued fever;" this title I have hitherto always applied to them myself. The thermometer, however, has led me

to regard them as only mild cases of enteric. Of course a means of diagnosis which only comes into play when the patient is getting better, can be of no practical utility so far as prognosis and treatment are concerned; and, fortunately, there is in these cases scarcely any chance of forming other than a most favourable prognosis, and no call for active treatment of any kind: our very ignorance of their real nature, by preventing us from conjuring up imaginary risks, and adopting a possibly injurious line of treatment, acts as a safeguard to the patient. Nevertheless, it is well that the exact nature of such cases should be known, as there are certain hygienic precautions which ought to be adopted in every case of enteric fever. I have constantly been in the habit, since I adopted the above view of their nature, of disinfecting the stools of every case of this "common continued fever" just as if I had to deal with a well-marked instance of enteric; if an error, it is one on the safe side.

Cases of enteric fever are so seldom admitted into hospital until they have been at least five or six days ill, that I have not had the opportunity to make, during the first week, a number of observations sufficient to form a foundation for any definite opinion regarding the utility of the thermometer at that stage of the disease. The remarks which I have to make will be understood as applying chiefly to the 2d and 3d weeks.

The facts which have been given are sufficient to show that the range of the temperature varies much in different cases: I know, indeed, of no disease whose thermometry shows so many variations, and so much diversity of range. I took all the well-marked cases which recovered, and in which the temperature had been noted, and tried, by striking the average for each day, to manufacture a case whose thermometry might be taken as a type of the whole; but the result bore so little resemblance to any of the cases which formed its basis that it was at once discarded. The explanation of this is not difficult to find. The morning fall and evening rise, which form the most notable feature in the thermometry of enteric fever, vary much both in degree and duration: in one instance (as in Case I.) this feature may characterize the whole course of the case; in a second, it may exist only for a few days, and at any period prior to defervescence; whilst in a third, it may never be such as to attract attention until defervescence commences. Although by no means constant, this character in the range is so much more frequent at the end of the 2d week than at any other time, that I am inclined to attach some importance to its occurrence at that period in certain cases. When it occurs throughout the whole course of a case, it is apt to be most marked then; when found only for a day or two, it will probably be about the 14th day; it is not, however, sufficiently constant or sufficiently early to be of use in diagnosis; its use in prognosis will be explained under that head. The only time at which this

marked difference between the morning and evening range is constant is during defervescence; then it is invariable and often most exaggerated. The most marked instance which ever came under my notice was one in which the average of the difference between the morning and evening range on the 23d, 24th, and 25th days was 6·9°; on the 24th day the morning temperature was 96·4°, the evening 103·8°,—a difference of 7·4 degrees. This mode of defervescence is characteristic of enteric fever, and in cases which have presented only equivocal symptoms becomes of great value in diagnosis, as in cases V. and VI. The pulse often shows a tendency to the same character, but rarely, if ever, in so marked a degree.

The occurrence of complications has an effect on the temperature which varies according to the nature of the secondary affection and the case in which it occurs. In cases having throughout a marked morning fall, the occurrence of an inflammatory complication reduces the extent of that fall more than it raises the evening range, as in case I., in which the occurrence of sore-throat at the beginning of the 3d week had a marked effect in that manner, and case II. in which the characteristic range of defervescence was interrupted by the onset of parotid swelling.

Pulmonary complications may have at first an elevating tendency, but when so fully developed as to interfere with the proper oxygenation of the blood, have an opposite effect.

Diarrhœa and hæmorrhages when slight have no effect at all, but when so severe as to be a source of immediate danger have a decidedly lowering tendency. Some observers regard a sudden and great fall of the temperature as a sign of intestinal hæmorrhage, by which the occurrence of that accident can be diagnosed before any blood is passed by stool; this I have never had the opportunity to verify, but have no doubt of its accuracy; a sudden and severe attack of intestinal hæmorrhage must have the same effect on the system, and on the temperature, as any other prostrating agency (severe epistaxis, for instance, which I have seen produce this effect), and if the blood be retained in the intestine for some time, a low temperature and other signs of prostration will be sufficient to enable us to diagnose what has happened before the actual appearance of that fluid in the stools; in such a case, however, the evidence of the thermometer would be merely corroborative (though still extremely valuable), as the palor, prostration, and other signs of sudden depression would tell their own tale before the thermometer could be used. Let us see how the facts given above bear on diagnosis, prognosis, and treatment.

Diagnosis.—There are two classes of cases in which difficulties in diagnosis are apt to be experienced: (*a*) those in which the head-symptoms are so prominent and so early as to cause the case to resemble typhus; and (*b*) those in which the symptoms are so mild and equivocal, and of such short duration, that a diagnosis is impossible during their continuance, and uncertain after their cessation. In the former, the range of the temperature is not sufficiently characteristic to be of use in the discrimination of the two diseases (see cases III. and IV. and the remarks thereon); in the latter, it gives little or no aid during the continuance of the febrile state, but with commencing defervescence supplies the only certain information which can be got regarding the true nature of the case (see cases V. and VI. with the remarks on them).

Prognosis.—This should always be guarded in enteric fever. In cases in which the head-symptoms are the source of danger, and in which death is threatened by coma, the thermometer is less valuable than in typhus, and is indeed less necessary; a case of enteric which proves fatal by coma generally does so by the end of the 2d or beginning of the 3d week, and has the cerebral symptoms marked and prominent from an early stage; if these during the first week are so bad as to cause the case to resemble one of typhus, the attack is a very severe one, and no further evidence is required to enable us to form a most unfavourable prognosis; though a high and irregular range of the temperature is generally found in such cases, it is not so constant as in typhus, and does not always precede the occurrence of the head-affection; it therefore does not always enable us to foretell the probable result at an early stage, and is chiefly of value in so far as it corroborates the more certain evidence of the general symptoms.

In a case which is imperilled by the severity of the intestinal affection, and in which death is threatened by asthenia, the amount of the danger is most accurately gauged by the extent of the abdominal mischief, as indicated by the presence of diarrhœa, hæmorrhage, tenderness, etc.; but as these symptoms may only be urgent for a few days before the fatal termination, and as this seldom takes place till the 3d or 4th week, it would be well if there were some other and earlier means of recognising and estimating the danger. Unfortunately there is no certain mode of doing so; nevertheless, if cases are carefully watched during the 2d week, we can in some instances get a clue to guide us in prognosticating their probable course during the 3d. The extent to which head-symptoms prevail (without being in themselves dangerous) is a tolerable index of the severity of the attack, and of the risk during the 3d week. A high and irregular range of the temperature during the 2d week often precedes bad symptoms during the 3d. If in a case presenting some features of anxiety we find the temperature during the 2d week persistently high, and showing no tendency to a morning fall, we get thereby corroborative testimony of the severity of the attack; but if, at the end of the 2d week, we find the morning temperature fall one or two degrees, we may (even though the evening range is high) give a hopeful prognosis, and should the temperature, after this morning fall has ceased (*i.e.*, during the 3d week), be persistently lower than it was before its

occurrence, a happy result may be expected,—Case II. is a good example of this. It must be confessed, however, that there is always a certain amount of hazard in giving a favourable prognosis in enteric fever; rapidly fatal symptoms may appear during the 3d or 4th week in a case whose general state, pulse and temperature, were not such as to lead to the expectation of such a result until within a short time of its occurrence. There exists no means by which we can tell those cases in which an ulcer in its progress is likely to open into a vessel, or into the peritoneal cavity. Prognosis, therefore, should be ever most guarded.

Treatment.—In this the thermometer gives no aid, unless in those cases in which it is said to give the earliest indication of intestinal hæmorrhage.

It may not be out of place to note here the chief points of distinction betwixt the thermometry of enteric and that of typhus fever, and also the points of resemblance. In mild cases of typhus the range is equable, and has a morning fall of rarely more than one degree. In mild cases of enteric it is also equable, but the morning fall is more decided, often two or three degrees. As many cases of each form of fever meet at and cross over this imaginary boundary line it is not of much use in diagnosis.

In cases with severe head-symptoms the range is high, and often irregular in both diseases; their more rapid development in enteric fever, when they are the real source of danger, prevents the thermometer from giving that timely warning of their approach which it very often gives in typhus. In connexion with this must be borne in mind the fact, that an amount of head-affection which would cause no great uneasiness in a case of typhus may be the source of much anxiety in a case of enteric.

Danger from asthenia is not indicated by the thermometer in either, unless there is great and alarming depression, which, in both diseases, causes an abnormal fall of the temperature.

Complications act much in the same way in both; inflammatory affections, as a rule, have an elevating, depressing agencies a lowering tendency.

The range during defervescence is the main point of distinction between the thermometry of the two diseases. In typhus the fall is often sudden, and the normal range reached in a few hours—no subsequent rise taking place; in most cases the process of defervescence is completed within 48 hours of the time at which it commenced; in most cases also the fall does not cease at 98·4°, but goes one or even two degrees below it. In enteric the temperature may fall to the normal standard on the morning of the 1st day of defervescence, and may never exceed that range on any subsequent morning; but in the evening it will for several days show a rise of 2, 3, 4, or even 7 degrees, having in fact, during the first few days of defervescence, an evening range little if at all below that which existed at the height of the fever; this nocturnal rise

may continue in an exaggerated degree for several days, it then gets less and less until (perhaps ten days after the commencement of defervescence) the normal standard is reached. The process is never sudden in enteric, it is never by crisis; it generally takes a week for its completion, often more. I never saw a case of uncomplicated typhus in which it took more than four days. During the early part of convalescence from enteric it is rare for the temperature to fall as low as 97°; in typhus it frequently does so.

In typhus, in consequence of the greater frequency of dangerous cerebral symptoms and their more gradual development, the thermometer is most serviceable in prognosis; in enteric, in consequence of the peculiar mode of defervescence, it is of most use in diagnosis. It must be borne in mind, however, that without any aid from the thermometer, the difficulties in the way both of diagnosis and prognosis are greater in enteric than in typhus.

When the thermometer is used in the study of enteric fever, care should be taken that the observations are made at the same hour every day. If only one observation *per diem* can be made, the afternoon or early part of the evening should be chosen, as the range is highest then; but the morning fall of mild cases, the irregularities of severe cases, and the commencement of defervescence cannot be detected unless two observations are made daily,—one in the morning, the other in the evening. This is a great obstacle in the way of the general employment of the instrument in private practice, the more so as it is only in exceptional cases that observations of the temperature do more than corroborate the testimony afforded by the other symptoms.

The above facts may be briefly summarized:—

1. The main feature in the thermometry of a typical case of enteric fever is a marked tendency to a morning fall and evening rise, which during defervescence is so great as to be characteristic of the disease.

2. The temperature during the continuance of the febrile state, however, does not enable us to distinguish enteric from other forms of fever.

3. In cases resembling, and apt to be mistaken for, typhus, the evidence given by the thermometer is uncertain, and not equal in value to that got from other sources, especially the condition of the eyes.

4. In cases which throughout have been doubtful, it often gives during defervescence the only certain testimony as to the nature of the malady.

5. When head-symptoms are severe, and death is threatened by coma, the range of the temperature is generally high and irregular; but the more rapid onset of the head-symptoms in such cases does not permit of the thermometer giving the same premonitory indications which it often gives in typhus.

6. Irregularities in the range, absence of the morning fall, and still more a morning temperature which exceeds that of the evening, are indications of severity, and specially so when the range is at the same time high.

7. A fall of the morning temperature at the end of the 2d week, and a subsequent range during the 3d, lower than that which existed during the 2d, are favourable signs.

8. The highest range occurs in cases which present marked head-symptoms.

9. Inflammatory affections springing up during the course of the disease have an elevating tendency.

10. Hæmorrhages and diarrhœa have, when profuse, a lowering tendency.

11. A fall of the morning range is generally the first sign of commencing defervescence.

12. Elevation of the temperature is one of the earliest indications of a relapse.

PNEUMONIA.

This is a disease whose course and extent can, in most cases, be so accurately determined by the physical signs to which it gives rise, that it has, almost ever since the stethoscope was introduced, been a favourite subject for clinical investigation and study. The same circumstances, which render the disease itself an interesting subject, impart interest to its thermometry. The extent of the pulmonary lesion, the exact condition of the affected portion of lung, and the various changes which take place in it, can be so accurately determined by the physical signs, that we get thereby a standard of comparison by contrast with which it might be supposed that the value of the information given by the other symptoms could be determined. Such, however, is not the case; the extent of the pulmonary lesion bears no definite relation to the severity of the symptoms. Pneumonia of the apex, as a rule, gives rise to more serious symptoms than pneumonia of the base, even though the extent of pulmonary tissue involved may be even greater in the latter than in the former. In the words of Dr Graves—"In estimating the danger of a patient labouring under pneumonia, it is not so much in proportion to the extent of lung engaged as to the quickness of respiration, and the presence or absence of symptoms of asphyxia." It might be interesting to determine whether the same statement applies to the temperature, whether its range bears any definite relation to the amount of the lung affection, and whether the information which it gives is of equal or greater value than that afforded by the pulse, respirations, and general symptoms.

By such an inquiry some definite idea might be formed regarding the utility of the instrument in diagnosis, prognosis, and treatment. The only way to investigate such a subject is carefully to study individual cases. The limits of this paper prevent me from giving more than a very few.[1]

I. A female, aged 14, came under observation on the 2d day of her illness.

2d day.—Has general febrile aspect; complains of cough and pain in right side; tongue furred; pulse 120, resp. 48, temp. 103°; in lower half of right back percussion is dull, breathing bronchial, and at close of inspiration are heard fine crepitant râles; to have poultice to side: *vesp.* sputum distinctly rusty; pulse 124, rather feeble, resp. 50, temp. 103·2°; to have four ounces of wine per diem.

3d day.—Passed rather a restless night; physical signs as last noted, except that the crepitation is less; has eruption of herpes on nose and lips; pulse 100, resp. 44, temp. 100·3°: *vesp.* breathing purely bronchial, no moist sounds heard; pulse 100, resp. 44, temp. 102·1°.

4th day.—Had a pretty good night; expresses self as feeling better, and has a clearer expression; pulse 100, resp. 50, temp. 101·1°: *vesp.* pulse 108, resp. 56, temp. 101·3°; physical signs unchanged.

5th day.—Percussion still quite dull; breathing purely bronchial, and unaccompanied by moist sounds; pulse 120, resp. 44, temp. 103·4°: *vesp.* pulse 116, resp. 64, temp. 103·4°; physical signs unchanged.

6th day.—Has much improved expression; skin moist; tongue cleaning; percussion note improved; bronchial character of breathing less marked; sounds more natural, and mingled both during inspiration and expiration with redux crepitation; pulse 96, resp. 36, temp. 98·6°: *vesp.* pulse 88, resp. 32; temp. 98·4°.

7th day.—Improvement in physical signs continues; pulse 84, resp. 32; temp. 96·8°: *vesp.* pulse 64, resp. 36, temp. 97·6°.

8th day.—Pulse 80, resp. 28, temp. 97·4°: *vesp.* pulse 60, resp. 36, temp. 98°; a few sub-crepitant râles are still audible.

The patient recovered rapidly, and was discharged perfectly well on the 20th of June. On the 24th of December, six months after date of discharge, she was readmitted, having been three days ill, and complaining of symptoms very similar to those from which she had formerly suffered.

II. *4th day.*—*Vesp.* skin hot and dry; tongue furred; complains of cough, and pain in left side; sputum rusty; in lower two-thirds of left back percussion is dull, and breathing purely bronchial; pulse 120, resp. 56, temp. 102·3°; to have poultice to side.

5th day.—Physical signs unchanged; pulse 120, resp. 56, temp. 103°: *vesp.* pulse 124, resp. 68, temp. 104·2°; hepatization complete.

6th day.—Expression much improved; tongue cleaner; skin moist; percussion less dull—woodeny in character; breathing less bronchial, and mingled with abundant sub-crepitant râles; pulse 96, resp. 38, temp. 99·1°.

7th day.—Physical signs improving; natural murmur everywhere audible, mingled with redux crepitation; in left dorsum there is also distinct pleuritic friction; pulse 72, resp. 32, temp. 96·4°.

8th day.—At extreme left base posteriorly percussion is again dull, and the breathing less natural—distant and somewhat bronchial; pulse 68, resp. 32, temp. 96·8°.

9th day.—Pulse 72, resp. 28, temp. 97·1°.

10th day.—Physical signs of pleuritic effusion still exist at base, but are not extending; pulse 72, resp. 36, temp. 97°.

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[1] In giving these cases, I have curtailed the notes as far as this could be done without making them bald.

The side was blistered, the fluid was absorbed, and the patient made a perfect recovery.

III. A male, aged 13, came under observation on the 6th day of illness.

6th day.—*Vesp.* skin hot and dry; tongue furred; coughs a good deal; sputum rusty; percussion and respiration everywhere normal, except at extreme left base posteriorly, where, over a space an inch and a half in extent, the percussion is dull, and the breathing bronchial, with a few fine crepitant râles at the close of the inspiratory effort; pulse 120, resp. 36, temp. 102·8°.

7th day.—Physical signs unchanged; pulse 120, resp. 48, temp. 104·2°: *vesp.* pulse 124, resp. 48, temp. 104·3°.

8th day.—Breathing purely bronchial; no moist sounds heard beyond a very few crepitant râles at close of inspiration; pulse 120, resp. 36, temp. 103·8°: *vesp.* pulse 96, resp. 30, temp. 105°.

9th day.—Has much improved aspect; percussion note less dull; breathing more natural, and mingled with redux crepitation; pulse 80, resp. 28, temp. 97·2°: *vesp.* pulse 72, resp. 24, temp. 98·5°.

Convalesced satisfactorily.

IV. A male, aged 21, came under notice on the 5th day, with the usual general symptoms of pneumonia.

5th day.—*Vesp.* over upper third of left back the percussion is dull, and the breathing purely bronchial; no moist sounds audible; patient is restless, and suffering from considerable depression; pulse 96, feeble, resp. 64, temp. 103·2°; to have poultice to back, and eight ounces of wine per diem.

6th day.—Passed a sleepless night; general condition and physical signs unchanged; pulse 92, resp. 64, temp. 101·6°: *vesp.* pulse 88, resp. 54, temp. 102·8°.

7th day.—General aspect improved; percussion in back less dull; breathing not so purely bronchial, and having a few sub-crepitant râles mingled with it; in infra-clavicular and upper mammary regions of affected side, however, the percussion note is impaired, woodeny in character, and the breathing is slightly bronchial; pulse 68, resp. 60, temp. 99·7°: *vesp.* pulse 68, resp. 42, temp. 99·8°.

8th day.—Perspired freely during the night; percussion everywhere improved; natural respiratory murmur, mingled with redux crepitation, everywhere audible; pulse 60, resp. 40, temp. 97·8°: *vesp.* pulse 56, resp. 44, temp. 98·6°.

9th day.—Improvement continues; pulse 56, resp. 40, temp. 97·8°: *vesp.* pulse 52, resp. 38, temp. 99·5°.

10th day.—In region of former pneumonia signs, percussion and auscultation reveal only normal sounds; in lower part of same side, posteriorly and laterally, percussion is clear, but distinct pleuritic friction is audible; pulse 52, resp. 32, temp. 98·2°: *vesp.* pulse 52, resp. 36, temp. 100°.

11th day.—Apex sounds good; friction as at last note; pulse 54, resp. 24, temp. 98·1°: *vesp.* pulse 52, resp. 28, temp. 98·2°.

12th day.—Pulse 54, resp. 28, temp. 98°: *vesp.* pulse 52, resp. 30, temp. 98·4°.

13th day.—Friction still audible, but much less distinct; pulse 52, resp. 22, temp. 98°.

The friction disappeared, no effusion took place, and the patient was discharged perfectly well.

V. A male, aged 26, came under observation on the 4th day of illness.

4th day.—Complains of cough, spit, pain in chest, and general febrile symptoms; sputum rusty; over the lower two-thirds of the right, and over the lower fourth of the left back, the percussion note is dull, the breathing tubular, and mingled, at the close of inspiration, with fine crepitant râles; pulse 84, resp. 40, temp. 100°; to have a poultice to the back, and six ounces of wine per diem: *vesp.* pulse 116, resp. 48, temp. 102°.

5th day.—Passed a restless night; breathing in both affected portions of lung still tubular; urine highly alkaline, contains a trace of albumen, chlo-

rides very deficient, throws down a copious deposit of phosphates and carbonates; patient is a good deal depressed; pulse 104, resp. 40, irregular, temp. 100·5°; to have wine to ten ounces per diem: *vesp.* pulse 120, resp. 42, temp. 101·6°.

6th day.—Physical signs unchanged; general depression more marked; pulse 116, weak and compressible, resp. 36, temp. 101·2°; to have four ounces of whisky in addition to wine: *vesp.* pulse 132, resp. 36, temp. 101·7°.

7th day.—Tongue dry in centre; urine has a trace of albumen, chlorides very deficient; in right back percussion is improved, the breathing more natural, and mingled with redux crepitation; in left back percussion is still dull, and breathing tubular; pulse, 112, resp. 40, temp. 99·6°: *vesp.* pulse 116, resp. 36, temp. 100·6°.

8th day.—Tongue moist and cleaning; improvement in right back continues; in left back percussion is still dull, but the breathing is not quite so bronchial, and is mingled with redux crepitation; pulse 88, resp. 28, temp. 97·8°: *vesp.* pulse 84, resp. 28, temp. 97·8°.

9th day.—Tongue clean; skin moist; natural respiratory murmur everywhere audible, mingled with sub-crepitant râles, most abundant in left side; pulse 92, resp. 20, temp. 97·6°: *vesp.* pulse 76, resp. 30, temp. 98·6°.

Improvement continued, and convalescence was satisfactory.

VI. A male, aged 50, came under observation on the 5th day.

5th day.—*Vesp.* complains of cough and pain in left side; breathing is considerably embarrassed; all over the left front, with the exception of the infra-clavicular region, percussion is dull, the breathing tubular, and mingled at the close of inspiration with fine crepitant râles; over the rest of the chest percussion is good, but the respiratory sounds are mingled with sibilation and a few mucous râles; pulse 100, feeble, resp. 30, temp. 100°; to have turpentine stupes applied to the chest, and half an ounce of whisky every three hours.

6th day.—Breathing still much embarrassed. Physical signs as at last note, with the addition of pleuritic friction in left lower back; pulse 120, resp. 32, temp. 102·5°.

7th day.—Is greatly depressed, too weak for examination; pulse 96, resp. 52, temp. 96·5°; to have half an ounce of whisky every hour.

8th day.—Still very low; tongue dry; pulse 100, resp. 60, temp. 96·8°.

9th day.—Has rallied somewhat, but still too weak to admit of examination of chest; tongue dry and brown; pulse 96, of better volume, but very compressible, resp. 60, temp. 96·6°.

10th day.—Percussion in region of former dulness much improved; auscultation reveals wheezing, with mucous râles all over the chest, and mingled in the left front with the finer sub-crepitation of a resolving pneumonia; expectoration copious, tenacious, and slightly rusty; pulse 92, resp. 44, temp. 96·4°.

11th day.—Bronchitic signs as at last note; pulse 84, resp. 48, temp. 95·7°.

12th day.—Pulse 96, resp. 52, temp. 97·1°.

Patient continued slowly to improve, and was ultimately discharged well, with the exception of a cough, to which he had been subject for years.

VII. A female, aged 38, came under observation on the 4th day of illness.

4th day.—*Vesp.* has troublesome cough and much embarrassed breathing; tongue dry in centre; sputum rusty; over lower two-thirds of left back and lateral region percussion dull, breathing bronchial, with fine crepitation at close of inspiration; over the whole of the chest rhonchus and sibilus, with a few coarse moist sounds; pulse 108, resp. 52, temp. 101·3°.

5th day.—Was delivered of a six months' child this morning. Physical signs as at last note; pulse 88, resp. 52, temp. 97·7°: *vesp.* pulse 136, resp. 60, temp. 102·8°.

6th day.—Breathing greatly embarrassed; patient is very restless, constantly tossing the clothes off; is with difficulty prevailed on to take either nourish-

ment or stimulant; physical signs unchanged; pulse 120, resp. 48, temp. 101·2°: *vesp.* pulse 108, resp. 50, temp. 98°.

7*th day.*—Face somewhat livid; breathing greatly embarrassed; sputum abundant, rusty and frothy; physical signs of hepatization distinct and perfect as on admission; abundant coarse mucous râles all over the chest; is much depressed; pulse 132, resp. 56, temp. 99°: *vesp.* pulse 128, resp. 60, temp. 99·6°.

Died during the night. No post-mortem obtained.

VIII. A male, aged 45, had been ill for several days with cough and general febrile symptoms; exact duration of illness could not be ascertained. On admission on 16th April was considerably depressed; tongue dry in centre; sputum rusty; conjunctivæ yellowish; urine slightly albuminous, and very deficient in chlorides. Over lower two-thirds of left back percussion dull, breathing tubular, and having fine crepitant râles mingled with it at the close of inspiration; pulse 92, feeble, resp. 32, temp. 102·8°; to have 8 ounces of wine per diem, with milk and beef-tea: *vesp.* pulse 84, resp. 34, temp. 100·5°.

17*th.*—Physical signs and general state unchanged; pulse 84, resp. 32, temp. 102·8°: *vesp.* pulse 112, resp. 32, temp. 103·2°.

18*th.*—Passed a restless night; is too weak for examination; skin and conjunctivæ yellowish; pulse 80, resp. 28, temp. 101·2°; omit wine; to have half an ounce of whisky every two hours: *vesp.* pulse 84, resp. 24, temp. 102·2°; breathing purely bronchial in region formerly indicated.

19*th.*—Retains nothing on stomach; is greatly depressed; pulse 84, resp. 24, temp. 102°; to have a pint of beef-tea and an ounce of whisky as an injection every two hours: *vesp.* pulse 84, resp. 42, temp. 100·6°.

20*th.*—Much sunk; respiration laboured; pulse 96, resp. 60, temp. 102·6°: *vesp.* pulse 96, resp. 56, temp. 100·6°.

21*st.*—Pulse 100, resp. 52, temp. 101·8°.

Died two hours after last observation. Post-mortem examination, twenty-four hours after death, showed red hepatization of about lower two-thirds of left lung. The other organs were healthy.

I. and II. were exquisite cases, which were allowed to run their natural course, uninfluenced by remedial agents. Treatment consisted solely in the application of a poultice to the side, and the administration of a light but nourishing diet, consisting chiefly of milk and beef-tea, with, in the case of No. I., 4 ounces of wine per diem. They are remarkable as both occurring in the same person, a robust healthy girl, who had not the slightest trace of a tubercular taint; an interval of six months intervened between the attacks. In each case the general febrile state, as indicated by the respirations, pulse, and temperature, was at its height on the 5th day; the physical signs of hepatization were then complete. On the 6th morning there was found, in both instances, a marked improvement in the general symptoms, and a very decided fall in the pulse, respirations, and temperature; whilst, at the same time, the physical signs indicated that absorption of the effusion had commenced—that the stage of red hepatization had terminated, and that of resolution set in. In No. I. it will be observed that the range of the temperature and pulse on the 3d and 4th days was much lower than on the 2d. At this time, also, the appearance of a herpetic eruption on the lips, and a change for the better in the feelings and aspect of the patient, seemed to indicate a commencing defervescence. The

physical signs, however, were still those of consolidation, and this, along with the increased frequency of the respirations, led to the belief that the diminution of the general state of irritation, as indicated by the fall in the pulse and temperature, was due more to the quiet and comfort of the hospital (the patient came from a bad and noisy locality, and was in a wretched state on admission) than to declension of the disease—an opinion which was strengthened by a re-elevation of the temperature and pulse on the 5th day, and their subsequent decline only when resolution of the pneumonia had set in.

				Pulse.	Resp.	Temp.	Max. Temp.
The mean range of	I.	in	morning	was 110	46	102·1°	...
,,	,,	,,	evening	,, 112	53	102·6	103·4°
,,	,,	II.	morning	,, 120	56	103·	...
,,	,,	,,	evening	,, 122	62	103·5	104·2

No. III. was also a well-marked case, which was not interfered with by treatment. On the 9th morning the physical signs of commencing resolution replaced those of perfect consolidation. The febrile symptoms subsided at the same time. The great fall of the temperature, from 105° on the 8th evening to 97·2° on the 9th morning (nearly 8 degrees), is noticeable. It will also be observed that on the 8th evening, when the temperature was at its highest, the pulse had fallen from 120 to 96. I have, on one or two occasions, found a fall in the pulse precede the fall of the temperature. The chief reason for which this case is instanced is to show that the range of the temperature bears no direct relation to the extent of the pulmonary lesion. Here there was only a very small portion of the left base involved, and yet the mean range of the temperature during the height of the disease was higher than that of any other case of which I have notes—the morning average was 104°, the evening 104·5°, the maximum 105°. In Case V., in which the pulmonary lesion was four or five times as great, the range was 3 degrees lower. The highest point which I ever found the mercury reach in pneumonia was 105·2°, and that was in a case which presented no alarming features.

In Case IV. the declension of the febrile symptoms, and the appearance of the physical signs of commencing resolution, are again simultaneous. It is chiefly interesting as showing that the former declined as soon as the latter were observed in the back, even though the physical signs in front indicated an extension of the pneumonia in that direction. The temperature, however, did not reach the normal standard until the physical signs of resolution were general, on the 8th day; the pulse was down to 68 on the 7th. No. V. was a case of double pneumonia, in which two-thirds of the lower part of the right back, and one-fourth of the corresponding portion of the left were involved. The case was an anxious one. It possesses, in its thermometry, several points of interest. The range of the temperature was far from high, and gave no indication

of the severity of the case; in this respect it was much inferior to the pulse and respirations.

	Pulse.	Resp.	Temp.
The average range was in morning,	101	39	100·5°
" " evening,	122	42	101·7
The maximum range was	132	64	102·

Whilst the stage of hepatization continued there was no tendency to a diminution in the febrile symptoms, but, as in No. IV., when (on the 6th day) there were observed signs of commencing resolution in the right back, though not in the left, the mercury fell more than a degree, but the temperature did not reach the normal standard, and the pulse and respiration showed no decided tendency to fall till the 8th day, when signs of commencing resolution were found in the left side also.

No. VI. was a case in which general bronchitis existed as well as pneumonia involving nearly the whole of the left lung. So great was the patient's state of depression, that he could not be disturbed by a daily examination; and to such an extent did the lung affection interfere with the proper aëration of the blood, that he was in imminent danger of being asphyxiated. Observations were made only once a day—at noon. On the 7th day, that on which the depression became extreme, the temperature fell from 102·5° to 96·5°—six degrees, and remained below the normal standard throughout the rest of the case; the pulse at the same time ranged from 90 to 100, and the respirations from 40 to 60. The case is instanced for the purpose of showing that a sudden and great fall of the temperature does not always indicate commencing resolution, but may be the result of alarming prostration and great pulmonary embarrassment.

Nos. VII. and VIII. proved fatal during the stage of red hepatization. Irregularity is the chief characteristic of their thermometry. They are instanced for the purpose of showing that, in pneumonia, a high temperature is not an essential feature of a severe case, and that irregularity of the range is of more prognostic value than mere height with regularity. When the fall which usually occurs in the morning does not manifest itself, when the evening temperature is lower than that of the morning (defervescence having not yet commenced), or even when the normal relation exists in an exaggerated degree, we have a range which, so far as my experience goes, occurs only in very bad cases. The severity of the general symptoms, however, quite throws into the shade the prognostic value of this feature of the thermometry; nevertheless, I am inclined to attach some importance to it, and to regard it as at least an interesting corroboration of the evidence afforded by the other symptoms.

In No. VII., the great fall on the morning of the 5th day was probably due, in part at least, to the depression consequent on the hæmorrhage which accompanied the miscarriage.

No. VIII. is remarkable for the low range of the pulse, respirations, and temperature, as well as for the great irregularity of the last. The symptoms in this case were of a markedly typhoid character; the patient died as much by asthenia as by asphyxia.

The above facts are sufficient for purposes of illustration. Before proceeding to indicate their exact bearing on the question of the value of the thermometer as an aid in diagnosis, prognosis, and treatment, it may be interesting to inquire whether or not they throw any light on the natural history of the disease. Indeed, before the instrument can be of use in prognosis, it is necessary that we should have our minds made up as to the exact stage of the disease at which improvement begins, and the particular changes in the physical signs which accompany and give evidence of commencing resolution.

Is the inflamed condition of the lung merely a local manifestation of the existence of a general disease of the blood, which is purified by the effusion that gives rise to the condition of hepatization; or is the local affection, the primary lesion, and the general febrile disturbance, only its result? In other words, does the disease begin to decline when effusion is complete—when the stage of hepatization, as indicated by dull percussion, tubular breathing, and the absence of moist sounds, has replaced the first stage of engorgement, with its fine crepitant râles? or does this not take place till the third stage has set in—till the diminished bronchial character of the breathing and the appearance of redux crepitation give evidence of commencing absorption of the effused matter? Most physicians in this country regard the pneumonia as the primary lesion. The question is well stated by Dr Parkes, in a lecture published in the *Medical Times and Gazette*, vol. i. for 1860, and quoted largely by Dr Aitken in his work on the "Science and Practice of Medicine":—"What is the exact connexion betwixt the lung symptoms and the general pyrexia? The course of the two is certainly not exactly parallel. The complete consolidation seems to be posterior, in point of time, to the height of the pyrexia. It has been even supposed by Wunderlich that the exudation into the lungs coincides with the end of the pyrexia—that is to say, that the defervescence commences when the lungs become completely hepatized. I have not been able to satisfy myself rigorously on this point. If it could be satisfactorily made out, it would certainly imply that the exudation into the air-cells relieved or cured the fever—in other words, that the lung disease is not a primary but a secondary condition, and that it succeeds to and brings to an end, by purifying the blood, a condition of general pyrexia arising from blood disease. Without believing that this relation is quite determined, there is no doubt that the fever ends spontaneously, or very greatly lessens, at the time when the inflammation of the lung is very great."

I believe that the course of the lung symptoms and the general

pyrexia is exactly parallel; that Wunderlich's supposition is unsupported by evidence; that the declension of the febrile symptoms is contemporaneous with commencing resolution of the pulmonary effusion, and, consequently, that the lung affection is the primary lesion. It is indeed true in many cases, as Dr Parkes says, "that complete consolidation, in point of time, seems to be posterior to the height of the general pyrexia;" that it is not always so will be seen by referring to the three first cases which I have instanced, in all of which the highest temperature was that noted on the last day on which the physical signs of hepatization were complete; nevertheless, it is very frequently so. I have on several occasions found the temperature as high on the second (before which I never had the opportunity to make an observation) as on any subsequent day of the illness; but it does not follow therefrom that the course of the lung symptoms and the general pyrexia in these cases was not parallel. There is no reason why the febrile symptoms should not attain on the second day a height little, if at all, below that of any subsequent period, for the inflammation is then as active as at any other time—the pneumonia proper is, so far as its febriforming effects are concerned, as severe then as when the affected portion of lung is fully hepatized. When this condition is established, there are added to the inflammatory risks those caused by effusion into the pulmonary tissue, of purely physical origin, and evidencing a more mature, but not necessarily a more active, inflammatory state than that which existed on the first day of the disease. From the manner in which the first, second, and third stages of pneumonia are talked of, one unacquainted with the disease would suppose that there were three distinct epochs in its history, separated from each other by a distinct line of demarcation, and having each its own definite period of existence. Such is not the case; they merge imperceptibly into each other, and each so overlaps the imaginary boundary line which separates it from its neighbours, that it is impossible to point to any particular time at which one ceases to be, and another comes into existence. This is specially true as regards the first and second stages; the latter is the mere completion of a process which was begun in the former; the effusion into the pulmonary tissue which characterizes it is the necessary and normal consequence of the inflammatory action which constitutes the so-called first stage of engorgement. The later development of complete consolidation is explained by the fact, that it is a physical condition depending for its existence on the inflammation which precedes as much as on that which accompanies it, and requiring a certain amount of time for its completion. Hence it is obviously illogical to say that the courses of the lung symptoms and general pyrexia are not parallel, because complete consolidation is posterior in point of time to the height of the pyrexia.

In sixteen cases of simple uncomplicated pneumonia (one or two of which have been instanced), which it was believed might safely be

left to take their natural course, and in which, beyond the application of a poultice to the affected side, and the administration of a light nourishing diet, no treatment was adopted, the pulse, respirations, temperature, and state of the chest, were noted morning and evening. It was found that the temperature (the best index of febrile disturbance) reached at a very early stage of the disease a point which served well to indicate its range throughout the case, and that it showed no decided tendency to fall till about the time at which the physical signs indicated that the stage of complete hepatization had ceased, and that of resolution commenced. To tell, from a physical examination of the chest, the precise moment at which one stage ceases and another begins, is beyond the power of the most accomplished stethoscopist — nay, the disease may commence at one part of the lung, and extend so gradually that the portion first affected may be undergoing resolution, whilst that last involved may still be in a state of consolidation. Under these circumstances, I think that we are warranted in saying that the declension of the febrile symptoms is contemporaneous with the commencement of the third stage, if we find the pulse and temperature high during the stages of engorgement and of red hepatization, and declining only about the time that the physical signs give the earliest indications of the onset of the stage of resolution. Perfect accuracy in such a case is impossible. If, at one observation, we find the affected portion of lung completely hepatized, and the pulse, respirations, and temperature maintaining the high range which has characterized them for some days previously; and if, on making another observation six or eight hours afterwards, we find that the general symptoms are improved, that signs of commencing resolution are perceptible in the pneumonic district, and that a very marked fall has taken place in the range of the temperature, and in the rapidity of the pulse and respirations; and if we find the same phenomena presenting themselves at the same time in every well-marked case in which the period of their occurrence is noted,—we are perfectly justified in concluding that defervescence and resolution of the pneumonia commence at the same time, even though we may not in a single instance have been able to determine the precise moment at which either began. My experience is, that the temperature shows no tendency to fall till the second stage terminates in the third, and that then the mercury indicates a great and rapid fall—so rapid that the normal standard is generally reached within twenty-four hours, often in much less. I never saw a case in which the pulse, respirations, and temperature showed any tendency to a progressive fall whilst the stage of hepatization was mature; and I never saw a case in which the pulse and temperature continued high after the physical signs gave evidence of its termination.

Let us turn to the general symptoms and physical signs themselves, and see what testimony they afford. I am sure that I only state what accords with the experience of all physicians when I say that the general state of the patient does not improve whilst the

lung remains consolidated, and that he would be most imprudent who would, on finding this condition of the lung, announce that the disease had attained its height and was now about to decline, ignoring not only the dangers of the second stage, but also the possibility of the red passing into gray hepatization, and subjecting the patient to all the risks of suppuration of the lung.

Let us now see whether or not the thermometer is of any use in diagnosis, prognosis, and treatment.

Diagnosis in pneumonia presents so little difficulty that one who has seen much of the disease can generally, from the mere aspect of the patient, say what is the matter with him; in addition, there is so much reliable information to be got from the state of the sputum, and the physical signs in the chest, combined with the general febrile symptoms, that a faulty diagnosis is most improbable. Under these circumstances, it is not to be expected that the thermometer could give information of much value in a diagnostic point of view; it simply indicates the presence of febrile disturbance.

Prognosis.—In forming an estimate of a case of pneumonia, and in giving an opinion as to its probable course, I have never received much aid from the thermometer; neither do I think that it can give information so definite and reliable as that which is derived from a careful study of the general symptoms, and a physical examination of the chest. Dr Aitken adopts Wunderlich's rule, that when the mean temperature is 104°, and the averages of the pulse and respirations above 120 and 40 respectively, the case is a severe one, and *vice versâ*. Rigid adherence to any such rule must lead to errors in prognosis, and possibly in treatment. There is no doubt that a case which habitually shows such a high range of pulse, respirations, and temperature, is likely to be a severe one; but the contrary does not hold good. I have instanced in No. VIII. a case of fatal uncomplicated pneumonia which occurred in a previously healthy man, 45 years of age, in whom the two lower thirds of the left lung were involved, who was under observation for five days before death, and in whom the average temperature was 101·8°, the average pulse 90, and the average of the respirations 38; the highest temperature was 103·2°, the highest pulse 112, and the respirations were never above 32 till within 36 hours of the fatal termination.

In No. III., which was never an anxious case, and in which only a small portion of lung was involved, the averages of the pulse, respirations, and temperature, before the commencement of defervescence, were as nearly as possible 120, 40, and 104°. As already remarked, irregularity of the range (see Cases VII. and VIII.) is a worse sign than mere height.

In ordinary cases of pneumonia the thermometer is an accurate index of the amount of febrile disturbance, and bears also early and reliable testimony to the commencement of defervescence; but in the formation of a prognosis it gives no aid which can for a moment be compared in value with that got from other sources, and more especially from the general symptoms. When we consider the usual mode of fatal termination, this is not matter of surprise. Death in this disease is generally due to asphyxia; when this is imminent, one result of the deficient aëration of the blood is a reduction in the temperature of the body; the thermometer, of course, declares the fact, and gives evidence whose value is directly as the extent of the abnormal fall; but be that fall ever so great, it can only serve as an interesting corroboration of the more reliable evidence of the general signs of threatened asphyxia. The words of Dr Graves, already quoted, are still applicable, "The quickness of the respirations and the presence or absence of symptoms of asphyxia" are the best means of estimating the danger in pneumonia.

The value of the pulse as an index to the amount of febrile disturbance and to commencing resolution is as great as that of the temperature. I know, indeed, of no disease in which the pulse and temperature run more regularly together, accompanying each other in their rise and fall in a manner which is quite remarkable, and renders their evidence mutually corroborative. As a sign of commencing defervescence, a fall in the frequency of the pulse occasionally precedes by several hours a diminution of the temperature; an instance of this is given in Case III.

The respirations may, in some cases, rise and fall with the pulse and temperature, but often during the first and second stages their range is, as compared with the natural rate, much higher and more often irregular. In prognosis, they generally give more valuable evidence than either the pulse or temperature; unusual frequency of the respirations is an invariable accompaniment of great pulmonary embarrassment; unusual rapidity of pulse, or height of temperature is not. They do not, as a rule, show a tendency to come to the normal standard until some days after the febrile symptoms have ceased; this is to be accounted for by the fact, that it takes some days to remove all the effused matter from the lungs and restore them to their natural state. The physical obstruction to the entrance of air into the cells of the affected portion of lung requires some time for its removal as well as for its formation. The same circumstances which explain Dr Parke's belief that "complete consolidation seems to be posterior in point of time to the height of the pyrexia," account also for his belief that "the fever ends spontaneously, or very greatly lessens, at the time when the inflammation of the lung is very great." With the onset of the local affection the general inflammatory symptoms are rapidly developed, whilst the changes in the lung substance require some days for their maturation; with its decline, the general inflammatory symptoms as quickly subside, whilst the restoration of the lung substance to its normal state requires again some days for its completion. The chief use of the thermometer in practice is to give early notice of the commencement of this reparative process.

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An Essay on the Contagion, Infection, Portability, and Communicability of the Asiatic Cholera in its relations to Quarantine; with a brief History of its Origin and Course in Canada, from 1832.

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A synopsis of the following Essay was laid before the American Medical Association at its annual meeting, held in Cincinnati, Ohio, in May, 1867.

My chief object in this Essay, is to bring within the reach of the members of the medical profession *authentic data* connected with what must ever be an important epoch in the medical history of this continent; and I have been induced to publish it from the fact that, statements connected with the history and progress of Asiatic Cholera, since its first introduction into Canada, have from time to time found their way into print, and been received as reliable data, many of which were mere fictions, and others exaggerations and misrepresentations. I therefore determined to describe some of the events and scenes in which I have been an active participator, and submit the record of *a living witness;* a kind of evidence that must daily become more valuable and interesting as time rolls on, and we examine such testimony from a greater distance.

To the medical gentlemen, both in this Dominion and in the United States, who have kindly responded to my appeal for facts connected with this subject, I return my most sincere and grateful thanks.

That fell fiend, and until very recently, almost intractable scourge, Asiatic Cholera, first set its deadly foot-print on the American continent at Quebec, in Lower Canada, on the 8th of June, 1832, whence it extended its ravages with the tide of "human travel and human traffic" in every direction, sowing the seeds of misery and ruin, and reaping a fruitful harvest of desolation and death.

I had the honour to submit a plan of Quarantine for Asiatic Cholera, which I originated in 1856, to the resident physician of New York, Lewis A. Sayre, M.D., in the autumn of 1865; of which he says in his annual report, presented 4th January, 1866: "The plan of Quarantine suggested by Dr. Marsden, with some slight modifications, is the most perfect I have yet seen that could be adopted; and I would earnestly recommend the Government to adopt it as a uniformity along the entire coast." That plan which has since received the endorsement and approval of the entire United States of America, including the Surgeon General and Army Medical Staff, and the leading physicians and surgeons of the Union, is declared to be "*the most perfect system of Quarantine yet known*," and is founded on the assumption that Asiatic Cholera is an infectious, portable, and communicable disease, and can be communicated and transmitted, both by persons and personal effects and is therefore a controllable disease.

My opinions on this subject are founded on my own experience and observations, which commenced with the first case of the pestilence that appeared on this continent, and extended over six separate and distinct visitations.

The result of that experience is, an indelible conviction that Asiatic Cholera is not endemic in Europe or America; but is an exotic, having its origin in India, where only *it is endemic* and indigenous, and whence it has constantly been imported into Europe and America by persons and personal effects tainted by its deadly poison; and *that its progress can be arrested by an efficient and uniform system of Quarantine, whether on sea or land, and its infectious properties can be destroyed by proper chemical agents.*

[Since I commenced this paper, the Metropolitan Board of Health of New York, has demonstrated the soundness of my principles; and internal Quarantine, or as it is expressively called "stamping out," has become a *fait accompli*. To Elisha Harris, M.D., Registrar of Vital Statistics, belongs the honour of having been the first to carry this successful practice into operation in America.]

One of the gravest errors into which a physician can fall, and one calculated to lead him into fatal mistakes, is the confounding sporadic or

common cholera, or cholera morbus, (which is common to every country and every clime,) with Asiatic cholera; and which Kennedy most properly calls "the contagious cholera."

So identical are the symptoms and characteristics of these two distinct varieties of diseased action, having a common name, and in malignant cases, a common termination—a sudden and hideous death, that it is extremely difficult, if not impossible, to diagnose accurately and at once between them, unless you accept the doctrine of contagion.

Common cholera or cholera morbus *never* becomes epidemic, but consists of isolated cases, often of very malignant type, and occasionally resulting in death; whereas the Indian or Asiatic variety being once sown, spreads from person to person, and from place to place; exactly in proportion to the nature of the intercommunication between the sick and healthy, the extent and character of the exposure to the disease, and the susceptibility of the persons exposed. Wherever the pestilence has appeared in point of time, to have been communicated from a continent to an island, or from an island to a continent, or from one continent to another, or from one part of a country to another, the same strong evidence of contagion or infection is still developed, as first marked its progress throughout India, and the Indian Ocean. In England it first appeared at a seaport town which had frequent intercourse with the Baltic, and on this continent it first appeared at Quebec, which had maintained frequent intercourse with the three kingdoms, as I will show hereafter.

A definition of the meaning intended to be conveyed by the term contagious is essential to the proper understanding of this subject, no less to the pathologist than the physiologist. The latter, stickling for the literal meaning of the derivative of *con* and *tango*, insists on absolute contact to propagate disease. This is not the sense in which the term ought to be used in reference to Asiatic cholera. On that account, it is that I have used the term infectious in preference to contagious, the disease being transmissible from individual to individual through the medium of the atmosphere at a very limited distance,—say a few feet,—without personal contact. Now, although contagion comprehends infection in the general acceptation of the term, and signifies the transmission of disease from one person to another, by direct or indirect contact, I generally apply the term infection to Asiatic cholera, because the disease is reproduced and communicated by the approximation of persons or effects tainted, poisoned or infected, by the pestilence *without actual contact*.

"Kennedy," in the preface to his admirable work on the History of the Contagious Cholera remarks on this subject,—The variety of

Indian Cholera under consideration has had several names assigned to it, as the "epidemic cholera," the "spasmodic cholera," the "epidemic spasmodic cholera," "cholera asphyxia," the "malignant cholera," &c. &c. It matters little what name is bestowed upon a disease, provided the name leads to a knowledge of its *identity;* none of the preceding, however, seem sufficiently expressive for that purpose. In India, the *species* of cholera to which this variety belongs, had existence from the earliest ages, and, occasionally, had prevailed to a great extent, in an exceedingly virulent form, even previous to the year 1817; during these periods, some, or all of the above names, might very well apply; but, in 1817, the disease assumed a contagious property, which there is no evidence to prove it ever before possessed; and a name was then wanting to distinguish the new variety. Writers convinced of its propriety, have abstained from using the title "contagious cholera," in deference to the opposition of the non-contagionists." With every respect for the ability displayed in this opposition, I cannot pursue a similar course. My defence must rest on the facts adduced in favour of contagion, and the common practice, in physical philosophy, of adopting the hypothesis which best explains the phenomena.

So much has been said and written on the subject of cholera, and especially on its character and treatment, that it is my intention in the following essay, to confine myself entirely to the consideration of the abstract question of contagion or infection, in reference to its bearings upon Quarantine. In doing so, I intend to deal with *facts*, and present them to you properly attested, leaving you to interpret them at your leisure, and in your own way, as I do not intend to extract from the countless volumes of published evidence that is is already within your reach, but will cull fresh cases from my note-books, and other authentic unpublished sources, within my reach.

On each of the six invasions of Asiatic Cholera with which Lower Canada has been visited, I have been enabled personally to trace the earliest cases of the disease to importation, and its subsequent extension to uninterrupted intercourse between the sick and healthy, and to a want of a proper system of Quarantine.

Among the inaccurate statements connected with the origin of Asiatic Cholera on this continent, I find the following in Dr. Milroy's Report to the Colonial Office, on the Cholera Epidemic in Jamaica, 1840—51; and printed by order of the House of Commons, on the 11th May, 1854.

"In the first visitation of Cholera, in 1832, in the New World, it has been very generally believed that the earliest cases occurred at Quebec, in Lower Canada, about the beginning of June in that year. They have been confidently ascribed by some writers to direct and traceable importation by vessels from Europe, but this point is far from having been distintly made out, and Dr. Douglas, the medical officer at the Quarantine station at Grosse Isle, informed me that he has serious doubts upon its accuracy."

The gentleman whose authority Dr. Milroy cites was not at the time referred to, a medical officer at Grosse Isle at all, nor was he then in the Province; therefore, any thing he said on that subject was mere hearsay, and not in accordance with the facts, which leave no doubt on the minds of unprejudiced persons that Cholera was introduced into America through Quebec, and was "traceable to importation." The first case of Asiatic Cholera on this continent occurred at Quebec, in Lower Canada, on the 8th June, 1832, in the person of an Irish emigrant. Two vessels, the "Elizabeth" from Dublin, and the "Carricks" from the same place, arrived at the Quarantine station, Grosse Isle; the former on the twenty-eighth of May, with 200 passengers, having had twenty-two deaths from Cholera on the voyage; and the latter, on the third of June, with 145 passengers, having lost forty-two from Cholera in fifteen days. I say "from Cholera," although the report of the Boarding Officer was from some "unknown disease." Yet the vessel having come from a port known to be infected, and a case of Cholera having occurred after the arrival of the vessel at the Quarantine station, which proved fatal in three hours, there can be no manner of doubt as to the nature of the "unknown disease." There was at that time no proper system of Quarantine, no separation of passengers from Cholera vessels, and other ships. The only separation consisted in removing those *actually sick* from among those who continued *apparently* healthy, and who were *at once* sent on their journey.

Constant and uninterrupted intercourse was permitted between the Quarantine station and the city, by boats and steamers; and passenger steamers were even permitted to proceed to Grosse Isle, and take passengers direct to Quebec and Montreal. The unusual number of 7151 emigrants had arrived in the harbour of Quebec from the Grosse Isle Quarantine station between the second and the fifth of June, all of whom had been more or less subjected to exposure and contact with the infected; and whatever might have been thought of such a system of Quarantine at that time, (a mere rope often being the only means used to separate passengers from each other) it would surprise no one in the present advanced state of our knowledge on this subject, that Cholera had "overleaped the bounds," (to use a favourite phrase of the non-contagionists), of such a system of Quarantine.

The history of the British barque "Brutus," from Liverpool, with 330

passengers, is highly interesting in reference to this visitation. It appears by the letter of W. W. Thompson, M.R.C.S.L., the surgeon of the vessel, that cholera broke out on board about eight days after leaving the river Mersey, which induced the captain to put back. It also appears from this letter that between the 27th of May, when the first person was attacked, and the 13th of June, the day on which the vessel arrived at Liverpool, 117 cases had occurred, with 81 deaths and 20 recoveries. Had the "Brutus" been less severely visited, the captain would, no doubt, have held on to his destined port, and the passengers for their own sakes would have spoken of the occurrence of cholera on board their vessel as little as possible, and so the matter would have been hushed up. The occurrence of 81 deaths at sea, among 330 persons on board the same vessel, cannot be accounted for, says the eminent authority Graves, unless on the supposition that the disease is contagious, and he adds, "*one such positive fact is worth a volume of negative evidence*"

On Thursday, the 7th of June, the steamer "Voyageur," of Montreal, Captain Morin, left the Quarantine station with an enormous number of passengers for Montreal, and when about nine miles above Quebec was found to be so dangerously overloaded, that she was compelled to put back during the night, and re-land 200 passengers. She proceeded next morning on her voyage, and cholera broke out on board, on her trip up to Montreal, and several deaths took place on the passage.

Before arriving at Three Rivers, an emigrant named Kerr was taken ill, and died before the vessel came into the Port of Montreal. Another emigrant named McKee had been seized on the afternoon of the same day (9th June), and was taken from the boat to a tavern on the wharf. The body of Kerr was exposed and visited by numbers of persons, (as also McKee's,) at the tavern. A soldier from the barracks visited and assisted in rubbing his body, and was among the first victims, and the first soldier in the garrison who died. It was reported at the time that some dead bodies were thrown into the river from the steamer "Voyageur" on going up, but this fact was never authenticated. However, after the vessel passed Sorel, a feather-bed belonging to a passenger, who had died of cholera, was thrown overboard, a man named Latour, who lived on one of the small islands in the vicinity, saw it, and going out in a canoe, took it up and carried home his prize, and hung it up to dry. He took cholera immediately, and died in twelve hours. His wife also took the disease and died.

A poor fisherman who lived at the village of Contrecœur, a little below Montreal, was out fishing in his canoe, when a raft came floating past.

The captain of the raft asked the old man to take one of his men ashore, who had died, and bury him. He had not heard about the Cholera, took the body ashore, and buried it. During the same night he took ill and died. His wife also sickened; and people passing by on Sunday morning, and seeing the house shut up, mentioned the fact to his nephew, whom they met at the parish church. Going to his uncle's house, he found his uncle dead, and his aunt dying. After doing his duty by his relatives, he went home to the second concession, where he took ill, and died. There were no other cases between these two points several miles apart.

A Canadian drover from *William Henry* left that place, where there had been a few cases of Cholera, to go to the Eastern Townships. His way lay through the unsettled forest for several miles. In the centre of this lay one of the way-side taverns, and was the only house in the forest. He arrived about midnight, rested, and took some refreshment, and in a couple of hours proceeded on his journey. The next day the tavern-keeper was attacked, and soon after his wife, and both of them died.

The history of such cases as the foregoing, together with the following, which I read in a Maine local paper in the early part of 1833, first awakened my suspicions to the possible contagion of Asiatic Cholera, and the reading of Kennedy's unanswerable work on the "Contagious Cholera" confirmed my impressions, that non-contagion was a dangerous and fatal doctrine.

A sailor belonging to the State of Maine died of Asiatic Cholera in 1832, in a northern European port in the Baltic, where Cholera prevailed. A chest containing his clothing and personal effects were sent home to his relations, who lived in a small straggling village on the Atlantic Coast, near Bangor, Me. It arrived about Christmas, 1832, and was opened immediately on its arrival. The inmates all remarked a peculiar heavy odour in opening the chest, and soon after began one by one to sicken, when the whole were within a few hours hurried into eternity by a disease resembling Asiatic Cholera in all its malignity. There had been no Cholera in the State previous to this, nor was there any after, until 1834. This last case is attested by Professor R. D. Mussey, late of Boston and now of Cincinnati, Ohio.

Let us now return to the steamer Voyageur.

A man who landed from her, died on the wharf on Saturday night, the ninth of June, in Montreal. Several other cases took place on the wharf, as well as in lodging-houses in the neighbourhood, and the disease spread rapidly.

Of the passengers who returned and remained in Quebec on Thursday night, a number went to the lodging-house of one Roche, in Champlain

street, where cholera broke out violently. Fifty-six persons died of Cholera, in this one house, during the season of 1832.

In most of the early cases of Cholera in Montreal, communication could be traced with Kerr and McKee, and the other passengers of the steamer "Voyageur." On the 11th, several new cases occurred, and a continued and regular increase took place until the 19th. From Montreal we next trace the disease West and South. It appeared at Lachine on the 11th, among emigrants from Montreal on their way to Upper Canada, on the 13th it reached the Cascades in the person of a clergyman from Montreal, who died at Côteau du Lac. On the same day a boatman direct from Montreal died of Cholera at Cornwall. On the 16th, it reached Prescott, the first case being from Montreal. On the 18th, a boatman from Montreal died at Brockville. On the 20th, it arrived at Kingston. On the 21st, the first case occurred at little York, now Toronto. The victim was a merchant tailor from Montreal, who had fled from fear of the pestilence. On the 22nd a vessel from Kingston, called the "Massasunga Chief," with 200 emigrants on board, arrived off the town of Niagara, but having Cholera on board, was not allowed to come into port, and Cholera did not get then into Niagara. Here we have conclusive evidence of propagation by man in exact proportion to the rapidity of his movements.

I will mention another fact of an official character connected with the garrison, which tells against the epidemic or atmospheric theory: "The garrison of Montreal, consisted of a portion of two regiments, in all 450 men, of whom 46 died of cholera previous to the 19th of June. Under orders from the Army Medical Department, the men were then removed and encamped under canvas on the Island of St. Helen's, near and opposite to the city. Strict non-intercourse was maintained, and the result was, that no more deaths occurred, although the city was decimated, and steamers were daily passing close to the Island, freighted with the sick and dying." In the same category, I may mention, "on official authority," that under an order of the Government, the Troops at Newcastle Sunderland, Edinburgh, and Glasgow, where cholera prevailed in 1831 and 1832, were confined to their quarters, and no communication or intercourse permitted with the citizens, and not a case of cholera occurred among them.

The disease then spread in a westerly direction along the course of the St. Lawrence to Montreal, but did not appear at *any of the intermediate places where no landing or communication had taken place;* thence to Upper Canada, and to the North-Western States of the American Union; and at the same time by Lake Champlain in a southerly direction to New York, Philadelphia, and other cities in the United States, till it reached New Orleans in the extreme South.

Having said that it did not attack any intermediate place between Quebec and Montreal where no passengers were permitted to land, I may mention that whilst these two cities were decimated the town of Three Rivers, equi-distant from each city, where steamers touched both ways daily, escaped entirely in 1832, by establishing a system of non-intercourse, and not allowing a solitary passenger to land there till the disease had subsided. Can this extraordinary exemption from the pestilence be accounted for excepting on the principle of contagion.

To be continued.

A case of Phlegmonoid Erysipelas commencing in the structures surrounding the Cæcum. By G. A. McCallum, M.D., Dunville, Ont.

J. H., æt. 60, the subject of the following rather interesting case, had been for some time previous to his present illness in a debilitated condition from frequent attacks of diarrhœa. April 5th, he was seized with severe rigours, followed by acute pain in the abdomen. He had throughout the day been complaining of some pain through the bowels, and on going to bed at night the chills commenced. After they had subsided, the pain was exceedingly severe; medical aid was obtained; and under the use of opiates and fomentations, the patient became easy; he was then ordered a purgative of turpentine and castor oil; however, as soon as the effect of the opiates wore off, the pain was still complained of, but more particularly now in the right inguinal region. His attendant next put him on a course of mercury, with counter-irritation over the tenderness, and this treatment was continued without benefit until the 19th, when I was called to see the case. It then presented the following symptoms—the patient was very weak and emaciated; pulse 100, small and feeble; complete loss of appetite; tongue coated; bowels constipated, and the urine high coloured; in the right inguinal region there was considerable tympanitis, which did not extend over the rest of the abdomen, it appeared as if the cæcum was distended with flatus; under deep pressure in the lower part of this region there was a good deal of tenderness, but I could discover nothing like a tumour; the pain was very much increased by moving the leg. Ptyalism had by this time been produced without any alleviation of the symptoms, and there was every indication for an opposite line of treatment; accordingly the calomel and opium were discontinued, and tinct. ferri chloridi and quinine with brandy, and beef tea, ordered in their stead, flax-seed poultices to be applied over the pain, and to have ¼ gr. doses of morph. sulph. to procure rest.

24th.—Not much improved, although he finds the poultices grateful, and is enabled to sleep pretty well at night under the influence of the morphia; has a sallow, anxious countenance; pulse still about 100, but very small; tongue coated with dark fur; and the lips and teeth with sordes; there is not much swelling in the right inguinal region, not so tympanitic, but the tenderness is about the same. On examining more closely, a resonant point was found below Poupart's ligament, apparently in the femoral canal; this was very tender under pressure and when he coughed. Treatment continued.

25th.—General symptoms about the same; the resonant tumour below Poupart's ligament, yesterday and last night, was very painful; had a poor night's rest; however, towards morning he became easier, and on taking off the poultice in the morning it was discovered that the whole thigh, but especially the anterior surface, was very much swollen; œdematous *tympanitic*, and except a space in Scarpa's triangle, which was very tender, the sensation was almost *nil*. Not so much tenderness above Poupart's ligament now. Treatment continued; ordered castor oil to move his bowels, as he had not had a passage since the 19th.

28th.—To-day my friend Dr. Mulvaney of H. M. gun-boat Britomart, saw the patient with me. We found him exceedingly low; the oil had not operated until yesterday, emptying the bowels of a quantity of dark scybalæ, but as its action seemed inclined to continue and causing a great deal of pain in the region of the cæcum, the diarrhœa and pain were allayed by opiates. The thigh now showed a blush of redness on its anterior surface, was quite tympanitic and very tender over a large extent of surface. At one point, about the junction of the upper with the middle third, it appeared somewhat boggy. A free incision was accordingly made into it; nothing but a sanious fluid escaped. Treatment continued, except that the brandy was increased from ℥ iv to ℥ vi per diem.

29th. Diarrhœa commenced again, and motions passed involuntarily, and to-day hectic symptoms have set in; a little pus was discovered about the edges of the wound, and small bubbles of fetid gas were escaping; this accounts for the resonant tumour below Poupart's ligament and the resonance over the thigh. A point higher up and more external, was found to be boggy, made an incision into it with the same result as before. Treatment continued.

30th. Pulse to-day very small and frequent, sweating profusely, &c., the opening in the thigh discharging freely a thin, dark-colored, very fetid pus, bubbles of gas with an odour feculent in character, are constantly escaping from the wounds and rendering the atmosphere of the

room almost intolerable. Still continued the iron and quinine with plenty of brandy and beef tea, and ordered a solution of permanganate of potash, as a disinfectant, to be sprinkled on the poultices and about the bed.

May 1st. To-day the whole integument on the anterior part of the thigh, from a short distance below Poupart's ligament to near the knee, seems to be loose from the muscles beneath, and when pressure is made above Poupart's ligament the flow of pus and escape of gas is very much increased, showing a communication; his pulse is rather better, as is also his general appearance; large portions of cellular tissue are sloughing away; the tenderness above Poupart's ligament has almost disappeared; made another incision at the most dependent part of the diseased tissues, and allowed of the free exit of a quantity of pus which had collected there. Treatment continued.

3rd.—Much better to-day; looks brighter and feels stronger; hectic symptoms diminishing; discharge from the incisions very profuse, but less fetid and more healthy in character. Continued treatment, and bandaged from the toes up to the incisions.

5th.—Still improving; hectic symptoms nearly gone; appetite middling, and strength improving generally. Treatment continued.

9th.—Able to sit up in bed; considerable discharge from the cuts yet, but the pus is laudable; has no pain anywhere, and is on a fair way to recovery.

This case presents many interesting points, the disease occurring, as it did, in a person debilitated by previous frequent attacks of diarrhœa was necessarily of an asthenic character from the beginning, and owing to its seat and peculiar course, renders the recovery rather remarkable.

It was evidently not a peritoneal abscess, from the fact that it had no tendency to point anywhere; the unhealthy inflammation, where we could see its action, as in the thigh, was inclined to spread rapidly, and large masses of dead cellular tissue were thrown off through the free incisions made. I am confident that it is to the free incisions and strong supporting treatment that the patient owes his life.

Case of Acute Rheumatism.—Pericarditis—Effusion on the Brain and death by Coma. Under the care of Geo. E. FENWICK, M.D., Prof. of Clinical Surgery, McGill University. Reported by Geo. Ross, A.M, M.D., House Surgeon, Montreal General Hospital.

Martha L., aged 26, was admitted into the Montreal General Hospital on the 26th of March, 1868, suffering from acute rheumatism.

The joints principally affected on admission were the ankles and the left knee. She has had one previous attack of the disease but was not aware of her heart having been then affected. There was to be heard at the base of the heart and for some distance along the course of the aorta, a soft, blowing systolic murmur; the action of the heart was quite regular and not at all excited, and there was no precordial pain; it was, therefore, thought that the murmur had probably existed prior to this attack.

She was immediately ordered the following treatment:

Haustus Niger, horâ somni sumend. and R. Potass Bicarbon ʒ vj. Aquæ ℥ xij. capt. coch: mag. duo secundâ quâque hora, together with extra beef-tea and milk. The joints to be swathed in cotton wadding and oil-silk. Under this management the case prospered favourably until the 1st of April, when the pulse rose somewhat in frequency and there was some slight pain and oppression in the precordia complained of. The pain and swelling had now almost completely left the lower extremities and had shifted to the elbows and wrists. On this day auscultation discovered an indistinct friction sound over the heart, but it could not be found to exist with the second sound, though sufficiently recognizable with the first.

April 2nd. Not so well to-day—pulse 110, tongue somewhat dry; complains of thirst; friction sound of the same character, but more distinct. No other joints affected, but the swelling remains unaltered in those previously inflamed. Omit the former medicine R. Acid Nitro-muriatici dil ʒ iv. Aquæ ℥ vi. capt. coch. mag. quartâ quâque horâ, and also R. Hyd. Submur. gr. ij. Pulv opii gr. ¼ quartâ quâque horâ—to be taken alternately.

April 3rd.—At noon to-day, the hour of visit, the patient expressed herself as feeling much better; had slept well and the expression of distress had quite disappeared from the face; tongue somewhat moister but slightly furred, and thirst less—pulse about 100—friction sound as yesterday. About 2 p.m., I was summoned by the nurse of the ward to see her, as she seemed suddenly much worse, and was said to be not quite "right in her head." On enquiry I found that for some time after the visit she had apparently dozed quietly, when she awoke and began talking incoherently and counting imaginary numbers on the wall. She appeared, however, when I saw her, rational enough, and answered me that she felt considerable pain in the chest. The pulse was now 145 and the respiration very rapid; on putting my stethoscope to the heart I found the heart's action very much increased in force, and the friction murmur exceedingly intensified. I ordered a large mustard plaster over the heart. At 3.30 p.m., saw her again with Dr. Drake, the house surgeon. She had now lapsed rapidly into a state of insensibility, from which she could only be partially aroused by a loud question or rough shaking, but could answer nothing—the pupils were strongly contracted and insensible to light; pulse 100; skin perspiring freely, and warm; respirations slower but no stertor; no vomiting and no especial heat of head. A messenger was at once despatched for Dr. Fenwick, but before his arrival, viz., at 5 p.m., she expired. The condition of profound coma remained until the end. When the insensibility was first noticed with the contracted pupil, narcotism was suspected, but on investigating we found that since 5 p.m. the evening before, she had taken but *four* of the powders, which would amount to only two grains of opium in all, in 22 hours, which rendered that suspicion nugatory.

Autopsy. Twenty hours after death. The *Brain* was first examined. The sinuses of the *dura mater* were found filled with exceedingly dark but tolerably fluid blood; the cerebrum itself, however, presented no especial marks of congestion. In the sac of the arachnoid was a considerable effusion of serum. The vessels of the *pia mater* were all intensely congested. The ventricles of the brain were found filled with clear transparent serum, in which floated the large and over distended choroid plexuses: the *amount* of serum could not have been less then *two ounces*. The base of the brain appeared healthy.

On opening the chest there were found to exist extensive and tolerably recent adhesions between the pericardium and the pleuræ of both lungs. The opposed surfaces of the pericardium were strongly adherent at the apex of the heart (apparently from previous inflammation), and more loosely over the auricles and base of the heart generally, where we found evidences of recently effused lymph. The membrane itself presented a most vivid congestion in patches with spots of apparent ecchymosis. The valves, both mitral and aortic, were found healthy, but the lining membrane of the aorta for a distance of two inches from the valves, was found extensively affected by atheromatous degeneration, roughened and worn away in patches, thus accounting for the basic murmur heard, without any disease of the semilunar valves.

The lungs and kidneys were healthy.

The foregoing case has been thought worthy of record, owing to the rarity of an organic lesion of the brain occurring during the course of acute rheumatism, also the suddeness with which alarming head symptoms developed themselves, and the rapidity with which they ended fatally.

Montreal General Hospital, 1st June, 1868.

A Case of Obstruction of the Bowels overcome by Electro-Magnetism.

By F. H. BRATHWAITE, M.D., of Prince Albert, Ontario Co., Ontario.

H. G——, shoemaker by trade, sent for me on the 5th inst. I found him complaining of much pain in the bowels, with bilious vomiting. He is of an eminently bilious habit, and is habitually costive. He suffered at times from bilious colic, and has been ruptured from boyhood,—the hernia has always been irreducible.

I at first endeavoured to allay the irritability of the stomach by chloroform and small doses of submurias with morphia, and by applying mustard sinapisms externally. As soon as the stomach would admit of a cathartic, I gave of submurias grs. x and jalapine grs. v, (at this time I did not suspect any obstruction, as his bowels had moved the previous morning, slightly.) This was retained for over five hours. Emesis again set in; no evacuations followed from the bowels. *Wednesday*, I gave four pil. cathartic æ co., retained seven hours, no evacuation. *Thursday morning*, I now gave four croton oil pills, one drop in each, one every hour. I waited two hours after they were finished and repeated the ol. tiglii in stronger doses.—No use. I had been continually employing fomentations of turpentine, hot water, vinegar, &c., poultices of bran, bread and mustard. I had been administering copious stimulating injections, with no result. *Thursday afternoon*—Inflammation of bowels evidently setting in, chills, great pain over bowels, pulse 110—120; full and hard; tongue furred. I requested a consultation. My friend, Dr. Ware, saw the case with me. We agreed that cathartics had been pushed far enough. Sedatives with large alterative doses of calomel were now given every three hours. A mercurial impression was desired. A large fly blister was laid on the bowels; at this time the vomiting, although not so frequent, was decidedly stercoraceous. This treatment, with injection, was kept up until the Monday following. *On Friday, 8th*, we noticed a tumour (fœcal) to the right of the umbilicus, which disappeared in a couple of days, and was evidently pressing against the bladder, as the patient was incessantly making water. I tried to find it *per anum*, but could not.

Tuesday 13th.—He was sinking fast; appearance cadaverous; pulse 110—120; weak and fluttering; intense thirst; bowels bloated; old rupture becoming much distended and painful, (the hernia had not hitherto caused any inconvenience.) The stercoraceous vomiting was still taking place at longer intervals. Cold clammy sweats would now and then break out; in short, the man was dying. My friends Drs. Ware and Warren were, with myself, in despair. A good, honest man was passing away before our eyes, and we were powerless for good. We thought of liquid mercury, but it frightened us. We thought of cold dashes of water on the bowels, but that was worse. We had done every thing; cathartics and stimulating injections had failed, sedatives and blisters had subdued the peritonitis, but that was not sufficient to save the man. Alteratives of mercury did not produce any appreciable results. All had failed. I would have bled the man at the outset, but he wanted more rather than less blood. As a *dernier resort* we applied electricity, a powerful electro-magnetic battery was employed, (one of Kidder's), one pole kept secure at the anus, and the other first moved up and down the spine, then passed slowly over the several parts of the colon. How the bowels twisted and tortured themselves! How they contracted into knots and relaxed again! The full strength of the battery was employed for nearly an hour. *And before the galvanic influence could pass away, we poured into him about an ounce of castor oil.* The man slept, and awoke to evacuate his bowels. Six or seven fearfully offensive motions followed, and the man is now (May 16) quite convalescent.

I have taken the liberty of recording this case, as an instance of life saved when death seemed inevitable, and as a lesson to my medical brethren never to give up *such cases* until the man is dead. I look upon this case as one of pure obstruction from fœcal impaction, (the accumulation perhaps of months), complicated with hernia. I am satisfied that the rupture had nothing to do with bringing about the obstruction; it threatened, however, at last to wind up the case summarily. Galvanism, I am aware, is recommended as a means of overcoming such obstructions, but it has generally failed. Could it have been because a cathartic was not given after the galvanism, or could it have been because the cathartic, if given, was given after the active effects of the battery had passed off?

REVIEWS AND NOTICES OF BOOKS.

Epidemic Meningitis or Cerebro-Spinal Meningitis. By ALFRED STILLE, M.D., Professor of the Theory and Practice of Medicine in the University of Pennsylvania. Philadelphia: Lindsay & Blakiston. Montreal; Dawson Bros.

This is an exceedingly well written volume of nearly 200 pages upon a disease which, within the last few years, has been attracting considerable attention both on this continent and in Europe. Fortunately, Canada

has, in a measure, been exempt from it; for although, we believe, isolated cases have occurred in various portions of the Province, so far as we are aware, we do not know of its having in any portion assumed an epidemic form. Dr. Stille's opportunities of studying the disease have been very extensive, several hundred cases having come under his observation while attending the Philadelphia Hospital. In giving its history, he confines his attention to the various epidemics which have occurred since the beginning of the present century. He says, "Its outbreaks have occurred almost simultaneously in regions as widely separated as Europe is from America, and annually it has made mid-winter attacks upon towns and rural districts, the salubrious and unhealthy alike, completing the cycle of its progress in a period varying from ten to fifteen years. Three such periods have occurred during the present century. The first of eleven years began in 1805, and terminated in 1816, the second of thirteen years, occurred between 1837 and 1850, and the third extended from 1856 to the present time. These two conditions, of simultaneous appearance in widely remote places, and of annual recurrence for a series of years, characterize no other disease whatever." The symptoms are given with much care, and a careful study of them would tend to prevent the numerous erroneous diagnoses which, we believe, have everywhere been made in connection with this disease. We have not the space to copy them at full, for they are very lengthy, but we give one or two extracts. After detailing the symptoms which usher in the attack, and which are in a great measure similar to those of ordinary meningitis, he says, "These phenomena more or less gradually assume a graver aspect, or usher in a heavy chill, which, in its turn, is followed by alarming symptoms, and especially by excruciating pain in the head, a livid or pale and sunken countenance, and extreme restlessness. The pulse is as often slow as frequent, and the skin but little, if at all, warmer than natural. The vague pains which opened the attack are now concentrated, and seem to dart in every direction from the spine, which is also, especially at its upper part, the seat of severe aching; and, in a large proportion of the cases, its muscles become more or less rigidly contracted, so that the head is drawn backwards, or the whole trunk is arched as in tetanus. * * In many cases eruptions appear upon the skin. During some epidemics the only one observed is herpes labialis; in others the eruption resembles roseola, measles, or the mulberry rash of typhus, or, from the first, it consists of petechiæ, vibices or extensive ecchymoses." Headache, Dr. Stille says, is a very constant symptom, and is usually felt in the forehead between the eyes. It varies in intensity in different epidemics, and is often relieved by dry cupping or blisters on the nape of the neck. He

alludes to two symptoms, which he states are thoroughly characteristic of the disease. The first is cutaneous sensibility, due to hyperæsthesia of the skin. The second is pain in the spine and limbs, and is almost uniformly present. The various modes of treatment are discoursed at good length; but we do not gather that any particular method has been more effectual than another. The little volume will well repay perusal.

Obstetric Clinic; a Practical Contribution to the Study of Obstetrics and the Diseases of Women and Children. By George T. Elliot, Jr., A. M., M.D., Professor of Obstetrics &c., in the Bellevue Hospital Medical College. New York: D. Appleton & Co. Montreal: Dawson Bros., 1868.

That Dr. Elliot is a close student, and a man of thoroughly practical ideas, the work from his pen, the title page of which we give above, proves beyond question. For fourteen years he has been connected with the obstetric department of the Bellevue Hospital, and during that time he kept full and accurate notes of every case of any clinical interest which was observed within its walls, and from these he has deduced a few facts and theories having a direct application to obstetric practice. Each fact or theory has a separate chapter devoted to its consideration, to which are appended the most interesting cases in illustration and the clinical remarks which were made on the cases reported. Perhaps the most interesting as well as the most important chapter in the entire volume is the first one, which is on the relations of albumin-urea to pregnancy, a condition so full of danger to the pregnant female. It is the custom in Bellevue to examine the urine of all cases admitted to the obstetric wards, and Dr. Elliot gives the results of four hundred and thirty-seven examinations, and in twenty-three albumen was present. In three of these cases, however, it was distinctly proved that the albumen present depended upon the admixture of pus. Deducting these, it gives us a ratio of one in nearly twenty-three cases. In making this calculation it is to be borne in mind that most of the women whose urine was examined were pregnant for the first time, and these especially liable to complications. Dr. Elliot urges early and continued examination of the urine, so as to detect the first trace of albumen. Its presence in this condition involves all the dangers associated with its appearance in other states of the system, but it entails a singular power, especially in some constitutions to that much dreaded complication of the puerpural state, convulsions, and also to mania. In summing up this chapter he hopes that experience and study will ere long enable us greatly to diminish the dangers which are associated with this condition of the kidneys.

Chapter iii. is on chloroform, and venesection in Puerpural Eclampsia, and consists almost entirely of cases, some thirty-three of them being reported. We have read a number of them, and they shew conclusively the almost wonderful controlling power of chloroform over this terrible complication. He says, "if only one method of treatment was given to me for these cases, my choice would unhesitatingly be for chloroform." He alludes to the fondness many New York Physicians have for Sulphuric Ether, but all things taken into consideration, he believes that chloroform is the most prompt and certain means we possess, of arresting and controlling the convulsions. Puerpural Mania occupies a brief chapter; he writes hopefully of the majority of cases, and recommends sending them to an asylum to be the last resort of the physician.

Chapter xvii is an article on "Kyesteine in the urine, as an indication of pregnancy" which was published some nine years ago in the *New York Journal of Medicine*. The experiments were made by Dr. Elliot and Dr. Van Arsdale. They examined the urine of one hundred and sixty pregnant women, and kept a tabulated statement of over one hundred and fifty-three. After careful examination of this large number of cases they came to the conclusion that they saw nothing conclusive as to recognize peculiarities in the urine of pregnancy." We think that there is nothing positive in its (Kyesteine) indications, and that its appearances can scarcely even be called "corroborative." In his preface, Dr. Elliot says that since he first published this paper, further experience has confirmed him in the opinion expressed.

We have had much pleasure in looking over this work, and are fully convinced it contains a mass of valuable information. We cannot avoid saying, however, that we do not admire Dr. Elliot's style of writing; we think many of his sentences a little ambiguous and difficult to understand. With this exception, which we hope to see improved in future editions, we have been much pleased with it. Messrs Appleton & Co. have printed it upon paper of very superior quality.

PERISCOPIC DEPARTMENT.

Surgery.

THE TREATMENT OF VENEREAL DISEASE.

By Dr. G. H. B. Macleod.

The treatment of venereal disease divides itself into the local and constitutional. The former is of course alone required in the case of the soft chancre, but both are necessary in true syphilis. It may, however, be here said that a mere excoriation demands the simplest possible application. Water dressing, or a very weak stimulant, or astringent solution applied on a thin teased-out flake of carded cotton; or what often succeeds better, a little chalk powder, or starch, dusted over the breach of surface, combined with attention to the bowels and the avoidance of wine, will probably suffice. In dressing this, and all such sores, the greatest gentleness should be enjoined. The old dressing should be removed by a stream of lukewarm water, and then the new application put on without any "scrubbing" of the part, and with as little irritation as possible. If the foreskin is returned over the dressing, the thinner the layer of lint or cotton put under it the better, and the patient should be forbidden to examine, as he is apt to do in his anxiety, the part during the intervals between the dressings, as the irritation thus caused is most pernicious. Twice a day is as often as the dressings should be renewed in any case.

"Herpes præputialis," as it may depend upon want of cleanliness, contact with leucorrhœal or other irritating discharges, stricture of the urethra, deranged bowels, and especially the acidity arising from excesses at table, or from the dyspepsia which attends gout, will, for its cure, demand the removal of whichever of these causes may be present. Locally the same applications may be used as are employed in excoriation, especially dry absorbent powders. Caustic is most injurious, and should never be applied. Lime water with opium forms a good wash, or some port wine, having water and tannin added, according to the requirements of the sore.

If a chancre is seen early it should be thoroughly and completely destroyed. This rule holds good as regards both sores. We destroy the virus of the non-infecting chancre in order to prevent local complications, to avoid it spreading or becoming phagedenic, to prevent the bubo of absorption, to hinder it from multiplying itself on the patient or being communicated to others. We destroy the hard chancre, *not* from any hope we can entertain thereby to prevent constitutional infection, because by the time we are able positively to say "this is a hard chancre," *the system is already infected*, but by adequately appplying caustic we convert the infecting chancre into a simple suppurating sore, we prevent its communication to others, we remove any depot of virus which may remain in the hard base to feed the disease in the system, we give the patient's anxiety a certain relief and inspire him with some confidence, and we also guard ourselves from the imputation of having omitted such application when afterward constitutional symptoms appear. It is probably true

that if the poison of the hard sore is inoculated on a breach of surface that the specific chancre may so quickly follow that we may be able to destroy the virus, while it is yet local, but it is the rare exception that the sore is thus seen by the surgeon. In the great majority of cases when the chancre comes under observation the time has gone past for any effectual action being taken to prevent the entrance of the poison into the economy, and if we have no other aim than to obviate *that* in applying escharotics to the sore, we would far better omit this painful step altogether. If we use means to destroy the chancre before the hard base, swelling glands, and other evidence of its infecting character, appear, we can have no possible grounds on which to say that we have had a true chancre to deal with at all, or that we have, by our caustic, prevented constitutional empoisonment; but, on the contrary, there are four chances to one that it is a sore which would never have been followed by any such evil results; whereas, if we wait till such evidence is supplied, then all hope of achieving the end in view is gone, as such indications as we can rely upon as demonstrating the nature of the sore are themselves evidence of the constitutional implication. This is the great obstacle to a decisive opinion regarding the effects of preventive treatment of syphilis. Men unconsciously deceive themselves. Caustic is applied, and no constitutional disease appears, then they conclude that it has been prevented by the local application; if, on the contrary, the constitution is invaded, then they think the escharotic was too late in being used, or it was not effectually applied. We are apt to forget how much more common the simple sore is than the infecting, and how many non-venereal lesions are aggravated into formidable and confusing ulcers by the applications which the patient, in his fear and anxiety, so often uses himself before he comes to us. Thus, then, we conclude that all suspicious sores should be effectually destroyed at as early a period as possible.

There are many caustics in use for the destruction of the chancre. Some of these are too deliquescent, others too weak, others too painful and slow in their action. The hot coal, or "dottle" from a tobacco pipe which are occasionally used by the vulgar, are more to be relied on than many of the escharotics employed by surgeons. I have experimentally tried all the caustics which have been recomended, and very much prefer strong nitric acid, or the acid nitrate of mercury to them all. I generally employ the former. It is very manageable, penetrating, and rapid. The sore must be well exposed, wiped dry, and a large drop of the acid put upon its centre by means of a spun-glass brush, or a bit of wood, and then the edges and whole surface rapidly destroyed. The patient should be placed with his back against the

wall, so as to prevent his withdrawing his person when he feels the sharp sting of the acid. Plunging the part into water or pouring a stream over the sore, quickly arrests further destruction, and allays the pain. Such simple means succeed in neutralizing the acid just as well as an alkaline solution. The spray apparatus should not be used to diminish the pain, as it hardens the tissues, and so prevents the caustic penetrating, and it renders the surface of the sore wet, and so neutralizes the acid. Caustic soda and potash; sulphuric, hydrochloric, acetic, and chromic acids; chloride of zinc; Velpeau's paste (sulphuric acid and saffron); alum; sulphate of zinc or copper; the hot iron; Ricord's application of animal charcoal six parts and sulphuric acid two parts, and many other escharotics which have been used, are in my opinion inferior to strong nitric acid and the acid nitrate of mercury, for the reasons before given. It requires no prolonged and painful applications, like chloride of zinc, or Velpeau's and Ricord's paste; it penetrates as no alum, or sulphate of zinc or copper, or chromic or acetic acids can do; it is not so formidable or painful as the actual cautery, and is not too deliquescent like caustic potash. Excision terrifies the patient, and is not effectual, as the records of the Dreadnought sufficiently prove, as not only is it very difficult to excise the whole sore, but the edges of the large wound are very apt to become inoculated, and thus matters are made worse than ever. Of all the local applications the most useless, in my opinion, is nitrate of silver. It is not powerful enough to destroy the sore effectually, and so fails in fulfilling its most important requirement; and it causes much irritation (especially if repeatedly used) and inflammation in the sore, gives a pseudo-hardness to the base, excites the lymphatics, and altogether does much mischief. It introduces features into the case which do not legitimately belong to it, and so occasions confusion, while, as has been said, it gives rise to unnecessary complications. No caustic should be applied to a chancre so long as it is inflamed. By the use of water, or some soothing application, or possibly by the aid of a leech and fomentations, or steaming, the excited action should be subdued before the escharotic is employed. One thorough application should suffice. The repeated use of a caustic does infinite harm. If it has once been properly applied, it should not again be required, unless phagedena set in. After the uses of the caustic a poultice, or, what is quite sufficient in most cases, warm water dressing, should be employed to cause the slough to separate, and then we have a simple ulcer to deal with, the applications to which will, like any other non-venereal sore, depend on its requirements at the moment. If it is slow to heal, or fails to heal from excess or defect of action, it will demand soothing or stimulant dressings; but if

it presents, as is usual, the characters of a healing granulation, then the simpler the applications the better. Water alone, or medicated by the addition of a small amount of metallic salt, or an astringent, or a solution of opium, will in very many cases suffice. As in the management of other ulcers, we may have occasion to change the remedies, as a "tolerance" is attained in the effect of any particular application; and it should always be remembered that black-wash is not a fit dressing, unless the ulcer requires a stimulant, and in that case it is not so cleanly as a weak solution of sulphate of zinc. Many would seem, by their unfailing use of black or yellow wash, to imply a belief in some specific effect of a mercurial on a venereal sore. So far from such a remedy being good for all chancres, it is supposed by not a few greatly to predispose the soft sore to phagedenism. Black or yellow wash are only useful when a metallic stimulant is demanded, and then, too, we may use the mercury in the form of vapour, if we please, though it has no special advantages, and is somewhat troublesome. Let not the ulcer be "oppressed" by remedies, but use the simplest dressing which will fulfil its wants. It matters comparatively little what wash is used, so long as it fulfils the requirements of the sore as to stimulation, soothing, etc. If its wants are properly recognized, there are plenty of agents which we can use. No stimulant application is, as a rule, better than Hey's red wash, Ricord's aromatic wine, or a solution of tartrated iron, or a weak solution of the muriated tincture of iron; nor does any astringent answer better than tannin and glycerine; nor is any soothing dressing superior to a watery solution of opium. All ointments are bad, as being apt to become rancid and poison the sore. After the destruction of the virus, the local management of soft and hard chancres is the same in all respects. They are both by the caustic reduced to the condition of simple granulating sores, and are to be treated accordingly. The hard chancre often heals up very quickly.

The bubo which may attend the soft chancre should be treated early, and with the aim of preventing suppuration. If it arise from the absorption of virus from the ulcer, our measures will fail to effect that end, as suppuration is sure to result; but we may by judicious measures diminish the area of inflammation and limit the amount of suppuration. If, on the other hand, the bubo be a simple adenitis, then, by the employment of the antiphlogistic treatment, we may, in most cases, prevent suppuration altogether. Rest is especially necessary, together with fomentations, preceded, it may be, by the application of leeches, and the administration of salines. Low diet should also be enjoined. Bubo is most frequently due to the repeated irritation of the ulcer by caustic or other applications, and our measures for preventing suppuration are not unfrequently thwarted by the irritable or scrofulous disposition of the patient. An opiate suppository at night is highly useful, by giving rest and preventing erections, by which the ulcer is irritated and the glandular complication augmented. When it is evident that pus is going to form, we apply poultices, and open the abscess freely parallel to Poupart's ligament. Small incisions are to be reprobated, and "multiple punctures" are worse than useless. A poultice will be employed till the pus is removed, and then the cavity of the abscess managed like a hollow ulcer. If the edges of the wound get inoculated by the pus, then we must apply caustic to them as to the primary sore; and if the skin get thin and undermined and so diseased as to be incapable of recovery, then we must remove or destroy it. If a gland project from the cavity of the abscess and prevent its closure, then we must excise it, or destroy it by caustic. Sinuses must be split up, and dressed so as to granulate from the bottom. The knife should always be preferred to caustic for opening buboes, and with the aid of the spray apparatus, we can accomplish it without pain. It is possible that the use of caustic may render the inoculation of the edges of the opening less likely to occur, but its slowness and painfulness more than counterbalance such advantages.

If the tendency to suppuration is checked, and enlargement and hardness in the gland alone remains, then the counter irritation should be employed. Blisters, or iodine, or a stimulant embrocation, may assist the removal of such deposits, but there is always great risk by their premature or inappropriate use of reviving the inflammation or inducing suppuration.

The hard rolling glands which accompany the infecting chancre demand no local application. They should be simply guarded from all irritation. Malpaquet's plan of using finely powdered bichloride of mercury, and pressure after vesication, is seldom of much use, and is no compensation for the possible harm resulting from the irritation it causes, and the laying up which it necessitates. So, too, all kinds of counter-irritation are apt to be injurious. The hard glands are here part of the constitutional affection, and are only legitimately amenable to the remedies against such constitutional disease, with the other traces of which they will disappear. If from any cause an abscess should form in the groin in connection with the hard chancre, then it must be treated like any other abscess in the same part.

Phagedena may affect either chancre, but it is infinitely more common in the soft than in the hard, and is in it much more destructive. It may prove a most terrible affair in broken-down intemperate persons, and demands most energetic treatment. The patient should be isolated, and

the most perfect cleanliness and ventilation enforced. If mercury was being administered it must be stopped, and that food which is most nourishing and easiest of digestion given, with a liberal allowance of wine, unless the patient be plethoric (which he very rarely is) and there is evidence of acute inflammation round the sore. Phagedena is usually a sign of depression and feebleness, and is to be managed accordingly. If, however, there is acute inflammation present, then it must be subdued in the ordinary way. The sore must be freely cauterized with strong nitric acid, and the application renewed as often as may be required to arrest the disease. When, by means of carrot or linseed meal poultices, combined with disinfectants, the eschar is separated, a strong solution of potassio-tartrate of iron (30 grains to the ounce) forms the best dressing, and a drachm of the same salt should be administered in the 24 hours internally, with a full opiate at night. It is some years now since we have had any bad cases of spreading sore to deal with in the Lock Hospital, and this I attribute mainly to the early and free use of the salt of iron just mentioned.

As regards warts and vegetations of a syphilitic origin, they should be removed with scissors, and the part from which they sprung touched with caustic or perchloride of iron. There is not the least fear of excessive hæmorrhage, and the spray apparatus will greatly diminish the pain. Warts can be very effectually destroyed by touching them with strong nitric, chromic, or acetic acid daily, till they are completely killed, and then removing them with a spatula or the nail. In the female, syphilitic vegetations occasionally attain an enormous size, hanging down in dendritic irritable masses, which exude a most nauseous discharge.—*Glasgow Medical Journal.*

DISLOCATION OF THE SHOULDER OF NINETY-TWO DAYS DURATION, REDUCED BY MANIPULATION.

By J. M. Boissot, M.D., of Philadelphia.

On September 10th, 1867, Mr. B., æt. 30, came into my office with an injury of his right shoulder. He had fallen upon his hands, and the efforts made to prevent this, added to his natural weight of 180 lbs., served to produce the condition herein described.

It is necessary to state here that the date of this visit was *thirteen weeks* subsequent to the receipt of the primary injury; he had consulted a physician, obeyed his instructions for the treatment of what he called, "*a bad sprain with a fracture of one of the little bones,*" and had even attempted to use the arm when some of the pain had subsided in order "to get rid of the stiffness."

My examination of the case showed a difference in the shape of the two shoulders, the injured having lost the roundness, so prominent a feature in the natural condition, presenting instead a slight elevation in front and beneath the outer third of the clavicle; the motions of the arm very few, imperfect, and attended by pain.

I diagnosed this condition as luxation of the humerus forward, and told him my further examination and treatment would be attended to on the following day, when I could place him under the influence of ether.

Sept. 11th, 1867, my friends Dr. Wm. M. Turner, and Dr. Thomas G. Morton, having examined the case, and agreeing with me as to its nature, gave their assistance in the work of reduction.

Ether was used, and upon total relaxation, the body was held firmly, and the arm so rotated by manipulation that the attachments formed were broken up, and the head of the humerus restored to the glenoid cavity, from which it had been absent just 92 days. The subsequent treatment consisted in keeping the elbow well in toward the middle line of the body, and supporting the head of the humerus in its natural position; for these purposes I applied the apparatus which I devised some time ago, for the treatment of fracture of the clavicle.

October 28th, 1867, he was discharged well, and with ability to use the arm freely and without pain; at this writing, March 17th, 1868, he has perfect use of the arm. This case is another plea for careful examination of patients who have received injuries, in order that a correct diagnosis may be made, without which, treatment becomes mere guess work; it shows that difficult and long standing dislocations of the shoulder joint may be reduced by manipulation; and lastly, as a matter of no slight importance, the necessity of proper after-treatment in the matter by rest and support, the appliances for which receiving as much attention in their adjustment, as those employed in cases of fracture.—*Philadelphia Medical and Surgical Reporter.*

CLINICAL REMARKS ON THE TREATMENT OF VARICOSE VEINS.

By Stephen Smith, M.D., Surgeon to Bellevue Hospital.

The treatment of varicose veins is palliative or radical. The palliative treatment is directed to the external support of the veins by means of such appliances as may be adjusted to the part, and will make equal pressure at all points. In the lower limb, a bandage properly applied will answer the purpose temporarily, but it is so easily displaced that it

serves no useful purpose where the patient leads an active life. The elastic stocking is a far better appliance, and is generally resorted to by those who can afford them.

The radical treatment aims at the obliteration of the vein, and hence, a permanent removal of the conditions on which the disease depends. In carrying out this treatment we must necessarily resort to operative measures, and no one of the various operations hitherto adopted has proved to be free from danger. Too frequently inflammation has occurred, and occasionally it has assumed a severe type and terminated fatally. The form of inflammation most dreaded was phlebitis, or inflammation of the vein itself. This disease was thought to be almost certainly induced when the vein was simply wounded, and but few surgeons had the hardihood to penetrate a vein in their operations. But inflammation also frequently occurred when the instruments employed were passed in the neighbourhood of the vein, or when excision of the vein was performed, and occasionally proved disastrous. These results have from time to time brought nearly every operation into more or less disrepute, and rendered surgeons timid about resorting to radical measures.

The obliteration of the vein by caustics has given more general satisfaction than any single method. And yet it is not free from severe if not dangerous consequences. We do not always sufficiently limit its local action, and it may then penetrate deeply and extend widely and do great harm. It is not, therefore, a remedy which can be placed in the hands of every practitioner with perfect safety.

Of the two methods of treatment, namely, the palliative and radical, the latter is infinitely preferable, provided our procedure is safe and effective. Those conditions I think have now been secured. The method to which I refer is the injection of the vein with persulphate of iron. The operation has been performed frequently in this hospital, and with the happiest results.

The attention of the profession of this city was first called to this method of treating varicose veins by Dr. Minor, of Brooklyn, in 1860. He reported five cases, in all of which the injection was successful, and in none were there unfavourable consequences.

It may seem strange that an operation which involves puncture of a vein should be attended with no severe inflammatory symptoms, when the older operation by transfixion was so frequently dangerous, and occasionally fatal. This is explained by the fact that in injections the vessel is itself medicated by the persulphate, which tends powerfully to arrest the inflammatory process.

It must be stated also in regard to the persulphate that it is a non-irritant to the internal membrane of the vein. However freely it is employed, the inflammation is still very inconsiderable, rarely amounting to more than a blush of redness, and slight swelling; and at the most giving but a small subcutaneous abscess, or, as in one case, a light erysipelas. You must remember that I speak now of the persulphate of iron. Some have mistaken, and have employed the perchloride, which, though powerfully hæmostatic, is nevertheless an irritant, and creates frequently considerable local inflammation.

The immediate effects which we obtain by injections of the persulphate are the same as those which we seek by other methods, namely, the formation of a clot. This clot is very firm, and at once perfectly occludes the vessel. It is much more firm and effective than those clots which form from external pressure, or other mechanical agencies.

The operation is very simple and can readily be performed by any one. A common subcutaneous syringe is first charged with the liquid persulphate, (Squibb's preparation); the patient takes the erect position so as to distend the veins of the leg; the needle of the syringe is then passed into the cavity of the vein, which is pressed by the finger, and five, ten, or fifteen drops injected. In a few minutes the clot is detected by external examination, and the needle withdrawn. The patient should remain in bed for several days, and cold applications be made to the puncture.

As a precautionary measure I always apply a compress and roller over the trunk of the vein on the cardiac side to prevent the possible escape of a coagulum from the mass into the general circulation. I usually inject the larger trunks, and generally inject at several points at one sitting.

In the treatment of varicose veins, therefore, you should, in my opinion, adopt radical measures. The time has passed when you should be satisfied with merely palliative treatment in a case which demands interference. Palliative measures, as the term indicates, are not curative; they leave the affected part in no better condition than when first employed; they are a constant source of annoyance, and to the poor a burdensome expense, which cannot long be endured.

In the method by injection of the persulphate we have a remedy which answers every indication, and may be regarded as entirely safe and efficient.—*Exchange.*

A NOTE ON CHRONIC URTICARIA.

Chronic urticaria is often a very troublesome affection, and a few hints regarding it, based on hospital experience, will be doubtless acceptable to our readers. Dr. Hillier, physician to the Skin Infirmary of this

hospital, considers the disease to be one requiring the utmost discrimination for its treatment. Occasionally a case will be found to depend on one article of diet, which it requires careful inquiries and observation to ascertain. One case was found to be caused by cheese, another by coffee, another by tea. In such cases the mere disuse of the offending article will sometimes cure the disease. When the patient is of a rheumatic tendency, alkaline medicines are of use. In very many cases colchicum is of great service: some of these are probably gouty in their nature. In many of them, however, it is not possible to find any indications of a gouty constitution. Dr. Hillier has seen great benefit from the use of quinine, especially when the attacks occur with marked periodicity. When there is no gastro-intestinal irritation, arsenic has been sometimes found useful. Unfortunately it is not always easy to decide what remedy shall first be tried. One case coming under Dr. Hillier's care from time to time is always cured by a few doses of cod liver oil. Dilute nitric acid has occasionally been found serviceable.

Of twenty-eight cases of which Dr. Hillier has notes, nine were either cured or received much benefit from the use of colchicum and alkalies. In four, quinine was given, of which three were cured, and of one the result was not known. In two cases nitric acid relieved the patients. Of three cases under alkaline treatment alone, two were cured, and in one the result was unknown. Arsenic cured one very obstinate case, and aggravated another case.

In all cases of chronic urticaria it is important to enquire as to the possible existence of bugs, fleas, pediculi corporis, or of the scarus scabiei. It is not uncommon for patients to suffer a long time from urticaria caused by one of these parasites, whilst other signs of their irritation are almost absent. In these cases ointment containing stavesacre or sulphur, with attention to cleanliness of bed and body linen, will cure the disease. Local applications in other cases appear of little permanent use; lotions or ointments containing chloroform, or nitric or acetic acid lotions, give momentary relief.

Pruriginous strophulus, a disease of infants, closely allied to urticaria, is usually relieved by the syrup of the iodide of iron.—*Lancet.*

Midwifery and Diseases of Women and Children.

THE OBSTETRIC HAND.

The following excellent suggestions from the pen of Dr. Robert Barnes, we find in the *Medical Times and Gazette*, and we fully indorse its teachings:

"In ordinary labour the obstetric hand is the only instrument required. It is also the only instrument called for in many of the greatest difficulties. In mal-presentations, in placenta prævia, in many cases of contracted pelvis, in not a few cases where, after perforation, the crochet and craniotomy forceps have failed to deliver, the bare hand affords a safe and ready extrication. One cannot help seeing that practice is often determined by the accidental perfection of, or familiarity with, particular instruments. Thus, a man who has only reached that stage of obstetric development which is content with a short forceps, will be armed with a good perforator and crochet. He cannot fail to acquire skill and confidence in embryotomy, and greatly to restrict the application of the forceps. Again, the preference generally given on the continent to cephalotripsy over craniotomy and extraction by the crochet or craniotomy forceps is the result of the great study directed to the perfecting of the cephalotribe. At the present day we may boast of having good and effective instruments of all kinds, each capable of doing excellent work in its own peculiar sphere, and, moreover, endowed with a certain capacity for supplanting its rival instruments. For example, the long forceps to supplant craniotomy in a certain range of cases of minor disproportion. Hence, it follows that it is of more importance to have a good forceps which can save life than it is to have a good perforator and crochet which destroy life. At the same time, it is eminently desirable to possess the most perfect means of bringing a fœtus through a very narrow pelvis, in order to exclude or to minimize the necessity of resorting to the Cæsarian section. Our aim should then be to get the most out of all our instruments—to make each one as good of its kind as possible. And admirable is the perseverance, marvellous and fertile the ingenuity, that have been brought to this task. I will not say it has all been misdirected; but certainly the cultivation of the hand, the study of what it can do in the way of displacing cold iron, has been much neglected. It would be not less instructive than curious to carry our minds back when the forceps and other instruments now in use were unknown, and to confront the problem which our predecessors, Ambrose Paré, Guillemeau and others had to solve—namely, how to deliver a woman with deformed pelvis without instruments. That they did successfully accomplish in many instances with the unarmed hand that we do now with the aid of various weapons, there can be no doubt. If this implies greater poverty of resources on their part, it not the less implies also greater manual skill. I am confident that the possession of instruments, especially of the craniotomy instruments, has led within the last century to a neglect of a proper use of the hands, which is much to

be deplored. We are only now recovering some of the lost skill of our ancestors."—*St. Louis Med. Reporter.*

Medicine.

ABSTRACT OF A CLINICAL LECTURE ON PYÆMIA AS A RESULT OF ENDOCARDITIS.

By Samuel Wilks, M.D., Physician to Guy's Hospital.

The following is an abstract of a very full report of the case taken by Mr. J. R. Stocker.

Alfred F—, aged 25, was admitted on January 1, 1868, for heart disease. He stated that he had rheumatic fever in 1851; and that the doctor then informed him that his heart was affected, but he had no symptoms referable to it until four months ago. He then became very ill, with shortness of breath, palpitation, etc., followed by some swelling and pains of the joints. On admission, he was seen to be very ill, having sallow countenance, and suffering great distress from shortness of breath and palpitation. The heart was most irregular and rapid in its action, with a loud systolic murmur, heard loudest over the apex. The urine had a good specific gravity, and was slightly albuminous. The legs were somewhat œdematous. He was ordered a saline mixture, with a pill of digitalis, squill, and mercury.

In four days he was much better; the breathing being less oppressed, and heart's action checked. He was then ordered to take ferrum tartaratum.

On January 14, he suddenly felt giddy, and afterward had intense headache.

On the 24th, there was an aggravation of the original symptoms, and great irregularity of the heart's action, with dyspnœa.

On the 31st, he found his right arm and leg very weak and numb. After four days this weakness had increased; and at the same time there was some hesitation in his speech and forgetfulness of words. He had also pain and swelling of the joints.

On February 14, he lay in a most precarious state. There was great irregularity of the heart's action. He had complete right hemiplegia, with partial aphasia; that is, a forgetfulness of many words. Thus, being told the name of a key, he would use the work "key" for every other object presented to him; and being shewn his grapes, and various names for them suggested to him, and among them the correct appella-

tion, he would not assent to any of them. He could read certain words on his bed-card, but not others. He thus continued in a barely living condition until March 1, when he died.

Post-Mortem Examination.—The left middle cerebral artery was plugged, and a large part of the left hemisphere disorganized by an abscess; the pus being green and thick. The lungs were in a state of splenization. The heart showed the mitral valve much diseased, the columns and cords covered with vegetations and shreds of fibrin. The liver contained throughout minute points of pus. The spleen had several fibrinous masses which were softening, and some were purulent—indeed, were distinct abscesses. The kidneys contained fibrinous masses not softening.

I bring this case before your notice, because it is the most marked which I have ever seen of the pyæmic process in connection with endocarditis. This disease is one of great interest pathologically, but has scarcely received a full recognition at the bedside of the patient. Although isolated cases of the disease may be found scattered through the journals, it has never been systematically treated of in the text-books of medicine. You know that, by the term pyæmia, we generally understand that form of disease in which the blood is infected by some purulent or kindred fluids; and that certain marked symptoms result, with a tendency to abscess in various parts of the body. The source of the infection is to be found on the surface of the body, and the deleterious matter is taken up into the veins. But now I have to tell you (in a clinical lecture, as I have been doing for many years past in the pathological lectures,) that the arterial blood may be in a like manner primarily infected at the very centre of the circulation. Just as, in ordinary pyæmia, the poisoned blood travels from the circumference to the centre, so here the converse process is in operation, the seat of the infection being the heart itself.

I should tell you that it has long been known that fibrinous masses have been found in the kidneys and spleen of those who have died of cardiac disease, and various theories have been mooted in explanation of their origin; the term capillary phlebitis having been much used of late years, after Rokitansky. We are indebted, however, more especially to the late much to be lamented Dr. Kirkes for unravelling this subject in a most masterly manner. If you refer to his paper, you will find that he had discovered the fact that, if particles of fibrin or vegetations were washed off the valves of the heart, they would be carried into the blood and plug up the vessels; they thus might lead to the destruction of any part, as of the brain or a limb, by the occlusion of the artery proceeding hereto. A case of this kind is now known by the term embolism. It

was also stated by Dr. Kirkes, that the fibrinous masses just spoken of as occurring in the kidneys and spleen, were also owing to small particles of fibrin blocking up the smaller arterial twigs; and he also showed that, with these formations, the blood was necessarily deleteriously affected, and that the patient suffered from symptoms of pyæmia. Now, it has so happened that the first-named facts contained in the doctrine inculcated by Dr. Kirkes have received the attention of the profession; but the latter have been too much disregarded, although equally important. The case of plugging of a large vessel and its effects are so manifest, that the case of blood-poisoning by smaller particles of disintegrating fibrin have been much overlooked except by a few pathologists, who have now and then published isolated cases of the affection. Thus cases by myself and others may be found in the Transactions of the Pathological Society and in the Guy's Hospital Reports. I might say that Dr. Kirkes and myself had, some years ago, some interesting correspondence on the subject.

In an ordinary case of pyæmia, death is most frequently due to a poisoned state of the blood, without any sufficient disease of a vital organ to account for the event; but we have no difficulty in pronouncing upon the character of the disease, from the peculiarity of the symptoms and the existence of a wound on the surface of the body. In the case, however, of pyæmia of the arterial system, arising from infection at the centre of the circulation, no such manifest cause may exist; and, after death, when the fibrinous masses or infractions are found in the viscera, they are believed to be inert, and the valvular disease is considered sufficient to account for all the symptoms and the ultimate issue of the case. Sometimes, however, the cardiac distress is but slight, while the symptoms of blood-infection are most marked, and then we begin to gain an insight into the importance of this variety of embolism. Thus, in a case which I published in the Transactions of the Pathological Society four or five years ago, the man had gangrene of the leg from the impaction of a plug of fibrin in his femoral artery; but, previous to this, he had several attacks of severe illness, accompanied by pains and swelling of the joints, called rheumatic, but which were in reality of a pyæmic origin.

In the case of ordinary pyæmia, an abscess may form in the brain, lung, or other organ, and so lead to death; but far commoner is it for these organs to show a lesser disease indicative of the morbid process in operation, while death is due directly to the altered state of the blood. So, in embolism, there are the striking instances of the imbedding of a plug in a vessel, leading to the destruction of the organ which it supplies; but there are also the other cases where the changes in the organ merely point to the blood-infection which is the real cause of the fatal

issue. Why in one case the symptoms are more severe than in another, may be due to the state of softening or disintegration of the fibrin. In one case the deposits are hard; in another they may have softened into a creamy fluid. I had until lately held the opinion that the material into which fibrinous matter softened was not true pus, but only pus-like; for, if examined by the microscope, no cells are seen; and that, if true pus were found either at the source of infection in the heart or in the viscera, endocardial ulceration must have taken place, and the tissues beneath must have been involved. In the present case, however, there was no proof of this deep-seated implication of the tissues; but yet the spleen and brain contained actual and well formed abscesses. I have never before seen so true an example of pyæmia from such a cause.

I wish you principally to remember the fact that the blood may be infected from disintegrating fibrin in the heart; and that all the symptoms of pyæmia may result, as violent rigours, followed by sweating, great prostration, sallow skin, pains and swelling of the joints, etc. I do not know that suppuration is necessary to the production of rigours, although it generally implies the introduction of a deleterious substance into the blood. Some of the most striking instances of this were those related in the London Hospital Reports, in which transfusion of fluid into the veins was performed. I have very little doubt that many of the symptoms which we witness in heart-disease are really due to the state of the blood, although overlooked from the greater attention given to the condition of the mechanism of the heart. Thus, in this very case, the patient is said to have had rheumatic pains and swelling of the joints, but these were probably pyæmic; and, carrying my memory back to other cases where death occurred after rheumatic endocarditis, I believe now that death was due to blood-poisoning, although at the time we thought the derangement of the affected valve sufficient cause for the event. In other diseases, too, it may give us a clue to the occurrence of certain symptoms; as, for instance, in scarlatina. Here there is the well-known rheumatic affection constantly occurring as a sequel to the disease, and at the same time endocarditis. Also, on post mortem examination, as I have elsewhere shewn, these fibrinous masses already mentioned may be found.

The purport of these remarks is that, in endocarditis or valvular disease of the heart, attended by the presence of vegetations of fibrinous coagula, a blood-poisoning may occur, giving rise to all the symptoms of pyæmia; and also that these may exist to a lesser degree in the form merely of pyrexia, prostration, pain in the joints. The facts are pathologically known, but are not sufficiently recognized from a clinical point of view, owing to the attention being too exclusively confined to the mere deranged mechanism of the heart.

I would also say that these symptoms by no means imply a fatal result. They come and go; the proof of this being found eventually in the cicatrices and remnants of deposits met with in the organs of the bodies of those who have died with heart-disease.—*British Medical Journal.*

PREVENTION OF EPILEPTIC ATTACKS.

M. Broca has presented to the Academy of Medicine an ingenious little apparatus, which has been invented by M. Rozier, of Bordeaux, with the object of exercising an instantaneous compression of the arm, and of thus preventing attacks of epilepsy whenever these are preceded by the aura epileptica. The apparatus had been contrived for an epileptic patient, a quarryman, who was never six weeks without being subject to a fit, and who was warned each time by a peculiar sensation which he felt in the right index. Scarcely a few seconds elapsed between the occurrence of this sensation and the explosion of the attack. It was consequently needful that the compression should be instantaneous. For eleven months the quarryman has constantly worn this little apparatus, and has never once had a complete attack. It is a sort of brasselet, and seems admirably adapted for all the purposes aimed at by the inventor.—*Lancet.*

MEDICAL NEWS.

The proposed alterations at the Royal Infirmary, Edinburgh, it is estimated, will cost £100,000. Mr. Kelley exhibited recently at a meeting of the London Pathological Society, a specimen consisting of two kidneys, both of which were found on the right side, none being on the left.

The distinguished Russian surgeon, Pirogoff, well known for his ingenious operation at the ankle, died recently.

Dr. Robley Dungleson, for many years Professor of Physiology in the Jefferson Medical College, Philadelphia, has resigned his appointment. Dr. James R. Wood has resigned his Professorship of Operative Surgery and Surgical Pathology in the Bellevue Hospital Medical College. The first medical college established in the United States, was the Medical Department of the University of Pennsylvania.

Nine students from the Dominion of Canada graduated at Bellevue College, New York, in March. The majority are from the Maratime Provinces. A Medical College has been established at Detroit, to be called the "Detroit Medical College." The citizens subscribed liberally towards its foundation fund.—Five nurses from the Nightingale—one of the institutions for training females as nurses—recently left London for Sydney, Australia, to accept situations as head nurses to the hospital in that town.—The Queen has subscribed £250 stg. towards the rebuilding of the Royal Infirmary, Edinburgh.—Sir James Clark has had a severe attack of Bronchitis, from which he is slowing recovering.

Canada Medical Journal.

MONTREAL, JUNE, 1868.

MEDICAL PRACTITIONER'S (COLONIAL) BILL.

The Medical Practitioners (Colonial) Bill introduced into the House of Lords, has attracted considerable discussion in the medical periodicals of Great Britian. The *Medical Times and Gazette* of April 25th, in an editorial article headed "Topics of the day," stated, "That this bill is nothing less than an attack on the rights and privileges of registered medical practitioners of the British Empire, which were secured to them by the Act of 1858. That persons whose names are upon the Imperial Register and who wish to practice in New Zealand, Canada or Australia, may be made to undergo the annoyance of fresh examinations as well as be mulcted in fresh fees." As regards our own colony, or rather that portion of the Dominion of Canada heretofore constituting Lower Canada, certain Legislative enactments have existed since the reign of George III., bearing on the practice of the profession of medicine and surgery in this Province.

In the year 1847 the Canadian Legislature granted an act of incorporation to the medical practitioners of the Province constituting them the College of Physicians and Surgeons of Canada East. By the provisions of that act no persons can practice the Profession of Medicine or Surgery in the Province without first obtaining the License of the College. The wording of the act bearing on all graduates of British Universities or Colleges is as follows: "But any person who has obtained a degree or diploma in any University or College in Her Majesty's Dominions shall be entitled to such license without examination as to his qualifications." As the law now stands, persons practising Medicine, Surgery or Midwifery in Canada East, and who do not hold the license of the College, are unable to enter a court of law and prosecute a patient for non-settlement of a professional claim; furthermore they are liable to summary conviction and fine before a magistrate for continuing to exercise their profession.

without having procured the necessary license. It appears to us that the question involved is whether we Canadians have the power to enact our own laws regulating these matters. If we have, and it is hardly to be believed that our right to enact and enforce our laws will be questioned, then is it impossible for any Imperial Act to become operative in our Dominion, so as to clash in any way with the enactments of our Legislature.

We have certainly extended to our brethren in the British Isles the right hand of fellowship by admitting graduates or licentiates from their Universities or Colleges who come amongst us, to the same privileges which we enjoy, the only requisite being that they shall appear in person, make oath that they are the parties mentioned in the Diploma which is submitted, that they obtained the same after a regular course of study, and having paid the registration fee the License of the College is granted.

It is time we should thoroughly understand our position, as it is unreasonable to expect us, although colonists, to make a one sided or blind bargain. We have Colleges and schools in Canada, in which the curriculum of study is quite equal to the best in the United Kingdom, and yet the right of registration in the Imperial register is not granted to our graduates or licentiates; or in other words, our Universities and Colleges are simply ignored as educational institutions. Our Colleges and Universities, many of them, possess Royal charters, they hold the same letters patent as those held by the time honoured institutions of the mother country, their prescribed curriculum is the same as that exacted by the Colleges at home, and in the matter of preliminary studies we have taken an exact copy of that prescribed by the General Medical Council of Education and Registration of Great Britain, and although we have done everything required of us, and of the educational institutions in the United Kingdom, still the privileges which they possess are withheld from us. Why is this? Surely not because we turn out less competent men, nor is it because of the fear of our flooding the avenues of practice with our graduates; in Canada, numerically, our graduates count by twenties, while the home Colleges turn out their thousands; what good and sufficient reason, then, can be advanced for refusing steadily and persistently to recognize those colonial educational institutions that have conformed in every particular to the wise and salutary enactments of the General Council of Medical Education and Registration of Great Britain? We have heard that it is because of the lack of supervision, the absence of any direct means of inspecting our method of conducting our examinations and of being satisfied that these examinations are fairly and honestly made. This is a matter which could be readily and satisfactorily attended to, without the necessity or expense of sending a special commissioner to inspect our schools.

There are men to be met with in any of the colonies, men unconnected with any of the schools, whose report would be perfectly reliable if that were needed, but we hold that the proficiency of the graduates of any given University or College should be sufficient to stamp the character of the instruction they received, but if more is needed, if an inspecting officer or several such be deemed necessary, they are to be had without the cumbrous and expensive method of sending out persons from the mother country to inspect and report on the character of our educational course. These observations are suggested because we regard the action of the home authorities as narrow-minded and unjust, and although alive to the necessity of caution in granting equal privileges to Colleges separated by thousands of miles, yet all things being equal, we think it would conduce to the credit of the authorities for liberality, and a desire to give to us, as Englishmen, equal rights and equal representation, if we are recognized on equal terms with those educational institutions which alone possess the superior advantage of having existed a half century or so longer.

THE AMERICAN MEDICAL ASSOCIATION AND THE CANADIAN MEDICAL ASSOCIATION.

The American Medical Association held its annual Session, May 5th, at Washington, D.C., which seems to have been one of great interest and harmony. We are pleased to see that delegates were appointed to represent that Association at the next meeting of the Canadian Medical Association, to take place in September. The gentlemen selected are C. C. Cox, M.D., LL.D., of Maryland; Drs. John L. Atlee, of Pennsylvania; N. S. Davis, of Illinois; Charles C. Dee, of New York; Grafton Tyler, of the District of Columbia; W. M. Wood, of the Navy, and S. D. Gross, of Philadelphia.

We are informed by Dr. Caniff, Secretary for Ontario of the Canadian Medical Association, that he received a letter from Dr. Atkinson, the Permanent Secretary of the American Association, inviting delegates from the Canadian association to be present at the late meeting. Dr. Canniff conferred with the General Secretary, Dr. Belleau, and they decided that in the present partially organised state of the Association, and in the absence from the country of the President, Dr. Tupper, C.B., the kind invitation proffered by Dr. Atkinson could not be responded to. Dr. Canniff communicated to that effect, and at the same time on behalf of the Canadian Association expressed a hope that the

American Association would feel it desirable to send delegates to Montreal in September. This invitation, as we have seen, has been gracefully responded to.

We hasten to assure the distinguished gentlemen who form the delegation, that the most fraternal greeting awaits them from the Canadian Medical Association. Nor shall the city of Montreal be found wanting in the most kindly courtesies.

CONVOCATION OF VICTORIA UNIVERSITY, COBOURGH, ONTARIO.

The annual Convocation at Cobourgh, last week, was the most imposing and successful that has been held. The Baccalaureate sermon on Sunday evening, the sacred concert on Monday evening, the Alumni Meeting on Tuesday afternoon, the Alumni Dinner on Tuesday evening, the Convocation on Wednesday afternoon, and the *conversazione* on Wednesday evening, were all occasions of peculiar interest.

THE ALUMNI ASSOCIATION.

On Tuesday afternoon a general meeting of the Alumni Association took place. B. M. Britton, Esq., M.A., of Kingston, occupied the Chair. The following officers were elected for the next year:—

Rev. A. CARMAN, President of the Albert College, Belleville, *President*, W. BEATTY, Esq., M. P. P., of Welland, *1st Vice President*, WM. CANNIFF, Esq. M.D., M. R. C. S. of Belleville, *2nd Vice President*, H. HOUGH, M.A., *Secretary*, REV. PROFESSOR BURWASH, M.A., *Treasurer*.

Graduates residing in Cobourgh compose the Managing Committee.

After choosing the officers, the attention of the meeting was called to the general interests of the University, and especially to the position resulting from the threatened withdrawal of the government grants to the colleges. The Alumni present were of one mind on this question. Decided and strong resolutions were passed, pledging the Association, whose members reside in every part of the province, to sustain the college, and claiming its right to continued legislative aid. The graduates and former students of Victoria College are thoroughly in earnest on the subject, they simply insist on the right thing being done; and we doubt not that they with the other friends of denominational colleges, will, if it should become necessary, make it manifest at the next elections that secularism must not enjoy a monopoly of public aid.

On Tuesday evening the Annual dinner of the Association was given, at which the Chair was occupied by the President, the Rev. A. Carman, M.A. Animated speeches were made by the Rev. W. M. Punshon, M.A., Rev. Dr. Ryerson, Rev. Dr. Nelles, Rev. A. Carman, M.A., W. Beatty, Esq., M.P.P., Dr. Boulter, M.P.P., Professor Wilson, William Kerr, Esq., M.A., B. M. Britton, Esq., M.A., W. W. Dean, Esq., M.A., W. H. Kerr Esq., of Montreal, Ashton Fletcher, Esq., M.A., Dr. Peltier, Dr. Canniff, Dr. Lavel, Dr. Potts, H. Hough, Esq., M.A. G. Young, Esq., M.A., J. Mills, B.A., Dr. Diamond, Dr. Fielding, and Dr. O'Rielly. It was exceedingly encouraging to observe the earnest devotion to the interests of the University, manifested by all present.

THE CONVOCATION.

The Annual Convocation is every year regarded with increasing interest. To the students the conferring of degrees is much more than a mere ceremony, to the graduates and their friends it is one of unmingled pleasure; the friends of our denominational college, see, in the annually increasing number of graduates, the importance of the work it is doing in the country; while the crowd of visitors and distinguished strangers from a distance, and the eloquent addresses delivered, heighten the *prestige* from year to year, of this important occasion. The Convocation last Wednesday was the best and the most encouraging that has ever been held. The proceedings were opened by an impressive prayer by the Rev. James Elliot, President of the Conference. Then followed the Latin Oration, by Mr. Edward Charlton, of Ilderton; the Greek Oration, by Mr. James Mills, of Penville; and the Valedictory Address, a very excellent oration on "Cowper," by Mr. James Roy, of Cobourg. Mr. Roy's oration was exquisitely tender and beautiful, and his elocution was very superior.

The degrees were conferred by the Rev. S. S. Nelles, D. D., President of the University. The following degrees were conferred:

B. A.—James Mills, *Gold Medallist.*– Jonathan B. Dixon, Thos. E. Morden, Edward Charlton, *Silver Medallist.* James Roy, W. H. Rowson, James E. Blair, John Scott, Henry Bleecker, Henry H. Shaler, Wm. Wilkinson.

M. A.—Ashton Fletcher, B.A., LL.D., J. R. Youmans, B.A., Osborne Lambly, B.A., Alfred McClatchie, B.,A., W. F. Morrison, B.A., Andrew Milne, B.A., W. C. Washington, B.A., Byron M. Britton, B.A., W. H. McClive, B.A., LL.B., Cyrus A. Neville, B.A., W. C. Henderson, B.A., David Kennedy, B.A., Wm. R. Parker, B.A., E. B. Ryckman, B.A., Jacob E. Howell, B.A., J. C. Wilmott, B.A.

L L. B.—J. J. McClaren, M.A., B.C.L., (*ad eundem*;) J. B. Doutre.

M. D.—Thos. Adams, B.A., R. Garneau, R. P. Aikman, J. Sylvestre, T. Brunskill, A. Marotte, C.H. Brereton, J. Gingras, J. S. Diamond,

J. Drainville, J. Fielding, A. Laferiere, P. D. Goldsmith, S. A. Longtin, W. A Hughson, J. Robitaille, F. King, J. Archambault, R. C. Lloyd, C. S. Stokes, C. Luiz, C. Williams, G. D. Loughead, B. Walden, A. MacLay, P. Valois, L. McAllister, L. Brodeur, A. N. McBrien, H. Choquette, D. Newkirk, E. Valcourt, J. B. Olver, J. Demers, M. O'Reilly, A. Beliveau, F. H. Pope, N. Richard, F. S. Sproule, S. McHenry, B. Vigneau, D. Martel.

D. D.—Rev. J. H. James, Govornor, Sheffield College, England.

Professor Geikie then addressed the Graduates in Medicine, with wise and impressive counsels.

The Prince of Wales Gold Medal was presented to Mr. James Mills, by the Rev. W. M. Punshon, M. A; *the Prince of Wales Silver Medal* was presented to Mr. Edward Charlton, by the Rev. L. Taylor, D. D.; *the Ryerson Prize* to Mr. A. G. Knight, by the Rev. Dr. Ormiston; *the Webster Prize*, to Mr. W. H. Rowson, presented by Dr. Browse; *the Hodgins Prize*, to Mr. James Roy, by Wm. Kerr, Esq., M.A.; *the Cooly Prize*, to Mr. J. W. Sparling, presented by Rev. Dr. Jeffers.

Literary Association Prize, for best English Essay, to Mr. James Roy, presented by W. W. Dean, Esq., M. A.; do. do. for Elocution, to Mr. John Scott, by Rev. A. B. Carman, Esq., M. A.; do. do. for English essay, to Mr. D. Robson, by Dr. Canniff; do. do. for Elocution, to Mr. James Allen, by W. Beatty, Esq, M.P.P. *The Punshon Prize* for Elocution and Composition, was founded at this Convocation by W. Kerr, Esq, M.A.; it was presented to Mr. James Roy, by Rev. Dr. Ryerson.

Each of these presentations was accompanied by suitable remarks, addressed to the successful competitor, and the variety of speakers and subjects sustained the interest of the meeting throughout. Rev. Dr. Ormiston was in one of his happiest moods. He was proud of being the first living graduate of Victoria College, and he cordially and earnestly bore testimony to the noble service the College had rendered to the cause of education. No description can do justice to the address of Mr. Punshon. Brilliant, strong, conclusive, practical, wise, he contributed greatly to the success and influence of this most important Convocation—important at this critical time,—and effectually rallying the friends of "OLD VIC." to the support of our educational standard. His remarks on the relations between Revelation and Science put the subject in its true light, and with remarkable vividness and force. Great as is his eloquence, the simple-hearted earnestness of his soul is, to us, the chief charm of his magical speaking.

PROCEEDINGS OF THE ANNUAL SESSION OF "THE MEDICAL ALUMNI ASSOCIATION OF VICTORIA UNIVERSITY," HELD AT COBOURG, ON TUESDAY AND WEDNESDAY, THE FIFTH AND SIXTH DAYS OF MAY, 1868.

First day's session commenced at 11 o'clock, A.M., John Hubert Sangster, M.A., M.D., President, not being present.

H. Peltier, M.D., Edin., Montreal, 1st Vice President, was called to the chair.

By direction from the chair the names of the Alumni present were recorded as follows, viz.,

S. S. Corbett, Perrytown; Benjamin Walden, Elginfield; Charles Williams, Glenwilliams; Thos Burnskill, Bondhead; John S. Diamond, Toronto; Laughlin McAllister, Duntroon; Chas. A. Breaston, Bradford; Thomas Adams, B.A., Tweed; R. P. Aikman, Ancaster; J. Fielding, Orono; Ralph E. Lloyd, Stouffville; Wm. A. Hughsan, Delaware; George D. Lougbead, Ballymote; Daniel Newkirk, Walsingham; Calvin Luter, Galt; Chas. S. Stokes, Toronto; Alfred N. McBrien, Newtonville; Miles O'Riely, Hamilton; Perry D. Goldsmith, Dundonald; Thos. S. Sproul, Maxwell; Archibald MacLay, Fingall; Frank King, Port Robinson; Samuel McHenny, Sandhill; Jobes B. Oliver, Ragles; Francis H. Pope, Bothwell; E. W. Tegart, Scotland, Co. Brant; Jas. D. Stewart, Ottawa; William Canniff, Belleville; J. Stuart Scott, Cobourg; George J. Potts, Belleville; George Burnham, Ashburnham; William A. Willoughy, Grafton; Marshall M. P. Dean, Reene; S. L. Nash, Amelissburgh; William Wade, Cobourg; Joseph A. Fife, Hastings; A. M. Rosebrugh, Toronto; George Alra Carson, Whithy; Robert A. Corbett, Perrytown; Walter Bayne Geike, Aurora; C. A. McRae, Erin Village, Wellington; George Abbott Norris, Omomee, James Stimson, St. George, Co. Brant; B. Vigneau, St. Gregorie de Nicolet; R. Garneau, St. Ann de la Perault; J. Sylvester, St Guillcaume D'Upton; A. Marrotte, Montreal; J. Gingras, St. Hyacinth; J. Drainville, Berthier; A. Laferrière, St. Cuthbert; S. A. Langtin, Montreal, J. Robitaile, Quebec; J. Archambault, Terrebonne; P. Valois, Montreal; L. Brodeur, Varennes; H. Choquette, Varennes; E. Valcourt, St. Simon; J. Demers, St. Bruneau; Remi Beliveau, Montreal; Nap. Richard, Montreal; A. N. Pelletier, Antrim; W. Sergius Bald, Antrim. Among the visitors present were Dr. G. H. Boulter, M.P.P., North Hastings; Rev. A. Carman, M.A., President Albert College, Belleville; Rev E. B. Ryckham, Kingston; W. W. Dean, Esq., A.B., Barrister, &c., Belleville.

Minutes of former Session held at Yorkville, near Toronto, 1st and 2nd days of October, 1867, were read and approved.

Draft of Constitution was read and submitted.

Moved by Dr. Rosebrugh, seconded by Dr. Canniff,—That the draft of the constitution, By-laws and rules of order now submitted for adoption by Dr. Potts, chairman of that committee, be adopted, provisionally only. Carried.

REPORT OF COMMITTEE ON ETHICS, SUBMITTED.

Moved by Dr. J. S. Scott, seconded by E. W. Tegart, M.D—That the consideration of the report of the committee upon "Medical Ethics" be deferred until the annual meeting at Toronto in October next; but that the adoption of a Code especially adapted to the requirement of this Association be recommended, and that Drs. Peltier and Rosebrugh be added to the committee. Carried.

Moved by Dr. Rosebrugh, seconded by Dr. Scott—That the election of officers be deferred until the current meeting in Yorkville in October next. Carried.

Moved by Dr. Nash, seconded by Dr. Tegart, that we adjourn to meet at 6 o'clock P. M. Carried.

AFTERNOON SESSION, 6 o'clock P.M.

Hector Peltier, M.D., Edin., Vice President, and thirty-one members present.

Minutes of morning Session read and approved.

Moved by J. S. Scott, M.D., seconded by Dr. Canniff—That the reading of papers be postponed until to-morrow morning at nine o'clock A.M. Carried.

Moved by Dr. Diamond, seconded by Dr. McAlister—That Dr. James Fielding, from the Yorkville Medical Branch, be requested to respond to the annual toast, "The Graduates in Medicine for the Session of 1867-68, at the Alumni Dinner." Carried.

Moved by Dr. Rosebrugh, seconded by Dr. Remi Beliveau—That Dr. Joseph Archambault, from the Montreal Branch Medical Department, be requested to respond to the toast, "The Graduates in Medicine for the Session of 1867-68." Carried.

Moved by Dr. Scott, seconded by Dr. Rosebrugh—1st. That this association has learned with pleasure of the engagement of Dr. Canniff as one of the Editors of the *Canada Medical Journal* and most heartily congratulate the Publishers in securing so efficient an aid to the Editorial Staff of that Journal.

2nd.—That the Secretary be requested to furnish the proceedings of this association to Dr. Canniff for publication in the *Canada Medical Journal*. Carried.

Moved by Dr. Willoughby, seconded by Dr. Burnham—That a list be now opened for subscribers to the *Canada Medical Journal*. Carried.

Moved by Dr. Rosebrugh, seconded by Dr. Willoughby—That we do now adjourn to meet here at 9 o'clock A.M., to-morrow morning. Carried.

SECOND DAY.

The adjourned Session of the Medical Alumni Association Victoria University met at the hour of nine o'clock A.M., Dr. Wm. Wade, Cobourg, fourth Vice President, in the chair. The Association having resolved to take into consideration charges which had been made against certain members of the association, of irregular and unprofessional practices, it was moved by Dr. Potts, seconded by Dr. Willoughby, that all persons present who are not members in good standing in the Medical Profession be requested to withdraw. Carried.

Vice-President Wade desiring to retire from the chair to take part in the debate, Dr. Canniff was requested to take the chair.

The first charge had reference to advertising by posting bills to announce that a certain Doctor would be in a certain place at such a time. It was shown that it had been done without the accused gentleman's knowledge and contrary to his wishes.

The second charge referred to the conduct of one who had associated himself with an "Eclectic." This case occupied two hours and was fully discussed by nearly every member present. The difficulty was finally met by the following resolution.

Moved by Dr. S. L. Nash, seconded by Dr. Tegart—That the Association does not approve of the principle of associating professionally with the so called Eclectics and Homœopaths, but are willing to abide by the decision of the Canadian Medical Association, which meets in September next in the city of Montreal. Carried.

The third case was in reference to a "Victoria Wine Bitters" advertised and exclusively vended by one who also professed to be an Eclectic. The individual in defence declared the article was sold not as a medicinal remedy, but simply as a Bitters, having for its object the reformation of drunkards, but he admitted when asked, that the Wine Bitters contained one-thirteenth of the whole quantity of pure alcohol.

Moved by Dr. Scott, seconded by Dr. Nash—That this Association disapprove of the course pursued by Dr.——in advertising his Wine Bitters, as calculated to bring reproach on the Profession of Medicine. Carried.

The fifth case was one in which an Alumnus was charged with resorting to the practice of "Cancer Curers" and adopting the usual course pursued by these quacks in the treatment of this disease. This case also elicited considerable discussion, during which Dr. Peltier entered the room. Dr. Canniff at once vacated the chair, and conducted the Vice President, Dr. Peltier, to the chair.

Moved by Dr. Miles O'Riely, seconded by Dr. Tegart—That in the opinion of this Association, the advertisement published by Dr—— as a cancer curer is detrimental to the dignity of the Profession and of this Association, and that he be requested to discontinue the same. Carried.

Dr. Peltier, Vice President, accordingly requested Dr.——to discontinue the practice and plan of his advertisement.

Moved by Dr. Rosebrugh, seconded by Dr. Potts—That whereas some of the members of this Association have published cards in the "public prints" of an irregular character, and whereas this association has not as yet adopted a Code of Medical Ethics, we cannot allow this occasion to pass without expressing our disapproval of the same.

Moved in amendment by Dr. W. B. Geike, seconded by Dr. M. P. Dean—That this Association views advertisements calling attention to special modes of treating special diseases, as cancers, chest affections, ruptures, &c., as at variance with the universally acknowledged rules of Medical Ethics, and hereby expresses its entire disapproval of the publication of such advertisements by any of its members. Amendment carried. The convocation exercises commencing at three o'clock P.M., it was moved by Dr. Scott, seconded by Dr. Diamond—That we do now adjourn to meet at six o'clock P.M. Carried.

The afternoon Session commenced at six o'clock P.M., Dr. Wm. Wade, fourth Vice President, in the chair.

Moved by Dr. Potts, seconded by Dr. Diamond—That in consequence of the prolonged Session of this morning, and attendance on the convocation exercises engaging the time of Dr. Potts, secretary, the minutes of this morning's Session, not being in readiness, are not required to be read, and that this afternoon's Session be considered a continuation of the morning Session. Carried unanimously.

Moved by Dr. Rosebrugh, seconded by Dr. Canniff.—That Dr. Potts of Belleville, Dr. Brouse of Prescott, Dr. Edmonston of Brockville, and Dr. R. A. Corbett of Perrytown, be appointed delegates of this association to attend the next meeting of the Canadian Medical Association to be held in the city of Montreal, in September next. Carried unanimously.

Moved by Dr. Caniff, seconded by Dr. Corbett, and resolved—That the thanks of this association be presented to the county council of the

united counties of Northumberland and Durham, for the favour accorded in placing their council chamber at the disposal of the Medical Alumni Association of Victoria University, and that the secretary furnish that body with a copy of this resolution. Carried unanimously.

Moved by Dr. Rosebrugh, seconded by Dr. Corbett—That the thanks of this Association are due and are hereby tendered to Dr. Potts, secretary, and the committee of management, for the zeal and efficiency with which they have discharged their duties in preparing the business of this meeting. Carried unanimously.

Moved by Dr. Potts, seconded by Dr. Dean—That the thanks of this Association is hereby tendered to the Grand Trunk, Great Western and Northern Railroad authorities for their liberality in furnishing return tickets at one fare, to members attending the Session of the Medical Alumni Association of Victoria University, and that the Secretary forward a copy of this resolution to each. Carried unanimously.

Moved by Dr. Dean, seconded by Dr. Burnham—That when the report of this Session of the Medical Alumni Association Victoria University, appears in the *Canada Medical Journal*, three hundred and fifty copies be procured and distributed among the Medical Alumni of Victoria University. Carried.

Moved by Dr. Corbett, seconded by Dr. Tegart—That the reading of the papers prepared by the members of this Association named in *circular*, and which should have been read during this morning Session, be deferred till the meeting of this Association in Yorkville on the 1st October next. Carried.

Moved by Dr. Diamond, seconded by Dr. Fielding—That the name of Dr. J. S. Scott, Toronto, be added to the delegates appointed to meet the Canadian Medical Association in September next in the city of Montreal.

Moved by D. Willoughby, seconded by Dr. Diamond.—That all members of this Association discountenance and discourage all reprints of an immoral tendency which sometimes appear in the public prints with regard to medical examinations, at Coroner's Inquests, &c., &c., as these have a tendency to lower the standard of morality among classes reading these journals in which they are published. Carried.

Moved by Dr. Scott, seconded by Dr. S. L. Nash.—That the truss presented by Mr. G. V. M. Relyea of Belleville, be referred to a committee, to consist of Drs. Diamond, Geike and Potts, to report at the next Session of this Association. Carried.

Moved by Dr. Potts, seconded by Dr. Corbett.—That we do now adjourn and stand adjourned until the next Session of the Medical

Alumni Association Victoria University, to be held in Yorkville on the 1st day of October next, of which all members shall receive due notice. Carried.

The Chairman congratulated the Association on the fraternal spirit and harmonious zeal for the general welfare of the profession, evidenced by the cordial and wholesome character of the friendly discussions held during the two days the Association had been in Session, and earnestly recommended that, in the future, continued effort for the advancement of our Profession and the uprightness of our conduct in our several fields of practice, might mark us as members of the Medical Alumni Association of Victoria University, thereby adorning the ranks of the honorable profession of which we are members, and honoring the University with which we are so intimately connected, and bidding all a hearty farewell, announced that the Session of the Medical Alumni Association of Victoria University is adjourned, to stand adjourned until the next regular meeting, to be held in Yorkville on the 1st day of October next, of which all members shall receive due notice.

GEORGE J. POTTS, M.D., *Secretary*
Medical Alumni Association, Victoria University.

Belleville, 15th May, 1868.

MEDICAL ASSOCIATION OF THE COUNTY OF HASTINGS, ONTARIO.

A meeting of the members of the Medical profession of the County of Hastings was held in the town of Belleville on Wednesday the 20th May. There were present, Drs. Lister, Holden, Bradley, Burdett, Hope, Potts, Wilson, Day, Oronhyatekha, Powers, Howell, Boulter, M.P.P., Stewart, and Canniff. Upon motion it was *Resolved*:—

"That we organise into an association to be called the Medical Association of the County of Hastings, to consist of the regular practitioners who are registered under the New Medical Act, residents in the said County."

Committees were then appointed to arrange the question of Medical Tariff, and to prepare a Constitution and By-Laws. The meeting now adjourned to give time for these Committees to prepare their respective reports.

The following Constitution was subsequently adopted.

CONSTITUTION.

NAME.

Article I.—That this Association shall be called the Medical Association of the County of Hastings.

Article II.—That the Members shall consist of the Regular Practitioners of the County of Hastings, who are registered under the New Medical Act, by paying the sum of One Dollar.

OBJECT.

Article III.—The object of this Association shall be to advance Medical Science and the interests of the Profession.

Article IV.—Each Member shall sign a declaration that he will observe the Constitution, By-Laws and Rules of Order of the Association, and be governed in his professional career by the Code of Medical Ethics adopted by the Association.

OFFICERS.

Article V.—The Officers of this Association shall consist of a President, First and Second Vice-Presidents, and Secretary-Treasurer.

Article VI.—The Officers shall be elected annually by Ballot.

Article VII.—The Majority of the votes cast shall determine the election of a candidate for office.

Article VIII.—Any Member who shall violate the Constitution or any of the By-Laws or Rules of Order, or who shall have been guilty of any gross violation of *Medical Ethics*, shall be censured or expelled by a two-third vote of the Members present, but no motion to expel a Member shall be acted upon unless he shall have been duly notified in writing.

Article IX.—That the Constitution and By-Laws may be altered by a two-third vote at the annual meeting, and the President may direct the Secretary to call a special meeting when required.

BY-LAWS.

Article I.—The Annual Meeting of this Association shall be held on the *Third Wednesday in May*, at Belleville, commencing at 10 o'clock A.M.

Article II.—To meet the expenses of the Association, a Tax on each Member shall be levied from time to time for that purpose.

RULES OF ORDER.

Article I.—During the Session of the Association the ordinary Parliamentary Rules of Order shall be observed in respect to debates.

The Association then proceeded to the elections of Officers, which resulted as follows: President, Dr. William Hope, Belleville; 1st Vice-President, Dr. G. H. Boulter, Stirling; 2nd Vice-President, Dr. James Lister, Belleville; Secretary-Treasurer, Dr. George J. Potts, Belleville.

The Association then adopted a Tariff to be observed by its Members.

The Association then adjourned, having passed a resolution that a

Report of the proceedings be furnished the *Canada Medical Journal* for publication.

TO OUR SUBSCRIBERS.

With this number, the fourth volume of the *Canada Medical Journal* is brought to a close. Those of our subscribers who are in arrear will receive their accounts with this issue, and we would earnestly request a speedy remittance. There are many subscribers in arrear, some indeed, who have never contributed one shilling towards our support. This is not right, and in mercy to themselves the publishers have determined to remove their names from the list of subscribers.

To those gentlemen who have fully recognized our usefulness, and also, the necessity of supporting a medical periodical in the Dominion of Canada, and who have regularly paid their subscription, we beg to tender our sincere thanks.

THE CHEMISTS' ASSOCIATION, MONTREAL.

The regular monthly meeting of the above association was held on the 4th of June. A very interesting and instructive paper upon Strychnine was read by Dr. Girdwood, who, we may add, has paid very great attention to this the subject. It is intended, we believe, to have the Association represented by delegates at the annual meeting of the American Pharmaceutical Association, which takes place this fall at Philadelphia. The Chemists' Association has had a very successful season, all the members taking an active interest in its support. It is now, we believe, thoroughly established, and we hope its next session will be even more successful than the one just closed.

THE AMERICAN MEDICAL ASSOCIATION.

This Scientific Association held its annual meeting in Washington on the 6th of May, and continued in session three days. A good deal of interesting matter was laid before the meeting and ordered to be printed in its transactions. A resolution to establish institutions in the United States for the training of nurses, similar to those in operation in London was referred to a special committee. The committee on Medical Ethics offered a resolution endorsing consultation with females who had received a regular medical education. A good deal of discussion took place upon this resolution, and the matter was indefinitely postponed. The Association decided to hold its next meeting in May, 1869, at New Orleans.

CANADA
MEDICAL JOURNAL

AND

Monthly Record

OF

MEDICAL AND SURGICAL SCIENCE.

EDITED BY

GEORGE E. FENWICK, M.D.,

Professor of Clinical Surgery McGill University; One of the Governors of the College of Physicians and Surgeons; Surgeon to the Montreal General Hospital; Consulting Surgeon to the Montreal Dispensary and Infirmary for Diseases of Women and Children; Honorary Local Secretary Anthropological Society of London, England.

AND

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VOL. IV.

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No. 879 of 1867.

Municipal Commissioner's Office,

Public Health Department,

Bombay, 30th April 1867.

To the MUNICIPAL COMMISSIONER

for the City of Bombay.

Sir,—With reference to paragraph 7 of Government Resolution No. 398 of 1867, dated 25th February 1867, forwarded to me for opinion, I have the honour to submit the following report on the measures which I consider necessary to prevent Bombay being regarded by European nations as a base whence Cholera habitually spreads westward by the sea.

2. Before entering into an inquiry of this kind, I think it well to bring to your notice the facts that have been proved regarding Cholera poison, as if we accept these as unquestionably true, our course in dealing with the question of prevention or counteraction of this disease will be rendered easier.

Dr. Simon, the Medical Officer to the Privy Council, states in his Eighth Report, published in 1866:—

"The doctrine on the subject of the contagiousness of Cholera, which, in my opinion, deserves, in the present state of knowledge, to be accepted as practically certain, sufficiently certain I mean to be made the basis for precautionary measures, may be stated in the following propositions:—that when Cholera is epidemic in any place, persons who are suffering from the epidemic influence, though perhaps with only the slightest degree of diarrhœa, may, if they migrate, be the means of conveying to other places an infection of indefinite severity; that the quality of infectiveness belongs particularly, if

not exclusively, to the matters which the patient discharges, by purging and vomiting, from his intestinal canal; that these matters are comparatively non-infective at the moment when they are discharged, but subsequently, while undergoing decomposition, acquire their maximum of infective power, that choleraic discharges, if cast away without previous disinfection, impart their own infective quality to the excremental matters with which they mingle in drains or cesspools, or wherever else they flow or soak, and to the effluvia which those matters evolve, that if the Cholera contagium, by leaking or soaking from drains or cesspools, or otherwise gets access, even in small quantity, to wells or other sources of drinking water, it infects, in the most dangerous manner, very large volumes of the fluid; that in the above described ways even a single patient, with slight choleraic diarrhœa, may exert a powerful infective influence on masses of population, among whom perhaps his presence is unsuspected; that things such as bedding and clothing, which have been imbued with choleraic discharges, and not afterwards fully disinfected, may long retain their infectious properties, and be the means of exciting choleraic outbreaks wherever they are sent for washing or other purposes.

"The precautions, generally, which may be taken against contagious diseases are of two kinds: *First*, if possible, to prevent the entrance of the contagion; *Secondly*, if the contagion be present, to annihilate, as far as possible, the circumstances which favour its spread."

3. I most thoroughly believe in the truth of the above doctrine, and all efforts in this city should be directed to the carrying out in the fullest extent the two recommendations Dr. Simon makes.

An examination of the Mortuary Reports, since they were established in 1848, proves that Bombay has always suffered more severely from Cholera when this disease

has been imported from the Moffussil into the Island, than when it is generated within the city itself.

I submit the following extract from Dr. Haines' Report for 1863:—

"In the Report for 1849 it is shown that up to the month of August in that year Cholera was all but extinct in Bombay, only six deaths having occurred in the first six months of that year. It suddenly appeared in the middle of August; in September 690 persons died, and by the end of the year it had destroyed 2,363. It was found that the disease had prevailed to some extent in the neighbourhood of Sholapoor in May of that year, but not enough to excite uneasiness. In the latter end of July or beginning of August it began to spread with the utmost rapidity, and appeared simultaneously in the Concan in villages far apart from each other, but all on the line of roads leading directly from the Deccan. In another week it had reached Bombay. It raged throughout the four following years, carrying off a maximum of 3,353 in 1854, after which the mortality gradually declined, until in 1858 only 105 persons died.

"In 1859 the deaths were again 2,265. The disease appeared about the second week in May in the Wace district of the Sattara Collectorate and about Bhor, and then slowly spread, travelling through the Concan to Bombay, where it appeared at the latter end of the month, and was so fully established in June, that in that month 843 deaths occurred.

"Cholera again slowly subsided. In 1861, during the first ten months, there were only 160 deaths, but in the month of December, it suddenly appeared and carried off 466 persons, and in the next month 625. In this case the spot where it originated in the town was pretty clearly ascertained. It commenced among the families of one of the Native Regiments on the Esplanade, and among a party which had just arrived from one of the largest towns

of the Deccan, where the disease was rife. After January 1862 it somewhat subsided, fluctuating about an average of 200 a month, and reached a minimum of 50 in February 1863. From that time the number of deaths slowly rose, until in June they were 161; then suddenly amounted, in the next month (July) to 412, and afterwards again fell. In December the number of deaths had crept up to 319, and in January sprang up to a maximum of 622.

"For days before the disease broke out here, it was notorious that the pilgrims returning from the fairs of Punderpoor and Alundee were dying by hundreds, and the introduction by them of the disease into the city of Poona was greatly dreaded, and not without reason. It soon appeared there, carrying off scores daily, and very shortly afterwards the rapid increase in the Cholera mortality in this town showed how quickly the contagion spread along the ordinary line of traffic."

Dr. Haines also gives the following extract from an account written by the Reverend Adam White,* which forcibly illustrates why the fair at Punderpoor should be regarded as the very *nidus* of Cholera:—

"I paid my annual visit to Punderpoor in November 1863. Great multitudes from all parts of the country come at that season, sometimes from incredible distances, to the shrine of Vittoba. In a flowing stream, in thousands, they pass on to the shrine from every direction, weary and worn, they then encamp near the town, mostly in the bed of the river. Some have tents, some light cloth sheds, many nothing at all to protect them. First comes a day of fasting, distinguished by eating either nothing or else unwholesome trash, and by shouting forth by day and by night, with clang of cymbals and violent gesticulations, up to the last ebb of strength, the names of Vittoba and his wife, and their

* Notes by the Rev. Adam White, dated 28th January 1864. Mr. White shortly afterwards died of Cholera at Sassoor.

'saints.' Add to this, that the whole atmosphere becomes frightfully tainted from the first, so that one cannot walk anywhere, far or near, without being sensible of the most sickening odours. Can it be wondered that Cholera is very often generated at the place. But the peculiar seed plot where that pestilence first germinates remains to be described. *It is believed that it is the very temple of Vittoba itself*, a small stone room, with no aperture but a small door, into which perhaps 50,000 persons in the course of a few days force their way, and in the immediate neighbourhood of which, in the temple court, may be seen thousands of men and women sitting closely packed, waiting for their turn to enter. So polluted does the air of the temple become, that the vapour from the breath and bodies of the worshippers condenses on the image, thus giving rise to the idea that the god miraculously perspires. The Sub-Assistant Surgeon now appointed by Government to Punderpoor, stated to me his belief that the disease, in its first origin, could usually be traced to *that spot*.

"The day after I left Punderpoor, Cholera in a bad form manifested itself among the pilgrims, but yet many did not die. The disease might have died out if it had not been cultivated. But the same pilgrims immediately resorted, as is their wont, to Alundee near Poona, and there it met with a favourable field in which to grow. The seed sown in Punderpoor ripened quickly and fearfully at Alundee; and the grain was then scattered broadcast over the length and breadth of the land by pilgrims on their way home. Poona was one of the first places to suffer, and a vast number died in it. The moment the returning pilgrims reached my field of labour (Sassoor) the disease appeared, and the people began to die. Every village almost had its sad tales to tell."

4. Although, it is to be hoped, since the publication of Dr. Haines' Report, and the special report on Pun-

derpoor by the late President of the Sanitary Commission, much improvement has taken place in the management and sanitary regulations of these fairs, I must yet express my opinion that while in certain years Government should forbid the assemblage of this and similar Juttras, it should at once compel the owners of each shrine to erect rest-houses for the accommodation of pilgrims outside the town or village in which it is situated, and that it should not be lawful for pilgrims to be lodged in houses within the precincts of the town in which the shrine might be, that these rest-houses should be daily sprinkled with McDougall's Disinfecting Powder, that trenches be excavated to leeward of the rest-houses to which male and female pilgrims might resort for natural purposes. That such trenches should be daily filled in by similar trenches dug parallel to them, that an uncontaminated water supply be provided in the neighbourhood of the rest-houses, and that at each celebration of the fair, a Government Official, who might be a medical man, should proceed to the place, where the fair was to be celebrated, at least ten days beforehand, to see that the sanitary condition of the place itself was in a satisfactory state. That this Officer should have under his command a sufficient number of sanitary Policemen, and that he should be temporarily armed with Magisterial powers to inflict penalties on all those who refused or neglected to obey his instructions ; and that above all he should be empowered, in the event of Cholera appearing at the place of celebration, to order the whole encampment of pilgrims to move to any named and suitable spot. I think also that the Patels of villages, situated near the great lines of communications, should be ordered to prevent pilgrims on their return from these Juttras from entering their villages.

5. As you are aware, we are looking forward with the very greatest apprehension to the celebration of the fair at Punderpoor during this season in July.

The Collectors of Sholapoor and Sattara have already notified to the Police authorities in Bombay, that from the scanty rainfall during last monsoon there is a great scarcity of water in the Punderpoor, Malseerus, and Mann Talookas, and that a pilgrimage to the Juttra held at Punderpoor or Shingnapoor will be attended with a very great risk from Cholera. We have done all we can do by having Battakees beaten throughout the Island, and distributing hand-bills notifying the above, but I cannot but think that Government should forbid, on the score of public health, the celebration of the fairs at these places, for until some such action is taken, whatever success we may have attained in destroying the disease in Bombay, all our efforts will be rendered nugatory in one day by the introduction of the poison into the city by pilgrims returning to their houses.

6. I would repeat the recommendations that I made in my annual report for 1866, viz., that *when Cholera is prevalent in the Mofussil*, a sanitary cordon should be drawn between the island and the mainland, and that sanitary encampments should be formed on the three great lines of Railway, say at Lanowlee and Egutpoora on the G. I. P. Railway Company's lines, and at Bulsar or Palghur on the B. B. and C. I., where all persons affected with Diarrhœa or Cholera should be compelled to stop before being allowed to proceed. I should also advise that Apothecaries be stationed at Coorla on the G. I. P. Railway Company's line, and Bandora on the B. B. and C. I., with orders to inspect all passenger trains, and especially all 3rd class passenger trains, and that power should be given to them to detain all persons affected with Cholera or Diarrhœa in buildings to be provided, and that again at Byculla and Grant Road Stations there should be buildings set apart, into which any persons sick with either of the above diseases might be detained. At the station on the line nearest to the place of celebration, Apothecaries should be placed, with orders to

examine every person coming from the direction of the fair, and to detain those that are sick.

At every Railway station there should be kept a supply of Cholera mixture and of McDougall's Disinfecting Powder.

The Guards of every train should be furnished with both these articles, and notices in the vernacular to this effect should be posted up in every 3rd class carriage and station.

Especial care should be taken to prevent over-crowding of 3rd class passengers waiting for the arrival of trains. The privies and ground adjacent to the station should be daily disinfected with McDougall's Powder, and should a passenger die of Cholera *en route*, the body should be taken out at the next station, covered with disinfecting powder, and buried as quickly as possible. The clothes should not be removed, but buried with the body. If removed, after disinfection, they should in all cases be immediately burnt; the bedding, if any, of the deceased, should likewise be burnt, and great care taken about any of the effects of the deceased, which ought to be exposed to a very high temperature before being given up to his friends. For this purpose I would recommend that disinfecting chambers, after the plan I lately brought to your notice, be erected at different places along the line, where such a precaution as I now advocate might be taken.

It should not be lawful for the Railway authorities to allow any carriage to be used in which a person sick with any zymotic disease had been carried until such carriage had been thoroughly disinfected.

7. These are the measures which I would advise should be adopted in the Mofussil, with a view of lessening the risk of importing Cholera poison into the city. I say advisedly lessening the risk, for I am aware

that although great benefit would follow the carrying out of the above measures, yet that nothing short of complete and absolute quarantine will prevent the introduction of undetected Cholera contagion, but regarding such complete isolation as impossible in a city which is connected with the main land by three lines of Railways, and which is the emporium of all the commerce of Western India, and rejecting as utterly impracticable any suggestion to disinfect every bale of cotton, every chest of opium, and to keep all visitors under medical supervision for at least ten days before they were allowed to enter the island, I have been forced to recommend what seems to me to be practicable, even if it may entail considerable trouble.

8. It may be urged that some of the measures above recommended, if carried out, would interfere too much with the liberty of individual pilgrims, but I cannot help thinking that if pilgrims will not listen to the force of argument, they should be compelled to listen to the argument of force, and as Government is at a vast expense to protect the lives of the community from violence, and its property from robbery, that surely the religious fanaticism of a few thousands of one particular section of it should not be permitted to imperil the lives and welfare of tens of thousands of their fellow subjects, embracing all castes and creeds, besides saddling our City with the reproach of being the great starting point of this terrible disease to the more civilized quarters of the globe.

9. Although it has been shown that these Juttras exert the most fatal influence as regards the production of Cholera in this Presidency, yet much remains to be done in the City itself to prevent its presenting, as it now does, a favorable home for the reception and propagation of the poison when imported. Doctor Leith, in his last Mortuary Report says:—"These returns have shown that here (Bombay) as elsewhere, Cholera is severest

in low humid localities, and that it is even more favoured by humidity than by the foul air of densely peopled but drier districts."

I need only refer to the report of Dr. Farr, of the Registrar General's Department, in which he shows that Cholera is essentially a low level disease, and that elevation of soil has a most marked influence in the mortality from it. His observations tend to prove that the very slightest rise in soil makes a most striking difference in the mortality.

In the case he quotes, viz, the Cholera outbreak in London in 1849, the first ten feet of elevation made a difference of more than 70 per cent. in the number of deaths.

According to Pettenkofer, to quote from Dr. Simon's Eighth Report—"The best soil for Cholera is a porous soil, easily penetrable by air and water, and in which water is to be found not far below the surface, and which is foul with excremental matters, and the times when such a soil is aptest to multiply Cholera contagion are times when the water level in it is falling after having reached an unusual height. The degree in which a given Cholera contagion when imported produces epidemic results is essentially determined by the degree in which its importation coincides with the fulfilment of those conditions of place and time."

10. The conditions that Pettenkofer lays down regarding soil are fulfilled in the calcareous sand, littoral concrete, and tertiary clay shale of Girgaum and Chaopattee, the districts which over and over again have been pointed out by Dr. Leith and other observers as the districts in which Cholera is always most severe in its ravages in this city.

The population of Girgaum, including Khetwadee and Chaopattee, amounted in 1864 to 56,866 persons.

In these three districts there are 345 privy cesspools, and 589 wells from which drinking water is drawn, and about 50 of which are within 25 feet of privies.

There is but the barest attempt of surface drainage, and this only along the sides of the roads. Only 774 Vehar water connections have been made, so that the drinking water of the inhabitants of the entire district is chiefly derived from wells dug in the calcareous sand. Now allowing the exceedingly moderate sum of 10 gallons per head, we have five hundred and sixty-eight thousand six hundred and sixty (5,68,660) gallons of water per day soaking into the ground around the habitations of the residents, and this calculation does not embrace the water used for all the horses and other cattle kept in this district, nor for irrigation purposes, so that it is not difficult to conceive how impure the water in the wells must be, and how extensively it becomes contaminated when the poison of Cholera is introduced.

The ventilation of the district is impeded by the existence of the belt of cocoanut trees along the shores of Back Bay. Dr. Leith has pointed out how injuriously this obstruction acts, and it is much to be wished that his wise recommendations were carried out. The poorer classes in this district for the most part reside in cadjan huts under the cocoanut trees, their ablutions are performed in the immediate vicinity of their huts, and they find it convenient to drain their refuse water to the bottom of any tree near their huts to be absorbed as best it may, and when the ground around any particular tree becomes too swampy to allow of approach to it, the drain is cut off and directed to another tree. The privies usually consist of spaces enclosed by matting, and are generally in close proximity to their huts. The filth from them indubitably finds its way into the wells which abound here. In 1864 I saw on more than one occasion persons in this district ill with Cholera, passing rice

water stools close to their huts adjacent to wells. I am carrying out Dr. Leith's suggestions of having a register kept of the levels of the water in the wells to see if any change takes place in them before an outburst of Cholera according to Pettenkofer's theory. The existence of gardens in this low lying district is also conducive to the spread of Cholera and the dangerous emanations from the grave-yards along the shore of the Back Bay have been alluded to by Dr. Leith as one of the reasons why Cholera is always severe in Girgaum and Chaopattee. There are three other districts in this City entirely without drainage, viz. Funneswady, Caval (which Dr. Leith well describes as a dirty and irregular labyrinth) in which there are 125 cesspools and 86 wells, and Mazagon which contains 148 cesspools and 53 wells. The Mortuary Reports since 1849 prove that it is in those districts of the City which are entirely unprovided with any drainage and where cesspools abound that Cholera is specially virulent.

11. The obvious remedy is to drain each of these localities, but whilst we are waiting for the interminable question of the drainage of this City to be settled, Cholera will be upon us some day, and finding us still undefended in our most vulnerable point, will infallibly add thousands to the large army of victims it has already consigned to a premature and unnecessary death. I cannot help expressing my strong conviction that immediate action should be taken without waiting for the solution of the drainage question, and that for each of the above districts at all events the system of disposing of this question that obtains in Holland be immediately adopted. I refer to what is known by the name of Captain Liernur's system of aeriform sewage, a system which provides for every particle of night soil being removed from amidst the habitations of man without any possible contamination of the soil in their vicinity.

To understand the details of this system, I attach the accompanying article by Mr. Bridges Adams, which I have had copied from the February Number of "Once a Week."

I have specified four districts as being absolutely without any drainage at all, yet I wish again to draw most earnest attention to the so-called drains that exist throughout the Native Town.

Those in Camatteepoora have been constructed at such a dead level that water will not flow in them. It is impossible to keep them properly clean. The existing drains, *i. e.* paved channels for the carrying off the wastage water from the houses in the Native Town should be immediately improved without waiting for any ulterior scheme which would propose to remove the night-soil as well. I cannot press this point too strongly.

12. As regards other measures, I would strongly recommend that public fountains with Vehar water be erected throughout the City, and especially in the districts above noticed, and that the existing impure dipping wells be abolished, and fountains substituted for them. Water absorbs any impurity that the atmosphere may bring over it, and it appears to me to be a matter of great marvel that after going to the immense expense of creating a source of pure water supply for this City, and after bringing it in a pure state to our very doors, the authorities should then do all in their power to make this water a means of disseminating, not health, but disease. The highest sanitary authority in this Presidency has, in his own forcible language, pointed this out, and I can only bring the matter again to your notice, trusting that it may be remedied.

13. Another point that should be attended to is the provision of a sufficient means of drainage to carry off the wastage water from places where Vehar water is laid on.

At present the condition of the ground around almost every Vehar water standard for filling the watering carts is most unwholesome, and infallibly will tend to promote the spread of Cholera. I have had the leakage from some of these pipes measured. From the one next to Mr. Cursetjee Furdoonjee's House, Middle Colaba, upwards of 4 gallons per hour escapes, at this rate nearly one hundred gallons per day run to waste from this one pipe alone. An attempt has been made to drain the standard on the Esplanade near the turning down to the Staff Lines. It prevents the grass being turned into an unwholesome puddle, but it fails to prevent the road being constantly wet. The ground where the carts are filled should either be paved on a slope leading to the drain, or an open iron grating should be placed for the carts to stand on so that the water might flow off at once.

14. As I before recommended, I advise the erection of at least three disinfecting chambers on different parts of the town, where the bedding, clothes, &c., which may have been fouled with Choleraic discharges might be exposed to an exceedingly high temperature if it were not thought safer to burn them.

15. I wish to direct particular attention to that portion of the extract from Dr. Simon's report, in which he says that the quality of infectiveness belongs particularly, if not exclusively, to the matters which the patient discharges by purging and vomiting from his intestinal canal, that these matters are comparatively *non-infective at the moment when they are discharged*, but subsequently while undergoing decomposition acquire their maximum of infective power, &c. From this it will be seen that it is a matter of the very highest importance to be prepared to disinfect these discharges as soon after they are passed as possible, as every minute that elapses after they have been evacuated renders them more intensely dangerous.

At present I am compelled to wait until I receive the report of a death from Cholera from the burial ground, and frequently more than 30 hours elapse before such report reaches my office; so soon as it does no time is lost and immediate intimation is sent to the European Inspector of the Ward in which the deceased died, whose orders are to leave off any other work he may be on and to take to the house McDougall's disinfecting powder to lime-wash with the powder the room in which the deceased died, to sprinkle his clothes and bedding with the disinfecting solution, to disinfect the ground adjacent to the house, and especially the privy, cesspools, drains, &c., and to report if the house requires lime washing internally or externally. If the privy is near a well for drinking purposes, the people resident in the house are warned to abstain from drinking the water in it for six weeks.

This course does do good, but it does not do the good it might, and I cannot hope to stamp out the disease, because I do not get sufficiently early intimation of the deaths, and this I cannot do until the present system of Registration of Deaths is put on a more satisfactory footing. Were my suggestions of having full-time men carried out, the Registrar would receive news of the death before the body was disposed of, and would immediately notify the same to the European Inspectors. This would effect a saving of many invaluable hours, and it ought to be done.

16. In times when Cholera is epidemic in this city, I should advise the formation of temporary additional Dispensaries and of Cholera Hospitals in every ward; trying, if possible, to select the staff of attendants from amongst those who have had Cholera before.

Persons sick with Cholera, and residing, let us suppose in Girgaum, will not consent to be taken to the Jamsetjee Jejeebhoy Hospital, even if it had sufficient accommoda-

tion for large numbers, as they reasonably object to the distance.

Cholera Hospitals would afford a splendid opportunity for trying different kinds of treatment, and we might be able to deduce most important results from the experience thus gained.

In conjunction with the establishments of these Hospitals there should be an organised system of house to house visitation by medical men. Other measures, such as the addition of carbolic acid to the water in the water carts would naturally suggest themselves.

17. The only point that now remains to be dealt with is the question as to the best means of preventing Cholera being exported from this City.

It is a matter of some interest to consider that as Cholera is principally generated in India through the fanaticism of one caste, so it is principally exported from India through the superstition of another.

During the cold season of each year, there is a steady influx of Mussulmen from Persia, Muscat, Kurrachee, and other parts of India, who are anxious to make the Haj.

Most of these persons are in very poor circumstances, many of them are beggars, who have collected just sufficient alms to pay for their transit fees; on arrival at Bombay these persons are in the habit of frequenting the Mosques, in the courts of which they lie about and live as best they can, supported by charity. They frequently import small-pox into the City, and are more filthy in their persons and appearance than it is possible to describe.

They are then ripe for disease when they embark, and hitherto the over-crowding in ill-ventilated ships has doubtless fostered the development of Cholera amongst them.

The suggestions contained in paragraphs 19, 20, and 21 of Dr. Leith's Report on the Cholera Conference regarding this question seems to me to embrace every point that is requisite, although it has struck me that it would be safer to require every intending pilgrim to perform a quarantine of 10 days on one of the islands of the harbour, where information regarding family residence, &c., antecedent circumstances of each pilgrim, could be registered before he would be allowed to be taken on board, and that every vessel, however small, if conveying pilgrims, should be compelled to carry either a qualified or unqualified medical practitioner, and to lay in a certain stock of medicines and McDougall's disenfecting powder.

These arrangements would naturally come under the supervision of the Health Officer of the Harbour.

18. In concluding this report, I would respectfully suggest for the consideration of Government whether it would be practicable to have in each Collectorate of this Presidency a Halalcore Battalion, which should be constantly employed in cleaning villages, first along the line of roads of communication over which troops pass, and secondly, in villages and towns throughout the country.

It has struck me that a city or village might be fined by having a company of Halalcores quartered on it whose maintenance it should be bound to find, and who in return would put the village into a thoroughly clean state.

I have the honour to be,

Sir,

Your most obedient Servant,

T. G. HEWLETT,

Surgeon, Bombay Army,

Health Officer, City of Bombay.

P. S.—Since finishing the above report I was informed by Colonel DeLisle that there was a full account of Captain Liernur's system published in the 7th December's number of the "Engineer." I have therefore had the article copied out, and submit it, as well as the other one; as this seems to me to be by far the most scientific scheme for dealing with the question of sewage on economical and sanitary grounds.

Its adoption even in the four districts I have named, would enable me to prove not only that it affords complete security against the contamination of air or water by the decomposition of night soil, but I could then demonstrate what I have so long advocated, that it would be possible to make a profitable manure out of the night-soil of this city.

In my letter No. 1, dated 30th May 1863, to the address of the Clerk to the late Municipal Commissioners, I proposed "that at the Chinch Bunder Tanks there should be some such arrangement of Engine Pumps and Agitators as is carried out at the Carlisle Irrigation Works. The two tanks would then be converted into one, and the night soil deodorised with carbolic acid, and driven out into large flat iron boats or reservoirs, when if liquid manure was not desirable, it might, by a very simple arrangement, be reduced to a powder by the application of artificial heat.

"The powder could then be conveniently packed in gunny bags and sent up-country with directions to be sprinkled over the khets previous to irrigation. By this method I think many of the objections that have been raised relative to the difficulty that is likely to arise from prejudice in the native mind would be obviated."

I now again bring this subject forward, and trust that you will do me the favour of submitting it to Government, as I believe this scheme is likely to prove a practicable, effectual, and economic disposal of the drainage question. A question than which there is none more vital for the interests of this City, and which appears to me to be as far off solution as ever.

I forward two specimens of pure night-soil and urine reduced to a powder. One I had mixed with a small quantity of McDougall's Disinfecting Powder (sulphite of lime and magnesia and carbolate of lime), and the other with a solution of carbolate of lime. My appliances were of the rudest description, and from these experiments I can form no proper idea of the cost of dealing with large quantities, but as you are aware an application is about to be submitted for sanction to conduct an experiment on a larger scale after the monsoon, when I shall hope to show the extraordinary fertilising properties of this manure. I have therefore refrained in this paper from enlarging on the details of the method I propose to adopt in dealing with the night-soil, should this scheme be sanctioned; but I am prepared to submit an especial report on this subject if required.

T. G. HEWLETT.

Whilst this report has been passing through the Press, I have received from England the latest work on the subject, entitled, "The Sewage Question;" by Mr. Krepp, in which the author proves the advantages to be derived from the adoption of Captain Liernur's system. I note with satisfaction that in new Zealand, Australia, and elsewhere measures have already been taken to introduce this, the most perfect, most economical, and most scientific method of dealing with Sewage. Should Government desire it, I will with pleasure forward the book, where the advantages I have spoken of can be perused at length.

Extract from "Once a Week," January 12th, 1867.

AERIFORM SEWAGE AND CAPTAIN LIERNUR'S SYSTEM.

Our modern system of dealing with excreta in towns very closely resembles the practice of our savage ancestors. Ancient towns were very commonly situated on streams and rivers for the convenience of water supply and also of transit, together with the advantage of fishing, and nothing would be simpler and easier in dealing with excreta than tumbling them into the stream to be washed away from their doors and consumed by fish. The ancient Lacustrian people were peculiarly happy in this arrangement, and it answerd very well so long as the inhabitants were not numerous. Our variation on their practice consists chiefly in making underground holes to convey the sewage to the streams. One of our modern Sewage Doctors once witnessed the washing out of a mass of dirt from a water reservoir by a stream from the pumping engine, and after calculating the hours of labour which it would have taken to do it by hand, proclaimed far and near that "water is your only cheap carrier." He forgot the water had carried the dirt into the reservoir as well as out, and so he urged the increased use of water; and having been told by Smith of Deanston of the wonder working "foul burn" of Edinburgh and its green crops, he thereupon concocted a theory that all sewage should be diluted to an unlimited extent, the more the better, and that this 100,000th dilution should be flooded on to the land. All the lore of Liebig was pressed into his service, cesspools were denounced, and sewers required to be thoroughly washed out.

And so the river was flooded with sewage, which deposited its solid matters on every shore and bank, to be stirred up and churned by every paddle that passed. So long as the weather was cool, this did not signify much; but with summer's heat, acetous, and then putrid, fermentation set in, with a stench that found out the legislators in their Westminster Palace, and induced them to commit the sewers to the charge of the Board of Works. Now one advantage in the river arrangement was, that the various gases evolved during the heat rapidly escaped into the upper atmosphere to be neutralised, and when the heat ceased the gases ceased to be produced. But when the Board of Works closed the sewers to the river, and confined the sewage to their large new brick tubes, they provided a perennial summer atmosphere, to keep the fermentation constantly going, and as dirty gas runs up hill while dirty water runs down hill, the streets and houses now get constantly flooded with noxious vapour, which in many cases forces its way with strong pressure through water, and forms a gilded pule on the surface of the water traps, and so long as this system continues, the only existing remedy is to build tall chimneys like those of manufacturing towns at every half mile along the course of the sewers, and this the Board of Works will put off as long as possible, because it would be a practical acknowledgment that they had not been foreseeing, and had to amend an error in their programme. The egotism of individuals is bad enough, but the egotism of Boards is insuperable. They would fain appear infallible, and to acknowledge an error, is to proclaim that they also are but men. The truths of nature are to them demonstrable fallacies when their infallibility is called in question.

The present writer has always considered the carriage of sewage in water a fallacy, and has frequently pointed it out; many common sense people have arrived at the same conclusion, and one of more than common sense had arrived at the same conviction in ages long past, when he enjoined on every man in the Israelitish camp to have a paddle or spade on the end of his spear. The Reverend Mr. Moule of Dorchester has been advocating the use of earth closets in opposition to water; and

he is right so far, though the structure of buildings in this great city is not well adapted for carrying away the mass of refuse; one valuable quality this system has, the prevention of fermentation, which is the great source of nuisance, and this fermentative or putrefactive process carries off a large portion of what is valuable as manure. If fermentation were carried on to exhaustion, the residue would be scarcely of any value.

Sewage is compounded of many substances, animal and vegetable, together with the refuse of living bodies, but none of these are noxious till putrefaction begins, and the great element for inducing putrefaction is water. If the substances be dried no putrefaction takes place, a fact long known to manure manufacturers; and the old cesspits were theoretically better than the modern sewers, inasmuch as they were not supplied artificially with a mass of water, the solid matter sinking to the bottom and the liquid running away; not a desirable condition of things, because percolation through the earth conveyed poison to the water springs.

Decaying vegetables and the refuse of stable-yards are easily handled, and are carted away because they are of sufficient value to pay for the labour; so are decaying flesh and bones; were they not of value, there would be one obvious mode of dealing with them, carrying them to the gas works and burning them under or in the retorts, so also might solid fæcal matter be treated, for it would make good gas as well as animal charcoal, a very valuable product. But how to obtain it solid without considerable cost in preparation is the difficulty.

Every day is brought into this huge city, in addition to permanent materials, fuel and food, both solid and liquid, for the purposes of consumption, and they are brought chiefly by wheeled vehicles, water carriage being but a small part, save in the case of coal and water; all these things are by the processes of burning and digestion reduced to a very small comparative bulk and weight, save only the water, which remains the same. The ashes

remaining from a burned ton of coal are very small, comparatively, both in bulk and weight, and the average solid refuse from human bodies is but one quarter of a pound each average person per day, or 335 tons per day, equal to 547 cubic yards in bulk for the whole of London. The fluids from the same source average one lb. and a half daily, or 2,000 tons per day, equal to 562,500 gallons, or 5,207 butts, about 3,280 cubic yards in bulk.

These two substances then are the real source of sewage nuisance, the solid forming in volume a cube of $1\frac{1}{2}$ feet, and the fluid a cube of about 9 feet per annum each person, total $10\frac{1}{2}$ cubic feet.

The dry ashes of the coals thoroughly mixed with the drained solids would perfectly neutralise them, and render them innoxious, and fit for manuring clay lands and corn land. The coal ashes in any case must be conveyed away, and the solid fæcal matter added to them would not appreciably enhance the cost. The fluids might be run into close reservoirs, and pumped into barrels for transport, and, freed from the bulk of water now used, the cost of conveyance would become a minimum.

In the olden time, the practice was to have a cess-pool large enough for the accumulation of seven or ten years. In Paris it extended to half a century. During all this time the valuable portions disappeared in gases more or less noxious, whenever the surface temperature was sufficiently high; now, inasmuch as fermentation does not usually set in till the fourth day after deposit, it follows that if the deposits were taken away day by day, or night by night, in the same proportions as the fuel and food which are their bases are brought in, there would be no nuisance or waste.

But to do this with the present structure of London dwellings seems almost an impossiblity. To have as pleasant and convenient a room as possible to sit in was always considered to be more or less a necessity, something very inferior was considered quite good enough to sleep in, the provision safe, and pantry needed some-

thing of light, and facile access, and absolute necessity enforce some provision for ventilation. The wine cellar also was, perforce, dry and tolerably warm and accessible under lock and key, but the names dust-*hole* and coal *hole*, clearly imply that any dark cranny was considered good enough for them, the space under the lower stairs, even in decent houses, being their locality ere the invention of cellars under the pavements. The refuse of food, it was thought, might be put away in any dark corner.

It is a maxim with all good housewives to have no dark corners about the house, "slut holes," as they were anciently termed, and it will be only when every portion of the house, both for the fresh provisions and the consumed provisions, are equally accessible to air and light, and open to examination, that the evil will be remedied. This means the alteration of most of the houses in London. Rich people, who keep carriages and horses, would think it very objectionable to have all their stable manure carried through the hall-door, and so their houses are commonly built back to back with a mews between them, and nothing can well be neater than the mode in which the refuse litter and stable manure is piled up each day in the open air, ready for carrying away. It is not thought advisable to keep this in a dark hole, and consequently being before people's eyes, there is no neglect in carrying it away before it begins to ferment.

These difficulties having been under the consideration of Captain Liernur, an Engineer of Holland, have led him to devise a new system for the conversion of the present water butts into air closets. It has long been a practice in various cities on the Continent to empty cesspools by means of a vacuum chamber carried on a waggon. The vacuum is produced either by an air pump or by an injection of steam, or by having spirits in the chamber; a metal pipe, with a stop cock, leads from the chamber to nearly the bottom of the cesspool, connected by a hose. The vacuum being complete, and the

stop-cock opened, the matter rushes up and fills the chamber without any need of hands; but this plan can only be available for a large mass of matter six or twelve months' collection, with nuisance during the period, unless mixed with earth, or coal dust, or other absorbent material, which would render it impracticable to empty it by the hose and vacuum, the Captain's plan is to empty every receptacle nightly.

The principle consists in discharging the fluids and solids through a large opening without a bottom, or valve, so that they may fall into a vertical pipe of cast iron, forming a curve or syphon in the ground, both ends of the pipe being open, the upper one above the house roof, and the other in the drain pipe in the centre of the streets, where they intersect each other. At the intersection is placed under ground, an air-tight wrought-iron vessel, some five feet in diameter and three feet in height, and this vessel receives four drain pipes from the streets; a stand pipe from this reaches nearly to the bottom, and rises to the level of the street. The whole system is air tight; a portable or traction engine is run over the opening of this pipe, and a vacuum is formed by an air pump worked by the engine; air valves are then opened in succession to every house connected with the cistern, and the atmospheric pressure drives both fluids and solids out of the closet pipes into the cistern. From this the exhaust carries it into a close barrel or tank, on the engine, and it is taken away to a railway station, or wharf, where it is discharged into barrels by a similar process for transport to cultivated lands; the dwellers in the house knowing nothing of it save that they have no fermented gases, and that the whole of the closets are swept out every night by a strong current of wind, while there is no valve to pay for or leaky pipes to overflow. The whole of the pipes are of one diameter, about five inches, and there is therefore nothing to stop the free passage of any thing that gets

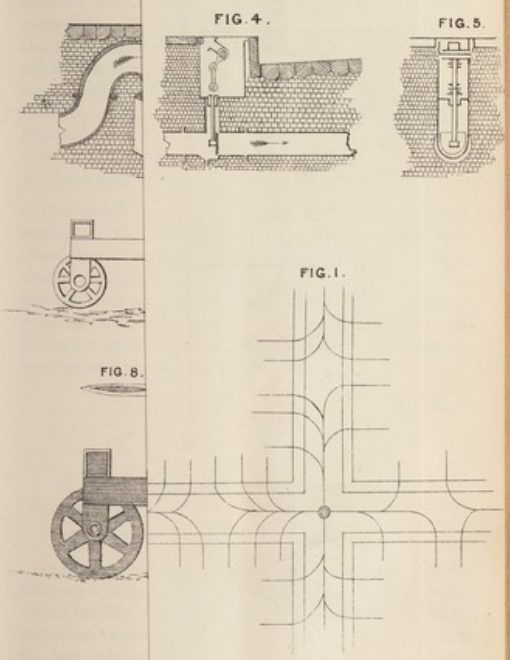
into them. The details have all been carefully considered by Captain Liernur, and experience seems to prove that the fæcal matter does not induce rust inside the pipes, but rather sheathes them, so that only the outsides need special guarding against rust. Captain Liernur, who is a member of the Royal Institution of Engineers of the Netherlands, is now engaged, in conjunction with Mr. Petersen, the City Engineer, in applying this system to a special district of the Hague, where the low level renders any ordinary system of sewage impracticable.

To bring any new system into use, requires the setting of a careful pattern in successful work; now this plan is especially adapted for division into localities independent of each other. It is, therefore, particularly worthy the attention of builders engaged in new localities erecting squares of buildings, or villas, where main drainage does not exist, and even the ordinary water supply depends on wells. For the larger country dwellings of noblemen and gentlemen, where a fixed or portable steam engine is at hand, every drain may be rendered perfectly free from gases. The whole system is independent of levels.

Towns situated on the sea side cannot possibly be made wholesome by a system of water-closets discharging into the beach or into the sea, and becoming a nuisance to bathers; but upon this system they may be wholly freed from nuisance, and without dealing with the enormous volume of dilution required in the water-closet system.

Captain Liernur's calculation is that one steam-engine of from ten to twelve horse power, such as are now common in agricultural districts, with three tenders, each of 90 cubic feet capacity, with about half a dozen men working from seven to eight hours nightly could dispose of the excreta of 10,000 inhabitants, say 1,000 houses, in a concrete form, unmixed with water, and weighing about six tons. At this rate it would need

PARATUS.



PNEUMATIC SEWAGE APPARATUS.

FIG. 2.

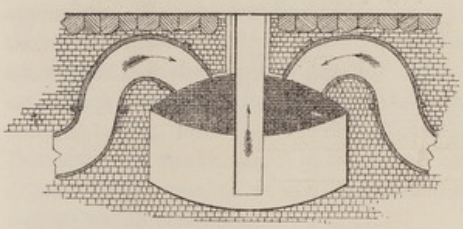


FIG. 3.

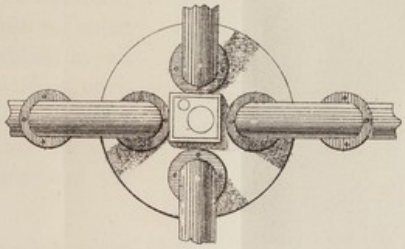


FIG. 4.

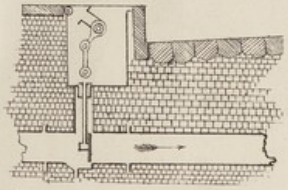


FIG. 5.

FIG. 7.

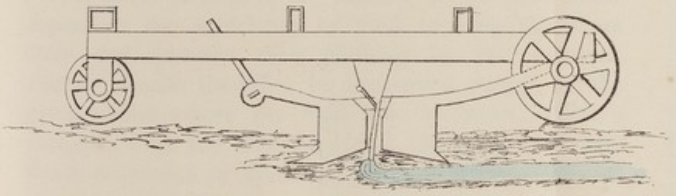


FIG. 1.

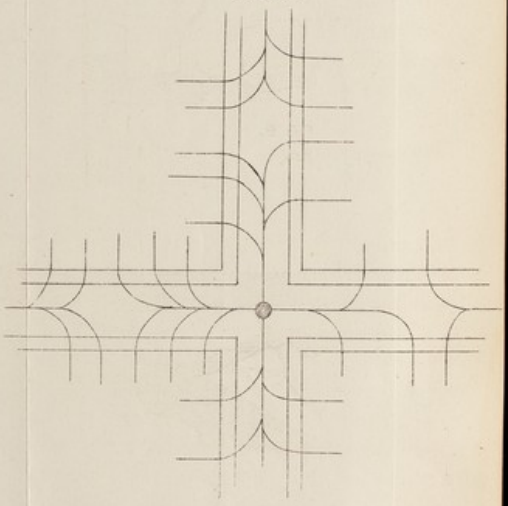
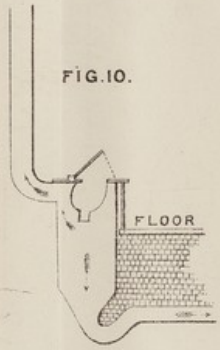


FIG. 8.

FIG. 9.

FIG. 6.

FIG. 10.



300 steam engines and a corps of 2,000 labourers to keep all London cleansed, supposing it effective, but it would be really cleansed and free from gaseous posion, and the manure of the value of which we have heard so much, and of which we know that in Belgium it exceeds ten shillings per head, is put into a saleable form, which every farmer can recognise and appreciate. But the first thing is to get rid of a nuisance, the next to make a profit of it if we can; though we must not assume that the excreta are of equal value in all cases; rich manufacturing cities yield more than agricultural towns, and Roman Catholic towns less than Protestant, for the reason that the value of the manure depends on the quality of the food that is eaten and the surplus which remains.

It is quite clear that the success of the plan must depend on the sufficiency of fluid to keep the pipes clean and prevent their choking by the material, contact should therefore be prevented except at the ground level where the fluid lies, as in an ordinary cesspool, with the difference that the cesspool is only five or six inches in diameter, and the whole contents are removed nightly.

(Signed) W. BRIDGES ADAMS.

Extract from the "Engineer," dated 7th December 1866.

THE PNEUMATIC SEWAGE SYSTEM.

In our last impression we described at some length the different systems of sewage employed in the principal towns on the Continent. From that article it will be perceived that the pneumatic system, or, in other words, the principle of exhausting air from a vessel in communication with the sewage to be removed, is extensively

used. We also stated that a system of sewage had been newly proposed, and, we may now add, tested, by which the defects of the pneumatic arrangement as now used appeared to be obviated. This system is the invention of Captain Liernur, of Holland, and it is at once so novel in its details, and so startling to English Engineers in the mode of applying the principle, that we have been at some pains to place it before them. The following description has been supplied to us by Captain Liernur, and is sufficiently elaborate to render the nature of the scheme perfectly comprehensible:—

This system includes with an improved method of collecting, also one for utilising, fæcal matters. The novelty of the first named process consists in the use of atmospheric pressure for cleaning and emptying closets of their excreta, instead of the large quantities of water thereby wasted in our present sewer system, and in the second process in the daily utilisation of the manure by means of immediately mixing the same with the topsoil of the grounds which constitute the sewage form, and which must for that purpose be under control of the corporation engaged in the works. The object of the combined project may be stated to be five-fold:—(1st) To remove effectually from towns an intolerable nuisance in the shape of fæcal matters and gases which are known to have a most detrimental influence upon the public health, and to effect such removal without annoyance to or hindrance from the inhabitants; (2nd) to prevent the present excessive waste of water in our water-closets; (3rd) to reduce the transportation of the excreta to its lowest possible cost, by leaving them unmixed with water, earth, chalk, coal, turf, and other materials which are known to have occasioned, whenever employed, a greater outlay than the value of the diluted compost obtained; (4th) to keep intact whatever fertilizing elements are contained in excreta, and not to allow any losses to be incurred by the usual dilution which gives the manure

such a form as to make it unfit for application on all arable land and most meadows, nor to allow the excreta time or opportunity to go over in fermentation, which they are known to do in from three to five days after their production, the fermentation resulting in the formation of volatile gases poisonous to animal life; (5th) to let the soil which is to be manured itself do the work of decomposition or disintegration of the raw materials, and convert it into fertilizing elements having such forms and combinations that the plants can take them up into their roots without injury.

Every Engineer and agriculturist at all conversant with the subject will know that these points represent fully all that can be desired in a perfect sewerage scheme, and, that when the mechanical arrangements whereby these ends are proposed to be accomplished are practical in the full sense of the word, there can be no doubt that a solution of the vexatious sewerage question has been therein found. Fig. 1 represents a street crossing; centrally under the pavement is a reservoir from which main pipes lead in the streets. From these main pipes branches lead up to the closets in the buildings right and left. Main and branches are 5″ cast-iron socket-pipes, and the reservoir (shown in plan and section in Figs. 2 and 3) is made of boiler plate, and is some 3 feet to 4 feet in diameter, depending upon the number of dwellings in the group; it is furnished with two vertical pipes, one of 3″ diameter for exhausting the air, and the other 6″ diameter for exhausting the fæcal matter collected in it during the scouring process, for which purpose it reaches nearly to the bottom. Where the branch-pipes cross the side walk, air-tight fitting side valves are placed under it, as shown in section at Figs. 4 and 5. These are so arranged that they can be opened and shut at will by an operator in the street. For this purpose he must lift the cover up off the casing containing the lever, and fit an iron stem or handle, which he carries with him, into the upper socket,

without this lever the valve cannot be opened; and the cover, which lies level with the side walk, is so adjusted that it cannot be closed unless the valve is closed also, this is for the purpose of drawing the attention of the labourer in case he has omitted to do his work properly. The lower end of the valve is case-hardened and sharpened to a chisel-edge, so that if any rags or old shoes, or such like matters, which often find their way into sewers, should by resting against the rabet of the valve seat remain fixed there, they will, by being cut through, not prevent the closing of the valve. The arrangement shown in the drawing is for countries where during the winter the side walks are not apt to be obstructed by much snow; where snow is heavy, the valve stem is prolonged, and the casing containing the lever fastened against the front or side of the dwelling, or against the fence, a foot or so above the side walk in such a manner that the operator can work the valve without removing any snow. The house closets are very simple in their construction (see Fig. 8), having instead of the ordinary basin and its valve and water-trap only a short pipe without bottom and made of strong glazed stoneware; this is placed in a vertical pipe so wide that matter dropping from the basin cannot soil its sides, which are thus kept clean, because there is nothing to make them dirty. This pipe is contracted below to a diameter of 5", and curved so as to form a cup or trap, from which with another upward bend, it leads to the branch-pipe containing the side-walk valve. Into this narrow curved portion or cup the excreta falls perpendicularly and free from the basin above, collecting together until the moment of removal. In the rear of the basin, and connecting with the wide vertical pipe, is another 5" pipe which is prolonged upwards to the roof of the house like a chimney, and serves not only for the admittance of the air which effects the scouring, but also for carrying off by ventilation the offensive watery vapour which rises from fresh excreta; the current of air which effects this, setting in downwards through the basin opening;

whenever the cover is removed, and thence upwardly through the chimney-pipe to the upper air; drawing then somewhat like a stove when the door or valve is opened. It is evident that a basin so constructed can never present the disgusting spectacle which water closets always do when the valve gear is out of order, or the water fails, as happens so often among the poorer classes, for here there is nothing to retain the matter, and thus nothing to soil. The basins are fitted closely in the wide pipe, so that they can be removed and cleaned like any other domestic utensil.

At night a steam-engine driving an air-pump is placed over the reservoir in the street, having behind it a tender carrying a cylindrical shaped vessel of some 90 cubic feet capacity to receive the contents of the reservoirs. The boiler of the Engine is upright and of the water-flue pattern, with a furnace arranged for burning mixed coals and gas-coke. Air-holes supplied with steam nozzles pass through the water space above the fire to prevent smoking. The engine stands upon three wheels, the single front wheel being furnished with shafts to its pivot-gear for one horse to work in, which is more required for steerage than for pulling. The boiler is placed between the front wheel and the rear or driving axles. The steam cylinder and air-pump work conjointly, vertically, one piston-rod uniting both pistons, and are securely fastened against the rear side of the boiler. The air-pump is 16" stroke and $16\frac{3}{4}$" diameter, equal to a capacity of two cubic feet, and as the pistons are driven to a speed of 100 double strokes per minute, developes in that time a space or volume of 400 cubic feet. To promote regularity of working, a connecting rod working from a small cross-head on the piston-rod gives motions to a crank axle and fly wheel. This axle, besides working the eccentrics for the two cylinders, also can be geared by means of cog-wheels to the rear axle of the carriage, furnishing thus the motive power to

transport the engine and tender from one reservoir to another. While the air-pump is working this running gear is unshipped. The exhaust steam passes through the hollow frame of the carriage before entering the chimney, so as to soften the noise of the blast; and the exhaust air is led in the boiler furnace for the purpose of purifying it. The boiler is fed by a Giffard injector. The tender is also carried upon three wheels, the two rear ones being large and carrying three-eighths of the weight, while the front wheel carries but one third, and serves for steerage. On top of the front end of the cylindrical receiver, immediately behind the driver's seat, is a dome, from which two 3″ pipes project, both having well-fitting valves; one of these can, by means of a flexible hose, be connected with the air-pump, and the other in a similar manner to the air-pipe of the reservoir under the pavement. At the bottom of the tender under the dome are two soil pipe-couplings, one on each side, which are also provided with good valves, and can be connected by means of a flexible hose to the 6″ suction pipe of the reservoir. The rear end of the receiver is partitioned off so as to form a tank of 10 cubic feet capacity for feed water for the engine. The fuel is carried in two baskets hooked on behind. The engine and tender having been connected in the above described manner to the street reservoir, the scouring process is as follows:—

The air pump, exhausting the air from the tender and the subterranean system of pipes and reservoirs at the same time, is kept at work until the vacuum gauge indicates the pressure within to be reduced to about one-fourth atmosphere, this partial vacuum of course extended up to the side-walk valves. The number of minutes required therefore depends of course upon the number of cubic feet contained in the sewer pipes, reservoir, &c. For an average group of dwellings around a street crossing this would amount to about 1,200 feet. The side-walk valves are then, one at a time, opened and shut immediately again. In order that this process should not occupy too much time it is done by two labourers working alternate valves, and continually passing each other. During this operation the air pump continues to work so as to maintain the proper internal pressure. The result of the opening of such a valve is of course that the air entering through the chimney-pipe forces the matters collected in the bend below through the street pipes into the reservoir, and does this with great violence, taking along whatever gases or vapours might be present.

To form an idea of the force here applied, it must be remembered that a hurricane, such as tears trees up by the roots, upsets buildings, and drives them before it, exerts a measured pressure of 50 pounds per square foot, or about 0·35 pounds per square inch; the pneumatic force applied to remove the small quantity of excreta collected daily in the lower part of the closet-pipe being about 11 pounds per square inch, is thus equal to about thirty-one hurricane winds concentrated in one. Repeated trials have shown how effective a mode of cleaning this is, and that the last vestige of dirt is utterly swept away by it as if by magic.

After all the side-walk valves belonging to the street-crossing have been turned once and shut again air is admitted into the reservoir by taking off the small flexible hose, and the valve of the large hose, connecting with the soil pipe opened, which causes the contents of the former to be at once discharged into the tender, all connections are then cast loose, and the engine with tender brought by its own motive power to the next reservoir, thence to the next, and so on until the tender is full. An empty tender, carrying only fresh fuel and water for the engine, brought there by a couple of horses is then exchanged for the filled one, which is immediately carried by the same train to buildings called "decanting stations." Then the manure is decanted into barrels, which are without delay shipped to the sewerage farm.

To cheapen transportation the buildings should be located in the immediate vicinity of a railway station or steam boat landing. During day-time the buildings serve for shelter for engines and tenders, stabling for the horses, fuel, feed, &c.

Upon its arrival on the land the manure is at once mixed with the soil by means of ploughs (see Fig. 6), which carry a filled barrel on a light three-wheeled waggon placed over them. A short pipe, fastened with leather straps over the bunghole of the barrel, discharges the manure in the furrow made by the ploughshare and is immediately afterwards covered again with loose soil by a sort of shovel trailing behind. This soil, having before been turned over by the ploughshare, lain on the surface, contains of course abundance of oxygen, which in coming thus in contact with the raw manure, rapidly decomposes it into elements which are absorbed by the earth on all sides. These elements are ammonia, carbon, phosphates, soda, potash, &c. or solutions of them, which the plants can take up by the capacity of selection due to their particular organism. The manure, composed as it is of fæces and urine in proportion of about one to nine, contains just water enough for forming such solutions. The mixture also flows evenly out of the barrels, having by the violent action to which it was subjected in the pipes, reservoirs, and tenders, been reduced to a sort of thick liquid of uniform consistency. The particular field to which this manure is to be applied depends upon the management of the farm; with rotating crops there is generally some field bare and ready for manuring, and crops can mostly be so worked, that while one field has growing plants, another is in a state of preparation. If, however, during spring time all the fields should be occupied, the manure is to be run in drills between the rows of the plants, they being for that purpose laid off in alternating planting beds and manuring paths of the respective width of 4 feet to 2 feet.

These paths are also very convenient for weeding. The following season these paths are to be reversed so that all soil used for planting is always manured a season previously. On meadow land the manure is placed under the sods by means of the plough (shown in Fig. 7). A sort of knife marks a running incision in the sods, while a hollow foot attached to it makes a cavity under them. In this cavity the manure is poured through a hole which passes through both the knife and its foot. After its passage the sods close immediately again, partly through their own weight and partly through elasticity. A layer of earth should be left between the sods and the manure, to decompose the latter and prevent its hurting the grass plants.

In the winter when the ground is frozen so hard or covered with snow, that the ploughshares cannot enter the soil, the manure barrels are housed in sheds erected on the sewerage farm, and kept as near as practicable at freezing point, so as to prevent fermentation. Every short spell of thawing is taken advantage of to empty the barrels.

The average produce of excreta per annum per head of a mixed population is known to be ten cubic feet, of which the urine makes up 8·60 feet. The combined mass contains $10\frac{8.5}{100}$ pounds ammonia, $4\frac{8.6}{100}$ pounds phosphate of lime, $1\frac{3.3}{100}$ pounds potash, and about 33 pounds of organic substance, which have a united agricultural market value of at least 10 shillings. A town of 10,000 inhabitants could therefore produce this kind of fertiliser if collected without the admixture with water, as above described, to the gross value of £5,000 in annual receipts, or increased produce of the farm where it is applied. Deducting £2,000 for working-expenses, management, and maintenance, there is still £3,000 left as revenue, which at 5 per cent. represents a capital of £60,000, nearly twice more than is required to build the

works completely, including changing every closet in town to suit the system.

Now considering that this mode of utilization can be applied to any arable land capable of being ploughed and of growing human food, it is evident that the sewage question which formerly was merely that of how to get rid of a nuisance has by this system been converted into an answer to that of how to save rivers and harbours from pollution, how to prevent the waste of water, how to increase the productiveness of the soil, and how to obtain thereby a good interest on capital seeking investment. The absolute practicability of employing pneumatic force for removing fæcal matters has been illustrated in a great number of Continental towns, where it is applied for the emptying of cesspools (as described in a former number), and the not less absolute practicability of the mode of utilisation of sewage, as here proposed, is proved by its success during centuries in China and Japan. The latter country, which is about the size of Great Britain, but owing to its mountainous formation, has only half its area fit for cultivation, not only comfortably supports a larger population than Great Britain, but even exports bread stuffs in considerable quantities. The cause is solely to be found in their mode of fertilising the soil, which is just the same as above described, namely, human excreta undiluted with water, undiminished in value by the composition, and mixed at once with the soil.

This, then, is Captain Liernur's system, on the merits of which, in the absence of experiments, it is impossible to pronounce a decisive opinion. It apparently possesses many advantages over even the London system, and disposes efficiently of several of the difficulties with which we have to contend in distributing sewage, and it is unquestionably better than the Continental system which we have described. The invention will, we believe, be soon tested on a large scale at the Hague, the capital of Holland, and we are pleased that the

authorities of that beautiful town are about to give an invention of such promise a fair start. At any rate the great and constant losses sustained by agriculture through the dilution and evaporation of fertilising matters, or their utter waste into streams, not less than the resulting sacrifice of health and life, fully warrant the policy of extending our experimental enquiries in this and other directions.

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NOTES

ON

THE TREATMENT OF CHOLERA.

BY

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Sanitary Commissioner for Bengal.

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PREFATORY NOTE.

THE annexed Schedule regarding "Results of personal experience as to the best means of treating Cholera," has, I believe, been generally circulated, amongst Medical Officers in India by the Principal Inspector-General, Medical Department.

As I was unable to embody the substance of my replies in the small space allotted in the blank form, I have, in the following brief "Notes" laid down my views on the points specially tabulated. I have presumed to publish these "Notes" chiefly in order to have an easy method of laying before those who are specially interested in the treatment of Cholera a few words in behalf of Calomel.

I do *not* produce them in my capacity of Sanitary Commissioner for Bengal, but simply as embodying the opinions and experiences of a single member of the Indian Medical Service.

D. B. S.

RESULTS OF PERSONAL EXPERIENCE AS TO THE BEST MEANS OF TREATING CHOLERA.

OPINION OF

STATION DATE

Whether experience of the Disease extends to Europeans as well as Natives.	Value of prophylactic treatment, and what? during prevalence of an Epidemic.	TREATMENT.				
		In premonitory stage.	When the Disease is fully developed.	In stage of Collapse.	In stage of Re-action.	In Secondary Fever.

NOTES
ON
THE TREATMENT OF CHOLERA.

In considering this subject, I shall adhere, as far as possible, to facts of which I have direct cognizance.

It may fairly be laid down that there appears to be *no* infallible *stereotyped* plan of treatment for this disease. We cannot as yet be guided in this matter by the *ipse dixit* of any man. Medicines which are reported by certain authorities to produce wonderful effects during one epidemic are found by others to be greatly less successful in the next outbreak ; and, indeed, it must be confessed that, at different periods of the same visitation, different effects are reported as being observed from the use of the same remedy. Nevertheless, in the face of such perplexing circumstances, it must be right that physicians should carefully record their personal experiences and their opinions regarding every remedy which they may prescribe, and the effects of which they have carefully watched.

I propose now to offer brief remarks on various means which have at different times been adopted for the cure of cholera.

I have seen numerous epidemics ; and my experience extends to Europeans as well as to Natives of almost every class in society.

From direct experience I have nothing of any value to record regarding the medical *prophylactic* treatment of cholera.

I confine myself to practical points bearing on actual treatment, merely premising that I believe a most important part of such treatment to consist in not allowing a patient, actually seized with cholera, to be carried any very great distance, even to a hospital,—rest and recumbency being vital considerations.

OPIUM.

The administration of opium in one stage of cholera or another is now almost universal. I believe its employment has been carried infinitely too far. No doubt, it acts as a sedative and astringent in the first stage of the disease, and its administration may then be called for. But I would dwell on the danger of considering a pill which contains opium, assafœtida, and black pepper, a specific for cholera. Truly it is *no* such thing. It checks diarrhœa ; but if trusted to in the stage of collapse, I feel convinced that in the vast majority of cases it fails altogether. I am decidedly of opinion that opium given in any period of the disease, except that of incubation, does more harm than good. For the time being it allays certain symptoms, but it afterwards complicates the disease, and lessens the patient's chance of ultimate recovery.

When a person is seized with *diarrhœa*, the so-called *cholera pill* is at once administered, and regarded as the best remedy that can be given. So, perhaps, it is ; but what I would insist upon is this : that it is a palpable misnomer for the remedy employed. It is by no means a *cholera* pill, because it checks diarrhœa ; on

the contrary, if trusted to in true cases of cholera, I am of opinion that it is not only useless but positively hurtful. The perfect appreciation of this fact is a matter of the greatest importance.

Orton, in his work on *Cholera*, declares :—" Opium is the sheet-anchor in the cure of cholera." I believe a more fatal dogma could not be enunciated by a physician.

I am constrained to dwell forcibly on this subject, because it is distressing to see how much danger is caused by the indiscriminate " doling out" of the so-called *cholera pill*, which has been made far too well known. It is a weapon which, handled without discrimination, produces disastrous results. As a rule, the oftener and the longer opium is given in true cholera, the less chance does there appear to be of the sufferer's ultimate recovery.

BLEEDING.

Venesection, which in former days was so much praised, I have never had recourse to, except to the extent of a few ounces. There can be no doubt that it has been found valuable in some cases, when not carried too far. Local bleeding, by leeches or cupping, has seemed to me to be seldom required. In some cases of cerebral congestion in the consecutive stage it may be advisable.

EMETICS.

My faith in emetics, employed for the treatment of cases of true cholera, in any of its stages, especially for those that are not marked by any distinct premonitory symptoms, is very limited. At the same time, it is but fair to confess that my experience of such a plan of treatment is also limited.

Emetics of salt or mustard have, however, been said to have roused patients from "an almost total lethargy."

STIMULATING AROMATICS.

If aromatics could cure cholera, the discovery must have been established half a century ago. But capsicum, peppermint, ginger, rose-water, cloves, and the much-vaunted camphor, although useful in their way, can be regarded merely as adjuvants.

ASTRINGENTS.

These are doubtless invaluable when they can be prescribed early, before the disease has produced extreme exhaustion. But even when given under the most favourable circumstances, they sometimes fail to check the regular course of the disease. I have seen many patients treated actively with powerful astringents from the very first, who gradually became collapsed. This is no reason, however, for abandoning their use in every case—very far from it. I believe that if they are employed *before prostration* occurs, it matters little what remedy is selected, whether acetate of lead, chalk, gallic acid, catechu, kino, or opium.

EXTERNAL APPLICATIONS.

There can be no doubt that it is absolutely necessary to do all that lies in our power to preserve the natural warmth of the body. All the ordinary means are useful in this respect: manual frictions with ginger, mustard and hot turpentine, sinapisms, hot bricks, bran-bags, and rubefacients generally. I think heat *passively* applied is preferable to active frictions, which, to a certain degree, exhaust the patient.

Application of hot water, of nitric acid, or of the cautery, has been proposed. I have no experience of these.

STIMULANTS.

I do not believe in the use of stimulants during the advanced stages of the disease. Very soon after seizure they may be called for, to assist nature in repelling the invasion of the disease; but once this has fairly occurred, they seem to have no decided effect.

When the pulse is weak, it appears but natural to administer restoratives, but experience teaches that it is of no avail after the early stage of cholera, and this particularly applies to brandy and other spirituous stimulants. I have come to this conclusion after prescribing them thousands of times, and carefully watching their effects. If, however, it is determined to give stimulants during collapse, those of a diffusible character are to be preferred.

Champagne is sometimes given in very hopeless cases, with apparently the desired effect. Such exceptional cases, however, do not, to my mind, affect the general rule.

DRINKS.

I believe it to be an essential part of treatment that the patient shall be allowed *very small quantities of fluid;* about a table-spoonful of water (iced, if possible) given every half-hour being sufficient. Excessive thirst may also be allayed by giving soda-water, magnesia and milk, cream of tartar drink, or water acidulated with nitric acid, in *small* quantities.

I cannot agree with Johnson that cold drinks ought to be allowed "*ad libitum*."

Orton says:—"The thirst is so excessive that it would be a great cruelty to refuse to gratify it."

We might almost with equal reason and kindness assert that delirium tremens should be treated according to the cravings of the drunkard; and that in cases of scabies, the irritation is so great that it would be cruel to recommend any cure but "scratching."

SULPHURIC ACID.

This remedy has been more lauded than almost any other. It is the main constituent of the celebrated Austrian specific. I have used it to a very considerable extent, and have found it a very valuable medicine, prescribed according to the rules laid down (*vide* Ranking's Half-yearly Abstract of Medical Science, Vol. XVIII, p. 262) by Mr. Buxton, of Great George Street, Westminster, *viz.*, in *half-drachm* doses every quarter of an hour in severe cases. I have been much pleased with its effects, although not so much so, perhaps, as Mr. Buxton. The panegyrics that have been lavished on this remedy are sometimes fulsome. Thus, Dr. Fuller (in the *Medical Times and Gazette* for October 1st, 1853) gives expression to the following opinion:—"My own conviction is, that in sulphuric acid we have an antidote—a specific—against choleraic diarrhœa, if not against the worst forms of cholera—as powerful, as energetic, and as certain in its effects, as in cinchona bark or quina against a paroxysm of ague!"

Would that Dr. Fuller's assertions could be endorsed by the profession generally! I have certainly seen sulphuric acid very valuable for choleraic diarrhœa, particularly in the case of children.

CARBONATE OF SODA.

This was one of the principal ingredients of Dr. Steven's famous saline treatment. It was given by him every quarter of an hour in half-drachm doses, with a scruple of common salt, and seven grains of chlorate of potash. I have seen it tried very frequently, but have, on the whole, been disappointed with it.

Dr. Lacy, at Agra, during the epidemic of 1856, used carbonate of soda in alternate doses every quarter of an hour, with sulphuric acid; and a considerable proportion of these cases were reported by Dr. Murray to have recovered.

The phosphate and sulphate of soda used also to be administered by Dr. O'Shaughnessy in ten-grain doses, with the same quantity of common salt, and five grains of carbonate of soda, given in six ounces of water.

CASTOR-OIL.

The treatment by castor-oil has at different times had fair trial in England. I am inclined to think that general professional opinion seemed to condemn it at last.

These remarks of course apply to its use (often in very large quantities) during the stage of collapse.

I believe there can be but little doubt in the minds of those who have seen most of cholera that the employment of aperients is hazardous during the regular course of the disease. After re-action has commenced, and the alarming symptoms of prostration and purging have decidedly abated, the advantage of an ounce or six

drachms of castor-oil is sometimes very marked, but it requires to be prescribed with much judgment and care.

CROTON-OIL.

This remedy, which was strongly advocated by Dr. Macgregor, seems to have fallen into disrepute. I have seen it given (although never to the extent of forty or fifty drops in twelve hours, as we read of in books), but cannot speak in its favour from personal experience.

ENEMETA.

In the case of Europeans, decided benefit often accrues from the use of warm saline enemeta, particularly in the later stages of the disease ; but it is a remedy which one is backward in having recourse to with the Natives of India, considering their exceeding aversion to it.

INJECTIONS INTO THE VEINS.

The injection of warm water and of saline solutions under the skin or into the veins, produces a marvellous effect in advanced cases. I shall never forget the first time I saw this plan of treatment adopted. It was in the case of a very old woman who was "moribund" from cholera. Her deeply sunken eyes, cold surface, shrivelled skin, and pulseless wrist, held forth no hope.

The fluid was injected ; within a minute the poor creature revived as if from death, sat up in her bed, and blessed those who were standing round her. In a few minutes she relapsed, and suddenly fell back on her bed. The injecting process was repeated ; again she sat up as before ; and so it continued until the injection had been used four or five times, when it ceased to take

effect, and the patient died almost immediately after. Dr. Kenneth Mackinnon, in his work "*On Public Health and Prevailing Disease in India*," has justly remarked that this treatment by saline injection "approaches more nearly to an immortal discovery than anything in medical practice of late years."

Dr. Owen Rees has recommended the following powder to be used for the purpose of injection into the veins, dissolved in water, and used when the solution reaches a specific gravity of 1,030, and a temperature of 98 deg. Faht:—

Chloride of sodium . .	. .	3 ounces.
Phosphate of soda . .	. .	1 ounce.
Carbonate of soda . .	. .	1½ ounce.
Sulphate of soda . .	. .	½ ounce.

Some have recommended a still higher temperature for the solution, *viz.*, from 106 deg. to 120 deg. Faht.

CHLOROFORM.

Chloroform is invaluable for the purpose of checking irritability of stomach, prescribed in doses of twenty minims with mucilage, camphor mixture, or tincture of catechu.

It seems much more efficacious than hydrocyanic acid or creasote.

IPECACUAN.

Those who have used ipecacuan in very large doses for acute dysentery, according to the plan suggested by Dr. Docker, Surgeon of the 2nd Battalion, 7th Royal Fusiliers (vide *Lancet*, July 31, and August 14th, 1858), must have been struck with its wonderfully satisfactory

effects. The value of such treatment is now fully recognised.

A dose of 90 grains may be given, according to Dr. Docker, "in the very worst cases, where the strength of the patient is almost exhausted," and rapid cure results in almost every case. From my knowledge of this plan of treatment, I was induced to try it in cholera, and in several cases it seemed to check purging, and better the condition of the patient. I have given as much as *forty grains* to a patient *in advanced collapse* with the result mentioned.

After prescribing the ipecacuan in this manner, I discovered that a still bolder administration of it formed part of the plan of the treatment of Dr. Carl Müller, of Vienna.

"When entire collapse has taken place," he recommends that "two drachms of the powdered ipecacuan be mixed with cold water, and gradually administered to the patient." "In half an hour or an hour," he declares, "re-action ensues." The rest of his treatment consists in the use of Haller's acid, (1 part officinal sulphuric acid to 3 parts rectified spirit). He reported that he treated 319 cases of cholera, without losing a single patient! The above has been extracted from a Pamphlet of Instructions to Army Medical Officers regarding cholera, issued from the War Department.

An extension of the ipecacuan treatment in India might be well worthy of trial.

LIQUOR ARSENICALIS.

Fowler's solution of arsenic was recommended many years ago by Dr. Black, of Chesterfield, in doses of 15

drops every quarter of an hour, until abatement of symptoms occurs; and after that in small doses.

I tried this in eight cases, but the results were by no means satisfactory: *six* of the patients died. It should be mentioned, however, in justice to Dr. Black, that these eight patients were very debilitated inmates of a pauper hospital.

NITRATE OF SILVER.

My attention was first drawn to the virtues of this remedy principally by an article in the second Volume of the Indian Annals of Medicine, by Dr. Barry, Civil Surgeon of Gowalparah, Assam. I have found it very useful, particularly amongst weak subjects in poor-houses. It was used under my direction in 16 cases, 11 of which recovered, it being prescribed as Dr. Barry recommends, *viz.*, one grain of nitrate of silver in a drachm of mucilage, after every dejection, until four, five, or six doses have been given, should it be necessary to continue it so long.

TURPENTINE.

This is a very valuable remedy for Natives during collapse. It seems to act as a stimulant and diuretic, and may be safely prescribed in doses of 1 or 2 drachms, repeated according to circumstances.

QUININE.

I have used quinine largely and with much satisfaction. Although it does not seem to have any specific effect in altering the character of the disease in any way, yet it braces the system, and carries the patient on from hour to hour. This alone is a point of vital importance in the treatment of cholera. I have never used it in very

large doses, but I am inclined to believe that, at any time, twenty grains would be better than ten, and thirty than twenty. In a poor-house eight cases were treated, under my supervision, with amorphous solution of quinine alone ; of these, six recovered.

Lastly, I come to speak of calomel.

CALOMEL.

This I believe to be at present our best remedy for cholera.

Having given it in very different doses, I think the best of all is Dr. Ayre's treatment of *two* grains, given regularly every ten minutes,—or perhaps *better still* the mode of giving *one drachm for a single dose*, placed on the tongue and washed down with a little water. Dr. Lithgow, of Her Majesty's 75th Regiment, first interested me in this plan. Out of 24 men treated so before Delhi, he only lost 4.

My experience of it is also very satisfactory. I have seen more permanent good result from its employment than from anything else. Without wishing to "cry it up" as a *catholicon*, I would simply state that I have seen hundreds of lives saved by it, and by it alone.

There are, as is well known, many other modes of treatment, each of which has had its partizans. Thus we have—inhaling of oxygen and of carbonic acid, cold affusion, the wet-sheet packing of the hydropathists, hot and vapour baths, galvanism, strychnine, cajeput oil, tartar emetic, Indian hemp, terchloride of carbon, nitrous acid, alum, naphtha, arnica, prussic acid, phosphorus, creasote, lemon juice, sesqui-chloride and sulphate of iron, hydro-sulphuret of ammonia, chlorine water, sulphur, charcoal, iodine, acetic acid, transfusion of blood, and quassia.

But of all these reputed *panaceas* I can say nothing.

Having made the above general remarks on the many remedies recommended for cholera, I will endeavour briefly to indicate my usual treatment of the different stages of the disease.

The preliminary stage. This stage, which is marked by general uneasiness, sense of exhaustion, ill-defined alarm, tendency to painless diarrhœa, and all the other well-known signs which have been termed the *prodromata* of cholera, appears to be best treated by stimulants and astringents. Of the latter, I have quite as much faith in compound chalk powder with opium, or in the lead and opium pill, as in the so-called *cholera pill.*

The 2nd stage of collapse is, I think, best met by calomel, giving either one drachm in a single dose at first, or ten grains quickly followed by doses of two grains every ten minutes. The single-drachm dose is very often quite sufficient to secure the patient's safety; the smaller doses must be continued very perseveringly, *until improvement takes place. Any single dose which may be rejected should at once be repeated.*

If there is much irritability of stomach, one or two doses (20 minims) of chloroform in mucilage may be administered with great advantage before giving the calomel, withholding stimulants altogether, and allowing only *a little* cold soda-water every half hour, and sinapisms, or the like, applied to the epigastrium, calves of the legs, &c.

At the same time that calomel is being prescribed, I sometimes give half-drachm doses of sulphuric acid every quarter of an hour. A dose of quinine, too, given with discrimination from time to time, is always of advantage. As before observed, other cases may be met by the ipecacuan treatment, giving doses of 40 grains or

more ; or by the use of nitrate of silver in single-grain doses.

If the disease advances in spite of all treatment, and the life of the patient is threatened, *I still continue perseveringly to give the calomel.* When almost all hope is gone, the disease (if this treatment is persevered in faithfully) often reaches a happy crisis.

When *re-action* sets in, it is well to discontinue the calomel, or, at any rate, to give it at longer intervals than before. Diuretics are now useful, as the nitrate or acetate of potash, demulcent drinks, and quinine ; occasionally also a small dose of castor-oil, when the motions are of dark tarry appearance.

In the stage of consecutive fever, nature is not more to be trusted than during the period of collapse.

Effervescing salines, carminatives, and diuretics, are now useful, and fomentations or stimulating frictions over the loins. The use of the catheter should never be omitted when urine seems to be secreted, (no matter in how small quantity,) and the bladder is powerless. Nourishment is now much required, and beef-tea, arrow-root, sago, rice-water, or barley-water, may be given with great advantage ; indeed, it is *very* important, both during and subsequent to the stage of collapse, to administer as much nourishment as can be retained.

If there exists great irritability of stomach, it is expedient to apply a blister over the epigastrium.

If head symptoms are urgent, and the breathing is laboured, counter-irritation is indicated ; for this purpose, a cap of blistering fluid on the vertex is often effective.

It is very important, indeed, that throughout this disease, the recumbent posture should be steadily maintained. During a single epidemic I saw two deaths result from neglect of advice, repeatedly given, on this point.

During *convalescence*, tonics and stomachics are generally required, and a rigid attention to matters of diet.

P. S.—The substance of the above notes was written in 1861. They still express my present opinions on the subject of the treatment of cholera. I would urge those who are sceptical of the value of any medicinal remedy in the stage of collapse to give the "heroic treatment" by calomel a fair trial, *persevering in its administration till the very last.* The satisfactory results will, I believe, astonish those who have never before used calomel, *boldly*, in severe attacks of cholera. The most hopeless cases I have ever seen on first admission into Hospital have terminated favorably when the calomel treatment has been *perseveringly* adhered to. I am glad to know that Dr. C. R. Francis, whilst he was officiating as Principal of the Calcutta Medical College and *Ex-Officio* Senior Physician to the College Hospital, took up, at my request, the idea of the calomel treatment, and that he has had reason to be pleased with the results.

The remedy possibly does not produce any *visible* and decided physiological effect. *All I can say is that I believe an unusual proportion of recoveries takes place ; and that consecutive fever and a typhoid condition are by no means usual after such treatment.*

In so far extolling the value of calomel in the treatment of cholera (as compared with other remedies), I offer no explanation or hypothesis as to its

mode of action. *I confess to using it empirically.* The results, nevertheless, are altogether inconsistent with the too common assertion that no medicine is of any value where cholera has reached the stage of collapse.

The above are but hasty notes. I cannot help believing, however, that they contain the elments of sound and not altogether usual practice : and one of my chief objects in publishing them will be served, if, on the one hand, I find that the general principles enunciated, particularly those relating to the use of calomel, are corroborated by men of extensive experience in Inda ; or, on the other, if they serve to attract the attention and to engage the thoughts of those who feel disposed to shake their heads and to shudder at the very name of this powerful and no doubt much-abused drug.

Lastly, I think it well to note that—except for the treatment of cholera—I scarcely ever prescribe a grain of calomel.

DAVID B. SMITH, M. D.

for Parkes F.R.S.
with the Translator's
kind regards

ON

HÆMOPTYSIS,

ESPECIALLY WHEN FATAL,

IN ITS

ANATOMICAL AND CLINICAL ASPECTS.

By Dr VALD. RASMUSSEN.

TRANSLATED FROM THE "HOSPITALS-TIDENDE,"

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HÆMOPTYSIS,

ESPECIALLY WHEN FATAL,

IN ITS

ANATOMICAL AND CLINICAL ASPECTS.

ALTHOUGH Hæmoptyses, or the hæmorrhages occurring through the air-passages, have from the earliest period attracted the general attention of medical men, as the very name Hæmoptysis, derived from Galen, testifies, it is nevertheless a fact that the source of these hæmorrhages, the absolute demonstration of the vessel or vessels whence the blood has come, has almost entirely escaped the observation of investigators, and the discussion of this point has therefore been almost exclusively confined within the uncertain domain of hypotheses. One of the most eminent clinical physicians of our time, Walshe,[1] has consequently, not altogether without reason, in treating of pulmonary phthisis, in which hæmoptysis most frequently occurs, indicated the precise demonstration of the ruptured vessel from which the blood has issued, as a desideratum.

Before I proceed to detail my own investigations in this direction, I shall give a short sketch of the views of the principal writers on pulmonary hæmorrhages in general, and especially on their sources.

The earlier literature is rich in reports of profuse hæmorrhages, and in many of them the quantity of blood brought up borders almost on the incredible, as 30 pounds in three hours (Rhodius). As to the source of the hæmorrhages, however, only rare and imperfect information is given, which is the more unfortunate, as in some writers indications are met with of circumstances similar to those which form the basis of the following essay. Thus varices are spoken of by Gilibert[2] as the cause of hæmoptysis and phthisis:

[1] Diseases of the Lungs, translated into Danish by J. Blicher, Copenhagen, 1861, p. 518.

[2] Adversar, p. 64. Quoted by J. Frank, Prax. Med. pars ii., vol. ii., sect. 1, p. 418, and elsewhere.

"Varices pulmonum sunt principium hæmoptysis et phthiseos; has etiam scalpello occupavi." Morgagni[1] says that, in the dissection of individuals who had died of hæmoptysis, he has seen tubercles with dilatation of the surrounding vessels. De Haën[2] says he saw an aneurism open into a cavity. Portal[3] states that he has observed ruptured vessels from the lymphatic glands opening into the bronchi. J. Frank[4] mentions a case where, in the dissection of a young man who had died during an attack of hæmoptysis, on injecting the pulmonary artery, he found that the injected mass had penetrated into a cavity.

While the earlier writers in general sought the cause of pulmonary hæmorrhages in rupture of larger vessels, observers in the present century began, supported especially by the authority of Laennec, to assume the existence of an exudation (diapedesis) of blood from the bronchial mucous membrane in those cases where no ruptured vessel was demonstrable; and this view has been maintained to our own day, modified only by the altered anatomical requirements, capillary hæmorrhages being substituted for exudation.[5]

What most essentially contributed to this change in the general opinion as to the source of the hæmorrhages, was the discovery that the vessels in the condensed tissue surrounding the cavity are obliterated. Baillie[6] was the first to call attention to this condition, in which he saw an effort of nature to prevent hæmorrhage. Laennec[7] demonstrated this by injections, and found at the same time that the trabeculæ which so frequently traverse cavities enclose obliterated vessels, which in rare cases, though usually only partially, may remain open. J. F. Meckel's[8] injections confirmed Laennec's,

[1] Quoted by J. Frank, *loc. cit.*, and in the Dict. des Sciences Méd., but without a reference; the matter is not mentioned in his De Sedib. et Causis Morborum. It is scarcely possible that Morgagni can have instituted very profound investigations in this direction, as he states (epist. xxii. artic. iii.) that he avoided and dreaded the bodies of those who had died of phthisis to such a degree that he never, even in advanced age, touched them.

[2] Quoted by J. Frank, but unfortunately incorrectly; it is scarcely to be found in his Ratio Medendi.

[3] Dict. des Sciences Méd., art. Hémoptyse, p. 319.

[4] *Loc. cit.*, p. 420, note.

[5] That diapedesis may in fact take place is shown, however, by Cohnheim's recent experiments on venous stasis (Virchow's Archiv, bd. 41, p. 220); after tying the femoral vein, he saw the red blood-corpuscles exude from the congested capillaries, whose walls were intact; probably this exudation takes place through Œdmansson's openings, yet the quantity of blood exuded in this mode must of course be only comparatively small, and is without importance in reference to hæmoptysis.

[6] The Morbid Anatomy of some of the most important Parts of the Human Body, London, 1793, p. 45. Baillie, as well as J. F. Meckel, mentions, however, that Stark had previously observed that the vessels are closed by coagulated blood.

[7] Traité de l'Auscultation Médicale, 4me édit., p. Andral, Paris, 1837, p. 185.

[8] Handb. d. pathol. Anat., Theil ii., Abth. ii., Leips., 1816, p. 375.

but showed that vessels without constriction of their cavity may pass in a cord-like form from one wall of the cavity to the other. Schroeder van der Kolk's[1] injections proved that the closing takes place from the smaller branches, and gradually extends to the larger. This obliteration of the vessels was subsequently confirmed by several, as Guillot, and the correctness of the observation was generally recognised; in accordance with this, most writers, with the exception of Rokitansky, assume that the hæmorrhage in the rare cases in which it comes from a cavity is due to erosion of such a non-obliterated vessel passing through the cavity. We shall at greater length refer to some of the more important writers.

Laennec[2] states that it is not impossible that a branch of the pulmonary artery or varices of the pulmonary veins may give rise to hæmorrhage, though he brings forward no regularly described case. Considerable, nay even fatal, hæmorrhages arise from rupture of a vessel passing through a cavity, as well as by the bursting of an aneurism of the aorta into the pulmonary passages. Most cases of slight or moderate hæmorrhage proceed from the bronchi by an exudation of blood from the mucous membrane of the latter. More violent hæmorrhages from the pulmonary parenchyma are due to pulmonary apoplexy.

Andral[3] enumerates three sources of pulmonary hæmorrhage:—1. From the bronchi, bronchial hæmorrhage, only in one case without coexistent pulmonary tubercles; he was not, however, in a position to demonstrate the starting point of the bleeding; the mucous membrane is stated only to present simple signs of bronchitis, and to be pale. 2. Pulmonary apoplexy. 3. From a cavity; such cases are however, on the whole, rare, and Andral has only once been able to point out the vessel from which the blood came; it was found in this case enclosed in a trabecula passing through the cavity; the vessel was eroded, and contained a little decolorized coagulum.

In opposition to Andral's view of bronchial hæmorrhage as the most frequent cause of hæmoptysis, which is that which has found most adherents, some authors assume that the hæmorrhages in phthisis—for it is only in this disease that the explanation meets with difficulties—stand in a more or less direct relation to tubercular deposition. Thus, according to Engelstedt,[4] the expectoration of blood depends not so much on the tubercles as on the altered collateral circulation in the inferior and not yet condensed portions of the lung, whose vessels remain distended with blood, and may therefore easily burst, when an exciting cause, as a fresh deposition of tubercle, a blow on the chest, etc., occurs. Of six individuals who

[1] Observat. Anatom. Patholog., fasc. i., Amstelod., 1826, pp. 75 *et seq.*

[2] Traité de l'Auscultat Méd., 4me edit., p. Andral, Paris, 1837, p. 81.

[3] Clinique Méd., t. iv. pp. 165 *et seq.*

[4] Phthisis, en anat.-klinisk Undersögelse (an Anatomico-Clinical Investigation), Copenhagen, 1853.

died shortly after expectorating blood, no trace of hæmorrhage was met with in or around the cavities; only in one per cent. did the hæmorrhage cause death.[1] In two instances non-obliterated, cord-like vessels were found passing through the cavity. Walshe looks upon hæmorrhage from the bronchial mucous membrane as exceedingly rare; according to him, molecular rupture of the capillaries of the parenchyma is the cause of hæmorrhage in tuberculosis, with the exception of the very rare cases where a vessel of more considerable size is perforated.

Rokitansky is certainly the only writer who describes cases such as those constituting the subject of the present essay. According to him, hæmorrhages during the course of phthisis are in the commencement rare and scanty; at a later period they are copious and frequent, and then proceed from branches of the pulmonary artery situated in the condensed walls of cavities, especially of bronchiectatic cavities, and in isolated cavities with aborted tubercles. The vessel becomes exposed partly by fresh deposition of tubercles, partly by a necrotic process in the wall of the cavity, so that it comes to want its accustomed support; it is soaked with the contents of the cavity, becomes thereby macerated, jelly-like, and soft, and at last bursts into the cavity. This takes place either by the formation of a split-like fissure in the vessel, or by the separation of a portion of the vascular wall giving rise to a round hole. Very frequently this is preceded by an aneurismatic dilatation of the vessel in towards the cavity. A preparation is mentioned as a rare case, where, in the wall of an immense cavity, a large branch of the pulmonary artery is found exposed to a certain extent, bulging in towards the cavity, and opened on a level with the bulging, a piece of the vascular wall of the size of a pea having separated, with the exception of a very small spot, so that a convex-concave lid is formed over the cavity.

Niemeyer's theory of pulmonary hæmorrhages, which is moreover quite that of Andral, I shall hereafter take occasion to state, and shall now mention only the most recent phthiseological work of Hérard and Cornil[2] which relates to hæmoptysis. The authors suppose that where, during the course of chronic phthisis, blood is expectorated, this is due to ruptures of small capillary vessels from little fungous excrescences in the walls of recent cavities; from cavities of longer standing no blood can come, as the vessels are obliterated in their immediate circumference. We are, they say, on the whole much more inclined to believe that the hæmoptysis is due to a fresh deposition of tubercles, which undergo the same changes as in the parts of the lungs first attacked. The authors indeed mention violent hæmoptyses, but no case where death occurred

[1] Louis (Recherches sur la Phthisie), among eighty-seven cases of phthisis, saw hæmoptysis only four times in the last days before death, and of these only two were to any considerable degree; there was no fatal case.

[2] De la Phthisie Pulmonaire, par Hérard et Cornil, Paris, 1867.

as the result thereof. On the whole, they do not give one positive contribution to the anatomy of the hæmorrhages. In chronic pneumonia hæmoptysis is, according to them, a symptom of the last period (ulceration), while in tuberculosis it occurs in the commencement of the disease.

The cases in which patients have died during violent hæmoptysis, and which form the basis of the following sketch, amount in all to 11, and fall into two groups:—1. Cases where the hæmorrhage proceeded from rupture of a vessel running in the wall of a cavity; and, 2. Cases where it was due to rupture of an aneurism of the aorta into a bronchus, or into the pulmonary tissue itself. The first group comprises eight cases; the second, three. Hæmorrhagic infarctions (pulmonary apoplexy), pulmonary gangrene and cancer of the lungs, in the course of which it is well known that hæmorrhages, though in general not profuse ones, also occur, are not unfrequently met with in the Municipal Hospital (Kommunehospitalet), but have never been attended with such considerable hæmoptysis as to cause death, and they have consequently not been the subject of anatomical investigation.

I.—Hæmorrhages from a Cavity.

These are due partly to ruptures of small sac-like aneurisms, developed on branches of the pulmonary artery, running in the walls of cavities, partly to dilatations (ectasias) of similar vessels with operculated rupture. These two forms occur with equal frequency—each of them, namely, in four cases.

The Aneurisms.—The size of these varies considerably, from that of a walnut to that of a pea and under; they are formed by the dilatation of a vessel in contact with the inner wall of the cavity, the part of the vascular wall touching the cavity at the point of contact being dilated, while the remainder lies firmly embedded in the condensed vascular wall. The form of the aneurism is at the same time given as sacculated with a tolerably uniform transition between the walls of the aneurism and those of the vessel, without any proper neck (*A. sacciforme verum*). The surface of the aneurism is smooth; only in one case where it was very large (as a walnut) was a small bulging of the size of a pea met with. In its cavity most frequently only freshly coagulated blood is found, and only in one instance, that just now mentioned, were the well-known, firm, decolorized, adherent coagula met with.

The walls are of various thickness. In aneurisms which have not burst, they are thickened, often two or three times thicker than the rest of the vascular wall; and when these, as was always the case, are small, the aneurism forms only a slight, dome-like dilatation on the vessel. In general, and invariably when the size is more considerable, the walls are however thin, only slightly or not

at all exceeding in thickness the vascular walls, and are particularly attenuated up towards the apex or point of perforation. Both forms may occur side by side (4th case). In some instances considerable fatty degeneration of the wall of the aneurism was met with, exhibiting itself as segregated and confluent, yellow, sharply-defined points; a circumstance to which I shall hereafter revert, when speaking of the causes of rupture.

The rupture takes place always at the most prominent point of the sac, and there is usually formed an irregularly fissure-like rent, rarely exceeding in width two or three millimètres, most frequently it is only large enough to allow the knob of an ordinary probe to pass. In the opening adhere loose, dark coagula, which are more rarely firm and somewhat decolorized. The edges are attenuated, yellowish, though the colour is often difficult to recognise on account of the imbibition of blood; but under the microscope distinct fatty degeneration, especially of the muscular coat, is visible.

The number of the aneurisms varies. In general only one is met with; in one case two were found close to one another; in another there were so many as four, two and two close one to another on the same branch. The vessels on which the aneurisms were located were on an average from one to three millimètres in width.

Case 1.—*Aneurisma ruptum et ectasia rami arter. pulmonalis in cavernâ bronchiectaticâ; Pneumonia chronica; Bronchitis; Arteriosclerosis art. pulmonalis (levi gradu); Cyanosis renum.*

H. T. Wulff, aged 64, was admitted on the 8th May 1866 into the second division of the Municipal Hospital. In 1853 he had cholera, but had never suffered from any other serious illness. For many years he has every spring had some cough, which has passed off toward summer, and never gave him further inconvenience. During the last month the cough has been a little more persistent, though without causing any pain in the chest, but his general health has suffered a little. Six days ago he expectorated, during a fit of coughing, which was at the same time accompanied with vomiting, four or five ounces of blood, some of which was coagulated, while the remainder was dark and fluid; previous to this he had no abnormal sensations in the chest; he cannot state whether it was the cough or the vomiting which gave rise to the expectoration of blood. A similar attack, during which a much smaller quantity of blood was brought up, took place the day before yesterday.

On admission the patient was found to be strongly built for his time of life; his expression was natural. He complains only of pain in the head during the cough, which is rather frequent and troublesome, and accompanied with a scanty, viscid, and coloured expectoration. The tongue is moist, and is but slightly furred; there has been no nausea or vomiting for the last six days. Pulse 80, full; appetite slight; sleep for the last few nights has been

disturbed by the cough. On examining the chest, the sound on percussion is found to be rather dull in the clavicular, infra-clavicular, supra-spinous, and in the upper part of the infra-spinous regions, and expiration is there rather prolonged; over the rest of the chest nothing abnormal is met with, but the respiration is on the whole rather feeble.

On the 9th, about six or seven ounces of dark, cherry-coloured, fluid blood, containing some coagulated lumps, and mixed with a quantity of mucus, were coughed up. The pulse was 80, and was very full. The patient was bled to twelve ounces, and was put upon a solution of watery extract of secale cornutum.

10*th.*—The patient felt relieved immediately after the bleeding, but towards night the cough returned, when a couple of ounces of expectoration mixed with blood were brought up.

11*th.*—The expectoration is again to-day freely mixed with blood. The patient had but little sleep at night, having been disturbed by the cough. Pulse 84, less full; syrup of acetate of morphia, one drachm three times a day.

12*th.*—Slept well, cough considerably less, but the expectoration, which has a characteristic bronchiectatic smell, is still mixed with blood. Pulse 84, full; appetite good.

On the afternoon of the 13th a violent fit of coughing came on, with expectoration of about twelve or thirteen ounces of light-coloured blood. The sanguineous expectoration continued more or less until the afternoon of the 16th, when, after being awoke from sleep, the patient got a violent fit of coughing, ending in profuse hæmorrhage, during which thirteen or fourteen ounces of blood were brought up. The attack lasted about ten minutes, at the end of which time the patient died.

Dissection.—Some cadaveric rigidity. Body strong and in good condition; subcutaneous fatty tissue everywhere abundant. Muscles of the natural appearance. Heart flaccid, covered with fat at the base and on the right side; in the pericardium was a small quantity of bloody fluid. The endocardium was strongly soaked with blood, as was the commencement of the aorta, where were some slight sclerotic changes. The valves were healthy, the muscular structure pale, opaque, slightly streaked with yellow, especially in the papillary muscles. The left lung was attached by old adhesions to the thorax; it contained air throughout, was somewhat coloured. In the apex were slight and circumscribed thickenings of the pleura. Only in the larger bronchi was there a small quantity of partly fluid blood. The right lung was in the apex very firmly adherent to the thorax; the two laminæ of the pleura were completely adherent to one another, so that the lung could be separated only with difficulty and not without injury. On section, the upper lobe was found to be in its greater and posterior portion condensed, almost completely void of air, especially superiorly. The surface of section was smooth, of a grayish yellow colour, with

pigment abundantly scattered over it; no lobular structure was perceptible, but in several places the yellowish coloration was seen to proceed from numerous small yellowish points (alveoli). About the middle of the posterior obtuse margin, and close to this, separated from the thickened pleura only by a thin, highly-coloured, condensed layer of pulmonary tissue, was a cavity rather larger than a walnut, filled with dark fluid or slightly coagulated blood. From the outer wall of this cavity projected a tumour about the size of a nut and of the shape of a bean. In the middle of the surface turned towards the cavity, was found a tolerably soft, pale red sanguineous coagulum, on removing which the subjacent part of the tumour appeared of a deep yellowish colour, and with a small perforation, through which only the button of a fine probe could pass. The opening was of irregular form, torn, and to its margins adhered loosely coagulated blood. This opening led into a cavity, which, on slitting up the pulmonary artery, proved to be an aneurismatic lateral dilatation of a branch of this artery, containing only a small quantity of freshly coagulated blood, without a trace of denser fibrinous deposit. The wall of the aneurism was attenuated, especially towards the perforation, and the yellowish colour occupied almost the whole of the outer surface, only close to the wall of the cavity was the colour somewhat reddish, though here and there studded with small, point-like yellow spots. The branch of the pulmonary artery leading to the aneurism was immediately in front of the latter, when slit up, six mm. in width; it was continued through the condensed wall of the cavity, but was so small on the other side of the aneurism, that only a horse-hair could be introduced into its cavity. In the trunk and some of the larger branches of the pulmonary artery, but by no means in all, were found small whitish yellow, sharply circumscribed points and spots, though only of small size, individually scarcely as large as the head of a pin, but here and there placed close together. In the vessel leading to the aneurism, as well as in its undilated wall, the inner parts were found perfectly healthy. On closer examination of the vessels leading to the cavity, it appeared that one, which when slit up measured scarcely three mm., ran in the upper wall of the cavity, somewhat above the aneurism; it projected there as a perceptible whitish yellow cord. When slit up its calibse was seen rather strongly arched in towards the cavity, especially in the middle, and the corresponding wall was thickened, whitish yellow, while the opposite one had its natural appearance. A larger bronchus, which was only four centimètres removed from the principal bronchus, terminated in the fundus of the cavity; it did not indeed pass continuously into the walls of the cavity, but neither was the transition abrupt. There was no other cavity but that just mentioned. The other parts of the lung were everywhere permeable to air; the surface of section was highly sanguineous, thin blood flowing abundantly from all the divided bronchi.

Nowhere were there traces of miliary tubercles, either in the lung or in the pleura. The mucous membrane of the bronchi was soaked with blood, thickened with abundant viscid mucus. The bronchial glands were scarcely swollen, and were strongly coloured. The stomach, as well as the transverse and ascending colon, was rather distended with air. The spleen was of the usual size, its parenchyma was of a brownish red colour, the follicles were few. The kidneys were of the usual size, rather rigid; the capsule was easily separated; their surfaces were smooth, of a dark red colour, as was the surface of section in both substances, though mostly in the cortical. The papillæ were pale; there was some papillary catarrh. The glomeruli were highly congested; the urinary canals were scarcely altered. In the stomach there was nearly a pint of bloody fluid; the mucous membrane was highly tinged with blood, but was otherwise unchanged. The liver was of the ordinary size; from the surface of section blood oozed abundantly from the larger branches of the vena portæ; the parenchyma was of a light reddish colour, and of a tolerably natural appearance. The intestinal canal was healthy.

Case 2.—*Aneurisma rupt. art. pulmon. Phthisis cavernosa (Bronchiectasis, Pneumonia chronica, Peribronchitis). Ulcerationes tuberculosæ ilei et cæci; Cyanosis renum et hepatis.*

Christ. Peter Carlsen, aged 34, labourer, was admitted to the Third Division on the 6th February 1866. Has for many years been subject to shortness of breathing, but otherwise enjoyed good health. Last summer he was under treatment in this hospital for hæmoptysis. A couple of months ago he began to cough more, and a fortnight since he felt a pain in the right side of the chest, particularly marked on deep inspiration. The cough at the same time became worse, and was often attended with vomiting. For the last week he has kept his bed, having altogether lost his strength. His appetite is slight, he sleeps but little, and during sleep perspires copiously. Bowels regular.

The examination of the chest gives a dull sound on percussion in the first intercostal space on the right side, also from the fifth rib downwards, where it passes into the hepatic dulness. Respiration is bronchial in the right infra-clavicular region, and is accompanied with some few subcrepitating râles; in the infra-mammary and lateral regions it is feeble. Posteriorly the sound on percussion is dull over the entire of the right side, the dulness increasing downwards and becoming total in the middle of the infra-scapular region. Respiration is very weak throughout the entire lung, but is still audible down to the base of the latter. The sound on percussion in the left side of the chest is not altered; over the whole of the left lung scattered subcrepitating and sonorous râles are heard. The liver does not extend beyond the margin of the ribs. Pulse 120.

On the 8th, the expectoration was streaked with blood; at the

right nipple there was a distinct friction sound. On the night of the 4th of March the patient began to expectorate red fluid blood; and in the afternoon of the 5th, during a fit of coughing, violent hæmoptysis came on, in the course of which the patient died.

The post-mortem examination took place on the 7th, in the forenoon. Cadaveric rigidity; body moderately emaciated; subcutaneous adipose tissue atrophic. Muscles pale, but very powerful. The heart contracted, of the usual size; the valves as well as the muscular structure healthy. In the cavities much fluid blood, and a few soft fibrinous coagula. The left lung is somewhat adherent along the posterior obtuse edge, but the apex is free. On section, a cavity is seen about the size of a pigeon's egg, filled with coagulated blood, situated in the middle of the upper lobe, four centimètres from the apex and three from the obtuse edge; it has tolerably smooth walls, though here and there they are somewhat uneven, and is invested with a grayish mass, capable of being scraped off. The wall is formed of condensed, coloured pulmonary tissue, about a quarter of a centimètre in thickness. An undilated bronchus is continued into the wall. On removing the contents of the cavity with a stream of water, a sac almost as large as a walnut is observed projecting from the outer wall of the cavity into the same. On the most prominent part of this sac some more solid dark coagula are adherent, which, on careful removal, are found to conceal a slit-like opening two or three mm. in length, with deep yellow attenuated and torn edges. On slitting up the sac it is seen to contain only a small quantity of freshly coagulated blood; the inside is smooth, blood-coloured; only inferiorly and posteriorly is a slight depression met with, corresponding to an external prominence as large as a pea, and only this is distended with firm, pale, yellow coagula. On slitting up the pulmonary artery a branch of the latter is seen, about one mm. in diameter, to open into this sac, which is formed by dilatation of the side of this vessel looking towards the cavity, while the other lies firmly in the wall of the cavity; a somewhat smaller branch opens superiorly into the sac, lying in the same axis as the inosculating one. Moreover, there are found in this lung, not merely in the apex, but also over the whole of the upper lobe, numerous miliary tubercles, partly scattered, partly collected into small groups, often of lobed or laminated structure, projecting on the surface of section, of a whitish colour, most frequently with a perforated or dark-coloured centre. The pulmonary tissue is everywhere pale and permeable to air, only superiorly towards the hilus is there some solidification, especially along the larger vessels and bronchi. In the lower lobe the tissue is congested with blood; miliary tubercles occur only in a very scattered and isolated manner. In the pleura a few traces of miliary deposition are seen. The mucous membrane of the bronchi exhibits a moderate, fine vascular congestion; in some places are small, partly cylindrical, partly diffuse, dilatations. Nothing abnormal is discovered either in the

other branches of the pulmonary artery or in the main trunk. The right lung is everywhere firmly adherent to the diaphragm. In the apex is a cavity about the size of a goose-egg, which reaches to the thickened pleura, or is separated from it only by a thin, highly coloured layer of fibrous tissue, which in general circumscribes it; into the cavity open, in many places, eyelet-hole shaped bronchi, and on slitting up these, many are seen to run a serpentine course, and to be dilated, with bands projecting between the dilatations, while others are natural with respect to their cavities. The mucous membrane is everywhere dark and uniformly red. The cavity contains a small quantity of chocolate-coloured fluid; the walls are uneven, in several places with pit-like, blind, eyelet-hole shaped depressions. The remainder of the lung is permeable to air, is congested with blood, and presents scattered miliary tubercles. The bronchial glands are highly swollen and coloured. In the throat and œsophagus, as well as in the larynx and trachea, is a large quantity of fluid or loosely coagulated blood; the mucous membrane is highly soaked with blood. Only on the under surface of the epiglottis are some swollen follicles. The spleen is about double its usual size; its capsule is thickened; its parenchyma is tolerably firm, of a pale red colour; the follicles and trabeculæ are in the usual number. The kidneys are firm, rigid; the capsules are easily separable, the surfaces are of a diffuse dark red colour; the surface of section is similar, the pyramids somewhat darker; the glomeruli are highly congested, the canals of the cortical substance exhibit only a slight opacity. The mucous membrane in the pelvis is natural. The liver is large, firm; its parenchyma is dark red; the central parts of the acini are slightly depressed; the peripheries are slightly loaded with fat. In the gall-bladder is a little thin, yellowish bile. In the stomach is a large quantity of loosely coagulated or cherry-coloured fluid blood. The mucous membrane is healthy, imbibition excepted. In the urinary bladder is a small quantity of turbid urine. In the small intestine the mucous membrane exhibits a light rose-red colour in the upper part; further down small round or elongated ulcers, with reddish swollen edges, and evident miliary deposition in the fundus, are met with. The ulcers occur only in a scattered manner, scarcely exceed the size of a four-skilling piece, and are all situated in the Peyerian patches; in other places yellowish tubercles of miliary size are seen. In the cæcum only a couple of elongated ulcers lying across the intestine are found; the rest of the large intestine is free. The mesenteric glands are slightly swollen.

[Having given two of the author's cases nearly *in extenso*, as examples of the accuracy with which his observations are reported, I shall be obliged in the remainder of his paper, in consequence of the length to which this translation would otherwise run, to confine myself to his general remarks, adding only the headings of his cases.—W. D. M.]

CASE 3.—*Pneumonia dissecans acuta; Pneumonia chronica; Aneurismata arteriæ pulmon., unum ruptum; Hæmorrhagia pulmonalis. Anæmia et Œdema cerebri.*

Bertha Nielsen, a widow, aged 50, admitted on 19th February 1866. The patient, who had usually enjoyed good health, was seized on the 16th with shivering, pain in the left side of the chest, and cough, with expectoration at first rusty and afterwards whitish. On 7th March the expectoration became decidedly bloody, and continued more or less so until the afternoon of the 23d, when, having previously felt very well, she was attacked with a fit of coughing, during which the blood gushed from her nose and mouth, and she died in the course of a few minutes.

CASE 4.—*Phthisis cavernosa pulmon.; Aneurismata arter. pulmonal., unum ruptum in Cavernam. Hypertrophia cord. dextr. Peribronchitis, Bronchitis, Laryngitis, c. Ulc. tuberc. Cyanosis renum.*

E. H., aged 48, resident in the General Hospital for nearly two years with chronic pulmonary phthisis. She was suddenly seized on the 14th November 1867 with violent hæmoptysis, which speedily proved fatal.

Ectasias are smaller aneurismatic dilatations of vessels, running in the walls of cavities. They occur under two forms: first, as cords of different lengths on the inside of the cavity, which on being slit up exhibit a slight dilatation of their calibre in towards the latter, with corresponding thickness of the walls; occasionally the thickness even becomes so considerable that the calibre of the vessel is at all events apparently diminished, so that we cannot properly speak of an ectasia. This form is the rarest. In the eight cases, which constitute the immediate basis of our description, it was observed only once in combination with a ruptured aneurism. I do not, however, mean to say that these ectasias are absolutely rare, but only in relation to hæmorrhages; thus, I have often seen them in the walls of cavities where no hæmorrhage had taken place during life. The second form is more frequently observed, in which the vessel comes in contact with the wall of the cavity only in a limited locality. In such a locality there is developed a rather oblong prominence, sometimes as large as a bean, though most frequently only as large as a pea, due partly to a dilatation of the calibre of the vessel, partly to a thickening of its wall. The perforation takes place always in a peculiar mode, a V-shaped slit forming in the wall of the vessel, whereby a kind of valve or lid-shaped flap is developed. The angle formed by the slit may be more or less acute, its sides shorter or longer, and the lid formed as the result thereof more or less easily movable. The rupture occurs in general in the boundary between the vessel and the wall of the cavity, and the apex of the lid lies always in the direction of the current of the blood. While

the walls of aneurisms proper are always thin at the seat of perforation, this is not invariably the case with the lid-shaped ruptures, as the lid is sometimes even remarkably thick and of fibrous consistence, so that its point, especially when at the same time the angle of the slit is large, can be raised only to a slight degree. But, as a rule, the lid forms a tolerably thin easily movable flap, of rather yellow colour. The vessel forming the ectasia is, as in the aneurism in general, empty or filled with freshly coagulated blood.

CASE 5.—*Phthisis cavernosa (Pneumonia chronica, Peribronchitis, Bronchiectasia). Dilatatio aneurismatica rami arter. pulmonal. perforat. (c. operculo), Hypertrophia cordis dextr. Nephritis interstitialis.*

Severin Peter Johnsen, aged 49, a labourer, was brought into hospital immediately after the occurrence of hæmoptysis, but died before admission to a ward, on the 28th December 1866.

CASE 6.—*Phthisis cavernosa c. Hæmorrhagia ex arteriola aneurismat., operculo rupta. Tubercula pleuræ; Hypertrophia cord. dextr. Cyanosis renum et hepatis.*

Juliane Kisling, aged 42, a patient in the third division of the Municipal Hospital, on the morning of the 20th October 1867, after having taken her tea as usual, and her condition not having altered essentially to all appearance from that of the previous days, sank suddenly back in the bed, and died in the course of a few minutes without any perceptible tracheal râle, and without any hæmoptysis.

CASE 7.—*Phthisis cavernosa pulm. dextr. Aneurisma parvum, operculo ruptum, arteriæ pulmonal. Hæmoptysis.*

Maren Andersen, aged 51, a widow, admitted to the General Hospital on 18th October 1866. Never had hæmoptysis until the afternoon of 20th October 1866, when, at half-past four, during a fit of coughing, a gush of blood suddenly came on, and proved fatal in the course of fifteen minutes.

CASE 8.—*Phthisis cavernos. pulmonum. Pneumon. chron. interstit. (et caseosa, Bronchiectasia). Hæmorrhagia e perforat. arteriolæ pulmon. c. operculo. Cyanosis lienis, renum, et hepatis. Hypertrophia ventric. dext. cord. Ulcerat. tub. intestinorum.*

Marie Nielsen, aged 28, admitted on the 25th of May 1864, having long suffered from chronic thoracic symptoms. On the 30th July, at 9 o'clock in the morning, very violent hæmoptysis suddenly set in, with symptoms of suffocation, and within ten minutes the patient was *in articulo mortis*.

Cavities.—Without in this place entering more fully into the so much debated question of the relation of miliary tubercle to chronic pulmonary phthisis, which I believe I am so much the more justified in omitting, as the detailed reports of the dissections are

presented in as far as possible an objective form, I shall only summarily state the occurrence of miliary tubercles in other organs than the lungs. In the eight cases tubercles were met with only in four in other organs—namely, in the intestinal canal, in two; in the larynx, in one; in the pleura, in one. In one other case there were yellow tubercles in the intestine, but without any characteristic miliary form.

In the four cases in which no trace of miliary deposition was found in other organs, in three (the 1st, 3d, and 7th cases) even the so-called peribronchitic depositions were wanting, and only one lung was attacked—in two instances the right, and in the third the left. Under such circumstances the affection must undoubtedly be designated as chronic pneumonia, with bronchiectatic cavities. In the fourth (the 5th case) a similar condition was met with in both lungs, but at the same time there were fresh lobular pneumonic infiltrations, peribronchitis, and yellow tubercles in the intestine. In the cases in which miliarly tuberculosis had supervened, this was only to a slight extent, and in such instances also the cavity retained its bronchiectatic character—that is to say, we had a cavity of greater or less size, surrounded by a more or less broad belt of condensed pulmonary tissue, into which a varying number of bronchi opened with eyelet-hole orifices. It may therefore be stated, quite apart from the existing contest respecting tuberculosis or non-tuberculosis, *that every cavity in the lungs whose walls are formed by condensed pulmonary tissue, containing non-obliterated vessels, may be the seat of aneurisms or aneurismatic dilatations with consecutive ruptures;* most frequently it is, however, perhaps those cavities whose walls are formed only of condensed tissue a few millimètres in thickness, directly adjoining permeable tissue, which belong to this category.

The size of the cavity does not stand in any relation to the formation of the aneurisms; the latter may occur in the very largest, and in very small ones. Thus we have, in our fourth case, a cavity almost as large as an ostrich's egg, with two closely adjoining aneurisms of the size of peas; in our second we have a cavity of the size of a pigeon's egg, with an aneurism as large as a walnut. Where, as is often the case, we have many cavities of different sizes, and possibly all filled with blood, the source of the latter may be found in a very small aneurism, which may easily be overlooked, as in our 5th case, where the aneurismatic dilatation was discovered in a cavity of the size of a nut.

Of changes in other organs, hypertrophy of the right side of the heart was met with four times. I shall content myself with noting this fact, as I do not find myself in a position, at least for the present, to contribute anything to the explanation of the circumstances under wheih this hypertrophy arises, or to state why it is found in some cases, while in others apparently quite similar it is wanting. In the four cases large cavities were discovered; but, at the same time, there were tuberculous affections in other organs (larynx, intestinal canal, pleura). In seven cases the hæmorrhage

proceeded in four instances from the left, and in three from the right lung.

With reference to sex, five were females, and three were males. The ages were: in two, between 28 and 34 years; in five, between 42 and 51; in one, 64 years.

Formation of the Aneurism.—We have in a very few cases found slight atheromatous changes—that is, small patches of fatty degeneration—in the inner coat of the pulmonary artery; but even these are met with chiefly in the main trunks, and never in the branches immediately adjoining the aneurism. We have, therefore, no reason to seek the cause of the formation of the aneurism in changes in the artery itself. Everything would, however, indicate that the development of the aneurism stands in a definite causal relation to the formation of the cavity.

Rokitansky is, as we have seen, the only writer who, although rather summarily, speaks of aneurismatic bulgings in the walls of cavities. He looks upon the dilatation as caused partly by the want of support to vessels in the progressive ulceration, partly by the imbibition of the wall of the vessel, which becomes soft, jelly-like, and finally bursts. According to my observations, there is no doubt that the want of support is one of the chief elements in the formation of the aneurism; softening such as Rokitansky mentions I have never seen, as is sufficiently evident from the description, but fatty degeneration. That this may stand in a certain relation to, and possibly be dependent on, an influence derived from the contents of the cavity, I would not absolutely deny; but when side by side with such an aneurism we may find another, which also has been exposed to quite the saue influence as the first, but whose wall is considerably thickened and very strong, it seems to me to be more reasonable to refer the starting-point of both these changes, both the fatty degeneration and the thickening, to the wall of the vessel itself independently of its surroundings; that, notwithstanding the contents of the cavity, this may even develop vigorous life, the considerable amount of hypertrophy decidedly proves. From a more theoretical stand-point, it may perhaps seem suitable to parallelize these two changes in the vascular wall with the two processes proceeding side by side in the wall of the cavity—the formation of connective tissue and the ulceration; but direct observation does not admit of such an assumption. The thickening, or, as it may properly be called, the compensating hypertrophy, consists in fact in an hypertrophy of the coats of the artery, especially of the muscular coat, not in a development of connective tissue such as takes place in the wall of the cavity,—for which reason also the surface retains its natural smoothness, and the fatty degeneration has its seat in the normal elements of the artery, and is not an ulceration of newly-formed connective tissue. It is, however, hard to say why the vessel in some cases develops an energetic activity to ward off the threatening danger, while in others it remains passive.

The defective support of the wall facing the cavity acquires still greater importance from the fact, that the rest of the vessel ies impacted in a firm unyielding connective tissue, consequently, as the second principal element in the formation of the aneurism, we have the increased intravascular pressure. This must, particularly during a fit of coughing, be very considerable, on account of the thereby augmented determination of blood to the lungs; but even under quieter circumstances it is increased, partly because so many vessels in the condensed pulmonary tissue are obliterated, partly because, especially in the larger aneurisms, the efferent branch is very small, and is speedily lost in the condensed tissue—a fact which we have repeatedly had occasion to confirm. Finally, it deserves to be mentioned, that the aneurism is sometimes formed on the most prominent point of a vessel running in a curve, but most frequently both the afferent and the efferent branches lie in the same plane. Ruptures would, moreover, certainly occur more frequently if the vascular wall were not greatly thickened by a compensating hypertrophy; but that even this is not always sufficient to resist the strong intravascular pressure is seen from our 6th case, where the thickened firm vascular wall was removed layer by layer.

A remarkable circumstance in these aneurisms is the absence of coagula, even in those which attain the size of a walnut. As to the smaller ones, which form only a dome-like dilatation of the vessel, this is easily explained; not so, however, with regard to the larger ones. Yet it must be remembered that the aneurisms never have any proper neck, so that their contents stand in tolerably free connexion with the current in the vessel. It is perhaps also possible that by filling the cavity with air, the blood may be expelled from the aneurism when its wall is thin and yielding.

Although the views here stated rest exclusively on my own observations, it will not be uninteresting to compare with the latter the few cases to be met with in foreign literature; they amount, so far as I have been able to ascertain, to only three rather brief communications, which I therefore reproduce *in extenso*. It is remarkable that although the cause of the formation of these aneurisms, cavernous pulmonary phthisis, is so universally met with, and common to all nations, all the three reports are from England, which, as is well known, is distinguished among many other things by its richness in aneurisms.

[The papers referred to by the author are a letter by Dr Fearn to the editor of the *Lancet* (Derby, Jan. 1, 1841), *Lancet*, 1840–41, vol. i. p. 679, describing a case of aneurism of the pulmonary artery; a case of phthisis, fatal hæmorrhage by the rupture of a small aneurism on a branch of the pulmonary artery, under Dr Cotton's care (*Medical Times and Gazette*, vol. i., 1866, No. 811, for January 13); a paper by Dr Peacock, in the *Edinburgh Monthly Journal*, vol. iii., describing a case brought by him before the Anatomical Society of Edinburgh, where he exhibited, in 1843, the right lung of a man,

aged 29, who died suddenly after violent hæmoptysis. The present communication is so very long, that I must refer to the original sources for the observations referred to.—*Translator.*]

Thus, in Fearn's case, no coagula whatever were met with, although the aneurism had attained the size of a nutmeg. In Cotton's no coagula were found in one part of the aneurism, but in the ruptured portion there was an adherent, partially decolorized, coagulum. It is possible that both in these and in my own cases a small coagulum may have slipped away during the rupture, and so have escaped our observation.

Fatal Hæmoptysis in a clinical point of view.—We have already seen from the anatomical description, that aneurisms and ectasias may form at every stage of cavernous pulmonary phthisis, if there be found only condensation of the pulmonary tissue surrounding the cavity without obliteration of the vessels. Thus are excluded the forms of phthisis clinically designated as florid, and anatomically as gelatinous, scrofulous pneumonia, as catarrhal lobular pneumonia with caseous change. This is, of course, not the place to give a sketch of the clinical progress of pulmonary consumption, nor to attempt to bring our individual cases under the various subdivisions into which writers have of late years, with more or less success, endeavoured to separate the formerly compact nosological idea of phthisis. For this purpose, moreover, the cases are too few, and for it an independent clinical material would be necessary, which I have not been in possession of. I shall therefore confine myself to more practical remarks.

Hæmoptysis due to aneurism or to an ectasia of a branch of the pulmonary artery usually occurs suddenly without either the patient or the physician having the slightest suspicion of its impending supervention; and this is true, as has several times been stated, both in those cases where physical examination has shown only a slight condensation in one of the apices of the lungs, and in those where large cavities are met with in both lungs. During a fit of coughing or violent bodily effort blood rushes from the nose and mouth, and the patient dies most frequently in the course of a few minutes with tracheal râle and asphyxia; in rarer instances he sinks back pale as a corpse on his pillow with slight râle; and in still rarer cases, especially those in which, in consequence of extensive destruction of both lungs, considerable dyspnœa has occurred, so that he wants power to bring up the blood, he dies indeed suddenly, but without hæmoptysis, and it is only on dissection that we find the trachea and bronchi filled with blood.

Of our eight cases, death took place suddenly in five, and among these are found all the four in which we met with ectasias with operculated rupture, and only one with aneurism. But hæmoptysis does not always occur so violently and cause so abrupt a termination of life. In some cases it assumes a remittent character. The

patient gets more or less, but always copious, hæmoptysis, consisting at one time of dark coagulated or fluid, at another of bright and frothy blood. Each hæmorrhage is ushered in by a violent fit of coughing, and after this is arrested the ordinary expectoration is for some time infiltrated with blood; finally this becomes quite free from blood, until, in general after the lapse of a few days, a fresh hæmoptysis sets in. If the patient is not much exhausted by his pulmonary disease, he may in the interval regain his strength very well, notwithstanding the considerable loss of blood; if, on the contrary, he be in a more advanced stage of his disease, he rapidly becomes anæmic, and often dies directly of anæmia, as the last hæmoptysis, although not more considerable, or even less than the preceding one, at length brings death. Such a course, with remittent hæmorrhages, obtained in the other three of our eight cases, and these specially deserve our attention.

In our first case we see a man aged 64, strong for his time of life, who had for many years suffered from cough in the spring, disappearing towards summer. A month before his admission into hospital the cough became persistent, without, however, particularly troubling him; he then suddenly got for the first time moderate hæmoptysis, which returned after the lapse of a couple of days. He then applied at the hospital. The cough became more frequent, more troublesome, interfering with sleep at night; the expectoration lost its bloody character, and became scanty and viscid. His general health was good, his pulse 80. Objective examination disclosed signs only of slight solidification in the apex of the right lung. The hæmoptysis returned at intervals of from two to five days, and on the sixth, in the course of fifteen days from the first attack, the patient succumbed. This result is certainly very striking. We have grown up in the opinion, which also accords with that entertained by the first clinical physicians of the present day, that the hæmoptysis in itself has nothing to make us uneasy, and that it scarcely ever is the direct cause of death, but that the danger lies in the ulcerative process in the lung, of which the hæmorrhage is only the expression. We were therefore led to form a comparatively favourable prognosis, although the increasing cough and the hæmoptysis, according to the view hitherto generally received, indicated the development of latent phthisis. We might, however, if we adopted the theory taken up anew by Niemeyer, as to the relation of hæmorrhage to pulmonary consumption, have anticipated a rapid development of the phthisis. The calculations did not, however, hold good; the patient succumbed to the hæmoptysis alone. Dissection exhibited only a circumscribed alveolar and interstitial pneumonia, and in this a bronchiectatic cavity with a ruptured aneurism of the size of a nut, and any other source of the hæmorrhage must be entirely excluded; there was no trace of any tubercular affection, nor was there any pneumonic infiltration proceeding from the hæmorrhages.

In our second case we have an ordinary phthisis, already tolerably far advanced, with intercurrent pleuritis. This latter had quite disappeared, when the patient in a fit of coughing began to bring up red fluid blood, which in the course of rather more than twenty-four hours increased to profuse hæmoptysis, of which he died.

The third case is rather an isolated one, and it was not until we had learned more accurately to recognise this formation of aneurisms that we were on the whole in a position to propose any probable explanation of it. A woman aged 50, stated to have previously enjoyed good health, was admitted into the hospital with all the signs of acute pneumonia in the left superior lobe, though accompanied with a whitish, moderately viscid expectoration. In the course of this, and after the general symptoms had perceptibly diminished, copious bloody expectoration, with dark coagula, set in nineteen days after the commencement of the disease, accompanied with increasing dyspnœa, but without fever or pain in the chest. Violent hæmoptysis returned almost daily, or at very short intervals, and seventeen days after the first occurance of the hæmoptysis the patient died in a profuse attack of it. On dissection the left lung was found attached to the thorax by old adhesions of connective tissue, strongest at the apex. In the upper lobe of the left lung was a large cavity filled with blood; among this were loose pieces of pulmonary tissue in a state of red and gray hepatisation, and the wall formed of broken-up pulmonary tissue; the cavity was traversed by numerous branches of the pulmonary artery, and on two of these were two aneurisms on each, close to one another, one of which had burst; the inferior boundary of the cavity was formed of pulmonary tissue in a state of chronic pneumonia.

In this certainly unique and interesting case we had most probably one, or perhaps two, central cavities of a bronchiectatic nature in the upper lobe of the left lung, surrounded by condensed pulmonary tissue. From the wall of this cavity the aneurisms were developed in the usual mode, but this circumscribed chronic pneumonia with bronchiectasis did not particularly inconvenience the patient. When in the surrounding hitherto healthy pulmonary tissue an acute croupy pneumonia arose, the pre-existing chronic pneumonia became dissecting, or, if we will, gangrenous, as happens in rare cases, for one or other reason, perhaps on account of the disturbances in nutrition. The following points are in favour of the development having taken place in this mode: the form of the aneurism, which in all was sacciform with a lateral dilatation of the vessel, just as we have seen it in the other cases; the thickening of the walls of the aneurisms, which decidedly indicated that they must have existed for a long time; the size of the cavity in proportion to the included pneumonic portions of lung, which seemed to lead to the supposition of the pre-existence of a cavity; the inferior boundary of the cavity, which was evidently formed of

condensed pulmonary tissue; finally, the chronic pleuritis in the apex of the lung may be mentioned. It is possible that the hæmorrhages may in part have proceeded from the dissecting pneumonia, and that only the last fatal one was derived from the ruptured aneurism. This question cannot be decided, for hæmorrhages may proceed also from such a pneumonia, as occurred in a well-marked case communicated a couple of years ago by Lector Reisz; but, on the other hand, we have also seen that such an intermittent hæmorrhage may be due exclusively to a ruptured aneurism. Some reasonable objection may, however, be made to this explanation: thus, I may mention the absence of the signs of chronic pneumonia, for the journal states that the patient had previously enjoyed good health; but what I lay still greater stress upon is, that the dissection did not exhibit firmly condensed pulmonary tissue, but only recent pneumonic, although the possibility that this may have been brought up with the copious bloody expectoration, and so have escaped observation, cannot be denied. Nevertheless, these objections do not appear to me to be so important as to shake the explanation above given, as it is supported by certain well-established facts.

In our three cases of aneurisms, therefore, death occurred not suddenly, but was preceded by hæmorrhages with greater or less remissions, and it consequently becomes a question which deserves to be considered, whether the anatomical investigation gives us any basis for the elucidation of this circumstance. It is evident that when a portion of the vascular wall is raised like a valve by the strong pressure of the blood, a profuse and fatal hæmorrhage may be the final result; and that the same may be the case with an aneurism which bursts at once, when it does not contain plugging coagula. We have, however, not been in a position to exhibit such firm decolorized coagula, but we have already stated that such may possibly have escaped through the perforation. On the other hand, we have in one case, and precisely in that (the first) where the remitting hæmorrhages occurred in the most characteristic form, found a condition which may possibly have some signification—namely, fatty degeneration of the wall of the aneurism. It occurred in small distinct points, and first became diffuse towards the seat of perforation; it is therefore possible that, before the more extensive rupture, very small openings formed through which the blood made its way, but in smaller quantity, into the cavity, and there got time to coagulate, until a fresh violent fit of coughing loosened the plugging coagulum or produced a new rupture, and so gave rise to fresh hæmorrhage. As the matter at this moment stands before me, I do not find myself in a position to give any more satisfactory explanations of these remitting hæmorrhages in aneurisms in the walls of cavities, but will await the result which coming investigations directed more precisely to this point shall give.

This peculiar, and in a practical point of view important, circumstance is scarcely purely accidental, as one might, from the limited number of cases here spoken of, be inclined to suppose; for if we include the two cases by other writers, above communicated, we find the same. In Fearn's case, violent hæmoptysis occurred from the 12th to the 19th December; then a long remission took place until the 25th, on which day the fatal hæmoptysis suddenly supervened. The aneurism, which was as large as a nutmeg, burst with a fissure, but contained no coagula. In Cotton's case, where the patient was in the last stage of phthisis, after previous moderate hæmoptysis, a very violent attack suddenly supervened; thereupon came a remission of three weeks' duration, when the hæmoptysis was repeated with the same violence, and proved fatal. The ruptured aneurism, which was in size less than a pea, contained an adherent, but only partially decolorized coagulum, while the other, which was separated from the first only by a slight constriction, did not contain coagula. The description of this coagulum, taken along with the total absence of any coagulum in the second aneurism, placed under precisely similar circumstances, decidedly indicates that the clot was not formed previously to the rupture, but that it proceeded from the last hæmorrhage, three weeks before death. This case seems, therefore, to be in favour of the explanation given above.

These instances, consequently, show decidedly that we must be cautious in our prognosis, when we have a profuse hæmorrhage before us, for even if it diminish or entirely disappear it may return, and in a few moments prove fatal. Upon this point I shall quote the following words of Fr. Hoffman, which seem to have been long forgotten: "In illa gravi et vera hæmoptysi, qua, ob ruptum majorem arteriæ pulmonalis ramum, magna sanguinis copia per brevia intervalla erumpit, caveat medicus ne promissis de restituenda sanitate sit dives, nisi fluxus sanguinis intra triduum convenientibus remediis compescatur."[1] The cases communicated by us show, however, that even if the hæmorrhage stops in the course of three days, we can by no means be easy. I believe that we must be the more cautious, as we are clinically scarcely in a position to distinguish an hæmoptysis due to rupture of a vessel in the wall of a cavity from another occurring during the course of pulmonary phthisis, and which may possibly have another cause. One of Niemeyer's pupils, Bürger,[2] has, as well as Niemeyer himself in the last edition of his manual, thought that the blood brought up, which proceeded from a branch of the pulmonary artery, must be absolutely dark, as this artery contains in fact the most venous blood in the whole body. Now since, as is well known, all observers are agreed that the blood in hæmoptysis is almost always of a light

[1] Fr. Hoffman Opera Omnia, t. ii. sect. i. cap. ii. Genevæ, 1748, p. 205.

[2] Ueber das Verhältniss der Bronchial und Lungenblutungen zur Lungenschwindsucht. Tübingen, 1864, p. 18. (On the relation of Bronchial and Pulmonary Hæmorrhage to Pulmonary Consumption.)

red, they see in this a proof that the blood in the vast majority of instances proceeds from branches of the bronchial arteries (broncho-hæmorrhagie); or, at all events, from the pulmonary veins, and only in extremely rare cases, where the blood is then dark, from an eroded branch of the pulmonary artery. This reasoning is, however, entirely theoretical, and they do not support it with a single anatomical fact; for, in the only example which they quote, the dissection, as I shall subsequently take occasion to show, by no means gives reliable information as to the source of the hæmorrhage. In our cases, where the hæmorrhage evidently proceeded from the pulmonary artery, the blood was most frequently of a light red colour; in a couple of cases (the first and third), it was alternately dark and light; in Fearn's case it was dark. This circumstance ought not, therefore, to be advanced as a certain diagnostic sign. The colour of the blood depends most probably on the rapidity with which it is brought up, and the quantity of air with which it is mixed. That it is precisely highly venous blood which rapidly takes up oxygen is shown by Alex. Schmidt's[1] recent experiments upon the blood of asphyxiated animals, in which he found that this, although in a different degree from the various organs, contains a substance which rapidly takes up and combines with the oxygen.

If we now ask what importance and extent we ought to award to these aneurisms and ectasias of vessels running in the walls of cavities, as the source of pulmonary hæmorrhages in general, this question is of course difficult to answer from the data hitherto made available. I can state only that, during the last two years, since I have had my eyes opened to these conditions, I have always been able to demonstrate the rupture of a vessel in the wall of a cavity in the mode described; while I earlier have made some post-mortem examinations in which I could not point out the source of the hæmorrhage, but in all these cavities were found, so that I have no doubt that it has had the same origin as in the later cases. Vessels enclosed in trabeculæ passing through a cavity ruptured by ulceration, I have never seen; but of course I do not venture to deny their existence, which is confirmed by too many reliable observers. As to the frequency of the fatal hæmoptyses occurring in this mode, they took place in the Municipal Hospital, during the last two years, in the following numbers:—

In 1866, among 79 dissections of phthisical patients, four times, or 5 per cent.

In 1867, among 104 dissections of phthisical patients, once, or 0·9 per cent.

There thus appears a considerable difference in the two years; but, rightly considered, these statistics do not prove much, for patients do not apply to the hospital on account of their aneurisms, and it is a mere chance if the rupture takes place during their stay

[1] Sächs. acad. Sitzungsberichte. Math. phys. Classe 1867. Centralbl. f. d. med. Wissenschaft, 1868, No. 3.

in the house. The sudden death which carries off the majority of these patients causes them only exceptionally to come upon the dissecting table, as death, even if they seek the hospital, most frequently occurs before they reach it.

That profuse and fatal hæmorrhages always proceed from cavities, appears to me, according to my observations, to be a legitimate conclusion. We have seen that a cavity of the size of a nut may give rise to fatal hæmorrhage, and that the cavity may be still less is probable, or at all events possible; but that such a cavity may be overlooked, and has been overlooked, especially formerly, scarcely any one will deny; for this kind of investigation requires great patience and perseverance.

The cases where copious hæmorrhages occur in apparently healthy individuals, and even cause death, and in which no changes are found in the lungs, ought, I believe, to be received with great caution. From what has been above brought forward, I can therefore by no means agree with Niemeyer when he thinks it improbable that cavities should be overlooked, and that it would be very strange if hæmorrhages from small overlooked cavities should be more frequent than from larger ones. This last is only a theoretical argument, and is quite without importance in presence of the certain demonstration that the occurrence of hæmorrhage is entirely independent of the size of the cavity.

Another question which naturally presents itself is, whether more considerable hæmorrhages, which do not cause death, and indeed after which the patients may even live for many years without any particular annoyance, may depend upon anatomical conditions similar to those we have met with in the fatal cases. The material in our hands affords no information in this respect; but as the question as to the source of pulmonary hæmorrhage has hitherto been all but confined to the domain of hypothesis, there can be the less hesitation in forsaking the beaten track of observers, as we have certain palpable facts on which to rest.

As we, in fact, in a certain number of cases of chronic pulmonary phthisis in its different stages, have established as the proximate cause of death a rupture of an aneurismatic dilatation of a vessel running in the wall of a cavity, it is not merely probable, but is even indubitable, that such aneurismatic dilatations may occur in another greater or smaller number of cases without giving rise to hæmorrhage, as in this instance death occurs before the rupture of the aneurism can take place, in consequence of the advanced nature of the consumption. We have, moreover, with ruptured aneurisms, found others which were not ruptured, and in many cases, without previous hæmorrhage, met with dilated, thickened vessels on the inside of the walls of cavities. The percentage of the aneurismatic dilatations may therefore really be fixed much higher than has been stated above; although I cannot give this proportion decidedly, I nevertheless believe that this conclusion is on the whole justified.

On the other hand, it is also probable that the aneurisms may be much smaller than we have observed in the fatal cases, and further, that the vessels on which they are developed may be less. Hence there is, it appears to me, nothing to hinder the assumption, that very small aneurisms may burst and give rise to a hæmorrhage which stops, partly on account of a spontaneous formation of thrombus in the ruptured vessel, partly on account of a defective escape through the cavity, whose eyelet-hole bronchial orifices may easily be obstructed, either by inspissated secretion or by swelling of the mucous membrane, thus giving the blood time to coagulate in it. Similar conditions may still more easily be supposed to be capable of taking place in those instances where the hæmorrhage proceeds from the eroded vessels situated in the walls of the cavity or passing through the latter; and such cases may, to judge from the statements of writers, be much more frequent than those described by me.

Such an explanation takes at least its starting point from undoubted anatomical facts, and seems to me, at least so far as the more violent pulmonary hæmorrhages are concerned, more satisfactory than that generally received respecting bronchial hæmorrhages. It is indeed curious that in our time, when so much prominence is given to anatomical investigation, so theoretical a view should be so strongly retained. The hæmorrhages cannot come from cavities, it is said, because the vessels in the walls and their immediate neighbourhood are obliterated, consequently they must come from elsewhere, and then most probably from the bronchi. But it is a fact that no one has been able, with the least probability, to demonstrate the place where the hæmorrhage has occurred in the bronchial mucous membrane; but, on the contrary, in every case in which the source of the hæmorrhage could be pointed out, it has always been a ruptured vessel in a cavity. Nevertheless, on the strength of the theory respecting the obliterated vessels, this only certain source has been reduced to a pure accident, to a rare exception, from which no conclusion whatever ought to be deduced respecting pulmonary hæmorrhages in general. To this only certain observation, the origin of pulmonary hæmorrhages from cavities, whose frequency, according to the cases above communicated, seems to have been much undervalued, I believe we ought to adhere; and in the revision, to which the whole subject of phthisis is at present subjected, the vessels also ought to be included. It is often easy, even by simply slitting up the vessels with scissors, to satisfy one's self that the vessels in some cases pass into the walls of cavities, especially in those cavities where the wall is very thin, and immediately adjoins the permeable pulmonary tissue. That the vessels are very rare, or are perhaps even altogether wanting in the highly condensed, almost cartilaginously hard, slate-coloured indurations surrounding old cavities, is undoubted; but we ought not from this to deduce any conclusion respecting cavities in general. I do not find myself in this place in a position to give decided

information on this point, which can be supplied only by comprehensive and careful injections; but I believe that the anatomical facts which are here brought forward afford strong proof of the necessity of instituting such investigations.

According to these views, therefore, pulmonary hæmorrhages, or at least the more violent ones, come to stand in a more remote relation to tuberculosis than has hitherto been admitted; indeed, are related to it properly only so far as chronic pneumonia with bronchiectasis is so frequently connected with tuberculosis. This is also the reason why even copious hæmorrhages in general do not affect patients except by the loss of blood, as chronic pneumonia with bronchiectasis is not, as such, attended with direct danger to life.

I do not, however, mean to say that we can explain every parenchymatous pulmonary hæmorrhage as proceeding from a cavity. In the more florid forms of phthisis this is certainly due to a breaking up of the infiltration. Nor is it probable that the slighter hæmorrhages, which occur in the commencement of chronic consumption, proceed from cavities; in this case I should be rather inclined to refer them to capillary pulmonary vessels, which have been perforated by reason of the development of tubercle in their adventitious tunics; but, as I have said, positive data in this direction are entirely wanting. I have on the present occasion been anxious only to give to hæmorrhages from cavities a much greater importance and extension than has hitherto been done. As it may possibly be objected that, in many cases of chronic phthisis, cavities are not met with, I will state my belief that this is an opinion which more careful investigation will not confirm; for it is, at least according to my observations, extremely rare in examining the bodies of phthisical patients not to find cavities, small though they may be, if the investigator will only not be satisfied with seeking them in the apex and in a single longitudinal section through the lung.

It cannot be expected that from the above materials, based exclusively upon cases which have terminated fatally, any direct advantage should result for treatment; but I believe that the facts here brought forward show the necessity of taking, if possible, still greater care than hitherto in every more considerable pulmonary hæmorrhage; and especially of employing suitable means, and, best of all, narcotics, to allay the fits of coughing, and to procure the patient in general the greatest possible quiet, so that the thrombosis in the ruptured vessel may have time to form. This, in connexion with a cooling regimen, is certainly the principal thing; and the different remedies, as digitalis, secale cornutum, acetate of lead, oil of turpentine, etc., to which recourse is usually had, are probably rather injurious than beneficial.

[The author appends the history of a corroborative dissection which he had an opportunity of making while engaged in writing the foregoing. The case was that of a student aged 17, both of whose parents had died of phthisis. Having himself laboured for some

time under the same disease, he died suddenly of hæmoptysis. In the upper half of the left lung were found cavities of various sizes, all of which, as well as the bronchi, even the smallest, were filled with freshly coagulated blood. On the inner wall of the largest of these cavities was found a projecting ledge, in the base of which ran a branch of the pulmonary artery of the size of a barrel of a small quill, giving off a smaller branch which ascended to the upper margin of the ledge, where a lid-shaped rupture had taken place.]

II.—Hæmorrhages from a ruptured Aneurism of the Aorta.

Of the twelve cases of fatal hæmoptysis which form the basis of this essay, the source of the hæmorrhage was, in three, a ruptured aneurism of the aorta. The rupture occurred once in the ascending aorta and the arch, twice in the descending thoracic aorta. The perforation took place once into the right principal bronchus, once into the left, and once into the left lung.

As to the hæmoptysis due to a ruptured aneurism of the aorta, most writers are contented with stating that it is profuse, and that it rapidly causes death. Lebert[1] who, in the 83 cases analyzed by him, met with rupture into the bronchi fifteen times, states that the profuse and rapidly fatal hæmoptysis is indeed the most frequent, but that in some cases also it may occur in the last two or three days before death in several violent attacks. Such violent attacks of hæmoptysis took place in one patient even three weeks before death.[2] The violence of the hæmorrhage depends in this instance, as in aneurisms in general, upon whether the rupture of the organ concerned takes place with a single large rent, or with one or more small openings, or upon whether the coagula enclosed in the aneurism are in a condition to offer a greater or less resistance to the pressure of the blood. Only in one of our three cases did the hæmoptysis occur suddenly and produce immediate death, and in this case coagula were completely wanting. In this, our first observation, the aneurism presented itself during life with all its characteristic signs, so that the diagnosis was beyond all doubt. The patient, a man aged 35, was comparatively well immediately before the rupture, which occurred just as he was about to go to bed, during a fit of coughing, and the quantity of blood brought up amounted to one or two quarts. On dissection, the ascending aorta and also the arch were found uniformly dilated; the boundary of the descending aorta was sharply defined. The walls of the sac-like dilatation thus

[1] Virchow, Handb. d. spec. Pathologie u. Therapie, 5 Bd. 2 Abth., 1855, p. 24.

[2] Slight attacks of hæmoptysis frequently occur during the course of aneurisms of the aorta, without any rupture having necessarily taken place into the air-passages, as they may be due exclusively to disturbances of the circulation in the lungs, produced by pressure of the aneurism. Thus, the celebrated English surgeon Liston, during the space of eight months, frequently brought up small quantities of blood.

formed were uneven, thickened, yellowish, of almost cartilaginous firmness, only here and there studded with some small calcareous plates, especially in the ascending aorta; in the arch the walls were thinner, and in some places small flat, soft, rather decolorized coagula adhered to the wall, but elsewhere the whole sac was free from these. The inner side of the sac was found adherent to the root of the left lung, especially to the posterior surface of the left principal bronchus, and the sac had burst into the latter with an irregularly torn opening with highly attenuated yellowish edges, and capable of admitting the point of the little finger. The adjoining larger bronchi as well as the trachea were filled with blood. The left lung was everywhere firmly attached to the thoracic wall by thick membranes of connective tissue of long standing. It was almost devoid of air, with numerous fresh lobular pneumonic infiltrations, which in the apex of both lungs presented a lardaceous appearance, while the intervening tissue was strongly condensed, slate-coloured, with some small smooth-walled cavities. The right lung was permeable throughout, except in the apex, where a slate-coloured condensation of more than the size of an egg was met with, but without the formation of cavity. Nowhere in the lung or in any other organ was there a trace of miliary deposition.

It is a generally received opinion, which is looked upon by almost all writers as correct, that aneurisms in general, but especially those of the aorta, do not occur together with pulmonary tuberculosis, and this opinion is supported even by Rokitansky's[1] ample experience. Lebert, however, states that tuberculosis was met with three times in his 83 cases. We have already, in the foregoing observation, seen chronic pneumonia with and without bronchiectasis, and a recent catarrhal pneumonia occur together with an aneurism of the aorta; but as an undoubted case, though not of tuberculosis in the modern rigorous acceptation of the term, yet of well-marked pulmonary phthisis, the following deserves to be communicated:—

Aneurism of the descending thoracic aorta, with perforation of the right bronchus; bronchial and pulmonary hæmorrhage, slight pulmonary emphysema, hæmorrhage from the stomach and intestines. Cavernous pulmonary phthisis (Chronic pneumonia, Bronchiectasis, Peribronchitis).

H. P. V. Jensen, a musician, aged 39, suffered for about twenty years from cough and dyspnœa, not so severe however as to prevent him taking part in the first Schleswig war, where he received a gunshot wound through the nates, opening the rectum, for which he was under treatment for nearly a year. Three years before his admission, which took place on the 3d April 1865, he had hæmop-

[1] Handb. d. patholog. Anat., 2 Bd. 1856, p. 333. This statement of Rokitansky stands, however, in striking opposition to another (*l. c.*, p. 330), where he mentions a rare case of the rupture of an aneurism of the aorta into a tuberculous cavity.

tysis, though not to any very considerable extent. In January 1865 he was for three weeks under treatment in hospital for the same symptoms. On 1st and 2d April, the latter returned with some violence. On the 7th, very violent hæmoptysis set in, and was arrested by the employment of the ordinary means. It returned, however, in the evening, and terminated fatally. [The author details the post-mortem appearances at full length, and proceeds to make the following remarks:—]

In this instance, the signs of chronic pulmonary phthisis completely masked the aneurism, which, as is frequently the case, especially with aneurisms of the descending aorta, did not reveal its presence by any more prominent symptom,—for even the pains in the back, which may often cause the existence of aneurism to be suspected, were also wanting. The dyspnœa was indeed very considerable, amounting even to orthopnœa; but was, during life, readily explained by the complication with severe bronchitis. As to the hæmoptysis, which clinically was necessarily placed in connexion with the established pulmonary phthisis, the careful post-mortem examination leaves us in doubt whether it proceeded both from the aneurism and from the cavity discovered. With respect to the hæmoptysis which occurred three years before, only conjectures can be formed; but it is probable that this proceeded from the cavity, and the later hæmoptysis from the aneurism, the softness of whose cogula permitted the blood to ooze through.

In our third case, a large aneurism of the descending thoracic aorta was met with in a man aged 45, attached by strong adhesions to the posterior surface of the upper lobe of the left lung, and opening, in a circumscribed situation, into the lung. The latter had thus become highly infiltrated with blood, which was met with also in the bronchi and trachea. At the same time there was exudative pleuritis on the right side, and incipient granular atrophy of the kidneys. There were very violent and increasing attacks of dyspnœa, and the hæmoptysis was present during the four days preceding death, at first in a moderate form, subsequently it was very violent.

In conclusion, I shall make some further remarks, although of a more aphoristic nature, and directly elicited by Niemeyer's work,[1] recently published and quoted in this Journal, namely, on the relation between hæmoptysis and phthisis.

It is well known that, until the time of Laennec, hæmoptysis was looked upon as one of the most frequent causes, though far from being the sole cause, of phthisis, and the so often repeated proposition, "Pus sanguinem sequitur," can be traced back to the Aphorisms of Hippocrates. Laennec described this theory as an inconsiderate application of the so frequently misused axiom, "Post ergo propter." He does not recognise any other phthisis than that due to tubercles, and hæmoptysis is for him an undoubted sign

[1] Klinische Vorträge über die Lungen-schwindsucht, mitgetheilt v. Dr Ott., 2 Aufl., 1867; Hospitalstidende, 1867, Nos. 37, 38, 39, and 40.

of the presence of tubercles, and one of the first symptoms by which the tuberculosis reveals itself. This view of the relation of pulmonary hæmorrhages to pulmonary consumption has obtained general acceptance, and is still, in the present day, that adopted by the most eminent clinical physicians in all countries, if, instead of tuberculosis[1] in Laennec's specific meaning, we substitute pulmonary consumption. The other view has, however, been again embraced by Niemeyer, who sees in hæmoptysis one of the most frequent causes of phthisis.[2]

According to Niemeyer, after an attack of hæmoptysis, signs of a more or less violent irritation of the lungs and pleura almost constantly set in. Clinically the latter manifests itself, nearly without exception, on the second or third day after the hæmorrhage, by increase of temperature and acceleration of the pulse, disturbed general health, stitch in the side, and the well-known physical signs of pleuro-pneumonia. The usual result of this, however, is resolution, and the patient is often convalescent after the lapse of even a few days; but in other cases the subjective and objective symptoms increase, and a well-marked florid phthisis, derived from the fact that the blood remaining in the bronchi and cells has given rise to pneumonia, with caseous metamorphosis of the retained blood and products of inflammation, is gradually developed. Niemeyer rests chiefly on a single anatomical investigation. This case was that of a man, aged 32, who was greatly reduced with tertiary syphilis, and had signs of cavities and of solidification in the apex of the left lung. He recovered very well under the use of iodide of iron, cod-liver oil, and nourishing diet, especially milk, when he suddenly got one night profuse hæmoptysis. For the next two days he was tolerably well, except that he was very weak; the physical signs were as before, only that a finely vesicular râle was heard over the whole of the left lung. On the third day the patient complained of lancinating pains in the region of the left nipple, where a friction sound was heard. The pulse and temperature were increased. Nine days subsequently, a slighter hæmoptysis came on, and now a florid phthisis was rapidly developed, to which he succumbed in four weeks from the first attack of hæmoptysis.

[1] This view of a phthisis ab hæmoptoe (Morton) is, however, adopted also by Andral (Clinique Méd., t. 4; Paris, 1834, p. 36) in certain cases where hæmoptysis suddenly occurs in hitherto healthy individuals, and is followed by signs of phthisis. The hæmoptysis is explained by a congestion of the lung (Apoplexia pulm. Laennec), and the tubercles are developed partly by reason of the disturbed nutrition in the affected parts of the lungs, partly by reason of predisposition. A German clinical physician, too, who would deserve to be mentioned in a German work, Schönlein (Algem. u. spec. Pathologie und Therapie, 2 Bd. 2 Aufl., 1832, p. 126), mentions phthisis among the results of idiopathic pneumorrhagia; he leaves it undecided whether the extravasated blood becomes the starting point for the formation of tubercle or not; in hæmoptysis in scrofulous individuals, at all events, this result can scarcely be doubted. Among other results, pneumonia also is mentioned, though only in its first stage.

[2] *Loc. cit.*, p. 51.

On dissection a slight hæmorrhagic, pleuritic exudation was found on the left side; in the apex of the left lung was a cavity as large as an egg, with considerable shrinking of the surrounding tissue; on the inside of this was a large arterial branch, "whose walls are considerably thickened, its cavity somewhat diminished, but not closed; no opening or erosion is demonstrable." The whole of the inferior lobe was filled with infiltrations from the size of a pea to that of a nut, lying close together, and partly confluent; the intervening permeable tissue constituted only about one-fourth of the whole tissue. On the surface of section the infiltrated parts exhibited a distinctly granular appearance, were of a red, gray, or grayish red colour, just like croupy pneumonia in a state of transition from red to gray hepatisation. In the centre of many of these foci the tissue was coloured yellow, in others it was at the same time caseous, dry, firm, and sharply contoured. Directly beneath the pleura lay numerous yellowish transparent cavities, about as large as peas, in the midst of a caseous tissue, and evidently formed by recent decay of caseous infiltrations. A medium-sized bronchus, which ran down towards the outer inferior margin, was filled with a tough puriform mass, constituting, through an extent of two inches, a firmer puriform plug, giving the whole bronchus the appearance of a vein plugged with a thrombus; it was adherent to the wall, where the branch of the air-tube ran through the infiltrated tissue; the wall was here considerably thickened and firm, the mucous membrane on the adherent part was as if diphtheritically infiltrated, so that after the separation of the plug there was loss of substance; in the finer ramifications the plug passed into a puriform fluid. In the apex of the other lung was a small cavity of longer standing, and in several places there were peribronchitic nodules, but nowhere in the lungs or other organs were there tubercles.

This case appears to me, however, not to be a very convincing one. First, as to the anatomical conditions, Niemeyer ought to have fully shown that the hæmorrhages proceeded from the large cavity; for they cannot possibly have been derived from the greatly thickened vessel described as running in its wall. According to the view I have given of the pathological anatomy of pulmonary hæmorrhages, I should incontestably have been most inclined to seek the starting point of the bleeding in the cavity; but this cannot be proved by the dissection, and I do not see how Niemeyer will be able to refute the assertion, that the hæmorrhage in this instance may have been derived from the decay of smaller pneumonic infiltrations,—these have in fact, as the record of the post-mortem examination distinctly shows, a very different standing, the greater part being recent, red or grayish, a smaller part having already fallen into detritus and forming cavities; for that hæmoptysis may arise in the course of a caseous pneumonia with the formation of cavity, can scarcely be denied, and that at least the last attack of

hæmoptysis in the case stated has had this origin, appears to me even to be most probable.

I shall next dwell for a moment on the peculiar thrombus formation in the bronchi, which was, however, met with only to a slight extent. This is a circumstance which has scarcely been before observed, but which cannot possibly be overlooked by any conscientious investigators, and it must therefore undoubtedly be considered as extremely rare, and most probably depending on peculiar circumstances in the concrete case. In general, the blood remaining in the bronchi after an attack of hæmoptysis is brought up in the course of some few days, intimately mixed with the ordinary expectoration; that this did not happen in the instance before us, may depend partly on the considerable languor and debility of the very much exhausted patient; but its presence seems to me to be with equal probability explicable from the fact that, previously to the hæmorrhage, an incipient infiltration of the lung existed, preventing the blood being discharged as usual; an infiltration which was very extensive, corresponding to the cavities and caseous parts met with on dissection, and that therefore it could not be clinically demonstrated.

While, therefore, in Niemeyer's case it must remain doubtful whether the hæmorrhage proceeded from the large cavity, or was due to the breaking-up of older scattered pneumonic infiltrations, we have, on the other hand, among our own observations, a number of cases in which, for a long time before death, there were abundant hæmorrhages, whose source we have anatomically shown to be a ruptured vessel in the wall of a cavity. This case seems therefore to be necessarily specially adapted to show this peculiar irritative effect of the blood, supposing that such actually exists. We shall consider this case more closely from this point of view. In our first case we have a chronic pneumonia, with a bronchiectatic cavity in the apex of one lung, from which, in the course of a fortnight, with a few days' interval, six copious hæmoptyses took place. After none of these attacks did we observe during life the slightest trace of this consecutive pleuro-pneumonia occurring "almost without exception,"—the general health was undisturbed, the pulse 80. On dissection we found no trace of any pneumonic infiltration. I attach special importance to this case, because it is not complicated, like Niemeyer's, in which there were found old solidifications with cavities, old and recent pneumonic infiltrations and peribronchitis, and because the source of the hæmorrhage was anatomically demonstrated to be entirely independent of any other destructive process in the lungs. Here, therefore, we have an opportunity of seeing the blood develop its irritative effect pure and simple. Neither in our third case did we find, after abundant hæmoptysis during seventeen days before death, any secondary pneumonia. In our second case of an aneurism of the aorta in connexion with chronic pulmonary consumption, where both old and recent hæmoptyses

occurred, we found nothing of the kind, nor in Fearn's and Cotton's cases, notwithstanding the copious hæmoptyses. I may here mention also an older case, which is not included in this review, because the source of the hæmorrhage was not discovered, but which, nevertheless, deserves mention in connexion with this subject. It is that of a man, aged 46, who for about a year had laboured under consumption, during which he had several times expectorated blood. On admission into hospital on the 28th September 1864, he was very much exhausted, and had signs of large cavities in the apex of each lung. On the 1st October he coughed up four ounces of blood, and almost every day afterwards until the 13th October, when the fatal hæmoptysis came on, he brought up considerable quantities of blood, even as much as a pint. His pulse was 84, his general health was unchanged, except that he had considerable debility. On dissection, large cavities were found in the apices of the lungs, miliary tubercles in the lungs and pleura, alternating hæmorrhagically-infiltrated and emphysematous lobules, but no trace of any pneumonic infiltration.

If we look next to the second factor in this condition,—the pneumonic infiltration,—it is clear enough, as Niemeyer himself states, that, together with chronic solidifications or ulcerative processes in the apices of the lungs, catarrhal pneumonias with caseous metamorphosis extremely frequently occur. But recent catarrhal pneumonias also frequently coexist with these older affections, and spread over greater portions of the lung, with reddish or reddish gray, in some parts perhaps even yellowish coloration, and a cribriform rather than granular surface of section. These may occur—nay, do so even most frequently—without any previous hæmorrhage. Even in the limited number of cases made use of in this paper, we have an example of this, namely in our first case of a ruptured aneurism of the aorta. Here we have old solidifications in the apices of both lungs, in the left combined with bronchiectatic cavities; in the same lung, and also in Niemeyer's case only in the one lung, numerous recent pneumonic infiltrations, which in the apex had already assumed a lardaceous appearance. In these cases, hæmoptysis had never occurred previously to the rupture of the aneurism which instantaneously caused death. It is worth observing what a large part the accidental may play in a single example; for, if the hæmorrhage from the aneurism had, as in our second case, preceded death by some time, this instance might have been quoted as a striking proof of the correctness of Niemeyer's theory.

When we thus find, as a frequent complication of chronic solidification in the apices of the lungs, with or without the formation of cavities, both old caseous and recent and diffuse pulmonic infiltrations, without any hæmorrhage having previously occurred; and, on the other hand, in a not very small number of cases, in which abundant hæmorrhages have for a long time preceded the fatal

hæmorrhage, have neither clinically nor anatomically observed the slightest trace of pneumonia,—I believe that we cannot be careful enough in our conclusions as to the mutual relations between hæmoptysis and pneumonia, if we wish to escape the rock *post ergo propter*. When, therefore, Niemeyer with so much vehemence casts it in the teeth of Laennec, that his doctrine rested on a very weak foundation, and was based upon theoretical reasoning, I believe that the same reproof may with equal justice be turned against himself, for he at once, with a theoretical dictum, decides the question which above all requires to be solved,—the anatomical foundation of the hæmorrhages,—setting up as almost the exclusive cause of the hæmoptysis a broncho-hæmorrhage, depending on a supposed brittleness (hæmorrhagic diathesis) in the arteries of the bronchial mucous membrane, and does not take into account that precisely the acute caseous (tubercular) pneumonia itself may undoubtedly be the cause of the hæmorrhage. Fully appreciating the interesting and talented views of the gifted clinical physician, I do not, however, believe that on this point he has satisfied the rigid requirements of our day for anatomical and clinical observation, or has on the whole laid so firm a foundation that any lasting structure can be reared upon it. A single observation, even if it were much more convincing than it really is, is quite too dependent upon the accidental, to admit of any general conclusion being drawn from it. "Ex paucis experientiæ manipulis ad axiomata generalia intempestive transcendunt," says Baglivi (*De Praxi Med.*, lib. i., cap. iv., § 2) of the physicians of his day; and these words may perhaps find their application in the subject before us, but of course completely separated from their other connexion.

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Dr Parker F.R.S.

with the Translator's

kind regards.

Some Cases of Aphasia, communicated by Dr. Œdmansson. Translated from the *Hygiea* for June, 1868, by William Daniel Moore, M.D., Dub. et Cantab.; M.R.I.A., L.K.Q.C.P.I.; Honorary Fellow of the Swedish Society of Physicians, of the Norwegian Medical Society, and of the Royal Medical Society of Copenhagen; Secretary for Sweden, Norway, and Denmark, to the Epidemiological Society of London.

The disease, or rather the morbid symptom, which has for some years borne the name Aphemia or Aphasia, was not unknown in ancient times. Trousseau quotes a passage from Pliny,[a] where the latter, after having given some examples of unusual memory, states that the memory may easily be affected by various causes, and quotes some instances in which persons have forgotten the names of their acquaintances, and even their own names, or the letters of the alphabet. Dax and, after him, many others, reproduce the following remark of Schenkius,[b] where the latter makes a certain Oethæus say: "Observatum a me est, plurimos post apoplexiam aut lethargum aut similes magnos capitis morbos, etiam non præsente linguæ paralysi, loqui non posse, quod, memoria facultate extincta, verba proferenda non succurrant."

Frederic Hoffmann[c] describes, under the name of aphonia, five cases which afford more or less distinct examples of aphasia. He defines aphonia to be an incapacity to speak or to produce articulate sounds, depending exclusively on a defect in the tongue, the cause of the latter being a lesion of innervation. That he does not mean an absolute loss of the power of articulation is shown by this, that one of his patients could the whole time say "mama." He distinguishes also certain forms of aphonia "ex vera linguæ paralysi," from others, but does not go farther into the matter. As the most usual form of aphonia he specifies that which attends apoplexy and hemiplegia, and which not unfrequently

[a] Historia mundi, Lib. sept., cap. xxiv.
[b] Obs. med. rar. Lugduni 1643, p. 180.
[c] Opera omnia. Tom. 3, pp. 249, et seq. Geneva, 1740.

may continue for some time afterwards. Among other causes he enumerates congestion of the head, the abuse of spirits, violent terror, cold, intestinal worms, all causes which, with the exception of the last, have been brought forward for aphasia.

The matter is, however, put much more clearly by van Swieten,[a] who expresses himself thus: Vidi plures, qui ab apoplexia curati omnibus functionibus cerebri recte valebant, nisi quod deesset hoc unicum, quod non possent vera rebus designandis vocabula invenire; manibus, pedibus, totius corporis nixu conabantur explicare miseri, quid vellent, nec poterant tamen. Malum illud per plures annos sæpe insanabile perstat. After adding, that subsequently to an apoplexy a loss of moral courage, a degree of effeminacy often occurs, he continues: Hinc videtur patere, singulares quasdam encephali functiones manere turbatas, vel et abolitas, tota vita, quia in solo principio primæ determinationis mentis in corpus quid mutatum fuit per apoplexiam prægressam. When van Swieten thus says, that some of the cerebral functions, and among them the power of speech, may be lost, while the rest remain undisturbed, he approaches the views of a later period, and is to be considered as a precursor in phrenology. The definition of aphasia, which may be deduced from the above statement, agrees most closely with that afterward given by Broca.

It was not until the entrance of phrenology upon the medical stage that the question of aphasia could assume the form in which it now appears. Gall referred the organ of the faculty of speech, which he calls "Memoire des mots," and which he would have separated from the "Faculté du Langage, Talent de la Philologie," to the antero-inferior part of the frontal lobes. Both he and Spurzheim[b] describe typical cases of aphasia, but had no opportunity of establishing by dissection the existence of any change in the central organ assumed by them. The phrenologists based their theory of the localization in the brain of the several intellectual and moral qualities principally on facts in comparative anatomy and cranioscopical observations, while they did not understand how sufficiently to estimate, or at least how to employ the control over the correctness of their views, which pathological anatomy could afford. Modern phrenology, if we may so call the recent attempts to divide the brain into many centres, arose properly precisely from the observation, that the loss of a higher faculty, the power of speech, coincided with lesion of a certain part within the brain, and recent investigations are directed to confirm or refute this observation. The question is still in its infancy, but once started it can never be left undecided. It is remarkable that the starting point of the more recent investigations is the same as that of Gall's. Even at school he began to study in his comrades their memory for words, compared with the form of their heads.

[a] Commentar. Tom. 3, p. 288.
[b] Observat. sur la Phrenologie. Paris, 1868, pp. 304, et seq.

In passing to the most recent period in the history of aphasia we meet with Bouillaud, who as a principle assumes unconditionally the plurality of cerebral organs, but says[a] that the only thing which his own investigations have taught him with respect to the localization of the intellectual organs of the brain, or the determination of the seat of these organs, is that the anterior cerebral lobes are the organs for the formation and memory of words, *i.e.*, the principal signs for the expression of our thoughts. To this conclusion he has been led by the observation, that loss of the power of speech and of the memory of words is always a result of a disorganization in the anterior cerebral lobes, and that this power may remain undisturbed in considerable changes in other parts of the brain. But loss of the power of speech may depend also on paralysis of the muscles which serve for the articulation of words, and in such cases he has also found an alteration in the anterior cerebral lobes. Now as he assumes the existence of distinct organs in the brain for a number of combined movements governed by the brain and the will—which he has inferred from the fact, that some of these movements may be found paralysed with a corresponding limited lesion in the brain—he asserts that the organ which governs the articulation in speech, and combines the muscular movements set in motion in speaking, has its seat in the anterior lobes in intimate connexion with the organ for the memory of words.

This centre he calls "organe co-ordinateur ou legislateur de la parole." The loss of the power of speech may, therefore, according to Bouillaud, depend either on loss of the memory of words or of the "legislative power." The place in the brain, which this double organ of the faculty of speech occupies, he did not, in his first essays on the subject, wish to indicate more precisely than in the anterior lobes, but in other places he says the anterior parts of these lobes.

Marc Dax[b] bases upon more than forty cases the assertion, that loss of memory of words occurs while intelligence is preserved and without paralysis of the tongue, and that the same is always combined with a lesion in the left hemisphere of the brain. To this latter conclusion he was led by observing that the hemiplegia so often occurring with loss of speech is always dextral. He explains the phenomenon itself with Lordat, by assuming an aberration of co-ordination between the muscles used in speaking. He likewise points out the interest the fact possesses in a medico-legal aspect, as patients so affected might easily be looked upon as insane. This is true principally of the form of aphasia, in which the patient can indeed speak, but uses words incorrectly and incoherently.

This paper at the time attracted no further attention, and quickly

[a] Traité de l'encéphalite. Paris, 1825, p. 284.

[b] Lesions de la moitié gauche de l'encephale coincidant avec l'oubli des signes de la pensée. Montpellier, 1836. Gazette Hebdom. 1865, No. 17.

lapsed into oblivion, from which a son of Dax, after the appearance of Broca's essay, brought it forward.

With the exception of some papers sent in by Bouillaud in 1839 and 1848 to the Medical Academy, in which he endeavoured with fresh cases to support his opinion, the question rested almost completely until 1861, when an animated discussion arose on the subject in the Société d'Anthropologie, in which Auburtin in particular defended Bouillaud's proposition, while Gratiolet contended for the unity of the brain.

Broca, who had previously questioned the theory[a] of the plurality of organs in the brain, went over in 1861, with a certain *eclat*, to the other camp, on account of two cases of aphasia which he witnessed immediately after one another. He goes still further than Bouillaud and Dax, asserting that aphasia is always connected with a lesion of the posterior third of the third (inferior, external) frontal convolution. In both cases the greatest change was found in the posterior part of the third frontal convolution, while in one almost the whole frontal lobe, the gyrus ascendens parietalis, the insula Reilii, &c., was more or less degenerated, in the other also the second (middle) gyrus frontalis was, although in a less degree, affected. Both were the typical cases where loss of the faculty of speech was met with without paralysis of the muscles of articulation, and at least for a long time without disturbance of intelligence. Broca considers this "symptom" to be so peculiar, that it deserves a special name, and he proposes for it the designation "aphemia." The patient both understands what is said to him, and can himself express his thoughts in writing; he has not lost the memory of words, nor the power of moving the muscles employed in phonation and articulation. To explain the nature of the aphemia he proposes two hypotheses. The one is that the patient has lost the memory of the combined movements necessary for the production of articulate sounds, and is therefore reduced to the condition of the child, who may have both the intelligence and the will, without the power of expressing its thought in words. The other hypothesis is to the effect that the aphemia is the result of an "ataxie locomotrice," limited to the part of the central nervous apparatus which governs the movements in the articulation of sound.

After Broca came forward the interest in the subject became extremely general in France. A very large number of cases were speedily published. The principal writer is Trousseau, who in his "Clinique Médicale" gives a full account of aphasia,[b] and brings forward many observations of his own. He assigns to aphasia a much more extensive signification than Broca did. While Broca defined it as a loss of the power of speech without disturbance of intelligence or of the motor apparatus, Trousseau says that such cases are indeed met with, but only exceptionally. As a

[a] Bulletins de la Société anatomique, August, 1861.

[b] Trousseau, for etymological reasons, changed the name aphemia to aphasia.

rule other modes of manifestation of thought are also wanting, especially the power of writing, and this usually in proportion to the defect in the faculty of speech, wherewith he assumes that the intelligence is in a greater or less degree diminished. We are not, he thinks, to allow ourselves to be misled by the patient's intelligent aspect, but we should always accurately investigate the condition of his mental powers. The principal cause of aphasia he places in a defective memory. He does not wish, however, completely to identify aphasia and amnesia, but entertains the view, that we must admit the existence of various kinds of memory, and that it is the memory of words and other means of expressing thought which is impaired, without the memory having in other respects necessarily suffered any damage. The cases of aphasia in which the mental powers are undisturbed, he explains, like Broca, by supposing that the memory of the combined movements in speech is lost. A patient so affected is, he says, in the same position as the deaf mute would be if he were suddenly to regain his hearing and endeavour to imitate the conversation around him.

G. Dax, in 1863, sent in to the French Academy of Medicine an essay,[a] in which he both referred to his father's work, and brought forward several cases in support of the view, that the faculty of speech has its seat in the left hemisphere, and according to his opinion probably in the middle lobe (its anterior,[b] external part). This paper gave rise to an animated discussion, in which the chief speakers were Trousseau, Bouillaud, Parchappe, and Baillarger. Trousseau repeated his formerly announced opinion, that all means of the expression of thought (speech, writing, gesture, singing, &c.) might in aphasia be more or less lost, and that in general a lesion of intelligence is present principally in the form of defective attention and dulness of memory. He brought forward statistics of known cases, by which he endeavoured to show, that both Bouillaud's and Broca's assumption of the localization of the faculty of speech in the anterior cerebral lobes wanted sufficient foundation, and he concluded by designating aphasia as a mere symptom, which almost always, if not constantly, depends on a lesion of various intellectual powers, while he thought that different parts of the brain co-operated in speech, even if the anterior lobes play the principal part in it. Bouillaud disputed the correctness of Trousseau's statistical calculations, as well as his entire theory of aphasia. He took up Broca's stand-point and recognized only such cases as aphemia or aphasia, where no lesions of intelligence or of the muscles of articulation were met with. With this form of aphasia, "Broca's aphemia," "aphemie exterieur," which according to B. depends on a change in the "organe legislateur de la parole," and the loss thence produced of the power of combining the muscular movements, he would yet always

[a] Gaz. Hebd. 1865, No. 17.

[b] Bulletin de l'Académié imp. de Med. 1865. Nos. 14–19.

have recognized his second form "aphemie interieur," which has its sole foundation in loss of memory.

Baillarger endeavours to define more accurately the several forms of aphasia and their causes. "Aphasia simple," where a more or less complete loss of the faculty of speech occurs, ought to be distinguished from the form in which the patient utters words without connexion with his thoughts. In it there is a perversion of the faculty of articulate language. Simple aphasia has two forms—1st, Where both the oral and written language have suffered; and 2nd, where only the faculty of speech is more or less lost. The former depends simply upon a loss of memory. Not so the latter. In this, he says, the memory cannot have suffered, as the power of expressing the patient's thoughts in writing is unchanged. Nor is there any lesion of a co-ordinating organ, as many can very well articulate certain, though often only a few words. Baillarger dwells on the well-known fact, that some of those affected with aphasia cannot find a word which they wish to express, while the same word often escapes them involuntarily a little later instead of another. He considers that this depends on an inability in the will to incite to the necessary muscular movements. "Incitation verbale volontaire" has been lost, while "incitation verbale involontaire" continues. As a proof of the existence of such involuntary incitation to speak—speech without voluntary determination—he adduces speech during sleep, as well as the fact, that men often speak to themselves without knowing it. The form of aphasia where one word is used for another without connexion, he would explain by a substitution of automatic, involuntary speech for that regulated by the will.

Jaccoud[a] revives the old name alalia, and by it designates every loss of the power of speech without reference to whether the cause of it is a defect of intelligence, a defect in the conducting apparatus from the brain, in the motor centre of speech (the medulla oblongata), or in the muscles of speech. By this jumbling together the question is thrown back into the obscurity from which Bouillaud, Broca, &c., endeavoured to rescue it.

Fleury has endeavoured to introduce special denominations according to the various causes of loss of the power of speech. To these belong aphthongia, or alalia, for the form which depends on diminished mobility of the tongue; aphrasia, where the cause is to be found in abolition of the faculty of thought; and aphasia, for the form which at present occupies us.[b]

[a] Gazette Hebdom. 1864. July and August.

[b] In the 40th volume of this Journal, p. 254, I have myself briefly recorded a case of aphasia in which the patient formed correct sentences, though not a word he uttered expressed the idea he meant to convey. In this instance the powers of co-ordination and of articulation were perfect; the intelligence also was, to all appearances, perfect,

In England the subject has latterly been followed up with interest. Hughlings Jackson (London Hospital Reports, 1864) gives an account of forty cases, in all of which, with one exception, the hemiplegia was dextral. The first case with *post mortem* examination, and confirming Broca's views, was brought forward by Sanders.[a] In a recent case by the same author the greatest change was found in the insula Reilii, which led him to express the opinion, that possibly the *insula Reilii* rather than the *gyrus frontalis tertius* is the point on whose integrity the power of speech depends. Sanders assumes, with Bouillaud, two forms of aphasia—amnemonic and ataxic; but believes, nevertheless, that in aphasia a certain degree of defect of memory is always met with. Fresh cases have been published by Fox, Jackson, Simpson, Ogle[b] (St. George's Hospital Reports, 1867), &c. Ogle describes no less than twenty-five cases, in all of which the left cerebral hemisphere was altered. He considers that cases of aphasia, depending on a change in the right hemisphere, may be explained by the assumption, that a corresponding point in the right hemisphere had been developed into an organ of speech in the same manner as we occasionally see the left hand developed to perform the functions of the right.

[It is to be regretted, that in the above bibliographical and historical sketch the author has altogether overlooked the many very valuable communications on the subject of his paper, which have from time to time appeared, in the former and present series of this Journal, from the pens of Dr. Osborne (so long since as November, 1833), Dr. Graves, Dr. Banks (May, 1865), and Dr. Popham (August, 1867).—TRANSLATOR.]

From the foregoing short account of the historical development of the subject of aphasia, it is easily seen that opinions are divided respecting almost everything belonging to it. It is remarkable that the idea of the brain as a complex of many centres, distinct from each other, has been disputed by only a few among the many who have recently stated their views respecting it, though it must at the same time be admitted that many have avoided giving any definite judgment in the matter, and that the degree of independence assigned to these centres has varied. All who admit the possibility of the existence of a distinct organ for the faculty of speech, or that the loss of the latter coincides with lesion of a limited point in the brain, must, however, be regarded as also recognizing the truth of phrenology in other respects, though not precisely Gall's phrenology, with its external craniological signs of the cerebral organs;

except that the patient had forgotten the proper connexion between his ideas and the words he ought to have used to express them. Such a case would be more adequately represented by the term *heterophasia* than by *aphasia*.—W. D. M.

[a] Edinburgh Medical Journal, 1866. February.

[b] Unfortunately, I have not seen the original, but only a short notice in the American Medical Journal for January, 1868.

but a phrenology based upon the theory of the various physiological action of the different parts of the brain, especially of the cerebral convolutions.

Among those who do not absolutely deny an organic plurality in the brain is Trousseau, who, however, thinks that the investigations hitherto made do not justify such an assumption. He argues, nevertheless, though not successfully, in favour of the possibility of a different action in the two hemispheres of the cerebrum by endeavouring to bring forward proofs of pathological and anatomical differences between the two sides of the body. Thus he would maintain that intercostal neuralgia, as well as hysterical hemiplegia, scarcely ever occurs except on the left side, and asserts, with Bouillaud, that the rheumatic affection of the heart is met with only in the left side of that organ. Further, he directs attention to the different mode of origin of the two common carotids.

Again, of the authors who demand a distinct organ for the faculty of speech, this is referred by the first to one, by another to a different part of the brain. Thus Gall and Bouillaud would place it in the anterior part of the frontal lobes; Dax, senior, in the left hemisphere; Dax, junior, in the middle left lobe; Broca, in the posterior part of the third frontal convolution; Sanders, in the left *insula Reilii*. Although opinions are divided, most writers, as we have seen, and all of those who have of late accurately studied the subject, agree in looking on the left hemisphere as the seat of that organ. Many cases collected of late years, both in France and England, are in favour of the point first indicated by Broca, but against this are other perfectly reliable observations, and among them one by Charcot, where Broca himself made the dissection, and found the third frontal convolution sound. In this, as in other instances, the change was met with in the insula, which, on the other hand, is often not mentioned in *post mortem* reports as diseased.

A support for the correctness of the assumption, that the organ for speech, if such exists, is placed in the left hemisphere, lies in the fact, that the hemiplegia, which often occurs in aphasia, is almost always dextral. There are, however, authentic cases where the hemiplegia was left-sided. An attempt has been made to explain this by showing that, simultaneously with the lesion in the third frontal convolution, which produced the aphasia, another is found in the right hemisphere, giving rise to the sinistral hemiplegia. This explanation passes as satisfactory so long as in such cases no *post mortem* examination has been made, and its incorrectness proved. Ogle supposes that in these cases the aphasia depends on a change in the pons Varolii.

The physiological explanations given of the nature of aphasia also vary greatly. Gall considers that the loss of the faculty of speech

depends on a loss of the memory of words. Bouillaud joins him for one form of aphasia, while he makes his second form depend on abolition or diminution of activity in his "organe legislateur de la parole"—*i.e.*, on destruction, through lesion of this organ, of the combination of the muscular movements necessary for speech. It is this latter form which the question at present bears upon. No one denies that a defect in the memory produces a corresponding limitation of the power of expression; but this defect, of a purely intellectual nature, ought not to be designated by the name aphasia, for this might easily give rise to confusion capable of injuriously affecting the development of the question. One reason, however, for giving the name aphasia also to this loss of the faculty of speech is, that we, unfortunately, cannot always distinguish this form from the other; but of this more hereafter. Lordat and Dax, senior, entertain an opinion similar to that of Bouillaud, as they consider that aphasia depends upon defective co-operation of the muscles of articulation, though at least Lordat does not think of any distinct centre in the brain as regulating and co-ordinating their movements. Broca looks upon this opinion of Bouillaud as possibly correct, but believes rather that the aphasia depends on loss of a special memory assumed by him—viz., the memory of the combined movements which are in operation in speaking. Nevertheless, the existence of such a memory is at least doubtful. Of those who hold the unity of the brain, Trousseau considers aphasia in general as an intellectual disturbance, but admits the existence of cases where the intelligence is undisturbed: for these he adopts Broca's last-mentioned hypothesis. Baillarger refers the cause of the affection to a loss of the power of the will to determine to speech, a loss of "incitation verbale volontaire."

Among these different opinions, it would appear that Bouillaud's view—that aphasia depends on a disturbance of combination of the muscular movements, which is about the same as Lordat's ("asynergie verbale"), and which Broca looks upon as in the end correct—reckons the most adherents, though all of these do not accept the cause of this disturbance assumed by Bouillaud—lesion of his "organe co-ordinateur." If this opinion be correct, Broca will have aphasia looked upon as an "ataxie locomotrice," which view has obtained acceptance in England, where Sanders and Ogle designate this form of aphasia as aphasia atactica; that, on the other hand, where the memory has suffered, aphasia amnemonica. It is remarked, in opposition to Bouillaud, that the co-ordination of the movements of the muscles of speech cannot be said to be removed, as many aphasics can articulate one or more words very well. The objection may, however, here be made, that in ataxie locomotrice many degrees are met with, and that even in the graver forms certain muscular groups can co-operate and combined movements take place, though not always, when the patient wishes, and often only for a moment. Broca's assumption, that aphasia depends upon a loss of memory of the movements of speech, is rejected by Baillarger, who believes that there is nothing to justify the assumption of this special memory. "Children endeavour to imitate and reproduce the sounds they hear spoken." "They try to exercise their muscles in combined movements rather than to acquire the memory of these movements." "The will can produce speech, but does not guide the several movements, which seem to be rather of a reflex nature."

As to the clinical signification to be attached to the name aphasia, writers have not been able to agree. Broca and Trousseau, the two principal authors, entertain, as has already been shown, far different views. Broca and his adherents will recognize as aphemia or aphasia only those cases where, with existing loss or limitation of the faculty of speech, no change is discoverable in the intelligence and motor apparatus. Trousseau indeed admits the exceptional existence of such cases, but will have aphasia, as a rule, looked upon as an intellectual disturbance, usually combined with other defects of the same nature, and often with other signs of cerebral lesion, as hemiplegia, contractions, eclampsia, &c. Both these views seem to me to be one-sided and unsatisfactory.

If we investigate a large number of cases we cannot avoid seeing the difficulty, nay, often the impossibility, of distinguishing cases of aphasia in a limited sense from those where actually a slight defect of intelligence—for example, a certain degree of dulness of memory—exists. When it is necessary to resort to the experiment of making the patient write, in order to study the degree of his intelligence, we need only remember how many, particularly old men of the lower classes, there are who are not able, or are only very imperfectly able, to write, and who easily forget what they could do in that way; and moreover, that the hemiplegia so often present frustrates the attempt; to see that this experiment is often made in vain. It must, therefore, no doubt, frequently happen that we cannot with certainty decide whether the case ought to be referred to pure aphasia or not. This will be still more true if we adopt Ogle's opinion, that the loss of the power of writing (agraphia) ought, as well as aphasia, to be distinguished into a. atactica and a. amnemonica.

If we adhere to the strict definition it may easily happen that we may in the same case at different times have an aphasia à la Trousseau and an aphemia à la Broca, nay, even an alalia à la Jaccoud. Suppose, for example, an apoplectic, who has regained intelligence but is still dull, has paralysis of the tongue and cannot speak, we have a case of alalia. After some time the tongue and the other muscles engaged in speech have entirely, or for the most part, regained the power of movement, but some dulness of memory, with inability to speak, continues; we have

now before us one of Trousseau's aphasics. The dulness gradually diminishes, the memory returns, but the speech is persistently imperfect. The case is now reduced to an aphasia in a limited sense—a Broca's aphemia. It seems incorrect to assert that aphasia exists only in the last case. It existed also in the second, though not alone, but combined with other symptoms of an originally more extensive cerebral lesion. In the first case we cannot, on the contrary, speak of an aphasia, for to it is necessary, on the one hand, sufficient intelligence to be able to think, and, on the other, sufficient mobility in the muscles of speech for the articulation of sound.

In my opinion we ought to be entitled to diagnose aphasia whenever the faculty of speech is more or less lost, and when this loss cannot be explained by dulness existing in the thinking power, or paralysis in the vocal muscles. We endeavour to recognize it, where it is met with, whether it be that the defective power of speech is the only symptom of a pathological change in the brain, or that it exists together with other symptoms of cerebral disease; in the latter case the same organic change may be the cause of the aphasia as in the former instance, although the lesion has also attacked other parts of the brain.

Aphasia is for us, however, only a symptom like certain neuralgias and paralyses of central origin, which, in one instance, at least for some time, may be the only sign of the central affection, in another, may be a link in a wider group of symptoms. This symptom deserves, however, when it stands alone, more than indeed to give a name to the disease, especially when we consider that the pathological process in the central organ may be of a different nature, as softening with or without a change in the vessels, hemorrhages from various causes, the pressure of tumours, encephalitis, &c., &c., and that the differential diagnosis may often be impossible.

With respect to Trousseau, it may be remarked that he endeavours to throw into the shade and to reason away the cases of aphasia, where loss of the thinking power stands entirely alone, although he must admit that such cases are met with. His view of both the physiological and clinical signification of aphasia he bases only on complicated cases, where, with loss of the faculty of speech, some intellectual defect or other is met with.

For the study of the physiology of the brain the pure cases of aphasia are incomparably the most important, but even the complicated cases are capable of affording information, as on *post mortem* examination we may find the same explanation of the symptom as in the simple cases. They teach us, moreover, to study this symptom in its relation to other symptoms of cerebral disease, and in general to investigate more accurately than hitherto the several symptoms.

I.—*Case of Epilepsy, with transient Aphasia after the attacks.*—Johansson,

a guard, was admitted into the Garrison Hospital in Stockholm, on the 16th December, 1863.

He had shortly before got a violent blow on the head, which deprived him of consciousness. In the journal from this time it is noted that he had a slight wound on the back part of the vertex, without any exposure of the bone or depression of the skull. He was quite confused, could not answer questions, nor even articulate any sound. No paralysis could be discovered, but the patient, on the contrary, moved with much violence, especially when his wound was dressed, whereupon he uttered a moaning cry of pain. Ice to the head. Enema aperiens. On the 18th he began to endeavour to answer, but his speech was still extremely confused and unintelligible. On the 20th he could enunciate short sentences clearly but slowly; in general he still lay in a lethargic condition. Blister to the nape of the neck. In the beginning of January it was observed that he had lost the power of reading. If a book were placed in his hand he could distinctly read a few words, but he then began to skip over some, and to introduce others quite foreign to the purpose, so that the whole was entirely unintelligible. His speech was at the same time imperfect, when he had to utter long sentences. These defects were gradually removed. Before the 28th February it was found that he was quite coherent, but rather sensitive and childish in his mind. On the 29th January he had an epileptiform attack, which was repeated a couple of times during the spring. Later in the season he was dismissed. During the first part of his stay in the hospital he was under the care of Dr. Edholm, subsequently of Dr. Lundberger.

After his return home he was for a short time free from attacks, but they soon came on again, and became gradually more frequent. His spirits were bad. In the beginning of 1865 he gave up the service. On the 9th of April he was re-admitted into hospital, having had ten or twelve fits during the preceding twenty-four hours. On his admission he was ordered a bladder of ice to the head and a turpentine enema. On the 10th the note of his case was—"Seven epileptiform fits since yesterday afternoon; a small cicatrix on the posterior part of the vertex, at the right of, and close to the mesial line." The patient lies in an apathetic and stupid state. He answers slowly "Yes" and "No," but on attempting to utter a long sentence, what he says is, with the exception of a word or two, quite unintelligible. On asking him if he feels any pain, he points to the left part of the forehead. The pupils are normal and movable. The pulse is about 100, of moderate fulness and strength. No fever. Bowels moved after the enema. Urine passed in the bed. Nothing else remarkable. Glauber's salt in cooling doses. On the 12th blister to the nape of the neck. His state continued the same until the 18th. He had four or five fits in the twenty-four hours, was in general dull and apathetic, but sometimes violent. In his rational moments he

complained of pain in the left side of the forehead. His speech continued scarcely intelligible, yet usually more so than on his admission. On the 18th the attacks began to occur only at night. He was now more wakeful, and spoke better, although thickly. On the 28th he had been for nine days free from attacks; was quite rational; spoke in general distinctly, though slowly—sometimes skipped over a word or uttered it indistinctly. During the night before the 29th he had at least 20 fits; in the morning was quite dull and stupid, and did not answer. In the afternoon of the 30th he began to regain consciousness. The power of speech was now at first completely removed, but gradually returned. His state continued in this manner alternately improving and getting worse, the power of speech on each occasion, when numerous attacks occurred in a short time, being completely lost, but gradually returning. The blister to the nape of the neck was repeated. The bowels were kept moderately open by means of Glauber's salt. For some time iodide of potassium was employed, though without effect. On the 28th of May I noted—"During the night numerous slight attacks occurred three or four times in the hour, characterized merely by twitchings in the head, distortion of the face, and fixing of the eyes. They lasted only a minute or two. The patient endeavoured to speak immediately after they were over. He himself says he retained consciousness during the attack. To-day at the morning visit he seemed lively and wakeful. He can only imperfectly articulate sounds. At one time a word passes his lips, at another he cannot, notwithstanding all efforts, utter the same word. A word or two he can often utter distinctly, but when he continues to speak he becomes entirely unintelligible. He is conscious of his incapacity. After he has in vain endeavoured to express his thoughts orally he shakes his head discontentedly and sadly, and endeavours to say "I cannot speak," in which he sometimes succeeds, although stammeringly and with difficulty. Sometimes he can utter only the first word. When asked if he means this or that, he makes an affirmative or a negative sign with his head. Objects shown to him he endeavours, though usually in vain, to name, and he knows their use well. He understands what he reads, and endeavours some time afterwards, when requested, to utter the words he has read. During all this he never employs words incorrectly. His writing is as bad as his speaking. He attempts to form letters, but soon throws away the pen. He can with ease protrude his tongue straight forward, but its pointing and other movements are rather slow and uncertain. There is no other trace of paralysis.

The attacks continued all day; in the afternoon the patient was particularly dull; the power of speech was entirely lost. The right arm was semiflexed, the right leg was extended; both were motionless, and maintained the position given to them. The left extremities moved easily. Even the following day the paresis had passed away. On the 30th, as

the attacks continued and were numerous, six leeches were ordered to the left temporal region.

31st.—Only one fit yesterday; several during the night. June 5th.—No attack for five days. The patient is quite coherent. The power of speech is restored, except that there is a slight hesitation. 9th.—One or two slight attacks during the last twenty-four hours. 10th.—Incessant attacks during the night, of various strength, though they were generally slight; these still continue to-day, and recur once in about every five minutes. His speech is extremely indistinct. Most words he cannot utter, though his gestures and bearing clearly indicate that he knows what he wishes to say. What he means is often plain from one or two syllables in conjunction with a gesture. Six leeches to the left temple, the bleeding to be encouraged as much as possible. Same day in the afternoon: Constant numerous slight attacks. Power of speech quite lost. All attempts fail. When he attempts to speak, movements of mastication and deglutition come on instead. The tongue can be protruded and moved quite easily and powerfully from side to side.

The attacks continued during the following days, and the patient became duller and duller. On the 12th the evacuations were involuntary. On the same day it was observed that the right naso-labial fold was obliterated, and that the right angle of the mouth was rather pendulous, but the tongue was movable. On the afternoon of the 15th six cupping glasses with scarification were ordered to the nape of the neck. From this time he had in general only one attack in the hour until four o'clock the following morning, after which the fits entirely ceased, not to return, so long as the patient remained in the hospital—more than six months.

The faculty of speech again improved quickly, and in the course of a fortnight was quite restored. The pronunciation of the consonants h, k, s, and x, was the last to be regained. Even afterwards a slight hesitation was sometimes observed. The patient's long delay in the hospital was caused by the fact, that during convalescence after the convulsive attacks atrophy invaded the muscles of the left forearm and hand, preceded and accompanied by violent neuralgia in these parts. The patient also occasionally complained of pain in the forehead.

He was dismissed on the 31st December, 1865, but came into hospital again on the 16th of February, 1866, in a state of unconsciousness. He had the same day had several violent epileptic attacks after taking some glasses of brandy. They were not repeated in the hospital, and he soon recovered. He was dismissed on the 9th of March. Later in the year he got his discharge and disappeared from Stockholm.

This case presents the not unusual example of aphasia occurring during epilepsy. It is less common to see aphasia come and go so often in a comparatively short time. On every occasion that several attacks

occurred soon after one another, the power of speech suffered in a greater or less degree, and was gradually completely lost. When the attacks ceased or became less frequent, the power of expression soon returned At the same time both intellectual disturbances, and occasionally also paralytic phenomena, set in and disappeared, but the aphasia always preceded them, and was the last to cease. Usually there was a diminished power of moving the tongue, but this certainly depended much on the contemporaneous dulness, and was moreover quite incapable of explaining the defect in the faculty of speech. We often found, especially during a temporary improvement, the movements of the tongue fully defined, while the power of speech had as yet scarcely begun to return. The tongue when protruded always deviated to one side. The other paralytic symptoms—at one time paresis of the right extremities, at another of the face—were dextral. In the attempts I made to get him to write he failed, but I unfortunately forgot to investigate his capability in the art of writing, after he had got better; probably it was even then inconsiderable. He was constantly, and even after the attack ceased, rather sensitive and easily excited, but his loss of the power of speech could not, of course, excepting at the period of the height of the exacerbation, be placed to the account of any intellectual defect. He knew very well what he wished to say, and made great efforts to bring out the words. When he failed he appeared unhappy and perplexed.

As to the etiology and more accurate diagnosis of the case, it is probable that originally the blow on the right side of the vertex produced, by *contre-coup*, a contusion with laceration of some point in the left frontal lobe. During the obliteration of this change, probably by cicatrization and atrophy of the part of the brain, the epilepsy set in. Besides the place which the blow struck, the obstinate pain in the left part of the forehead would show, that the mischief was located in the left frontal lobe; in favour of the left side were the dextral paralytic symptoms. Now, as since the first appearance of Broca's statement, not a single case has been brought forward, where a lesion of the posterior part of the third frontal convolution has occurred without a corresponding and persistent loss of the faculty of speech, and in our case the aphasia was only temporary, came and went, we may rest satisfied that Broca's region was not, in this instance, directly injured. It is possible, on the other hand, that the change was to be found somewhere in the neighbourhood, and that during the repeated epileptic attacks the disturbance of the cerebral circulation affected, in the first place, the immediate surrounding of the previously altered part.

In a therapeutical point of view, the great effect which cupping the nape of the neck had upon the attacks deserves to be noted. After having lasted for many months, and having latterly been often so numerous, that the one attack followed directly on the other, they

entirely ceased immediately after the cupping. The minor abstractions of blood from the left temple I prescribed on the supposition that cerebral lesion existed beneath the part.

2.—*Case of Miliary Tuberculosis; Tubercles in the Brain; Aphasia with Hæmorrhage in the Island of Reil.*—Carlsson, aged twenty-one, admitted into the garrison hospital on the 19th September, 1865.

The patient had sickened a week previously with general indisposition, want of appetite, some thirst, disturbed sleep, to which, after a few days, supervened headache. The bowels were torpid during the whole time, with the exception of a couple of days, when aperient medicine was taken. *Status præsens* Sept. 20.—The patient is pale and languid, complains of the above symptoms, which, nevertheless, he believes to be slighter to-day than they were yesterday. The temperature of the skin is little, if at all, elevated. Pulse 72, rather weak. The tongue almost clean; to-day some little appetite. Abdomen sluggish; no motion during the last two days. Spleen not enlarged. The urine contains a small quantity of albumen. Nothing else noteworthy. Phosphoric acid.

Sept 25th.—The above slight symptoms have constantly increased. The patient has generally lain silent and reserved, but when asked how he is he says he is rather better than before. Yesterday evening he began to speak indistinctly, without anything unusual having been previously remarked, and without his state having otherwise indicated the slightest confusion of mind. This continues to-day. He is wakeful and apparently clear in his mind, but silent. His countenance is pale. Pulse 58. Temperature in the axilla, 100°·04 F. When questioned he endeavours to answer with suitable words, but his speech is particularly indistinct, especially certain consonants—k, s, h. Usually he can utter, so as to be tolerably well understood, the first word, or a couple of words, of a sentence, but the rest becomes merely a mumble, impossible to understand. The tongue, rather dry and furred, he protrudes when desired, and moves it with ease in all directions. The two sides of his face are quite symmetrical. The pupils are normal, and quite movable. He shows that he suffers from pain in the forehead and weakness.

26th.—During the course of yesterday he became more drowsy and apathetic. The power of speech diminished more and more, and from the afternoon he ceased to answer. He usually lay upon his back, but sometimes he bent himself up, sometimes stretched himself out in his entire length. Yesterday afternoon he got an aperient enema, which produced a loose motion. To-day he lies in a semi-comatose condition, from which he cannot be completely roused. It is only after several vain efforts that he can be got to stammer out a yes or a no to a question. His appearance is dull; his mouth and his eyes half open; his eyes

at one time quiescent, at another they move slowly from side to side. His otngue is dry. No vomiting. Abdomen hard, moderately swollen. Over the postero-inferior parts of both lungs numerous finely vesicular loose râles are heard. The spleen is not enlarged. The urine is free from albumen. Blister to the nape of the neck.

30th.—The patient's state has continued about the same. No lesions of motility. Speech very imperfect, but he now generally tries to answer. When he is not understood he becomes annoyed and turns away. No vomiting. No motion since the 27th, when he got Glauber's salt, which he afterwards took in doses of a teaspoonful twice a day.

Oct. 2nd.—Increased stupor. Now and then he makes an attempt to answer questions, but succeeds only very imperfectly. Passes urine involuntarily.

3rd.—In the course of yesterday the temperature began to be somewhat higher. The face, previously pale, occasionally flushed. The pulse was increased in frequency. To-day he seems rather more wakeful than before; shakes his head when asked if he slept during the night. When desired he slowly protrudes his tongue; its tip is directed somewhat to the right, but it can, however, with tolerable ease, be brought into another direction. His face is slightly flushed. The conjunctivæ are rather injected. The right pupil is considerably larger than the left, and is not perfectly round; both are slightly, though somewhat movable. After an enema yesterday he had a slight motion; a second followed involuntarily in the night. Yesterday evening a bladder of ice was ordered to the head, which is still continued.

5th.—The patient's state during the last two days is as before. The pupils are as large, tolerably wide, little movable; the right continues to be not entirely round. The right upper eyelid hangs lower than the left, but can be completely raised. When the patient is left to himself, a slight strabismus divergens sometimes comes on. The respiration is so far altered that a number of slow inspirations are followed by a number of more rapid ones. The gum has a tendency to bleed. The tongue is dry. The abdomen is more sunk in.

The patient died the same day at half-past 8 p.m. Temperature and pulse since the 25th:—

		Morning.	Pulse.	Afternoon.	Pulse.
Sept.	25	100°·04 F.	58	101°·56	60
	26	101°·84	50	100°·04	54
	27	98°·96	52	99°·68	54
	28	99°·32	48	102°·92	52
	29	99°·68	52	99°·68	50
	30	101°·48	58	102°·2	56
Oct.	1	100°·4	60	101°·56	62
	2	98°·6	90	101°·84	120

		Morning.	Pulse.	Afternoon.	Pulse.
Oct.	3	101°·12	84	102°·56	120
	4	101°·84	125	102°·2	120
	5	100°·4	120	100°·04	160

Post mortem examination on the 7th October, thirty-eight hours after death:—

Considerable emaciation. General rigidity. Skull of the usual shape. Dura mater congested. On the inside of its left half are found a large number of partly distinct, partly agglomerated, shining, transparent, grey granulations, raised above the surface, as large as the heads of pins. Most of these are situated in the middle of the lateral portion of the left hemisphere; the others are grouped around this point. On the right half of the membrane only two such granulations are met with; they are located in small depressions in the cortical substance of the brain. Corresponding to the part of the left half of the dura, where the granulations are most dense, a number of similar tubercles are found in the soft membranes, which follow chiefly the course of the vessels, and are here and there conglomerated into large irregular masses. Beyond this surface only a few granulations are met with over the convex portion of the left hemisphere. Around the chiasma, and as far as the pons, the soft membranes are infiltrated with a yellowish green turbid fluid. The surfaces of both optic nerves exhibit a tolerably intense redness, the right oculo-motor nerve a general rose colour both on its surface and—although in a somewhat less degree—in its interior; the left abducent nerve is for the length of an inch dark red on the surface. Scattered miliary tubercles are found in the soft membranes on the under surface of the frontal lobes. In the commencement of the right fossa of Sylvius they are met with, though only in small number. In the left fossa, on the contrary, the membranes are sprinkled and thickened with numerous granulations. In the upper part of the fossa of Sylvius a coagulum of blood larger than a nut is found. Corresponding to this part the cortical substance on the posterior portion of the island of Reil, with its immediately surrounding, is softened and discoloured, with a number of point-like hemorrhages. The soft membranes of the brain are in general rather congested. They are easily separated from the surface of the organ. When they are removed a not inconsiderable number of tubercles, of the size of hempseed or less, appear proceeding from the intergyral portion of the pia lodged in small depressions in the cerebral substance. Similar tubercles, even somewhat larger, are found scattered, to the number of from twelve to fifteen, in the cortical substance, mostly in the convex part of the left hemisphere. The majority are situated in the innermost part of the cortical substance, or on the boundary between this and the white substance, but a couple are met with on the outer surface, some within the white substance but quite close to the grey.

Both lateral ventricles are rather dilated; their contents are almost clear, and are of a light red colour. The surrounding parts are in general loose and pale. The white substance is otherwise pale, but, as well as the grey, is of ordinary consistence; the grey is rather congested. The central cerebral ganglia are free from any remarkable change.

On the upper surface of the cerebellum are seen three cheesy, yellowish tumours, as large as peas, of brittle consistence, occupying the grey substance in its entire thickness, and passing into the white. The medulla oblongata exhibits a considerable injection in the corpora restiformia.

The more accurate description of the other organs I shall here omit. Numerous miliary tubercles were found in the lungs, liver, spleen, and kidneys, some few in the serous investment of the heart and on the peritoneum. In the liver a number of cavities, varying in size from that of a hempseed to that of a pea, were met with, filled with a thick yellow fluid, and surrounded by a more or less thick membranous boundary. The solitary glands in the large intestine, as well as, though to a less degree, in the lower part of the small intestine, were considerably swollen, and were here and there ulcerated; in the large intestine were some ulcers as large as Swedish farthings (öre). In their fundus no tubercles were met with.

It is possible that some, on reading the above case, may be inclined to question its title to be brought forward as an example of aphasia. It is, however, to be borne in mind, that during the first twenty-four hours, when the power of speech was affected, the patient's inability to express himself was the only prominent symptom, while no paralysis was met with in the tongue, and there was evidently sufficient intelligence to form ideas and to wish to express them; we are, therefore, I think, quite justified in singling out the defect in the faculty of speech, and of speaking of the case, during the above-mentioned time, as aphasia. During the further development of the case, also, we occasionally found the power of speech altered to a higher degree than was proportionate to the intellectual decay. The patient endeavoured to form words, and from a syllable or two we could perceive that the word was clear to himself, that speech took place within him, while the articulation of the sound, the outer speech, was extremely limited. On *post mortem* examination I first sought the cause in the third frontal convolution, but found it instead in the posterior part of the island of Reil and its immediate neighbourhood (the very bottom of the fissure of Sylvius), that is, in about the place which Dax, junior, and Sanders consider to be most closely connected with the faculty of speech.

3.—*Case of Dextral Hemiplegia and Aphasia.*—This case has been communicated to me by Dr. v. Sydow, of Gefle, who has already, on several occasions, made valuable contributions to the Journal.

Dr. S. was called on the 23rd July, 1866, to the widow of a seaman, aged sixty-two, who the day before, while out walking, on stooping down to tie one of her shoes, suddenly lost the power of speech, became distorted in the face, and began to drag the right leg, so that she was obliged to be supported home. Dr. S. found the right extremities paretic, the mouth drawn obliquely to the left. Her speech was thick, her answers incoherent. Even on the 25th of the same month the distortion of the face was much diminished and the power of movement in the right arm increased, but the patient seemed not to understand rightly what was said to her. In consequence of pressure of business during the then prevailing epidemic of cholera, Dr. S. did not see her until the 27th August, when he observed that her speech was limited to unconnected syllables, mixed with single short words without meaning. On the 25th September the power of motion was considerably increased in the extremities, the obliquity of the face had disappeared, but the tongue still deviated, when protruded, somewhat to the right. Her speech was also very imperfect. By gestures and her general manner, however, she showed unequivocally that she understood what those about her said to her, and she could, in ordinary cases, make herself understood. She listened with an attentive aspect when she was spoken to, and endeavoured to answer, but her answer was only a word or two or a short sentence, which she pronounced clearly and plainly, often repeatedly, after which she ended with the well-modulated enunciation of certain syllables. Her efforts, at the same time, resembled not the endeavour of a stammerer to get the vocal organ to express a definite word, but rather the delight of a child in speaking long series of unconnected syllables and composing words of them, which signify nothing. She had never been able to write. That she to the last retained at least a certain degree of mental powers is shown by the fact, that she then had the Bible read to her, and that before her death she received the sacrament of the Lord's Supper.

On her cerebral affection an extensive bronchial catarrh supervened towards the end of September. She died on one of the last days of the same month.

Dr. S. had not found any affection of the heart.

The brain was sent to me for examination. The preparation had suffered some injury from the long journey, but the following could, without difficulty, be established:—While the upper parts of the brain have their normal arching, the posterior part of the third left frontal convolution, the inferior part of the ascending frontal convolution, and the anterior part of the superior temporal convolution, are collapsed, loose, and easily shaken. The same is the case with the whole of the fissure of Sylvius met with between these two points, including the entire of the island of Reil. The soft membranes can with care be

separated from these parts. The outer portion of the cortical substance has a certain consistence, can be cut, has a yellowish colour, while the inner part, as well as the adjoining white substance, is often softened to the depth of half an inch into an unctuous mass, presenting a white ground colour studded with red points and streaks. Nowhere are there traces of any hemorrhage. The vessels in the left fissure of Sylvius are everywhere permeable. In some places at the divisions a slight thickening is seen, as in some of the other larger vessels at the base of the brain, but the inner surface of the vessel is even and smooth, without any deposition. The other parts of the brain exhibited no remarkable change.

In the outer part of the cortical substance, on the altered part, a large number of fat corpuscles were met with; also small one-nucleated cellular bodies, with or without fatty degeneration of their contents; in the walls of the vessels were often fat globules in variable quantity; in the foci of softening were also found free fat, detritus, and myelin.

The cause of the symptoms in this case is, therefore, found in a considerable change in the left cerebral hemisphere. This occupied both Broca's region and the island of Reil, and the adjoining parts throughout no inconsiderable extent. In the cases of aphasia which have been submitted to dissection, more or less extensive softening has often been found, with or without emboli, in the afferent vessels; more rarely hemorrhages or other changes. Not unfrequently a cardiac lesion has proved to be the cause of the embolism.

It is not easy to decide how the above case commenced. The paralytic attack points to embolism or hemorrhage; but, according to Dr. v. S., no cardiac lesion was met with, and on dissection the vessels were found permeable, without deposition or any remains of hemorrhage. It is possible that in the commencement a capillary hemorrhage occurred in a limited point, and that subsequently softening took place in the surrounding part.

Nor in this instance is the question of simple aphasia, but both a disturbance of intelligence in the form of general dulness, and diminished mobility of the tongue were observed, but neither was, as appears from the history of the case, so important as to be capable of explaining the greatly-diminished power of articulating sounds in speech.

REPORT

OF THE

CITY OF GLASGOW

FEVER HOSPITAL,

From 1st May, 1867, to 30th April, 1868.

BY

DR. JAS. B. RUSSELL,

PHYSICIAN AND MEDICAL SUPERINTENDENT.

PRESENTED TO THE FEVER HOSPITAL COMMITTEE OF THE BOARD OF POLICE, 3RD JULY, 1868, AND ORDERED TO BE PRINTED.

GLASGOW:
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1868.

Committee on Fever Hospital.

THE LORD PROVOST.	DAVID CARSON.
BAILIE GILKISON. *	JAMES MARTIN.
" WILLIAM MILLER.	PETER DALLAS.
MATTHEW DICK.	JOHN URE.
JAMES COUPER.	WILLIAM BROWN.

THE LORD PROVOST, *Convener.*

BAILIE GILKISON, *Sub-Convener.*

Three a Quorum.

Meets every alternate Thursday, at 2.30 P.M.

Physician-Superintendent.

JAMES B. RUSSELL, B.A., M.D., 278 BATH CRESCENT.

Resident Medical Officer.*

GAVIN P. TENNENT, M.B., C.M.

Matron.

MISS JANE GIBSON.

Clerk and Storekeeper.

JOHN MUNRO.

* This arrangement only came into operation on 1st July, 1868.

CONTENTS.

REPORT.

It seems unnecessary to report upon this, the third year of the existence of the City of Glasgow Fever Hospital, at the same length, and with equal minuteness of detail, as upon the two preceding years. In accordance with the recommendation of Dr. Gairdner, Medical Officer of Health for the City, as expressed in a letter prefixed to my last Report, a Special Committee of the Board of Police was appointed in October, 1867, to overlook the affairs of the Hospital, and guide me in its management. Since then I have had the advantage of periodical inspections and reports by members of this Committee, so that the business of the Hospital has for great part of the year been made public in the minutes of the Board of Police; and the sanction thus obtained from time to time removes my chief motive for a review of the year so minute as I have hitherto striven to present.

The preceding year was remarkable for enforced idleness, and yet enforced expenditure, so that the most apparent feature was the disproportion between the work done and the money expended. But in that respect it was anomalous, and not to be referred to except to guide future action in similar circumstances. This year, as was anticipated in my estimates of expenditure submitted to the Committee in October, I hope to show that nearly double the amount of work has been done, while the outlay has increased by less than a twentieth. The Hospital was never so full as in January and February, 1868. Looking to the chief criterion of success, that which ought amply to justify any reasonable expenditure, the results of the treatment of the patients, this Hospital excelled itself in lowness of mortality at all periods of life as compared with

the two previous years, and contrasts more advantageously than ever with the practice of the Royal Infirmary, as given in the Annual Report for 1867, p. 20. While I wish to remain satisfied with the bare statement of this pleasing fact, I must say, for the sake of coming years, that, especially to non-medical judges, a mortality so low as 9 per cent. over all cases (that of the Infirmary being 16·9 per cent.) is not a rate which we can hope to maintain. It depends more on the nature of the cases than on the treatment to which they were subjected, or the general hygienic condition of the Hospital. Especially must I beg that no one will take 9 per cent. and 16·9 per cent. as representing fairly the ratio of mortality in the two institutions; but will, to determine this in a just way, resort to comparison at similar periods of age, as is done in Table No. IV. Still, after all deductions of this sort, seeing that pauper patients are now admitted, that we have had, as we shall show, many cases sent in moribund to swell the mortality, and yet that at every age there is a decided decrease in the death-rate, we may venture humbly to credit the Hospital with some share of the merit of lives saved. Further, I would venture to attribute a large share to the excellence of the nursing during the past year. The following is Miss Gibson's return of the changes in this department:—

On Staff, 1st May, 1867, 8; subsequently engaged, 13, ...				21
Dismissed for	Drink, ...	3	12	
	Inefficiency, ...	2		
	Services not required, ...	7		
Resigned, ...			2	
				14
Remaining on Staff, 1st May, 1868, ...				7

As compared with returns in former reports, we have here a decided decrease in the dismissions for misdemeanours. The changes arose chiefly from the variations in the numbers of patients; so that I am left at the end of the year almost with the same individuals on the staff as at the beginning. These constitute what is called the "permanent staff," who are six in number, chosen with care, employed by six-month written

engagement, and methodically instructed. During the summer they are sufficient for the ordinary work as well as for any emergencies; and during the winter I adopted the following method of utilizing their services to the utmost. In Wards I. (female) and II. (male), four of the permanent staff were stationed, one by night in each ward, and one by day. Thus the best nursing at all hours was ensured to the patients in those wards; and into them were put all above thirty years of age, or who were evidently serious cases when admitted. Another member of the staff took charge of the nursery by day, where infants and young children were sent; while the remaining nurse had charge by day of a ward where boys and young men were treated. In these two wards night-duty was taken by carefully chosen night-nurses. During the height of the winter's pressure, all the wards in the Hospital were opened, and the nurses employed numbered 13; but even then, by following out as far as possible this principle of classification, the best of the nursing was reserved for those who most needed it, and every patient had one trustworthy and intelligent person at least during a portion of the day in attendance. The general result was, that personally I felt pleased with the nursing in the Hospital during last year, as I never was before; and, indeed, so far as the "permanent staff" is concerned, it was as nearly perfect as I hope to see. That it was good is shown by the fact that it is in the adult periods of life where we have improved most upon former practice, and where the general excellence is most marked. This subject of nursing, especially in the department of fever, is one on which I have dwelt in former reports, and which may seem to some unduly obtruded—indeed, to be a mere hobby. But it is a conviction established in my mind by observation, and daily strengthening with experience, that success in the treatment of continued fever can be gained only by faithful, minute, and intelligent attention from hour to hour on the part of the nurse, and to a certain extent also on the part of the medical attendant. The nursing is the most important. It must be had, cost what it may, for all cases;

while it is merely in selected cases, and at certain stages, that the medical man ought to see his patients frequently. Even in those he is helpless unless aided by a nurse, and powerless readily to detect carelessness, as he would in ordinary diseases when the patient is sensible and can report delinquencies. Therefore, while nursing in all its branches demands attention, fever-nursing I hold to be the most important of all. Through a variety of causes, especially from the dangerous nature of the duties, it tends to fall into the hands of the worthless or incapable. In place of such, we require the worthy and the intelligent. I very much desire the Board of Police and general public to understand clearly why a fever nurse should not only be a good nurse, but if possible the best of nurses. Her patients suffer from a disease which cannot be checked—which passes deliberately through its stages—which creates wants, while it deprives of the power of supplying them, and ultimately destroys the sense of their existence, just when the real need is at the greatest—which so stupefies the faculties that neglect is not observed, and ill-treatment is forgotten. A nurse in a fever ward, as a rule, can never be at rest, whether her watch be by day or by night. Indeed, when a ward is rapidly filled, so that all the beds contain patients in the heat of the fever, one nurse is quite insufficient for 11 patients, as I found once or twice last winter. Food must be given judiciously in small quantities from hour to hour, day and night, without distinction. One patient is stupidly sleepy, and must be roused now and then to take milk or beef-tea, not waiting until he asks it; another is delirious, perhaps violent, and must be soothed, and only as a last resource restrained, which is the first notion of a bad and lazy nurse; another is obstinate, or full of notions of poison in his drinks, and must be coaxed, or cheated, or overcome, in any way which will result in getting the nourishment into his stomach. Apart from such peculiar incidents, the majority, even in the ordinary course, are restless—tossing the clothes off, and requiring them to be put kindly on again—crying for drinks, and needing that they be given with a soft word, not with a scold for the trouble of it—in many respects being as helpless as infants, while yet having the weight and unwieldiness of adults. There is, I assert, no sort of nursing so laborious and exacting as fever-nursing. Let any one who would be convinced of this look into a ward in each department of the Royal Infirmary at any time, but especially at night. As a rule, in the medical and surgical wards things are quiet. The night-nurse may keep her seat, and give a draught of water when it is asked—help to shift out of an uneasy posture, or administer the medicine at the prescribed hour, and do her duty well with little labour. But in the fever wards there generally are noisy delirium, and distracting calls from various quarters, and constant occasions for lifting and turning, and patients for whom there is no motion of the body for any purpose, or to any extent, without aid or actual lifting as of a dead thing—when duties have to be performed, gently and with tenderness, from which naturally one would shrink. Indeed I often have stood at midnight and looked upon a ward full of males, mostly adults—one strong and violent, and strapped down—others helpless and dependent as infants—others restless, and no sooner calmed than up again, burning with some wild fancy; and then wondered at the courage a woman must have to pass the long hours of a night watch alone in such company, and still more at the rarer gentleness, where such courage exists, which must combine with and soften its manifestations. Women enough are to be found who are callous and hard, equal to anything; who flock to a fever hospital, who protest their want of fear, who have had "the fivver" repeatedly, and whose faces seem incapable of kindly emotion; who by sleeping draughts and strait-jackets make things snug, and enjoy a quiet sleep during the night—who nevertheless, through the activity of their imagination and the extent of their "experience," are able to give a marvellously natural and graphic account of how the patient "passed the night," to the physician at the morning visit. We have depicted the harassing attentions

required of the fever nurse. But suppose her to be one of this sort—suppose she neglects her duty, goes to sleep, gives no drinks, no medicine, no wine—suppose she even drinks the wine: how can the delinquency be detected? The fever patient is usually stupid and incoherent; and even when most intelligent, his statements cannot be safely trusted. But, indeed, one never hears of a complaint even in such circumstances. A shrewd eye may see it written on the sunk face and parched tongue, and on their poor bodies in bed-sores; but otherwise a bad fever nurse is without a witness against her. I might show further how much independence of judgment, how much manual dexterity in certain delicate operations which she ought to perform, are required to make a good fever nurse; but I have already said enough to direct the attention of those who have fever wards or hospitals under their care, in the most pointed manner, to a scrutiny of their nursing as a part of the treatment of fever which lies close to the secret of success.

The requests for private nurses have been frequent and urgent during the year, but only on two occasions could be complied with—once by sending out a nurse, and once when a medical practitioner seized with fever became unmanageably delirious and a male attendant was applied for, and one of the porters sent. The gentleman recovered, and the assistance given was gratefully appreciated. On one occasion also a poor woman recently confined, and unfit therefore for removal, although ill of fever, was nursed by a nurse from the Hospital at the request of the District Medical Officer.

During the past year, in pursuance of the policy advised in my last Report, the Hospital has been thrown open to patients for payment to a greater extent than formerly—the charge being £2 per head. On these terms, 366 patients were treated for the City Parochial Board. Besides these, 6 private patients were treated at same rate; and one, who occupied a private room, paid £6. As it would divert attention from the proper objects of the institution to treat patients anywhere save in the open wards, I receive those private

cases only on condition that they enter the wards. In the one case mentioned exception was made, as separate nurses also were not needed. On these terms, so long as the patients whom the Board are bound to treat do not require all the accommodation, any person may be admitted. We have removed both Police and Pauper cases after hours, sometimes late in the night; and no person presenting a medical certificate at any hour is refused attention. If within Sanitary Office hours, the case is referred to the officials there; but if not, then the certificate is immediately acted upon, and the patient is removed.

During the period 1st May, 1867, to 30th April, 1868, the total number of patients admitted was 969. At the close of last year 24 were still under treatment, making a total of 993, who are accounted for thus:—832 dismissed, 96 died, and 65 remaining to be carried to next year. Summing up the statistics of the Hospital since its erection, we find that 2834 persons have been admitted, 2455 dismissed, and 314 have died. Table No. I. shows the number admitted, dismissed, and died, with the highest and lowest number in Hospital for each month in the year. The highest monthly admission was 131, in January; and the highest number under treatment at one time was 108, on the 31st of January, 1868. These numbers are the highest in the history of the Hospital. The highest number ever in the Hospital before this year was 89, on 1st December, 1865. At this time I found it impossible to carry on the general management of the Hospital and the treatment of so many patients unaided; and accordingly I applied for assistance, which was immediately granted by the Board. Joseph Coats, M.B., acted as resident assistant from 1st February for seven weeks. Rapid oscillations in the number of cases have always been a puzzling feature in the management of this Hospital. It is well to point this out to the Committee with reference to the adjustment of the staff to the requirements. In the month of January just spoken of we find a striking instance. On the 14th there were 50 patients in Hospital, and by the 31st

there were 108. In those 17 days, 96 patients were admitted. To anticipate and prepare for such emergencies is part of the duty of a superintendent; so that it is neither prudent nor even possible to vary the staff *pari passu* with the patients. Nor can the relation between number of patients and number of attendants be taken simply as a numerical proportion. The nature of the cases—as, whether they suffer from mutually infectious diseases—must also be enquired into. At two distinct dates there might be the same number of patients; but at one time homogeneous, so to speak, at another time heterogeneous, and therefore the number of attendants might be doubled in the latter case. To show how far I have been able to adjust the staff to the number of patients, I add to Table No. I. two columns showing the daily average for each month, and the number of nurses on the pay-bill for the corresponding period. During the summer months small-pox and scarlet fever were under treatment; so that 8 nurses were as necessary in June for 24 patients as in November for 52.

Having thus dismissed the round numbers of the Hospital work, I shall in the following paragraphs speak of each class of cases with reference to the final result, because at the time of writing that is with certainty known. In Table No. II. are displayed the monthly admissions of various diseases, with number of deaths from each disease *after treatment*. Of the 969 patients, 795 were cases of typhus, 55 of enteric fever, 35 of scarlet fever, 14 of small-pox, 3 of measles, 22 of febricula, and 45 of other diseases.

Typhus.—Table No. III. exhibits in quinquennial periods of age the usual data with regard to mortality and stimulation. The general mortality was 9·05 per cent., against 12·5 last year, and 11 the preceding. These percentages ought not, however, to be taken simply for any purposes of comparison. In Table No. IV., my own statistics for the three years of the Hospital are given in parallel columns with those of the Glasgow Royal Infirmary for 1867, and the London Fever Hospital for same year, the figures being in both cases from the published reports. From this Table I wish all comparisons to be made, and made in accordance with this principle —"that hospital is most successful which can show *the least mortality at the greatest number of periods*." (Report 1865-6, p. 29.) Comparing ourselves with ourselves, I find that at 9 out of 14 periods of life the mortality is less than that of previous years. Comparing this Hospital with the Royal Infirmary during almost the same space of time, our mortality is considerably lower at all periods save three. Finally, as compared with the London Fever Hospital, Glasgow is lower at all periods save two. Many of the cases were hopelessly gone when admitted, as will appear when it is stated that 1 died 4 hours 40 minutes after admission, 1 within $8\frac{3}{4}$ hours, 1 within 13 hours, 1 within 24 hours, 1 within 36 hours, and 3 within 48 hours; in all, 8 cases moribund on admission.

The *proportion stimulated* in the course of their illness is less this year than last. Owing to the admission of the pauper element amongst my patients—many being dissipated creatures accustomed to drink—I had to use whisky more frequently and freely than on previous years; so that, while a less number required to be stimulated, those who did, required the alcohol less diluted. As this was the first occasion on which I had treated parochial side by side with non-parochial cases, the opportunity seemed a favourable one for testing the accuracy of certain impressions of difference which the treatment of them separately had produced on my mind. The numbers are rather few to give safe ground for induction, except in a very broad way; but such as they are they will be found in Table No. V., which gives the number treated, died, and stimulated at the various periods of age, "Parochial" and "Non-Parochial" in contrast, with percentages. Of the 297 parish cases of typhus, 10 per cent. died, and 35 per cent. were stimulated; while of the 498 non-parochial cases, 8·4 per cent. died, and 36 per cent. were stimulated. But here, as in all such statistical enquiries, we must take a survey of these facts in relation to age. Running the eye over the different quinquennial periods, we discover an evident pre-

dominance of fatality, with increased need of stimulation, among the parochial cases above thirty-five, while below that age there is even a more marked contrast in the opposite direction. This striking fact is brought clearly out by taking all parochial cases below thirty-five, and contrasting them with all non-parochial cases below thirty-five; and so also with all cases above thirty-five. We then find that on the youthful side of thirty-five the parochial mortality is only 2·6 per cent., stimulation 23 per cent.; while the non-parochial mortality is 5·6 per cent., stimulation 33·3 per cent. On the aged side of thirty-five, on the contrary, the parochial mortality is 35·8 per cent., the stimulation 76 per cent.; while the non-parochial mortality is only 23·6 per cent., the stimulation 67 per cent. The lesson broadly gathered from these interesting facts I believe to be this, that it is the dissipation and abandoned life which are so generally the basis of pauperism, and not the effects of poverty and privation, pure and simple, which render typhus more dangerous to paupers than to the best fed and clothed of its victims. Indeed these figures confirm in the gross what every fever physician must have observed in single instances, that the half-fed and emaciated, if not also debauched, pass with greater impunity through fever than the highly nourished and muscular.

The Average Residence of typhus cases who recovered was 19½ days, of those who died nearly 8 days, and over all cases 18½ days. Last year those averages were lower. The increase in length of residence arises partly from the low mortality, and partly from the necessity of feeding up well the pauper patients, who had no home-comforts awaiting them.

Enteric Fever was more prevalent this year than last in Glasgow. 55 cases were treated, with 9 deaths, or 16·3 per cent.—a high mortality, being 2 per cent. above the Royal Infirmary and the London Fever Hospital. Two of the cases were moribund when admitted—one dying within 36 hours; and one remarkable case expired from abdominal hæmorrhage 4 hours after admission. Of the 9 deaths,

4 arose from exhaustion, either from long drain on the system, or from the vitality being borne down early by the impact of the poison; 2 were from perforation and consequent peritonitis; 1 from perforation killing by primary shock; 1 from peritonitis, its origin not being clear—probably splenic embolism; and 1 from hæmorrhage. As might be expected, as enteric fever haunts different localities* from those frequented by typhus, so it prevails in a different grade of society. This is slightly indicated in those cases by the circumstance that only 13 out of the 55 were parochial cases. The *average residence* of those who recovered was 28½ days, of those who died 10, and over all cases 25½ days. All were treated as usual in the open wards, without being infected by typhus.

Scarlet Fever.—35 cases were admitted, and 8 died, or 22·8 per cent., a mortality which is extremely high. But when we separate the parochial from the ordinary cases, and find that 17 were sent in by the City Parochial Board, and 7 of those died, while of the remaining 18 received from various parts of the city only 1 died, we have an indication of the causes which contributed to this mortality. Two of the parish cases were sent from the Lying-in Hospital, Rottenrow, and were cases of puerperal scarlatina, which is almost always fatal. The others were children from the City Poorhouse—puny, sickly creatures, with latent constitutional diseases, most discouraging and vexing to treat. Of the deaths, 4 were from the direct effects of the poison, including the two puerperal cases; 3 were from pneumonia, and 1 from double pleurisy. *The average residence* of those who recovered was 28½ days, of those who died 8 days, and over all cases 24 days.

Small-pox.—14 cases were treated, with one death; 9 were unmodified by previous vaccination, 4 adults and 5 children,

* I have made an investigation into the distribution of enteric fever in the city, which will be published elsewhere. The broad result is to show that, so far as hospital experience goes, the Infirmary included, it has no special habitats in Glasgow.

of whom 1 died; 5 were modified, 4 adults and 1 child. 5 of the cases were members of one family—3 unmodified, and 2 modified. The history of those cases is interesting, as illustrative of the practical difficulties, apparent to all who are actually acquainted with sanitary operations, especially in large towns, which would render Sir J. Y. Simpson's "stamping-out" process practically ineffectual. G. C., living at 80 High Street, took headache on 24th May, pain in his back on the 27th, and on the 28th observed spots on his face. They gradually developed themselves; but, feeling well, he went about his work as usual until the neighbours became alarmed, and said he had small-pox. A doctor being got, the patient was sent in to Hospital on 3rd June, presenting a copious eruption of distinct modified small-pox, the pustules beginning to burst. On 11th June this man's wife and two children, an infant of three months and a boy of three years, were admitted. The woman had well-marked modified small-pox. The baby, although unvaccinated, recovered from a severe confluent attack; but the boy, who took ill on the 8th, and was also unvaccinated, died. The house was now empty of its inmates; but on 13th June a young woman, aged twenty-two, was admitted, who had visited the wife occasionally after the husband's removal, and who sickened on the 10th. She also had not been vaccinated, and passed through a severe attack. Here therefore was a man going about for a week, during any day of which a doctor could have told him he had small-pox, but never suspecting it. Even after the disease was recognized, we have here parents, knowing that these two children were unvaccinated, taking no precautionary measures; and an adult woman, knowing herself to be unprotected, continuing to expose herself. While such ignorance is so common as it is, and the wrong-headedness founded upon it which leads people even to conceal a disease known to be infectious, no process of "stamping-out," though elaborate and perfect in print, and much to be desired in fact, can be prosecuted with success, Indeed any appearance of compulsory power of removal, or

of the interference of authority, even in the way of enquiry, is always found to render discovery more difficult, and to increase the effort to conceal, even from the neighbours, who are generally ready to tell from selfish motives. "Stamping-out" therefore would result practically in stamping-in; and so would the original process as applied to cattle, had they only as much power of combined action, and as much to say in the disposition of their own affairs, as human beings, and especially the British, have.

Measles.—The three cases treated were all sent in as small-pox.

Febricula.—Of the 22 cases so classified, many might more properly be called simple continued fever, as being longer and sharper than febricula, but yet not possessing the characters of any specific fever. Some cases were distinctly associated with typhus, but could not be so classified. The one death is due to cystitis, induced by over-distension of the bladder. When the patient (a male, seventy-four years of age, with enlarged prostate) was admitted, it was enormously distended; and he died exhausted, after being five weeks in Hospital. There never was any evidence of specific fever, nor could the case be connected with such fever.

Other Diseases.—These are cases sent in under the belief that they were labouring under one or other of the specific diseases already noticed. They were not sent in with the knowledge that they are what they here figure as being. The number of such cases this year was 45, or only 4·6 per cent. of error in the total number admitted, which is nearly one-half less than I have ever known before, and is, considering the difficulties attending diagnosis, and the propriety also of rather sending in a patient by mistake than allowing a patient to lie at home by mistake, very creditable to the diagnosis of the medical practitioners of the city. The proportion of error in the London Fever Hospital was 18 per cent. last year. Still, in looking over this list of cases, it is evident that there is yet some room for improvement; and as a ready help in eliminating error, I venture to urge the

use of the thermometer, and to state shortly the nature of the help to be expected.

The thermometer will not by one observation tell the practitioner what is the nature of the disease, but it decides the question—febrile or non-febrile, abnormally or normally hot. There may be every constitutional sign of fever—quick pulse, dry, brown tongue, flushed face, ambiguous eruptions, even a deceptive sensation of heat to the touch—and yet the temperature may be normal. *There is no unusual production of heat, and therefore there can be no fever.* On the other hand, there may be no prominent constitutional signs of fever, especially there may be a quiet pulse, and otherwise only such delicate indications as a practised observer would detect—for these are never wanting, even in such a case—and yet the temperature may be high. *There is unusual production of heat, and therefore there may be specific fever.* I give a few illustrations from this table of "Other Diseases." "Destitution:" temperature in arm-pit on evening of day of admission, 98·2° Fahr. "Purpura:" temperature as before, 98·6°. "Mercurial salivation:" temperature as before, 99·6°, the pulse being 150. "Post-partum Debility:" temperature as before, 98·4°. Of course the cases entered as "Nothing" were equally destitute of the one essential property of fever, abnormal heat. It may be that some of those cases were seen by the certifying practitioner a day or more before admission. It must also be remembered that feverishness in any degree, without local cause, is sufficient to warrant the removal of a person from a large family or a crowded stair. But, all allowances being made, there can be no doubt that some of those cases might have been detected by the thermometer. To ensure greater accuracy is not merely an object of professional ambition. The result of a mistake to the unfortunate patient is, that the stamp of fever is most likely placed upon him or her by the removal of the hair, unless in the rare cases when it is tidy, and not infested with vermin. The majority of such people live in lodgings; and so branded, they become

homeless—no lodging-house keeper will admit them. Some of the most vexing scenes I have witnessed have occurred in connection with such cases.

Disease.	Admd.	Died.	Disease.	Admd.	Died.
Pneumonia,	15	4	Brought forward,	31	7
Cerebral,	5	3	Acute Eczema,	1	...
Bronchitis,	1	...	Itch,	1	...
Phthisis,	2	...	Purpura,	1	...
Hepatic,	2	...	Drink,	2	...
Bright's,	1	...	Delirium Tremens,	1	...
Uterine,	1	...	Phlegmasia Dolens,	1	1
Ovaritis,	1	...	Post-partum Debility,	1	...
Choleraic Diarrhœa,	1	...	Destitution,	1	...
Dysentery,	1	...	Doubtful,	1	1
Mercurial Salivation,	1	...	Nothing,	4	...
Carry forward,	31	7	Total,	45	9

Little can be said, which would be of general interest, regarding this list. All these cases were treated to some extent. Of the 15 cases of *pneumonia*, 4 died. The age of one of the fatal cases was 54; of another, 65. The site of the disease in the other two was the apex of the lung affected; and in one it had supervened during a drinking-bout. The case of *choleraic diarrhœa* occurred in August, 1867. It was specially reported upon to Dr. Gairdner, and differed from the cholera of October, 1866, in hardly any respect save the recovery of the patient. The case recorded as "*doubtful*" very much resembled enteric fever in many respects, but yet could not be so classified. It proved fatal, after an illness of eight days.

General Remarks regarding Patients.—366 were paupers, 7 paid their own expenses, and 596 were treated by the Board of Police. Of the pauper patients, 43 died, and 22 were interred by the parish; of the others, 57 died, and 10 were interred by the Board of Police; the remainder in each case by the friends. Exclusive of the officials of the Hospital, 12 employés of the Board were treated, and 2 died—viz., 1 sanitary inspector, 6 constables, 3 scavengers, and 2 lamp-

lighters. Of these, 8 were suffering from typhus, 2 from small-pox, 1 from measles, and 1 from pneumonia. The Hospital staff supplied only 3 cases—all of typhus: 1 nurse, who recovered; 1 scrubber, who died; and my domestic servant, who recovered. The scrubber was interred in the Hospital lair at Sighthill, at the expense of the Board.

The Royal Infirmary Dorcas Society has during the past year enabled us to supply comfortable clothing to such as were needy and seemed to be deserving. The Matron states that she has issued from their branch store at the Hospital 483 articles of clothing to 148 individuals—viz., 310 articles to 83 females, and 173 articles to 65 males.

The Chaplain of the Royal Infirmary, Mr. Topping, has again obliged us during the year, more especially by officiating at the interment of the scrubber, Mrs. Finlayson. The Roman Catholic patients lost the services of a faithful and judicious minister by the promotion of the Rev. Mr. Dwyer to a separate charge, early in the year. His duties are now discharged by the Rev. Mr. Oswald.

CHOLERA HOSPITALS.

The Greendyke Hospital was maintained during the past year under the charge of private watchmen. Notwithstanding the extremely wet and boisterous winter, and the temporary nature of the erection, it continued in a perfectly serviceable condition. Under the pressure of January on the Fever Hospital, I found it necessary to employ the stock of bedding stored at Greendyke, as well as other portions of the stores.*

The Cranstonhill Hospital remains as it was, and is in good condition, and ready for any emergency.

* *June.*—Since the close of the year on which I report, this hospital has been entirely removed, and the remaining stores distributed partly to the Fever Hospital and partly to the Cholera Hospital at Cranstonhill, where they are held in reserve for future emergencies.

FINANCIAL STATEMENT.

In October, 1867, various causes combined to induce the Board of Police to consider minutely the financial affairs of the Hospital, with reference especially to the comparative expense of using it for treatment, and of shutting it up. By the "Glasgow Police Act, 1866," the position of this Hospital, or of some hospital as a *permanent building* in possession of the Board, for the treatment of infectious diseases when required, seems to be secured. (Sect. 266.) There are thus certain charges in the annual expenditure which cannot be got rid of, which would continue supposing the Hospital to be closed, and which therefore, not being created by the treatment of patients, ought not to be charged to treatment. The total amount of this *permanent outlay* is £300 per annum on an average. This includes £150 for rent of land, £84 for watching, and the balance for coal, gas, painting, and other repairs necessary to "maintain" the building. Taking the requisite data from the Report for 1866–7, by the advice and with the aid of the Medical Officer for the city, Dr. Gairdner, I submitted to a Special Committee of the Board an estimate of the annual *working expenditure* of the Hospital, calculated for daily averages of patients over a year from 20 up to 60. These calculations were minutely examined by Bailie Miller and others; and the result of the investigation was to show, that so soon as the daily average over the year passes 40, the average cost of treatment falls within £2 per patient. The appended financial statement of the year 1867–8, which I now submit, will be found to confirm the estimates referred to. Indeed it must be explained that of the £300 which ought to be deducted as *permanent outlay*, I have deducted only the £150 paid as rent of the Hospital site. If we deduct the other half of the sum, we get both a more favourable and a more strictly correct result, which shows the financial gain for the year, of treating our own patients, to have been £182 on a daily average of 48 inmates.

Without this additional deduction, the working expenditure, as given in the Abstract appended, is £1909 16s. 4d. The receipts from the City Parochial Board and private patients were £750. The actual working cost of the Hospital to the Police Board is thus reduced to £1159 16s. 4d., which represents the outlay in the treatment of 596 patients.

It is difficult to estimate, from the aggregate sums, in what departments of expenditure there has been an increase or a decrease relatively to the work done. In all it has been my aim to reduce expenditure to a minimum, always having regard, in the first place, to the efficiency of the Hospital. The courteous advice and aid always rendered by the fortnightly visitors, and the Hospital Committee at its meetings, have made my efforts more successful than would otherwise have been possible. The Matron has managed the kitchen with minute care and economy. I have checked all the transactions of the storekeeper for the year, and compared the purchases with the recorded consumption, finding a sufficiently close agreement between the stock in hand and the balances. The actual figures will be found in the books, which are open to inspection, with the percentage of error noted, and also the average cost of each article for the year. Referring for a moment to a few of the departments, I may state that in household expenditure there is a decrease of £10; in stationery, a decrease of £8. One item, the expenses of horse and van, which drew down special criticism last year, suggests the record of a fact. The item *horse hire* was, by the purchase of a horse, done away with—31st October, 1867. So early as September, 1865, I recommended, in a memorandum addressed to the Sanitary Committee, the purchase of a horse, on the ground that "at the present rate of hiring its cost would be cleared in six or seven months." As seven months of last year had elapsed before the purchase, it will be observed that there still is an item of £32 2s. for "horse hire," which will not appear again. A cart has also been provided by the Committee, which is found to be very useful, and will also enable us to save various items, such as cartage

of coal. This will be another deduction in future statements. In the item Salaries, there is £14 additional for my temporary assistant in February and March. The item Wages is remarkable, as, last year, with a daily average of 24 patients, it was £350, while this year, with double the average number of patients, it is only £35 more. This arises from the circumstance that with six nurses 50 or 60 patients can be treated as easily as 20, and fewer nurses will not do for the smaller number.

The item Provisions still shows the influence of unusually high prices. Fortunately, very favourable contracts for beef and milk—the staple articles of our consumption—more than counterbalanced a rise in such articles as bread, potatoes, oatmeal, &c., and enables me to show a decrease in the aggregate cost per patient. The official dietary was somewhat improved last year,* so that its money-value cannot justly be compared with previous years. The cost per week for food was: of nurses, 4s. 6d.; of scrubbers, 4s. 1¾d.; of porters, 5s. 8½d.

The following are the usual data, with calculations of the expenditure, in various aspects, founded thereon:—

		£	s.	d.	q.
Average Daily Number of Patients, ...	48				
" Residence of Typhus Cases, ...	18·5 days.				
" " Enteric Cases, ...	25·5 "				
" " Small-pox Cases, ...	20·5 "				
" " Scarlet Fever Cases, ...	22 "				
" " All Cases, ...	18 "				
" Daily Expenditure, $\frac{£1910}{366}$ = ...		5	4	4½	
" " Cost of Patients, $\frac{£5\ 4\ 4½}{48}$ = ...		0	2	2	37†
" Cost of Typhus Case, ... (2s. 2d. ·37q.) × 18·5 =		2	0	2½	
" " all Cases, ... (2s. 2d. ·37q.) × 18 =		1	19	1½	

The only useful and really comparable view of the expenditure is to be gained from the "Classification of Expenses

* Reprinted in this Report, p. 26, as amended.

† All the other Averages may readily be got in the same way.

with regard to Patients," given on page 38; and the following Table founded thereon.

		Average Expense of the Hospital per Day.	Average Expense of a Patient per Day.	Average Expense of Treatment of a Patient.
		£ s. d. q.	£ s. d. q.	£ s. d. q.
Direct.	Food,	1 1 0 3·84	0 0 5 1·08	0 7 10 3·4
	Stimulants,	0 3 1 2·42	0 0 0 3·14	0 1 2 0·3
	Medicine,	0 1 1 3·65	0 0 0 1·16	0 0 5 0·7
Indirect.	Official,	2 14 7 0·73	0 1 1 2·59	1 0 5 2·6
	Conveyance,	0 5 2 3·16	0 0 1 1·23	0 1 11 2·1
	Firing, &c.,	0 12 9 1·44	0 0 3 0·78	0 4 9 2·1
	Various,	0 6 4 2·56	0 0 1 2·39	0 2 4 2·8
	Totals,	5 4 4 1·8	0 2 2 0·37	1 19 1¼

As strictly comparable items, I have tabulated in parallel columns the "direct" expenditure per patient for the three years of the Hospital.

	Average "Direct" Expense per Patient per Day.			Average "Direct" Expense of Treatment per Patient.		
	1865-6.	1866-7.	1867-8.	1865-6.	1866-7.	1867-8.
	s. d. q.	s. d. q.	s. d. q.	s. d. q.	s. d. q.	s. d. q.
Food,	0 5 0·14	0 6 1·33	0 5 1·08	7 4 0·0	8 4 1·0	7 10 3·4
Stimulants,	0 1 0·00	0 0 3·94	0 0 3·14	1 5 2·0	1 3 2·0	1 2 0·3
Medicine,	0 0 2·02	0 0 2·25	0 0 1·16	0 8 3·0	0 9 0·0	0 5 0·7
Total "Direct" Expense,	0 6¼	0 7¾	0 6¼	9 6¼	10 4¼	9 6

An examination of this table suggests various remarks. For instance, the expense for stimulants has decreased each year, and was last year only a mere fraction over three farthings per day to each patient. The expense for medicine was also less last year than ever it has been. Indeed, the entire "direct" expense was lower, being 6¼d. per day. In estimating the average "direct" expense for the terms of treatment, the difference in length of residence last year counterbalances the daily saving; but not quite, as the total

cost of food, stimulants, and medicine for each patient was 9s. 6d.; while in 1865–6 it was 9s. 6¼d., and in 1866–7 it was 10s. 4¾d.

Expenditure—Greendyke Cholera Hospital.

I have appended a note of the expenditure in connection with the watching of Greendyke Hospital during the year. There is also an account for earthenware, which was supplied during the furnishing of the Hospital.

OFFICIAL DIETARY.—CITY OF GLASGOW FEVER HOSPITAL.

NURSES.

	Sunday.	Monday.	Tuesday.	Wednesday.	Thursday.	Friday.	Saturday.
Breakfast and Supper,	Egg. Sweet Milk, ½ pint.	Bread, 2 lbs. Sweet Milk, ½ pint.	Sweet Milk, ½ pint.	SweetMilk, ½ pint.	Bread, 2 lbs. Sweet Milk, ½ pint.	Sweet Milk, ½ pint.	Bread, 2 lbs. Tea,3oz.,Sugar,¾lb. Salt Butter, ½ lb. Sweet Milk, ½ pint.
Dinner,.....	Broth, 1½ pints.	Steak, ½ lb. Potatoes, 1 lb.	Fish, ½ lb. Potatoes, 1 lb.	Broth, 1½ pints.	Steak, ½ lb. Potatoes, 1 lb.	Fish, ½ lb. Potatoes, 1 lb.	Steak, ½ lb. Potatoes, 1 lb.

SCRUBBERS.

Breakfast,	Egg.	Porridge.	Bread, 2 lbs. Porridge.	Porridge.	Bread, 2 lbs. Porridge.	Porridge.	Bread, 2 lbs. Tea,2oz.,Sugar,½lb. Salt Butter, ½ lb. Porridge.
Dinner,.....	Broth, 1½ pints.	Steak, ½ lb. Potatoes, 1 lb.	Fish, ½ lb. Potatoes, 1 lb.	Broth, 1½ pints.	Steak, ½ lb. Potatoes, 1 lb.	Fish, ½ lb. Potatoes, 1 lb.	Broth, 1½ pints. Potatoes, 1 lb.

PORTERS.

Breakfast,	Ham or Fish.	Bread, 2 lbs. Porridge.	Egg.	Bread, 1 lb. Porridge.	Bread, 2 lbs. Ham or Fish.	Porridge.	Bread, 2 lbs. Egg. Tea,3oz.,Sugar,¾lb. Butter, ½ lb.
Dinner,.....	Broth, 1 pint. Steak, ½ lb. Potatoes, 1 lb.	Broth, 1½ pints. Potatoes, 1 lb.	Fish, ½ lb. Potatoes, 1½ lbs.	Steak, ½ lb. Potatoes, 1 lb.	Broth, 1 pint. Steak, ½ lb. Potatoes, 1 lb.	Fish, ½ lb. Potatoes, 1½ lbs.	Broth, 1½ pints. Potatoes, 1 lb.
Supper,.....	Porridge.	Porridge.	Porridge.	Porridge.	Porridge.	Same as Breakfast.	Porridge.

Diseases.—Porridge as in No. 1 Diet; Broth, Tea, according to directions in Sick Dietary, excepting that the Boiled Meat shall be served up by itself.

APPENDIX TO REPORT.

TABLES REFERRED TO IN REPORT.

ABSTRACT OF WORKING EXPENDITURE.

TABLE No. I.

Monthly Admissions, Dismissions, and Deaths from all Causes, during Year 1867–8.

Month.	Admitted.	Dismissed.		Number in House.		Average Number in Hospital.	Number of Nights on Pay-Sheet.
		Well.	Died.	Highest.	Lowest.		
1867.—May,	37	37	9	31	15	23	6
June,	45	35	2	29	16	24	8
July,	24	31	2	20	9	15	5
August,	60	37	2	35	15	25	6
September,	63	54	4	43	34	39	6
October,	81	70	9	49	38	43	7
November,	102	71	10	68	36	52	8
December,	109	103	13	79	54	68	11
1868.—January,	131	67	12	108	50	66	10
February,	118	129	12	105	81	91	13
March,	104	128	11	86	50	75	11
April,	95	70	10	65	43	52	8
Total 1867–8,	969	832	96				
" 1866–7,	547	478	79				
" 1865–6,	1318	1145	139				
Grand Total,	2834	2455	314				

TABLE No. II.

Monthly Admissions of various Diseases, with number of Deaths from each, after Treatment.

Month.	Typhus.		Enteric Fever.		Scarlet Fever.		Small-pox.		Measles.	Febricula.		Other Diseases.		Total.	
	Admd.	Died.	Adm.	Died.	Adm.	Died.	Adm.	Died.		Adm.	Died.	Adm.	Died.	Adm.	Died.
May,	28	4	2	...	3	2	1	...	...	1	...	2	1	37	7
June,	25	1	1	...	1	...	9	1	...	...	...	9	1	45	3
July,	18	1	...	...	1	...	3	...	...	...	...	2	...	24	1
August,	53	2	...	...	...	...	1	...	...	3	...	3	1	60	3
September,	57	3	1	...	...	...	...	...	...	...	...	5	...	63	3
October,	59	9	12	2	6	...	...	...	1	1	...	2	1	81	12
November,	88	8	6	2	3	1	...	..	...	1	...	4	...	102	11
December,	89	9	8	1	8	3	...	...	...	1	...	3	...	109	13
January,	102	8	14	3	7	...	...	...	1	4	1	3	1	131	13
February,	99	8	6	...	5	2	...	...	...	4	...	4	1	118	11
March,	94	7	1	...	...	...	...	...	...	3	...	6	1	104	8
April,	83	12	4	1	1	...	..	...	1	4	...	2	2	95	15
Total,	795	72	55	9	35	8	14	1	3	22	1	45	9	969	100
Former Yrs.,	1538	176	81	12	26	4	41	3	1	37	...	101	12	1865	220
Grand Total,	2333	248	136	21	61	12	55	4	4	59	1	146	21	2834	320

TABLE No. III.

Statistics of Typhus, 1867-8, showing Number Treated and Stimulated at Quinquennial Periods of Age in each Sex, with Totals and Percentages for each Age.

Age.	Treated.		Died.		Stimulated.		Total Treated.	Total Died.		Total Stimulated.	
	M.	F.	M.	F.	M.	F.			℘ Cent.		℘ Cent.
0— 4,.....	27	35	....	3	3	3	62	3	4·83	6	9·7
5— 9,.....	61	63	...	...	2	7	124	...	...	9	7·2
10—14,.....	89	63	2	4	15	16	152	6	3·94	31	20·4
15—19,.....	55	61	1	2	18	23	116	3	2·58	41	34·4
20—24,.....	49	40	2	6	23	15	89	8	8·98	38	41·5
25—29,.....	25	39	2	4	16	21	64	6	9·37	37	57·8
30—34,.....	17	28	2	2	8	12	45	4	8·88	20	42·2
35—39,.....	17	26	4	5	9	14	43	9	20·93	23	53·5
40—44,.....	19	22	6	5	14	18	41	11	26·82	32	78·
45—49,.....	14	17	6	5	11	11	31	11	35·48	22	70·9
50—54,.....	15	2	5	...	12	2	17	5	29·41	14	82·3
55—59,.....	3	4	2	1	3	3	7	3	42·85	6	85·7
60—64,.....	...	2	...	1	...	2	2	1	50·	2	100·
65—69,.....	1	1	1	1	1	1	2	2	100·	2	100·
All Ages,..	392	403	33	39	135	148					
All Ages and both Sexes,	795		72		283		795	72	9·05	283	352

TABLE No. IV.

Comparative Table of Mortality of Typhus at Quinquennial Periods of Age in this Hospital, Years 1865–6, 1866–7, and 1867–8; London Fever Hospital, 1867; Glasgow Royal Infirmary, 1867.

Age.	City of Glasgow Fever Hospital, 1867-8.			City of Glasgow Fever Hospital, 1866-7.			City of Glasgow Fever Hospital, 1865-6.			Glasgow Royal Infirmary, 1867.			London Fever Hospital, 1867.		
	Trtd.	Died.	Per Cent.	Trtd.	Died.	Per Ct.	Trtd.	Died.	Per Cent.	Trtd.	Died.	Per Ct.	Trtd.	Died.	Per Ct.
0— 4,	62	3	4·83	14	...	...	48	6	12·5	10	...	...	14	1	7·1
5— 9,	124	...	...	49	1	2·	172	2	1·16	39	2	5·12	76	1	1·3
10—14,	152	6	3·94	73	...	...	245	3	1·22	87	3	3·44	174	2	1·1
15—19,	116	3	2·58	60	3	5·	204	15	7·3	158	13	8·22	237	8	3·4
20—24,	89	8	8·98	53	6	11·3	126	16	12·6	136	14	10·29	194	22	11·3
25—29,	64	6	9·37	25	3	12·	78	11	14·1	89	14	15·73	153	33	21·6
30—34,	45	4	8·88	27	5	18·5	80	15	18·7	79	21	26·58	106	17	16·
35—39,	43	9	20·93	22	5	22·7	68	15	22·	52	14	26·92	100	37	37·
40—44,	41	11	26·82	23	6	22·2	55	17	30·9	43	14	32·55	98	30	30·6
45—49,	31	11	35·48	16	7	43·7	33	7	21·2	34	12	35·29	83	37	44·6
50—54,	17	5	29·41	8	4	50·	17	6	35·2	15	8	53·33	69	33	47·8
55—59,	7	3	42·85	5	3	60·	18	9	50·	12	8	66·66	33	23	69·7
60—64,	2	1	50·	7	4	57·	5	3	60·	7	6	85·71	16	14	87·5
65—69,	2	2	100·	1	...	...	4	2	50·	...	...	...	15	12	80·
70—74,	...	...	...	1	1	100·	...	...	...	...	...	...	2	2	100·
75—79,	...	...	...	...	...	...	1	1	100·	...	...	...	1	1	100·
Not specified,	...	...	...	...	...	...	...	...	...	...	...	...	10	...	...
	795	72	9·05	384	48	12·5	1154	128	11·09	761	129	16·95	1381	273	19·7

TABLE No. V.

Comparison of Parochial and Non-Parochial Cases as to Stimulation.

Age.	Parochial.					Non-Parochial.				
	Treated.	Died.	℘ Cent.	Stimulated.	℘ Cent.	Treated.	Died.	℘ Cent.	Stimulated.	℘ Cent.
0— 4,.....	31	2	6·4	4	12·9	31	1	3·2	2	6·4
5— 9,.....	45	...	...	1	2·2	79	...	...	8	11·2
10—14,.....	47	1	2·1	11	23·4	105	5	4·7	20	19·
15—19,.....	47	2	4·2	17	36·1	69	1	1·4	24	34·7
20—24,.....	21	...	...	6	28·5	68	8	11·7	32	47·
25—29,.....	19	...	...	6	31·5	45	6	13·3	31	68·8
30—34,.....	20	1	5·	8	40·	25	3	12·	11	44·4
35—39,.....	15	3	20·	9	60·	28	6	21·4	14	50·
40—44,.....	27	9	33·3	22	81·4	14	2	14·2	10	71·4
45—49,.....	13	5	38·4	10	77·	18	6	33·3	13	72·2
50—54,.....	6	3	50·	4	66·	11	2	18·1	10	90·
55—59,.....	4	2	50·	4	100·	3	1	33·3	2	66·6
60—64,.....	...	...	...	...	...	2	1	50·	2	100·
65—69,.....	2	2	100·	2	100·	...	...	...	...	...
70—74,.....	...	...	...	...	...	...	...	...	...	...
	297	30	10·	104	35·	498	42	8·4	179	36·

ABSTRACT

OF

WORKING EXPENDITURE

OF

THE CITY OF GLASGOW FEVER HOSPITAL,

From 1st MAY, 1867, to 30th APRIL, 1868.

Page					
34.	Provisions,*			£611	17 5
35.	Wines and Spirits, ...			34	5 6
35.	Malt Liquors, ...			27	13 6
35.	Aerated Drinks, ...			13	7 0
36.	Household Expenses and Matron's Sundries, ...			55	14 6
37.	Firing, Lighting, and Cleaning, ...			233	17 6
38.	Medicines, ...			21	4 4
38.	Stationery, ...			12	2 10
39.	Expenses of Horse and Van :—				
	Horse Hire, ...	£32	2 0		
	Provender, ...	56	7 6		
	Miscellaneous, ...	7	5 8		
				95	15 2
40.	Salaries, ...			314	0 0
40.	Wages, ...			385	7 6
40.	Repairs, and Jobbing Accounts, ...			34	16 2
40.	Sundry Furnishings, ...			58	5 11
41.	Miscellaneous Accounts, ...			11	9 0
				£1,909	16 4
	Less Receipts from City Parochial Board and others,			750	0 0
	Actual Expenditure, ...			£1,159	16 4

* Details will be found at the pages indicated.

PROVISIONS.

Table showing Quantity and Value of Purchases, Stock at 30th April, 1868, Consumption and Proportion to Patients and Officials, for period 30th April, 1867, to 30th April, 1868.

Article.	Purchased and in Stock.		In Stock 30th April, 1868.		Consumed.		Patients.		Officials.	
	Quantity.	Cost.	Quantity.	Cost.	Quantity.	Cost.	Quantity.	Cost.	Quantity.	Cost.
		£ s. d.		£ s. d.		£ s. d.		£ s. d.		£ s. d.
Beef, Boiling,	3178½ lbs.,	75 14 2	26½ lbs.,	0 12 9½	3152 lbs.,	75 1 4½	2641 lbs.,	62 18 0	511 lbs.,	12 3 4½
Beef, Steak,	1040 „	40 17 1½	...	...	1040 „	40 17 1½	297 „	11 13 4	743 „	29 3 9½
Beef, Bones,		1 1 6	...	...	...	1 1 6	...	...	...	1 1 6
Beef, Ham,	384 „	11 2 10	...	...	384 „	11 2 10	...	...	384 „	11 2 10
Milk, Sweet,	3380 gallons,	183 15 0	...	...	3380 gals.,	183 15 0	3260 gals.,	177 2 0	120 gals.,	6 13 0
Milk, Skim,	899 „	22 17 8	...	...	899 „	22 17 8	661 „	16 16 0	238 „	6 1 8
Milk, Cream,	746½ gills,	6 4 5	...	...	746½ gills,	6 4 5	...	...	746½ gills,	6 4 5
Bread,	376½ doz. 2 lb. loaves,	76 0 6	14 lbs.,	0 2 6	9022 lbs.,	75 18 0	3820 lbs.,	32 2 9	5202 lbs.,	43 15 3
Oatmeal,	11 loads, 60 lbs.,	27 19 2	212 „	1 17 2	2928 „	26 2 0	2000 „	17 16 6	928 „	8 5 6
Potatoes,	3 tons, 10 cwt., 3 qrs.	20 3 3	28 „	0 1 9	7896 „	20 1 6	2612 „	6 13 0	5284 „	13 8 6
Barley,	7 cwt., 2 qrs., 20 lbs.	5 19 6	3 qrs., 16 lbs.,	0 14 1	760 „	5 5 5	475 „	3 5 11	285 „	1 19 6
Rice,	3 cwt., 2 qrs., 26½ lbs.	3 7 5	1 qr., 7 lbs.,	0 5 4	383½ „	3 2 1	360 „	2 18 4	23½ „	0 3 9
Pease,	1 cwt., 3 qrs., 11½ lbs.	1 8 8	1 qr., 10 lbs.,	0 5 5	170 „	1 3 3	116 „	0 15 10	54 „	0 7 5
Arrowroot,	6½ lbs.,	0 4 10½	4½ lbs.,	0 3 4½	2 „	0 1 6	2 „	0 1 6	...	...
Corn Flour,	1 qr., 4½ lbs.,	0 12 8½	6 „	0 2 5½	26½ „	0 10 3	26½ „	0 10 3	...	...
Tea,	294 lbs.,	39 4 0	28 „	3 14 8	266 „	35 9 4	125 „	16 13 4	141 „	18 16 0
Coffee,	56 lbs., 12 oz.,	3 17 4	3 „ 8 oz.,	0 5 0	53 lbs. 4 oz.	3 12 4	3½ „	0 4 6	50 „	3 7 10
Sugar,	9 cwt., 6 lbs.,	17 13 2	3 qrs., 3 lbs.,	1 10 4	927 lbs.,	16 2 10	195 „	3 7 11	732 „	12 14 11
Butter,	6 cwt., 3 qrs., 9 lbs.,	40 5 5	20 lbs.,	1 2 1	745 „	39 3 4	261 „	13 14 0	484 „	25 9 4
Eggs,	358 doz.,	14 13 3	6½ dozen,	0 4 1	351½ doz.,	14 9 2	139½ doz.	5 14 8	212 doz.,	8 14 6
Fish, Salt,	2½ barrels,	5 7 0	...	...	2½ brls.,	5 7 0	...	...	2½ brls.,	5 7 0
Fish, Fresh,	3 cwt.,	3 2 0	...	...	3 cwt.,	3 2 0	...	...	3 cwt.,	3 2 0
Vegetables,	...	13 0 2	...	...	...	13 0 2	...	5 0 2	...	8 0 0
Concentrated Beef & Liebig's Extr.,	...	6 0 2	...	...	...	6 0 2	...	6 0 2	...	...
Pepper,	9½ lbs.,	0 4 9	3½ lbs.,	0 1 9	6 lbs.,	0 3 0	6 lbs.,	0 3 0	...	...
Mustard,	31 „	1 7 7	2½ „	0 2 1	28½ „	1 5 6	28½ „	1 5 6	...	...
Sundries in Grocer's Account,	...	0 18 8	...	...	...	0 18 8	...	0 18 8	...	...
		£928 2 3½		£1 4 10½		£913 17 5		£895 18 4		£290 2 1

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WINE AND SPIRITS.

Article.	Stock Last Year and Purchased. Quantity.	Cost.	In Stock. Quantity.	Cost.	Consumed. Quantity.	Cost.
Port Wine,	10 1/12 dozen,	£12 2 0	1 1/12 dozen,	£1 6 0	9 dozen,	£10 16 0
Whisky,	16½ gallons,	13 1 4	½ gallons,	0 13 4	15 1/12 gallons,	12 8 0
Brandy,	9½ „	11 12 0	½ „	0 12 0	9½ „	11 0 0
Claret,	½ dozen,	0 9 0	5 bottles,	0 7 6	1 bottle,	0 1 6
		£37 4 4		£2 18 10		£34 5 6

MALT LIQUORS.

Article.	Stock Last Year and Purchased. Quantity.	Cost.	In Stock. Quantity.	Cost.	Consumed. Quantity.	Cost.
Ale,	118 dozen,	£14 13 6	—	—	118 dozen,	£14 13 6
Porter,	130 „	13 0 0	—	—	130 „	13 0 0
		£27 13 6				£27 13 6

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AERATED DRINKS.

Article.	Purchased and Consumed. Quantity.	Cost.
Lemonade,	25½ dozen,	£7 13 0
Soda Water,	24½ „	6 2 6
Carb. Loch Katrine,	1 „	0 5 6
		£14 1 0
Less Discount,		0 14 0
		£13 7 0

HOUSEHOLD EXPENSES AND MATRON'S SUNDRIES.

Month.	Butcher.	Grocer.	Petty Cash Book.	Total.
	£ s. d.	£ s. d.	£ s. d.	£ s. d.
1867.				
May,	2 5 6½	0 14 9	1 17 9	4 18 0½
June,	1 17 8½	0 13 5	2 4 9	4 15 10½
July,	2 2 6½	0 12 8	2 4 7	4 19 9½
August,	2 4 8	0 16 9	2 3 7	5 5 0
September,	2 5 8½	0 14 11	2 0 4	6 0 11½
October,	1 6 8	0 11 2½	2 4 1	4 1 11½
November,	2 6 10½	0 9 10	2 1 7	4 18 3¾
December,	1 15 4	0 11 7	1 18 11	4 5 10
1868.				
January,	1 16 1½	0 11 6	1 19 10	4 7 5½
February,	1 15 2	0 8 9	1 16 11	4 0 10
March,	1 18 8	0 11 1	2 1 0	4 10 9
April,	1 10 11	0 8 8	1 10 2	3 9 9
	23 5 11	7 5 1½	25 3 6	55 14 6½

FIRING, LIGHTING, AND CLEANING.

Coal.—Purchased and Consumed—Coal, 227 Waggons,.....£124 17 0

" " Dross, 46 " 16 2 0

Gas.—Charge for Year,..... £140 19 0

Cleaning.— 49 17 6

	In Stock and Purchased. Quantity. Cwt. Qrs. Lbs.	Cost. £ s. d.	In Stock. Quantity. Cwt. Qrs. Lbs.	Cost. £ s. d.	Consumed. Quantity. Cwt. Qrs. Lbs.	Cost. £ s. d.
Soft Soap,	39¼ Firkins,	£18 19 9	¼ Firkin,	£0 6 1	29¼ Firkins,	£18 13 8
Hard Soap,	3 1 20	5 4 8	1 0 19	1 15 9	2 1 1	3 8 11
Soda,	14 1 18	4 12 9	3 0 0	0 18 0	11 1 18	3 14 9
Sundries,	—	0 8 0	—	—	—	0 8 0
		£29 5 2		£2 19 10		£26 5 4

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Straw for Hospital Bedding.—Purchased, 388½ stones,.....£18 17 4

In Stock, 50 " 2 1 8

Consumed, 338½ stones,..... 16 15 8

£233 17 6

MEDICINES.

Amount of Druggist's Accounts during year,	£25	14	1
Less for "Liebig's Extract," charged to Provisions,	£4	8	6
Less Discount on Balance,	1	1	1
	5	9	7
		£20	4	6
Charged to this Item from Superintendent's Sundries Account—Ice, &c.,	£0	10	10
Cotton Wadding,	0	9	0
		0	19	10
		£21	4	4

STATIONERY.

Total for the year,	£12	2	10
	£12	2	10

EXPENSES OF HORSE AND VAN.

HORSE HIRE.—From 1st May to 30th November, 1867, at 21s. per Week, ... £32 2 0

PROVENDER.—	In Stock. Quantity.	Cost.	Consumed. Quantity.	Cost.
Straw,	113 cwt. 2 qrs.,	£57 7 4	1 cwt.,	£0 6 6
Hay,	6 bolls 3 balls.,	8 13 9	112 cwt. 2 qrs.,	£36 16 10
Oats,	3 bolls 1 bah.,	4 6 10	2 bushels,	0 0 0
Beans,	3 bolls 1 bah.,	5 3 11	6 bolls 1 bah.,	8 4 9
Barley,	3 bolls 1 bah.,	5 3 11	1 "	0 4 8
Bran,	6½ bags,	2 5 6	3 bolls,	4 2 2
			1 "	0 5 8
			3 bolls,	4 18 3
			3 bolls,	4 18 3
			6½ bags,	2 5 6
	£57 13 4		£1 5 10	£56 7 6
				56 7 6

MISCELLANEOUS.—Tolls, Pontages, Boys for holding Horse, and Sundries entered in Vanman's Pass-Book, and charged in Superintendent's Sundries Account,	£4	8	6
Saddler—Sundries,	1	3	8
Shoeing, Sharpening, &c.,	1	17	6
Repairs executed on Van,	2	18	6
	£10	8	2
Less Credited to this Account from Petty Receipts Book,	3	2	6
	7	5	8
	£95	15	2

SALARIES.

Physician and Medical Superintendent,	£240	0	0
Assistant Physician—seven weeks, at £2 per week,	14	0	0
Matron,	60	0	0
	£314	0	0

WAGES.

Storekeeper,	£65	10	8
Gatekeeper,	41	18	11
Vanman,	41	18	11
Under Porter and Barber,	34	15	0
Cook,	15	0	0
Laundry Maid,	12	3	0
Private Servant,	12	0	0
Nurses,	136	19	1
Scrubbers,	27	1	11
	£387	7	6
Less Credited to this Account from Petty Receipts Book,	2	0	0
	£385	7	6

REPAIRS AND JOBBING ACCOUNTS.

Plumber,	£7	7	5
Glazier,	0	14	7
Printer—including printing Report,	19	15	0
Slater,	2	11	2
Repair of Heating Apparatus,	4	8	0
	£34	16	2

SUNDRY FURNISHINGS.

Brushes,	£4	7	0
Books for Nurses and Patients,	2	8	2
India Rubber Sheeting, &c.,	11	14	6
Clothing for Convalescents,	15	7	8
Upholsterer's Items,	6	7	3
Pottery,	4	1	9
Shroud Cloth, Washing Flannel, &c.,	2	0	10
Ironmongery,	3	0	9
Cordage,	0	9	10
Window Blinds,	2	7	4
Cutlery,	1	16	10
Shoes for Convalescents,	4	4	0
	£58	5	11

MISCELLANEOUS ACCOUNTS.

Interment of Nine Bodies,	£7	13	0			
Interment Dues at Sighthill for Scrubber,	0	6	0			
				£7	19	0
Advertising for Nurses, &c.,				0	16	0
Superintendent's Sundries,	£7	13	4			
Deduct Charged to Horse and Van Account, £4 8s. 6d.; to Medicines Account, 10s. 10d.,	4	19	4			
				2	14	0
				£11	9	0

ACCOUNTS PROPERLY NOT CHARGEABLE TO ANNUAL OUTLAY.

Price of Horse,	£30	0	0
" Harness for do. (two Sets),	27	18	9
" Cart,	11	10	0
Rent of Ground,	150	0	0
	£219	8	9

CLASSIFICATION OF EXPENSES WITH REGARD TO PATIENTS.

DIRECT.	Food,				£385	15	4
	Stimulants,	Wine and Spirits,	£34	5	6		
		Malt Liquors,	9	14	6		
		Aerated Drinks,	13	7	0		
						57	7	0
	Medicines,					21	4	4
INDIRECT.	Official,	Provisions,	£226	2	1		
		Domestic Expenses,	55	14	6		
		Malt Liquors,	17	19	0		
		Salaries,	314	0	0		
		Wages,	385	7	6		
						999	3	1
	Conveyance to Hospital,					95	15	2
	Firing, Lighting, Cleaning, and Straw for Beds,					233	17	6
	Various,	Repairs and Jobbing,	£34	16	2			
		Stationery,	12	2	10			
		Sundry Furnishings,	58	5	11			
		Miscellaneous,	11	12	8			
						116	17	7
						£1910	0	0

EXPENDITURE—GREENDYKE CHOLERA HOSPITAL.

Wages.—Day Watchman,	£41	18	11	
Night "	41	18	11	
				£83 17 10
Firing and Lighting.—Coal—13 Waggons,	£7	4	2	
Gas,	10	7	10	
				17 12 0
Pottery,				8 9 2
				£109 19 0

With the kind regards of J. W. B.

STRUMA EXOPHTHALMICA

(VASCULAR BRONCHOCELE AND EXOPHTHALMOS).

By PROFESSOR VIRCHOW.

TRANSLATED, WITH NOTES AND OBSERVATIONS,

By J. WARBURTON BEGBIE, M.D.

EDINBURGH: PRINTED BY OLIVER AND BOYD.

MDCCCLXVIII.

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STRUMA EXOPHTHALMICA.[1]

[In a recently published part of the important work of Virchow on "Tumours" (Die Krankhaften Geschwulste),[2] there occur some statements and reflections in regard to a peculiar form of disease which, both in this country and on the Continent, has of late years attracted very considerable attention. The opinion, as to the nature of so complex a disorder as "Vascular Bronchocele and Exophthalmos," entertained by the eminent professor of Berlin cannot fail to prove of interest to the members of the profession in general. I have therefore thought it worth while to offer a translation of the author's observations, and to this have added some remarks suggested by their perusal. The latter, for the sake of clearness, and in order to avoid all risk of confusing the reader, are thrown entirely by themselves at the close of the translation. The foot-notes and references are those of the author unless otherwise distinguished.]

Finally, that is an exceedingly remarkable connexion of goitre with affection of the heart (Herzreizung, literally heart irritation), and large staring eyes (Glotzaugen). So far as is yet known, Flajani[3] was the first to notice the coincidence of goitre with lasting palpitation of the heart. He mentions three cases of this nature, all of which occurred in men, two of the three being youths. In all of these cases, chiefly by means of external treatment of the bronchocele, a cure was obtained. Of the condition of the eyes he says nothing, but visible enlargement and varicosity of the veins over the thyroid gland were noticed. Parry[4] appears to have been the earliest to observe the third symptom—prominence of the eyes. Next to him, I find two descriptions with post-mortem examinations of accurately investigated cases by Adelmann,[5] in which considerable goitres appeared with enlargement of the heart. During life there existed continued violent palpitation in the region of the

[1] The word "Struma," as used by Virchow in the text, and employed by various German writers, means simply Bronchocele or Goitre—the enlargement of the thyroid gland from whatever cause—and has no relation to that morbid condition or cachexia known as the strumous, or Scrofula.—(*Translator.*)

[2] Zweiundzwanzigste Vorlesung, iii. band, i. halfte, s. 73.

[3] Guiseppe Flajani, Collezione d'osservazioni e riflessioni di Chirurgia, Roma, 1802, t. iii. p. 270.

[4] Caleb Hillier Parry, Collections from the unpublished Medical Writings, London, 1825, vol. ii. p. 3, quoted by Stokes. "Die Krankheiten des Herzens u der Aorta." Uebers, Von Lindwurm, Würzb. 1855, s. 232.

[5] Adelmann, Jahrbücher der philosophisch. medicinischen Gesellschaft zu Wurzburg, 1828, bd. i. ii., s. 104-108.

heart, great dyspnœa, pain in the abdomen; and in one of the cases it is mentioned that, in addition to these symptoms, "the staring look of the large eyes caused a very remarkable aspect." These facts, notwithstanding, remained nearly unknown till the new experience of Pauli,[1] Von Basedow,[2] and Graves,[3] was published. Although the first communication of Graves appeared in 1835,[4] that of Von Basedow has, notwithstanding, the advantage; the history of the disease having been, in the first place, completely given by him, and, through his knowledge, much information regarding it supplied. By Von Basedow was originated the designations of exophthalmos, of the staring eye (Glotzauges) as the most striking feature, and of the cachexia exophthalmica, large staring eye cachexia (Glotzaugenkachexie), which has more recently become so universally known; and it is from this circumstance that, according to the proposal of G. Hirsch,[5] it has been customary to style the whole assemblage of complex symptoms "Morbus Basedowii." Trousseau,[6] on the other hand, maintains that the disease should be termed "Graves' disease." I hold that this is not correct; for Graves regarded both the palpitation of the heart and the goitre as essentially symptoms of hysteria, while the condition of the eyes is only incidentally mentioned by him; moreover, he is not the first who observed the complex symptoms. By recent writers, prominence has been assigned at one time to one, at another time to another of three principal symptoms; and, according to it, the choice of a designation for the disease has been made. Of late, the name "Struma exophthalmica" (goitre exophthalmique) has become very generally disseminated; but Lebert,[7] having reference to the affection of the heart, suggests for the disease the appellation of "Tachycardia Strumosa."[8]

The condition of the Thyroid gland is, during life, subject to variety. As a general rule, the swelling is not so great as that of the ordinary goitre, still a very marked enlargement is discovered. The most conspicuous feature of the swelling is an increased development of the vessels, with which, not unfrequently, a diastolic beat and "souffle" (ein diastoliches Klopfen und Rauschen) are perceived, so as to be directly styled "Struma aneurysmatica,"[9] or

[1] Pauli, Heidelberger Medic. Annalen, 1837, s. 218.
[2] Von Basedow, Casper's Wochenschrift, 1840, No. 13, s. 198.
[3] Robert James Graves, Klinische Beobachtungen, Deutsch von Bressler, Leipz., 1843, s. 409.
[4] Stokes, a. a. O. S. 234.
[5] G. Hirsch, Klinische Fragmente, Königsberg, 1858, heft ii. s. 224.
[6] Trousseau, Gazette Hebdomadaire de Médecine, 1862, No. 30, p. 472.
[7] Lebert, Die Krankheiten der Schildrüse, s. 307.
[8] "Tachycardia" (Ταχυς, quick, Καρδιά, the heart), in reference to the rapid and excited action of the organ. The exact term suggested by Lebert is "tachycardia exophthalmica strumosa." See his "Grundzüge der Artzlichen Praxis," 1867, s. 250.—(*Translator.*)
[9] Henoch, Casper's Wochenschrift, 1848, No. 40, s. 629. Romberg und Henoch, Klinische Wahrnehmungen und Beobachtungen, Berlin, 1851, s. 191. (Of this interesting paper, an abridged rendering by the translator will be found in the "Edinburgh Medical and Surgical Journal," April, 1854.) Bullar, Medico-Chirurgical Transactions, 1861, vol. xliv. p. 37.

"Bronchocele vasculosa."[1] Sudden appearance of swelling, and its rapid subsidence, have been associated. The results of anatomical inquiries are not agreed upon.[2]

In the only case investigated by me, and of which Traube and Von Recklinghausen have given an account, the gland moderately increased in size, exhibited simply excessive plastic without any gelatinous, nodular, or cystic formation. The lobes of the thyroid reached very distinctly forwards, the interstitial structure was abundant, and the veins only were generally enlarged. Very similar was its condition in the case given by Reith, also in one by Trousseau, which Peter has described, except that, in the last, there is no mention made of the enlargement of the veins. Smith found a very considerable augmentation, especially of the right lobe of the gland, while the arteries were much enlarged and strangely tortuous. Markham remarked the gland as being large and firm, at the same time (in a woman of 26 years of age) he found an enlarged and persistent thymus gland. In an instance recorded by Hirsch, the thyroid gland was big, hard, and externally covered by enlarged vessels. Heusinger describes the thyroid as double the natural size and uniformly hypertrophied, but without the presence of any abnormal formation. Very similar was its condition, according to the observation of James Begbie. In the case of Schleich, related by Laqueur, Runge found a large gelatinous goitre. Naumann describes the thyroid as very large, its structure uniformly red, and presenting hæmorrhagic spots, the arteries greatly developed. Von Basedow discovered the gland enormously enlarged with hydatid and varicose degenerations, and Marsh (Sir Henry) saw the thyroid irregularly lobed, containing cysts, which were occupied by a clear fluid, and the jugular veins very greatly distended. Analagous to this was the case recorded by Banks. Lastly, Praël found a ponderous goitre which stretched downwards into the cavity of the chest, its right lobe embracing

[1] Laycock, Edinburgh Medical Journal, 1863, p. 1. J. Warburton Begbie, ditto, September 1863, p. 211.
[2] Marsh, Dublin Journal of Medical Science, 1842, vol. xx. p. 471. Von Basedow, Casper's Wochenschrift, 1848, No. 49, s. 775. Heusinger (in Braunschweig), Casper's Wochenschrift, 1851, No. 4, s. 53. Naumann, Deutsche Klinik, 1853, No. 24. s. 269. Smith, bei Stokes, a. a. O., s. 239. Banks, Dublin Hospital Gazette, 1855 (quoted by W. Moore, Dublin Quarterly Journal, 1865, November, p. 347). James Begbie, Edinburgh Medical and Surgical Journal, 1855. Case-book, p. 33. F. Praël, sen., Archiv f. Ophthalmologie, 1857, bd. iii. 2, s. 199. Markham, Transactions of the Pathological Society of London, 1858, vol. ix. p. 163. Hirsch, a. a. O., s. 224. L. Laqueur, De Morbo Basedowii nonnulla, adjecta singulari observatione, Dissertatio inaugularis, Berol., 1860, p. 12. Traube und Von Recklinghausen, Deutsche Klinik, 1863, No. 29, s. 286. Trousseau et Peter, Gaz. Hebdom., 1864, No. 12, p. 181. Archibald Reith, Medical Times and Gazette, November 1865, p. 521.

the trachea, and having passed into a state of cartilaginous degeneration.[1]

It appears from the foregoing comparison of observations, that it is not a determinate variety of goitre, or a fixed enlargement of the same, or definite course, which settles the appearances. Indeed, in many cases the alteration of tissue is so trifling, that we may ask, as Graves did, whether it is in reality a bronchocele, or merely a swelling (intumescentia) of the gland which exists. From this consideration, there arises a direct refutation of the opinion entertained by some, that the cause of the exophthalmos is the pressure of the thyroidal tumour on the vessels of the neck.[2] Further, it shows, that at first a simple swelling of the gland exists from which a true bronchocele is formed, and that the goitre runs its usual course from a very moderate, chiefly plastic formation, or advances to a fibrous induration of nodular form. The same series of changes, however, occurs with sufficient frequency in the ordinary goitre, without the appearance of the other symptoms, and accordingly the alteration of the thyroid gland is to be regarded as a secondary phenomenon. That the persistent enlargement of the vessels, and especially of the veins, plays a decided part, may already be conjectured from clinical details. It seems to depend less upon the condition of the arteries; at least in all cases in which these were remarkably changed, there existed also considerable disease in other parts of the vascular system.

In nearly all the cases the Heart is greatly enlarged, for the most part dilated, even where the valves are healthy; the left ventricle being chiefly affected. The aorta and great vessels were, in most instances, but by no means in all, atheromatous. Clinical investigation demonstrates that the hypertrophy of the heart belongs to an advanced stage of the disease; accelerated motion (100 beats and upwards in the minute) is the ordinary phenomenon.

The earliest entertained notion in regard to the Eyes was that a hydrophthalmos existed; this is now on all sides abandoned. Naumann alone has found a trifling enlargement of the eyeball. The essential change lies in the fatty tissue of the orbit, which is sometimes hypertrophied, but is for the most part expanded by a hyperæmic swelling, capable of being overcome during life by pressure, and readily disappearing after death.[3] Reith alone, besides greatly distended veins, found a small quantity of partially coagulated

[1] Besides the names already mentioned in the text, that of "Cardiagmus strumosus" (*Καρδιαγμὸς*, a Hippocratic term, synonymous with Cardialgia) has been applied to this disease by Hirsch.—(*Translator.*)

[2] Piorry, Gazette Hebdomadaire, 1862, No. 30, p. 477. A. Cros, Gazette Hebdom., No. 35, p. 548. Nunneley, Medico-Chirurgical Transactions, vol. xlviii. p. 32.

[3] Dechambre (Gazette Hebdomadaire, 1862, p. 482) quotes an interesting parallel observation by Decès (Thèse inaugural sur l'Aneurysme cirsoïde, 1857), where a transitory exophthalmos appeared in a woman who suffered from alternating arterial dilatations in different parts of the body.

blood effused over the eyeball.[1] If to this be added a fatty degeneration of the muscles of the eye, as Von Recklinghausen detected, we are enabled to understand how so considerable a prominence of the eyeballs occurs, that in fact the eyelids can no longer be closed,[2] and that in the uncovered portion of the eye inflammation may be induced, which, in turn, may lead to a complete destruction of the cornea and wasting of the eyeball.[3] As a rule, the prominence of the eyes is on both sides and also symmetrical; still it does happen that the protrusion is either earliest seen, or, at all events, is more marked in one eye than the other.[4]

For the present it must be left undecided, whether the majority of observers have found Enlargement of the Spleen to be an essential or merely an accidental result. At all events, we cannot on *à priori* grounds lightly estimate the disturbance of the digestive function, more particularly the vomiting and tendency to diarrhœa, which are often observed. There always remain the three intimate symptoms or triad: affection of the Heart, the Thyroid gland and Eyes (Orbital-polsters, orbit cushions), as the regular, although, in relation to each other, not constant phenomena, and it may be asked what the explanation of this combination is. That the lesion of the thyroid gland is not to be considered as the centre or mainspring of this complex disorder, I have already made apparent. Individual observers indicate that the bronchocele may be altogether absent.[5] Still less can we regard the affection of the fatty tissue in the orbit as of principal importance, particularly as the protrusion of the eyes is sometimes wanting, or else it only becomes apparent at a later stage.[6] Neither can it be held that the hypertrophy of the heart is itself to be looked upon as the point of departure. On the one hand, hypertrophy is not always present; and on the other, considerable hypertrophy of the heart often exists without the staring eyes and without the goitre. The anatomical changes of all these elements cannot then be regarded as diagnostic.

We come therefore to the question of the functional disturbances. Here I must specially call attention to the fact, that there exists a peculiar combination of affections of the thyroid gland and heart. This combination, which was formerly mentioned (vol. i. p. 114), is the so-called Iodism, or the goitre cachexia (Kropfcachexie). Here we observe, with the disappearance of the goitre as the consequence of a slight iodism, a most remarkable acceleration of the pulse, not

[1] A. Reith, l. c., p. 521.

[2] Graves, a. a. O., s. 411. Stokes, a. a. O., s. 231.

[3] Casper's Wochenschrift, 1840, No. 14. s. 221. Von Gräfe, Archiv f. Ophthalmologie, 1857, bd. iii. 2, s. 282. Teissier, Gazette Méd. de Lyon, 1863, No. 1, 2.

[4] Von Basedow, a. a. O., 1848, No. 49, s. 772. Henoch und Romberg, Klinische Wahrnehmungen, s. 182. Reith, l. c., p. 521. Prašl, a. a. O., s. 206, 207.

[5] Prašl, a. a. O., s. 209.

[6] Henoch und Romberg, Klinische Wahrnehmungen, s. 179, 180.

unfrequently the production of annoying palpitation. Only the exophthalmos is wanting; instead of it there is another prominent symptom rarely present in the "struma exophthalmica,"[1] to wit, the association of rapid and great emaciation with voracious appetite [mit Bulimie].[2] The point now mentioned, at all times worthy of notice, is so much the more so, from the circumstance that in a case of Oliffe's,[3] the moderate exhibition of iodine in "struma exophthalmica" produced the worst effects. Trousseau[4] himself has made similar observations, and on this account has not hesitated, in the discussion on iodism in the French Academy of Medicine, to regard as cases of "struma exophthalmica" instances which, by Rilliet, had been described under the name of iodism. Rilliet[5] has, on the contrary, in the most decided manner claimed as examples of iodism recorded cases which had been described under the name of "struma exophthalmica." Extended observations are required in order to clear up this dispute. Neither bronchocele nor iodine produce the phenomena of the "cachexia exophthalmica," or of the "cachexia iodica;" in both cases there must, in addition, be the presence of something peculiar. In reference to this, we must go back to an original predisposition; and I may mention that Bednar[6] has repeatedly found in newly born children the co-existence of an enlargement of the thyroid gland and hypertrophy of the heart. Yet these facts, supposing them to possess a general importance, which is unlikely, do not exclude from the inquiry the existence of a further cause.

In the acceptation of the humoral pathology, it is concluded that there is always a blood derangement when several organs are together affected, without there appearing to be a simple dependence of the disease on one or other of these. Von Basedow[7] has forthwith extended this view to the establishment of an independent dyscrasia, which he has expressed as a hidden scrofula. At a later period, he has pointed out this dyscrasia as being similar to the chlorotic.[8] This opinion has been subsequently embraced by many other observers,[9] and Anæmia has become the theoretical foundation of the complex symptoms, while Mackenzie has gone so far as to indicate the condition of the eyes as being neither more nor less than "exophthalmia anæmica." In favour of this view, there is not merely the frequent occurrence of pulsations, palpitations, and

[1] Trousseau, Union Méd., 1860, tome 8, p. 437–456.

[2] Emaciation and bulimia do sometimes co-exist in cases of "struma exophthalmica."—(*Translator.*)

[3] Trousseau, Union Méd., 1860, tome 8, p. 513.

[4] Trousseau, Gazette Hebdom., 1860, Avril, p. 219–267.

[5] Rilliet, Mémoire sur l'Iodisme constitutionnel, Paris, 1860, p. 83.

[6] Bednar, a. a. O., s. 79. [7] Von Basedow, a. a. O. 1840, s. 225.

[8] Von Basedow, in the same place, 1848, s. 772.

[9] L. Gros, Gaz. Med., 1857, p. 232. Hervieux, Union Méd., 1857, No. 117, p. 477. Beau, Gaz. Hebdom., 1862, No. 34, p. 539. Fischer, Arch. Génér., 1859, Dec., p. 671. Begbie, Edin. Med. Jour., 1863, Sept., p. 201. Praël, a. a. O., s. 210.

murmurs in the vascular system, as in chlorotic patients,—not merely the circumstance that the majority of cases of staring eyes with goitre (Glotzaugen-Kropf) are observed in women,[1] and that on several occasions pregnancy and child-bearing have exerted a remarkably favourable influence on the removal of the malady,[2] but also very specially the experience which we possess in relation to the satisfactory operation of an invigorating treatment.

It is, however, undoubted that the Anæmia, granting its existence, cannot directly produce such an effect. At the very least, we must assume that, through the disordered blood, an injurious influence on the nerves takes place. In returning, however, to the nerves, the question arises, whether anæmia is required in order to produce such a condition of the nervous system. Different observers[3] have been content to look upon it as a feeble state of the nervous system (einen Schwächezustand des Nervensystems). Graves and Brück[4] considered it hysterical. Stokes[5] limited himself to pointing out that the essence of the disease consisted in a functional disturbance of the heart, upon which organic change is apt to follow. More recently a further advance has been made, and attention has been directed to the nerves of the heart, and especially to the Sympathetic,[6] with perhaps also participation of the spinal cord.[7] Köben, the first to offer this conjecture, supposed that the sympathetic was compressed and irritated by the bronchocele; since then the bronchocele has justly been regarded as pertaining to the Neurosis. In support of this view, some not unimportant facts in pathological anatomy have been advanced. Peter[8] found the lowest cervical ganglion enlarged and greatly reddened, its interstitial tissue increased, the nerve fibres diminished. Somewhat similar is the account given by Moore[9] of the inquiry conducted by Cruise and M'Donnel. Reith describes the middle and lower cervical ganglion on both sides, especially the left, as enlarged, hard, and firm; and, when viewed under the microscope, filled with a granular material resembling a lymph gland in the first stage of tuberculosis. The trunk of the sympathetic itself, as well as its branches proceeding to the "arteria thyroidea inferior" and "arteria vertebralis," were enlarged. He held these changes to be tubercular. Directly op-

[1] Trousseau, Union Médicale, 1860, t. viii. s. 437. Charcot, Gaz. Med., 1856, Sept., p. 584. Gaz. Hebdom., 1862, Sept., p. 564. Corlieu, Gaz. des Hôpitaux, 1863, p. 125.

[2] Von Basedow (Casper's Wochenschrift, 1848, s. 774) mentions the following remarkable facts—that the mammæ of a man were found greatly enlarged, the left being hard, congested, and painful, yielding colostrum.

[3] Handfield Jones, Med. Times and Gazette, Dec. 1860, p. 541. Fletcher, British Med. Journal, 1863, May. (Hyperneurie.)

[4] Graves, a. a. O., s. 410. A. Th. Brück, Casper's Wochenschrift, 1840, No. 28 (Baphthalmus hystericus), 1848, No. 18, p. 275. [5] Stokes, a. a. O., s. 244.

[6] Köben, De Exophthalmo ac Struma cum Cordis affectione; Diss. inaug., Berol., 1855. Von Gräfe, a. a. O., s. 280. Trousseau, Union Méd., 1860, t. viii. p. 487. Arau, Gaz. Hebdom., No. 49, p. 796. Reith, l. c., p. 522.

[7] Laycock, Edin. Med. Jour., 1863, Feb., p. 681; July, p. 1.

[8] Peter, l. c. p. 182. [9] Moore, Dublin Quar. Jour., 1865, Nov., p. 348.

posed is the observation of Von Recklinghausen, in so far as the cord and ganglia of the sympathetic were small, as if atrophied, but without histological changes. All now stated was undoubtedly insufficient to explain the real nature of this interesting affection, especially as the appearances of the "struma exophthalmica"—if we appeal to the familiar physiological experiments of Cl. Bernard—correspond in part to the paralysis, in part merely to the irritation of the sympathetic; while, again, there appears to be no true connexion, as some of these constant phenomena in the pupil have not been noticed.[1] Only in isolated cases has enlargement of the pupil been observed. Stromeyer[2] compares the "exophthalmia strumosa" with the temporary incomplete prominence of the globe, which he had noticed in connexion with habitual spasm of the head (Krampfe des Kopfnickers). When this cramp occurs either from maintaining the erect posture, or from mental emotion, he seeks the foundation of the staring eyes in spasm of the oblique muscles of the eye, and the levator muscles of the eyelids. Demme[3] has also frequently observed with the ordinary goitre partial changes in the pupils, especially "Mydriasis," and a notable elevation of the upper lids. He gives as an anatomical condition at the same time (besides serous swelling and interstitial connective tissue in the recurrent nerve) marked reddening and serous swelling of the sympathetic. These statements, however, suffice just as little as the older descriptions of different changes in the vagus connected with the goitrous condition. Certainly the direction of the inquiry turns, even as in the question of the connexion between disease of the supra-renal capsules, bronze-skin,[4] and other cases, more and more to the nerves themselves; while there still exists too little material for enabling us to arrive at a decision.[5] After all, the question resolves itself not so much into an examination of the cases in their later stages, which can be cleared up by autopsies, but into an investigation of the earliest determining cause.

At least, there can no longer be any hesitation in acknowledging the intimate nervous dependence of the complex symptoms as the

[1] Henoch und Romberg, Klin. Wahrnehmungen, s. 182. Reith, l. c., p. 251.
[2] Stromeyer, Handbuch der Chirurgie, ii. 2, s. 389.
[3] H. Demme, Würzb. Med. Zeitschrift, bd. iii., s. 269, 273, 297.
[4] *Bronzing of the skin* occurs in connexion with "struma exophthalmica." It exists in a marked degree on the face of a patient (a man) of Dr Begbie's presently under observation.—(*Translator.*)
[5] Only very recently there died, in my division, a man who had long suffered from very violent palpitation of the heart, with great dyspnœa. His eyes, without being precisely exophthalmic, had an unusual glare (glanz), and gave the impression of being increased in size. A few months previously, Herr Von Gräfe, on account of a commencing glaucoma, had performed iridectomy. Near the close he was affected by dropsy, with very diminished secretion of urine, which was albuminous and rich in uric acid; also with obstinate and violent pain, associated with bloody diarrhœa, great restlessness and fever, with other symptoms. On post-mortem examination I found hypertrophy of the heart, with very extensive myocarditis, a goitre, and very considerable enlargement and interstitial thickening of the sympathetic in the neck, especially of the uppermost and lowest ganglion.

only probable view. With justice has reference been made to the existence of great derangement of the general nervous system, to the loss of sleep, the often noticed epigastric pulsation, the sensation of heat,[1] and, lastly, to a macular eruption occurring on the head after a slight mechanical irritation.[2] In what particular portion of the nervous system the original seat of the disturbance, and what the disturbance itself is, that must first be more accurately established; and the inquiry must be made also, whence, from what source (whether from the blood) the disturbance has been developed. At all events, it is a step in advance to become acquainted with these complex symptoms, and the jeers of M. Piorry are unable to deter us from acknowledging the entity of the disease.[3] In the history of goitre, it forms an episode as remarkable as it is important; for although this variety of bronchocele seems of itself to have little importance, yet it constitutes a part of a grave malady, and one not unfrequently fatal, although, in other circumstances, readily curable.

As to the ætiology, there remains little more to be said. According to the observations hitherto advanced, it is by no means in goitre-districts of country that this variety has frequently occurred. Still more does the "struma exophthalmica" plainly constitute one of the most important species of sporadic goitre. Females in a greatly preponderating degree suffer,[4] more so in the early period of life, especially about puberty, and in childbed. Uterine derangements act by no means always as exciting causes; and while serious diseases, such as typhus, and colds particularly affecting the throat, exert an influence, chlorosis is chiefly to blame. Since the latter disease, according to my understanding, is one of early life—is even a disease of development[5]—we are led to assume the existence of an original predisposition. Romberg and Juncken[6] observed the disease in two sisters.[7] We are still further removed from understanding the cause of the disease in men. Exhausting labour, great and long continued depression of the mind, and weakening diseases, have at times preceded it. According to the comparison instituted by Von Gräfe,[8] the disease appears at a later period, on the

[1] Von Basedow, Casper's Wochenschrift, 1840, No. 13, s. 202; No. 14, s. 220. Teissier, Gaz. Méd. de Lyon, 1862, No. 29; 1863, Nos. 1, 2. Trousseau, Gaz. Méd., 1864, No. 12, p. 180. Warburton Begbie, Edin. Med. Jour., 1863, Sept., p. 216. [2] Trousseau, Gaz. Méd., 1864, No. 12, p. 180.
[3] Piorry, Gaz. Hebdom., 1862, p. 477.
[4] It is not without interest that Rorie (Edin. Med. Jour., 1863, Feb., p. 696) frequently found prominence of the eyeballs in persons of weak intellect, and this particularly in women (35 per cent.); inequality of the pupils, also, he noticed not unfrequently. Foderé remarks, concerning Cretins:—"Aux uns les yeux sont enfoncés dans la tête, aux autres ils sont très en dehors. En général leur régard est fixe et égaré, et il y a toujours un air d'étonnement."
[5] Virchow, Cellular pathologie (3d edition), s. 211.
[6] Henoch, Casper's Wochenschrift, 1848, No. 40, s. 627.
[7] Dr Begbie informs me that, quite recently, he has observed the disease in two sisters, both married. In both the malady had assumed its unequivocal characters.—(*Translator.*) [8] Von Gräfe, a. a. O., s. 292.

average, in men, but, at the same time, is more serious. It is worthy of remark that, not unfrequently, the commencement of the malady has been noticed to be quite sudden, for example, after a fright or hard labour.

Death results with an increase of the appearances, sometimes very quickly, accompanied by great uneasiness and disturbance of the brain, mostly in a gradual manner, with decay of nutrition and strength, which is hastened by urgent diarrhœa, sometimes dysenteric in character, and mucous catarrh of the lungs. At another time, on the other hand, chiefly in recent cases, a complete cure results; the goitre, however, it is true, not always entirely disappearing. Sometimes the preparations of iron have effected this, sometimes digitalis,—it is seldom that iodine is useful. The best consequences have succeeded the employment of cold-water treatment, sea-bathing, and an invigorating diet.[1]

Observations by Translator.—It is necessary, in the first place, to correct an error into which Virchow has fallen when offering the interesting historical summary regarding "Struma Exophthalmica," with which his observations commence. Parry, he remarks, appears to have been the earliest to observe the prominence of the eyes. The fact is, however, that Parry noticed the enlargement of the thyroid gland in connexion with disease of the heart, but in the whole course of his statement regarding that connexion there is only a single, and that evidently casual, reference to the condition of the eyes. Unquestionably the earliest observer in our own country of the peculiar affection of the eyes, believed by him, at the time, to be a real enlargement, was Dr Stokes, and next in order to him was the late Sir Henry Marsh, of Dublin. Antecedent to 1835, the date of his first publication on the subject, Dr Graves had incidentally had his attention directed to the coincidence of cardiac disease and enlargement of the thyroid gland. In 1839, Dr Begbie had evidently, in a manner altogether independent, noted the association of the three peculiar symptoms. During the succeeding ten years other instances of the kind occurred to him in practice, and, in 1849, he published an account of them. In doing so, Dr Begbie was the earliest in this country to assert the entity of the disorder, to advance a theory of its cause, namely, its dependence on anæmia, to indicate its amenability to treatment, and capability of perfect cure, and, lastly, to suggest a plan of treatment, the success of which has, happily in many instances since

[1] I may take the opportunity of directing attention to a brief but very interesting account of the disease by a physician of Heidelberg, Dr Theodor Von Dusch, in his recently published volume, entitled "Lehrbuch der Herzkrankheiten." Leipzig, 1868.

Dr Von Dusch's observations on the "Basedow'sche Krankheit" are illustrated by a woodcut representing an example of double exophthalmos, but without goitre, in a man of thirty-two years. The portrait, in the first instance, was photographed from nature.—(*Translator.*)

that time, been most satisfactorily proved. Five years subsequently to the incidental observation by Dr Graves, of enlargement of the thyroid gland with disease of the heart, a German physician, Von Basedow, published a paper, in which terms now sufficiently familiar in connexion with the disease, were for the first time employed. For example, "Exophthalmos," "Cachexia exophthalmica," and, in the German, "Glotzaugen" (staring eyes), "Glotzaugenkachexie" (staring-eye or goggle-eye cachexia). In these phrases it will be noticed that no particular reference is made to the condition of the thyroid gland, which, in connexion with heart enlargement, had already attracted the attention of Parry and others; but Von Basedow was familiar with the bronchocele as well, and accordingly, in consideration of the correctness of his observation so far, there can be no objection, as has been proposed by Hirsch, and followed by German physicians generally —although scarcely receiving the sanction of Virchow's high authority—to designate the disease "Basedow's disease" ("Basedow'sche Krankheit," "Morbus Basedowii"). The late distinguished and lamented physician of the "Hôtel Dieu," whose unbounded admiration for the character and writings of Dr Graves is as well known as it is highly appreciated by all readers of the "Clinical Lectures," has styled the disease, "Graves' disease" ("Maladie de Graves"), and whether rightly or wrongly so—Virchow, as we have seen, thinks wrongly—there can be no doubt that under this name, as originally employed by Trousseau, it will long be familiarly known and described. Were it not that I entirely agree with the late Dr Todd, of London, in regarding it as no compliment to the great names of our profession "to attach them to any of the numerous ills which flesh is heir to,"[1] I should feel disposed to suggest the appellation of "Stokes' disease." Such, too, is a sufficient reason for not encouraging the use of that name which my filial respect had otherwise most cordially approved, to wit, "Begbie's disease," as already proposed by more than one writer. There is little to be said in favour of the other nomenclature adverted to in the translation, the "Tachycardia strumosa," or "Tachycardia exophthalmica strumosa," of Lebert, and the "Cardiagmus strumosus" of Hirsch and Von Dusch; and it must be confessed that a really good and serviceable title for the disease is still a desideratum.

Like other observers, it is worthy of note, that Virchow indicates no special form of disease as incident to the thyroid gland in this complex disorder. The most marked or characteristic feature is its increased vascularity, and the peculiar pulsation, or pulsatory thrill, which is, at all events, distinguishable over it. Hence the application to this variety of goitre of the terms "vascular bronchocele" and "struma aneurysmatica." While increase of gland

[1] In referring to facial palsy as "Bell's paralysis," in Clinical Lectures on Diseases of the Nervous System, Lecture 4.

structure, and various kinds of degeneration, as, for example, the fibrous and cystic, have been detected in such goitres, there is no doubt whatever that the augmented activity and chronic enlargement of the vessels, particularly the veins in them, is of chief importance.

The central organ of the circulation invariably and, in point of time primarily, suffers. At first, however, and usually for a lengthened period, the heart is only functionally disturbed. Of this disturbance its greatly accelerated action is the prime, as it is the unmistakable, indication. After a time, structural change ensues, and that is, for the most part, of the nature of dilatation, or hypertrophy with dilatation. The left ventricle is the chamber chiefly affected. Valvular disease of the heart is certainly rare; when it exists, the imperfection is secondary, never primary. In other words, the increased size of the mitral and tricuspid orifices, discovered on the post-mortem examination of some cases of the disease, bore an intimate relationship to the greatly augmented capacity of both ventricles. This, the earlier result, was evidently the cause on which the insufficiency of the auriculo-ventricular valves depended.[1]

In the great majority of cases, a disturbed action of the heart, generally spoken of as palpitation, is the *first* symptom to attract attention. If, however, a very careful inquiry into the previous history of such patients be made, it will be found that for some time before the distressing action of the heart was noticed—possibly at a considerable period antecedent to the acknowledged existence of any departure from health—there had occurred a diarrhœa or lientery, a menorrhagia or leucorrhœa, an epistaxis, rather a frequently recurring loss of blood from the nose, or a hæmorrhage from piles; and although, in many cases, none of these may have been sufficient to produce the more manifest indications of anæmia, it will, I firmly believe, be further found that impoverishment of blood—readily enough recognisable—exists.

Virchow, like all recent observers of this disease, rejects the notion of the prominence of the eyes being due to "hydrophthalmos." He looks upon a change in the fatty tissue of the orbit—a view originally maintained by Heusinger—as being the essential morbid condition upon which the very strange aspect of the eyes depends. An hypertrophy of the fatty tissue, or its congestion, are the states more particularly indicated. Enlargement of the spleen is referred to by Virchow as having been noticed by some observers, but he truly remarks that the degree of importance to be attached to its occurrence has not been as yet accurately determined.

There are then, it may be stated, *four* essential symptoms in this most interesting malady — namely, 1. Disturbance of the Heart's action, prone to terminate in structural change; 2. Enlargement—vascular in its nature—of the Thyroid gland; 3. Prominence, with

[1] See, for example, the case of J. K., as recorded by Dr Begbie, in "Contributions to Practical Medicine," pages 143, 148.

peculiar expression, of the Eyes; and, 4. Remarkable visible pulsation and vibratory thrill throughout the whole Arterial system. Of this "tetrad" of symptoms, the first and fourth are always present; the second and third may each be absent. Without cardiac and vascular derangement, the disease has no existence. An exquisite illustration of the malady, however, presumes the presence of all the features now mentioned.

In discussing the essential pathology of "struma exophthalmica," Virchow has justly observed that anæmia, granting its existence, cannot directly produce the results which are witnessed. While the "primum mobile" is, however, seated in the blood, it is abundantly evident that an injurious influence is largely exerted on the nerves. A decided advance in our knowledge on this point has recently been made, for Peter, Reith, and Von Recklinghausen have each discovered a distinct lesion of the sympathetic nerve and its ganglia, while Virchow himself so far confirms their interesting and probably important observations as to have found in a case not exactly of the "struma exophthalmica," but bearing a certain resemblance to it, a lesion of the same nervous trunk.

Further observations are, however, required in order to determine, with any amount of accuracy, what value is to be attached to these morbid appearances in relation to the intimate pathology of that strange disease with which they have been found connected.[1]

It cannot be too distinctly stated, nor too carefully borne in remembrance, that, in this disease, *iodine* is an unsuitable remedy —its administration, so valuable in ordinary goitre, is in the "struma exophthalmica" not only useless, but injurious. From *iron*, *digitalis*, and *belladonna*[2] discriminately employed, and from the steady perseverance with an invigorating plan of treatment, the best effects have been found to follow.

[1] I have observed a decided feebleness in the lower extremities, almost amounting to paraplegia, in two or three aggravated cases of the disease. This also points to the implication of the nervous system. In two of these instances, the patients walked with considerable difficulty, and had acquired a peculiar rotatory movement in progression.

[2] On Vascular Bronchocele and Exophthalmos, by the translator; Edinburgh Medical Journal, 1863, page 217.

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ON SOME

ANALOGIES OF CHOLERA,

IN WHICH

SUPPRESSION OF URINE IS NOT ACCOMPANIED

BY SYMPTOMS OF URÆMIC POISONING.

BY

WILLIAM SEDGWICK, M.R.C.S., L.S.A.

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The indirect aid afforded by analogy in the investigation of disease may be said to correspond with that of a reflecting mirror, in which there is presented a more or less clearly defined image of what would otherwise be obscure or hid; and, therefore, as an assistance towards lessening the difficulty which attends the investigation of cholera, which, although uniformly well marked as regards its symptoms, is exceptionally obscure as regards its nature, analogy, in so far as it enables us to trace out and establish a resemblance between it and other morbid conditions of the system, which admit of being more readily explained, will be useful, provided the principle be sound, on which the analogical reasoning is based. For it has become a necessary and, in some respects, an unpleasant duty to direct attention to the fact that, in the investigation of this as of other baffling diseases, attempts have on many occasions been made to establish false analogies in support of unfounded theories of the disease, or of ill-

devised plans of treatment, which, after attracting popular attention, have for the most part been soon slighted and forgotten. It may, moreover, be remarked that in some cases the attempt to establish an analogy, even if successful, would not be attended with any useful result, for with respect to our knowledge of the nature of the disease, it would be but the substitution of one difficulty for another; as, for example, when cholera is compared with tetanus, or alleged to be an epilepsy of the organic nervous system, or the cold stage of an incomplete attack of ague. In like manner the analogies which have been urged in support of some of the proposed remedies for the disease are equally defective in the information they are intended to convey, as when cholera is compared with sea-sickness, in consequence of its having been assumed that both admit of being prevented and cured by ice to the spine; or when, in support of the castor-oil or eliminative method of treatment, cholera is compared with smallpox. Even the term eliminative, as used in this latter case, is objectionable, since there is nothing in common between the two processes by which the characteristic discharge in smallpox is excluded from the system by the skin, or the characteristic discharge in cholera by the gastro-intestinal mucous membrane; and if, in support of this theory of so-called elimination, and apart from any preconceived theory of treatment, it were desirable, in consequence of the sympathetic connection which exists between the skin and the alimentary mucous membrane, to establish an analogy between cholera and some disease characterised by abnormal and excessive discharge from the skin, the most appropriate for that purpose would decidedly be the sudor anglicus, or sweating sickness of the Middle Ages, which has apparently prevailed though to a less noticeable extent in modern times, and which Dr. Roupell[1] has referred to as approaching nearer than any other epidemic disease to cholera. But it must be confessed that inquiry even in this direction, notwithstanding the parallelism presented by the profuse and characteristic discharges, could not be expected to lead to

[1] 'On Cholera' (the Croonian Lectures for 1833), p. 72.

any useful result, in consequence of the sudor anglicus being quite as great if not a greater pathological mystery than cholera itself. In order, therefore, to avoid the errors above referred to, and to check the ever-recurring tendency, in a discussion of this kind, to introduce assumption in the place of fact, it is proposed in the following attempts to trace an analogy between cholera and allied but less obscure affections of the system, to limit the inquiry to a comparison between the well-defined and characteristic group of symptoms and pathological changes which accompany the collapse of this disease, with special reference to suppression of urine during life, and an empty and contracted state of the bladder after death, and such cases of collapse resulting either from poison or from disease, in which a corresponding condition has been developed and observed.

It is a noteworthy fact that when cholera first attracted general attention, and before the public mind had become familiarised with the peculiar and fatal character of the disease, it was very commonly ascribed to irritant poisoning, and popular tumults were in many places the result of this belief. It is of some importance with reference to the present inquiry to remark that this now almost forgotten but once widespread delusion was suggested by the analogy which prevails between the chief symptoms of cholera and many of those poisons, the effects of which are more or less commonly known; and that, like other errors of a similar kind, the hasty and imperfect reasoning on which it was based contained, as it were, the germs of truth. For if we proceed to analyse the earliest of the analogies of cholera, it will be found that most of the symptoms which are met with in the collapsed stage of the disease occur also in the case of certain poisons, the action of which is essentially referable to the gastro-intestinal mucous membrane; and it may in consequence be not unreasonable to expect that if the inquiry be pursued in this direction, it will furnish us with a clue to the primary seat of the disease. In addition, however, to exciting early and popular attention, this analogy between cholera and cases of irritant poisoning seems to

have specially attracted the notice of many scientific observers in subsequent epidemics of the disease, by whom the importance of urging the inquiry in this direction has been fully recognised. In an able article on "Diseases which may simulate Poisoning," Devergie[1] assigns the first place to cholera, and mentions suppression of urine as one of the symptoms common to the collapse of both. Tardieu[2] has also published numerous illustrations in support of this statement. And among other observers whose evidence might be cited on this occasion is Dr. Horace Jeaffreson, who has had favorable opportunities for studying cholera at the London Hospital during the outbreak of the disease in the eastern districts of London in 1866; and who, in a lately published and instructive paper "On the Pathology of Cholera Collapse"[3] has justly remarked, that "inquiry in this direction will give us some remarkable analogies."

In this preliminary stage of the investigation it will be sufficient to state that the cases in which such an analogy can be more or less clearly established, are acute poisoning by corrosive sublimate, by arsenic, and by mineral acids, especially nitric acid; the effects which follow the eating or drinking of poisonous animal matters, such as tainted or simply unwholesome meat or fish, and milk which has undergone some injurious but as yet unknown change, decomposing vegetables and some of the poisonous fungi; and the excessive action of certain drugs, for the most part belonging to the class of drastic purgatives, especially croton oil. After a brief consideration of some of the more important cases of this description, in which the collapse resulting from the gastro-intestinal action of an irritant poison has been noted as closely analogous to that of cholera, attention will be directed to a well-defined group of cases in which the symptoms from disease referrible to the same part of the system so far simulate choleraic collapse, as to have been occasionally and almost unavoidably mistaken for it.

[1] 'Médecine Légale,' 3e édit., tom. iii, chap. xxi, 1852.
[2] 'Etude Médico-Légale et Clinique sur l'Empoisonnement,' 1867.
[3] 'Edinburgh Medical Journal,' Dec., 1866, pp. 520-538.

In cases of poisoning by corrosive sublimate, both the symptoms during life and the appearances after death are in many important particulars, and especially as regards the urinary secretion, closely analogous to those of cholera. This is well shown in the report of a case recorded by the late Dr. William Henry,[1] of Manchester, in which a woman, aged twenty years, died from the effects of this poison on the sixth day, in which it is mentioned that "no urine was voided after the third day; and on introducing the catheter repeatedly, the bladder was found empty;" and that after death "the bladder was empty and exceedingly contracted." In five cases in one family, recorded by the late Mr. J. W. Valentine,[2] of Bolsover, in which a mother and three children died, and one child recovered from poisoning by corrosive sublimate, suppression of urine was in like manner a characteristic symptom in the collapse, which in other respects also closely resembled that of cholera. In two of the fatal cases, the urinary bladder was contracted to "the size of a walnut;" in a third case, it was of very small size, and empty; and in a fourth case it was found "closely contracted to the size of a marble." The child who recovered was a girl, aged fourteen years, who swallowed only a small portion of the poison, and had suppression of urine for three days. Dr. Robert Venables[3] has recorded a case of poisoning by corrosive sublimate, in which a servant girl died from the effects of the poison on the eighth day. In this case it was specially noted that "there was a total and permanent suppression of urine; and that there might be no room for doubt upon this subject afterwards, Mr. Marten, at my suggestion (adds Dr. Venables), introduced the catheter, but no urine flowed." After death "the bladder was (found) perfectly empty, and very much contracted in size." In a case of poisoning with corrosive sublimate, reported by Mr. Archibald Blacklock,[4] of Dumfries, in which a man, aged

[1] 'Edinburgh Medical and Surgical Journal,' vol. vii, 2nd edit., pp. 150, 151, 1811.
[2] Ibid., vol. xiv, pp. 468-474, 1818.
[3] 'London Medical Gazette,' vol. viii, pp. 616-623, 1831.
[4] 'Edinburgh Medical and Surgical Journal,' vol. xxxvi, pp. 92-4, 1831.

fifty years, died on the fifth day; the urine was suppressed from the time the poison was taken, and we are informed that there was no inclination to pass it even when at stool. The pulse in this, as in many other cases recorded, was almost imperceptible for some hours before death. In Mr. (now Sir James) Syme's 'Clinical Report for the Winter Session 1834-5,'[1] there is the case of a man named James Maxwell, aged thirty-five years, who suffered severely, but did not die from the poisonous effects of mercury externally applied. In this case the urine was suppressed for five days, in consequence of which "the catheter was introduced repeatedly without allowing any to escape, except two or three teaspoonfuls of mucous-looking fluid destitute of urinous smell." * * * "The patient drank freely during the suppression of urine; he was quite sensible; and lay quiet without any disposition to coma." In commenting on this case, Mr. Syme remarks that it "is interesting on several accounts. In the *first* place, it affords an example of the suppression of urine, which results from poisonous doses of the oxymuriate of mercury given internally, occurring from the external application of a mercurial salt; *secondly*, the suppression of urine was not accompanied with coma; and *thirdly*, the patient recovered after suffering complete suppression of urine for five days." In a case of acute poisoning by corrosive sublimate, which was under observation in Guy's Hospital in February, 1843, it is recorded that there was "complete suppression of urine, and symptoms closely resembling those of cholera." * * * The patient lived four days, and did not pass any urine during the whole of this time;" and after death "the bladder was empty and contracted."[2] According, also, to Dr. Copland,[3] it appears that in this form of poisoning there is suppression of urine or dysuria during life, and that after death "contraction of the urinary bladder is always observed." Ample evidence has, moreover, been cited by Dr. Taylor[4] to show that suppression of urine, which, when unaccompanied by head-symptoms, has been thought to be almost peculiar to choleraic collapse, is a frequent result of poisoning by corrosive sublimate, both in those cases in which the poison has been swallowed, and in those in which it has been externally applied.

[1] 'Edinburgh Medical and Surgical Journal,' vol. xliv, pp. 26, 27, 1835.
[2] 'Guy's Hospital Reports,' vol. ii, pp. 24-27, 1844.
[3] 'Dictionary of Practical Medicine,' vol. iii, part i, pp. 349, 350.
[4] 'On Poisons,' 2nd edit., pp. 446-459, 1859.

Among continental observers whose authority might be cited on this subject, is Casper,[1] who has specially noted that in cases of poisoning by corrosive sublimate, there is "suppression of urine;" and after death he has also found "the urinary bladder small and contracted." In like manner, Flandin[2] includes among the signs of poisoning by mercury "scantiness or total suppression of urine;" and in his summary of the comparative effects of mercury and arsenic (in poisonous doses) on the animal economy, which present, as he justly remarks, "great analogies," adds that "arsenic, in paralysing nervous action, diminishes or suspends the secretion and the emission of urine. Mercury produces the same effect; but, moreover, it irritates the bladder and urethra, and gives rise to fictitious and painful efforts to pass water."[3] In one of the cases recorded by this observer,[4] in which a young girl died from poisoning by corrosive sublimate on the third day, there was complete and prolonged suppression of urine, which was demonstrated by passing a catheter on the first, and again at the end of the second day, on both of which occasions no urine flowed. In another case recorded by the same observer,[5] in which a lad, aged fifteen years, died five days and six hours after poisoning by corrosive sublimate, it was ascertained that "the urinary secretion, during all this time, was suspended," and the bladder after death was found to be contracted. Lastly, the evidence which has been published on this subject by Tardieu[6]

[1] 'A Handbook of the Practice of Forensic Medicine,' Sydenham Society, vol. ii, p. 62, 1862.
[2] 'Traité des Poisons,' tom. ii, p. 140, 1853.
[3] Ibid., p. 113.
[4] Ibid., pp. 114-116.
[5] Ibid., pp. 116, 117.
[6] Op. cit., 1867. See Obs. 2, p. 596; Obs. 4, p. 597; Obs. 5, p. 598; and Obs. 8, p. 605.

may be referred to as very conclusive in favour of the collapse from poisoning by corrosive sublimate being associated with suppression of urine, and other symptoms similar to those of cholera.

It is important, moreover, to notice that although in the preceding cases there was suppression of urine during collapse from this poison in the human subject, yet the opposite condition of a copious and free discharge of urine has been observed to result from corrosive sublimate given in large quantities to horses. This apparent discrepancy in the effect of the poison on the urinary secretion in men[1] and horses is not due, as perhaps might be hastily assumed, to difference in the composition of their urine, but chiefly, if not altogether, to the fact (which it would be useful to consider in connection with the comparative influence of epidemic cholera on man and the lower animals) that corrosive sublimate has no irritant effect on the stomach of the horse corresponding with what has been observed in man; and that it does not produce any analogous collapse. This has been satisfactorily established by the experiments of the late Mr. Stevenson,[2] veterinary surgeon, of Norwich, who found that considerable quantities of corrosive sublimate might be given with impunity to a horse; and that in this animal neither disorganization nor inflammation of the coats of the stomach

[1] In connection with this subject it should be mentioned that in a case observed by Mr. John Ward, of the Royal Cornwall Militia, in which two brothers were poisoned by the external application of corrosive sublimate, in one of them, aged 24 years, death occurred on the eleventh day, and it is stated that he passed urine freely on different occasions during the illness; but there was no opportunity of knowing what was the condition of the gastro-intestinal mucous membrane, as no examination of his body after death was made. In the other brother, aged 19 years, death occurred on the fifth day; the symptoms of collapse were more acute than in the former case, and throughout the illness the urine was completely suppressed, none being obtained on passing a catheter. After death the stomach was found highly inflamed and ulcerated, and the bladder was "healthy in appearance, but did not contain the least drop of urine."—('London Medical Gazette,' vol. iii, pp. 666-667, 1829.)

[2] 'Edinburgh Medical and Surgical Journal,' vol. v, pp. 254-256, 1809.

resulted from repeated doses, varying from two drachms to two ounces, of the poison.

In cases of poisoning by arsenic, as in the preceding class of cases, it has been observed that, in addition to the more common results of poisoning, there are others which sometimes closely assimilate its effects to cholera; in consequence of which Devergie[1] refers to it as "one of the poisons which best simulates cholera;" whilst Tardieu[2] emphatically states that of the spontaneous diseases most closely allied with arsenical poisoning, "the first of all is incontestably cholera." Among the symptoms and pathological appearances which have been noticed in some of these cases, and to which more especially attention should be directed, are suppression of urine, cyanosis, conjunctival injection, and a physical alteration in the condition of the blood closely analogous to that which occurs in cholera; together with such a failure of the circulation that, occasionally even for some hours before death, neither pulse nor beating of the heart can be perceived: in addition to which Tardieu[3] mentions that there is sometimes "a serous and white diarrhœa." In a case of this description, reported by Dr. Wilks,[4] the patient was a man, æt. 50, who was admitted into Guy's Hospital on May 3rd, 1855, and who died three hours and a half after admission, and nine hours after taking the poison. The symptoms throughout closely resembled those of cholera; the urine was suppressed, and after death the bladder was observed to be firmly contracted, and it contained only two drachms of opaque urine. Dr. Wilks, in commenting on this case, remarks, "it would have required more than ordinary observation and skill at such a time, and in the absence of a history, to have avoided a suspicion that the patient was not the subject of this disease (cholera). The collapse, the pulselessness, the coldness of the surface, the lividity, the restlessness, the violent cramps, and, moreover, the character of the stools (which might have passed well for choleraic after the ad-

[1] Op. cit., p. 774.
[2] Op. cit., p. 337.
[3] Op. cit., p. 328.
[4] 'Guy's Hospital Reports,' 3rd ser., vol. i, p. 364, 1855.

ministration of the eggs) all closely resembled the symptoms of that malady. The post-mortem examination also further carried out the similarity, and probably would have confirmed the diagnosis of cholera, if it had been superficially made, and the stomach had not been examined as is sometimes the case." Dr. Wilks in a note informs us that "during the epidemic of 1849, a practitioner in the neighbourhood of the hospital mistook, during the life of the patient, a case of arsenical poisoning for one of cholera, and his faulty diagnosis was severely animadverted upon by the coroner's jury." Tardieu,[1] in a paper on cases of death from natural causes which may be attributed to poisoning, mentions a mistake of an opposite character to that referred to by Dr. Wilks, in which a case of cholera, occurring in 1849, was at first assumed to be a case of suicide by arsenical (Schweinfurt's) green. A well-marked suicidal case of arsenical poisoning simulating cholera has been recorded by M. Flandin,[2] in which a prisoner on hearing his condemnation to death, swallowed twelve grammes of arsenious acid, and died in thirteen hours. There was immediate and violent vomiting and pain; and within little more than two hours after swallowing the poison the pulse was imperceptible, not the least movement of the artery being detected, and not the slightest beating of the heart could be felt on applying the hand to the præcordial region. The surface of the body became icy and cyanosed; whilst the intellectual faculties, up to the last hour, remained unimpaired. With regard to the urinary secretion it was noted that the patient, in the course of the illness, experienced a desire to pass water, but without the ability to satisfy it; and when the catheter was passed only a few spoonfuls of *clear urine* were obtained. After death the stomach was found to be in a state of complete disorganization; the bladder contained about a "glass of urine," which presented no special character; and it was moreover remarked that all the divisions of the pulmonary artery were filled with incoagulable blood, whilst, on the contrary, the

[1] 'Annales d'Hygiène Publique,' 2e sér., tom. ii, pp. 150-178, 1854.
[2] Op. cit., tom. i, pp. 494-501.

pulmonary veins were nearly empty. The same observer has recorded a case[1] of arsenical poisoning through a wound in the skin, in which the patient died seven days after the application of the poison. The intelligence in this case was preserved up to the last moment of life; the conjunctivæ were injected with blood; and the urinary secretion was very scanty. After death the bladder was found to be contracted and empty; and "the lungs, collapsed above and behind, presented hypostatic sanguine congestion at their base," similar to what is usually observed after death from choleraic collapse. In another case of "acute poisoning by cutaneous absorption" of arsenic, recorded by Flandin,[2] it was noted on the day following the poisoning that the urine, "since the beginning of the illness, was totally suppressed;" and after death, which occurred on the sixth day, the bladder was found to be empty; it is not, however, distinctly stated that the suppression of urine continued during the whole of the six days. The chief effects of the poison absorbed in this case appear to have been on the alimentary canal at the junction of the duodenum with the jejunum, where the ulceration had led to perforation. Among other cases of poisoning by arsenic to which reference might with advantage be made, are Casper's case No. 185,[3] in which death occurred in twenty-four hours, and the urinary bladder was found to be empty; M. Delsol's[4] case, in which there was marked cyanosis as well as suppression of urine; and some cases recorded by Tardieu,[5] including that of the duc de Praslin.

With reference to the suppression of urine, which has been noted in the preceding and in other cases of arsenical poisoning, it is necessary to state that very opposite opinions have been published on the subject; for whilst Devergie[6] mentions "complete suppression of the emission of urine," as one of

[1] Op. cit., tom. i, pp. 545-551.
[2] Ibid., pp. 502-509.
[3] Op. cit., pp. 72, 73.
[4] 'Bull. de la Soc. Anatom. de Paris,' 2e série, tom. vii, pp. 362-364, 1862.
[5] Op. cit., 1867, pp. 379-380, 384-385, and 385-401.
[6] Op. cit., tom. iii, pp. 505 and 552-553.

the symptoms produced by this poison; and especially refers to the re-establishment of the urinary secretion as the turning point in favour of recovery in the case of a woman whose appearance had previously been such that "one would have said she had been attacked with most violent cholera;" and, in like manner, Flandin,[1] supported by the evidence of many other observers, includes suppression of urine "among the general physiological effects" of poisoning by arsenic, and further remarks that it has been noted at all times and by all observers: on the other side, Orfila stated at the celebrated trial of Madame Lefarge that the flow of urine in these cases is absolutely free;[2] and Dr. Taylor[3] has remarked that, "with respect to the urinary secretion (in cases of poisoning by arsenic) there is no certain rule; it is sometimes suppressed, as in several cases reported by Flandin; at other times it is natural or only slightly diminished." This apparently conflicting testimony admits of being to some extent if not altogether accounted for by the fact that arsenic has a two-fold action on the living body, and produces, as Dr. Christison[4] assures us is now generally acknowledged, two classes of phenomena. One action, which is local and purely irritant, affects chiefly that portion of the alimentary mucous membrane which corresponds with what appears to be the primary seat of disease in cholera; and this action is manifested both in those cases in which the poison is swallowed and consequently applied directly to the mucous membrane, and in those in which it enters the system through the skin.

[1] Op. cit., tom. i, p. 514.

[2] In the latest publication of Orfila "On Poisoning by Arsenic" ('Dictionnaire Encyclopédique des Sciences Medicales,' tom. vi, p. 224, 1867), he informs us that there have been more frequently than otherwise noted "disorders which are observed in the algide period of cholera, smallness and irregularity of the pulse, coldness of the skin, prostration, cramps, cyanosis;" and, with regard to the urinary secretion, that "the emission of urine is in general suspended, but the secretion is only diminished, and, save in exceptional circumstances, it is possible to excite the urinary secretion by means of diuretic drinks."

[3] 'On Poisons,' 2nd edit., p. 359, 1859.

[4] 'A Treatise on Poisons,' 4th edit., p. 288, 1845.

The other action is of a more complex character, producing results which are to some extent secondary and remote; for whilst a state of collapse resembling that of cholera is caused by the irritant effects of the poison on the abdominal nervous system, injurious effects are produced on organs remote from the primary seat of mischief, through the irritant effects of the poison on the circulating blood. In the one case the irritant action of arsenic is circumscribed, and, in affecting chiefly that portion of the alimentary mucous membrane which corresponds with what may with great probability be assumed to be the primary seat of disease in cholera, produces, through the influence of the abdominal nervous system, a condition analogous to choleraic collapse; in the other case, through its presence in the blood, secondary and remote effects are produced beyond those which are developed in that disease.

The recognised difference in the action of arsenic on the animal economy may perhaps to some extent account for the contradictory character of some of the experimental evidence derived from the effects of arsenic on horses and dogs; although part of the difference which has been observed in these cases may possibly be due to arsenic, like corrosive sublimate, not having the same poisonous influence on man and the lower animals. For M. Delafont[1] in his elaborate memoir, in answer to the question "Is the urinary secretion suppressed in acute poisoning by arsenious acid?" which was read at the French Academy of Medicine, on July 5th, 1842, states, as the results of twelve experiments with arsenic on horses and dogs, that "in none of the animals submitted to the experiment has the secretion of urine been suppressed." At the same time it was observed that the urine passed in all these cases of fatal poisoning contained arsenic.

Besides the influence of this poison on the secretion of urine, the effects of arsenic on the vascular system and the blood afford further corroborative evidence in favour of the

[1] 'Gazette Médicale de Paris,' 2e sér., tom. x, p. 446, 1842.

analogy with cholera. Casper[1] has remarked that "the blood generally, in acute arsenical poisoning, is deficient in coagulating power, and never forms a firm clot;" which accords with the experiments of Brodie,[2] and the evidence[3] also of Morgagni, Ruysch, and other observers. Novati,[4] who has had the opportunity of opening the bodies of many individuals poisoned by arsenic, and has carefully studied the physical and physiological characters of this form of poisoning, states "that the blood in these patients is liquid and black as it is in cholera," and without any trace of coagulation. "It should not (Novati very justly adds) be forgotten meanwhile, 1st, that many diseases may give to the blood characters analogous to this; 2nd, that other poisonous substances will be capable of giving the same result; and 3rd, that when death has not occurred promptly, the inflammatory reaction determined by the eschars may quite change these characters of the blood." Lastly, both Orfila and Flandin have cited, with reference to the same subject, the evidence of Rognetta and other members of the Italian school, who agree in stating that "the blood in poisoning by arsenic is deliquescent, syrupy, and nearly the same as in cholera; and this state is not the result of a direct chemical action on the blood; it is altogether a physiological effect, a consequence of the hyposthenic action of the arsenic;" such hyposthenic action being referrible to the lowering influence of the poison on the nerves supplied to the vascular system, and not to any specific effect of arsenic on the blood. And this theory of action quite agrees with the latest opinion of Orfila[5] on the subject, who states that "at the present day, after the beautiful researches of M. Claude Bernard and the ingenious experiments of M. Marey, establishing that inflammation and fever are associated with a state of depression or

[1] 'Op. cit., vol. ii, p. 55.
[2] 'Phil. Trans.,' 1812, part i, p. 214.
[3] Cited by Orfila, 'Traité de Toxicologie,' 4e édit., tom. i, p. 344, 1843.
[4] Cited in 'Gazette Médicale de Paris,' 3e sér., tom. vi, p. 571, 1838.
[5] 'Dictionnaire Encyclopédique des Sciences Médicales, tom. vi, p. 224, 1867.

paralysis of certain parts of the great sympathetic, one would not know how to consider the action exercised by arsenious acid otherwise than as an action really hyposthenic."

With respect to poisoning by the mineral acids, it has been specially noted in some of the more carefully recorded cases that, apart from any absorption of the acid and consequently abnormal irritation of secreting organs from its presence in the blood, the corrosive action of the acid on the gastro-intestinal mucous membrane and the resulting collapse lead to the urine being suppressed: and this analogical evidence is the more valuable from the fact that the suppression in cases of this description cannot be accounted for by a complementary discharge from the bowels, as there is usually obstinate constipation at the same time.

In a case of poisoning by hydrochloric acid, reported by Dr. Collas,[1] in which the patient was a Hindoo, æt. 28, in addition to coldness of the skin, small and frequent pulse, and other symptoms of collapse, "there was suppression of urine, but no purging." And Devergie[2] cites the case of a boy, æt. 3½, who was poisoned by hydrochloric acid, and in which after death it was noted that "the bladder contained no urine."

Among the symptoms of poisoning by sulphuric acid, Tardieu[3] mentions that "the urine is suppressed," and that after death "the bladder is usually empty." Casper[4] has recorded the case of a boy, æt. 16 (No. 194), in which the stomach internally appeared quite black, and perforation had occurred. There is no allusion in this case to the urinary secretion during life, but it was observed after death that the urinary bladder was empty; that the lungs were remarkably anæmic; and that the whole of the blood in the body, as is usual in such cases, was dark and syrupy. In Casper's case (No. 197) of a girl, æt. 20, the lungs were anæmic, and the bladder

[1] 'Annales d'Hygiène,' Janvier, 1858, p. 209, cited by Dr. Taylor, 'On Poisons,' 2nd edit., p. 290, 1859.
[2] Op. cit., tom. iii, pp. 290-296, 1852.
[3] Op. cit., pp. 175 and 180.
[4] Op. cit., vol. ii, 1862.

empty; and again in a case (No. 198) recorded by the same observer, of a child, aged six weeks, it is stated that the urinary bladder was empty and that the lungs contained no blood. Mr. Stanski[1] and Mr. Robinson[2] have also recorded cases in which the symptoms of poisoning by this acid were analogous to those of cholera.

From the elaborate researches of M. Tartra[3] on poisoning by nitric acid, it appears that the corrosive action of this poison is chiefly noticeable in the stomach and upper portion of the intestinal canal, which are inflamed and disorganized, whilst "the large intestines are ordinarily filled with fæcal matters excessively hard;" and, as M. Tartra adds, "it is useful to remark that the urinary bladder does not contain urine, although the patients have not passed any urine." In two of the patients who came under the notice of this observer, one of whom lived twenty, and the other thirty hours after taking the poison, perforation of the stomach occurred. In the first of these cases (Obs. 10) the patient was a lad, aged sixteen years, in whom "the pulse became extremely small, the skin cold, the flow of urine was suspended, and the constipation resisted the use of lavements." In the other case (Obs. 14) the patient was a woman of the town, aged thirty-five years, in whom the symptoms were similar as regards the superficial coldness and imperceptible pulse, but the state of the urinary secretion is not mentioned. In the case of a painter, aged thirty-two years (Obs. 9), who died in nineteen hours from poisoning by nitric acid, M. Tartra informs us that "there escaped towards the end some drops of urine;" and it was remarked that whilst the "hideous aspect of the body of this unfortunate sufferer resembled that of a corpse," the mind was unimpaired. Lastly, in the case of a man, aged forty-five years (Obs. 8), who recovered from the immediate effects of the poison, but who died after a long interval from marasmus, the usual urgent symptoms, such as repeated vomiting, extreme cold-

[1] 'Gazette Médicale de Paris,' 2e sér., tom. v, pp. 188, 189, 1837.
[2] 'London Journal of Medicine,' vol. iii, p. 443, 1851.
[3] 'Traité de l'Empoisonnement par l'Acide Nitrique,' An. 10—1802.

ness of the surface, burning heat internally, and smallness of the pulse, were accompanied, M. Tartra informs us, by "absolute impossibility to pass urine," which from the history of the case appears to have been suppressed.

Poisonous and decomposing animal and vegetable matters have in like manner occasioned symptoms of collapse closely resembling those of cholera, if not the disease itself; but, as a rule, no attention has been paid to the urinary secretion in this class of cases. Dr. Christison[1] has remarked that "Game only decayed enough to please the palate of the epicure, has caused severe cholera in persons not accustomed to eat it in that state;" and similar cases have, some years since, come under my own observation, in which several members of the same family, after eating tainted mutton, suffered from cholera, three of whom died. On examination, it was found that the meat which had caused the mischief in this case, instead of being bad only on the surface, had undergone a rapid and peculiar decomposition near the bone. It is often, however, difficult, if not impossible, to recognise any morbid change in the animal matter to which this choleraic or pseudo-choleraic poisoning has been referred; and it is a curious fact that even milk itself, the secretion of which is commonly supposed to be altogether exempt from any choleraic influence, may in like manner undergo some unrecognisable change by which it acquires deleterious properties capable of producing symptoms of poisoning, which are sometimes analogous to and even identical with those of cholera. Among the more noticeable cases of this description is that which occurred in the year 1826 at Aurillac, a village in France, and which Professors Orfila and Marc were commissioned by the French Academy of Medicine to investigate. In this case fifteen or sixteen customers of a particular dealer in goat's milk were attacked on June 13th, with all the symptoms of violent cholera; and about twenty-four hours afterwards the goat too was taken ill with the same affection, and died in three days. Professors Orfila and Marc in their report stated that, in parallel cases which

[1] Op. cit., 4th edit., p. 635, 1845.

had been reported to them by the police of Paris, they had been unable to detect any mineral poison; that none of the received explanations were in their opinion satisfactory; and that they were disposed to ascribe the poisonous alteration of the milk to new principles formed by a vital process.[1] As an appendix to this case, it may be mentioned that on October 13th, 1856, about twenty persons in Malta were seized with violent vomiting and other symptoms of cholera, which were ascertained by the police physician, Dr. Felice Calleja, to be caused by drinking the milk of two goats, which was said to have acquired poisonous properties from the animals having fed on some wild herbs belonging to the genus oxalis or wood-sorrel.[2]

Among vegetable matters which are capable of producing injurious effects analogous to cholera are poisonous fungi, which in some cases produce narcotism, whilst in others the symptoms are those of gastro-intestinal irritation and collapse.[3] Tardieu[4] in his description of the symptoms produced by poisonous fungi, mentions that "the pulse is weak, the skin cold, and covered sometimes with violet-coloured blotches, and the urine suppressed;" death occurring in these cases "in from one to two or three days." He cites two cases in which a mother and daughter were poisoned by *amanita bulbosa;* the urine having been suppressed in the daughter from the time of the accident, whilst in the mother it was passed in the beginning of the illness, but subsequently her case like that of the daughter assumed the character of choleraic collapse. Flandin[5] has cited a case in which four persons in the same family were poisoned by eating a quantity of *amanita muscaria;* and M. Girard, in his report on this case, remarks, "We have found much analogy between the symptoms of this poisoning and those of

[1] 'Archiv. Gén. de Méd.,' tom. xv, pp. 460-462, 1827, cited by Dr. Christison, op. cit., 4th edit., p. 644, 1845.

[2] 'Medical Times and Gazette,' vol. xiii, new series, p. 456, 1856.

[3] Pereira, 'The Elements of Materia Medica and Therapeutics,' 4th edit., vol. ii, part i, p. 59, 1855.

[4] Op. cit., pp. 821-822, and 828-829, 1867.

[5] Op. cit., tom. iii, pp. 464-469, 1853.

Asiatic cholera. The alvine dejections have nearly always been white like rice-water and flaky." It is important moreover to notice that the analogy is strengthened by the post-mortem examination of the bodies in some of these cases, especially with reference to the condition of the blood, which has been observed to be "black and fluid."

There is little need to dwell on the analogy between cholera and the excessive action of drugs, for, like the poisons already referred to, it is chiefly those drugs whose normal action is limited to the gastro-intestinal mucous membrane which produce, when given in over-doses, effects similar to those of cholera. Among the more noticeable of these is croton oil, which in poisonous doses produces collapse closely analogous to that of cholera; but there is unfortunately no mention in the published records of such cases, either of the urinary secretion during life, or of the state of the bladder after death.[1] And similar omissions with respect both to the urinary secretion and the bladder occur in the records of other cases in which there has also been collapse resembling that of cholera; as, for example, in a case mentioned by Dr. Taylor,[2] in which a girl, aged eighteen years, was poisoned with castor-oil seeds, and whose appearance is described as "like one affected with malignant cholera;" and in the cases reported by Scot,[3] in which "three men having eaten, at the same time, the seeds of the coral plant, had all the symptoms of severe cholera."[4]

[1] 'Journal de Chimie Médicale,' tom. v, 2e sér., pp. 509-511, 1839; Orfila, op. cit., tom. ii, p. 108; Dr. Adam, 'Edinburgh Medical Journal,' vol. i, p. 932, 1855-6; and Dr. Greenhow, 'Medical Times and Gazette,' August 11th, 1866, p. 142.

[2] 'Principles and Practice of Medical Jurisprudence,' p. 266, 1865.

[3] 'Madras Report on Cholera,' p. xxxix, 1824.

[4] The evidence in favour of the excessive action in these cases being due to local irritation of the gastro-intestinal mucous membrane is confirmed by the observations of Pereira (op. cit., vol. ii, part i, pp. 401-402 and 406-407), 'On the Inhalation of the Dust of Euphorbium and of Croton Seeds.' In the latter case, owing to the local irritation being primarily limited to the pulmonary mucous membrane, several doses of castor oil had to be given before the bowels could be induced to act.

With reference to the various cases which have been as yet cited, it must be admitted that the closeness of the analogy is, at first sight, not altogether unfavorable to the theory which refers cholera to a specific blood-poison; and that it would consequently be allowable to assume, if the reasoning from analogy were limited to such cases, that the abdominal centre of the sympathetic nervous system may be only secondarily affected through the ramifications of the vaso-motory nerves. For such a theory of action might to some extent, though not altogether, be reconcileable with the irritant and corrosive effects produced on the gastro-intestinal mucous membrane, and which, in all of the preceding cases, were associated with, if they did not occasion and develop the collapse simulating that of cholera. But there remains to be considered a group of cases characterised by a closeness of analogy approaching in some respects almost to identity with cholera, in which it would not be allowable to assume that a blood-poison was the cause of the collapse. Well-marked illustrations of this occurrence of collapse, altogether independent of any possible connection with blood-poisoning, are to be met with in cases of perforation, rupture, and laceration of the stomach and small intestines; over distension of the stomach; and impassable obstruction in the small intestine. In all of these cases, with some others of an allied character, which will be incidentally noticed, there is usually a condition of collapse associated with suppression of urine, which not unfrequently has been, and, almost unavoidably, is very liable to be, mistaken for that of cholera; and if, as is sometimes alleged to be the case, such diagnostic errors are comparatively more often noticed in hospitals than elsewhere, it may be inferred that in private practice they are sometimes overlooked or concealed, and that in consequence the published records of their occurrence are incomplete.

The first cases of perforation of the stomach in which I noticed suppression of urine during life, with an empty and contracted state of the bladder after death, occurred before my attention had been specially directed to cholera; and

these earlier observations were subsequently embodied in a paper "On Perforating Ulcers of the Stomach," which was read at a meeting of the now extinct North London Medical Society in 1854. In this paper, of which an abstract was published in one or two medical journals, it was stated that "suppression of urine, although it does not appear to have attracted much notice, is as marked a symptom (in perforation) as it is in cholera, and that after death the urinary bladder is usually found to be empty and contracted." Among the cases occurring in my own practice which were cited on the occasion was that of a journeyman coach-painter, aged fifty-three years, in whom alarming hæmorrhage from the stomach had immediately preceded perforation; and although the patient survived nearly two days, no urine was passed, and the bladder after death was found empty and contracted. In the case of a Chelsea pensioner, aged fifty-nine years, employed as a potman, and in whom the immediate cause of perforation was a "dinner-treat" of roast pork and brandy, death occurred in thirty hours, during which time only six ounces of urine were passed, and after death the bladder was found empty and contracted. Whilst in the case of a bricklayer, aged sixty-six years, who died in sixteen hours and a half, and in the case of a retired publican, aged eighty-two years, who died in twenty-two hours, and whose case is of additional interest as one of the oldest on record,[1] there were no notes taken of the state of the urinary secretion during life, but in both of these cases, which occurred in the St. Marylebone Infirmary, the bladder after death was found empty and contracted.

Further inquiry in the same direction, and a more extended acquaintance with the literature of the subject, having convinced me that there is a very close relation between perforation and cholera, I endeavoured, in 1856, to prove in my work 'On the Nature of Cholera as a Guide to Treatment,'

[1] Dr. Brinton, "On Ulcer of the Stomach," 'Med.-Chir. Review,' vol. xvii, p. 171, 1856, states that the oldest case of perforation of the stomach he is acquainted with is that of a man who was, as in the above-mentioned case, aged 82 years.

that "the theory which ascribes cholera to an impression on the sympathetic nervous system, conveyed through the medium of the stomach or intestines," is very strongly supported by the analogical evidence of these and other allied cases which could be cited in its favour; and I proposed to include them with cholera, under the head of "Disorders of the Sympathetic Nervous System, depending on diminution or arrest of its functions, and produced chiefly through the medium of the stomach." There is no need for me to enter again into an exhaustive examination of the nature of this evidence, or to notice all the successive cases of mistaken identity, in which perforation and other morbid affections belonging to the same group have been from time to time confounded with cholera. In all of these cases there is essentially the same condition of collapse which is usually accompanied, as in cholera, with suppression of urine during life, and an empty and contracted state of the bladder after death; and although in a large number of the recorded cases of this description, including those which have and those which have not been mistaken for cholera, there is no allusion, after the perforation has occurred, to the secretion of urine during life, or the condition of the bladder after death; yet in all cases, so far as I can at present remember, in which I have sought and have been enabled to fill up this omission by subsequent inquiry, I have ascertained that suppression of urine, unaccompanied by any head-symptoms, is as well marked in such cases as it is in cholera. As an illustration of the extent to which this important condition in the analogy prevails, it may be mentioned that in the two cases of perforation simulating cholera, lately reported in the medical journals, and in neither of which is there any allusion to the urinary secretion or bladder, it has been ascertained on further inquiry that although in the St. George's Hospital case[1] of perforation of the stomach closely simulating but not apparently mistaken for cholera, no note was made of the state of the urinary secretion or bladder, and no information on the subject could be obtained; yet in

[1] 'Medical Press and Circular,' March 20th, 1867, pp. 271-272.

the other case[1] referred to, in which the patient died in the Cholera Ward of the London Hospital, after an illness of about two days and a half, from perforation of the duodenum near the pylorus, the acting resident medical officer who reported the case subsequently informed me by letter that there was "undoubted and continued suppression of urine." The additional information which was obtained in this latter case derives increased importance from the fact that in another case[2] of pseudo-choleraic collapse, which occurred in the same hospital only seven days after the last-mentioned case, and in a young female patient, who at the time had been an inmate of the hospital for more than a week on account of disease of the elbow-joint, the symptoms which led to her removal to and subsequent death in the Cholera Ward, were supposed to be indicative of "sudden cholera collapse;" her condition at the time being described as follows: "almost pulseless, quite cold, with general lividity of the surface, and blueness of the finger-nails." But it is important to notice in the history of this case, that whilst "several ounces of clear urine were drawn off," yet the patient was only semi-conscious. This difference in the symptoms, accompanying the collapse, of these two pseudo-choleraic cases in the London Hospital during the same month, admitted of being readily explained after death by the fact that the fatal collapse in the last case was due to Addison's disease, and that all the organs, with the exception of the supra-renal capsules, were found to be healthy; whilst in the other case, in which there was complete suppression of urine, the fatal collapse was the result of perforation of the duodenum near the pylorus.

So far as regards the literature of this subject, it must be acknowledged that the positive evidence in favour of suppression of urine during the collapse consequent on perforation is somewhat incomplete; for many of those who have published papers and cases descriptive of this lesion, have not apparently been aware of the significance of the symptom,

[1] 'Lancet,' Nov. 24th, 1866, p. 577.
[2] Ibid., March 9th, 1867, p. 299.

even when, as seems to have been more often than otherwise the case, it has not been altogether overlooked. Devergie[1] indeed in his valuable work on legal medicine distinctly states that in perforation of the stomach, the urine is suppressed; and in like manner, Valleix[2] has remarked "that the suppression of urine is sometimes very remarkable" in peritonitis from perforation; but there are very few writers of importance who have recognised that it is a usual, if not a constant symptom in such cases. Dr. Reeves[3] has indistinctly noticed that "the bladder is the organ most frequently affected" in perforation of the stomach; and that its implication is marked "by an incessant desire to pass water —a few drops only escaping;" and he has, moreover, remarked that "the bladder will be found on the introduction of a catheter, empty;" yet he refers to it merely as evidence of the peritoneal inflammation having become general, and does not connect it with shock to the abdominal sympathetic nervous system, although he has cited five cases of more or less sudden death from perforation of the stomach, and has further remarked that patients are sometimes liable to die suddenly in attempting to sit up after perforation has occurred. Dr. Stokes in his paper on "Peritonitis from Perforation of the Serous Membrane,"[4] does not even allude to suppression of urine in connection with perforation of the stomach; and in the abstract published of Mr. Leonard's paper on "Perforating Ulcer of the Stomach,"[5] which was read at the Medical Society of London in 1852, there is likewise no allusion to the urinary secretion, or to the state of the bladder. Dr. Crisp[6] has so far overlooked both the frequency

[1] 'Médecine Légale,' 3e édit., tom. iii, p. 776, 1852.
[2] 'Guide du Médecin Practicien,' 3e édit., tom. iii, p. 308, 1853.
[3] 'Diseases of the Stomach and Duodenum,' pp. 108, 109, 1856.
[4] 'Cyclopædia of Practical Medicine,' vol. iii, 1834.
[5] 'Medical Times,' vol. xxv, pp. 476, 477, 1852.
[6] 'Cases of Perforation of the Stomach from Simple Ulceration,' pp. 3-7, 1843. In one other case recorded by this author (case 4, pp. 10-12) there is indirect evidence of the urine being suppressed, as it is stated that the patient, a girl, æt. 15½, on the evening before her death "had a small quantity of gin and water, and about as much nitre as would lie on a sixpence.'

and the importance of this symptom, as merely to state, without comment, that in the case of a woman who had died at 10.30 a.m., on December 27, 1837, from perforation of the stomach, no water had been passed "since 12 o'clock last night; bladder not distended;" and it was found after death that "the bladder was empty."

Among French writers to whom it will be useful to refer is M. Cazeneuve,[1] who has bestowed much attention on this diagnostic symptom, more especially in perforation of the small intestines, and who was at one time disposed to claim the merit of having been the first to draw attention to it in this latter class of cases. He relates a very impressive case of cancerous perforation of the stomach in a man, æt. 28, which came under his observation in the Military Hospital at Lille, in 1833. As no urine was passed, after the perforation in this case occurred, a catheter was introduced, but only two spoonfuls of urine were obtained, and it was found to be impossible to turn the catheter about in the bladder, owing to the very strong contraction of that organ. Subsequently the catheter was again passed, but no urine was obtained, and after death it was noted that the bladder was contracted and hidden behind the pubes. Laisné,[2] Gerard,[3] Lefevre,[4] and many other observers, who have written on this lesion, have generally failed to notice the state of the urinary secretion; although they have sometimes incidentally mentioned without comment, in what appears to have been the only case in which any note was made of this patho-

[1] 'Gazette Médicale de Paris,' 3e sér., tom. vi, p. 814, 1838.
[2] (Fr. Chaussier) 'Médecine Légale,' par MM. Lecieux, Renard, Laisné, et Rieux, 1819. A second edition of the Thesis on Perforation, translated by Dr. Weatherhead and dedicated, by permission, to Lord Eldon, was published in 1821.
[3] 'Des Perforations Spontanées de l'Estomac,' 1803. In a case cited by this author (Obs. 17, pp. 62, 63), in which a woman, aged 30 years, died from spontaneous perforation of the stomach, it may be inferred from the history of the case that there was suppression of urine during life; after death it was observed that "the bladder was very small."
[4] 'Archives Générales de Médecine,' 3e sér., tom. xiv, pp. 377-428, and tom. xv, pp. 28-59, 1842.

logical fact, that the urinary bladder after death was found contracted.

Among the isolated cases which have been recorded of perforation of the stomach, in which there is any reference to the urinary secretion and bladder, is a case, observed by Dr. (afterwards Sir Philip) Crampton,[1] of perforating ulcer of the stomach in a lady, æt. 29, in which death occurred from collapse twelve hours after the accident. The condition of the urinary secretion during life is not mentioned; but it is stated that after death "the urinary bladder was empty and contracted." In a case recorded by Dr. Carson,[2] of rupture of the stomach from rapid development of gas, which occurred in a sailor æt. 20, and who lived for fifteen hours, the condition of the urinary secretion after the occurrence of the accident is not referred to; but it was ascertained after death that "the bladder was contracted and empty." This case appears to have been somewhat complicated, as a slight trace of arsenic was detected in the stomach. M. Desgranges[3] has recorded the case of a young woman, æt. 18, who died in twenty hours from spontaneous perforation of the stomach, and in which the symptoms noted twelve hours before death were as follows:—"icy coldness of all the body, especially of the extremities; no pulse; eyes dull and hollow; temples sunk; face pale and cadaverous; and aphonia." There was desire but constant inability to pass urine, and later in the history of the case M. Desgranges refers to the fact of there being no urine. An instructive case of perforation mistaken for cholera has been recorded by Dr. Rigaud,[4] in which the patient died in twenty hours. Dr. Hérard[5] has recorded a case of perforation of the stomach in a woman, æt. 34, in which it was noted that the urine was suppressed. A female domestic servant, æt. 48, was admitted

[1] 'Transactions of the Association of Fellows and Licentiates of the King's and Queen's College of Physicians in Ireland,' vol. i, pp. 1-10, 1817.
[2] 'Edinburgh Medical and Surgical Journal,' vol. lxvi, pp. 27-31, 1846.
[3] 'Journal Général de Médicine,' 2e sér., tom. xv, pp. 145-164, 1821.
[4] 'Gazette Médicale de Paris,' 3e sér., tom. xxviii, p. 768, 1858.
[5] 'Bull. de la Soc. Méd. des Hôp. de Paris,' tom. iii, pp. 149-152, 1864.

into the Cholera Wards of St. Bartholomew's Hospital, Nov. 7, 1866, in whom, after death, there was found to be peritonitis from perforation of the stomach. Lastly, Dr. Barlow[1] has cited a case observed by Dr. Gull, of chronic ulcer of the stomach, with constriction of the pyloric extremity, in which there was excessive vomiting, and no urine was passed for forty-eight hours before death. In this case the bladder was found, after death, empty and contracted.

If from cases of perforation and injury of the stomach the attention be transferred to the corresponding class of cases affecting the small intestines, it will be found that suppression of urine is usually associated with the resulting collapse; and that in consequence of the common occurrence in such cases of this symptom, it has been referred to by some writers as diagnostic of the seat of mischief. There will not, however, be much difficulty in recognising, on further inquiry, that whether the mischief be the result of perforation or of obstruction of the small intestines, the suppression of urine which results from it must be regarded chiefly as an indication of the intensity of the collapse, consequent on the comparative suddenness of the mischief, and its nearness to the abdominal centre of the sympathetic nervous system; in the same way that a corresponding suppression occurs in severe cases of cholera, whilst in mild cases of this disease, which it has been customary to refer to as choleraic diarrhœa, there may be, and usually is, the characteristic cholera flux without any suppression of urine. If, for instance, the clinical history of those cases in which the mischief has been limited to one of the three divisions of the small intestines be carefully examined, it will be found that whilst suppression of urine is a very constant symptom in the collapse consequent on perforation or rupture of the duodenum, and also in corresponding cases affecting the jejunum, of which, however, there are but few examples on record, it is less constantly, although still very commonly observed in the strictly analogous cases in which the ileum has been the seat of mischief. With regard, moreover, to the comparative

[1] 'Medical Times and Gazette,' April 28th, 1866, p. 445.

value of this diagnostic symptom, it is important to notice that in a large proportion of the latter class of cases, in which the perforation has occurred in the course of typhoid fever, there has been diminution of the urinary secretion as a consequence of the fever, previous to and apart from any severe affection or injury of the ileum, and that this diminution, being associated with the continued formation and accumulation in the system of the urinary constituents, is pathologically a distinct condition from the diminution and suppression which occurs in those cases which are analogous to cholera, and in which, as in cholera, the failure of excrementitious elimination is an index of the extent to which the sympathetic nervous system is affected during collapse.

The following illustrative cases referrible to the duodenum may be cited in favour of these remarks:—1. The case already mentioned of spontaneous perforation of the duodenum, three lines from the pylorus, which was lately admitted into the Cholera Ward of the London Hospital, and in which there was "undoubted and continued suppression of urine." 2. A case, reported by M. Petrequin,[1] of spontaneous perforation of the duodenum, eight lines from the pylorus, in which the collapse very closely resembled that of cholera, for there was suppression of urine, cyanosis, the Hippocratic face, and the pulse was imperceptible at the wrist. The state of the bladder after death was not noted. 3. A case, reported by Dr. Humphrey Sandwith,[2] of perforation of the middle of the duodenum by a worm, in which the pulse at times was scarcely perceptible at the wrist, and when a catheter was passed only a few ounces of urine were withdrawn. After death the bladder was found empty and contracted. 4. A case, reported by Dr. Symes,[3] of laceration of the duodenum, from external violence, which the patient survived only twenty hours. In this case, which differs somewhat in character from the preceding, as the injury occurred from without inwards, there was a constant desire

[1] 'Archiv. Gén. de Méd.,' 2e sér., tom. xii, pp. 483-485, 1836.
[2] 'British Medical Journal,' March 9th, 1861, pp. 249, 250.
[3] 'Dublin Quarterly Journal,' vol. xlii, pp. 497-498, 1866.

to pass water, but it is not stated that any was passed. After death the bladder was found empty and contracted. 5. A case, recorded by Prof. Rembold,[1] of strangulation of the first portion of the duodenum, in which, on passing a catheter, only some drops of urine were obtained. 6. A case of perforation of the duodenum in a clerk, aged thirty years, recorded by M. Oulmont,[2] in which there was suppression of urine with vesical tenesmus; and when, owing to the patient believing that the bladder was about to burst, a catheter was passed, not a drop of urine could be obtained. After death the bladder was found strongly contracted and quite empty. In this case there was for some hours before death persistent delirium, which appears, however, to have been due, not to the suppression of urine, but to the patient's previous habits of excessive drunkenness, which had led to occasional attacks of insanity, during which he had sought one day to strangle his child, and on another occasion to kill his wife. Lastly, in a small series of cases of duodenal perforation, recently published by Dr. Andrew Clarke,[3] there is, beside the case already referred to as having died in the Cholera Ward of the London Hospital, a well-marked case (Obs. 2), of a lad, aged nineteen years, who was admitted into the London Hospital "in a state of partial collapse, and alleged to be suffering from retention of urine." By means of a catheter, easily introduced, the bladder was found empty. After death it was ascertained that the perforation of the duodenum had occurred about one inch and a half from the pylorus. "The right cavities of the heart were gorged with blood; the left empty." Dr. Clarke, in commenting on this case, remarks that "one could scarcely have wondered if it had been mistaken for cholera." In the case of a watchmaker, aged twenty years, which is the third case in this series, the urinary secretion and bladder are not referred to, but the appearance of the patient is described as "like that

[1] 'Gazette Hebdomadaire,' 28 Avril, 1865, pp. 271, 272.
[2] 'Archives Médicales de Strasbourg,' Août, 1836; cited by M. Forget, 'Gazette Médicale de Paris,' 2e sér., tom. v, p. 229, 1837.
[3] 'British Medical Journal,' June, 1867.

of a choleraic in collapse."[1] There are two other cases of perforation of the duodenum recorded by Dr. Clarke, which it may be useful to notice in connection with this subject, as they illustrate the relatively milder effect of a slight and gradual leakage compared with a more sudden and extensive escape of the duodenal contents. In one of these cases the patient was a man, aged thirty-five years, who died from perforation of the duodenum, which was situated about half an inch from the pylorus. When first seen, only a slight leakage had apparently occurred; the extremities were warm, and some healthy urine had, a short time previously, been passed. Six hours before death there was undoubted evidence of peritonitis from perforation, with the characteristic collapse. The perforation of the duodenum in this case was ascertained to be about half an inch from the pylorus; but the state of the bladder after death is not mentioned. In the other case (Obs. 6), which is of a somewhat doubtful character, a man, aged forty-three years, was supposed to have had a slight leakage from duodenal perforation, and it is stated that the urine was normal; but the diagnosis in this case could not be verified, as the patient recovered.[2]

Cases of perforation, of obstruction, or even of traumatic lesion of the jejunum, are, as already stated, rare; but among the few which have been recorded, there is one of considerable interest observed by M. Cazeneuve,[3] in which a man, æt. 24, suffered from two attacks of peritonitis from

[1] Corresponding cases have been published by M. Poisson ('Bull. de la Soc. Anat. de Paris,' tom. xxx, pp. 300, 301, 1855;) by Mr. Stilwell ('Lancet,' July 18th, 1846, p. 67); and by Sir David J. H. Dickson ('Med.-Chir. Review,' vol. xxxiii, pp. 586, 587, 1840.)

[2] In connection with these cases it may be useful to notice that acute ulceration mostly limited to the first portion of the duodenum, and to which Mr. Curling ('Med.-Chir. Trans.,' vol. xxv, 1842) has directed attention as a not unfrequent sequel to burns, especially in young subjects, appears to be an occasional consequence of what at present can only be vaguely referred to as shock, which appears to act by causing an arrest of nutrition, and which would therefore be strictly allied to, though differing in degree from, what occurs in cholera and in some of its analogies.

[3] 'Gazette Médicale de Paris,' 3e sér., tom. vi, obs. 2, p. 813, 1838.

perforation, which occurred in January and in April of the same year. It is mentioned in the report of this case that there had been, at the time of the first attack in January, vesical tenesmus with difficulty in the urinary secretion: whilst in the fatal attack in April there had been suppression of urine. After death, perforation of the ileum near the ilio-cæcal valve, and of the jejunum near its middle were found; and the bladder was empty and hid behind the pubes. Dr. Barlow[1] has recorded a case of fatal obstruction of the jejunum, in which there was complete suppression of urine for six days; a catheter having been passed and no urine obtained. And a case of traumatic rupture of the jejunum in a labourer, æt. 31, which was followed by death in forty hours, occurred lately in the practice of Mr. Curling[2] at the London Hospital. In this case the collapse closely resembled that of cholera; there was intense pain in the hypogastric region; vomiting; coldness of the extremities and also of the tip of the nose; pulse imperceptible at the wrist, and very feeble in the brachial artery; and cyanosis. There is no reference, in the report of the case, to the state of the urinary secretion during life, but after death the bladder was found empty and contracted.

Although it must be admitted that comparatively little attention has been directed to the state of the urinary secretion in perforation or in other lesions of the stomach, duodenum, and jejunum, yet there has been great and, in some respects, undue importance assigned to it in those cases in which the ileum has been affected; and whilst some distinguished writers have referred to it as in some degree diagnostic of perforation, others have regarded it as an almost certain indication of obstruction in the small intestine, chiefly at this part. Among the former may be cited M. Cazeneuve,[3] who has recorded three cases of perforation of the ileum (Obs. 1, 2, and 5) occurring in the course of typhoid fever, in which

[1] 'Guy's Hospital Reports,' vol. ii, pp. 378-380, 1844; and 'Medical Times and Gazette,' April 28th, 1866, p. 444.

[2] 'London Hospital Reports,' vol. ii, pp. 287-288, 1865.

[3] 'Gazette Médicale de Paris,' 2e sér., tom. v, pp. 818-821, 1837.

there was more or less vesical tenesmus, with suppression of urine; and in each of these three cases the bladder was found empty and contracted. Subsequently, however, M. Cazeneuve[1] has remarked that "suppression of urine has not occurred in all cases; often, also, it is not complete. I should only have (he adds), in order to prove this, to cite the silence of authors on this point. In effect, it is not mentioned by M. Andral, Louis, Chomel. M. Broussais cites only two or three examples of it; moreover, cases have been related in which the urinary secretion has continued." Among those who have bestowed special attention on this symptom is M. Judas,[2] who, when Assistant Physician to the Gros-Caillou Military Hospital at Paris, recorded two cases of perforation of the ileum which occurred in that hospital, and in both of which there was suppression of urine. In one of these cases, that of a soldier aged twenty-three years, a catheter was passed without, however, obtaining a drop of urine; and after death the bladder was found empty and very much contracted. In the other case, when a catheter was passed, it could not be depressed, and only a few drops of clear and inodorous urine were withdrawn: the bladder, after death in this case, was found empty and contracted to the size of a pigeon's egg. In a case which occurred in the practice of M. Piorry,[3] in which a man, aged thirty-one years, had perforation of the ileum in the course of typhoid fever, there was for some time suppression of urine as a result of the perforation, but subsequently, during the last few days of life, the urine was again secreted and passed; showing that the suppression in these cases is not so much due to either the fever or the inflammation, as to the shock consequent on the perforation. Whilst in cases unconnected with typhoid fever, as, for example, in those produced by a worm, or by external injury, suppression of urine, with an empty and contracted state of the bladder after death, has been noted by Mr. Kell,[4]

[1] 'Gazette Médicale de Paris,' 3e sér., tom. vi, p. 814, 1838.
[2] Ibid., pp. 722, 723.
[3] Ibid., pp. 44, 45.
[4] 'London Medical Gazette,' vol. ii, pp. 649-650, 1828.

by M. Andral,[1] by M. Baraduc,[2] and by some other observers.

But far more importance has been assigned to suppression of urine in the collapse which results from intestinal obstruction than, perhaps, in any other class of cases; and the following case of strangulation of the lower part of the ileum, which has been lately recorded by M. Dexpers,[3] affords a good illustration of the analogy between such cases and cholera. The patient was a soldier, who, when brought to the hospital, presented, we are informed, the appearance of a patient suffering from cholera; his eyes were sunk; his complexion cyanosed; his hands blue and cold; the skin wrinkled; and the pulse small, hard, and very frequent. The succeeding night was passed in mortal agony; in spite of frequent desires, it was impossible for him to pass either fæces or urine, and the introduction of a catheter brought away only a few drops of urine. The intellectual faculties were unimpaired. After death the bladder was observed to be empty and completely hid behind the pubes. Dr. Besnier,[4] in a valuable memoir on intestinal obstruction, has recorded a fatal case (Obs. 128) of strangulation of the small intestine in a woman, aged forty-seven years, who, "in consequence of a bath too hot taken immediately after having eaten, was suddenly seized with a very sharp and sudden pain in the abdomen." There was the usual collapse with constipation and suppression of urine from that time, and it was ascertained on the day after her admission that the bladder was empty. And Mr. Robinson[5] has recorded the case of a woman, aged sixty-three years, who died in thirteen hours from collapse with suppression of urine, which was supposed to be cholera, but after death it was ascertained that there was strangulation of the ileum. With reference to such cases it is curious to notice

[1] 'Clinique Médicale,' tom. ii, 3e édit., pp. 602-604, 1834.
[2] 'Bull. de la Soc. Anat. de Paris,' tom. xiii, pp. 336-340, 1838.
[3] 'Recueil de Mémoires de Médecine, de Chirurgie, et de Pharmacie Militaires,' tom. xviii, 3e sér., pp. 195, 196, 1867.
[4] 'Des Etranglements Internes de l'Intestin,' Paris, pp. 194-196, 1860.
[5] 'London Journal of Medicine,' vol. iii, pp. 443-446, 1851.

the great difference of opinion which has prevailed respecting the importance and the meaning of this suppression of urine; for whilst, on the one side, the late Dr. Barlow,[1] in common with some other observers, alleged that it might be considered diagnostic of that form of constipation which results from obstruction of the bowels situated high up, and that when the obstruction is low down then the urine is not suppressed in these cases of obstinate constipation, owing, as was erroneously supposed, to a sufficient amount of fluid for carrying on the urinary secretion being allowed to enter the system in the latter class of cases, but not in the former; on the other side it will be found that a different explanation of the fact has been given in the lately published and posthumous work of Dr. Brinton[2] on intestinal obstruction, in which the diagnostic value attached to this symptom by Dr. Barlow is altogether denied, and Dr. Brinton, in admitting the frequent occurrence in these cases of suppression of urine, expresses his belief that "the amount of vomiting is the truest symptomatic correlative of the diminution of the urine," and consequently agrees to some extent with Dr. Besnier,[3] who, in directing attention to the opinions on this subject of "English physicians," remarks, that the state of the urinary secretion, although "useful to consult, nevertheless does not furnish any indication which has an absolute value;" and that to enable us to infer something from the small quantity of urine secreted there must be neither peritoneal inflammation nor any considerable or frequent vomiting; "for these conditions (he adds) are sufficient to bring about diminution or even suppression of the urinary secretion." It may here, perhaps, be allowable to remark that any correlation which may exist between vomiting and suppression of urine in these cases appears to be simply the result of both conditions being primarily dependent on the affection of the abdominal

[1] 'Guy's Hospital Reports,' 1844; and 'Medical Times and Gazette,' April 28th, 1866.

[2] 'Intestinal Obstruction,' by William Brinton, M.D., F.R.S., edited by Thomas Buzzard, M.D., London, 1867.

[3] Op. cit., pp. 305, 306.

sympathetic nervous system; and this seems to have been subsequently, though very imperfectly, recognised by Dr. Brinton, who refers with evident hesitation and doubt to the connection between this form of suppression of urine and what he terms "mere collapse."

The opinions which have been expressed by the above-cited and other authorities respecting the diagnostic value of suppression of urine in such cases as the preceding appear to be all defective, in so far that the suppression is alleged to be connected with or dependent on something which is not essential to its occurrence, although it may be, and perhaps often is, associated with it. Hence the importance of carefully collating the clinical history of all those cases in which, as in cholera, it characterises the collapse; for by thus extending the range of the inquiry it will be conclusively proved that this symptom may occur in corresponding conditions of collapse, independent, for example, of any one of those four cardinal phenomena, of pain, vomiting, intestinal obstruction, and intestinal flux, which have each been referred to by distinguished writers on the subject as its correlative, since by means of such extended inquiry it can be clearly demonstrated that suppression of urine is not necessarily dependent on any of these associated conditions. This will become more and more evident as the inquiry proceeds, but it will be convenient on this occasion to remark that its occasionally more or less complete independence of pain is fully proved by the fact that there may be epigastric pain quite equal in intensity to that of cholera, or any analogous disease, without any suppression of urine; that cases of painless choleraic diarrhœa have been observed in which the urine has been suppressed; and that in cases of cholera itself the suppression is often maintained for a long time after all pain has ceased in patients who ultimately recover. Whilst its alleged dependence, either on intestinal obstruction or on flux from the stomach or bowels, is in like manner fully disproved by cases in which neither of these two apparently opposite conditions has been observed, the term apparently being here introduced in consequence of these two conditions,

so far as the suppression of urine is concerned, admitting of being reconciled. For the suppression of urine in these cases is supposed to represent simply a want of water, and this deficiency is alleged to be consequent on the supply being so completely shut off in cases of intestinal obstruction, and so completely drawn off in cases of intestinal flux, as to reduce the remaining fluid in the body very considerably below what is required to carry off the excrementitious matters usually eliminated by the kidneys. Ample evidence of a very conclusive kind could be cited in opposition to this theory of the suppression of urine during collapse being due to a reduced supply of fluid; but it will be sufficient for the present to quote the two following cases to show that the urine may be suppressed during collapse from structural lesions in the immediate neighbourhood of the stomach, without the integrity of the gastro-intestinal mucous membrane being impaired. In the first of these illustrations, which has been recorded by Dr. McSwiney,[1] a man, aged forty-five years, died from collapse with suppression of urine, fifty-eight hours after the spontaneous rupture of the common bile-duct. The lesion, which occurred about three quarters of an inch from the entrance of the duct into the duodenum, was probably connected with over-distension of the gall-bladder from starvation; and although the lower portion of the ileum in this case was much diminished in calibre, it was pervious to the passage of fluid. In the second illustration, which was observed by Dr. Wolf,[2] of Bonn, there was a corresponding rupture of the hepatic duct in a woman, aged sixty years, who died twenty-four hours after the occurrence of this lesion. The resulting collapse in this case closely resembled that of cholera, and only a few drops of urine were discharged. The urine has in like manner been sometimes observed to be suppressed in severe abdominal operations, and among the more distinguished observers who have specially noted this fact is M. Claude-Bernard,[3] who informs us that in the operations

[1] 'Dublin Quarterly Journal,' Nov., 1866, pp. 518, 519.

[2] 'Journal des Progrès des Sciences et Institutions Médicales,' tom. xiv, pp. 245, 246, 1829; cited from 'Journal von Graafe,' 12e, B. S., 370.

[3] 'Comptes Rendus,' tom. xlvi, p. 161, note, 1858.

required to expose the renal vein, in which it is necessary to open largely the abdomen, there is "nearly always (as a consequence of the operation) in the dog and in the rabbit, if not immediately, at least after a few moments, suppression of urine."

The importance which has been attached to suppression of urine in cases of collapse simulating that of cholera must not, however, lead to other points in the analogy being undervalued or overlooked; for in some recorded cases belonging to the preceding group, in which there is no mention of the urinary secretion or bladder, much useful information on some other facts in pathology may not infrequently be obtained. With reference more especially to the condition of the vascular system, it should be observed that there is often a very close resemblance between these cases and cholera, not only as regards the frequent occurrence of cyanosis, and the failure, often for several hours before death, of the pulse at the wrist, which have been noted in many of the cases already cited, as well as in others in which there is no mention of the urinary secretion; but even the physical condition of the blood and the spasmodic contraction of the blood-vessels have occasionally been observed to be also similar. In a case, for example, of spontaneous perforation of the stomach in a girl, aged twenty-one years, observed by M. Ubersaal,[1] Senior Physician to the Orphan Asylum at Strasburg (a preparation of which has been preserved in the Museum of the Faculty of Medicine), it was found, on bleeding the patient from the arm to the extent of eight ounces, that the blood drawn from the vein was so thick as to flow with difficulty; and although the collapse in this case was not at first so well marked as it usually is after perforation has occurred, yet only a small quantity of urine was passed. The state of the bladder after death was not noticed. And Dr. Ebermaier,[2] in an ably written article on perforation of the stomach, states that in two of the cases which he has cited (cases 2 and 8) the patients were ordered

[1] 'Archiv. Gén. de Médecine,' tom. xxvi, pp. 412-414, 1831.

[2] 'Journal Complémentaire du Dictionnaire des Sciences Médicales,' tom. xxxi, pp. 34 and 164, 1828.

to be bled, and that in each case the attempt to obtain blood from a vein failed.

Another point in the analogy between these cases and cholera, to which it may be useful to refer, is the spasmodic contraction of the muscles, which has been specially noticed by M. Gerard[1] in a well-marked case of spontaneous perforation of the stomach in a man, from twenty-eight to thirty years of age, in whom "the muscles of the abdomen were in a state of such violent contraction that the anterior wall of this cavity appeared glued to the vertebral column, and offered the hardness of a board." The patient was ordered to be bled, in accordance with the usual practice at that time, and it was noticed that "as soon as the lancet was withdrawn from the vessel the blood spurted out more than ten feet, and immediately stopped, without the possibility of the surgeon, who was very dexterous, being able to obtain more." M. Gerard, in commenting on this case, informs us that these violent contractions are not an infrequent symptom also in wounds of the stomach, and he has cited a remarkable instance which occurred in the military hospital at Strasburg, into which a soldier had been admitted apparently dead from a large wound in the abdomen below the zyphoid cartilage, and through which the contents of the stomach escaped. The patient rallied, but died the next morning, after suffering great pain; and in dissecting the body it was found that in the muscles of the arm, but especially in those of the thighs, an innumerable number of the muscular fibres were broken. In like manner it has been remarked by Orfila,[2] and other observers, that spasmodic rigidity of the muscles during life is "among the lesions of tissue produced by arsenious acid."

Well-marked pulmonary collapse, which has been already noticed in connection with irritant poisoning, was observed by Dr. Cotton[3] in a case of large perforating ulcer of the stomach communicating with the transverse colon. The

[1] Op. cit., pp. 1-4, 1803.
[2] Op. cit., tom. i, 4e édit., p. 344, 1843.
[3] 'Medical Times and Gazette,' Jan. 12, 1856, pp. 38, 39.

patient in this case was a shoemaker, aged forty years, who died in the Brompton Hospital, eighteen days after fæcal vomiting had occurred, from "exhaustion or collapse;" and the lungs, which were found after death to be free from tubercle, were "very much collapsed." In connection, moreover, with pulmonary collapse it may be remarked that an alteration characterised by diminution or absence of the voice, such as is common in cholera, is also very noticeable in many of these cases of analogous collapse, and it has been specially referred to by Dr. Murchison[1] in his elaborate paper on gastro-colic fistula.

In directing attention to these and other accompanying conditions in collapse, it is necessary at the same time to state that even if they could all be detected and analysed with the same facility as suppression of urine, which, generally speaking, is not possible, yet none of them indicates either so clearly or so exactly the character and the extent of that general affection of the system which, in the case of cholera and its analogies, has been inferred to result from a central arrest of organic function. Failure of the circulation, cyanosis, and superficial coldness, are none of them so distinctive as suppression of urine is of such an arrest of organic function, for they may to some extent occur without it. In like manner, the pulmonary function also fails to supply any corresponding amount of information; for even if, in relation to organic life, carbonic acid could reasonably be assumed to be the correlative of urea, yet there is not the same facility for ascertaining either the extent to which its development has been arrested or the extent to which it has been unduly retained in the blood. With reference, therefore, to the alleged connection between the lungs and cholera through the medium of the blood, to which great and apparently undue importance has sometimes been attached, although there is evidence to show that the development of carbonic acid during intense collapse is more or less completely arrested, and that consequently the air inhaled only partially enters the lungs, and is expired almost, if not occasionally

[1] 'Edinburgh (Monthly) Medical Journal,' vol. iii, p. 132, 1858.

altogether, unchanged, still it must be admitted that the lungs can never, like the urinary bladder, be empty and contracted for an equally lengthened period of life, nor can the formation of carbonic acid, like that of urea, be so completely interrupted for the same time. It may consequently be affirmed that the state of the urinary secretion is the best guide that could be selected in investigating the nature of choleraic and allied forms of collapse, the assistance afforded by it being twofold, and derived partly from direct observation and partly from analogy. With respect to the former, it has been satisfactorily demonstrated by the researches of Dr. Garrod[1] and other able investigators[2] of the chemical pathology of cholera, that the formation of urea is checked or suspended during choleraic collapse; and although there are no corresponding observations on the suppression of urine in analogous cases of collapse, and consequently no direct authority for stating that the suppression in such cases is due to a like arrest of organic function, yet it must be admitted that the presumptive evidence is very great, if not almost conclusive, in its favour. For unless we are prepared to admit that the suppression of urine in these cases of analogous collapse is connected with the arrested formation of urea, it would be impossible to avoid coming in contact with one of two dangerous fallacies, which represent Scylla and Charybdis in the pathology of collapse. For if, in these cases, urea is uninterruptedly formed during collapse, it must be either discharged or retained; in the one case we should have to face the insurmountable obstacle of vicarious secretion, insurmountable at least on this occasion, because it would be necessary to assume that the amount of fluid might be reduced below that required for renal secretion, and yet suffice, not only for a distant and extra-visceral secretion like that from the mammary gland, but also for an adjoining and vicarious secretion; whilst in the other case we should have

[1] "On the Pathological Condition of the Blood in Cholera," 'London Journal of Medicine,' 1849.

[2] Dr. O'Shaughnessy 'On the Chemical Pathology of Malignant Cholera,' p. 61, 1832.

to admit that urea might accumulate in the blood during collapse without causing mischief, although all past experience and all direct observation on the subject lead to the inference that uræmic poisoning would follow with unusual rapidity the accumulation of urea in blood which, although too inspissated, as it is alleged, for renal secretion to occur, is yet not so inspissated as to arrest the mammary secretion or to impede the circulation through the brain.

Apart from analogy it would, perhaps, be scarcely possible to estimate correctly the value of this symptom as indicative of collapse, for it is still very generally supposed that the suppression of urine, if not the collapse itself, is essentially dependent in cases of cholera on the flux from the stomach and bowels; and Dr. Copland,[1] who agrees on this point with Sir Thomas Watson,[2] Dr. Wood,[3] of Philadelphia, and other distinguished writers, may be said to represent very fairly the prevailing opinion when, in his latest publication on the subject, he states without reserve that "the ischuria (in cholera) is a consequence of the excrementitial fluid of the blood being all discharged by the digestive canal and skin, none being left that can be removed by the kidneys." In reply to this assertion it will be sufficient to mention that, not only is there ample evidence derived from direct observation to refute it, in addition to the evidence which has been adduced from analogy, but that there is, moreover, a self-destructive fallacy in thus supposing that the digestive canal could serve for the complete escape of excrementitial matter usually discharged by another outlet, whilst the elimination of excrementitial matter proper to the canal itself is altogether checked. For just in the same way that jaundice results from the increasing accumulation of the essential elements

[1] 'Dictionary of Practical Medicine,' vol. iii, part ii, p. 1006, 1858.

[2] 'Lectures on the Principles and Practice of Physic,' 4th edit., vol. ii, p. 642, 1857.

[3] "In epidemic cholera, in which the urinary secretion is sometimes entirely arrested, the brain remains remarkably clear, in consequence, no doubt, of the copious discharges from the skin and alimentary mucous membrane."—'A Treatise on the Practice of Medicine,' 4th edit., vol. ii, p. 599, 1855.

of bile in the blood, and uræmic poisoning from the increasing accumulation in like manner of urea, so the absence of these two morbid conditions shows that during collapse there is no excess of excrementitial matter for either the kidneys or the liver to eliminate, and therefore no urine is conveyed to the bladder and no bile to the alimentary canal. It is, indeed, chiefly in consequence of this parallel arrest of the organic functions presided over by the abdominal sympathetic nervous system that the primary seat of the disease in cholera, as in analogous diseases, has been referred to the digestive canal, for no corresponding arrest of function usually occurs in those organs which are remote from this nervous influence; and the analogy which has been traced in the preceding groups of cases tends, without exception, to this conclusion. But before any just estimate can be formed of the value of analogy in cholera, it is necessary that preconceived opinions in favour of a lung theory of the disease should be given up, so as to allow the attention for a time to be transferred from the outworks to the centre of organic life, for otherwise it would be impossible to give any physiological explanation of the fact that lactation continues and the urine is suppressed, whilst the condition of the brain itself is undisturbed; and although analogy can only be referred to for the purpose of supplying indirect evidence, yet, on the present occasion, this is so strongly in favour of cholera being primarily due to an affection of the sympathetic nervous system, developed through the medium of the digestive canal, as scarcely to need any further evidence to support it. It may, however, before concluding, be useful to state that such experimental evidence as the subject admits of strengthens the argument from analogy; and although evidence of this kind, when based on a protracted and complicated operation, is usually very questionable, there is some which appears to be free from any such objection, as, for example, Dr. Davey's[1]

[1] 'The Ganglionic Nervous System,' p. 196, 1858. The pressure of an aneurism, by interfering with the nervous supply to the small intestine, appears to have caused "excessive diarrhœa" in Dr. J. W. Ogle's case of aneurism of the superior mesenteric artery at St. George's Hospital. ('Path. Trans.,' vol. viii, pp. 168-170, 1857.)

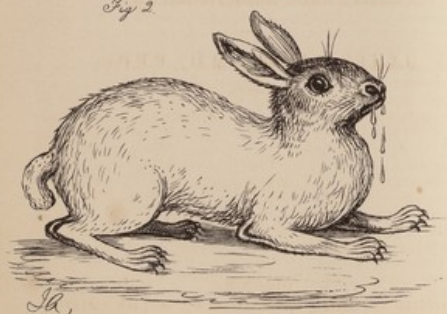
important observation, that "the fatal depression in cholera, consisting in the complete annihilation of the action of all the vital organs, may be at any time simulated by pressing the solar ganglion on the fore part of the bodies of the vertebræ over which it lies."

P.S.—Since the above was written some important evidence in support of the argument contained in this paper has been supplied by M. Moreau's experiments on section of the abdominal nerves in relation to intestinal flux. ('Comptes Rendus des Séances de l'Académie des Sciences,' tom. lxvi, pp. 554-557, 1868; cited in 'Medical Times and Gazette,' April 11, 1868, p. 397.)

Fig 1.

Under influence of genuine Battley's Solution of Opium

Fig 2.



Under influence of "Pritchard's Battley."

ON THE

DETECTION OF ACONITE

BY ITS

PHYSIOLOGICAL ACTION;

BEING

NOTES OF EXPERIMENTS MADE IN CONNECTION WITH THE TRIAL OF DR. E. W. PRITCHARD.

BY

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ON THE

DETECTION OF ACONITE

BY ITS

PHYSIOLOGICAL ACTION.

THE recent trial of Dr. Pritchard for the murder by poison of his wife and mother-in-law, is likely to take a high place in the *causes célèbres* of this country. Irrespective of those circumstances which distinguished the crime as peculiarly cruel and revolting, there existed in the singular combination of poisonous agents employed, and in the means used for their detection, considerations of special interest to the medical profession; and it is with the view of satisfying the numerous inquiries which have been addressed to us that we have prepared the present communication.

Besides conducting an independent examination of the organs of both ladies, similar to that committed to Dr. Douglas Maclagan of Edinburgh, and the results of which are fully described in Dr. Penny's reports to the Crown, and read by him in giving evidence at the trial, there was entrusted to Dr. Penny another, and a very extensive and difficult, investigation. He was required to report upon the nature and properties of a large number of articles of food—of various substances, labelled to indicate that they were officinal preparations of well-known drugs—and of various other articles, regarding the nature of which there was no clue. These were not delivered at one time, but were brought at irregular intervals up to a date very shortly preceding the criminal trial.

Among other articles undergoing examination, there were several phials, some of which were labelled and some not, containing "Tinct. Conii.," "Tinct. Digital.," "Tinct. Hyoscy.," "Tinct. Aconit.," "Battley's Sedative Solution," &c., &c., all of which were clearly and satisfactorily identified. The contents of the phial labelled "Battley's Solution," which, as shown at the trial,

had been found in Mrs. Taylor's pocket, was tested for mercury, antimony, and various other metallic poisons, and the results showed the presence of antimony in a soluble form, in the proportion of one grain and a half of tartarized antimony to the fluid ounce. The discovery of an extraneous admixture directed special attention to this Battley's solution, which we shall hereafter call "Pritchard's Battley," and led to its chemical examination for the leading organic alkaloids, other than those existing in genuine Battley. After a fruitless search for the presence of strychnia, veratria, conia, and hyoscyamia, a small quantity of the liquid was evaporated in a watch glass, and the soft extract applied to the tip of the tongue, when there was felt quickly supervening upon the taste of *genuine* Battley's solution a peculiar benumbing and tingling sensation, increasing in intensity for a short time, and persisting for some hours. In numerous comparative trials with genuine Battley's solution, obtained from various sources, it became very clearly evident that there was a something characteristic in the taste of the Pritchard Battley, and suspicion turned to the probability of an admixture of aconite. Observations made with an evaporated soft extract of Fleming's tincture of aconite, with and without a mixture of genuine Battley, gave sensations so perfectly corresponding, that strong presumptive evidence was now obtained of the presence of aconite in the suspected fluid.

By Stas's well-known process, and by other chemical means, more distinct indications of aconite were obtained; but the unreliable and altogether inconclusive character of all known processes in chemistry for the detection and unequivocal identification of certain vegetable poisons, especially when contained in organic mixtures, and also the necessity of husbanding what remained of the suspected liquid, were sufficient reasons for determining that no time or material should be wasted in further chemical analyses, and a series of experiments upon animals was therefore determined on and planned.

It was at this stage of the investigation that Dr. Penny, finding that he would not be able, unaided, to overtake so extensive an inquiry, owing to the limited time at his disposal, and having a part of the analysis still on hand, solicited the assistance of Dr. Adams. Ere this date, however, the list of witnesses against the prisoner had been closed and served, and hence Dr. Adams was precluded by the statutory limitations from appearing in evidence at the trial.

In publishing the notes and results of our observations and experiments, we wish it understood that we do not claim for them the character of a scientific or complete physiological investigation. They are intended simply to convey to the medical profession such details as may show the practical nature of the inquiry which we conducted, and at the same time enable a judgment to be formed by others as to the value of those conclusions at which we arrived as a result of that inquiry.

We have also to add, that the very limited time at our command before the trial, debarred us from attempting an exhaustive physiological course of experiments, and at the same time prevented us from making microscopic examinations, analyzing the blood, &c., &c.

In view of the fact that different kinds of animals are differently affected by the same agent, we resolved to restrict our observations to one class, and several satisfactory considerations led us to select the rabbit. It appeared to us of little moment, that the phenomena manifested in our trials with the rabbit should differ from those exhibited by the human subject when under the influence of the same agent. For our purpose it was sufficient if the action of the toxic agent was uniform and characteristic, when employed on the same animal.

Our object in these altogether practical experiments was twofold—first, to determine if the suspected liquid contained anything destructive to life, when employed in a dose which would have been safe, assuming it to contain no foreign substance; and second, to determine what that foreign substance was, of the presence of which we were already clearly satisfied.

The details of these experiments, although somewhat tedious, can scarcely be omitted without a corresponding loss of practical usefulness. For that which may seem unimportant to one observer, and perhaps only casually introduced in the record, becomes often to another the single or strongest point of interest, and the clue which may lead either to separate investigation or to another conclusion. It is not sufficient, unless for popular comprehension, to state that certain agents are destructive to life in certain doses, and that paralysis or convulsions is a common effect; but for satisfactory reference and comparison it is necessary to describe the exact form in which the agent has been employed, and the order and precise nature of the phenomena observed.

For several sufficient reasons we thought it advisable to make a number of concurrent experiments with atropia, veratria, conia, hyoscyamia, strychnia, digitalis, &c.; but as these do not properly come within the scope of our present communication, we shall not allude to them further than to say that some of them proved extremely interesting; and as our inquiry widened, there grew in our minds a conviction that the physiological test in its application to the detection of vegetable poisons presented a more inviting field for toxicologists than had hitherto been admitted,

and we hope at another date to present some of the conclusions we have reached regarding certain of the alkaloidal poisons. Meanwhile we restrict ourselves to the special investigation of Pritchard's Battley. The agents we employed were—

1. Genuine Battley's solution of opium procured from Murdoch Brothers, and taken from the same stock bottle out of which the phial containing Pritchard's Battley was filled.
2. Tincture of aconite (Fleming's).
3. Mixtures of tincture of aconite with genuine Battley in various proportions.
4. Pritchard's Battley.

These agents were prepared for the purposes of experiment, by being evaporated, either at a gentle heat, or by evaporation *in vacuo* over sulphuric acid, to a soft extract, which was then dissolved or suspended in a measured quantity of pure water.

In a number of experiments, the extract prepared from genuine Battley, and that from tincture of aconite, was either introduced into the stomach or dropped upon the eye; but while the characteristic effects were unequivocal, we were satisfied that so much more agitation and alarm were produced by the greater and more prolonged handling of the animal, that we soon abandoned this mode of operating. Moreover, we found that the quantity of the agent required to produce the same effects was more variable—the interval occurring before the symptoms became manifest more uncertain, and the duration of the experiment prolonged to so tedious a degree, that observation became difficult and less reliable. To these experiments we therefore only refer as corroborative of the phenomena otherwise generally observed.

In the very large proportion of instances we injected the agents beneath the skin, selecting the fold of integument at the flank or between the skin and muscles of the back. In this operation we employed a small graduated glass syringe, armed with a needle, perforated through its entire length; taking care in every instance to avoid injury of the large vessels and nerves, or penetration of any of the cavities.

This method of hypodermic injection is almost literally painless, and in execution is easy, rapid, and complete, while the action of like quantities of the same agent is better contrasted as regards the time within which the characteristic effects begin to be noticeable. These variations were generally within small limits, and seemed to bear a close relation to the age and strength of the animal.

The following table shows the number of experiments of which the results were systematically observed:—

1. GENUINE BATTLEY'S SOLUTION.

Introduced into stomach,.....	2	9
" " eye,.....	2	
" under skin,.....	5	

2. TINCTURE OF ACONITE (FLEMING'S).

Introduced into stomach,.....	3	8
" " eye, ...	3	
" under skin,.....	2	

3. GENUINE BATTLEY'S SOLUTION AND TINCTURE OF ACONITE (FLEMING'S), MIXED IN VARIOUS PROPORTIONS.

Introduced into stomach,...	2	13
" " eye,.....	1	
" under skin,.....10		

4. PRITCHARD'S BATTLEY.

Introduced under skin,..... 5

Of the facts observed in these experiments we shall now give a summary, referring for more precise details to the Appendix, where we record such a selection of experiments as will indicate the general character of the whole.

GENUINE BATTLEY'S SEDATIVE SOLUTION.

In one instance ten grains were introduced into the stomach. In another, 100 grains were employed. The quantities injected under the skin varied from ten to fifty grains. In no instance did death occur.

Within 10 minutes of the introduction of the poison by injection under the skin the animal seeks a prone position, lying fully extended on belly and chest—the head resting on the ground—the legs sprawling—the claws concealed (Fig. 1). If undisturbed, it inclines to retain this position, rarely shifting its place or attitude. When urged to motion, it crawls a few paces in a tortoise-like manner, but speedily subsides into its former passive, motionless condition. When lifted by the ears it struggles, and when laid on side it recovers its legs. A sharp noise or heavy footfall attracts its notice. The eyes remain open, and only tend to close at occasional long intervals. The pupils are dilated. The respiration is slow and gentle. In from 5 to 8 hours the effects begin to pass away, and in about 14 hours the animal eats freely, and exhibits vivacity in its movements.

In carrying out these trials with Battley's solution, an impression gained force in our minds that there is still much knowledge to be acquired, and much of error to be removed, regarding the action of the nervine poisons generally. We are led, therefore, to request particular attention to the fact that no convulsions, or tendency to such, was exhibited in any of the animals experimented upon with Battley's solution. This is opposed to common

belief, for, without specifying names, we may simply observe that every authority we have consulted mentions, in express terms, the occurrence of convulsions as a usual and characteristic action of opium in mammiferous animals, and in many instances rabbits are referred to as among the animals subjected to experiment. Such a mistake, as we hold it to be, may be accounted for in part by the circumstance that authors of systematic treatises cannot reasonably be expected to verify, by personal experience, the statements of observers of good repute, from whose writings quotations are usually made. Re-copied and re-transferred these statements are at last, in many instances, accepted as beyond cavil. In the case of opium, familiarity with the drug and the usual faithful description of its ordinary effects on man, have probably assisted in securing a too hasty acquiescence in the common statement of its effects on animals. Be this as it may, we have satisfied ourselves that, as regards rabbits, no convulsive seizures attend the action of opium. As Battley's liquor is a watery solution from which a certain portion of the drug is excluded, we made three experiments with laudanum, a soft extract of which, prepared by evaporation, as in the other instances, was employed. In one case we injected the extract of twenty grains by weight beneath the skin of the back of a six weeks' rabbit; in the second fifty grains, and in the third one hundred grains, in both cases on full-grown rabbits. The effects produced were identical with those caused by Battley's solution, with this exception, that while there was an equal indisposition on the part of the animal to move its position until stimulated and urged, there was a difference in the character of the progression of the animal along the floor. Under laudanum the animal moved with its customary leaping action, and seemed to have entire power of its limbs, while Battley's solution induced a condition in which the movements were of a crawling nature. But in no other respect was there any contrast.

TINCTURE OF ACONITE (FLEMING'S).

In one instance five grains, and in a second seven and a half grains, were introduced into the stomach; the animals recovered. Fifty grains introduced into the stomach caused death in 15 minutes. Half a grain dropped into the eye produced all the characteristic effects. Two grains injected under the skin resulted in recovery in 1 hour 15 minutes, and four grains applied in the same way caused death in 3 hours 15 minutes.

The symptoms vary in the order and in the rapidity of their production according to the strength of the dose. In a case proving fatal, and where there has been time for the development

of the effects, there is first an interval of restless uneasiness during which the animal shifts its position, and occasionally couches, with one or both hind legs extended laterally. It remains for a short interval in an attitude of expectancy or watchfulness, during which a twitching of the lips and a chewing action of the jaws is observed. Gradually the limbs become awkwardly stiff —the claws protruded, and the feet sprawling, as if grasping the floor—the animal seemingly steadying itself. The head is carried erect and somewhat retracted—the nostrils expanded. Occasional spasmodic movements of the ribs and belly occur as if retching.* Suddenly the animal staggers and rolls over, quickly regaining its feet. Saliva begins to flow from the mouth, and soon after peculiar chirping or stridulous cries are emitted (Fig. 2). The severe convulsions which quickly and invariably follow, are of an opisthotonic character, the limbs being extended in a straight line with the body—the back arched, and the head excessively drawn back. In a few seconds the convulsions subside, and the limbs become relaxed, the animal lying helplessly on its side. After a longer or shorter interval, it is then occasionally affected with an impulse to rush madly forward or make violent leaps, without regard to obstacles; but the motions are of an abortive paralytic character—the legs sprawling about in a floundering, jerking sort of action, altogether different from the usual leaping motion of the animal. After one or more convulsive seizures, it lies on its side with head retracted—nostrils distended—flanks heaving—the forepaws spread apart and held in the air. The respiration becomes very evidently laborious, is quite audible, and seems altogether diaphragmatic or abdominal. In this utterly helpless condition it is still sensible to stimuli; it winces when the cornea is touched, and is roused to momentary effort on pulling or squeezing the ears, legs, or tail. Salivation continues, but no longer accompanied by the twitching, chewing movements; the eyelids are half-closed or drooping; the pupil usually contracted. In this prostrate state, an interval of many minutes, or even of some hours, may elapse, during which occasional convulsive seizures may occur. Frequently, at the termination of such seizure, the urine is forcibly discharged. At length a strong convulsion takes place, and the animal dies. The limbs immediately relax, and the pupil begins to dilate. *Rigor mortis* is nearly complete in from 40 to 60 minutes.

GENUINE BATTLEY'S SOLUTION AND TINCTURE OF ACONITE.

Fleming's tincture of aconite was mixed with genuine Battley's solution in proportions varying from one of the former to from

* A rabbit does not vomit.

four to eighteen of the latter. In one instance death was caused by one grain mixed with nine; in another, two grains mixed with ten. In both instances the poison was injected beneath the skin.

There was caused by a mixture of these agents an evident blending of the symptoms produced by both, but the action of the aconite predominated. When, however, the Battley was employed in the large proportion of eighteen to one of the aconite, the conditions were reversed. When a full dose of both in a state of mixture was used, there was at first less of the active uneasiness than was noticed with the aconite alone, on the contrary, a stronger disposition to couch or rest was manifested; but when doing so, the head was invariably carried erect. On being urged to motion, the hobbling or crawling progression, as opposed to the spasmodic jerking action observed with aconite, was more strongly marked. The convulsive seizures did not, however, seem to be lessened in severity, but the intervals of relaxation were longer, and during these intervals the action of the Battley became evident.

PRITCHARD'S BATTLEY.

The symptoms are so closely similar to those of Battley's solution mixed with aconite, that, *nomine mutando*, the one will pass for the other. No possible distinction could be drawn from the appearances presented by an animal suffering from the effects of a dose of Pritchard's Battley and of another undergoing, at same time, the effects of a mixture of Battley and aconite.

We think it preferable to indicate thus shortly the action of Pritchard's Battley, instead of summarizing its effects, as we have done with aconite and Battley's solution respectively, because, in all experiments wherein vital action is exhibited, there will invariably be found some points of difference in the occurrence, order, or character of individual phenomena. We trust that the description we have given of the effects of aconite and of Battley's solution, when used alone, is sufficiently clear for the purpose of comparison and reference; and with that circumstance kept in view, we believe that the larger number and fuller details we have given, in the Appendix, of experiments with Pritchard's Battley, will enable our readers better to appreciate the nature of those points of similarity by which we ourselves were satisfied regarding the composition of Pritchard's Battley.

Referring, for example, to experiments No. 27 and No. 34, it will be seen that, although there was a difference in the strength of the animals operated on, probably also in the dose of the agent employed, these influencing, no doubt, the duration of the experiments, there was yet such a close correspondence in the general

effects that no essential difference in the character of the phenomena can be detected.

Having thus by clear and satisfactory evidence established the characteristic and essential differences in the effects produced on the rabbit by genuine Battley and Pritchard's Battley respectively; and having found that the action of Pritchard's Battley in a fatal dose corresponded in every respect with that of aconite, either alone or when mixed with genuine Battley; and bearing in mind the peculiar and distinctive sensation communicated to the organ of taste by the above liquids—we had no hesitation in arriving at the following conclusions:—

1st. That Pritchard's Battley contained a poisonous substance.

2nd. That that poisonous substance was aconite.

We may also state, that from a careful consideration and comparison of the intensity, the period of access, and the duration of the symptoms exhibited in our trials with the mixtures of tincture of aconite and of Battley's solution in known quantities, we were led to infer that the proportion of the former in Pritchard's Battley was from five to ten per cent. We disregarded the infinitesimal proportion of antimony discovered in Pritchard's Battley, being satisfied that it could not in any way influence the symptoms we witnessed.

Our conclusions were confirmed by the fact of our finding no effects analogous to those of aconite produced by any of the other agents (veratria, strychnia, &c., &c.) employed by us in the concurrent experiments already referred to, or by any other poisonous agent with the action of which we were familiar.

With the medical aspects of the criminal trial our inquiry had no necessary connection, and we, therefore, purposely avoid any commentary on the peculiar symptoms manifested by Mrs. Taylor and Mrs. Pritchard. Neither have we attempted to draw any analogy between the action of the poisonous agent as displayed upon animals and those produced on the human subject. These considerations form sides of the case altogether apart from those which we attempted to uncover, and were otherwise dealt with by the special medical and general evidence. That the unfortunate ladies were poisoned was clearly proved, and the prisoner ultimately admitted that he did put aconite into the Battley's solution. The detection of that aconite was the problem that our experiments were intended to solve; and although the confession of the criminal has no doubt aided the complete acquiescence of the public in the justice of the verdict, yet his admission or denial could not in any way influence our convictions, nor the conclusions to which we had already committed ourselves.

APPENDIX.

A.

TABLE OF EXPERIMENTS,

SHOWING PROPORTION OF AGENTS EMPLOYED, HOW INTRODUCED, AND RESULTS.

No.	Agent.	Introduced.	Results, &c.	
	BATTLEY'S SOLUTION.			
1	10 grains,	Into stomach.	Recovered.	Large rabbit.
2	100 "	"	"	"
3	2 "	Into eye.	"	"
4	10 "	"	"	"
5	10 "	Under skin.	"	Young rabbit.
6	10 "	"	"	Large rabbit.
7	20 "	"	"	"
8	40 "	"	"	"
9	50 "	"	"	"
	TINCTURE ACONITE.			
10	5 grains,	Into stomach.	Recovered.	Large rabbit.
11	7½ "	"	"	"
12	50 "	"	Death in 15 minutes.	"
13	¾ "	Into eye.	Recovered.	"
14	¾ "	"	"	"
15	2 "	"	"	"
16	2 "	Under skin.	"	"
17	4 "	"	Death in 3 hrs. 30 min.	"
	MIXTURE OF BATTLEY & ACONITE.			
18	Battley, 8 grains; Aconite, 2 grains.	Into stomach.	Recovered.	Large rabbit.
19	" 10 " " 2 "	"	"	"
20	" 4 " " 1 "	Into eye.	"	Young rabbit.
21	" 2½ " " ½ "	Under skin.	"	Large rabbit.
22	" 9 " " 1 "	"	"	"
23	" 9 " " 1 "	"	Death in 44 minutes.	Young rabbit.
24	" 18 " " 1½ "	"	Recovered.	Large rabbit.
25	" 18 " " 1 "	"	"	"
26	" 8 " " 2 "	"	Death in 49 minutes	Full grown buck rabbit in strong condition.
27	" 36 " " 4 "	"	Death in 15 minutes.	Large rabbit.
28	" 5 " " 5 "	"	Death in 13 minutes.	"
29	" 45 " " 5 "	"	Death in 6½ minutes.	"
30	" 50 " " 10 "	"	Death in 2 minutes.	Young rabbit.
	FRITCHARD'S BATTLEY.			
31	10 grains,	Under skin.	Recovered.	Young rabbit.
32	16 "	"	"	Large rabbit.
33	20 "	"	"	"
34	40 "	"	Death in 53 minutes.	"
35	50 "	"	Death in 22 minutes.	"

B.

SELECTED EXPERIMENTS.

BATTLEY'S SOLUTION OF OPIUM.

Expt. No. 6.—Ten grains Battley's solution was injected in the fold of integument of right flank of a full-grown buck rabbit. Within 10 minutes the animal lay down prone and fully extended on its belly and chest, the head resting on the ground, the hind legs apart and sprawling. At the end of 45 minutes it continued in the same position, capable of being roused, but evidently indisposed to exertion. When urged to motion it would crawl a few paces in a tortoise-like manner, never attempting the leaping motion characteristic of the animal's ordinary movements, and quickly subsiding into a torpid or lethargic condition. When lifted by the ears it struggled actively, and it resisted being laid on its side. The eyes remained widely open, the pupils dilated. The respiration very gentle. Watched closely for three hours, no change in condition was observed. At the end of 6 hours it was recovering its natural manner, and moved with a leaping action, but was still somewhat lethargic.

Expt. No. 8.—Forty grains Battley's solution was injected in the fold of integument of the left flank of a full-grown buck rabbit. It moved about with ordinary activity for about 5 minutes, but within 7 minutes lay down, with the head resting on the ground, quite torpid and helpless; the whole body subsided, flattened out and extended at full length; the tail flaccid and drooping, but the ears erect, the claws retracted. (Fig. 1.) The entire frame seemed thoroughly relaxed and prostrate, and seeking the support of the floor—a condition, in short, of perfect inanity. At the end of 22 minutes, as it showed no disposition to move, although evidently wide awake, as is eyes were fully expanded and it noticed and watched any threatening gesture applied to it, it was urged to motion and it then *crawled* a few paces like a tortoise. It permitted itself to be shoved along the floor readily enough, but would not lie on its side when so placed, invariably recovering its feet when the attempt was made. The pupils seemed rather dilated; the respiration was slow and gentle. Watched closely for 2 hours, no change was observed. Next day it had quite recovered.

TINCTURE OF ACONITE (FLEMING'S).

Expt. No. 16.—Two grains by weight of Fleming's tincture of aconite, was injected under the skin in the left flank of a full-grown doe rabbit. A small quantity was spilled in the operation. The animal moved actively and in its usual manner for 3

minutes, when it couched, but almost instantly got up again, showing signs of uneasiness. During the next 5 minutes it was very restless, shifting its position as if under sudden impulses. At the end of 10 minutes it placed itself in a position half couching, but the body not resting on the ground; the head was retracted, the paws spread as if grasping the floor; the tail incurved, and the whole attitude expressive of alarm. (Fig. 2.) An active twitching of the lips and chewing motion of the jaws began. After 13 minutes, faint gasping cries were uttered, the breathing became laborious and performed with deep fetches, and distinctly audible. After 38 minutes, copious salivation began. The animal frequently shifted its position, generally with one movement, and only a few inches at a time. The head was kept continually upright, and somewhat retracted. After 48 minutes, the twitching and chewing motion of the jaws and lips, the salivation, the occasional utterance of faint cries, and the retracted position of the head, continued prominent symptoms. But the animal's condition was otherwise improving—the breathing was less laboured, and at intervals it ran about actively, coming occasionally to an abrupt stop, sitting up on its haunches, and carrying on actively the chewing motions for a few seconds. After one hour and a quarter it had nearly recovered its ordinary condition.

Expt. No. 17.—Four grains Fleming's tincture of aconite, was injected between the skin and muscles of the back of a large brown buck rabbit. It immediately displayed lively agitation, shifting its position incessantly, and frequently licking the wound made in its back by the injecting needle, which this time seemed to have found an extra-sensitive spot. At the end of 8 minutes, it fell or threw itself prostrate on its flank, with hind legs extended far from body, but speedily got up again and continued its active uneasy movements. After 13 minutes, it placed itself in the peculiar position described in preceding experiment; the twitching of jaws commenced and the head was retracted, with at same time a marked aspect of alarm. After 17 minutes, it uttered choking cries—the nostrils expanded, and the respiration was performed with laborious catches. The motions of the animal continued untiringly active. After 22 minutes, active salivation was manifest. After 35 minutes, there was extreme retraction of head. The animal continued still actively moving about, but in a paralyzed manner, occasionally staggering and falling on its flank. At intervals it seemed to be delirious, and made violent struggling efforts to force itself through openings in the basket cage in which it was confined. These efforts were made in spasmodic rushes, sometimes a spring being made from the prone posture. When successful in forcing itself through an opening the animal darted wildly forward with a floundering staggering movement for

several yards, occasionally striking in a regardless manner against obstacles. The head continued violently retracted, the nostrils expanded, but the breathing was becoming gradually slower and feebler, and still performed in a laborious manner. At intervals it lay on its side in a very prostrate condition. After 57 minutes, there occurred marked opisthotonic convulsions, followed by extreme prostration, the animal lying on its side—the limbs and muscles of chest relaxed, but the head always stiffly retracted. After 60 minutes, while lying exhausted it was quite sensitive to the ears and legs being pulled or squeezed hard, and under continued and active rousing it was capable of making efforts to get away, but quite incapable of regaining the upright posture. After 1 hour 35 minutes, it continued lying on its side in the same helpless condition—the breathing slow, distressed, and quite audible. After 2 hours 30 minutes, it discharged urine with force. After 2 hours 35 minutes, sharp convulsions occurred, at termination of which the animal rolled over on its back—the fore paws extended in air, the head stiffly retracted, the nostrils expanded, the breathing laborious. After 3 hours 25 minutes, no change in condition, excepting that the respiration became gradually feebler and slower, being 10 per minute. After 3 hours 30 minutes, violent opisthotonic convulsions occurred, accompanied with choking cries, and followed by death. The limbs immediately became flaccid, and the pupil, which was contracted, began to dilate. *Rigor mortis* was complete, 40 minutes after death.

BATTLEY'S SOLUTION, WITH TINCTURE OF ACONITE.

Expt. No. 24.—Eighteen grains of Battley's solution, mixed with one and a half grains Fleming's tincture of aconite, both by weight, were injected under the skin of the back of a large and very strong buck rabbit. It moved about with customary activity for 5 minutes, when it lay prone on its belly. During the next 25 minutes, it occasionally shifted its position in an uneasy manner. After 30 minutes, it lay in same position, disinclined to move, breathing slowly, audibly, and with deep fetches. After 40 minutes, retching movements occurred. The head was maintained in a retracted position. Occasionally it moved a few paces in a crawling manner—staggered, and then came to a stop. After 50 minutes, salivation was observed, but no twitching of lips or chewing movements of jaw. At times spasmodic twitches affected the limbs, and the animal braced itself, as if to avoid falling. After 65 minutes, the breathing was very laborious and slow, being 10 per minute; the nostrils were widely expanded, and the head retracted. After 85 minutes, salivation continued, and the animal was much disinclined to move, but, when lifted, used its muscles

actively. After 90 minutes, when urged to motion, it attempted to do so in the customary manner, by leaps, but staggered, and was evidently paralyzed, particularly in the hind legs. Its manner indicated distress—the breathing was still slow—the head carried erect. After 2 hours, was evidently improving. After 5 hours, was much improved, and capable of moving about in its natural manner.

Expt. No. 27.—Thirty-six grains Battley's solution, with four grains Fleming's tincture, both by weight, was injected under the skin of the back in a small-sized doe rabbit. The animal moved about with fidgety celerity for four minutes, when chewing motions of the jaw took place, with twitchings of the lips. After 7 minutes, the position was half sitting, half crouching, with head erect and retracted—a look of watchful expectancy. A slight degree of salivation was noticed, and occasional retching efforts were made. After 9 minutes, great uneasiness was manifest—the head was strongly retracted, the eyes occasionally closing, stridulous or choking cries were emitted at intervals, and from time to time the animal moved a few paces in a staggering manner, trailing the hind legs. After 12 minutes, it lay prostrate on the side and back—the fore paws raised in the air—the head upturned and retracted—the nostrils widely expanded—the breathing laborious, audible, and performed with deep fetches—the limbs and entire body, with exception of neck, very relaxed. At the 13th minute, there occurred severe opisthotonic convulsions, at intervals the animal bounding upwards in the air like a fish—then propelling itself in darting movements, while still lying on the side, round the interior of the basket cage—then lying with hind legs extended stiffly in a line with the body—the back arched and the head retracted. This convulsive attack lasted one minute and a half, after which followed half a minute of utter prostration, during which the urine was forcibly discharged, and immediately after the animal died quietly—the pupils being contracted during the convulsions, and becoming dilated in the short interval of relaxation preceding death, and again contracting for some time after death; 15 minutes precisely elapsed from beginning of the experiment till death. After death the limbs were quite relaxed. *Rigor mortis* was complete, 40 minutes after death.

PRITCHARD'S BATTLEY.

Expt. No. 32.—Ten grains by weight of Pritchard's Battley was injected under the integument of both flanks of a full-grown buck rabbit, a very small quantity being lost in the operation. During 5 minutes the animal seemed very uneasy, inclining frequently to crawl, but almost immediately, and as if moved by

a new impulse, getting up and shifting to a fresh position. Within the next few minutes it lay down twice very suddenly, with both hind legs freely extended sideways, and carrying the head very erect. After 22 minutes, a strongly marked twitching of the lips and a chewing motion of the jaws began. The animal at same time placed itself in what seemed an attitude of expectancy, or watchfulness—half sitting on its haunches, the fore legs spread in front, and the paws of both fore and hind legs somewhat retracted, so that the weight of the body was supported on the heel, or palmar surface of the feet, the claws protruded a little as if to grasp the floor, the head was thrown back, and the eye expressed alarm. After 23 minutes, it suddenly staggered and fell on its side. Retching efforts now became frequent, and respiration seemed difficult. After 32 minutes, saliva began to flow rather abundantly, and the chewing action of jaws continued actively. At this time the pupils were contracted, the eyelids occasionally drooping. After 35 minutes, the breathing was very laborious, and performed with deep catches. The animal still rested on its side, seemingly unable to vary its position, and when shifted from one to the other side, it retained the position in which it was placed. The head was carried erect and retracted, and the nostrils expanded widely, in correspondence with the laborious respiratory efforts. After 55 minutes, a tetanic convulsion of short duration occurred. At end of one hour, the animal still lay on its side, or flank, but it now made frequent efforts to raise itself, using with greatest power the fore legs. In these efforts it shifted its position and moved in a retrograde manner, as if shrinking from some offensive or threatening object. On being urged to motion, it made staggering and awkward efforts to hobble away. At the end of 3¼ hours, it continued in a resting attitude, prone on belly, and with head retracted; the breathing very slow; evidently disinclined to move. The pupils continued contracted, the eyelids occasionally drooping and closing. After 8 hours, was so far recovered as to take food.

Expt. No. 31.—Ten grains by weight of Pritchard's Battley was injected between the skin and muscles of back of a rabbit six weeks old. Within a few seconds it exhibited marked uneasiness, frequently and in an agitated manner shifting its position. After 3 minutes it lay prone at full length, the hind legs extended laterally, the head erect. After 6 minutes it was roused to exertion, when it crawled about for a few seconds. After 8 minutes the peculiar twitching of lips and chewing action of the jaws, as in foregoing experiment, were observed. Almost instantly after, severe retching movements began, and the head was strongly retracted. After 11 minutes the animal lay prone and fully extended, the breathing laborious and catching, the head

strongly retracted, and the nostrils widely expanding in correspondence with the respiratory movements. After 13 minutes it uttered piteous stridulous, or choking cries, made frequent convulsive leaps, but abortive as if from paralysis, and then lay on its side utterly prostrate. After 15 minutes violent tetanic convulsions came on, the limbs extended in straight line with body, the back arched, and the head extremely retracted. The choking cries became very frequent, and at intervals, as the spasms relaxed, the animal made frantic leaps, bounding from its side, like a stranded fish, upwards. The pupil at this time was contracted, and the cornea sensitive to stimuli. Between the 15th and 19th minutes there was an interval of extreme prostration, during which the limbs were very flaccid and relaxed, and the animal lay in whatever position it was placed. The breathing continued distressingly laborious, but no cries were uttered. At the 26th minute the piteous cries were renewed, and the opisthotonic convulsions recurred with extreme severity, and continued with little interval for about 10 minutes; the animal at times rolling over on its side or back, with fore legs extended in the air, or making struggling abortive leaps, and at intervals of about half a minute lying prostrate, and breathing heavingly, and with evident distress. At this time the animal was sensitive to stimuli, and made efforts when the ears were pulled, or the feet squeezed, or the eyeballs touched. After an hour and three quarters, its condition began to improve, and it was able to crawl a little when urged, but it could only progress a few inches at a time, and that in a crawling hobbling manner. After 2 hours 20 minutes its condition was so far improved that when rolled over on its back it regained its legs, but it was still unable to change its position further than a few inches. It inclined to lie couched, breathing slowly and with effort, the head retracted so that the face looked directly upwards, the eyelids drooping and inclining to close at intervals. After 3½ hours there occurred little change, excepting that there was now more of a torpid condition, and that all tendency to convulsive seizures had passed away. No considerable degree of recovery or return of the natural action of the animal was noticeable until fully 6 hours after the beginning of the experiment.

Expt. No. 33.—Twenty grains of Pritchard's Battley was injected under the skin of flank of a large and strong full-grown buck rabbit. In a few minutes afterwards it moved about in an uneasy manner, but within 5 minutes it placed itself in a semi-couching attitude, braced up, with head erect and thrown back as if watchful and expectant; the fore paws as if grasping the floor, the toes spread and a little upturned, the claws protruding. After 7 minutes it fell, or seemed suddenly to throw itself on its side, in a staggering, paralyzed manner. During the next 3

minutes it moved occasionally with a hobbling kind of effort, lying down from time to time prone on its belly—its breathing performed with deep fetching action and very slowly. After 11 minutes the peculiar twitching of lips and chewing action of jaws began. The pupils at this time were markedly dilated. After 15 minutes there occurred frequent spasmodic retching. After 20 minutes the animal sought the prone posture, was disinclined to move, even when disturbed and urged; its legs lay sprawling, and, when one of them was drawn from under it, it remained, and there was no effort to regain its former position. The chewing action of the jaws occurred with occasional intervals of cessation. After 40 minutes active salivation commenced. Watched closely for about 2 hours, no further change was observed, and at end of 6 hours it still seemed very ill and refused food; next day it had quite recovered.

Expt. No. 34.—Forty grains of Pritchard's Battley was injected under the skin, partly of both flanks, of a large and very strong full-grown buck rabbit. Within 5 minutes, and after showing active uneasiness, there began a chewing motion of jaws and twitching of lips, the head was strongly retracted, the respiration laborious, the nostrils expanding in correspondence with breathing efforts. During next 2 minutes the animal shifted, and turned its position frequently, but without removing from the same spot of ground. After 7 minutes peculiar stridulous cries were emitted. At end of 8 minutes it staggered and fell, and was unable to recover its legs; its movements evidently quite paralyzed, but the greatest debility was manifest in the hind legs. After 10 minutes there occurred frequent retching efforts. After 13 minutes it made several violent and convulsive leaps, bounding quite from its feet upwards in the air, and these were immediately followed by strong opisthotonic spasms. The pupil at this time was much contracted. This convulsed condition continued 5 minutes, after which the animal lay helplessly on its side, the fore paws extended in the air, the nostrils expanded, the breathing distressingly laborious. The pupils were now dilated, the cornea was sensitive, and the animal winced when the paws were squeezed. After 52 minutes there had occurred no material change, excepting that the breathing was becoming gradually feebler. At the 53rd minute there came on very violent convulsions of an opisthotonic character, during which the animal propelled itself in darting movements twice round the large basket cage in which it was confined; the urine was discharged with force, the limbs suddenly relaxed, and the animal was dead. At the instant of death the pupils were contracted, but gradually began to dilate; the *rigor mortis* began, and was well marked in about 40 minutes.

C.

EXPERIMENTS ON RABBITS IN EDINBURGH.

After the foregoing investigation was completed, it was considered desirable by the Crown authorities that the effects produced upon rabbits by "Pritchard's Battley," and by genuine Battley, should, for the purpose of evidence, be seen by the other medical and scientific witnesses engaged in the case; and with this view Dr. Penny was requested to carry out, conjointly with Dr. Christison, Dr. Maclagan, Dr. Littlejohn, and Dr. Gamgee, in Edinburgh, the requisite experiments with these liquids, and with tincture of aconite.

The experiments, which were four in number, were made with full-grown rabbits, and by injection, as in our trials; but as they formed no part of our inquiry, and were not distinguished by any results different from what we had witnessed in Glasgow, we have not deemed it necessary to give the details of them in a separate form. It will be sufficient to state generally, that the effects in the case of each agent—though more intense in degree and shorter in duration, arising from the largeness of the dose, and therefore not permitting time for the development of some of the minor symptoms—coincided in every essential particular with those exhibited in our own trials, and completely corroborated the conclusions at which we had arrived.

ADDENDA.

CHEMICAL REPORTS.

Report of Analysis in the Case of the Death of Mrs. Pritchard.

"Andersonian University, Glasgow, *9th May*, 1865.

"On Monday, the 10th of April last, I received from Dr. Douglas Maclagan, at his laboratory in Edinburgh, the following articles, all of which were certified to have been taken from the body of Mrs. Pritchard:—

"No. 1. Pyloric half of stomach.
" 2. Nearly half of kidney. } These four articles were contained
" 3. Portion of rectum. } in a stoneware jar.
" 4. Portion of spleen. }
" 5. Portion of liver in a glass jar.
" 6. Portion of brain in a glass jar.
" 7. Portion of heart in a glass bottle.
" 8. Portion of blood in a glass bottle.
" 9. 225 grains of dried contents of intestines in a glass bottle.

"The several vessels containing these articles were securely closed, and duly labelled. I brought them direct to Glasgow on the day referred to, and, in accordance with instructions from the Crown-Agent, Edinburgh, I have, at my own laboratory, carefully analyzed and chemically examined each and all of the said articles, for the purpose of ascertaining whether they contained any poisonous substance.

Dried Contents of Intestines.—The investigation was commenced with the contents of the intestines. From the information which I received, my attention was particularly directed to the detection of antimony; but deeming it desirable to search for the presence of other metallic poisons, I subjected a portion of the said contents to the usual course of qualitative analysis for the detection of various metals of a poisonous nature. The results of this exhaustive examination gave distinct indications of the presence of antimony and mercury. For the purpose of establishing unequivocally the presence of these metals, and at the same time of estimating their quantities respectively, the following experiments were then carried out:—A known quantity of the said contents was dissolved with the usual precautions in hydrochloric acid, with the addition of chlorate of potash, and the solution being properly diluted with water, was subjected to the action of sulphuretted hydrogen gas. An abundant black precipitate was obtained, which, by proper treatment, was separated into sulphide of antimony and sulphide of mercury. The sulphide of antimony, which was obtained of a fine orange-red colour, was washed, dried, and weighed. Its weight corresponded to a quantity of metallic antimony equal to 2·1 grains in one thousand parts of the dried contents of the intestines. The same sulphide was found to be readily soluble in sulphide of ammonium, and also in hydrochloric acid, and the acid solution, when poured into water, give a white precipitate, and when boiled with copper-ribbon, deposited a violet-coloured coating on the surface of the copper. The coated copper, on being heated in a glass tube, gave no distinct crystalline sublimate. All these results are eminently characteristic of sulphide of antimony when thus treated. The sulphide of mercury was black; it was dissolved in nitric and hydrochloric acids, and the solution, being appropriately prepared, was treated with chloride of tin. A precipitate of metallic mercury was obtained, which, after being suitably washed and dried, was found to correspond to three grains in one thousand grains of the dried contents. A portion of this precipitate, on being heated in a dry glass tube, gave a sublimate of mercury in brilliant and

mirror-like globules. Another portion was dissolved in nitric and hydrochloric acids, and the solution, after the removal of the excess of acid, was tested with caustic, potash, ammonia, and iodide of potassium, and with other reagents and methods for the detection of mercury. In every case the peculiar reaction of that metal was satisfactorily produced. In order to corroborate the results of the foregoing experiments, another portion of the said contents of the intestines was subjected to Reinsch's process, and this was supplemented by Marsh's process. By the former process copper-foil was coated with a deposit which presented the peculiar violet colour and the general appearance of metallic antimony; and, by continuing the process till the copper foil ceased to be coated and the liquid was exhausted of separable matter, pieces of the copper foil were obtained with a grey coating, which, on being rubbed, became silvery and lustrous, like metallic mercury when similarly deposited. The coated copper was then digested in an aqueous solution of pure potash, and after being well washed and dried, it was cautiously heated in a small tube. A sublimate of metallic mercury in minute lustrous globules was obtained; and this sublimate, when dissolved in the proper acids, yielded with the well-known tests the chemical reactions of metallic mercury. The potash solution from the coated copper was then treated in the usual manner for the separation of antimony in the form of the orange-red sulphide, which, when collected and weighed, was found to correspond very closely with the proportion obtained by the process previously described. The sulphide of antimony was soluble in sulphide of ammonium and in hydrochloric acid. The solution in hydrochloric acid gave a white precipitate when poured into water, and on being subjected to Marsh's process, deposited on a porcelain slab the characteristic stains of metallic antimony. In another experiment, a portion of the said contents was distilled with concentrated hydrochloric acid, and antimony was detected in the distillate. With a view of ascertaning whether the antimony and mercury existed in a form soluble in water in the said contents of the intestines, a portion of these was macerated in distilled water, and the solution carefully tested for both metals. The presence of antimony was distinctly detected, but no mercury. The said contents were also examined by Stas's method for aconite, morphia, and other organic poisons, but not the slighest evidence of the presence of such poisons was obtained.

Stomach.—The stomach was analyzed by the same methods as those applied to the dried contents of the intestines. It yielded antimony in appreciable proportions, but no mercury. The quantity of antimony obtained was equal to ·05 of a grain in one thousand parts. The stomach was also minutely examined for morphia and aconite, but not a trace of these substances was obtained.

Liver.—The liver was found to contain antimony, but no mercury. The proportion of antimony amounted to one-tenth of a grain in one thousand grains.

Spleen.—The spleen yielded antimony in about the same proportion as that found in the liver, and it also contained mercury in well-marked quantity.

Kidney.—The kidney yielded about the same proportion of antimony as the liver, and it was also found to contain an extremely minute trace of mercury.

Heart.—The heart yielded antimony in a proportion rather larger than that found in the liver. It also contained mercury in smaller quantity than the spleen.

Brain.—The brain contained antimony in less quantity than the liver, but it yielded no mercury.

Blood.—The blood contained a small quantity, and also a faint trace of mercury.

Rectum.—The rectum yielded antimony, but in less quantity than the liver. It afforded no indications of mercury.

Having deliberately considered the results of my experiments upon the articles subjected to analysis, I have arrived at the following conclusions:—

1st, That all the parts of the body examined by me contained antimony. 2nd, That in the dried contents of the intestines the antimony was partly in a form soluble in water, and most likely in the state of tartar emetic or tartarized antimony. In the liver, kidney, and other viscera, the antimony was deposited in a state insoluble in water. 3rd, That the contents of the intestines contained the largest proportion of antimony, next the heart, then the liver, kidney, and spleen; less in the stomach; and the smallest quantity in the rectum, brain, and blood. Not knowing the total weight either of the contents of the intestines, or of the several organs here enumerated, I was unable to calculate the total quantity of antimony in these matters, either separately or conjoined. 4th, That the contents of the intestines, the spleen, the heart, the blood, and the kidney, contained mercury; but that none of this metal was present in the liver, stomach, rectum, and brain. That, in all these matters, the mercury was in a state insoluble in water; and this result is quite consistent with the known property of mercury to form insoluble combinations with animal substances, even though it had been taken or administered in a soluble form during life. 5th, That the largest quantity of mercury was contained in the contents of the intestines, next in the spleen and heart, and extremely minute traces in the blood and kidney. 6th, That the presence of antimony and mercury in the contents of the intestines, indicates that these metals were being passed from the deceased up to the time of death. 7th, That no other metallic poison was contained in the matter examined. 8th, That no aconite, morphia, or other vegetable poison, discoverable by chemical processes, was contained either in the contents of the intestines, or in the stomach. 9th, Not having detected any organic poison, either in the said contents of the intestines or in the stomach, it was not necessary to examine the other articles for such poisons, and more especially as the quantities of these matters received for analysis were too small to hold out any prospect of a successful result.

All this I certify on soul and conscience.

Glasgow, *9th May*, 1865. Frederick Penny.

Report of Analysis in the Case of the Death of Mrs. Taylor.

Andersonian University, Glasgow, *9th May*, 1865.

On the same day and occasion that I received the articles in the case of the death of Mrs. Pritchard, Dr. Douglas Maclagan delivered to me the following articles, certified to have been taken from the body of Mrs. Taylor:—1. Portion of liver in stoneware jar; 2. Portion of stomach in glass bottle; 3. Portion of heart in glass bottle; 4. One kidney in glass bottle; 5. Portion of rectum in glass bottle; 6. Portion of blood in glass bottle; 7. 100 grains of dried contents of intestines.

The vessels containing these articles were securely closed and duly labelled, and were, on the day referred to, brought by me direct to Glasgow.

I have subjected all the articles above enumerated to a course of analysis and chemical examination similar to that applied to the articles in the case of Mrs. Pritchard. The following were the results obtained:—

Liver.—In the liver the presence of antimony was unequivocally detected, and a quantitative estimation gave ·047 of a grain in 1000 grains of this organ. A careful analysis was also made for the presence of mercury, but not the slightest trace was detected.

Stomach.—The stomach yielded about the same proportion of antimony as that found in the liver. No mercury was detected. The stomach was also minutely examined by Stas's process for aconite and morphia, but not a trace of these poisonous alkaloids was obtained.

Heart.—The heart was found to contain antimony in less proportion than the liver. It yielded no mercury.

Kidney.—The kidney yielded about the same quantity of antimony as the heart. It gave a marked quantity of mercury.

Rectum.—The rectum gave antimony, but no mercury.

Blood.—In the blood, antimony was detected in rather larger proportion than in the heart. No mercury was detected.

Dried Contents of Intestines.—In the dried contents of the intestines, antimony was found to the extent of ·583 parts in 1000 parts by weight. It was partly present in a form soluble in water. No mercury was detected. The said contents were also carefully analyzed for aconite and morphia, but no evidence of the presence of these poisons were obtained.

From a careful consideration of the results of the analysis and examination of the above-named articles I am clearly of opinion that they are conclusive in showing:—1st, That all the articles subjected to analysis contained antimony. 2nd, That the dried contents of the intestines contained the largest proportion of antimony; next, the liver and stomach; then the blood, and in less quantity in the heart, kidney, and rectum. 3rd, That part of the antimony in the contents of the intestines is in a form soluble in water. 4th, That the kidney was the only article in which mercury was detected. 5th, That neither the stomach nor the contents of the intestines contained aconite or morphia in quantity sufficient to be detected by known chemical processes. 6th, That the articles subjected to analysis contained no other metallic poison than antimony and mercury as reported above.

To the truth of this report I hereby certify on soul and conscience.

Glasgow, 9*th May*, 1865. Frederick Penny.

Report of Analysis of certain Articles referred to in the case of Dr. Pritchard.

Andersonian University, Glasgow, 17*th May*, 1865.

On Thursday, the 13th of April last, Alexander M'Call, Superintendent of Police, delivered to me, at my laboratory, the following productions, having sealed labels attached, referring to the case of Dr. Pritchard:—A glass bottle, labelled "Battley's Sedative Solution," [B.] A bundle of seven small paper packages, [C.] A quart wine bottle, containing ginger wine, [D.] A small glass vial, containing a white powder, [E.] Three small vials, two corks, and one stopper, securely tied together, [F.] Six small vials and six corks, attached with string, [I.] On the same day and occasion, John Murray delivered to me a paper package, having labels attached, marked A, and containing tapioca. On Friday, the 21st April last, Alexander M'Call delivered to me a small glass phial, with label attached, marked G, and also a piece of cheese, marked H. On Thursday, the 11th inst., John Murray delivered to me a paper package, with label attached, marked K, and containing tapioca. In accordance with instructions received from John Gemmel, Esq., Procurator-Fiscal, I have made a careful analysis and chemical examination of the contents of the several productions above enumerated. My experiments and investigations gave me the following results, which, for the facility of reference, are reported in alphabetical order:—This paper package [A] contained 2850 grains of tapioca. The presence of antimony, in the form of tartarized antimony, was unequivocally detected. Its amount was found to be equal to 4·62 grains in the pound of tapioca. Not a trace of mercury was detected. This bottle [B] contained one ounce and five drachms of a dark brown liquid, having the odour and general appearance of Battley's solution of opium. It was found to contain an appreciable quantity of antimony in a soluble form. The amount was equal to 1·5 grain per fluid ounce of the liquid. It contained no mercury. (I am at present engaged in examining it for other substances.) The seven paper packages [C] comprised in this production were marked No. 1 to No. 7 inclusive. No. 1 contained a small lump of crystallized nitrate of silver, weighing 16·5 grains. It contained no antimony. No. 2 contained 132 grains of cummin seed in powder. Neither antimony nor mercury was found in it. No. 3 contained 143 grains of sugar of lead. Nothing extraneous was detected. No. 4. The contents of this package consisted of a mixture of mercury and chalk, weighing together 6·5 grains, and it was evidently the medical preparation called "Hydrargyrum c. Creta." No antimony was found in it. No. 5 contained a lump of opium, weighing 110 grains. No. 6 contained 13·5 grains of morphia, contaminated with a small quantity of nitrate of silver, which, from the appearance of the paper package, had manifestly enfiltered accidentally from without. No. 7 contained 1350 grains of a white, gritty, crystalline powder, which was found to have all the physical and chemical properties of sugar of milk. It was carefully tested for mercury, antimony, and other substances, but the results were entirely negative. This bottle [D] contained 18 fluid ounces of ginger wine. No antimony or mercury was detected. This phial [E] contained 3·5 grains of a white powder, which was found by analysis to be tartarized antimony. The three phials [F] included in this production were labelled respectively 1, 2, and 3. No. 1 contained one ounce and three drachms of tincture of conium. No. 2 contained five drops of the same tincture. No. 3 contained two and one half drachms of the same preparation. This phial [G] contained nine drachms and a half of a light yellow-coloured liquid, having the taste and odour of cinnamon, and consisting of a mixture of medicinal substances. It contained no antimony and no mercury. This cheese [H] was tested for antimony and mercury, but no evidence of the presence of these metals was obtained. This production [I] included six small phials, which were found to contain as follows:—No. 1. Four drops of tincture of aconite; No. 2. Twelve drops of the same tincture. No. 3. Thirty drops of the tincture of conium. No. 4. Fourteen drops of the tincture of conium. No. 5. Empty. No. 6. Nine drops of the tincture of digitalis. This paper package [K] contained 1695 grains of tapioca. Not the least trace of either antimony or mercury was detected in this tapioca.

All this I certify on soul and conscience.

17*th May*, 1865. Frederick Penny.

Report of Analysis of certain Articles referring to the Case of Dr. Pritchard.

Andersonian, Glasgow, 19*th May*, 1865.

This is to certify that I have subjected to careful analysis and chemical examination the following articles, which were delivered to me on the 15th inst. by Alexander M'Call:—No. 1. A brownish-coloured and turbid liquid, measuring three fluid ounces, contained in a glass bottle, labelled chloroform. It was tested for antimony and mercury, but not a trace of either metal was detected. It contained no aconite. No. 2. A white crystalline powder, contained in a small cylindrical wooden box, with screw cover. It weighed 15·5 grains, and was found to consist of a mixture of tartarized antimony and arsenious acid (that is, the common poison of arsenic) in nearly equal proportions by weight. No. 3. About ten drops of colourless liquid, contained in a quart wine bottle. It was found to be an aqueous solution of corrosive sublimate. No. 4. (A.) A white powder, contained in a circular red pasteboard box. It weighed five grains, and was found to be calomel. No. 4. (B.) A white powder, weighing 35 grains, contained in a green pasteboard box. It was found to be tartarized antimony. All the productions containing the articles subjected to analysis were securely closed, and had sealed labels attached.

Frederick Penny.

MEDICO-CHIRURGICAL SOCIETY OF GLASGOW.

Tuesday, 11th July, 1865.

DR. ADAMS ON THE DETECTION OF ACONITE.

Dr. Adams read a paper entitled "Notes of experiments made with a view of determining by a physiological test the presence of aconite in a suspected liquid."

The *Chairman*, after thanking Dr. Adams for bringing this most interesting subject before the Society, proceeded to say—It is novel, I have no doubt, to most of us. The experiments are extremely instructive, and I think open up an entirely new field of inquiry.

Dr. Steven—I think it would be well if Dr. Adams would let us know the effect of Battley combined with aconite, as well as that of aconite alone.

Dr. Adams—I have already stated that by a mixture of both there is produced an effect nearly corresponding with that produced by either of the agents alone—that it is only when Battley's liquor is used in the very large proportion of 20 to 1 that the effect of the aconite seems to be subdued, and that of the Battley seems to predominate. In any other proportion, the aconite decidedly predominates. In *no case* does the one overpower *completely* the other, but the action of the aconite generally predominates. In cases where the dose was not very deadly, we found that the markedly noticeable effects commenced in about three minutes from the time of the fluid being injected.

Dr. Coats—Were those peculiar motions of which you have spoken in connection with conium—rubbing on the nose, &c.—the same in all the animals? I can quite understand how vital action should be exhibited, but I would like to know whether these little motions were all identical.

Dr. Adams—As regards the extract from tincture of conium, they were identical. But our observations on the various alkaloidal poisons to which I have referred are as yet incomplete, and that is what has made me do no more than allude to some of the peculiar effects noticed by us, because Dr. Penny and myself think it advisable to continue our inquiries. As regards the action of aconite and Battley's liquor, we are satisfied we have little to learn. In every form in which these can be varied, we are perfectly satisfied what the result will be. In some of the others we have noticed peculiarities that are equally characteristic, and that differ entirely from many of the vegetable poisons, and we hope to arrive at results in regard to a number of these poisons which will be serviceable afterwards for reference.

Dr. Gairdner—Suppose you were condemned to death by poison, and that you had absolutely to take ten or twenty drops, as it might be, of Fleming's tincture of Aconite, would you rather take that plain, or along with Battley?

Dr. Adams—I would decidedly take it along with Battley.

Dr. G. H. B. Macleod—Having been present at the recent lamentable trial during the time these experiments were detailed, I must say that I was exceedingly struck with the effect which their recital appeared to produce upon the jury and the public who were in court. I think it was an exceedingly fortunate circumstance that Dr. Adams and Dr. Penny fell upon this mode of experimenting, because unquestionably it had a very great influence indeed in deciding the opinion of the jury with regard to the action of these medicines upon the unfortunate ladies who were poisoned. The paper which has been read by Dr. Adams opens up one of the most important fields for farther researches which can well be suggested; but at the same time, those experiments must be conducted with exceeding care, and be repeated a very great number of times, before they can be of absolute value to us as demonstrating the action of these poisons. There is another circumstance which I think commends itself to us

all, and that is, that while these poisons are shown to act in a particular way in the case of rabbits, yet the experiments, after all, do not teach us a great deal of how the human subject is affected by these poisons. I do not wish to detract from the importance of these experiments so far as they go, and some points may yet be expiscated by Dr. Adams which will give us some information with regard to the action of these poisons upon the human subject. There were one or two facts in the medical evidence that were exceedingly interesting. One of these was the curious contraction of the hands which took place in the case of Mrs. Pritchard, and of which I can find no explanation in any book that it is in my power to consult. Now, we all know that Dr. Pritchard had in his possession ten grains of strychnine, to which no reference has been made; and it would be a curious thing to know whether, if strychnine had been mixed with these poisons, it would have modified the action of the aconite. We know that aconite produces paralysis of the muscular system generally, and of the heart, and so produces death; whereas, on the other hand, strychnine produces, to a certain extent, a contrary effect. Now, how far the symptoms of either of these two poisons would be modified by their admixture is, I think, a point worthy of Dr. Adams' attention. There is little doubt, from the evidence, that Mrs. Taylor died from opium. I think it was the opium unquestionably, in her case, that seemed to produce the symptoms that were observed. But in the case of Mrs. Taylor there must have been a large dose of aconite, as we know it was mixed with Battley in this large proportion, because evidence was led to show that she got this bottle (capable of containing five ounces of Battley's solution) filled in Edinburgh before coming down to Glasgow, and it was filled on the Monday preceding her death, and found half empty at the time of her death. Now, the quantity of the Battley that she took in this way must have been very large, and if it contained even five per cent. in every drachm of the Battley, with five per cent. of Fleming's tincture of aconite, that would be equal to something like three minims of Fleming's, or eighteen minims of the ordinary tincture of aconite. Besides, the probability is that she did not content herself with a drachm or anything like a drachm, so that we naturally conclude she must have taken a large quantity of the aconite. Another point in the case was the action upon the pupil. There is no doubt that when Dr. Paterson saw Mrs. Taylor, her pupil was contracted to a point, and we know that aconite has the very opposite effect. It tends to paralyze the eye, so as to cause dilatation of the pupil; and in these experiments made by Dr. Adams I find that it produced contraction of the pupil till the animal was about dead, or altogether dead. That certainly is different from the symptoms as observed in the human body. I may say I made the selection of the bottles which were examined by Dr. Penny, and there was a very large quantity of conium in Dr. Pritchard's repositories. I do not know how far that may have been administered in this case. The chewing and twitching of the mouth is a curious symptom in these rabbits. We know that aconite produces a very curious feeling in the mouth—a tingling of the lips and tongue. It would be a very odd thing if, when injected into the back, it produces a similar sensation. I certainly would be disposed to think that the sensation is due to the immediate application to the lips and tongue; but is it possible that it will give rise to the chewing and twitching, even when injected under the skin?

Dr. Gairdner—I entirely concur with Dr. Macleod in the tribute which he has paid to Dr. Penny and Dr. Adams' experiments, as explained in court. No one could hear that testimony without feeling that it was evidence of the most clear and trustworthy character—guarded at every point most religiously, so as to place it beyond the possibility of being confuted. The admirable character of that evidence, and the experiments that Dr. Adams announced, rather led me to expect that I would get something from them tending to explain more than has been done those very curious spasms that occurred in the case of Mrs. Pritchard. I must confess that I am almost as much puzzled now as then to

tell what poison could have been the cause of those peculiar spasms. They were not general convulsions in any sense; they were constant. Certainly Mrs. Pritchard was delirious and intoxicated at the time, but the spasms were quite constant, with the retention of a certain amount of sensibility and power to speak, and with no material dilatation or contraction of the pupils, and therefore they were not such as are ordinarily produced in extreme narcotism. Moreover, they were not like the ordinary effects of any narcotic poison. At the same time, I do not think that anything we read about antimony or any of the metallic poisons explains such very peculiar spasms, which I may say were more like the carpo-pedal spasms of children. Next day she was perfectly sober and sensible, but she still had the remains of these same spasms, which were at that time the only thing to be observed. At various times since then strychnine has crossed my mind. We know Dr. Pritchard had strychnine. I think it quite likely she was under strychnine; I think it still more likely she was under a combination of poisons, five or six of them together, so that it is extremely difficult to detect them. So impressed was I on this subject that I was at one time on the point of sending a warning to the analysts that if one poison was found there might be half a dozen. It certainly puzzled me very much then, and I confess that it puzzles me very considerably now.

Dr. Adams, in answer to Dr. Robertson, said that atropine had little effect on rabbits. In doses of half a grain and a whole grain of pure atropine injected under the skin, there was little noticeable effect, beyond rendering the animal fidgety and uncomfortable.

Dr. George Robertson—This is strange; for on one occasion I injected the sixteenth part of a grain of atropine under the skin of a medical man, and for fourteen hours, commencing very soon after the injection, he was rambling and apparently raving, and quite unable to pronounce a single articulate sentence. He was a middle-aged gentleman, and a Free Church elder. (Laughter.) Almost immediately after the injection under the skin of the fore-arm, he said he felt it at the top of his head, and that he felt the peculiar taste of it in his mouth. In a few seconds he said he felt a stiffness about his jaw. In a few seconds more he said, "I cannot speak, and, demmit, I cannot curse." (Laughter.) I tried to soothe my friend, saying it was no great loss, as he was not in the habit of cursing, but he could not reply. To me the effect seemed so alarming that I should not like to inject a larger dose than that under the skin of a middle-aged medical man. This injection was used at the very urgent request of a medical man for the cure of lumbago and sciatica, which had rendered him unable to follow his business for about eight weeks. He walked about very well next day, and told me afterwards that ever since he had been quite cured of his lumbago. He remained in my house some four and twenty hours after this; but I was so alarmed at the effect of that sixteenth part of a grain, that I would not like to repeat the injection, even for sciatica or lumbago.

Dr. G. H. B. Macleod—I must protest against any inference being drawn from the peculiar effects produced upon Free Church Elders, and the analogy extended to individuals of other persuasions. (Laughter.)

Dr. Adams—And I must warn the society against believing that either Dr. Penny or myself has fallen into the error of supposing that there is a strict analogy to be drawn from the symptoms produced upon animals and extended to human beings. We selected one class of animals, and it was sufficient for our purpose to find that the characteristic effects of any one agent were uniform in that animal. With regard to the peculiar chewing, twitching movement of the lips, and the salivation produced by the aconite, I find that Headland has noticed it, and that he attributes it, very strangely as I think, to paralysis of the fauces.

This closed the discussion.

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Fig A.

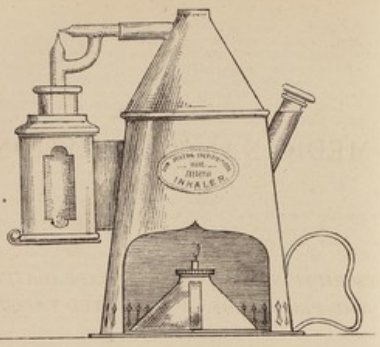
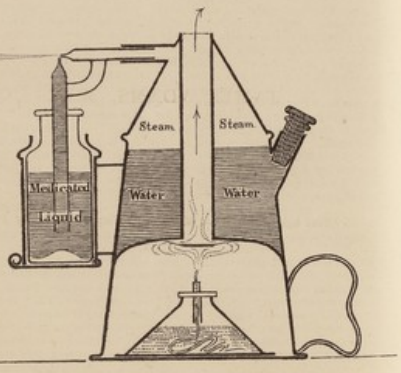


Fig B.



Apparatus for the production of Medicated Vapours.

Fig A, External appearance.

Fig B, Sectional view.

ON

MEDICINAL INHALATIONS,

WITH

DESCRIPTION OF AN IMPROVED APPARATUS FOR THE PRODUCTION OF MEDICATED VAPOURS.

BY

JAMES ADAMS, M.D.

(Read before the Glasgow Med.-Chir. Society, 7th Feb. 1868.)

GLASGOW:
JAMES MACLEHOSE, 61 ST. VINCENT STREET,
Bookseller to the University, and to the Faculty of Physicians and Surgeons.
1868.

MEDICINAL INHALATIONS.

In the treatment of affections of the lungs and air passages, the inhalation of medicated vapours has been practised from the earliest times. The chiefest medical authorities speak in terms of high commendation of such agency, whether as remedies bringing immediate relief to the patient, or as a branch of therapeutics, through which specific modes of treatment for some diseases may ultimately be reached.

But owing to several causes it happens that, unless by a limited number of medical men, the use of medicated inhalations is only occasionally practised. Among these causes, there is no doubt the great trouble and loss of valuable time that is involved in instructing a patient or his friends in the proper use of an apparatus, or in the most suitable arrangements for making the inhalations efficacious. But I am inclined to think that the inefficiency of most of the contrivances that have hitherto been easily accessible, and the cost of others, has had a greater deterring influence. And still greater influence must be accorded to the fact, that until a late period—until the present time I may say—there has not existed a means by which medicaments, however likely and however potent, could be applied to the mucous lining of the air passages, unless that these substances were either volatile in themselves, or were capable of being dissipated in the air by the action of heat artificially applied. It is to this difficulty, or rather to the methods that have been successfully contrived for overcoming it, that I now wish to direct the attention of the Society. And, under the pretext of submitting to your inspection a convenient modification of an apparatus, on which I have been expending my mechanical powers, and which I have very largely used, I

hope to elicit the experience of some who can speak from actual observation of the effects of medicinal inhalations, and to enlist the thoughtful consideration of others to whom this mode of treatment is comparatively new, or altogether untried.

It may be well that I should first refer to some of the contrivances which have been hitherto in use.

Hippocrates describes an apparatus consisting of a pot, the lid of which had an opening for the reception of a reed, through which the vapours escaped and were inhaled through the open mouth, while moistened sponges were employed to protect the mouth from being scalded.

In later times the methods employed have been various, but usually of a very simple kind, and—discarding all consideration of complicated costly arrangements, such as apartments specially constructed,—have been often extemporized with a reference to the agent made use of. Thus we have Tar heated with certain precautions in an iron ladle, or pot, as recommended by Sir Alexander Crichton, who saw it employed in Russia in the treatment of bronchitis and phthisis. I have often so used it myself, employing for the vaporization of it and other solid substances, as sulphur, &c., such a simple apparatus as I now place on the table. The fumes arising from undressed Wool, burned on an iron plate, were at one time much used on the recommendation of Dr. Physick of New York. He found these fumes extremely serviceable for stimulating and healing external sores, and he thought he had found them of utility in phthisis when inhaled by the lungs. I have not unfrequently made use of strips of bibulous paper soaked successively in solution of Nitre and tincture of Tolu or Benzoin. These, when dry, are ignited on a plate, and a vapour is given out from which patients affected with Asthma and chronic coughs say they often experience relief. Pastilles of varied composition are occasionally employed with similar results. But the most common method of employing inhalations consists in pouring boiling water upon Vinegar, Chlorine, Æther, Camphor, any of the Balsamic resins—or Narcotic extracts, or their Tinctures. These substances are placed in vessels variously constructed of block tin, pewter, glass, or earthenware, but all essentially consist of a vessel of large capacity, having a tubular orifice to which the patient applies his mouth and makes forcible inspiration. I place several of these instruments on the table; but I feel constrained to say that, notwithstanding their various names and modifications of form, material, and cost, a large-sized tea or coffee pot, or common tea-kettle, will at any time form an efficient substitute. To all these contrivances I have an objection, that their use is fatiguing to the patient, who, I rarely find

can avoid making use of distressing and exhausting efforts at inhalation, not that there exists such necessity, but that the form and mode of using the instrument seems to suggest instinctively the effort. There are various other objections which I need not take time to enumerate, such as the rapid cooling of the hot water, and the triflingly small amount of vapour that is produced, &c.

The steam of hot water, without any admixture, is often considered a desirable application to a dry and inflamed throat, trachea or bronchial lining; and in illustration of the rough and ready means that are usually extemporized, I mention that on two recent occasions I knew a medical man, than whom there are few so full of practical resource, directing a basin of warm water, with a piece of heated iron or common fire brick dropped into it, to keep up the steam; and another gentlemen, one of our most accomplished physicians, instructing a roll of stiff paper to be tied to the spout of a kettle, so that the steam might be projected in a direction to reach the patient.

The necessity of having recourse to such crude expedients, in the course of practice among the more comfortable classes, where the mere question of cost for even a passing want is no real obstacle, shows that a convenient and portable contrivance has been a desideratum. Such instruments as were available applied only to substances in their nature volatile, but until the present time, so to speak, there has existed no means by which a non-volatile chemical body could come in contact with the organs of respiration.

And this brings me to mention, and with respect, the name of Sales-Giron of Pierrefond in France, who, after two years consideration of the subject, laid before the Academy of Medicine of Paris, in 1858, an apparatus by means of which fluids containing dissolved medicaments, whether volatile or non-volatile, could be converted into a fine spray or mist capable of reaching the extreme bronchial tubes when inhaled. This proposed addition to practical medicine met, of course, with that careful and searching consideration that has ever been accorded by medical men to all proposals having for their object the relief of suffering humanity—a consideration, the spirit of which is commonly misunderstood by the general public, and, partly from combined ignorance, interested motive, and malicious intent, is commonly misrepresented by the charlatan.

Sales-Giron, in the construction of his apparatus, carried out an idea which in some measure had been already practically applied, although in a coarse and little effective manner, in the vaporatory of certain sulphur baths. His instrument consisted of a vessel filled with fluid, in which was dissolved certain

medicaments; and an air-pump was attached over the vessel. By the pressure of the air-pump, the fluid was forcibly projected through a narrow aperture, and made to strike against a metal disc, when the stream was broken up and converted into a very minute vapour or spray. This he designated an *atomized* or *pulverized* fluid.

When this portable apparatus was placed before the Academy it became a question, how far, if at all, such fluids could affect the respiratory tubes. A committee of investigation was appointed; and in 1862 the committee reported as a result of their labours, that the various substances used by them in their experiments reached not only the trachea, but penetrated to the minute tubes and air cells. And it was during the discussion of this report that Trousseau said, "I have applied the inhalations in many instances, and derived great benefit from them. They form a medicament of great value in affections of the pharynx, larynx, treachea, and the large bronchi. . . . In short, Sales-Giron has rendered a great service to the world at large by his invention of the treatment by means of pulverization."

This statement of the veteran Trousseau has been emphatically endorsed by the experience and commentaries of the numerous eminent medical men on the Continent and in this country, who have in a special manner given their attention to the subject.

At the hands of several individuals who approved of the new invention, the apparatus of Sales-Giron underwent various modifications, the most important of which consisted in the contrivance of Dr. Bergson, who connected at right angles the capillary openings of two glass tubes, one of which dipped into a vessel containing the liquid to be "pulverised"—the other tube having attached to it an india-rubber pipe with two india-rubber balls, one of which acted as a bellows, the other a reservoir of air,—the identical instrument, in short, that is now advertised as "Dr. Dewar's Patent Spray Producer" the difference consisting in the tubes being made of Vulcanite, with metal points instead of glass.*

This very ingenious contrivance of Dr. Bergson has undergone various modifications. As a toy instrument, consisting of the tubes alone, it is used for sprinkling perfume, by simply blowing in one of the tubes while the other dips into the phial of perfume. When the horizontal tube is blown through with some force, the effect is to exhaust the air in the other which dips into fluid, the fluid rises to fill the vacuum, and,

* Several months before Dr. Dewar's patent, the instrument, but without metal points, was in regular course of sale by Thornton & Co., manufacturers of vulcanite.

reaching the capillary orifice at the top, is caught by the rushing blast, and blown into fine vapour.

It is sometimes used in the same arrangement for sprinkling the larynx and fauces. Sometimes it is attached to a regularly constructed bellows, but the common construction is the original one, that of an india-rubber ball that requires to be compressed by the hand. Its action is, however, intermittent, producing alternately a coarse or a fine spray just as the pressure of the hand upon the ball relaxes or contracts. Under this pumping action the patient is liable to receive alternately the full blast of an uncomfortable cold sprinkling shower on his face or to catch merely the extreme or furthest projected particles of the spray. To most individuals this cold blast is disagreeable—to many intolerable. To obviate this drawback a spirit lamp is sometimes applied to heat the medicated fluid; and I have found it occasionally necessary to recommend that the vessel of medicated fluid should be placed in hot water and gently warmed, where the patient has happened to possess himself of an india-rubber ball apparatus. But the use of this india-rubber ball is very difficult. Practice and dexterity is required before anything like a continuous spray can be produced. The limited and monotonous movements of the fingers soon become irksome, and can only be maintained under a sense of fatigue or of cramp even by persons possessing considerable muscular power. On this point I have made experiments assisted by various individuals, and the result satisfies me that, even where the patient can have an assistant willing to give the requisite time and effort, that effort can not be maintained in cases where the inhalations require to be frequent and of prolonged continuance. The efficient use of the apparatus by the invalid himself is, I think, quite out of question.

Unless, therefore, for the object of sprinkling the fauces or larynx, the use of any contrivance requiring manual exertion will not be continued by any practitioner or patient; and it was from a knowledge of these drawbacks that the latest improvement, that of producing a continuous and steady current of vapour by the action of a jet of steam, was applied by Dr. Siegle.

It is this combination of Bergson and Siegle's several apparatus, known by the name of Dr. Siegle, that is now in approved use; and it consists in a small boiler heated with a spirit lamp the capillary tubes being attached to the boiler. The steam produced in the boiler issues from one of the tubes with considerable force, and in a steady current, producing the same effect as a blast of compressed air, so that the medicated

fluid is projected in a gentle warm mist or vapour, very much finer than could possibly be previously obtained by any other arrangement. Contrasting the vapour produced by this apparatus with that of the air-pump or bellows arrangement, I should liken the first to a fine Scotch mist, and the second to a plump of rain.

Experiment has shown, what indeed was already very obvious, *i.e.*, that this very minute division, or pulverization, of the medicated fluids, is a matter of prime importance, as the finer the vapour the less the irritation to which it gives rise in passing the larynx, and the more effectually it penetrates to the minutest and furthest removed air-tubes. The patient requires no assistant in working the instrument; he inhales without fatigue or flurry, and at leisure; and the inhalations can be repeated and prolonged indefinitely and at pleasure.

I have during the last fifteen or eighteen months had very extensive trials of this apparatus; and I think that, with some modifications it has undergone at my hands, and to which I will immediately refer, it leaves little to be desired. I was led to a consideration of these modifications on account of the high cost of the instruments, ranging from 15s. to 50s., and the occasional delay of many days, and even weeks, before they could be obtained. These were serious obstacles to a fair trial, or to the general use of the instrument, and I was unable to stimulate either instrument-maker or druggist to find a remedy. I at length got a smart tinsmith to work several patterns under my directions, and to produce an instrument equally effective with the most costly, and a respectable druggist* to give him an order for a supply. These within the last few months, under the stimulus of the sulphur mania, have been sold in large numbers by several druggists at an average cost of 4s. One druggist tells me he alone has sold upwards of 200.

I felt interested in my attempts to improve the instrument, and you would be amused if I showed you all the patterns from first to last, and surprised if I were to go into a description of little details which required consideration or remedy before the instrument I now place on the table was produced. Suffice it that I feel warranted in saying that it is the most efficient, ready, and cheap instrument that can at the present moment be obtained. Let me state shortly some of the qualities wherein I think that it contrasts with, and is superior to, others.

It is compact and ready for use, and is so put together that there is no necessity for frequent and nice adjustments,—consequently the risk of troublesome disarrangements, or of accidental injury of the instrument, is exceedingly small.

* Mr. D. P. Walker, 125 New City Road.

The form of the boiler is distinctive and peculiar, and ensures several advantages. Thus, the position of the water inlet enables the boiler to be filled to the proper height and no more. Above the water line and inlet there is a reservoir for steam sufficiently large to maintain a continuous current of spray, and to project it to any distance that may reasonably be desired. This arrangement of the water inlet, steam chamber, and steam outlet, prevents a very annoying and even dangerous accident of frequent occurrence in instruments with the ordinary form of boiler, namely, the forcible projection of spirts of scalding water in the face of the patient, caused by the boiling liquid coming over with the steam. The heat from the spirit lamp is carried up through the centre of the boiler, thus reaching a larger heating surface of the boiler, generating steam more rapidly, keeping up a full supply of the steam, and at same time *super-heating* and *drying* the steam so generated. The steam escapes by a short horizontal nozzle at the top of the boiler, and necessarily is subjected at the instant of its escape to the action of the flue of the lamp, thus ensuring such a dry condition of the steam that it quickly becomes dissolved or dissipated in the air, so lessening the risk of annoyance to the patient, and at same time avoiding in a great measure the dilution of the medicated fluid with watery steam. By carrying the flue of the lamp through the centre of the boiler, the body of the instrument is not so hot but that a handle attached to the lower and cooler portion of the case can be grasped with comfort and safety even when in use. An alarming-looking and costly safety-valve, very liable to become stiff and unworkable, has been dispensed with, as I have found that a simple cork or india-rubber plug is equally efficient and more convenient. The box containing the phial of medicated fluid is soon warmed by the mode of its attachment to the case of the instrument, and there are other details of minor importance which, when combined, make the apparatus more convenient in use.

Before parting with my reference to this instrument I may mention that I have experienced annoyance from finding my patterns and directions departed from occasionally by workmen who did not understand what I was aiming at. An old friend of mine,* who had opportunities of knowing this fact, suggested a patent, which I of course rejected, my ideas on medical patents being at one with my friend Dr. Gairdner.† I did not object, however, that he should do so; and, associating

* Mr. J. C. Stuart, manufacturing chemist, Dundas Hill.

† See *Certain Moral Aspects of Money-Getting*, p. 23.

with himself a respectable druggist,* he has taken out a patent. I hope thus to ensure accuracy and good quality of workmanship with cheapness of manufacture. *This will certainly gratify me; but beyond this, I beg emphatically to say that I have no right of property in the instrument, and no interest whatever in its sale.*†

Having said so much regarding instruments, I have little time to refer to the medicaments, which may conveniently, and with probable advantage, be employed in the form of vapour. Those with the use of which I am most familiar are solutions of Morphia, Digitalis, Stramonium, Squill, Tannin, Alum, Nitrate of Silver, Sulphate of Zinc, Chloroform, Acetic Acid, and Sulphurous Acid. I extend and combine this list of agents as seems to me desirable. Each drug has of course its special properties, and it would open too large a discussion to enter, however shortly, upon their consideration. I might say much to show why I have reason to be satisfied with the results I obtain from time to time; but I think it sufficient to indicate the fact that I am so satisfied, and that I believe that the inhalation of medicated vapours is likely to be a more familiar and a more important therapeutic agent in the hands of the physician in time to come.

Those who are inclined to pursue this subject will find ample details of the history and full directions for, and illustrations of, treatment by medicinal inhalations in the work of Dr. Beigel of London—an admirable work in my opinion, and showing in its author the possession of sound common sense and professional ability.

At the last moment, it has occurred to me that it might give point to the object of my communication, and open an easy inlet to the observations of the members, if, before concluding, I should make special reference to one agent, with the name and alleged miraculous virtues of which the public of Glasgow has been made very familiar during the last few months. I need scarce say that I allude to sulphurous acid gas, or, as it has been termed in the popular epistles through which it has gained notoriety, "the Great Sulphur Cure."

The statements in Dr. Dewar's original pamphlet regarding the value of sulphur as a great remedial agent, seemed to me at the time sufficiently moderate and circumstantial to warrant a candid examination; and during the last twelve months, and of course long before the recent excitement, I have given to it a fair trial in a spirit of patient observation. I may at once say

* Mr. P. Harrower, 136 Cowcaddens.

† The instrument, price 5s., is now sold by the three parties I have named, and by other chemists and druggists.

that I have satisfied myself that it has its uses—that it is not an innocuous agent—one to be pooh-poohed and laid aside without trial, or to be contemptuously stifled in a cloud of "chaff," scattered over the columns of a newspaper. But, to qualify this allusion, I admit that it is very difficult to be amazed and yet temperate when reading the loose statements, crude deductions, and far from satisfactory cases recently published by Dr. Dewar; or, to refrain from disparaging the rhapsodical style of his enthusiastic follower, Dr Pairman.* Yet a medical man, having in recollection the history of medicine and of popular credulity, as the latter is manifested upon the occasion of every new appeal that is made to its faith in nostrums and universal cures, should ever bear in mind that a question of Science, or of facts in Medicine, is not to be discussed in an arena and before a jury that in all time has pronounced, as if by intuition and in popular acclaim, a favourable deliverance upon every kind of wonderful 'cure' that successively crops up—ranging from Sulphur, through Cold Water, Hot Water, Turkish Baths, Mineral Baths, Homœpathy, Mesmerism, St. John Long's Liniment, or Perkin's Metallic Tractors. Neither truth nor professional credit is advanced by such discussion, and we would do well to imitate the example of lawyers, who are never drawn into newspaper columns to wrangle over questions of law.

Passing from this digressive comment, and returning to Drs. Dewar and Pairman, I am well satisfied that they are both sincere men; and, believing as I do, that they have made their statements in perfectly good faith, and alleged, as matters of fact, occurrences that are open to daily and familiar experiment and observation, I think it preferable to sift these statements before troubling myself about misty theories, or rather hypothetical conjectures—or in denouncing pretensions however apparently absurd, If these gentlemen are competent observers, and of sound judgment, then the occurrences they have recorded must have been repeated under like circumstances; and this recurrence of powerful and novel effects has no doubt been observed, and will be corroborated by the testimony of medical men whose position as competent observers is established. We have such observers among us this evening, and I hope they will express such a judgment as their experience warrants them in giving. As regards myself, I repeat that I have observed with patience and without prejudice, and as a result of my observations I felt warranted, at the commencement of

* Dr. Pairman has, however, admittedly "a method in his madness," and his statements of positive facts are, when closely examined, found to be more moderate and reasonable than is at first sight apparent,—certainly much more so than those of Dr. Dewar.

the late sulphur mania—for it deserves no other name—in expressing my opinion that the agent was useful; and that Dr. Dewar deserved great credit for his earnest and persevering efforts in directing the attention of the profession to several novel and useful applications of the remedy; but that it was most preposterously over-lauded—that its excessive popularity would have a short day—and that some injury and many grievous disappointments would remain *en souvenir*.

My experience of the remedy, in some of its principal applications to medicine in the way of inhalation, and without any reference to surgical ailments, may be shortly stated.

Individuals whose general health was good, and who have resorted to the sulphur inhalations on account of such slight forms of Catarrh as usually receive little or no medical treatment, except it may be a sweating powder, or a warm bath, have been the most liberal in their acknowledgments of benefit. It did really seem to me that some cases were relieved or shortened in their progress by the treatment.

In Acute Bronchitis I have seen a copious secretion of serous or watery fluid cast off from the air passages at an unusually early stage of the disease, and this effect was evidently induced by the inhalations, and was followed by marked relief.

In Chronic Coughs it has frequently acted powerfully in exciting forcibly expiration, and in inducing a more copious expectoration.

From these results I am of opinion that in Catarrh, acute Bronchitis, and Chronic Coughs, the remedy stimulates the minute exhaling vessels, the bronchial surface seems to be both sweated and purged, and the tough, viscid phlegm which collects in the bronchi is dislodged more freely and effectively than occurs under the use of ordinary expectorants, administered in the usual way. But I have found no notable difference nor any advantage in use in the Sulphurous Acid over common Vinegar or solution of Chlorine administered by inhalation in like cases.

In a few opportunities I have tried it in Asthma, but have not found that the inhalations were tolerated or continued, partly on account of marked distress which they occasioned, and partly because no evident relief was obtained during the paroxysms.

In Phthisis its effect seems to be altogether that of a topical expectorant, but I have not seen any decided lessening of the amount of expectoration.

Cases of inflammatory sore throat have not been benefitted, but in the hoarseness following the acute stage of a cold I have seen improvement and relief.

I have not seen, in any of its applications, the alleged "sedative" or "calmative" action of the remedy.

Troublesome and even alarming consequences are of occasional occurrence after prolonged inhalations of the dry sulphur fumes, or of the aqueous solution of sulphurous acid gas in its full strength,—such as great tumefaction of the tonsils and pendulous palate, husky voice,—difficulty in swallowing,—pain in larynx spitting of blood, &c.; and I have seen all these effects occur under circumstances where no blame could be justly imputed on the score of carelessness.

It is not, therefore, a remedy to be administered indiscriminately or without precaution, and I deprecate the practice of fumigating a patient's room by throwing a quantity (no limit as to quantity) over a heated shovel or live coal, and filling an apartment (which may be large or small, well ventilated or otherwise) with the dry fumes of an indefinite quantity of sulphurous acid gas,—an agent of known powerful chemical properties, and alleged to possess very potent influence upon the living organism. As well tell a patient to take "a large dash of laudanum" in his gruel when going to bed, or "a good pinch of calomel" occasionally.

My remarks on the sulphur cure have gone to a greater length than I anticipated, and I will now only add that I have not seen any effects from its use that have led me to think that it has any *specific action, when inhaled in any disease*, or that it has any other effect than that of a local stimulant, tonic, and astringent. As such I believe it is likely to prove a useful adjuvant in the treatment of various maladies.

(From the Glasgow Medical Journal, March, 1868).

MEDICO-CHIRURGICAL SOCIETY.

MEETING VI.—SESSION 1867-68. 7TH FEBRUARY, 1868.

DR. GAIRDNER, Vice-President, in the Chair.

DR. ADAMS read a paper on Medicinal Inhalations, with description of an improved apparatus for the production of Medicated Vapours. Dr. Adams placed before the Society a large number and variety of apparatus, used for medicinal inhalations,—including specimens of Siegels, Bergsons, Dewars, and the instrument constructed under his own directions. Dr. Adams also exhibited several of the apparatus in action, using perfumed instead of medicated liquids.

DR. ANDREW BUCHANAN thanked Dr. Adams for bringing forward these interesting mechanical contrivances, and giving a brief history of this increasingly important mode of medication, which he had no doubt would yet be applied with very beneficial effects in diseases of the lungs and air passages. Of course the modes of application would require to be thoroughly tested, and that in the same spirit in which this had been done by Dr. Adams. He (Dr B.) had first made acquaintance with these new mechanical appliances through the instrument of Dr. Dewar, which had been introduced to his notice by a very intelligent man, who stated to him that Dr. D. had taken a patent for it. He was surprised, however, on investigating the matter, that there was really nothing original in this patent of Dr. Dewar's except the gold points. Dr. Adams had told them that Dr. Dewar had been anticipated in the introduction of vulcanite in the instrument, by the manufacturers of that substance. This appeared to point decidedly to the fact that the present law of patent was essentially defective, when an instrument could be patented which in principle and detail had nothing original in it. Dr. Buchanan believed that the importance of this mode of treatment consisted in its bringing medicines directly to act upon the mucous membrane of the lungs. Formerly the only means they had of acting on this membrane was by expectorants, which were very uncertain in their operation, and could not therefore be depended on. The first step to the application of medicaments by inhalation was in the introduction of such substances as ether, chloroform &c., into the body through the lungs. By means of medicinal inhalation it was now possible to influence directly the pathological condition of the membrane of the lungs, through a mode never before thought of. The medicines were by this means introduced in a form so bland—the vapour or the nearest approximation to vapour was so finely divided, that the risk of injury was indefinitely lessened. Such a mode of application was precisely what was desiderated in an organ of such delicacy. He had tried sulphurous acid spray in a few cases of chronic bronchitis, and he had found it serviceable in some of these cases.

DR. RITCHIE, after remarking on the great expenditure of time, thought, artistic talent, and money which it must have cost Dr. Adams to have brought his Spray Inhaler to its present perfection, said that it was to be regretted, however, that there did not exist more materials of a strictly professional kind on which to found an intelligent judgment of the utility of medicinal inhalation. This mode of curative treatment being indicated

on several principles, and capable of application in a variety of forms, it would be well to have them clearly known. He (Dr. R.) must confess that he knew little of pulmonary inhalation of medicinal substances, in the treatment of disease. He had for many years, in common with others, been in the habit, as suggested by the late Dr. Robert Watt, of Glasgow, of using tar in the second stage of whooping-cough, and bronchitis, either by burning it or evaporating it by the heat of boiling water, or of a spirit lamp. Another application of the principle, though with a different object, was the mercurial vapour bath, in the use of which in secondary syphilis he had had great satisfaction. He had also, like Dr. Adams, used nitre papers, but without benefit, and he had frequently tried the inhalation of naphtha and petroleum in phthisis, but always with unfavourable results.

The application of atomized fluids to the treatment of disease, and especially the introduction of these by the air passages, had given a new aspect to the therapeutics of various affections; and, looking at the extreme facility with which aqueous or medicated spray could now be introduced into the most remote portions of the lungs; the amazing extent of absorbing surface presented by that organ, and the immediate admixture of whatever is introduced with the whole mass of the blood, there surely was a call for caution in the exhibition of such an agent as Sulphurous Acid spray. It would be valuable could any competent observer devote himself to the investigation of all the circumstances connected with the inhalation and injection of gaseous and atomized fluids. The induction of certain effects on the cerebro-spinal system as by chloroform; the modifying the action of the respiratory organic nerves as by stramonium, lobelia, and opium; the dilution of the blood with simple or with slightly saline water, as might perhaps be practised in cholera and collapse of fever, and other cases; the stimulation of the mucous membrane of the bronchi by nitrate of silver in chronic bronchitis, and the destruction of parasitic growths in the larynx, as in diphtheritis, by sulphurous or carbolic acid, were all so many different principles on which the vapour inhalations might be employed. In closing, he would express the hope that the Chairman, and other gentlemen favourably situated for such investigations, would direct their attention to this interesting subject, more especially as related to the introduction into the blood by the spray, of water, or of weak solutions of muriate of soda, in the state of vital collapse, which occurred in fever, cholera, and other states.

MR. POLLOCK, of Mearns, stated that he could corroborate Dr. Adams' observation about the impossibility of working Dr. Dewar's instrument any length of time, owing to the hand becoming powerless. He thought Dr. Adams' instrument a great improvement on all its predecessors.

DR. MORTON said that as regarded the general question of inhalation, there could be no doubt that for some years it had been gaining ground in the estimation of the profession, as a means of introducing medicinal agents into the system, and very probably they would have greater recourse to it as they became more familiar with the use of it. Dr. Ritchie had alluded incidentally to the caution alleged to be necessary in introducing agents into the lungs, owing to the rapidity with which they sometimes acted upon the system. They had no doubt been taught that this was the case, but it was open to doubt whether experience completely bore out the statement. The mucous membrane of the lungs seemed able to bear a much greater amount of irritation than was commonly believed. Every one who had experience in the frequent use of chloroform must have noticed that, in rare cases, its administration gave rise to an inflamed condition of the conjunctivæ. Now, he was not aware that any one observed any similar

effect on the lining membrane of the lungs, or of the air tubes. He also remarked that ethereal substances introduced into the cavity of the peritoneum very soon manifested themselves in the blood, and, *a priori*, it might be expected that the absorption of such substances would take place even more rapidly by the lungs. He thought it a circumstance to be wondered at that the list of substances which had been tried to be introduced into the system by means of inhalation was as yet so small. A tanner in town had informed him of the alleged curative effects of Valonia Galls on certain forms of bronchial affections. This man employed in his work girls from fourteen to twenty years of age: and he had observed those suffering from chronic bronchitis—popularly believed to be labouring under severe chest disease—on entering, suffer keenly for the first few days, and in some cases obliged to discontinue working for some time, from a feeling of suffocation; but when they got over this period, they were greatly benefitted, so much so that this employment was regarded as a kind of cure. Doubtless the astringent effect of the tannic acid was the cause of the curative action. This was perfectly in consonance with the old practice of giving cinchona, the efficacy of which was ascribed to its astringent effects. All the substances which had been used in the way of inhalation—chlorine, stramonium, sulphurous acid, &c., could only act by stimulating the bronchial mucous membrane—increasing the secretion, and getting it drawn off. That they could cure disease in this way he did not believe; but it was quite possible, in the way indicated, to influence the membrane very materially.

Mr. John Reid said that he agreed with many of the conclusions to which Dr. Adams had come in respect of the inhalation of different substances, and particularly of the value of what had been called the great Sulphur Cure. Very early in his practice he had been in the habit of prescribing different agents for inhalation in bronchial affections, such as iodine, stramonium, &c., but he must say he never saw any marked benefit from such treatment. He had used for eight or nine years one of the old inhalers, of which a specimen was exhibited, with iodine, but he had now discontinued its use from never observing any benefit from it. In regard to the effect of sulphur they were well aware that it could only act as a stimulant upon the bronchial mucous surface, and that in a very disagreeable way. He had no faith whatever in its acting as a cure in bronchial affections. It was a fact well known to medical men attending on chemical works, that the men exposed to sulphur fumes were particularly prone to bronchial affections, and indeed were all more or less affected with bronchitis; showing that Sulphur produced the very reverse of a sedative effect on the mucous membrane. He did not think it creditable to any member of the profession to have made such a noise about this matter as Dr. Dewar had done. He first introduced it as a remedy in rinderpest, then in diphtheria, and now it was trumpeted as a remedy for almost every disease. He looked on the way that he had puffed himself into notice as nothing less than quackish and quite unwarrantable. The inhalation of sulphurous acid could have no other effect than to bring up a quantity of phlegm, which was not curing disease at all. It would not be found a specific in complaints of the lungs any more than in skin diseases. He had no faith in its curing bronchitis, catarrh, or any of that class of complaints.

Dr. Lyon, after complimenting Dr. Adams for the ingenuity displayed in his instrument, said that he had been carried along by the entreaties of some of his patients to try the Sulphur Cure, and had allowed some of them to use it, but in no case had he found it to come up to their expectation, and indeed in nearly all the cases no benefit had resulted. One patient, a severe sufferer from very frequent paroxysms of asthma, followed by bronchitis, had tried it but with no advantage. He thought that its day was nearly out. He had no doubt, however that medication of the mucous membrane by inhalation could be employed with advantage. He had been in the habit of doing the same thing in another way, viz., through the stomach. He had often given Copaiba in this manner; and it gave decided proofs of its presence in the lungs by the exhalations which resulted. He believed that it was possible to act with decided efficacy on the respiratory organs in the manner indicated by Dr. Ritchie, with mercury; and he doubted not that medicated vapours might thus be carried into the system and exhaled from the lungs with advantage, in bronchitis, &c.

Dr. Eben. Watson said that having had some experience of the use of one of the instruments exhibited, he had listened with much interest to the paper, and especially to the excellent short history of the instruments which Dr. Adams had given, and he was pleased with Dr. Adams' modification of a rather expensive instrument. The principle of it was nearly the same as that of Siegle's instrument, though the tubular boiler was certainly a great improvement. The piece of mechanism, however, showing greatest ingenuity was Bergson's tubes. With regard to the use of these instruments his own experience was that they had two effects to expect from inhalations, the local effect and the general effect; and the medicines which Dr. Adams had named could only benefit from their general effect. There was no doubt that the mucous membrane of the bronchial tubes had a large surface, was very vascular, and afforded a ready means of absorption into the system generally. Great caution ought, therefore, to be exercised in regard to such medicines as digitalis or morphia, owing to their rapid and powerful action. The medicines which he had chiefly applied in solution through Siegle's instruments were such as produced a local effect, such as carbolic acid, a weak solution of which had been long known to have a beneficial effect in ulceration of the larynx.

He had also used borax and sulphate of zinc in ulcerations of the windpipe with some advantage. But the agent he had most frequently tried was warm water, which had a good calmative effect on the mucous membrane of the throat in ulceration of the larynx, and was extensively useful as a preparative to other treatment. He had not used nitrate of silver in this way, but it had been done in Paris, and he believed that it had had an excellent effect when applied with a sponge or brush to the windpipe. With regard to the Sulphur Cure he might state his experience. He had heard of a great many miraculous cures as having been effected, but on coming into contact with the subjects of the alleged cases, he found there had been a great deal of exaggeration. He had used it in several cases of different kinds—ordinary sore throats, inflamed tonsils, and inflammations of different kinds, and in all these certainly with great disadvantage, for sulphur was a local irritant. In its calmative action he did not believe at all. There was every reason to think that the Sulphur mania would be shortlived. But he believed that the use of medicinal inhalations would go on increasing, and that in many cases it would be of advantage, both when applied for a local and a general effect.

Dr. Perry said he believed that the discussion had proved that as a profession they were ignorant of the atomisation of fluids, though they had been long familiar with the use of medicated substances, more especially Iodine. In affections of the respiratory organs they were only beginning to understand that this might become an important mode of treatment. He had had a little experience, and, without entering into details, he might state that the cases in which he had tried inhalation of atomised fluids embraced croup, haemoptysis, scarlatina, chronic laryngitis, phthisical

laryngitis, and a variety of other diseases principally in connection with the throat and lungs. He was in a position to bear witness to the great trouble which Dr. Adams had given to bring this instrument to a state which he (Dr P.) considered one of perfection. He had been going along with Dr. A. in his experiments, and he would say, that no one could have any idea of the immense amount of labour involved. To point out the advantages of the improved instrument were needless; for both as regards cheapness, portability, and every useful quality, it had the advantage of the other form of inhalers. Siegle's last instrument, he might mention, from want of a handle, could not be held in the hand at the bedside, as it soon became too warm for the hand, and there were various other disadvantages in its construction which made it comparatively an inefficient instrument.

Dr. Gairdner said that he regretted that he could not respond to the request of Dr. Richie with regard to the use of inhalation in cholera, as he had never tried it in that disease, but he was disposed to think favourably of the principle of applying it in that way. In regard to the general question of inhalation, he had long been an employer of inhalation of hot water, charged with all sorts of medicated substances which could be received in vapour. He had, however, been extremely *fidgetted* in the want of a good instrument to furnish the spray in sufficient quantity, with a well regulated temperature, and for a proper length of time. The instrument which, on the whole, he had found the most useful was one invented by his father, Dr. John Gairdner of Edinburgh, and consisted of a spirit lamp, and a long tube through which the vapour was diffused. He had, however, long expected satisfactory results from the new method. He believed that this instrument of Dr. Adams' would be of great advantage to persons in general practice and to hospital physicians, &c., from being so cheap, portable, and not liable to be easily put out of gear, and he had no hesitation in saying that no other would now be in use in his own hands. Two weeks ago a friend of his came from Edinburgh for the express purpose of showing him a recently devised form of an instrument sold for a guinea, and which he had picked up in a visit to Vienna. The only advantage which he could see the Vienna instrument had was from the use of a long vulcanic tube to disperse or collect the spray as required. In fine, he had no hesitation in saying the thanks of the Society were due to Dr. Adams for the great personal trouble he had taken to improve the inhaler, for the impartial view he had given of the history of the instrument, for the admirable spirit and careful wording of his remarks on the therapeutics of inhalation, and for the delightful perfumes by which he had brought a rather questionable subject into good odour in the Society.

Dr. Adams said that in reference to the remarks of Dr. Ritchie it was obvious to every one that time would have failed him to enter at any length on the subject of treatment. His reason for bringing forward the subject was that in his intercourse with brother practitioners he had found that as a profession they were imperfectly acquainted with the new treatment of the inhalation of 'atomised fluids,' and he thought the subject at once important and interesting. He had found initial difficulty in all experiments on the subject—the instruments were always breaking, or going out of gear, or proving somehow unmanageable, and he had conceived it the first step in making any further advance, to endeavour to improve the apparatus so as to make it at once cheap, simple in construction, and thoroughly serviceable. The Society was now in a position to judge how far he had removed this difficulty out of the way of further progress.

PAMPHLETS

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