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Die Phosphorsaure Kalk

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Ziegler
Zoo-dynamia.

Macpherson.
Bengal Dysentery.

Fallot
Dysenterie Epidémique

Prichard
Life of

Der

phosphorsaure Kalk

in physiologischer und therapeutischer

Beziehung.

Ein Beitrag

zur physiologischen Heilkunde

von

Friedr. Wilh. Benecke, M. D.
Hausarzt am deutschen Hospitale in London.

Göttingen
bei Vandenhoeck und Ruprecht.
1850.

Den

Herren Hofräthen und Professoren

Dr. Rudolph Wagner

und

Dr. Friedrich Wöhler

zu Göttingen

im Gefühle aufrichtigster Dankbarkeit und
Hochachtung

der Verfasser.

Ich wage es, Ihnen, hochverehrte Lehrer, dieses kleine Schriftchen zu widmen; möchte es Ihnen einen Beweis liefern, daß ich im fernen Lande der deutschen Wissenschaft nicht vergessen habe, daß mich die freudigste Erinnerung oft an den Ort führt, wo ich vor einigen Jahren das Glück hatte, zu Ihren Schülern zu gehören, daß ich stets dankbar der Stunden eingedenk bin, in denen Sie Lust und Liebe zu den medicinischen Studien in mir anregten. Glücklich an einer Anstalt thätig zu sein, welche mir kein zu großes, aber ein sehr reichliches Material zu wissenschaftlichen Beobachtungen liefert, hoffe ich Ihnen im Verlaufe der folgenden Jahre weitere Beweise meiner Thätigkeit vorlegen zu können; nicht in der großen Menge der

Beobachtungen, sondern in der Qualität derselben werde ich dabei einen Werth suchen. Bei der Mangelhaftigkeit des physiologisch-chemischen Theils unseres medicinischen Wissens, scheint es mir in der That eine der Hauptaufgaben unserer Zeit zu sein, sich genauer, als es bisher geschehen ist, mit den zugänglichen Angriffspunkten pathologischer Processe bekannt zu machen, die Anfangs- und Endpunkte der pathologischen Stoff-Metamorphosen im Organismus zu studiren, um von da aus dann auf unzugängliche Mittelglieder Rückschlüsse versuchen zu können. — Unsere Aufgabe ist nicht mehr, mit der Kenntniß pathologisch-anatomischer Grundlagen, mit der Fähigkeit durch physikalische Untersuchung und Auffassung des objectiven Thatbestandes richtige Diagnosen zu stellen, mit dem Erlernen erfahrungs- und nicht erfahrungsmäßiger Heilmethoden erfüllt; selbst die weite Ferne des hohen Zieles einer Erkenntniß des gesammten einzelnen Krankheitsprocesses darf uns nicht abschrecken von dem Versuche, auf allen möglichen Bei- und Umwegen demselben näher zu kommen.

Sollten die vorliegenden Zeilen eine nachsichtige, freundliche Beurtheilung Ihrerseits finden, dann möchte ich Sie bitten, nicht mir, sondern vielmehr den Männern, auf deren geistreiche Forschungen ich meine Untersuchungen stützte, das etwa darin enthaltene Verdienst zuzuschreiben; v. Liebig und Schmidt sind es, denen wir jene Forschungen danken. — Das Einzige, was mir oblag, war, diese Erfahrungen auf dem

Gebiete der vergleichenden Physiologie für die Praxis brauchbar zu machen, eine Aufgabe, deren Erfüllung bei solchen Vorlagen, wie ich sie hatte, nicht schwierig ist. —

Nehmen Sie, hochverehrte Lehrer, diesen kleinen Beweis meiner steten Dankbarkeit freundlich auf, und schenken Sie ihm dieselbe Nachsicht, welche Sie mir stets zu Theil werden ließen.

London, den 8. November 1849.

Der Verfasser.

Als ich im vorigen Jahre in Griesingers Archiv für physiologische Heilkunde einige Bemerkungen „über den phosphorsauren Kalk in physiologischer und therapeutischer Beziehung“ veröffentlichte, stellte ich das Resultat meiner damaligen Beobachtungen als fraglich hin und gab die Entscheidung über dasselbe künftigen Erfahrungen anheim. Leider habe ich eine längere Zeit hindurch durchaus von der Verfolgung jenes Gegenstandes abstehen müssen; nur in den letzten 3/4 Jahren ist es mir vergönnt gewesen, am deutschen Hospitale in London die Beobachtungen mit wünschenswerther Genauigkeit fortzusetzen. Allein diese kurze Zeit hat genügt, einerseits die früheren Resultate zu bestätigen und andrerseits Erweiterungen und Berichtigungen der damaligen Ansichten zu ermöglichen. Ich sehe mich daher im Stande schon jetzt einzelne feststehende Resultate zu veröffentlichen und beeile mich damit um so mehr, als sie mir einer weitern Verbreitung werth zu sein scheinen. — Es ist nicht sowohl der Gewinn eines einzelnen feststehenden Factums, eines einzelnen Heilmittels für gewisse pathologische Zustände, welcher mich zu dieser Veröffentlichung antreibt; ich möchte vielmehr die durch denselben vervollständigte und abgeschlossene Erkenntniß eines durchgreifenden, höchst interessanten Naturgesetzes auf der einen, auf der andern Seite aber das Princip, nach welchem jenes Heilmittel gefunden wurde und dessen weitere Verfolgung die schönsten Resultate für die Therapie zu versprechen scheint, als die wichtigeren Punkte der vorliegenden Abhandlung bezeichnen.

Ich beginne auch hier wieder mit dem Hinweis auf die unbegreifliche Vernachlässigung, mit welcher man in Betrachtung humoralpathologischer Vorgänge die unorganischen Bestandtheile des Blutes behandelt hat; es kann dieser Punkt nicht dringend genug hervorgehoben werden. Stehen auch der richtigen Erkenntniß jener Bestandtheile im physiologischen sowohl, als pathologischen Zustande unendliche Schwierigkeiten entgegen, sie müssen überwunden werden, wenn wir jemals in den Besitz einer rationellen Humoralpathologie gelangen wollen; wir können einmal keine Blutkrankheit beurtheilen ohne die Verhältnisse der organischen und unorganischen Bestandtheile an und für sich, so wie ihre gegenseitigen Abhängigkeitsverhältnisse zu kennen. Es walten im Bereiche des organischen Geschehens, welches von der ersten Bildung organischer Verbindungen aus unorganischen Grundstoffen in der Pflanze bis zur Wiederauflösung der tausendfältig variirten organischen Verbindungen im Thierreich zu den ursprünglichen unorganischen Grundstoffen eine ununterbrochene Kette von Bildungsprocessen darstellt, bestimmte, durchgreifende, große Gesetze. Wir kennen zur Zeit nur wenige derselben; eins scheint sich jedoch mit Bestimmtheit dahin aussprechen zu lassen, daß eine gewisse Anzahl der unorganischen Bestandtheile des Erdbodens überall die Herstellung und Umsetzung organischer Verbindungen vermittelt; dieselben Salze, welchen wir in der Pflanze begegnen, finden wir im niedern, wie im höhern Thierreiche wieder; eine gleich wichtige Rolle, wie sie nach Liebig's unzweifelhaften Nachweisen in der Pflanze spielen, müssen sie ohne alle Frage auch im Thierreiche übernommen haben. — So lange wir dieses Gesetz nicht anerkennen, so lange wir es nicht auf die Humoralpathologie anwenden und bedenken, daß mit dem Mangel oder Ueberschuß dieses oder jenes anorganischen Bluthestandtheils ein pathologischer Zustand herbeigeführt werden muß, daß viele noch dazu auf hypothetische Abnormitäten des Fibrins

und Albumins reducirte Blutkrankheiten auf pathologischen Verhältnissen der unorganischen Bestandtheile beruhen können, so lange sind wir auch von einer richtigen Auffassung der Humoralpathologie entfernt. Nur eine sehr oberflächliche Kenntniß der Physiologie ist erforderlich, um einzusehen, von welchen bedeutenden praktischen Consequenzen eine Erfahrung sein muß, wie z. B. die, daß bei dem Genuß von Fleisch und Brot sich die Menge der phosphorsauren Alkalien mehrt, während die der kohlensauren abnimmt, daß andrerseits bei den Herbivoren geradezu ein umgekehrtes Verhältniß stattfindet. Schon ein flüchtiger Blick in die folgenden Blätter wird zeigen, welch' wichtige Rolle der phosphorsaure Kalk, wie im Pflanzen- und niedern Thierreich, so in den höchsten Organismen spielt.

Ich kann mich hier nicht weiter mit einer Beweislieferung von Seiten der allgemeinen und vergleichenden Physiologie beschäftigen; gleich wenig Zweifel an der hohen Bedeutung, welchen die unorganischen Bestandtheile des Blutes im gesunden und kranken Zustande haben müssen, lassen aber directe Untersuchungsresultate aufkommen. Allgemein ist der Einfluß der Salze auf die Löslichkeit des Albumins und Fibrins, so wie der Blutkörperchen bekannt; wir wissen, daß die Gerinnung des Fibrins in einer Salzlösung schwer oder gar nicht zu Stande kommt; wir wissen, daß ein bestimmtes Verhältniß des Salzgehaltes zum Serum erforderlich ist, wenn die Rinde der Blutkörperchen im normalen Zustande verharren, d. h. fest bleiben soll *). Die höchst interessanten Versuche Magendies lehren uns den decomponirenden Einfluß von Injectionen alkalischer Flüssigkeiten in das Blut kennen; die Bemerkung Zimmermann's **), daß im entzündlichen Blute die Salze vermindert sind, ist in der That

*) Lehmann, physiologische Chemie, pag. 202.

**) Analyse und Synthese pseudoplastischer Processe. pag. 207.

nicht minder wichtig, als die Kenntniß von der gleichzeitigen Vermehrung des Fibrins. Die gröbsten Versuche reichen hin, um uns auf die hohe Wichtigkeit der Salze hinzuleiten; brauchen wir doch nur einen eiweißhaltigen Urin, welcher alkalisch reagirt, zu kochen, um zu sehen, daß der Alkaligehalt desselben die Gerinnung des Albumnes verhindert, um weiter schließen zu können, daß ein zu großer oder zu geringer Alkaligehalt des Blutes Abnormitäten der Löslichkeitsverhältnisse des Albumens herbeiführen muß. — Es bedarf keiner weitern Beweise für die obige Behauptung; daß aber in der That in den humoralpathologischen Schriften fast gar nicht an die anorganischen Bestandtheile des Blutes gedacht ist, daß der Vorwurf der Vernachläßigung ein durchaus gerechter ist, dafür kann ein Jeder in jenen Schriften selbst den Beweis finden. Sämmtliche Blutuntersuchungen von Andral, Becquerel und Rodier (hier finden wir Angaben über die fraglichen Bestandtheile, aber so oberflächlich, daß sie kaum brauchbar sind), Simon, selbst Zimmermann zum größten Theil berücksichtigen fast nur die organischen Bestandtheile; in Haeser's Schriftchen „über die gegenwärtigen Standpunkte der pathologischen Chemie des Blutes", worin die Untersuchungen von Andral und Gavarret, Becquerel und Rodier und Popp in alle möglichen Proportionen gebracht und zu zum Theil sehr unzuverläßigen und unpraktischen Schlußfolgerungen benutzt werden, finden wir nur am Schlusse die kurze Bemerkung, daß es „sehr zu wünschen sei, daß bei künftigen Analysen das Verhalten der alkalischen Salze sorgfältig berücksichtigt werde"; erwähnt wird aber weiter nichts über das Wie und Warum. Selbst in den noch mehr compilirenden und kritisirenden Schriften neuerer Zeit, wie u. a. in Wunderlich's pathologischer Physiologie des Blutes — ein inhaltsschwerer Titel — vermissen wir jene Berücksichtigung; ein Mangel, der hier um so fühlbarer ist, als es eine Hauptaufgabe solcher Bestrebungen bildet, die Dürftigkeit des vorliegenden Thatbestandes aufzudecken. Daß aber die pathologische Anatomie, welche sich auch an die Begründung einer Humoralpathologie gemacht hat, eo ipso der obigen Anforderung nicht entsprechen kann, daß sie, eben weil die eine Hälfte der Blutbestandtheile gar nicht in das Bereich ihrer Betrachtung fällt, gradezu außer Stande ist eine Humoralpathologie zu schaffen, bedarf kaum der Erwähnung.

Es ist nicht die Aufgabe der vorliegenden Blätter näher auf die Standpunkte und Leistungen der heutigen Humoralpathologie, die in der That diesen Namen kaum verdient, einzugehen; der oben erwähnte Mangel derselben ist aber jedenfalls als einer der größten zu bezeichnen. Ich hoffe um so mehr, daß dieser kleine Beitrag zu einer allmäligen Beseitigung jenes Mangels willkommen ist; er kann nur einen neuen Beweis für die Wichtigkeit der anorganischen Bestandtheile des Blutes liefern.

Die Art und Weise, in welcher der phosphorsaure Kalk als Heilmittel für gewisse pathologische Zustände hier gefunden ist, ist von frühern Arten der Auffindung von Heilmitteln verschieden; nicht der Zufall, nicht das Experiment, sondern der vernunftgemäße Schluß hat dazu geführt. Von einer Betrachtung der organischen Bildungsprocesse im Pflanzen- und Thierreiche ausgehend, habe ich die Analogie eines dieser Processe im menschlichen Organismus vermuthet, und diese Vermuthung ist durch den Versuch und die Erfahrung zur Gewißheit erhoben; eben jene Vermuthung gab aber unmittelbar das Heilmittel an die Hand. Ich erwähne dies, weil ich glauben möchte, daß sich auf demselben Wege noch Vieles thun läßt, weil wir auf diese Weise Heilmittel finden, deren Rationalität keines Beweises bedarf, deren Einfachheit die beste Empfehlung ist. Immer wird die Empirie über den Werth therapeutischer Principien die allein entscheidende Stimme haben, allein es ist Sache der Theorie, ihr die Fragen vorzulegen. — —

Bekanntlich beschäftigt sich Liebig in einem großen Theile seiner Agriculturchemie mit dem schon erwähnten höchst wichtigen Nachweise, daß die unorganischen Bestandtheile des Erdbodens unerläßliche Requisite für die Bildung organischer Stoffe im Pflanzenreiche sind. Die Beweise für diese Behauptung finden sich in großer Menge vor und die zum Grunde liegenden Thatsachen lassen uns an ihrer Gültigkeit keinen Zweifel hegen; es handelt sich hier nicht um erschaffene oder theoretische Formeln, die ein Zweiter oder Dritter wieder anders geben kann; es sind vielmehr Facta, mit denen wir es zu thun haben, Facta, welche keinen Widerspruch erlauben.

Wir heben dem Zwecke dieser Bemerkungen gemäß nur Einiges aus jenen Beweisen hervor und verweisen hinsichtlich der weitern Ausführung auf das benannte Werk selbst. —

In dem Abschnitte über „die anorganischen Bestandtheile der Vegetabilien" beginnt Liebig mit dem Nachweise, daß dieselben, und insonderheit die Alkalien durchaus erforderlich sind zur Bildung der ersten organischen Verbindungen, der Säuren. „Alle diese Säuren", heißt es pag. 84 (5. Aufl.), „sind an Basen gebunden, an Kali, Natron, Kalk oder Bittererde; nur einige Pflanzen enthalten freie organische Säuren; diese Basen sind es offenbar, welch: durch ihr Vorhandensein die Entstehung der Säuren vermitteln; mit dem Verschwinden der Säure beim Reifen der Früchte nimmt der Kaligehalt des Saftes ab". Im Fortgange dieser Untersuchungen wird sodann über die Bildung des Amylons, des Zuckers, des Gummis und andrer stickstofffreier Verbindungen gehandelt, bis zuletzt auch die stickstoffhaltigen Substanzen in das Bereich der Betrachtung gezogen und auch für ihre Bildung die unorganischen Bestandtheile als höchst wesentlich nachgewiesen werden. In Bezug auf diese stickstoffhaltigen Verbindungen — Verbindungen, die in ihrer Zusammensetzung unsern organischen Blutbestandtheilen durchaus gleichartig sind, so daß

zwischen Pflanzenfibrin und Blutfibrin, Pflanzenalbumin und Blutalbumin u. s. w. die chemische Analyse keinen Unterschied mehr zu entdecken vermag — in Bezug auf sie, sage ich, kommt Liebig aber alsbald zu dem wichtigen Resultate (s. auch den Absch. über Wechselwirthschaft), daß es insonderheit die phosphorsauren Salze sind, deren Gegenwart für die Bildung jener „Blutbestandtheile" durchaus erforderlich ist.

Nur einige von den Bemerkungen Liebig's in Bezug auf diesen Punkt möchte ich erwähnen. „Keine von unsern Getreide- und Gemüsepflanzen", heißt es pag. 100, „kann ausgebildete Samen tragen, Samen, welche Mehl geben, ohne eine reichliche Menge von phosphorsauren Alkalien und phosphorsaurer Bittererde, und, eben weil sie stickstoffhaltig sind, ohne Ammoniak zu ihrer Ausbildung vorzufinden. Wir finden in der Knospe, in dem jungen Blatte Salze mit alkalischen Basen, wir finden die stickstoffhaltigen Bestandtheile stets begleitet von phosphorsauren Salzen und wir müssen annehmen, daß auch sie in den Lebensfunctionen der Pflanze eine gewisse Rolle spielen". „Die Alkalien", lesen wir ferner pag. 251, „sind vorzugsweise zur Erzeugung der stickstofffreien Bestandtheile, des Zuckers, des Amylon, des Pectins und Gummis nöthig; die phosphorsauren Salze wirken vorzüglich auf die Bildung der Blutbestandtheile". — „Wir müssen annehmen, daß zur Bildung der Halme, des Krautes, zur Fixirung des Kohlenstoffs, zur Erzeugung von Zucker, Amylon und Holzfaser eine gewisse Quantität Alkali (bei den Kalipflanzen), oder ein Aequivalent Kalk (bei den Kalkpflanzen) nöthig ist, allein wir müssen uns denken, daß mit aller Zufuhr von Ammoniak und Kohlensäure sich nur eine, den phosphorsauren Salzen entsprechende Menge der s. g. Blutbestandtheile in dem Organismus der Pflanzen bilden kann. Die Erzeugung der stickstoff- und schwefelhaltigen Bestandtheile des Saftes steht mit ihrer Gegenwart in der engsten Beziehung". (pag. 212).

Es ermangeln diese geistreiche Bemerkungen nirgends der interessantesten Beweise; eines weitern, auf ihnen basirten Umstandes, der eben für meinen Zweck der wesentlichste ist, haben wir jedoch jetzt besonders zu gedenken. Es bemerkt nämlich Liebig pag. 138: „Wir wissen, daß der Stärkegehalt der Kartoffeln in einem humusreichen Boden wächst, daß bei kräftigem, animalischem Dünger die Anzahl der Zellen zunimmt, während sich der Amylongehalt vermindert; in dem ersten Falle besitzen sie eine mehlige, in dem andern eine seifige Beschaffenheit. Die Runkelrüben, auf magerm Sandboden gezogen, enthalten ein Maximum von Zucker und kein Ammoniaksalz, und im gedüngten Lande verliert die Teltower Rübe ihre mehlige Beschaffenheit, denn in diesem vereinigen sich alle Bedingungen für Zellenbildung". Es lassen diese auf Erfahrung gestützten Behauptungen keinen Zweifel zu, daß sich eben in dem animalischen Dünger Stoffe vorfinden müssen, die der Zellenbildung günstig sind; diese Stoffe selbst sind aber die phosphorsauren Salze. Die Erfahrungen und Untersuchungen über den Dünger, vereinigt mit den obigen Bemerkungen über die Bedeutung der phosphorsauren Salze für die Bildung stickstoffhaltiger Bestandtheile der Pflanze rechtfertigen diesen Ausspruch; die positiven sowohl, als negativen Beweise liefert aber Liebig selbst. Jene werden hergeleitet aus der anerkannten Wirksamkeit der Knochen- und Aschendüngung überhaupt, diese werden durch die Ausforschung der Quellen der Kohlensäure, des Ammoniaks und des Wassers geliefert. — „Wir wissen", sagt Liebig pag. 239, „daß die thierischen Excremente in der Agricultur ersetzbar sind durch Materien, die ihre Bestandtheile enthalten. Da nun ihre Hauptwirksamkeit in ihrem Gehalte an den mineralischen Nahrungsstoffen beruht, welche die Culturpflanzen zu ihrer Entwickelung nöthig haben, so ist klar, da die Ernährung und das Gedeihen der wildwachsenden Pflanzen an die nämlichen Ursachen und Gesetze geknüpft

ist, daß wir mit den mineralischen Nahrungsstoffen der wildwachsenden Pflanzen, dies will sagen, mit ihrer Asche, unsere Felder in ganz gleicher Weise düngen können, wie mit Thierexcrementen, daß wir damit, wenn eine zweckmäßige Auswahl getroffen wird, unsere Aecker mit allen den Bestandtheilen wieder versehen können, die wir in der Ernte der Culturpflanzen hinweggenommen haben. — — Die Wichtigkeit der Aschendüngung fällt in die Augen, wenn man in Erwägung zieht, daß die mit kaltem Wasser ausgelaugte Holzasche kieselsaures Kali gerade in dem Verhältniß, wie im Stroh enthält, daß sie außer diesem Salze beträchtliche Mengen phosphorsaurer Salze enthält." Und ferner heißt es pag. 245: „Geben wir der Pflanze Kohlensäure und alle Materien, die sie bedarf, geben wir ihr Humus in der reichlichsten Quantität, so wird sie nur bis zu einem gewissen Grade der Ausbildung gelangen; wenn es an Stickstoff fehlt, wird sie Kraut, aber keine Körner, sie wird vielleicht Zucker und Amylon, aber keinen Kleber erzeugen. — Durch die Zufuhr von Ammoniak und damit von Stickstoff werden die Zwecke der Agricultur ebenfalls nicht erfüllt; so nothwendig das Ammoniak auch für die kräftige Entwickelung der Pflanze ist, so reicht es dennoch für sich allein nicht aus zur Erzeugung von vegetabilischem Casein, Fibrin und Albumin, denn ohne die begleitenden Alkalien, ohne schwefelsaure und phosporsaure Salze kennen wir diese Stoffe nicht; wir müssen voraussetzen, daß ohne ihre Mitwirkung das Ammoniak auf die Entwickelung und Bildung der Samen nicht die geringste Wirkung ausübt, daß es ganz gleichgültig ist, ob wir Ammoniak zuführen oder nicht, es wird keinen Antheil nehmen an der Bildung der Blutbestandtheile, wenn die andern Bedingungen zu ihrer Erzeugung nicht gleichzeitig vorhanden sind. In den flüssigen und festen Excrementen haben wir alle diese Bedingungen beisammen, keine fehlt; wir haben darin nicht nur das Ammoniak,

sondern auch die Alkalien, die kieselsauren, phosphorsauren und schwefelsauren Salze."

Wissen wir demnach, um es kurz zu wiederholen, daß die Production stickstoffhaltiger Substanzen gebunden ist an die Gegenwart der phosphorsauren Salze, daß die Zellenproduction zunimmt mit der Kraft und Menge des Düngers, daß diese Kraft wieder abhängt insonderheit von der Gegenwart jener Salze, (denn die andern Salze scheinen in der That unwesentlich für die Erzeugung der s. g. Blutbestandtheile zu sein), so kann, wie ich meine, kein Zweifel an der obigen Behauptung mehr obwalten, und wir gelangen demnach zu dem für uns wichtigen Resultate, daß die phosphorsauren Salze nicht nur für die Bildung stickstoffhaltiger Bestandtheile der Vegetabilien von der größten Wichtigkeit, sondern daß sie auch die Vermittler des Zellenbildungsprocesses im Pflanzenreiche sind.

Es kommen nun in den Pflanzenstoffen verschiedene phosphorsaure Salze vor; phosphorsaures Natron, phosphorsaurer Kalk, phosphorsaure Bittererde und phosphorsaures Eisenoxyd. Haben wir, so müssen wir demnach weiter fragen, Grund zu der Annahme, daß eine dieser Verbindungen insonderheit dem Zellenbildungsprocesse förderlich ist, oder sind sie es alle in gleichem Maaße? Ich glaube, daß die vorliegenden Erfahrungen entschieden zu Gunsten der ersten Annahme ausfallen; sie lassen kaum einen Zweifel übrig, daß es der phosphorsaure Kalk ist, welcher die Bildung der stickstoff- und schwefelhaltigen Bestandtheile, so wie namentlich der Zellenmembran vermittelt. Wir führen, um einen Beweis für diese Behauptung zu liefern, nur die eine höchst wichtige Erfahrung an, daß in England wie durch einen Zauber der Ertrag der an phosphorsauren Salzen erschöpften Felder um das Doppelte erhöht wurde, als man die Einfuhr von Knochen und die Düngung der Felder mit denselben betrieb

(vergl. Liebig pag. 216). Die Knochendüngung hat sich jetzt zu einer allgemein anerkannten Wichtigkeit in der Agricultur emporgehoben, und wir können nur um so weniger an der hohen Wahrscheinlichkeit jener Bedeutung des phosphorsauren Kalkes zweifeln. Fehlen auch zur Zeit noch ganz directe Untersuchungen, Untersuchungen wie die, von denen sogleich die Rede sein wird — die vorliegenden die Bildung der Pflanzenzelle und ihres Inhaltes betreffenden Thatsachen greifen so harmonisch ineinander, die daraus zu ziehenden Folgerungen besitzen einen so hohen Grad von Wahrscheinlichkeit und Zuverlässigkeit, daß auch eben die Folgerung, um welche es uns zu thun ist, mit großer Gewißheit gezogen werden darf, daß, mit andern Worten, der phosphorsaure Kalk als durchaus nothwendiges Erforderniß für den Zellenbildungsproceß im Pflanzenreiche betrachtet werden muß. —

Die diesem letztgezogenen Schlusse zu Grunde liegenden, in aller Kürze von mir angeführten Hauptpunkte finden, wie gesagt, sämmtlichst ihre weitere Begründung und Ausführung in Liebig's ausgezeichnetem Werke. Ich muß es einem Jeden überlassen, dieselben dort weiter nachzusehen. Für die Behauptung jedoch, daß es von den phosphorsauren Salzen insonderheit und fast ausschließlich der phosphorsaure Kalk ist, welcher jene Bildungsprocesse vermittelt, entnehmen wir weitere Beweise aus den vielfach angestellten Pflanzen-Aschenanalysen, deren Resultate von Enderlin, Fresenius, Will u. A. in mehren Jahrgängen der Annalen der Chemie und Pharmacie von Liebig und Wöhler mitgetheilt sind. Der Gehalt an phosphorsaurem Eisenoxyd stellt sich in diesen durchweg als sehr unbedeutend heraus, während der phosphorsaure Kalk immer in einer gewissen, je nach dem Boden größern oder geringern Quantität zugegen ist. Daß es aber von dem phosphorsauren Kalk und dem phosphorsauren Natron lediglich der erstere ist, dem wir die fragliche Bedeutung zuschreiben müssen, geht, die große Fruchtbarkeit der Felder in Folge der Knochen-

düngung berücksichtigt, daraus hervor, daß die Quantität des Natron in den Knochen nach Analysen von Berzelius u. A. nur eine sehr geringe ist. Dasselbe läßt sich von der Magnesia sagen, die noch dazu aller Wahrscheinlichkeit nach als kohlensaure in den Knochen enthalten ist. (Vergl. Berzelius Lehrbuch der Chemie. IV. 1. pag. 446. 1831.)

Anmerkung. Nachträglich füge ich noch eine für diesen Gegenstand sehr wichtige Bemerkung aus Dr. C. Schmidt's Entwurf einer allgemeinen Untersuchungsmethode der Säfte und Excrete des thierischen Organismus, Mitau und Leipzig 1846. pag. 61 hinzu. Es heißt daselbst: „Bei Gelegenheit einer Untersuchung über die mit dem Namen Pflanzenschleim, Bassorin, Cerasin bezeichnete Gruppe von Stoffen, sämmtlich die Elemente von Kohle und Wasser enthaltenden morphologisch-chemischen Zwischenstufen des Uebergangs von Gummi und Stärkemehl in Holzfaser, richtete ich besonders meine Aufmerksamkeit auf das Verhältniß des phosphorsauren Kalkes zu diesen Materien. Er ist stets in löslicher Form, mit einem Albuminat verbunden, dem Zelleninhalt eingemengt, nie durch's Microscop krystallonomisch zu diagnosticiren oder nur wahrzunehmen. Concretionen, s. g. Verkalkungen von Kalkphosphat finden sich übrigens in der Pflanzenzelle nie; ein schlagender Beweis für den erwähnten Satz, daß der phosphorsaure Kalk in sehr innigen Beziehungen zum Zellenbildungsproceß steht, also in der Pflanze, wo dieser überall und zu jeder Zeit fortdauert, nirgends entbehrt, als unnützer Ballast, gleich andern Excreten, oxalsaurem, schwefelsaurem, kohlensaurem Kalk, in krystallisirter Form abgelagert werden kann."

Hiemit die uns interessirenden Verhältnisse im Pflanzenreiche beschließend, wenden wir uns zum niedern Thierreiche. — Was dasselbe anbetrifft, so stehen gegen die bedeutenden Fortschritte in der Kenntniß seiner Anatomie und Physiologie, Fortschritte, die wir bei der bedeutenden Vervollkommnung der Beobachtungswerkzeuge einem Müller, Wagner, v. Siebold, u. A. verdanken, die Kenntnisse bei weitem zurück, welche die chemischen Verhältnisse der Bildungsprocesse und des Stoffwechsels angehen. — Die große

Schwierigkeit der einschlagenden Untersuchungen giebt einen hinreichenden Grund für die dürftige Förderung dieses Theiles der Zoochemie ab. Mit um so größerer Freude aber dürfen wir Arbeiten begrüßen, deren Verfasser in dem Verfolgen geistreicher Aufgaben keine Mühe gescheut haben und nach überwundenen Schwierigkeiten die Wissenschaft mit den wesentlichsten Resultaten bereicherten. Zu diesen Arbeiten rechnen wir mit vollstem Rechte einen im Jahre 1845 erschienenen Beitrag „zur vergleichenden Physiologie der wirbellosen Thiere. Eine physiologisch-chemische Untersuchung von Dr. Carl Schmidt." Sie ist für unsern Zweck von hoher Wichtigkeit, und die schon aus mehrfachen Artikeln in Liebig's und Wöhler's Annalen bekannte Tüchtigkeit des Verfassers in chemischen Untersuchungen bürgt uns für ihre Zuverlässigkeit.

Schmidt spricht nun in diesem Werke die Vermuthung aus, daß der phosphorsaure Kalk in inniger Beziehung zum Zellenbildungsprocesse stehe, und erhebt diese Vermuthung durch seine eigenen Versuche zur höchsten Wahrscheinlichkeit, wenn nicht zur Gewißheit. Er kam zu dem höchst interessanten Resultate, daß bei den Gliederthieren, von denen er den Flußkrebs, die Squilla und den Hummer zu Untersuchungen gebrauchte, der Gehalt an phosphorsauren Erden proportional der Quantität des organisirten Chitingewebes (eines in Wasser, Alkohol, Aether und Kali unlöslichen, farblosen, durchscheinenden, in der Reihe der wirbellosen Thiere als Hauptbestandtheil des Skelettes weit verbreiteten Stoffes) steigt; er fand damit Resultate früherer Untersuchungen von Mérat, Guillot, Chevreul und Göbel bestätigt. „Das Chitinfasergewebe ist aber," wie Schmidt sagt, „das Resultat eines lebhaften Zellenbildungsprocesses beim Schalenwechsel; die Quantität phosphorsauren Kalkes steigt also mit der Intensität dieses Processes; die relativen Mengen geformten Gewebes geben für sie den Maaßstab ab. Der phosphorsaure Kalk muß demnach in inniger Beziehung zum Zellenbildungsproceß stehen." Noch deutlicher und

mit Bestimmtheit geht dies aus dem folgenden Versuche Schmidt's hervor. Es heißt pag. 44: „Ich leitete bei einigen Krebsen durch schichtweises, vorsichtiges Abtragen eines Theils des Brust- oder Scheerenpanzers bis auf die oberste Pigmentschichte der darunter liegenden Membran einen Neubildungsproceß ein. Dieser erfolgt rasch; nach acht Stunden fand sich schon eine dicke, zähe, klare Masse ausgeschwitzt (Cytoblastem); in dieser zahlreiche, in Kali und Essigsäure unlösliche Kügelchen (Fettbläschen) und andere darin lösliche Molecüle (Albuminate), sonst keine körperlichen Theile; eingeäschert hinterblieb eine bedeutende Menge phosphorsaurer Kalk (nach approximativer Bestimmung 8%) nebst etwas phosphorsauren Alkalien und kohlensaurem Kalk, der als solcher nicht präexistirte. Dieser phosphorsaure Kalk war in gelöster Form darin, denn Ammoniak trübte die unter dem Microscope befindliche Masse sehr stark. Nach 14—16 Stunden hatten sich die löslichen Molecüle (Albuminate, vielleicht auch phosphorsaurer Kalk) um die Fettbläschen zu kuglichen Massen angehäuft; einige dieser Kugelhaufen hatten sich bereits mit einer Membran umgeben (primäre Zellen), andere noch nicht; gleichzeitig befanden sich zahlreiche rhomboëdrische Krystalle (von kohlensaurem Kalk) darin, die mit Säuren aufbrausten. Bei Behandlung mit Kali quollen die primären Zellen nebst körnigem (Albuminat?) Inhalt stark auf, wurden durchsichtig und lösten sich; in jeder kam das Fettbläschen als Kern zum Vorschein; sie bestanden demnach noch nicht aus Chitin, wenn dies sich nicht vielleicht im frühen Zustande wie Gummi zu Zellenmembran verhält, d. h. löslich ist. — Nach 24—36 Stunden fanden sich unter denselben Elementen viele dieser primären Zellen lang gestreckt, spindelförmig, die in Kali noch aufquollen, sich jedoch nicht mehr lösten, demnach schon aus Chitin zu bestehen schienen. Ich konnte den Proceß nicht weiter verfolgen, da mir die Thiere aus

Unvorsichtigkeit starben und es zu spät im Jahre war, um neue zu verschaffen."

Diesen für den Zellenbildungsproceß im Thierreich so sehr interessanten Versuchen giebt Schmidt im Verfolge seiner Arbeit auch noch den negativen Beweis für die aufgestellte Behauptung bei. Er fand nämlich bei Helix (pomatia, nemoralis und hortensis) bei Untersuchung des Kalkgehäuses und dessen innerster Schichte, einer glashellen, strukturlosen Membran fast gar keinen phosphorsauren Kalk und dem entsprechend fast gar keinen Zellenbildungsproceß, lauter amorphe, erhärtete, von Kalkschichten getrennte Schleimmassen (Albuminate); Schmidt selbst sagt: „das Zusammentreffen ist zu auffallend, als daß man es nicht als Bestätigung der aufgestellten Ansicht über die physiologische Bedeutung jenes Salzes ansehen sollte." „Ich glaube," heißt es pag. 56, „wie gesagt, daß eine bestimmte Verbindung von Albumin mit phosphorsaurem Kalk, oder besser, eine mit einer gewissen Portion des letztern gesättigte Albuminlösung vorzugsweise die Fähigkeit besitzt, sich in Berührung mit heterogenen Körpern zu relativ festen Membranen um diese herum zu verdichten, d. h. die Wand primärer Zellen zu bilden — doch ist mir's bis jetzt nicht gelungen, experimentell mit genügender Schärfe das Wie? und Warum? zu ermitteln."

Wenn wir hiernach nun zu dem Resultate gelangt sind, daß der phosphorsaure Kalk in dem Pflanzen- und niedern Thierreiche ein für den Zellenbildungsproceß nothwendiges Requisit sei, liegt dann nicht die Vermuthung sehr nahe, daß es sich in den Organismen der höhern Thierklassen und des Menschen ebenso verhalte, daß weiterhin mit dem Mangel desselben ein zur Erzeugung einer der Zufuhr von Nahrungsmitteln entsprechenden Quantität organisirten Gewebes höchst wesentliches Erforderniß hinweggenommen sei? — Wir kennen die durchgreifende Einfachheit und Gesetzmäßigkeit der Lebensprocesse der Mikrokosmen und ihr Verhält-

niß zum Makrokosmus nur zum kleinen Theil; dennoch, so weit wir sie kennen, berechtigt sie uns zur Aufstellung solcher Fragen, und stellt sich eine bejahende Antwort derselben heraus, so wird unsere Erkenntniß und unsere Bewunderung jener Einfachheit zur weitern Verfolgung ähnlicher Fragen antreiben. — Das Blut, das Bildungsmaterial sämmtlicher Bestandtheile unsers Körpers, enthält sämmtliche Stoffe, die irgend zur Bildung jener Bestandtheile erforderlich sind; fehlt irgend einer dieser Stoffe, ist er nicht in seiner normalen Quantität als Nahrungsmittel eingeführt, so muß sich eine Anomalie im Stoffwechsel, in der Anbildung neuen Gewebes, in der Reproduction, kurz es muß sich ein pathologischer Zustand herausstellen; diese nothwendige Consequenz erhebt ohne Zweifel die Humoralpathologie zu unserer wichtigsten Lehre. Tausendfältig mögen jene Zustände in der Art vorkommen, daß sie unsern kurzsichtigen Augen verborgen bleiben; wir werden erst dann Zeugen pathologischer Processe, wenn sie eine bestimmte Höhe erreicht haben.

Ich habe mir nun die obige Frage vorgelegt und die Beantwortung derselben wird sich aus den folgenden Blättern ergeben. Wenn ich aber auch selbst der festen Ueberzeugung bin, daß die aus den vorliegenden Beobachtungen zu ziehenden Resultate zuverlässig und fest begründet sind, so möchte ich mich dennoch vor dem Vorwurf der Leichtfertigkeit und Voreiligkeit insofern verwahren, als das dem Einzelnen zu Gebote stehende Material immer zu gering ist, um über Fragen, wie die meinige, mit ganz entschiedener Sicherheit zu entscheiden; von weitern Beobachtungen, und zwar von fremden, müssen meine Resultate ihre Bestätigung erwarten.

Auf experimentellem chemischem Wege zu prüfen, ob der phosphorsaure Kalk zum normalen Zellenbildungsprocesse durchaus erforderlich sei, war mir nicht in der Weise möglich, wie ich es wünschte; es gehören hierzu die feinsten, chemischen Untersuchun-

gen, Aschenanalysen des Blutes, Untersuchungen frischer Blasteme u. s. w. Der Versuche, welche ich in dieser Beziehung angestellt habe, wird weiter unten Erwähnung geschehen. Ich habe mich deshalb sogleich an die Praxis gewandt und mir die Frage vorgelegt, ob nicht durch innere Darreichung des phosphorsauren Kalkes der Heilungsproceß der langwierigen s. g. atonischen Geschwüre, namentlich der scrophulösen, gefördert werden könnte. Wenn man bedenkt, daß eben jene Stoffe, die das eigentliche Bildungsmaterial für alle plastischen Processe hergeben, ich meine das Fibrin (?) und Albumin, nach den Untersuchungen verschiedener Chemiker chemisch mit phosphorsaurem Kalk verbunden sind, wenn wir schon daraus für die wahrscheinliche Richtigkeit unserer ersten Frage eine Stütze herleiten können, so ist auch a priori gegen die Aufstellung dieser zweiten Frage nichts einzuwenden*).

Daß sich der phosphorsaure Kalk im Magen löst, leidet keinen Zweifel; denn einmal löst er sich überhaupt in Säuren und wird nur bei einem Ueberschuß von Alkalien, d. h. wenn die Flüssigkeit neutral oder alkalisch wird, wieder gefällt, und dieser Fall möchte wohl selten im Magen vorkommen, andrerseits aber

*) Lehmann giebt in seinem Handbuche der physiol. Chemie an, daß das Albumin immer mit phosphorsaurem Kalk in der Zusammensetzung der Knochenerde ($Ca^3 \ddot{P}^2$) verbunden ist; es ist diese Verbindung unstreitig eine chemische, da der phosphorsaure Kalk nicht nur in dem alkalischen Eiweiß aufgelöst ist, sondern auch dem Eiweiß bei der Coagulation durch Hitze oder Säuren, so wie bei der Fällung durch Metalloxyde folgt; genau ist indeß sein normales Verbindungsverhältniß mit dem Eiweiß noch nicht ermittelt; man hat im frischen Eiweiß 1, 8—11,0% Knochenerde gefunden. — Das Fibrin findet man immer von phosphorsaurem Kalk begleitet. Mulder will ziemlich constant im Blutfibrin 6,70% davon gefunden haben. Auch das Globulin ist mit phosphorsaurem Kalk verbunden; Lehmann fand in der Asche desselben 0,213%.

löst er sich auch im Albumin auf und wird deshalb, bald nach der Mahlzeit genommen, ohne Zweifel in gelöster Form in das Blut übergehen.

Ich ließ nun den phosphorsauren Kalk so rein, als möglich darstellen; anfangs geschah dies durch Digeriren des Cornu cervi ust. ppt. mit Phosphorsäure und nachheriges Trocknen des Präparates; später wurde er aus phosphorsaurem Natron und Chlorcalcium dargestellt, und dies letztere Präparat namentlich in Anwendung gebracht.

1ste Beobachtung. Es betraf diese ein fünfjähriges Mädchen, L. G., welches schon seit zwei Jahren an einem beständig eiternden und nach Abweichung der Kruste wieder verschorfenden Geschwüre mitten und oben auf dem Kopfe litt.

Das Kind trägt deutlich ausgeprägt den scrophulösen Habitus, lebt in sehr ärmlichen Verhältnissen und erhält fast nur die ihm am schädlichsten Speisen, als Schwarzbrod, Kartoffeln u. s. w. zur Nahrung. Am 8. Februar 1847 sah ich das Kind zuerst. Ueber den Gesundheitszustand im Allgemeinen keine Klage; das Geschwür selbst hatte die Größe eines Thalers, die dasselbe umgebende Haut war zwar etwas härtlich anzufühlen, allein tuberkulöse Ablagerungen in dieselbe hatten nicht stattgefunden. — Drei Monate hindurch blieb nun trotz aller möglichen Versuche das Geschwür in unverändertem Zustande. Der innerliche Gebrauch des Ol. jec. aselli, so wie zeitweilig gelinder salinischer Purgantia, die örtliche Anwendung einer Natronsalbe (Natr. carbonic. ʒij Axung ℥ß), die mir oft nützlich war, der Versuch durch mehrfache Scarificationen und Ugt. basilic. den Granulationsproceß zu fördern, Derivantia im Nacken, Ueberschläge von einer Auflösung des Kali caustic., Cauterisation mit Lapis infernal., Zink- und Bleisalben, Alles blieb ohne Erfolg. Hiernach entschloß ich mich beim herannahenden Sommer eine Zeit lang nichts

anzuwenden; ich sah das Kind längere Zeit gar nicht. Als ich doch mittlerweile auf die Frage nach der Wirkung des phosphorsauren Kalkes stieß, suchte ich es wieder auf; das Geschwür war ganz unverändert. — Ich ließ nun die Kruste abweichen und verordnete den 26. July Calcar. phosphoric. gr. iß Sacch iß Dos. xij. Täglich zweimal ein Pulver zu nehmen. Schon nach drei Tagen bemerkte ich eine unzweifelhafte Veränderung der Suppuration; der bis dahin mehr dünnflüssige Eiter verwandelte sich in ein pus bonum et laudabile, und ich war nicht wenig erstaunt, als am 4. August die Vernarbung des Geschwürs von den Rändern aus begann. Die Verordnung wurde wiederholt, pr. dosi 2 gr. Calcar. phosph. gegeben und bei gleichmäßigem Fortschreiten des Vernarbungsprocesses war das Geschwür am 17. August total verheilt. — Bei der reinen Anwendung des Kalkes, bei der unveränderten Fortsetzung der gewöhnlichen Diät und Lebensweise des Kindes konnte hier keine Täuschung obwalten, und ich mußte schließen, daß es allein jenes Mittel war, welches einen normalen Zellenbildungsproceß herbeigeführt hatte. Nach nicht langer Zeit (etwa einem halben Jahre) brach jedoch die vernarbte Stelle von Neuem durch und es wollte mir nicht so rasch gelingen, die Schließung herbeizuführen; auch weiß ich leider nicht, ob die Heilung zu Stande gekommen ist, da ich in der Beobachtung plötzlich unterbrochen wurde und das Kind nicht weiter gesehen habe; erwähnen möchte ich aber, daß das zweite weniger günstige Resultat sicher zum Theil durch die sehr mangelhafte Pflege, durch die sehr ärmliche Lebensweise, Aufenthalt in einer ganz dunstigen, von einer Menge Menschen überfüllten Wohnstube, lediglichen Genuß von Schwarzbrod und Kartoffeln u. s. w. herbeigeführt ist. Was das Aufbrechen der Narbe selbst anbetrifft, so war mir dasselbe nicht eben wunderbar; denn mag es auch im scrophulösen Blute an phosphorsaurem Kalk fehlen, so braucht die Dyskrasie als solche durchaus nicht auf diesem einen Deficit zu

beruhen und mit Darreichung des Kalkes können wir wohl die Erscheinungen vertreiben, welche sein Mangel herbeiführt; deshalb aber noch nicht die Gesammtdyskrasie heben.

2te Beobachtung. In derselben Zeit, wo ich mit der ersten Beobachtung beschäftigt war, wurde mir ein anderes Kind, E. B., sieben Jahr alt, zugeführt. Die kleine Patientin, die den scrophulösen Habitus nur in geringem Grade trug, litt seit vier Jahren an scrophulösen Geschwüren. Zwei solcher Geschwüre im Gesichte wurden vor zwei Jahren von einem Arzte operirt, die in der Haut liegenden Tuberkel entfernt und die Wunde verheilte. Alsbald jedoch zeigten sich an dem Dorsum jeder Hand zwei neue Geschwüre, die zwei Jahre lang bestanden und erst vor zehn Wochen, mit dem Aufbruch zweier neuen Ulcera an der innern Seite eines jeden Oberarms, vernarbten. Mit diesen beiden Geschwüren kam das Kind zu mir; sie hatten die Größe eines Sechsgroschenstückes und sonderten ein eitrig-jauchiges Fluidum ab; unter dem des rechten Armes lag ein Tuberkel von der Größe einer Haselnuß. Am 13. August wurde sogleich Calcar. phosphoric. gr. iv Sacch. ℥ß, täglich zweimal ½ Pulver, verordnet. Ich war nicht wenig überrascht, schon am 16. August in den tiefgreifenden Geschwüren eine gute Eiterung und auf ihrem Boden die üppigsten Granulationen zu sehen. Am 19. August waren beide bis auf eine erbsengroße Stelle verheilt und ohne daß örtlich irgend etwas in Anwendung gebracht wurde, war das Geschwür des linken Arms am 31. August total vernarbt; das des rechten Arms blieb längere Zeit in Eiterung, verheilte aber auch im Laufe des folgenden Monates zur Freude der Eltern gänzlich. Im Monat December öffnete sich das letztere noch einmal wieder; der unterliegende Tuberkel war zwar kleiner geworden, aber doch noch vorhanden; allein jetzt ist nach wiederholter Anwendung des Calcar. phosph. auch dieses wieder verheilt. Es ist diese Beobachtung

eine durchaus reine und ich mußte deshalb auch hier dieselbe Vermuthung hegen, wie bei der ersten Beobachtung.

3te Beobachtung. Diese wurde an einem Kinde in einer durch und durch scrophulösen Familie gemacht: J. F., fünf Jahre alt. Die Patientin wurde schon seit einem halben Jahre von mir an einer Impetigo achor, die sich über die ganze Kopfhaut erstreckte, behandelt. Neben der innern Darreichung bekannter Antiscrophulosa wurden örtlich anfangs zur Losweichung der Krusten Mandelemulsin-Ueberschläge angewandt; allein so oft sie sich lösten, erzeugten sie sich auch wieder. Der ganze Kopf war mit einem Schorfe bedeckt; die Haare waren natürlich gleich anfangs gänzlich abgeschoren. Obstructionen, Hyperämien des Kopfes machten oft die Anwendung salinischer Purgantia bei dem vollsaftigen Kinde erforderlich. Der Erfolg dieser Behandlung war jedoch sehr wenig erfreulich, bis endlich örtlich eine Natronsalbe und innerlich zunächst Natr. carbonic. und dann Natr. phosphoric. angewandt wurde. Dabei trat, wiewohl die Diät des Kindes nur eine sehr kümmerliche war, alsbald Besserung ein; die Krusten fielen allmählig ab und eine neue gesunde Epidermis bekleidete alsbald den ganzen Kopf. Nur an zwei Stellen, über beiden Ohren, blieben Ulcera von der Größe eines Thalers zurück und diese wollten bei der bezeichneten Behandlung nicht verschwinden. Am 29. July wurde daher Calcar. phosphoric. (wie oben) verordnet und nach Verbrauch von 48 Gran derselben am 25. August waren die Geschwüre gänzlich verheilt und vernarbt. Das Kind befindet sich bis jetzt sehr wohl und die gesunde Kopfhaut ist mit jungem Haarwuchs bedeckt. — Ich überlasse die Epikrise dieses Falles dem Leser, da mir selbst bei dem dubiösen post hoc, ergo propter hoc, die Möglichkeit vor Augen schwebt, daß in dieser Beobachtung eine Nachwirkung der vorbezeichneten Behandlung hinsichtlich der Heilung der beiden Geschwüre stattgefunden haben könne. Der Umstand jedoch, daß

die letztere erst grade dann eintrat, als die Calcar. phosphoric. in Anwendung gebracht wurde, rechtfertigt vielleicht die Vermuthung, daß sie auch hier einen Antheil an dem Zellenbildungsproceß gehabt habe.

4te Beobachtung. Ann Mate, vierzehn Jahre alt, trägt in hohem Grade den scrophulösen Habitus. Zunächst vor $1^3/_4$ Jahren bekam sie Anschwellungen und Ulcerationen der dritten Phalangen der rechten Hand; dann stellten sich vor etwa $1^1/_2$ Jahren Geschwüre des rechten Enkels und der Zehen ein; daran wurde sie anfangs privatim, dann in zwei verschiedenen Hospitälern mit Cataplasmen, Heftpflasterverbänden, Ol. jec. Asell. u. s. w. behandelt. Am 13. December 1848 wurde sie im German-Hospital als Out-patient aufgenommen; ich sah das Kind zuerst am 17. Februar 1849. Bis dahin war Leberthran verordnet; allein die Geschwüre am rechten Malleolus und zwischen dem Hallux und der 2. Zehe des rechten Fußes blieben unverändert. Beide waren sehr tiefgreifend und sonderten einen schlechten, jauchigen Eiter ab. Am 17. Februar wurde Calcar. phosphoric. täglich dreimal 2 gr. verordnet. Am 3. März waren die Geschwüre schon in der Heilung begriffen. — Es litt das Kind zugleich an einer Olecranarthrocace des rechten Arms; das Gelenk war bedeutend geschwollen, ein fistulöser Canal führte von außen auf das Gelenk. Am 10. März bemerkte ich Folgendes: „das Ulcus am rechten Malleolus ist verheilt; der fistulöse Canal am Ellenbogen ebenfalls; das Ellenbogengelenk selbst ist dünner geworden. — Schöne Granulationen in dem Ulcus an den Zehen. Das Aussehen des Kindes gewinnt bedeutend an Frische". Diese Wirkungen konnte ich nicht umhin lediglich dem phosphorsauren Kalke zuzuschreiben; das Geschwür an den Zehen verheilte in der nächsten Zeit fast ganz. Allein die Dyskrasie des Blutes war, wenn auch verändert, doch nicht getilgt; das Kind bekam einen enorm großen jauchigen Abscess am rechten

Oberschenkel, einen kleinern unter dem processus zygomaticus an der Wange; auch wich die Olecranarthrocace nicht ganz. — Der erstere wurde geöffnet und sehr bedeutende Mengen jauchigen Eiters täglich entleert; der letztere schwand nach und nach von selbst; auffallend aber war, daß das Kind, trotz der starken Eiterungen eine gesunde und frische Farbe behielt. — Während die Eiterung noch fortdauerte, wünschten die Eltern die Patientin nach Margate, an die Seeküste, zu schicken; es wurde diesem Wunsche nachgegeben, und, wie ich höre, soll es dem Kinde dort wohl gehen.

5te Beobachtung. Isabella M., eilf Jahre alt, hat ein scrophulöses Geschwür in der rechten Parotidealgegend; es ist bemerkenswerth, daß das Kind erst im dritten Lebensjahre anfing zu gehen. Am 9. July werden 12 Pulver aus Calcar. phosphoric. gr. iv Sacch. gr. vj. dreimal täglich ein halbes Pulver zu nehmen, verordnet. Am 16. July war der Verheilungsproceß schon im Beginne; weitere zwölf Pulver führten die Heilung herbei.

6te Beobachtung. George Tailor, zwei Jahr alt, litt seit längerer Zeit an scrophulöser Ophthalmie, welche zu Geschwürbildung auf der Cornea führte, und gleichzeitig an Ulcerationen des Gesichts. — Der anfängliche Gebrauch des Ol. jec. Asell. führte keine Besserung herbei; am 25. August wurde sodann Calcar. phosphoric. verordnet. Am 8. September waren die Geschwüre des Gesichts verheilt, die Ophthalmie gebessert und die Ulcera cornea in der Verheilung begriffen. Aus einem Abscess am Arm wurde ein wäßriger Eiter entleert. Der retardirte Stuhl wurde durch salinische Purgantien bethätigt. Am 29. September waren alle Geschwüre verheilt.

7te Beobachtung. Clara J., dreißig Jahr alt, unverheirathet, scrophulöse Gesichtsbildung. — Neben vielfachen hysterischen Erscheinungen findet sich bei der Patientin ein kleiner Ab-

seß an der Nase; derselbe wird geöffnet. Die Oeffnung verheilt aber nicht, sondern es bildet sich ein Ulcus. Ol. jec. Asell. wird längere Zeit ohne Erfolg gegeben; auch Calcar. phosphor. führte anfangs keine Veränderung herbei, außer daß der Grund des Geschwürs besser wurde; es ergab sich aber, daß Patientin die Krusten immer abkratzte und dadurch die Verheilung verhinderte. Am 13. September wurde sie im Hospital aufgenommen und das Geschwür verheilte jetzt bei Anwendung des Kalkes binnen zehn Tagen gänzlich; die Kruste fiel dann von selbst ab. — Ich bemerke übrigens, daß die Patientin im Hospital auch Ol. jec. erhielt; dennoch schreibe ich die Verheilung des Ulcus der Calcaria zu, da mehrfache Erscheinungen der scrophulösen Dyskrasie, wie z. B. Harnsedimente von oxalsaurem Kalk ꝛc., noch nach der Verheilung fortbestanden und das Ol. jecor. dieselbe durchaus noch nicht gehoben hatte.

Diesen von mir selbst angestellten Beobachtungen füge ich einige hinzu, die ich der Güte des leider jüngst verstorbenen Herrn Dr. Schmidt sen. in Bremen verdanke. Sie bestätigen in sehr erfreulicher Weise meine Erfahrungen, und ich kann nicht unterlassen, die briefliche Mittheilung des Herrn Dr. Schmidt wörtlich wiederzugeben.

„Versuche mit Calcaria phosphorica:

8te Beobachtung. „Marie Struve, sechs Jahr alt, ein bleiches, cachektisches Kind, von lymphatischem Habitus, hatte schon früher anhaltend an scrophulösen Augenentzündungen, namentlich an Ulcera corneae, gelitten, die nur sehr langsam zur Heilung gelangten. Seit sechs Monaten genesen, wurde sie neuerdings befallen, und erschien jetzt mit einer scrophulösen Conjunctivitis und einem nicht unbedeutenden Ulcus corneae, zu dem ein ansehnlicher Streifen stark aufgetriebener Blutgefäße vom äußern Augenwinkel sich hinzog. Große Lichtscheu und starker Thränenfluß waren damit verbunden. Die Nase war aufgeschwollen und beide Nasenlöcher durch scrophulöse Geschwürbildung wund. Appetit war sehr gering und die Stimmung verdrossen und weinerlich. Eine schon früher gebrauchte Augensalbe ℞ Hydrarg. oxyd. alb. gr. iij. Butyr. rec. insuls. ʒij hatte die Mutter wieder in Gebrauch gezogen. Ich verordnete am 16. September 1847: ℞ Calcar. phosphoric. gr. v. Sacch. alb. β. Tal. dos. xij. S. Nach dem Frühstück und Mittagessen ein Pulver mit Wasser zu nehmen. Am 23. September war das Ulcus corneae kleiner, der Gefäßstreifen weniger intensiv geröthet und schmäler; die Lichtscheu geringer. Die Nasenlöcher waren weniger wund und das ganze Aussehen des Kindes hatte gewonnen. Der Appetit war vermehrt und das Kind fing an, heiterer zu werden. Am 18. September hatte die Augenkrankheit noch mehr abgenommen, die Nasenlöcher waren beinahe ausgeheilt. Die Gesichtsfarbe des Kindes ist sehr viel frischer und reiner, seine Fröhlichkeit und Heiterkeit setzt die Mutter in Erstaunen (der Mutter eigene Worte). Die Pulver werden nochmals wiederholt. Am 5. October konnte ich das Kind hergestellt entlassen. Nicht nur, daß das übel aussehende Ulcus schnell verheilt war, sondern es war bis auf eine leichte Trübung keine Spur desselben zurückgeblieben. Das Kind hatte sich im Allgemeinen vortheilhaft verändert und an Aussehen und Fülle gewonnen."

9te Beobachtung. „Martin Röber aus Weserdeich, im siebenten Jahre, blond, blauäugig, für sein Alter klein und unbeholfen, dagegen pastos, mit dickem Kopf, aufgedunsenem, stark gefärbtem Gesicht u. s. w. leidet schon seit 3½ Jahren an ausgebildeter Scrophulosis, die sich namentlich durch fast beständigen Catarrh und eine Menge Drüsen und Ulcera, sowohl in der Gegend der Ohren, als am Halse, bei dicker Nase und entzündeten Augen, ausspricht. Während dieser drei Jahre war zur Abhülfe durch Diät und anhaltenden Gebrauch von Ol. jec. Asell., Jod,

etc. Manches geschehen, ohne jedoch Heilung, wenn auch Besserung, zu erreichen. — Am 27. September hatte er noch 12 verschiedene Wunden und viele Knollen an der angegebenen Stelle. Dieselbe Vorschrift von Calcar. phosphoric. Dos. xxiv, wie oben, wurde auch ihm gegeben. Am 25. October erschien er wieder; alle seine Geschwüre hatten sich vernarbt, auf einigen fand sich noch eine Kruste, ähnlich der Lepra alphosa. Die Drüsenverhärtungen waren kleiner, das gedunsene Gesicht war mehr beigefallen und sichtlich war das Kind lebhafter und behaglicher geworden; überhaupt schien sich das Allgemeinbefinden so gebessert zu haben, daß ich jetzt die Mutter auffordern konnte, den Knaben eine eine halbe Stunde von seiner Wohnung entfernte Schule besuchen zu lassen, was ich am 27. September auf ihr Befragen noch verweigern mußte. Ich verordnete nochmals für 24 Tage die Calcar. phosphoric. und empfahl, mit dem Kinde nochmals zu mir zu kommen, was aber nicht geschah; wahrscheinlich ist die Besserung nachhaltig geblieben."

10te Beobachtung. „Fräulein P., 46 Jahre alt, litt seit einigen Jahren an lymphatischen Geschwülsten an der Wangenseite, am Halse, am Schultergelenk, am Ellbogen, am Knie u. s. w., von denen mehre in Eiterung übergegangen sind und üble Geschwüre bilden. Ein Versuch mit Calcar. phosphoric., zweimal täglich 8 Gran, erregte unangenehme Leibschmerzen, die auch eintraten, als die Dosis um die Hälfte herabgesetzt wurde. Nach 18 Tagen mußte ich von dem Versuche abstehen. Einen Einfluß auf das Uebel habe ich nicht bemerkt."

11te Beobachtung. „Zwei Kinder mit Ausfluß aus den Ohren, welcher den äußern Gehörgang ziemlich excoriirt hatte, genasen nach kurzer Zeit, wie mir scheint in Folge des Mittels."

12te Beobachtung. „Louise B. litt an scrophulöser Auftreibung der Nase, Schnupfen, leichter Ophthalmie in Folge des-

selben, Wundsein der Nase. Ein fast achtwöchentlicher Gebrauch der Calcaria hatte den guten Erfolg, daß alle oben angegebenen Beschwerden gewichen sind und das Kind viel wohler, als vor der Cur aussieht. Auch sie gab einigemal Leibweh als Folge (?) der Pulver an."

Nach diesen Erfahrungen handelte es sich um die Entscheidung der wichtigen Frage, ob der phosphorsaure Kalk in der That nur den Zellenbildungsproceß fördere oder ob er eine specifische Einwirkung auf die scrophulöse Dyscrasie habe. — Die folgenden Beobachtungen werden dazu dienen, mit ziemlicher Bestimmtheit hierüber entscheiden zu können, und ich werde zunächst die Fälle von allgemeiner Scrophulosis, und sodann die von verschiedenartigen Geschwüren, welche ich mit Calcaria behandelte, zusammenstellen.

Die obige vierte Beobachtung hätte zunächst ebensowohl hier, als dort ihren Platz finden können. Es verheilten die scrophulösen Geschwüre, die Dyscrasie selbst aber wurde nicht getilgt. Ein Gleiches läßt sich von der 1. und 7. Beobachtung sagen.

13te Beobachtung. Priscilla W., 16 Jahre alt, trägt den scrophulösen Habitus und leidet an einer Conjunctivitis. Sie wurde vom 9. bis zum 27. Februar an derselben behandelt; eine fortgesetzte Anwendung kalter Ueberschläge, salinischer Purgantien und die Application eines Empl. cantharid. im Nacken führten Genesung herbei. — Am 3. Mai stellte sich jedoch das Mädchen wieder ein; es war seit einigen Tagen die Oberlippe stark geschwollen, eine Blepharadenitis mit beträchtlicher Anschwellung der Augenlieder entstanden, und auch die Conjunctivitis zeigte sich in geringem Grade wieder; zugleich fand der Ausbruch eines impetiginösen Ausschlags an einigen Stellen des Gesichts Statt. Es wurde jetzt Calcar. phosphor. gr. iv. Sacch. alb. gr. vj. M. f. p. Disp. tal. dos. xij S. dreimal täglich ein halbes Pulver

zu nehmen verordnet *). Am 4. Mai sah ich die Patientin wieder, aber sowohl Impetigo, als Lippengeschwulst, als Conjunctivitis und zum Theil auch die Blepharadenitis waren verschwunden. Die Calcaria wurde wiederholt, und es kann als ein Zeichen des andauernden Wohlbefindens betrachtet werden, daß sich Patientin bis jetzt nicht mehr hat sehen lassen.

14te Beobachtung. Mary G., 25 Jahre alt, Tochter eines tuberkulösen Vaters und einer gesunden Mutter, Schwester zweier an „Auszehrung" verstorbener Brüder, trägt durchaus nicht den scrophulösen Habitus, leidet aber seit längerer Zeit an Anschwellungen der Halsdrüsen und an drei bis vier Geschwüren am Halse, über dem Sternum, und über der Clavicula. Dabei wird über großes Schwächegefühl geklagt; anaemisches Colorit. — Ol. jec. Asell. ist seit kurzer Zeit ohne Erfolg gebraucht. Am 26. Juny wird Calcaria verordnet; am 4. July ist das Ulcus über dem Sternum bedeutend kleiner. Der abgesonderte Eiter, der früher, namentlich aus einem sehr tiefgreifenden Geschwüre, ein sehr wässeriger war, wird jetzt gut und rahmähnlich. — Am 25. July zeigen sich alle Geschwüre in der Heilung begriffen; eine beträchtliche Drüsengeschwulst in der Submaxillargegend bleibt zurück; nach längerer Abwesenheit der Patientin endlich, aber bei zeitweiliger Fortsetzung der Calcaria, sah ich sie am 10. October wieder; alle Geschwüre sind verheilt, die Geschwulst der Drüse ist aber noch vorhanden. An der einen Stelle derselben hat sich ein kleiner Abscess gebildet, aus dem einige Tropfen sehr guten Eiters entleert wurden. Die Schwäche hat sich verloren; Patientin sieht kräftiger und wohler aus.

15te Beobachtung. Eliza J., 18 Monate alt, hat kürzlich gleichzeitig 6—8 Zähne bekommen. Kurz darauf bricht eine

*) Diese Verordnung habe ich hier immer beibehalten und führe sie deshalb weiterhin nicht wieder an.

Tinea capitis aus; dieselbe ist ganz frisch, als das Kind zum Hospital gebracht wird. Es wird sogleich (am 27. Juni) Calcaria und daneben eine Natronsalbe für die Eruption am Kopfe verordnet. Am 4. Juli zeigt sich Vernarbung der Haut unter den Krusten; eine wiederholte Dosis der Calcar. führte vollkommene Genesung herbei.

16te Beobachtung. Mary Wh., 10 Jahr alt, trägt den scrophulösen Habitus, hat aber bis dahin weder an Hautausschlägen, noch Drüsenanschwellungen, noch Augenentzündungen gelitten; nur seit zwei Jahren ist nach und nach eine nicht unbedeutende Anschwellung des linken Carpus entstanden und dieselbe bis dahin mit Compressivverbänden ꝛc. behandelt. Am 29. März kam das Kind zuerst zum Hospital. Es wurde sogleich Calcaria, aber wegen der Bedenklichkeit des Leidens auch Ol. jecor. gegeben. Allein es trat durchaus keine Besserung ein, im Gegentheil, es bildeten sich Abscesse, und das Vorhandensein von Caries war keinem Zweifel unterworfen. — Im Laufe des Monats April wurden drei Abscesse geöffnet, es wurden warme Cataplasmen mit Infus. Chamomill. verordnet, die obigen Verordnungen noch festgesetzt, allein ganz ohne besondern Erfolg. Namentlich war es mir auffallend, daß, was sonst nie der Fall war, der Eiter nicht besser wurde; er blieb stets wässerig, saniös; nur erst als einige Ligaturen applicirt wurden, glaubte ich an den starken Granulationen der Incisionsstellen eine Einwirkung des Kalkes erkennen zu können. Das allgemeine Befinden blieb übrigens gut und erforderte keine besondere Behandlung; das Kind gewann sogar an Frische und Fülle. Am 26. Mai trat aber plötzlich eine Veränderung ein; bei mäßigem Fieber, bei den Erscheinungen eines acuten Magencatarrhs, schwoll die Hand beträchtlich auf, die Eitersecretion vermehrte sich, ein neuer Abseß bildete sich nach der Vola zu, während sich die frühern auf dem Dorsum befanden. Blutegel wurden vergebens applicirt; Purgantia, anfangs etwas

Calomel mit Jalappe, später Natr. sulphuric. wurden verordnet, im Ganzen blieb aber die Geschwulst ziemlich die frühere. Endlich wurde beständige Fortsetzung kalter Bähungen mit Seesalzwasser empfohlen, und diese hatten einen herrlichen Erfolg. Die Hand fiel immer mehr zusammen, die Eiterung hörte auf, die Ligaturen waren schon entfernt und ihre Wunden vernarbten, das subjective Befinden war ganz nach Wunsch. Es wurde jetzt wieder Ol. jecor. angewandt, daneben auch Calcar. mit Natr. phosphor. gegeben, und, ob in Folge davon, oder nicht, im Monat September konnte das Kind als genesen betrachtet werden. Die Hand war zwar im Carpus und Metacarpus anchylosirt, ein Fistelcanal noch nicht ganz geschlossen, jedoch der Krankheitsproceß selbst jedenfalls erloschen. — Ich entnehme dieser Beobachtung keine weitere Bemerkung, als daß die Calcar. nicht im Stande war, die scrophulöse Caries allein zu heilen.

17te Beobachtung. Daniel Sharp, 4½ Jahr alt, in sehr ärmlichen Verhältnissen lebend, kommt am 19. März zum Hospital. Seit 8 Wochen hat der Knabe die Fähigkeit zum Gehen verloren und ist sehr abgemagert. Er hat ein sehr anaemisches, erdfahles Colorit; die Hautvenen sind stark entwickelt und scheinen überall durch die Haut hindurch. Auf den Wangen unschriebene Röthe, sehr frequenter, kleiner Puls; Phantasieen im Schlaf; Klagen über Kopfschmerz. — Aus dem rechten Ohre wird eine reichliche Menge sehr unangenehm riechenden, jauchigen Eiters entleert; der Nervus facialis ist in seinem Verlaufe durch den canal. Fallop. comprimirt oder zerstört, denn die sämmtlichen von ihm versehenen Muskeln der rechten Gesichtshälfte sind gelähmt. Aus dem linken Ohr findet ebenfalls ein Ausfluß Statt, welcher aber nicht so beträchtlich ist. Der Digestionsapparat bietet die Erscheinungen beginnender Darmgeschwüre dar; 3—4 flüssige Sedes täglich, belegte Zunge, sehr starker Appetit, aufgetriebener Unterleib u. s. w. — Da augenblicklich die Calcar.

phosphorica nicht vorräthig war, sondern erst im Laboratorium präparirt werden mußte, so erhielt der Patient zunächst 3 Unzen Ol. jec. Asell. und Aq. Calcis, dreimal tägl. einen Eßl. voll zu nehmen; am 29. März wurde dann aber der Kalk allein, zu 2 Gran dreimal tägl. gereicht. Am 5. April zeigte sich die Veränderung, daß der Ausfluß aus dem linken Ohre ganz aufgehört hatte, der aus dem rechten aber geringer und der Qualität nach bedeutend besser wurde. Das Kind sollte der Aussage der Mutter nach heiterer sein; die Diarrhoe hatte aufgehört; das Fieber ließ nach. Am 12. April stand das Kind schon wieder auf seinen Füßen, das Colorit und der gesammte Habitus hatten sich bedeutend gebessert, das Kind war stärker geworden, aus dem rechten Ohre wurde noch ein dicker, guter, rahmähnlicher Eiter entleert. Am 30. April war die Besserung in gleichem Maaße fortgeschritten und am 24. Mai endlich war das Kind genesen. Es ging prächtig, war heiter und lebhaft, der Digestionsapparat war geregelt, der Ausfluß aus dem Ohre hatte aufgehört, an seiner Statt war aber eine wuchernde Excrescenz der Schleimhaut entstanden. Es gehört diese Beobachtung unstreitig zu den erfreulichsten, welche ich gemacht habe, und es leidet keinen Zweifel, daß die schönen Resultate dem Kalke zuzuschreiben sind.

18te Beobachtung. Caroline R., 9 Jahr alt, von scrophulösem Habitus, leidet seit 3 Wochen an einer bedeutenden Anschwellung der untern Epiphyse der Tibia des rechten Beines. Die Haut darüber ist sehr gespannt, die Venen stark entwickelt. Es wird lediglich Calcar. verordnet; der Erfolg ist nicht bedeutend; jedoch hatte sich binnen 4 Wochen die Geschwulst etwas verkleinert und die acuten Erscheinungen derselben, Röthe, Hitze u. s. w. waren gewichen. Die Beobachtung wurde durch die Abreise des Kindes unterbrochen.

Ich füge diesen Beobachtungen einige andere hinzu, welche ich einer brieflichen Mittheilung des Herrn Dr. Lorent in Bremen

verdanke; zwar sind dieselben keineswegs rein, indeß beweisen sie die Unzulänglichkeit der Calcar. zur gänzlichen Hebung einer scrophulösen Dyscrasie.

19te Beobachtung. „Wilhelm K., 2½ Jahr alt, wurde vor 15 Monaten im Kinderkrankenhause aufgenommen; er litt im hohen Grade an Atrophie und hatte einen großen rhachitischen Schädel, dessen große Fontanelle noch jetzt ¾ Zoll im Durchm. geöffnet ist. — Bei der bessern Pflege, dem Gebrauche von Ferr. carbon. cum Rheo et Magnes. carbon., Ol. jecor. Asell. und häufigen Bädern von Mutterlaugensalz besserte sich das Kind. Die letzten 3 Monate wurde neben den Bädern Calcar. mit Zucker, viermal täglich eine Messerspitze voll genommen, von dessen Gebrauche aber keine auffallende Wirkung bemerkt worden ist; das Allgemeinbefinden besserte sich eben so langsam, wie bei den übrigen Mitteln."

20ste Beobachtung. „Anna H. wurde im April 1847 im 7. Jahre mit bedeutender Spondylarthrocace und über den ganzen Körper verbreiteten scrophulösen Geschwüren im Kinderkrankenhause aufgenommen. Unter dem Gebrauche von einem Pulver aus Rheum cum Antimon. crud., Ferr. alcohol., Magnes. carbon. und Jod, Ol. jec. Asell., unter Anwendung von Cataplasmen und häufigen Bädern von Rehmer Mutterlaugensalz besserte sich die Patientin in dem Grade, daß im September der ganze Körper rein von Geschwüren und die Paedarthrocace an den Fingern und Fußzehen geheilt war, während die Spondylarthrocace, wenn gleich der Rücken spitzer geworden, in der Heilung begriffen schien. Große Abmagerung zeichnete die Kranke aus, derentwegen die schon seit September angewandte Calcaria fortgesetzt wurde. Jedoch äußerte bis jetzt der Gebrauch des Mittels keinen sichtlichen Erfolg, im Gegentheil ist die Abmagerung größer geworden und im Februar 1848 stellte sich Porrigo favosa capitis ein; bald darauf bildeten sich wieder an den frühern

Stellen unter der Haut liegende Knoten, die Neigung zum Aufbruch haben und tuberkulös scheinen und die verheilten Stellen der Finger und Fußzehen fangen wieder zu eitern an." (Es fehlen die Data der eingetretenen Geschwürsbildung und des Beginns des Gebrauchs der Calcaria.).

21ste Beobachtung. Gesine B. kam im Alter von 1½ Jahren mit Atrophia infantum und rhachitischer Verbiegung der Rippen im August 1847 in das Kinderkrankenhaus. Sie erhielt eine kräftige Diät, Mutterlaugenbäder und seit September Calcar. phosphor. — Die rhachitische Verbiegung und die damit zusammenhängende Kurzathmigkeit besserte sich, wenn gleich die Atrophie sich noch mehrte, bei regelmäßiger Verdauung und heiterer Stimmung.

22ste Beobachtung. Rebecca B., ein starkes, pastoses Kind von 4 Jahren, litt seit einem Jahre an Arthrocace des Ellenbogengelenkes, aus deren drei Abscessöffnungen eine profuse Eiterentleerung stattfand. Cataplasmata, Mutterlaugensalzbäder und Calcaria wurden angewandt. Die Eiterung und die Geschwulst des Gelenkes nahm im Verlaufe von 3 Monaten ab. Das Kind wurde magerer.

Ein ähnlicher Fall wurde von meinem Freunde Dr. Scuhr in Celle ohne Erfolg behandelt; dagegen sah wieder Herr Professor Oppolzer, der die Güte hatte den Kalk zu versuchen, in einem gleichen Falle ein sehr günstiges Resultat; die Fistelgänge verheilten und das Gelenk wurde dünner. Beide Bemerkungen verdanke ich kurzer mündlicher Mittheilung und kann deshalb die nähern Verhältnisse nicht angeben.

Weitere hierher gehörige Beobachtungen verdanke ich der Güte meines Freundes, Stadtphysikus Dr. Brandes in Hannover. Er wandte den phosphorsauren Kalk in drei Fällen an, und theilt mir darüber Folgendes mit: „In einem Falle gab ich das Mittel bei scrophulöser Drüsenanschwellung des Halses neben einer

langwieriger scrophulösen Ophthalmie, in einem zweiten bei Anschwellung der Halsdrüsen und chronisch gewordener Eiterung in der Parotideal- und Unterohrgegend. Die Individuen waren 4 und 19 Jahre alt und zeigten den scrophulösen Typus unverkennbar. Ich weiß nicht, ob dieß die rechten Fälle waren, wo man Erfolg erwarten konnte, aber daß ich nach 4—5wöchentlichem Gebrauche von täglich 3—10 Gr. Calcar. durchaus keinen Erfolg sah, kann ich versichern.“ Der dritte Fall betraf ein Kind mit scrophulöser Impetigo. Nach etwa 6wöchentlichem Gebrauch der Calcar., zuletzt in der erhöhten Dosis von dreimal täglich 10 Gran, war die Krankheit gehoben und vollständige Heilung trat ein. „Es wurde daneben nichts Anderes angewendet. Aber nach etwa zwei Monaten trat der Ausschlag wieder auf und besteht noch fort. Ich lasse jetzt wieder dasselbe Mittel nehmen. In andern Fällen habe ich gar keinen Erfolg gesehen, doch sind darunter keine Geschwüre, was ich von vorn herein bemerke.“

In Fällen der ganz gewöhnlichen Scrophulosis ohne jede Geschwürsbildung habe auch ich in der letztern Zeit mehrfach die Calcaria versucht; ich habe jedoch keinen entschiedenen Einfluß auf die Dyscrasie bemerkt, namentlich bestanden die Drüsenanschwellungen meistens fort.

Wenn nun auch nicht mit evidenter Gewißheit, so geht doch mit größter Wahrscheinlichkeit aus den vorstehenden Beobachtungen hervor, daß der phosphorsaure Kalk die scrophulöse Dyscrasie in toto nicht, daß er dagegen die in dieser Dyscrasie begründete mangelhafte Zellenbildung in den meisten Fällen hebt. Zweifelhaft muß ich es lassen, wie sich der Kalk zu den dyscrasischen Gelenkentzündungen und deren Ausgängen verhält; künftige Beobachtungen müssen darüber ebenso entscheiden, wie über einige scheinbare Widersprüche, welche sich in den mitgetheilten Fällen finden.

Ich wende mich hiernach zu Geschwüren nicht scrophulösen, sehr verschiedenen Ursprungs und deren Verhalten bei der Darreichung des Kalkes. — Sie haben mir in der That unträgliche Beweise für den zellenbildungsfördernden Einfluß desselben geliefert. Ich glaube es jedoch erwähnen zu müssen, daß man hiebei von richtigen Prämissen ausgehen muß. Wenn z. B. ein Patient an s. g. varicösen Geschwüren leidet, die Varices die Veranlassung zu Exsudaten ins Zellengewebe geben, und die Gegenwart dieser wieder die Geschwürsbildung veranlaßte, so darf man nicht hoffen, mit Darreichung der Calcar. die mechanischen Entstehungsursachen des Geschwürs und eventualiter das Geschwür selbst hinwegzuschaffen. — Wenn dagegen in syphilitischen, secundären, tiefgreifenden Geschwüren bei Anwendung des Kalkes der Zellenbildungsproceß auf eine eclatante Weise gehoben ist und die Geschwüre rasch zur Heilung gebracht sind, so liegt darin gewiß eine dringende Aufforderung den Kalk in gleichen Fällen mehrfach zu versuchen. Die Frage nach dem Wie? einer Heilindication kann nur dann richtig beantwortet werden, wenn man weiß, was man zu heilen hat und was man heilen will. Nur bei der jedesmaligen Vorlage dieser Frage, deren Antwort nach dem Stande unseres Wissens freilich oft traurig genug ausfällt, können die Heilindicationen einige Rationalität erlangen.

23ste Beobachtung. Am 27. März wurde im Hospital Peter W., 34 Jahr alt, mit den Erscheinungen eines sich bildenden Abscessus psoae der rechten Seite aufgenommen. Es bildete sich bald eine Geschwulst neben den Lumbarwirbeln und sobald Fluctuation in der Tiefe wahrgenommen werden konnte, wurde eine Incision gemacht und eine beträchtliche Menge Eiters entleert. Täglich wurde in der ersten Zeit eine gleiche Quantität ergossen; die Sonde drang nach unten gegen vier, nach oben gegen fünf Zoll weit unter die Bedeckungen ein. Es wurde eine Ligatur applicirt. Der Patient aber magerte ab, litt an sehr copiösen Schweißen, fieberte beständig, die Eiterung blieb sehr beträchtlich; kurz, es sah um die Prognose sehr mißlich aus. Am 17. Mai

wurde Calcaria phosphorica verordnet. Decoct. chinae wurde schon längere Zeit genommen. Der Erfolg war in der That ein sehr günstiger. — Der Eiterabfluß wurde nach und nach geringer, die Schweiße verschwanden, der Patient nahm an Kraft und Fleisch zu und bis zum 26. Juni waren die weiten Fistelgänge sämmtlich mit Granulationen ausgefüllt und vernarbt. Das Allgemeinbefinden war sehr zur Zufriedenheit. — Es war in diesem Falle namentlich interessant, schon kurze Zeit nach Anwendung des Kalkes die üppigste Granulationsbildung an den Oeffnungen des Ligatur-Canals zu beobachten, eine Beobachtung, welche ich später öfter gemacht habe. Ich konnte nicht umhin, die rasche Herstellung des Patienten zum größten Theile der Calcaria zuzuschreiben.

24ste—29ste Beobachtung. Ich fasse, um ermüdende Krankengeschichten zu vermeiden, diese Beobachtungen zusammen. Es betreffen dieselben Ulcerationen der verschiedensten Art. — Im ersten Falle hatte der Patient Johann K., 40 Jahr alt, in Folge einer Lymphangioitis einen beträchtlichen Abseß in der Gegend der Malleoli; dieselbe wurde geöffnet. Aber die Vertheilung trat nicht ein, es bildete sich vielmehr ein großes Geschwür, dessen Ränder nach und nach callös wurden. Es schien anfangs, am 13. Mai, als ob die Natur die Heilung allein zu Stande bringen würde, es bildeten sich scheinbar gute Granulationen auf dem Grunde des Geschwürs; allein es blieb bald bei diesen Granulationen stehen. Vergeblich wurden belebende Cataplasmen angewandt, Scarificationen der Ränder und des Bodens vorgenommen u. s. w.; am 24. Mai wurde Calcar. verordnet. Es trat die Granulationsbildung jetzt in der That sehr rasch ein und bis zum 9. Juni war das Geschwür durchaus verheilt. — Einem zweiten Patienten war vor etwa dreiviertel Jahren die vordere Hälfte des Fußes amputirt; es hatte sich jetzt an der Stelle der Vernarbung der Wunde bei etwas stärkerer Anschwellung als gewöhnlich eine Ex-

ulceration gebildet, und es ist nicht unwichtig zu bemerken, daß eben die Vernarbung dieser Stelle auch zum ersten Male eine bedeutende Zeit erforderte. Es wurden zunächst Cataplasmen mit Infus. Chamomill., Ugt. basilic. u. dgl. mehr angewandt; allein wenn es auch gelang, einige Granulationen hervorzurufen, es wollte sich durchaus keine eigentliche Narbe, kein Hautgewebe bilden, und die Granulationen selbst waren sehr schwammig und unkräftig. Es wurde darauf Calcar. versucht; zuerst am 10. Mai. Am 15. Mai habe ich bemerkt, daß seit den letzten 36 Stunden die Bildung eines weißlichen Narbengewebes an den Rändern des Geschwüres sichtbar wurde; am 19., daß die Granulationen sehr üppig seien und das Narbengewebe weiter vorschieße; am 30. Juni endlich, daß das Narbengewebe, nach mehrmals intermittirtem Gebrauche der Calcaria fest und vollendet sei, womit denn der Patient entlassen wurde. Die dritte und vierte Beobachtung betrafen zwei mit aus Furunkeln entstandenen Geschwüren behaftete Patienten, Heinrich Lahmann, 41 Jahr alt, und Sigm. Binkowsky, 21 Jahr alt. — Die anfangs mit Cataplasmen längere Zeit vergeblich behandelten, tiefgreifenden Geschwüre bekamen sehr bald nach Anwendung der Calcaria ein gutes Ansehen; die Granulationen waren üppig und von bester Beschaffenheit; ich konnte diese Wirkung ganz entschieden dem Kalk zuschreiben. Es ist eine schon mehrfach erwähnte, gewiß durchaus richtige Ansicht, daß der Furunkelbildung, wo sie in reichlichem Maaße stattfindet, eine bestimmte Blutkrase, die als furunculöse bezeichnet wird, zum Grunde liege. Es war mir nun sehr interessant in den bezeichneten Fällen das Verhalten dieser Krase bei der Darreichung des Kalkes zu beobachten. (Gleiche Beobachtungen wurden auch an 3—4 andern Subjecten gemacht). Es erlosch nämlich die Krase keineswegs — dieses wurde meistens erst durch die nachherige Anwendung des Solut. arsenical. Fowler. erreicht, allein sie schien sich in Etwas zu ändern. Es fan-

den noch pathologische Ablagerungen in das Zellgewebe verschiedener Körpertheile statt, allein selten kam es zu einer Vereiterung des Exsudates und Zerstörung des involvirten Zellgewebes, vielmehr wurde in einigen Fällen das Exsudat hart, vielleicht organisirt, und in andern Fällen wurde beim Einschnitt nur sehr wenig Eiter und etwas Serum entleert; nie aber beobachtete ich wieder einen normalen Furunkel und ein eventuelles, tiefgreifendes Geschwür; bildeten sich Ulcerationen, so waren sie mehr oberflächlich. — Es scheint nach allen vorliegenden Thatsachen ziemlich außer Zweifel, daß die der Furunkelbildung zu Grunde liegende Krase zu den auf qualitativen Abnormitäten der schwefel- und stickstoffhaltigen Bestandtheile des Blutes beruhenden Krasen gehöre. Wir sehen aus den vorliegenden Beobachtungen, daß das Quale der Krase durch Darreichung eines Stoffes geändert wird, welcher in der engsten Verbindung mit jenen Bestandtheilen steht, und es erhält dadurch meine weiter unten zu erwähnende Vermuthung, daß die qualitativen Alterationen der organischen Blutbestandtheile meistens die Folge von Abnormitäten im Quale oder Quantum der unorganischen, sicher aber immer davon begleitet seien, eine nicht unbedeutende Stütze. — Eine 5te Beobachtung betrifft einen Cholera-Reconvalescenten, Heinrich Mohr, 30 Jahr alt. Ein um die Unterschenkel geschlagener Sinapismus hatte an dem rechten Unterschenkel eine Ulceration herbeigeführt, und wiewohl Patient damit in der Hoffnung aus dem Hospital entlassen war, daß es in einigen Tagen verheilen werde, kehrte er nach 8 Tagen wieder zurück und beklagte sich, daß das Geschwür noch immer offen sei. Es war circa 3 Zoll lang und 1 Zoll breit. — Anfangs wurden Chamillenumschläge, Ungt. basilicum u. s. w. angewandt, allein es wollten sich keine gute Granulationen bilden, vielmehr behielt das Geschwür einen speckigen, unfruchtbaren Grund; nach etwa 12 Tagen versuchte ich den phosphorsauren Kalk. Schon am dritten Tage begannen Granulationen aus dem

schlechten Grunde hervorzuschießen; es wurde absichtlich äußerlich nichts mehr angewandt, als ein Heftpflasterstreifen zur Bedeckung; mit jedem Tage nahmen die Granulationen zu; es war eine Freude, dieses frische Leben zu sehen. Wenige Tage darauf begann dann auch die Narbenbildung; aber wunderbar genug, nicht, wie gewöhnlich, vom Rande des Geschwürs aus, sondern mitten auf dem Geschwürsgrunde; wo die Granulationen zuerst erschienen waren, überzogen sich dieselben auch zuerst mit dem Narbengewebe. Nach und nach entstanden auf diese Weise mehre Hautinseln, welche dann endlich in eins zusammenflossen. In etwa zehn Tagen war der Heilungsproceß vollendet.

An diese Beobachtungen reihe ich diejenigen, welche ich an secundären syphilitischen Geschwüren gemacht habe; dieselben lieferten mir sehr überraschende Resultate, und habe ich auch bis dahin nur zu drei Beobachtungen Gelegenheit gehabt, so glaube ich doch aus ihnen mit ziemlicher Gewißheit auf den entschiedenen Einfluß des Kalkes schließen zu können. Auch hier liegt natürlich jeder Gedanke an die Hebung der Dyscrasie selbst fern, allein es bieten die secundären und tertiären Formen der Syphilis manche Verhältnisse dar, die einerseits unzweifelhaft die Existenz einer (oben erwähnten) qualitativen Alteration der zu den Bildungsprocessen nothwendigen Blutbestandtheile darthun, andrerseits aber schon a priori an die interessanten Beziehungen des phosphorsauren Kalkes zum Zellenbildungsproceß und an sein Verhältniß zu jenen Blutbestandtheilen selbst denken lassen. Ich erinnere in Bezug auf diese beiden Punkte an die Ablagerung krankhaften Bildungsstoffes, die der Geschwürsbildung vorausgeht, überhaupt; bemerke, daß auch hier, wie eben bei allen Geschwürsbildungen kein normaler Zellenbildungsproceß stattfindet, weil das für ihn nothwendige Requisit, ein normales Material, fehlt; erinnere, daß wir neben der Geschwürsbildung allgemeine Abmagerung, also allgemein gehinderten Zellenbildungsproceß beobachten, und

glaube endlich, daß, wenn nicht schon die erwähnten Punkte, die besondern Beziehungen der Syphilis zum Knochensystem auf abnorme durch sie herbeigeführte Verhältnisse des phosphorsauren Kalkes wenigstens hinweisen. Ganz im Vorübergehn möchte ich auch hier der gewiß nicht ohne Grund aufgestellten Behauptung erwähnen, daß Kinder von syphilitischen Eltern meistens scrophulös seien; die in beiden Krankheitsprocessen beobachteten Affectionen des Knochensystems, Geschwürsbildungen, Ablagerungen pathologischer Stoffe in gleichen Systemen, der mangelhafte Ernährungsproceß überhaupt, u. s. w. geben in der That zu manchen interessanten, hierhergehörigen Betrachtungen Anlaß. Ich sage, ich erwähne dies im Vorübergehn, und in der That möchte ich nur einen gelegentlichen Gedanken ausgesprochen haben; unsere Kenntniß von dem Quale der verschiedenen, und selbst der allergewöhnlichsten Dyscrasieen ist ja noch zu mangelhaft, als daß wir uns irgend schon mit theoretisirenden Betrachtungen beschäftigen dürften; gehen wir daher sogleich zu den mitzutheilenden Factis über.

29ste Beobachtung. Elizabeth M., 24 Jahr alt, Näherin, kam am 6. August zum Hospital, selbst durchaus unwissend, daß ihr Leiden ein syphilitisches sei, und bis dahin ganz ohne ärztliche Behandlung geblieben. Sie war vor fünf Monaten inficirt und bei zunehmender Schwäche, bedeutender Abmagerung, vielfachen Digestionsbeschwerden und Verlust jeder Spur eines gesunden Colorits hatten sich nach und nach sehr bedeutende Geschwüre und zwar in beiden Ellenbogengelenken, an der Stirn, im Halse und auf dem Kopfe gebildet. Die Patientin war in der That so herabgekommen, daß sie nicht allein gehen konnte. Das Geschwür auf dem Kopfe war größer als ein Zweithalerstück und trichterförmig, die am Ellenbogen (in der Beugeseite) Thalergroß und das an der Stirn von der Größe eines Viergroschenstückes. Eine Menge kleiner Papeln und Hauttuberkeln, von deren beginnender Vereiterung an bis zur vollständigen Geschwürsbildung eine schöne Reihe pathologischer Bilder vorlag, waren vorhanden. — Der Dringlichkeit des Falles wegen wurde auf eine zunächst ausschließliche Anwendung des phosphorsauren Kalkes verzichtet; es wurde neben demselben in der gewöhnlichen Form täglich 3—4 mal 1/4 Gran Protojoduret. hydrarg. verordnet. 13. August. Die Geschwüre im Ellenbogen beginnen zu verheilen; es wird schon Narbengewebe sichtbar. Der Patientin eigene Worte „I feel much stronger" gaben einen Beweis für das gebesserte Allgemeinbefinden. 16. August: die Geschwüre in den Ellenbogengelenken und an der Stirn sind verheilt; die im Halse sind in der Verheilung begriffen; der Boden des großen Geschwürs auf dem Kopfe beginnt sich mehr und mehr zu heben. 23. August: Es hat sich über den frühern Geschwüren des Ellenbogengelenkes ein starkes Narbengewebe gebildet. 27. August: die Geschwüre im Halse sind verheilt. Das Geschwür auf dem Kopfe hat seine Trichterform verloren; an den Rändern beginnt die Hautbildung. — Die Fortsetzung der Behandlung wird für mehre Tage durch einen intercurrirenden Cholera-Anfall unterbrochen. 9. September: das Geschwür auf dem Kopfe hat noch die Größe eines Shilling. 24. September: das Geschwür auf dem Kopfe ist verheilt; in der Mitte befindet sich noch eine kleine trockene Kruste. Das Aussehen der Patientin hat sich im Verlaufe der Zeit total verändert; sie sieht wieder frisch und blühend aus, fühlt sich sehr kräftig; der Digestionsapparat bietet keine Krankheitserscheinungen dar. Am 15. October sah ich die Patientin wieder; das allgemeine Wohlbefinden bestand fort; unter der Kruste des großen Kopfgeschwürs hatte sich aber wieder etwas Eiter gebildet; wir glaubten dies als ein Zeichen der noch nicht gänzlich erloschenen Dyscrasie ansehen zu müssen, gaben noch einige Protojoduret-Pillen, und die Patientin ist jetzt als genesen entlassen. Ein sehr bemerkenswerther Umstand ist der, daß Patientin seit der Behandlung nicht

menstruirt war, während früher nie Unregelmäßigkeiten in dieser Hinsicht stattfanden. Die Menses haben zweimal cessirt. Ich habe dieselbe Beobachtung bei zwei andern Mädchen, welche die Calcaria bei sichtbarer Zunahme der Kräfte gebrauchten, gemacht, und möchte geneigt sein, diesen Umstand, falls er sich fernerhin bestätigt, durch einen gesteigerten Verbrauch von Bildungsmaterial in der Reproduction organisirten Gewebes zu erklären.

30ste und 31ste Beobachtung. In diesen beiden Fällen, welche zwei junge Männer betrafen, lagen Geschwüre vor, welche sich aus vereiterten Bubonen gebildet hatten. Das eine war von sehr bedeutendem Umfang, das andere etwa von der Größe eines Achtgroschenstückes. In dem erstern Falle war die frische Dyscrasie bald gehoben; die Bildung von Granulationen in dem Geschwüre, das Anlegen der unterminirten Ränder ging bei Anwendung des Kalkes sehr rasch vorwärts. In dem zweiten Falle war die Dyscrasie bedeutend hartnäckiger, das primäre Ulcus blieb lange Zeit indurirt. Nun war es sehr interessant bei Anwendung des Kalkes das Verhalten des aus einem Bubo entstandenen Geschwürs zu beobachten. Auf dem Grunde desselben, der immer mit Eiter bedeckt war und ziemlich tief lag, verhinderte, wie es schien, der syphilitische Eiter selbst die Bildung organisirten Gewebes, an den höher gelegenen Rändern aber, die bei der beständigen horizontalen Lage des Patienten der Eiter nicht berührte, bildete sich ein ganzer Kranz der üppigsten Granulationen, in einer Art, wie ich sie nie beobachtet habe. Endlich, nachdem der gänzliche Schwund der Induration des primären Geschwürs das Erlöschen der Dyscrasie anzeigte, entstanden auch auf dem Grunde des aus dem Bubo gebildeten Ulcus sehr schöne Wucherungen, und in sehr kurzer Zeit war es dann verheilt.

Ich wende mich hiernach zu einer andern Reihe von Beobachtungen und zwar solchen, welche an tuberculösen Individuen angestellt wurden. Es lag nach den bei scrophulösen Subjecten gemachten Erfahrungen nahe, den Kalk auch hier zu versuchen, denn mag man auch die Frage nach der vollkommenen Identität der Scrophulosis und Tuberculosis nicht geradezu bejahen wollen, so steht doch so viel fest, daß beide die nächste Verwandtschaft haben. Die Beobachtungen lassen im Allgemeinen die bei der Scrophulosis gemachten Erfahrungen bei der Tuberculose bestätigt finden.

Die beiden ersten hierhergehörigen Fälle lieferten so interessante Resultate, daß ich nicht unterlassen kann, ihrer umständlicher zu erwähnen.

32ste Beobachtung. Carl H., ein Arbeitsmann von 26 Jahren, war seit dem 17. Februar 1847 in meiner Behandlung. Der Patient litt als Knabe an Drüsengeschwülsten und trägt den tuberkulösen Habitus in ausgezeichneter Weise. Unter der rechten Clavicula findet sich ein gedämpfter Percussions-Ton und unbestimmtes Athmen. Bei der Inspiration erfolgt rechts fast gar keine, links eine unbedeutende Einziehung der Intercostalräume. Seit längerer Zeit trockener Husten; vor einigen Tagen Hämoptoe. Die Blässe der Hautdecken, das beständige Frösteln, die große Hinfälligkeit des Patienten, namentlich bei warmer Temperatur zu diesen Erscheinungen hinzugerechnet, rechtfertigten die Diagnose einer Lungentuberkulose. Untersuchung des Unterleibes: Leber namentlich nach rechts und unten vergrößert; ganz matter Percussionston, so daß auf eine die Vergrößerung bedingende Hyperämie geschlossen werden kann. Die Funktionen des Darmkanals durchaus krankhaft. Der Leib ist eingezogen; beständige Diarrhoeen quälen den Patienten und namentlich muß er stets sogleich nach dem Essen dem Drange zum Stuhlgang folgen. Die Entleerungen sind wässerig oder breiig, stets mit Schleim untermischt; oft zeigen sich Beimischungen von Blut und Eiter. Das Vorhandensein tuberkulöser Darmgeschwüre war hiernach nicht eben zweifelhaft. — Im Uebrigen keine besondere Erscheinungen außer einem eigenthümlichen Kältegefühl im Penis, welches ich kaum

anders als eine Nesterscheinung im Bereiche der sensitiven Nerven zu deuten wußte. Es wurde nun der Patient mit Oel-Emulsionen, Opium, Morphium, Plumb. acetic. u. s. w. regalirt; doch, wurden die Erscheinungen auch zeitweilig dadurch gemäßigt, einmal sogar ganz gehoben (durch Plumb. acetic. mit Opium), so traten sie dennoch immer wieder hervor, und nach Verlauf von vier Wochen fand sich Patient immer wieder bei mir ein. Ol. jec. Asell. konnte Patient durchaus nicht vertragen; die Diarrhoe wurde schlimmer darnach. Am 19. August wurde Calcar. phosphor. gr. iv. Sacchar. alb. $j\beta$ Dos. xij. zweimal täglich $^1/_2$ Pulver zu nehmen verordnet. Eine noch vorhandene Oel-Emulsion mit Opium und Aq. lauroceras. wurde anfangs dabei geleert; alle Arbeit wurde untersagt, die größte Ruhe anempfohlen. Eingedenk der Empfehlung von Stokes, das Opium bei Darmgeschwüren, deren Heilung man bezwecke, in Anwendung zu bringen, weil es die peristaltischen Bewegungen des Darmkanals verringere, ließ ich die einfache Tinct. Op. fortgebrauchen. Ich kann mich jedoch, da sie früher fruchtlos war, nicht dem Gedanken hingeben, daß sie zu der Verheilung der sicher vorhandenen und nicht unbedeutenden Geschwüre das Beste gethan habe, wenn ich auch damit für meine Meinung nicht mehr Glauben beanspruche, als für die eines jeden Andern. Schon am 30. August erschien Patient wieder bei mir, um mir die große Zufriedenheit mit seinem Befinden zu erkennen zu geben. Er meldete mir, daß das Poltern und Kollern im Leibe immer mehr aufhöre, daß er sich etwas kräftiger fühle, daß er seit langer Zeit in der letzten Nacht zum letzten Male wieder geschwitzt habe, daß seine Ausleerungen consistenter, und normale Faeces mit geringen Beigaben eines eitrig-schleimigen Fluidums entleert werden, daß endlich auch das Gefühl im Penis verschwunden sei. Im Verlaufe des Septembers wurde die obige Verordnung dreimal reiterirt, und am 29. d. M. konnte ich ihn aus der Behandlung entlassen. Der Patient erholte sich wirklich wunderbar; er nahm im ganzen Körperumfange zu, bekam einen gesunden Gesichtsausdruck und rothe Backen, und er selbst erzählte mir mit Freude, daß seine Freunde, Leute niedern Standes, sich über sein verändertes Aussehn wunderten. Seiner Arbeit konnte er ohne Beschwerde nachgehen. Im November (am 16.) kam jedoch der Patient wieder und bat sich seine Pulver aus, da die Diarrhoe wiedergekehrt sei. Es wurden ihm zunächst einige Dosen Plumb. acetic. mit Op. pur. gegeben und sodann die Calcaria; am 28. November und am 15. December ist die Gabe reiterirt, seitdem erschien aber Patient nicht wieder. Ich sah ihn oft zur Arbeit gehen, sein gutes Aussehen erhielt sich; — allein es ist meine Ueberzeugung, daß früher oder später Patient sicher von einem neuen localen Ausbruch der gewiß nicht getilgten, ursprünglichen Dyscrasie befallen werden wird. Ich selbst war nur noch vier Monate nach der Zeit der Wiederherstellung an dem Orte der Beobachtung, kann daher von dem weitern Verlaufe nichts berichten; sei dem aber, wie ihm wolle, jedenfalls ist der Fall für unsern Zweck sehr interessant. Ich zweifle nicht, daß die Darmgeschwüre verheilt waren; und wenn der Kalk einmal die Fähigkeit besitzt, den Zellenbildungsproceß zu heben, Geschwüre zur Verheilung zu bringen, so ist es in der That einerlei, ob das Geschwür in der äußern Haut oder in der Darmschleimhaut seinen Sitz hat. Die Lungentuberkulose, muß ich noch erwähnen, forderte in dem vorliegenden Falle durchaus nicht zu weiterm Einschreiten auf.

33ste Beobachtung. Ein junger Mann, Posamentirgehülfe R., 24 Jahre alt, der eben von einer linksseitigen Pleuritis genesen war; bekam plötzlich eine Pneumonie in beiden obern Lungenlappen. Diese Diagnose stand durchaus fest und da der Patient den scrophulösen Habitus trug, so zweifelte ich nicht, daß das Exsudat ein tuberkulöses sei. Meine Befürchtung traf ein; das begleitende Fieber nahm nach und nach den Charakter des

s. g. hektischen an, die Dämpfung des Percussionstons blieb, der Auswurf wurde eitrig, hatte jenen bekannten, süßlich widrigen Geruch, und betrug nach meiner eigenen Messung täglich durch vierzehn Tage hindurch 3/4 Quartier, zweimal sogar darüber. Dabei magerte Patient in drei Wochen bis zum Skelet ab und kein Mensch glaubte an sein Aufkommen; abwechselnde Diarrhoeen, beständige Schweiße und ganz unzweifelhafte Cavernenbildung, Oedem der untern Extremitäten u. s. w. ließen mich selbst jede Hoffnung verlieren. Versuchshalber leitete ich indessen folgende Behandlung ein: Gegen den beständigen und heftigen Hustenreiz und die Schlaflosigkeit: Morph. acetic.; Extr. hyoscyam. in Inf. digital. u. s. w.; zwei starke Fontanellen auf der Brust. Dabei Morgens, Mittags und Abends Bouillon und zwar in der Weise bereitet, wie Liebig in seiner bekannten, ausgezeichneten Schrift: Chemische Untersuchung über das Fleisch und seiner Zubereitung als Nahrungsmittel, Heidelberg 1847 vorschreibt. Zum Frühstück ein weichgekochtes Ei; Mittags außerdem ein wenig gekochtes Obst und leichte Fleischspeisen; dabei täglich zweimal 3—4 Gran Calcar. phosphor. Nach vierwöchentlicher Fortsetzung dieser Behandlung war der Patient ein anderer geworden; der Auswurf ließ nach, und sichtlich nahm Patient im ganzen Umfange zu, so diese Zunahme war nach sechs Wochen so bedeutend, daß R. das Bett verließ, die Schweiße und Fieber ganz aufhörten und Leute, die ihn auf seinem Krankenlager gesehen hatten, kaum glauben wollten, daß er derselbe sei. Jetzt war noch eine geringe Dämpfung oben unter beiden Clavikeln nachweisbar und das Respirationsgeräusch fast gar nicht hörbar; eine Einziehung der Intercostalräume erfolgte bei der Inspiration nicht. Seit acht Wochen kam Patient seiner Arbeit wieder nach, ging spazieren und sah frisch und wohl aus. Der etwas zu große Uebermuth zog ihm Ende Januar einmal einen Rückfall zu; er hatte sich in heftigem Ostwinde erkältet und in der Diät kein Maaß gehalten; nach dem

Schwunde eines abermaligen, mit eigenthümlichen membranösen Fetzen vermischten Auswurfes und bei Fortsetzung der Calcaria erholte er sich jedoch auch diesesmal und ich entließ ihn in sehr zufriedenstellendem Zustande aus der Behandlung.

So erfreulich dies erste Resultat der hierher gehörigen Versuche war, so habe ich mich doch eines gleichen nicht wieder zu erfreuen gehabt und bin deshalb entfernt, es irgend zu überschätzen. Die folgenden Beobachtungen haben mich vielmehr gelehrt, daß auch bei den Tuberculosen wohl eine Bethätigung des Zellenbildungsprocesses im Allgemeinen, eine Hebung der Kräfte durch die Calcaria bewirkt, daß aber die Dyskrasie selbst nur wenig davon influencirt wird.

Ich hoffe zu genügen, wenn ich mich bei den hierhergehörigen Fällen auf die Mittheilung der Resultate beschränke; es würde nutzlos und ermüdend sein, die sämmtlichen Krankengeschichten aufzuführen. — Im Ganzen liegen mir jetzt zwölf Beobachtungen vor, denen sich eine von Herr Dr. Lorent in Bremen hinzugesellt.

34ste—46ste Beobachtung. Die drei ersten Fälle betreffen Individuen, welche an sehr vorgeschrittener Tuberculose mit bedeutender Cavernenbildung litten. Koch und Kronenberg, resp. 55 und 37 Jahr alt, hatten Cavernen in beiden obern Lungenlappen; der Verlauf der Krankheit war ohne besondere Eigenthümlichkeiten und endete mit dem Tode. Die Calcaria hatte keinen Erfolg, als höchstens den, daß die Abmagerung den bedeutenden Verlusten durch Schweiße und Sputa durchaus nicht entsprach und die Kranken fast nie über großes Schwächegefühl klagten. Dasselbe war bei dem dritten Kranken, Leilig, der Fall, doch nahm hier in der letzten Zeit der Körperumfang bedeutend ab. Der Patient litt vor einem halben Jahre an Pleuritis sinistra, dieselbe hatte ein Exsudat zurückgelassen, es war der untere Lungenlappen mit ergriffen und neben bedeutenden Cavernen in den obern Lappen fand sich in der Leiche statt des untern

linken Lappens ein großer Eitersack, in den die offenen, größern Bronchialstämme hineinragten. Wunderbar genug, daß sich während des Lebens hier niemals bedeutende Blutungen einstellten; beim Auscultiren nahm man Flaschenklingen in ausgezeichneter Weise wahr. Was die Leichenbefunde selbst anbetrifft, so waren sie im Uebrigen die gewöhnlichen, und die Tuberkel oder Cavernen zeigten keine Eigenthümlichkeiten, die sich etwa auf die Anwendung des Kalkes hätten zurückführen lassen. — Ein vierter Patient, Trost, wurde lange Zeit an einem chronischen Geschwüre des rechten Unterschenkels behandelt, es verheilte dies bei der Darreichung des Kalkes; nach kurzer Zeit kehrte er mit beginnender Tuberculose in beiden Lungenlappen in's Hospital zurück, das Geschwür brach nach wenigen Tagen wieder auf. Der tuberkulöse Proceß schritt unaufhaltsam zur Cavernenbildung (links oben) fort; der Kalk hatte keinen Einfluß, außer vielleicht den, daß auch hier die Abmagerung bei bedeutenden Verlusten durch Sputa, Schweiß, Diarrhoe u. s. w. sehr unbedeutend war; der Patient behielt stets eine frische, gute Farbe; — den endlichen Ausgang der Krankheit erlebte ich nicht, da Pat. von seinem Wunsche, nach Deutschland zurückzukehren, nicht abzubringen war. — 5ter Fall. Der Patient, Null, leidet seit einigen Jahren an Tuberculose, ist 32 Jahr alt, in der letztern Zeit sehr abgemagert. Oben, links, hinten ist eine bedeutende Caverne sehr leicht nachweisbar; ein ausgezeichnet schönes Flaschenklingen wird wahrgenommen. Ich habe diesen Patienten fünf Monate lang beobachtet und wenn auch jeden Tag eine gleiche, bedeutende, gegen 1/4 Quartier betragende Menge eitriger Sputa entleert wurde, so veränderte sich bei dem Gebrauch der Calcaria das Allgemeinbefinden nur vortheilhaft; die Kräfte wurden in dem Maaße wieder hergestellt, daß Patient das Hospital zu verlassen wünschte. — Obgleich in diesem Falle gleichzeitig Ol. jec. Asell. gebraucht wurde, so muß ich doch das im Ganzen günstige Resultat auf Rechnung der Calcaria und der

stets sehr nahrhaften Diät schreiben, da sich früher unter dem alleinigen Gebrauch des Ol. jecor. der Zustand nur verschlimmert hatte. — Ganz ähnliche Resultate erhielt ich in dem 6ten und 7ten Falle. Die Patienten Stegmann und Rosenblum, resp. 42 und 49 Jahr alt, litten, der erste an noch im Beginne befindlicher, der letztere an schon zur Cavernenbildung vorgeschrittener Tuberculosis pulmonum. Bei jenem wurde der Proceß nicht aufgehalten, doch war die Kräfteabnahme sehr gering und dem Fortschreiten des Krankheitsprocesses nicht entsprechend; er verließ das Hospital, um zu seiner Familie zurückzukehren, ehe sich eine bestimmte Prognose stellen ließ. Bei dem letztern stellte sich ein sehr günstiges Resultat heraus; die anfangs copiösen Sputa, Schweiße und die allgemeine Hinfälligkeit schwanden, und Patient wurde, fähig seine Beschäftigung als Schneider fortzusetzen, aus dem Hospital entlassen. Sein Körperumfang nahm zu. Die Beobachtung ist übrigens nicht ganz rein, insofern auch Leberthran angewandt wurde. — Im 8ten und 9ten Falle lagen zwei frisch beginnende, beide Male zunächst unter dem Bilde eines leichten Typhus auftretende Tuberculosen vor. — Nach Beseitigung der ersten acuten Erscheinungen wurde alsbald Calcaria gegeben; die Kräfte hoben sich dabei sichtbar, der Körperumfang nahm zu, in keinem Falle kam es zur Cavernenbildung. Bei dem einen Patienten, Schley, 22 Jahr alt, der den tuberkulösen Habitus nur in sehr geringem Maaße trug, ist meiner Meinung nach bei später hinzugefügter Anwendung des Leberthrans die Dyscrasie total erloschen; er verließ das Hospital sehr kräftig und arbeitsfähig und noch vor Kurzem (ein halbes Jahr nach seiner Entlassung) habe ich ihn im besten Wohlbefinden angetroffen. Der zweite, Bernhard, 20 Jahr alt, litt ohne Zweifel auch an beginnenden Darmgeschwüren, magerte anfangs sehr ab, erholte sich dann aber sehr sichtbar, beim Schwinden der Diarrhoeen, des Hustens und der allgemeinen Mattigkeit. Die Zunahme des Kör-

perumfangs war in diesen Fällen in der That sehr auffallend und ungewöhnlich; ich halte es für unnöthig hinzuzufügen, daß der Kalk natürlich wohl nicht viel nützen würde, wenn seiner Darreichung nicht ein passendes diätetisches Verhalten zur Seite ginge. Vor Allem ist hier die Liebigsche Bouillon zu empfehlen. — Der 10te Fall betraf eine junge Dame, L. H., 23 Jahr alt, Tochter einer an Tuberculose verstorbenen Mutter. Sie wurde plötzlich von Husten, bedeutender Abmagerung und sehr heftiger Diarrhoe ergriffen; ein s. g. hektisches Fieber, Schweiße u. s. w. stellten sich ein, der Krankheitsproceß localisirte sich namentlich auf der Darmschleimhaut, die Prognose konnte in der That auf nichts Anders, als ein baldiges lethales Ende gestellt werden. — Es waren die Diarrhoeen schon länger vergeblich behandelt; sobald Patientin ins Hospital kam, wurde sogleich eine ähnliche Behandlung als bei dem Posamentiergehülfen R. eingeschlagen. Die heftigen Diarrhoeen erforderten anfangs zugleich Plumb. acetic. mit Opium, stellten sich, sobald dies weggelassen wurde, auch zu Anfang wieder ein, allein nach und nach hörten sie auf. In den Lungen machte die Tuberculose keine Fortschritte, dagegen erschien links neben den Lumbarwirbeln und dem Os sacrum, scheinbar von diesen ausgehend, eine harte Geschwulst, welche sich in der benachbarten Musculatur, namentlich unter dem Glutaeus, verbreitete. Anfangs schien diese in Eiterung übergehen zu wollen, man fühlte in der Tiefe eine leichte Fluctuation; auch kam nach einem gemachten Einstich und bei Anwendung von Cataplasmen eine geringe Quantität Eiter zum Vorschein, (ein Eiter, der, nach microscopischer Untersuchung bedeutende Mengen Fett enthielt) allein dennoch schwand die Geschwulst bis auf ein unbedeutendes Residuum. Nach Sistirung der Diarrhoeen ist auch Ol. jec. Asell. in Anwendung gebracht, der Kalk stets fortgesetzt und das ganze erfreuliche Resultat ist jetzt folgendes: die Kräfte im Allgemeinen sind gehoben, Patientin geht wieder spazieren, die Affection der Lungen ist sistirt, die Sedes sind regelmäßig und normal, der Leib nicht mehr aufgetrieben und tympanitisch, die Geschwulst am Dorsum ist sehr verringert, Patientin kann wieder auf der linken Seite liegen, die Incisions-Wunde ist stark vernarbt. Eigenthümlich waren in diesem Falle noch beständige, ganz oberflächliche, circumscripte silbergroschengroße Ulcerationen der sehr rissigen Zunge; auch diese sind fast total verschwunden, und die ganz reine Zunge bekommt mehr und mehr ein glattes Ansehen. Ich kann nicht unterlassen zu erwähnen, daß die früher stets regelmäßigen Menses während dieser Behandlung cessirten. — Eine sehr eigenthümliche Beobachtung bot der 11te Fall dar; er betraf einen 23jährigen Bäcker, Jung. — In Folge einer Gonorrhoe hatte der in seiner Jugend scrophulöse Patient eine Orchitis des linken Hoden bekommen; das von derselben gesetzte Exsudat tuberkulisirte; es bildeten sich Oeffnungen nach außen und lange Zeit andauernde Eiterungen; der Patient magerte bei dem einfachen Gebrauche von Cataplasmen ab. Plötzlich traten die Erscheinungen einer Miliartuberkulose auf, Patient wurde sehr hinfällig, fieberte sehr bedeutend, allein die acuten Erscheinungen schwanden wieder und in der folgenden Zeit blieben als Folgen nur oftmalige Oppression der Brust, ein geringer, oft für lange Zeit ganz aufhörender Husten und ein zäher, schleimiger, glasiger Auswurf zurück. Nun aber traten auffallende, von mir bis dahin nur zweimal beobachtete Vorgänge auf. Es bildeten sich nämlich im Verlaufe von etwa vier Monaten an den verschiedensten Stellen bedeutende Abscesse über und unter den Fascien. Ein sehr bedeutender an der Wade, über den ganzen Bauch des Gastrocnemius verbreitet, ein anderer an der Volarfläche des Vorderarms, ein dritter auf dem Dorsum des Fußes, ein vierter an dem Dorsum der rechten Hand, ein fünfter am Ligam. Poupart. Aus dem ersten wurde eine enorme Menge jauchigen Eiters entleert, der Ausfluß ließ dann nach und sehr, sehr langsam trat eine Ver-

heilung der großen Abseß-Höhle ein; drei derselben bestehen unverändert fort, ohne sich jetzt noch zu vergrößern, der fünfte an der Hand ist geöffnet und entleert fortwährend viel Eiter. Patient ist dabei schon seit langer Zeit ganz fieberfrei; der Digestions- und Respirations-Apparat bieten keine Krankheitserscheinungen mehr dar, der Urin macht aber fast beständig starke Sedimente von harnsaurem Ammonium, in der ersten Zeit von phosphorsaurer Ammoniak-Magnesia und phosphorsaurem Kalk. (Das harnsaure Ammonium-Sediment weicht jetzt auf Anwendung des Natron sesqui carbonicum). — Während der langwierigen Krankheit hat sich nun in Bezug auf den phosphorsauren Kalk Folgendes herausgestellt: der Patient ist trotz der beträchtlichen Eiterungen kräftiger geworden, sein Colorit hat sich verbessert, der früher wässerige und jauchige Eiter ist in ein pus bonum verwandelt, an den Oeffnungen des Handabscesses zeigen sich kräftige Luxuriationen, die Tuberculose, als solche, macht keine Fortschritte. Es wurde eine Zeit lang auch Ol. jecor. gegeben, allein, anfangs vertragen, rief es später jedesmal Diarrhoeen hervor und mußte verlassen werden. — Jod innerlich und äußerlich, China und Chinin, Mittelsalze, Eisen, Alles ist vergeblich versucht, und ich muß gestehen, daß wir jetzt ziemlich rathlos dastehen. Daß die Tuberculose als solche erloschen ist, ist kaum zweifelhaft; allein welcher Art die jetzt zum Grunde liegende Dyscrasie sein mag, ob an ihrer eigenthümlichen Gestaltung der lange Gebrauch des Kalkes Schuld trägt, darüber mag ich kaum eine Vermuthung wagen. Dennoch habe ich diese Beobachtung hier aufgeführt, da auch sie die Erfahrungen in Betreff der Calcaria bestätigt hat. — Schließlich theile ich den von Herrn Dr. Lorent beobachteten Fall mit und unterlasse die weitere Ausführung eines durchaus ähnlichen von mir bei einem 18jährigen Mädchen D. V. angestellten Versuches.

„Meta M., ein 17jähriges, noch nicht menstruirtes Mädchen, hatte mehre Male Hämoptoë gehabt und anhaltend einen kurzen Husten. Mitte Sommers bekam ich sie in Behandlung, fand sie sehr abgemagert, mit anhaltendem Husten, fieberhaft. Lungentuberkeln scheinen zum Grunde zu liegen. Nachdem der Husten durch die gewöhnlichen und demulcirenden Mittel gemäßigt war, wurde im Herbst Calcaria angewandt, viermal täglich eine Messerspitze voll. Nach längerem Gebrauche schien die Vegetation sich zu heben, Patient wurde bei regem Appetit kräftiger; das Hüsteln dauerte an und selten ist eine Hämoptoë eingetreten; das Allgemeinbefinden ist bedeutend gehoben." — Die ersten 11 dieser Fälle von Tuberculosis sind die, welche ich im Hospital genau beobachtet habe und in denen mir die Anwendung von Calcaria erfreuliche und nicht erfreuliche, jedenfalls aber zuverlässige Resultate lieferte. Mehrfache andere Beobachtungen sind bei den Out-patients unsers Hospitals angestellt (Patienten, welche zweimal wöchentlich gesehen werden und ihre Arzneien vom Hospitale erhalten); sie haben dieselben Resultate geliefert. Ich bin gewiß, daß der phosphorsaure Kalk allein durchaus nicht die tuberkulöse Dyscrasie hebt, aber ich glaube, daß wir in ihm eins der mächtigsten Adjuvantia zur Unterstützung der gegen dieselbe eingeschlagenen Behandlungen besitzen. Ob und inwiefern seine Anwendung, deren Erfolg sich in einer gehobenen Nutrition ausspricht, in dieser Hinsicht auf die tuberkulöse Dyscrasie einen irgend wie alterirenden Einfluß ausübt, vermag ich nicht zu entscheiden. Ich bemerke, daß das hier hauptsächlich gegen Tuberculosis angewandte Mittel das Ol. jec. Asell. ist, und daß wir bei seiner Anwendung in Verbindung mit der Calcaria oft recht erfreuliche Erfahrungen machen.

Ich komme zu der letzten Reihe zusammenhängender Beobachtungen, zu den meistens auf scrophulöser Dyscrasie beruhenden Atrophieen und damit verbundenen Diarrhoeen der Kinder, so wie zu den Diarrhoeen in der Dentitionsperiode. Die erzielten Resul-

tate sprechen selbst am besten für die Wichtigkeit des Kalkes in dieser Beziehung.

46ste Beobachtung. J. T., ein 1½jähriges Mädchen, litt an einem hohen Grade von Atrophie; die Haut hing in großen Falten und Runzeln an den Extremitäten, das Gesicht hatte jenen eigenthümlichen Ausdruck alter Leute, der Leib war mäßig stark entwickelt, ohne daß man jedoch Geschwülste durch die Bauchdecken hindurch fühlte; Diarrhoeen waren nicht vorhanden, dagegen Helminthen (Asc. lumbric.), von denen eine große Menge bei Gebrauch des Electuar. Anthelminth. entfernt wurde. Der Appetit war sehr stark, das Kind verlangte beständig zu essen. Am 20. August wurde die Calcaria zu zweimal täglich 2 Gran verordnet; das seit ganz kurzer Zeit gebrauchte Ol. jec. Asell., so wie Syrup. ferr. jodat. wurde ausgesetzt. — Wiewohl nun die Beobachtung zu Anfang September durch eine intercurrirende leichte Dysenterie, welche der Zeit epidemisch war, unterbrochen und auf etwa 8 Tage der Kalk ausgesetzt wurde, so war dennoch nach Verlauf von vier Wochen schon eine beträchtliche Besserung bemerkbar. Die Mutter sagte mir, das Kind nehme stark zu und habe angefangen zu laufen. In der That hatten sich die schlaffen Hautdecken etwas gehoben und die Runzeln derselben wurden durch die Neubildungen ausgeglichen. Noch andere vier Wochen wurde mit der Behandlung fortgefahren; dann hatte sich aber das Kind so erholt, daß eine weitere Behandlung unnöthig war. So lange ich das Kind sah, erfreute es sich der besten Gesundheit, sein Aussehen war frisch und blühend.

47ste Beobachtung. Mary R., 1½ Jahr alt, trägt den scrophulösen Habitus. Die Lippen sind entzündet, dick geschwollen, mit Krusten bedeckt; das Kind ist atrophisch, leidet, bei geringem Appetit, seit 3—4 Monaten an Diarrhoe; die täglichen 10—14 Stuhlgänge sind oft mit Helminthen vermengt. Zunächst wurde ein Elect. Anthelminth. gegeben und am 26.

Februar dann Calcaria verordnet. Am 1. März waren die Lippen dünner geworden, die Diarrhoe aber nur sehr wenig gebessert. Am 8. März hatte sich das Befinden bedeutend verändert; die Lippen waren fast ganz normal, der Appetit gehoben, täglich fanden 4—5 Stuhlgänge statt, das Kind ist, nach Aussage der Mutter, heiterer und lebendiger, als zuvor. Am 15. März war die Diarrhoe ganz geschwunden, die Lippen waren gesund, der ganze Gesichtsausdruck frischer, das Colorit besser, die Heiterkeit anhaltend. Mit einer neuen Dosis Calcaria wurde das Kind als genesen entlassen.

48ste Beobachtung. Thomas L., 16 Monat alt, leidet an Atrophie. Seit drei Monaten soll er nach Aussage der Mutter täglich abgenommen haben, ist sehr weinerlich, hat seit drei Monaten die Fähigkeit zum Gehen, welche er schon im zehnten Monat besaß, verloren. Der Digestionsapparat bietet catarrhalische Erscheinungen dar, wiewohl die Sedes nicht relarirt sind. Das Colorit ist blaß, anaemisch; die Muskulatur schlaff und welk. Am 29. Juni wurde zum ersten Male die Calcaria verordnet, ohne daß vorher noch nebenher etwas Andres gebraucht wurde. — Nach vierwöchentlicher Fortsetzung derselben war eine ganz bedeutende Besserung sichtbar. Das Kind ging wieder, die Farbe war besser, die Blässe hatte sich verloren und die Muskulatur wurde kräftiger. Das Kind wurde heiterer. Es ist als genesen am 24. Juli entlassen und bis dahin (Ende October) nicht wieder gekommen.

49ste Beobachtung. William C., 14 Monat alt, ist in der Zahnungsperiode, leidet an Diarrhoe und wird mager. Am 3. September wird sogleich Calcaria gegeben. Am 6. September hat die Diarrhoe schon etwas nachgelassen, die Geschwulst des Zahnfleisches beginnt zu schwinden, das Allgemeinbefinden ist besser. Am 17. September ist die Diarrhoe geschwunden, die Heiterkeit ist auffallend, die Mutter sagt „he gets much strong-

er.“ Am 4. October zeigte sich die Besserung anhaltend, die Diarrhoe ist nicht wiedergekehrt, das Kind wird als genesen entlassen.

50ste Beobachtung. George G., 3 Jahr alt, trägt den scrophulösen Habitus und leidet namentlich an einer Atrophia extremitatum infer., so wie an einer chronischen Entzündung im rechten Kniegelenk. Es dauert diese seit etwa drei Wochen; das Kind geht stets mit auswärts gedrehtem, rechten Fuße. Bei Anwendung der geeigneten localen Mittel schwand diese Entzündung bald, und es wurde dann am 3. April Ol. jec. Asell. verordnet. Allein der gesammte Zustand besserte sich fast gar nicht dabei; der scrophulöse Habitus, das gedunsene Gesicht blieben dieselben, die Schwäche der untern Extremitäten blieb so groß, daß Patient kaum darauf gehen konnte, „er wackelt,“ wie die Mutter sich ausdrückte. Am 12. Juni wurde deshalb das Ol. jec. ausgesetzt und Calcaria gegeben; im Ganzen wurden drei Drachmen und zwölf Gran verbraucht. Am 10. September erschien das Kind zuletzt. Es konnte fest und sicher auftreten, das Gesammtbefinden war bedeutend gebessert, die Abmagerung der Extremitäten war geschwunden, kurz es blieb nichts mehr zu wünschen übrig. Patient wurde als genesen entlassen.

Ich könnte die Zahl dieser Beobachtungen leicht vermehren, da mir noch mehre vorliegen; ich glaube jedoch die vorstehenden werden meinem Zwecke genügen und ich enthebe mich gern der weitern Aufführung von Krankengeschichten. — Einige sehr interessante Fälle von Atrophia, so wie auch von gleichzeitiger Rhachitis und Arthrocacen sind noch in der Behandlung und ermuthigen ebenfalls nur zu weitern Versuchen. Nicht weniger werden noch verschiedene Geschwüre u. s. w. mit gutem Erfolge mit dem Kalk behandelt.

Noch einer Beobachtung erwähne ich hier, welche an Individuen gemacht wurde, die Fontanellen trugen oder denen solche

applicirt waren. Wenn diese nämlich eine Zeit lang die Calcaria genommen hatten, so bildeten sich an den Rändern der Fontanelle stets sehr beträchtliche Granulationen so, daß sie oft die eingelegten Erbsen ganz überragten und verdeckten. Da ich dies in so bedeutendem Maaße nicht bei andern Fontanellen gesehen habe, so trage ich kaum ein Bedenken, den regern Zellenbildungsproceß auch hier von der Einwirkung der Calcaria herzuleiten.

Ich schließe damit die Reihe der so kurz als möglich zusammengefaßten Beobachtungen ab, und glaube mich durch sie zu der Aufstellung der folgenden Hauptresultate berechtigt:

1) Gleich wie im Pflanzenreiche und niedern Thierreiche die Abhängigkeit des Zellenbildungsprocesses von der Gegenwart des phosphorsauren Kalkes erwiesen ist, so ist der phosphorsaure Kalk auch im menschlichen Organismus ein nothwendiges Requisit für die Bildung der Zelle.
2) Der phosphorsaure Kalk, als Heilmittel angewandt, hat sich zunächst bei oberflächlichen scrophulösen Geschwüren als den Zellenbildungsproceß fördernd und die Vernarbung der Geschwüre herbeiführend erwiesen.
3) Eine gleiche Einwirkung habe ich bei andern chronischen Ulcerationen, insonderheit auch bei tiefgreifenden syphilitischen Geschwüren beobachtet; daß aber eine hier zum Grunde liegende Dyscrasie des Blutes als solche durch den Kalk nicht gehoben werden kann, bedarf kaum der Erwähnung.
4) Der phosphorsaure Kalk hat sich bei meistens auf scrophulöser Grundlage beruhenden Atrophieen und den sie oft begleitenden Diarrhoeen

der Kinder, namentlich auch in der Dentitionsperiode, sehr wirksam gezeigt.

5) Es ist nicht unwahrscheinlich, daß der phosphorsaure Kalk in einer besondern Beziehung zur scrophulösen Dyscrasie steht, und es möchte in dieser Beziehung sehr der Beachtung werth sein, daß sämmtliche Exsudate (mit sehr seltenen Ausnahmen) bei scrophulösen Subjecten amorph sind; falsch scheint jedoch jedenfalls die Vermuthung, daß der Kalk, als Arzneimittel dargereicht, die Dyscrasie selbst hebe. Ein etwaiges, durch chemische Analysen näher zu entwickelndes pathologisches Verhältniß des phosphorsauren Kalkes würde eben nichts als ein constituirender Theil jener Dyscrasie sein; diesen kann der Kalk heben, einen andern nicht.

In einer Kritik der Dietl'schen Gehirnkrankheiten in Schmidt's Jahrbüchern sagt Dr. Scuhr sehr treffend, daß eine jede therapeutische Entdeckung ein empirisches Stadium durchlaufen müsse, um überhaupt gemacht oder als wahr bewiesen zu werden. Wenn ich nun meinerseits auch einen großen Theil dieses Stadiums in Bezug auf den phosphorsauren Kalk durchgemacht zu haben glaube, wenn ich mir von „feststehenden Resultaten" zu sprechen erlaubt habe, so lege ich dennoch mit einer gewissen Schüchternheit diese kleine Beigabe zur Therapie der Oeffentlichkeit vor, und werde mich erst dann ganz frei von Täuschungen wähnen, wenn von gewichtigern Seiten Bestätigungen meiner Erfahrungen erfolgen. Sollten sich diese, wie ich hoffe, herausstellen, so haben wir in einem den Zellenbildungsproceß im kranken Organismus fördernden Heilmittel einen nicht unwichtigen Beitrag, der in jeder Hinsicht auf Rationalität Anspruch machen darf. Keinem aufrichtigen Arzte ist die große Dürftigkeit unserer

therapeutischen Zustände verborgen; in der Mangelhaftigkeit physiologischer Anhaltspunkte liegt der Grund zu derselben. Das Streben, solche Anhaltspunkte zu gewinnen, der Therapie eine mehr rationelle Basis zu geben, ist deshalb in jeder Weise gerechtfertigt. Feststehende, rein empirische Thatsachen behalten immer ihren Werth und wenn man dem rationellen Therapeuten so oft entgegenhält, er könne ja doch nicht einmal erklären, weshalb das Chinin das Wechselfieber heile, so kann dies allgemein ausgestellte testimonium paupertatis eben so wenig dazu dienen, ihn von seinem rationellen Streben abzubringen, als die Achtung vor jenen Thatsachen zu verkleinern. Das aber wird wohl Keiner bezweifeln, daß in dem Verhältniß von Chinin und Intermittens, sei es, welches es wolle, doch eine ratio obwalten muß; es sei denn, daß sich Jemand zu dem nur zu unphysiologischen Geständnisse verstände, zwischen Heilmittel und Organismus finden überhaupt keine rationellen Verhältnisse Statt. Möchte eine richtige Abschätzung der Resultate himmelweit verschiedener Therapieen diejenigen, welche ein solches Geständniß haben laut werden lassen, erinnern, daß sie ein durchlöchertes Kleid mit durchlöcherten Fetzen flicken, und daß ein Hungernder von der Ueberzeugung, daß er Mangel leidet, nicht satt wird.

Kehren wir nach dieser kurzen Abschweifung zu einer weitern Betrachtung den phosphorsauren Kalk betreffender Verhältnisse zurück.

Es ist bekannt, daß die Humoralpathologie, welche zunächst die Betrachtung des Blutes zu ihrem Gegenstande hat, vornehmlich zwei verschiedene Classen von Alterationen desselben anerkennt. Es sind dies die quantitativen und die qualitativen Alterationen einzelner Blutbestandtheile. Die quantitativen erstrecken sich sowohl auf die organischen Verbindungen, als auf die unorganischen; in beiden Beziehungen sind sie von gleicher Wichtigkeit, und die Alterationen der unorganischen Bestandtheile dürfen um so weniger vernachlässigt werden, als die Abhängigkeit des

physiologischen Zustandes der organischen Verbindungen von dem normalen Verhältnisse der letztern keinem Zweifel unterworfen ist. Der Ueberschuß von Alkalien im Blute muß eben sowohl einen Krankheitsproceß erzeugen, als der Ueberschuß des Fibrins; der Mangel an diesen oder jenen Salzen eben so bestimmte Störungen in der Stoffmetamorphose herbeiführen, als der Mangel an Eisen, welche Ursachen er auch immer haben mag, in der Chlorose seinen Ausdruck findet.

Was jedoch die qualitativen Alterationen, namentlich der organischen Verbindungen, anbetrifft, diese Alterationen, deren Existenz erst in neuerer Zeit durch die pathologische Anatomie über allen Zweifel erhoben ist, so muß ich noch einmal auf den schon erwähnten Punkt zurückkommen, daß nämlich das Albumin und Fibrin unseres Blutes nicht nur Verbindungen von CHON, sondern daß sie stets zugleich mit Schwefel und phosphorsaurem Kalke verbunden sind. So lange, bis das Gegentheil bewiesen ist, ist durchaus die Vermuthung gerechtfertigt, daß jene qualitativen Alterationen auf quantitativen Mißverhältnissen ihrer integrirenden Bestandtheile beruhen, und es muß demnach die Möglichkeit zugegeben werden, daß auch diese oder jene Alteration des Fibrins oder Albumins in einem quantitativen Mißverhältnisse des phosphorsauren Kalkes begründet sein könne. Ehe nicht durch die chemische Untersuchung dieser Vermuthung ihr Recht geschehen ist, sind in der That jene pathologisch-anatomischen Befunde nur von geringen praktischen Consequenzen und lassen der Erklärung einen weiten Spielraum; daß aber Erklärungen, wie die Zimmermann's: es beruhen jene qualitativen Alterationen auf einer abnormen Lagerung der Molecüle, gänzlich hypothetisch sind und unsere Einsicht auch nicht um eine Haarbreite fördern, bedarf wohl kaum der Erwähnung. — Ich habe diesen Punkt hervorheben zu müssen geglaubt, weil er zeigt, wie schon die einfache Anschauung der physiologischen Verhältnisse des Blutes zu bestimmten Fragen

über pathologische Verhältnisse, wie aller Bestandtheile desselben, so auch des phosphorsauren Kalkes, hinführt. Bis dahin sind aber dergleichen Fragen und noch mehr die Versuche ihrer Beantwortung gänzlich hintangestellt gewesen.

Ein zweiter Punkt, auf welchen ich aufmerksam machen möchte, ist das Vorkommen des phosphorsauren Kalkes in Urinsedimenten. Das Vorkommen selbst, namentlich in Kinderkrankheiten, welche mit Zersetzungsprocessen einhergehen, bei bedeutenden Abmagerungen, langwierigen Eiterungen u. s. w. ist wohl allgemein bekannt. Nicht bekannt ist aber meines Wissens die Beobachtung, welche ich über die Erzeugung eines phosphorsauren Kalksedimentes bei kranken Individuen in Folge der innern Darreichung des kohlensauren Natron gemacht. Ich bin mit diesen Untersuchungen noch nicht ganz zu Ende gekommen, so viel aber habe ich als feststehend erkannt, daß man namentlich in Fällen von Rheumatismen, und insonderheit von solchen, die sich in anaemischen Individuen nicht selten finden, durch das Natron alsbald (etwa nach Verlauf von 48 Stunden) Sedimente von phosphorsaurem und kohlensaurem Kalk meistens nebst phosphorsaurer Ammoniak-Magnesia hervorrufen kann. — Wir haben das kohlensaure Natron hier nicht selten und mit gutem Erfolge bei Rheumatismus acutus angewandt. In einigen Fällen, die in sehr robusten, jungen Leuten vorkamen, gelang es mir oft nur sehr langsam, das Sediment zu erzeugen, ja, was mir kaum erklärlich ist, es kam ein Fall vor, in welchem trotz lang fortgesetzter Anwendung des Natr. sesquicarbonic. der Urin nicht einmal alkalisch wurde; in den meisten Fällen dagegen und namentlich solchen, welche, wie erwähnt, in anaemischen (chlorotischen) Individuen auftraten, gelang es fast ohne Ausnahme. Es wurden in 24 Stunden 6 Drachmen Natron gegeben, nach 48 Stunden oder auch nach 3—4 Tagen erschien dann sicher Kalk im Urin, und sobald das Natron wieder ausgesetzt wurde, schwand das Se-

diment. Oft war das letztere mit Fett und Eiterkörperchen vermengt, diese letztern Beimengungen sind jedoch inconstant und zum Theil von individuellen Verhältnissen abhängig. Auch bei andern Krankheiten, als Scrophulosis, Catarrhus ventriculi ꝛc. ꝛc., habe ich dasselbe Resultat erreicht; es ist mir durchaus nicht unwahrscheinlich, daß es sich in den allermeisten Fällen, wenn nicht immer, erreichen läßt. Die Diagnose des Sedimentes war chemisch und mikroskopisch festgestellt, namentlich in letzterer Beziehung jedesmal durch Zusatz von Schwefelsäure und eventuelle Bildung von Gypskrystallen dargethan, eine Methode, welche mir die sicherste und schnellste für die Diagnose des Kalksedimentes zu sein scheint. — Ueber das Wie? und Warum? dieses interessanten Verhältnisses können wir natürlich nur sehr oberflächliche Vermuthungen hegen; ich beschränke mich deshalb auch auf die bloße Mittheilung des Factums, hoffend, daß eine Zeit, in der wir genauer als jetzt mit den Verhältnissen der bildenden und rückbildenden Stoff-Metamorphose des Körpers bekannt sind, auch diese Vermuthungen zur Wahrscheinlichkeit und Gewißheit bringen wird. — Die bekannte Einwirkung einer hinreichenden Quantität Alkali auf das Fibrin und Albumin, die dadurch einerseits herbeigeführte Auflösung dieser Stoffe selbst, so wie die eventualiter gehinderte bildende und direct oder indirect beschleunigte rückbildende Metamorphose andrerseits möchten jedenfalls die aufmerksamste Berücksichtigung verdienen. Wir wissen, daß das phosphorsaure Kalksediment bei erschöpfenden, mit Abmagerungen einherschreitenden Krankheitsprocessen natürlich vorkommt, wir können es künstlich durch kohlensaures Natron erzeugen und nach Allem, was wir über die Wirkung des letztern erschließen können, tritt es hindernd der Entwickelung eines bildungsfähigen Materiales und damit der Bildung organisirten Gewebes selbst entgegen. In beiden Fällen sehen wir also gleiche Resultate im Organismus — i. e. eine Abnahme der Neubildungsprocesse, der Zellenbildung —

und in beiden Fällen sehen wir ebenfalls den phosphorsauren Kalk als unbrauchbares Material ausgeschieden in dem Urin. — Kann es einen bessern und interessantern Beleg für die ausgesprochene Bedeutung des phosphorsauren Kalkes geben? Können wir freudiger, als durch Auffindung ähnlicher Facta, angeregt werden zu der weitern Verfolgung der so unendlich interessanten Verhältnisse der Stoffmetamorphose? Wir besitzen, das leidet keinen Zweifel, in dem kohlensauren Natron und ihm verwandten Stoffen (den meisten Alkalien) Mittel, die progressive Metamorphose zu hindern oder die regressive zu fördern; der phosphorsaure Kalk bildet ihren Gegensatz, denn er fördert jene, während er diese beeinträchtigt.

Was die Betheiligung des phosphorsauren Kalkes beim Zellenbildungsprocesse selbst anbetrifft, so hat Schmidt darüber, wie oben erwähnt, gewiß eine sehr richtige Vermuthung ausgesprochen. Er meint, um es kurz zu wiederholen, daß eine mit einer gewissen Portion des phosphorsauren Kalkes gesättigte Albuminlösung vorzugsweise die Fähigkeit besitze, sich in Berührung mit heterogenen Körpern zu relativ festen Membranen um diese herum zu verdichten, d. h. die Wand primärer Zellen zu bilden. Insofern es nicht an Beispielen fehlt, daß sich in Flüssigkeiten, welche das nothwendige Material enthalten, auch außerhalb des Körpers und ohne Zusammenhang mit organisirten Körpertheilen Zellen gebildet haben, Beispiele, unter denen namentlich die von Helbert über die Eiterbildung in der einer durch ein Canthariden-Pflaster erzeugten Blase entnommenen klaren Flüssigkeit, sehr schlagend zu sein scheinen*); so ist, wie ich meine, auch die Möglichkeit vorhanden, daß sich in einem künstlich gemischten Fluidum, welches die nothwendigen Requisite zur Zellenbildung, so weit wir sie

*) Vgl. Vogel, Pathologische Anatomie des menschlichen Körpers. Leipzig 1845. p. 84. — Das Werkchen von Helbert ist betitelt: De exanthematibus arte factis fragmenta. Gottingae 1844. Ich habe bei eigenen Untersuchungen die Resultate Helbert's einmal bestätigt gefunden

kennen, enthält, außerhalb des Körpers in einer geeigneten Temperatur Zellen bilden. — Versuche, welche die Lösung solcher Fragen betreffen, tragen allerdings der wunderbaren Zartheit aller im Organismus selbst vor sich gehenden Bildungsprocesse gegenüber einen hohen Grad von Rohheit an sich, indeß einerseits kennen wir von der Physik des Organismus noch viel zu wenig, als daß wir nicht auch auf diesem Wege Aufklärungen zu erhalten suchen dürften, und andrerseits lassen uns einzelne Blicke in die großartige Einfachheit organischer Bildungsvorgänge nicht ganz grund- und hoffnungslos dem Experimente entgegentreten. Es ist zur Zeit schon höchst wahrscheinlich, daß der Act der Bildung der Zelle selbst ein rein physikalischer, der Beihülfe weiterer organischer Thätigkeiten nicht bedürftig sei, und es dürfte dieser Gegenstand gewiß weiterer und gründlichster Untersuchungen werth sein.

Ich möchte die besondere Aufmerksamkeit auf Versuche dieser Art, welche ich angestellt habe und welche mir sehr überraschende Resultate geliefert haben, hinlenken; ich glaube in der That entschieden, daß es mir gelungen ist, wirkliche Zellen, die von den Exsudatzellen und auch von größern Eiterkörperchen durchaus nicht zu unterscheiden waren, künstlich darzustellen. — Ich hatte zu diesem Zwecke einem Theile eines Hühnereiweißes etwas phosphorsauren Kalk und reines Fett zugesetzt: diese Mischung wurde dann in einem Sandbade einer andauernden Temperatur von 32 Grad R. ausgesetzt und ich beobachtete nun mikroskopisch die in der Flüssigkeit vor sich gehenden Veränderungen. — Meine ersten Versuche gaben mir schon, bei 4—6stündiger Fortsetzung des Versuches die Ueberzeugung, daß ein Niederschlag von Albuminat mit phosphorsaurem Kalk um Fetttröpfchen stattfinde, in spätern, 10—12 Stunden lang fortgesetzten Beobachtungen gelang es mir denn die in Fig. 1. und 2. dargestellten Bildungen zu beobachten. — Eine Reihe neben einander liegender Gebilde dieser Art ließen mich namentlich über die

Art und Weise der Bildung Aufschluß bekommen. Ich sah ein reines Fetttröpfchen, ein anderes, dessen lichte Mitte schon getübt, matt war und schließlich in Folge weitern Niederschlages das Bild einer Zelle, welches in Nichts von einer Exsudatzelle zu unterscheiden war. Wenn ich diese Zellen mit Schwefelsäure behandelte, so erhielt ich durchaus dieselben Resultate, welche ich bei Behandlung von Exsudat- und Eiterzellen, erlangte; es bildeten sich nämlich auf der Oberfläche der Zelle sehr kleine, dunkele Körnchen, Bildungen, welche ich nach den weiter unten anzuführenden Beobachtungen für schwefelsaure Kalk-Crystallchen gehalten habe (cf. Fig. 2). Sehr interessant waren mir hier auch namentlich einige Zellen, welche, selbst etwas größer, eine zweite kleinere einschlossen, ein Bild, welches am meisten der Zeichnung des primitiven Eies von Wagner im Prodromus histor. generat. gleichkam. Es schien sich hier um eine gebildete kleinere Zelle von Neuem Fett und um dieses eine Kalkalbuminat-Verbindung abgelagert zu haben. — Bei Behandlung mit Essigsäure klärten sich die Zellen etwas auf und ließen Körnchen, ähnlich wie bei der Behandlung mit Schwefelsäure hervortreten. — Da nun im Hühnereiweiß schon eo ipso phosphorsaurer Kalk enthalten ist, so frug ich, ob überhaupt der Zusatz von phosphorsaurem Kalk erforderlich sei. Ich setzte deshalb eine einfache Verbindung von Eiweiß mit etwas Fett der erhöhten Temperatur im Sandbade aus, und auch hier fand ich nach etwa 6—8 Stunden sehr vereinzelte kleine Zellen; es waren deren aber nur außerordentlich wenig, ein Resultat, welches sich im Voraus erwarten ließ. Ich bemerke noch, daß ich diese Versuche mehrfach wiederholt habe und in den letzten drei Malen stets zu gleichen Resultaten gekommen bin; ich wünsche weiter nichts, als daß sie ihrer hohen Wichtigkeit gemäß von umsichtigen, und mit ähnlichen Versuchen vertrauten Händen der Prüfung unterzogen werden.

Hier berichte ich endlich auch über meine unlängst mit fri-

schen Blastemen, Eiter, Muskelfasern u. s. w. angestellten Versuche, welche in so schlagender Weise mit Allem, was ich über den phosphorsauren Kalk mitgetheilt habe, übereinstimmten, daß sie mir in der That das höchste Interesse gewährten. Es wird allgemein bekannt sein, daß während der phosphorsaure Kalk nie in crystallinischer Form vorkommt, der schwefelsaure Kalk in den schönsten Nadeln, welcher sich bei weiterer Ausbildung unter dem Mikroskope als rhomboidale Tafeln darstellen, in einer durchaus unverkennbaren Form crystallisirt. Hat man unter dem Mikroskope eine einigermaßen beträchtliche Menge phosphorsauren oder kohlensauren Kalkes, so bilden sich beim Zusatz eines Tropfens concentrirter Schwefelsäure sofort eine Unmasse von Nadeln und rhomboidalen länglichen Tafeln, die zum Theil sehr rasch zu sehr schönen, runden, strahlenförmigen Drusen zusammenschießen, zum Theil als einfache Kreuze (unter rechten oder spitzen Winkeln), oder auch ganz isolirt erscheinen (cf. Fig. 3). — Hat man hingegen nur eine sehr unbedeutende Menge der obigen Kalkverbindungen und setzt nun Schwefelsäure hinzu, so geht langsam vor den Augen des Beobachters der ganze Entwickelungsproceß des Gypscrystalles vor sich, und diesen ganz genau zu kennen, ist durchaus erforderlich, um die folgenden Beobachtungen anzustellen. — Bei dem von mir gebrauchten Prichard'schen Mikroskope habe ich diesen Proceß bei etwa 350facher Vergrößerung folgendermaßen beobachtet. Zunächst bilden sich kleine, schwarze Molecüle oder Körnchen mit deutlicher Contour, welche durchaus noch keine crystallinische Form besitzen, vielmehr eine Urbildungsform für sehr viele Crystalle, wenn nicht, wie die Zelle für organisirte Bildungen, für alle, darstellen; ich habe sie auf gleiche Weise in der Bildung des Harnsäurecrystalls, der phosphorsauren Ammoniak-Magnesia u. s. w. wahrgenommen (cf. Fig. 4 b.); nach und nach erkennt man eine Winkel- oder Kreuzform unter diesen Punkten und Körnchen, es zeigt sich hie und da ein klein-

stes, von zwei sich kreuzenden Gypsnadeln gebildetes, im Mikroskope etwa $^1/_8$—$^1/_{12}$‴ großes Kreuz, oder eine von den allmählig an Umfang gewinnenden Körnchen gebildete kleinste Rosette, und bei außerordentlich kleinen Mengen von Kalk bleibt bei diesen Formen oft schon die Bildung stehen (cf. Fig. 4 c. und Fig. 5 a.). — Ist jedoch die Quantität des Kalkes bedeutender, so geht es jetzt weiter; man sieht deutliche Kreuze mit einfacher oder doppelter Contour, einzelne Nadeln oder kleine aus ihnen zusammengesetzte Drusen, Formen, die sich endlich zu länglichen, rhomboidalen Täfelchen ausbilden. Diese erhalten dann schließlich, je nach der Menge des Kalkes, eine mehr ausgebildete Gestalt, bis die oben erwähnten Formen erscheinen. — Ich habe gefunden, daß man auf diese Weise die kleinste Menge Kalkes nachzuweisen im Stande ist, und die Form der mit Schwefelsäure gebildeten Crystalle erlaubt approximative Schlüsse auf jene Menge.

Anmerkung. In Bezug auf die hier einmal näher erwähnte Bildungsgeschichte des Gypscrystalls bemerke ich noch, daß ich bei der Bildung der spitzen Winkel der ausgebildeten, rhomboidalen Tafeln denselben, oder wenigstens einen ganz ähnlichen Vorgang beobachtet habe, wie ihn Schmidt in seinem „Entwurf einer allgemeinen Untersuchungsmethode der Säfte und Excrete des thier. Organismus" pag. 49 von der Winkelbildung der phosphorsauren Ammoniak-Magnesia Crystalle beschrieben hat. — Die Gypsnadel ist anfangs ein reines Oblongum; bei ganz langsamer Ausbildung habe ich dann an den schmalen Enden eine lichte, hellere Umgebung, grade wie in der Schmidt'schen Abbildung Fig V. b. und c., wahrgenommen, und aus dieser bildet sich der spitze Winkel hervor; es wird gleichsam ein rechtwinklichtes Dreieck dem Oblongum an beiden Seiten angesetzt; einige Crystalle waren ganz von einem lichten Mantel umgeben. Cf. Fig. 6.

Ich versuchte nun, ob man durch Behandlung mit Schwefelsäure Gypscrystalle aus frischen Exsudaten u. s. w. erhalten und dadurch den Gehalt an phosphorsaurem Kalk in denselben nachweisen könnte. Diese meine Vermuthung ist vollkommen be-

stätigt, und überall, wo wir Zellenbildung finden, läßt sich auf diese Weise der Kalk darstellen; ja ich fand weiter, daß je reger der Zellenbildungsproceß Statt fand, desto schönere und größere Crystalle erschienen; es mußte mithin hier die Quantität des Kalkes eine größere sein. (Sollte Jemand einwenden, es hätte kohlensaurer Kalk sein können, so bemerke ich, daß auch nie nur ein Bläschen in dem Objecte entwickelt wurde). Zunächst untersuchte ich das frisch aus einer, durch ein Empl. cantharid. erzeugte Serum. Frisch, so wie es genommen war, untersucht, fand ich nur die allerkleinsten, nur dem damit vertrauten Auge erkennbaren, oben beschriebenen Molecüle; eine Crystallform war daran nicht zu entdecken; sobald ich aber das Serum etwas abdampfte, und nun einen Tropfen der concentrirten Flüssigkeit mit Schwefelsäure in Berührung brachte, entstanden bald früher, bald später (oft erst nach einigen Stunden) deutliche, schöne Crystalle von schwefelsaurem Kalk; Kreuze, Nadeln und sehr feine Drusen. — Von demselben Individuum untersuchte ich 24 Stunden später ein Stückchen des frisch auf dem entblößten Corium gebildeten Exsudates, das sich zum Theil schon als ein Häutchen darstellte, und aus Zellen verschiedener Größe und Eiterkörperchen bestand, und hierin fand ich nun nach etwa zweistündiger Einwirkung der S̄ eine Menge schöner Crystalle, Crystalle, die sich insonderheit auf den Zellen selbst accumulirt vorfanden. — Ich bemerke beiläufig, daß ich unter diesen jüngst gebildeten Zellen Gebilde vorfand, die ganz täuschend denen ähnelten, welche ich bei dem künstlichen Zellenbildungsversuche wahrnahm. Es schienen mir Fetttröpfchen zu sein, um welche sich ein Niederschlag von Albuminat in Verbindung mit Kalk (was durch die Darstellung der Gypscrystalle erwiesen) gebildet hatte. — Diese Versuche habe ich mehrfach wiederholt und habe jedesmal die Bildung von Crystallen in gleicher Weise wahrgenommen. Ich untersuchte jetzt wieder Eiterkörperchen und Eiterserum. Der erste war einer sehr gut granulirenden Wunde am Arm eines von einer Zellengewebsentzündung reconvalescirenden Patienten entnommen. Hier nahm ich nur sehr, sehr kleine Crystallformen, fast nur die obigen Molecule wahr, jedoch wurden auch einzelne kleine Nadeln und Kreuze beobachtet. Aus einem andern jauchigen sehr dünnflüssigen Eiter eines an einem Abscesse leidenden scrophulösen Subjectes, so wie aus dem dünnflüssigen, schlechten Eiter und einer kleinen schwammigen Granulation aus dem Boden des Ulcus eines mit Krebs des untern Augenlides behafteten Patienten gelang es mir nicht, nur ein Crystall darzustellen; ich nahm nur einzelne wenige der kleinsten Molecüle wahr.

Unter den vielen interessanten Bemerkungen, welche Liebig in seinem Aufsatze über die Constitution des Harns der Menschen und der fleischfressenden Thiere in Liebig's und Wöhler's Annalen 1844. Band 50. über die Verhältnisse der phosphorsauren Salze im Organismus mittheilt, finden wir pag. 178 auch die durch Verbrennungsprocesse bewiesene, daß „bei dem Uebergange des Blutes in Muskelfaser offenbar der größte Theil der phosphorsauren Alkalien in die Circulation zurücktrete, während eine gewisse Menge phosphorsauren Kalkes in chemischer Verbindung in den Organen bleibe." Diese Menge nun, so unendlich gering sie auch in einem Stückchen frischer Muskelfaser sein mag, welches auf das Objectglas gebracht der Art ausgebreitet ist, daß die Querstreifen der Fasern deutlich erkennbar sind, diese Menge, sage ich, läßt sich ebenfalls auf die obige Weise nachweisen. Ich habe ein kleines Stückchen frischen Muskels 24 Stunden lang mit S̄ befeuchtet stehen lassen und nach diesem Zeitraume finde ich, in einem unter das Mikroskop gebrachten Minimum desselben an einer Stelle deutliche Gypscrystalle, Nadeln, zarte Drusen und die zu Kreuzen vereinigten Nadeln, Crystalle, deren viele selbst eine deutliche doppelte Contour zeigten. Es sind besonders schön an

dieser Art von Präparaten die verschiedenen Entwickelungsstufen der Crystalle nachweisbar. —

Daß phosphorsaurer Kalk im Fleische, ja in allen organisirten Bildungen vorkomme, wußten wir lange; er ist aber meines Wissens auf diese Weise und in diesen kleinsten Quantitäten bisher nicht nachgewiesen. Die vorstehenden Beobachtungen müssen uns einen neuen Beweis für seine weite Verbreitung und seine daraus zu erschließende eminente Bedeutung für Bildungsprocesse organisirter Stoffe liefern. Die Beobachtungen über seine allergeringste Quantität in bildungsunfähigen Materialien erlauben einen weitern Schluß auf diese Bedeutung, und es möchten fortgesetzte Untersuchungen dieser Art die letzten positiven Beweise für die oben aufgestellten Behauptungen beizubringen im Stande sein.

Ueber eine weitere medicinische Anwendung des phosphorsauren Kalkes habe ich selbst nur eine sehr geringe Erfahrung; dennoch möchte ich in den folgenden Punkten einige Andeutungen für weitere Beobachtungen geben.

Zunächst halte ich die Calcaria für ein wichtiges Mittel zur Reconvalescenz von schweren, allgemeine Abmagerung herbeiführenden Krankheiten. Ich habe sie angewandt bei Typhus-Reconvalescenten und sah vorhandenen Decubitus außerordentlich rasch verheilen, die allgemeine Hinfälligkeit bald gehoben, die Zunahme des Körperumfanges auffallender als gewöhnlich. — Ich habe sie ferner gebraucht bei Patienten, welche an bedeutenden Zellgewebsvereiterungen gelitten hatten, und sah die Neubildung des verloren gegangenen Bindegewebes außerordentlich rasch entwickelt. Ich werde nicht anstehen, sie in allen ähnlichen Fällen zu versuchen; in Krankheiten, während deren Verlauf wir phosphorsauren Kalk in den Urinsedimenten beobachten, ergiebt sich, wie ich meine, die Indication zu Versuchen von selbst; bei den erwähnten und sehr bekannten Zellgewebsvereiterungen habe ich bei der Anwendung von Ligaturen und der sofortigen Darreichung von

Calcaria phosphor. nebst China und Säuren die herrlichsten Resultate beobachtet. —

Eine zweite Indication scheint dem ersten Blicke nach bei der Rhachitis vorzuliegen und, so viel ich weiß, ist der Kalk hier namentlich in früherer Zeit oft gegeben. Wenn man aber bedenkt, daß bei der Rhachitis mit ihrer Osteomalacie u. s. w. jedenfalls ein Ueberschuß von Säure vorhanden ist, sei er nun primär oder secundär entstanden, so, sollte ich meinen, wäre es gerathener, bei ihr den kohlensauren, als den mit der stärkeren Phosphorsäure verbundenen Kalk anzuwenden. Wenigstens werde ich in allen künftigen Fällen den Anfang damit machen und den Erfolg abwarten. Ich habe bis jetzt leider wenig Gelegenheit zur Beobachtung der Rhachitis gehabt; in einem letzthin vorgekommenen, sehr eclatanten Falle habe ich jedoch, Versuchs halber, lediglich den phosphorsauren Kalk angewandt. Der dreijährige Knabe gebraucht denselben jetzt seit fünf Wochen, ist dabei im Ganzen kräftiger geworden und geht wieder (was er längere Zeit nicht konnte); allein die Anschwellungen fast der sämmtlichen Epiphysen der Knochen sind noch nicht geschwunden.

Ueber die Caries der Knochen und ihr Verhältniß zum Kalk habe ich bis dahin auch keine genügenden Erfahrungen gemacht; es liegen aber Untersuchungen vor, die zur Anstellung von Versuchen auffordern. In den Annalen der Chemie und Pharmacie von Liebig und Wöhler, Band 57, Heft 3, 1846, befindet sich ein Artikel von v. Bibra: über die Zersetzung, welche die Knochen durch Caries erleiden. Das Hauptresultat dieser schönen Untersuchungen ist das, daß die Zersetzung, Auflösung und Entfernung der Knochensubstanz von einem bedeutenden Schwunde des phosphorsauren Kalkes begleitet sei. Während sich z. B. in dem obern Theile einer wegen Caries im Fußgelenke amputirten Tibia 51,02% Kalkerde fanden, ward in einem Knochenstück aus der spongiösen Substanz der cariösen Gelenkfläche nur 25,83 und in

einem Knochenstücke der am stärksten ergriffenen Theile nur 12,90% Kalkerde gefunden. Der in der Norm nur 1—3% betragende Fettgehalt der Knochen steigt dabei sehr bedeutend, in dem angegebenen Falle z. B. von 1,21 auf 29,41. — Mehrfache andere Untersuchungen führten zu ähnlichen Resultaten. Auch Valentin's Analysen cariöser Knochen *) ergeben dasselbe. Während die Tibia eines gesunden Mannes von 38 Jahren 61,98% Asche und in dieser 84% phosphorsauren Kalk lieferte, wurde in einer cariösen Tibia eines gleichalten Mannes 44,12% Asche und darin 77,93% phosphorsaurer Kalk gefunden. Auch Davis Untersuchungen erweichter Knochen (Lond. med. Gaz. 1847. Febr.) gaben ähnliche Befunde. Er fand in 100 Theilen der pathologischen Knochen Kalkphosphat 16,40%; Kalkcarbonat und Magnesiaphosphat 4,88%; Fett 20,35%; Gelatina 58,37%; in gesunden Knochen hingegen: erdige Materien 66,70% und thierische Stoffe 33,30%. Das Schwinden des Kalkes in cariösen Knochen leidet nach diesen mehrfachen Untersuchungen keinen Zweifel, und es ist gewiß von hohem Interesse, die Frage, ob und welchen Einfluß eine innere Darreichung des phosphorsauren Kalkes hier ausübe, durch Versuche zu entscheiden.

Eine fernere Anwendung möchte ohne Zweifel bei Fracturen zu versuchen sein. Es sind uns schon einige hiezu ermuthigende Mittheilungen von Fletcher in der Lancet 1846. Vol. II. № 15. gemacht. Gestützt auf die Beobachtung, daß ein Huhn mit einem gebrochenen Fuße Eier ohne Schaalen lege, stellte Fletcher an einem Canarienvogel, der ein gebrochenes Bein hatte, Versuche an. Zur Begünstigung der Ablagerung der Knochenmaterie bekam der Vogel eine ziemliche Menge Kalk. Er konnte schon am 6. Tage sein zerbrochenes Bein wieder gebrauchen, und da nach dem gewöhnlichen Gange der Dinge drei Wochen vergehen, bevor eine

*) Repertorium für Anat. und Physiol. 1838. p. 306.

so günstige Veränderung eintritt, so war der Erfolg gewiß überraschend. Im Hinblick auf diese Thatsachen wendete Fletcher den Kalk bei Fracturen an. Er reichte von gebranntem Knochen oder präparirtem Kalk täglich dreimal einen Scrupel in Form einer Mixtur und verdünntes Kalkwasser zum Getränk. In zwölf Fällen wurde die herrliche Wirkung dieser Behandlung constatirt, sieben dieser Fälle werden mitgetheilt. Die Kranken konnten meistens ihre gebrochenen Glieder in 8—10 Tagen bewegen und rotiren, und in 14 Tagen wieder gebrauchen. Schlechte Constitution, Kacherien, wird hinzugefügt, verhindern auch hier die Heilung. — Ich habe in Folge dieser Mittheilungen den Kalk in vier Fällen, welche mir bis dahin zu Gebote standen, angewandt. Zunächst bei einem 8jährigen Knaben, welcher an einer Fract. femoris litt. Er erhielt täglich Calcar. phosphoric. ʒβ und konnte in der dritten Woche den Schenkel rotiren und bewegen; gehen konnte er in der vierten Woche. — In dem 2. Falle lag eine complicirte Fractur des Unterschenkels bei einem 28jährigen Arbeitsmann, Thomas Lee, vor. Die Eiterung, welche sich sehr weit unter und zwischen den Muskeln verbreitete, dauerte lange Zeit und es trat während derselben keine Verbindung der Knochenenden ein. Sobald sie indeß aufhörte, wurde sehr rasch ein Exsudat von den letztern geliefert und die vollständige Callusbildung war etwa in 14 Tagen vollendet. Patient ging dann leicht und fest einher und wurde entlassen; es war aber der Callus selbst ein sehr luxuriöser geworden. — Der dritte Fall betraf ebenfalls eine Fractura cruris, bei einem 38jährigen Arbeitsmann. Es konnte derselbe am 18. Tage nach der Erkrankung das Bein schon aufheben, in der vierten Woche ging er umher. Der vierte Versuch endlich wurde bei einer Fractura ulnae an einem 30jährigen Manne gemacht; die vollständige Verheilung und Genesung wurde binnen drei Wochen erzielt. Aber auch in diesen beiden letzten Fällen wurde trotz der geringern angewandten

Quantität Kalkes eine bedeutende Callusbildung beobachtet und eben von dieser Seite her möchten weitere Versuche große Vorsicht erfordern. Eine Gabe von täglich 4 Gran phosphorsauren Kalkes wird, glaube ich, durchaus hinreichend sein, die Callusbildung zu beschleunigen, und mit ihr werde ich künftige Versuche anstellen.

Weitere für diesen Gegenstand wichtige Beobachtungen verdanken wir v. Bibra in seinen „chemischen Untersuchungen über die Knochen und Zähne des Menschen und der Wirbelthiere ꝛc. 1844. Schweinfurt." — Er fand bei eierlegenden Thieren nach der Entziehung von Kalksalzen Verschwinden der Kalkdeposita auf der Eierschaale und endlich Cessation des Eilegens, so wie Chofsat bei Tauben nach längerer Entziehung von Kalksalzen Knochenbrüchigkeit und Diarrhoe (NB!) bemerkte. Bei der Untersuchung der Knochen des Huhns, welches der Kalkinanition unterworfen war, ergab sich eine Abnahme der anorganischen Substanz um etwa 10%, eine Abnahme der phosphorsauren Kalkerde um 6—10%.

Damit will ich hier die Reihe der Fragen, welche ihre Antwort durch die Praxis erhalten können, begränzen. — Daß sich der phosphorsaure Kalk mit der Zeit mehr und mehr als ein höchst wichtiges Heilmittel herausstellen wird, daran hege ich nicht den geringsten Zweifel; möchte aber auch die Chemie bald im Stande sein, uns über die physiologische und pathologische Bedeutung des Kalkes weiter aufzuklären! Ohne ihre kräftige Beihülfe werden wir noch lange Zeit tüchtiger Fortschritte in der Therapie entbehren müssen.

Haben sich erst unsere Kenntnisse hinsichtlich der chemischen Verhältnisse und Veränderungen der integrirenden Blutbestandtheile in pathologischen Processen erweitert, so muß sich damit eine Menge neuer und höchst wichtiger Aufgaben für die Therapie stellen; es wird namentlich nur auf diese Weise der wichtigste Theil derselben,

die Diätetik, zu einer so durchaus nothwendigen Rationalität gelangen können. Für sie ist dann aber noch ein Zweites nöthig, und das ist die Kenntniß der Zusammensetzung der Nahrungsmittel selbst. — Was der Dünger dem Felde, ist die Nahrung dem Menschen; fehlt dort ein nothwendiger Bestandtheil, so fehlt das Produkt; es erzeugt sich kein Amylon, kein Gummi, kein Zucker bei dem Mangel von Alkalien, bei dem Fehlen der phosphorsauren Salze bildet sich kein Albumin und Fibrin aus. Nicht anders ist es im thierischen Organismus; der Mangel irgend eines Bestandtheiles der Nahrung muß einen pathologischen Zustand herbeiführen, und mit der entsprechenden Hinwegnahme oder mit der vermehrten Zufuhr dieser oder jener Nahrungsmittel müssen wir eben diese Zustände heilen können.

Ich möchte zum Beleg der interessanten Abhängigkeitsverhältnisse zwischen Nahrung und Bildung der einzelnen Bestandtheile des Organismus ein Beispiel anführen, welches uns die Untersuchungen Boussignault's über die Entwickelung der mineralischen Bestandtheile in dem Knochensysteme des Schweines liefern*). So lange die jungen Schweine gemischte Nahrung erhielten, nahm die Entwickelung des Knochensystemes einen raschen Fortgang; die Quantität des phosphorsauren Kalkes erhöhte sich binnen 8 Monaten von 84,1 auf 91,3%. Sobald dann aber die gemischte Nahrung mit einer Nahrung von Wasser und Kartoffeln vertauscht wurde, hörte die rasche Weiterentwickelung auf, und in $3\frac{1}{2}$ Monaten nahm der Gehalt an phosphorsaurem Kalk nur von 91,3 auf 92,4% zu. Höchst interessant ist dabei das Resultat, daß dennoch mehr phosphorsaure Kalkerde in den Organismus aufgenommen, als durch die Kartoffeln (der Analyse zufolge) eingeführt wurde. Es konnte dieser Ueberschuß durchaus

*) Liebig und Wöhler's Annalen der Chemie u. Pharmacie Bd. 59 Heft 3.

keine andere Quelle, als das Wasser haben. Die Untersuchung dieses letztern ergab nun auch, daß es sehr kalkhaltig sei (in 900 Litres fanden sich 179 Grammes), und wir gelangen demnach zu dem Resultate, daß auch die anorganischen Bestandtheile des Wassers zur Bildung von Bestandtheilen des thierischen Organismus verwendet werden. — Aehnliche Betrachtungen erheben die Kenntniß von der Zusammensetzung der Nahrungsmittel, ohne welche wir z. B. nicht erklären können, warum denn Schwarzbrod und Kartoffeln scrophulösen Kindern schädlich sind, zur höchsten Wichtigkeit, und wenn es unzweifelhaft ist, daß die Bestandtheile der Nahrungsmittel, je nach dem Boden, welcher sie erzeugte, verschieden sind, so ist die Kenntniß dieser Bodenverschiedenheit selbst in der That von um so höherer Wichtigkeit, als sie uns im Allgemeinen das bietet, was die Analyse der einzelnen Pflanzen einer bestimmten Gegend im Speciellen darlegt. Diese Kenntniß ist von durchaus gleich hoher Bedeutung, als die der atmosphärischen Verhältnisse verschiedener Länder, Gegenden und Districte, als die der durch langjährige Gewohnheiten stabil gewordenen Sitten und Gebräuche verschiedener Nationen — Kenntnisse, die sämmtlich erforderlich sind bei der Erforschung des Wesens einzelner Krankheitsprocesse, so wie bei der Aufsuchung neuer, auf festen Grundlagen stehender therapeutischer Principien. Wir müssen auf diese Verhältnisse recurriren, um uns die endemischen Verschiedenheiten in der Entwickelung des menschlichen Organismus zu erklären; wir müssen sie vor Allem in Betracht ziehen bei der Nachfrage nach der Ursache endemischer Krankheiten. Das hier und dort endemische Auftreten von Scropheln ist nicht auf andere Weise zu erklären, und wenn wir in Frankreich die Tuberculosen so häufig, in England dagegen sehr selten mit Fettleber combinirt sehen, so müssen auch diese Verhältnisse ihre endliche Grundlage in der Verschiedenheit des blutbildenden Materiales, so wie in der Verschiedenartigkeit diätetischer Gebräuche und atmosphärischer Ver-

hältnisse finden. Die Beschaffenheit der Oberfläche des bebauten Bodens ist aber keineswegs eine ganz stabile; sie wird mannigfach durch die Verhältnisse der Agricultur, wie Liebig in seiner Agriculturchemie so schlagend nachgewiesen, verändert, und auch diesen Punkt dürfen wir in Bezug auf Krankheitserscheinungen nicht ganz unberücksichtigt lassen. Wenn wir im Laufe vieler Jahre gewisse chronische Krankheitsprocesse eine weitere, zunehmende Verbreitung finden sehen, ein Verhältniß, welches sich ohne Zweifel in unserer Zeit bei Scrophulosis oder Tuberculosis herausstellt, so liegt es nicht fern, auch hier in Veränderungen der Beschaffenheit der Bodenoberfläche einen Grund zu jener Verbreitung aufzusuchen. So ist z. B. offenbar — wenn auch vielleicht nicht direct mit den erwähnten Krankheiten im Zusammenhange —, daß die Bodenoberfläche mit der Zeit an dem Gehalte des phosphorsauren Kalkes verlieren muß, denn von den Millionen von Knochengerüsten, zu denen dieser das Material liefert, kehrt ein nicht unbeträchtlicher Theil nicht wieder zu den fruchttragenden Feldern als Dünger zurück; das oben erwähnte Beispiel von der in England durch Knochendüngung herbeigeführten Veränderung der Fruchtbarkeit der Felder mag einen Beweis liefern, daß nicht Jahrtausende erforderlich sind, um in dieser Hinsicht sichtbare Effecte herbeizuführen.

Es liegen uns nur sehr vereinzelte Arbeiten vor, welche sich mit Umsicht mit der Aetiologie endemischer Krankheiten beschäftigen; im Allgemeinen sind die hierhergehörigen Studien und namentlich die Nachweise einer Uebereinstimmung und Abhängigkeit pathologischer Processe mit endemischen Verhältnissen bis dahin vernachlässigt; es finden sich in den Hand- oder Lehrbüchern der Pathologie zwar eine große Menge von Angaben, die aber in der That oft nichts mehr als Phrasen sind und jeder wissenschaftlichen Begründung entbehren. Wir sind zur Zeit meines Wissens nicht in dem Besitze einer guten medicinischen Geographie im weitesten Sinne des Wortes; so schwierig aber die Ausführung eines sol-

chen Werkes auch ist, der dadurch zu stiftende Nutzen würde eines reichen Lohnes gewiß sein; denn es kann nicht fehlen, daß ein genaues und gründliches Studium der in verschiedenen Ländern verschiedenen tellurischen und atmosphärischen Verhältnisse ein bedeutendes Licht über viele Krankheitsentwickelungen verbreitet.

Es werfen sich hier, als in das Gebiet allgemeiner Physiologie gehörend, eine Menge von Fragen auf, für deren Beantwortung ein Zeitraum von mehreren Generationen kaum hinreichend sein wird. Dennoch, abgesehen von dem großen Reize, welchen wissenschaftliche Betrachtungen dieser Art an sich tragen, müssen auch sie einmal näher in's Auge gefaßt werden, als es bis dahin der Fall war; sie müssen es im Gebiete der praktischen Heilkunde sowohl der Theorie halber, als namentlich wegen der unendlich wichtigen praktischen Consequenzen, welche sich aus ihnen gewinnen lassen. — Der einzelne Organismus, ein Theil der gesammten Schöpfung, steht mit allen übrigen Theilen derselben in so unmittelbaren Beziehungen, daß seine Lebenserscheinungen im gesunden und kranken Zustande nicht begriffen werden können, ohne eine gleichzeitige Betrachtung dieser übrigen Theile. Der Mikrokosmus steht nicht in dem schroffen Gegensatze zum Makrokosmus, welchen man ihm früher vindicirte; Entdeckungen großer durchgreifender Naturgesetze liefern den Nachweis überall existirender gleichartiger und ineinander fassender Processe. Nur die Kunst hat bisher oft getrennt, was die Natur in eine ununterbrochene Kette von Erscheinungen zusammengefügt hat. Seit Dujardin's Untersuchungen über die Infusorien, seit der wichtigen Entdeckung Wöhler's über die Sauerstoff-Entwickelung der Frustulien in der Rodenberger Saline, seit dem neuerdings bekannt gewordenen Nachweis der Kohlensäure-Exhalation der Schwämme und Pilze von Döpping und Schlossberger, seit Schmidt's gehaltreichen Forschungen über die Identität der Substanz der Pflanzenzellenmembran mit der des Ascidien- und Frustulien-Mantels

sind, um hier ein Beispiel anzuführen, die bisher gezogenen Grenzen zwischen Pflanzen- und Thierreich gefallen; ja Schmidt hat weiter in der neuesten Zeit den Versuch gemacht, auch die schroffe Scheidung zwischen organischen und unorganischen Stoffen abzuweisen und ruhmvoll, auf Schwann's und Schleiden's großartigen Forschungen fußend, eine neue Naturanschauung durchgeführt. Er bewundert es mit Recht, „daß weder Physiologen noch Physiker „es versuchten, den Gestaltungsproceß einst organisirt gewesener, „jetzt in's s. g. anorganische Reich zurückgetretener Stoffe, jener „Vermittler der organischen und unorganischen Natur, mit denen „die Chemie der zusammengesetzten Radicale sich beschäftigt, au„ßerhalb des Organismus, für sich, zu beobachten. Grade die„ses Studium," sagt er, „ist von der höchsten Wichtigkeit für „den Inductionsbeweis eines Hauptsatzes der Physiologie, den näm„lich, daß wir es in der unorganischen, wie organischen Natur „mit denselben chemischen Grundstoffen und den ihnen immanen„ten, von ihrem Begriff untrennbaren Kräften, d. h. Ursachen „gewisser Bewegungserscheinungen zu thun haben, deren Summe „wir als individuelles Leben bezeichnen."

Von der hohen Wichtigkeit dieser allgemeinen Naturerscheinungen durchdrungen, habe ich es zu Anfang ausgesprochen, daß die durch die vorliegenden Beobachtungen abgeschlossene Erkenntniß eines allgemeinen Naturgesetzes mir das wichtigere Resultat derselben zu sein scheine. Der mangelhafte Zustand einer allgemeinen vergleichenden Physiologie macht es erforderlich, die wenigen gleichlautenden Facta im Bereiche des organischen Geschehens zusammenzustellen und das Wenige, was wir besitzen, auszubeuten und zu benutzen. — Wir finden in dem Resultate der vorliegenden Untersuchungen weiterhin einen Beleg für die Einfachheit der Mittel, durch welche die Natur ihre Zwecke erreicht; dieses Einfache zu erkennen, ist aber auch die letzte und höchste Aufgabe aller Naturforschung. —

Erklärung der Zeichnungen.

Fig. 1. Künstlich aus Fett, Eiweiß und phosphorsaurem Kalk dargestellte Zellen. Dieselben bildeten sich nach Verlauf von 6—7 Stunden. (Cf. pag. 72.)

a. Reine kleine Fetttröpfchen und ein größeres mit schon etwas mehr als normaler matter Oberfläche.

b. Fetttröpfchen, auf denen sich ein Niederschlag von Albuminaten mit Kalk gebildet hat; zu Anfang gröber, in der weitern Ausbildung feiner gezeichnet.

c. Kleine Zellen, welche durchaus den Eiterkörperchen gleichen.

Fig. 2. Dieselben Zellen mit Schwefelsäure behandelt, umgeben von schwefelsauren Kalk-Crystallen. Es ist zu bemerken, daß die Schattirung dieser Zellen dunkler ist, als die der nicht mit Schwefelsäure behandelten.

Fig 3. Schwefelsaure Kalkcrystalle, wie sie sich bei rascher Bildung aus Schwefelsäure und phosphorsaurem Kalk aus Urinsedimenten darstellten.

Fig. 4. Dieselben Crystalle und deren langsame Entwickelung aus mit Schwefelsäure behandelten Urinsedimenten von phosphorsaurem Kalk.

a. Ein Theil des Sediments von phosphors. Kalk (körnchen- und perlenartig) und phosphorsaurer Ammoniak-Magnesia ohne S.

b. Frühestes Stadium der Gypscrystalle (wie alle diese Angaben, bei 350facher Vergrößerung).

c. Zweites Stadium.

d. Drittes Stadium. — b, c und d wurden auf ganz gleiche Weise im Serum aus durch Empl. canthar. erzeugten Blasen dargestellt; ebenso im Eiter verschiedener Wunden und aus frischem Fleisch.

Fig. 5. Gypscrystalle, aus frischen Exsudaten, eingedampftem Serum, auch hie und da aus phosphors. Kalk-Sedimenten im Urin dargestellt, fast sämmtlich aber sehr langsam entwickelt. Namentlich die Formen sub a. scheinen wahre Hemmungsbildungen des Crystalls zu sein. (Es kommt hiebei viel auf die Quantität der zugesetzten S an).

Fig. 6. a. Eine der Abbildung von Schmidt durchaus ähnliche schwefelsaure Kalk-Crystall-Druse.

b. Die einzelnen Crystalle als Oblonga.

c. Die oben beschriebene Bildung der spitzen Winkel nur einmal in dem mit S behandelten Eiter von einem durch Empl. cantharid. entblößten Corium beobachtet.

Fig. 7. Gypscrystalle, welche sich in der raschesten Weise aus mit S behandeltem phosphorsauren Kalk darstellten. Die anfänglichen Bildungsstufen sind hier gar nicht wahrnehmbar.

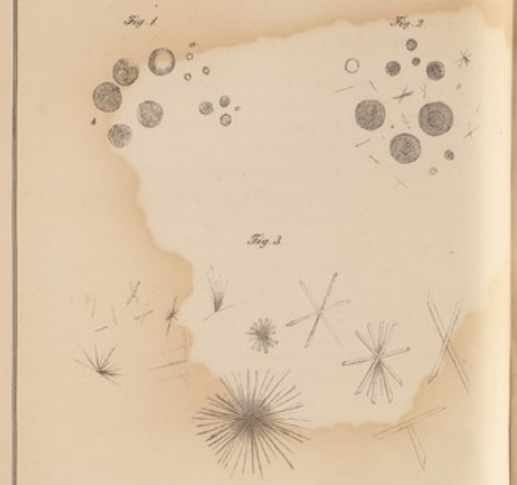
Fig. 1.
Fig. 2.

Fig. 3.


Fig. 4.


Fig. 5.
Fig. 6.


Fig. 7.

GESCHICHTE

UND

CHRONOLOGISCHE UEBERSICHT

DER

GESAMMTLITERATUR

DES

SCORBUTS.

Von

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SEINEM LIEBEN FREUNDE

DR. A. v. KYBER,

Kaiserl. Russ. wirkl. Staatsrath, Medicinalchef der Flotte und Hospitäler des schwarzen Meeres, correspondirendes Mitglied der Akademie der Wissenschaften und Ehren-Mitglied der Gesellschaft deutscher Aerzte zu St. Petersburg, Ritter des St. Wladimir-Ordens 3-ter Klasse, des St. Annen-Ordens 2-ter Klasse mit der Krone, des St. Stanislaus-Ordens 2-ter Klasse, des Griechischen Erlöser-Ordens, und Inhaber der Medaille für den Russisch-Türkischen Feldzug von 1828 und 1829.

I. Geschichte des Scorbuts.

Das Wort Scorbutus wird von einigen Schriftstellern vom Dänischen Schorbeet, Schorbuck, Schoerbuch (Olaus Magnus, hist. de gent. septentr. L. IX Romae 1555.) abgeleitet, andere wieder thun das vom Holländischen Scheurbuyk, Scheurbeck, noch mehrere legen das Sächsiche Schorbock zum Grunde, welches in seiner eigentlichen Bedeutung Grimmen und Reissen bezeichnet, während die richtige Ableitung von Slavischen Scorb (Krankheit überhaupt) sein dürfte.

Betrachten wir den Scorbut in Verbindung mit der Entwickelungsgeschichte des krankhaften Lebens zu verschiedenen Zeiten überhaupt, so müssen wir Häser's (Volkskrankh. 1 Th. S. 176.) geniale Ansicht als die richtige anerkennen: dass nämlich der Scorbut durch eine gewisse allgemeine Lebensstimmung zu epidemischer Entwickelung heranwuchs. Allein nicht allein dadurch, sondern auch durch solche zu der Zeit allgemeiner verbreitete gelegenheitsur-

sächliche Momente, wie sie jetzt noch an bestimmten Orten die Krankheit nicht vollkommen erlöschen lassen, waren dazu erforderlich. Ohne dieselben würde zwar die Richtung der krankhaften Lebensstimmung dieselbe geblieben sein, allein die Gestaltung wäre eine andere geworden. Den Beweis für diese Behauptung wird jeder finden, wenn er die Entwickelung und Ausbreitung an den Orten verfolgt, wo die Krankheit auch jetzt noch zu epidemischer Gestaltung gelangt und wo sich individuell das wiederholt, was in der früheren Zeit allgemeiner auf die specielle Richtung des krankhaften Lebens überhaupt seinen Einfluss übte.

Bis zum Jahre 1250 n. Ch. finden sich keine bestimmten Angaben über denselben (K. Sprengel's Gesch. d. Med. B. 2 S. 690. B. V. S. 522. F. Schnurrer's Chronik d. Seuch. T. II). Die Seefahrten der Alten waren an und für sich nicht sehr weit und fanden mehr in der Nähe der Küsten, als im offenen Meere statt, so dass durch die immerwährende Erneuerung der frischen Nahrungsmittel und den kürzeren Aufenthalt auf dem Meere die Entwickelung nicht gefördert werden konnte. Bei den verschiedenen Heereszügen und Belagerungen der Griechen und Römer fanden allerdings solche Verhältnisse statt, welche der Entwickelung des Uebels günstig sind, allein in den Angaben, welche für die Kenntniss des Scorbuts bei den Alten angesprochen werden, (Hippocrates, Dio Cassium, Plinius, Strabo, Celsus, Aetius, Paulus Aegineta, Aretaeus, C. Aurelianus, von welchen wieder Celsus, Aetius, Paulus Aegineta, Aretaeus und C. Aurelianus Abschreiber des Hippocrates waren. Vergl. Gruner, morbor. antiquitat. p. 132—141. Auch die Oscedo des Marcellus gehört nicht den scorbutischen Mundaffectionen an, sondern ist als Stomacace zu betrachten.) stellt sich eine solche durchaus nicht heraus und diese können leichter auf Stomacace, Typhus, Icterus niger, Milzleiden etc. als auf Scorbut bezogen werden. Uebrigens mag ich nicht in Abrede stellen, dass auch scorbutische Erscheinungen von denselben beobachtet worden sind, allein als einer eigenthümlichen Krankheit angehörig, wenn auch mit anderer Bezeichnung, sind dieselben von den Alten nicht geschildert worden. Erwägt man dabei: dass bei den gebildeten Nationen der alten Zeit Vegetabilien den Haupttheil der Nahrung ausmachten und dass die Römer überhaupt, besonders aber die Soldaten und der gemeine Mann, das Wasser vermischt mit Wein oder Essig genossen, so kann man um so mehr sich zu der Meinung hinneigen: dass der Scorbut nur sehr selten und nicht bedeutend entwickelt sich gezeigt hat und daher nicht beschrieben wurde.

Die erste deutlich sprechende Aufzeichnung der Krankheit trifft mit der Belagerung von Damiette zusammen, im Jahre 1249. Nachdem nämlich Ludwig IX im August mit seinem Heere nach Egypten aufgebrochen war und Damiette eingenommen hatte, zog er 1250 nach Cairo, an dessen Einnahme er jedoch durch die gewöhnlichen Nilüberschwemmungen verhindert wurde. Als nun im Heere Unthätigkeit und Unordnung herrschte und dasselbe, nach einigen glücklichen Siegen von Seiten Meles Sala, eingeschlossen wurde, und grosser Mangel im Heere folgte, so zeigte sich eine Krankheit, bei welcher die weichen Theile der Glieder hart wurden, blaue Flecken bekamen, das Zahnfleisch faulte und die Kranken an Blutungen starben. (Joinville. Hist. de Louis IX. (Paris) 1261 p. 324.) Als Gelegenheitsursache für die Krankheitsentwickelung wirkte wohl auch die sumpfige Gegend, in welcher das Heer lagerte.

Nach dieser Zeit findet man erst wieder um die Mitte des 15. Jahrhunderts Beobachtungen über Scorbut aufgezeichnet z. B. von Echt, nach seinen zu Cöln gemachten Beobachtungen. Ausgebreitet herrsch-

te das Uebel im Jahre 1446 im nördlichen Europa (G. Fabricius Annales urbis Misniae. Ed. an. 1446. Spangenberg's Mansfelder Chronik. Cap. 342. 393. J. Burchhard's Thüringsche Chronik. Leipz. 1613. B. 3. S. 25. Ch. Lehmann's Historisch. Schauplatz etc. Leipz. 1699. S. 849. Dreihaupt's Beschreibung des Saalkreises. B. 2. S. 764). Ebenso fallen in diese Zeit die ersten grösseren Entdeckungsreisen zur See, wo dann, bei der Unvollkommenheit des Seewesens überhaupt und den geringen Hülfsmitteln: um die Mannschaft gesund zu erhalten, sich der Scorbut, zumal auf den nördlichen Meeren, häufiger zeigte und bösartiger gestaltete, als dies im Allgemeinen in der neueren Zeit der Fall gewesen ist. So wurde die Mannschaft eines Venezianischen Kaufmanns P. Quirino im Jahre 1431 vom Scorbut ergriffen und hatte viel zu leiden, nachdem sie zwischen Norwegen und Island verschlagen worden war. (Forster's Gesch. d. Entdeck. im Nord. S. 273.) Eben so raffte das Uebel viele von der Mannschafft des Vasco da Gama weg, als er im Jahre 1498 auf seiner Entdeckungsreise nach Indien sich an der östlichen Küste Afrikas, zwischen Mozambique und Sofola, um seine Schiffe auszubessern, verweilte. (Barros Gesch. d. Entdeck. etc. A. d. P. von D. W. Soltau Th. 1. S. 107.) Im December 1535 befiel das Uebel die Mannschaft Cardier's, als sie auf der Fahrt nach Neufundland durch den grossen Meerbusen auf dem Flusse Canada sich befand. Im Februar hatte dasselbe so um sich gegriffen, dass von 110 Mann nicht 10 mehr gesund, 8 bereits gestorben waren und mehr als 50 dem Anscheine nach ohne Hoffnung darniederlagen; später noch blieben nicht 3 Mann gesund. Nachdem 25 gestorben und die übrigen Kranken sich in einen so elenden Zustande befanden, dass man ihr Aufkommen bezweifelte, lernte man von den Einwohnern den Nutzen der Canadischen Fichte gegen den Scorbut kennen,

vermittelst welcher denn auch die übrigen Kranken hergestellt wurden. (Hakluits collect. of Voyage V. 3. p. 225. Brief recit et succincte narration de la navigat. faite es ysles de Canada etc. Paris. 1545. p. 34.) Ebenso wurde die Reisegesellschaft v. Monts, Pongraves und Poutrincourts auf ihrer Reise durch den Theil von Neufrankreich (Canada, Norembergen, Hochelage und Labrador), welcher an Virginien gränzt, vom Scorbut heimgesucht. Es starben 36 Menschen und 36—40 erholten sich wieder im wiedergekehrten Frühlinge. Die grösste Sterblichkeit fand im Januar, Februar und März statt: es starben nämlich fast alle in diesen Monaten Erkrankte, während die später Befallenen leichter genasen. Wie wenig gekannt aber die Krankheit zu der Zeit war, geht daraus hervor: dass v. Monts, nach seiner Rückkehr, sich vergebens Aufschluss darüber zu verschaffen suchte. Im Jahre 1553 herrschte der Scorbut unter den Einwohnern von Stadogana (Hakluit a. O. Vol. 3. p. 225). Im Jahre 1556 wurde er epidemisch gleichzeitig mit Raphanie in Brabant, Böhmen, Schlesien und Genf beobachtet (Dodonaeus obs. méd. ex rar. c. 33) und eine solche Complication wird um so wahrscheinlicher, da manche Gelegenheitsursachen beiden Krankheitsformen als solche anheim fallen. Auch bemerken Aerzte der damaligen Zeit (Wyerus, Forest, Schenck etc.) im nordwestlichen Deutschland, vorzüglich aber an den Küsten, wo das Uebel häufig auftrat (R. Solenander. Consil. med. V. p. 501.), die Complication mit Gicht, nachdem der nasse Sommer von 1553 sehr nachtheilig auf die Vegetation eingewirkt hatte. Während der Jahre 1556, 1558 und 1562 beobachtete Ronseus (Dodonaeus l. c. C. 23.) den Scorbut in Holland und Brabant in Folge des aus Preussen eingeführten verdorbenen Getreides und der Witterung. Wahrscheinlich trug der allgemeine Mangel in Folge von Misswachs wesentlich zur

epidemischen Ausbreitung bei. Ronseus erwähnt zugleich: dass der Scorbut immer in Holland in Folge von ungesunder feuchter Luft und schlechten Trinkwassers herrsche und nur bei nasser Witterung dann epidemisch werde, wenn noch andere Ursachen begünstigend mitwirkten. Der Frühling und Herbst waren die Jahreszeiten, wo er am gewöhnlichsten vorkam und im letzterem zeigte er sich hartnäckiger als im ersteren. Er verschonte zwar kein Alter, aber suchte doch vorzüglich Männer im höheren Alter heim. Was Albertus Angaben über Scorbutepidemien in Brandenburg, am Harz, in Böhmen, Schlesien und Obersachsen anlangt, so sind dieselben nicht zuverlässig, da viele der aufgezählten Krankheitserscheinungen so wenig dem Scorbut eigenthümlich sind, dass man nicht ohne Unrecht behaupten kann: er habe andere Krankheitsformen mit demselben verwechselt, oder doch die zufällige Complication mit Scorbut als das Hauptleiden angesehn.

Sehr verheerend zeigte sich der Scorbut während der Jahre 1562, 1563—1573 in einzelnen Ortschaften Schwabens, Hollands etc., nachdem in Folge von Kälte und Nässe Misswachs vorausgegangen war. G. Horst führt an: dass wenn gleich zu der Zeit in Niedersachsen und der alten Mark Brandenburg der Scorbut sehr häufig beobachtet wurde, er doch wieder an andern Orten weit seltener vorkam, was er der mehr oder weniger gesunden Nahrung, Witterung und der Beschaffenheit des Bodens zuschreibt. Im Jahre 1569 wurden die Holländischen Küsten von grossen Ueberschwemmungen heimgesucht, besonders aber Löwen und die Umgegend, worauf der Scorbut ausgebreitet auftrat. Im Jahre 1573 wurden in Holland, nach vorausgegangener Hungersnoth ganze Dörfer durch die Krankheit entvölkert. Im Jahre 1631 richtete die Krankheit in Verbindung mit Typhus unter dem Schwedischen

Heere während der Belagerung von Nürnberg grosse Verheerungen an. (J. Rotenbeck et C. Horn. Specul. scorbut. etc. Norinb. 1633.) Auch kam derselbe in den früheren Zeiten häufig in London vor und richtete, zumal im Jahre 1675 grosse Verheerungen an. Nach den Londoner Todenlisten von 1671—1686 starben 9451 an Scorbut. Von 1686 — 1701 hatte sich bereits die Zahl auf 569 verringert und von 1701—1776 finden wir nur 226 an Scorbut Verstorbene aufgezählt. (Black. Sterblichkeit des menschl. Geschl. A. d. E. Leipz. 1798. S. 50. 305.) Die Abnahme des Uebels zu London geht Schritt vor Schritt mit der Verbesserung der Strassen und Wohnungen, so wie auch mit der einer gesunderen Nahrungsweise. Ebenso finden wir die Krankheit unter der Besatzung des belagerten Breda, entstanden nach einer Pestepidemie in Folge von Hungersnoth und ungesunden Wohnungen. Zuerst erschien dieselbe um die Zeit des Aequinoctium und bis zum 20. März waren bereits an 1608 Soldaten befallen. (Mye. de morb. et symptomat. popul. Bredan. etc. Antwerb. 1627. Edidit Gruner. Jenae 1792.) Mit dem Eintritt der wärmeren Jahreszeit und nach der Vertheilung von Wein und Taback verlor sich die Krankheit immer mehr und mehr. Bei einigen Kranken entwickelte sich vorzüglich die Zahnfleischaffection, bei andern die Purpura scorbutica. Viele litten an Herzklopfen und starben plötzlich. Am heftigsten wurden die trägen Holländer ergriffen, die gleich im Beginn der Hungersnoth Hundefleisch verzehrten und dabei in feuchten Quartieren lagen; seltener dagegen die Wallonen und Flanderer, welche im Allgemeinen mässig lebten, sehr selten Franzosen, deren froher Muth, selbst in den Tagen der grössten Noth, sich durch Körperbewegung und Gesang aufrecht erhielt.

Im Jahre 1703 wüthete die Krankheit unter der Besatzung des belagerten Thoren so bedeutend, dass 5—6000 Mann dadurch weggerafft wurden. Im Jahre

1710 kam sie häufig in Verbindung mit Pest zu Coppenhagen vor. (J. G. Böttiger. Morb. malign. etc. explicat. cui pestis per sex mens. Haffniae saevient etc. Hamb. 1713.) Im Jahre 1731, 1732 und 1733 wüthete das Uebel bei nasser Witterung zu Cronstadt (Sinopaeus), 1732 beobachtete Nitsch dasselbe in Wiburg, 1733 zu St. Petersburg und 1742 in Finnland zu Wiburg während 8 Monate (Januar—August) in einer solchen Bösartigkeit, dass von den Befallenen fast Niemand gerettet wurde; allein nicht alle Krankheitsfälle waren Scorbut, sondern andere nur mit demselben complicirte Krankheitszustände. (R. Krebel. Erkenntn. u. Heil. d. Scorbut. Leip. 1838. S. 31. f.) In den Jahren 1734, 1735 herrschte derselbe in Verbindung mit Wechselfiebern in Ungarn und Oberitalien unter den Oesterreichischen Truppen. (Kramer D. epist. de scorbut. Norimb. 1737.) Desgleichen in den Jahren 1732—1742 häufig in den Russischen Armeen. Während der Belagerung von Asow, im Jahre 1736, wo die Witterung abwechselnd kalt und regnicht war, und die Soldaten Mangel an Holz hatten, griff die Krankheit ungemein um sich. Darauf folgte Regen abwechselnd mit grosser Hitze und ausser den Ueberschwemmungen der Donau, wodurch die Truppen sehr belästigt wurden, litten sie auch Mangel an guten Nahrungsmitteln. Ebenso kamen viele Scorbutische unter den Regimentern vor, welche nach Otschakow marschirten, wozu die grossen Strapazen, die strenge Kälte und Nässe, die schlechten Quartiere und die mangelhafte Ernährung Veranlassung gab. Ganz dasselbe gilt auch von der grossen Anzahl Scorbutischer, welche im Feldzuge am Dniester vorkamen; doch mag sie auch dadurch angewachsen sein: dass viele abgemattete Rekruten zu den Regimentern kamen, wenigstens scheint dies dadurch eine Bestätigung zu finden: dass im Jahre 1793, in dem Feldzuge bei Chozim, wo die Rekruten so zeitig anlangten, dass sie sich vor dem Beginn des Feldzuges erholen konnten, das Uebel weit seltener vorkam. Um diese Zeit fand man die Hospitäler zu Asow, St. Amera, Kobilak und Abo immer mit Scorbutischen gefüllt und halbe Regimenter wurden Opfer der Krankheit. In den J. 1749—1751 richtete dieselbe, in Folge von feuchten Wohnungen und schlechter Kost, grosse Verheerungen unter der Besatzung von Riga an; 1758 war der Scorbut in der Umgegend von Breslau die herrschende Krankheit (Baldinger's Krankheit. einer Armee etc. Leipz. 1774. Th. 3.) und im J. 1759 wurden Viele unter den Englischen Truppen in Canada ein Opfer derselben. Während der J. 1761 u. 1762 litten die Oesterreichischen Truppen in Schlesien, Ungarn und Piemont ungemein durch denselben, eben so auch im letzteren Jahre die Englischen Truppen in Bremen. Theils die niedrige Lage von Bremen, theils aber auch die feuchten Quartiere und der Mangel an frischen Vegetabilien sind als wesentliche Gelegenheitsursachen zu betrachten. Eben so verheerend finden wir ihn in den J. 1789 und 1790 unter den Russischen Truppen während des Feldzuges in Finnland (E Enncholm's Taschenb. d. Kriegshyg. etc. St. Peterb. 1813). Im Jahre 1801 während des Feldzuges der Franzosen in Egypten folgte der Scorbut im Lager am See Madieh auf eine Augenepidemie (Larrey's med. chir. Denkw. etc. A d. f. Leipz. 1813. S. 263 f.). Zuerst wurden einige Verwundete heimgesucht, nachdem Mangel an Lebensmitteln und frischem Trinkwasser eingetreten und die Soldaten durch den Durchstich des Sees Madieh grosser Nässe ausgesetzt waren. Darauf breitete sich die Krankheit so sehr aus, dass ein grosser Theil der Armee und der Einwohner gleichzeitig ergriffen wurden und sich bereits im August 1500 Scorbutische in den Hospitälern befanden, von welchen täglich 3, 4 bis 5 starben, von den erkrankten Einwohnern sogar 6—7, da sie gar kein gutes

Trinkwasser und nur verdorbenen Reis zur Nahrung hatten. Anfangs schien zwar die Vertheilung von Weinessig, Datteln, Kaffe, und Zuckersyrup das Uebel zu beschränken, als aber das Trinkwasser zu mangeln begann, machte das Uebel reissende Fortschritte. Bemerkenswerth ist übrigens: dass während der Dauer des Scorbuts kaum 2—3 Pestfälle vorkamen. Die Officiere welche eine bessere Nahrung hatten, blieben verschont. Das Uebel befiel Personen jeden Alters, vorzüglich aber solche, welche eben eine schwere Krankheit überstanden hatten. Nachdem man anfing gesundes frisches Pferdefleisch und Brod, aus gutem Reis bereitet, zu vertheilen, nahm das Uebel immer mehr ab, wozu auch wohl noch der Umstand beitrug: dass die Armee mit Medicamenten, vorzüglich Tamarinden und Citronen versorgt wurde. Von ohngefähr 3500 Scorbutischen, die in die Hospitäler von Alexandrien kamen, starben vom Juli bis 18. October, wo die Einschiffung der Kranken erfolgte, 262 und mehr als 2000 begaben sich vor und während der Einschiffung zu ihren Bataillonen; 700 davon gelangten mit der Krankheit behaftet nach Frankreich. Alle waren bei der Ankunft in die Quarantaine entweder geheilt, oder auf dem Wege der Besserung mit Ausnahme von 6—7, die auf der Ueberfahrt nach Frankreich gestorben. Hundert und einige der am schwersten Ergriffenen blieben in Alexandrien zurück, kamen aber später nach Frankreich zurück, ohne dass unter ihnen verhältnissmässig mehr Todesfälle stattgefunden hatten. Nach der Capitulation von Alexandrien, versorgten die Engländer die Franzosen mit frischem Fleisch, Wein und Gemüse, wodurch natürlich die Heilung der zurückgebliebenen Scorbutischen bedeutend gefördert wurde.

In den Jahren 1802 und 1803 herrschte die Krankheit epidemisch im Temescher und Werschezer Kreise in Ungarn. (Schraud's Scharb. in Ungarn etc. Wien 1805.) Es lässt sich mit Bestimmtheit nachweisen: dass das Uebel auch früher an diesen Orten, jedoch in keiner so bedeutenden Ausbreitung angetroffen wurde, um die specielle Aufmerksamkeit der Behörde in Anspruch zu nehmen. Die Epidemie begann in den letzten Monaten des J. 1802 und erreichte im Februar 1803 ihre Höhe. Während derselben blieben die deutschen Einwohner verschont, gleich viel, ob sie getrennt, oder mit Wallachen vermischt wohnten. Von den Wallachischen Ortschaften litten am meisten die, welche an den morastigen Ufern des Temesch lagen. Dabei litten mehr Frauen als Männer und von den letzteren wieder mehr alte. Als vorzügliche Gelegenheitsursachen, müssen, nächst den Lokalitätsverhältnissen, die gebräuchlichen Fasten und sonstige ungesunde, ärmliche und unreinliche Lebensweise gelten. Bei der ersten Untersuchung im Februar belief sich die Krankenzahl auf 4000 und bis zum 4. Juli 5560 bei 91,499 Einwohnern; davon genasen 4740 und 820 starben, und zwar ⅔ Frauen und ⅓ Männer. Uebrigens ist die Berechnung in so fern nicht ganz genau, als sie nicht mit dem Anfange der Epidemie begann. Das Verhältniss der Todten zu den Genesenen ist 1 : 6.

Auch im benachbarten Tarantales Comitat brach die Epidemie in den von Wallachen bewohnten Ortschaften aus, vorzüglich in den morastigen Gegenden an den Ufern des Temesch, und Begaer Canals und in den mehr an der Moresch gelegenen Dörfern. Sowohl die Ausbreitung als Heftigkeit des Uebels war im Allgemeinen mit der an den angegebenen Orten gleich. Die Anzahl der Erkrankten belief sich auf 2566, der Gestorbenen auf 314. Auch überstieg die Zahl der erkrankten Frauen die der Männer. Ausserdem beobachtete man die Krankheit in den benachbarten Gespannschaften am Arad, Bekesch und Baes, wo die Einwohner theils Wallachen,

theils Deutsche und Ungarn sind, letztere jedoch in geringer Anzahl. In der ersteren, welche theils gebirgig und waldig, theils morastig ist, zeigte sich der Scorbut überall, jedoch häufiger im sumpfigen Theile; am wenigsten Kranke zählte die Gebirgsgegend der Processe von Arad und Borus-Jenö; das Verhältniss der Genesenen zu den Gestorbenen war 22 : 1. Im Processe von Arad waren nur 9 Ortschaften, mit 225 Kranken befallen; im Boros-Jenöschen 11 mit 270 Kranken. Ungleich häufiger trat die Krankheit im Wilagoscher und Zarander Processe auf. Im ersteren zählte man in 12 Ortschaften 1225 Kranke, im letzteren in 18 Ortschaften 1879. Noch zahlreicher war die Krankenzahl in Vilagos, Magyarat, Syndudvar, Sziklo und Nagy-Pel, denn es kamen auf 13,112 Einwohner 1542 Scorbutische, mit 232 Verstorbenen. Die Bekescher Gespannschaft, meist von Ungarn und Deutschen bewohnt, hat nur eine Wallachische Ortschaft, Kategyhza; während nun unter den ersteren nur vereinzelte Erkrankungen vorkamen, war dagegen in der letzteren die Krankheit so verbreitet, dass von 2149 Einwohnern 432 ergriffen wurden (234 Männer und 109 Weiber), Genesene zählte man 394. Als Ausnahme finden wir hier weniger Weiber befallen. Ausserdem kamen auch noch in der Baeser Gespannschaft Erkrankungen vor. Die Bewohner derselben sind Illyrier, Ungarn und Deutsche. Die Krankheit trat einige Monate später auf, als in den früher angegebenen Ortschaften, erreichte jedoch keinen bedeutenden Grad. Die Gegenden an der Theis und Donau waren der Sitz der Krankheit, während der mittlere Theil der Gespannschaft verschont blieb. Die grösste Krankenzahl lieferten die Illyrier, die kleinere die Ungarn und von den Deutschen erkrankten nur Bewohner von Cseb.

In den Jahren 1807 und 1808 finden wir den Scorbut unter den französischen Truppen in Preus-

sen. Im Winter und Frühjahre 1820 unter den Truppen zu Council Bluff's und St. Peter's am Missouri. Die Krankenzahl bis zum 1 März war 503, wovon 168 starben. Von den Officieren erkrankte nur einer. Die Gesammtzahl der Besatzung beider belief sich auf 1016 Mann. (F. Frey, Americ. Journ. 1842.) In den Jahren 1828 und 1829 unter den Russischen Truppen in der Türkei, (Seidlitz u. Petersen, Med. prakt. Abh. deutsch. A. in St. Petersb. B. I. S. 97. 155. 1835) und in zwar so bösartiger Gestalt, dass mehrere Regimenter, welche zu Sizeboli in Garnison lagen, den grössten Theil ihrer Leute dadurch verloren. Im Jahre 1836 unter den Englischen Truppen in der Provinz Adelaide (J. M. Grigor, Lond. med. Gaz. V. 20). Im Jahre 1840 unter den Russischen Truppen an der Ostküste des schwarzen Meeres, so ausgebreitet, dass binnen 3 Monaten 1080 Kranke im Hospital von Phanagoria aufgenommen waren. Bösartig gestaltete er sich durch häufige Complication mit Febr. gastr. nervos., Febr. intermitt., Ruhr und Hydrops (Нарциссъ: Военн. Журн. V. 58), ferner in der ersten Hälfte desselben Jahres zu Moskau, (W. v. Samson-Himmelstiern: Haeser's Arch. V. 4. 1844.) zu Troizk im Orenburgischen Gouvernement (Th. Schütz: Med. Zeit. Russl. 1846. No. 1 u. 2) und ausserdem noch an vielen andern Orten, vorzüglich in den Fluss- und Wassergebieten. Im Jahre 1843 im Prager Provinzialstrafhause. Es erkrankten im Mai und Juni von 777 Sträflingen 379 (302 Männer und 77 Frauen) mit 12 Pct. Sterblichkeit. Ebenso verderblich ward der Scorbut der Armee des Ibrahim Pascha in Arabien, denn der grösste Theil der Verstorbenen waren Scorbutische und wenn von mehr als 100,000 Mann nur wenige zurückkehrten, so muss er arg gewüthet haben. (Lacheze. Du scorbut d'Arabie, dit improprement plaie du Hedjaz: Bullet. de l'acad. de Méd. de Paris. Juin. 1844.) Auch in Ir-

land fehlt es nicht an Scorbutepidemien und über eine in den letzten Jahren berichtet J. O. Curran (Dublin Quaterly Journal of Med. Sc. 1847).

Als constanten verderblichen Begleiter finden wir den Scorbut auf den Schiffen, welche zumal früher grosse Reisen machten, vorzüglich bei solchen auf dem Eismeere. So z. B. bei der in den Jahren 1734—1743 von Owzyn, Laptew und 1809 auf der von Hedenströrm, (F. v. Wrangel's Reise längs d. Nordk. Sibiriens etc. Th. 1. S. 40. 62. 63. 101.) jedoch fehlt es auch später bis auf die neusten Zeiten nicht an Beispielen, wo die Krankheit beobachtet wurde. Wie verheerend übrigens die Krankheit den früheren Seefahrern gewesen ist, sehen wir bei den ersten Reisen, welche die Schiffe der Ostindischen Compagnie unternahmen, wo beinahe ¼ der Bemannung durch denselben weggerafft wurde (Black a. O. S. 305); bei Dellons Reise (un voyage aux Indes orientales 1638) und bei Anson's Reise, auf welcher über ⅔ von seiner Mannschaft am Scorbut hinstarber. Anson ging am 18 September 1740 von St. Helena aus mit 5 Kriegsschiffen unter Segel; allein die ganze Ausrüstung war höchst mangelhaft, die Lebensmittel spärlich und schlecht, die Mannschaft ungeübt und kränklich. Bereits nach einigen Monaten zeigte sich der Scorbut mit solcher Heftigkeit, dass von der 510 Mann starken Besatzung nur 130 mit Anson im Juni 1744 nach England zurückkehrten. Aus dieser Reise lernte man: dass zu Erhaltung der Gesundheit der Schiffsmannschaft überhaupt und zur Vorbeugung des Scorbuts nicht allein die grösste Reinlichkeit der Verdecke, Hangematten etc. nöthig sei, sondern dass man auch für jeden einzelnen Mann einen hinlänglichen Raum im Schiffe zu berechnen habe, dass eine zweckmässige Eintheilung der Schiffsarbeiten nöthig sei und dass ausserdem noch auf das Vorhandensein von diätetischen und medicinischen Hülfsmitteln Rücksicht

genommen werden müsse. Die bereits oben erwähnten Umstände, welche das Auftreten des Scorbuts herbeiführten, wurden noch durch die häufigen Unwetter unterstützt, womit die Schiffe zu kämpfen hatten. Die Stürme zerstreuten nämlich die Flotte und nach und nach mussten 3 Schiffe, als unbrauchbar, verlassen werden. Dabei umlagerten Nebel die Segel, der Schnee fror an der Takelage fest und während die vom Scorbut befallenen Leute in ihren Hängematten starben, vollzogen andere mit halberfrorenen Händen und Füssen die nöthigen Arbeiten. Endlich, nachdem die halbe Mannschaft durch den Scorbut aufgerieben war, und längst schon der Centurio, das Hauptschiff, als Hospital diente, erreichte Anson mit Mühe die Insel Juan Fernandez und ging in der Bucht von Cumberland vor Anker. Der Scorbut verschonte zwar von jetzt an die noch übrige Mannschaft, allein die noch vorhandenen Kranken, für welche keine Rettung mehr möglich war, fanden alle ihr Grab unter den Felsen der Insel. Auf der noch übrigen Fahrt, wo Anson auch mit vielem Missgeschick zu kämpfen hatte, brach zwar das Uebel auch wieder aus, nachdem er, 7 Wochen zurück, mit ganz gesunder Mannschaft die Küste Mexikos verlassen hatte; allein es stieg doch nicht wieder zu der früheren Bösartigkeit.

Ebenso litten die Schiffe, welche in den Jahren 1746 und 1747 die Reise nach der Hudsonsbay machten. (Ellis. A voyag. to Hudsonsbay etc. Lond. 1748.) Ferner die Besatzung des englischen Schiffes Talbot, welches am 23. März 1768 von England aus nach Bengalen segelte. (C. Clark's Beobacht. über Krankh. auf lang. Reis. etc. A. d. E. Coppenh. 1798.) Zuerst zeigte sich der Scorbut im Juni, nachdem der Talbot bereits das Vorgebirge der guten Hoffnung umschifft und theils mit heisser, theils mit feuchter nebliger Witterung gekämpft

hatte, und Wechselfieber unter der Mannschaft vorausgegangen waren. Im Juni zählte man von der 240 Mann starken Besatzung 8 Scorbutische, und obgleich es nicht gelang die Krankheit zu beseitigen, so unterlag doch derselben keiner. Am 11 Juli, wo das Schiff Madagaskar verliess, waren alle Kranke genesen und im August ankerte der Talbot im Flusse Culpee in Bengalen. Nachdem nun derselbe am 22 März 1769 seine Rückreise angetreten hatte, stellte sich im Juni abermals der Scorbut ein, wahrscheinlich in Folge der schlechten Nahrungsmittel, welche die Mannschaft in Bengalen erhalten hatte und des im Anfange eingetretenen nasskalten Wetters, welches auch verderblich auf die Schiffsprovision einwirkte. Obgleich nun das Uebel rasch um sich griff, so dass bereits am 18 Juli 20 Mann zur Arbeit gänzlich untauglich waren, und selbst Officiere, zumal die durch Fieber geschwächten, nicht verschont blieben und bei der Ankunft in der Augustinsbay auf Madagaskar von der Besatzung (87 Mann mit Officieren) bereits 33 hart daniederlagen, so wurden doch alle hergestellt. Dies glückliche Resultat wurde dadurch erzielt, dass man auf Madagaskar Pomeranzen, Gemüse, Wein etc. im Ueberfluss vertheilen konnte.

Ferner sehen wir die Krankheit auf der Englischen Flotte, welche im Jahre 1773 nach Algier segelte, (U. B. Askow. Diar. med. naval. etc. Lond. 1774) und während der Jahre 1774 — 1783 auf der Schwedischen Flotte, bald in grösserer, bald in geringerer Ausbreitung. (Faxe.) Im Jahre 1780 und 1781 herrschte der Scorbut auf dem Russischen Geschwader des Viceadmirals Borissow in einer solchen Ausdehnung, dass er dadurch genöthigt ward auf der Rhede von Lissabon vor Anker zu gehn, um für die Kranken am Lande ein Lazareth zu errichten; wo alsdann binnen 40 Tagen an 2000 Kranke, viele selbst mit dem höheren Grade behaftet, hergestellt wurden. Die wesentlichste Gelegenheitsursache scheint ungünstige Witterung zur See gewesen zu sein. Die Mannschaft hatte nämlich mit Staubregen, Kälte und Stürmen zu kämpfen. Als hierauf die Flotte von Lissabon aus, nach einer langwierigen Fahrt, in Livorno anlangte, war man abermals genöthigt, viele Scorbutische an das Land zu setzen.

Eben so stellte sich der Scorbut in den Jahren 1782—1784 auf der Flotte unter Admiral Tschitschakow im Mittelmeere ein, allein derselbe nahm nicht überhand. (Spedicati's Th. zur Beurtheilung d. Scorb. A. d. J. St. Petersb. 1787 S. 112.). Auch kamen im Kriege mit Amerika auf der Englischen Flotte mehr Menschen durch den Scorbut um, als durch Schiffbrüche und die vereinigten Schwerdter der Feinde. (F. Milmann's Unters. über d. Scorbut. A. d. E. Berlin 1795. S. 1. f.)

Eben so litt die Englische Flotte, welche in den Jahren 1778—1783, während des Krieges mit Frankreich, Holland und Spanien, in den Antillen stationirt war, viel durch Scorbut. (G. Blanc's Beobacht. über Krankh. d. Seeleute. A. d. E. Marb. 1788. S. 1—135.). Nachdem nämlich bis 1781 im Allgemeinen keine bedeutende Anzahl Scorbutischer auf der Flotte vorgekommen war, erreichte das Uebel im Monat April, nachdem die Flotte 6 Wochen hindurch auf der Windseite von Martinique gekreuzt hatte, eine solche Ausdehnung, wie zuvor niemals auf der Englischen Flotte vorgekommen war. Als Ursache dürfte gelten: dass die Flotte lange Zeit selbst einen Theil der 6 Monate hindurch weder frisches Fleisch, noch auch Vegetabilien erhalten hatte. Diejenigen Schiffe, welche zu St. Eustach und St. Lucie ankerten und frische Lebensmittel erhielten, litten weit weniger. Als die Flotte am 23. Mai in Barbados anlangte, war der Scorbut bei 1600 Kran-

ken fast die einzige herrschende Krankheit. Uebrigens ist zu erwähnen: dass der Alcid und der Invincible, welche sich mit Citronen versorgt hatten, nur wenig durch den Scorbut litten. Die Malzabkochung wurde zwar regelmässig auf der Flotte vertheilt, jedoch ohne besondere sichtbare Wirksamkeit, am meisten leistete sie noch auf dem Gibraltar, Centaur, Torbay und Alcid. Am 10. und 12. Juni verliess die Flotte Barbados und die Bemannung blieb gesund, bis der grösste Theil der Flotte im August nach Nordamerika segelte. Am 25. Februar 1782 vereinigten sich die aus England angekommenen Schiffe mit der Flotte, die im December 1781 aus Nordamerika zurückgekehrt war und den zu Barbados zurückgebliebenen Schiffen, deren Gesundheitszustand sehr befriedigend war, da sie sich mit frischen Vegetabilien versorgt hatten. Die vereinigte Flotte betrug 34 Linienschiffe und ging am 1. Mai zu St. Lucie vor Anker. Die vorzüglichsten Krankheiten in den letzten Monaten waren: Ruhr, Blattern und Fieber, aber auf 1884 Kranke kamen nur 130 Scorbutische, von welchen 2 starben. Im April war im Allgemeinen der Gesundheitszustand befriedigend und Blane schreibt dies den beiden glücklich abgelaufenen Seetreffen und den guten Lebensmitteln zu, womit die Flotte versehen war. Uebrigens war dennoch die Zahl der Scorbutischen grösser als im vorigen Monat. Die ganze Flotte (24 Schiffe), welche in Jamaika zurückgeblieben, hielt einen grossen Theil des Mais die See und kehrte am 25. Mai nach Port Royal zurück, wo sie bis Ende Juni im Hafen blieb, ausgenommen 3 Schiffe. Trotz der grossen Sterblichkeit auf der Flotte durch Fieber, war die Zahl der Scorbutischen im Mai und Juni gering und dass dieselben nicht gänzlich fehlten, ist der Fortdauer der Ruhr und Fieber zuzuschreiben und dem Umstand, dass die Schiffe nicht alle mit frischen Lebensmitteln

versorgt werden konnten. Am 17. Juni verliess die Flotte Jamaika, um nach den Küsten von Nordamerika zu segeln und langte am 17. September zu Neuyork an. Der Scorbut fing sehr bald nach der Abfahrt, besonders aber gegen Ende August um sich zu greifen. Weniger war dies auf den Schiffen der Fall, welche sich reichlich mit Wein, Molasses und Sauerkraut versorgt hatten. Obgleich im October der Gesundheitszustand im Allgemeinen gut war, so fehlte es doch nicht an Scorbutischen. Am 25. October segelten 13 Linienschiffe nach Westindien, während die übrigen zurückblieben und langten am 20. November in Barbados an; nachdem auf der Fahrt ausser einem 2-tägigen Sturm günstiges Wetter stattgefunden, nahm während November der Scorbut immer mehr ab. Im December blieb die Flotte in Barbados und wurde durch 8 aus England angelangte Schiffe vermehrt. Die grosse Anzahl von Scorbutischen aber, welche man im December auf der Flotte zählte, rührte daher: dass der Magnificent, aus England angelangt, sehr viele unter seiner schlecht bekleideten Besatzung hatte. Im Januar zählte man auf der alten Flotte nur 44 Scorbutische, auf den neu angekommenen dagegen 320. Im Februar auf der ersteren 63, auf den letzteren 212. Im März auf der ersteren 46, auf den letzteren 123, ausser den Scorbutischen, welche man in das Hospital geschickt hatte. Bis zum 12. Januar blieb der Haupttheil der Flotte in Barbados, kreuzte dann 4 Wochen lang bei Martinique und am 8. Februar ging die ganze Flotte bei St. Lucie vor Anker und im April 1783 endete der Krieg.

Ferner finden wir den Scorbut in bedeutender Ausbreitung auf den Englischen Schiffen Alexander, Friendship, Lady Pynrhyn, und Scarburg auf der Fahrt von Neusüdwallis über Batavia nach England. (Forster's Magaz. von merkwürdig. Reis. B. 1. S.

125. 154.). Am verbreitesten und verderblichsten war die Krankheit auf dem Alexander und der Friendship. Bereits am 10. August, nachdem die Schiffe am 14. Juli Port Jackson verlassen, und bei bald schwüler bald stürmischer Witterung in der Nähe von Neuseeland gelangt waren, begann die Krankheit sich zu zeigen, und hatte zu Anfange November so sich ausgebreitet, dass ausser den Officieren kaum ein Mann dienstfähig blieb. Weniger breitete sich die Krankheit auf der Lady Pynrhyn und dem Scarbourg aus, weil erstere auf Otahaiti und letzterer auf Tinian sich mit frischem Fleische und Vegetabilien versorgt hatten. Uebrigens mangelte es dieser Flotte gleich beim Beginn der Reise an Aerzten und der zur Verhütung des Scorbuts nöthigen Provision.

Auch litt die französische Schiffsmannschaft, welche im Jahre 1788 von Frankreich aus nach Neufundland des Stockfischfanges halber gesegelt war und daselbst überwinterte, durch den Scorbut. Die wesentlichen Ursachen waren hier mangelhafte Bekleidung, feuchte kalte Witterung, Nebel und Mangel an frischer Nahrung. Auch auf der Rückfahrt nach Frankreich, wo sie mit stürmischer Witterung und Mangel zu kämpfen hatten, nahm das Uebel so überhand, dass man von 80 Kranken 40 Scorbutische zählte (Larrey).

In dem Jahre 1794—95 zeigte sich auf der Englischen Flotte kein Scorbut, obgleich der Typhus auf derselben herrschte und auch Blattern und Wechselfieber vorkamen. Im Monat zählte obige 32 Linienschiffe, 8 Fregatten, 1 Brander, 1 Schaluppe, 1 Kutter, 2 Lastschiffe und ein Hospitalschiff. Der Grund für das Nichterscheinen des Scorbuts mag allerdings in dem grossen Vorrath von Zucker und Citronensaft zu suchen sein, womit die

Schiffe versehen waren. Zuerst erschien im März 1795 eine allgemeine scorbutische Diathese unter der Mannschaft der 40 Segel zählenden Flotte, die bereits Ende Januar ausgelaufen war und im Kanal kreuzte, als wahrscheinliche Folge des vorhergegangenen harten Winters, wodurch viele Schaafe und Rinder umkamen und die Preise des Fleisches so stiegen, dass die Lieferungen für die Flotte verringert wurden. Am 17. März gingen die Schiffe London, Valiant, Hannibal, Colossus, Robust, nebst 2 Fregatten unter Segel und erhielten eine Quantität Citronensaft. Ebenso segelte eine Eskadre unter Elphinstone nach dem Vorgebirge der guten Hoffnung, die durch den Ankauf von Citronen grösstentheils vom Scorbut befreit ihre Rückfahrt antrat. Auf den im Canal kreuzenden Schiffen dagegen zeigte sich nicht allein die Krankheit sehr bald und die Krankenzahl wuchs schnell, sondern sie erreichte auch schnell ihre höchsten Grade. In Folge des Verbrauches des noch vorräthigen Citronensaftes wurden viele Kranke hergestellt, die übrigen aber ins Hospital geschickt. Am 16. April lief eine Eskadre unter Waldegrave, bestehend aus den Schiffen Minotaur, Invincible, Excellent, Tremendous, La Nymphe und Blonde aus. Am Bord des Excellent befanden sich nicht nur viele Scorbutische, sondern bei der ganzen Besatzung eine scorbutische Diathese. Am 7. Juni lief sie wieder in den Hafen ein und ein reichlicher Vorrath von Citronen und Pomeranzen während der Zeit, verhinderte nicht allein das Auftreten des Scorbuts auf den davon befreit ausgelaufenen Schiffen, sondern machte es auch möglich, von den Scorbutischen keinen durch den Tod zu verlieren. Günstig wirkte mit darauf das schöne Wetter hin, welches diese Eskadre während der ganzen Fahrt genoss.

Die Flotte, welche unterdessen im Canal ge-

kreuzt hatte, kehrte am 17. April unter Colpoys nach Spithead zurück. Sie war zwar auch mit Citronensaft versehen worden; allein wenn man auch keinen Kranken durch den Tod verlor, so gab es doch auf allen Schiffen viele Scorbutische. Im Durchschnitt litt die Mannschaft der grösseren Schiffe mehr, als die der kleinern. Das Schiff Hannibal musste zu Plymouth einlaufen, weil die ganze Besatzung vom Scorbut befallen war. Wahrscheinlich trug wohl der Umstand dazu viel bei, dass das Schiff neu und dass der grösste Theil der Besatzung aus Landsoldaten bestand, die des Seelebens ungewohnt waren. Die Kranken genasen übrigens mittelst grüner Vegetabilien theils auf dem Lande, theils auf dem Schiffe selbst.

Eine andere Eskadre kam unter Harvey aus der Nordsee zurück, deren Besatzung noch mehr durch den Scorbut litt, als die der oben erwähnten Eskadre. Das Schiff Prince of Wales war genöthigt, 50 Scorbutische zu Deal ins Hospital zu schicken, von denen 5 auf dem Wege dahin starben und ausserdem brachte das Schiff noch viele Scorbutische mit nach Spithead. Das Schiff Thunderer war das einzige, welches ohne Scorbutische heimkehrte. Es hatte sich nämlich hinreichend mit grünen Vegetabilien, lebendigem Vieh etc. versorgt. Zu Ende April erfolgten die Fleischlieferungen in der gewöhnlichen Quantität und ausserdem, zur Vertheilung an die Scorbutischen, Citronen, Pomeranzen und grüner Gartensallat. In Folge davon fiel nicht nur die Zahl der Scorbutischen bedeutend, sondern die Genesung erfolgte auch auf den Schiffen.

Am 2. Mai segelten die Schiffe Leviathan, Hannibal und Swistsure nach St. Helena und St. Domingo, aber versehen mit Citronensaft verloren sie keinen Scorbutischen durch den Tod, obgleich die Krankheit auf allen allgemein herrschte. Obst und Gartengewächse brachten es jedoch bald dahin, dass man den Gesundheitszustand der Mannschaft als befriedigend ansehen durfte. Auch brach der Scorbut im Mai auf dem Royal George aus, wo man 30 Kranke zählte. Auf der Queen Charlotte und den übrigen Schiffen sprach sich die scorbutische Diathese deutlich aus. Am 26. Mai gingen der Royal Sovereign, Mars, Triumph, Bellerophon, Brunswick mit der Fregatte Phaëton und Pallas unter Cornwallis von Spithead in See. Nur der Mars war vom Scorbut frei, die übrigen Schiffe hatten einen Vorrath von Citronen und Sallat, von Früchten und Citronensaft in Fässern eingenommen. Mittelst derselben gelang es viele Scorbutische auf der See herzustellen, so z. B. 100 auf dem Triumph. Am 24. Juni lief diese Eskadre in Plymouth ein und ging, nach beendigten nöthigen Ausbesserungen, nach Bellisle, um einige Schiffe der Eskadre Bridports abzulösen. Während des Monats Mai nahm der Scorbut auf den Schiffen, welche von verschiedenen Eskadren nach Spithead zurückgekehrt waren, immer mehr zu. So zählte London 99, Colossus 86, Barfleur 60, Prince of Wales 80, Robust 50, und Russel 64. Dass der Scorbut nicht gänzlich erlosch, muss der ungenügenden Quantität grüner Gemüse zugeschrieben werden, indem nach hinreichender Lieferung zur allgemeinen Vertheilung der Scorbut so abnahm, dass man keine Kranke mehr ans Land schickte. Es wurden überhaupt von der Mitte des März bis zum 12. Juni 3000 ganz zum Dienst unfähige und 6000 leichter befallene Scorbutische hergestellt.

Am 12. Juni gingen unter Bridport der Royal George, Queen Charlotte, Queen, Prince of Wales, London, Prince George, Prince, Barfleur, Sans Pareil, Valiant, Colossus, Irresistible, Russel, Orion nebst mehreren Fregatten etc. in See und kreuzten

in der Nähe von Quiberon. Sie waren reichlich mit Vegetabilien, Citronensaft und gutem Biere versehen. Am 10. Juli vereinigten sich mit denselben die von Cornwalis befehligten Schiffe: Royal, Sovereign, Formidable, Triumph, Bellerophon, Invincible und Brunswick. Im August fing der Scorbut aufs Neue an sich einzufinden und griff während des August mehr oder weniger um sich. Zu bemerken ist: dass zwar die Provision in jeder Weise zweckmässig war, dass es aber an gutem Trinkwasser zu mangeln anfing. Der Robust, welcher im Juni mit Warrens Eskadre gesegelt war, blieb so lange vom Scorbut befreit, als Früchte und Citronensaft ausreichten; darauf nahm er aber so überhand, dass er 69 Kranke, mit den höchsten Graden behaftet, zu Haslar in das Hospital schaffen musste, von welchen 3 starben ehe sie noch das Land erreichten.

Am 20. September kehrte die Eskadre von Bridport nach Spithead zurück, wo der Royal George 160 Scorbutische zählte, der Royal Sovereign 250, die Queen 78, Sans Pareil 100, Invincible 260, Valiant 100, Triumph 30, Bellerophon 30, Pallas 17, Megäre 60. Nach der Vertheilung grosser Quantitäten Aepfel und frischer animalischer und vegetabilischer Nahrungsmittel erlosch die Krankheit in kurzer Zeit. (Frotter.)

Häufig findet sich die Krankheit auch auf den Schiffen ein, welche den stillen Ocean wegen des Spermacet-Wallfischfanges besuchen. (W. Bower. Noval. Avantur. II. B. Lond. 1833) In der neusten Zeit litt die Mannschaft des Kapitain Lasarew so bedeutend, dass er, ohne seine Erforschung von Nowaja-Semlja begonnen zu haben, zurückkehren musste, da die Mannschaft das Schiff nicht mehr bedienen konnte. (Bullet. scient. de l'acad. de St. Petersb. B. II. S. 139. 1837.) Ebenso auch die Mann-

schaft Pachtussow's bei seiner Ueberwinterung auf Nowaja-Semlja im Jahre 1832 und 1833. Die ersten Scorbutischen kamen im März und die ersten Todten im Mai vor. (Bullet. sc. a. O. S. 143. 144. 148.) Ferner die Mannschaft und Officiere der französischen Schiffe Astrolabe und Zelée, commandirt von Dumont d'Urville, in den Südpolargegenden. Unter 58° S. B. begegneten sie am 15. Januar den ersten Eisbergen und brachten bis zum 7. März theils vom Eise eingeschlossen, theils mit Nebel kämpfend zu; worauf sie am 7. April in den Chilischen Hafen Conception einliefen. Bis zum 1. April waren von der Mannschaft der Zelée ein Scorbutischer gestorben und 8 lagen mit den höheren und 30 mit den milderen Graden darnieder, dagegen hatte die Astrolabe nur 15 Scorbutische. (Moniteur Aout. 1838.) Ebenso wurde sämmtliche Mannschaft, mit welcher Black seine letzte Nordpolexpedition machte, vom Scorbut befallen. (G. Friedberg's Journ. d. neust. Lond. u. Seereis. H. 8. S. 70. 1837), und auf der Reise des berühmten Ross fehlte es auch nicht an Scorbutischen (Second. voyage of discovery to the arct. reg. etc. Lond. 1835). Ebenso unter der Besatzung der Fregatte Columbia auf der Reise um die Erde. (F. Coale: Ammeric. Journ. 1842.)

Im Allgemeinen drängt sich nun hier die Ueberzeugung auf: dass zwar in früherer Zeit, namentlich zu Ende des 15-ten, während des 16-ten und einen Theil des 17-ten Jahrhunderts hindurch, der Scorbut weit häufiger und verbreiteter, in Folge einer individuellen Lebensstimmung, herrschte, als späterhin; dass man aber keineswegs berechtigt ist, denselben als die allgemein herrschend gewesene Dyscrasie zu betrachten, wie der Recensent meiner Schrift: über den Scorbut, Leipzig 1838, in der Berliner litterarischen Zeitung 1839 No. 24 anzunehmen scheint. Das Uebel war bei seiner Entstehung und bei dem später erneuten Auftreten an bestimmte

Gelegenheitsursachen gebunden, die niemals so allgemein einwirkten, dass bei der Mehrzahl (ausgenommen zur See) diese Lebensstimmung allgemein zur Krankheit erhoben worden wäre. Selbst wenn sich dieselbe über Länderstriche ausbreitete, finden wir nur immer einen bestimmten Theil der Bewohner, die ärmeren, ergriffen, und die reicheren nur dann, wenn sie den Gelegenheitsursachen anhaltend ausgesetzt waren. Ferner kann man nachweisen: dass das allmälige Erlöschen, oder das seltenere Auftreten der Krankheit, durch verbesserte Bodenkultur, durch gesundere Wohnungen und zweckmässigere Lebensweise, nicht aber durch Erlöschen der Dyskrasie überhaupt herbeigeführt wurde und dass der Scorbut in den Gegenden, wo er noch jetzt angetroffen wird, nächst den klimatischen Verhältnissen, aus den Ursachen hervorgeht, welche die Lebensweise mit sich führen, wie wir dies namentlich im Jahre 1840 in Russland an vielen Orten, z. B. in Moskau, unter den Truppen an der Ostküste des schwarzen Meeres, zu Sewastopol etc. etc. beobachtet haben.

Ferner dass, ohnerachtet aller Hülfsmittel, er dennoch auf langen Seereisen in den kalten Regionen nicht immer ausbleiben, wohl aber durch dieselben gelingen wird, der Ausbreitung und Steigerung zu begegnen. Wie erfolgreich umsichtige Vorkehrungen in dieser Beziehung gewesen sind, sahen wir bei der französischen Expedition, bestehend aus den Fregatten Revanche, Syrene und Guerrier, welche im Jahre 1806 im nördlichen Eismeere unter dem 72—77° N. B. kreuzte, um den Wallfischfang der Engländer in diesen Regionen zu stören, und bei O. v. Kotzebue's Entdeckungsreise in der Südsee und nach der Beringsstrasse, in den Jahren 1815—1818, wo kein Mann der Besatzung vom Scorbut befallen wurde.

II. Chronologische Uebersicht der Gesammtliteratur des Scorbuts.

Lind hat zwar in seinem Werke über Scorbut eine Uebersicht der wesentlichen Schriften gegeben, die noch vom Uebersetzer, J. Nath. Pezold, vervollständigt wurde; allein immer aber ergaben sich, wie auch bei andern, noch Lücken, die hier nicht allein für die ältere Zeit, sondern auch für die spätere vollständig ausgefüllt sind. Dass so eine Menge Dissertationen, Streitschriften etc., welche keinen besondern Werth haben, mit aufgeführt werden mussten, bedarf keiner weiteren Rechtfertigung.

1539. Agricola. Medic. herbar. 1539.

Hält das Uebel für Stomacace, wie sie Plinius beschrieben hat.

1541. I. Echtius. De scorbut. vel scorbut. passion. epitome Witteberg. 1541. 1624.

Wahrscheinlich der erste Arzt, welcher eine Abhandlung über den Scorbut geliefert hat. Bereits in der angeführten Schrift findet man die Frage aufgeworfen: ob das Blut im Scorbut nicht verdorben sein könne, ohne dass die Milz, oder ein anderes Eingeweide angegriffen sei? E. hält das Uebel für ansteckend.

Obgleich Haller diese Schrift keiner Beachtung werth hält, so scheint mir dies Urtheil im Allgemeinen zu hart, da wir in derselben die gewöhnlichen Krankheitserscheinungen nicht allein richtig aufgefasst finden, sondern auch die charakteristischen von den auch bei andern Krankheitszuständen vorkommenden geschieden sind.

Unter den Symptomen finden wir varicöse Ausdehnung der Gefässe unter der Zunge und an der Unterlippe erwähnt.

1554. J. Langius. Epistol. miscellan. var. etc. Basil. 1554. 1560. Hanau 1605. L. II. de scorbuto.

L. sucht den Beweis zu führen: dass der Scorbut von den Alten nicht gekannnt worden ist.

1564. B. Ronseus. De magn. Hipocrat. lien. Plinique stomacace ac scelotyrbe, s. de vulgo dicto scorbuto. Antwerp. 1564. Witteberg. 1585. Echtio, Wiero et Langio 1624. 1654.

R. behauptet, dass der Scorbut den Alten bekannt gewesen sei. Als Ursache des Uebels in Holland betrachtet er den häufigen Genuss von fetten Seevögeln u. s. w., vorzüglich aber die feuchte Luft.

Die Beschreibung des Uebels ist ziemlich kurz ausgefallen. Das Wesen sucht er in einer Krankheit der Milz, daher er auch Aderlässe anräth mit eröffnenden und verdünnenden Abkochungen antiscorbutischer Kräuter, mit einem Zusatz von Fol. Sennae und andern purgirenden Substanzen. Als er aber später die Erfahrung gemacht hatte: dass die einfachsten Zusammensetzungen die heilbringendsten waren, so glaubt er Cochlearia und Absynthium als hinreichend zur Beseitigung des Uebels aufführen zu dürfen. Zum Beschluss der Kur räth er eine gelinde Abführung zu geben und untersagt dabei die Anwendung aller heftigen scharfen Mittel, bis sie der Kranke verträgt. In Bezug auf Diät empfiehlt er leichte Nahrung, Brod, Wein; gesunde Wohnung etc. Als Prophylacticum soll man im Herbst eine leichte Purgans verordnen. Auch erwähnt er Scorbutepidemien in den Jahren 1556 u. 1562.

1567. J. Wyerus. Observat. rar. Lib. I. de scorbut. Basil. 1567. Amstelod. 1657.

W. sucht die nächste Ursache des Scorbuts in kranken Eingeweiden und in verdorbenem Blute; die entfernten Ursachen dagegen: in schlechter, verdorbener Nahrung, in vorhergegangenen Fiebern und Kummer. Er bezweifelt zwar die Ansteckbarkeit im Allgemeinen, nimmt sie aber doch auch da an, wo das Zahnfleisch bedeutend ergriffen ist. Die

Krankheitserscheinungen sind von W. genau beschrieben und bis Eugalen wurden sie von den verschiedenen Schriftstellern über den Scorbut nur abgeschrieben. Ebenso findet man ein weitläufiges Verzeichniss der Antiscorbutica. Aderlässe untersagt er, sobald das Uebel vorgeschritten ist. Abführungen und Diaphoretica empfiehlt er mit darauf folgenden antiscorbutischen vegetabilischen Mitteln, verbunden mit Milch, Wein und Bier.

1580. J. Wyerus Arzneib. von etlichen besond. etc. Krankh. Scharboch etc. Frankf. 1580. 1583. Leip. 1693. 1696.

1581. R. Dodonaeus. Medicinal. observ. exempl. rar. C. XXIII. de scorbut. Colon. 1581. Hardenov. 1584. Antwerp. 1585. Hardenov. 1621.

Als vorzügliches ursächliches Moment betrachtet D. schlechte Nahrung. Aderlässe wandte er nur ein Mal bei einem Vollblütigen an. Zur Beseitigung benutzte er antiscorbutische Vegetabilien, verbunden mit einer nährenden Diät. Beim Beginn des Uebels gebrauchte er zuweilen Abführungen, erwähnt aber dabei: dass man mit dem Gebrauch im spätern Verlauf des Uebels vorsichtig sein müsse.

1588. S. Eugalenus. De morb. scorb. lib c. observ. quibusd. breviq. et succinct. cujusq. curat. indicat. Brun. 1588. Lips. 1604. Jenae 1624. Ed. Stubendorph et Brendel. Jen. 1634. Haag 1658. Amstelod. 1720.

E. beobachtete Zahnfleischaffectionen und die localen an den Schenkeln nur sehr selten, und es scheint: als wären in den von E. beobachteten Fällen, die Brustorgane die vorzüglich ergriffenen Theile gewesen. Als Symptom führt er überhaupt auf: faules Zahnfleisch; Purpura et Petech. scorbuticae; bösartige Geschwüre; Brand; schweres Athemholen; Erbrechen, Würgen, Durchfall, Ruhr, Fieber, Ohnmachten, Schmerzen in fast allen Körpertheilen; harte Beulen am Schaamberge; Schwäche, Beugung

und Lähmung der Schenkel; Hemiplegie, Schwäche des Nervensystems, Zuckungen, Epilepsie, Kolik, Apoplexie; Podagra, Hydrops; Atrophie; Caries dentium; Erisipelas; pestartige Fieber und Beulen. Carus, tiefer Schlaf; Entkräftung, Salivation und colliqualiver Schweiss. Beigefügt sind 72 Krankheitsgeschichten, wodurch E. die angeführten Erscheinungen, als dem Scorbut anheim fallend, zu beweisen sucht.

1589. B. Bruner. Tractat. duo. Rostoch 1589. Jen. 1624. Haag 1658. Lips. 1622. Amstelod. 1720.

B. hat im Allgemeinen Wyerus abgeschrieben. B. bemerkt, dass den übrigen Erscheinungen des Scorbuts zuweilen heftige Schmerzen in den Fuss- und Kniegelenken so wie im Ober- und Unterschenkel vorhergehn. Ferner: dass zu seiner Zeit, als Weintrinken allgemeiner geworden war, der Scorbut seltener wurde. Als Ursachen betrachtet er: schlechte Kost, feuchte Witterung; Sorgen; Nachtwachen und Unterdrückung der natürlichen Absonderungen. Als vorzügliches Prophylacticum rühmt er den Senf und Molken mit Succ. nasturt. aquat. bereitet.

H. Brucaeus. De scorbut. proposit., de quibusd. publ. disputat. est. Rostoch 1589. Jen. 1623.

B. sucht den Grund: dass das Uebel einigen Ländern eigenthümlich ist, in dem häufigen Genuss gesalzenen und geräucherten Fleisches; in der Lage und der feuchten kalten Witterung.

Die Erscheinungen des Scorbuts sind nach Wyerus abgefasst. Die nächste Ursache soll in einer Verstopfung der Leber und Milz liegen. Als Complicationen führt er: Wassersucht, Schwindsucht, Durchfälle und Wechselfieber an. In diätetischer Hinsicht rühmt er: frische animalische und vegetabilische Kost, Wein und Bier; in therapeutischer: scharfe und aromatisch-bittere Mittel.

1591. H. a Bra. Epistol. de novo morbo Frisiis et Westphal. peculiari de Varen in P. Foresti. L. XIX. Leid. 1591.

B. führt als Symptome herumschweifende Schmerzen durch alle Glieder, vorzüglich aber im Rücken und der Lumbalgegend mit Stuhlverstopfung an.

E. Hettenbach. Diss. de scorbuto. Witteberg 1591.

1593. S. Albertus. Scorbut. histor. cui in observat. v. saltem indicat. sympt. genar. coarctat. Witteb. 1593. Plat. 1594. In collet. Witteb. 1624.

A. scheint das Uebel nur aus wenigen eigenen Beobachtungen gekannt zu haben. Unter den Symptomen erwähnt er zuerst der Steifigkeit und Unbeweglichkeit der untern Kinnlade. Erblichkeit und Ansteckung nimmt er an. Als diätetische Mittel rühmt er: saure herbe Früchte, Essig- und Weinsuppen und Bewegung; als therapeutische, bei Vollblütigkeit Aderlässe; gelinde Abführungen, (Purgantia drastica verwirft er als schädlich) und antiscorbutische Vegetabilien (um die dicken Säfte aufzulösen). Die Krisen werden nach A. entweder durch die Nieren, oder die Haut vermittelt, je nachdem die eine, oder die andere Richtung obwaltet, muss jedoch durch passende Mittel unterstützt werden.

1595. P. Forestus. L. XX. de lienis morb. et de scorb. nov. mor. Leid. 1595. Tr. 1611.

F. hat die Beschreibung der Krankheitserscheinungen dem Echtius entnommen, allein er erläutert sie durch Krankheitsgeschichten. Die nächste Ursache sucht er in einer Krankheit der Milz. Der Syrupus Forestii (Vid. Pharm. antiscorbut) diente lange Zeit in Flandern, Brabant und Holland als Heilmittel wider den Scorbut. Nach Wechselfiebern beobachtete er häufig das Auftreten des Scorbuts. F. widerstreitet auch der Annahme: dass den Alten das Uebel bekannt gewesen sei.

1600. H. Reusner. Diexodicar. excercitat. sive de scorbut. Fkf. 1600.

Diese Schrift ist blos in Bezug auf die aufgeworfene Theorie bemerkenswerth, der denn auch die Medicamente angepasst sind, ohne dass eben dabei die Erfahrung wesentlich berücksichtigt worden wäre.

1606. M. Backmeister. Disputat. de scorbuto. Rostoch 1606.

1608. J. Coler. De scorbut. et hypochondr. affection. Basil. 1608.

J. Wancker. De scorbuto. Basil 1608.

1609. G. Horst. Tractat. de scorbut. s. magn. Hippocratis lienibus Pliniique stomacace et scelotyrb. Giess. 1609.

Ausser vielen Widersprüchen, findet man nur das bereits bei Eugalenus und Forest angeführte.

1614. J. Albinus. Praecidanea de scorbuto. Basil 1614.

1616. R. Dodonaeus. Prax. med. L. II. C. 62. Amstelod. 1616.

Ueber den Verlauf des Uebels in Middelburg während der Belagerung im Jahre 1555.

R. Dodonaeus. Histor. stirp. p. IV. L. V. c. XVI. Antwerp. 1616.

Diese Schrift enthält nächst der Beschreibung des Uebels die Anempfehlung der Vegetabilien als Heilmittel.

1618. G. Hambergerus. De stomacace et scelotyrbe vulgo nuncupat. scorbut. L. 1618.

F. Kest. De scorbut. Basil 1618.

J. Wolf. De scorbut. Helmst. 1618.

1620. D. Sennert. Tract. de scorbut. Witteb. 1620. 1624. Jen. 1661, cui access. ejusd. argument. tract. epistol. Balduini, Ronsei, Echtii, Wyeri Witteb. 1654. C. M. Martini ejusd. argument. libell. Jen. 1624.

Diese Schrift enthält im Wesentlichen das, was bereits von Eugalen angeführt wurde.

1621. A. Dreyer. Disputat. de scorb. Basil. 1621.

1623. J. H. Meibom. Diss. de scorb Helmst. 1623.

J. Stubendorph. Diss. de scorb. Lips. v. Jen. 1623. Ed. Haller. Jen. 1624.

Enthält das bereits bei Eugalen Angeführte und die Behandlung nach S. Albertus.

1624. A. Falconet. Du scorbut. Lyon. 1624.

J. Langius. De scorbut. epistol. duae Frankf. 1624. Lind. 1654.

M. Martini de scorbut. commentat. Witteb. 1624.

Abschreiber des Eugalen. Erwähnt der Blindheit, Geschwulst der Augen als Symptom und der Wechselfieber als Complication.

D. Sennertus. Tractat de scorbut. ejusd. pract. med. L. III. p. 5. 1624. Witteb. 1631. 1648 Paris. 1632. 1662.

J. Wyerus. Fragment. de scorbut. cura Sennerti, in Witteb. collect. edit. 1624.

1626. A. Weikard. Thesaur. pharm. Galenchem. v. tract. pract. L. III. c. s. de stomacace sive scorbut. 1626.

Diese Schrift ist Compilation des bis dahin über den Scorbut Bekannten.

1627. J. Fabricius. De scorbut. diagnos. et therap. Rostoch. 1627.

J. Zeidlerus. Diss. de scorbut. Lips. 1627.

F. van der Mye. De morb. et sympt. popular. Bredan. etc. Antwerp. 1627. Ed. Gruner's Biblioth. d. alt. A. Leip. 1780—1782.

Auch M. erwähnt der deprimirenden Gemüthsaffecte als ursächliche Momente und dass das Uebel durch die Jahreszeiten und die Nahrung modificirt werde. Als selten vorkommende Complication erwähnt er die Fieber, dagegen häufig beobachtete er die mit der Ruhr, bei welcher nur wenige genasen und auch dann cachectisch oder wassersüchtig wurden. Den Tod sah er meist unter colliquativen Durchfällen erfolgen. Die Geschichte des Scorbuts

in Breda während der Belagerung ist ausführlich erzählt.

1629. G. Jourdouyn, A. G. Guerin. An scorbut. victus aerisque mutat. Paris 1629.

1633. J. Hartmann. Prax. hymiatr. P. 345 de scorbut. Genev. 1633.

H. spricht zuerst von den Nachtheilen des Mercurgebrauchs im Scorbut. Als Heilmittel rühmt er Tartarus vitriolatus und Spiritus vini tartarisatus.

J. Rötenbeck et A. C. Horn. Specul. scorbut. oder Beschr. d. Scharbocks in zwei Tractätlein abgefasst etc. Nürnberg 1633.

Beschreibung des Scorbut im Schwedischen Heere, während der Belagerung von Nürnberg im Jahre 1631.

1634. Z Brendelius. De scorbut. Jen. 1634.

J. Placcius. De scorbut. Jen. 1634

1636. A. Rhodi. Diss de scorbut. Hafn 1635.

1636. Ch. Tinctorius. De scorb. Prussiae jam frequent. Regiomont 1636.

1639. T. Citasius. Opuscul med. P. 168. Paris 1639.

C. hält den Scorbut für eine Krankheit der neuern Zeit.

G. Clauder. N. C. D. II. an 7. Obs. 164

Beobachtungen von Risus Sardonii während des Verlaufes des Scorbuts entstanden

Ch. Sanctorius. Diss. de scorb. in Pruss. jam. frequent. Regiomont. 1639.

1640 M. Banzerus. De scorb. mult. morb. sarragine. W. 1640. 48 1652.

L. Riverus. Prax med. L. XII. C. 6. de scorbut. affect 1640.

R. sucht das Wesen in einer bösartigen Hypochondrie und meint, dass beim Scorbut die Glandulae meseraicae häufig mit ergriffen wären. Uebrigens scheint er nicht aus eigenen Beobachtungen geschöpft zu haben.

1642. J. van Beverwyck. Van de blaauw Schuyt. Dordr. 1642.

1643. J. H. Arcularius. Diss. de scorbut. Argent. 1643

1644 H. Conringius. Diss de scorbut. Helmst. 1644. 1659. 1672.

J. Haberstro. Diss. de scorbut. Jen. 1644

G. Moebius. Diss de scorbut. Jen. 1644. 1662.

1645. Consilium medic. facultat. Hafniens de scorbut. Hafn. 1645 in Th. Bartholini cista medic. Hanf. etc. Hafn. 1661.

Handelt von den endemischen Charakteren des Scorbuts in Dänemark und in andern nördlichen Ländern. Als nächste Ursache wird eine schwarzgallige verdorbene Beschaffenheit der Säfte in Folge von schlechter Nahrung angegeben, wodurch die Thätigkeit des Magens und die Blutbereitung geschwächt werde. Als entfernte Ursache giebt es an: kalte feuchte Luft; schlechte Nahrung; wenig Körperbewegung; Krankheiten (z. B. Infarcten der Leber und Milz und Wechselfieber); Ansteckung und Erblichkeit. Der angeerbte Scorbut soll unheilbar sein. Ebenso soll er verderblicher für Alte, als für Junge sein. Zur Vorbeugung werden gerühmt: trockene Wohnungen, aromatische Räucherungen in denselben; leicht nährende Kost, Wermuthwein, Körperbewegung und Offenhalten des Leibes, so wie auch der Gebrauch von Abführungen im Herbst und Winter. Als therapeutische Mittel empfiehlt man: den Syrupus Forestii, die Aqua antiscorbutica und den Succus cochleariae mit Wein.

1646. H. Botter. Tractat. de scorcut. Lubec. 1646.

1647. V. H. Vogler. Diss. de scorb. Helmst. 1647.

G. Drawitz. Bericht und Unterricht von d. Krankh. des Schmerzen machenden Scharbocks. Latein. 1647. A. d. L. Leipz. 1658. 4-te Aufl. von J. Michaelis. Leipzig. 1704.

Enthält die Beschreibung einiger D. eigenthümlichen Medicamente, ist aber ausserdem ohne besondern Werth.

1688. St. Blandcard. Nauwkeurige Verhandlinge van de Scheuerbuk. 1648.

St. Blancard. Prax. med. C. XV. de scorbut.

Die Gährungstheorie nach Cartesianischen Grundsätzen auf den Scorbut angewandt.

W. Rolfinkius. Diss. de scorbut. Jen. 1648.

1649. F. Sourez-Feyo. Tratado de scorbuto a qne a vulgé chamamal de soneda. Lisboa 1649.

J. Michaelis. Diss. de varis, arthritide vag. scorbut. Lips. 1649.

J. Schmidt. Stud. med. Montpil. 1649. D. 1. an. 4. 5. obs. 155. a scorbut. acromia dysuria.

1651. G. Charleton. De scorbut. libel. singular. Acced. epiphomena in medicastr. London. 1651. 1672.

Ch. beschreibt den Scorbut nach Eugalen, Sennert und Willis und nimmt drei Arten an: 1) den brenzlichen, vom Ueberschuss des Schwefels im Blute; 2) den salzigen vom Ueberschuss erd-salziger Theile und 3) den sauren, von einer Säure im Blute. Als Heilmittel rühmt er Aderlässe, Abführungen, harntreibende Mittel und eisenhaltige Mineralwasser bei Kranken mit hitzigem Temperament, zuletzt Analeptica.

1652. St. H. Cravelius. Diss. de scorbut. Jen. 1652.

Ch. Henningius. Diss. de scorbut. Arg. 1651 oder 1652.

1653. A. Mengering. Diss. de atrophia scorbutica. Lips. 1653.

1658. J. A. Grabe. Cas. labor. affect. hypochondr. c. sympt. scorb. Giess. 1658.

1660. S. Pauli. De var. caus. febr. Coppenh. 1660. Im Anhang.

1661. J. A. Grabe. Kurzer Unterricht vom Scharbock. Erfurt. 1661.

A. Guyot et C. Brisset. Estne scorbut. ab aquar. vitio. Paris. 1661.

C. Gemma. Cosmocrit. L. II. C. 2.

1662. B. Timaeus. Opera med. pract. 1662. T. I. p. 3. 7. 15. T. III. 24. 32. 35. 36. T. IV. 15. TVIII. 15. 18. Lib. 3. epist. 10—12. 20. 28. L. V. 9. Unzuverlässige Beobachtungen.

1663. V. A. Möllenbrock. De var. s. arthr. vag. scorb. Lips. 1663. 1672.

Nach M. soll das Uebel alle Menschen heimsuchen. Man findet nur eine Art Scorbut beschrieben, dagegen recht viele Irrthümer auf eine pomphafte Weise erzählt.

G. B. Metzger. Diss. de scorbut. Tubing. 1663.

L. Ursinus. (Beer) Diss. de scorbut. Lips. 1663.

J. Schuter. D. de scorbut. Leid. 1663.

1664. J. Th. Schenkius. D. de scorbut. Jen. 1664.

1665. Barbette. Praxis. Amstelod. 1665. 1669. Roterd. 1665. Cum mult. notis ed. F. Dicker. Leid. 1669. Amstelod. 1678. 1702. Patav. 1676. Venet. 1732. A. d. L. Frankf. 1693. Lübeck 1700. 1718. Engl. 1718.

B. hat die Beschreibung nach Eugalen abgefasst. Bei der Behandlung verwirft er Aderlässe und starke Abführungen und rühmt Murias ammon., Spir. sal. dulc. und Hb. cochlear.

Th. Bardolin. De medicin. Danorum domestic. diss. X c. vindic. et addidament. Hafn. 1665. Fuse de scorbut.

Nach B. behandelten Franzosen und Italiener den Scorbut unrichtig, obgleich er in Arles oft vorkam. In Norwegen gebrauchte man die Multbeere zu B. Zeiten, noch früher Radix angelicae zur Beseitigung des Scorbuts. B. räth bei Complication mit anderen Krankheiten, zunächst den Scorbut zu beseitigen.

D. Westelier. De scorbut. Leid. 1665.

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D. Westelier. De scorbut. Leid. 1665.

1666. F. Plater. Prax med. L. III. C. 4. Basil. 1666.

P. gehört auch zu den Abschreibern des Eugalen. Nach P. soll der Scorbut, so wie die Lues durch Seefahrer uns zugeführt worden sein. Unter den Symptomen zählt er Beulen, bald schmerzhaft, bald unschmerzhaft auf, die ihren Sitz in drüsigen Theilen, oder zwischen den Muskeln haben. Nach der Beschreibung derselben zu urtheilen, waren diese Beulen: theils Drüsenanschwellungen, theils Furunkeln oder solche Ablagerungen, wie sich bei Neigung zur Geschwürsbildung finden. Als Heilmittel rühmt er den Senf mit Honig und Succus Aurantior.

J. N. Pechlin. E. N. C. D. 1. an. 9. 10.

Bei einer scorbutischen Frau, die an Zahnschmerzen litt, wurden Würmer, die den Läusen (?) nicht unähnlich waren, ausgezogen.

S. R. Sulzberger. Diss. de scorbut. Liss. 1666.

M. Lyser. De scorbut. Lips. 1666.

1667. E. Maynwaring. The cur. of Scurvy. Lond. 1667.

Als Ursachen führt er den Gebrauch des Tabacks und die übermässige Befriedigung des Beischlafs an.

H. Ch. Alberti de essere scorbuti Erf. 1667. v. 1692.

J. Schmidt's Pest etc. u. Scharbock Augsb. 1667. 1692. 1702.

Th. Willis. Tract. de scorbut. 1667.

W. weicht zwar bei der Beschreibung des Scorbuts von den frühern Schriftstellern ab; sie ist jedoch bei alle dem ganz falsch. Nur tadelt er mit Recht: dass man die Formen des Scorbuts ohne Recht vervielfältigt habe. Als Ursache führt er ungesunde Luft, Entartung der Blutmasse und des Nervensaftes an. Die erstere sei bald schwefelicht-salzig,

bald salzig-schwefelicht. Bei der erstern seien Aderlässe und kühlende Mittel, bei der letztern Irritantia und flüchtige Salze enthaltende Mittel angezeigt.

1668. E. Maynwaring. Util. obs. fact. in quibusd. scorbut. curat. Lond. 1668.

F. Albinus. D. de scorbut. Leid. 1668.

J. D. Major. Progr. lect. de scorbut. privat. praemiss. Kiel. 1668.

H. Meibom. De arthr. vag. scorbut. Helmst. 1668.

W. Rolfinkius. Diss. de scorbut. Jen. 1668.

G. Rolfinkius. Diss. de scorbut. Jen. 1668.

1669 R. Lower. Bromograph. oder Haberkur. Amsterd. 1669. von J. Frank. Ulm. 1715. 1722. Strassb. 1754. Schwed. v. J. Ch. Nordenheim. Stockh. 1724.

Ueber den Nutzen des Habertranks von Lower im Scorbut.

1670. G. Frank. D. de scorbut. Heidelb. 1670.

1671. O. Borichius. D. de scorbut. Hafn. 1671.

O. Borichius. Act. Hafn. V. I. obs. 134. V. II. an. 4. obs. 72. an. 5. obs. 87.

Ueber den Nutzen des Spiritus antiscorbut. von Dr. Protten.

H. Stubbes. Epist. discours concern. phlebotomy in oppos. of G. Thomson at the effects of blood letting the etc. scurvy etc. Lond. 1671.

J. H. Schmidt. Discurs. med. de cachex. scorbut. Placent. 1671.

J. Thevart. An ex aeris et diaet. vit. scorbut. Paris. 1671.

N. Vernette. Tract. du Scorbut. ou mal de ter. et de tout. l. malad. qui arriv. s. la mer. Rochelle. 1671.

V. giebt eine Geschichte des Scorbuts. Nach V. soll durch Anhäufung von Scorbutischen Infec-

tion der Luft erfolgen und in kälteren Jahren das Uebel epidemisch werden. Krankheiten und Alles was die Kräfte aufreibt, wirkt als prädisponirendes Moment. Der Sitz des Uebels ist im Blute. Nach V. Beobachtungen fand sich die Milz nur selten (8:500) ergriffen, dagegen das Pancreas, Omentum und Mesenterium häufig zerstört. Auch V. nimmt einen heissen und einen kalten Scorbut an. Unter den Symptomen führt er Erbrechen und Durchfall an, durch welche blutabsondernde Geschwülste ausgeschieden werden sollen. Als prophylactische Mittel werden Reis, Wein, Scherbet und Körperbewegung gerühmt. Während der Belagerung von Rochelle leistete der Senf bei den Scorbutischen gute Dienste. Als Heilmittel finden wir aufgezählt: Ptisanen, Rad. armorac, Arum, Nasturt. aquat. etc. Gegen Geschwüre im Munde Decoct. Tamar. mit Alumen. Bei angeschwollenem Zahnfleisch machte er Scarificationen und liess Decoct. Aristoloch. und Gentian. als Mundwasser brauchen. Gegen Durchfälle rühmt er Rheum, Myrob. und bei der Complication mit Rheumatismus Theriac, Diascord. und Opium zu gebrauchen.

S. Wirdig. Diss. de scorbut. Rostoch. 1671.

1672. R. W. Krause. Diss. de scorbut. Jen. 1672.

J. A. Fridericus. Diss. de laesion. oris scorbut. Jen. 1672.

M. Friebe. E. N. C. D. III. an. 4. obs. 91.

F. meint eine scorbutische (?) Affection sei durch fliessende Hämorrhoiden beseitigt worden.

1673. J. F. v. Cappeln. De scorbutic. sanguin. intemperie D. Leid. 1673.

M. Sennert. D. de scorbut. Witteb. 1673.

J. Schouten. D. de scorbut. Leid. 1673.

1674. V. A. Möllenbrock. Cochlear. curios. Lips. 1674.

M. erwähnt das häufige Vorkommen des Scor-

buts in Westphalen. Die nächste Ursache sei ein scharfflüchtiges Salz der Säfte; die entfernte: unzweckmässige Lebensart. Auch neigt er sich zu denjenigen, welche den Scorbut für ansteckend hielten. Heilmittel: zunächst Purgantia und Diuretica; dann Antiscorbutica z. B. Hb. cochlear., Oleum de pinn., Martialia.

O. Worm. D. de scorbut. Hafn. 1674.

Ueber den Gebrauch der Multbeere bei den Norwegern als Antiscorbuticum.

A. B. Angli. D. de scorbut. L. B. 1674.

Ph. Haechstetter. Obs. rar. med. D. VII. 10. Lips. 1674.

J. J. Waldschmidt. De scorbut. in astrolog. med. Ann. non addit.

G. W. Wedelius. De arthr. vag. scorbut.

1675. H. Cellarius. Bericht von Scharbock. Halberst. 1675.

C. nimmt einen heissen und einen kalten Scorbut an. Unter den Eingeweiden leidet am häufigsten die Milz mit; dann das Pancreas, deren Saft widernatürlich scharf wird.

G. Harrey. Diseas. of Lond. or a discovery of scurvy. Lond. 1675.

Das Uebel in London. H. theilt den Scorbut in zwei Classen: den Mundscorbut, entstanden in Folge von Säure; und in den Scorbut der Beine, in Folge einer seifenartigen Beschaffenheit der Säfte entstanden.

J. D. Muller. D. de cruentat. gingivar. scorbut. Altdorf. 1675.

G. Stein. Diss. de cardialgia scorbut. Altdorf. 1675.

G. Stein. D. de paralys. scorbut. Altdorf. 1675.

J. M. Sulzer. D. de scorbut. Hafn. 1675.

1677. F. Clamette. Prax. med. Riverian.

non absimil. T. II. Montpell. 1677. Lyon. 1704. 1714. Engl. Lond. 1706.

Im 2-ten B. spricht C. über den ranzigen und sauren Scorbut.

A. Lamb. D. de scorbut. Leid. 1677.

1678. J. Zipfel. Scharbock etc. Dresden 1678.

1679. C. Patin. Orat. de scorbut. Patav. 1679.

P. Pena. Advers. stirp. p. 121. 122.

E. Renaudot. Conferenc. publiq. ou quest. academiq. sur l. sc. par le plus beaux espr. de ce temps. Paris. 1679. Scorbut. T. V.

F. D. Sylvius. Opera omnia med. Amstelod. 1679.

Enthalten wenig über den Scorbut. Als Heilmittel findet man empfohlen: Hb. Cochlear. Sem. Sinapeos, Succ. Aurant. Spir. nitr. dulc. u. Spir. sal. dulc.

M. Ziervogel. D. de scorb. Leid. 1679.

E. Maynwaring. Treatis. on the scurvy. Lond. 1679. 1085.

1680. A. Curtius. D. de scorbut. Marburg. 1680.

B. Fischer. D. de scorbut. Leid. 1680.

1681. P. Ammann. De stomacace sive scorb. oris. Lips. 1681.

E. Leichner. D. de scorbut. Erf. 1681.

A. Muntingi. De ver. antiquor. herb. Britannic., ejusd. efficat. contra stomacac. s. scelotyrb. Frisiis et Batav. de Scheuerbuyck. D. hist. med. 1681.

M. rühmt Rumex aquaticum, die Hr. Britanica der Alten, als bestes Heilmittel.

1682. G. F. Hildanus. Observ. et curat. chir. Obs. V. C. 5. Fkft. 1682.

Zwei Krankengeschichten.

1683. Cameau. Trait. curieux du scorbut, de ses sympt. et demonstr. de la ferment. ou mouvement.

intestin. d. corps s. l. princip. d. Decrates. Lond. 1683.

A. H. Fasch. D. de arth. vag. scorbut. Jen. 1683.

L. Chameau. Trait. du scorbut. Paris 1683.

Ueber das endemische Auftreten des Scorbuts in England. Das Wesen soll in einer contagiösen Auflösung des Bluts liegen und als Heilmittel rühmt er die Milch.

M. Dellon. Un voyage aux Indes orient. M. D. Suppl. ch. 2. 1683.

Nach D. ist der Scorbut auf langen Seereisen die gefährlichste Erscheinung, da er nicht allein ansteckend, sondern auch zur See nicht zu heilen sei. Das Blut des Meerschweins soll specifische Kräfte gegen den Scorbut besitzen.

1684. J. Dolaeus. Med. theor. pract. encyclopaed. L. III. C. 12. 1684.

Unsinnige Behauptung über den Calomel, als Heilmittel wider den Scorbut. Das Wesen sei eine saure Schärfe und daher sei er auch mit der Hypochondrie verwandt.

Cameau. Moyens praeservat. et method. p. l. guerison du scorbut. Lyon. 1684.

1685. J. Broen. De duplic. bile veter. Leid. 1685.

B. führt als Symptome des Scorbuts Hydrops und Pleuritis auf.

A. Hochmuth. Diss. de purpur. epidem. scorb. Witteb. 1685.

Geschichte des Scorbuts. Ursächliche Momente: der Genuss verdorbenen Getraides.

G. Harvey. Of the small pox etc. a diseas. of the scurvy. Lond. 1685.

Th. Sydenham. Opera universal. Lond. 1685.

Enthält wenig über den Scorbut.

J. Thiele (?) De purpura epidemica scorbutica. Viteberg. 1685.

Enthält die Geschichte der Krankheit, welche aus dem Genusse des Mutterkornes entstanden war.

1687. G. W. Wedelius. Diss. de scorbut. Jen. 1687.

B. Boni. D. de scorbut.: Journ. des Savans 1687.

1688. G. W. Wedelius. D. de colica scorbut. Jen. 1688.

G. Wölffel. D. de febre scorbut. Erf. 1688.

J. Vesti. D. de scorbut. Erf. 1688.

1690. R. Huntley. D. de scorbut. Traject. 1690.

1691. J. F. Decker. D. de arthr. vag. scorb. Leid. 1691.

G. Budens. D. de scorbut. Leid. 1691.

1694. M. Lister. Sex exercitat. de quibusd. morb. chron. E. V. de scorb. Lond. 1694.

Nach L. soll der Scorbut einzig in Flandern geherrscht haben und sich nur erst dann überall ausgebreitet haben, als die Engländer Reisen nach Indien ausführten. Zur Zeit L. suchte das Uebel die Seeleute aller Nationen häufig heim. Durch die Salztheile der Seeluft soll eine salzige Entartung der Säfte und so der Scorbut bewirkt werden. Cochlear., Succ. citr., säuerliche Früchte und Kräuter, Acetum und Spirit. vitrioli rühmt er als Heilmittel.

Leigh. Phthisiolog. Lancastrens. Lond. 1694. Genev. 1736.

Ueber phthisis scorbutica.

S. Sedel. E. N. C. D. II. an. 2. obs. 34.

Beobachtung eines blutigen Schweisses, der durch Körperbewegung hervorgerufen wurde.

S. Ch. Chomberg. D. de paralys. scorbut. Erf. 1694.

1695. Ph. J. Rumpel. Diss. de tabe scorbut. Utrecht. 1695.

1696. W. Cockbrun. Seadiseas., or a treat. of the nat., caus. and cur. etc. Lond. 1696. 1706. Leid. 1701. A. d. E. Rostock. 1726.

Enthält die Hypothesen über das Wesen des Scorbuts und belegt durch Beispiele den Nutzen von Gartengewächsen (Kohl, Rüben etc.). Das Wesen sucht er in unvollkommner Verdauung und Mangel an Hautausdünstung.

M. Ettmüller. Colleg. pract. de morb. corp. hum. P. II. Fkf. 1696.

Der Scorbut sei der höchste Grad von Hypochondrie.

M. Friccius. D. de colica scorbut. Ulm. 1691.

A. Pitcarn. Element. med. phys. mathem. L. II. c. 23. de scorb. 1696.

P. macht auf den Nachtheil der Blutentziehungen im Scorbut aufmerksam. Als Heilmittel empfiehlt er Milch und wo sie nicht vertragen wird, Eisen mit tonischen Mitteln. Auch die Transfusion von Thierblut bespricht er.

J. Verbrugge. Examen van Land end Zee Chirurgie de voornaamste Hoofdstukken etc. van aller Ziekten op Ost en Westind. Grönland. Amsterd. 1696. 4-e A. 1714. v. J. D. Schlichting. Amsterd. 1748.

Ueber den Scorbut unter den Holländischen Seeleuten in Grönland, Spitzbergen etc.

J. Colbatch. Physico medical essays concerning alcali and acid in the cure of disempers. Lond. 1696.

Alkalische Beschaffenheit der Säfte sei Ursache des Scorbuts.

J. Floyer. Praeternatural state of animal humors etc. Lond. 1696.

Salzige Entartung der Säfte als Ursache des Scorbuts.

J. Baggaart. Over de Scheurbuyk. Middelb. 1696.

1698. Le Clerc. La chir. complet. etc. Paris 1698. Belg. 1695. 1706. 1793. Hal. 1724.

1699. P. le Jonnellier et A. Lippi. Non est scorbut. aegritud. nova. Par 1699.

1700. G. B. Hoffmann. D. de scorbut. Argent. 1700.

1701. Y. Gaukes. Genes. an heelkonstige van de Scheurbock. Utrecht. 1701.

A. L. Vroling. Tractat. v. Scheurbock. Deventer. 1701.

1702. Th. Balthasar. De sale commune. Altorf. 1702.

Als Heilmittel Säure empfohlen, da der Scorbut Folge von Fäulniss sei.

M. de la Vigne et J. Depyney Peschard. Ergo omni scorbuto volatil. Paris 1702.

1704. J. Ph. Euselius. D. de febre scorbut. exanthem. Erf. 1704.

1705. L. F. Jacobi. D. de scorbut. haereditar. Erf. 1705.

N. Buchner. D. de scorbut. Daniae endemico. Leid. 1705.

1706. St. Blancard. De scorbut. et luis vener. divers. sign. et med. Lips. 1706.

G. E. Stahl. D. de scorbut. et vener. luis sign. et med. Halae 1706. in Haller. Diss. pathol. T. VI. No. 197.

1707. S. Roeseler de Rereseer. De scorbut. mediterran. Cibim 1707.

Carrichterus. Prax. German. L. 1. C. 41.

J. Crauford. D. de scorbut. L. B. 1707.

1709. H. Boerhave. Aphorism. de cognosc. et cur. morb. L. B. 1709. T. III. Aphor. 1148. — 1165. Mit Erläuter. v. G. van Swieten. B. III. Abth. 2. S. 389. — 465. Fkft. u Leipz. 1769.

Nach B. war der Scorbut den Alten bekannt und wenn gleich van S. dieser Meinung nicht unbedingt beistimmt, so räumt er ihnen doch eine beschränkte Kenntniss ein. Aph. 1149. v. S. tadelt die grosse Anzahl der Formen. 1150. Die vorzüglichsten Ursachen: nasse Kälte; Mangel; unverdauliche, schwere Nahrung; Trägheit; Melancholie und Missbrauch von China. 1151. Erscheinungen und Complicationen. Nach B. soll sich beim Scorbut schnell Ansteckbarkeit entwickeln, was jedoch v. S. gründlich widerlegt hat. 1153. 1154. Nach B. liegt die nächste Ursache in dicker scharfsalziger, laugenartiger, oder saurer Beschaffenheit des Bluts. 1155—1159. Die Heilanzeigen sind der Ansicht über das Wesen angepasst. 1159. v. S. tadelt das Blutlassen und den Gebrauch von Purganzen. 1160 — 1165. Auch Mercurialia werden verworfen.

J. Frank. Herb. Hallelujah botanice considerat. in corpor. prop. prax. cum nupera febre epidem. Ulmae Obs. an. 1703. 1708. Ulm. 1709.

Khcern. Affectio scorbutica mulieribus Hartbergensibus ante aliquot annos (1709) epidemic. Ephem. N. C. D. I et II. p. 324.

Das hier beschriebene Leiden gehört nicht dem Scorbut an, sondern entspricht mehr einem epidemischen Fieber mit Milzleiden und Stomacace.

1710. G. Magiri. D. de tabe scorbut. Lips. 1710.

G. Magiri. D. de paralys. scorbut. Lips. 1710.

1711. J. R. Deutgen. D. de scorbut. Hardenov. 1711.

Ch. de Geyter. D. de scorbut. L. B. 1711.

J. Bing. De scorbuto nautico 1711 grassato, Hafn. 1712.

1712. J. H. de Heucher. Caution. in cognoscend. curandoq. scorbut. necess. 1712.

Erzählt nur Irrthümer.

J. M. Hoffmann. E. N. C. D. III. an. 2. Osv. 207.

Geschichte einer Paralysis scorbut., durch Decoct. lignor. geheilt, und eine, wo nach heftigen Schmerzen im Arm der Tod apoplectisch erfolgte.

1713. J. G. v. Bergen. D. de scorbut. Francof. 1713.

1714. T. Zwinger. Examen plantar. nasturtiar. Basil. 1714.

Ueber den Nutzen von Nasturt. im Scorbut.

1716. J. Ph. Euselius. De aquilegia scorbut. asylo. Erf 1716.

Ueber den Nutzen des Succ. aquileg. vulgar. recent. als Heilmittel des Scorbuts; allein derselbe ist durch spätere Versuche nicht bestätigt worden.

1717. J. A. Fischer. Diss. de scorbut. ejusd. tum genuenis tum contraversis caus. symptomat. praecipuis et cura. Erf. 1717.

1718. F. Hoffmann. Med. rational. system. Halae 1718. T. IV. C. 1. p. 5.

H. rühmt als Heilmittel das gewöhnliche Wasser, noch mehr aber die natürlichen Mineralwässer. Auch warnt er gegen den Mercurgebrauch.

1719. J. A. Wedelius. D. de cachex. scorbut. Jen. 1719.

1720. M. Albertus. D. de scorbut. praeservand. Halae 1720.

L. Lambert. D. de scorbut. Lugd. B. 1720.

1724. P. Chirac. Obs. sur l'equipag. de vaisseaux. Paris 1724.

1725. J. Bodel. D. de scorb. Lugd. B. 1725.

J. Freind. Hist. of phisick. from the time of Galen. etc. T. II. p. 387. Lond. 1725.

F. zählt den Scorbut zu den neuen Krankheiten.

1726. Ch. M. Burchhard. Progr. de scorbut. septentrional. Rost. 1726

1727. G. Thiesen. D. de morb. marin. Lugd. B 1727.

1729. H. P. Juchius. D. de scorbut. Erf. 1729.

H. P. Juchius. D. de scorbut. summo morbor. et caus. morbific. genere. Erf. 1729.

1730. G. D. Albertus. D. de scorbut. Lugd. B. 1730.

M. Albertus. D. de scorb. Daniae non epidemic. Halae 1730.

1731. P. Duret. D. de scorbut. Lugd. B. 1731.

1732. S. B. Meyer. Scorbut. considerat. med. Giess. 1732.

A. Nitsch. Histor. Scorbut. Wiburgi regnantis. 1732.

1734. J. F. Backström. Observat. circ. scorbut. ejusd. indol., caus., sign. et cur. Leid. 1734. Halleri l. c. No. 195.

B. hält den Mangel an Vegetabilien für die einzige Ursache des Scorbuts, daher er sie auch als Heilmittel rühmt.

D. Sinopaeus. Parerga medica. Petropol. 1734.

Diese Schrift gehört, obgleich nicht eben sehr bekannt, zu den klassischen. Die Beobachtungen über den Scorbut machte S. im Seehospital zu Cronstadt, während der Jahre 1730—1733.

D. Schulz. Hist. scorbut. Wiburg. regnant. communicavit ab A. Nitzsch. Commerc. liter. Norimb. an. 1734. p. 162.

J. A. Wedelius. D. de scorb. Jen. 1734.

1735. Ch. M. Burchhard. D. de scorbut. mar. balthic. acolio non epidem. Rost. 1735.

J. J. Daebel et Waller. D. scorbut. Suecis non esse endemic. Lond. 1735.

J. G. H. Kramer. Medicin. castrens. Norimb. 1735.

Scorbut complicirt mit Febris intermittens unter den Oesterreichischen Truppen in Ungarn und Oberitalien, in den Jahren 1734 und 1735.

1736. J. Schmid. D. de scorb. Prag. 1736.

1737. J. G. H. Kramer. D. epist. de scorb. Norimb. 1737. Haller l. c. No. 197.

K. liefert eine Beschreibung des Scorbuts unter den Oesterreichischen Truppen in Ungarn. Er läug-

net die Ansteckung und verwirft die vielen Zufälle, welche fälschlich dem Scorbut zugeschrieben wurden. Im Allgemeinen giebt er die Erscheinungen als in allen Fällen gleich an. Sumpfluft; Fieber; schlechte, schwere Nahrung waren die ursächlichen Momente. K. bestimmt das Frühjahr als diejenige Jahreszeit, in welcher der Scorbut auftritt; allein er irrt in der Behauptung: dass dies weder im Herbst noch Winter der Fall sei. Als Heilmittel erwähnt er: antiscorbutische Vegetabilien, flüchtige, harzig-ätherische und Eisenmittel; bemerkt jedoch dabei: dass die China sich am wirksamsten bewiesen habe.

1738. F. Hoffmann. De scorbut. ver. origin. indol. et curat. Halae 1738.

Die Beschreibung ist nach Willis abgefasst.

J. Hummel. De arthr. tam tartar. quam scorb. Buding. 1738.

E. F. Pelgrom. D. de scorb. Lugd. B. 1738.

Petit. Mal. de os. T. II. p. 443.

Ueber exostosis scorbutica.

Pugh. Med. obs. and. inquir. V. II. p. 241. 1738.

Anempfehlung der China.

1743. P. Briscow. Trait. du scorbut. Paris. 1743.

1744. G. Barkeley. A chain of philos. refect. and inquir. concerning the virt. of tar-water. Lond. 1744.

Theerwasser soll für sich allein den Scorbut beseitigen.

G. C. Richter. Progr. in Hippocrat. scorb. antiquitat. Götting. 1744.

Hippocrates soll bereits den Scorbut erwähnt haben.

Reus. in Ejusd. opusc. V. III. p. 110–117.

J. F. Schreiber. Vom Scharbock St. Petersb. (?)

1747. S. P. Hilscher. Progr. de gravi scorb.

symptomate scelotyrbe dicto memorabili casu illustrat. Jen. 1747.

S. P. Hilscher. D. de scorbut. ejusq. remed. Jen. 1747.

A. Nitzsch. Theoret. pract. Abhandl. des Scharbocks. St. Petersb. 1747.

N. tadelt die Schriftsteller, welche den Begriff von Scorbut zu weit ausgedehnt, aber auch ebenso die, welche ihn zu sehr beschränkt haben. Als Grund für das seltnere Auftreten an manchen Orten führt er mit Hoffmann den häufig gewordenen Thee- und Kaffegebrauch an und glaubt: dass an die Stelle des Scorbuts das Friesel getreten sei. N. läugnet den Einfluss des gesalzenen Fleisches, der feuchten Seeluft, des Mangels an Vegetabilien zur Erzeugung des Scorbuts und nimmt verschiedene für den heissen und kalten an. Für die erstere Art: Körperschwäche in Folge von Anstrengungen und Krankheiten, deprimirende Gemüthsaffecte, feuchte heisse, oder kalte, oder mit animalischen Ausdünstungen geschwängerte Luft, die vereinigt das Uebel ungemein steigern können. Die nächste Ursache sucht er in einer Cacochylia putridinosa vappida in primis viis mit zäher fauliger Beschaffenheit des Blutes. Die Ursache der zweiten, weit seltener vorkommenden Art, soll in einer laugenartigen Schärfe und Auflösung des Blutes bestehen und immer von Fieber begleitet sein.

N. hat seine Beobachtungen nicht zur See, sondern nur bei Landarmeen gemacht.

Beim kalten Scorbut finden wir bei N. 4 Arten aufgezählt.

1) Scorbutus lividus, der blaue Scorbut. In den Jahren 1732 und 1733 zu Wiburg und St. Petersburg beobachtet. Als charakteristische Zeichen werden angegeben: blaue Flecken auf den Beinen, zuweilen auch auf dem Rücken, der Brust, den Au-

genlidern und der Conjunctiva, wodurch die letztere sackförmig hervorgedrängt werde und oft eine chronische Augenentzündung zur Folge habe. Ferner bedeutend geschwollenes, blasses, weiches Zahnfleisch, aus welchem beim Druck blutige oder eiterartige stinkende Materie ausfliesse; angeschwollene Parotis, rheumatische Schmerzen und Fieber.

Betrachten wir nun die aufgezählten Erscheinungen, so ergiebt sich leicht: dass sie entweder einer Stomacace oder Hydrargyrosis febrilis mit scorbutischer Diathese verbunden, aber nicht als eine eigene Species des Scorbuts gelten dürfen.

2) Scorbutus petechialis vel lenticularis. Im Jahre 1732 zu Wiburg und 1737 in den Verschanzungen zu Ust-Samara beobachtet. Die Flecken sollen, gleich den Flohstichen oder Petechien, dunkelroth, nur an der vordern Fläche der untern Extremitäten und über dem Knie und in der Kniekehle als Vibices gefunden werden; und mit der Zunahme derselben die gleichzeitig bestehende Geschwulst und Schmerzen in diesen Theilen sich verstärken und der Puls frequenter werden. Auch sollen sich dazu an der innern Fläche der Wangen, bald harte knollige, bald schwammige Geschwülste gesellen; zuweilen auch Anschwellung und Verhärtung des Musculus temporalis über den Jochbogen, ohne jedoch die Parotis mit zu ergreifen. Das Zahnfleisch weniger weich, als bei der ersten Art, fände sich an seinem obern Theil excoriirt. Auch sei die Salivation und der üble Geruch bedeutender als bei den übrigen Arten. Als nächste Ursache für diese Species soll Zähigkeit der lymphatischen und wässrigen Bestandtheile des Blutes anzusehen sein.

Finden sich nun auch unter den aufgezählten Erscheinungen einzelne dem Scorbut angehörig, so zwingen doch wiederum die Angaben über den Puls, die Mundaffectionen etc. diese Species nicht für

Scorbut, sondern für Hydrargyrosis febrilis zu halten; zu der sich entweder der Scorbut gesellte, oder aber die Hydrargyrosis wurde durch unzweckmässigen Mercurialgebrauch bei scorbutische Diathese herbeigerufen.

3) Scorbutus pallidus, der blasse Scorbut mit zwei Abarten: Scorbutus pallidus mucosus und S. p. muriaticus, wurde von N. in Finnland 1742 und 1743 beobachtet. Dieser soll aus einer Verderbniss des Fettes, oder der öligen Bestandtheile des Blutes hervorgehen. Alle Flecken auf der Haut fehlten, weil die wässrigen Bestandtheile des Blutes nicht zähe würden und die rothen nicht gerönnen. Dagegen finde sich eine bleiche allgemeine Geschwulst ein, die, sobald die übrigen Theile des Blutes brenzlicht würden, in das Gelbe übergehe. Sobald das Fett aber die Härte des Talgs annehme, erschiene die Geschwulst der weichen Theile der Arme und Füsse, die allmälig so hart werde, dass sie dem Druck des Fingers nicht nachgebe und zugleich treten auch wahre Tophi auf den Händen und der Tibia auf. Da aber nun die wässrigen Theile des Blutes weit schneller fade und die salzigen der übrigen salziger und schärfer würden, so fände man auch hier die Wangen stärker geschwollen, die Knie zusammengezogener, das Zahnfleisch weicher u. s. w. Wenn sich das Blutwasser im Zellgewebe anhäufe, so entstehe Hydrops; wäre dies in den Drüsen der Fall, so bilde sich Durchfall aus. Entstehe aber durch Vermischung des Blutwassers mit den ölig-salzigen Theilen eine Schärfe, so erschienen in den verschiedenen Theilen, z. B. den Gelenken der Rippen, am Brustbein, die heftigsten Schmerzen, erstickende Engbrüstigkeit, Brand der Wange, colliquativer Durchfall u. s. w.

Auch bei dieser Art suchen wir vergebens das eigenthümliche Bild des Scorbuts, sondern finden

vielmehr eine Complication mit Wassersucht, Rheumatismus und Gicht.

4) Scorbutus ruber, nur in den Verschanzungen von Ust-Samara beobachtet. Als eigenthümliche Symptome dieser nur ein Mal beobachteten Form, sind: grosse Schwäche, rothe Flecke über den ganzen Körper, aufgedunsene herabhängende Wangen, schwammiges, faules, stinkendes Zahnfleisch und im Kniegelenk zusammengezogene Extremitäten aufgezählt.

Diese Art entspricht am meisten dem zweiten Grad des Scorbuts.

Scorbutus calidus, der hitzige Scorbut. Wurde von N. an verschiedenen Orten, vorzüglich oft aber zu Wiburg und Cobilak beobachtet. Der hitzige Scorbut soll sich durch allgemeine Abmagerung charakterisiren. Das Zahnfleisch soll weder sehr schwammig noch auch übelriechend, sondern schmerzhaft aufgeschwollen, brennend sein, zuweilen so stark, dass Berührung desselben Ohnmacht bewirkte. Die Schmerzen in den verschiedenen Theilen seien wandernde und bewirkten grosse Beklemmung, Colik, Strangurie u. s. w. Das begleitende Fieber sei zwar anhaltend, aber von unregelmässigem Typus. Weder Kniegeschwulst, noch auch Purpura scorbutica kämen vor und als Hauptunterscheidungszeichen habe man den Urin zu betrachten, da derselbe beim kalten Scorbut zwar eine dunklere Farbe habe, sich aber nur wenig verändere, während beim hitzigen Scorbut der Urin sich mit einer Fetthaut bedecke und einen dicken sandigen Bodensatz fallen lasse. Auch komme diese Gattung niemals epidemisch vor und man fände in den Leichen in der Regel scirrhöse Geschwülste der Unterleibsdrüsen, vorzüglich des Pancreas und der Leber.

Auch diese Gattung gehört nicht hierher, sondern vielmehr zu den febrilen Krankheitsformen.

Uebrigens ergiebt sich das Unrichtige dieser Eintheilung von selbst, wird aber noch deutlicher bei Betrachtung des Krankheitsbildes heraustreten.

Im noch übrigen Theile der Schrift spricht N. von den Ursachen, welche während der Belagerung von Asow, 1736; während des Feldzuges am Dniester und bei Chozim, 1739 etc. das Uebel hervorriefen.

Die Behandlung bietet nichts besonderes dar.

1748. G. Anson. A voyage round the world in the years 1740—44. Lond. 1748.

Nächst Belehrendem in Bezug auf Prophylaxis, findet man auch Lesenswerthes in Bezug auf die Erscheinungen des Scorbuts.

H. Ellis. A voyage to Hudsonsbay etc. in the years 1746. 47. Lond. 1748.

E. schreibt dem Missbrauch des Branntweins das Erscheinen des Scorbuts unter seiner Mannschaft zu; allein zuverlässig wirkte die Kälte und der Mangel an frischen Vegetabilien gleichzeitig mit darauf hin. Der Tod erfolgte meist mit colliquativen Durchfällen und Wassersucht bei übrigens gewöhnlichen Erscheinungen des Scorbuts. Die gewöhnlichen Mittel leisteten keinen Nutzen, dagegen soll das Theerwasser sich so heilsam bewiesen haben, dass selbst die Kranken noch im letzten Stadium durch dasselbe gerettet wurden. Die Kriesen erfolgten durch den Harn.

M. Law. D. de scorbut. Edinb. 1748.

Bigot de Morogues. Auserl. Abh. d. d. Acad. zu Paris zugeschickt wurden. A. d. f. v. Beer. T. I. S. 145.

Handelt von der verdorbenen Schiffsluft als Ursache für den Scorbut.

1749. C. Bisset. A treat on the scourvy. Lond. 1749.

Die Schrift ist voll von Irrthümern.

Cadet. D. sur le scorbut. avec obs. Paris. 1749.

R. Mead. On the scurvy in the works of Mead p. 437—50. Lond. 1749.

M. beschreibt das Uebel genau; betrachtet feuchte Luft, Salztheile, schlechte Nahrung als Hauptursachen und Vegetabilien, Milch und Landluft als Heilmittel.

1750. Ch. Alston. A dissert. on quick-lime and limewater. Lond. 1750.

Kalk und Kalkwasser als Heilmittel im Scorbut.

A. Cocchi. Bagni di Pisa. p. 253 Firenz. 1750.

C. bemerkt dass der Scorbut durch Mangel an frischen Vegetabilien entstehe und durch dieselben auch zu beseitigen sei.

M. Gmelin. A journ. of voyag. made by ordre of the court of Russia in the Ramavatzin, by the coast of Sibiria etc. 1736—1750.

Beschreibung des Scorbuts unter zwei Schiffsbesatzungen, während sie unter 71° N. B. überwinterten.

1751. G. E. Hambergerus. D. de scorbut. frigid. Jen. 1751.

Brescou Dumouret. Trait. du scorbut. Paris. 1751.

1753. A. Addington. An essay on the Seascurvy. etc. Lond. 1753.

Empfehlung von Acid. muriatic. als vorzügliches Heilmittel.

J. Lind. A treat. of the scurvy. Edinb. 1753.

1754. G. Ch. Dethardingius. De scorbut. Meyapolens. Rostoch. 1754.

H. M. Missa. Quest. med. An a divers. virus scorbut. in dole et sede morb. divers. Paris. 1754.

J. Pringle. Beobachtung über die Krankheit einer Armee. A. d. E. von J. E. Greding. Altenb. 1754.

S. 10. Der Scorbut ist die vorzüglichste und langwierigste Krankheit des sumpfigen Theiles der Niederlande und ist vorzüglich durch feuchte Luft bedingt. Allein dass dies doch nicht in so grosser Ausdehnung anzunehmen ist, geht daraus hervor, dass die Reichen, oder überhaupt die, welche eine gute nahrhafte Diät beobachten, nicht befallen werden. P. macht keinen Unterschied zwischen Land- und Seescorbut. Das seltenere Vorkommen des Scorbuts in den letztern Jahrhunderten, S. 328—336, schreibt er, wie Black, der grössern Reinlichkeit der Städte, den gesunderen Wohnungen, dem allgemeinern Verbrauch des Weins, Bieres, der Gartengewächse, des Thees und des Zuckers zu. S. 338. Im sumpfigen Theil der Niederlande, wo der Scorbut häufig vorkam, will er niemals Scabies gesehen haben, allein auf englischen Schiffen fehlte es nicht an dieser Complication. Viele Krankheitserscheinungen soll die in den Gefässen frei werdende Luft bedingen. Das Krankheitsbild hat er nach Eugalen entworfen. Die nächste Ursache liegt in Fäulniss des Blutes. Auch nimmt P. nur eine Art, den fauligen Scorbut, an.

Die Schrift enthält wohl Belehrendes, wenn man es nur aus der Hülle der Fäulnisstheorie herauszieht.

1755. J. de Gotter. System. de prax. med. Fkf. et Leip. 1755. T. I. p. 95.

Nach Boerhave, wenigstens im wesentlichen abgehandelt.

1756. J. Huxham. Abhandl. v. d. Fiebern A. d. F. 1756. Anhang.

Als Ursachen betrachtet H. schlechte Nahrung, faule Schiffsluft und feuchte Atmosphäre. Das We-

sen sucht er in laugenartiger Schärfe des Blutes. Die besten Heilmittel: Acida vegetabil. et mineral.

E. Rosen. D. de symptom. purpur. scorbut. chron. Lond. 1756. Haller l. c. No. 198.

1757. J. Lind. An ess. on the most effect. means of perserv. the health of seam. etc. Lond. 1757. 1762. 1713. 1774.

Bemerkenswerth in Bezug auf die Prophylaxis.

1758. A. de Haen. Problema de scorbut. in ration. medend. Vindob. 1758—79. P. VII. c. 4. P. VIII. c. 14.

G. v. Swieten. Beschreib. und Heilart. der Krankheit., die am öftersten in den Feldzügen beobachtet werden. Triest. 1758. S. 144.

Kurze Beschreibung des Scorbuts.

1759. L. G Klein. Interpr. clin. Erf. et Lips. 1759. Ed. nova 1826. p. 138.

Besonders genau ist die Prognose abgehandelt.

J. Astruc. Traité de tumeurs etc. Avec deux lettres etc. Paris 1759. T. II.

Die erste Epist. handelt vom Nutzen des Vinum Mouralti und andern Medicamenten im Scorbut.

1760. R. Russel. A dissert. on the use of Seawat. in the diseas. Lond. 1752. 1760.

Das Seewasser als bestes Prophylacticum empfohlen, weil das Salz die Fäulniss verhindere.

1761. J. a. Bona. Tractat. de scorbut. Veron. 1761.

B. zählt eine ungemeine Menge Krankheitsformen, als dem Scorbut eigenthümlich, auf. Auch bekennt er sich zur Annahme: dass die Alten den Scorbut gekannt haben und erwähnt das Vorkommen des Uebels in Italien.

1764. D. Monro. An account of the diseases, which were most frequent in the Brit. milit. hospit.

in Germain. Lond. 1764. p. 250. A. d. E. v. D. J. E. Wichmann. Altenb. 1766. S. 203., v. Begue de Presle. 2 B. 1771.

Ch. E. Endter. Cur des Scorbuts. Hamb. 1764.

D. Macbride. Experimental essays on the following subjects No. 4. on the scurvy etc. Lond. 1764.

1766. C. N. Altmann. Analys. plantar. antiscorbut. Vien. 1766.

S. Eugalenus. De scorbut. select. tract. Boerhave, Huxham, Lind, Addington. Venet. 1766.

F. Home. Princip. medic. Ed. 3. Amstelod. 1766. p. 188.

Anempfehlung einer einfacheren Behandlungsweise des Scorbuts, als bisher befolgt wurde.

A. Severinus. Lib. de scorbut. ed. G. F. Bachström et Huxham. Venet. 1766.

Charl. Bisset. Medical. essays and observations. Lond. 1766.

Beschreibt unter den Namen Landscorbut eine Krankheit, die offenbar Syphilis gewesen ist.

1767. Raymond. Hist. de l'elephant., contenant aussi l'orig. du scorb. etc. Lausan. 1767. Montpel. 1783.

W. Chelmsky. De scorbut. exercit. caesar. reg. in Siles. an. 1760 et 1761 gravit. afficient. Prag. 1767.

1768. N. Hulme. De natur. caus. et curat. scorbut. libel. Lond. 1768.

Die Beschreibung der Zufälle ist nach Lind abgefasst. H. erzählt einen Fall, wo durch schnelles Oeffnen der Schiesslöcher auf einem Schiffe plötzlich der Tod bei einem Scorbutischen bewirkt wurde. Auch erwähnt er der Nyctalopie als Symptom. Die nächste Ursache sucht er im Zurückbleiben und Anhäufen einer fauligen Materie im Körper. Die Gelegenheitsursachen findet er einzig und allein in der

Nahrung. Als Heilmittel empfiehlt er Zitronen, Pomeranzen, China etc.

T. B. de Sauvage. Nosolog. method. etc. Amstel. ed. II. T. 1768.

T. I. p. 307. Synochus scorbutic. p. 352. Tertiana scorbut. p. 446. Miliaris (Purpura) scorbutica. p. 532. Trismus (Rigor) genarum scorbutic. p. 538. Contractura scorbut. p. 546. Catochus scorbut. p. 661. Dyspnoea scorbut. p. 676. Orthopnoea scorbut. p. 684. Pleurodyne scorbut. p. 790. Paralysis scorbut. p. 803. Asthenia scorbut. p. 813. Syncope scorbut. p. 836. Coma scorbut. somnolentum. Cataphora scorbut. T. II. p. 23. Arthrit. scorbut. p. 28. Osteocopos scorbut. p. 30. Rheumatismus scorbut. p. 116. 139. Lumbago scorbut p. 224. Pica scorbuticorum. p. 290. Haemoptysis scorbut. p. 299. Haematemasis scorbut. p 322. Hepatirrhoea scorbut. p. 330. Dysenteria scorbut. p. 333. Melaena scorbut. p. 369. Ephidrosis scorbut. p. 379. Ptyalismus scorbut. p. 455. Phthisis scorbut. p. 464. Aridura scorbut. p. 563. Ascites scorbut. p. 563—567. Scorbutus. S. nimmt 3 Perioden bei der Entwickelung des Scorbuts an: den Scorbutus incipiens, den S. crescens und den S. inveteratus und schildert die Erscheinungen treffend. p. 577. Scabies scorbut. p. 593. Melasicterus scorbut., Icterus niger scorbut. p. 626. Necrosis scorbut. Bei den einzelnen Formen findet man auch die entsprechende Literatur angeführt.

1770. G. V. Zeviani. Sopra lo scorbuto, coronat. della reale Acad. etc. di Manlova. Veron. 1770.

Z. rühmt als bestes Prophylacticum die Milch; da jedoch diese zur See nicht anzuschaffen ist, so setzt er an die Stelle: Gartengewächse und Früchte und belegt den Nutzen derselben durch Beispiele. Unter andern erzählt er: wie Milch das Uebel bei

den Soldaten eines Venetianischen Regiments, und als Besatzung eines Schiffes, schnell beseitigte.

1771. Le Roi. Melang. de phys. et de méd. Paris. 1771.

1772. J. Anderson. D. de scorbut. Eding. 1772.

E. G. Baldinger. D. de scorbut. Jen. 1772.

R. Broeklesby. Oeconomical and med. obs. Lond. 1764. A. d. E. v. Selle. Berlin. 1772.

Empfehlung der China beim Scorbut.

R. A. Vogel. Acad. praelect. de cognosc. praecip. c. h. affect Gött. 1772. p. 576.

Behauptet: dass Hippocrates den Scorbut gekannt habe.

1773. D. Macbride. Einleit. in d. theoret.-prakt. Arzneiw. A. d. E. Leipz. 1773. S. 1001—1031.

J. Lind. Vers. üb. d. Krankh. d. Europ. in heiss. Climat. etc. A. d. E. Riga u. Leipz. 1773 v. Petzold 1775. Mit Anmerk. v. Thion de la Chaume. Leipz. 1792.

Erwähnt zwar den Scorbut, aber ohne sich weiter über denselben hier auszusprechen. Spricht von der Trinkbarmachung des Seewassers und im Anhang über eine Formel zu antiscorbutischen Mischungen.

1744. U. B. Askow. Diar. med. naval. in expedition. Algirens. An. prim. Lond. 1774.

Der Scorbut befiel während dieser Expedition vorzüglich unreinliche, faule oder solche Subjecte, die kürzlich von andern Krankheiten genesen waren. Der Malztrank leistete bald gute Dienste, bald wurde er ohne Erfolg gegeben, jedoch schreibt dies A. dem Verderben des Malzes durch langes Liegen zu. Der Succ. citri versagte niemals seine Dienste, daher A. ihn dem erstern vorgezogen wissen will.

E. G. Baldinger. Krankh. e. Armee etc. Leipz. 1774. T. III. Absch. 3. C. 3.

Ch. G. Gruner. Analect. ad antiquitat. med. Vratislav. 1774.

Poupart. Auserl. Abh. Lübeck u. Leipzig. 1774. S. 289.

Enthält den schriftlichen Nachlass über Sectionsbefunde am Scorbut Verstorbener, mitgetheilt v. N. G. Lesko.

1775. J. A. Brambilla. Chir. pract. Abhandl. v. d. Phlegmone etc. Wien 1775. B. II. S. 331—354.

Mittheilungen über den Scorbut unter der Oesterreichischen Armee in Schlesien während 1762. Er huldigt der falschen Ansicht: dass sich das Uebel unter unzähligen Gestalten verbergen könne. Als Ursachen des sich sehr verheerend bewiesenen Uebels, betrachtet er die schlechten überfüllten Quartiere, wo die Soldaten bei strenger Kälte auf der Erde und auf wenigem Stroh schlafen mussten; den Mangel an Bier, Branntwein und Sauerkraut. Das Uebel erschien zuerst im Februar und hatte bereits im April so um sich gegriffen, dass bereits ⅓ von manchen Regimentern im Spital lagen. Die Armee in Sachsen, welche bessere Quartiere und gute Nahrung hatte, blieb so lange verschont, bis sie sich mit der in Schlesien vereinigt hatte. B.'s Ansicht von der Ansteckung beruht auf einen Irrthum, denn durch das Zusammenleben ward nicht die Ansteckung, sondern nur gleichzeitiges Einwirken der ursächlichen Momente bedingt. Die Erscheinungen, unter welchen das Uebel verlief, waren die gewöhnlichen, nur dass bei robustgebauten Männern, also wo das Uebel mehr örtlicher Natur, oder nur scorbutische Diathese vorhanden war, sich an den Schenkeln starke harte, blaurothe, entzündete (?) Geschwülste ausbildeten mit heftigem Fieber und vollem, harten Pulse. Beim allgemeinen Scorbut war

der Puls schwach. Diese Geschwülste gingen in Brand über und zogen den Tod nach sich. Auch Caries der Maxilla kam vor. Die Heilmittel waren: Körperbewegung, frische animalische und vegetabilische Nahrung, Wein, Getränk von Wasser und Essig, Spir. nitr. dulc. oder Spir. sulphur. aether. Rad. calam. aromat., Rad. armorac., Spir. cochlear., Succ. recent., Hb. beccabung., Elix. Switenii und Molken. Gegen die Zahnfleischaffection wurden Scarificationen und Mundwasser aus Decoct. malt. mit Wein, Oxymel, Borax, Alumen angewendet. Bei den Geschwülsten an den Extremitäten, wenn das sie begleitende Fieber entzündlicher Natur war, leisteten Blutentziehungen gute Dienste. Trat Brand ein, so bekamen die Kranken Milch und China innerlich, und äusserlich wurde eine Abkochung der China mit Wein angewandt. Auch der Complication des Scorbuts mit Dysenterie, Pleuritis geschieht Erwähnung. B. warnt gegen den Gebrauch der Mercurialia et Martialia, allein er verordnete die letztern zu früh, daher sie auch nichts nützen konnten.

J. Lind. Abh. v. Scharbock. Nach d. 2-ten Aufl. A. d. E. v. J. N. Petzold. Riga. Leipz. 1775.

L.'s Schrift gehört zu den klassischen Werken. Sie beginnt mit der Kritik der verschiedenen Schriftsteller über den Scorbut, erörtert umfassend die Ursachen, Diagnose, Prognose, Prophylaxis, vorzüglich zur See; die nächste Ursache und die therapeutische Behandlung. Die erstere sucht er in einer Fäulniss des Blutes und sucht dies durch Sectionsergebnisse zu beweisen. L. verwirft die Ansteckung und den Unterschied von See- und Landscorbut. Beigefügt sind Briefe von C. Cork, über das Uebel in Russland und der Tartarei und von Linée über dasselbe in Schweden. L. verneint die Kenntniss des Scorbuts bei den Alten und weiset

zugleich auf einige der ältesten Mittheilungen über das Uebel hin. In der Litteratur, welche von 1541 bis 1774 fortgeführt ist, sind dennoch Lücken.

L. Rouppe. Abh. v. Scharbock. A. d. E. v. J. Ch. F. Schlegel. Gotha 1775.

Diese Schrift steht der obigen zwar weit nach, allein sie gehört dennoch zu den bessern Schriften über den Scorbut. Schlegel tadelt die Aerzte, welche überall Scorbut finden wollten. Zu den prädisponirenden Ursachen zur See zählte R. den Missbrauch des Rauch- und Kautabacks, eine Constitution mit zähem, dickem, erdigem Blute; schwere Kost; Mangel an Bewegung und Mangel an frischen, grünen Vegetabilien und Missbrauch des Branntweins. Ferner bemerkt R., dass in der Regel die Einwirkung mehrerer ursächlichen Momente zum Auftreten des Uebels nothwendig ist. Als Gelegenheitsursachen sieht er an: Nässe und Kälte, und deprimirende Gemüthsaffecte. R. nimmt auch 3 Grade des Scorbuts an. Als pathognomisches Zeichen führt er die Gänsehaut (allein diese fehlt doch auch bei Neigung zu Wassersucht), Purpura scorbutica und die Zahnfleischaffection an. Die Untersuchung des Blutes in den verschiedenen Stadien des Scorbuts ist sorgfältig angestellt und von Krankheitsgeschichten begleitet. Die nächste Ursache sucht R. auch in zähem, dicken Blute und bemüht sich dies auf verschiedene Krankheitserscheinungen anzuwenden. Die Angaben über das diätetische Verhalten sind beachtenswerth, dagegen die über das therapeutische nur theilweise zu befolgen. Er warnt mit Recht gegen den Gebrauch von Brech- und Abführmitteln. Wenn etwa bestehende Stuhlverstopfung nicht auf den Genuss von Früchten weicht, so räth er zu Tamar. und Cremor. tartar. Zum Schluss der Kur werden Martialia empfohlen. Auch das Heilverfahren der Complication mit Brustbeschwerden, Wassersucht erörtert R.

1776 J. Oduardo. D'una spec. partic. di scorbut. Venez. 1776.

In Belluno soll der Scorbut endemisch sein.

1777. Michaelis. Briefe aus Neujork. Richter's chirurg. Biblioth. B. IV. 1777. S. 135—137. 739.

Ueber den Nutzen des Carottenbreis und des Sprucebiers im Scorbut. Vertheidigt den unzweckmässigen Gebrauch der Vesicatorien im Scorbut, und schildert den Nachtheil, welchen Mercurialia bei der Complication der Lues mit Scorbut angewandt, nach sich ziehn.

Macbride. Nachrichten v. e. neuen Art d. Seescorb. zu behand. A. d. E. Leip. 1777.

Ueber den Malztrank.

1778. H. Callisen. Abhandl. über d. Mittel d. Seefahr. gesund zu erhalten. A. d. L. v. J. P. G. Pflug. Coppenh. 1778.

In Bezug auf die Prophylaxis bemerkenswerth.

Mertans. Philosoph. transact of the royal. soc. of Lond. V. LXVIII. P. 2. 1778.

Als vorzügliche Gelegenheitsursache sieht M. den häufigen Genuss von gesalzenem Fleisch an und empfiehlt als Heilmittel: Vegetabilien, zumal Sauerkraut.

Oft bemerkte M. in Russland, dass der gemeine Mann, trotz schlechter Wohnung und Unreinlichkeit, vom Scorbut befreit blieb und schreibt dies einzig und allein dem häufigen Genuss des Sauerkrauts, der rohen Zwiebeln, des Rettigs, der Heidelbeeren etc. nicht mit Unrecht zu. Als einst in Moscau im Findelhause das Uebel so um sich gegriffen hatte, dass die gewöhnlichen Mittel keine Hülfe schafften, so liess M. die obigen Mittel in grossen Quantitäten geniessen worauf bald Besserung folgte.

J. G. Hempel. Eigene Erfahrung u. Wahrnehm. v. Scharb. Coppenh. u. Leipz. 1778.

1779. Hell. Zucker als Präservativ geg. d. Scorbut. Leipz. 1779.

1780. Poisonnier Desperrieres. Trait. des malad. des gens de mer. T. II. 2-e Ed. 1780.

Beweise über Nichtansteckung des Scorbuts.

1781. N. Hulme. Heilart. d. Steins u. d. Scorbuts. A. d. E. Wien. 1781.

Empfehlung des kohlensauren Gases gegen den Scorbut, entwickelt durch Kali carbonicum und Acidum sulphuricum.

Th. Kirkland. Abhandl. f. p. A. B. XI. S. 714.

Ueber den Mercurialscorbut.

1782. Ch. L. Hoffmann. Scharbock etc. Münster 1782.

Als vorzügliches Heilmittel Rad. Calam. aromat. empfohlen.

J. Ch. A. Theden. Bemerk. und Erfahr. Th. 1. S. 166. Berl. 1782.

Beim örtlichen Mundscorbut erfolgte auf die Anwendung von Acid. sulph. dilut. cum Mel. rosar. die Heilung.

Fr. Milmann. An inquiry into the scurvy from whence the symptoma of the scurvy etc. Lond. 1782.

Kramer diss. epist. de scorbut. etc. pertinent ad ann. 1 20, in ea enim scorbut. describitur, qui ann. 1720. in Dacia ripensi grassatus est. in Crell. Samml. B. III. S. 184. f. Berlin 1782.

1783. Th. Garnett. Samml. auserl. Abhandl. f. p. A. B. VIII. St. 4.

Eine Krankengeschichte, wo zuletzt die Zufälle auf die Anwendung des mit Aqua oxymuriatica gesättigten Alcali vegetabile verschwanden. Täglich wurden 3—6 Gran 4 Mal mit Wasser verbraucht.

Onomatologia medico-pract. V. IV. Norimb. 1783 — 1786.

V. III. p 21. Wird der Vorschlag gemacht, Ziegen mit antiscorbutischen Kräutern zu füttern und die Milch als Heilmittel zu benutzen.

J. B. Forster. Bemerk. auf einer Reise um die Welt. Berl. 1783. S. 531.

Ueber den Nutzen des Malztranks und des Sauerkrauts nebst Bemerkungen über die Gelegenheitsursachen.

Lombard. Diss. sur l'import. d'evacuans d. l. cure de playes recent etc. Suivie d'observat. raison. s. l. complicat du virus vener et scorbut. Strassburg. 1783.

J. Hunczovski. Medic. chirurg. Bemerk. auf einer Reise durch England etc. Wien. 1783.

Behandlungsweise des Scorbuts in den Hospitälern zu Bordeaux, Brest und Portsmuth. Im letztern verschaffte man den Kranken so viel wie möglich reine Luft, und verordnete innerlich Electuarium acidum (zusammengesetzt aus Electuar. lenetiv. Ph. L. Conserv. absynth. und Acid. sulphur.) und äusserlich Waschungen und Umschläge der Ecchymosen und der Purpura scorbutica mit erwärmtem Essig. Auch soll die äusserliche Anwendung des Malztrankes gute Dienste geleistet haben. Viele Kranke (wahrscheinlich nur leicht befallen) genasen, einzig und allein beim Verbrauch grüner Gemüse und beim Genuss von reiner, frischer Luft. Im Hospital von Brest, das schlecht eingerichtet war, benutzte man vorzüglich Rumex acetosum, oder auch Succ. citri vel aurant. in Form von Limonade, oder Punsch. Einen andern Trank, den man vorzüglich bei scorbutischen Lungendefecten rühmte, bereitete man auf folgende Weise: 2 Pf. Tannenholz und 2 Pf. Honig liess man mit 30 Maas Wasser ½ Stunde kochen und 2—3 Pf. täglich davon verbrauchen. Zu

Bordeaux behandelte man mit China und geschabten Kartoffeln.

1786. Th. Trotter. Observ. on the scurvy. Edinb. 1786.

Abhandlungen d. Schwedisch. Academ. für d. Jahr 1785. B. VI. Leipz. 1786.

Faxe über den Scorbut auf der Schwedischen Flotte in den Jahren 1774—1783.

H. Bacheracht. Pract. Abhandl. über d. Scharb. Petersb. 1786.

1787. Duncan. Med. comment. etc. 1787—1790. V. V. 2—4.

Bemerkungen über Bacheracht's Schrift und Blane's Krankheiten der Seeleute.

H. Bacheracht. Phys. diätet. Anleit. die Gesundh. der Seeleute zu erhalten. Petersb. 1787. Französisch. Reval. 1787.

Prophylaxis nach Lind's, Rouppe's, Monro's etc. Erfahrungen für die Russ. Marine entworfen.

Solenander. Consil. medic. L. V. p. 501.

D. Spedicati. Theoret. practisch. Beurtheil. d. Scorb. A. d. J. Petersb. 1787.

Das Uebel soll als Folge von Schlaffheit der festen Theile und der allmäligen Verminderung der Ausdünstung, bewirkt durch kalte feuchte Luft entstehn, wozu die Schiffskost nur bedingungsweise mitwirke. Auch finden wir Belege für die nachtheilige Wirkung der Vesicatorien bei Scorbutischen. Ausserdem enthält die Schrift nichts Bemerkenswerthes.

Th. Trotter. Abhandl. über d. Scharbock A. d. E. v. Michaelis. Leipz. 1787. Vergl. Journ. d. Erfind. etc. Gotha 1793. B. I. St. 3. S. 35.

Die Schrift steht zwar auch der von Lind nach, enthält jedoch manches Beachtenswerthe, untern andern auch kritische Bemerkungen der Ansichten über die Ursachen.

1788. G. Blane. Beobachtung über d. Krankheit. d. Seeleut. A. d. E. Marb. 1788.

Die Betrachtungen über den Scorbut selbst sind kurz, dagegen findet man den einzelnen Abschnitten der Schrift umfassendere Bemerkungen über die Ursachen und die Prophylaxis einverleibt.

J. Marsowsky. D. de scorbut. in M. Stollii diss. med. ad morb. chron. spect. ed. Eyerel. V. 1. Vindob. 1788. p. 24—72.

De l'Humeau. Journ. de méd. chir. etc. Avril. Juin. 1788. T. LXXV.

Ueber eine besondere scorbutische Affection.

Marcq. Am angeführten Ort.

Geschichte einer Phthisis, zu der sich Scorbut gesellte, welcher durch frisch ausgepressten Saft der Kressen, binnen 3½ Monat beseitigt wurde.

Guthrie et Brown. Duncan a. O. V. III. 1788.

G. erwähnt, dass bei dem gemeinen Manne in Russland, in Folge des Climas und der ungesunden Lebensart, der Landscorbut sehr häufig und in so hohem Grade vorkomme als zur See; und dass auch wieder in seiner Lebensart, nämlich im häufigen Genuss des Kohls, der Gurken, der Zwiebeln etc., der Grund zu suchen sei, warum das Uebel nicht allgemeiner um sich greife. Im Winter 1785, wo der Scorbut ungemein häufig vorkam, fand, in Folge von Misswachs, grosse Theuerung der genannten Gemüse statt.

B. bemerkt: dass Trägheit und Faulheit, so wie Schwäche nicht immer als Ursache, sondern eben so auch als Symptome des Scorbuts zu betrachten sind. Die Meinung aber: dass der Puls bei Scorbutischen in der Regel lebhaft, ja sehr oft kräftig gefunden werde, muss als durchaus falsch ange-

sehen werden. Auch macht B. auf die Complication mit Brustentzündungen aufmerksam und zeigt den Nutzen kleiner Aderlässe bei den intensiv verlaufenden an. Als häufig vorkommende Erscheinungen nennt er; Blutungen aus der Nase und Ophthalmia scorbutica. Scorbutische Geschwüre sah er aus Ecchymosen entstehn. Sie erhoben sich nämlich, nachdem sie einen grösseren Umfang erreicht hatten, in Gestalt von Blasen, gefüllt von einer dunkelrothen Flüssigkeit, unter welchen sich, nach dem Oeffnen, ein sinuöses Geschwür fand.

W. Cullen. Medicin Nosolog. A. d. E. B I. S. 385. Leipz. 1788.

1789. W. Cullen. Anfangsgr. d. pract. Arzneiwissensch. A. d. E. 2-te Ausg. B. IV. S. 420—467. Leipz. 1789.

C. theilt die Erscheinungen in drei Grade. Verwirft die Ansteckung. Betrachtet den Genuss des Salzfleisches als wesentliche Gelegenheitsursache (indem er dasselbe als in Fäulniss begriffen ansieht) und frische Vegetabilien als bestes Prophylacticum. Giebt aber doch auch den Einfluss der Kälte und Nässe, der Schwäche und der vernachlässigten Hautcultur zur Erzeugung des Uebels zu. Das Wesen sucht er: in einer Auflösung der Säfte und die beste Heilkraft in Vegetabilien. In den Anmerkungen des Uebersetzers findet man Mancherlei über die Prophylaxis und über einzelne therapeutische Mittel.

Leeder. Duncan a. O. Vol. III. Edinb. 1789.

Krankheitsgeschichte eines entwickelten Landscorbuts mit denselben Symptomen, wie sie der Seescorbut liefert. Die Ursachen waren: unreine Luft, sitzende Lebensweise und schlechte Nahrung bei schwächlicher Constitution. Als nächste Heilindication soll man die Wiederherstellung der Hautausdünstung betrachten und dann den gesunkenen Ton wieder heben. Ersteres durch Antimonium mit

Opium, letzteres durch Elix. acid. Haller mit bittern Mitteln.

Terras. Journ. de méd. et chir. etc. Aout. T. LXXX. 1789.

T. rühmt bei Complication des Scorbuts mit Lues den Sublimat in Verbindung mit Cort. chin. und Stip. Dalcamar.; allein weder ist diese Verbindung rationell, noch auch die Anwendung des Sublimat überhaupt zulässig.

S. G. Vogel. Handbuch d. p. Arzneik. 2-te Aufl. Stendal. 1789. Th. II. S. 140.

Complication des Scorbuts mit Rheumatismus.

1790. Th. Fowler. Duncan a. O. V. IV. Edinb. 1790.

Eine Krankheitsgeschichte von scorbutischer Blutung durch Stuhl und Harn, welche durch Alumen, Gum. Dracon. und Decoct. cort. Chin. mit Elix. acid. Halleri gehoben wurde.

F. Thomson. An essay on the scurvy. Lond. 1790.

1791. D. F. V. Guldner. Beobachtung. über die Krätze etc. Prag. 1791.

G. macht darauf aufmerksam, dass bei vielen Schriftstellern, wo von Scabies scorbutica die Rede ist, nur die häufig als Symptom des Scorbuts vorkommende Gänsehaut gemeint ist. Da aber auch die Scabies neben der letztern vorkommen kann und beide einige Aehnlichkeit haben, so muss man bei der Diagnose sorgfältig verfahren. G. will übrigens niemals bemerkt haben, dass beide einen Einfluss (?) auf einander ausüben.

Guthrie. Journ. de med. chir. etc. Juclet. 1791.

Ueber die Wirkung eines kalten Climas auf den Scorbut.

Lowitz. Auswahl ökonom. Abhandl. etc. B. II. S. 217. Petersb. 1791.

Trinkbarmachung des verdorbenen Wassers.

F. Schraud. Abhandl. von d. Verbind. d. Lustseuch. mit d. Scorb. Wien 1791.

Buchhave. Acta medic. Hafniens. V. III. Hfn. 1791.

Ein Fall von fieberhaftem Scorbut bei einem 7-jährigen Knaben nach überstandenem Scharlach. Mir scheint jedoch das Uebel keineswegs Scorbut gewesen zu sein.

1792. Th. Trotter. Observ. on the scurvy, with a review of the opinions lately advanced on that disease and a new theory defended on the approved method of cure, and the induction of pneumatic chemistry. Lond. 1792.

Soll die 2-te Aufl. der 1786 erschienenen Schrift sein; allein sie unterscheidet sich durch die gänzliche Verschiedenheit der aufgestellten Theorie und durch die Anpassung an das antiphlogistische und Brownsche System gänzlich von derselben.

1793. G. Musgrave. De arthrit. scorbut, in arthr sympt. Oxon 1793.

1794. Th. Beddoes. Medic. Schrift. A. d. E B. I. Leipz. 1794.

S. H. Jackson. Dermapathologie etc. über die Theorie vom Scorbut in besonderer Absicht die Eichenrinde gegen die Krankheit zu empfehlen. A. d. E. Erf. 1794.

1795. F. Milmann. Untersuch. über d. Ursprung, d. Sympt. d. Scorbuts etc. A. d. E. v. H. W. Lindemann. Berlin 1795.

1796. C. G. Th. Kortum. Beiträge zur pract. Arzneiwissensch. Götting. 1796. S. 39. 69. 101.

Ueber den Scorbut zu Stollberg bei Aachen. Als das beste Prophylacticum empfiehlt K. die Milch, sobald sie nur vertragen wird.

1797. Aasheim. De scorbut. secund. accurat. theor. med. phys. explicand. Hfn. 1797.

Hartenkeil. Medicin. chir. Zeit. 1797. B. III. S. 319.

Hales u. J. Ventura. Abhandl. d. Schwedisch. Academ. B. XXVIII.

Mittel zur Luftreinigung auf Schiffen.

F. Oloff. D. de scorbut. et necros. Lemb. 1797.

Th. Trotter. Medicin. nautic., an ess. on diseas. of seam. V. II. Lond. 1797 A. d. E. v. E. Werner. Erf. 1798.

Ueber den Skorbut auf der Englischen Flotte in den Jahren 1794 und 1795.

J. G. Leidentrost. Opuscula physico-chem. et medic. V. II. De cancro scorbutico, ejusq. differentiis a cancero carcinomatoso Duisb. 1797.

J. Clark. Beobachtung. über d. Krankh. auf langen Reisen nach heissen Climat. etc. 2-te Ausg. A. d. E. Koppenh. u. Leipz. 1798. Abth. I. C. I. Abth. II. Abschn. I. C. I. Abschn. 2. C. 6. und Anhang.

Beschreibung des Scorbuts auf dem Schiffe Talbot, von 240 Mann Besatzung, während einer Reise von England nach Bengalen und zurück nach England. C. betrachtet den Scorbut als dasjenige Uebel, welches am häufigsten zur See nach Kälte und Nässe entsteht. Selten oder niemals soll er früher erscheinen (?), als bis die Schiffe auf der stürmischen Höhe des Vorgebürges der guten Hoffnung gelangt sind. Wirksamer, als der Malztrank, erwies sich Citronen- und Pommeranzensaft verbunden mit China, Wein und Gewürzen; und C. behauptet sogar: dass wo von guter Wirkung des erstern die Rede ist, diese den gleichzeitig gebrauchten Mitteln zuzuschreiben sei. C. schlägt, als die zweckmässigste Diät, in kalten Climaten die vegetabilisch animalische, in heissen dagegen, die vegetabilische für sich allein vor; allein dies dürfte doch wohl nur dann der Fall sein, wenn man mit der letztern den Genuss des Weins verbindet, da eine vegetabilische

Diät für sich allein, als zu bland, leicht Fieber und Ruhr herbeiführt. Als Prophylacticum empfiehlt C. den Thee zum Frühstück mit Citronen- oder Pommeranzensaft und Branntwein vermischt. Wofür der Umstand spricht, dass die von China kommenden Schiffe nur selten vom Scorbut heimgesucht werden. C. hat auch lesenswerthe Krankheitsgeschichten beigefügt.

1799. J. Th. K... Entw. von d. Entsteh. etc. alter Geschwüre etc. Leipzig 1799.

Zur Heilung der scorbutischen Geschwüre soll die stärkende Heilmethode verbunden mit Acid. miner. und vegetal., frischer Luft, Bewegung etc.; aber niemals Mercur in Anwendung gebracht werden.

A. Portal. Beobacht. über d. Natur etc. d. Lungensucht. A. d. F. v. G. F. Mühry. B. I. Absch. 9. Hannov. 1799.

Ueber den schädlichen Einfluss der Seereisen bei Phthisis scorbutica. Uebrigens ist die sogenannte scorbutische Schwindsucht Portals nur als eine Complication der ersteren mit dem Scorbut und dadurch herbeigeführten rapideren Verlaufe zu betrachten.

1800. Davis. Phys. med Journ. April. 1800. p. 270.

Ein Fall von fieberlosen Petechien, der nach den Erscheinungen zu urtheilen zum Scorbut zu zählen ist.

1801. Journal, phys. medicin. nach Bradley und Willich. von Kühn. Febr. 1801.

Ueber das Trinkbarerhalten und Trinkbarmachen des Wassers auf langen Seereisen.

T. Trotter. Ueber den Gebrauch der Citronensäure im Scharbock. Physic. med. Journ. Dec. 1801. No. 3.

1802. J. D. Herhold. Uebersicht der verschied. mechan. und chem. Mittel zur Reinigung der Luft. A. d. V. v. Tode. Koppenh. 1802.

Humb. Millioz. Essai sur le Scorb., qui a regné à Alexandrie en Egypte en 1801. Paris 1803.

C. D. Balme. Observat. et reflex. sur le scorb. et après celui qui a régné parmi les troupes françaises, formant la garnison d'Alexandrie, pendent le blocus et le siège de cette ville en 1801. Montp. 1803.

1804. E. Horn. Medicin. Chirurg. B. 1. A. 3. L. 29. Leipz. 1804.

Behandlung der scorbutischen Geschwüre.

Keraudren. Reflexions sommaires sur le scorbut. 1804.

1805. F. v. Schraud. Nachricht. v. Scharb. in Ungarn 1803 etc. Wien 1805.

Beschreibung des Scorbuts im Temescher und Werschezer Kreise. Als Heilmittel benutzte man Semen Sinapeos mit Wein und Bier; äusserlich bei den Mundaffectionen Infus. hb. Salviae mit Acid. muriat., Alumen, Tinctur. myrrh. und Honig; ferner Waschungen der Glieder mit warmem Essig. Auch innerlich angewandt leistete Acid. mur. gute Dienste. Sch. betrachtet eine gesunde zweckmässig gewählte Diät mit Recht als den wesentlichen Theil der Cur. Die Krankheitserscheinungen waren die gewöhnlichen, auch kamen keine Rückfälle vor. Die nächste Veränderung beim Scorbut trifft den Cruor und den Faserstoff-Bestandtheil des Blutes unter allmäliger Auflösung desselben, wodurch Sch. auch die verschiedenen Krankheitserscheinungen zu erklären sucht. Als Complicationen erwähnt er, in Folge einer allgemeinen catarrhalischen Krankheitsconstitution, Durchfälle; Lungenentzündungen; Wassersucht etc. Als besonders wohlthätiges Agens sieht Sch. die Sonnenwärme an. Als ursächliche

Momente führt er an: schlechte schwere Nahrung; Mangel; ungesunde Wohnungen und vieles Fasten (238 Tage im Jahr).

Bemerkenswerth sind die Mittheilungen: dass bei scorbutischen Schwangern kein Abortus erfolgte, sondern die Frucht ausgetragen und übrigens gesund geboren wurde. Ferner, dass Säuglinge von scorbutischen Müttern genährt, nicht scorbutisch wurden, sondern meist gesund blieben.

1807. H. Wolter. Quaed. observ. de nat. et usu plant. acrium in scorbut. util. Gron 1807.

1808. L. F. B. Lentin. Beiträge zur aus üb. Arzneiw. Supplementb. v. H. Sachse. Leipz. 1808. S. 363.

L. betrachtet sowohl den Scorbut als die Lues als die gewöhnlichen Ursachen für Herpes; allein wenn gleich nicht geläugnet werden kann, dass bei dem Scorbut Ausschläge vorkommen, so kann dennoch derselbe nicht als Ursache der Flechten angesehen werden, ohne allen rationellen Haltpunkt für die Behandlung aus den Augen zu verlieren.

1812. Krusenstern. Reise um die Welt. St. Petersb. 1812. 3 Th. S. 184—232.

C. Espenberg liefert bemerkenswerthe Mittheilungen in Bezug auf den Scorbut.

1813. J. D. Larrey. Medicin. chir. Denkwürdigk. A. d. f. Leipz. 1813. S. 8—10. 263—75.

Bemerkungen über den Scorbut an der Küste von Neufundland und unter der französischen Armee in Egypten. Die Krankheitserscheinungen waren die gewöhnlichen, nur blieben bei den Kranken in Neufundland die Extremitäten verschont und die Erscheinungen beschränkten sich auf den Mund und die Brust. L. nimmt einen acuten und einen chronischen Scorbut mit drei Stadien an. Als Ursachen führt er an: Mangel an frischer animalischer und

vegetabilischer Nahrung; schwere, gesalzene und unverdauliche Kost; Cisternenwasser und feuchte Luft. Die Ansteckung bestreitet er. Die Cur beginnt L. im ersten Stadium mit einem Brechmittel, worauf er zwei Abführungen folgen lässt; dann aber verordnet er Decoct. Tamarindor. mit Melass. versüsst; mit Zuckeressig versetzte Klystiere, 1—2 Tassen Kaffe am Morgen; leichte Nahrung, Wein und Körperbewegung. Beim vorgeschrittenen Uebel ward dem Decoct. Tamar. am Abend Campher und Extr. opii aquos. zugesetzt; am Morgen aber Infus. cort. chin. spirituos. (mit schwachem Rum bereitet) gegeben. Als gewöhnliches Getränk erhielten die Kranken Oxycrat, oder Tamarindenwasser. Sobald das Uebel das letzte Stadium erreichte, so verstärkte L. die Gaben der China, des Camphers und des Opiums. Auch findet man Bemerkungen über die Behandlung der Geschwüre und über die nachtheilige Wirkung der Vesicatorien bei Scorbutischen.

F. Schnurrer. Geograph. Nosologie. Stuttg. 1813. S. 439—448.

Vergleichung der Radesyge, des Mal de Rosa und des Pellagra mit dem Scorbut. S. 518—542. Bemerkungen über den Scorbut selbst. Der Scorbut hat in so fern Aehnlichkeit mit dem Aussatz, dass auch bei ihm die beträchtlichsten Veränderungen im materiellen Theil des Körpers vor sich gehn können, ohne dass die Geistesthätigkeit wesentlich dabei leidet und dass, wenn die Krankheitsursachen fortdauern, der pathologische Process, ohne alle Reaction von Seiten der Naturheilkraft, fortschreitet. Vom Aussatz und allen übrigen Krankheiten zeichnet sich der Scorbut dadurch aus, dass alle Individuen, welche sich unter den geeigneten Umständen befinden, das Uebel fast auf gleiche Weise bekommen und auch eben so, nach abgeänderten äusseren Verhältnissen, wieder genesen. Die nächste Ursache

soll nicht sowohl Auflösung und Hydrogenisation der Blutmasse, sondern vielmehr eine einseitige Oxydation derselben sein, so dass es minder frei und gleichförmig das Gehirnleben und die Thätigkeit der willkührlichen Muskeln anfachen kann. Als Beleg für dieses (das Unrichtige wird später erörtert werden) hat Sch. einseitig die Beschaffenheit des Blutes im ersten Stadium ohne eigene Anschauung angeführt. In Bezug auf Gelegenheitsursachen, Symptome und Therapie liefert er bereits Bekanntes.

1815. Schillito. London. Reposit. IV. Decemb. No. 2. 1815. Med. chir. Zeit. B. II. S. 325. B IV. S. 347. 1816.

Warnung gegen die Anwendung des Sal Acetosellae, da dasselbe, mehr als die andern Säuren, die Verdauungsorgane angreift und selbst Vergiftungszufälle erregen kann.

Thomson. London. Reposit. V. III. No. 7. 1815.

Erörterung des eben angegebenen.

1816. F. Holst. Commentat. de Acid. nitric. usu med. Christian. 1816.

Ueber den Nutzen der Salpetersäure bei Complication des Scorbut mit Lues.

Joh. Demschick. D. de scorbuto. Landshut. 1816.

1817. R. W. Bamfield. A pract. treat. on the trop. a scorbut. dysenter. etc. Lond. 1817. 1819.

Ueber den Nutzen eines aus gegohrenem Zucker bereiteten säuerlichen Getränks im Scorbut.

F. Jahn. Chronische Krankh. v. H. A. Erhard. B. II. S. 297—336. 1817.

Ziemlich ausführliche Betrachtung über den Scorbut.

F. Buchholz. Medic. topograph. Nachricht. aus d. Olonezischen Gouvernem. Russ. Samml. 2-r B. H. 2 S. 227. 1817.

B. führt den Scorbut unter den endemisch vorkommenden Krankheiten auf und erwähnt zugleich, dass er häufig im Olonetzischen mit tödtlich ablaufender Wassersucht verbunden vorkomme.

L. Vogel. Die Heilk. d. venerischen Krankheit. Gotha 1817.

Die Syphilis soll eine Vermischung des Scorbutes mit der Lepra sein, daher sie auch mit den gegen beide Krankheiten empfohlenen Mitteln (Metalle und Lign. Guajaci) zu behandeln sei. Uebrigens hat diese Meinung nur wenig Anklang gefunden.

1819. Balme. Trait. hist. et practiq. du scorb. chez l'hom. et l'anim. Lyon 1819.

H. v. Martius. Abhandl. v. d. krimmschen Krankheit etc. Freib. 1819.

S. 68 erwähnt M., dass die Complication mit Scorbut die häufigste sei; allein die Frage, ob die krimmsche Krankheit nicht der höchste Grad des Scorbuts sei, ist dahin zu deuten, dass durch das erstere Uebel das letztere in der Entwickelung bedeutend begünstigt wird.

1822. J. R. Köchlin. Medic. chir. Zeit. B. II. S. 400—432. 1822.

K. empfiehlt als Heilmittel im Scorbut Salze und Salpetersäure.

J. M. Mac-Carthy. D. sistens scorb. theor. etc. Vindob. 1822.

R. Sichting. Diss scorb. aethiolog. sistens. Halae 1822.

Unbedeutend.

1823. E. A. G. Himly. Diss. Cachex. et Cacochym. Gotting 1823.

H. handelt zwar auch die nächste Ursache des Scorbuts ab, ohne jedoch eine besondere Ansicht darüber auszusprechen.

C. L. Moritz. Specimen topograph. medic. Dorpatens. Dorpat. 1823.

S. 63 sagt M., dass der Scorbut im Frühjahr beim Nachlass der Winterkälte in den Hütten der Armen herrsche und durch das Zusammenwirken mehrer ursächlichen Momente, vorzüglich aber durch unreine Luft bei heissen Wohnzimmern und durch den Genuss von gesalzenen Fischen hervorgerufen werde. Selten überschreitet er den ersten Grad, kommt aber häufig mit andern Uebeln z. B. der Lues complicirt vor.

1824. Ф. Гейротъ. Военно-Медицинскій Журн. Ч. IV. No. 2. 3. Ч. V. No. 1. Санктпетерб. 1824. 1825.

Liefert eine ausführliche Beschreibung des Uebels, wobei er vorzüglich Lind etc. benutzt hat.

v. Wedekind. Hufeland's Journal. B. LVIII. St. 1. 1824.

Zieht den Sublimat in Pillenform allen andern Präparaten vor, da er antiseptisch(?) wirke; sobald aber der Athem übelriechend wird, so räth er den Gebrauch des Mercur auszusetzen, indem er das Mercurialfieber als den Anfang des venerischen Scorbuts betrachtet.

Mere Latham. An account of the disease lately prevalent at the general penitentary. Lond. 1825.

Ueber den 1823 und 1824 epidemisch verlaufenen Scorbut.

1826. E. Götte. D. scorb. theor., sympt., aetiol. et therap. Dorp. 1826.

1827. L. A. Struve. Erkenntn. u. Cur acut. und chron. Krankh. S. 408. Dorp. 1827.

Mercurialscorbut.

J. Ch. Dressler. D. de diagnosi affectionum cutanearum scorbuticarum. Dorp. 1827. 8.

1828. C. A. W. Behrends. Vorlesung. über p. A. v. C. Sundlin. B. V. S. 179—94. Berl. 1828.

H. J. Leithann. Adumbratio medico-topogr. urbis Rigue. Dorp. 1828.

Enthält nur in der allgemeinen Krankheitstabelle S. 68 die Angaben der binnen 5 Jahren von den

Aerzten in Riga behandelten Scorbutischen und aus dieser Angabe scheint hervorzugehn, dass die in den Hospitälern behandelten nicht mit aufgeführt sind.

R. Richter. Versuch. e. medicin. Topograph. von Archangelsk. Dorp. 1828.

Nachdem R. S. 119—121 die Annahme: als sei die Kälte die vorzüglichste Gelegenheitsursache, zu widerlegen sucht, (übrigens wurde dies nicht so allgemein angenommen, als R. ausgesprochen hat) behauptet er S. 121, dass die Hauptursache des zu Archangelsk unter den untern Volksklassen endemisch vorkommenden Scorbuts, im Allgemeinen weder im Clima, noch in der Kälte, sondern im häufigen Genuss von gesalzenen Fischen, bei durch Alter und Krankheiten geschwächter Constitution liege. Allein R. hat die ursächlichen Momente sowohl im Allgemeinen, als auch in Bezug auf Archangelsk zu einseitig aufgefasst. S. 135 bemerkt R. kurz: dass er dann und wann die Complication des Scorbuts mit der Lues beobachtet habe. S. 149 finden sich 2 Krankheitstabellen, aus welchen ich hier die für den Scorbut angegebenen Zahlen beifüge, wo sich ein Deficit der Aufgenommenen zu den Geheilten von 17 herausstellt.

1822.

	Aufgen.	Genes.	Gest.
Im Seehospitale . . .	496	463	24.
Im Landhospitale . . .	561	523	26.
	1057	986	50.

1823.

	Aufgen.	Genes.	Gest.
Im Seehospitale . . .	46	46	2.
Im Landhospitale . . .	57	65	2.
	103	111	4.

1824.

	Aufgen.	Genes.	Gest.
Im Seehospitale . . .	66	59	1.
Im Landhospitale . . .	19	19	—.
	85	78	1.

1825.

	Aufgen.	Genes	Gest.
Im Seehospitale . . .	614	609	6.
Im Landhospitale . . .	270	261	6.
	884	870	12.

1826.

	Aufgen.	Genes.	Gest.
Im Seehospitale . . .	180	180	—.
Im Landhospitale . . .	22	21	1.
	202	201	1.

1830. J. Belard. D. de morb. quem Radesyge nominant. Berol. 1830,

Vergleichung der Radesyge mit dem Scorbut.

1832. Neumann. Schnelle und sichere Heilart scorbutisch. Krankheit. Hufeland's Journ. d. p. H. Febr. 1832.

Nach N. soll der Scorbut durch Ansteckung und freiwillig entstehn und die nächste Ursache in einem eigenthümlichen scorbutischen Gift liegen. Bei dem durch Ansteckung erzeugten Scorbut soll der Kranke zunächst im Munde ein eigenthümliches Brennen bemerken, worauf das Zahnfleisch anschwelle und an den Armen blaue Flecke erschienen; auch seien die letztern viel kraftloser und in der Achselgrube fühlten sie Schmerzen ohne Anschwellung der Drüsen und es gesellten sich schneller Durchfälle dazu. Ebenso behauptet N., dass jeder Arzt, der

in ein Lazareth von Scorbutischen komme, davon ergriffen werde. Von allen diesen ist uns nichts bekannt worden, obgleich wir häufig unter Hunderten vom Scorbutbefallenen verweilten. Ebenso ist auch unsere Zunge nicht so fein wie N's Zunge organisirt, um wie er das Gift durch den Geschmack wahrnehmen zu können, (wenn es nämlich überhaupt irgend wo anders als in N's Kopf existirt,) auch schwoll uns das Zahnfleisch nicht auf und unsere Extremitäten blieben auch frei von Flecken. Mit Unrecht verwirft N. die Acida mineral. et vegetabil. und rühmt die Bierhefen als specifisches Heilmittel.

1833. Kleinstein. Med. Jahrb. d. K. K. öster. St. B. XIII. St. 2. 1833.

Eine Krankheitsgeschichte, die zwar den Scorbut im letzten Stadium schildern soll, wohl aber eher den putriden Fiebern anzugehören scheint.

A. Kikin. D. de scorbuto. Mosq. 1833. 8.

1834. R. Krebel. Hecker's Annal. Octobr. 1834.

Rochoux. Dict. de Med. A. d. f. v. F. L. Meissner u. C. C. Schmidt. B. XI. Leipz. 1830—1834.

B. schreibt den Alten Kenntniss des Uebels zu und will, dass es bereits von Hippocrates und Plinius beschrieben worden sei. Die nächste Ursache liegt in beträchtlichen Mischungsveränderungen des Blutes, und bei der Entwickelung müsse den Witterungs- und climatischen Verhältnissen ein grosser Einfluss zuerkannt werden.

C. Forget. Medicine navale. Paris. 1834. T. II. p. 233—257.

1835. J. M. Föhr. D. de vin. Moselan. vi medicatr. in morb. scorb. Berol. 1835.

J. Hutchison. Transact. of the med. etc. of Calcutta. V. VII. Calcutta. 1835.

Beschreibung eines Falls von Landscorbut und Bemerkung dabei: dass der Palmwein wirksamer als vegetabilische Säuren sei.

O. G. A. Rosenberger. D de scorb. Halae 1835.

J. M. Slaviero. D. de scorb. Patav. 1835.

T. W. G. v. Tilesius. Verhärtung des Zellgewebes beim Scorbut.

Horn's Archiv. Juni 1835.

Das Blut soll im Scorbut nicht aufgelöst sein und durch Ergiessung in das Zellgewebe die zuweilen vorkommende Verhärtung desselben, als Vorbote des Ausbruchs der vollkommenen Krankheit, bilden. Weder Seeluft, noch auch das Salz, sondern Mangel an frischer vegetabilischer Nahrung bedingen das Uebel.

Eisenmann. Vegetative Krankheiten. Erlang. 1835. S. 73. 227.

In dem von E. aufgestellten natürlichen System findet man den Scorbut als eine Krankheitsfamilie, Porophyra, mit 5 einzelnen Arten der Sippe Bracheosen, flüchtige Krankheiten, die er wieder zu der Ordnung Parablasten, Nebensprossen, zählt, aufgeführt. Unter den letztern begreift übrigens E. solche Krankheiten, die anatomische Veränderungen, durch abnorme Entwickelung des einen oder des andern organischen Gewebes, hervorbringen und flüssige Producte liefern, die sich different, ja selbst feindselig gegen den Organismus verhalten. Die einzelnen Arten des Scorbuts sind 1) Ophthalmoporophyra, Augenscorbut. 2) Stomaporophyra, Mundscorbut. 3) Gasteroenporophyra, Bauchscorbut. 4) Dermatoporophyra, Peliosis, Hautscorbut. 5) Pneumoporophyra, Lungenscorbut.

Wenn nun gleich im Allgemeinen E. sein natürliches System geistvoll entwickelt hat, und die einzelnen hier angeführten Arten des Scorbuts durch

einzelne Fälle als scheinbar selbstständig nachzuweisen wären, so lassen sich doch wieder die Mehrzahl der einzelnen Krankheitsfälle und selbst die mit den Erscheinungen eines hervorstechenden Ergriffenseins der Schleimhaut des einen oder des andern Theils, welche E. als den Focus des Krankheitsprocesses ansieht, nicht so isoliren, dass man berechtigt wäre sie in die angeführten Arten einzureihen. Die verschiedenen Symptome, an welchen Theilen sie auch, mehr oder weniger sichtbar, erscheinen, sind nur der örtliche Reflex des eigenthümlich materiell veränderten Reproductionsprocesses etc. und nur in wenig einzelnen Fällen scheint derselbe ursprünglich ein örtlicher zu sein, oder sich auch nur deutlich an einer Stelle z. B. an der Conjunctiva, am Zahnfleische etc. auszusprechen und wenn dies der Fall ist, so geschieht es immer nur Anfangs und bei der weiteren Entwickelung des Uebels treten dann auch die Symptome an den übrigen Theilen hinzu. Was die Annahme eines örtlichen Scorbuts anlangt, so steht zwar dieselbe mit der Ansicht über das Wesen in Widerspruch; allein dennoch dürfte es sich mit Wahrscheinlichkeit nachweisen lassen: dass sich der Scorbut, wie andere Krankheiten auf ein örtliches Keimen, wenigstens Anfangs, beschränken kann; wenn nämlich durch besondere Disposition eines Theils z. B. der Haut des Zahnfleisches etc. das Capillargefässsystem und dessen Blut primär ergriffen wird, während der übrige Organismus hinlänglich zur Ausgleichung der nachtheiligen Einflüsse reagirt. Vielleicht entwickelt sich selbst in einzelnen Fällen die allgemeine Krankheit aus dem örtlichen Uebel, vermittelst der Fortdauer der Gelegenheitsursachen und durch Absorbtion des krankhaft veränderten Blutes.

1837. M. Marjolin. Cours de patholog. chirurgic. T. I. Paris 1837.

Nach M. ist der Scorbut, wenigstens in einer

Epoche seiner Dauer, ein Morbus totius substantiae. Unter den Ursachen hebt M. das Heimweh hervor und erzählt: dass junge Soldaten aus der Bretagne, welche, so wie die Auvergnaten, am meisten zum Heimweh hinneigen, an Scorbut und Heimweh leidend, zu Wagen nach ihrer Heimath abreisten, unfähig ihre Glieder zu gebrauchen, (wohl nicht sehr genau zu nehmen) schon am dritten Tage ihre Reise zu Fuss fortsetzen konnten. M. nimmt drei Grade, oder Epochen der Krankheit an. 1) Störung des Gemeingefühls, Schwäche, schmutzige Blässe des Gesichts, Erweiterung der Pupillen und dicke belegte Zunge. 2) Auflockernde und übelriechende Absonderung der Schleimhaut des Mundes; aphthöse Geschwüre, Anschoppungen im Zellgewebe der untern Extremitäten, Extravasat unter der Haut und zwischen den Muskeln, und Geschwüre. Auch fand er bei Injection von Leichen: dass sich die Injectionsmasse reichlich in die Extravasationshölen ergoss. 3) Grosse Schwäche, Fieber (?), Hämorrhagien, Muthlosigkeit bis zur Verzweiflung mit Delirien verbunden; Tod durch Schwäche der Respiration und Anschoppung der Lungen.

C. Oernstrup. Bibliothek for Laeger Kjoebenh. 1837. No. 3.

Ueber die scorbutische Krankheitsconstitution und ihren Einfluss auf die unter derselben herrschenden Krankheiten. Der Scorbut soll in der Reihe der Epidemien eine bedeutende Stelle einnehman. Der Typhus im Heere des Aet. Gallicus und die Krankheit des Römischen Heeres in Westphalen unter Germanicus hält er für Scorbut und behauptet zugleich: dass Hippocrates das Uebel gekannt habe.

Im 15-ten Jahrhundert entwickelte sich ungefähr gleichzeitig mit der Scorbutischen Constitution der Englische Schweiss, und die Fieber traten dann unter einen bösartigen asthenischen Character auf.

Auch das Petechialfieber und die Pest sollten aus der scorbutischen Constitution und Fieberepidemien hervorgegangen sein. (Warum nicht umgekehrt?) Bei entwickelter scorbutischer Constitution bezweifelt er den Uebergang zur Pest, da die letztere ein febriles Element habe. Ebenso schreibt er die fauligen Exantheme und Anginen auch fälschlich derselben Quelle zu.

Eisenmann. Wundfieber etc. Erlang. 1837 S. 108.

Der Genuss gesalzener Nahrungsmittel, die Kälte, die Feuchtigkeit und Mangel an Bewegung will E. nicht als Gelegenheitsursachen für den Scorbut gelten lassen, sondern nur als prädisponirende. Der Scorbut sei vielmehr das Geschöpf einer eigenen, der typhösen etwas verwandten, Luftconstitution, die sich über dem Meere und über gewissen Gegenden befinde, aber auch künstlich, durch das Zusammenleben vieler Menschen auf Schiffen, in Kerkern und belagerten Festungen erzeuge. Zuweilen sei sie, ohne prädisponirende Ursachen, hinreichend zur Erzeugung des Uebels. Der scorbutische Krankheitsprocess bestehe, ähnlich dem typhösen, in einer vegetativen Anomalie der Capillarität, auf die Blutzersetzung erst dann folge, wenn die Krankheit den adynamisch-putriden Character angenommen habe. Obgleich nun durch jene eine Vergiftung der allgemeinen Blutmasse bewirkt werde, so besitze dennoch das Uebel eine bestimmte Verlaufsstelle und zwar die Conjunctiva et Sclerotica oculi, die Schleimhaut des Mundes, des Rachens, der Respirationsorgane, des Magens und der dünnen Därme; wobei noch die äussere Haut in Mitleidenschaft gezogen werde. Uebrigens sei die Schleimhaut des Nahrungskanals, namentlich die des Magens und der dünnen Därme, in der Regel der vorherrschende Sitz der Krankheit; doch könne Anfangs das örtliche Leiden auf jeder der genannten Schleim-

häute ausschliesslich, oder vorherrschend auftreten; sobald es aber den putriden Charakter angenommen habe, würden alle Schleimhäute mehr oder weniger ergriffen. Die so ergriffene Schleimhaut käme Anfangs in den Zustand der scorbutischen Congestion oder Stase, sie färbe sich dunkelblauroth und könne einige Zeit in diesen Zustand verharren. Allmälig komme es aber zur pathologischen Secretion, und da auch beim scorbutischen Process, wenigstens Anfangs, der sthenische, hypersthenische und asthenische Charakter vorkommen könne, so wären auch die krankhaften Aussonderungen verschieden, so beim sthenischen: eistoffigserös; bei dem sich den hypersthenischen nähernden: (wahre Hypersthenie zieht er doch in Zweifel) gerinnstoffig Pseudomembranen bildend; und beim asthenischen: viscös-gauchigt, wo alsdann immer der Tod folge. Als Zeichen der Krankheitsnarcose betrachtet er: das Schwächegefühl und die Gemüthsverstimmung. Es soll auch zwei verschiedene Krankheitsprocesse mit gleichen Erscheinungen geben; nämlich den gewöhnlichen Scorbut und eine scorbutische Krankheit unter den Negern Südamerikas; allein das Nähere der letzteren sei noch zu ergründen. Als bestes Heilmittel sei das Jod zu betrachten.

Fassen wir nun diese von E. aufgestellte Ansicht fest in das Auge, so hat ihm beim Niederschreiben derselben nicht die Erfahrung als Richtschnur gedient, sondern er hat das Bekannte ganz willkührlich nach seinem System geformt.

J. L. Schönlein. Allgem. u. spec. Pathol. und Therap. B. II. S. 39—54. Herisau 1837.

Sch. zählt den Scorbut zu der Krankheitsfamilie der Cyanosen und zwar als zweite Gattung. Mit Unrecht tadelt er die Annahme von 3 Stadien, weil sich nämlich kein bestimmter Verlauf für alle Fälle nachweisen lasse; allein dem ist nicht so, und

eben so wenig wie bei andern Krankheitsformen, wo man Stadien angenommen hat, dieselben zu verwerfen sind, weil ein einzelner Fall nicht vollkommen hineinzupassen ist, eben so wenig ist es auf den Scorbut anzuwenden. Unter den Ursachen finden wir die mit Wasser-, Jod- und Chlordämpfen überfüllte Atmosphäre aufgeführt, was aber davon zu halten ist, werden wir später sehen. Der Scorbut soll nur mit der Lues Combinationen eingehen und nur den Krankheiten, die sich zufällig mit ihm verbinden, einen bösartigen Charakter verleihen, worin diese aber eigentlich besteht, ist nicht gesagt und daher wollen wir sie in den scorbutischen Charakter setzen. Auch verläuft das Uebel nicht immer ohne Krisen, wie Sch. meint. Ebenso wenig erfolgt die Genesung nach wenigen Tagen auf die Ausschiffung bei Seescorbut und beim Landscorbut nicht immer sehr langsam und die Neigung zu Recidiven ist bei beiden gleich und nur in so fern ein Unterschied zu gestatten, als den Ursachen zur See ein grösserer Spielraum offen steht.

H. U. L. v. Roos. Erster med. Jahresber. v. Marienkrankenh. zu St. Petersburg v. Jahre 1836. Petersb. 1837.

Die Zahl der stationär und ambulant behandelten Scorbutischen belief sich auf 592, von welchen 426 genasen, 86 starben und 121 wegblieben. Das Uebel begann mit Anfang Juni und währte bis Mitte August und herrschte so ausgedehnt, als es R. 12 Jahre nicht beobachtet hatte. Auf 25 männliche Kranke kam nur 1 weibliches Individuum. Als Complicationen kamen Fieber, Entzündungen und chronische Brustleiden vor, die meist tödtlich abliefen, während selbst die höchsten Grade des reinen Scorbuts mit Genesung endigten. Spontane Blutungen erleichterten oft sehr (bei welcher Form ist nicht gesagt). Nach Probeaderlässen war dies nicht der Fall; allein auch bei den letztern sind die Umstände

nicht angegeben, unter welchen sie gemacht wurden. Die Wirksamkeit der Mineral- und Pflanzensäuren in Verbindung mit aromatisch-bittern und adstringirenden Mitteln, frischer Nahrung und Luft, so wie von Reinlichkeit, wird durch R. bestätigt.

1838. H. U. L. v. Roos. Zweiter med. Jahresb. etc. Petersb. 1838. S. 78. 199–202.

Die Anzahl der stationär und ambulant behandelten Scorbutischen war 51, mit 40 Genesenen, 1 Todten u. 10 Ausgebliebenen. Die ersten Kranken kamen im März und die letzten Ende August vor. Als Complication in 4 Fällen zeigte sich Wassersucht. Die grösste Zahl der Kranken kam in dem Alter von 17—50 Jahren vor. Je zarter die Jugend und je höher das Alter, um so seltener soll nach R. das Uebel auftreten. Vom 60-sten Lebensjahre und unter den 12-ten Jahre beobachtete R. keinen Fall von Scorbut. Uebrigens enthält der Bericht nichts Bemerkenswerthes.

Keraudren. Annal. d'Hygiene publiq. No. 37. 1838.

Als Ursache betrachtet K. feuchte kalte Atmosphäre, als prädisponirende überhaupt alle schwächende Momente. Durch die in Folge der Feuchtigkeit unterdrückte Hautaussonderung wird das Blut in den Capillargefässen in seiner Mischung wesentlich verändert und dies alsdann auf die ganze Blutmasse übertragen. Wenn er aber sagt: die Untersuchung des Bluts von Scorbutischen habe nichts Besonderes nachgewiesen, so sind ihm die Erfahrungen darüber unbekannt geblieben.

J. J. Ott. De Scorbuto. Lugd. Batav. 1838.

Langheinrich. Scorbuti ratio historica. Berol. 1838.

Beachtenswerthe Schrift.

1859. Eisenmann. Krankheitsfamilie Typosis. Zürich 1839. S. 666. 667.

Der Land- und Seescorbut sollen ihrem Wesen nach verschiedene Krankheiten sein, wenn gleich die äussern Erscheinungen sehr ähnlich sind. (Früher betrachtete er beide als nicht verschieden.) Als Beweis führt er an: dass in manchen Gegenden des südlichen Amerikas und der unter den Ungarn herrschende Scorbut durch das Reisen zur See (!!!) gehoben werde. Allein bei E.'s Behauptung ist nur der kleine Umstand zu beachten, dass das Uebel, was er als Scorbut ansieht, gar nicht dieser Krankheit anheim fällt. Eben so wenig kann man mit E. darin einverstanden sein: dass der Morbus haemorrhagicus Werlhofii eine Art Landscorbut sei. Man berücksichtige nur die Verschiedenheit der allgemeinen Blutmasse bei beiden und das Unrichtige tritt sogleich hervor. Denn während bei dem Morb. h. W. das aus der Ader gelassene Blut eine natürliche Beschaffenheit hat, selbst wenn das durch Blutungen entleerte missfarbig und zersetzt erscheint, so findet man beim Scorbut in allen Theilen die Beschaffenheit der Blutmasse gleich. Ferner befällt der letztere nie so plötzlich als der erstere. Ferner sind Blutungen beim Scorbut immer sehr nachtheilig, während sie beim Morbus h. W. den Kranken nur wenig schwächen. Der Beweis: dass Roborantia, Tonica und Acida in beiden Formen wesentliche Heilmittel sind und folglich auch das Wesen ein gleiches sein müsse, kann unmöglich Gültigkeit haben, da man alsdann alle die Krankheiten hierher rechnen müsste, wo diese Mittel überhaupt Hülfe leisten.

Lond. medic. Gazet. V. XX. p. 906. 940. V. XXI. p. 160. 295. C. Ch. Schmidt's Jahrb. B. XXIII. S. 35—37.

Bericht Englischer Militärärzte über den Scorbut, welcher im Jahre 1836 im Capland herrschte, von J. M'Grigor mit Bemerkungen von Murray. Das Uebel brach 1836 unter den Truppen in der Pro-

vinz Adelaide aus, welche zur Vertreibung der eingefallenen Amakosekaffern verwendet wurden. Nach M. soll der Scorbut auf den Schiffen weniger durch den langen Gebrauch gesalzener Speise, als vielmehr durch den Mangel frischer Vegetabilien hervorgerufen werden. Auch habe man zur Erklärung der Entstehung nicht nur ein einzelnes Moment, sondern alle Umstände genau zu erwägen. Was aber die von M. angenommene Verwandschaft der melänischen und dyspeptischen Krankheiten mit dem Scorbut anlangt, so kann man derselben nicht beistimmen, eben so wenig auch darin: dass weniger eine tonische Behandlung angezeigt sei, als vielmehr eine solche, wie gegen subinflamatorische oder congestive Zustände der chylopoetischen Organe und zur Beseitigung der Magen und Leberstörungen angezeigt wäre, um die fehlerhafte Blutmischung zu verbessern.

Was nun die Mittheilungen von S. Bailey und D. Armstrong anlangt, die gleichsam als Belege für M. gelten sollen, so muss ich geradezu bezweifeln, dass sie Scorbutische vor sich hatten, sondern vielmehr entzündliche Affektionen der Leber, Milz und Brustorgane, zu welchen sich nur zufällig oder in Folge der Behandlung das eine oder das andere Symptom des örtlichen Scorbuts, oder Morbus maculosus Werlhofii gesellt hatte. Die Behandlung von B. u. A. war nämlich strenge Diät, kleine Aderlässe, Calomel, Antimonialia und alle Morgen eine volle Dosis Magnesia sulphurica.

Ford entwickelt recht klar die ursächlichen Momente, welche das Uebel hervorriefen: nämlich die im Juni und Juli herrschende Kälte, bei welcher die Soldaten in schlechten Hütten ohne Feuer auf dem Erdboden schlafen mussten und zugleich nicht allein Mangel an Vegetabilien und gutem Brode litten, sondern auch durch Krankheiten und den übermässigen Genuss des Branntweins geschwächt

waren. Alle welche bessere Wohnungen inne hatten und bessere Kost genossen, blieben verschont. Auch keine Spur von Contagiosität war aufzufinden. Warum das 75-ste Regiment, unter gleichen Verhältnissen, weit mehr litt als die andern Truppen, ist nicht zu ermitteln (Sollten nicht B u. A. vielleicht die Schuld tragen). Das Verschontbleiben der Caffern und Hottentotten bei dem 75-ten Regiment ist wohl darin zu suchen, dass sie mehr an die Einflüsse der Witterung gewöhnt waren, dass sie durch Jagen, Tanzen etc. die Spannkraft des Geistes und Körpers aufrecht hielten, dass sie Feuer unterhielten und dass sie durch Einsammeln von essbaren Wurzeln und Kräutern ihre Lage verbesserten. F. setzt das Wesen des Uebels in eine krankhafte Umänderung der Blutmasse und daraus entstehender Atonie der festen Theile. Die aufgezählten Erscheinungen sind die gewöhnlichen. Nutzen brachte eine tonische Behandlung bei nährender stärkender Diät. Wo Abführungen angezeigt waren, gebrauchte er Magnes. sulphuric. mit einem Aufguss von bitteren Kräutern, oder ein Brausetränkchen.

Morgan erwähnt solcher Erscheinungen, die auf Complication mit entzündlichen Leiden der Brust, namentlich auf Pericarditis, Pleuritis etc. exsudatoria hinweisen und wofür auch der von Malcolm beigefügte Sectionsbefund spricht.

Delmege sucht die Ansteckung durch zwei Fälle zu belegen, irrt jedoch offenbar.

Die Berichte der übrigen Aerzte stimmen im wesentlichen mit dem von Tod überein.

A. Schleifer. Medic. Jahrb. d. O. St. B. XIII. St. 1. 1839.

Einzelner Krankheitsfall. In Folge melancholischen Temperaments, träger Lebensweise, ungesunder Wohnung und schwerverdaulicher Nahrung, stellte sich Scorbut ein, später verbunden mit Gehirnaffectionen und der Tod erfolgte durch Darm-

lähmung und Entzündung. Der Gebrauch des Chinins mit Opium hat durchaus nachtheilig gewirkt.

G. Marcusson. D. de scorbut. Berol. 1839.

H. Haeser's hist. pathol. Unters. etc. B. I. Leipz. 1839.

Betrachtet den Scorbut als Volkskrankheit, hervorgegangen aus der Entwickelung des allgemeinen (typhosen) Krankheitscharakters zu seiner Zeit.

P. Mittel. Beknapte genees en heelkundige Verhandeling over den Scorbutus. Groening. 1839.

1839. Henderson. Edinb. med. and surg. Journ., Juli 1839.

Spricht von den Mischungsverhältnissen des Blutes bei Scorbutischen und welchen Einfluss sie auf Blutungen haben.

1840. Budd. Tweedies Library of Med., Practical Med. Vol. V. Lond. 1840. p. 58. f.

Geschichte und Behandlung des Scorbuts werden besonders hervorgehoben. Mangel an frischen Vegetabilien als die einzige Ursache angegeben und das frische Fleisch für sich allein als kein Prophylacticum betrachtet.

Tilesius. Brandis und Wackenroden's Archiv. f. Ph. B. XXIII.

Russische Volksmittel gegen den Scorbut.

J. F. C. Hecker. Milderung der Feldkrankheiten, in Medic. chirurg. Zeitung. Berlin. 1840. No. 8.

Der Scorbut soll ein steter Begleiter des Typhus gewesen sein; nicht zufällig, sondern als Ausdruck eines und desselben Grundleidens; allein nicht nur fremde, sondern auch eigene Beobachtungen lassen diese allgemein hingestellte Annahme nur beschränkt oder bedingungsweise gelten.

1841. C. Canstatt. Ueber antiscorbutische Mittel: Med. Corresp.-Bl. f. Bayer. A. 1841. No. 4. S. 60.

Die antiscorbutischen Kräuter, Wurzeln etc. wirken nach C. nicht sowohl durch besondere Eigenschaften, als durch die Menge rohen Pflanzensaftes, welchen sie enthalten. Im frischen Zustande heilen frische Gräser den Scorbut nicht minder, als Cochlear. Nasturt. Letztere zeichnen sich nur durch den grösseren Gehalt an frischen Pflanzensaft aus; so verhält es sich auch mit den Citronen, Pommeranzen, Sauerkraut, Kartoffeln etc. C.'s Meinung möchte wohl nur sehr leicht basirt sein; denn welchen Unterschied der grössere oder geringere Zukkergehalt, des scharfen Prinzips etc. in Bezug auf die Verdaulichkeit und Reproduction liefert, hat er ganz übersehn und wohl diese Meinung ohne direckt-gemachte Erfahrung niedergeschrieben.

Braun. Ebendas. No. 6. S. 10.

B. wirft die Frage auf: auf welche Weise die mineralischen und vegetabilischen Säuren ihre antiscorbutische Wirkung äussern; und erwähnt dabei mehrere sich widersprechende Ansichten. Nach Oken soll nämlich durch die Nahrungsmittel so viel wie gar kein Sauerstoff zugeführt werden. Nach Schill wirken der Citronensaft und die übrigen Pflanzensäfte dadurch wohlthätig: dass sie dem Blute keinen Stickstoff, dagegen viel Sauerstoff in einer organischen Verbindung zuführen, die Plasticität des Blutes vermehren, die Ernährung befordern, also dem Blute Kohlenstoff entziehn und so der Venosität entgegenwirken. Nach Geigel kann die Thätigkeit der Gangliennerven dadurch erhöht werden, dass in das Blut solche Stoffe gefördert werden, die vorzüglich belebend auf das Gangliensystem wirken. Dagegen bemerkt Eichhorn, dass wenn auch die vegetabilischen Säuren gleich Anfangs eine kühlende Wirkung haben, doch bald darauf eine erhitzende eintritt, also das Blut decarbonisiren, den Athmungsprocess anregen und die thierische Wärme vermehren. Grundfalsch ist nach denselben, dass der Sau-

erstoff das Blut decarbonisire. Das Ganze dreht sich hier um primäre und secundäre Wirkung der Mittel und fasst man dies nur in das Auge, so schwinden auch die Widersprüche.

W. Dalton. The Lanzet. Septb. 1841.

Rühmt die rohen Kartoffeln, geschält mit einem Zusatz von Essig, nach seinen Erfahrungen, welche er während 3 Jahren auf einem Schiffe gemacht hat.

Парникель. Воен. Журн. Ч. XXXVIII. (Journ. für Militärärzte. B. 48.)

Der Scorbut ist eine gewöhnliche Erscheinung unter den Truppen an der Ostküste des schwarzen Meeres. Im Sommer 1840 herrschte er so ausgebreitet, dass binnen 3 Monaten 1080 Kranke im Hospitale von Phanagoria aufgenommen waren und durch die häufigen Complicationen mit Febr. gastr. nervos., Febr. intermitt., Dysenter. und Hydrops sehr bösartig wurde.

1842. Guislain: Annal. de la Soc. med. de Gand. Januar 1842.

Nach G. wird jetzt der Scorbut nur noch unter den Matrosen angetroffen; auch erörtert er: dass zum Entstehen nicht allein klimatische Einflüsse, sondern auch mangelhafte Ernährung etc. nöthig sind.

Medico-chir. Review. April 1842. No. 72. p. 358.

Auf der Englischen Flotte ist die Sterblichkeit durch den Scorbut, in Folge der besseren Bekleidung, der grösseren Reinlichkeit, der besseren Ernährungsweise, gering.

Chomenko. Journ. f. Militärzte (Russisch) B. XXIX. No. 2. 1842. Vergl. Canstatts Jahrb. B. III. H. 2. S. 190. 1844.

Als wirksam gegen Scorbut wird der Kumis gerühmt.

J. Forni. D. de scorbut. Ticini 1842.

J. Magrini. D. de scorbut. Ticini. 1842.

Beide Schriften sind ohne Bedeutung. Der erstere schildert den Scorbut als Angiosthenie, der zweite zählt den Wasserkrebs zu dem Scorbut.

F. Coale. Ammerican. Journ. 1842.

Liefert Bemerkungen über den Scorbut auf der Fregatte Columbia, mit 480 Mann Besatzung, auf der Reise um die Welt. Die Provision war reichlich und gut, ausgenommen Fleisch und Wasser. Der erste Fall kam beim Cap der guten Hoffnung vor, wurde jedoch bald hergestellt. Die zu Bombay eingenommenen Lebensmittel, Fleisch und Zwieback waren schlecht und nachdem an der Küste von Surinam die Ruhr ausgebrochen, an der Küste von China aber nur noch als Durchfall fortdauerte, stellte sich auf der Fahrt nach den Sandwichinseln (August) der Scorbut ein. Denselben voraus gingen Nachtblindheit bei der grösseren Anzahl der Mannschaft. (Coale irrt in der Angabe: dass nur Bonnet (Lond. med. Gaz. B. IX.) allein das gleichzeitige Vorkommen der Nachtblindheit und des Scorbuts erwähnt habe. Man vergleiche Bampfield, Delford, Blane, Hulme etc. etc.)

Unter den Erscheinungen walteten gastrische Symptome und die Neigung zur Geschwürbildung vor. Die habituellen Grogtrinker befielen zuerst. Es starben am reinen Scorbut 23. Von den Officieren litten von 28 nur 3 und leicht, bei übrigens gleicher Kost. Nur nach beendigter Reise bei frischer Nahrung liess das Uebel nach. 7 starben bald nach der Landung und einer nach 6 Monaten.

Budd. Lond. med. Gaz. Aug. 1842.

B., sich auf die Untersuchungen von Busk stützend, meint: dass die Quantität der Hämatosine im Blute Scorbutischer vermindert sei, während die Menge des Faserstoffs, (?) des Eiweisses und der Salze

vermehrt wäre; dass die eigentliche Beschaffenheit in den Salzen zu suchen; dass irgend ein salziges Prinzip (?), klein vielleicht in Betrag, aber bedeutend im Einflusse und nothwendig für die Ernährung, fehle. Diese Ansicht ist ohne Basis und würde nur dann Berücksichtigung verdienen, wenn ihr gleiche Resultate in allen Stadien des Skorbuts zur Seite ständen, obgleich auch dann noch durch Constitution, Lebensart, Complicationen etc. gar manche Verschiedenheit bedingt sein muss.

Die folgenden Nummern enthalten einen Aufsatz über mangelhafte Ernährung, der wenigstens indirect hierher zu zählen ist. Dabei findet man angeführt: dass nach Malcolmson bei den Gefangenen in Indien, welche nur Brod und Wasser erhielten, der Skorbut auftrat. Ebenso dass im Milbaer Penitentiary die Gefangenen, als im Juli 1822 die Kost geändert und sie fast gar keine animalische Nahrung erhielten, kraftlos wurden und sich später Durchfälle und Scorbut einfanden, der nach weissem Brode, Reiss, Fleisch und 3 Zitronen täglich, wieder verschwand.

F. Forry. Ammerican Journ. 1842. p. 77. f.

Ueber den Scorbut unter den Truppen der vereinigten Staaten zu Council Bluffs und S. Peters am Missouri im Winter und Frühjahr 1820. (Nach Mower und Gale). Die Zahl beider Besatzungen betrug 1016 Mann. Die Zahl der Kranken bis zum 1 März 895, wovon 503 am Scorbut litten und 168 starben. Als Ursachen hat man anzusehn: Kälte, Feuchtigkeit, grosse Anstrengungen und Mangel an Fleisch und frischen Vegetabilien. Von den Officieren erkrankte nur einer. Die Jäger, welche in den Wäldern von Wild lebten, blieben gesund. Als mit dem April wilde Vegetabilien (Alliumarten) genossen werden konnten, so erholten sich auch die schwer Erkrankten bald.

Chomel. Gaz. des hôpit. 13 Febr. 1842.

Einzelner Krankheitsfall, wo das Zahnfleisch so bedeutend geschwollen war, dass die Zähne gänzlich von demselben bedeckt wurden und deren Plätze nur noch durch eine längs des Kieferrandes hinlaufende Vertiefung zu erkennen war.

Antonio Carnevale Arella. Giornale delle scienze mediche di Torino. 1842.

Der Verfasser hält das Uebel irrig für ansteckend und als Hauptmittel führt er Nasturtium aquaticum an.

1843. G. Andral. Essai d'hematologie pathologique. Paris 1843.

Andral hat im Blute Scorbutischer eine geringere Menge Faserstoff bei gleicher Quantität Blutkügelchen gefunden und betrachtet die Verminderung des ersteren und das relative Vorherrschen der letzteren für das Charakteristische.

G. v. Samson-Himmelstiern. Beobacht. üb. d. Scorbut etc. Berlin 1843. Recens. in d. Med. Zeit. Russl. 1844. No. 2. (Kr) — C. Ch. Schmidt's Jahrb. 1844. B. XXXXIII. H. 1. S. 125 (Radius.)

Der Verf. theilt den Scorbut in folgende Stadien ein: 1) *in das der Infiltration* oder Transsudation, welche entweder mit Zerreissung der Capillargefässe oder ohne dieselbe geschieht; 2) *in das der Maceration*. Diesem entspricht ein faserstoffarmes Blut und folgt meist dem der Infiltration ohne Faserstoffablagerung. Noch ist 3) *das der Restitution* möglich, indem oft sehr bedeutende Zerstörungen zurückgebildet werden. Allein diese Eintheilung wird eine lichtvolle consequente Entwickelung des Krankheitsbildes schwerlich zulassen. Als nächste Ursache betrachtet der Verf.: Zerfallen des Blutes in seine Bestandtheile, beginnendes Absterben desselben im lebenden Körper und gehemmter Einfluss der Gefässnerven.

Er betrachtet in seiner Schrift die pathologisch-anatomischen Veränderungen, welche durch den Scorbut in den verschiedenen Geweben des Körpers, in den Eingeweiden, Flüssigkeiten, und der Psyche entstehen, auf eine sehr belehrende Weise und jeder einzelne Abschnitt bietet Interessantes dar. Als Anhang sind noch Krankengeschichten beigefügt und zwar als Belege für die einzelnen aufgezählten Thatsachen.

1843. Radius. De scorbuto. Lips. 1843.

Diese kleine Schrift liefert im Allgemeinen manche Belege zu den Erfahrungen, welche wir bei G. v. Samson finden.

C. A. Reuss. Ueber den Scorbut. Würzb. 1843.

W. Blay. Lond. med. Gaz. Vol. XXXI. 1843.

Sucht durch statistische Tabellen nachzuweisen: dass die in Wasser abgekochte Kartoffel ein gutes Prophylacticum gegen den Scorbut in Gefängnissen abgiebt. Aus den Tabellen ergiebt sich ein sehr häufiges Vorkommen des Scorbuts in den Englischen Gefängnissen, veranlasst durch die Ernährungsweise mittelst Brod und trockner Vegetabilien. Als Grund für die antiscorbutische Wirkung der Kartoffeln giebt Blay das darin enthaltene Acidum tartaricum (nach Einhoff: Gehlen's Journ. B. IV. S. 455), oder citricum an (Vauquelin, Journ. de physiol. T. 85. p. 118).

Canuti. Bullet. di Bologna. Maggio e Giugno. 1843.

Will das Secale cornutum bei zwei Scorbutischen mit ausgezeichnetem Erfolge gebraucht haben, allein seine eigene Furcht vor dem Mittel liess ihn bald zu antiscorbutischen Kräutersäften greifen, welchen offenbar die Beseitigung des Scorbuts anheim fällt.

1843. Grancelli. D. de scorbut. 1843. (Unbedeutend.)

Taylor. Prov. med. Journ. 1843. No. 129.

Ein einzelner Fall.

Borchard. Journ. de med. de Bordeaux. Mai 1843.

Ein einzelner Fall.

1844. H. Thielmann. Die scorbutische Augenentzündung, in d. Med. Zeit. Russl. 1844. No. 1 u. 2.

Behandelt den Gegenstand auf reiche Erfahrung gestützt so gründlich, dass nicht nur die bisjetzt gültigen falschen Ansichten berichtigt sind, sondern dass auch diese Lücke in den Handbüchern über Augenkrankheiten vollkommen ausgefüllt wurde.

J. A. Lange. Zincum muriaticum gegen Syphilis mit Scorbut complicirt. Ebendas. No. 12.

Zugleich erörtert der Aufsatz gegen Hunter etc. dass Scorbutische von primärer Lues inficirt werden können.

Pupke, Wirkung des Citronensaftes gegen den Scorbut. Vergl. Preuss. Vereinzeit. No. 18. 1844.

Ein einzelner Fall, der aber wohl der Stomacace zuzuzählen ist.

Cejka. Prager Vierteljahrschr. I 2. 1844.

Behandelt eine Scorbutepidemie im Provinzialstrafhaus von Prag im Mai und Juni 1843. Von 777 Sträflingen wurden 397 (320 Männer 77 Frauen) Individuen mit 11 Pct. Sterblichkeit befallen. Die Krankheitserscheinungen waren im Allgemeinen die gewöhnlichen. Bei den hydropischen Erscheinungen kam eiweisshaltiger Urin vor; übrigens traten dieselben eben so wie Pyämie (pustulöser Ausschlag, Furunkeln, Abscesse, Pseudoerysipelas, Lobularentzündung der Lungen, Milz und Nieren, Ruhr etc. als Nachkrankheiten auf. Unter den Complicationen wurde häufig die mit Tuberculosis pulmonum beobachtet, ohne dass jedoch der Scorbut auf die letztere einen besondern Einfluss ausgeübt. Acne rosacea

erlitt Verschlimmerung, während Mentagra für die Dauer des Scorbuts verschwand. Die Ergebnisse der pathologischen Anatomie sind reichhaltig, wurden aber nicht alle durch den Scorbut bedingt. Bei der Aetiologie bemerkt der Verf. ganz richtig: dass die krankhafte Haematose von mehreren Ursachen abhängig gewesen. Als vorzügliche Gelegenheitsursachen hat man den Mangel an frischem Fleische und frischen Vegetabilien zu betrachten. Die Behandlung ward mit Umsicht mittelst der bekannten Mittel durchgeführt.

W. v. Samson-Himmelstiern. Häser's Archiv. V. 4. 1844.

Resultate einer Scorbutepidemie, welche er in der ersten Hälfte des Jahres 1840 zu Moskau beobachtete. Sowohl die ursächlichen Momente, als auch die Krankheitserscheinungen, so wie die Ergebnisse in Bezug der pathologischen Anatomie, bieten nichts Besonderes dar, jedoch sind die Untersuchungen genau angestellt. Aus dem niedergelegten Material dürfte Folgendes hervorgehn. Ueberall zieht der Scorbut materielle Vorgänge nach sich, mit Ablagerung eines Stoffs an verschiedenen Stellen, der entweder in den serösen Bestandtheilen, oder verbunden mit dem Faserstoffe des Blutes besteht. Leider fehlen auch hier die chemisch-mikroskopischen Untersuchungen des Bluts, der Exsudate etc. und so auch die Feststellung des wichtigsten Theiles, nämlich: ob die durch das Capillargefässsystem vollbrachte Ablagerung die gewöhnlich im Blute enthaltenen Bestandtheile darbieten oder eigenthümlich-pathologische.

Hebra. Oestreichische Wochenschrift. 1844.

Liefert die Beschreibung von 5 Fällen von Purpura scorbutica.

C. Novellis. Omodei's Annali universali. Nov. 1844.

Beobachtungen des Scorbuts im Militärhospitale zu Alessandria. Das Uebel soll auf phlogistischer Basis beruhn, zumal wenn es akut auftritt und sonach hält er auch die antiphlogistische Behandlung für die entsprechendste.

Lachéza. Du scorbut d'Arabie, dit impropre-ment plaie du Hedjaz. Bullet. de l'acad. de Méd. de Paris. Juin. 1844.

Beobachtungen unter dem Heere des Vicekönigs von Egypten, wo das Uebel häufig und verderblich ist und den Namen Wunde von Hedjaz trägt. Die Ursachen sind die bekannten.

1844. L. Beer. Ueber die in öffentlichen Straf- und Correctionshäusern vorkommende scorbutische Cachexie: Oestreich. Jahrb. Dec. 1844.

Besonders in Bezug auf die Prophylaxis in solchen Anstalten wichtig, von der er weit mehr Erfolg sah als von Zitronensaft, Säuren und Bierhefe.

1845. C. Lingen. Anatomische Veränderungen der Rippen und Rippenknorpelverbindungen durch Scorbut. Med. Zeit. Russl. 1845. No. 39.

J. Kirby. Hydriodate of potash in Land-Scurvy: Dublin med. Press. Octbr. 1845.

Obgleich nicht zu entscheiden, wie viel Diät oder Kal. hydrojod. hier gewirkt haben, so verdient der Fall dennoch in Bezug auf das letztere Beachtung.

Stöber. D. de l'influence que l'analyse chimique et la micrographie ont exercée sur la Pathologie et sur la Therap. Strasb. 1845; Vrgl. Mem. de Med. chir. et Pharm. milit. T. 59, p 179.

Ueber die veränderten Mischungsverhältnisse des Bluts im Scorbut, nebst Erörterung der Resultate, welche Rhodes, Busk und Budd aus angestellten Versuchen zogen und die der Ansicht von Henderson: über die Ursache der scorbutischen Blutungen, direkt widersprechen.

1846. Th. Schütz. Ueber die Wirkung des Kruts im Scorbut; beobachtet während einer Scorbutepidemie zu Troizk im Orenburgischen Gouvernement, im Jahre 1840. Med. Zeit. Russl. 1846. No. 1. 2.

C. Novellis. Sullo Scorbuto. Indagini, osservazioni ed esperienze: Annali univers. di med. April. Maggio 1846.

Verbreitet sich vorzüglich über die Ursachen, welche den Scorbut unter den Gefangenen zu Alessandria hervorrufen. Beweist die Nichtansteckbarkeit des Scorbuts und rühmt besonders den Salpeter bei der Behandlung.

Guyon. De la nature de la maladie connue des Anciens sous le nom de Scelotyrbe ou Scelotyrben: Comptes rendus de l'Academie des Sc. T. XXII.

Sucht zu zeigen, dass die Scelotyrbe veterum dem Scorbut zugehöre und scorbutische Schwäche und Steifigkeit der Glieder bezeichne.

1847. A. v. Kyber. Bemerkungen über den Morbus cardiacus (Pericarditis scorbutica) und über Paracentese des Herzbeutels in demselben. St. Petersb. 1847. gr. 8. Vergl. Med. Zeit. Russl. 1847. No. 21—25.

Nach den Beobachtungen im Cronstädter Seehospitale, mit besonderer Rücksicht auf die Paracentese und darauf folgenden grossen Gaben von Chinin. sulphuric. bearbeitet.

Th. Schwank. D. i. pathol. therap. de Haemopericardio scorbutico. Dorpat. 1847.

Enthält nur dürftig dasjenige, was bereits von Seidlitz, G. v. Samson, W. v. Samson und A. v. Kyber über Pericarditis scorbutica bekannt gemacht wurde.

J. O. Curran. Ueber Scorbut: Dublin Quaterly Journal of Medical Science 1847. Vergl. Froriep's Notiz. d. Nat. und Heilk. Januar 1848. No. 6. B. V.

C. liefert eine umfangreiche Abhandlung, die, ausser historischen Erörterungen über das Alter, Geschlecht, Natur des Scorbuts, auch noch die Diagnose der Purpura vom Scorbut erörtert.

Marchal (de Calvi): note sur la composition du sang dans le scorbut.: Gaz. méd. de Paris. No. 34. — Froriep's Notiz. d. N. u. H. 1847. No. 5. B. IV.

Die Untersuchungen theilte M. der Akademie in Paris mit und dieselben liefern folgende Ergebnisse. 1) Es giebt zwei Arten von Blutungen beim Scorbut: Infiltration und eigentliche Blutungen, welche sowohl äussere als innere sein können. 2) Bei der interstitiellen Blutung können sich die Gewebe entzünden; 3) diese Reaction soll zugleich erklären, dass bisweilen das Verhältniss des Fibringehalts nicht abnimmt; allein nach meiner Meinung möchte hier wohl die Folgerung umgekehrt die richtige sein, zumal da die praktische Erfahrung die Belege dazu liefert. 4) Bei der eigentlichen Blutung kann man mit Sicherheit annehmen, dass weil jede lokale Reaction fehlt, auch das Verhältniss der Fibrine geringer werde. (Auch hier ist die Folgerung umzudrehen.) 5) Bis jetzt hat sich nichts ergeben, was die Annahme von Armuth an Faserstoff im Blute als unrichtig herausstellt. 6) Bei der beobachteten Epidemie, hat, ausser den diätetischen Verhältnissen, noch eine andere unbekannte Ursache mitgewirkt. 7) Eiweiss und Blutkügelchen sind im Scorbut vermindert, und dennoch treten weder (nicht immer) Wassersuchten noch Arteriengeräusche auf. Was das Fehlen der ersteren anlangt, so leuchtet ein, dass ein Mangel an Eiweissbildung sehr verschieden ist von einem Verluste dieses Stoffes. 8) Scorbut und Typhus sind nicht analog; bei dem einen besteht Verarmung des Bluts, bei dem andern Vergiftung desselben.

1847. Becquerel et Rodier, de la composi-

tion du sang dans le scorbut.: Gaz. méd. de Paris. No. 26.

Die Ergebnisse der Analyse des Blutes von 5 scorbutischen Individuen. Die Blutkörperchen und das aufgelöste Eiweis waren vermindert, und der Wassergehalt entsprechend vermehrt. Der Faserstoff nicht vermindert, sondern theils in normaler, theils vermehrter Quantität vorhanden. Positiv verändert und zwar verringert war das specifische Gewicht. Dissolution des Blutes und grösserer Gehalt dieser Flüssigkeit an Alkalien oder Salzen nicht vorhanden.

A. Fauvel, mémoire sur le scorbut observé à la Salpetrière en 1847, et sur la composition du sang dans cette maladie.: Archives générales de méd. Juillet.

Genaue Schilderung der Krankheit in der genannten Epidemie. Die allgemeinen Bemerkungen über die Beschaffenheit des Blutes reihen sich an die von Andral an.

Andral, note sur l'état du sang dans un cas de scorbut. Comptes rendus. T. 24.

Ein Fall, der ganz analoge Resultate mit den von Becquerel und Rodier angegebenen lieferte.

Beaugrand, nouvelles observations sur l'état du sang dans le scorbut. Journ. des connaissances méd. Août.

In den sogenannten neuen Beobachtungen des Hrn. B. findet man nur Reflectionen über die von Becquerel, Rodier, Andral und Fauvel mitgetheilten Thatsachen, die jedoch etwas Neues oder Bedeutendes nicht enthalten.

R. Christison, über epidemischen Scorbut im Generalgefängnis zu Perth und in Edinburg etc. unter den Eisenbahnarbeitern. Monthly Journ. of med. Sc. Juni und Juli.

Wichtig in Bezug auf die Genesis der Krankheit. Als wichtigste Krise beobachtete er verschiedene chronische Hautausschläge. Die Krankheit herrschte so ausgebreitet, dass ¼ der männlichen und ⅟₁₀₀ der weiblichen Kranken im Gefängniss zu Perth scorbutische waren. Als Ursachen führt er nur Bekanntes an. Als Heilmittel empfiehlt er Milch.

Ch. Ritchie, Beiträge zur Pathologie und Therapie des gegenwärtig in verschiedenen Theilen Schottlands herrschenden Scorbuts. Monthly Journ. Juli und August.

R. zählt 4 Arten von Scorbut auf: 1) durch Anämie, Abmagerung, Durchfall, Blutungen, Wassersuchten, geringe Affection des Gefässsystems und das Fehlen der eigentlichen Scorbutsymptome charakterisirte Form. 2) Diejenige Form, welche durch Anämie, häufigen Durchfall, beschleunigten Puls, epigastrischen Schmerz, Traurigkeit, Petechien, rothe urticariagleiche Eruption und Blutungen gebildet wird. 3) Eine Form mit neuralgischen, den rheumatischen ähnlichen Schmerzen, Zahnfleisch-Affection und Ecchymosen. 4) Die gewöhnliche Form mit Zahnfleischblutungen und Ecchymosen an den Gliedern.

Bei uns hier, und so weit mir die Beobachtungen aller Zeiten zugänglich gewesen, habe ich diese Formen mit ihren angeführten Erscheinungen nicht so abgegränzt gesehen und gefunden.

Lonsdale, über den Scorbut in Cumberland. Ibid. August.

Die Ursachen des Scorbuts in Cumberland und der Umgegend wie auch in Schottland überhaupt während des Winters von 1846 zu 1847 sind der Misserndte zuzuschreiben.

Th. Shapter, über das neuerliche Vorkommen des Scorbuts in Exeter und seiner Umgegend. Lond. med. Gaz. Mai.

Enthält nur Bekanntes.

O. B. Bellingham, Fälle von Scorbut. Dublin med. Press. Juli.

Bei den Symptomen sagt der Verf.: niemals habe er an der Stelle der grossen Arterien Blasebalggeräusche, noch an den Halsvenen Nonnengeräusche gehört, obschon das anämische Ansehn der Kranken solches hätte erwarten lassen.

Moore Neligan, Fälle von Seescorbut. Dublin quart. Journ. November.

3 Fälle beobachtet auf einem Russischen Schiffe während der Reise von Odessa nach Dublin. Auch theilt er 2 Analysen scorbutischen Blutes mit.

Blot, über die Gehirnzufälle im Scorbut.: Union. méd. No. 50.

Gehirnerweichung, die in keiner Beziehung zum früher überstandenen Scorbut steht.

Gouzée, Beobacht. v. Scorbut.: Arch. de la méd. belge.

Einzelner Fall.

Allgemeiner Ueberblick der Scorbutepidemie, welche 1846 und 1847 in Grossbritannien geherrscht hat. Union medic. No. 125. 127.128 und 131.

Gaz. méd. de Paris. No. 38.

Sucht die Verwandschaft des Scorbuts, der Purpura, des Oedems und der Elephantiasis nachzuweisen.

Scoutetten, über eine Scorbutepidemie in Givet.: Bullet. de l'Acad. roy. de Méd. T. XII. No. 21.

Mangelhafte Ernährung nach einer Typhusepidemie brachte im Militärhospital zu Givet unter 2 Regimentern den Scorbut zum Ausbruche, indem gleichzeitig feuchte, ungesunde Beschaffenheit des Hospitales selbst wesentlich mitwirkte.

A. Anderson, über die verschiedenen Meinungen bezüglich der Ursachen des Scorbuts: Monthly Journ. of med. Sc. Septbr.

Sucht die zu allgemein ausgesprochene Behauptung Christisons zu widerlegen, dass Milch in allen Fällen antiscorbutisch wirke. Die Frage A.'s ob Scorbut aus verschiedenen Ursachen, nämlich bald aus dem Mangel frischer Vegetabilien, bald wieder aus dem von stickstoffhaltiger Nahrung entspringen könne, dürfte dahin zu beantworten sein, dass weder der eine noch der andere für sich allein, wohl aber in Verbindung mit andern prädisponirenden und Gelegenheitsursachen das Uebel hervorrufen kann. Dass die Milch mehr durch die während der Verdauung erzeugte Milchsäure, als durch ihren Kaseingehalt antiscorbutisch wirke, ist als Hypothese anzusehen. Auch hier sind beide Bestandtheile wirksam und nicht einer, und die Milch ist wie jedes anderes kein absolutes Specificum. Was auch von den vom Verf. gerühmten Kartoffeln gilt, obgleich sie die 3 zur Ernährung nothwendigen Elemente enthalten. Ueber die Beschaffenheit des Blutes im Scorbut bemerkt der Verf., dass derselbe Anämie herbeiführe, in Folge deren die Menge der Blutkörperchen und des Eiweisses abnehme, während Faserstoff und Salze unverändert blieben.

Hier ist wie bei allen in der letzteren Zeit veranstalteten chemischen Untersuchungen des scorbutischen Blutes wohl zu bemerken, dass es von Kranken, mit den leichteren Graden behaftet, genommen wurde, dass also nur die beginnende Umänderung und auch diese nicht eben vielfältig erörtert ist.

Th. Coycock, klinische Vorlesungen über Purpura oder Landscorbut.: Dublin med. Press. April.

Schreibt die in England herrschende Scorbutepidemie von der Kartoffelmisserndte her; auch meint er, der Anbau derselben habe das seltenere Auftreten der Krankheit in England überhaupt bewirkt.

Nonnulla de Pericardii paracentesi. Acc. 1 Lith. Dorpat 1848.

Die Schrift ist in so weit als eine Ergänzung der Schrift von A. Kyber anzusehen, als sie die Operationsmethode genauer erörtert und beschreibt.

CONTRIBUTIONS

TO THE

PATHOLOGY OF THE KIDNEY.

BY

WILLIAM T. GAIRDNER, M.D.

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INTRODUCTION.

ON THE NORMAL STRUCTURAL ANATOMY OF THE KIDNEY.

THE researches of modern anatomists have shown, that the secreting substance of the kidneys may be considered as essentially consisting of the ramifications of a mucous membrane, which is disposed in the form of minute tubes, clothed internally with epithelium, and closely embraced by a network of capillary vessels. These tubes, which in the cortical substance have an exceedingly irregular and tortuous distribution, pass through the pyramids, forming straight converging ducts, which unite together two by two, and finally open on the surface of the papillæ, where their epithelial lining becomes continuous with that of the calyces, pelvis, and ureters. In every part of their course, these tubes are accompanied by vessels, the ramifications of the renal artery and vein being distributed copiously in the substance of the pyramids, and over the external surface of the organ; while the cortical substance is chiefly occupied by the capillary plexuses surrounding the tubes, and by the Malpighian bodies, which have been ascertained to consist of globular tufts of vessels arising from the arteries, but whose anatomical relations and function are still the subject of much discussion.

The labours of anatomists having been thus far successful in elucidating the normal structure of the gland, it became necessary for

pathologists to follow in their steps, and by the same modes of investigation to trace out the changes induced by disease in those structures which had been found to be the most essential anatomical elements of the kidney. This branch of the investigation was first taken up by Valentin,[1] Hecht,[2] and Gluge,[3] who from 1839 to 1842 published various microscopic observations on Bright's disease and other morbid conditions of the kidney. These became extensively known in Germany, and gave rise to several other contributions of much interest, but appear to have excited no attention in this country till after the publication of the papers by Dr Johnson and Mr Simon of London, in the Medico-Chirurgical Transactions for 1846 and 1847. It is proper, however, to state, that in 1842, Mr Goodsir addressed to the Medico-Chirurgical Society of this place a communication on the anatomy of the kidney, and the changes in Bright's disease, which was, so far as I am aware, the first contribution to the literature of that subject in this country; but of which an abstract only was published in the *Monthly Journal* for that year.[4]

The researches which I am now about to publish, were at first undertaken, and indeed had proceeded a considerable way, with a very imperfect knowledge, on my part, of what had been previously observed by others. In consequence of this circumstance, I was led to the minute and careful investigation of some minor points in regard to the normal anatomy of the gland, which I might perhaps have neglected had I possessed the confidence which the examination of Gluge's drawings would have given me. Nevertheless, as no one appears to me to have observed and defined with sufficient exactness the *standard of health* in the human kidney with relation to microscopico-pathological researches, I have thought it might be desirable to introduce the strictly pathological part of this inquiry by a few observations on the healthy structure, premising that I mean to notice only such points as are important in reference to pathology, and by no means to attempt any thing like a complete account of the normal anatomy of the gland.

1. *On the Vascular System of the Kidney.*—It is well known to all who have given attention to the subject, that nothing is more variable, even in cases where no disease can be suspected, than the vascularity of the kidney. Nevertheless, there is no doubt that in some instances its abnormal character is most important as leading

[1] Valentin's Repertorium, Bd. II. 290.
[2] De Renibus in Morbo Brightii Degeneratis, Berolin. 1839; and in Casper's Wochenschrift, 1839.
[3] Observationes Anatomico-Pathologicæ.
[4] Dr H. Bennett informs me that he has, for several years, explained in his classes the views on steatosis of the kidneys contained in Gluge's work; which he had also verified himself.

us to detect disease of the organ. It is therefore of some consequence to understand the conditions under which the most marked alterations of vascularity take place.

The veins of the kidney are disposed chiefly in two situations; viz. on its surface, and in the substance of the pyramids. The cortical substance contains exceedingly few. On the surface, they form a peculiar net-work, visible with the naked eye, and known to anatomists from the time of Ferrein, forming, by their intersections, the boundaries of small pentagonal or hexagonal spaces, in the interior of which the natural pale colour of the cortical substance appears, about the size of a very small pin's head (half a line). The larger veins are scattered over the surface, and tend to a stellate distribution. In this situation, the venous injection is liable to the greatest irregularity of distribution and amount (as has been well described by Rayer). The veins may appear in the highest degree distended, or, on the other hand, perfectly anemic, in correspondence with the degree of fulness of the general venous system; and no change is more frequent than the distension of veins on the posterior side of the organ, from gravitation of the blood. On the other hand, great irregularity of injection, amounting to marbling of the surface, and great increase in the size of the stellar vessels, are generally tokens of disease, as they are the result either of partial obliteration of the venous network, or of the extrusion of the blood from it through over-distension of the loops of tubuli, which form the intervening pale spaces.

The engorgement of the capillaries and Malpighian tufts gives rise to two conditions:—*first*, a generally diffused heightened colour of the cortical substance; and, *second*, increase and greater distinctness of the vascular striæ running from the base of the pyramids to the external surface. This latter species of injection often exists, to a great extent, without any corresponding injection of the rest of the kidney, and, in some instances, the red points composing the striæ are so much increased in size as to form considerable petechiæ (one line in diameter or upwards), in which case the petechiæ usually extend to the surface, occupying the intervening spaces of the venous polygons above mentioned. This appearance was supposed by Rayer to occur from simple hypertrophy and vascular injection of the Malpighian bodies; but Bowman,[1] who has shown that the Malpighian bodies do not exist on the surface of the kidney, has also given a better explanation of such petechiæ, which he holds to arise from rupture of the Malpighian tuft, with extravasation of blood into the neighbouring tubes. He argues that the petechiæ are of irregular form, and of much larger size, than the Malpighian bodies have ever been observed to acquire. He gives, also, a figure, representing the occurrence of a similar appearance

[1] Philosophical Transactions, 1842.

from artificial injection at the surface of the kidney. In this figure the loops or knuckles of the tubuli are seen filled with injection, presenting themselves at the surface, and surrounded by the venous network. The correctness of this explanation cannot be doubted, and it is therefore evident, that the occurrence of these petechiæ must be considered as invariably morbid.

The blanched and anemic state of the cortical substance is a very frequent condition.[1] In many cases, no trace of red colour is to be seen, and the vascular striæ and points are wholly imperceptible; in others, a uniform light rose colour mingles with the pale yellowish tint peculiar to the renal substance, and the vascular points indicating the Malpighian glands are faintly visible. These conditions may coexist with abundant vascularity on the surface, and in the pyramidal portions of the kidney; and they have, since the publication of Dr Bright's researches, been looked upon by all pathologists as of the greatest importance in estimating the healthy and diseased states of the gland, particularly in reference to the granular degeneration. Rayer points out that inflammatory and other diseases of the kidney, are the most frequent of all causes of decoloration of the cortical substance; but he has also noticed the occurrence of anemia as an independent lesion in the kidney, and says, with great justice, that both in the anemic and hyperemic conditions of the organ, the partial character of the vascularity is much more decidedly indicative of the presence of a morbid product than its absolute amount.

I have had numerous opportunities of examining, microscopically, kidneys in which the cortical substance was decolorized, both where this occurred independently, and where it was connected with abnormal deposits in the organ. In such cases, the Malpighian coils of vessels, which, in a strictly normal specimen, may be observed filling the capsule, particularly towards its circumference, with red injection, are pale, bloodless, and compressed, sometimes maintaining their rounded form,—at other times, more or less angular. Along with this condition of the Malpighian bodies, I have generally observed distension of the urinary tubules, either by morbid deposit, or by the accumulation of their own secretion. In the latter case, the kidneys have usually been above the normal size, and of more or less diminished consistence. On the other hand, in cases in which the kidneys have been about or under the usual size, and firmer in texture than ordinary (without morbid deposit), I have several times observed the amount of vascular injection to be greater than usual.

In considering these phenomena with reference to their cause, it is not difficult to show, that from the anatomical constitution of the kidney, the fulness of the urinary tubules must of necessity induce, as its first consequence, compression and emptying of the Malpighian vessels. For whether we adopt the view of Bowman, who asserts the capsule of the Malpighian body to be the dilated extremity of the urinary tubule—or that of Gerlach,[1] who regards it as a diverticulum—or of Toynbee,[2] who considers it as a separate membrane retaining the tubule and the vascular coil in contact with each other, we find that the close connexion of the Malpighian vessels with the urinary tube is maintained by the majority of modern observers (although denied by Reichert and Hyrtl). If this be admitted as probable, then it follows that fluid pressure arising within the tubules must fall back upon the Malpighian vessels. Moreover, from the exceedingly firm character of its fibrous investment, the kidney cannot be suddenly increased in bulk without considerable pressure being exerted on its substance; so that, as a consequence of the anatomical disposition of the gland, the sudden engorgement of its secreting tubes must necessarily be followed, even in health, by the diminution of its vascular supply. I shall afterwards have to adduce numerous instances of the occurrence of this in the pathological states of the gland.

On the whole, it appears from the analysis of the variations in the vascular system, that the most interesting of these, in reference to pathological inquiries, are those of the Malpighian bodies; and that the varieties of the superficial venous plexus are of little consequence, excepting in the case where it is so unequally filled as to give rise to mottling or marbling of the surface.

2. *On the Tubuli Uriniferi.*—The tubes, within which the urine is secreted, are composed of an extremely delicate, translucent, and brittle membrane, the exterior of which is in contact with the capillary vessels, and the interior with a layer of nucleated cells. That these cells are intimately connected with the function of secretion has long been considered probable; and the researches of Goodsir comprise observations extending over so wide a series of secreting structures, and so apposite, as almost to amount to demonstration,[3] that the epithelium of the ultimate glandular ducts is the immediate agent in the process of secretion. Hence the pathological alterations of these structures have become of peculiar importance.

In the kidney of the human subject, the appreciation of the normal characters of the tubuli, and of their epithelium, is a task of no small difficulty. It is not always easy, especially in hospitals situate among the population of large towns, to find organs which can be relied upon as furnishing a standard of health; and, even in those which present no obvious marks of disease, the variations

[1] Muller's Archiv. 1845, No. IV.
[2] Med. Chir. Tran., Vol. XXIX.
[3] This is peculiarly evident from his observation on the testis of the Squalus Cornubicus, where the actual process of secretion may be said to take place under the eye.—*Anat. and Path. Observations*, No. V., *and Trans. Royal Society of Edin.* 1842.

observable in a minute examination of the tubes, are so frequent and considerable, as to present the greatest difficulties to the unpractised observer. With the view of familiarizing myself with these variations, I examined, during nearly two months (with the kind concurrence, and frequently also the valuable aid of Dr Bennett), all the kidneys, with few exceptions, which were removed at the *post-mortem* examinations in the Edinburgh Royal Infirmary. The following results of this inquiry may be useful to those engaged in similar observations, by preventing the mistake of healthy for diseased conditions.

The lining membrane of the tubuli, which is the homogeneous or basement membrane of Bowman, is never seen in the fresh and healthy kidney uncovered by epithelium-cells, the nuclei of which are ranged, at pretty nearly equal distances, over its internal surface. In certain diseased states, and also as the effect of maceration, there may sometimes be seen in the kidney considerable portions of tube having a perfectly homogeneous character, and perfect transparency, with no appearance of structure. Much more commonly the tube is seen destitute of epithelium-cells, but retaining in its walls a few scattered oval nuclei, about one-third smaller than the nuclei of the epithelium-cells. These are the young epithelium-nuclei of Bowman, the germinal centres of Goodsir. They appear imbedded in the substance of the membrane, and are very rarely separated from it even when, in diseased conditions of the kidney, the tube has ceased altogether to perform its function. Observations illustrative of these facts will be given in a succeeding part of this memoir.

The membrane of the tubuli appears to be possessed of considerable elasticity, so as to be capable of accommodating itself to the greater or less amount of secretion within them. In no case is it thrown into folds when the tubes are *in situ*, even when the calibre of the tube is very much narrowed. In the strictly normal kidney, however, the diameter of the tubuli varies much less than might be supposed, being generally, in all parts of the organ, from 1-25th to 1-15th of a millimetre. This is no doubt owing to the constant nature of the secretion, and the freedom with which it escapes as it is secreted, on account of which the tubes are not, like the ducts of the mammary gland, subject to alternate distension and relaxation.

The epithelium-nuclei of the tubule are, as above stated, in the normal state arranged at somewhat regular intervals on the inner surface of the membrane, the intervening spaces being occupied, and entirely filled up by the cell-walls, which, when *in situ*, assume an irregularly polygonal form from mutual pressure, according to the amount of distension of individual cells. The cavity of the tubule appears to be entirely filled up by these cells and by the secretion which distends them, and which, when freed, filters away between them.

The size of the nuclei is pretty constantly from 1-120th to 1-100th of a millimetre. They are circular, and have an extremely clear, well-defined edge, which is perfectly smooth when the kidney is fresh; but occasionally, from putrefaction or other causes, becomes slightly irregular, destroying the circular form of the nucleus. They appear quite flat by every arrangement of the light, and when seen sideways become oval or nearly linear. By transmitted light they have a slight uniform shadow, and present one or two central dark points, which, however, are not constant in their occurrence and position. I have not observed the nuclei to present the phenomena of endosmosis and exosmosis. The addition of water produces little change on them; acetic acid generally makes them clearer than before, but rather by dissolving away surrounding obscurities than by any change in the nucleus itself.

The cell-wall is extremely delicate, sometimes indeed so much so as to be scarcely visible, even with the most careful management of the light; but if a current be produced in the fluid, when the nuclei are floating free on the field of the microscope, the presence of the cell may always be recognised, even when it is most delicate, by its preventing the complete approximation of the nuclei to one another. In a certain proportion of the nuclei, also, it appears to be absent even in the most healthy kidneys; and I have frequently seen organs presenting no other apparent change, in which the proportion of free nuclei was so large that it was difficult to find a complete cell among them. Of this circumstance, and also of the extremely different degrees of tenuity of the cell-wall, where it is present, I am not able to offer an explanation, further than that the latter seems to have a relation to the rapidity of development of the cell; inasmuch as when the cell-development is evidently sluggish, and the tubes obstructed with granular matter, the cell-wall is in the majority of instances denser than usual.

The size of the entire cell varies considerably; it being sometimes but little larger than the nucleus, while at others it attains a diameter of 1-50th or even 1-40th of a millimetre. Its shape, when free, is spherical; within the tubule, however, this shape is modified by the pressure of surrounding parts. When floating free in fluid, the cells frequently roll over, showing the position of the nucleus, which is attached to the side.

The fluid contained in the cells of the tubules, being in fact their own secretion, is, in the strictly normal state, perfectly transparent. Nevertheless, it is exceedingly common to find it clouded and rendered opaque by a minutely molecular deposit, which may be so abundant as entirely to obscure the nucleus, or may even appear distinctly granular, being at the same time scattered over the field of the microscope, and resembling very closely some of the morbid deposits to be hereafter noticed. This molecular shading of the cells is, in the greater majority of cases, owing to a deposit of lithate of ammonia, which is removed almost instantaneous-

ly by the addition of an excess of acetic acid. Such a deposit, when moderate in quantity, can scarcely be called morbid, as it takes place from the cooling of the urinary secretion under the most various circumstances, and without any other trace of the presence of disease.

It is not yet certain whether the act of secretion implies the disappearance and subsequent removal of the cell-wall, or whether the cell gets rid of its contents by a process of exosmosis, in the same way as by endosmosis it receives them from the vessels. But if the former view be correct, it is clear that the effete particles must be removed by the urine in a molecular form or in solution; as no epithelial debris of any kind can be detected in the tubes of a perfectly healthy kidney, and the existence of such debris is one of the most unequivocal and ordinary signs of disease. It is not at all improbable that the molecules of effete secreting epithelium may constitute a considerable part of that impalpable sediment which subsides from normal urine, and which is so fine as frequently to present, even under the microscope, nothing but a cloud of almost invisible molecules.

Whatever be the destiny of the cell-wall, the nucleus must be regarded as a permanent structure, whose function is the perpetual renewal of the membranous cell-wall, and of its secretion. Accordingly, the nuclei have a greater power than any other part of the organ of resisting decomposing agencies; and they are never observed in the urine except when the tubes are the seat of disease.

3. *On the connecting Tissue or Parenchyma, and the general Structural Arrangement of the Kidney.*—Toynbee, who ascribes great functional importance to the parenchyma, describes it as consisting in part of peculiar cells, similar to those within the tubes, to which bloodvessels and nerves are distributed, and which he supposes may have the office of effecting some change in the blood preparatory to the secreting process. Bowman and Goodsir describe the different anatomical elements of the kidney as connected together by a delicate fibrous tissue, which forms a sustaining skeleton for the organ.

The general arrangement of the tissues of the kidney is readily seen by making careful sections through the cortical and tubular substance with Valentin's double knife. Where such a section is made through the cortical substance, the tubes are seen sometimes in section, and sometimes presenting to view more or less of their sides, enclosed in the areolæ of an extremely delicate and lax fibrous tissue, which is so disposed as completely to fill up the interspaces. Here and there a Malpighian body is seen surrounded by its capsule, and enclosed in an areola two to four times the size of that of the majority of the tubules. By rubbing the section between plates of glass, some of the tubes may often be displaced,

leaving the areolæ clear and empty, and displaying the fibrous network unaccompanied by the other tissues. These appearances are represented in the woodcuts below, although it is extremely difficult to delineate this very delicate tissue without some degree of exaggeration.

Fig. 1. Fig. 2.

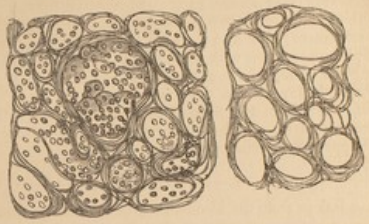


Fig. 1. Section of the cortical substance (by Valentin's knife), treated with acetic acid, showing the tubes and a Malpighian body, with the intervening delicate areolar tissue. The nuclei are seen scattered over the field, being brought out in strong relief by the acetic acid. Magnified 180 diameters.

Fig. 2. Portion of a similar section, from which the tubes have been squeezed out. The areolæ are seen empty. Magnified 180 diameters.

When the section is made through the striæ of the cortical substance, a divided bloodvessel is occasionally visible, and, whether filled with blood or not, is known by the large amount of fibrous tissue which enters into its walls. In the pyramids a similar structure is seen when they are cut across the axis of the tubes; but, as might be expected, the fibrous tissue is much more abundant, from the greater number of large vessels included in the section.

When the capillaries are distended by natural or artificial injection, they are seen to form a close network round the tubes, running in the midst of the intertubular areolar texture above mentioned, and almost filling up the intertubular spaces. Indeed, so much of these spaces do they occupy, that I think it not improbable, that the whole, or nearly so, of the delicate fibrous tissue alluded to, is made up of the walls of the capillary plexus of vessels. Mr Goodsir and Mr Bowman, however, seem to consider it as an independent structure, the former regarding it as analogous to the capsule of Glisson in the liver.

As to the parenchymal cells of Mr Toynbee, I have not been able to observe them either in the healthy or diseased states of the kidney, and must hesitate about admitting their existence, especially as they are not described with such minuteness as to enable us to distinguish them from the epithelium of the tubes.

PART I.

ON THE PATHOLOGICAL ANATOMY OF THE KIDNEY.

In the following observations, I have endeavoured to keep constantly in view the connexion between the pathological elements and the normal structures; and also to reconcile, in as far as possible, the microscopic appearances with those visible by the unaided eye. In describing the latter, I have availed myself extensively of the works of Bright and Rayer; and the reader, to whom those works are accessible, will find in the present memoir numerous references to their excellent plates, which will serve to prevent misapprehension as to the appearances referred to.

I.—Exudation.

The exudations from the blood-vessels of the kidney, being at once the simplest and the most common of its structural changes, and being connected, either as cause or effect, with most of the other more complex conditions, require to be considered first of all.

Exudations into the substance of the kidney give rise to a great variety of external appearances. These have of late years, especially since the observations of Dr Bright upon their connexion with some of the most severe and fatal diseases, been objects of much interest to the pathologist; and have been represented and described in the works of Bright and Rayer, with a completeness and accuracy which has scarcely a parallel in the anatomy of any other organ in the body. Nevertheless, the cultivators of a more minute pathological anatomy have found the lesions of the kidney involved in greater mystery and confusion than those of any other organ whose structure and functions are equally well understood; and although some of the best modern observers have endeavoured to bridge over the chasm between our physiological and pathological knowledge, much remains still to be done.

Exudation may take place from the blood-vessels into all the tissues of the kidney. Its most common seat is the interior of the tubes; but it also occurs frequently within and around the Malpighian bodies, and in the inter-tubular tissue, the tubes being quite clear. I have also seen it infiltrated through all the tissues in the form of a homogeneous mass, which contained within it the whole of the anatomical elements of the kidney.

1. *Exudation within the Tubes.*—The process of secretion in the kidney being in fact a normal process of exudation from the blood-vessels into the tubes, and one which, from its extreme complexity, is liable to very frequent derangement, it is not surprising that, of all

the lesions of the kidney, the presence of foreign matters in the tubes should be the most frequent. The greater part of such exudations, however, are either soluble in the urine, or readily carried away by it, and only come under the notice of the pathological inquirer in connexion with the alterations in that fluid. Of this kind are the albumen in Bright's disease, and the sugar in diabetes, besides a number of soluble substances, which, although connected with morbid processes in the kidney or elsewhere, find a ready exit from the system, and do not remain to block up the tissue of the organ.

When, from any cause, the secreting cells of the kidney receive from the blood substances which do not remain in solution, obstruction of the tubes very readily takes place; much more readily indeed than in most other glands, owing to the remarkably tortuous course and narrow caliber of these tubes, and their complete occupation by the normal epithelium. If the abnormal character of the secretion continues, the obstruction is progressively increased; while, at the same time, there accumulates within the obstructed tubes a quantity of insoluble exudation, which modifies the appearance of the gland, and interferes materially with its function.

The appearance of the kidney, as altered by the presence of exudation in the tubes, is subject to variations depending on the amount of engorgement, and its partial or general character. I have already stated (See p. 5), that the almost invariable effect of the repletion of the tubes throughout the kidney, is a corresponding diminution in the fulness of the vessels of the cortical substance; particularly of the Malpighian vessels and the capillaries surrounding the tubes. Indeed, when the accumulation is considerable, the Malpighian vessels are rarely to be traced with the naked eye, as they generally are in a healthy kidney. I have shown above (*loc. cit.*) the anatomical reason of this phenomenon.[1]

However characteristic of morbid exudations into the tubes, this paleness of the kidney on section is by no means peculiar to such diseased states. It sometimes occurs as a consequence of general anæmia, and much more frequently in organs turgid from retained secretion,

[1] I take this opportunity of stating, that on this point I am compelled to differ decidedly from the views of Dr Johnson of London. It appears to me, that he has fallen into an error both of observation and theory, in ascribing the albuminous urine of Bright's disease to secondary congestion or rupture of the Malpighian bodies, caused by the distension of the tubes from accumulated fat. Not to insist further on the anatomical argument, his view is opposed by every one of Bright's own plates, which show the cortical substance uniformly pale and bloodless, with the exception of Plate V., which would probably not be admitted by Dr Johnson to be a case of Bright's disease at all, inasmuch as it presents none of the appearances of a fatty kidney. In Rayer's work, also, every plate representing the section of a granular kidney (See Plate VIII. Figs. 3 and 5—Plate IX. Fig. 8) presents a pale uninjected cortical substance; and this concurs with the descriptions of Rayer, Bright, Rokitansky, Christison, &c. It will be seen hereafter, that my views of the relation of albuminous urine to the fatty and other degenerations of the kidney, are different from the above.

arising from accidental causes. Such kidneys are also frequently very soft and easily torn, often œdematous, and present a remarkable similarity in their general appearance to some of the earlier stages of exudations. In these cases the microscope is of the greatest service in enabling us to form a positive opinion; and I have so frequently been enabled to correct my own first impressions, as well as those of others, by this means, that I have ceased to repose confidence in the judgment of the unaided eye on kidneys of this description.

The consistence of kidneys containing exudation in the tubes is very various, depending chiefly on the amount and character of the morbid deposit. The colour also varies considerably according to the kind of exudation. When this is very white and opaque, it presents itself in marked contrast to the intervening tissues, giving to the kidney on section a minutely and irregularly speckled appearance, which extends through the cortical substance, and sometimes affects also the tubular cones. It is also seen very distinctly in the intervals of the superficial venous polygons, when these have not been obliterated by pressure. This form is admirably seen in some cases where the exudation consists of salts deposited from the urine. On the other hand, when the exudation approaches nearly in colour to the kidney itself, it is frequently distinguished with great difficulty, the organ presenting a uniform paleness, without any further apparent change.

The volume and weight of kidneys containing exudation in the tubes, are frequently much increased; but this circumstance is so much under the influence of accidentally coexisting diseased conditions, that I prefer to leave it to be treated of afterwards in a separate section.

The above remarks indicate the appearances produced by exudation uniformly diffused through the tubes of the kidney; but these, though common in the slighter forms of the affection, seldom persist when the abnormal deposit has become such as to crowd any portion of the organ. It then tends to accumulate in certain sets of the convolutions in which the urinary current is least active. These, becoming partially blocked up, and ceasing entirely to secrete, are thrown aside from the general outward current of secretion, and become a centre of attraction for further deposit, just as the eddies and still water at the sides of a rapid stream receive from it the foam and floating bodies brought down from above. In this way more and more of the adjacent loops of tubuli are filled with the abnormal deposit, and become added to the former nucleus, until the masses of exudation, thus imprisoned within tubules through which no secretion passes, form irregularly rounded bodies in the cortical substance, visible to the naked eye, more or less prominent on the surface of the organ, and usually of an opaque yellowish colour. These are the granulations first described by Dr Bright, and figured in his 1st and 3d plates, and in Rayer's work (Plate

VIII. Figs. 1, 2, 5, 6, and Plate IX. Figs. 1, 5, 8). The admirable descriptions of these bodies by the last-mentioned pathologist are now well known in this country, and supersede the necessity of further detail in this place.

In 1842, Mr Goodsir described the granulations of Bright as formed by the accumulation of secreted matter within the tubes. In Germany, Gluge,[1] Hecht,[2] Eichholtz,[3] and other observers, have given descriptions of the structural relations and composition of the granulations, which, though differing in detail, concur in representing them as formed within the tubes; and in the excellent work of Lebert on microscopic pathology, a description of them will be found, which, although short, is not surpassed by any of the others in accuracy. In this country several recent observers have taken up the same view. In a paper by Mr Toynbee in the *Medico-Chirurgical Transactions*, Vol. XXIX., there are excellent plates of the anastomoses and convolutions of the tubes, and the granulations in Bright's disease, which afford valuable aid in the understanding of this subject.

The peculiar seat of the renal granulations is the cortical substance; the flow of urine through the pyramids being too constant to permit of the accumulation there of exudation in large quantity. The tendency to form granulations is generally first displayed in the neighbourhood of the surface, and also in the deep-lying convolutions between the pyramids; in both of which situations the tubes are remote from their orifices, and the pressure from behind is consequently small.

It may easily be understood, that the tubes involved in a granulation are in general permanently lost to the kidney as secreting structures; for, having ceased to perform their function, and the stream of secretion having been diverted into new channels, the re-establishment of the former ones is in the majority of cases impossible, and the useless granulations become absorbed and obliterated. The mode in which this occurs will be hereafter described.

The special characters of intra-tubular exudations next fall to be considered. Excluding tubercular and cancerous deposits, which are rare, and in regard to which I have no new observations to offer, these may be considered under three heads, viz. *a*. Crystalline or saline matters deposited from the urine; *b*. Oleo-albuminous or granular exudations from the blood-plasma; *c*. Exudations forming pus.

a. Exudations consisting of Crystalline or Saline matters deposited from the Urine after secretion.—I have already alluded to the fact, that the urate of ammonia, which so frequently occurs as a sediment in

[1] Atlas der Pathologischen Anatomie. [2] Op. cit. [3] Müller's Archiv. 1845.

urine out of the body, is no less frequently deposited from the urine contained within the tubes of the kidney. This occurs in most cases simply as a *post-mortem* appearance, consequent upon the cooling of the body; and, when it is small in amount, it is only appreciable by the microscope. Occasionally, however, it is found in such quantity as to present to the naked eye the appearance of a distinct deposit. In such cases the cortical substance, which, when otherwise healthy, generally retains its normal vascularity, appears occupied by a white or yellowish-white opaque deposit, which presents itself also in a very marked form between the vascular striæ of the pyramids, particularly in the half nearest the cortical substance, where the vessels are more abundant than towards the apex. Such a deposit, which in reality is consistent with a perfectly healthy state of the organ, might easily be mistaken for a diseased condition. The following observation will illustrate this.

Observation I.—A man, æt. thirty-five, was admitted into the Royal Infirmary, March 9th, labouring under symptoms of concussion. He had fallen from a window while in a state of intoxication, and had fractured his left tibia. There was a severe lacerated wound of the scalp. He died next day.

The body was examined March 13th. It was that of a tall and unusually robust man, perfectly well formed, and presented every appearance of perfect health. Every organ in the head, thorax, and abdomen, presented its usual appearance, except the kidneys. These were of normal size, the capsule was easily stripped, the vascular arrangement perfectly normal; but in every part of the cortical substance were seen irregular opaque white specks, contrasting strongly with the vascular redness around, and giving the organ a minutely mottled appearance. This white deposit penetrated between the striæ of the pyramidal substance. I was led to suspect its true nature by the normal character of the vascularity, and also by its penetrating so freely into the converging ducts, which are comparatively rarely the seat of other deposits. On examination by the microscope, the tubes were seen clouded and obscured by a molecular deposit, which was likewise scattered over the field, but which was completely removed by a drop of dilute acetic acid. The structure of the kidney was perfectly normal.

The distinguishing character of this deposit is its ready solubility in dilute acids, such as the acetic or nitric. Under the microscope it presents the appearance, when within the tubes, of a fine molecular shading, which entirely obscures the nuclei. That part of it which floats free on the field of the microscope, may be observed to be composed of fine molecules and granules, which, when large enough to have a defined edge, may be observed to be amorphous or angular, sometimes approaching the circular form, but never accurately rounded. Sometimes these granules cohere together in the form of opaque masses, dark by transmitted light, and of irregular form. The addition of a drop of acetic acid produces instantaneous clearness, unless the deposit be very abundant, in which case more must be added.

The following case, in which a deposit similar to the above took place to a much greater extent, and produced a distinct morbid condition of the kidney, presents many features of interest, and is one of

by no means frequent occurrence. It is the only case of the kind which I have had an opportunity of observing.

Observation II.—*White Deposit in Tubes of Kidney—Cavities in Right Kidney filled with Deposit—Dysentery.*—Alexander Crichton, æt. seven, admitted into the Royal Infirmary, March 1, 1848, under the care of Dr Andrew.[1] He was excessively emaciated, and had been for ten weeks affected with constant purging and pain of the abdomen. He had been in a state of great destitution. He died March 3d, two days after admission.

The thoracic organs were healthy. The mucous membrane of the colon was thickened and ulcerated, and there were patches of lymph on the surface. At the lower end the caliber was much diminished, so as barely to admit a finger. The kidneys were much enlarged; the right weighed 4½ oz., the left 6½ oz.; surface smooth; venous injection unequal. In the left kidney cortical substance voluminous (four lines broad), mostly developed between the pyramids, protruding towards the pelvis, and closely packing in the apices of the cones. Cortical substance infiltrated throughout with small white opaque granules. On the surface these were also visible in the intervals of the venous polygons, but without disturbing the smoothness of the surface. Tubular substance compressed; but in some parts the white infiltration was seen at the base of the pyramids, at others extending nearly to the mammella. The right kidney was broken up superiorly into several anfractuous cavities, from the size of a hazelnut to that of a walnut; these cavities were filled with a diffluent white substance, which had much of the appearance of softened brain. The cavities were lined by a false membrane, which contained numerous gritty particles, and varied from two to four lines in thickness. It appeared to be composed of the condensed tissue of the gland, lined by a layer of concrete matter from the deposit contained within the cavities. In other respects, the right kidney presented the same appearance as the left. Both kidneys were tolerably firm in texture; the venous network of the surface was well injected, and the veins of the pyramids were in some parts full of blood. The cortical substance contained little blood; the vascular striæ and the Malpighian bodies were obscure.

Microscopic Examination.—The tubes were seen to be completely filled with an opaque matter, which obscured the nuclei within. Diffused in water, this matter was observed to be composed of molecules and amorphous granules (Figs. 3, 4), and of a nebulous obscuration, which under a power of 350 diameters was not wholly resolved into distinct parts. The cells, many of which were well formed and entire, were filled with a similar obscuration, which prevented the nuclei from being distinctly visible—(Fig. 4). The white opaque fluid from the cavities was composed of similar granules and molecules, in addition to which were seen some dark opaque amorphous bodies, evidently composed of aggregated granules and molecules—(Fig. 3); and in the midst of the deposit there were numerous nuclei, which were not surrounded by a cell-wall. The whole of this amorphous deposit, and all the molecules, were dissolved on adding a drop or two of dilute acetic or nitric acid; the tubes, with their cells and nuclei, then became clearly visible; nevertheless, after the addition of the acid, some of them were found to contain a few fatty granules and globules, some of which were also scattered over the field. The Malpighian bodies were mostly destitute of blood; a few were slightly injected.

[1] I take this opportunity of expressing my thanks to the physicians of the Royal Infirmary, who have kindly permitted me to make use of every source of information as to the cases under their charge. It is right, however, to state, that I am alone responsible for the *selection* of facts in relation to the histories of disease, and for the whole account of the pathological investigations.

Fig. 3. Fig. 4. Fig 5.

Fig. 3. Some of the white semi-fluid matter from the cavity in the kidney. It is seen to be composed of molecules and granules, interspersed with free nuclei, the debris of the epithelium cells. Some of the granules are aggregated into masses of irregular form. The perfectly spherical granules are composed of fatty exudation, which is present in limited quantity. The nuclei are slightly and uniformly shaded in their interior. (250 diameters.)

Fig. 4. Fluid scraped from the surface of the cortical substance in the same kidney. It differs from the last in containing entire epithelium cells, which are obscured and filled with granules and amorpheous exudation. Part of this, as in Fig. 3, is composed of perfectly spherical fatty granules.

Fig. 5. The same. The urate of ammonia has been removed by acetic acid, leaving only the fatty granules and epithelium cells.

In a case in the *Medico-Chirurgical Transactions of London*, Vol. XXIX. p. 272, in which one kidney was deficient and another very extensively diseased, Mr Busk describes a deposit in the tubes of "a semi-opaque white granular material, soluble or rendered transparent by acetic acid, and presenting none of the characters of oil." In this respect, and also in the small opaque specks and white striæ described as existing in the cortical and pyramidal substances of the kidney, the case has many points of resemblance to those which I have given above; and, in as far as appears from the description, I should incline to consider this a deposit of the same kind, occurring in a kidney otherwise diseased and atrophied. Mr Busk seems to have considered it albuminous in its nature; but albuminous deposits, when amorphous or granular, are not generally found to present the ready solubility in acetic acid which is described in this case, and is always found with urate of ammonia.

Although the deposit of urate of ammonia in the tubes is of little or no pathological importance in the majority of cases, yet it occurs so frequently, and presents an appearance so much like other deposits to the unaided eye, and in some cases readily mistaken even in a microscopic examination, that I am satisfied it must occasionally have been a source of erroneous impressions to pathologists. At least I am conscious, in my own case, that I must have been frequently misled, before I was aware of the importance of applying the test of acetic acid to every deposit occurring in the kidney. This test is particularly necessary, when, as not unfrequently happens, the urate of ammonia deposit is mixed with a certain quantity of fatty granules; in which case, an idea of the relative amount of the two forms of exudation can only be obtained by the removal of one of them in the way described. (See Figs. 4, 5.)

Crystalline deposits within the tubes are of much greater rarity than the amorphous urate of ammonia. I have repeatedly seen in the tubes of diseased kidneys, small groups of perfectly circular

bodies, with a clear distinct edge, of a yellowish colour, and varying in size from 1-200th to 1-80th of a millimetre. As they did not present the peculiar glistening refraction which distinguishes fatty granules and globules, I was disposed to consider them as crystals, more especially as they were very similar to those described by Dr Golding Bird (*Urinary Deposits*, Fig. 8, p. 72) as crystalline urate of ammonia. I have lately seen reason to alter this opinion, having found them to resist the action of acetic acid, and to present characters by no means compatible with the supposition of their crystalline nature. I have now ascertained, from observing their generation in urine, that they are in all probability formed out of the body as a product of decomposition; but as my observations have not yet led to any precise knowledge of the mode or circumstances of their development, I shall for the present do no more than record their occurrence.

A less questionable form of crystalline deposit has been observed by Gluge in the kidney of a dog, whose bladder contained a sanguinolent urine. The kidney presented marked capillary injection, and was considered by Gluge as being inflamed. The tubes were in some places crowded with semi-transparent crystals of very irregular form (see *Atlas d. Path. Anat. Lieferung* 10. P. II. Figs. 5-7.) Gluge has not stated the probable nature of these crystals.

In the following case I had an opportunity of observing a deposit of crystals very like those mentioned by Gluge; and, from their appearance and colour, I have little doubt that they were uric acid, although from their minute quantity they could not be submitted to chemical examination.

Observation III. *Nephritis? Pneumonia. Crystalline Deposit in the Tubes of the Kidney.*—Edward Graham, æt. twenty, labourer. Admitted into the Royal Infirmary, February 29th, under the care of Dr Douglas, on account of epigastric pain and tenderness, nausea and vomiting, accompanied by obstinate constipation, scanty high-coloured urine, and a peculiar typhoid oppression of aspect, less marked on admission than it became afterwards. Two months before admission he had a gonorrhœal discharge, for which he appeared to have taken mercury; the gums and mucous membrane of the mouth were extensively ulcerated. Three days after admission the urine was examined, and found to contain a considerable quantity of albumen, with blood corpuscles; its density was 1·017. The urine continued scanty (10-15 oz. daily), and had to be drawn off by the catheter, on account of its retention in the bladder; the typhoid depression increased; and the day before his death the physical signs of extensive pulmonary affection, without any marked symptoms, were observed. The pulse gradually became weaker, and he died on the 9th March.

On dissection, the heart and great vessels were loaded with very dark blood. Both lungs were much engorged, and a considerable portion of the left lung had passed into the state of red hepatization. The kidneys weighed 7 oz. each, and were of large size, the increase being chiefly apparent in the cortical substance, which was of a much deeper colour than natural. The radiated vascular striæ of the cortical substance were also much injected, and the points

indicating the Malpighian bodies were turgid and dark-coloured. The surface showed the venous network in a state of congestion, but no other change.

Microscopic Examination.—On making a section of the cortical substance with Valentin's double-bladed knife, the gorged capillaries were seen surrounding the tubes, and apparently completely filling up the inter-tubular spaces. The Malpighian bodies were large, and injected throughout, instead of merely at their edges, as is commonly the case. In one or two places the tubes appeared full of blood; but this was not at all general. Here and there were scattered among the sections of the tubes well-defined angular bodies of different sizes, from 1-100th to 1-30th of a millimetre. Their shape was by no means regular, being sometimes imperfectly rounded, at others distinctly angular, and when so, inclining to the rhomboidal form. When isolated, their thickness appeared to be considerable, and their colour was a moderately deep amber yellow. The larger ones presented the appearance of cracks or fissures in their interior, the cleavage being generally more or less accurately parallel with the sides. On adding acetic acid, which did not affect in any way the bodies above described, many of the tubes were seen isolated, their nuclei plainly visible, and their cells free from deposit; the crystalline bodies could be seen in a few; and one, which is figured below (Fig. 6), was completely crowded by them. Comparatively few, however, were in this condition; the greater number being apparently healthy.

Fig. 6.

Fig. 6. Irregularly crystalline deposit, probably of uric acid, in a tube from kidney of Graham. Some of the crystals are seen loose. (250 diameters.)

In this case we have partial suppression of the urinary secretion, which was of low specific gravity, and albuminous (partly, no doubt, from blood): in connexion with which circumstances there were symptoms of gastric irritation, such as frequently accompany disorders of the kidney, and a marked typhoid depression and stupor. It seems every way probable that this last condition was owing to the retention and accumulation of urea in the blood. These circumstances entitle us to look to the kidney as having a share in the disease, notwithstanding the absence of pain on pressure in the lumbar region, which is indeed a circumstance of little moment, when we consider that all uneasy sensation seemed to be masked, owing to the typhoid condition. Notwithstanding these functional alterations, however, there is no apparent exudation within the substance of the gland, except this minute sabulous matter in the tubes. May we not regard this last as one of the exciting causes of the disease, taken in connexion with the urethral irritation, and the state of the system induced by the action of mercury, to which he appears to have been subjected before admission?

b. Oleo-Albuminous Exudations from the Blood-plasma.—I employ this term as including, in one extended series, the whole of those exudations recognised both by German and English pathologists as fatty in their nature (the fatty granules, globules, and corpuscles of authors), together with many of those which have been distinguished as more properly inflammatory, such as the inflammation globules, granular corpuscles, or exudation granules and corpuscles of different writers. The necessity of a classification founded on the elementary structure and chemical composition, rather than on the accidental structural varieties of such exudations, is every day becoming more apparent. On the one hand, the use of the term "fatty" by pathological anatomists, though in itself most descriptive and apposite, has frequently been the means of vitiating their conclusions, and even their descriptions, when employed, as it has very frequently been, with the preconceived view that it is applicable only to chronic changes. On the other hand, the observations contained in the present memoir will be found to add new links to the chain of evidence which has been accumulating for several years past, that the so-called "inflammation" or "exudation" corpuscles, masses, and granules, are by no means characteristic of acute inflammatory processes. This conviction, which from the first induced many of the most eminent and well-informed pathological writers to withdraw the original term "inflammation globules" employed by Gluge, and to substitute for it those of granular cells, or exudation corpuscles (see the works of Henle, Vogel, and Hughes Bennett), has received a most complete confirmation from the valuable researches, lately published by Reinhardt,[1] on the nature of the granular corpuscle; in which it is proved by numerous and well-founded observations, that the corpuscle in question arises, in many instances, from the deposition of granules, consisting of fatty and protein elements, in the natural epithelium cells of different organs; and that its origin is not only in some cases independent of the inflammatory process, but frequently a purely physiological change, as in the *membrana granulosa* of the Graafian vesicle of the ovary. The occurrence of the granular corpuscles in almost every species of pathological product, is mentioned in the systematic works of Lebert and Vogel; and the readers of the *Monthly Journal* need scarcely be referred to the observations of Dr Bennett on cancerous structures, for numerous proofs of their formation in connexion with this form of chronic disease.

The fatty nature of the granules occurring in inflammatory products is known to most histologists, and is easily proved by observing the reaction of ether. Vogel[2] states, that they are composed partly of fat and in part of protein, and carbonate or phosphate of lime (the mineral elements are, however, very variable in amount). The relations of the oil to the albumen or protein, in these and other structures, has been minutely studied by Ascherson and Hughes Bennett,[3] who have shown that an albuminous membrane surrounds the oil granules, and prevents them from coalescing, as they would otherwise necessarily do; and that the formation of emul-

[1] Archiv. für Phys. Pathologie, by Virchow and Reinhardt, No. 1. 1847; analyzed in *Monthly Journal*, February 1848,—Retrospect, p. 6.

[2] Path. Anatomy, Dr Day's translation, p. 157.

[3] See the paper of the latter "On the Structural Relation of Oil and Albumen;" in the *Monthly Journal* for September 1847.

sions, where oil exists in a minute state of division, depends on a similar arrangement.

I have myself had repeated opportunities of observing the formation of granular corpuscles in diseased structures. In pneumonic exudation, in which they can be traced very frequently from their earliest stages, I have invariably found them to be formed from epithelial cells, according to the law laid down by Reinhardt. I have likewise satisfied myself, by repeated observations, that in some of the forms of so-called pneumonia, the quantity and size of the fat globules and granules is such as to constitute a true fatty degeneration of the lung, in the same sense in which the term has hitherto been more familiarly applied to the corresponding lesions of the liver and kidney; and that these lesions present no structural difference from the more ordinary forms, except the greater number and the larger size of the globules which accumulate in the tissue. Finally, in the case of the lung these views have been fully borne out by chemical analysis. In a series of researches by Guillot (*Gazette Médicale*, No. XXIX. 1847), it is shown, that in all diseases of the lung giving rise to obstruction of its tissue by exudation, there is an increase in the relative quantity of fatty matter, which, in the adult healthy organ, is about six per cent., but in the diseased states (such as pneumonic or tubercular infiltration) rises frequently to fifteen, and sometimes to fifty per cent. No similar analysis has yet, so far as I know, been applied to the kidney; but considerations, deduced from histological observations, give the strongest reason to suppose that in this respect, as in others, an analogy would be found between the pathological conditions of the two organs.[1]

I have entered thus far into the general pathological anatomy of this form of exudation, with the view of reconciling my own observations with those of previous writers on this subject; as also to explain my adoption of a classification which annihilates, or, at least, very much modifies, distinctions which many conceive to have a pathological significance corresponding to their practical importance. However satisfactory it might be to point out the inflammatory and non-inflammatory lesions of the kidney as presenting strongly marked pathological distinctions, I am satisfied that such an attempt would fail, from not being founded in nature or truth; and I am confirmed in this view, by the free admission, on the part of the most skilful pathological anatomists, of the extreme difficulty of making the distinction in question in the case of the kidney. Indeed, in considering the terms inflammatory and non-inflammatory as being more applicable to the modes of invasion of diseases of the kidney than to differences in their pathological anatomy, I am only following out the ideas of Rayer, who included most of them under one patho-

[1] Rokitansky enumerates inflammatory exudations as among the circumstances under which fat is deposited pathologically.—*Handbuch d. Path. Anat.*, and *British and Foreign Med. Chir. Review*, Jan. 7, 1848, p. 287.

logical name (Nephrite), and then distinguished them into acute and chronic.

The application of these principles to the explanation of various well-known diseases of the kidney, will be treated of in the sequel (Part II).

Oleo-albuminous exudations are distinguished by their being partially soluble in ether, which leaves an amorphous residue insoluble in cold mineral acids. The amount of this residue relatively to the whole mass, differs much in different cases, and indicates the relative amount of the protein element; it is sometimes in minute quantity, but is never entirely absent. The form assumed by such exudations is that of granules or globules, which are perfectly spherical, and present a dark distinct edge. These spherical bodies vary in size, being sometimes exceedingly minute, at other times as large as 1-60th of a millimetre, or even much larger; the variation is generally considerable in a single portion submitted to examination. Owing to their powerful refraction of light, they present a brilliant white centre and a dark circular rim, which is darkest towards the external edge.

The composition of these granules and globules has been already adverted to. The oil which forms their central portion is probably derived from the serolin, and the protein envelope from the fibrin or albumen of the blood-plasma. When the albuminous element is in large quantity relatively to the oil, the granules found are small, verging into the minutely molecular appearance; when the reverse of this is the case, they occur mostly in the form of large globules, of which the investing membrane is thin and readily ruptured by pressure. The prolonged action of acetic acid also frequently dissolves the membrane, and allows the contained fatty particles to coalesce. Caustic potass dissolves, after a time, both the albuminous and the oily element.

The mode in which the fatty granules or globules are disposed within the tubes of the kidney, next demands attention. The existence of fat in this situation was first distinctly recognised and described by Gluge as a diseased condition of the kidney, to which he gave the name of cirrhosis[1] or stearosis, with the view of distinguishing it from the states which he has described as inflammatory. In Germany, although many authors have written upon this subject, none appears to have in any way added to Gluge's later researches in his *Atlas der Pathologischen Anatomie*. In this country, the memoir of Dr Johnson of London is the only one, I believe, yet published on this subject.

[1] Gluge uses the term cirrhosis, both in the liver and kidney, to denote the pale and yellow rather than the granulated state of the organs.

Fig. 7. Fig. 8.

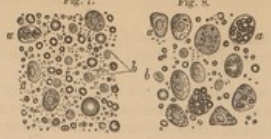


Fig. 7. Fatty granules and globules as seen in fluid scraped from the cortical substance of a very pale and soft kidney (slightly granulated). The existence of a large number of free nuclei of epithelium cells, as at b, b, *indicates the imperfect formation or disruption of these cells. Some of them are, however, seen entire, as at* a, *and contain fatty granules in different proportion. In this exudation the oil is relatively in large, the albumen in small quantity. (250 diameters.)*

Fig. 8. Fluid scraped from cortical substance in kidney of Biggie (see p. 811). The entire epithelium cells (a a) *are in much larger proportion than in the last case. Some of them are nearly clear, others filled in different proportions with granules, constituting the forms of cell described by authors as inflammation globules, exudation cells, fatty corpuscles, granular cells, &c.. Free nuclei* (b) *and free fat granules are also seen in considerable numbers. The proportion of oil is in this case also relatively large. (250 diameters.)*

According to my observations, fatty exudations from the tubes present themselves under two different aspects: *First*, free molecules, granules, and globules, intermingled with the cells and nuclei of the secreting structures; *Second*, similar granules, &c., enclosed within the cell-wall, between it and the nucleus. In regard to the first of these forms it is not necessary to repeat what has been already said; but the second demands a few words of explanation.

Dr Johnson is certainly the first who has stated distinctly, and kept constantly in view, the fact of the accumulation of fatty granules in the glandular epithelium of the kidney, having been guided in doing so by the facts previously stated by Bowman with respect to the liver. In regard to these facts there is no doubt; nor is there any doubt that their disregard by the continental writers on the kidney, has introduced much needless complexity into their descriptions.

Fatty deposit may exist in the cells of the kidney in large or small granules. It sometimes takes the form of a nearly molecular deposit; in this case it forms a mere shading, obscuring the nucleus, and rendering the cell more or less opaque. At other times, cells may be seen in different states of fulness, their contents being granules of nearly equal size, and not larger than from 1-500th to 1-300th of a millimetre. This is the granular corpuscle or inflammation globule of German writers. Again, the contained granules may be very unequal in size, the cell being irregularly distended or partially filled with granules and globules, from 1-500th up to 1-100th of a millimetre. It is rare in the kidney to find the contained granules so large as they are observed in the liver, where they not unfrequently fill nearly the entire cell. These appearances are illustrated in Figs. 7 and 8.

Fig. 9.

Fig. 10. Fig. 11.

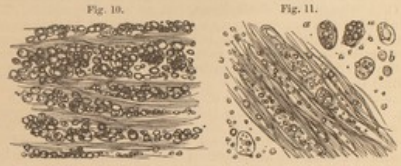


Fig. 9. Section of the cortical substance in a granulated kidney; the tubes are filled with granules and molecules of fatty or oleo-albuminous exudation. A Malpighian body is also occupied by exudation. The granules are mostly of small size. The tubes appear somewhat shrunk, the fibrous tissue having contracted around the deposit. (250 diameters.)

Fig. 10. Exudations, consisting of fatty globules of considerable size, in tubes of kidney of Biggie (see p. 811). (250 diameters.)

Fig. 11. Scattered granules in tubes of the pyramidal substance, in a kidney in which many of these were obliterated; a a, *cells from cortical substance of the same kidney;* b, *nuclei from do. (250 diameters.)*

In the tubes the fatty granules may appear to occupy the whole cavity (Figs. 9, 10), or to be sparingly disseminated (Fig. 11). Here, too, they may present every variety of size—from the smallest perceptible points or molecules, up to twice the size of the nuclei, in which case they are generally free. The tubes containing the fatty granules sometimes appear distended, at other times smaller than natural, as if they had contracted around the fat (Fig. 9). All these appearances are best seen in sections by Valentin's knife; and, when the tubes are displaced or roughly handled, the fatty deposit is very apt to be squeezed out of them.

It never happens that the whole of the tubes and cells of a kidney are equally the seat of fatty deposit. Even in the most diseased kidneys some healthy cells and tubes can generally be found; and, on the other hand, it is not unusual in kidneys apparently quite normal, to meet with a few cells containing fatty granules in greater or less number. This is, however, a departure, though a trivial one, from the strictly normal state of the cell.

The general description of the appearances produced in the kidney by the occurrence of deposits in the tubes (pp. 796-8), is applicable with peculiar force to the oleo-albuminous exudations, which are of

all others the most persistent, and the most frequently disorganizing in their effects.

The following cases are selected with a view to exhibiting the different stages, and most characteristic appearances presented by this form of exudation into the tubes.

Observation IV.—*Mottled Smooth Kidney—Partial and slight Exudation of Fatty Granules in the Epithelium Cells—Deposit of Urate of Ammonia—Phthisis—Pericarditis.*—John Young, æt. thirty-eight, printer, admitted into the Royal Infirmary, October 26, 1847, under the care of Dr Robertson: had for three years suffered from cough and dyspnœa, latterly attended with night sweats, diarrhœa, and rapid emaciation. He had observed slight hæmoptysis on one occasion two years before admission. He had never had pain in the chest. On admission there was aphonia, but no urgent symptom. The normal sounds of respiration were obscure, and supplanted by mucous and subcrepitant râles, particularly in the left lung, where there was comparative dulness on percussion at the upper part as well as at the lower dorsum. He died on the 12th November, having suffered from increased cough and dyspnœa, constipation and nausea.

On examination the lungs were found extensively affected with miliary tubercle. There was dense fibrous induration, of an iron-gray colour, at the upper part of both lungs, affecting most extensively the left; and a few small dry caverns existed in the upper lobes. The pericardium contained a turbid serum, with a few shreds of lymph. The liver weighed above 4 lbs., and presented on section a pale waxy appearance; it contained rather small fatty granules in nearly all the cells. There were a few tubercular ulcers in the ileum. The kidneys weighed each above 5 oz. They presented a smooth surface, and were of firm consistence. The cortical substance was of uniform density, but slightly mottled throughout from the greater vascularity of some parts than others; the gradations of colour, however, passed insensibly into one another. The vascular injection was nowhere absent, but was on the whole diminished.

On microscopical examination, the tubes and their contained nuclei were very obscure till after the addition of acetic acid, which had the effect of rendering them perfectly distinct, apparently by the removal of a fine molecular haze. The cells were then seen to be well formed and mostly entire, but they contained in many instances fatty granules from 1-300th to 1-120th of a millimetre in diameter, which were also seen to fill some of the tubes. These could be removed entirely by ether. (When a thin section of a kidney is to be treated with ether, it should be placed, carefully spread out by needles, on a plate of glass, which should be gently inclined, and ether should then be poured drop by drop over the whole surface of the section; this will generally remove much of the oil. The section should then be taken up with the forceps, spread on a clean plate of glass, and treated with water or acetic acid in the ordinary manner. The process may require to be repeated.)

This case presents a good example of the slightest form of the fatty exudation. The organ is mottled from the unequal distribution of the vascular redness; it is slightly increased in volume; its consistence is not altered; its vessels are nowhere obliterated. The secreting cells are well formed, and the presence of an oily deposit in them is the only abnormal appearance.

The fatty exudation was in this case, as very frequently occurs, common to the liver and kidney, and was probably indicative of no special morbid tendency in either organ, but rather of a cachectic state of the system, in which the respiratory, secreting, and nutrient

functions suffer a common decay. There was no symptom calculated to lead to an examination of the urine.

Observation V.—*Pale Kidney, with general Fatty Deposit in the Tubes—No Granulations. Fever? Delirium Tremens.*—Robert Kirkwood, æt. fifty-six, was admitted into Dr Paterson's ward in the Royal Infirmary, on December 7, 1847, with symptoms of delirium tremens. He was ascertained to have been of intemperate habits. He died three days after admission, December 10th.

On examining the body (December 12th), there was considerable serous effusion into the subarachnoid spaces and the ventricles of the brain. The spleen weighed fifteen ounces and was softened, as were most of the organs. The kidneys weighed each four and a half ounces; they were exceedingly pale; the surface was smooth and pale, presenting only a few stellar veins; the Malpighian corpuscles were much obscured, and under the microscope appeared destitute of blood. The tubes obviously contained, throughout the cortical substance, a number of oil granules and globules of different sizes, up to the 1-100th of a millimetre. On examining the contents of the tubes separately, the oil granules were found to be mostly floating loose; the cells imperfectly formed; their membrane thin and delicate, and many of the nuclei free. (See similar appearances in Fig. 7.)

This case differs from the last chiefly in the imperfect development of epithelium, and in the more general diffusion of the exudation, which gave rise to greater paleness of the organ.

Observation VI.—*Very general Deposition of Fatty Granules in the Tubes of the Cortical and Pyramidal Substances—No Granulations—Incompetency of Aortic Valves—Hypertrophy of Heart.*—Alexander Durham, æt. twenty-nine, plumber, admitted into Edinburgh Royal Infirmary under Dr Douglas, on March 5, 1848. He had been affected from the beginning of the year with cough, dyspnœa, and palpitation, and had on one occasion expectorated a small quantity of blood. There were on admission accelerated respiration and orthopnœa, sibilant râles in the chest, and the physical signs of aortic regurgitation, with hypertrophy of the heart in a very marked form. On the 11th March there was observed slight swelling of the limbs; the urine was tested and found to be albuminous, though faintly so; it was of good specific gravity, and 18 oz. (had been generally not more than 10 oz). The œdema increased till his death, which happened rather suddenly on the 15th. On the 13th he had slight hæmoptysis, with temporary relief to the symptoms; but the orthopnœa continued to the last.

The heart weighed 22 oz., through hypertrophy chiefly of the left ventricle; the aortic valves were incompetent; the lungs were extensively studded with hemorrhagic extravasations. All the abdominal organs were normal, except the kidneys; these were above the normal weight, and dense. They were, however, very friable, so that in removing the capsule small portions of the cortical substance adhered to it. The vascular striæ opposite the bases of the pyramids were distinct; the vascularity of the capillaries and the Malpighian bodies not appreciable. The cortical substance was slightly and minutely mottled; the striæ of the pyramids, from their bases through two-thirds of their length, were marked by the presence of a white opaque deposit.

Microscopic Examination.—The greater part of the deposit proved to be granules and globules of fat, which appeared to fill the tubes in every part of the organ; it was mostly unconnected with cells. The epithelium was very imperfectly developed, many of the nuclei being free. The tubes themselves seemed in no way displaced or deformed.

In this case there was albuminous urine, constantly deficient in

quantity, and generally loaded with urate of ammonia, which formed a portion of the deposit in the tubes of the kidney. There was no marked permanent disorganization of the organ; but the deposit in the tubes was as general as I remember to have seen it without such disorganization taking place.

It is to be remarked, that in the urine there was no apparent sediment except that of amorphous urate of ammonia, which disappeared on heating, leaving the urine clear. It is therefore probable that it did not contain oil in any appreciable quantity. It is to be regretted, however, that a chemical examination of it was not made.

Observation VII.—*Fatty Granules and Globules in the Tubes, extending into the Pyramidal Portion—No Granulations—Fever?*—John Biggie, æt. twenty-six, Irish labourer, admitted July 26, 1847, into Ward 3, Edinburgh Royal Infirmary, in a state of extreme prostration, with impaired intelligence, weak pulse, brown and dry tongue. He was stated to have been eight days ill of fever. His abdomen presented slight swelling, with indistinct fluctuation. On the 28th, abdominal swelling and fluctuation increased; prostration greater; some yellowness of the conjunctivæ and surface. Died. The urine could not be procured.

On dissection, the cellular tissue, which contained within its substance a good deal of fat, was infiltrated with serum. The serous cavities also contained more or less of fluid. The liver weighed 4 lbs.; it was of a nut-brown colour; its surface presented numerous mammillary projections, varying from the size of a mustard seed to that of a pea; a similar granulated disposition pervaded the whole organ. The gall-bladder contained a small quantity of bile. The spleen weighed nearly 3 lbs.; its consistence was diminished, and its colour dark. The kidneys were of normal size, flabby, and soft; the capsule brought away with it small portions of their tissue. Both cortical and pyramidal substances were very pale in colour. (Condensed from the report of Dr Waters, then pathological clerk.)

The microscopical appearances of the kidneys in this case are delineated in Figs. 8 and 10. The tubes were for the most part full of oil globules of considerable size, mixed with smaller fatty granules. Some of them were, however, nearly healthy. The deposition of fatty matter extended to the pyramids, from a section of which Fig. 10 was drawn. A section of the cortical substance presented an appearance under the microscope very like Fig. 9, but with occasionally larger globules. The epithelium cells were mostly entire and well formed, but in their interior presented various degrees of granular exudation.

In the two last observations, the deposit was connected with extreme diminution of the activity of the renal function, and this without any obvious destruction or disorganization of the substance of the organ. So far as can be judged from the pathological appearances alone, there seems to be no adequate reason why a kidney in the state above indicated should not return to health, the deposit in the tubes being removed or reabsorbed. If, on the contrary, the tendency to this deposition should continue, it seems to be inevitable that the consequence must be a complete breaking up of the structure of the organ, and such obstruction to its functions as must lead to speedy death. I have several times seen kidneys which appeared to have undergone this form of degeneration to a greater or less extent; but none so

marked as in the following case, where both the liver and the kidneys seemed converted into a species of atheroma.

Observation VIII.—*Complete Atheromatous Degeneration of Liver and Kidneys—Pus in Hepatic Vein.*—Mary M'Gonagil, æt. thirty, admitted Jan. 26, 1848, into Edinburgh Royal Infirmary, under the care of Dr Douglas, with marked enlargement of the liver, apparently of five years' standing. Before this she had nine still-born and premature children. She had been subject to obstinate costiveness, and pains of the abdomen and loins. The constipation and tympanitic distension of the abdomen were overcome by remedies, and she continued pretty well till the 3d February, when marked tenderness of the epigastrium and right chest became developed, with pain on motion of the corresponding arm. On the 7th there was great and indefinite distress; the right front of the chest was the seat of a firm, diffused, and painful swelling, seated apparently in the cellular tissue. Tenderness extended from this to the axilla. The same day she died. (The urine was always very pale and scanty.)

On dissection, the surface was very pale; the thoracic organs were healthy; the liver was of large size, and exceedingly irregular form; its surface was every where uneven, at some places puckered and depressed; its substance was soft, friable, in some parts almost diffluent; it presented no appearance of structure or vascularity; scattered throughout were dense semi-transparent masses, which had the appearance on section of pieces of fibro-cartilage. The hepatic vein contained a good deal of pus. The kidneys were of the normal size, soft, and flabby; on stripping the capsule, the smoothness of the surface was seen to be diminished; this appearance, however, was found not be the result of the development of granulations of Bright, but of a peculiar condition of the cortical substance, which had lost its characteristic appearance, and became converted into a homogeneous friable mass; this, however, possessed greater consistence than that of the liver above described.

Microscopic Examination.—The texture of both liver and kidneys seemed converted into fatty granules and globules of different sizes, amid which very few secreting cells could be seen. In the kidney, however, by diluting the mass with water, fragments of tubes could be observed greatly attenuated, sometimes consisting of basement membrane alone, sometimes with a few nuclei attached. Many of these fragments were empty, others contained deposit in large quantity, and were very irregularly distended. A few loose Malpighian bodies, empty and shrivelled, were also seen. The cartilaginous-looking matter in the liver was composed of peculiar thin and delicate transparent laminæ, wholly unlike any of the normal tissues of the organ; they were insoluble in hot ether and in strong nitric acid, but I did not pursue the investigation into their nature further.

The preceding observations afford sufficiently characteristic examples of the fatty deposit in that form in which it is infiltrated in various degrees through the whole of the tubes. Very frequently, however, especially when the disease is very chronic in its character, the exudation tends to occupy particular points of the organ, leaving many of the tubes free; the points so occupied are the granulations of Bright, the formation and progress of which I have already explained. The observation which follows will illustrate this.

Observation IX. *Irregular Granulations* (early stage) *throughout the Cortical substance of Kidney—Exudation consisting of small Fatty Granules—General Dropsy.*—George Whitnall, a weaver, æt. thirty-seven, admitted January 14, 1848, into the Royal Infirmary, under the care of Dr Paterson. He had laboured under general dropsy for about twelve months, which became ex-

cessive shortly before admission. He was much prostrated, and died next day (January 15th). The urine was not procured.

The lungs were highly emphysematous; the heart slightly hypertrophied. The abdominal viscera were healthy, with the exception of the kidneys. One kidney weighed 8 oz., the other 9½ oz. The capsule was easily stripped. The surface, which was pale, approaching to a flesh-colour, with a few stellar veins, was interpersed with yellowish opaque granulations, irregular in form, and very slightly elevated. They did not exceed the size of a small pin's head, and were pretty uniformly scattered throughout the cortical substance, which was completely anemic, and sharply divided from the highly injected pyramidal substance. The latter contained a few points of yellow deposit, near the bases of the pyramids. Both cortical and tubular substance were hypertrophied.

Microscopic Examination.—The organ was so friable as not to admit of a moderately thin section being made with Valentin's knife. Owing to this circumstance the structure was very indistinctly discovered; but there was seen, disposed in irregular masses throughout the cortical substance, an exudation composed of very fine and small granules, having the refraction of oil, and not affected by acetic acid. The nuclei were abundant, the complete cells few.

This case presents an instructive instance of the earliest form of the granulations of Bright. It is that figured by Rayer (Atlas, Plate VIII. Figs. 1 and 2), and except in the slight prominence of the granulations on the surface, and their somewhat yellowish colour, corresponds exactly to his fourth form of Bright's disease, in which he describes the granulations as "petites taches d'un blanc laiteux," and as appearing "sous la forme de lignes irrégulières, comme floconneuses, qui semblent se continuer avec les stries divergentes des cônes tubuleux." The small size of the granules, forming the exudation in this case, is probably owing to the absorption of part of the oily matter, in consequence of which there is an altered relation between the proportions of the constituent parts of the exudation—(See ante, p. 21). The irregular form of the granulations, their small size, their copious diffusion through the cortical substance, and their existence even at the bases of the pyramids are all accounted for, if we consider this kidney to have passed through a stage of general fatty infiltration, similar to that of Durham or Biggie (Obs. VI. and VII). If, in either of these kidneys, the deposit had been absorbed from some of the tubes, and persisted in others, and had then become consolidated and opaque from the removal of part of its fluid constituents, they would have presented exactly the appearances here indicated.

May, then, the early stage of the granulations of Bright be considered as, in some instances, the retrograde movement of a still more threatening condition,—viz. the universal fatty infiltration?

The progress of the granulations of Bright is connected with other changes of a different character, such as atrophy of the surrounding tubes, obliteration of vessels, &c.; and will therefore come under consideration in the other sections of this memoir. Enough has been said for the present to show their connexion with the fatty exudations in the tubules.

c. Exudations in the form of Pus.—The occurrence in the cortical substance of deposits having all the external characters of pus, is not very uncommon. Their most usual form is that of small abscesses, rarely exceeding the size of a pea, and frequently much smaller, sometimes confluent, and irregularly disseminated through the cortical substance. They are generally surrounded by more or less deep vascular redness; this, however, is limited to a narrow rim around the deposit; the remaining portions of cortical substance being either natural in appearance, or paler than usual. These appearances are well delineated in Plate II. Figs. 1 and 2, of Rayer's work.

The formation of abscesses having a distinct limiting membrane, or surrounded by condensed tissue, is, in the kidney, of extremely rare occurrence. I have already related a case (Observation II.) where a cavity of this sort was found; but the appearances of the contained matters to the naked eye and under the microscope had no resemblance to those of pus.

The following case is of considerable interest in several points of view, and tends, in connexion with other observations, to elucidate the formation of pus in glandular organs:—

Observation X.—*Purulent Deposits in Kidney and Lung—Sloughing Abscess of Spleen—Peritonitis.*—Cecilia Hall, æt. twenty-seven, shoebinder, admitted Dec. 23, 1847, under Dr Douglas. Had been affected for eight days before admission with considerable fever, with rigors, vomiting, and abdominal pain. On admission the vomiting had ceased, the other symptoms continued. There was tenderness with dull percussion in the hypogastrium and left iliac region. There was also a good deal of cough. This last symptom increased considerably during the next three weeks, and the right lung became somewhat dull on percussion, with tubular respiration posteriorly, and mucous and subcrepitant râles in various parts of the chest. The abdomen became tympanitic, but the tenderness disappeared. She died much exhausted, but without pain, on Jan. 30, 1848, one month after admission.

On dissection, the heart was soft, but not altered in structure. The lungs were emphysematous, and much engorged; the upper lobe of the right lung scarcely crepitated on pressure, but floated in water. In various parts of both lungs were nodules of pulmonary tissue, which were quite dense, of a greyish colour, and some of which contained in their centres yellow creamy pus. The cavities containing the pus were found by the probe to communicate with the smaller bronchi, and were lined by a membrane having an exact resemblance to mucous membrane. The spleen was slightly enlarged and soft; in its substance, towards the convex surface, there were two or three masses of soft exudation, one of which was surrounded by a line of ulceration, and was in part detached, projecting from the surface of the organ. Around this part, and between the surface of the spleen and the diaphragm, a layer of soft yellow lymph was thrown out, which connected the opposing surfaces of the peritoneum. Stomach and intestines healthy. The kidneys were of the natural size; one of them contained in the cortical substance numerous small abscesses, from the size of a pin's head to that of a pea; several of these occurred in groups towards the surface of the kidney. The abscesses were not surrounded by any indurated substance, but by a vascular rim of a rose colour, and about half a line in diameter. They contained a bright yellow pus. A little pus was also in one or two places infiltrated into the tubular cones, near their base. The pelvis of the kidney was slightly vascular, but contained no fluid. The peritoneum

lining the bladder and pelvis was somewhat vascular, and blood was extravasated in considerable quantity in the sub-peritoneal cellular tissue, both in the pelvis and about the situation of the umbilicus.

The microscopical examination of the fluid from the minute abscesses in the kidney showed the following objects:—1st, Spherical granules and molecules insoluble in acetic acid. 2d, Rounded corpuscles (Fig. 12, *b b*) of the usual size of pus corpuscles (1-100th of a millimetre), and of a dark granular appearance. On being treated with acetic acid, they became more transparent, and showed in their interior a greater or smaller number of spherical granules, and occasionally an ill-defined nucleus. On the whole, however, they underwent less change than is usual in pus from an ordinary abscess on the addition of acetic acid. 3d, Larger corpuscles from 1-30th to 1-50th of a millimetre in diameter (Fig. 12, *a a*), of nearly spherical form, and crowded with granules; on adding acetic acid they underwent little change. 4th, Extremely delicate cells (Fig. 12, *d d*) of the same size as the last mentioned, and containing a granular nucleus, about 1-100th of a millimetre, and exactly similar to the pus corpuscles described above (2d); the cell wall extremely attenuated, readily yielding to pressure, and disappearing completely after the addition of acetic acid; the space between the cell wall and the nucleus perfectly free of granules and molecules, and filled apparently with transparent fluid. On scraping the walls of one of the small abscesses gently with the point of the knife, and examining the adhering tissue, fragments of tubes were discovered, which contained all the above elements, and in which the cells and nuclei described appeared to have taken the place of the normal epithelium. The smaller corpuscles above mentioned (2d) were in greater abundance than the other elements. In the other parts of the kidney, the microscope showed nothing abnormal.

In the lung the pus presented appearances so exactly similar to the above, that the same description will apply to both.

Fig. 12. Fig. 13.

Fig. 12. *Pus and fragment of tubule from small abscess in the kidney of Hall, 250 diameters.* a a, *Granular cells of the size and appearance of granular epithelium.* b b, *Smaller pus corpuscles, presenting a very granular structure (most of them have been represented too flat by the engraver).* d d, *Corpuscles like the preceding, but surrounded by a cell with clear contents and an exceedingly delicate wall. The tube is seen to be filled mostly with the bodies* b.

Fig. 13. *The corpuscles treated with acetic acid.*

In the *Monthly Journal* for February 1848 (p. 589), Dr Bennett has described and figured pus corpuscles, which appeared as granular nuclei, surrounded by a delicate and transparent cell-wall. These corpuscles he has found in abscesses of the lung and kidney, and also in grey hepatization of the lungs; and he thinks that at a later stage of their formation the cell-wall disappears, leaving the nucleus as the mature pus corpuscle. From having seen Dr Bennett's previous demonstrations, and had my attention turned to the subject, I had no difficulty in recognising the bodies, *d d*, as identical with those described by Dr Bennett. But what appears to me worthy of attention in the present case is, 1st, the coexistence of these bodies with the cells *a a*, which are undoubtedly the granular epithelium cells described in a previous part of this memoir (see ante, p. 22); 2d, the existence both of these bodies and the smaller pus corpuscles *within* the tubules, where they appeared to take the place of normal nuclei and cells; 3d, their existence in the lungs, in cavities formed by dilatations of the smaller bronchi: in short, their formation both in the lung and kidney in connexion with a mucous surface.

It is well known to microscopic observers, that the pus formed on the surface of mucous membranes seldom presents the clear and definite reaction with acetic acid characteristic of normal pus corpuscles. It has even been at different times supposed that the pus corpuscle is formed from the epithelium cell; and though this doctrine is undoubtedly attended with many difficulties, and has never been held by pathologists generally, it seems to be worthy of further investigation in cases like the present. The resemblance in size and form of the bodies *b b* to a granular epithelium nucleus, and of *a a*, *d d*, to granulated and non-granulated epithelium cells, appears, especially when taken in connexion with their position *within* the tubules, and the complete absence of normal epithelium, to be somewhat more than an accidental circumstance.

The symptoms in this case were referrible to the abdominal and pulmonary lesions described; but they were attended from the first by a marked typhoid depression, and a continued languor and exhaustion, which justified a suspicion of idiopathic fever superadded to the local disease.

In several other cases which have occurred in the Royal Infirmary of purulent deposits in the kidneys, a similar typhoid state has existed; in all, however, there have been numerous other lesions, and sometimes abscesses in other parts of the body, indicating a general tendency towards the formation of pus. The blood in these cases presented no unusual appearance.

2. *Exudation within the Malpighian Bodies.*—The granular (oleo-albuminous) form of exudation above described as so frequently occupying the tubes of the kidney, is also occasionally found within the capsules of the Malpighian bodies. When in large quantity in this situation, the tuft of vessels which normally fills the capsule, is completely compressed and shrunk, in most cases invisible. Where the exudation is in smaller quantity, however, it frequently adheres to the interior of the capsule and the exterior of the tufts, without materially affecting their form.

Exudation in this situation is generally accompanied by similar exudation, in greater or less abundance, within the tubes. The

anatomical relations of these parts, as now generally understood, would, indeed, entitle us to expect that the pathological conditions of the one should be shared by the other. An exceedingly good illustration of these associated conditions will be found in Fig. 9 (see ante, p. 23). While, however, many cases of this sort have occurred to me, I have met with a still larger number which confirm the statement of Dr Johnson (*Med. Chirurg. Trans.*, vol. xxix. p. 4.), that the exudation within the tubes often occurs to a very great extent, without the Malpighian bodies being at all involved. More rarely a limited amount of deposit occurs within the latter, when there is comparatively little within the tubes. The cause of these differences is very obscure; nor does the examination of it appear to promise any results of importance, in the present extremely imperfect state of our knowledge as to the special functions of the Malpighian bodies.

3. *Exudation in the Inter-Tubular Tissue.*—In cases where oleo-albuminous exudation is in small quantity, it frequently appears to be disposed without any distinct relation to the tubes; and, where it is in very large quantity in the tubes, it sometimes appears in the interstices of the areolæ, as is represented in Fig. 9. In kidneys which are the seat of firm opaque granulations, a section of these frequently presents a dark opaque mass, covering a large portion of the field of the microscope, and showing no trace of arrangement; the deposit must, therefore, either have broken up the structure entirely, or completely occupied every vacant place. In all these cases, however, it is extremely difficult to determine by actual observation that the exudation is external to the tubes; and I am not a little disposed to doubt the occurrence of this condition, or at least to consider it as secondary to the complete occlusion of the tubes by exudation.

4. *Partial Distribution of the Oleo-albuminous Exudation.* (*Plaques Blanches de Lymphe Plastique*, Rayer.)—I have already described the formation of granulations as dependent on the accumulation of deposit in particular groups of tubules in the cortical substance. In such cases, however, the affection is probably at first general; they are very different from the form now to be described, in which the deposit is quite limited in extent, and isolated.

There are occasionally met with on removing the capsule from the surface of a kidney, irregular patches of a paler colour than the rest of the organ, sometimes a little elevated, sometimes depressed below the general surface. Their boundary is quite abrupt, and they are frequently surrounded by a well-marked rose-coloured areola, extending more or less into the surrounding substance. On making a section of these patches, they are found to penetrate into the cortical substance, and sometimes even a certain way into the pyramids. The vascular areola, when present, extends round them in every direction, and is found on examination to consist of highly

injected Malpighian bodies and capillaries, with or without extravasation. The colour of the patches varies from yellowish-gray to a gamboge-yellow; their consistence is generally firm. On microscopic examination, they present a large amount of exudation, varying from the molecular to the large granular form. In some cases the tubes may be seen filled with exudation; in others, they appear to be in great part obliterated. In one case I found the Malpighian bodies quite free of exudation; they preserved their usual arrangement, and were readily discoverable by a simple lens on the surface of the section. The parts of the kidney not involved in the deposit, generally present no abnormal appearance.

Various illustrations of this species of deposit are to be found in Rayer's work. (See Pl. I. Fig. 6. Pl. V. Fig. 2. Pl. XXXIV. Figs. 2, 6.) He has figured it in various stages and under different names, as *Nephrite simple* and *rhumatismale*, and *Hemorrhagie*. Its origin and progress are very obscure, and it has not been satisfactorily connected, either with other morbid states, or with any peculiar symptoms. I have seen it in connexion with fever, with puerperal convulsions, with erysipelas and dementia, and in several cases where no account of the symptoms could be procured. Dr Bennett possesses a most remarkable preparation and drawing of a case in which such deposits were most extensively present, and left very little intervening sound tissue. The affected kidney had a most singularly variegated appearance.

II.—Lesions affecting chiefly the Vascular System.

In passing to the consideration of the morbid changes which occur in the vascular system of the kidney, the conditions of sanguineous congestion and extravasation on the one hand, and anæmia on the other, would fall to be described first in order. But the simple hyperemic and anemic states of the organ have been noticed so fully in the anatomical introduction (see pp. 2-4), that little more remains to be said on this subject.

Congestion followed by permanent obliteration of the Capillaries of the Cortical Substance.—Under this head I have to describe a form of lesion in the kidneys which, although certainly of less frequent occurrence than those characterised by exudation, is of a pathological and practical interest in no way inferior to any other.

The appearances most characteristic to the naked eye of this form of lesion, are those so admirably figured and described by Rayer as the second form of his "néphrite albumineuse." The kidneys are generally increased in size, sometimes very remarkably so. Their consistence varies; they are sometimes more flaccid than in the natural condition, but always preserve considerable tenacity. The surface is either quite smooth, or more or less depressed and furrowed. The venous vascularity assumes to a considerable extent the stellate form; the polygons are mostly absent; and the extreme

irregularity and abruptness of distribution of the superficial veins gives to the surface a variegated or "marbled" appearance, which is quite characteristic of this stage of the affection. (See Rayer, Plate VI. Figs. 2, 3, 5; Bright, Plate II. Fig. 1.) Occasionally, also, amid this unequal injection there are to be found scattered petechiæ, indicating recent extravasations of blood into the tubes. On section the cortical substance has considerable volume, and presents a smooth, glistening, almost semi-transparent appearance, which cannot be better distinguished than by the term *waxy*. It may partake in a slighter degree of the variegated character of the surface; more commonly it is of uniform appearance, and of a yellowish or fawn-colour, sometimes verging into a pale flesh tint. The vascular striæ of the cortical substance are generally to be traced by a more or less distinct injection, and a few injected Malpighian bodies, or petechiæ of extravasation, are sometimes dispersed through the section. (See Rayer, Plate X. Fig. 3.) In other cases a little further advanced, both the striæ and the Malpighian bodies are nearly destitute of blood. (Rayer, Plate X. Fig. 1; Bright, Plate II. Fig. 1.) The pyramids frequently retain their normal vascularity; sometimes, however, they are of a pale colour, and their bases are indistinctly marked,—a condition which indicates the progress towards a further disorganization.

When a kidney in this condition is carefully and minutely injected, the greater proportion of the cortical substance remains impervious; the injection, however, can frequently be made to penetrate as far as the cortical striæ, and even to some of the Malpighian bodies. (See Rayer, Plate X. Fig. 2; Bright, Plate II. Fig. 3.)

From these circumstances it is obvious, that the lesion above described consists in an obliteration or obstruction of the capillary system of vessels throughout the organ, and a partial obliteration of the veins on its surface. There is also every probability that this condition is secondary to one in which there is a high degree of congestion of the organ. The extravasations, the occasionally injected Malpighian bodies, and the highly injected, though partially distributed, stellar veins, leave no doubt that the state of congestion described as the first form of albuminous nephritis by Rayer, is really the antecedent of the present or second form.

To any one who is familiar with the *marbled* and *waxy* kidney here described, there can be no difficulty in recognising a further stage of the same lesion, in which the organ is perfectly pale both on the surface and on section, with the exception, perhaps, of a very few stellated superficial veins. The kidney in this stage (the transition to which seems to be represented in Rayer, Plate VI. Fig. 4) is still heavy and voluminous; it acquires additional firmness and elasticity, and assumes much of the general appearance of a true non-vascular texture. It varies from a light yellow to a fawn-colour, which extends to the pyramids, the bases of which become still more confused and intermingled with the cortical substance than in the

marbled kidney. The capsule is frequently more firmly adherent to the external surface than in health.

From the pale and yellow appearance of the kidney in this stage, it is very apt to be mistaken, even by a practised eye, for an extreme degree of the fatty degeneration. A well-marked example, indeed, will hardly give rise to this error, if attention be directed to the degree of firmness of the organ, the peculiar lustrous character of the cut surface, and the entire absence of the opaque granulations of Bright, or of that dull tint which distinguishes the excessive degrees of the fatty disease. The appreciation of these characters is, however, more difficult where, as sometimes happens, exudation is also present; and the distinction which has escaped the acute observation of M. Rayer, has undoubtedly been overlooked by many other observers.[1]

The microscopic characters of this lesion are chiefly negative. There is not unfrequently an entire absence of exudation; indeed, in the most marked cases of the lesion, I have seldom found even the slightest trace of any abnormal deposit. Occasionally, however, there is a very minute quantity of fatty exudation in the tubes, generally in very small granules, and scattered throughout the organ. The tubes are either natural, or in the advanced stages pass into some of the states hereafter to be described. The capillary vessels surrounding the tubes are not visible, and in their place there is fibrous tissue, which in this form of lesion always appears somewhat exaggerated. The Malpighian bodies are also frequently seen in process of obliteration, and surrounded by dense capsules of fibrous tissue. The epithelium is frequently altered in character, but its changes follow no fixed rule.

The absence or scantiness of exudation, taken in connexion with the extent of degeneration appreciable by the naked eye, are amply sufficient characters to distinguish this lesion from the extreme stages of the fatty disease.

I shall add three observations which illustrate the different stages and varieties of this important form of renal degeneration. I do not at present mean to enter on a consideration of the symptoms, further than to say that both Bright and Rayer have figured it as being connected with albuminous urine and dropsy; and these facts entirely concur with some of my own observations. This lesion is therefore undoubtedly one form of what is commonly called Bright's disease.

Observation XI.—*Marbled and Waxy Kidneys* (without exudation) *Bronchitis—Large Abscess in Labium.*—A woman was admitted into the Royal Infirmary in a state of great exhaustion from an abscess, the size of a child's

[1] Plate VI. Fig. 4 of Rayer's work, is probably an example of the waxy pale kidney; Plate VII. Figs. 2, 3, 4, of the fatty disease. The distinction is sufficiently evident even in the plate. All of these are referred by Rayer to the third form of "Néphrite Albumineuse."

head, connected with the external parts of generation. This was opened, but a few days after admission she died.

On dissection, November 28, 1847, the lungs were found much engorged, and the bronchi full of fluid. The kidneys were greatly enlarged, and weighed 8¼ and 6¼ oz. They were nearly of the usual consistence. The surface was very uneven, from being marked all over with irregular depressions and furrows. There was a considerable amount of venous injection, but very irregularly distributed; so much so, that the surface, which was at some parts quite bloodless, had an irregularly variegated appearance (not unlike the case of Sallaway, in Dr Bright's work, Pl. II. Fig. 1.) The depressions in the surface were mostly filled with stellated veins. On section, the cortical substance was of increased volume, particularly between the pyramids; these were broad at the bases, and the line of separation from the cortical substance was quite distinct. The pyramids were well injected; the cortical substance contained, in the line of its striæ, a few points of unusually distinct injection, but was generally anemic, being of an exceedingly pale and clear salmon-colour. The surface of the section was very smooth, and neither the section nor the surface presented the slightest trace of granulations. The mucous membrane of the pelvis of both kidneys was deeply injected with arborescent vessels. The renal veins were distended with dark blood.

On examination by the microscope, a few injected Malpighian vessels were seen; the majority were bloodless. The capillaries were uninjected. The normal epithelial cells were in great abundance; but neither in these, nor in the tubes, could any fatty granules be observed, although numerous sections were made for the purpose of determining this point.

The remarkable similarity in appearance of the section of this kidney to that in Rayer, Pl. X. Fig. 3, was noticed at the time. Both of them may be considered as good and characteristic examples of this affection, in a moderately early stage. The marks of recent vascular excitement were observable in the irregular injection of the surface, the congested state of the pelvis of the kidney, and the points of injection in the cut surface. On the other hand, the absence of exudation, and the smooth waxy appearance of the section, distinguished it from the fatty kidney. The increase in size and weight must have been owing to the great abundance of secreting epithelium within the tubes.

Observation XII. *Mottled Waxy Kidneys* (with slight exudation)—*Fatty Liver—Softened Spleen—Fever and Scurvy.*—Daniel Kean, æt. twenty-three, was admitted May 19, 1848, into a fever ward. In addition to the ordinary symptoms of eruptive typhus, he suffered from painful induration and ecchymosis in the calves of both legs, with spongy and ulcerated gums (symptoms in every way similar to those of the endemic scurvy of last year). For some time before admission he had lived very poorly, chiefly on bread and coffee. He died on June 6th.

On dissection, the lungs were congested, the heart and liver paler than usual, the spleen very soft, and slightly enlarged. The kidneys were slightly above the normal size, their capsules more firmly adherent than usual. Their surface was rendered uneven by the presence of shallow grooves and irregular dimples. The venous polygons were very obscure, being mostly supplanted by stellated and arborescent vessels, which were so distributed as to give the surface an irregularly mottled appearance. Over different parts of the surface were scattered petechiæ, varying from a dark purple to a slate colour. On section, the cortical substance appeared generally of a somewhat bright fawn

colour. The cut surface was perfectly smooth, and had a waxy lustre. Here and there were seen points and lines of injection, corresponding with the vascular striæ of the cortical substance; also a few larger and deeper-coloured petechiæ. The whole of the injection was very irregularly distributed. The pyramids were paler than usual, and the line of demarcation with the cortical substance was slightly irregular. The pelvis and calyces were normal. At one point of the cortical substance, in the left kidney, there was found a patch of rather light yellowish colour, about the size of a pea, which was surrounded by a very distinct and abrupt rose-coloured border; this, on minute examination, was obviously composed of highly injected Malpighian bodies.

On examination by the microscope, the fibres of the heart were found in most places occupied by small granules, which, however, did not generally completely obscure the striæ. The liver contained oil-globules in considerable numbers, some free, and others within the epithelium cells. In the tubes of the kidneys there could be seen in many parts of the cortical substance a few minute and scattered granules, but they were mostly filled by perfectly clear cells and nuclei, which were in preternatural abundance. The nuclei were mostly smaller than usual. The inter-tubular fibrous tissue appeared unusually dense, and no injected capillaries could be seen. A few Malpighian bodies were partially injected. In the part above described as surrounded by an injected border, the tubes and cells contained granular exudation in a quantity much more considerable than elsewhere.

In this kidney, we have the waxy degeneration accompanied by a certain degree of exudation, although not to such an extent as to give any special character to the lesion. The petechiæ are interesting in connexion with the scorbutic diathesis which existed; nevertheless, the state of the superficial veins, and the injected state of some of the Malpighian bodies, along with the fact of extravasation into the tubes, appear to indicate, as in the former case, a recent condition of vascular excitement. Whether the exudation occurred as a consequence of this condition, or of the general tendency to fatty degeneration, as shown in the liver and heart, is open to question. The former view appears, from the character and partial distribution of the exudation, to be not improbable.

The succeeding case is an example of the more confirmed form of this degeneration.

Observation XIII.—*Pale yellow waxy Kidney* (without exudation)—*Enlargement of Liver and Spleen—Bronchial dilatation—Pneumonia.*—Isabella M'Kinlay, æt. twenty-three, admitted November 15th, 1847, into Ward 15 as labouring under fever. She was weak and emaciated, suffering from severe dyspnœa and cough, with fever and pain of the left side of the chest. Her complaints were of long standing, and she had had occasional diarrhœa. There were distinct physical signs of cavities in the left lung, and of general bronchitis. She was supported by wine, but continued in a febrile state, with much depression, and slight jaundice, and died December 1st.

On dissection, the heart was pale, but of natural size. In the left lung, which was much diminished in size, there were numerous large cavities. The vesicular structure of the lung was wholly obliterated, and the cavities were found to consist of dilated bronchi, the walls of which were much hypertrophied. The right lung was slightly condensed in the upper part. The liver was enlarged, pale, and firm. The spleen was large and soft. The kidneys weighed 9 oz. and 8 oz. The capsule adhered with unusual firmness. The surface was uneven and furrowed, at some points roe-like or botryoidal; but the projections here indicated had not the opacity and whiteness characteristic of the

granulations of Bright (for an illustration of this point, see Rayer, Pl. X. Fig. 10). The surface was perfectly pale, with the exception of a very few straggling arborescent veins. The whole cortical substance was of a pale straw colour, tumid, and perfectly anemic ; the pyramids were very slightly mottled from vascular injection, and appeared to present straggling radiations, which were prolonged indefinitely into the cortical substance towards the surface ; the bases of the pyramids were thus rendered very indistinct. The whole kidney was of much firmer consistence than usual. Pelvis and calyces normal.

The principal microscopic appearances were dilatation of the tubes, and entire absence of all appearance of vessels. The epithelium was in considerable abundance, and had a tendency to cohere in masses, bearing the form of the tubes. Many of the cells were also compressed. A doubtful trace of granular exudation was here and there observed ; but, after a most careful search, nothing very definite could be seen.

Some of the alterations in this kidney will be considered under the head of lesions of the tubes and epithelium. At present it is sufficient to say, that this case was considered by many persons who were present to be one of fatty degeneration of the kidneys, as well as of the liver and heart. Microscopic examination proved, however, that none of these lesions existed in any appreciable degree ; the paleness of the heart and liver being, I believe, the result of anæmia, while that of the kidneys was the indication of an advanced stage of the waxy degeneration.

Connexion of Congestion with the Process of Exudation.—I have already stated, that in general the degree of fulness of the vessels stands in an inverse ratio to that of the tubes ; and that accordingly an amount of exudation, so great as to produce distension of the tubes, generally produces a corresponding depletion of the vascular system. I have likewise shown, by reference to the works of Rayer and Bright, as well as by original observations, that paleness of the cortical substance is one of the most frequent characters of kidneys containing exudation.

A moderate or small amount of exudation, however, sometimes occurs in organs which present considerable vascular injection, and sometimes even a distinctly hyperemic condition. Such cases occur not unfrequently in the latter stages of heart disease. In only one or two cases have I found the vascularity much greater than usual ; in these the kidneys were of large size (weighing from six to eight oz.), and contained very little exudation. In one instance they presented on the surface the petechiæ indicative of extravasation into the tubes (as in Bright's fifth Plate) ; in this case there had been albuminous urine and dropsy during life, with obstinate hæmaturia during two months before death ; there was also intense dysentery and disease of the liver. The kidneys were otherwise diseased ; but the exudation was in small quantity, and in very minute granules. In another case (a woman who died of convulsions in the eighth month of pregnancy, and in whom an apoplectic clot was found in brain), the kidneys weighed four and four and a half oz. ;

they were firm in texture, and of a tawny orange colour, pretty generally diffused. This was found to proceed from the presence of blood in the tubes, intermixed with small fatty granules, which were in considerable abundance. There was, however, little capillary injection, and the Malpighian bodies were mostly bloodless. The urine in this case had not been examined.

The analogy between the results of exudation in the kidney, and those in the lung and other organs, would naturally lead to the idea, that, as in the red hepatization of the lung, the deposition of granular exudation is preceded and accompanied by vascular turgescence, so in the kidney there may be a form of the exudative process in which active congestion of the organ plays a part. Almost all pathologists since the time of Dr Bright, who have written on the diseases of the kidney, have dwelt more or less strongly on the forms of renal disease, accompanied by acute symptoms and a congested state of the organ, as being the precursors, in some instances, of the more ordinary changes, in which no increased vascularity occurs ; and Bright himself has indicated "an inflammatory state of the kidney" as a probable cause of the structural changes which he described and classified.—(See Bright's *Reports*, Vol. I. p. 3). This remark is strongly borne out by the case of Evans (p. 33), to which Pl. V. refers. Christison and Rayer concur in describing a state of congestion as the commencement of many of the chronic changes, and have established the coincidence of such a state, in some instances, with symptoms of acute dropsy and albuminuria. Even supposing the opinions of these authors as to the connexion between the hyperemic and anemic changes to be, as they probably are, founded partly on theoretical views as well as on direct observation, they must be admitted to be of great weight in a question which requires for its solution so large a field of experience.

From the comparatively small number of cases of acute disease of the kidney, and more especially of acute dropsy, which have occurred in the Edinburgh Royal Infirmary since my attention has been turned to this subject, I am unable to add any observations bearing more directly upon this question than those to which I have already alluded. Whether, in the two cases above mentioned, the evidences of vascular excitement, and the exudation in the tubes, can be considered as any thing more than accidental concomitant circumstances, I am not prepared to say without further opportunities of investigation ; but if these cases can be considered as examples of a state of the kidney analogous to red hepatization of the pulmonary texture, I am satisfied that this state is of much rarer occurrence, or at least more rarely fatal, in the former organ than in the latter.

I have alluded incidentally to Dr Johnson's views on this subject. He considers the deposit of fatty granules in the kidney as being invariably a chronic process, never preceded, in any case, by congestion or extravasation. On the other hand, admitting that these

states are frequently found in connexion with fatty exudation, he considers them as secondary results of the distension of the tubuli uriniferi.—(*Med. Chirurg. Transactions*, Vol. XXIX, pp. 4, 8, 9.) I have already shown (see p. 11), that this view is opposed both by anatomical considerations and by pathological facts; and, in particular, that the confirmed fatty degeneration of the kidney is usually accompanied by nearly complete depletion of the vessels. The comparative rarity of congestion in connexion with fatty exudation, under all circumstances, and its greater frequency while the exudation is still small in amount, sufficiently show the inadequacy of Dr Johnson's hypothesis to explain the phenomenon. The assertion of the exclusively chronic nature of the deposit in the kidney, is probably founded on an imperfect theoretical view of the nature and origin of fatty exudations in general.—(See pp. 18-20.)

On the whole, the supposition which appears to harmonize best with the analogies of other organs, and also with what has been hitherto observed in the kidney, is, that the oleo-albuminous or fatty exudation is sometimes preceded and accompanied by a congestive stage of short duration, in the course of which extravasation of blood into the tubes may occur. When, however, the exudation has accumulated within the tubes to such an extent as to cause fluid pressure by obstruction, the vascular system of the organ is emptied of its blood in a degree proportionate to the amount of distension; and, as the exudation continues to increase, the stage of congestion is rapidly superseded by the development of the pale yellowish colour so frequently mentioned in connexion with the fatty degeneration. This view appears to be strongly supported by the cases formerly mentioned, in which the oleo-albuminous exudation occurs in scattered whitish patches, surrounded by a distinct vascular rim.—(See p. 32.) On examining microscopically a section of such patches, I have observed the line of demarcation between the congested and the depleted Malpighian bodies to correspond accurately with the boundary of the exudation, so as to render it probable that the congestion, originally present throughout the diseased portion, had been superseded by the presence of the abnormal deposit.

Whether any connexion exists between the development of the congestive form of exudation and the presence of acute symptoms, is a question for further clinical experience, united with careful pathological investigation, to determine. As it is evident that the earlier stages of disease in the kidney have been hitherto to a great extent overlooked, both at the bedside and in the dissecting-room, it is highly probable that many of those affections which have been considered as most obviously chronic in their nature, may in reality be the advanced stages of processes more or less acute, which have not been fatal in the first instance, or which, if fatal, have not presented lesions appreciable by the unaided eye.

III.—Lesions of the Tubes and Epithelium.

Some of these lesions have been already fully described under the head of exudation (I.); but there remain others which are not less important in themselves than those formerly alluded to, and which are very frequently found in connexion with them.

Imperfect Development of the Epithelium Cells and Nuclei.—The natural condition of the epithelium cells has been fully described in the anatomical introduction to this memoir.—(See *ante*, p. 5.) The size of these cells, and the thickness of the cell wall, has been stated to vary within certain limits in organs apparently healthy. The size of the nuclei is less variable than that of the cells; but in all kidneys, whether healthy or diseased, the nuclei which are most closely adherent to the basement membrane are less perfectly circular, and of considerably smaller size, than the majority of those lining the tubes, and surrounded by complete cells.

Notwithstanding these differences in the normal condition, the physical characters of the epithelium is capable of affording important information as to the diseases of the kidney. In very many pathological conditions of the organ, the nuclei occur in various places almost wholly devoid of cell walls. They may be more abundant or more scanty than usual; and often appear in great profusion, huddled together in confused masses, and mixed with shreds of membrane and amorphous molecular matter, not soluble in acetic acid. This appearance of debris, which no doubt results from disintegration of the cell walls, most frequently occurs in kidneys which are abnormally soft and large, and from the cut surface of which an unusually large amount of turbid whitish juice may be scraped. It is usually impossible to obtain a satisfactory microscopic section; the cohesion and elasticity of the organ being so much impaired as to present no resistance to the pressure of the glasses. Such softened and altered kidneys occur frequently in fever and other diseases, and have appeared to me frequently to concur with deficiency in amount, or alterations in character, of the urinary secretion. I have not been able to arrive at any very definite conclusion, as to how far the disintegration in question may be the result of post-mortem change. However this may be, it is clearly abnormal, and ought always to suggest careful examination, as it is undoubtedly often connected with other morbid characters.

A more unequivocal pathological change (often occurring along with the above) is the small size and altered form of the nuclei throughout the organ. I have frequently observed the majority of the nuclei to be not more than half the usual size (some of them being even less); in this case they have always been destitute of cell walls, and have presented a more or less oval or slightly angular form. Sometimes they float scattered and solitary in the field of the

microscope; at other times they appear aggregated together, either by two and three, or in much greater numbers. When a few of these aggregated nuclei are observed, it can usually be seen that they are not actually in contact, but are inclosed in a very delicate and transparent filmy substance, which is readily twisted about in all directions by currents in the fluid, but which, nevertheless, has sufficient tenacity to prevent the nuclei from being torn asunder. Occasionally, in the midst of this connecting substance, obscure marks of cell walls can be observed around the nuclei; and, from repeated observation of these varieties, I am convinced that this transparent and homogeneous film is nothing else than the nascent or undeveloped cell membrane, which has separated from the basement membrane along with the half-developed or young nuclei above detailed. These aggregations of young nuclei are sometimes mingled with the amorphous debris of effete epithelium, or with granules and molecules of oleo-albuminous exudation, or of lithate of ammonia, which communicate to them a dark and confused appearance. Not unfrequently also these masses, when freed from the tubes, retain more or less of their form, and present so exactly the appearance of the casts of the tubuli seen by Franz Simon, and many other observers, in the urine, as to leave no doubt of their identity with these bodies. —(See Figs. 17, 18.)

Desquamation of the Epithelium.—The changes above described are generally accompanied by an extremely rapid generation of nuclei, which are separated from the basement membrane in an imperfect state, and carried away along with the urine. I shall not at present enter into the subject of the changes in the urine, further than to say, that the appearance in that fluid of the immature nuclei and cells, as well as the aggregations above mentioned, forms one of the most sure and undoubted signs of a diseased condition of the urinary tubules. There is reason also to think that disease may be detected in this way, long before the kidney has undergone disorganization at all evident either to the unaided eye or to the microscope.

The analogy of the anatomical changes now described, with those which occur in diseased mucous membranes in general, deserves to be adverted to in this place. Henle was the first to show satisfactorily that the essential phenomenon of mucous catarrhs and inflammations is usually the increased formation of epithelium cells in various stages of growth, and their separation along with an increased quantity of fluid secretion. The same fact has also been clearly elicited by Lebert, in his examination into the microscopic character of expectoration.—(*Physiologie Pathologique*, Vol. I.) The analogy of the phenomena in these cases, with those presented by urine in the various disorders of the kidney, in which desquamation takes place from the tubuli, cannot fail to be appreciated, when it is remembered that the basement membrane of the tubuli is essentially a mucous structure, differing only from mucous membranes in general by its

anatomical arrangement, and by its containing none of the accidental or non-essential parts of mucous tissue.

The desquamation of the epithelium of the tubuli uriniferi, has been recognised by several continental pathologists as an important characteristic of renal disease. Vogel (*Icones Histologicæ Pathologicæ*, p. 108), in particular, has furnished important observations on this subject; and several other authors might be cited as having alluded to the different forms and sequelæ of this morbid process.

In the memoirs of Dr Johnson and Mr Simon, published simultaneously in the London *Medico-Chirurgical Transactions* for last year, the desquamation of the epithelium and its anatomical results, are described, from independent observations, as characteristic of the inflammatory affections of the kidney, and as distinguishing these from the chronic *fatty* degeneration of the organ. According to Mr Simon, the latter is the exclusive result of scrofulous disease; while the desquamative disease is the consequence, in general, of a rheumatic or other *inflammatory* diathesis. Dr Johnson describes the desquamation as occurring both in an acute and chronic form, to which he gives the names of *acute* and *chronic desquamative nephritis*; and these are held to be true inflammatory affections, giving rise to organic changes of a peculiar kind. In the description of these changes, and of their relations to the different forms of renal degeneration described by former pathologists, there are considerable differences between the memoirs of Dr Johnson and Mr Simon, some of which will be presently adverted to.

In detailing the results of my observations on the anatomical changes in the kidney, I have avoided as much as possible all speculations as to the pathological causes or symptomatic phenomena of the lesions described. This branch of the subject I hope to take up at a future period; in the mean time, however, it is necessary, to prevent misconception, that I should repeat as regards the alleged scrofulous and rheumatic forms of renal degeneration, what I have formerly said in reference to the inflammatory and non-inflammatory,—that the application of such names to the anatomical changes in diseased kidneys is subject to this objection, that none of the lesions which I am engaged in describing, can be correctly considered as having an exclusive connexion with any specific pathological cause. In particular, I believe that there is no disorder of the system in general, or of the kidney in particular, with which the desquamative process is not liable to be connected, and that its connexion with the oleo-albuminous exudation is the most frequent of all.

I have therefore thought it necessary to exclude all names having reference to such vague pathological theories; believing that the lesions of an organ must be made the subject of purely anatomical consideration before its pathology can be rightly apprehended.

In some cases of desquamation of the epithelium, it is scarcely possible to recognise any departure from the usual condition of the kidney, either with or without the assistance of the microscope. The

degree of vascularity is very various in different specimens, and the epithelium thrown off is so quickly resupplied, that there is no very observable change in the microscopic condition of the tubules. In one very intense case, in which ten pounds of very watery urine, loaded with an epithelial sediment, were passed daily for some weeks before death, the kidneys were small, flaccid, and bloodless; many of the tubes were quite full of nuclei heaped closely together; some of the nuclei were undersized; the cells, when entire, were much compressed and angular.—(See Fig. 15.) In another instance, where urine was passed in large quantity and full of epithelial debris, during the last two months of life, the kidneys were found in an opposite condition; viz. large and congested, and with a firmness and smoothness of section like the first stage of the waxy degeneration formerly described.—(See pp. 33, 34.) In this case the condition of the tubuli was in most parts quite natural; in some, however, there was extravasated blood, and in others the epithelium had accumulated to an abnormal extent. In both these cases there was imperfect development of the epithelium; but cases have occurred to me, in which this character was by no means well marked. The crowding of the tubes with nuclei, although frequently found in the earlier stages of desquamation, is not invariably present; and I have seen the tubes gorged with epithelium, in a case where none had been separated with the urine for weeks before death.

Fig. 14. Fig. 15.

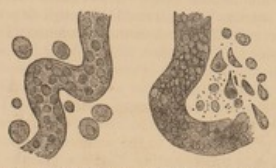


Fig. 14.—A portion of a tubulus uriniferus normally filled with cells. It presents an obviously cylindrical form, and the nuclei are disposed on the internal surface so as to leave considerable intervening spaces. The cells and nuclei from such a tube are unaltered in form. (250 *diameters.*)

Fig. 15.—A tube crowded with nuclei and compressed cells. Some of the cells altered in form, and nuclei are seen mixed with debris, outside the tube. The nuclei are somewhat smaller than those of the healthy tube. (250 *diameters.*)

So long, therefore, as the epithelium is freely regenerated, the kidneys may preserve a tolerably healthy appearance even on minute examination. The principal characters of the disease in this stage are derived from the urine. After prolonged disease, however, further changes take place. The epithelium becomes more sparingly generated, and is thrown off in the coherent masses above described (p. 42), leaving the basement membrane in portions bare, or

with a few scattered oval nuclei, much smaller than those cast off, adhering to its inner surface.—(See Fig. 16.) In the microscopic examination of organs in this condition, there are frequently seen films of such exceeding delicacy and transparency as to be only visible by very careful management of the light. They preserve the shape of the tubules, and contain no nuclei or structures of any kind. Similar films are occasionally seen in the sediment of urine. They are probably thrown off from the denuded basement membrane.—(See Fig. 18.)

Obliteration of the Tubes.—The basement membrane, which, with the few closely adherent oval nuclei above described, is now the sole remaining structure of the tubes, soon undergoes a change.—(See Fig. 16.) It loses the cylindrical form proper to it in the fresh and natural kidney, and becomes flattened by the pressure of the surrounding parts. Its cavity is thus obliterated, and what was a tube assumes the appearance of a transparent riband, dotted here and there with small oval nuclei, which, when seen at the edges, appear to be enclosed between two layers of membrane. These riband-shaped portions of membrane appear to preserve considerable tenacity and elasticity; by their greater density, and by the constant presence of the small oval nuclei so often mentioned, between their layers, they are in most cases readily distinguished from the delicate films which have been referred to above. They are very various in diameter, but are always inferior in this respect to the normal tubes; and they appear to break up spontaneously into smaller portions, each of which contains from one to six, or even more, nuclei. These portions are of various sizes, from 1-10th to 1-4th of a millimetre in length, and from 1-120th to 1-30th in breadth. They are usually

Fig. 16. Fig. 17. Fig. 18.

Fig. 16.—Fragments of tubes from which most of the cells have separated, and which are undergoing the change mentioned above. Some small nuclei are seen adherent, and others free. (250 *diameters.*)

Fig. 17.—Debris of epithelium separated from the interior of the tubes, and presenting a mould of their form. (250 *diameters.*)

Fig. 18—Delicate films presenting the form of the tubes, and probably separated from their interior. Along with them a few nuclei and cells, which, however, are not imbedded in the membrane, as in Fig. 16. (250 *diameters.*)

broadest in the middle, and taper to a point at both ends. The smallest of them contain only a single nucleus, and present an ap-

pearance in every respect like that of the young fibres of areolar texture, or those fusiform cells which have been called *fibro-plastic*. I think it probable that the whole of the diseased basement membrane ultimately splits up into fibres of this kind.[1]

While these changes are proceeding, the capillary vessels, which have ceased to be subservient to secretion, are usually obliterated. The consequence of this double obliteration of vessels and tubes, is a considerable degree of atrophy in the diseased parts; and, as the atrophy takes place at first chiefly in the cortical substance, great irregularities of the surface generally supervene. Thence arises the appearance so well described and figured by Dr Bright (Plate III. Fig. 2), in which, from the atrophy of the cortical substance, the bases of the pyramids "are drawn towards the surface of the kidney."

When oleo-albuminous exudation supervenes on the above derangement of the tubes, or when desquamation supervenes on the former (circumstances which I conceive to be of very common occurrence), the exudation most commonly takes the form of the granulations of Bright, which are deposited chiefly in the diseased tubes; and the atrophy proceeding around these they become salient, and the surface generally irregular, giving rise to the tuberculated state of the surface, so common in all the later stages of the granulated kidney (Bright, Plate III. Fig, 1; Rayer, Plate VII. Fig. 6; Plate IX. Fig. 8.) As the atrophy, however, proceeds, the granulations are gradually absorbed; and when the kidney has become extremely contracted and irregular, they often in great part disappear.

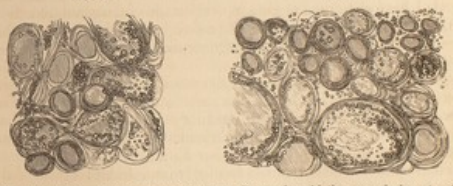
The atrophied portions of the kidney are usually exsanguine and of a tawny or drab colour; they have considerable hardness and toughness. Examined microscopically, they appear to consist of fibres and fusiform cells in great abundance, and more or less granular exudation, according to circumstances. According to Henle, Eichholtz, Gluge, and others, these fibres are in great part new formations; Johnson and Simon consider them as nothing more than the compressed parenchyma of the gland, from which all the other normal elements have disappeared. I look upon them as formed in great part by the breaking up of the basement membrane of the tubes (as above-described), as well as from the parenchyma and obliterated capillaries. It is not improbable, however, that, in addition to these elements, some new fibrous tissue is formed.

The extreme stage of the atrophied kidney is nearly the same whether exudation have existed or not.

[1] I have never seen any reason to believe, with Mr Simon, that the tubes in diseased kidneys burst from over-distension, discharging their contents into the inter-tubular tissue. The separation of the epithelium from the tubes, under the pressure of glasses, takes place to a considerable extent even in healthy organs, and much more in disease; but it is the result of the manipulation, not a pathological appearance.

Microscopic Cyst-formation.—It occasionally happens, on examining the section of a kidney with the microscope, that we see scattered through some parts of the section a few small clear vesicles of nearly circular or oval form; they are either of a very pale straw-colour, or nearly colourless, and are perfectly clear and translucent, with a very distinct shadowed margin, which causes them to stand out in bold relief from the other textures composing the section. Their diameter is usually from 1-40th to 1-15th of a millimetre, but in this respect they vary considerably; sometimes they appear to lie in the tubular areolæ, and at other times to be unconnected with these. Very rarely they have appeared to contain a few granules; most commonly, even when there is granular exudation around them on every side, they contain nothing but clear fluid. Their refractive power is not so great as that of oil, while it is much greater than that of the spherical cells of the tubes. Hence their distinct and characteristic shadowed outline.—(See Figs. 19 and 20.)

Fig. 19. Fig. 20.



Figs. 19 and 20.—*Vesicles described above, dispersed amid the normal elements of the section of the kidney. There is a considerable quantity of granular exudation in the Malpighian capsules and tubular areolæ. One vesicle in Fig. 20 contains two or three granules, but all the rest are quite clear. In Fig. 19, a tube contorted, and here and there apparently much constricted, is seen to pass from the left (below) to the right (above). One of the vesicles lies over a constricted portion, and two others are seen in contact with the tube below.*

These bodies (which, however, have never appeared to me to present distinct nuclei) are probably the same with the "nucleated cells or vesicles" described by Mr Simon, as resulting from the extravasation of the epithelial cells into the intertubular tissue, and as progressively enlarging so as to form the cysts visible to the naked eye, which are so common in diseased kidneys. To these structures he attaches great importance in the pathology of the kidney, conceiving them to be the invariable result of the desquamative disease when of long standing; the kidney being, in Mr Simon's opinion, changed more or less into an aggregation of microscopic cysts, which either undergo absorption, and lead to atrophy of the organ, or increase in size and monopolize its texture. Thus, according to Mr Simon, the serous cysts so common in the kidney result from an enormous development and hypertrophy of extravasated epithelium cells, which

assume the character of the vesicles he describes, and acquire the power of increase and endogenous development.

Whether the bodies described by me above, are the same with the vesicles of Mr Simon, I have some difficulty in determining; but they are the only objects I have seen which correspond at all closely with his description, unless, indeed, it were possible to suppose, as Dr Johnson appears to hint,[1] that he may have mistaken the normal disposition of the tubuli (See Fig. 1, and *ante*, p. 9) for a cystic structure.

However this may be, I am satisfied that the vesicles above described are exceptional productions, and by no means invariably connected, as Mr Simon describes his vesicles to be, with the progress of the desquamative degeneration. They are seen in comparatively few cases; on referring to four, of which I have drawings or memoranda, I find two to have been congested and waxy kidneys, with slight exudation, one to have been a soft and desquamating kidney, also with slight exudation, and one a granular kidney, with numerous cysts, from the size of a pea to that of a hazel-nut. On the other hand, I have examined organs in every stage of desquamative disease without finding these bodies, the production of which cannot therefore be an essential step in the degeneration and atrophy of kidneys so affected.

The origin and progress of these vesicles is very obscure. It is not improbable that, as Mr Simon asserts, they are transformed into the larger cysts visible to the naked eye; though I confess that I have not been able to trace the intermediate steps of their progress in a satisfactory manner. On the other hand, their origin from extravasated epithelial cells seems exceedingly improbable; indeed, I have already stated that I do not think the epithelium ever becomes extravasated. Moreover, the vesicles in question have all the appearance of being formed *within* the tubes, although they afterwards become separated from them.

From the occasional appearances of alternate distension and constriction presented by the tubes when undergoing obliteration, I am induced to believe that cysts may be formed by the occlusion and isolation of portions of tube which have not yet lost their power of secretion. Whether the vesicles in question are formed in this way, can only be determined by close and repeated observation; and I have not been able to obtain demonstrative evidence on this point. The observation in Fig. 19, however, though not free from sources of fallacy, appears to me to favour this view.

The larger cysts in the kidney present very strong evidence of being formed in connexion with the secreting membrane. In one instance I found their inner surface to be lined at some points with tesselated epithelium, in the form of pentagonal or hexagonal flat-

[1] See first part of article Ren, in Todd's Cyclopædia of Anatomy and Physiology, just published and not yet completed.

tened cells, with circular nuclei; in another case there were oval nuclei without any distinct cells, and a large number of free oil-globules of considerable size. The existence of oil in these cysts has also been observed by Dr Johnson. Other products of secretion are also occasionally found. On one occasion I found several cysts in a kidney otherwise healthy in appearance, which contained a turbid ochrey-coloured liquid, presenting under the microscope numerous minute crystals of uric acid. Mr Simon mentions having found on two occasions xanthic oxide in considerable proportion. I have more than once observed them to contain blood in large quantity, and I have likewise found them full of a matter like stiff glue.

The occurrence of cysts in kidneys presenting a generally healthy structure is so frequent, as to lead to the idea that they must be in such cases the result of disease which has been arrested before any considerable disorganization has taken place. Many of the cases of partial atrophy of the kidneys figured by Rayer (see Plate V. Figs. 5, 6; Plate XXXV. Figs. 8, 9, 10), are probably due to the rupture or obliteration of these cysts.

Before leaving the subject of cyst-formation, I may state, that in one instance I have observed the Malpighian capsules to be occupied by distinct cysts. This case will be presently detailed.

Dilatation and Thickening of the Tubes.—This condition, although by no means a very frequent one, is important as being characteristic, so far as I have observed, of the extreme stages of what I have called the "waxy degeneration." I have scarcely ever seen it unaccompanied by entire obliteration of the vessels, and by enlargement and increased density of the kidney. The organ has the dense, resistant feeling of fibro-cartilage, and both cortical and tubular portions have the light yellow colour, and the appearances described above, pp 34, 35, as those of the waxy degeneration in its last stage. The striæ of the pyramids appear to radiate indefinitely towards the surface, and meet the cortical substance in digitations, instead of being marked off by a sharp semicircular line, as occurs in the healthy kidney. When examined with a simple lens, or even the naked eye, the pyramidal striæ are seen to pursue an unusually sinuous course; this is peculiarly the case where they pass into the cortical substance. Moreover the pyramids are unusually broad at the bases; and the length of the straggling digitations is sometimes so great, that I have measured fully an inch and a half between the extreme end of the striæ and the corresponding papilla. Nevertheless the cortical substance is not usually diminished in quantity, being developed to a great extent between the pyramids.

This condition I have ascertained to proceed from dilatation and thickening of the tubuli uriniferi throughout the organ. The dilated tubes are usually twisted and varicose, as may be seen by inspecting a section of the pyramids with a low power.—(See Fig. 21, A.) When examined with a higher power, the section presents an

appearance very similar to some tumours (of the fibrous or fibro-cystic kinds); viz. a number of compressed areolæ, enclosed by fibrous tissue, and presenting an appearance of irregular concentric rings of various distinctness, (an effect apparently due to the peculiar refraction of light by the thickened membrane.)—(See Fig. 21, B.) The nuclei are obscured or invisible, owing to the thickness of the intervening wall, but nevertheless exist in considerable numbers. The Malpighian bodies and capillaries are usually obliterated. The kidney has in fact become, like the tumours whose structure it resembles, a true non-vascular texture.

Fig. 21.

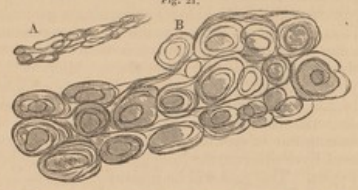


Fig. 21. A.—*Tubes in the pyramidal substance of the kidney, in the case of Campbell.* (Observation XIV.) (40 *diameters.*) B. *A similar section* (*as described above*). (170 *diameters.*)

The explanation of the peculiar extension of the pyramidal striæ towards the surface in these cases, is to be found in the fact, that even in the normal condition the convoluted tubuli have a general disposition from the bases of the pyramids towards the surface, in the direction of the striæ of the cones. This is evident from the facility with which the gland tears in that direction; although in the normal state this disposition is masked by that of the vessels, which, passing in straight lines through the cones, break into a complicated net-work of capillaries at the bases of the pyramids. In the present lesion, the vessels having disappeared, and the course of the tubes being strongly marked, their disposition towards the surface becomes manifest, and the abrupt line of demarcation between the cortical and pyramidal substance, caused by the presence of the vessels, is obliterated.

A very characteristic instance of this lesion is found in Observation XIII. It may be worth while to remark, that in this case the lesion above described occurred in common with bronchial dilatation, there being thus a precisely analagous state of the pulmonary and renal texture. In the following case there are several unusual circumstances—the amount of exudation—the persistance of some of the vascularity of the surface and pyramids—and the cysts of the Malpighian bodies.

Observation XIV.—*Waxy Kidney* (*last stage, with Granular Exudation*). *Cysts in Malpighian Capsules.*—Agnes Campbell, æt. thirty, was admitted into the Royal Infirmary, February 8, 1848, under the care of Dr George Paterson. She laboured under great general anasarca, and was extremely anemic. There was a murmur with the first sound of the heart. The urine was found to be highly albuminous, specific gravity 1·012, acid reaction. She died February 20th.

On dissection, the external aspect was excessively anemic; the face was puffy, the legs œdematous. The cavities of the chest and abdomen contained a considerable quantity of fluid. All the organs were sound, except the kidneys. These were enlarged by about a fourth, and were firm and dense. The capsule was easily stripped; the surface not uneven; the venous vascularity less than usual, and irregularly distributed. The cortical substance generally was of a yellowish colour, with some opacity, and destitute of vascularity. The yellowish opaque colour penetrated between the tubular striæ towards the bases of the pyramids, which were broad, and displayed a good deal of vascular injection. The Malpighian bodies were prominent on the surface of the section, and presented an unusually transparent and pale appearance.

On examining microscopically a section by Valentin's knife, there was seen dispersed among the tubes of the cortical substance a considerable quantity of very finely granular exudation. A few cells presenting the appearance of the granular or exudation corpuscles were seen. The nuclei in the tubuli were mostly free from cell-walls. There were numerous fusiform cells and rudimentary fibres. The Malpighian capsules were thickened, and no blood-vessels could be seen in their interior, which was divided into loculi.

On separating the Malpighian bodies from the capsules (which was readily done by scraping the cortical substance), and viewing them either with strong or weak powers, they were seen to be composed of pale semi-transparent cysts, from 1-15th to 1-12th of a millimetre in diameter, and compressed together. When floated out, they presented the appearance of a cluster of grapes, except as regards the form of the individual cysts, which was circular. They were apparently retained together by a very slight medium, as by a little manipulation several of them could readily be separated from the mass. They appeared to contain transparent fluid.

The above transformation of the Malpighian bodies has not escaped the attention of Rayer, who says that in an advanced stage of the *Néphrite Albumineuse* "the glandules of Malpighi resemble small serous vesicles, mingled with others a little larger, which still later became true cysts."—(See Rayer's Atlas, Pl. IX. Figs. 6. and 7.) In the case just detailed, however, there was no enlargement of any of the Malpighian bodies.

CONCLUSION.

With the view of enabling the reader to place the foregoing observations in relation with the descriptions found in systematic pathological works, I subjoin the following short remarks on the principal physical characters usually ascribed to diseased kidneys.

Increase of Size and Weight—Hypertrophy.—Enlargement of the kidney occurs chiefly in consequence of three conditions; 1st, from sanguineous engorgement; 2d, from distension of the tubes by secretion or exudation; 3d, from permanent dilatation and thickening of the tubes. Of all these causes, the second is by far the most common. The last is characteristic of the waxy degeneration formerly described.

The quantity of liquid in the tubes is at all times subject to so much variation, that it is difficult to say what amount of increase of weight may be thereby occasioned without the existence of any positively morbid condition. It is not very uncommon to find kidneys otherwise not differing from the healthy standard, about double the usual weight, or between seven and eight ounces each. I have more than once found them to weigh nine ounces each, with very slight marks of disease. When the weight much exceeds this, it is probable it arises from the rare combination of vascular and tubular engorgement.

In kidneys containing oleo-albuminous exudation, the greatest increase of size is attained when the exudation is universal, and unaccompanied by desquamation.

Cystic degeneration of the kidneys, dilatation of the pelvis and ureters (Hydronephrose, Rayer), &c., also give rise to great increase of size and weight.

Diminution of Size and Weight—Atrophy.—This condition sometimes occurs to a certain extent in emaciated subjects, without any disorganization, owing to the diminished activity of secretion. More frequently, however, it is the result of separation of the epithelium, followed by contraction and obliteration of the tubular structure.

Atrophy, from this cause, is liable to supervene in all other varieties of renal lesion, except the waxy degeneration, which appears to lead to a permanently hypertrophied condition of the organ. In kidneys enlarged from exudation, the occurrence of desquamation and its consequences is frequent; and the diminution of size in such cases, is often not followed by a return to the natural condition but by permanent atrophy.

The course of all disorganizing diseases in the kidney, is to produce first enlargement, and then contraction of the organ. In the extreme stages of the atrophy which results from exudation, exudation is often nearly absent. When exudation therefore, even in very sparing quantity, accompanies a contracted condition of the kidney, there is a probability that it has been abundant at some former period.

Irregularities of Surface—Tuberculated and Granulated Kidneys.—The smoothness of the surface in the kidney is destroyed either by unequal dilatation, or unequal contraction of the tubuli of the cortical substance. The former takes place in the waxy degeneration, the latter in the desquamative processes.

The most frequent irregularities of surface are formed in connection with the granulations of Bright (the origin of which is described, See *ante*, p. 12). These are invariably formed when exudation is deposited in kidneys tending to the desquamative lesion; and, as this runs its usual course, the granulations become prominent from the destruction of the tubes around them. An extreme degree of the irregularities thus produced constitutes the tuberculated kidney.

The puckering and partial atrophy occasionally seen in kidneys

otherwise not morbid, or comparatively slightly diseased, are probably in many instances the result of the obliteration of cysts.

The more remarkable changes in colour and consistence are described very fully in many parts of the preceding memoir.

On reviewing the whole of the observations, the result of which I have now laid before the public, I am induced to regard the following conclusions as especially important in relation to the pathology of renal diseases:—

1. By far the greater part of the pathological lesions of the kidney arise from, or are connected with, the exudation of oleo-albuminous granules into the interior of the tubes and epithelial cells.

2. The oleo-albuminous exudation is probably often preceded, and certainly occasionally accompanied, by vascular congestion; but when the quantity of exudation is considerable, more or less complete depletion of the vascular system invariably occurs. This is a secondary result of the obstruction of the *tubuli uriniferi*.

3. The oleo-albuminous exudation occurs in two chief forms; viz. *first*, Universal infiltration of the tubes throughout the organ; and *second*, Infiltration of particular sets of tubules, the rest remaining free, or nearly so. In the latter mode arise the granulations of Bright.

4. There is no essential anatomical difference between the exudations in the kidney which are the result of chronic processes, and those which have been considered as the result of inflammation.

5. The capillary vessels of the kidney are subject to spontaneous obliteration (unaccompanied in the first instance by any visible lesion of the tubes), giving rise to the peculiar affection which I have called the *waxy degeneration*. This obliteration of the vessels is probably in all cases preceded by a stage of congestion.

6. The consequence of the waxy degeneration is thickening and varicose dilatation of the tubuli throughout the organ.

7. The tubes of the kidney are subject to contraction and obliteration, in consequence of the desquamation of their epithelium; a condition resulting in atrophy, and complete disorganization of the organ.

8. The desquamation of the epithelium occurs very frequently in all the other diseased conditions of the kidney. When sufficiently long-continued and extensive, it produces contraction, and this indifferently whether exudation be present or not. It is sometimes accompanied by vascular congestion in every stage of its progress.

9. The earlier stages of the exudations can only be discovered by means of the microscope. The progress of the waxy degeneration, on the contrary, is best traced by the unaided eye. The desquamation of the epithelium is only to be discovered with certainty by means of the microscope, and is particularly apt to escape attention, under all circumstances, if the *kidney* only, and not the *urine*, be looked to. It results that careful investigation, both by the micro-

scope and the naked eye, both of the kidney after death and the urine during life, are indispensable to enable the pathologist to determine with exactitude the presence or absence of disease.

[I propose to publish the results of my observations on the urine, and on the pathology of Bright's disease, and other diseases of the kidney, as soon as some investigations suggested by the present series of papers are completed.]

ON THE

PATHOLOGICAL ANATOMY

OF

BRONCHITIS,

AND THE

DISEASES OF THE LUNG

CONNECTED WITH

BRONCHIAL OBSTRUCTION.

BY

W. T. GAIRDNER, M.D.,
FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS, AND PATHOLOGIST TO THE ROYAL INFIRMARY OF EDINBURGH.

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ON THE

PATHOLOGICAL ANATOMY OF BRONCHITIS, &c.

NOTWITHSTANDING the great frequency of bronchitis in European latitudes, and the attention which has always been given to this disease by physicians, especially since stethoscopic examination came into use, it has appeared to me that the commonly received doctrines as to its ultimate effects upon the pulmonary texture are not expressive of the importance of the subject, and in many points not consistent with the truth of nature. Having become deeply impressed with the truth of the views to be advocated in the following papers, and having taken much pains to satisfy myself as to their correctness, I venture to lay them before the profession, in the hope that, if corroborated by the further researches of others, they may lead to improvements of some importance, both in pathology and practice.

As to the sources from which my materials are drawn, it is only necessary to state here, that while I have relied chiefly on personal observation, I have endeavoured to combine and harmonise the conclusions thus arrived at with those of prior observers, so far as known to me; nor have I willingly omitted any species of evidence which seemed to bear upon the subject. It would have been, indeed, an unsatisfactory task to have brought forward the results of personal experience on this subject, had they not been found to agree in many, even in most points, with those of some former authors in respect to matters of detail. In placing these pages in the hands of the public, therefore, I profess myself anxious for judgment as to the *truth*, more than as to the absolute *novelty*, of their conclusions; and shall be satisfied if enough of originality be found in them as a whole, to rescue them from the charge of being a superfluous or burdensome addition to our literature.

PART I.

PRIMARY RESULTS OF BRONCHITIS.

In the present part, the direct and primary results of bronchitis will be discussed; comprising under this head those effects which follow, almost constantly, the accumulation of mucus and inflammatory products in the bronchial tubes, when these are sufficient in quantity to cause serious obstruction. A second part will include the secondary and more permanent disorganizations of the pulmonary texture, which result from the former under peculiar circumstances; being induced either by long-continued intensity of the original disease, or by constitutional states unfavourable to the removal of its results.

Obstruction of the Bronchi.

The ordinary effects of bronchitis upon the mucus and other contents of the bronchial tubes, are, for the most part, well understood and clearly described by authors. The vascular engorgement of the mucous membrane, which at a later stage becomes much softened and thickened, and the loading of the tubes with an altered mucous secretion, intermixed to a greater or less degree with purulent matter, are familiar to every one. The connection of these sources of obstruction with the auscultatory and other signs is also so well known as to require but few observations.

The varieties in the character of the secretion from the diseased membrane are very great. In the earlier stages of the affection, the mucus is profuse, thin, watery, and frothy, being mixed with air bubbles of all sizes. At this period it contains but few microscopic elements, a few altered epithelium-cells being only visible, and these in much greater number in the mucus procured from the dead body than in that expectorated by the living patient. At a later period the mucus has become yellowish, more tenacious and viscid, containing numerous pus corpuscles, and more of the altered epithelial elements. Occasionally, but more rarely, the expectoration, or the fluid in the bronchi after death, is composed of pus nearly pure, but always viscid and thick, like that which flows from old abscesses. In these cases the pulmonary tissue is rarely unaffected.

When the disease has been of considerable duration, and has nevertheless been the result of an acute attack, there are usually found, on incising the bronchi, numerous yellowish pellets, or irregular flocculent masses of somewhat curdy consistence, which float in the more recent and thinner mucus, without in any degree becoming amalgamated with it. These masses are found of all sizes, and constitute a well-known form of the expectoration of bronchitis in its advanced stages. They are no doubt formed by the evaporation of the more fluid parts of the mucus, in consequence of the constant passage over it of dry air; the inspissated material clinging to the walls of the air passages, until detached and washed away by renewed exudation from the mucous membrane. Such a mass is not unfrequently seen plugging the whole caliber of one of the larger or smaller bronchi, in such a position as to leave no doubt that in the act of inspiration it must have acted the part of a ball-valve, completely preventing the access of air to the part of the lung involved, by falling back upon the orifices of the smaller bronchi, into which its size would prevent it from entering.

When bronchitis, or bronchial catarrh, has existed for some time in a comparatively slight form, and with the expectoration only of an increased quantity of mucus unmingled with pus, portions of this secretion become inspissated in the form of a thick, glairy, tenacious, semi-transparent material, sometimes resembling raw white of egg. In one or two cases I have pressed out of the smaller bronchi plugs of altered mucus of a still more tenacious character, like cold glue, and nearly, if not quite, transparent. But this amount of inspissation is not common.

The effects of these morbid accumulations in producing obstruction of the bronchial tubes are well known to the auscultator. So long as the mucus is thin and watery, or even more or less purulent, there is no serious or complete impediment to the passage of air; which, as the fine and coarse mucous rales, accompanied by vesicular respiration, indicate, finds its way through the fluid to the ultimate bronchi and pulmonary vesicles. The fluids, too, at this stage, move freely throughout the bronchial tree even to its minutest branches, and when in excessive quantity, are readily expectorated in the act of coughing. This stage of bronchitis, therefore, is comparatively little apt to be accompanied by urgent dyspnœa, or by changes in the condition of the pulmonary texture.

It is otherwise, however, when after a time the secretions within the bronchial tubes have become inspissated; the mucus having either become purulent and formed itself into the tenacious pellets above mentioned, or assumed the stringy, tenacious consistence proper to the more chronic forms of the disease. When, under these circumstances, expectoration is hindered, either by the tenacity of the mucus itself, the weakness of the patient, or any other cause, the tubes become really obstructed, the sound of vesicular respiration is at some points of the lung much diminished, or it may be altogether

lost, and the bronchial râles are correspondingly modified, indicating the passage of a smaller quantity of air with a much greater amount of resistance; the ordinary mucous rattles being supplanted in part by the sharp "clicking" and valve-like sounds so well described by Dr Williams.[1]

It is quite evident to the observer of a patient in this condition, or, indeed, in any stage of bronchitis attended with laborious breathing, that the chief difficulty is invariably in *inspiration*. This act is accomplished only with the aid of all the accessory muscles; and, even then, the contracted state of the chest, especially in children, and the imperfect descent of the diaphragm, show that in proportion to the power employed, the amount of air entering is small. The expiratory act, on the contrary, is always accomplished with comparative ease; and any serious obstruction to the performance of this act is at once got rid of, or dislodged, by the additional impulse given to the expired air in coughing or hawking. These phenomena of laborious breathing, particularly the long-drawn, exhausting, inadequate inspiration, are probably quite peculiar to obstructive bronchitis. They occur, it is true, in diseases of the heart; but in them, I believe, only secondarily, from the accumulation of mucus in the respiratory passages. The dyspnœa of *pure* pneumonia, on the other hand, is something quite different, being a mere *acceleration* of the respiration, without any of the heaving or straining inspiration observed in bronchitis, or in cases where the two diseases are combined. So much is this the case, that I have repeatedly observed patients affected with a great extent of pneumonia in both lungs, and in whom the extreme lividity, and the respirations numbering 50 or 60 in the minute, showed infallibly the amount to which the function of the lung was interfered with: and who nevertheless lay quietly in bed, breathing without any of the violent effort, or the disposition to assume the erect posture, so constantly accompanying the more dangerous forms of bronchitis. If this freedom from orthopnœa and laborious breathing be not uniformly characteristic of true pneumonia, it is because that disease comparatively seldom exists uncomplicated by some degree of bronchial affection.

The cause of the inadequate and laborious inspiration in bronchitis, while the expiration is comparatively easy, is to be found, I believe, not merely in the smaller power of the inspiratory muscles to dilate the chest, nor in the advantage which the expiratory forces derive in the dislodgement of obstructive mucus from the sudden impulse of coughing, but in the mechanical relations of the tenacious inspissated mucus to the caliber of the bronchial tubes. On this subject I have more to say in the next division of my subject.

One fact of considerable importance, in connection with the total obstruction of bronchi by muco-purulent matter, has been adverted to by Andral, and is illustrated by two cases in the "Clinique

[1] Diseases of the Chest, 4th edition, p. 80.

Médicale." This is, that the accidental position of such an obstructive plug may be the cause of a very rapid and unexpected fatal issue, in a case by no means threatening from the violence of inflammatory action. In each of the two instances given by Andral, the respiratory murmur became completely suppressed in the upper part of one lung, the patients having been previously affected with moderate bronchitis, and dyspnœa having become suddenly increased after a fit of coughing. The post-mortem examination showed the signs to be due to an obstructive accumulation in the bronchi leading to the upper lobe of the lung; but the absence of respiratory murmur, combined with clearness on percussion, had led during life to the diagnosis of emphysema. There can be, I think, little doubt that many of the paroxysmal accessions of dyspnœa in persons affected with bronchitis are due to accidental change of position of the pellets and ropy masses of inspissated mucus, which accumulate in the tubes. At least stethoscopic examination frequently reveals the signs of obstruction in particular parts of the lung, supervening rapidly, and disappearing again with equal suddenness, in consequence of the accession of cough.

I have now to advert to a condition of the pulmonary texture which appears to me, from the results of my own dissections, to spring more directly than any other from obstruction of the bronchi by mucus of a certain degree of tenacity. As this condition, the collapse of the air-cells, has been but little noticed by authors in this connection, at least in the adult lung, the subject will perhaps be best introduced by a narrative of my own observations with regard to it, which I shall endeavour afterwards to connect with the facts furnished by others.

Collapse of the Lung as connected with Bronchial Obstruction.

During the epidemic of continued fever which prevailed in Edinburgh in the greater part of the year 1847, it was frequently observed that the lungs, in persons of all ages, were the seat of a form of condensation, characterised by the absence of the friability, and granular appearance on section, of pneumonic consolidation, and also by the peculiarity of its microscopic elements, the large granular cells which form so common an ingredient in ordinary red hepatization being either very sparingly or not at all present. The condensed portions were usually scattered over both lungs, and often very limited in extent, being accurately circumscribed by the margins of the lobules; in most other respects they corresponded with the descriptions to be hereafter given. These appearances occurred very commonly in typhus fever, whether of the ordinary form or the abdominal typhus (now commonly called *typhoid fever*), accompanied by intestinal ulceration; which latter form had at that time a prevalence quite unusual in Edinburgh. Similar lesions were occasionally, though less frequently, seen in the relapsing fever, which was also

epidemic during that year. This state of the pulmonary tissue was the subject of frequent conversation, and repeated microscopic examination, among those chiefly engaged in the pathological theatre of the Infirmary during this period; although my own notes of fever cases at this time are but few, I am fortunately able to compare my recollections with several sufficiently characteristic descriptions of the lesion in question by Dr Waters, whose examinations were recorded with the utmost care and fidelity, with a view to the preparation of a history of the epidemic, unfortunately not yet published.[1]

The nature of this pulmonary affection appeared to me, at the time, very doubtful. By some it was looked upon as corresponding with the "typhoid deposits" in the lung described by German authorities; and the imperfect cell-forms often seen under the microscope seemed to favour this view. On the other hand, careful examination showed that there was frequently little or no real *deposit;* the cell-forms differing very little from those which might be procured from the normal lung, or still better, from one compressed by pleuritic effusion, or any other cause. At this time, my experience in pathological studies was not such as to enable me to form a decided opinion; but towards the close of the epidemic, the result of observations inclined me to believe that the lesion so frequently witnessed in fever was not connected with any specific form of morbid deposit, but was, in its purest form, a condition of imperfect expansion or collapse of the pulmonary tissue, similar to that described in the lungs of infants under the name of *atelectasis*, and differing only in its distribution in patches, and in other accessory circumstances, from the condition of *carnification* described by Laennec as the result of compression of the organ.

That the fever of 1847 was not, in any degree, *specially* characterised by these lesions is fully proved by the researches of other authors, as well as by my own subsequent experience. In fact, in the course of the two succeeding years, I had various opportunities

[1] From one of Dr Waters' reports I quote the following:—"Inferior lobe of left lung felt condensed, and contained less air than ordinary; its section was smooth, not granular, and its consistence somewhat tough, not breaking down under the finger." On another occasion, "The lower lobe of the left lung was much gorged with blood, and of a dark colour, and smooth section, not granular, void of air, and sinking entirely in water." The reader may compare these with the characters which follow (p. 12) of the bronchitic collapse. The following description from the case of a child dying of fever, corresponds to the characters of lobular collapse, combined with the bronchial abscesses to be hereafter described. There were condensed nodules in the lung which "presented at the surface of the organ, where they were recognised by their bluish-black colour and resistent feel; they were not elevated above the surrounding surface, but rather very slightly depressed." * * "On section, the great majority of these masses presented a reddish-blue colour and smooth surface. They were not friable but of firm consistence. A few of them had degenerated into a reddish-gray somewhat friable substance, and two or three were excavated by central cavities, which were empty and collapsed."

of becoming familiar with collapse, or carnification, of the pulmonary tissue, as a disease distinct from pneumonic consolidation, occurring in various forms, and under circumstances where no external compression of the lung could be conceived to account for it. Many of these lesions occurred in the lungs of children, but those of adults and old persons were scarcely less frequently affected. The disease presented itself, also, frequently in combination with other affections, which gave rise to no small difficulty in forming an opinion on the true nature and mode of origin. Neither was it always very easy to distinguish it from pneumonia, more especially in some of the combined conditions to which I shall hereafter allude; and I am satisfied that the terms *lobular pneumonia*, *red hepatization*, or the more indefinite expression, *condensation*, have often sufficed in my own case, as well as that of others, to cover ignorance or imperfect knowledge of the condition in question.[1]

Notwithstanding these difficulties, however, observations made in the years 1848-9 left me no longer in doubt, that the lungs of adults not unfrequently display portions more or less limited or diffused, which are so perfectly condensed as to sink rapidly in water, and yet differ widely, in appearance and microscopic character, from truly inflamed lung, as well as from all the atrophic conditions of the organ which can be clearly traced to inflammation or structural disease as their cause. In some instances, such portions presented exactly the appearance of fœtal lung; and only the manifest absurdity of the proposition could have prevented the observer from ascribing their state to the same cause as the congenital non-expansion of the lung, or atelectasis of Jörg. In the absence, therefore, of any manifest explanation of the compression or collapse of the tissue observed in these cases, I was obliged, provisionally, to rest satisfied with the knowledge of the fact that most of the lobular and many of the more diffused forms of condensation usually ascribed to pneumonia, in the adult as well as the child, were really the result of some other and unknown condition.

Some time after this collapsed condition of the lung had become tolerably familiar to me, the following case occurred, presenting a marked example of the coincidence of the affection with obstructed

[1] Had opportunities of examining the lungs of very young children frequently occurred to me, I could scarcely have failed to have soon become familiar with all the phases of this lesion; and to have recognised much sooner than I did, the identity of the congenital atelectasis, the "lobular pneumonia," and the carnification or collapse of the adult lung. But owing to the exclusion of all children under five years of age from the Infirmary, my observations were confined almost entirely to the lungs of persons above the age of infancy; and it was only at a comparatively recent period that the observation of a few infantile lungs, and the perusal of the work of M. Legendre (Recherches Anatomo-pathologiques et Cliniques sur quelques Maladies de l'Enfance, Paris, 1846), revealed to me clearly the immense importance and frequency, though not, I believe, the true significance, of this state of the pulmonary texture in the early periods of life.

bronchi, *both lesions being limited to a very small and circumscribed space in the lung.*

Case I.—*Lobular collapse of Lung—Obstructed bronchi.*—(P. R. 257.[1])

A man, æt. 18, affected with necrosis of the femur, anasarca, and some degree of abdominal dropsy from disease of the liver, spleen, and kidneys, died in the surgical hospital in the last stage of exhaustion and emaciation. No pulmonary symptoms had attracted notice.

The lungs were generally normal in appearance. At one or two places, however, they crepitated imperfectly over spaces not larger than an inch in diameter; these portions were quite circumscribed by abrupt margins; the bronchi leading to them yielded on pressure a very tough gelatinous mucus (like thick calves'-foot jelly), which contained only ciliated epithelium, and had otherwise the ordinary appearance of mucus under the microscope.

In this case there was little or no room for fallacy in judging of the connection of the pulmonary collapse with bronchial obstruction. The parts affected, with the bronchi leading to them, were quite capable of being isolated; and there was no trace of an inflammatory affection in any part of the pulmonary tissue. The co-existence of the collapse with bronchial obstruction thus accurately limited, could only, in all probability, be explained by the dependence of the one upon the other as its cause. The following case, however, which occurred during the present year, is still more conclusive:—

Case II.—*Extensive Collapse of Lung—Obstruction of Bronchi by a Tubular Membrane.*—(P. R. 301.)

A girl, aged about 21, was attacked, after a surgical operation upon the tongue, with urgent dyspnœa having the character of a laryngeal affection. Tracheotomy was performed, but failed to save her. She died about twenty-four hours afterwards.

Dissection performed Feb. 22, 1850. The appearances in the air passages and lungs were the following:—

The right lip of the glottis was infiltrated with serum, which distended the epiglottidean fold of mucous membrane so much as nearly to close the opening. The larynx and trachea were occupied by a tubular false membrane throughout their whole length; it was about a line in thickness, friable, and of a yellowish white colour. This membrane was continued at the bifurcation into the right bronchus, but the left was free from it. It could be traced throughout the bronchi of the right lung even into the minuter ramifications, in which it assumed the form of a very soft opaque matter, like a thick emulsion. On examination with the microscope, the membrane presented the usual appearance of coagulated fibrin, with some pus or mucus corpuscles, but these not in very large numbers.

Both lungs were more collapsed, and contained less air than natural. The left, however, was much less affected than the right. The latter was at some parts completely flaccid and free from air, while others presented an imperfect crepitation. The upper lobe crepitated more than the others, and its colour was

[1] These figures affixed to a case, refer to the numbering in the Pathological Register of the Royal Infirmary, vol. xii., where the details which I have omitted, as not bearing on the argument, will be found, usually in a pretty extended form.

mostly natural; but in its lower portions it approached more in colour to the middle lobe. This was perfectly non-crepitant, of a grey colour, having a smooth flesh-like section, and infiltrated with a thin serosity, having a faint reddish tinge, nearly transparent, and presenting under the microscope only epithelium, pigment cells, and a very small amount of pus cells, and granular matter. The lower lobe was not quite so much compressed, and contained a good deal of blood. The bronchial glands were slightly enlarged, and the whole pulmonary mucous membrane highly vascular.

In this case, as in the former, there were no pneumonic appearances; for the small amount of pus cells which existed in the serosity scraped from the lung, were probably derived from the small bronchi, and were, at any rate, quite inadequate to account for so remarkable a consolidation. Neither can it be supposed that there was any older structural lesion of the pulmonary substance, as the girl was undoubtedly in good general health at the period of her admission into the hospital, and the whole stages of the fatal disease were too rapid to admit of any complicated structural change. The collapsed lung presented exactly the appearances so commonly seen in organs compressed by pleuritic effusion, except that the middle lobe had rather more of an œdematous appearance than is common under such circumstances. Here, then, is an instance of collapse of the entire lower lobes, and part of the upper in the right lung, as complete as it could have been from external pressure, and coinciding with a manifest cause of obstruction in the whole of the bronchi of that lung; whereas on the opposite side, where the obstruction had been much less considerable, the lung was comparatively expanded and normal. It is impossible not to see here a relation of cause and effect; and as it will not be maintained that the deposit of false membrane was the result of the collapse, the converse proposition is, I think, scarcely to be avoided.

The form of bronchial obstruction in this case is a sufficiently rare one in the adult, but by no means unexampled. It is not at all clear whether the membrane had never extended to the bronchi of the opposite lung, or had been dislodged by expectoration. In the former case it is not easy to understand the non-symmetrical character of the fibrinous exudation, while the mucous membrane appeared equally inflamed on both sides of the chest; in the latter it is still more incomprehensible, how the complete expectoration of an extensively ramified membrane could take place, without being observed during life. But of the fact of its presence on the right, and its entire absence on the left side, there is no doubt.

The observation of these two cases, and the conclusions which appeared naturally to follow from them, as to the connection between collapse of the air cells and bronchial obstruction, tended to throw light on many of the obscurities of the preceding observations. More particularly, it seemed to me that they furnished the connecting link necessary for the explanation of the peculiar forms of "lobular pneumonia" (as they were generally considered), which had frequently occurred both in the lungs of children and adults. The

peculiar connection of lobular condensation with bronchitis was a matter of common observation with me before this period; the more recent literature on the diseases of the lung in children also seemed to point to the same fact; and it seemed every way probable that many of the more complex phenomena of pulmonary disease, would receive explanation from the more careful consideration of their connection with obstructed bronchial tubes. I therefore renewed my observations with great interest, directing them particularly, and with more care than formerly, to the relative condition of the bronchial tubes and pulmonary tissue, in all cases where either pulmonary collapse or bronchitis existed to any considerable extent. Some of the illustrative cases which have occurred, will hereafter be detailed. The general result to which I have been led is, 1st, that in all cases of collapse of the lung not caused by external pressure, the bronchi have presented unequivocal appearances of obstruction; 2d, that in most, if not all, the instances of severe and fatal bronchitis, especially if the secretions had become ropy or inspissated, more or less collapse of the pulmonary texture has also been present; that under peculiar circumstances, which will be presently adverted to, a much less amount of obstruction may be attended with collapse of the pulmonary texture, the symptoms in such cases probably attracting little attention.

In order to justify these conclusions, it will be necessary to describe a little more fully the forms under which I have observed the lesions referred to.

Bronchitic collapse of the lung occurs under two distinct aspects: the *diffused* form, and the limited or *lobular* form. Of these, the latter variety is the more striking and characteristic, and has been, especially in the lungs of children, the subject of more discussion than the former; but the diffused form is by far the more common, and is in fact of very frequent occurrence, at least in its slighter degrees. Both forms present the same fundamental changes of the pulmonary tissue, which is usually of a dark violet colour externally, as seen beneath the pleura; and internally of a more or less deep brownish red, or mahogany tint. The colour, however, is by no means an invariable criterion, depending almost entirely on the amount of blood in the collapsed tissue. The affected parts are always more or less condensed; this condensation may amount to a mere diminution of the crepitation, or to a total absence of it, in which case portions are usually found to sink readily in water. These latter portions are both more flaccid and much less friable than the pulmonary tissue when in a state of red hepatization; and they differ greatly from this lesion in the aspect of their section and the nature of the fluid it yields to the knife. In every variety of true pneumonic consolidation, in which the lung is completely void of air, the air cells are occupied by a deposit, presenting to the naked eye (and still more distinctly to a power of 20 to 30 diameters) the well-known granular aspect of the hepatized lung. If the deposit is

fluid, or semi-fluid, it is capable of being pressed out, or scraped off, in the form of a thick opaque emulsion-like matter, of yellowish, orange, or grey colour; and in all cases it shows, under the microscope, abundant granular elements and cell-structures, of the kinds usually found in inflammatory exudations in parenchymatous organs. In the collapsed lung, on the contrary, the section is comparatively smooth, having somewhat the appearance, as described by Laennec, of muscular flesh; it presents no trace of granulations, and yields, on pressure, or to the knife, only a semi-transparent bloody serosity, which, under the microscope, is seen to contain little or nothing besides blood-corpuscles, epithelium, and other portions of normal tissue, and possibly a small amount of pus from the interior of the bronchi.

In the *diffused* variety, the collapsed condition may be found affecting a more or less considerable portion of either or both lungs, usually at their posterior part, and passing quite gradually into normal tissue; the supple, dense, tough feeling being exchanged for the normal spongy, elastic crepitation; and the violet, or deep purple colour, shading off into the usual hue of the surface. Even in this form of the lesion, however, a tendency of it at some points to be circumscribed by the interlobular divisions may often be observed; this tendency being, so far as I have observed, quite characteristic of the bronchitic, as opposed to the pneumonic, consolidations.

In the *lobular* forms of bronchitic collapse, which often occupy the anterior edges, as well as all other parts of the lung, the affected portions are everywhere accurately and abruptly marked off by the interlobular septa, the portions so limited being various in size and form, but always manifestly shrivelled, and sunk in below the level of the surrounding parts. This is peculiarly manifest when they occupy the anterior edges. When they are scattered through the lung, they communicate to the fingers, in feeling the organ externally, much the same sensation as clustered tubercles in the midst of crepitant tissue. This is the form which was so often described as "lobular pneumonia" in young children, till the experiments of MM. Legendre and Bailly clearly showed it to be nothing but a collapse of the air cells.[1] These observers were in fact the first to apply to this condition of the lung the same test as Jörg had long before used in respect to the congenital atelectasis, and to show that the lung affected with "lobular pneumonia" could be generally restored nearly to its natural condition by forcible inflation, which occasionally requires, however, to be continued for some time. I have employed inflation in both the diffused and lobular form of collapse, as observed in adults, and as above described, with precisely the same results as those of Bailly and Legendre. I may state, however, that though this test is very useful in demonstrating the nature of the lesion, in a favourable case, to one not familiar

[1] "Archives Generales de Médécine," 1844; and Legendre's work before cited.

with its character, I do not believe it to be applicable to the determination of the presence or absence of pneumonia in those mixed cases in which alone there is any difficulty. For I have observed that on the one hand, the partially pneumonic lung may be inflated when the affection is recent, and combined, as it frequently is, with bronchitic collapse; and, on the other, that in the latter lesion in its purest forms, complete inflation is often very difficult or impossible after the collapsed state has been of some duration. In fact, the lung then begins to undergo a modification in its nutrition and structure, which ultimately leads to permanent atrophy.

With regard to the combined forms of lesion above alluded to, they are, I believe, by no means of rare occurrence. The collapsed lung, especially in the incomplete and diffused form in which it most frequently presents itself in adults, may become the seat of a true inflammatory exudation into the air-cells, giving to the section an obscurely granular aspect. The exudation has in such cases the usual microscopic characters, but the shrivelled state of the lung, and the minuteness of the granulations, together with the state of the bronchial tubes, demonstrate the participation of these in the inflamed condition. Occasionally, also, the collapsed lobules are subject to a œdematous infiltration of fluid, when this state prevails in the other parts.

I think, however, that I have also observed the collapsed parts, particularly in the well-marked lobular forms of this lesion, to escape to a certain extent, the inflammatory or œdematous condition prevailing in the parts around them. At least, I have observed cases where these remained comparatively dry, containing only blood, when other parts were bathed with frothy serum or pus. This subject, however, requires further investigation.

In maintaining (as I have no hesitation in doing) that some degree of collapse of the lung is an almost invariable concomitant of bronchitis of a certain degree of intensity, it must not by any means be supposed that *complete* loss of crepitation is to be looked for in any part of the tissue in the majority of cases. The usual fact is, that the collapse is in the incomplete and diffused form; but I believe, nevertheless, that dulness of percussion during life from this cause, and complete lobular or diffused collapse after death, especially in the posterior parts of the organ, will be found to be much more common in the bronchitis, as well of adults as of children, than is commonly supposed. Of the truth of these opinions, both recorded observations, and unrecorded recollections, appear to me to furnish no inconsiderable amount of evidence.

The following two cases are good examples of collapse from bronchial obstruction,—the former in its diffused, the latter in its limited or lobular, form. The accounts during life are, in both cases, very inadequate; but they have, nevertheless, some points of great interest, apart from the lesion now immediately under consideration, which will render reference to them necessary in the sequel.

Case III.—*Extensive Collapse of Posterior Portions of Lungs—Emphysema of Anterior Parts—Obstruction of Bronchi by Ropy Mucus—Death by Exhaustion.*—(P. R. 362.)

A woman, æt. about 30, died of exhaustion from the combined effects of Bright's disease and ulcerating cancer of the uterus.

The body, examined July 1st, 1850, was extremely pale, and the limbs, with the depending parts of the thoracic and abdominal parietes, were anasarcous. The abdomen was not remarkably distended, but a little fluid was found in the peritoneal cavity, and the surface of the membrane was studded over with small miliary granules. All the pelvic organs were more or less involved in cancerous disease, and small nodules, slightly larger than those on the serous membrane, were infiltrated into the muscles and fat of the abdomen. The kidneys were very pale, and presented an early stage of the waxy (non-granular) degeneration.

On opening the thorax, which appeared contracted and flattened laterally, the anterior edges of the lungs covered the heart more than usual. Both lungs were evidently emphysematous anteriorly. In the left lung large bullæ existed at the lower part of the upper lobe in its anterior prolongation; the dilated air cells forming a portion connected with the rest of the lung by a narrow process, composed of pulmonary tissue in part emphysematous, and in part completely collapsed and flaccid. The anterior edge of the lower lobe was also emphysematous. The pulmonary tissue at the posterior part of both lobes was perfectly collapsed and flaccid, tough, and of a violet colour; not containing enough of air to give the feeling of crepitation. The crepitant tissue passed very gradually into the collapsed. In the right lung there was diffused emphysema of the anterior edges, with here and there collapsed portions. Fully one-third of the posterior portion of this lung was completely collapsed; the rest more or less crepitant,—the non-crepitant tissue at some points shading off gradually into the crepitant; at others it being pretty sharply divided from it by the interlobular septa. On inflating the lungs artificially, the collapsed portions, for the most part, admitted air pretty freely when considerable force was employed; some of them, however, even after several repetitions of this process, retained their collapsed appearance. On making incisions into the lungs, the dense portions were seen to be smooth on section, and of a deep mahogany colour, and contrasting remarkably with the brighter colour of the crepitating parts. The collapsed tissue yielded nearly pure blood to the point of the knife; the crepitating portions a frothy sanguinolent serum. Some of the denser portions sunk in water; other parts were indifferent; and many floated freely. A large quantity of tough, semi-transparent, stringy mucus existed in the bronchi of both lungs. The mucous membrane was of a purple colour. Heart healthy.

Case IV.—*Lobular Collapse of Lung from Bronchial Obstruction—Interlobular Emphysema—Death from Tubercular Hydrocephalus.*—(P. R. 360.)

A child, aged about 5 years, much emaciated, died with symptoms of hydrocephalus. The state of the lungs not noted during life.

The body was examined June 30th, 1850. Inflammatory lymph was deposited beneath the arachnoid at the base of the brain, and very minute tubercles existed over some of the convolutions. The ventricles were distended with serum. The root of both lungs was occupied by enlarged bronchial glands, which, on being cut into, were seen to be infiltrated with crude yellow tubercle. The right lung was healthy, with the exception of partial and imperfect collapse of some parts of its tissue, and considerable redness of the mucous membrane of the bronchi. The enlarged bronchial glands in the left lung pressed upon some of the bronchi going to the lower lobe, so as distinctly to diminish their caliber, as ascertained by a probe. The upper and lower lobes were glued together by adhesions, and in the upper margin of the lower lobe was a rounded

portion of condensed lung of the size of a walnut, which, on being cut into, showed a thick cluster of yellow tubercles, mostly confluent, and at some parts breaking down into pus. The anterior extremity of the lower lobe was completely collapsed and violet coloured, evidently sunk below the rest of the lung. The anterior end of the upper lobe showed a very few slightly enlarged air-cells at the extreme margin; the interlobular septa were emphysematous over a considerable extent of this margin, even where the air-cells were normal. On inflating the left lung, it was found that the collapsed tissue could be imperfectly inflated by using considerable pressure, but subsided again when the pressure was removed.

In the last case, besides some general bronchitis, there was a special obstructing cause in the bronchi going to the collapsed lobules; their caliber being diminished by the encroachments of enlarged bronchial glands, and of the isolated tubercular mass in the lung itself, which was situated immediately in contact with the collapsed part, between it and the root of the lung. In both these cases, the inflation of the diseased parts was effected with difficulty, and in some parts imperfectly; showing that their nutrition was already becoming modified, and the state of permanent atrophy was supervening on that of temporary collapse.

I have now to advert to the observations of the authors who have described collapse of the lung as a state distinct from pneumonic consolidation, or pleuritic carnification, with the view of showing in how far their researches have tended to throw light on the question of its origin. Laennec described the diffused form of collapse only in connection with pleuritic effusion and compression; and there can be little doubt, from some expressions in his descriptions of the first and second stages of pneumonia, that he must, to a considerable extent, have confounded the two conditions, especially in their more mingled and less characteristic forms. That he has not altogether overlooked the peculiarities of the lobular collapse, however, is evident from a passage in which he speaks of meeting with *carnified* portions of the size of a filbert or an almond in the midst of very crepitant pulmonary tissue. The occurrence of these he ascribes to "a slight inflammation in the first stage, the resolution of which, hastened perhaps by compression of the lung, has taken place in an irregular and imperfect manner."[1] This expression, while it shows that this great pathological anatomist had been puzzled to account for the lesion in question, will not be accepted as anything more than a hypothetical explanation of it. Very many writers, following Laennec for the most part in their pathology, have thrown this passage out of view altogether.

In 1829, M. Louis described the condition of the lung in a variety of cases of typhoid fever. These descriptions were repeated in his work on fever in 1841, in which the state of the lung in fevers was compared with that found in other diseases. The result of these inquiries was a most accurate description of the collapse of the lung

[1] De l'Auscultation Mediate, vol. i., ch. v., art. 1, sect. 366.

as a state altogether different from pneumonia, and which was found not only in typhoid fever, but in a variety of other diseases; chiefly at the posterior part of the lung, but sometimes *disseminated* (lobular.) M. Louis offers no speculation or remark as to the origin of this affection.[1]

In 1830, Dr Alderson, in a paper on the "Pathology of Hooping-Cough,"[2] pointed out most clearly the distinction between the lobular condensation observed in that disease, and true hepatization of the lung. "In hooping-cough the lung is always dense and contracted, as if the air had been expelled, and from the throwing out of adhesive matter the sides of the air cells had been agglutinated together; while in hepatization the lung is less dense than in hooping-cough, and is rendered more voluminous than in its natural state."[3] The inflation of the lungs would probably, in some cases at least, have clearly disproved this supposed agglutination of the air-cells. The state of the bronchi is carefully described by Dr Alderson. In one case, for instance, "most of the tubes were filled with a light yellow secretion, which, in the greater number, had assumed a concrete form, having very much the character of fibrine; in others, it was in the form of a thick puriform mucus; where it occurred in the concrete form, it adhered, though slightly, to the lining membrane of the tubes."[4] It will not be questioned that these descriptions concur entirely with the views already laid before the reader in this paper, as to the origin of the lesions in question.

The researches of Jörg, in 1832,[5] gave a new impulse to infantile pathology, by showing the frequent occurrence in new-born children of a state of deficient expansion of the pulmonary lobules, having all the appearance of being congenital, but often persisting for some time after birth. This state, which Jörg called *atelectasis*, was identical in its characters with the collapse described above, and, like it, was often lobular in its distribution. It was figured as a disease of the new-born child by Cruveilhier,[6] who, however, understood nothing as to its real nature. Jörg was the first who pointed out the effects of inflation in this form of pulmonary affection as contradistinguished from pneumonia. He ascribed the collapse to various causes, but particularly to weakness on the part of the child, and to

[1] Recherches sur la Gastro-enterite, I. 361; and Recherches, &c., sur la Fievre Typhoide, I. pp. 328-334. It is worthy of remark that, from the observations in the preceding pages on the Edinburgh epidemics of 1847, as well as from Dr Jenner's recent careful descriptions of cases observed in London (Monthly Journal, Feb. 1850, p. 115, et seq.), this form of pulmonary lesion does not appear to be peculiar to any type or form of continued fever, at least in this country.

[2] Medico-Chirurgical Transactions, vol. xvi. p. 78.

[3] Loc. Cit., p. 91.

[4] Loc. Cit. p. 85.

[5] De Pulm. Vitio Organico; Leipz. 1832; and Die Fötus-Lunge im Gebornen Kinde; Grimma 1835.

[6] Anat. Pathologique, livraison 15, plate 2, fig. 1.

the circumstances of a too precipitate birth, which he conceived, somewhat fancifully, to militate strongly against the establishment of respiration. In this last view he has had few supporters.

In 1838, MM. Barthez and Rilliet published a monograph on infantile pneumonia, which afterwards became the basis of the extended treatises in their great work on the diseases of children.[1] While these authors describe with great care and general accuracy the appearances of the carnified lung, whether lobular or diffused, they fall into the error, at that time universal in France, of considering it as a form of pneumonia; and their reference to the researches of Jörg is so slight as to render it probable that they were not aware of the important facts established by him. An interesting remark in relation to the present subject, is, however, made by MM. Barthez and Rilliet, viz.:—that bronchitis, especially of the smaller bronchi, is a frequent concomitant of the pneumonia of children, *especially the lobular form* (collapse); the connection of bronchitis with the lobar pneumonia being more rare.[2] Similar observations had been made by M. Fauvel and other authors.

Rokitansky, writing in 1842, does not allude to any of the researches hitherto mentioned; nor does he appear to be sufficiently aware of the distinctive marks and real nature of collapse of the lung. In his description of *lobular, typhoid,* and *catarrhal pneumonia,*[3] however, the reader will trace many of the characters of this condition. In regard to the last of these affections, he says, "It is always lobular, concurring with catarrhal affection of the bronchial ramifications leading to the diseased lobules, and occurring frequently in catarrhal attacks in children, especially hooping-cough and catarrhus suffocativus." * * "The lobuli affected are blueish-red, dense, and rather tough; the walls of the air-vesicles are swollen, so as to obliterate their cavities, and contain, when less swollen, a sero-mucous, slightly frothy secretion; there is no trace of granulations." Rokitansky also notices the sinking in of the affected lobules, which he ascribes to an emphysematous condition of the surrounding parts.[4]

In 1844 were published the important researches of MM. Bailly and Legendre, before alluded to, which demonstrated the identity of the "lobular pneumonia" of children with the congenital collapse, or *atelectasis,* of Jörg. These authors also describe the catarrhal affections of infants as often attended with this change. But it is singular, that notwithstanding their own application of inflation of the lung to show the nature of this lesion, they consider it as produced, in some instances, by distension of the blood-vessels causing closure of the air cells. It is clear that, if this were the case, inflation could not effect any considerable change.

[1] Traité Clinique et Pratique des Maladies des Enfans. 1843.
[2] Op. Cit., vol. i. p. 75. [3] Path. Anat. vol. iii.
[4] Path. Anat., vol. iii., p. 106.

The greater number of these authors, in so far as concerns the diseases of children, are passed in review by Dr West in his late carefully elaborated, and, at the same time, original lectures.[1] The descriptions of Dr West well deserve to be read, on account of their clearness and accuracy of detail; and his views as to the frequent connection of the collapse of the lung with bronchitis, and its dependence, in many cases, on that affection, harmonize so closely with my own, that I can scarcely regret the late period at which I became acquainted with them,[2] as it enables me to point out more clearly the identity of the conditions in the adult with those in the child. "In the child," says Dr West, "nothing more is needed than a copious secretion of mucus into the bronchi, or a feeble condition of the vital powers, to prevent the air from freely entering the pulmonary vesicles, and thus to induce the collapse of a large portion of the lung."[3] I have already endeavoured, in part, to show that in the adult the same causes are capable of producing the same effects; and I shall hereafter explain more at length what I conceive to be the exact mechanism of this change, both in the one and in the other. Dr West adduces three cases, examined by Dr Baly, of lobular collapse of the lung in the adult, in persons who died in a state of great exhaustion from fever and dysentery. In two of these cases there was much dyspnœa and distinct signs of bronchitis. The third is said to have presented no complication, and is considered by Dr West to be the "result of simple debility." I shall refer to this point in the sequel.

On reviewing the whole of the facts here presented to the reader, I think that the frequency of collapse of the pulmonary tissue, both in the adult and the child, must be considered as established, and its connection with bronchial obstruction rendered at least extremely probable. I have adduced evidence that the condensation of the pulmonary tissue thus produced, which in its slighter degrees is often overlooked by anatomists, has in its more marked forms been described by many careful and exact observers under different names, and with various ideas of its pathological significance,—and that, especially in the case of children, it has been accurately distinguished from ordinary pneumonic condensation. I have described the forms in which this lesion has occurred under my own observation, and showed that, in all essential characters, it is the same in children and adults; that in both a certain degree of pulmonary collapse may be almost invariably found as a concomitant of fatal bronchitis; and that, in some cases, this state of the lung bears so obvious and undeniable a

[1] Lectures on the Diseases of Infancy and Childhood: Lond. 1848.
[2] Dr West's researches have come to my knowledge only since this paper was read, in its original form, to the Medico-Chirurgical Society.
[3] Op. Cit. p. 183.

relation to obstruction of the tubes, as to lead to the almost unavoidable inference of the dependence of the former upon the latter.

That this conclusion from anatomical data is, in all respects, consistent with clinical experience, and with correct views of the mechanism of respiration, I shall presently endeavour to prove; and, in the second part of this memoir, I propose to show the probable dependence of many important chronic alterations of the pulmonary texture on the condition of collapse from bronchial obstruction. The pathological history of this lesion, however, in relation to acute bronchitis, would not be complete without some notice of a condition which occurs very frequently in connection with it, and leads to some of its most important secondary consequences.

Bronchial Abscess.

It not unfrequently happens that, in the centre of the collapsed lobules of a lung affected with acute bronchitis, there are found small collections of pus, varying in size from that of a hemp-seed upwards to double or treble that volume. These small abscesses present, on section, an appearance so like that of softening tubercles, as to be very readily mistaken by many persons for these bodies; and the resemblance is all the greater on account of the peculiar limited form of the condensation by which they are generally surrounded, which, when felt by the touch from the exterior of the lung, is exceedingly deceptive. In their interior, however, these little abscesses contain, in the recent state, a very fluid pus; moreover, they are often met with as acute lesions produced by a few days of illness, and without a trace of tubercle in any other organ. This is peculiarly characteristic in the young child, in which tubercle of the lung so very rarely occurs without extensive deposits in the bronchial glands, whereas the present lesion is accompanied in its pure form by nothing more than slight enlargement. When the pus contained in these abscesses, in their recent form, is pressed or scraped out, they are seen to be lined with a fine villous false membrane, very different from the thick curdy mass which generally surrounds softened tubercles; in others they are not abruptly limited at all, the pus appearing to lie in contact with the surrounding pulmonary tissue. When the bronchi leading to the lung so affected are carefully incised, they are found much inflamed; the mucous membrane vascular, thickened, and covered with pus; and some of the bronchi thus affected can be observed to communicate with these purulent collections, the mucous membrane having evidently been, at the point of communication, destroyed by ulceration, and either stopping short abruptly, or becoming gradually incorporated with the false membrane lining the abscess. Sometimes these abscesses are found to break into one another, and form more considerable excavations; in one instance (Case VII.) I found them connected with a gangrenous condition; more commonly, however, they remain

of limited size, preserving perfectly the direction and relations of the bronchial tubes.

These abscesses occur in the diffused as well as in the lobular form of condensation from collapse; and both forms may sometimes be seen in the same lung. A similar lesion may accompany true pneumonia, but always in those cases where it is combined with intense bronchial inflammation. In this case, also, it tends to the formation of more considerable and more irregular excavations.

Such cavities have been pretty accurately described by Barthez and Rilliet (as well as by other French writers on the bronchitis and pneumonia of children), under the name of "*vacuoles*." They are, however, far from uncommon in adults, though, perhaps, more frequent in the so-called "lobular pneumonia" of children. As these "vacuoles" unquestionably arise from the accumulation of pus primarily in the extreme bronchial tubes of the collapsed lobules, the name of bronchial abscesses may serve to distinguish them from other forms of excavation of the lung.

That Laennec should have overlooked these small excavations is singular enough. Probably he may have considered them a form of tubercle, a term which has been used by him and others with sufficient vagueness to cover a multitude of anomalous lesions. His assertion that in many hundreds of pneumonic lungs, he had only met with collections of pus five or six times, can only be explained upon this principle. It is, indeed, quite true that in simple pneumonia there is little tendency to the formation of abscess, the pus escaping rapidly by the pervious bronchi; but as we have seen that Laennec has probably confounded the pneumonic with the bronchitic condensation, I think it also probable that he has not distinguished the bronchial from the tubercular abscess, especially as these lesions often resemble each other so much as to have led me habitually, for some time, to call the former *tuberculoid disease* of the lung, which name is inscribed over several dissections in the pathological register from which the following cases are taken.

In three of the four cases of fatal hooping-cough recorded by Dr Alderson, in the paper formerly alluded to, these lesions are described shortly as dilatations of the smaller bronchi by thick mucus or muco-purulent secretion. Most of the French writers on the pneumonia and bronchitis of children since Barthez and Rilliet, refer to them more or less distinctly; and in Dr West's lectures they are described as a *true* lobular pneumonia—the result, however, of bronchitis. As the work of Dr West is probably accessible to most readers, I do not think it necessary to transcribe the passage, which contains, however, a very accurate description of the lesion in question—(see p. 174, op. cit.) In regard to the characters of these abscesses, as distinguished from tubercle, my observations, as detailed above, almost exactly concur with those of Dr West. In one point alone I am disposed to differ from him. He seems to regard the abscesses as not formed in truly *carnified* lung, because the condensed por-

tions are not always *exactly* circumscribed by the interlobular tissue. I believe this appearance will be fully accounted for by the fact that when bronchial abscesses occur, the bronchitis is usually general and intense, and the collapse correspondingly diffused. Besides, I have found bronchial abscesses, and their results, in exactly circumscribed lobules, in repeated instances; and that the tissue in which they occur is mostly collapsed there can be no doubt, from the whole of its ordinary and microscopic characters. (See Case V.)

With these remarks I shall lay before the reader a few examples of this interesting lesion, which undoubtedly forms one of the most frequent consequences of intense bronchitis in children, and is also not rare in adults. With the exception of Case V., these observations, like many others which I have recorded, were made without the knowledge of those of the other authors mentioned above.

Case V.—*Death from Dysentery—Extensive Collapse of Lung, with Bronchial Abscesses—Emphysema of Lung.*—(P. R. 350.)

A woman, æt. 30,—body examined June 8, 1850.

The body was much emaciated and pale.

Heart normal. Both pleuræ contained a small quantity of fluid, with some floating and adherent soft lymph. The lungs presented great variations in density; the anterior edges were partially emphysematous, but between the portions thus affected could be felt numerous condensed parts, which, when superficial, presented a somewhat sunk collapsed appearance, and a deep purple colour. At the posterior part of the lungs were considerable masses similarly condensed. On cutting into the pulmonary tissue, there were seen, throughout the condensed portions, numerous small yellow points resembling softened tubercles, but more irregular in outline; these, when scraped with the knife, were found to be bronchial tubes, or small cavities filled with and surrounded by pus. Except at these points, the condensed tissue yielded to the knife only a little sero-sanguinolent fluid, which, when examined under the microscope, contained mostly blood corpuscles, with a few epithelial cells and pus corpuscles.

The colon presented a marked example of follicular dysenteric ulcers, which had destroyed a considerable portion of the mucous membrane.

This case presents an example of the very first stage of the disease, the mucous membrane of the bronchi being as yet scarcely destroyed. It is the same affection of the lung as appears to be figured by Dr Addison in Guy's Hospital Reports, Series 2d, Vol. III., Plate 3. Its resemblance to tubercle, as well as its distinguishing characters, have been perfectly correctly appreciated by Dr Addison.

Case VI.—*Bronchitis after Fever, with Collapse of Lung—Death from this cause and Dysentery—Bronchial Abscesses.*—(P. R. 62.)

A boy, æt. 11, admitted under Dr Bennett, 13th Dec. 1848, in a delirious state, and with the symptoms of typhus fever. He continued in this state with weak pulse up to the 22d, when the tongue was cleaner, the pulse still weak, and there was slight erythema on the back. Next day cough attracted attention; the chest was somewhat dull at the lower part; there were sibilant râles, and on the 24th slight crepitation over the lower lobes of either lung. On the 27th dysenteric symptoms supervened, and continued till his death. Erythema also spread over the face. On the 6th January there was dyspnœa with clogged bronchial tubes. He died in the course of the night.

The dissection was performed on the 9th January. The body was considerably emaciated.

The posterior part of the left lung was gorged with blood and serum, and crepitated very imperfectly; in most parts of the inferior lobe, and at the lower border of the upper, it was quite dense and contained no air. The condensed portions were not granular on section, but gave out on pressure a considerable quantity of reddish serum; throughout them were irregularly disposed yellowish opaque tubercular-looking points, which could be seen and felt through the pleura, being more resisting than the rest of the tissue; they did not exceed in general the diameter of 2 or 3 lines, and were irregularly circular in form. On pressure, they yielded a quantity of yellowish fluid mixed with a small quantity of air; and most of them could be seen to be perforated by an oval aperture admitting a probe, by which the communication of these cavities with the bronchi could be easily ascertained in all the larger ones. On slitting up a few of them, they were found lined with a reddened injected mucous (?) membrane. The lower edge of the right lung was partially condensed, and contained enlarged and thickened bronchial tubes (?) in the condensed portion.

The mucous membrane of the colon presented extensive ulceration, the characters of which need not be particularly detailed.

It will be seen from the expressions now marked (?) that this, like some of the other cases given, contains what I now consider the results of imperfect observation. The "thickened bronchial tubes" and the "mucous membrane" here adverted to, are in fact not normal structures, but false membranes supplanting those which have been destroyed by ulceration. Of this I am now well assured by other observations.

Case VII.—*Intense Bronchitis—Bronchial Abscesses and Gangrene of Lung—Emphysema of Anterior Edges.*—(P. R. 284.)

A labourer, æt. 40, admitted under Dr Bennett, Jan. 6, 1850, with intense cough and dyspnœa. Had been sometime affected with these symptoms, but more severely for a week before admission. Face swollen, lips livid, difficulty in inspiration. Chest arched, very resonant on percussion. Expiration prolonged. Sibilant râles, with inspiration, occasionally. Expectoration tenacious, frothy, muco-purulent. On the 20th he was relieved; on the 25th much worse—feeling of "smothering." 26th, Quiet at night, but dyspnœa very great in the morning. Died at 9½ A.M.

On dissection, the heart was found soft, and its fibres granular under the microscope. Both pleuræ presented adhesions, not very firm. The bronchi were filled with frothy sanguinolent pus, and their mucous membrane was very red. The crepitation of the upper part of both lungs was impaired, but was nowhere completely destroyed over a large space. Distributed throughout the lung were small condensed patches, which gave to the touch somewhat the feeling of tubercles, but on section appeared to be less consistent and more diffused. A few condensed patches of larger size than the majority existed in the lower and anterior part of the left lung, and one of these on section displayed a distinct gangrenous cavity, capable of containing a cherry-stone, lined by a tolerably distinct membrane, and containing a fetid sanies intermixed with debris of fibrous tissue. Anterior edges of both lungs emphysematous.

Case VIII.—*Measles, with intense Bronchitis—Collapse of Lung—Bronchial Abscesses—Partial Emphysema.*—(P. R. 272.)

A boy, æt. 8, admitted December 19, 1849, under Dr Bennett, suffering under measles, with mucous râles over the chest, which was clear on percus-

sion. On the 24th, he had an attack of croupy breathing, relieved by an emetic. On the 26th, chest symptoms aggravated, with loss of appetite, and fever; crepitating râles all over chest; but no dulness on percussion. *5th January* 1850.—Much dyspnœa, pulse quick and feeble. *6th*.—Dulness observed behind on both sides of the chest. Died on 10th January.

Dissection performed January 11th. The lungs collapsed imperfectly on opening the thorax; they were emphysematous at their anterior edges, and presented some irregularities of surface. Condensed lobules were felt in various parts,—frequently in close contact with, or in the midst of, the emphysematous portions. On section, the lower lobes presented irregularly disposed and irregularly formed yellowish deposits,—the largest of the size of a small bean, frequently perforated by bronchial tubes, and less sharply circumscribed than tubercles.

The last three cases, the history and symptoms of which during life are extracted from the clinical case-books of the hospital, will, I think, appear to the observant physician the type of many others which have come under his notice, especially if his field of observation have embraced the diseases of children, or of hospital patients enfeebled by fevers and chronic diseases. The rapid supervention, and sometimes equally rapid evanescence, of dulness on percussion, limited or diffused, in the midst of attacks of general bronchitis of greater or less intensity, and when the respiratory sounds are muffled or supplanted by large and small mucous râles in every part of the chest, is an occurrence which has impressed itself on my recollection in numerous instances,—of most of which, however, I have no records, or only notes too meager to be placed before the reader. Such an event comparatively seldom occurs in the acute catarrh of healthy adults; but, as a sequela of measles and hooping-cough in children, it must be familiar to every one; and I have seen it again and again in adults, in the bronchitis of fever,—in that which often terminates Bright's disease, or disease of the heart,—and still more frequently in those obscure and treacherous chest affections, which supervene in the last stage of exhaustion from hectic or malignant disease, and which are the accompaniments, rather than the active causes, of breaking-up of the enfeebled frame. Such affections have commonly been called latent pneumonia, and they undoubtedly may be so in some instances; but much more commonly they are nothing more than bronchitic collapse, determined by obstruction so slight as would in an otherwise healthy individual be easily overcome, and which, even in these emaciated and bloodless subjects, is only *not* overcome because the exhaustion is great and the need of respiration small,—the circulation being at a low ebb, and the attenuated and sluggish blood requiring very much less air, and consequently less lung, for its renovation than under ordinary conditions. Again and again has it occurred to me, under such circumstances, to open the bodies of persons in whom no suspicion of a respiratory affection existed during life, and in whom, nevertheless, condensation, abruptly lobular or diffused, having often the characters of the "peripneumonie des agonisans," or the "peripneumonie hypostatique" of M. Piorry (both of them varieties of the bronchitic collapse), has been found after death affecting large portions of one or both lungs.[1] On the other hand, I can remember instances in which a superfluous zeal, or *nimia diligentia*, in stethoscopic studies, has detected the signs of these lesions, when not a single rational symptom existed to call for such examination; and very many cases where the extent of the lesion was altogether out of proportion to the gravity of the symptoms that attended its accession.

Apart, however, from such latent, or very obscure cases, it has often been remarked of late years, that the pneumonic affections springing from or accompanying bronchitis, are apt to present a peculiarly asthenic or typhoid character. So much has this been the case in France, that within the last few years "pneumonie catarrhale" and "pneumonie typhoide" have become almost synonymous terms with some authors. Seeing that a great number of cases of so-called broncho-pneumonia which have come under my notice have been instances, not of true pneumonia, but of bronchitic collapse, sometimes combined with engorgement or œdema of the lung, and sometimes with bronchial abscesses, I am inclined to believe that the solution of the asthenic or typhoid character of this disease will be found in a modified view of its pathology. The fact is, that in adults the tendency in bronchitis to complete collapse of the lung is comparatively small, excepting in exhausted or debilitated constitutions, or under the influence of typhoid affections of the system. In these the asthenia is not a result of the disease, but one of its most essential predisposing causes.

In the child, again, owing to causes which will be presently mentioned, collapse, in its most complete form, very readily occurs under all circumstances, as a consequence of acute bronchitic attacks. In commenting upon this peculiarity, Dr West, whose opinion on this subject I have before referred to, remarks that it is by the collapse of the lung "that we must explain many of the instances in which urgent dyspnœa, and all the symptoms of serious pulmonary disease, have developed themselves in the course of a few hours out of what had seemed to be a severe cold, or a bronchitis of moderate intensity. This, too, accounts for the occasional supervention of dulness on percussion, and of bronchial respiration in the child; so that you may discover them in the morning in a situation where, overnight, the percussion was good, and no sound of graver import than large crepitation."[2] To this remark I would only add, that dulness on percussion supervening under such circumstances is not necessarily attended with any increase of fever; and that careful attention to the state of the patient in this respect may be in some cases the only mode of forming an opinion with any degree of accuracy as to the presence or absence of true pneumonia in a case of bronchitis. Of

[1] Instances of this are to be found in Cases I., III., IV., V.
[2] Op. Cit. p. 183.

this fact a striking instance occurred to me lately in the case of a little boy, three years old, whom I attended during an attack of acute bronchitis. He was a delicate child, and had suffered several times from bronchitic attacks. His chest presented a good deal of deformity, particularly on the left side, of the kind called "pigeon-breast." On the fourth day of the attack, which had all the usual acute characters during the first three days, decided dulness on percussion, with obscurity of respiration, appeared in the right back; the dyspnœa continuing considerable, but *the fever rather abating than increasing*. Under the influence of an emetic, and a forced decubitus on the opposite side to that on which the dulness existed, almost every trace of it had disappeared in thirty-six hours, and the dyspnœa was entirely relieved.

If these views be correct, they cannot be without practical importance. The rules, both of diagnosis and treatment, in regard to bronchitis and its complications, must probably undergo some modifications, in order to be safely followed. Such an axiom as the following, at least, which is one very generally conceived to be unquestionable, will not, I believe, stand the test of renewed observation, if the fact of collapse of the lung from bronchial obstruction be kept in view. "The stroke-sound of the chest," says Dr Williams, "is not materially impaired by catarrh; and, accordingly, the partial suspension of the breath-sound in a part of the chest in this disease *cannot be mistaken for that caused by hepatization*."[1] The reader will at once perceive that the clearness of the stroke-sound here alluded to is not always, perhaps not even generally, to be expected under such circumstances; nor can the opposite condition, even when combined with diminished or suppressed respiratory murmur, or with bronchial respiration, be allowed necessarily to indicate hepatization. The judicious physician has doubtless often been saved from error in the application of his remedies in this, as in many other cases where an unsound pathology has prevailed, by adhering to the great principles of our art, and the teaching of personal experience, rather than to received formulas and rules on matters of detail. But it is difficult to escape from the conviction, that the influence of a name, or the vanity of an *exact* diagnosis, may have misled many into such errors; and that in cases of supposed broncho-pneumonia or typhoid pneumonia, the practice may frequently have embraced blood-letting, heroic antimonials, or calomel and opium, when emetics and expectorants, with suitable stimulation, would have been better adapted to the circumstances of the case. This will be still more evident on considering the mechanism of these affections.

Since the preceding observations were in type, I have received the

[1] Williams on Diseases of the Chest, 4th edition, p. 80. Similar rules of diagnosis are given by Laennec, Skoda, Watson, and most other writers.

recent work of Dr Fuchs, on the bronchitis of children,[1] which in its elaborate descriptions, both of symptoms and post-mortem appearances, furnishes additional evidence of the connection of collapsed lung with bronchitis. The state of the lung, called "lobular pneumonia" by most authors, and "état fœtal" by Legendre, is regarded by Fuchs as a direct consequence of bronchitis; and its relation to bronchial obstruction on the one hand, and to peculiarities in the infantile system on the other, is certainly more clearly stated than by any previous author. To distinguish this lesion from the congenital form of collapse, as well as from other varieties of condensation, Fuchs proposes the term *apneumatosis*;—but, although evidently anxious to point out a sufficient anatomical ground of distinction between the *unexpanded* and the secondarily *collapsed* lung, he admits that the *diagnosis* must rest chiefly on a consideration of the cause—the one being congenital and the other acquired (See pp. 112, 113). Under these circumstances it will probably, I think, appear to English readers unnecessary to burden the science of pathology with another scholastic term; and I am convinced that careful examination of both lesions will convince most observers that Legendre and Bailly are correct, and that there is no real ground for distinguishing them, excepting what may be inferred as to their mode of origin.

The simplicity and clearness of the pathological views entertained by Fuchs, as compared with most other writers on this subject, and the highly original character of his work, render it a most important contribution to the history of bronchitis; and as such, I shall have occasion to refer to it in the sequel. In the meantime, it is only necessary to say that he enumerates three stages of *apneumatosis*, each of which is described at great length. Into the anatomical description of these I do not mean to enter, as it refers exclusively to children below the age of five years. The reader of the original work will see many points of similarity between the characters there given and those which I have indicated as distinguishing bronchitic collapse in the adult as well as the child. In the case of children under five years, Dr Fuchs, indeed, denies having seen a true pneumonic condensation; and he appears also to have passed over, very lightly, the lesion above described as bronchial abscess; the only allusion to it being in p. 114, where he remarks, cursorily, on "the accumulation of yellow mucus in the bronchi and air-cells, *and their dilatations*;" this being, as he declares (in opposition to Friedleben, as well as to other authors), the only form of suppuration in the lung observed in early infancy. These assertions are entitled to due consideration, but can scarcely be accepted without further, and even more careful, examination.

[1] Die Bronchitis der Kinder, &c., von Dr Caspar Friedrich Fuchs. Leipzig. 1849. The reader will find a notice of this work in the last No. of the British and Foreign Med. Chir. Review.

It appears from the work of Dr Fuchs, that, in 1837, a year before the publication of the monograph of Rilliet and Barthez, Seifert[1] had given an excellent description of infantile bronchitis, and of the peculiar pulmonary lesions in which it commonly terminates, which he considered to be a peculiar form of pneumonia. This work seems to have excited some attention in Germany, and it is not a little remarkable, under these circumstances, that the close relation of these lesions to the congenital affection described by Jörg, should have been overlooked until the observations of Legendre and Bailly, in France, seven years afterwards. Seifert also noticed the resemblance of the "broncho-pneumonia" of children, to the "peripneumonie des agonisans" of adults; and seems to have described the bronchial abscesses as a stage of suppuration.

Mechanism, Causes, &c., of Bronchitic Collapse.—The object of the remarks hitherto made, in reference to this subject, has been chiefly to establish the frequent coincidence and probable relation of cause and effect between the obstruction of the bronchi and the collapse of the air-vesicles. The nature and rationale of that connection now fall to be more particularly considered.

When a bronchial tube is in any way obstructed, or much diminished in caliber, at one or more points, the question arises, what is the mechanical effect of the movements of respiration upon the amount of air thus partially imprisoned behind the obstructed part? If the obstruction be complete, of course no change can take place, at least mechanically; but in the case of its being, as it usually is, incomplete, it may be supposed that the inspiratory act tends to draw in more air than the expiration can expel, and consequently that the air tends to *accumulate* in the vesicles; or, *vice-versa*, that the air behind the obstruction tends constantly to *diminish* in amount, owing to the comparative inefficiency of the inspiratory act; or finally, that the forces equalise each other, and the quantity of the enclosed air *remains unaltered*.

Now, it cannot be denied that from the pathological anatomy of bronchitis, a *prima facie* case might be made out for each or all of these theories; for, although only one side of the question has hitherto been brought prominently forward in the preceding part of this paper, the reader will not have failed to remark that in several cases in which bronchitic collapse of the lungs existed, other parts of the same organs were affected with emphysema or dilatation of the air-cells. (See Cases III., IV., V., VII., VIII.) Indeed, so familiar is this conjunction of emphysema with bronchitis, as to have suggested to Laennec, long ago, the first of the three theories above mentioned as to the cause of dilatation of the air-vesicles in that disease. Again, fatal cases of bronchitis undoubtedly occur, at least in adults, in which

[1] Die Bronchio-pneumonie der Neugebornen. Philipp Seifert. Berlin, 1837.

there is no change of the pulmonary texture so marked as to afford support to either of the first two theories.

Laennec's view of the consequences of obstruction was founded on the idea of the comparative weakness of the expiratory, as compared with the inspiratory, forces. "The mucus secreted into the bronchi, in consequence of pulmonary catarrh, must, especially if it is very viscous, present a great resistance to the free passage of air in inspiration and expiration; and we shall show, in speaking of the râle, that this resistance often goes the length of producing complete, though momentary, obstruction of a part of the bronchial ramifications. Now, as the muscles which subserve inspiration are strong and numerous, while expiration is produced only by the elasticity of the parts, and the weak contractions of the intercostal muscles, it must necessarily happen that the air, which has been forcibly driven beyond the obstruction in inspiration, will not be able to overcome it in expiration, and will be in a manner imprisoned, by a mechanism not unlike that in the butt-end (condenser) of an air-gun." I have given this passage from Laennec entire, because, notwithstanding the palpable fallacy it contains, it has been referred to, and its conclusion adopted without comment, by almost every systematic writer in this country as well as in France. The fact is, however, that though *ordinary* inspiration is more of a muscular act than *ordinary* expiration (merely because in the latter there is little or no resistance to be overcome, to which the elastic subsidence of the parietes is not adequate), yet the residual effective force for overcoming adventitious obstruction is very considerably greater in expiration. The *forced* or *muscular* expiratory act is, in fact, about one-third more powerful, as measured by its effect upon a pressure-gauge, than the extreme force of inspiration;[1] and it is this force which is thrown into action when obstruction in the tubes is to be overcome. In the act of coughing, moreover, we find a beautiful mechanism, by which the air within the vesicles is discharged outwards at a *maximum amount* of pressure, and brought to bear with all the additional mechanical advantage of a sudden impulse, on every obstructing substance within the bronchial tree,—a cumulative provision which does not exist in the case of the inspiratory force. There can be no great difficulty, therefore, on these grounds, in coming to the conclusion, that the data of Laennec's hypothesis are quite erroneous, and that the practical efficiency of the expiration in forcing air through obstructions must be, *cæteris paribus*, far greater than that of the inspiration. I have already alluded to the fact, that this is consistent with general experience; for while the inspiratory act is always, in bronchitis of considerable intensity, attended with extreme difficulty, the expiration is never so.

[1] See the numerous experiments of Hutchinson and Mendelsohn, quoted in Dr J. Reid's article on Respiration: Cyclop. Anat. and Physiol. Part 32, p. 336.

The question of the origin of emphysema of the lung will be considered in the second part of this memoir, in which I shall endeavour to account for its connection with bronchitis, by referring its production to a totally different mechanism from that just mentioned. In the meantime, I would remark that, in order to establish a direct relation between this lesion and bronchial obstruction, it is necessary to show not merely that emphysema occurs in connection with bronchitis, but that it occurs especially or exclusively *in those parts of the bronchitic lung where obstruction can be shown to exist.* This is the proposition which I conceive the preceding pages have tended to establish as regards bronchitic collapse, and on the ground of which I have argued for the relation of cause and effect between this lesion and obstruction. In how far does emphysema fulfil these conditions?

It is well known to every one who has studied the anatomy of this pathological state, that the emphysematous portions of a lung can generally be inflated from the bronchi with the greatest ease. Indeed, so far as my own experience in this matter is concerned, I cannot recal any instance in which the pressure of the air was not found to reach the emphysematous parts with as great rapidity as the rest of the lung. In the collapsed lung, on the contrary, as I have already shown, very considerable resistance is often opposed to its inflation from the bronchi—a resistance only to be overcome by pressure many times greater than can ever occur in the vital act of inspiration. If this observation be correct, it is plain that the emphysematous parts of the lung are usually free, the collapsed parts obstructed.

Further, I cannot find that any unequivocal instances have been adduced, to prove that an obstruction, confined to a part of a lung, or to one lung only, is commonly accompanied by a corresponding distribution of emphysematous portions—a proposition which, if true, might surely be easily verified from the records of pathology. So far from this being the case, the habitual seat of emphysema leads to an inference of a directly opposite kind—a point which appears to have been overlooked in the discussion of this question. Let the reader reflect that, in the vast majority of instances, the seat of election of emphysema is the *anterior* border of the lungs, while the stethoscope, as well as the results of *post-mortem* examination, show that accumulations of mucus in bronchitis occupy in an equally numerous proportion of cases, the *posterior* and *lower* parts, which are also, especially in the adult, the principal seat of the bronchitic collapse.

Cases of the impaction of foreign bodies, and other palpable obstructions of the bronchi, are generally recorded with too little attention to the condition of the lungs to be available for the present discussion. Carswell has, however, figured the case of a monkey, in which the left bronchus was much compressed, or rather obliterated, by a mass of tuberculous glands; in this case the corresponding lung had diminished to less than a third of its normal bulk, while

the *opposite* lung, of which the bronchus was free, presented emphysema in several places.[1] Andral has adduced, as before mentioned, two cases of obstruction of the upper lobe of one lung, where, from the stethoscopic phenomena, he supposed emphysema to be present, but where the examination after death proved that this was not the case.[2] He has also recorded a case[3] in which the bronchi of the right lung were compressed by a melanotic mass, and the respiratory murmur greatly enfeebled. In the short note of the appearances after death, no notice is taken of any abnormal condition of the lung in this case. Andral, indeed, states (p. 196) that emphysema is one of the consequences of stricture of the bronchi; but adduces nothing whatever in proof of this assertion, which evidently rests on the ground of Laennec's theory.

All doubt, however, as to the real effect of a solid obstruction in the bronchi on the air in the lung is removed by the direct experiments of Mendelsohn and Traube on animals.[4] The former inserted a leaden shot into the trachea of a dog, pushing it down as far as possible into the bronchus with a probe. In another instance he inserted a ball of paper. In both cases, the parts to which the obstructed bronchi led were red and void of air. In the former there were emphysematous portions in the other parts,[5] and in the opposite lung.[6] Traube's experiments were similar, but more numerous. The general result was, that the artificial obstruction of a bronchus always produced expulsion of the air from the corresponding part of the lung, which had a dark-red colour, and presented the characters of collapse.[7]

It is clear, therefore, from experiment, as well as from pathological observation, that the most usual and most direct effect of obstruction, or of diminished caliber of the bronchi, however caused, is not accumulation, but diminution in quantity, of the air beyond the obstructed point. It is probable that this is due in part to the comparative weakness of the inspiratory power, and that the proposition of Laennec may, therefore, correctly enough be inverted. There is also, however, another mechanical condition which comes into play in producing collapse from obstruction, especially in the case of a viscid

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[1] Illustrations of the Elementary Forms of Disease—Atrophy. Plate iv., fig. 3.
[2] Clinique Médicale, v. 2, pp. 187-190. [3] Ibid, p. 193.
[4] For an account of these experiments I am indebted to the work of Fuchs, not having access to the original sources.
[5] The expression of this passage is not quite clear, but this is certainly the meaning, and corresponds with the author's inference.
[6] Der Mechanismus der Respiration und Circulation, p. 37. Berlin, 1845. Mendelsohn also threw a solution of gum into the air passages of an animal, with the result of collapse of some portions of the lung. In one instance I tried this experiment upon a rabbit, with a similar result; but the difficulty of limiting the fluid to particular parts of the lung makes these experiments less valuable.
[7] Beitrage zur experimentellen Pathologie und Physiologie, 1 Heft.

plug of mucus, which is most commonly, in bronchitis, the source of this affection. This condition is to be found in the form of the tubes.

The bronchi are a series of gradually diminishing cylinders, dividing, for the most part, dichotomously. If a plug of any kind, but especially one closely adapted to the form of the tube, and possessing considerable tenacity, be lodged in any portion of such a cylinder, it will move with much more difficulty towards the smaller end, and in doing so will close up the tapering tube much more tightly against the passage of air, than when moved in the opposite direction into a wider space. If such a plug be placed over a bifurcation, it will, even if freely moving in the larger space in which it lies, be of sufficient bulk to fall back upon one or other of the subdivisions during inspiration, in the manner of a ball-valve upon the orifice of a syringe, and thus completely to occlude it. (See diagram.)

The consequence of this mechanical arrangement must inevitably be, that at every expiration a portion of air will be expelled, which, in inspiration, is not restored, partly owing to the comparative weakness of the inspiratory force, and in part to the valvular action of the plug. If cough supervene, the plug may be entirely dislodged from its position, or expectorated, the air of course returning freely into the obstructed part; but if the expiratory force is only sufficient slightly to displace the plug, so as to allow of the outward passage of air, the inspiration will again bring it back to its former position, and the repetition of this process must, after a time, end in perfect collapse of the portion of lung usually fed with air by the obstructed bronchus.

It is not a little surprising that this simple and clear mechanical mode of explaining the collapse should not have occurred to Dr Fuchs, who, in accounting for the disappearance of the air in the experiments of Mendelsohn and Traube, finds himself reduced to the theory of its absorption into the blood-vessels. (Op. Cit., p. 63.) Surely nothing can be more superfluous than such an explanation.

In considering, as a whole, the causes which tend to produce bronchitic collapse (as revealed in the preceding investigation), they seem to resolve themselves into the following:—*Firstly*, the existence of mucus in the bronchi, which is more liable to produce obstruction according as it is tenacious and viscid; *secondly*, weakness, or inefficiency of the inspiratory power, however caused; *thirdly*, inability to cough and expectorate, and thus to remove the obstructing mucus. Of these conditions the first must be considered as the immediate exciting cause, the others as predisposing causes, co-operating with the first, but incapable without it of producing collapse. Of the exciting cause enough has already been said. The inability to expectorate is obviously enough a formidable condition, and may be owing either to simple debility, or to a laryngeal affection. But I have

still a few remarks to make on the circumstances producing inefficiency of the inspiratory act, and their bearing on the present subject.

The inspiratory act is apt to be rendered inadequate from several causes. Of these the most obvious is weakness of the muscles of inspiration, usually concurring with general debility. I have already pointed out the great predisposition to bronchitic collapse which arises from an exhausted frame; so much so, that a barely appreciable amount of bronchitis, nay sometimes, I believe, the mere accumulation of the natural mucus in a debilitated subject (as in an individual near death), will give rise to a considerable extent of the pulmonary lesion. I cannot, however, see reason to believe with Dr West, that mere debility, apart from any obstruction in the tubes, is a sufficient cause for collapse in the child. The very fact of the lesion being usually more or less lobular, or partial in its distribution, appears to indicate special circumstances of a local kind, as having a marked influence on the production of this affection; and on this ground, as well as that of theory, I am disposed to think that, in the cases ascribed to debility alone (including the third case of Dr Baly, before alluded to, p. 19), the evidences of more or less obstruction might have been detected during life or after death. That this, however, is often of a very slight character, leading to scarcely any symptoms, and probably in some cases undiscoverable, except by physical examination, I have already indicated. (See p. 24; also, Cases I., III., and IV.)

A second circumstance tending to render the inspiratory act ineffective is distension of the abdomen, impeding the descent of the diaphragm. The influence of this condition in the production of collapse I have repeatedly witnessed; having had occasion to observe that when ascites, or any other cause of similar distension, is present, a very slight amount of bronchitis will determine extensive pulmonary collapse. In Case I. this cause was in action, combined with great exhaustion; and one of the first cases that awakened my attention to the subject of this memoir, was that of a boy of 17 years of age, who died of an enormous medullary tumour of the abdomen, and in whom a most marked form of lobular collapse was found distributed over both lungs, with a good deal of thick mucus in the tubes.

A third cause of inefficiency of the inspiratory act, and one of the greatest importance in relation to this subject, is the want of due resistance on the part of the thoracic parietes. The full dilatation of the lungs is only effected when the depression of the diaphragm is accompanied by the elevation of the ribs and widening of the thorax; and if the bones of the latter be very yielding, the external muscles of inspiration cannot, of course, act effectively under an obstruction. This is obviously the reason of the greater tendency in children to collapse of the lung as a consequence of bronchitis. The respiration of the child is at all times, even in health, more diaphragmatic than that of the adult; and the observations of Rilliet

and Barthez[1] afford satisfactory evidence of the comparatively small dilatation of the thorax in children, particularly of its lower part. When any obstruction exists to the entrance of air into the chest, even this small dilatation ceases, and collapse of the lung very readily takes place. Under such circumstances, Dr Rees[2] has pointed out that in very young children the motions of the chest are absolutely reversed, and instead of the descent of the diaphragm being accompanied by expansion of the chest, the ribs give way beneath the exhaustion caused by it within the thoracic cavity, and bend inwards to accommodate themselves to the collapsed lung in inspiration. This altered movement of the chest in infants is regarded by Dr Rees as pathognomonic of *atelectasis*. It is also a prolific source of that permanent deformity of the chest which, in the early years of life, is often ascribed, with too little discrimination, to rickets.[3] Of this deformity I shall have something to say in the second part of this paper.

As to the so-called *atelectasis*, I have had but few opportunities of observing it accurately during life. I may, however, remark that in respect to its causation, it probably differs but little from the acquired collapse, and close examination would probably show that mere debility, without some obstruction in the bronchi, is as inadequate to prevent the expansion of the lung as to cause its collapse. The cases published by Jörg himself, although very imperfectly observed as regards physical signs, will, I think, on perusal, convince a careful reader that there is abundant ground for this opinion; but undoubtedly the subject requires renewed investigation.

Origin of Bronchial Abscess.—The mechanism of this lesion it is not difficult to explain satisfactorily. When pus accumulates in the central bronchi of a collapsed lobule, the evacuation of that pus is prevented from occurring, firstly, in consequence of the absence of the expiratory *vis a tergo*; and secondly, from the resistance opposed by the thickened mucous membrane and its secretion, closing up the bronchus in front. The coats of the ultimate bronchi, therefore, softened and injured by disease, gradually give way to ulceration; and the pus, which thus accumulates in still larger quantity, may at first scarcely be circumscribed, but soon begins to be surrounded by a false membrane exactly similar to that of an abscess in any other part of the body. The continuity of this membrane with that of the original bronchus, may be either maintained from its first formation, or it may be secondarily established. I believe, however, that

[1] Op. cit., vol. iii., pp. 643, 644.
[2] Atelectasis Pulmonum. Lond. 1850.
[3] Rilliet and Barthez describe a reversed movement of the ribs in inspiration as taking place in rickets. There can be little doubt that rickets, combined with chest affections, forms a frequent source of the deformity; but the presence of the latter is probably essential. Vol. iii., p. 646.

the first of these views is the correct one; and that the bronchus acts the part of an obstructed fistulous opening, not sufficiently pervious to prevent accumulation entirely, but not permitting of its increase beyond a certain amount.

When the bronchial abscess has been of some standing, and the patency of the tube leading to it has become re-established in time to prevent its obliteration, a process of repair takes place, analogous to the cicatrization of a wound, and perfectly similar to that which is observed in all healing excavations in the lung, however formed. The false membrane which lines the cavity becomes intimately blended with the bronchial mucous membrane, and indeed comes to resemble it so closely that it is almost impossible to tell where the true mucous membrane ceases and the new structure begins.

This reparation, however, is rarely, if ever, accompanied by restoration of the perfect function and structure of the lung; and on this account it will be considered under the permanent effects of bronchitis.

The length to which these remarks on collapse of the lung have extended, can only be excused by the immense importance of this lesion in relation to the pathology of bronchitis; the whole of the organic affections following from which seem to me more or less dependent on that which has formed the principal subject of these observations. These secondary effects of bronchitis and bronchitic collapse will form the subject of the second part of this memoir.

PART II.

SECONDARY RESULTS OF BRONCHITIS; OR, PERMANENT DISORGANISATIONS DEPENDING ON COLLAPSE OF THE LUNG, ETC.

In discussing the primary affections of the pulmonary texture resulting from Bronchitis, I have treated, at considerable length, of the collapse of the air-vesicles connected with obstructed tubes,—a lesion of which, as I have endeavoured to show, the true pathological significance has been much neglected or misapprehended, even by authors who have correctly enough described some of the morbid appearances. The more chronic and permanent lesions connected with long-continued bronchitis have now to come under consideration; and although these affections cannot be said, for the most part, to have engaged less than a due share of the attention of pathological writers since they were made familiar by the descriptions of Laennec, yet it will appear from the sequel, that the links which bind them together in a series, cannot be understood without reference to those primary results of bronchitis to which I have already adverted. In describing these affections, therefore, I shall avoid as much as possible dwelling on descriptions already familiar to well-informed medical readers, confining myself, for the most part, to the pathological considerations which flow more or less directly from what has already been advanced in the first part of this memoir.

Results of Bronchitic Collapse of the Lung—Curability of Pulmonary Collapse.—There can be little doubt that the condition of collapse of the air-vesicles, from obstruction of the bronchi, may, when recent, be completely removed, and give place to the normal condition of the pulmonary texture. The imitation of this result, by forcibly inflating the lung so affected after its removal from the body, not only proves the absence of any organic change, but shows conclusively that it is in many instances only a sufficiently strong inspiratory force which is required to disperse the obstructing mucus, and make a free passage for the entrance of air into the lung. The collapsed lung, however, is placed under a most serious disadvantage, as compared with that which contains air, in freeing the bronchi from causes of obstruction. The latter can render available the expiratory

force, and this, when aided by the impulsive effort of coughing, is by far the most efficient agent in displacing and removing the bronchial plug, which it carries outwards, and expels altogether from the system. The completely collapsed lung, on the other hand, can only bring into play the inspiratory force, a weaker power acting against a greater resistance, and able, at the best, only to disperse inwards, never directly to remove, the cause of obstruction. Under these circumstances, it might seem probable, that a lung when once brought into a state of complete collapse, should be mechanically incapable of perfect recovery; and even in minor degrees of the affection, it would appear that the tendency *inwards*, or towards the lung, of mucus, and all other obstructions, must greatly increase in proportion as the residual quantity of air in the vesicles diminishes. Again, where the obstruction is much localised, as in limited and lobular collapse, the mechanical forces tending to remove it, whether inspiratory or expiratory, will at all times be apt to diffuse themselves over the surrounding normal or comparatively unobstructed lobules, so that the removal of the bronchial plug under such circumstances is not easily understood, if we take into account only the forces we have hitherto been considering.

Does collapse of the lung, then, necessarily, or in the majority of instances, lead to organic and permanent change of structure? This doctrine I should be very slow to admit. A consideration of the cases in which bronchitis occurs, and is even repeated frequently in the same individual, without appreciable permanent change, while we know, from post-mortem appearances, and understand on mechanical grounds, that accumulation even to a very moderate extent in the bronchi, is often sufficient to cause a certain amount of the lesion, will, I think, even in the absence of more detailed clinical experience, constitute a strong case for believing in the existence of some more active remedial and conservative mechanism in such cases than that of the inspiratory and expiratory forces. Such a view is altogether borne out by the observations of writers on the bronchitis and lobular pneumonia of children, which, though often a grave, and even a fatal affection, is never regarded as being, in favourable cases, less capable of perfect resolution than any other form of pulmonary condensation. The remarks of most other practical writers are so much governed by pathological views, differing from those we have been considering, that it is quite impossible to eliminate the information they may contain as to the results of pulmonary collapse. I have already remarked that a great number of the varieties of so-called catarrhal and typhoid pneumonia are undoubtedly affections of this kind, sometimes combined with genuine pneumonia, and sometimes uncomplicated; and in particular, that the hypostatic pneumonia of M. Piorry, and the "peripneumonie des agonisans" of Laennec, are generally instances of the diffused form of pulmonary collapse. The former observer has devoted so much attention to the observation of this particular form of disease, as to render his remarks valuable,

even though probably modified by an erroneous pathology. He says:—"The first stage of hypostatic pneumonia, while the blood is still contained within the vessels (*i.e.*, while no exudation has occurred into the air cells,) is *very susceptible of cure;* indeed, it may be said that this state exists *in a large number of invalids* (chez beaucoup de malades), and *is dissipated during convalescence.*"[1] The other stages, especially the third and fourth, in which solid or purulent deposits exist in the air-cells, are, according to M. Piorry, more grave, and even generally incurable; but these again are obviously not uncomplicated instances of pulmonary collapse. I shall not at present enter more fully into the discussion of M. Piorry's views on this subject, than to remark, that the passage above quoted is in harmony with all that has been already submitted to the reader, more particularly with the observations alluded to in the last volume of this Journal, pp. 234–6. Indeed, I cannot entertain a doubt, judging from the facts there mentioned, that a more extensive and exact clinical experience bearing on this subject will demonstrate the extreme frequency, and in many cases the easy and rapid removal, of a certain degree of pulmonary collapse, which may or may not have led to serious symptoms during life.

De-obstruent Function of the Bronchial Tubes.—Supposing these views correct, the mechanism by which the viscid mucus is expelled to such an extent as to permit the return of air into the occluded vesicles, demands further consideration. We have seen that the expiratory forces are, under such circumstances, thrown out of action; while those of inspiration, even if strong enough to displace the obstructing plug, can never permanently remove it. Under these circumstances, it seems to be reasonable to ascribe to the bronchi themselves an active part in the expulsion of obstructive mucus, by means of the slow contraction of those circular fibres, the muscular character of which was demonstrated by Reisseisen, and whose physiological properties have been fully illustrated by the experiments of Dr Williams and others. It is now well established, that these fibres have no such vital endowments as would enable them to co-operate with the movements of respiration, influenced as these are by the will. "The contractility," says Dr Williams (of the bronchi), "resembles that of the intestines or of the arteries more than that of voluntary muscles or of the œsophagus, the contractions and relaxations being gradual and not sudden. They are, however, much less tardy than those of the arteries."[2] This kind of contractility is precisely that which empties the arteries of their blood after death, and which, in all probability, contributes to the passage of calculi along the ureters or gall-ducts. It is also more or less analogous to the peristaltic contraction of the intestines, or of the elongated tubular uterus of many of the lower animals, by which the

[1] Piorry—Pathologie Iatrique, vol. iv., p. 411.
[2] Williams—Diseases of the Chest, 4th Edition, p. 330.

solid or fluid contents of these viscera are gradually expelled towards their outlet. The experiments referred to appear to prove that the contractility of the air tubes is readily excited, not only by galvanism applied externally, but by mechanical and chemical stimuli in contact with their mucous membrane. It is easy, therefore, to understand, that the bronchi (or at least those which have not cartilaginous walls) may have a most important power of dislodging obstructions, altogether independently of the forces of respiration. When these forces are in active operation indeed, the tonic or slow contraction will be in abeyance, or very slightly manifested, as the air-tubes will then be dilated to their full extent at each inspiration and expiration. But, according as the admission of air to any part of the lung becomes less from obstruction, the detrusive action of the bronchial muscles will increase, being thus called into effective action precisely at the period when most required. Perhaps, also, the slighter contractions of these muscles may be in almost constant operation in the normal condition, to aid, by a kind of peristaltic movement, the outward passage of the physiological secretion. This secretion, comparatively small in quantity as it is, would almost necessarily tend to accumulate in the air-tubes (seeing that no efforts of coughing or forced expiration are made for its removal); and this would take place, particularly in the smaller bronchi, which we know to be especially subject to mechanical obstruction, and in which the ciliated epithelium, so abundant in the cartilaginous bronchi and trachea, gradually gives way to transition forms, not constantly furnished with cilia.

It may not be easy to adduce direct proof of the theory here proposed, as to the function of the bronchial muscles in health and disease; but as *no* theory upon this subject has yet been found consistent with our present physiological knowledge, and as the above speculation appears in all essential points to correspond with what is already known of the action of these muscles, it may be worth while to give it consideration, were it merely to rescue us from the unphilosophical predicament of supposing the circular fibres of the bronchi to be endowed with contractility, solely for the purpose of producing the asthmatic paroxysm. That these fibres are probably perfectly passive, as regards the respiratory act, is now generally admitted (contrary to the ancient opinion) by physiologists; and under these circumstances the theory of their de-obstruent action, even in health, but more especially in the diseased states of the pulmonary texture above described, appears to supply a gap in the chain both of physiological and of pathological phenomena.

The ordinary form of the paroxysm of spasmodic asthma, of the humoral kind, is full of instruction, when considered by the light of the preceding views. Notwithstanding the extremely doubtful and difficult pathology of this disease, it seems impossible to avoid referring its most obvious symptoms to some kind of irregular action of the muscular apparatus of the air-tubes. The copious expectoration, with which the attack concludes, and by which it is immediately relieved, appears to indicate that undue accumulation of mucus has been taking place; while the absence, in some instances, of all considerable catarrhal symptoms, appears to demonstrate that this accumulation is directly connected with the spasmodic derangement which produces the paroxysm. The connection of these two phenomena it is by no means difficult to understand, according to the principles already laid down; in fact, if the removal outwards of the pulmonary mucus depends, in the normal state, upon the regular peristaltic contraction of the bronchial muscular fibres, it is obvious that accumulation must accompany the derangement of that action, just as constipation is the invariable concomitant of the analogous derangement of colic or ileus. In both cases the paroxysm ceases when the normal action is restored; and in general there is in both a copious discharge of the previously retained excretions.

Asthmatic persons are often subject to a slight habitual wheezing in some part of the chest, and also to an occasional cough, with or without slight expectoration, but with no other symptom of catarrh. These symptoms have been described to me as occurring on exertion in the open air after prolonged rest; they are accompanied with slight dyspnœa, and this, together with the rest of the symptoms, ceases when the exertion is continued long enough to produce some degree of re-action. These phenomena are unquestionably the minor degree of the paroxysm; they are probably caused by the same irregular action of the bronchial muscles as causes the latter, but do not reach the climax, because the nervous centres are awake to the first approaches of disorder, and the excitement and quickened respiration consequent on exertion produce the cure. The aggravated asthmatic paroxysm always occurs during sleep, when the energy of the nervous system is at the lowest, and the comparatively quiescent condition of the respiratory function favours the accumulation of mucus. It seems probable that the asthmatic paroxysm is attended with more or less of pulmonary collapse, the consequence of the accumulation in the bronchi; but I have not had an opportunity of direct observation on this point. It is certain, however, that this accumulation must seriously contribute to the production of the most distressing symptoms of the paroxysm. The spontaneous cure in the real paroxysm, as in the minor attack, or threatening of asthma, above referred to, usually takes place when the nervous centres have been thoroughly roused, and the whole system brought into a state of re-action by the exertion consequent on the dyspnœa.

An interesting fact, in connection with asthma and other spasmodic respiratory diseases, is the frequent occurrence of vomiting during the paroxysms,—a fact which points to the probable dependence of all these affections on some morbid condition in the communication of which the pneumogastric nerve and the medulla oblongata are the principal parts concerned. A phenomenon exactly the converse of that just alluded to, is the profuse and immediate expectoration

in cases of obstructive bronchitis after the administration of an emetic. Now, it is interesting to observe, in relation to both these facts, and their bearing on the subject we have been considering, that Volkmann has apparently succeeded in demonstrating the influence of stimuli applied to the trunk of the vagus nerve upon the muscular contraction of the bronchi,—a point left open to doubt, both by the experiments of Williams and by the subsequent ones of Longet.[1] The expeditious and complete relief afforded by an emetic in cases in which there has been extreme difficulty of expectoration, is one of the most striking phenomena connected with bronchitis; and one of which, I believe, no sufficient explanation has yet been afforded. It appears, however, to be completely in harmony with the theory I have advanced in the preceding pages.

Another fact tending still furthur to illustrate this view, is found in the experiments of Reid, Longet, Schiff, and others,[2] on the effects of section of the pneumogastric trunk or of its visceral branches, on the lungs and bronchi. All experiments concur in proving that these operations are followed by a very large accumulation of frothy mucus in the bronchi. Changes in the lungs have also been observed, which seem to be of the nature of congestion and collapse, but are imperfectly described. M. Longet has also found emphysema of the lungs, the relations of which to pulmonary collapse will hereafter be considered; and there can be little doubt that we have in these cases all the phenomena of bronchial obstruction and collapse following the division of the nerve which, according to the views above proposed, is the chief regulator or excitor of the bronchial de-obstruent function.

To sum up the results of this discussion, as respects bronchitis, I would recapitulate the following points, which, if not established, seem to be at least rendered highly probable. *Firstly*, That pulmonary collapse from bronchitis, when recent and uncomplicated, appears to be susceptible of cure, on removal of the bronchial obstructions. *Secondly*, That this is usually effected, not so much by the agency of respiration, as by the muscular contractions of the obstructed bronchi themselves. *Thirdly*, That the derangement or paralysis of this de-obstruent function becomes a cause of bronchial accumulation even in the normal state of the mucous membrane, and, *a fortiori*, in cases of bronchitis. *Fourthly*, That the de-obstruent function of the bronchial tubes may be impaired by various causes

[1] Volkmann introduces into the trachea of a decapitated animal a tube having its outer end tapering, and perforated by a rather small opening. This being placed opposite a flame, he isolates and galvanises the vagus nerve, when, at every application of the stimulus, the flame is observed to be blown aside.—*Wagner's Handwörterbuch der Physiologie*, vol. ii., p. 586.

[2] Edin. Med. and Surg. Journal, April 1839; or Reid's Anatomical and Physiological Commentaries. Monthly Retrospect, 1849, p. 3. Longet—Systeme Nerveux.

acting on the pneumogastric nerve, either directly or through the nervous centres. And *Fifthly*, that it may be stimulated by remedies or other agents acting in a similar manner.

The application of these principles to pathology might be almost indefinitely expanded, if it were desirable at the present stage of the inquiry to indulge in much farther speculation. But enough has probably been brought before the reader to show that the symptoms, causes, and cure of bronchitis and other allied affections, even when not resulting in demonstrable or organic disease, are illustrated by a clear conception of the phenomena of pulmonary collapse and its attendant conditions.

I now proceed to the consideration of some permanent disorganisations, for the most part well known to anatomists.

Permanent Lesions of the Air-Vesicles and Bronchi depending on Bronchitis.

Relation of Bronchitic Collapse to Pulmonary Emphysema.—I have discussed above the mode in which the collapsed lung, under favourable circumstances, reverts to its natural condition. The mechanism by which this is effected, and particularly that portion of it which I have theoretically suggested under the name of the deobstruent function of the bronchial tubes, must be considered as of vast physiological interest, if we reflect that there is scarcely a case of fever, or any other debilitating disease, in which the signs of mucous accumulation and of partial pulmonary collapse may not be discovered at one period or other at the lower and back part of the lungs; and that the same forces which under these circumstances restore the lung to its normal state, by throwing off the load in the bronchi, are probably perpetually in action to prevent a similar accumulation in the state of health. A little careful reflection on the mode in which the free and unembarrassed play of the lungs is maintained in health, notwithstanding the constant presence of a viscid secretion from the bronchial mucous membrane, will probably satisfy every one that a special function for the removal of this secretion must form an element of the highest importance in normal as well as morbid respiration; and the ciliary apparatus, as I have already mentioned, is not calculated fully to perform this office, being less abundantly distributed to the smaller bronchial tubes, where its presence is apparently most required.

In many persons the removal of the bronchial mucus is habitually ill-performed. The quantity of mucus is not materially greater than natural, but it is not discharged as rapidly as it is secreted, owing to some defective condition of the deobstruent apparatus, or perhaps

a defective innervation of the bronchial muscles. In this condition, whatever be its cause, the tranquil and insensible processes of the normal economy are exchanged, sometimes for fits of coughing, which bring up pellets of tenacious pearly mucus, the result of undue accumulation; and sometimes for paroxysms of dyspnœa, which end in more copious evacuations of bronchial mucus, and a return to comparative health. These persons are the subjects of the *dry* or *humid asthma* of English authors, and of the *catarrh sec* of Laennec. Their disease, though simple and free from danger in its outset, is, according to the judgment of all physicians, apt to lay the foundation of organic disorder, which usually assumes the form of pulmonary emphysema. This consequence is peculiarly apt to occur, if care be not taken to guard against the supervention of bronchitis, which in these individuals generally assumes characters of great intensity, and is uncertain and protracted in its cure.

There are other persons who, with a habitually normal state of the respiratory functions, are subject to repeated acute bronchitic attacks of great severity. They are careless in their mode of life, frequently exposed to cold, or endowed with a peculiar sensitiveness of the pulmonary mucous membrane, while they are at the same time free from the tuberculous constitution, with its organic sequelæ. In such persons, also, pulmonary emphysema is known to be a frequent disease; being left behind as the legacy of the bronchitic attacks, and aggravated after each successive invasion.

Not unfrequently a considerable, or even an extreme, amount of pulmonary emphysema is observed to follow a single attack of acute disease in the chest. Thus, emphysema frequently arises in the earliest years of infancy and childhood, as the consequence of some form of severe infantile bronchitis; and all practitioners can bear witness to many cases in which shortness of breath and incapacity for exertion can be traced distinctly back to the date of an attack of hooping-cough or measles. Some of the most marked instances of emphysematous lungs in young subjects that have fallen under my notice in dissection, have had a similar history; and all authors on the diseases of children, who have carefully investigated the morbid anatomy and history of these affections, concur on this point. Again, in adults otherwise healthy, the severer forms of epidemic influenza are peculiarly apt to be attended with, or followed by, the development of emphysematous lesions; a fact which has been well observed and carefully recorded by Dr Peacock, in his excellent history of the last London epidemic of that disease.[1]

[1] The Influenza or Epidemic Catarrhal Fever of 1847-8. By Thomas Bevill Peacock, M.D., &c.: London, 1848. See pp. 31-32, 134-135, 143-144, for graphic descriptions of the morbid appearances after death from influenza. Dr Peacock has favoured me with a letter on this subject since the publication of the first part of this memoir, and I am happy to be able to state, that this experienced pathologist—my predecessor in my present office—is convinced of the correctness of my explanations of the bronchitic collapse of the lung, and satisfied of the identity of that affection with many of those indicated by him in the pages to which I have referred.

Finally, a certain amount of emphysema of the lungs is of so frequent occurrence in the aged, as to be scarcely entitled to the name of a disease, distinct from the other evidences of corporeal decay. This fact was first pointed out by Magendie, and the form of emphysema here alluded to has since been described by many pathologists as a peculiar one, constituting a kind of senile atrophy of the pulmonary tissue. But there can be little doubt, that here also the pulmonary lesion is the concomitant of a bronchial affection,—the chronic bronchitis or bronchorrhœa,—which is almost constantly the companion of the more advanced periods of human life. In cases where this has been absent, I have repeatedly found the lungs of very aged individuals quite free from all trace of emphysematous lesion.

Considerations like these have, ever since the accurate descriptions of emphysema by Laennec became generally known, given rise to a general belief among practitioners that emphysema is related to bronchitis as effect to cause; and that it is indeed the organic lesion of the lung of all others most closely and invariably connected with long-continued or severe bronchial affections. In taking it, therefore, as the starting-point of the following researches on the permanent lesions of the lungs connected with bronchitis, I shall have the advantage, not only of beginning with a disorganisation so palpable and well-known as to be rarely overlooked at the present day by any one acquainted with pulmonary pathology, but one, the relation of which in some way or other to bronchitis, is almost universally admitted, notwithstanding the numerous differences of opinion as to its mechanism and causation. The observation of it somewhat more rigorously will serve, therefore, as a criterion of the correctness of the observations in the first part of this memoir, and at the same time will lead naturally to the consideration of other subjects.

TABLE OF FORTY CASES OF PULMONARY EMPHYSEMA, SHOWING ITS CONNECTION WITH OTHER AFFECTIONS, ESPECIALLY OF THE LUNGS.

Vol. and No. in Path. Reg.	Age.	Sex.	Amount, Position, and Extent of Emphysema.	Collateral Affections of Lungs.	Collateral Affections of other Organs.	Duration and Character of Symptoms.
XII. 10	37	F.	In L. L. sub-pleural bullæ at apex, &c. In R. L. irregular emph. of inferior borders.	Condensation, non-granular at posterior portion of R.L. Lobular condensation. Cretaceous tubercle in apices.	None mentioned.	Cholera. No detailed history of symptoms. Died in reaction, with considerable dyspnœa.
XII. 25	31	F.	"Anterior part of both L. highly emphysematous."	"Posterior lobes much congested. Lower and back part of R. L. *somewhat hepatised* (?) and studded sparingly with miliary tubercles." Muco-purulent matter in bronchi.	None mentioned. "The abdominal organs healthy."	"Subject to want of breath from childhood." Dyspnœa, cough, lividity. Thick white sputa.
XII. 32	39	M.	The sub-pleural vesicles mostly solitary,—not larger than a barley-corn.	None mentioned. Upper lobe of R. L. compressed by a neuromatous tumour.	Neuroma of vagus nerve. Necrosis of scapula.	Frequent difficulty of respiration. Great exhaustion.
XII. 36	49	M.	Position and extent not mentioned. (The chest arched in front.)	L. L.—a cavity at apex, with irregular condensation. R. L.—miliary tubercles with condensation, as in L. L.	Cysts in kidneys.	Fever succeeded by bronchitis. No old history.
XII. 47	69	M.	Anterior edges of both lungs.	Chronic gray induration of upper and posterior parts of both L., with cavities and cicatrices of pleura (non-tubercular).	Hypertrophy of heart.	Six months' (?) cough, dyspnœa, &c.
XII. 67	22	M.	"Ant. edges very emphysematous." (Chest arched in front.)	Adhesions. L. L. hepatised in lower part of upper lobe. R. L., scattered lobular condensation in posterior part. Bronchi containing much mucus.	None mentioned.	Acute attack; 8 days. An *equestrian* in Batty's circus—intemperate, and exposed to cold.

XII 108	19	M.	"Exceedingly emphysematous at ant. edges."	Sulci between emphysematous portions. Gray condensation. Tubercle, with small cavities.	Extreme emaciation. Hypertrophy of liver and spleen. Tubercle of intestines.	Chronic phthisis; 4 years.
XII. 169	17	M.	Anterior portions. (Sternum arched.)	Tubercular condensation with cavities, scattered through both lungs, except a small part of anterior edges. Adhesions.	Tubercle of mesenteric glands, and slight ulceration of intestines.	No history. Symptoms acute.
XII. 220	—	M.	Whole anterior part of both lungs.	Some recent tubercle. Chronic and irregular condensation. Old puckering in apex of L.	Aneurism of aorta opening into œsophagus.	No information.
XII. 222	—	—	"Highly emphysematous at some points."	"Lobular condensation, alternating with crepitating and emphysematous lung. Outline very irregular, from sinking of the condensed, and prominence of emphysematous parts."	Softening of cerebellum.	No information.
XII. 272	—	—	Emphysematous at anterior edges; presenting irregularities of surface.	Lobular condensation "frequently in the midst of the emphysematous portions." Bronchial abscesses.	None mentioned.	Acute affection, succeeding measles. Fatal in 3 weeks.
XII. 274	38	F.	"Considerable emphysema of both lungs anteriorly."	Calcareous deposits scattered through both L., especially at apex. No condensation or tubercle. Adhesions.	Calculus in left kidney.	No information.
XII. 275	38	M.	"Both lungs very emphysematous anteriorly." (Chest considerably arched in front.)	"Slight condensation of both L. posteriorly." "R. L., a portion completely condensed. Bronchi full of muco-pus."	Hypertrophy of heart.	No information.
XII. 288	50	M.	"Air-vesicles much enlarged beneath the pleura at whole anterior part."	"L. posteriorly condensed, and scarcely crepitating." In the midst of the crepitating and emphysematous portions small lobular condensations. Adhesions.	Hypertrophy of heart.	Repeated attacks of chest symptoms for many years.
XII. 317	45	M.	"Ant. and lower edges excessively emphysematous."	Lobular condensation. Bronchi clogged with thick mucus. Soft lymph on pleura.	None of importance.	No information.

TABLE OF FORTY CASES OF PULMONARY EMPHYSEMA, SHOWING ITS CONNECTION WITH OTHER AFFECTIONS, ESPECIALLY OF THE LUNGS.—(*Continued.*)

Vol. and No. in Path. Reg.	Age	Sex.	Amount, Position, and Extent of Emphysema.	Collateral Affections of Lungs.	Collateral Affections of other Organs.	Duration and Character of Symptoms.
XII. 323	60	F.	"Both lungs emphysematous anteriorly."	Bronchi contain much mucus. "Right side yields copious frothy serum. No condensation."	Hypertrophy of heart (right auricle).	No information.
XII. 326	—	—	Both lungs very emphysematous.	Irregular lobular condensations. Bronchi contained much mucus. Bronchial abscesses. Adhesion at apex of R. L.	Hypertrophy of heart.	No information.
XII. 327	35	M.	"L. emphys., particularly the anterior borders."	Lobular condensations; bronchial abscesses; cretaceous matter in bronchial glands.	Granular kidney. Necrosis of thigh.	No information.
XII. 345	45	M.	Both L. remarkably emphysematous. (Sternum very arched; sides of chest flattened.)	Hepatisation and collapse. Cicatrices at apex of R. L. Bronchi congested and thickened. Adhesions.		Cough *constant* for a year. Haemoptysis. Emaciation.
XII. 349	36	M.	L. L. generally emphysematous.	L. L. containing miliary tubercle. R. L. contracted. Empyema, with contraction of right side of chest.	None mentioned.	3 months. Acute attack. Signs of empyema.
XII. 350	30	F.	Anterior edges partially emphysematous.	Between emphysematous portions, collapsed lobules; and a considerable amount of condensation post. Bronch. abscesses.	Ulceration of colon.	No information.
XII. 352	17	M.	R. L.,—intense emphysema of upper lobe; marked also in lower. L. L.,—much less emphysema, but in same situations as in R. L.	R. L.,—deep furrows between emph. portions, corresponding to dark atrophied parts. L. L.,—upper lobe much atrophied, and containing calcareous matter. Adhesions.	Hypertrophy of heart. Firm indurated deposits in spleen.	No information.

XII. 355	—	—	Great dilatation of air-cells (to size of a bean) in the anterior prolongation of upper lobe of right lung.	Collapse of upper lobe of R. L., with bronchial abscesses. Diffused collapse posteriorly, and lobular anteriorly, along with emphysema. Adhesions.	Slight hypertrophy of right side of heart. Stricture of urethra, &c.	No information.
XII. 360	7	M.	Interlobular emphysema confined to anterior prolongation of upper lobe of left lung.	R. L. healthy, except imperfect and partial collapse. L. L. contains a mass of tubercle in lower lobe, obstructing some of the bronchi. At the anterior edge, opposite this mass, lower lobe completely collapsed.	Tubercular lymph at base of brain.	Sub-acute hydrocephalus.
XII. 364	30	F.	Both lungs very emphysematous at anterior edges. Some air-cells dilated to size of walnuts.	Posterior portions of lungs completely collapsed. Emphysematous prolongation of upper lobe of L. L., connected with the rest by partially collapsed tissue.	Cancer of uterus, ovaries, and peritoneum.	No information.
XII. 390	16	M.	Both L. highly emphysematous at anter. borders, especially at the anterior part of lower lobes.	Condensation of posterior portions. Adhesions.	Contraction of mitral orifice. Slight hypertrophy of right side of heart, with granular degeneration of fibres.	No information.
XII. 400	50	F.	Lungs very emphysematous anteriorly.	Atrophied portions mingled with the emphysematous. Posterior parts but slightly crepitant.	Contraction of mitral orifice. Hypertrophy of heart. Disease of kidneys.	No information.
XIII. 1	40	M.	Anter. portions in both; in left, all the lobes anteriorly, and the lower and outer border of lower lobe.	Collapsed, intermixed with emphysematous portions. Firm concrete pus in some parts, forming small roundish masses of nearly cartilaginous consistence. Partial hepatisation. Adhesions.	Purpura spots on surface. The intestines healthy.	No information.

TABLE OF FORTY CASES OF PULMONARY EMPHYSEMA, SHOWING ITS CONNECTION WITH OTHER AFFECTIONS, ESPECIALLY OF THE LUNGS.—(*Continued.*)

Vol. and No. in Path. Reg.	Age.	Sex.	Amount, Position, and Extent of Emphysema.	Collateral Affections of Lungs.	Collateral Affections of other Organs.	Duration and Character of Symptoms.
XIII. 9	—	F.	Highly emphysematous in anterior half.	Lobular collapse at edges, alternated with emphysematous portions. Posterior part of left lung crepitated imperfectly. Bronchi contained much muco-purulent matter. Adhesions.	Tubercle in bronchial glands and kidney.	No information.
XIII. 10	—	M.	In both lungs, but greatest in left; in upper part, anterior and lower borders, and at the diaphragmatic surface.	Emphysematous parts had furrows corresponding to parts affected with lobular collapse. A part of the lower margin of R. L. crepitated very sparingly. Bronchi filled with pus, and dilated.	Bronchial glands enlarged and dark. An old sinus opening into right bronchus at the root of the lung. Old miliary tubercle of kidney.	No information.
XIII. 18	40	M.	"Highly emphysematous in their anterior half."	Lobular collapse and atrophy at edges. Posterior half of both L. slightly collapsed, but still crepitating. Bronchi contained much muco-purulent matter.	Commencing cirrhosis of liver. Very slight and doubtful hypertrophy of right ventricle of heart.	Bronchitis. Duration not stated.
XIII. 27	45	M.	Both lungs very emphysematous anteriorly.	Posteriorly, imperfect collapse, with œdema. Small points of extravasation.	Hypertrophy of heart (20 ozs., chiefly left). Bright's granulations of kidneys.	No information (Report of examination imperfect in many points.)
XIII. 30	45	M.	Lungs emphysematous over anterior third.	Atrophied portions in emphysematous edges. Collapse of posterior third. Mucus in bronchi.	Clot in left hemisphere of brain.	No information.

XIII. 34	40	F.	Lungs highly emphysematous over ant. third, and in lower border. Air-vesicles dilated to size of mustard seed.	Completely atrophic portions in midst of emphysematous. Mingled collapse and atrophy in post. two-thirds. Hard encysted nodules scattered through both L., containing much carbonaceous pigment.	None mentioned.	No information.
XIII. 49	68	M.	Both lungs extremely emphysematous anter. In upper lobes, bullæ size of a hazel-nut.	Diffused and limited condensation, collapse, atrophy, induration, with encysted concretions in the midst of emphysematous parts. Bronchi loaded with muco-purulent matter.	Superficial fibrous degeneration of brain, &c.	No information.
XIII. 74	40	M.	Lungs emphysematous anteriorly.	Emphysematous edges present scattered collapsed lobules. Posterior—incomplete collapse of right, and hepatisation of left lung. Lymph on pleura.	Hemorrhagic ulcers of stomach, &c.	No information.
XIII. 81	22	M.	Lungs at anterior part pretty generally emphysematous.	Considerable collapse at posterior part, and scattered in midst of emphysematous portions. Tubercles—miliary, yellow, softened; small cavities, with puckering of pulmonary tissue (atrophy). Encysted masses, scarcely cretaceous.	Ulcers of intestines. Bright's disease of kidneys.	Symptoms acute; lasted 10 days.
XIII. 84	30	F.	Extensive emphysema at ant. edges. In left L. several pedunculated lobules, and greater dilatation than in right.	Collapse, diffused and scattered among emphysematous parts. Hemorrhage into bronchi.	Hypertrophy of right side of heart.	Pain in chest, cough, and expectoration, for 6 months.
XIII. 86	54	M.	Extensive and extreme emphysema; at upper part of left lung, one bulla, size of a pigeon's egg.	Much atrophy, with induration at apices, and scattered through emphysematous parts. Some concretions, cicatrices, a single small cavity (bronchial abscess) in left lung. Chest almost cubical in form.	Bright's granulations of kidney, &c. &c.	Symptoms of asthma of many years' standing.
XIII. 88	25	F.	Extensive and tolerably uniformly diffused emphysema of ant. edges. Nowhere great dilatation of air-vesicles.	Traces of atrophied lobules at anterior edges. Diminished crepitation of lungs behind.	Other organs not examined.	No history beyond the ultimate attack.

Analysis of Cases of Pulmonary Emphysema.—The preceding six pages contain a table of forty cases of emphysema of the lungs, the object of which is chiefly to show the connection of that lesion with other collateral affections of the pulmonary tissue. The state of the bronchi is not always noted, nor indeed can it be fairly assumed that, in a chronic lesion such as emphysema, the condition of the bronchial mucous membrane at the period of death has any direct relation to it in the majority of cases. The statements in the table accordingly show, not the evanescent and inappreciable conditions of the bronchial membrane, but the more permanent and evident affections of the air-vesicles and pulmonary tissue. The other columns are added for the satisfaction of those who may be studying the same subject under different aspects, and references are given in all the cases to the Registers of Dissections in the Royal Infirmary, where more detailed reports of them may be found. It is right also to state, that no cases of conside rable emphysema have been excluded from the table, excepting a few, in which the report was, from one cause or other, considered to be inadequate or untrustworthy.

The most cursory inspection of this table will show that pulmonary emphysema is in by far the greater number of instances accompanied by other lesions of the air-vesicles and pulmonary tissue; and that, in fact, its occurrence as an isolated affection of the lung, is not only uncommon but doubtful. In every instance it was found connected with some mode or form of condensation of the pulmonary tissue, except in the two cases marked XII. 32, and XII. 323. In one of these the report was not drawn up by me; and in neither of them do I now feel certain of its accuracy in this respect, as some of the lesions which I shall have to describe in the sequel as concurring with emphysema are easily overlooked, and have, in fact, been constantly overlooked by Laennec and other writers on this subject.[1]

The appearances in the other cases may be arranged as follows:—

Hepatization in four cases, or 10 *per cent.*, viz., XII. 67, 345; XIII. 1, 74.

Tubercle (or tubercle with condensation) in eight cases, or 20 *per cent.*, viz., *tubercle without excavation*, XII. 25, 220, 349, 360; *with excavation*, XII. 36, 108, 169; XIII. 81.

Condensation (presumably *bronchitic collapse*, and often described as such) in 27 cases, or 67·5 *per cent.*, viz., XII. 10, 25 ?, 67, 222, 272, 275, 288, 317, 326, 327, 345, 350, 355, 360, 364, 390; XIII. 1, 9, 10, 18, 27, 30, 34, 49, 74, 81, 84.

[1] In Louis's essay on Emphysema, in the Mem. de la Societé d'Observation, he describes at considerable length numerous cases of this lesion affecting the *entire* lung, without any concurrent affection,—a condition which, I do not hesitate to say, is not found in nature.

Bronchial abscesses, or non-tubercular ulcerations, in seven cases, or 17·5 *per cent.*, viz., XII. 47, 272, 326, 327, 350, 355; XIII. 86.

Chronic induration or atrophy, in ten cases, or 25 *per cent.*, viz., XII. 47, 108, 345, 352, 400; XIII. 30, 34, 49, 86, 88. Besides some of the tubercular cases, and the following:—

Contraction of opposite lung (absorbed pleuritic effusion) in one case, XII. 349.

Concretions in eight cases, or 20 *per cent.*, viz., XII. 10, 274, 352; XIII. 1, 34, 49, 81, 86.

Even a superficial inspection of this catalogue of morbid appearances will serve to corroborate many of the views previously expressed, and will conduct us to conclusions simplifying very much the whole subject. We find, in the first place, that 67·5 per cent. of the whole cases of emphysema were connected, at the time of death, with those forms of pulmonary condensation which have been ascertained in the preceding pages to be most frequently the result of bronchitis, and which, indeed, concur with it in the great majority of cases. The per-centage of bronchitic lesions, however, as we shall see in the sequel, will be increased from those found under other heads. Contrast with this the proportion of cases referred to hepatization or tubercle, two of the commonest of pulmonary lesions, the former of which is, nevertheless, found only in 10, the latter only in 20, per cent. of the emphysematous cases; and it will be at once evident, to what a large extent the bronchitic lesions predominate over all others. Nor is this apparent predominance merely the result of their greater absolute frequency; for I find, by my manuscript returns of the results of 502 post-mortem examinations of all kinds of disease performed in the hospital, during the periods referred to in the table, that, among these mixed cases, hepatization occurred 48 times, or 9·8 per cent., and tubercle of the lung 100 times, or 20 per cent.; while the other forms of condensation alluded to occupy a medium position in frequency, viz., 59 times, or 11·8 per cent., being only a little more frequent than hepatization, and very considerably less so than tubercle.[1]

[1] As all the circumstances tending to affect the accuracy of these returns ought to be mentioned, so far as known to me, I may here indicate that I believe the frequency of these bronchitic lesions to be understated in both classes of cases, from the slighter forms being not always recognised or recorded, especially before I became quite familiar with their character and significance. These omissions would affect both classes of cases quite equally, and therefore be of little moment, were it not that the distinction between hepatization and collapse of the lung is not always sharply drawn in the earlier cases; and I think it possible that, especially in the larger series of returns, a few cases of the latter may have found their way under the head of the former. Even if we suppose, however, the per-centage of hepatization *slightly diminished*, and that of collapse of the lung *slightly increased*, in the series of mixed cases, the difference between the two affections in regard to the production of emphysema, will remain too broad and well-marked to be explained by any accident.

I am aware it will be said, that tubercle stands in a wholly peculiar relation to this subject, as several pathologists of great eminence have maintained the doctrine of the incompatibility of emphysema and tubercle; supposing, on the ground of their comparatively rare co-existence, that the former confers on those attacked an immunity from the latter affection. Rokitansky has, indeed, given the sanction of his high authority and immense experience to a doctrine which may be considered inclusive of this assertion of the French pathologists; viz., that all affections producing *venosity*, or imperfect oxygenation of the blood, such as cyanosis, curvature of the dorsal spine, emphysema, &c., confer an immunity from tubercle. Without entering here on the discussion of this doctrine, in its more general relations, it may be confidently stated, that the portion of it relating to emphysema gains no support from the numbers just quoted. According to this doctrine, it might reasonably be expected, that among persons dying with emphysematous lungs, tubercle would bear a decidedly lower proportion to the whole numbers than in a mixed hospital mortality; whereas, by a remarkable enough accident, it happens that the proportion is, in the above numbers, precisely the same, viz., 20 per cent. in both classes of cases.[1] And although it would be too much to argue from this coincidence, that emphysema and tubercle exert no influence upon each other, yet I think it may justly make us pause before accepting a doctrine which has not, *a priori*, much argument in its favour, and the evidence of which has never been presented to the public under a form approaching to exactness. I shall have occasion hereafter to state my own views on this subject.

The following table exhibits, in one view, the per-centage of most of the lesions referred to above, in emphysematous and in mixed cases of disease,—the numbers from which it is calculated being derived from the same hospital returns, so as to assimilate the conditions of observation as nearly as possible.

	In mixed Cases.		In Emphysematous Cases.	
Hepatization,	9·8 per cent.		10·0 per cent.	
Tubercle,	20·0	—	20·0	—
Condensation (collapse),	11·8	—	67·6	—
Bronchial abscesses,	5·5	—	17·5	—
Induration and atrophy,	7·5	—	25·0	—
Concretions,	4·1	—	20·0	—

It will be seen that while the first two lesions in the preceding table appear to have no special numerical relation whatever to

[1] Rokitansky admits the conjunction of obsolete or cretaceous tubercle with emphysema. But, in the cases above referred to, all the instances of obsolete tubercle have been excluded from both lists. In the cases conjoined with emphysema, it will be seen that there existed cavities in four cases; the others were miliary or yellow tubercle without excavation.

emphysema, their per-centage being nearly the same in this affection as in the general returns, the remaining four are found to be greatly more frequent in connection with emphysema than under other circumstances. But this is not all; for, as tubercle is almost invariably connected with some form of condensation, and was so connected in many of the cases here referred to, and as all the cases of hepatization are also to be found under the head of bronchitic condensation, it becomes nearly certain that, of the whole forty cases of emphysema, not one had any direct connection with either hepatization or tubercle, as such, but only through the medium of the other lesions mentioned. Tubercle and hepatization, therefore, are in all probability merely the accidents, and not either the causes or effects, of emphysema of the lungs.

If now we consider the all but invariable connection of emphysema with one or other of the remaining lesions of the lungs, and the frequency with which all of them occur in emphysematous as compared with mixed cases, we shall be driven almost inevitably to the conclusion, that some circumstance, common to them all, and not necessarily present in hepatization and tubercle, is closely connected with the production of emphysema, if not, indeed, its real pathological cause. What that circumstance is, we may now endeavour to discover.

Mechanism of Emphysema.—Emphysema of the lungs was said by Laennec, in one of the most original and accurate of his descriptions, to have two varieties: the one being a dilatation of the air-cells, and finally a rupture of them one into another by removal of their septa; the other, a rupture of the air-passages directly into the interlobular areolar tissue. It is needless to repeat these descriptions, the distinction of vesicular and interlobular emphysema being well known to every one, or at least accessible to all, in words which cannot be improved. It is only necessary to add, that the microscope and other modern means of investigation, which have done so much for morbid anatomy, have scarcely availed here to augment our knowledge; having only succeeded in demonstrating more clearly the fact, known to Laennec, of the gradual breaking up of the vesicular septa, and the obliteration of their capillary network.[1]

Emphysema, therefore, is an abnormal distension of the pulmonary tissue with air. In its earliest stages, whether interlobular or vesicular, or, as frequently happens, both combined, nothing can be more certain than that it is essentially a mechanical lesion: in fact, the distension of the air-cells, giving the peculiar cushion-like and

[1] After frequent personal observation on this subject, I am compelled to regard the late theory of Mr Rainey, in regard to the dependence of emphysema on fatty degeneration of the lung, as fallacious. The granules described by him certainly do not always occur in emphysematous parts; and when they do so, they are so few, and so little characteristic of this particular lesion, that it is plain Mr Rainey's views have been founded on an imperfect appreciation of the relations of the so-called "fatty granules" to morbid tissues.

pale appearance to the lung, can be exactly imitated by inflating it with undue force artificially. Moreover, the whole of the subsequent structural changes implied in the gradual removal of the septa and obliteration of the capillaries, are readily explained by the mechanical effects of distension. Upon this subject M. Poiseuille, to whom we owe so many interesting facts in mechanical physiology, has a very beautiful experiment.

An instrument being adapted to the pulmonary artery of an animal, by which a given quantity of liquid was propelled with a given force through the capillaries of the lung, he found that this was effected, in the normal condition, in 29 seconds. M. Poiseuille now inflated the lungs so as exactly to fill the cavity of the chest; the time was still 29 seconds. On distending the lungs, however, farther, so as to produce the appearance of a partial emphysema, the time required for the passage of the fluid became lengthened to 62 seconds; when the emphysematous appearance was increased, 95 seconds; when it pervaded the whole lung in consequence of excessive distension, 129 seconds were required, and the fluid returned from the pulmonary veins mixed with some bubbles of air.[1] From these results, it is evident that whenever the air-cells are distended beyond the amount required or possible in the healthy condition, the flow of blood through the ultimate capillaries of the lung must be retarded or obstructed;—a condition not only corresponding with the appearances observed in emphysema, but readily accounting for the structural changes, the absorption of the walls of the air-cells, and the tension and obliteration of vessels observed in the latter stages of the disease.

It is, therefore, nearly certain that the source of emphysema is to be sought in a derangement of the mechanism of respiration, and not in any previously morbid condition of the affected part. Every thing denotes that the emphysematous parts of a lung are usually free from all diseased changes, with the exception of those which are the result of inordinate distension. The freedom from œdema and from morbid deposits, when other parts of the lung are so affected; the absence of accumulation in the bronchi, or at least its comparatively slight character, allowing of the perfect and easy inflation of the emphysematous parts when others are collapsed; finally, the habitual seat of emphysema in those parts of the lung which are usually most exempt from other disease,—all tend to prove what I have now stated. The diminished elasticity, the dryness, the anæmia, which have all of them been supposed to be the predisposing cause of this lesion, are manifestly nothing more than the effects of the distension with air upon the circulation and nutrition of the compressed walls of the delicate pulmonary air-cells. Even the

[1] Bulletin de l'Academie Royale de Médecine, vol. viii., p. 705.

small accumulations of granular deposit found by Mr Rainey may be accounted for by these secondary nutritive changes.

But emphysema is not merely a lesion resulting from inordinate distension of previously sound portions of lung; it is, as we have already seen, the product of mechanical derangement in the *sound parts* of lungs *otherwise diseased*. The existence of bronchitic condensation, of induration, of concretions, &c., if not a necessary cause of the production of emphysema in the sound air-vesicles, is at least in some way related to it. The theory of emphysema by Laennec, besides the objections offered to it in the former part of this memoir, in no way accords with the facts now adduced. Mucous obstruction of the bronchi, even if proved to exist, cannot determine, *directly*, both condensation and rarefaction of the lung; and we have already learned, from unquestionable and multiplied evidence, which of these two is its real result. The opinion of Louis, derived, apparently, chiefly from a consideration of the seat of election of emphysema as compared with that of bronchitis,[1] is opposed to the idea of any precise relation between these two affections; but this negative opinion would appear to be sufficiently answered by the numerical facts above adduced.

Some writers, conceiving, like Laennec, that emphysema is produced in the act of expiration, believe it to be the result of violent efforts of coughing, or other forcible expiratory acts. But have we really any direct proof whatever that cough, however violent, or any similar act, can produce emphysema, apart from the other accidents of bronchitis? In croup, in laryngitis, in aneurism of the aorta, we have cough even more violent and distressing than that of bronchitis; yet these affections are not known usually to cause emphysema, and I have repeatedly seen cases opposed to the idea of their having any such influence. The alleged unusual frequency of emphysema among players of wind-instruments is likewise totally devoid of proof, and rests upon one unsupported assertion of Laennec; whereas, if the real cause of emphysema were such as above described, no singer or wind-instrument player could in all probability remain long exempt from this disease. But it would require further to be known whether an increased liability to emphysema in this class is not accompanied by a similar proclivity to other pulmonary affections, before the question could be decided on such grounds.

But the most serious objection to the expiration-theory of this disease is, that the expiratory act is *mechanically* incapable of producing distension of the lung, or of any part of it. The act of expiration tends entirely towards emptying the air-vesicles by the

[1] "Si l'on se rappelle que le *maximum* de l'emphysème ordinairement a son siège au bord tranchant des poumons et dans leur voisinage, tandis que le catarrhe pulmonaire aigu intense a le sien en arrière et en bas, on sera forcé de conclure que si ce catarrhe a une influence quelconque sur la développement de l'emphysème, cet influence est peu considerable et ne s'exerce sans doute que bien rarement."—*Memoires de la Societé Médicale d'Observation*, tome premier, p. 253.

uniform pressure of the external parietes of the thorax upon the whole pulmonary surface; and even when the air-vesicles are maintained at their maximum or normal state of fulness by a closed glottis, any further distension of them by the expiratory force is as much out of the question as would be the further distension of a bladder blown up and tied at the neck, by hydrostatic or equalised pressure applied to its entire external surface. The air-vesicles can sustain no distending pressure from the column of air *within* the tubes, as that air only becomes compressed in virtue of a force acting on the *exterior* of the lung, which opposes exactly as much resistance without as it creates pressure within. It is singular that a theory so radically unsound, and so devoid of direct proof, as this of the production of emphysema by expiration, should have been allowed to maintain a place in medical literature.

The only theories of emphysema which remain, are those which refer it to the act of inspiration. The most usual form assumed by these theories, is the supposition that emphysema of the lung is a physiological *compensation* for the occlusion of a diseased portion of lung;—a view not only giving no real explanation, but totally inconsistent with the fact, that in truly morbid emphysema there is always a diminished respiratory surface and consequent dyspnœa. Dr Williams, however, and some others, have placed the inspiration-theory in a more tenable position,—supposing that, when certain portions of the lung are occluded, the air is brought by inspiration to penetrate with greater force, and in greater volume, into the remaining parts.

This view is certainly near the truth, and is quite consistent with clinical stethoscopic experience. But it is clogged in Dr Williams' work with a reference to the incompetent expiration-theory of Laennec, as if the author did not see his way clearly to the explanation of all cases of emphysema by his own. Moreover, it is not the whole truth; because certain obstructive lesions have, as we have seen, no appreciable influence in causing emphysema; and also because it is evident that the inspiratory or expansive power of the chest is exactly limited by its capacity, and that even when a portion of lung is impervious to air, as in hepatization, the inspiratory force can no more distend the sound air-cells to the degree observed in emphysema than it can do so in the normal state. This fact will appear more clear from the following observations.

It appears to me that none of the writers on this subject have clearly apprehended, or at least clearly expressed, the single obvious condition which is necessary to the mechanical completeness of the inspiration-theory of emphysema. Emphysema is, according to this theory, a *complementary* lesion, dependent upon the previous existence of some form of occlusion of the vesicles, and invading the remaining sound portions of lung. Thus far it corresponds with all that we have hitherto seen, to an extent certainly not anticipated by Dr Williams, when, after enunciating his own view, he brings

forward Laennec's theory to account for residual unexplained cases. But there is yet another condition necessary, besides mere occlusion of the air-vesicles in a part of the lung: this is *partially diminished bulk*;—in other words, collapse or permanent atrophy of a portion of the lung.

The operation and importance of this condition will be at once seen by the aid of a diagram. Suppose that in the accompanying fig. 1 the three equal partitions represent the *maximum* air-space, in

Fig. 1.

Fig. 2.

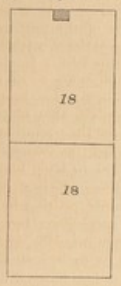


Fig. 3.

the normal condition of full inspiration of three lobes or portions of a lung (represented equal for the sake of simplicity). Each lobe holds, on a full inspiration, say 12 cubic inches or other measures of air; and it is adapted normally to hold this quantity, without pressure on the capillary circulation, or risk of violence to the texture of the organ. It is at once obvious that no amount of lesion, which leaves the upper partition or lobe of its normal volume, can at all affect the maximum expansion of the other two. They will continue, under all circumstances, to be capable of receiving their normal 12 measures of air; they will be prevented from receiving more, not by the tendency of the pulmonary texture to *resist* further expansion, but by the inadequacy of the mechanical apparatus *for producing* further expansion. No strain can in this case be thrown upon the walls of the air-cells; these still preserve their normal relation to the capacity of the chest which contains, and, by its dilatation, expands them. The inspirations will indeed be multiplied,—they will also be increased in fulness and force beyond the ordinary condition; but this can have no more effect in producing emphysema in the free air-cells of a diseased lung (under the above conditions) than running or violent exercise can have in relation to a healthy well-organised chest. This is the state which occurs in pneumonia, tubercle, and

all other lesions primarily affecting the air-cells themselves; it is represented in fig. 4, where the upper partition is supposed to be blocked up, at its full volume, with some abnormal deposit occupying the air-spaces. It may be granted that this diagram differs from what occurs in nature thus far, that even in the purest instances of hepatization the volume of the lung is seldom fully maintained; but it will be found, that exactly in proportion as it is so, the liability to emphysematous lesion is less.

Fig. 4.

But now suppose the occurrence of a lesion, in which the air-spaces of one of the partitions are closed by the collapse of its parietes, with diminution of bulk of the lung in this lobe. In this case, it is obvious that the expanding forces of inspiration will act inordinately upon the remaining lobes, and tend to attract into them the air which is prevented from entering the occluded one. If these forces were sufficiently powerful to overcome the resistance offered by the tissue of the sound lung under these circumstances, and if the sound portions of lung yielded equally in all directions, it is obvious that the condition established would be that in fig. 2, in which the lung is expanded to the normal maximum; but the air is differently distributed, being excluded from one lobe, and present in the others to the extent of 18 measures in each, instead of 12 as formerly. In like manner, the occlusion of two lobes, if accompanied with collapse of the tissue, would necessarily lead, in the event of the lung being fully dilated, to the accumulation of the whole 36 measures of air in the remaining lobe, as in fig. 3. A lobe thus distended would certainly suffer obstruction of the capillary circulation, as in the experiment before mentioned of M. Poiseuille; and the original purely mechanical condition would pass into one complicated by those structural changes which are actually produced in chronic emphysema.

It may be well to explain here, that a certain amount of overdistension, *when gradually effected*, is sometimes borne by the lung without the supervention of a distinctly morbid condition. The lung, under those circumstances, probably undergoes a genuine hypertrophy, the air-vesicles becoming slightly enlarged, but with a nutritive adaptation of the vascular and other structures to the changes thus effected. This enlargement of the lung, without the pathological characters of emphysema, is sometimes observed in disease, when the whole of one side of the chest has been contracted from pleurisy, the opposite lung passing, as the stethoscopist well knows, for an inch or two across the median plane in front, and having all its parts seemingly adapted to its increased size and function. A large power of adaptation of the lung to external circumstances is also shown (as has been pointed out by an acute

critic of my original communication on this subject to the Medico-Chirurgical Society of Edinburgh) "among the inhabitants of such lofty situations as the high table-land of South America," in whom "the chest becomes of a size considerably beyond its ordinary dimensions,"[1] owing to the permanent and constant necessity for inspiring a greater volume of air than in less elevated situations.

True hypertrophy of the lung is most readily produced when, as in the above cases, the distending force acts equally on the whole or a large part of the pulmonary tissue, and when it is so gradual as to give time for corresponding changes in the nutrition and circulation of the parts. For this reason, emphysema seldom arises to a marked extent when one lung replaces the function of another destroyed by pleurisy; the expansion of the sound lung remaining limited by the normal conditions until all the structures have gradually accommodated themselves, under the influence of exercise and habit, to the altered circumstances of the system. That emphysema may be produced, however, to a certain extent in such instances, is shown by the case marked XII. 349, as well as in others of a similar kind which I have witnessed. The forms of pulmonary disease in which emphysema is most readily produced, on the other hand, are those where the primary lesions have been much disseminated, so that every part of the chest, in its expansion, acts at once directly upon corresponding portions of lung partially collapsed or atrophied, and yet containing many comparatively unobstructed lobules, which yield readily to the distending force. Hence the most frequent of all combinations with recent emphysema, as may be seen by reference to the table, is a certain extent of collapse of the posterior portions of the lungs, with a number of disseminated lobular condensations between the emphysematous parts. As these lesions are also very rapidly produced, and give rise to dyspnœa extremely urgent, they are apt to induce accelerated and laborious efforts at inspiration, in the midst of which emphysema, either of the interlobular or vesicular kind, or both combined, very readily arises.[2]

The theory here proposed has already been advanced by various writers, and with different degrees of precision of statement, to account for those cases of emphysema which are connected with the cicatrisation of tubercular cavities and other kinds of pulmonary atrophy. It is obvious, however, that its true significance, and the extent of its application, cannot be understood, till it is clearly apprehended that all cases of considerable obstruction in bronchitis bring with them, *as a necessary consequence*, a certain amount of diminished volume in the obstructed parts of the lung; and, therefore, that the connection of emphysema with bronchitis need present no difficulty

[1] Med. Times, July 20, 1850, p. 72.

[2] The relation of emphysema to the violence of the inspiratory efforts, rather than to the apparent importance of the pulmonary lesion, is noticed by Rilliet and Barthez, "Maladies des Enfans," vol. i., p. 139.

to the pathologist, even when the latter affection has not been so violent or long-continued as to lead to any considerable amount of permanent and evident occlusion. That emphysema prevails in the opposite parts of the organ to those in which the direct effects of bronchitis are observed, becomes, in this point of view, one of the strongest evidences of its connection with that affection. That in the great majority of cases it is found in company with bronchitic collapse, or some lesion implying diminished size of the organ, amounts, I think, almost to demonstrative proof of the correctness of the theory here advanced.

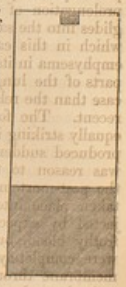
I am prepared, then, to maintain, that emphysema of the lung may, in all cases which I have witnessed, be satisfactorily accounted for by considering it as *a secondary mechanical lesion, dependent on some condition of the respiratory apparatus leading to partially diminished bulk of the pulmonary tissue, and consequently disturbing the balance of air in inspiration.* I therefore submit this principle to the judgment of the profession, in the confident anticipation, that it will prove no less constant and satisfactory in the hands of other observers, and will establish itself as the exclusive law of the production of this most important lesion.

A very few facts, in addition to the evidence already adduced, appear to be so striking as to deserve to be placed in an isolated form before the reader. One of these is found in Case IV., formerly narrated (p. 15), and also inserted in the table (XII. 360.) A child, in whom the right lung was normal, excepting imperfect bronchitic collapse, had in the left lung a mass of tubercular bronchial glands pressing on the bronchi passing to the anterior prolongation of the lower lobe, which was accordingly perfectly collapsed, void of air, and flaccid. The corresponding prolongation of the upper lobe, which in the act of inspiration glides into the same angle of the pleural cavity, and the bronchi of which in this case were free, presented very marked interlobular emphysema in its early and perfectly recent condition, and the other parts of the lung were normal. Nothing can be more clear in this case than the relation of the collapse to the emphysema, both being recent. The following case, which occurred to me lately, is an equally striking illustration of this point. An aneurism of the aorta produced sudden death, by bursting into the air-passages. There was reason to think, however, from the symptoms, as well as the post-mortem appearances, that bleeding to a less extent had taken place internally some tin e before death, without being rejected by expectoration. The bronchi on both sides contained frothy blood, but the *lower* bronchial branches of the left lung were completely stopped up with coagula of blood. The mucous membrane throughout the air-passages was quite healthy, though stained purple. The right lung appeared externally uniformly emphysematous, or at least distended with air throughout the upper and middle lobe, and less so in some parts of the lower lobe. The

surface was marked with purplish irregular mottlings, which could be seen to be quite beneath the pleura, and shining through from the substance of the lung (blood-stains, without condensation). Considerable portions of the lower lobe presented distinctly-marked lobular collapse. The left lung was also generally emphysematous in the upper two-thirds of the superior lobe. The inferior third was partially condensed and flaccid. The whole of the lower lobe was violet-coloured, completely condensed, and flaccid, having all the external characters of carnified lung. The whole sequence of the phenomena is here again most evident: the coagulated blood in the lower air-passages, especially of the left lung, producing obstruction and collapse, while in the upper part of both it had merely produced staining or mottling of the tissue, the bronchi being free, and the tissue generally emphysematous. In like manner, in emphysematous lungs having, as is usually the case, distinctly marked collapsed lobules or portions in the anterior edge, I have frequently been able to demonstrate the excess in the bronchi of the latter of muco-purulent matter; and in all cases the greater amount of obstruction may be demonstrated by the attempt to inflate the lungs, when the emphysematous portions will be found to yield at once, while the others follow slowly and often imperfectly.

Relation of Emphysema to Hepatization and Tubercle of the Lung.—It has already been shown, that no apparent numerical relation exists between emphysema and hepatization or tubercular deposit in the lung; the per-centage of cases of emphysema accompanied by these affections being nearly the same as in the general hospital dissections. These facts agree in all respects with the theory just stated, which shows that morbid deposits, affecting the ultimate tissue of the lung, can have no direct connection with the production of emphysema, unless they lead, in the first place, to diminution of bulk, or atrophy of the parts involved. This is not the case either with tubercle or hepatization in their recent condition, except when connected with bronchitis, in which case they may lead to the condition represented in the diagram, fig. 5. If a lobe of lung be, in the first place, completely hepatised, a subsequent attack of bronchitis may produce the collateral lesions of collapse and emphysema in the remaining lobes, the hepatized part remaining indifferent both to the one and the other tendency. On the other hand, bronchitis and its attendant phenomena may be succeeded by hepatization or tubercle. In either case they exert no direct influence upon the mechanical conditions under which respiration is accomplished.

Fig. 5.



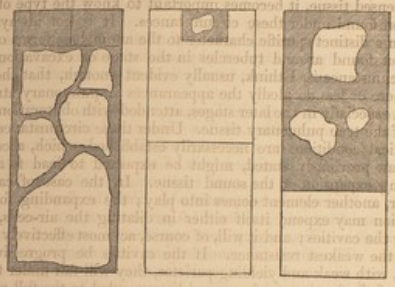
Relation of Emphysema to Excavations, and to Tubercular or Bronchial Abscesses.—The above remarks apply to tubercle in the

state simply of morbid deposit, before it has proceeded to excavation. The formation of cavities, however, in the lung, from whatever cause, is calculated to modify so considerably the mechanical conditions for the production of emphysema, that it is necessary to devote some consideration to this subject. As cavities are usually surrounded by condensed tissue, it becomes important to know the type of condensation found under these circumstances. It is not always easy to assign a distinct specific character to the anomalous forms of condensation found around tubercles in the stage of excavation; but one circumstance is, I think, usually evident enough, that they assume more or less decidedly the appearances of pulmonary atrophy, and are, especially in the later stages, attended with obliteration of all trace of the true pulmonary tissue. Under these circumstances, the mechanical conditions are necessarily established, which, according to the law previously stated, might be expected to lead to a compensating expansion of the sound tissue. In the case of cavities, however, another element comes into play; the expanding force of inspiration may expend itself either in dilating the air-cells, or in dilating the cavities; and it will, of course, act most effectively where it finds the weakest resistance. If the cavities be progressive, ill-defined, with weak and yielding parietes, they will fall under the influence of the inspiratory force, and be expanded to the full amount necessary to compensate the atrophy; and this result will follow the more easily, in proportion as they occupy, collectively, a larger proportion of the lung. On the other hand, if the cavities be healing, and surrounded, as they usually are in such circumstances, by very dense fibrous tissue; and if there be much sound pulmonary tissue, the cavities being few in number, and small, the expansion during inspiration will principally take effect upon the air-vesicles, and emphysema will be produced. These conclusions harmonise in all respects with the results of experience. Most pathological writers have noticed the frequent connection of emphysema with cicatrised cavities and healed tubercle; and even Rokitansky, whose opinions on the incompatibility of the two affections I have already noticed, admits that, with healing or healed (obsolete) tubercle, emphysema is frequently found. Is it too much to suppose that this whole doctrine of alleged incompatibility has been founded on the fact, that rapidly ulcerating and extending cavities, form a condition unfavourable to the development of emphysema; and that consequently, *in this stage*, they rarely co-exist? Such is, in truth, the whole extent to which, I believe, experience will be found to corroborate this hypothesis.

The relation of cavities to emphysema, then, may be shortly stated as follows:—1st, Large or numerous cavities, with flaccid walls, are, even when accompanied by atrophy—(see fig. 6.)—unfavourable to the development of emphysema; 2d, Cavities in process of cicatrization, if few or small in extent, and surrounded by firm atrophied walls—(see fig. 7.)—are extremely favourable to

the production of this lesion; 3d, Cavities, not surrounded by atrophied walls, whether large or small, exercise no particular influence in relation to pulmonary emphysema.—(Fig. 8.)

Fig. 6. Fig. 7. Fig. 8.



The great confusion which has hitherto existed on this subject, may be shown by reference to the best pathological works of the present day. The theory of Rokitansky, which derives one of its chief proofs from the relation of emphysema to tubercle, has been already noticed. Not to mention the absurd propositions made at various times to cure tubercular lungs by establishing emphysema, the following passage, from the careful and truthful work of Hasse, will show conclusively the correct apprehension he had of the facts, and the difficulty of finding a sufficiently general solution for them. "Those much in the habit of examining the dead body, cannot but be struck with two circumstances,—First, The almost invariable existence of emphysema in lungs, which bear the characteristic marks of recovery from phthisis;—and, Secondly, The *proportionate* rareness of tubercular deposits in emphysematous portions of lung. This would seem to show, that dilatation of the air-cells constitutes one of the conditions under which the cure of phthisis is possible, and again, that it forms *an obstacle to the development and progress of tubercle*." Hasse then gives an accurate picture of the mode in which the cure of phthisis tends to the production of emphysema; and it is therefore clear, that, from the same series of facts, he makes two distinct inferences: one, that the cure of tubercle causes emphysema; the other, that emphysema causes the cure of tubercle. One of the two is, as I need not point out, obviously superfluous. In a subsequent paragraph he says:—"The different forms of active hyperemia are likewise subject *to the control of emphysema*." Emphysematous portions of lung seldom become

affected with œdema. Pneumonia, for the most part, *leaves exempt such lobules as happen to be emphysematous.*" [1] There is here the same inverted inference as in the other case; but no hint of the correct solution of the phenomena. The principle that emphysema is always a secondary lesion, and always complementary of other states involving diminished bulk of the lung, at once solves all such difficulties.

There remains for consideration the relation of emphysema to the chronic forms of pulmonary atrophy and induration, including concretions, and of these lesions to affections of the bronchi.

Permanent Atrophy of the Lung in relation to Bronchitis.— In the preceding investigation of the causes and mechanism of pulmonary emphysema, the connection of that lesion with all causes leading to partially diminished bulk of the lung has been fully displayed. Among these causes of emphysema, bronchitic collapse of the lung and its sequelæ unquestionably hold the first place in importance and frequency; about 67.5 per cent. of the cases of emphysema in the table, having been manifestly connected with these affections. The forms of condensation here referred to may be divided into the more recent and the more chronic; the former being pure instances of bronchitic collapse, usually capable of easy removal by artificial insufflation of the dead lung, and therefore probably remediable in their character; the latter, on the other hand, having acquired more or less of a permanent type. The latter kind of condensation I distinguish by the name of *pulmonary atrophy*, a condition of the lung, the various forms of which are very imperfectly described by systematic writers, and by no one, so far as I am aware, except by Dr Stokes, ascribed to bronchitis.

The connection of atrophy with bronchitic collapse of the lung, can require but little explanation to the reader of this memoir. That the lung affected with collapse should after a time become altered in its structural relations, and be the subject of a permanent contraction or even obliteration of the air-vesicles, is no more than might have been apprehended from the knowledge of what takes place in the pulmonary tissue when subjected to long-continued pressure from pleuritic effusion. In such cases, it becomes, after a time, more or less impermeable to air, and incapable of its former expansion, even when the fluid has disappeared from the pleura, and all the mechanical conditions are favourable for its return to the normal state. Observation and experiment on the dead body also shew, that in those cases the proper tissue of the lung has in part disappeared, and that the air-vesicles which remain are incapable of assuming their original volume by any amount of expanding force.

[1] Hasse's Path. Anat. (Sydenham Society), pp. 313, 314.

In some instances, indeed, where compression has not been too long continued, we have good evidence that time will do much towards restoring the lung to its former condition; but in the majority of cases of chronic pleurisy, a permanently contracted side, with some degree of hypertrophy in the opposite lung, form the nearest approach to a perfect cure.

A precisely similar series of changes may be observed in the collapsed bronchitic lung, and has, indeed, been already alluded to in the first part of this memoir (Cases III. and IV., p. 15). In the dead body, the following gradations may be traced:—1. In the quite recent forms of the affection, the collapsed lobules yield before a force somewhat greater than that which inflates the sound portions of the lung; when inflated to the utmost, they are pale, emphysematous in appearance, and of volume equal to the surrounding parts; and when allowed again to subside to the ordinary condition of the dead lung, they are undistinguishable from the originally sound portions. This is the condition of simple collapse without atrophy. 2. The collapsed lobules cannot be inflated without the application of considerably greater force than in the former case; they then yield, however, and though perhaps not gaining altogether the full volume of sound lobules, are, on subsidence, not very easily distinguishable from them. 3. On insufflation of the lung, the collapsed lobules yield after very considerable resistance, but evidently not to the full extent; on allowing the lung to subside, they return more or less completely to the collapsed and non-crepitant condition. These phenomena are of course best observed in the well-defined lobular collapse which affects the anterior and lower edges of the lungs, as in Case IV., already alluded to. 4. The collapsed lobules cannot be inflated with air, except perhaps by a force sufficient to rupture the tissue; and then the air passes more readily into the interlobular spaces than into the obliterated air-vesicles. This is the condition of complete simple atrophy.

In *simple atrophy of the lung*, the result of uncomplicated bronchitic collapse, the affected parts usually present somewhat different characters from other forms of pulmonary atrophy. They are, in fact, reduced to a lax fibrous or areolar texture, inclosing the remains of bronchi and vessels; perfectly flaccid, free from all induration or abnormal exudation, and very frequently, in the purest form of the lesion, free even from that excessive deposit of carbonaceous pigment, which is so apt to accompany all chronic affections of the lung. Such atrophied lobules will almost invariably be found, on examining the free anterior or lower margins of old emphysematous lungs; and, in more recent specimens of emphysema, the anatomist will generally be able to trace several of the stages which I have indicated above, as intervening between collapse and atrophy. The atrophied lobules at the edge of the lung, correspond to the indentations and grooves between the emphysematous parts. On examin-

ing them closely, there will often be found a thin lamina, spread out between two emphysematous prominences, like the web of a frog's foot, and composed of the two pleural layers, enclosing the attenuated remains of the pulmonary tissue. Such portions are generally clearly and definitely marked off by the interlobular septa from the emphysematous lobules in their neighbourhood. In other instances, scarcely even this amount of tissue can be traced, and the two pleural layers may appear to be almost in contact over a small space, with little or no intervening substance. To any one who attentively studies a variety of such specimens, it will be apparent that simple atrophy of the lung, in its most complete form, is a lesion only to be distinguished by negative characters. The proper and special elements of the pulmonary tissue have disappeared; but they are not replaced (as in atrophy from other causes) by any adventitious structure, or even by the thickening or induration of the fibrous basis. For this reason, simple atrophy is sure to be overlooked, unless its traces be sought for in the manner I have described. In the centre of a lung, very many lobules may be entirely atrophied, and leave no visible or tangible evidence of their previous existence.

Simple atrophy, like the lesion which gives rise to it, occurs in the lobular and the diffused form. The latter is chiefly found in the posterior portions of the lungs, near their root, among the great bronchi and the bronchial glands, which are often, in these cases, dark coloured from infiltrated carbon, even when the lungs are by no means remarkably so. In diffused simple atrophy, the lung is rarely entirely condensed, generally retaining a certain degree of crepitation, but being dense, tough, and fibrous; sometimes dark slate-coloured, at other times not so; and in the most marked and exaggerated examples, crossed in every direction by fibrous processes, or septa of considerable thickness and density, corresponding to numerous depressions and irregularities on the surface of the lung, which is usually in these cases very emphysematous in front, and over the surface generally. Such lungs will always be found, when a fresh section is inspected with or without a lens, to present the most remarkable varieties in the size of the air-vesicles; some of which are entirely obliterated, or very small, and others greatly expanded beyond the normal volume; the latter condition prevailing, of course, towards the anterior margins in the most emphysematous parts.

The changes impressed upon the form and movements of the chest, by the chronic sequelæ of bronchitis, form so marked an illustration of the doctrines above recorded concerning the supervention of atrophy on bronchitic collapse, that some reference to them here is quite necessary to the complete treatment of this subject. I have already alluded in the first part of this memoir to the modifications of respiratory movement which take place in acute bronchitis in children, while the bones and cartilages are, as yet, inadequate to the task imposed on them of expanding the chest under conditions of increased resistance. Under such circumstances, it is not very unusual to find the movements of the lateral regions of the chest actually reversed, the parietes being, as it were, sucked inwards at each descent of the diaphragm, owing to the external atmospheric pressure overcoming their power of expansion. This yielding of the ribs I have indicated as probably one of the causes of the extremely frequent occurrence and great extension of bronchitic collapse in very young subjects. In rickety individuals, it is not only more marked, but apt to become permanent, especially when such subjects are affected with any considerable or persistent bronchitic affection. In such cases, the reversed movement of the ribs is stereotyped, as it were, in the form of chest called *pigeon-breast*, in which the sternum is protruded, particularly below, and the whole lateral region, including also the lower costal cartilages in front, flattened, or even at some points rendered irregularly concave.

Many slighter and more partial permanent irregularities in the form of the chest are no doubt owing to infantile bronchitic attacks, either modifying the original expansion of the lung, or producing subsequent partial collapse of its tissue. The immense frequency of such diseases in childhood, and the unquestionable tendency which they are now shown to have towards structural changes, will probably go far to account for many of those disorganizations in the lung, revealed by morbid anatomy in a large proportion of cases, and which often seem to have no connection with any thing in the history of the individual. To a similar source may, in all probability, be justly traced most of the so-called "physiological heteromorphisms" of the chest, described and investigated with such elaborate minuteness by M. Woillez.[1] According to this writer, these slight and trivial deviations of the apparently healthy chest, occupy, for the most part, the same situations as those which are known as the results of disease; and, indeed, it would appear that the "physiological" and "pathological" irregularities are by no means separated by a very distinct line of demarcation. It is quite true that, in many of the individuals presenting these changes, no history of chest-disease can be procured; but every one accustomed to the task knows that the elimination of information in regard to diseases of early life, is, in the cases of most hospital patients, nearly impossible, even where the disease has been of considerable importance. In so far, therefore, as these "physiological" irregularities are worthy of consideration at all,[2] I cannot but

[1] Recherches Pratiques sur l'Inspection et la Mensuration de la Poitrine. Paris, 1838.

[2] This qualification is not unimportant, seeing that M. Woillez, by means of rather ponderous statistical machinery, has arrived at the singular conclusion, that only 1 in 3 of the *healthy* chests, and about 1 in 5 of *all* the chests examined by him, present a strictly *regular* conformation. It is obvious that, with a few more refinements such as those to which this observer has devoted so much labour, the ideal of regularity would require to be sought altogether beyond the

think that infantile bronchitis may probably have a large share in their production.

In adults, the motions of the chest are altered to a considerable extent in bronchitic affections, though not nearly to the same degree as in children. The greater solidity and firmness of the bones and cartilages opposes an effective resistance to that abrupt and well-marked retraction of portions of the thoracic wall which has been noticed as occurring in infantile bronchitis; the chest expands more uniformly and forcibly over its whole surface, and the phenomenon which gives rise to the pigeon-breast is not observed when the bones are healthy. That the respiratory motion may be seriously limited, however, and sometimes even reversed, at certain points in the adult chest during bronchitis, is demonstrated by the observations of Dr Sibson, made with the aid of his ingenious and useful instrument, the chest-measurer.[1] These observations I regret that I have not yet been able to repeat, but of their general accuracy I can entertain no doubt. It is evident, indeed, to the eye (which, when employed with the requisite care, is in this case a far less deceptive, and more instructive instrument, than the ordinary measuring tape) that, while even in the severer forms of bronchitis, the chest on the whole expands both in its upper and lower zones, the movement of the latter is much more restricted than that of the former; and that while the *lateral* expansion of the thorax is circumscribed, the *anterior* movement of projection of the sternum and costal cartilages is usually even exaggerated. The result of this curious modification of respiration is, that in cases of long-continued chronic bronchitis, even during the intermissions of accumulation in the air-tubes, an altered habit of breathing is acquired and permanently retained; and the stethoscope, as well as the inspection of the chest, can often determine in such cases that respiration is effected chiefly by the upper and anterior portions of the lung, and by the movements of elevation and projection of the sternum; while the parts of the lung corresponding to the lateral and posterior regions of the chest, remain comparatively little affected by the respiratory act. The modification in the permanent form of the chest which supervenes upon this condition, is tolerably well-known as the "cylindrical" or "emphysematous" chest; it is marked by increased fulness and prominence of the whole anterior thoracic vault; often also, but not invariably, by increased arching of the sternum from above downwards; and perhaps yet more characteristically by a diminution in the lateral, and a relative increase of the antero-posterior, diameter of the thorax.

pale of humanity. It appears very doubtful whether even the Apollo or the Antinous could withstand the search for "physiological heteromorphisms" by M. Woillez. At all events, artists and anatomists are well aware that, among the poor sons of Adam, strict symmetry and regularity in every point of form, is an occurrence of almost fabulous rarity. The very general lateral curvature of the dorsal spine, and the all but invariable lateral deviation of the nose, are glaring instances known to every one. How often do the phrenologists find a *regular* head?—or would any two of them agree upon the subject?

[1] Medico-Chirurgical Transactions, Vol. xxxi.

The true relation of these changes to the existence of collapse posteriorly, and emphysema anteriorly, in the lungs, is not altogether so clear as it may at first sight appear. That the permanent modification of form is the consequence of the peculiarly altered movement of the chest which I have described above, will admit of little doubt to those who have witnessed this movement in characteristic cases of acute and chronic bronchitis. It may also be freely admitted that the diminished lateral motion is the direct effect of the diminished expansion of the lung in consequence of bronchitic accumulation, with partial collapse, and perhaps subsequent atrophy of its tissue. But to ascribe the increased movement and consequent deformity of the anterior part of the chest to the production of emphysema, appears to me an error both of logic and of observation. I believe, on the contrary, that whatever be the relation of emphysema of the lung to the "emphysematous chest," *it is not directly or indirectly the cause of that deformity*. And this conclusion appears to be borne out by the following considerations:—

In the first place, the increased respiratory movement in the anterior part of the chest, which appears in all cases to be connected with the generation of emphysema, as well as of the deformity above-mentioned, exists in a large number of instances of bronchitis, before either the one or the other condition has yet arisen; indeed, in its slighter degrees, I believe the increased anterior thoracic movement to be an almost constant concomitant of that affection. In the second place, the existence of very well-marked emphysema, though unquestionably concurring with the highest degrees of the deformity, has always appeared to me to tend to diminution of the abnormal excess of motion; this excess being always detected most characteristically in company with simply puerile, not emphysematous, respiration. It appears, therefore, impossible that the generation of emphysema can be the cause of that exaggerated motion. Lastly, according to arguments and observations already laid before the reader, it appears that emphysema is a lesion directly due to the forcible expansion of the chest under peculiar circumstances, which seems fairly to exclude the opposite proposition, that undue permanent expansion of the chest can ever be owing to the existence of emphysema.

From observations on this subject, it appears to me susceptible of demonstration,—that the abnormal motion of the chest, in the cases above alluded to, always precedes both the deformity and the emphysema; that the emphysema frequently precedes the deformity, but in its more chronic and exaggerated forms generally follows in its wake; that a certain amount of emphysema may exist without deformity, and a certain amount of deformity without marked emphysema; and that, in any given case, when emphysema

supervenes on exaggerated anterior movement, with or without deformity, its natural effect is to diminish that excessive movement. This last proposition corresponds with the state of the lung in extreme emphysema, in which the emptying of the air-vesicles is effected with great difficulty, or even may be absolutely impossible, owing to the existence of an apparently valvular obstruction to the egress of air; a condition which suggested to Laennec his theory of emphysema, but which I believe to be a secondary effect, and not a cause of that structural alteration.

Were I to hazard a speculation as to the mutual connection of this complicated series of phenomena, it would be that indicated in the following propositions, which I submit to the reader, not as ascertained truth, but simply as being the most probable conclusions at present attainable in relation to this subject:—1. The direct tendency of bronchitis is to produce bronchial accumulation, and thereby to restrain the expansion, or even to produce retraction, of the whole lung, and consequently of the chest. 2. To overcome this tendency, forced respiration is at once thrown into action, and the breathing, from being, as in the normal state, mostly diaphragmatic, becomes in a high degree costal and thoracic. 3. In overcoming resistance, by means of costal superadded to diaphragmatic respiration, those parts of the chest, whose movements are performed by the most powerful muscles, acting at the greatest mechanical advantage, tend to assume the principal function, while the remaining portions fall into abeyance, or yield in part to the opposing resistance. 4. On this principle the elevation of the sternum, and of the anterior ends of the true ribs, which is effected by the powerful aid of the cervical muscles in addition to the intercostals, becomes the predominating movement along with the descent of the diaphragm; while the motions of the posterior and lateral parietes of the chest, which are maintained, in the normal state, by a much weaker force, tend to fall into abeyance. 5. The respiratory forces, instead of acting equally on all parts of the pulmonary surface, and tending to expand it from all points at once, are thus spent in greater measure upon the anterior edge and upper part of the lung, which are in contact with the most mobile parts of the thorax, as well as upon the lower edges and diaphragmatic surface; and these parts, therefore, become the principal seats of respiratory movement, while the root of the lung and its lateral and posterior surfaces only receive the inspiratory impulse secondarily, or in greatly diminished ratio. 6. The consequence of the inferior power of movement in the posterior and lateral parts of the lung, is accumulation and stagnation of mucus in the tubes; thence a greater liability to pulmonary collapse and atrophy as the consequence of bronchitis. 7. The consequences of the superior power and greater extent of movement at the edges and upper parts of the lung, and on the diaphragmatic surface, are comparative freedom from mucous accumulation, and consequently from pulmonary collapse and its consequences, and on

the other hand, much greater tendency to the development of emphysema from violent and repeated forced inspiration, when partial collapse or atrophy is present elsewhere. 8. The irregularities of movement of the thorax tend ultimately to affect its form, producing in the child the *pigeon-breast*, by lateral flattening of the yet flexible and soft ribs, with depression of the lower costal cartilages, and protusion of the sternum; in the adult or older child, slighter lateral flattening, with expansion or bulging of the cartilages, and arched protrusion of the sternum; and in both the child and the adult, increase of the antero-posterior diameter relatively to the lateral, and of the upper zone relatively to the lower. 9. The deformity of the chest usually accompanying emphysema of the lungs is neither a cause nor an effect of that lesion, but both emphysema and the "emphysematous chest" depend on the altered respiratory movements in bronchitis, and the exaggerated respiration necessary to overcome the tendency to bronchitic collapse of the lung.

It may appear to some readers that the above explanation of the seat of election of pulmonary collapse and emphysema is superfluous, and that the gravitation of the mucous obstructions in bronchitis to the posterior portions of the organ, is a sufficient reason for the occurrence of collapse in that situation, and of emphysema in the opposite region. To this opinion, however, some facts stand in direct opposition. The most important is that in the horse, in which emphysema and the other diseases of the lung are common, and in which the position of the lung as respects the effect of gravitation is precisely the reverse of what occurs in man, the seats of election of emphysema and of pulmonary condensation are nevertheless nearly as in the human subject. In various experiments on the rabbit, also, I have noticed the same tendency of emphysema to the borders of the lung, and of collapse to its root, although the animals were allowed to maintain the natural position, in which the force of gravitation ought to have had an opposite tendency. For these reasons, I have been induced to ascribe very much less effect than most observers to the simple statical condition of the fluids in pulmonary diseases, and to look for some dynamical cause which would explain the position of the lesions found in bronchitis, pneumonia, and emphysema, in a more satisfactory manner than hitherto. To what extent the preceding paragraph is a successful attempt at such an explanation, must be left to the judgment of the reader, and to the future observation of facts bearing on the subject.

Pathological alterations of the Bronchi in Pulmonary Atrophy and in Emphysema.—The memoir of M. Reynaud[1] on obliteration of the bronchi, has been referred to by most subsequent writers as having enumerated and described with great completeness all the more

[1] Memoires de l'Academie de Médecine. Tome iv.

ordinary varieties of permanent contraction or dilatation of the air-passages. Indeed it is difficult to conceive anything more completely exhaustive than this memoir, when considered purely in an anatomical point of view, and solely with reference to the air-passages; and having frequently had opportunities of verifying nearly all of his observations, I find it, like most others who have referred to them, not easy to state anything novel upon this subject. But M. Reynaud's researches, though full of anatomical truths, are strangely barren, at least in his own hands, of real pathological interest; which arises chiefly from his having too exclusively pursued the inquiry relatively to the bronchi themselves, and not having sought to connect their alterations with those of the pulmonary tissue, with which they are, according to my experience as well as that of others, constantly and indissolubly associated. Somewhat of the same objection applies to Laennec's observations on dilatation of the bronchi, which first gave to this disease a place in pathological anatomy. Accordingly it has been reserved for future observers to discover, that both the dilatation and the contraction of the bronchi are almost always secondary lesions, or at least invariably connected with some kind of disorganization of the pulmonary air-cells. Several of the later pathologists have adopted these views, with more or less decision; among whom may be mentioned Hasse, Rokitansky, Stokes, &c.; but the ideas of Laennec and of Reynaud have still been adopted by many writers on the diseases of the chest, with perhaps too little discrimination, and very little real advance has been made in the pathology of these affections.

The forms of obliteration and contraction described by Reynaud are numerous. It is scarcely necessary to enter into the consideration of the anatomical varieties. The different kinds of dilatation, as described by Laennec and Reynaud, and their relations to obliteration of other parts of the same bronchial divisions, are of more interest. The most frequent are the following:—A small bronchus of normal caliber suddenly opens out into a sacculated dilatation, lined by smooth thin membrane, and of more or less rounded form. This dilatation sometimes terminates all trace of the bronchus; in other specimens, the contracted and obliterated remains of bronchi pass from its opposite end towards the circumference of the lung. Again, a bronchus may show a succession of marked irregular dilatations through its whole length, at some parts having the sacculated character, at others being irregularly cylindrical, with partial annular projections, and transverse septa arising from the walls of the dilated tube. Finally, the whole of a lung or of a lobe may be broken up into a series of cavities, having free communication with each other, and with the main bronchi of which they are presumed to be dilatations.

On all the changes here described, there is one important remark to be made—they are invariably found in close connection with atrophied lung, either of that kind which results, as we have seen,

from bronchitic collapse, or some of the more complex varieties which proceed from other lesions, such as tubercle or chronic hepatization. With regard to obliteration or contraction of a bronchus, it is indeed self-evident that this must be the case; and the reader of the foregoing parts of this memoir will see without difficulty by what steps the obstruction of the air-tubes leads to their contraction, along with the collapse and gradual atrophy of the vesicles to which they lead. In the case of dilated tubes, it is an observation of all the later pathologists, in which I fully concur, that the pulmonary tissue around these dilatations is usually impermeable, and in a condition of fibrous atrophy, most commonly without marked induration. It was this circumstance which gave rise to the theory of bronchial dilatation by Dr Corrigan, which led him to call this disease "cirrhosis of the lung,"[1] conceiving that the formation of a peculiar contractile fibrous tissue in the interstices of the bronchi, and the obliteration of the air-cells, led to the expansion of the tubes, by the gradual operation of the inspiratory forces, and constituted the true pathological condition of the disease. Although I have little doubt that Dr Corrigan's theory is fallacious, in so far as regards the existence of any *new* or *peculiar* fibrous element in this affection, yet there is no doubt that his observations were in other points correct, and that in particular he has the merit of being the first to draw attention in a decided manner to the morbid alteration and obliteration of the air-cells, a fact singularly enough nearly overlooked by Laennec.

What, then, is the origin of bronchial dilatation? The explanations of Laennec and others, which ascribe it to violent coughing, to distension by accumulated mucus, &c., are clearly unsatisfactory, on precisely the same grounds as have been already indicated in the case of emphysema, and which there is therefore no occasion again to repeat here. As in the case of emphysema of the vesicles, it seems more consonant with reason to ascribe these dilatations (as is done by Dr Corrigan) to the expansive forces of inspiration acting upon the bronchi of atrophied lung. But it is difficult to understand, on this principle, the occasional partial character of the lesion—the expansion of one portion of a bronchial tube into a sacculated globular enlargement, while adjoining tubes and adjoining portions of the one affected, retain their natural size. In such cases it becomes necessary to suppose the existence of some more local affection, rendering the bronchial tube dilatable at the point in question.

To those who have studied this subject only in the light of Laennec's description, the following remarks will probably appear too bold and sweeping a generalization. They are, nevertheless, the result of much consideration, both of the descriptions of authors,

[1] Dublin Journal. May 1838.

and the facts observed in very numerous dissections, as well as in most of the public pathological collections of this country. The conclusion to which I have been led by this survey is, that almost all the so-called bronchial dilatations, and all of those presenting the abrupt sacculated character here alluded to, are in fact the result of *ulcerative excavations* of the lung communicating with the bronchi. That such ulcerations are not uncommon in bronchitis, especially in the case of children, has been already sufficiently indicated in the first part of this memoir, under the head of bronchial abscess. I have, in fact, incised the bronchi with great care in cases of recent bronchitis with bronchial abscess, and have found the small cavities so described to occupy precisely the same relations to the caliber of the tubes as the larger dilatations which are found in connection with chronic atrophy. The expansion of these small cavities, either by increase of ulceration, or by the act of inspiration, would clearly in these cases have led to an appearance closely resembling the so-called dilatations of the bronchi in everything except the fine, smooth, and consistent lining. On the other hand, the examination of a very great number of unquestionable instances of chronic pulmonary excavation from tubercle and other causes, has satisfied me that cavities originally formed by ulceration, may become lined by membranes exactly resembling those found in the "dilated bronchi" of Laennec. In no instance that I have seen, has this membrane exactly resembled the mucous membrane of a bronchus; even in the cases of so-called true bronchial dilatation, it is thin, dense, very smooth and glistening, and with comparatively few vessels; in fact, more resembling a serous than a mucous tissue. This description altogether concurs with those of Hasse[1] and others as applied to bronchial dilatation. It is, I think, almost conclusive upon this question, that in chronic cavities, evidently of tubercular origin, I have been able to trace quite satisfactorily the gradual assimilation of their lining to this type; and in several instances to observe cavities, which, but for the existence of others in the same lung in a different condition, would scarcely have been distinguishable from the so-called bronchial dilatations; they being lined by membrane perfectly smooth and glistening, and gradually passing into that of the undilated portion of the tubes leading into them. I have also observed that in such cases there is even the formation of an incipient epithelium upon this new membrane; or, at least, of numerous cells which, under the microscope, sufficiently resemble the columnar epithelium of various parts of the air-passages, differing, however, from that of others, and especially of the trachea and larger bronchi, in being of inferior size, and never, so far as I have observed, furnished with cilia. The opportunities of making such observations under the requisite conditions to ensure accuracy,

[1] Pathological Anatomy—article, Bronchiectasis.

are too rare to permit of my entering into this subject at greater length.

If these observations are admitted as bearing on this question, it will, I think, become probable that the usual origin of bronchial dilatations is in cavities formed in atrophied lung, in consequence of bronchitis or tubercle, and afterwards expanded beyond their original dimensions by the inspiratory force. The conditions that conduce to such expansion have been already considered under the head of emphysema, and its relation to excavations. It will be at once evident that the tendency to expansion of a cavity must be great in proportion to the flaccidity of its walls, and the absence of crepitant lung in its neighbourhood. It is owing to this circumstance, that bronchial dilatation and emphysema of the lungs have been found, to a certain extent, mutually exclusive. The cases most fitted for the development of such permanent excavations, are those in which the whole of one lung has been converted into a series of cavities, with no intervening crepitant tissue. Of this I have seen several examples. One such instance, figured by Cruveilhier,[1] forms an admirable illustration of Corrigan's cirrhosis, and of the real mode of its origin, viz., by ulceration. The whole upper lobe of the lung is converted into the condition of chronic cavities, lined by smooth membrane, and communicating freely with each other; while in the lower lobe are found recent excavations; and every intermediate condition between the two varieties can be readily traced. Of this case I have on one occasion seen an almost exact counterpart, in a boy, aged about 12 years, affected for a long time with hepatic and pulmonary disease, under the care of Dr Renton.

Pulmonary Concretions and Cicatrices.—The existence of cicatrices and puckerings in the pulmonary tissue, sometimes accompanied by distinct induration, with much thickening of the pleura in the neighbourhood, and sometimes by rounded whitish masses of atheromatous, chalky, or stony consistence, imbedded in the tissue of the lung, and surrounded by a fibrous cyst, has been long known to morbid anatomists, although more attentively studied of late years. Morgagni, summing up his own experience with that of his predecessors, signalised their existence in connection with asthma and other symptoms of disease of the respiratory organs.[2] Portal considered calculous concretions of so much importance, that he indicated, by means of them, a particular species of phthisis, the "phthisie calculeuse." He maintained the entire dissimilarity between the calculi of gouty and those of scrofulous origin, showing

[1] Anatomie Pathologique, Livraison, 32. Pl. 5. Fig. 3. The case was considered to be of tuberculous origin, yet "the cicatrization (of the excavations) was perfect; the parietes presented the appearance of accidental mucous membranes."

[2] De Sed. et Causis Morb. Epist. xv. 17, ad finem.

their frequent connection with phthisical symptoms, cough, hemoptysis, dyspnœa, &c.,[1] which, however, he did not consider as exclusive of the gouty diathesis, inasmuch as he has a "phthisie arthritique et rhumatismale." Portal also considered these calculi as formed in some instances by inspissation of the bronchial humours, as well as by dust inhaled from without, — an opinion which has received some recent confirmation in the special case of the stone-hewer's phthisis, but which, as a general deduction, is contradicted by the chemical nature of these bodies; the pulmonary calculi being now known to consist in a great part of phosphate of lime, and other calcareous and magnesian salts, evidently of organic origin. Laennec was the first distinctly to protest against the opinion that these pulmonary concretions were necessarily attended by symptoms; having, as he says, frequently found them "in subjects who had presented no sign of oppression or embarrassment in the respiratory organs." The description by Laennec of these bodies bears the marks of a very attentive observation of all their pathological relations. He notices their frequent occurrence with or without the accompaniment of other lesions, and remarks that they are occasionally found in the centre of tubercles, and very frequently along with pulmonary cicatrices, like those which are found in tuberculous individuals; from which circumstance he concludes, that, "in the greater number of instances, they are the result of a cured tubercular affection." He does not, however, deny that osseous and cretaceous concretions may be developed independently of tubercle, but regards this as occurring very rarely.[2]

Since the enunciation of the above opinion by Laennec, as to the source of these pulmonary lesions, it has been for the most part acquiesced in by pathological authorities; and, as usually happens when a doctrine gains general support, even the prudent reservations of its author have been in some danger of being consigned to oblivion. The accuracy of Laennec's observation as to the frequency with which these lesions occur, is more than justified by the later researches of M.M. Rogée and Boudet of Paris, and Professor Bennett,[3] who have found that pulmonary contractions and puckerings, with or without concretions and thickenings of the pleura, occur in a very large proportion of the bodies subjected to examination in hospitals. In the cases of Dr Bennett, the proportion is about 40 per cent. of the whole; while M. Rogée and M. Boudet give respectively 51 and 86 per cent. in their different

[1] Phthisie Pulmonaire. Edition of 1809. Vol. i. 478 et seq.; vol. ii. 321, 349. The connection of calculi with phthisis was also maintained by Bayle, as well as other still older authors. See Sauvages' Nosologia Methodica—article Phthisis.

[2] Auscultation Mediate; tome 2, chap. 4.

[3] See the paper of Dr Bennett, in Ed. Med. and Surg. Journal, vol. lxiii., p. 406.

spheres of observation. It thus becomes a matter of considerable importance to determine whether the connection of these lesions with obsolete tubercle be subject to no exception; more especially as none of the observers above noticed have, in drawing inferences from the facts adduced by them, indicated any doubt upon this subject. I have thought it right, therefore, to make the following remarks, tending to limit the application of the doctrine of Laennec, which in being made to include all, or nearly all, pulmonary cicatrices and concretions, under the designation of healed or obsolete tubercle, appears to me to have been scarcely warranted by the facts of the case. That I may not be suspected, however, of an equally exclusive bias upon the other side, I may state that the healing of tubercles in this particular manner admits, in my opinion, not of the smallest doubt; and that to any one who has seen, on a sufficiently large scale, the progress of these lesions, as exemplified in the lungs of those dying of unquestionable tubercle, the conclusions of Laennec as to the frequent cure of tubercular lesions, especially in their early stages, must appear perfectly irrefragable. Nor is there anything in the doctrines of Carswell, Cruveilhier, or other subsequent writers, from which I feel called on to dissent, in so far as they illustrate the different modes in which this cure is accomplished. But, as regards the precise frequency with which the early stages of tubercle become obsolete, I believe that we are not yet in possession of accurate statistical results; and that neither the 40 per cent. of Dr Bennett, nor the 51 and 86 per cent. of the French observers, represent correctly the proportion of such cases in our hospital bills of mortality.

That simple bronchitis must be responsible for a certain number of pulmonary contractions and puckerings, will be at once evident to the reader of the preceding pages. Every instance of pulmonary atrophy, from whatever cause, which is abruptly defined and surrounded by normal or emphysematous tissue, will necessarily present the appearance of a cicatrix,—more especially if the pleura over it be, as often happens, thickened. It might be supposed that such cicatrices would in simple bronchitis occur chiefly or exclusively at the back part of the lungs; but this is by no means the case; for while partial atrophy occurs with extreme frequency at the back part of the lungs, it is rarely complete, and almost always in the diffused form; while the obvious pulmonary cicatrices arise from lobular atrophy, which occurs chiefly at the edges and upper parts of the lung. I know of no means by which a simple cicatrix, formed by bronchitis or broncho-pneumonia, surrounded (as such cicatrices often are) by a certain amount of induration and carbonaceous infiltration, could be distinguished from a tubercular lesion, unless the absence of tubercular traces in other organs, and presence of lobular atrophy along the whole edges of the lung with diffused incomplete atrophy behind, and the comparative exemption of the summit of the lung, be considered to indicate such a distinc-

tion. Even to one aware of all the characteristics of both forms of disease, cases will constantly occur in which no distinct opinion can be formed; at least such is the result of my experience since my attention has been particularly drawn to this subject. Nor can I for a moment doubt, considering the frequency of bronchitis at all periods of life, that a proportion of pulmonary cicatrices, quite large enough to modify considerably the statistical results alluded to, has been included among the tubercular lesions without having a just claim to this designation. This is particularly the case with M. Boudet, whose enormously high proportion of 86 per cent. could only have been attained, as it appears to me, even among old subjects (and it is not asserted that his were exclusively such) by diligently seeking out every trace of pulmonary contraction, by whatever cause produced. By such a method the traces of extinct pulmonary disease may indeed be discovered in a very large proportion of cases; but certainly not of extinct tuberculous disease.

With regard to concretions, which were found by Dr Bennett in about 22 per cent. of the bodies opened by him, I have little doubt that they have a tuberculous origin in a large proportion of cases. But something in the way of reservation requires even here to be kept in view. The occurrence of such obsolete masses of exudation in connection with old-standing bronchitis is far from uncommon; and as Laennec, and after him many others, have clearly traced tuberculous matter through all its stages into that of complete calculous induration, so has it occurred to me repeatedly to see old bronchial abscesses, having evidently the characters and the usual distribution of bronchitic lesions, in every stage of conversion into these bodies. Even when the conversion was complete, it has appeared to me that a certain proportion of those I have met with might be reasonably referred to this source, from their prevalence at all the borders of the lung, or from their being surrounded rather by what I have called *simple lobular atrophy*, than by any considerable induration. But in this, as in the former case, the observer who looks to all the possibilities of the case is apt to find instances of a sufficiently equivocal and doubtful character in the present state of our knowledge.

Without being prepared to defend the following conclusions as absolute, and without at present entering at length into the grounds for some of them, I may here endeavour to state briefly the inferences to which I have been led by the preceding and other researches in regard to such lesions of the lung as may be suspected to be connected with the extinction of tuberculous disease. 1. There can be no reasonable doubt that open excavations, one or many, completely cicatrised on their internal wall, and lined by a membrane possessing the appearance, and in some degree the minute structure, of an epithelial membrane, may be of tuberculous origin;[1] but such excavations (the *dilated bronchi* of Laennec) may also originate in simple bronchiitis, broncho-pneumonia, simple isolated abscess (a very rare disease), multiple or metastatic abscess, syphilitic ulceration, gangrene of the lung,—which last lesion also may have several causes. Of these forms of healed excavation, the tuberculous is probably the most frequent; but there are as yet no good characters to distinguish it from the others, unless collateral lesions, sufficiently characteristic, be found in other organs, or unless the original lesion of the lung be, in other parts of the same or opposite organ, in an earlier stage of its development. 2. Concretions of inspissated pus, atheromatous matter, cretaceous or calculous matter, surrounded by atrophy, with or without induration, are in the great majority of cases the remains of obsolete tubercles, softened or miliary; but they may also arise from any other form of pulmonary ulceration or abscess, as above enumerated. 3. Pulmonary atrophy, simple or with induration, and carbonaceous deposit, accompanied or not by adhesions and thickening of the pleura, may arise from obsolete tubercles, from bronchitis, from broncho-pneumonia, and probably also, though more rarely, from simple pleuro-pneumonia; and pulmonary cicatrices arise frequently from all these sources except the last. The *relative* frequency of these lesions as leading to cicatrices is as yet undetermined. 4. The lesions above mentioned (1, 2, 3) *are probably tubercular*, if they occur exclusively or chiefly at the apices and back parts of the upper lobe of both lungs at once; or in the apex of one lung only, without trace of a lesion elsewhere; or generally diffused throughout both lungs, but chiefly in their upper lobes, and especially at their back part and apex; or in any case in company with characteristic traces of tubercular lesions in other organs. In reference to this last clause, it is doubtful whether cretaceous or other deposits in the bronchial glands can, in a case of such pulmonary disease, be considered as a characteristic indication of the tuberculous taint; but such deposits in other parts of the lymphatic system, especially in the mesenteric and cervical glands, or the traces of old deposit or ulceration in the mucous membrane of the small intestine, would necessarily determine the tuberculous nature of the affection, except in some cases, rare enough in this locality, but of which I have seen examples, in which typhoid or cancerous disease might throw doubt upon the diagnosis. 5. All these lesions *are probably non-tubercular* if they occur in one lung in a generally diffused form, without traces of tubercle, even obsolete, in the other lung; or in the lower lobes to the exclusion of the upper; or at the edges of the lung in both lobes and not at its apex; or at the root of the lungs only; being in all these cases unaccompanied by tubercles, or the traces of tubercles, elsewhere. 6. After employing all these characters of distinction, a certain number of cases of all the lesions in question will remain of indefinite or unknown origin.

In concluding this contribution to the history of bronchitis, I am

[1] See the text of Cruveilhier, loc. cit.

well aware that many important practical relations of the various subjects discussed have been necessarily left almost untouched; but if I have succeeded in throwing any light on these relations, or in giving the practitioner a key to their apprehension in any degree simpler and of more extensive application than has hitherto been accessible, I am confident that the numerous minds at work in this country and elsewhere in the furtherance of practical medicine, will not allow these researches to remain destitute of the assistance which they are calculated to derive from more extensive opportunities of clinical research. With this conviction, I leave the foregoing pages in the hands of the profession.

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CASE OF ANEURISM OF THE AORTA,

ARISING FROM THE BACK PART OF THE ARCH,

SIMULATING LARYNGEAL DISEASE, AND FATAL BY SUFFOCATION.

By W. T. GAIRDNER, M.D., F.R.C.P.,
PATHOLOGIST AND ASSISTANT-PHYSICIAN TO THE ROYAL INFIRMARY OF EDINBURGH.

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(*Read to the Medico-Chirurgical Society of Edinburgh, 18th June* 1851.)

The following case of aneurism of the aorta is interesting, 1st, from the absence of physical signs, and the prominence of the symptoms of a laryngeal affection; 2d, from the free communication of the sac with a mucous canal, without causing serious hemorrhage; 3d, from the termination of the disease by suffocation, and the remedial measures suggested by this termination. On these grounds I beg to lay it before the Medico-Chirurgical Society, along with a preparation showing the parts involved in the disease.

Thomas O'Brien, æt. 46, a robust labourer, was admitted into the hospital at the hour of visit on the 30th May 1851. He complained of great dyspnœa, which, in the recumbent posture, was so extreme as to threaten suffocation. The breathing was sonorous, with a distinctly stridulous character on inspiration. The countenance anxious and flushed; no fever or pain complained of. Expectoration considerable; the chest was examined as well as his state permitted, and revealed only slight bronchitic râles, the harsh laryngeal breathing being heard over all the bronchi. The voice was evidently produced with effort, scarcely husky, but having a somewhat muffled character; there was no tenderness over the larynx; the epiglottis and throat were natural. Shortly after his removal to a ward, the paroxysm subsided to some extent. I then learned that this was only an accidental exacerbation of a state which had existed for some months, and for which he had undergone active treatment. A blister was applied to the nape of the neck; and he was ordered ipecacuan wine 3ss every second hour.

On the 31st he had slept well; but the dyspnœa had returned in

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the morning; at visit he was better, but not able to lie down. The chest was examined with more care; no abnormal percussion at any part; the respiratory murmur every where abundantly audible, and natural; some coarse mucous râle in both backs, and a few dry bronchial râles elsewhere; the sounds over the region of the heart and of the great vessels strictly normal. The pulse was hurried, but natural in character.

He continued in much the same state till June 4th; paroxysms of extremely difficult breathing occurring three or four times a-day, and lasting generally from twenty to thirty minutes. He expectorated daily from six to eight ounces of frothy mucus, tinged of a distinct rusty colour, with streaks of purple. Repeated examination of the lungs, however, showed that they admitted air abundantly in every part, and were free from every physical sign of disease. The treatment was not altered; the operation of tracheotomy was proposed, and, though urged as a certain means of relief from the paroxysms, was objected to in the strongest terms by the patient, who said he would rather die than submit to it.

On the morning of the 4th June, between 7 and 8 A.M., he had an attack of laryngeal suffocation of peculiar intensity, accompanied by dull pain in the lower part of the chest. He was seen by the resident clerk, and again refused the operation; he seemed to obtain relief by being supported in the erect position, and walking up and down the ward, friction being also applied to the front of the chest. Another paroxysm, not so severe, occurred in the afternoon. At half-past 7 P.M. there was a return of the paroxysm. He was seen at 8 o'clock, when he was livid and exhausted; he expressed a desire to be bled, and again refused to permit tracheotomy. At half-past 8 the dyspnœa was intense, the lividity of lips very great; the face generally pale; the skin covered with cold sweat. The operation was performed by the resident surgical clerk in attendance, but the patient was nearly asphyxiated before the tube was introduced. He continued after the operation to breathe slowly and at long intervals; the pulse continued perceptible for about ten minutes, but he did not rally, and died about a quarter of an hour after the tube was introduced. Artificial respiration was employed without effect. A small quantity of blood was lost during the operation, some of which entered the trachea, and was apparently expelled again with considerable force.

Post-mortem examination, 6th July.—The body unusually robust; post-mortem lividity considerable; rigor mortis well marked. No emaciation either of fat or muscle.

Pleuræ containing little fluid; adhesions at apex of right lung, corresponding to a few encysted cretaceous concretions, little larger than a barley-corn. A little emphysema in the anterior parts of both lungs, and slight collapse of the tissue posteriorly, otherwise they were healthy. The greatest bronchi had the mucous membrane slightly congested, and contained a considerable quantity of tough mucus and muco-purulent matter rather deeply tinged with blood; but nowhere any distinct coagula.

The heart weighed 12½ oz.; its muscular tissue much congested. On the aortic valves, which were quite competent, and not at all deformed, there were one or two very minute granulations, and a few similar ones on the inner membrane of the vessel near its origin. The other valves perfectly normal.

The thoracic aorta had its inner membrane throughout uneven and thickened, but with little distinct abnormal deposit. The arch presented no general dilatation; it was, however, slightly dilated upwards at the root of the innominata; and this vessel, as well as the origin of the right subclavian, was uniformly large relatively to the vessels on the opposite side. The two carotids were of equal size; but both of them, as well as the left subclavian, were very slightly expanded at their origin.

At the back part of the arch, half an inch below and between the origin of the innominata and left carotid, was an oval opening, through which a hazel nut might be passed lengthways. Its edges were tolerably smooth and rounded; and it was three-quarters occluded by a mass of firm granular coagulum, which passed from this opening into the aneurismal sac beyond. This was of the size of a walnut, and was situated between the aorta and the trachea, being adherent to the perichondrium of some of the tracheal rings; the sac was nearly full of laminated, decolorized coagula, with a little fluid blood.

The left recurrent nerve, emerging from below the aorta, passed immediately to the left of the sac, and rather behind it, being bent over it, and at one point almost imbedded in the thickened cellular tissue which surrounded it; at this point there were also one or two indurated lymphatic glands around the nerve, dark from carbonaceous deposit. The pneumogastric nerve on both sides, and the recurrent on the right, had their normal relations, excepting that the subclavian artery, where it was surrounded by the right recurrent, was, as before mentioned, somewhat dilated.

The tongue rather brown, and dry in front. Its root, and the fauces natural.

The epiglottis normal in size and form; its mucous membrane faintly rose-coloured on the posterior aspect, and displaying a somewhat granular surface, from prominence of the mucous follicles, especially in the neighbourhood of the arytænoid cartilages. Ventricles of larynx and vocal cords natural.

The cricoid cartilage and three upper tracheal rings divided by a perpendicular incision in the middle line.

The mucous membrane in the larynx and upper fourth of the trachea nearly natural in colour and appearance. Below this the mucous membrane presented rose-coloured vascularity, deepening towards the bifurcation, on the left side, into purple. The mucous membrane slightly granular throughout this injected part from hypertrophy of the follicles.

About an inch and a quarter above the bifurcation on the left side there was a circular opening, admitting readily a crow-quill, and

passing into the aneurismal sac before mentioned, which lay in contact with the outside of the costal cartilages.

Nearer the bifurcation there were three or four small points slightly elevated, and of an opaque yellowish colour, as if the mucous membrane were stretched over some abnormal deposit. The cartilages of the two tracheal rings immediately behind the opening were entirely separated from their perichondrium at the part opposite the aneurismal sac.

The abdominal viscera were congested as usual in asphyxiated persons, but had no other morbid appearance. The abdominal aorta was not so uneven internally as the thoracic, but presented more distinctly atheromatous opaque deposit in its inner membrane.

The first question which suggests itself in connection with this case is, *What was the cause of death?* On this point, I think, a consideration of the whole circumstances will leave no doubt that the patient died chiefly from laryngeal suffocation, induced by pressure of the sac on the recurrent nerve of the left side. The occurrence of suffocation from this cause is too well attested by numerous cases of aneurism and tumours of the chest now on record, to admit of reasonable doubt. The evidence adduced by Dr Hugh Ley upon this subject in his work on laryngismus stridulus, although certainly insufficient to establish his exclusive theory of that disease, is strongly confirmatory of the correctness of the views entertained nearly two centuries ago by Willis as to this source of death in some intra-thoracic tumours. The experiments of Legallois, and the far more elaborate and satisfactory ones of Dr John Reid, have demonstrated, in the most unquestionable manner, the production of laryngeal suffocation by various kinds of interference with the recurrent nerve on one or both sides of the neck. "From the experiments we have detailed," says Dr Reid, "it is apparent that severe dyspnœa, amounting to suffocation, may arise both from irritation and compression of the inferior laryngeal nerves, or the trunks of the pneumogastrics. For when both, or *even one recurrent nerve*, was irritated, the arytenoid cartilages were approximated, so as in some cases to shut completely the superior aperture of the glottis."[1] Section of the vagi, also, according to Dr J. Reid, produced "sudden and violent attacks of dyspnœa, which generally went off in the course of a very few minutes, when they did not terminate in suffocation;" leaving, however, the animals liable to renewed paroxysms on the occasion of a violent struggle, or any exertion tending to hurry the respiration. It is unnecessary to enter into the physiological details and principles connected with these curious results; it is sufficient for the present purpose to observe, that they fully explain the numerous cases recorded in pathological and practical works from the time of Bonetus, in which tumours involving these nerves (in the great

[1] Physiological, Anatomical, and Pathological Researches, p. 120. See also pp. 167 and 272.

majority of cases aneurismal) have been shown to produce death by sudden orthopnœa, often independently of any pressure directly on the air-tubes. Indeed it is worthy of remark, that spasmodic dyspnœa is a cause of death in a very considerable proportion of cases of aneurism of the aorta.[1] It is sufficient to refer, in illustration of this point, to the cases by Drs Graham and Alison, communicated to the Edinburgh Medico-Chirurgical Society in 1835,[2] in which aneurisms of the aorta were accompanied by marked laryngeal dyspnœa from this cause, in Dr Graham's case altogether simulating a primary laryngeal affection; to a similar case under the care of Dr Todd,[3] in which the recurrent nerve of the left side, and all the muscles to which it was distributed, had undergone atrophy from the pressure of the tumour; to the case of aneurism of the innominata, detailed by Mr Lawrence,[4] in which death took place from suffocation, tracheotomy being proposed but not performed; and to several examples of this form of dyspnœa detailed in a paper by Dr Henderson,[5] and in the work of Dr Ley before referred to.[6] I shall only say farther, that the violent paroxysms of dyspnœa experienced by my patient on many occasions before they were actually fatal, the highly stridulous respiration, the difficulty he evidently had in producing vocal sounds, and their altered character, pointed unquestionably to the glottis as the source of his danger; and the absence of any physical signs or morbid appearances indicating serious pressure of the aneurism on the trachea render it not probable that death can be ascribed to this cause. There remains only one other possible cause of suffocation, and to this I am willing to allow its due influence. The exhaustion consequent upon the numerous attacks of dyspnœa during the last days of life evidently told severely upon his strength; and the considerable and increasing quantity of mucus in the bronchi must have been expectorated far less freely in the last hours of his existence. The accumulation of this mucus, which was found in the larger tubes after death, fully explains why the operation of tracheotomy performed *in articulo mortis*, was followed by so imperfect a result. It is worthy, however, of remark, that up to the last visit which I paid him, the evidence of obstruction in the bronchi continued to be not greater than in cases of very slight bronchitis, and fully warranted the idea, that the greater part of the mucus expectorated came from the upper part of the air-passages. This idea corresponded also with the morbid appearances in the dead body.

The muco-purulent matter found in the larger bronchi after death

[1] It may even be said that this symptom is rarely absent in those aneurisms which spring from the back part of the arch of the aorta. See Dr Greene's collection of cases of this kind in the Dublin Quarterly Journal, No. 3, new series; and Mr Crisp's table of aneurisms, Treatise on the Blood-vessels, p. 235; for numerous instances bearing on this point.

[2] Edin. Med. and Surg. Journal, vol. 43, p. 292, *et seq.*

[3] Lancet, June 1841, p. 400.

[4] Medico-Chirurgical Transactions, vol. vi.

[5] Monthly Journal of Med. Science, 1841, p. 10.

[6] On Laryngismus Stridulus, &c., p. 453, *et seq.*

was considerably more tinged with blood than that expectorated at any period during his fatal illness. But it is very doubtful whether this increased hemorrhage was from the sac; as I am told that a good deal of blood was drawn into the trachea during the operation. At all events, it is clear that hemorrhage was not connected with the fatal event, nor did it ever form a serious complication,—never amounting to more than was sufficient to give a purplish, and often only a rusty tinge to the expectoration.

With this absence of material hemorrhage, it is important to ask, *how long the communication of the sac with the trachea had existed before death?* The lungs having presented no symptom of disease, and being found after death free of all serious lesion, it is difficult to suppose that even the small quantity of blood in the sputa had any other source than the aneurism; and yet we have evidence that, if this be so, the opening must have continued for months, yielding only these small quantities, as the patient distinctly stated that he had at no time coughed up clots of blood. When we consider the nearly complete occlusion of the sac by coagula, this phenomenon will appear less difficult to understand; at all events, it is far from rare in the histories of aneurisms opening on mucous surfaces, and especially into the air-passages, to find, even after one serious hemorrhage has given evidence of a rupture of the sac, that the hemorrhage reduces itself to a very trifling amount, and sometimes is altogether suspended. The case of Mr Liston at once suggests itself as an illustration of this fact. In the only record published of the fatal illness of this distinguished member of our profession, it appears that the first hemorrhage was followed by a period of exemption from symptoms, and that when these recurred, it was in the form of a cough attended with expectoration, which was "difficult, small in quantity, and of a rusty colour;" no further material hemorrhage occurring till his death, which was from orthopnœa.[1] In the remarkable case of abdominal aneurism which I read to the Society last year,[2] it is more difficult, owing to the situation of the first opening into the duodenum, to judge of the amount of blood that may at different times have been ejected, and it is highly probable that some bleedings may have taken place unobserved; but it seems in every way probable that no considerable hemorrhage occurred during twenty-two months, from an aperture which had evacuated gallons of blood in a few days, and which was found very nearly, if not entirely, sealed up after death.

Could the aneurism have been discovered during life? On this point we have the following data;—no dull percussion, abnormal pulsation, or tremor at the upper sternum; no abnormal sound over the heart and great vessels in front or behind; normal and symmetrical percussion over the lungs in every part; no abnormal respiratory sound over the trachea in front or at the root of the lung behind; abundant and symmetrical respiratory murmur in both lungs and over every part of them, mixed with slight mucous râles behind, and a very little sonorous râle in front. These physical signs, in regard to which full and careful examination may be relied on, form the elements of a tolerably complete negative diagnosis of aneurism, the suspicion of which was certainly entertained at the second examination of the chest, but soon dismissed, the case being treated as one of laryngeal affection. Perhaps it is still doubtful if more than a bare guess could have been formed under the circumstances; but the event showed that the suspicion thus negatived by physical diagnosis was allowed too hastily to be driven from the mind by the apparently greater probability of an ulcerative lesion of the larynx and trachea; and during the short period he was under my care no time was given for considering the case in all possible points of view, especially as the indications of practice appeared sufficiently distinct. After a careful consideration of the diseased parts, however, I am led to think it probable that while no sure sign of aneurism of the aorta could have been made out, a very close and accurate examination at the root of the neck, and in the course of the arteries, *might possibly* have discovered the dilatation of the innominata and subclavian vessels. Circumstances which did not come to my knowledge till after the patient's death were also calculated to arouse suspicion. The long persistence even of a trifling amount of blood in the expectoration would certainly have justified the belief in something more than an ordinary ulceration in the larynx; and a pain which he is said to have suffered at one time at the upper part of the sternum, but which was not complained of during the last part of his illness, would have confirmed the diagnosis of some fault in the thoracic region. The absence of any marked tenderness on pressure over the larynx, and of swelling of the epiglottis, was calculated to attract, and did attract, attention from the first; but this negative circumstance was considered as outweighed by the rest of the evidence.

It is worth while to remark, although it is difficult to obtain any accurate data on the subject, that the combination of symptoms presented by this case may probably be expected not unfrequently to occur in chronic or acute ulceration of the laryngeal mucous membrane. Local pain and tenderness are by no means of constant occurrence in these cases; neither can alterations of the epiglottis and upper vocal cords be recognized in all cases, though some kind of local symptom will doubtless be accessible in the great majority. On the other hand, the presence of blood in the sputa, though of course a suspicious circumstance when long continued, is neither universally present in aneurism, nor always absent in laryngeal ulceration. I have lately seen a case almost precisely parallel to this one in every important feature, in which paroxysmal laryngeal dypsnœa, apparently very little under the influence of remedies, and accompanied for a considerable period by blood in the expectoration, is probably due to a primary laryngeal affection, of which the local

[1] Lancet, December 11th, 1847.

[2] Aneurism of the Superior Mesenteric artery opening into the duodenum, twenty-two months before death.—*Monthly Journal*, vol. x. 1850, p. 82.

symptoms have lately become more distinct, while physical signs of aneurism remain, after repeated and careful examination, undiscoverable. If this man remains under observation for a sufficient length of time, it is probable that a more secure diagnosis may be formed; but at first it would have been impossible to act on an assured conviction either of thoracic or laryngeal disease, while the state of the patient has been, and continues such, as may render a recourse to tracheotomy an extremely necessary expedient for his security, or even his rescue from impending death.[1]

In reference to diagnosis, the practical conclusions which follow from the above remarks are no less evident than important. The mistake of an intra-thoracic tumour for a laryngeal affection is one of those accidents which has probably occurred in practice far more frequently than it has been accurately recorded; although a sufficient number of instances have been published to show that it may readily occur in the most careful hands, in the absence of stethoscopic examination. It cannot, therefore, be too strongly insisted on, that a physical examination of the chest should take place in all cases of supposed laryngeal disease. This is indeed an invariable rule with all careful practitioners, on many grounds; although it may be doubted whether the lungs and air-passages do not often too exclusively absorb attention in such examinations. But the present case, while it proves still more strongly that no amount of caution in the examination of the chest, and especially of the great vessels, is superfluous, also shows, I think, conclusively, that the absence of the physical signs of aneurism or tumour should not suffice to remove completely the suspicion that they may be concerned in the

[1] Since the above was published in the Monthly Journal, this case has terminated fatally by profuse haemoptysis and consequent suffocation; the source of the symptoms having been shown on dissection to be an aneurism, arising at the back part of the innominate artery, and bursting into the lower third of the trachea. The aneurism pressed on the right recurrent nerve, which was flattened and involved in the sac, and the internal muscles of the larynx on the right side were slightly atrophied; the mucous membrane of the larynx, as in the case at the beginning of this paper, being nearly normal. The branches of the innominata were normal; the arch of the aorta not dilated; the heart 9½ ounces in weight, normal. The sac was nearly full of coagulum; and this circumstance, with its deep situation and projection backwards, must account for the absence of appreciable pulsation at the root of the neck, and of all the other physical signs, to which the expansion of a sac, the size of a large date, communicating with nearly the entire length of the innominata, would assuredly have given rise under any other conditions. The case forms a most instructive addition to the facts recorded in this and other papers on the diagnosis of aneurism. I may therefore state, that although not recorded in detail during life, it was most sedulously and accurately examined by three physicians, under the express suspicion of aneurism, and that every known physical sign of that disease was sought for in vain. The patient was likewise seen by a fourth physician, not of Edinburgh, but of large special experience in regard to affections of the throat and larynx, who unhesitatingly pronounced the epiglottis and neighbouring parts ulcerated and thickened,—a diagnosis which I am by no means prepared to justify, but which, with some slight redness of the mucous membrane, plainly enough discernible to every one concerned, gave a bias to the opinion expressed in the text.

affection of the larynx. It is obvious that the part of the aorta most apt to be affected in these cases is the middle or transverse portion of the arch, and particularly its posterior or inferior surface, where it is most removed from the possibility of physical diagnosis. It is also evident that a very small tumour in these situations is enough to give rise to all the symptoms of laryngeal obstruction. Dr Todd correctly remarked, in his clinical lecture upon the case above alluded to as having occurred under his care, that "most observers had attributed these symptoms (those of chronic laryngeal affection) to compression of the trachea and bronchi, and had overlooked the condition of the recurrent nerve." In the present case, as in that of Dr Todd, "there was the most ample evidence that the pressure upon that nerve occasioned the laryngeal distress."

With regard to the treatment of such cases, the present narrative seems also not devoid of instruction. Had an aneurism been discovered or strongly suspected in this case, it seems probable that general blood-letting, together with such remedies as would have contributed to control the heart's action, might have been pursued farther with advantage to the patient; whereas the chronic nature of the supposed laryngeal affection, and the active treatment to which he had already been subjected, were accepted as sufficient reasons for foregoing these remedies and trusting to blistering, ipecacuan, and the performance of tracheotomy. This operation was absolutely refused by the patient while he had sense and vigour, and the case accordingly adds one more to those in which the operation was performed too late to be of any service; but I think it is impossible not to admit that it would probably have prolonged life had it been performed at any period before the final agony, and that the patient's sufferings throughout his illness would have been greatly less severe had advantage been taken of one of the earliest threatening paroxysms of dyspnoea to place a tube in the trachea. In a clearly ascertained case of aortic aneurism, such a proceeding could of course only be proposed as a temporary relief from immediate and pressing danger; and even in this point of view it could only be prudently recommended after careful examination had ascertained the freedom of the lung and of the air-tubes from any considerable pressure; but under these circumstances, I should certainly not hesitate in offering to the patient the benefit, even though temporary, which this little operation is calculated to afford. Much less should I feel justified in withholding it, on the ground of the uncertainty of diagnosis, in cases like the present, where an obvious laryngeal spasm exists, the source of which cannot be discovered, but which is unconnected with any other ascertainable affection of the respiratory passages. For further remarks upon this subject, however, I beg to refer the reader to the following report of the debate on this paper in the Medico-Chirurgical Society.

CASE OF ANEURISM OF THE THORACIC AORTA, WITH OBSERVATIONS ON THE PROPRIETY OF PERFORMING TRACHEOTOMY IN SOME CASES OF ANEURISM.

Dr W. T. Gairdner read a case of aortic aneurism, which presented the following peculiarities:—The sac, about the size of a walnut, arose from the back part of the arch, which was but slightly dilated; the tumour pressed upon the left recurrent nerve, and had in this way determined frequent attacks of orthopnœa, with laryngeal obstruction; at the same time that there was no evidence nor appearance of serious pressure upon the air-passages. The aneurism had opened into the trachea, but without important hemorrhage; nothing but a tinge of the expectoration had resulted, the sac being full of coagulum. There was no physical sign of the presence of a tumour, and the aneurism remained undiscovered till the patient's death, which occurred, not from hemorrhage, but from spasmodic suffocation. Tracheotomy was projected from the first, and repeatedly urged, but the patient would not submit to it until he was moribund, when accumulation in the bronchi had taken place to such an extent as to render it unavailing.

Dr Gairdner discussed several points in the pathology and diagnosis of this case, and concluded by observing that the state of the parts as found after death, fully justified the attempt to relieve the patient of his distressing symptoms by tracheotomy. He argued that there was distinct evidence of the frequent termination of aneurisms in this situation by spasmodic laryngeal suffocation; and under such circumstances, if it was ascertained that the lower air-passages were tolerably free from impediment, he thought that tracheotomy was not only justifiable, but imperatively demanded as a means of prolonging life. That the operation of tracheotomy might have the effect of prolonging existence, even in aneurisms, more advanced, and circumstances far more desperate than the present, was proved by a case recorded in the Lancet for 1844, in which Mr Judd performed it upon a patient in articulo mortis, with the effect of prolonging life thirteen days, although a large aneurism of the aorta had been pressing upon, and had burst into the trachea, causing hemorrhage to the extent of thirteen ounces. And although few would be inclined to covet an opportunity of following this practice under the like circumstances, the case was instructive as pointing out how much might be done for the relief of those in whom the symptoms were chiefly or exclusively laryngeal. At all events, as the propriety of tracheotomy in aneurism was not, so far as he knew, formally recognised by any authority, and as in several well known works it appeared to be discouraged, or even viewed as unjustifiable, Dr G. had thought it his duty to lay the subject before the Society, with the view of eliciting the opinions of members present.

Dr Seller thought that the presence of an aneurism should in general counter-indicate the operation. In this particular case, however, had Dr G. been allowed to act at an earlier period, the life of the patient would, in all probability, have been prolonged. But he repeated his conviction that tracheotomy, though justifiable in this case, was not applicable in cases of aneurism in general.

Drs Keiller and *Andrew*, who had had the care of the patient before Dr Gairdner took charge of the case, had detected no physical sign of aneurism, and quite concurred in the propriety of performing tracheotomy.

Professor Miller concurred in the main with the statements of Dr Gairdner, and thought that he had laid an important as well as interesting communication before the Society. Cases of aortic aneurism, with reference to the performance of tracheotomy, seemed to be divisible into two classes,—1. Those wherein the tumour was so circumstanced in situation and bulk as to compress the air-passage, narrowing its calibre, and producing a permanent obstruction to breathing. In those the dyspnœa would be continuous; liable, it may be, to aggravations and remissions, but never wholly absent; and, in most examples also, diagnosis both as to the existence of aneurism and of such mechanical interference with the air-passage, would not be very difficult to the experienced and careful observer. In such cases he would be inclined to refrain from operation, anticipating little or no benefit from it, even of a temporary kind; and fearful lest, by obvious failure, the

department of operative surgery should be brought into discredit. Into this class the melancholy case of the late Mr Liston, alluded to by Dr G., evidently came; the windpipe compressed, the breathing continuously laborious, and no likelihood of satisfactory relief from artificial opening of the windpipe.

The case quoted by Dr G. from the Lancet, might seem to oppose this opinion. But he (Professor Miller) felt inclined to regard that case as quite an exceptional one. The man could breathe only through a small tube or catheter thrust down from the wound past the aneurismal swelling; and that tube—so small, so liable to displacement, so liable to obstruction, so certain to cause advance of ulceration in the part of the tumour on which it lay—seemed to him too miserable and slender a tenure of existence to warrant its being had recourse to with any hope of success. It was only surprising that through such a precarious access to the lungs, the man continued to live so long.

2. The second class included those cases in which the tumour, not so large as to compress and narrow the air-passage, is yet so circumstanced as to affect the larynx dangerously; as in Dr Gairdner's case, irritating or compressing the recurrent branch of the eighth nerve, so as to excite paroxysmal attacks of intense laryngeal dyspnœa, similar in their nature to those of laryngismus stridulus. These tumours, from their small size and deep locality, would be difficult of detection; and yet there were means, too, as Dr G. had shown, both positive and negative, by which their existence might be at least strongly suspected. And, under such circumstances, he (Professor M.) would have no hesitation in saying, as his own belief, that—provided the paroxysms of dyspnœa were such as to threaten instant extinction of life—he would have recourse to tracheotomy at a comparatively early period of the case, not waiting till the patient was in *articulo mortis;* and for the following reasons:—The operation in itself was not formidable, not difficult of performance, and not dangerous to life. By having recourse to it, he would relieve the patient at once from intense agony and suffering. Besides, at comparatively little cost, he would prolong existence—for hours, weeks, or months; and all well knew how vastly important even a comparatively short period of reprieve might be found to be under such circumstances. Further, it was not wholly utopian to suppose that such relief and prolongation of life might, in some rare cases, contribute towards the actual occurrence of spontaneous cure. In listening to the details of the post-mortem examination in Dr Gairdner's case, one could not fail to be struck with the near approach made in that case to cure. The small aneurism was almost entirely filled with clot, and the oval aperture by which it communicated with the aorta was almost wholly obstructed by a mass of fibrin. Very little more might have silenced the tumour. And that little might have been greatly contributed to by the relief of respiration afforded by tracheotomy timeously performed; while, on the other hand, the oft-recurring attacks of dyspnœa, with the attendant excitement and distress of the patient, could not fail not only to oppose all attempts at spontaneous cure, but also to hurry on the fatal advance of the tumour.

Hitherto his creed had been—(he had taught it in his class, and published it in his System of Surgery)—in regard to the performance of tracheotomy in connection with aortic aneurism, that the operation should not be had recourse to, the cause of the suffering being apparently beyond relief by tracheal wound. But now he was bound to say, that he saw reason to alter that opinion, and to hold that, while the rule of non-interference still held good in the first class of cases which he had specified, in the second there was not only a warrant but a call to operate, with the hope of certainly relieving suffering and prolonging life, and with the chance of even contributing towards the occurrence of spontaneous cure. There might be a third class of cases (as suggested by Dr Gairdner) in which pressure on the windpipe was slight, but yet with a certain degree of contraction of its calibre, and in which *paroxysmal* laryngeal dyspnœa was also urgent. In such circumstances he would hold that the latter part of the case, or major, would rule the former part, or minor; and that, therefore (more especially if urged to it by the patient), he would be inclined to operate, though, of course, with a less favourable prognosis than when no compression and contraction of the windpipe existed.

On the whole, he considered that Dr Gairdner had made an important contribution to practical surgery; extending its operative interference to a class of cases—few in number, doubtless, and not holding out much hope of permanent benefit—hitherto supposed by many to be excluded from its range. It was the province and duty of the surgeon, as well as the physician, to palliate by every reasonable means where he cannot cure; and if unable to save life, at least to protract and soothe its closing day.

After some remarks by Dr D. Maclagan,

Dr W. T. Gairdner said that he had alluded to Mr Liston's case as conclusively showing that an aneurism of the aortic arch might remain without physical signs, or at all events, might present an extremely obscure diagnosis, up to a period when it had threatened life by one or other of its secondary accidents. The present case, as well as others, proved that spasmodic dyspnœa, a frequent cause of death in aneurism in this situation, might occur and prove fatal while the air-passages were exempt from pressure; and while the larynx, involved through its nerves, was the sole cause of dangerous symptoms. In such a case, the practitioner was surely not warranted in making the probable or certain fatality of the disease an excuse for non-interference, especially when the operation was one not in itself dangerous, and almost certain to secure to the patient not only a comparative immunity from distressing symptoms, but a longer lease of life and a slower progress of his disease. Nothing was more likely to accelerate the increase and fatal result of an aneurismal tumour, than the violent inspiratory and other effects which accompanied the paroxysms of stridulous breathing in these cases; and if these paroxysms could be entirely removed in many cases of aneurism by so simple a proceeding as tracheotomy, it would surely be allowed that this operation presented a fair claim to be admitted into the legitimate treatment of such cases; and to be resorted to, not unwillingly and as a last resource, but *as early as it could be ascertained that laryngeal symptoms were the source of the more immediate danger*. No doubt the operation became less and less eligible in proportion as *other* sources of danger became imminent; but so long as these did not reduce the laryngeal symptoms to a manifestly secondary importance, Dr G. did not feel that he would be justified in refusing to a *willing* patient, *after due explanation*, the chance of life and comparative comfort for an additional day or hour. The amount of pressure of an aneurism on the air-passages could always be estimated by careful stethoscopic examination with considerable precision; and though other sources of danger might remain unknown, this fact ought not to be made an argument against interference for the removal of the palpable and imminent hazard. And if these rules of conduct were sound when an aneurism was *known* to exist, they were all the more evidently so in the case where such a tumour was only suspected, or where, as in the present case, the suspicion had been allowed on the strength of negative evidence, to drop too hastily from the mind.

An authority so justly and so universally known as Dr Watson, had stated that no man with a stethoscope in his hand, and the power to use it properly, ought to be seduced into the error of performing tracheotomy in a case of aortic aneurism. If the principles adopted by him (Dr G.) were correct, there was here an opinion extremely calculated to mislead; and no doubt there were many physicians who would be alarmed at the idea of opening the trachea, not for a laryngeal, but for a thoracic disease. For his own part, Dr G. had no desire to screen himself from criticism, if it was thought that by any reasonable precaution, a correct and secure diagnosis might have been formed in the present case; but on general, and not on personal, grounds, he thought that a different bias ought to be given to the practical precepts current on this subject, and that practitioners should neither be taught to be too sure of the diagnosis of thoracic aneurisms, nor too apprehensive of the performance of tracheotomy.

MURRAY AND GIBB, PRINTERS, EDINBURGH.

ON

THE THREATENINGS OF APOPLEXY AND PARALYSIS; ETC.

BEING

THE CROONIAN LECTURES DELIVERED AT THE ROYAL COLLEGE OF PHYSICIANS IN MARCH, MDCCCLI.

"In these facts we have the *Proof* that a slight degree of contraction of the muscles of the neck, induced by the electric current, induces, in its turn, heightened colour of the face, of a florid hue; and that a greater degree of that contraction induces a deeper colour of the face, the lips and angles of the mouth being livid, and the eyes suffused, with confusion of thought, headache, dimness of sight, alternating with flashes of light; these latter remaining for a few minutes after the cessation of the current, and then disappearing. They present the *Demonstration* of the nature both of trachelismus and phlebismus, and of their *effects*."—§ 107; compare § 232 and § 300.

ON

THE THREATENINGS OF APOPLEXY

AND PARALYSIS;

INORGANIC EPILEPSY;

SPINAL SYNCOPE; HIDDEN SEIZURES;

THE RESULTANT MANIA; ETC.

BY

MARSHALL HALL, M.D. F.R.S.

L. AND E.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS;

FOREIGN ASSOCIATE OF THE NATIONAL ACADEMY OF MEDICINE OF FRANCE;

ETC. ETC. ETC.

LONDON:

LONGMAN, BROWN, GREEN, AND LONGMAN, PATERNOSTER ROW.

MDCCCLI.

TO

HENRY SMITH, ESQ.

LATELY OF TORRINGTON SQUARE,

MEMBER OF THE ROYAL COLLEGE OF SURGEONS;

ETC. ETC.

IN GRATEFUL REMEMBRANCE OF

HIS DISINTERESTED, UNTIRING, AND INVALUABLE

ASSISTANCE,

THIS LITTLE VOLUME IS INSCRIBED,

BY HIS ATTACHED FRIEND,

THE AUTHOR.

38, *Grosvenor Street, March* 20, 1851.

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ADVERTISEMENT.

THE following pages contain a very brief and imperfect outline of a most momentous subject—that of the *principles* on which all *prevention* of the seizures about to be noticed depend. And, in these cases, prevention is the all-important object of the physician.

These seizures, when they have once occurred, are so apt to recur, the susceptibility to recurrence is so difficult to remove, the *effects* of attacks are so dire, whether we regard mind or limb, that *the one* object of the patient and of the physician must be to watch the dawn, as it were, of the malady, and adopt with energy and constancy every means of obviating such a calamity as a first or second seizure.

With this view, no premonitory sign should be neglected, however apparently slight. A flush, a sense of constriction about the throat, a momentary vertigo, a momentary loss of feeling or of power about the lips or the fingers,—should strike us with such terror as may, at least, awaken our utmost attention. This is the occasion for the maxim—'venienti occurrite morbo.' The best physician is he who *watches* his patient most carefully. The wisest patient is he

who submits—for *the rest of his life*, it may be—to his physician's injunctions, asking, not—'how little may I do?' but—'how much can I do?'—in my perilous case.

The regulation of the ingesta and of the egesta, of the occupations and emotions of the mind, of the exercises of the body, of the sleep especially, of the posture during sleep, of the circulation in the head and in the hands and feet,—these and many others are topics never more to be forgotten by the threatened patient.

It is not mere *doses* of medicine, which may indeed ward off an attack for the moment, but mild, yet efficient, *courses* of medicine, to which we must trust.

The *Emotions* and the *Irritations* are so frequently associated with undue secretion of gastric acid, that I cannot sufficiently recommend the due administration of antacids. With these, stomachic aperients, alterative mercurials, frequently gentle tonics, and especially, I think, the spinal tonic, strychnia, and every plan for the improvement of the general health,—the shower-bath, change of air and scene, but especially travelling,—must be combined.

The physician and the patient should be prepared for the recurrence of the threatening, or of the actual seizure,—and provided with the due and energetic means and instant remedies proper for the emergency.

In a word, the strictest regimen must be enjoined, with the view of prevention, and the promptest remedies in the case of threatening or of seizure.

The *means* of prevention are suggested entirely by the *pathology*. In *all* the affections treated of in these Lectures, certain causes and principles, emotions and irritations, act directly or diastaltically upon the muscles of *The Neck*, inducing what I have ventured to designate *Trachelismus*. If this *spasm* can be dissolved, all its *effects* cease, more or less perfectly. How important then is this view of the subject! I think that spasm *is* dissolved by an antacid emetic and antacid aperients.

In this manner we are enabled, I believe, in many instances, to prevent attacks of apoplexy, of paralysis, of epilepsy, and even of mania! Surely this is an important result.

And this result is the more interesting to myself, because it has flowed directly from a physiological principle, which has been both evolved and applied by my own labours. I would draw especial attention to the *Synoptical View* given at page 35.

The application of *Physiology* to *Pathology*, to *Diagnosis*, to the establishment of a *Class* of Diseases, and to their *Prevention* and *Treatment*, had never, I imagine, been so made before.

Man lives a life of Emotion. No moment of that life is passed in absolute tranquillity of mind. Every emotion has its influence on every muscle of his frame. It is written on the countenance, on the posture, on the very hands. The muscles of the neck do not escape; grief and anger choke; shame and indigna-

tion flush the face and neck. But what we term *expression*, as it affects the neck, is the first stage of trachelismus; and blushing and flushing are forms of phlebismus. Extremes of these become cerebral or spinal seizures.

Similar remarks may be made with regard to the Irritations. Errors in diet and deranged states of the alimentary canal, and excited conditions of the uterine system, are causes of disease to which mankind is most subject in a state of civilisation. I have explained the manner in which these and other causes of irritation act diastaltically on the neck, its nerves, its muscles, and its veins.

I beg, once for all, to state that I have excluded from the following pages, as from the Lectures themselves, all that did not bear on my argument. I propose in due time to put my subject into a more *systematic* form. The present state of the inquiry will be at once known by adding to this little work my *Synopsis of the Diastaltic Nervous System*, published a year ago.

ON

THE THREATENINGS OF APOPLEXY AND PARALYSIS; ETC.

LECTURE I.

PRELIMINARY FACTS AND OBSERVATIONS.

GENTLEMEN,

1. In the Croonian Lectures of last year, I had the honor of bringing before the College the subject of *The Spinal or Diastaltic Nervous System*, in its relation to Anatomy, Physiology, Diagnosis, Pathology, Therapeutics, and Obstetrics. I purpose, on the present occasion, to treat of a branch of that System, in its relation and application to the pathology of a peculiar Class of the Diseases of the Nervous System.

2. The physician is frequently summoned to cases in which, with or without an actual seizure, there is the *Threatening* of an attack—of Apoplexy, of Paralysis, of Epilepsy, or of Syncope. It is to these *Threatenings*, to these *Minæ*, according to the expression of

Heberden,—affections which equally alarm the patient, the patient's friends, and the physician,—that I beg to call your most serious attention.

3. On the occasion of excitement or emotion, or of gastric irritation, or in the midst of the most usual occupations, the patient is seized with vertigo, or a momentary oblivium or delirium, or various affections of the senses, or loss of muscular power, especially of the speech, of the hand, or of one side, and flushes, or turns pale, with intense alarm for fear of an apoplectic or paralytic seizure;—or the eyes and head may become suddenly fixed, the pupils dilated, the countenance flushed, with obvious loss of consciousness, and there is the threatening of epilepsy.

4. These *minæ* may last for a minute or two, and subside. But the patient is evidently in danger of a 'fit,' or seizure, and that of an apoplectic, paralytic, epileptic, or syncopal character; or such a seizure may actually take place.

5. What are the hidden springs of action which have been called into play in these various circumstances? What is the rationale, what the modes and means of prevention?

6. Some of these affections are of the slightest kind—"nihil aliud æger sentit præter oblivium quoddam et delirium adeo breve, ut ferè ad se redeat, priusquam ab adstantibus animadvatatur*." It is their

* Heberdeni Commentarii, ed. 1807, p. 139.

obvious and fearful *tendency* which gives them importance.

7. I have known mere *blushing* to become intense, constantly recurrent on every slight occasion, and attended by varied mental distress, and even to pass into an epileptoid affection; just as we have all known the flush of anger or indignation to pass into apoplexy. Mere 'sick-headache' sometimes passes into an affection of a far more formidable character. One patient experienced a sense of 'tightness' about the throat, with flushing, and the dread, and danger too, of some seizure, after taking indigestible food.

8. In one gentleman, sudden attacks of loss of speech, or of the power of the hand to write, took place from time to time, at varied and rather distant intervals, for ten or twelve years, leaving, at length, permanent inability to speak distinctly, or to retain the saliva perfectly, and a degree of paralytic weakness of one side. Ultimately an attack was attended by a degree of stupor and stertor; he was "quite unconscious to what was passing around him for ten or fifteen minutes, with loud snoring breathing, and then imperfectly conscious; and again, the next day, the mind seemed to wander at intervals, becoming, however, afterwards perfectly clear and composed."

9. Epilepsy itself could not be more paroxysmal.

10. Another gentleman became liable to attacks of loss of the power of articulation, so that he was compelled to *point* to the objects he required. At one

time he was seized with loss of the power of writing; at another, with transient hemiplegia. At length the inability to write occurred from the mere flurry occasioned by the loss of his spectacles at a moment when it was his duty to sign some official papers The next day he threw up his office,—and, a day or two afterwards, he committed suicide!

11. These are examples of what I have ventured to designate *paroxysmal* apoplectic and paralytic affections. Of epilepsy, as a paroxysmal affection, I need not now speak. But sometimes these seizures, instead of being apoplectic, or paralytic, or epileptoid, are *syncopal* in their external form and character. With or without previous flushing, the patient may become pale and faint, and exclaim—'I am dying!'

12. In some instances, again, these seizures take place *unobserved*,—in the night,—or in the absence of friends; and the effects and results of such *hidden* seizures are of the most puzzling character, until the occurrence of those seizures is detected, or at least suspected. These effects may be—a degree of stupor, of loss of memory, or of delirium; or actual *Mania*, or amentia!

13. One such case I shall hereafter lay before you in all its deeply interesting details. Obscure, and indeed not to be understood, until the fact of hidden seizures was discovered, all was made plain when that discovery was made.

14. Before I proceed, I must beg to be permitted to recall to your recollection the following extracts from the no less practical than classical work of Heberden:

15. "Invadente *apoplexia* aut *paralysi*, continuo laxare opertet omnes istas vestium partes, quæ *collum* cingunt; *id* enim nonnunquam his morbis advenientibus adeo *tumet*, ut ab arctiore quovis vinculo strangulationis periculum instaret." And—

16. "Instante accessione *epileptica* diligenter providendum est, ut omnes illæ vestium partes, quæ *collum* cingunt, quamprimum laxentur; *hoc* enim interdum adeo *tumet*, ut strangulationis metus impendeat." And again—

17. "Plerique *capitis dolores* vacant periculo; ubi autem ad hoc accedant stupor, aut *colli universi tumor*, aut mentis alienatio, aut distentiones membrorum, res ægri nequaquam in tuto sunt; hujusmodi enim mala subsecutæ sunt epilepsiæ, paralyses, et apoplexiæ*."

18. Abercrombie too notices—"the flushing of the face, turgidity of the features, throbbing of the external vessels, and other appearances, which have been referred to the doctrine of determination of blood to the head;" and adds, "numerous writers have remarked the unusual quantity of blood which is discharged from the integuments in opening the heads of

* Op. cit. pp. 299; 144; 86.

persons who have died of apoplexy. In some of Dr. Cheyne's dissections, upwards of a pound was collected in this manner." And again—"The remarkable turgidity of the features and the neck, which often occurs in apoplectic cases, must indeed be familiar to every one; and I think it appears to be most remarkable where the disease has proved rapidly fatal, without any means having been employed. A gentleman, whom I saw with Mr. Whyte, after some symptoms shewing an apoplectic tendency, was one morning found dead in bed, his body being scarcely cold. *His head and features were of a deep* PURPLE *colour, and* TURGID *in a most uncommon degree;* but no turgidity was observed in the vessels of the brain*."

19. To these extracts I must add the following observations from the same admirable work:

20. That writer observes—"The apoplectic attack is a sudden deprivation of sense and motion," "the face being generally flushed, and the breathing stertorous. In further tracing the history of such an attack, the following circumstances deserve our particular attention:—

21. "I. In many cases the patient speedily and perfectly recovers.

22. "II. In many cases the disease is speedily fatal; and we find, on inspection, extensive extravasation of blood.

* On the Brain and Spinal Cord; ed. 3; p. 303.

23. "III. In other cases, which are fatal, usually after a longer interval, we find only serous effusion, often in no great quantity.

24. "IV. In many fatal cases, no morbid appearances can be detected, after the most careful examination.

25. "Thus," he adds, "the phenomena of the disease appear fully to establish the important fact, that there is a modification of *apoplexy*, depending upon a cause of a temporary nature, without any real injury done to the substance of the brain; that the condition upon which this attack depends, may be removed almost as speedily as it was induced; and that it may be fatal without leaving any morbid appearance in the brain*."

26. The same remarks, according to the same authority, apply to *paralysis*:—

27. "The attack may, under proper treatment, pass off speedily and entirely, leaving, after a very short time, no trace of its existence."

28. "Many of the cases seem to bear a close analogy to simple apoplexy; and, when they are fatal, present either no satisfactory appearance, or only serous effusion, often in small quantity."

29. "The whole phenomena of *palsy* do indeed bear evidence that certain cases of it depend upon a cause which is of a temporary nature, and capable of being speedily and entirely removed. We see hemi-

* Op. cit. p. 205.

plegia take place in the highest degree, and yet rapidly disappear," &c. *

30. These then are the important subjects of the present Lectures:

1. The paroxysmal form of certain apoplectic and paralytic, as well as epileptic, seizures;

2. The various degrees of lividity and tumidity of the integuments of the face and neck in these;

3. The frequent speedy and entire recovery from them; or the absence of morbid appearances in the cases which prove fatal.

31. I shall have further to advert to the *cerebral* form of some cases of epilepsy, to the *syncopal* form of other seizures, and to the possibility of such attacks being *hidden*, and their effects mysterious.

32. It is obvious that, after much consideration given to the subject, Abercrombie felt the want of some principle on which to explain the occurrence of attacks of what he designates simple apoplexy. He asks, at the close of his interesting chapter entitled "*Conjectures in regard to the Circulation in the Brain*," "Why is not apoplexy produced by every increase in the mass of the blood, and why is it not excited by every instance of intemperance, violent exercise, or strong mental emotion? Is there any provision by which the effects of these causes are averted in their daily occurrence, though, in a certain condition of the

* Op. cit. pp. 247; 249.

system, each of them may be capable of producing perfect apoplexy*?"

33. It is to *this* great question, hitherto left unsolved, that I hope to present the answer.

34. I hope to show that, whatever the *violence* of the *arterial* circulation, there is no danger, no tendency to morbid action, as long as there is no impediment to the return of blood along the *veins*;—that the idea of '*tendency*' or '*determination*' of blood *to* the head, is a fiction and a chimæra; and that the real state of things in the condition which has been so designated, is, in fact, its IMPEDED RETURN *from* the head;—that this impeded return of blood from the head is induced by a *spasmodic* action of the *muscles* of the neck on the *veins* of that region,—an action *evident* in a vast many instances, though *latent*, perhaps, and *to be inferred* from the similarity of its effects, in others. *See* § 106.

35. To these conditions of the muscles of *The Neck*, I venture to give the designation of *Trachelismus* (from τραχηλον, the neck). Its effect on the veins may be termed phlebismus. It is frequently to be *felt*, when it is not to be *seen*, on applying the *finger*. It is still more frequently to be traced and inferred by observing the *lividity* and *tumidity* of the integuments of the face and neck.

36. Having made these preliminary observations,

* Op. cit. p. 310.

I proceed to the more detailed discussion of my subject.

1.—*The Paroxysmal Form of this Class of Diseases.*

37. The first characterictic of the Class of diseases of which I am about to treat, is—*their Paroxysmal form.*

38. Simple apoplexy, simple paralysis, not less than epilepsy and spinal syncope, may occur, recede, and recur, promptly, repeatedly, at varied intervals. Trachelismus, with its effect, phlebismus, is, indeed, to paroxysmal apoplexy and paralysis, what laryngismus is to epilepsy. Both are equally spasmodic, and subject to the laws of spasmodic affection.

39. In the first instance, the remission or recovery from these seizures may be perfect. Afterwards, some permanent effect remains, and there may be a degree of inarticulateness of speech, a little tendency to the flow of saliva over the lip, or a little debility in the movements of an extremity; or the mental faculties, the power of attention, of apprehension, of memory, may be somewhat impaired,—and nothing more.

40. These effects are equally the result of apoplectic or of epileptic seizures, though more speedily of the former than of the latter.

41. The causes too of these two forms of disease of the nervous system are the same—and chiefly, mental emotion and gastric irritation.

42. The difference appears to be, that in one case the cerebrum, in the other the medulla oblongata, is, principally, affected.

43. This result may depend on the different susceptibilities of these different portions of the nervous system, or on the different channel through which the cause may operate, in different individuals.

44. In a third instance, that modification of action obtains, which leads to ghastly pallor and apparent syncope, frequently with sickness.

45. Indeed, this sickness frequently plays an important part in paroxysmal diseases, occurring, as it does, in its slightest form of 'sick-headache,' or of what may be termed 'sick-giddiness,' or in the form or in the course of an apoplectic seizure.

2.—*The Lividity and Tumidity in Paroxysmal Affections.*

46. After their paroxysmal form, lividity of the countenance, either with flushing and turgescence of the face and neck, or with pallor, is the most characteristic phenomenon of these seizures. How is this phenomenon produced?

47. Augmented flow of arterial blood, as in violent exercise, may induce vivid, florid flushing; but how different is the hue of this flushing from the *lividity* observed in the threatening of apoplexy and

epilepsy! Impeded return of venous blood, observed in the case of effort, as in lifting, induces a deeper flush, somewhat mingled with lividity, and much more nearly resembling, in its hue, the pathological flush of those diseases.

48. Nor could *tumidity* arise from undue impulse of arterial blood, unaccompanied by impediment in its ulterior course. But admit the existence of impeded return of the venous blood, and tumidity is the evident, the immediate, and the inevitable effect of distension of the blood-channels placed immediately between the last branches of the arteries and the first roots of the veins, and of the veins themselves.

49. The lividity and tumidity of the face and neck, observed in certain diseases of the heart, and of the lungs; the livid flush of anger, of efforts, of stooping, are scarcely to be distinguished from the lividity and tumidity of the apoplectic or epileptic seizure. In the former cases, the lividity and tumidity are distinctly owing to impeded *arrière* or venous circulation. What is their nature in the latter?—what their cause and rationale?

50. We have all observed the livid flush of anger, and of gastric load and irritation. We have all known that livid flush to *pass into* the apoplectic threatening or seizure, as we all know that the excitement of the comitia of the Roman forum was apt to give rise to the epileptic attack, whence its ancient designation of *morbus comitialis*.

51. But what is the rationale of this venous turgescence of the face and neck? How do emotion, gastric irritation, &c. act in inducing this singular effect?

52. This—this is the deeply interesting question to which I beg your attention. It is the reply to this question which, I believe, presents the *Key* to all the difficulties in regard to the nature,—source and origin,—of *paroxysmal* apoplexy, paralysis, epilepsy, &c. And thus I am led to treat of the immediate and principal subject of these Lectures.

3.—*On Trachelismus; or Contraction of the Muscles of the Neck.*

53. We are all familiar with the phrase—'choked with grief or with anger,' and we have all witnessed the blush of shame and the deep flush of anger; and I have already stated that I have distinctly traced mere intense blushing into epilepsy, and that the still more intense flush of anger has passed into apoplexy.

54. With this blush of shame and this flush of anger, there are frequently, and in proportion to their degree, a purple lividity and tumidity of the face and neck, and even of the upper part of the thorax.

55. I have *seen* the same flush of the countenance, whilst the patient has *felt* a degree of stricture of the throat, with the fear of some seizure, as the effects of an indigestible meal.

56. The emotion of disgust, and gastric irritation, frequently issue in actual sickness and vomiting, involving closure of the larynx, or laryngismus, a partial trachelismus.

57. In epilepsy, the state of the neck is obvious to the eye; the head is *fixed*, or there is torticollis; and there is the 'facies nigrescens*' of Heberden. In the threatening of apoplexy, there is the same livid or purpurescent hue of the face,—and the same paroxysmal character. Is it possible to doubt that, what is evident in the former affection, exists, though in a latent form, in the latter, the *effects* and the recurrent character of the affection being the same?

58. The lividity and tumidity are not to be explained by any hypothetical 'tendency' or 'determination' of blood to the head, as I have already stated, and as I shall show more at length hereafter. They can only arise from *impeded venous return*. It is presupposed that there is no disease of the heart or lung to induce impeded venous circulation; and it is to be remembered that the affection recurs in paroxysms, that, in epileptoid cases, it *is* to trachelismus that the phenomena are traced, and that even in the apoplectoid, there are, in some instances, sensations about the throat, of no equivocal character.

59. History informs us that violent emotion and gastric irritation may issue in apoplexy. How is this

* Op. cit. p. 139.

phenomenon to be explained—for we have ceased to be satisfied with the vague and unmeaning expression of *sympathy?* I believe that trachelismus intervenes as the connecting medium between the cause and its dire effects.

60. The occasional *sensation* of strangulation, the purpurescence and turgescence of the face and neck, the loss of consciousness, &c. the sudden accessions and recessions, or the paroxysmal form, of the affection,—such then are the evidence of trachelismus.

61. This trachelismus probably occurs in the more deeply seated muscles of the neck, and, according to the *degrees* or *kind* of impeded venous circulation, may lead to further *cerebral*, or *spinal* symptoms; whilst the external evidence of its operation in the condition of the face and neck, varies from similar causes.

62. I may now observe that the *first* stage of trachelismus is probably always *latent;* being *inferred* from the turgescence of the face and neck. The *second* is inferred from *cerebral* symptoms in some cases, and from *spinal* symptoms in others.

63. The importance of this view will be seen when I come to treat of the further pathology of paroxysmal apoplexy; but still more when I proceed to discuss the treatment. There has long been, for instance, a question as to the propriety and safety of administering emetics in apoplectic affections. It is evident to me that this question must be solved by determining the previous question as to the *Diagnosis*

between paroxysmal and therefore secondary apoplexy, and apoplexy arising from organic lesion of the cerebrum. In the former, the first effect of ipecacuanha, or of nausea, is to resolve the spasm of the neck and break the first link of the chain of disordered actions. In the latter, the expiratory efforts of vomiting might augment the lesion already sustained by the tissues within the encephalon.

64. It is an important question—how far the action of the muscles of the neck may be specific in different instances. Are the various phenomena of external blushing, or flushing, of the apoplectic seizure, and of the epileptic attack, the varied effect of the compression of the external and internal jugular and of the vertebral veins respectively? These questions must, I think, be answered in the affirmative. But the satisfactory *proofs* of these facts may still be wanting. The act of sickness—the effect of emotion, or of gastric irritation—is, however, perfectly specific and distinct.

65. That action of the muscles designated *expression*, takes place in the *neck* not less than in the face; and it is thus the first stage of trachelismus, as the blush of shame and the flush of anger are the first shades of phlebismus, and, if I may venture to say so, of paroxysmal nervous affection.

4.—*Of Sleep, and its Trachelismus and Phlebismus.*

66. There is another topic, which I must briefly notice, in connection with that of trachelismus. It is the condition of the muscles and veins of the neck during *Sleep*. As the orbicularis contracts on the principal of *tone*, and closes the eye-lids, so the muscles of the neck contract and compress the veins of this region —inducing slight turgescence of the countenance, vivid suffusion of the eyes, and a sub-apoplectic state of the cerebrum and medulla oblongata. It is a slight trachelismus, and frequently concurs with other causes in inducing the apoplectic and epileptic seizure.

67. For the following interesting observation I am indebted to Dr. W. Tyler Smith:

68. "The person I observed kept falling asleep and waking every few minutes. In the course of a long ride, I had opportunities of seeing that, when he became unconscious, the external jugulars became full and strongly marked, and that these disappeared on the instant of waking from his brief sleep. There did not seem to be any change in the respiration, or in the position of his body, sufficient to account for the distension of the jugulars."

69. It would be well if some accurate index and measure of this condition of the muscles and veins of the neck could be discovered. I imagine that a little instrument like that devised by Dr. R. Quain, and

termed by him the *Sthethomoter*, might be employed for this purpose, and that it would indicate both the trachelismus and the subsequent phlebismus. The *facts* themselves, generally speaking, are however sufficiently obvious to the observant eye.

70. The relation between sleep and the apoplectic or epileptic seizure is well known. It is scarcely less remarkable than that of emotion and irritation with these seizures.

71. On this subject Heberden observes—

72. "Somnus est imprimis necessarius ad renovandas vires animosque, labore et curis exhaustas; et tamen procul dubio hominem opportuniorem reddit omnibus illis affectibus, qui ex nervorum infirmitate oriri existimantur; in quibus quoque numeranda est *apoplexia*, quæ sæpe per quietem crescit, vel tum primum invadit. Illos itaque omnes qui in his morbis sunt, et cupiunt amoliri præsentia mala, vel futura præcavere, oportet abstinere a nimio somno: optimus ejus modus erit, qui minimus salva valetudine capi potest."

73. "Somnus distentionibus amicus est, ut et omnibus malis quæ ex nervorum affectibus oriuntur. Itaque hæ quoque noctu præcipue molestæ sunt. Alios invadunt in somnum labentes, alios expergiscentes, multos etiam dormientes excitant*.

* Op. cit. pp. 304; 353.

5.—*Turgescence of the Face and Neck, and of the Conjunctiva.*

74. To return to the subject of turgescence, I may observe that, under the influence of trachelismus, the veins, the arteries, and the intermediate blood-channels, become turgid, and the tissues of the face, head, neck, and eye, are suffused, and assume, in a greater or less degree, the hue imparted by venous blood. The veins on the forehead are enlarged, the temporal arteries become tense and throbbing.

75. The degree of these appearances marks the degree of impediment to the return of venous blood.

76. In many cases, of some duration, the conjunctiva becomes of a deep venous red colour, with enlargement of its veins. To observe these, I evert the lower eye-lid and use a lens of about an inch focus. Sometimes they admit of being traced in considerable number, and are of considerable size.

6.—*Effect of a Tight Collar or Cravat.*

77. It is here, I think, that I may most appropriately introduce the question of the baneful and dangerous influence of a tight collar or cravat.

78. It was observed by Dr. Donald Monro that soldiers were liable to be carried off by apoplexy, in

consequence of stricture of the veins of the neck, from being obliged to wear their cravats too tight*.

79. Abercrombie quotes a case from Zitzilius, of "a boy who had drawn his neckcloth remarkably tight, and was whipping his top, stooping and rising alternately, when, after a short time, he fell down apoplectic. The neckcloth being unloosed, and blood being drawn from the jugular vein, he speedily recovered†."

80. The following case occurred in the person of a most intelligent member of our own profession. I give it in his own words:

81. "A few weeks ago, my shirt collar was made too tight, and felt rather uncomfortable; yet not so much so as to induce me to change or slacken it. On looking into the mouth of a patient, in such a position as to twist my neck a little, I dropped down in my surgery as if I had been shot, in a moment, as helpless as a dead man. I soon got up; but my head was giddy for some time. I changed my shirt, and lost all fear of a return of the accident. There can be no doubt that it arose from compression of the veins."

82. I saw a patient, a very short time ago, whose face and ears were purple from the influence of too tight a collar and cravat. I was consulted from the occurrence of oneirodynia and subsequently maniacal

* See Cheyne on Apoplexy, p 41.
† Op. cit. p. 202.

delusion. (Such a case is described by Heberden:—"Qui conflictantur cum arthritide, paralysi, aut malis hystericis, interdum expergiscuntur maxime perturbati, et quasi territi exclamant." "Pueri hoc modo experrecti interdum desipiunt horam integram priusquam ad se redeant*.") I loosened the collar, and the lividity of the complexion disappeared. I do not yet know whether the oneirodynia and its consequence also ceased.

83. The influence of a tight collar or cravat is not duly appreciated. It may be slight, in a state of repose. But on moving the head variously, the muscles of the neck expand; this expansion cannot take place *outwardly;* it therefore takes place *inwardly*, and so compresses the subjacent veins! It is on this principle, not, I think, generally acknowledged, that a moderately tight cravat may prove an unsuspected source of danger. Under the influence of such a cravat or collar, the not unusual actions of the muscles of the neck become a sort of trachelismus, perhaps more frequently than is imagined. The cravat, too, which is not tight generally, may become so under the influence of sleep, of emotion, or of gastric repletion.

* Op. cit. p. 151.

7.—*Results of Experiments.*

84. I have long projected a series of experiments with the view of illustrating the effects of the impeded return of blood from the head.

85. 1. I propose, in the first place, to ascertain the effect of a ligature, of various degrees of tightness, applied round the neck.

86. 2. I propose, in the next place, to determine the effect of a ligature round the neck, of extreme tightness, tracheotomy having been previously performed;

87. 3. In the second place, I propose, having applied a thick and soft ligature round each jugular and vertebral vein, under the influence of chloroform, to tighten these, first, one by one, then two by two, and, lastly, three by three;

88. 4. I next propose to trace the effect of a current of electro-magnetism variously across the neck, so as to induce artificial trachelismus and phlebismus.

89. An experiment of the second kind was performed, at my request, by my friend, Mr. Henry Smith, and Mr. Coates of Salisbury:

90. "On December the 17th, 1850, a full-grown greyhound was placed under the influence of chloroform, and an opening was made into the lower part of the trachea.

91. "Five hours were allowed to elapse, a double tracheotomy tube was inserted, and a cord was tightened round the upper part of the neck. After a momentary struggle, the animal became still, and the respiration slow; the eye-balls protruded, the pupils gradually dilated until the iris was a mere line, and the nearest approach of a taper induced no contraction. The diastaltic actions, as indicated by the closure of the eye-lid and retraction of the eye-ball when touched, were perfect.

92. "After the lapse of an hour and a half, the respiration had become short and feeble, the expirations being longer than the inspirations; there were occasional convulsive inspirations, and the sphincter ani was relaxed. The pulsation of the heart was audible at the distance of a yard, and induced a movement of the flame of the taper held near the orifice in the trachea. The disastaltic actions became feeble, and at length ceased. The cornea began to appear hazy and shrivelled. The tail was occasionally moved convulsively from side to side, and the anterior extremity became raised and the posterior extended powerfully, and then relaxed as suddenly.

93. "After the lapse of another hour, the respiration and the action of the heart continued as before; the tongue hung out of the mouth.

94. "In another hour and a half, the respiration and the action of the heart ceased, amidst slight convulsive movements of the posterior extremities.

95. "On examination, about six hours after death, the membranes and substance of the brain and the pia mater of the medulla oblongata were found gorged with blood, and *bloody serum* was found in the ventricles and at the base of the brain."

96. It is obvious, from this experiment, that impeded flow of blood along the veins is instantly followed by insensibility—apoplexy, in fact,—and afterwards by epileptoid affections. It is impossible, I think, to imagine an experiment more replete with instruction.

97. The following experiment I quote from a paper of the late Sir Astley Cooper, Bart.:

98. "In one rabbit I tied the jugular veins on each side of the neck. When it was set at liberty, it ran about, cleaned its face with its paws, and took green food.

99. "Its respiration was reduced to 68 inspirations in a minute, which is about half the natural number. After four hours, it ran about as if nothing had happened; and eventually recovered.

100. "When it was killed and injected, I found, on each side, three anastomosing veins passing from the anterior to the posterior part of the jugular vein, and conveying the blood from the head to the heart; the vertebral vein had remained whole, and become enlarged, and passed, on the fore part of the vertebræ, from the head to the space between the fourth and fifth cervical vertebræ, where it entered the vertebral canal.

101. "In a second rabbit, I tied the jugular veins on each side of the neck, as before. The animal's respiration became slow; but it ate green food, ran about, and was difficult to catch: but, for five days after, it appeared dull; its ears had dropped. On the seventh day, it was seen to be convulsed, and frequently rolled over. Its voluntary powers were lost, as well as its sensation, in a great degree. On this day it died. On examination, *a clot of blood* was found extravasated in the left ventricle of the brain.

102. "Hence it follows, that apoplexy will occasionally result from an obstruction to the return of blood in the jugular veins; and this I have known to happen from enlargement of the glands in the neck of a boy*."

103. Sir Astley Cooper was also in the habit of shewing an experiment in which he compressed, as he supposed, the carotids and vertebral arteries in a rabbit. It was doubtless the jugular and vertebral veins.

104. For the following most valuable fact I am indebted to J. Russell Reynolds, Esq. of University College, a gentleman of great talent and promise:

105. "A girl, nineteen years of age, was admitted into University College Hospital for aphonia; and, amongst other things in the treatment, she was ordered to have galvanism applied to the larynx daily, by the electro-magnetic machine.

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* Guy's Hospital Reports, vol. i, p. 471.

106. "While using this machine, I observed the effect upon the muscles of the neck, and remarked that, when the wheel was turned slowly, and the superficial muscles were alternately contracted and relaxed, *the colour of the face was heightened*, and of a florid hue, and no unpleasant feelings (further than those arising from the shocks) were experienced; but when the wheel was turned rapidly, with a less powerful current, and the muscles were maintained, during the rapidly intermitting action, in a state of almost permanent contraction, *the face became of a deeper colour*, *the lips and angles of the mouth livid*, *the eyes suffused*, and some feelings of confusion of thought, headache, and dimness of sight, alternating with flashing of light, were induced. The latter effects remained after the cessation of the current, for a few minutes, and then disappeared."

107. In these facts we have the *Proof* that a slight degree of contraction of the muscles of the neck, induced by the electric current, induces, in its turn, heightened colour of the face, of a florid hue; and that a greater degree of that contraction induces a deeper colour of the face, the lips and angles of the mouth being livid, and the eyes suffused, with confusion of thought, headache, dimness of sight, alternating with flashes of light; these latter remaining for a few minutes after the cessation of the current, and then disappearing. They present the *Demonstration* of the nature both of trachelismus and of phlebismus, and of their *effects*.

108. Apoplexy may depend on a first degree of the effects of compression, and convulsion or epilepsy upon a second; or—apoplexy may depend upon interrupted flow of the blood along the jugular veins principally, and epilepsy, upon interrupted flow of blood along the vertebral: this at least appears to me to be probable. But experiment must determine the interesting questions.

109. Whatever may be the rationale of epilepsy in this respect, the *effect* of the paroxysm is greatly seen in the condition of the integuments, in the extreme lividity of the countenance, the frequent ecchymoses, especially about the temple, the occasional blood-shot eye, and the not unfrequent epistaxis. The condition of the face and neck, therefore, however it may be an indication of the condition of the encephalon, is by no means a measure of that condition. There is more of lividity in epilepsy than in apoplexy; but there is a greater *degree* of stupor and of the tendency to paralysis in the latter than in the former, though these occur in both.

8.—*Pallor, Sickness, Faintishness, &c.*

110. Instead of flushing and turgescence of the face and neck, we very frequently observe pallor, with or without sickness, and faintishness, in cases of seizure.

111. Pallor may be produced by a syncopic impression on the heart itself alone, and directly.

112. But pallor and sickness conjoined *must* depend on a common cause, and that cause is doubtless seated in the medulla oblongata.

113. This affection may follow the opposite state of flushing, and be the effect of fear. One patient, to whose case I have already adverted as an example of paroxysmal paralysis, exclaimed—'I am dying!' He turned pale with terror.

114. In other cases there are pallor and ghastly lividity, probably as the immediate effect of trachelismus on the vertebral veins, inducing *irregularity* of circulation in the medulla oblongata. Faintishness, sickness, and vomiting, frequently ensue. The event may be compared with what is experienced by some persons from the movement of a carriage or a swing, and by almost all from that of a vessel on a rough sea. Irregular impulses of the blood on the medulla oblongata induce the effect of shock on the heart, and of irritation on the muscles of expiration combined in the act of vomiting. In the cases to which I have alluded, the cutaneous pores are frequently relaxed, and a cold perspiration bedews the patient's surface.

115. There is frequently, in this case, as well as in that of suffusion of the countenance, loss of consciousness, and the fear of falling, or actual falling.

116. It is a case to be most carefully distinguished from ordinary syncope from sources of exhaustion, dis-

ease of the heart, &c. and I propose to characterize it by the term—*Spinal Syncope*.

117. Sickness is sometimes associated with headache, thus constituting 'sick-headache,' sometimes with giddiness. It is frequently an effect of the emotion of disgust; sometimes, of a fall on the head; sometimes, of an apoplectic seizure.

9.—'*Tendency*' *of Blood to the Head; in reality, its Impeded Return.*

118. There is no physiological principle on which we can found the idea of 'tendency' or 'determination' of blood to the head.

119. If the circulation be accelerated by any cause, it is still accelerated equally or proportionally in every artery of the body. This result flows from the important experimental researches of M. Poiseuille.

120. And if there were such a principle of unequal distribution of the arterial blood, it would not explain the phenomena of *venous* turgescence and purpurescence observed in the cases of apoplectic and epileptic seizures.

121. But impeded venous return may be partial, and the cause at once of turgescence and of purpurescence; it is explained by the *fact*, frequently evident, however it may be sometimes latent, of spasmodic contraction of the muscles of the neck.

122. The most violent action of the heart and arteries can only induce throbbing and flushing; impeded venous return induces these, with the turgescence and purpurescence to which I have adverted, and various symptoms, such as headache, vertigo, loss of consciousness, &c.—symptoms produced equally by trachelismus and by too tight a cravat.

123. I shall never forget the interesting phenomena which I witnessed in a little boy, an American, whilst his father, an intelligent physician, and myself were discussing the questions involved in his case:—suddenly the eyes and head became fixed; the pupils dilated; the conjunctiva suffused; the cheeks deeply flushed: the little patient was obviously unconscious:—in a moment the spell was broken, the natural colour, the natural look, and consciousness, returned. The muscles which had fixed the head, had compressed the veins of the neck!

124. The doctrine of tendency or determination of blood to the head, is therefore both unfounded in fact and principle, and incapable of explaining the phenomena. Impeded venous return is both in itself the obvious effect of a familiar event, and affords the ready explanation of a subsequent series of events, hitherto unexplained.

10.—*Congestion and Softening of the Brain.*

125. In all cases of the apoplectoid or epileptoid seizure, whether hidden or observed, the cerebrum is congested, the intervening links being trachelismus and phlebismus.

126. If this congestion be extreme, and greater in one hemisphere than the other, hemiplegic paralysis is observed.

127. If the cerebral affection be limited to *congestion*, and if this congestion disappear, the paralysis disappears too. It is paroysmal and transitory.

128. But if this congestion leads to ecchymosis (as we *see* in the face), this cannot subside; *softening* is the result; and, with this, a greater or less degree of permanent hemiplegia.

129. Or there may be effusion of serum into the ventricles, and its consequences—loss of memory, &c.

130. If, with the paralysis, there be spasm (if it be spasmo-paralysis), the medulla oblongata is *irritated*, by pressure or counter-pressure from the tumefied cerebrum.

131. This series of events is of the deepest interest, and presents a new subject of investigation to the pathological anatomist. It will be necessary to trace and distinguish the different links of the chain of cause and effect: the morbid appearances are *not the disease*; they may be the *effect* of one of its sym-

ptoms, and the *cause* of others. They may be intra-vascular and evanescent during life, and therefore absent on the post-mortem examination; or they may become extra-vascular during life, and therefore be detectible by the anatomist.

132. I believe this view of softening, as the result of congestion, to be at once new to the pathologist and deserving of his most serious attention. Mere morbid anatomy, unconnected with the *history* of the case, is like the caput mortuum of the alchymist; it is only of real value when traced backwards to its living cause or causes, and forwards to its effects.

133. The flow of venous blood, on opening the cranium, the condition of the veins in the extra- and intra-cranial tissues, the effusion of serum or of blood, must all be viewed in connection with the chain of morbid processes during life. It is *living* pathology which alone can serve us in relieving the sick.

11.—*The Diagnosis between Paroxysmal and other Attacks of Apoplexy.*

134. There is still no medical topic of such value, and importance, and difficulty, as Diagnosis.

135. Hitherto, I think, the distinction between the different attacks of apoplectic character has only been one of *degree*. But I believe there is an essential difference between the *Threatenings* of apoplexy which

occur and recede paroxysmally, and even the slightest inroads made by organic disease, whether of the arteries, or veins, or membranes, or the substance of the encephalon. Whilst the former are repeated, at first leaving little or no ill effect behind, the latter proceed insidiously, and at last there is perhaps a crushing attack of pain, of pallor, and of apoplexy; or, of hemiplegia;—a large laceration of the substance of the brain and extravasation of blood being discovered on making a post-mortem examination.

136. It must be borne in mind that a first attack may assume the form of paroxysmal apoplexy, the patient recovering speedily and entirely; and yet the second may be of the most deplorable character. In the former case, there are generally turgescence of the face and neck—the effect of trachelismus; in the latter, there is pallor—the effect of *shock*. An interesting case of this kind was recently published by Mr. Dunn, in *The Lancet*.

12.—*The Treatment of Paroxysmal Nervous Affections.*

137. I have already hinted at the difference in the treatment of paroxysmal and of organic apoplexy:

138. It would be a very dubious measure to administer an emetic in the case of a violent attack of apoplexy or paralysis. It is more than probable that

greater congestion, or greater effusion of blood, would be excited by the acts of vomiting.

139. But if the case were one of paroxysmal apoplexy or paralysis, the nausea and sickness induced by a mild emetic would probably dissolve the spasm on the muscles of the neck, and so remove the consequences—the impeded return of the blood along the veins of the neck and head, and the congestion of the encephalon.

140. It is in this manner that the long-continued discussion between Fothergill and Cheyne and other physicians, is to be terminated. There are forms of the apoplectic seizure, for which a mild but effectual emetic is the appropriate remedy; there is another in which the administration of an emetic would not be unattended with the danger of aggravating the disease.

141. The principles of the treatment of the paroxysmal forms of apoplectic, paralytic, and epileptic diseases are indeed totally different from those of the similar diseases of *organic* origin. But I shall have to recur to this important topic.

PINAL

THE NECI

I. THE CAUSES. I. THE MODE US.

I. *Centric;*

1. *Emotion, Excitement;*
2. *Sleep; awaking, &c.;*
3. *Posture; Effort; &c.;*
4. *Abnormal Conditions of the Blood; plethora; anæmia; &c.*

} *Catastaltic or Direc*

of

ugular—

ugular—

Veins.

II. *Ex-centric;*

1. *Dental—*
2. *Gastric—*
3. *Intestinal—*
4. *Uterine—*
   1. *Catamenial—*
   2. *Puerperal—*

} *Irritation;* } *Diastaltic, or Refle*

ral.

(*To face page* 35.)

LECTURE II.

RATIONALE; SYNOPTICAL VIEW; ETC.

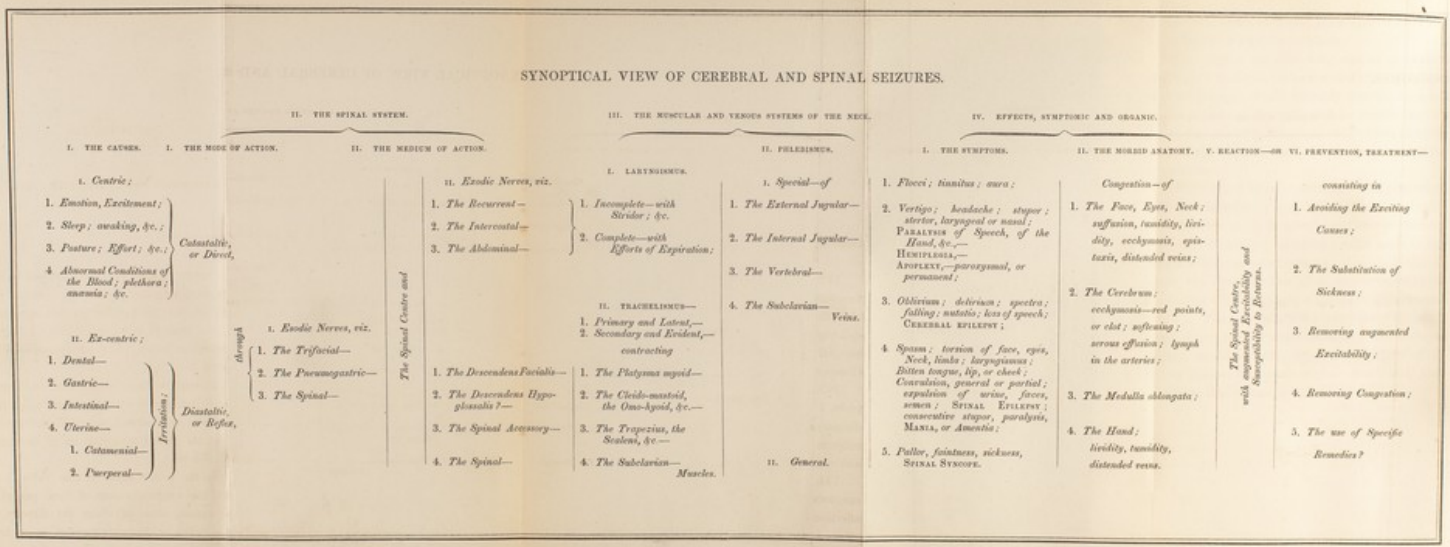
GENTLEMEN,

142. In the present Lecture I propose to lay before you a more *connected* view of the chain of causes and effects, in the cases of paroxysmal apoplexy, of epilepsy, &c. than in my former one. I beg your especial attention to this *Table* or *Synoptical View* of those events.

143. It will be remembered that, in these Lectures, I leave out of view entirely all originally organic diseases of the encephalon or spinal marrow. My subjects are the paroxysmal diseases of the cerebral and spinal systems.

144. In the first column in this *Table*, I have enumerated the exciting *causes* of these paroxysmal affections; causes, some of which act directly, and others in a reflex or diastaltic manner, in regard to

SYNOPTICAL VIEW OF CEREBRAL AND SPINAL SEIZURES.



(To face page 35.)

the spinal centre. Of the former class, are *Emotion* and mental excitement principally; of the latter, the *Irritations.*

145. Why these causes should select the muscles of the neck and throat principally for the display of their influence, is a mystery; but it is not the less a fact that they do so. I have already remarked, I think, that *Expression* is as much *seen* in actions about the throat as in the countenance, whilst the effects of emotion are *felt* in that susceptible region.

146. The effect of *Sleep*, again, is still manifested in the same region, though less directly. Volition being removed, the muscles of the neck are delivered over, like the orbicularis, to the influence of *tone*—a spinal action, as proved by the experiment on the turtle, of withdrawing the spinal marrow and watching the effect in relaxing the sphincter. The result of this trachelismus is a *sub*-apoplexy, and the disposition to paroxysmal seizures. I have this day seen a patient who occasionally experiences an attack of a suffocative character, with a sense of constriction about the throat, on falling asleep. See § 66. Another patient was liable to awake in a state of confusion, and this confusion, on one occasion, lasted for many minutes. This is, in fact, a species of *Oneirodynia.*

147. The other causes enumerated in the first part of the first column, act also directly upon the medulla oblongata, and thence on the muscles of the throat and neck.

148. The Irritations act in a reflex or diastaltic manner. Sometimes there is a feeling of constriction or of 'a spike' (for so it has been expressed) about the throat or neck; sometimes there are, sooner or later, sickness and vomiting; the latter of which involves closure of the larynx. In other instances there are flushing, vertigo, headache, and other threatenings of apoplectic or paralytic seizure; or, perhaps, of a seizure of an epileptoid character; facts, all of which may now, I think, be adduced as proofs of the existence of trachelismus, to which laryngismus, trachelismus in other form, is so apt to be conjoined.

149. The irritations act through the medium of incident or *Esodic* nerves; viz. the trifacial, in the case of teething; the pneumogastric, in that of gastric irritation; and the spinal, in those of irritation of intestinal or uterine origin.

150. The irritations of these nerves are, by a mysterious agency, diastaltic through the medulla oblongata, and thence through certain *Exodic* nerves upon the muscles which they supply, and specially upon those classed in this part of my *Table;* viz. the recurrent, the intercostal, the abdominal; the descendens facialis, the descendens hypoglossalis, the spinal accessory, and the other spinal nerves.

151. The course of action along these nerves is traced in its effects on special muscles. These are arranged in *this* column. They are the muscles which close the larynx, and the muscles of the neck,—espe-

cially the platysma myoid, the cleido-mastoid, the omo-hyoid, the trapezius, the scaleni, the sub-clavian.

152. As proofs of these actions, I must here adduce some most interesting facts:

153. For the first of these I am indebted to J. Russell Reynolds, Esq. to whose talents I have already paid a well-merited tribute of praise, at page 25. He states, in a note addressed to me in June 1849,—"I have been watching, with great interest, during the last five days, a case of Epilepsy, in University College Hospital. The patient, a stout woman, aged twenty-six, was brought in early on the morning of June 1, in a fit. She had several attacks before I saw her, which was about half-past ten, a. m. She was then lying very restlessly, her face a little flushed, and some convulsive twitches were playing around the mouth. I placed my finger on the *omo-hyoid* muscle, which I could at times see distinctly in the 'posterior triangle' of the neck. It contracted and relaxed several times under my finger; then some of the surrounding muscles were strongly contracted, and a general, but not severe, convulsion followed. There was *total loss of consciousness*, but *not* any great *turgescence of the external veins.*

154. Two days after this, I was again watching her. She had had several severe attacks in the night; and there were now the same convulsive twitches of the muscles of the lower part of the face. I placed my finger in the direction of the omo-hyoid muscle,

but could not distinguish it. As I was doing this, the *platysma myoides* contracted violently; its fasciculi stood out in full relief; it was exceedingly rigid; *the veins of the neck became much distended, the face deeply livid;* the surrounding muscles of the neck were *then* strongly contracted, the thorax was drawn towards the head, and the general convulsion which followed was one of the most violent I have ever seen."

155. For the next case I am indebted to W. J. Bryant, Esq. of Bathurst Street. I give his graphic sketch in his own words:

156. "Jane D. aged 82, has been under my care for the last fourteen years. For many years she was severely attacked with bilious sick-headaches, of an agonizing character. The attack was always accompanied by severe bilious vomiting. This state of things continued for a year or more, when, during an attack of vomiting and headache, she was seized with a mixed character of fit (apoplectic epilepsy), which more particularly attacked the left arm and leg, and the left side of the face, the tongue being wounded. The fit passed off, and was succeeded by a profound sleep. I have now seen her so attacked twenty-eight times. She has diminished power of the left hand and arm after each attack.

157. "The last attack was very severe, the patient being insensible and unable to swallow. Having occasion to apply a mustard plaster to the nape of the neck, I was struck by observing the peculiar manner

in which the skin of the neck was drawn, as it were, into a band. *I could distinctly observe this band arresting the flow of blood through the external jugular vein, which, with the veins of the face of the left side, was turgid*, and, in one part, dilated into a *varix*. To ascertain whether this band was influencing the circulation, I raised it up, and immediately the veins emptied themselves, and the patient was able, for the first time, to reply to a question from me. My patient being of a very spare habit, I had an excellent opportunity of witnessing the remarkable part which the muscles of the neck were playing. The genio-hyoid, the omo-hyoid, the sterno-cleido-mastoid, and the platysma, were prominently shown and rigid.

158. "The difficulty of swallowing and the insensibility were greater in this attack than usual; but they are always present, more or less, in all.

159. "I had written this, and from circumstances had been prevented further detail, when I received a summons to visit my patient, who was again in convulsions. Upon my arrival, I found my patient but slightly attacked. There were twitches of the muscles of the face, side, and leg, and a slight difficulty in swallowing; the face, as usual, was suffused, the veins slightly turgid, and, to my satisfaction, I found the same band of skin raised by the contraction of the *platysma;* and it was now that I was able at once to arrest the phenomena of convulsive action, by raising

the band. The paroxysm ceased almost immediately. The omo-hyoid was not so distinct; but still it was prominent. In fact, viewed as a whole, it was an admirable natural dissection of the triangle of the neck."

160. An interesting case, in which there was contraction of the omo-hyoid, was communicated to me by T. A. Henderson, Esq. of Portman Place, Edgeware Road:

161. "Miss H. aged 67 or 68, was attacked, nearly four years ago, with symptoms of commencing apoplexy, which subsided, but left the left leg and arm very weak, and liable to very constant and peculiar muscular action—the great toe of that foot being painfully drawn away from the others, and the left arm being in a tremulous, twitching condition almost continually, and much weaker than the other. I must remark that all these symptoms were much better when the patient was recumbent. The head has been lately drawn downwards and to the left side, and she *feels* a *pulling* in the throat on that side. On putting the fingers along the lower part of the neck, the *omo-hyoid* can at times be felt twitching and drawing—in fact, in a kind of irregular spasmodic action; just as the tendons of the muscles of the arm can be felt at the wrist of the left arm, and indeed, lately, of the right arm also, twitching and catching in a very irregular manner. I should also add, that these symptoms are at times much less violent, varying with the state of the general health; but never entirely absent.

When they are severe, I have remarked that pressure on the omo-hyoid causes pain, and sets up the same spasmodic action and pain in other parts, as the great toe of the left foot, the back of the neck, &c. Pain is also at such times felt in the anterior portion of the trapezius muscle, which I once or twice thought I could feel in the same irregular state of action as the other muscles."

162. In a case of epileptoid seizure, I had, some time ago, an opportunity of observing the clonic contractions of the *omo-hyoid*, with my friend Mr. Martin.

163. The late Professor Gregory used to mention, in his Lectures, the fact of a man who, being in a boat, and suddenly turning his head to look at an object in the opposite direction, fell down apoplectic.

164. One patient, subject to epileptic attacks, cannot turn her head extremely to the left side without a strange feeling of vertigo, or confusion, or threatening of a seizure. A similar position of the head turned towards the right side produces no such effect. The phenomenon is obviously the effect of the action of certain muscles and the compression of certain veins.

165. These facts are sufficient for illustration. The subject is proposed for investigation; for it is new, and still insufficiently explored.

166. The subject of the anatomy and physiology of trachelismus, with its *varied* effects on the circulation, and on the functions of the face, neck, encephalon, and medulla oblongata, will require years of cautious observation. § 168.

167. Of the influence of compression of the veins of the neck in inducing apoplectic symptoms, we have an example in a case of Sauvages, quoted by Abercrombie*:—"A man, after execution, was recovered by three bleedings, and sat up and talked, his breathing and deglutition being natural. After a short time, the part of his neck where the cord had been applied began to swell, so as evidently to impede the circulation in the veins of the neck; he then became drowsy, his pulse and respiration slow, without dyspnœa, and in a few hours he died apoplectic."

168. In blushing and flushing, in the suffused and blood-shot eye, in ecchymosis, in epistaxis, we have the effect of impeded return of blood along the external jugular; in apoplectic symptoms, we have the evidence of impeded return of blood along the internal jugular; in epileptoid symptoms, we have the same evidence in regard to the vertebral. At least, this I believe to be true in general terms. These points must be submitted to cautious *observation and experiment.*

169. The transition of congestion into ecchymosis, and of this into softening, as displayed in this column of the *Table*, is also a further subject for careful investigation, equally new and important.

170. In the succeeding column another topic is noticed. It is the susceptibility or tendency left by previous attacks, to subsequent attacks of the same

* Op. cit. p. 202.

character. The nature of this may be either of a nervous or vascular character, or both,—that is, there may be either *nervous or vascular exhaustion and reaction*. Time, and the avoiding of the exciting causes, and tonics, and especially such a tonic as will act on the spinal marrow, appear to me to be means of cure.

171. These remarks illustrate the last column of the *Table*, in which I have enumerated the principles of treatment of paroxysmal seizures.

172. To remove and avoid the causes; to avoid the obstacles to the cure, by regulating all the functions; to restore the due tone of the system; are our great objects.

173. Now there comes a question as to any specific remedy; and, in this respect, it becomes a question whether strychnia, which we know to possess the power, in large doses, of singling out and *stimulating* the centre of the spinal system, would, in minute doses, act as a *tonic* upon this organ specially—an event which, from some cautious trials, I think probable. It would present us with an example of a *Spinal Tonic*.

174. A second question is not of less interest. Might sickness and vomiting be so timeously induced, as either to anticipate or supersede the paroxysmal seizure? Such a seizure is frequently terminated by a fit of vomiting. If there were any premonitory circumstances or symptoms, might not an emetic ward off the coming attack? Are sickness and vomiting

compatible with the paroxysmal apoplectic or epileptic threatening?

175. Lastly, do the new principles which I have unfolded lead to any other modes of prevention or treatment?

176. I could give a goodly list of cases which have been brought, by dietetics, by mental and physical regimen, and by the remedies and means to which I have adverted, to a happy issue. But time, and patience, and steadiness of purpose, are required in the physician, in the patient, and in the patient's friends; and many are the disappointments in the course of the case which may yield to your efforts favorably at last. The susceptibility to attacks may be extreme; exposure to the exciting causes scarcely to be avoided.

177. The first link in this extraordinary chain of causes and of effects is a cause either of direct or catastaltic, or of reflex or diastaltic, action; this cause acts through the spinal system upon the muscles of the throat and neck, and perhaps of the larynx; these upon the veins of this region; congestion of the *intermediate* blood-channels, intermediate between the last arterial branches and the first venous roots, and congestion and perhaps ecchymosis, of the exterior or interior parts of the head, take place!

178. In all this chain, each link is essential and the series complete! Is it not a unique instance of a living pathology so traced, and of the practical appli-

cation of a physiological discovery? And how does it call forth our knowledge of anatomy!

179. Indeed, I propose to seize the opportunity of again dissecting *The Neck*, regarded as a *Medical Region*, with peculiar care. The nerves, esodic and exodic, the muscles, the veins, must be displayed; and their relative actions should be traced in a series of well-devised experiments and cautious observations.

1.—*The Relation of Apoplexy, Paralysis, Epilepsy, and Mania.*

180. The patient affected with paroxysmal apoplexy sometimes becomes epileptic. The epileptic patient, on the other hand, sometimes experiences attacks which gradually assume the more apoplectic character. The fit of apoplexy is sometimes attended with convulsion, as observed by Abercrombie*. The fit of epilepsy usually terminates in an apoplectic stupor, and this sometimes in mania.

181. Both the apoplectic and the epileptic seizure are equally prone to issue in hemiplegic paralysis. This event is both more frequent and more apt to be permanent in the former case than in the latter; and, I believe, for this reason:—The cerebrum is more congested in apoplexy than in epilepsy, though it *is*

* Op. cit. p. 203-4.

affected in both. Epilepsy is more apt to become complicated with spasmo-paralysis than apoplexy, for a similar reason: the medulla oblongata is more affected in the former disease than in the latter.

182. The apoplectic and especially the epileptic seizure is apt to pass into stupor or mania; and, in the case of *hidden* seizure, it may be very difficult to form an accurate judgment of the nature of these events.

183. Heberden, whom I have so often quoted, observes—"Modo insania et paralysis eundem vicissim occupaverunt. In nonnullis epilepsia tam prope abest a paralysi, ut difficile dictu sit ad utrum morbum signa sint referenda." And—"Inter plurima autem mala, quæ secum ferunt affectus apoplectici, aliquid inde boni semel visum est oriri: nam epilepticus quidam, attonitus factus, deinde revixit, et veterem suum morbum nunquam postea expertus est. Contra, aliis contigit, ut ex hemiplegia assurgentes, tum primum cœperint cum epilepsia conflictari*."

184. Every fact leads to the inevitable conclusion, that the apoplectic, paralytic, epileptic, and maniacal affections are allied intimately together.

185. The same remarks relate to puerperal cases: convulsion, apoplexy, paralysis, mania, are so linked together, that they may not only occur singly, but in various succession, before, during, or after, parturition.

* Op. cit. pp. 287; 297.

The difference is, in reality, but the difference of vein principally compressed.

186. In one lady, the subject of repeated epileptic seizures, these assumed gradually more and more of the apoplectic character, until one terminated fatally and suddenly. On a post-mortem examination, the integuments of the face were of "a dark blue colour," dark blood flowed on dividing the scalp and separating the dura mater; the sinuses of the veins on the surface of the brain were gorged with dark-coloured blood; the substance of the cerebrum was healthy, but greatly congested. The vertebral arteries presented "a pouchy appearance." There was a fatty heart.

187. In a gentleman, several epileptic seizures occurred, the effect of *fear*,—the fear of cholera. After each, a hemiplegic paralysis of the *right* side took place; but this yielded completely, except that the patient could never divert his mind from the idea that the feeling of the affected side was somewhat different from that of the other. At length a further attack proved fatal; and, on a post-mortem examination, the arachnoid was found slightly opaque, the ventricles containing serum, whilst in the *left* corpus striatum there was the remnant of a small clot of blood, in a cyst slightly discoloured. The arachonoid was raised in one spot by serum, resembling a vesicle, and a small cyst was attached to the plexus choroides.

188. In both these cases, the arteries at the base of the brain contained a little opaque fibrine;—the

effect of the seizures and of impeded flow of blood along their course?

189. Every day brings forth some new illustrative fact. For the following sketch I am indebted to W. F. Barlow, Esq. of the Westminster Hospital:—"A woman, 38 years of age, who had been some time labouring under chronic bronchitis and a laryngeal affection, which was occasionally aggravated by spasm, was one day seized with a violent spasmodic action of the glottis, in which she appeared nearly suffocated. It relaxed, and she recovered, without ill consequence; but shortly afterwards she was attacked with another such spasm, on the subsidence of which, the left side of the face, the left arm, and the left leg, were found completely *paralysed*. The patient was going on, to all appearance, very well, when she was seized with an *apoplectic* fit, and speedily died."

2.—*Of Paroxysmal Diseases of the Cerebral and Spinal Systems, as a Class.*

190. In concluding this Lecture, I may observe that I am persuaded that I have stated enough of fact to effect the establishment of a *Class* of paroxysmal diseases of the nervous system, each and all of which involve an excitant of diastaltic action, on muscles of the neck, and compression, by these, of the veins of that region, and the consequent congestion of the tissues

without or within the encephalon and spinal cavity, perhaps with ecchymosis or softening, or serous effusion.

191. These events are variously translated into apoplectic, paralytic, epileptic, syncopal, or maniacal seizures, which constitute the *Class* of Cerebral and Spinal Paroxysmal Affections.

192. In some instances, the *first stage* of these seizures is *hidden;* in others, the seizure assumes the form of *Oneirodynia;* in others again, it is mere blushing, 'sick-headache,' 'sick-giddiness,' &c.

192. What a momentous subject for fresh inquiry!

193. In our daily visits to the sick, our first duty is to establish an accurate *Diagnosis.* Diagnosis, in these diseases, is unfortunately not of the physical kind, as in diseases of the thorax, but the interpretation of symptoms. In this manner it is that the *physiology* of the nervous system and the *diagnosis* of its diseases, meet and coalesce. And yet the physiologist is still calumniated by the 'mere practical man,' that is, the empiric, as a *theorist.* Such is still the deplorable condition of our profession!

LECTURE III.

DIAGNOSIS; CASES; TREATMENT.

GENTLEMEN,

194. In this concluding Lecture I propose to illustrate my subject quite practically, and by the detail of a few *Cases*, with such observations as they may suggest, and with special reference to the diagnosis and treatment.

195. The basis of all scientific medicine is—the *Diagnosis.* The next steps are the physiology, the living pathology of the disease; and the next, the therapeutics.

1.—*Apoplexy and Paralysis.*

196. The great question, in regard to the diagnosis of apoplectic and paralytic seizures, is that of their Inorganic or Organic character, primary or secondary.

197. I consider that form of apoplexy or paralysis which arises from emotion, or irritation, as primarily *inorganic*. That form of these affections which arises out of disease within the encephalon, and especially from rupture of the substance of the brain, of course, as *organic* in its character.

198. The former of these is characterized by varied flushing of the countenance, and perhaps of the neck, and by various symptoms, such as headache, vertigo, loss of consciousness; loss of the power of speech or of the hand; or more decided apoplexy or hemiplegia. Of this kind of attack there is every variety, every degree, every duration from the most transitory to the permanent, every kind of recurrence and remission. It may be slight and transitory, and recurrent during many years. It may lead to organic apoplexy or paralysis. It may prove fatal even, in any of its attacks, early or late.

199. I now beg, Gentlemen, to lay before you another extract from Abercrombie, which I consider as amongst the most important in medical writings—a sufficient apology, I hope, for its length;

200. "The apoplectic attack is generally preceded by symptoms indicating some derangement of the circulation in the brain. The most remarkable of these are the following:—headache, giddiness, sense of weight and fulness in the head, violent pulsation of the arteries, and confused noises in the ears. These symptoms are often accompanied by *epistaxis*, which

may give a partial and temporary relief; by loss of recollection, and incoherent talking, resembling slight intoxication; by affections of the sight, double vision, and temporary blindness; by drowsiness and lethargic tendency. We also frequently observe *indistinct articulation*, and other *partial paralytic* affections. These are sometimes confined to one limb, or part of a limb; sometimes affect the eyelids, producing inability either to shut the eye, or to open it; and frequently impair the muscles of the face, producing a slight distortion of the mouth. These symptoms, and others of a similar kind, mark the *tendency* to the apoplectic state, and often appear for a considerable time *before* the attack actually takes place. The attack itself occurs chiefly under three distinct forms, which it is of importance to distinguish from each other.

201. "I. In the first form of the attack, the patient falls down suddenly, deprived of sense and motion, and lies like a person in a deep sleep; his face generally flushed, his breathing stertorous, his pulse full, and not frequent, sometimes below the natural standard. In some cases *convulsion* occurs, in others *rigid* contraction of the muscles of the extremities; and sometimes contraction of the muscles of the one side, with relaxation of the other. In this state of profound stupor, the patient may die after various intervals, from a few minutes to several days; or he may recover perfectly, without any bad consequence of the attack remaining; or he may recover from the coma, with

paralysis of one side. This paralysis may disappear in a few days, or it may subside gradually, or it may be permanent. Other functions, as the speech, may be affected in the same manner, being speedily or gradually recovered, or permanently lost; and recovery from the apoplectic attack is sometimes accompanied by loss of sight.

202. "II. The second form of the disease begins with a sudden attack of pain in the head; the patient becomes pale, sick, and faint; generally vomits, and frequently, though not always, falls down in a state resembling syncope; the face pale, the body cold, and the pulse very feeble. This is sometimes accompanied by slight convulsion. In other cases, he does not fall down, the sudden attack of pain being only accompanied by slight and transient loss of recollection. In both cases he generally recovers in a few minutes from the first effects of the attack, is quite sensible and able to walk, but continues to complain of headache; after a certain interval, which may vary from a few minutes to several hours, he becomes oppressed, forgetful, and incoherent, and then sinks into coma, from which he never recovers. In some cases paralysis of one side occurs, but in others, and I think the greater proportion of this class, no paralysis is observed.

203. "III. In the third form, the patient is suddenly deprived of the power of one side of the body, and of speech, without stupor; or if the first attack be accompanied by a degree of stupor, this soon disappears; he seems sensible of his situation, and endeavours to express his feelings by signs. In the farther progress of this form of the disease, great variety occurs. In some cases, it passes gradually into apoplexy, perhaps after a few hours; in others, under the proper treatment, the patient *speedily* and *entirely* recovers. In many cases the recovery is gradual, and it is only at the end of several weeks or months that the complaint is removed. In another variety, the patient recovers so far as to be able to speak indistinctly, and to walk, dragging his leg by a painful effort, and after this makes no farther improvement. He may continue in this state for years, and be cut off by a fresh attack, or may die of some other disease without any recurrence of the symptoms in his head. In a fifth variety, the patient neither recovers, nor becomes apoplectic; he is confined to bed, speechless and paralytic, but in possession of his other faculties, and dies gradually exhausted, without apoplexy, several weeks or months after the attack.

204. "These three forms of disease frequently pass into one another; but they are very often met with, as they are here described, forming affections which differ remarkably from each other; and they appear very naturally to arrange themselves into the three classes which have here been referred to;—first, those which are immediately and *primarily apoplectic;* secondly, those which begin with a sudden *attack of headache*, and pass gradually into apoplexy; thirdly,

those which are distinguished by *palsy*, and loss of speech, without coma*."

205. It is obvious that the form of seizure described in the *first* of these paragraphs, is the *paroxysmal*, and that it may be *apoplectic* or *paralytic*.

206. It is not less obvious that the attacks described in the *second* and *third* paragraphs are alike in their *organic* origin, that of the former being *apoplectic*, that of the latter *paralytic*.

207. There ought then to have been, *not three* paragraphs, but either *two* or *four*; and such is the division I would propose. Thus cerebral seizures may be divided into

1. *The Paroxysmal*, and
2. *The Organic*;

and each of these may be subdivided into

1. *The Apoplectic*, and
2. *The Paralytic*;

whilst each of these may present itself in the form of

1. *The slightest Threatening*, or
2. *The severest Seizure.*

208. Paroxysmal cerebral seizure is for the most part distinguished by the flushing of the countenance, the recurrent form of the seizures, the partial nature of these, &c. &c whether they be apoplectic or paralytic, and the absence of *severe pain of the head.*

209. The organic cerebral seizure is generally

* Op cit. p. 203—5.

denoted by pallor of the countenance, faintishness, sickness, sometimes with *severe pain of the head.* This kind of attack is generally severe, and the apoplexy and the paralysis are comparatively little under the control of remedies.

210. In the paroxysmal seizure there is little of the appearance of *shock*; in the organic, the shock is frequently extreme, and traced in the condition of the countenance, the general surface, the pulse, &c.

211. In the case to which I have already referred, published by Mr. Dunn, the first attack was of the paroxysmal kind; the second was obviously of the organic character. And thus it is obvious that the *effect* of a paroxysmal seizure to-day, may prove the *source* of an organic seizure to-morrow.

212. The great and real distinction between paroxysmal and organic apoplexy and paralysis is this:—in the former, the condition of the encephalon is first one of congestion, and afterwards of ecchymosis, rupture, softening; in the latter, the condition of the encephalon is one of organic disease, rupture, and compression;—with their respective consequences on the functions of the nervous system; apoplexy or paralysis being the effect of the congestion, or of compression, general or partial, and transitory or permanent like their cause; and paralysis, of congestion or of rupture or softening, and transitory or permanent like its cause.

213. Abercrombie speaks of paroxysmal apoplexy

as "simple" or "primary," and of the organic as "not primary," and as "accompanied with exhaustion." Of the latter he says—"They are not at first apoplectic; or, if there be at the very first attack loss of sense and motion, this state is recovered from in a few minutes, or perhaps seconds, without any remedy. The prominent symptom, at the commencement of the disease, is a sudden attack of violent headache, the patient often starting up and screaming from the violence of it. Sometimes he falls down, pale, faint, and exhausted, often with slight convulsion, but recovers from this state in a very short time. In other cases he does not fall down, but feels a sudden and great uneasiness in his head, generally with paleness, sickness, and often vomiting. The first attack being so far recovered from that the patient is often able to walk home, the symptoms go on under various modifications. The fixed pain in the head generally continues, often referred to one side of the head; and generally there is vomiting. The patient continues for some time, perhaps an hour or two, cold and feeble, with cadaverous paleness of the countenance; his pulse weak and generally frequent. He is quite sensible, but oppressed. By degrees he recovers heat and the natural appearance of the countenance, and the pulse improves in strength. The face then becomes flushed; he is more oppressed; he answers questions slowly and heavily; and at last sinks into coma, from which he never recovers." And—"As far as my observation extends,

the cases which belong to this class are generally fatal. They form a modification of the disease, remarkably different from the simple apoplectic state; and, on inspection, we find none of those varieties and ambiguities which occur in the apoplectic cases, but uniform and extensive extravasation of blood. From the whole history of them, I think there is every reason to believe, that they depend upon the immediate rupture of a considerable vessel, without any previous derangement of the circulation, the rupture probably arising from disease of the artery at the part which gives way. At the moment when the rupture occurs, there seems to be a temporary derangement of the functions of the brain; but this is soon recovered from. The circulation then goes on without interruption, until such a quantity of blood has been extravasated as is sufficient to produce coma*."

214. Cheyne observes—"I have never known a patient recover, who, in the beginning of the attack, complained of sudden pain in the head," &c.†

215. There are then paroxysmal and organic apoplexy. How essential that the *diagnosis* should be vividly impressed on our minds!

216. The following case, for which I am indebted to Mr. Coates, of Salisbury, is full of interest, as displaying some feelings and appearances of trachelismus with cephalic symptoms:

* Op. cit. pp. 218; 219.
† Cases of Apoplexy and Lethargy; 1812; p. 13.

217. "A gentleman, aged about 70, of full habit, and having suffered from hæmorrhoids, with occasional loss of blood, and from gout, sustained a severe affliction in the loss of his son. He became liable to awake in the night with a suffocative feeling in the throat, making a peculiar noise. In the day too he was subject to giddiness, with a slight cloudy appearance before the eyes, and a sense of tightness about the throat.

218. "He had, at the time of this report, frequent headache and giddiness, and dimness of sight; his neck was thick, the external jugulars and the temporal arteries prominent."

219. I extract the following important case from the useful work of Dr. Cheyne*:

220. "August 26, 1804. Mr. A——n, æt. 65. I was called to visit this gentleman, in lodgings, at Bath Street, where he was residing for the convenience of sea-bathing, which he had been advised to use for some weeks. He had dined in Edinburgh, and had afterwards walked home. On his arrival, his daughter observed only that he was exceedingly flushed and warm, and that he was perspiring very copiously about the head and face. While she was preparing some drink for him, he fell from his chair insensible. On my arrival, he was laid in bed, his head and shoulders supported by his wife, and my father in the act of

* Op. cit. p. 94.

bleeding him. The state of apoplexy was complete, and unequivocal: the respiration was deep and sonorous, and the pulse was slow and full. *His face was flushed*, or rather *livid*, for he was a big and corpulent man, with a thick short neck, and the superadded signs of a bon vivant. The blood flowed freely from a large orifice; and, as the fourth cup was nearly filled, our patient became sick, and vomited very freely the half-digested remains of a plentiful dinner. Shortly after this, our patient opened his eyes, and turned round his head; and after two hours, he seemed nearly completely recovered. His wife and daughter were much less surprised than we were, for this was the gentleman's *third* attack of apoplexy; and the former fits had also terminated by vomiting. After the first attack, the right arm continued paralytic for twelve weeks, but gradually recovered, after a course of sea-water bathing.

"(Signed) GEORGE KELLIE, M.D."

221. In the treatment of the apoplectic and paralytic attack, the great questions relate to the administration of blood-letting and emetics.

222. In the decided paroxysmal seizure, our practice may be, and ought to be, energetic. We should promptly take away blood, and we should induce sickness and vomiting.

223. If the attack be slight and repeated, an antacid aperient draught, properly repeated, may be all

that is immediately required. If it be severer, an emetic with an antacid should be first given, and then an antacid aperient. If severer still, bloodletting, by cupping or by venesection, must be premised.

224. In the midst of these measures, the head should be raised, a cold lotion applied to the crown of the head, sinapisms behind the ears and to the nucha, and fomentations of the feet, and an enema should be administered.

225. Afterwards, the tenth part of a grain of the chloride of mercury, two grains and a half of the pilula hydrargyri, and half a grain of squill and of ipecacuanha, should be given thrice a day.

226. But, in organic apoplexy or paralysis, it may be a question whether we should take blood; but there can be no question in regard to the administration of emetics.

227. The propriety of bloodletting and its measure, must depend upon the state of the pulse and of the patient generally. The condition of the pulse must be ascertained as the blood flows. Sometimes its strength improves, and then we venture to proceed. On having taken what is deemed the due quantity of blood from the arm, we may prescribe cupping behind the ears, or at the nucha.

228. Emetics ought, I believe, and for the reasons stated, to be avoided.

229. The other remedies are those which have been already noticed as proper in the other form of apoplexy or paralysis.

230. The cupping instrument applied to the nucha, making crossed incisions, but taking very little blood, presents us with a very efficacious mode of counter-irritation.

2.—*Epilepsy and Epileptoid Affections.*

231. The epileptoid or epileptic seizure is still more distinctly characterized by trachelismus. In some cases the whole attack consists in a fixed state of head and eye, dilated pupil, and a deep flush. In other instances, unusual flushing of the face, with suffusion of the eye or eye-lid, is the forerunner of a decidedly epileptic seizure. Every thing tends to prove that the earliest effect, whether in apoplexy or epilepsy, is a state of trachelismus.

232. In the slighter forms of these maladies, there is, in reality, *no* difference. The threatening of apoplexy is so far spasmodic, that is, *spinal*, that it consists in trachelismus with its effects on the countenance and encephalon; the *petit mal* has even been designated *cerebral*, from its principal symptoms. The condition of the countenance and of the brain is identical. I repeat, there is no difference. The real difference between apoplexy and epilepsy is only seen in their severer forms. It is then that, whilst apoplexy is only attended by the simpler trachelismus, in epilepsy, to this simpler trachelismus is superadded an-

other form or degree of the same affection, with all the peculiarity it induces, laryngismus, and, in its train, it may be, odaxismus, or the—trachelismus, shall I call it?—involved in the *bitten tongue*. Now it is that, whereas the further phenomena in apoplexy are *cerebral*, those in epilepsy are *spinal*.

233. The first stage or first degree of both apoplexy and epilepsy consists then in trachelismus,—a spasmodic or spinal action, manifested in its effects on the venous circulation of the countenance and of the encephalon. The second stage or degree of these maladies, is augmented cerebral affection in the former, of spinal affection in the latter; the difference consisting in the different forms assumed by the trachelismus, or of the muscles contracted, and of the veins compressed and obstructed. If these muscles are those which compress the jugulars, the case is apoplexy; but if they are those which compress the vertebrals, and close the larynx, it is epilepsy! At least, I have not been able to resist the train of thought which has forced itself upon me, and which I lay before you with the utmost frankness, trusting to you to give it your most candid consideration.

234. Both paroxysmal apoplexy and epilepsy are, then, first *spinal* or spasmodic, only in different degree and extent; both become *cerebral*, both leading to *coma* and, it may be, to *paralysis*; both terminating, occasionally, in *mania* or *amentia*. See § 183.

235. Gentleman, I commend these views at once

to your indulgent consideration. I am persuaded I have taken a real step in the pathology of these dire and herculean affections. But if I have failed, I have failed in that which the celebrated Esquirol, after a life devoted to the subject, declared to be impossible!—"Les symptomes de l'épilepsie sont tellement extraordinaires, *tellement au dessus de toute explication physiologique*; les causes de cette maladie sont tellement inconnues, que les anciens ont cru qu'elle dépendait du courroux des dieux*."

236. The great fact is — that trachelismus, a spasmodic affection of the muscles of the neck, is the first, or rather the second, link in the chain of actions which lead to paroxysmal apoplexy or paralysis, or mania, as well as epilepsy and the epileptoid affections.

237. I think I need not insist further on this fact, so important in the pathology. And it is precisely the same fact which leads us into the true path of treatment.

238. May a fit of sickness and vomiting, timeously induced, be made to anticipate and supersede, and take the place, as it were, of a fit of epilepsy? How full of the deepest interest is this momentous question!

239. And then there is another question—When ought this emetic to be given?

240. There are, I believe, two periods when this

* Les Maladies Mentales, tome i, p. 274.

is proper. The first, is when an attack is imminent, as ascertained by premonitory signs; the second, when, without premonitory signs, we may be anticipating the attacks generally.

241. Another remedy of great moment, which may or may not be combined with the emetic, is a large dose of antacid, as twenty or thirty grains of the bicarbonate of potass.

242. Both emotion and gastric irritation are apt to induce excessive secretion of the hydrochloric acid in the stomach; and this, I suspect, is a frequent cause of attack. This cause is effectually removed by the antacid, which should be administered whenever any symptom, nervous or gastric, seems to call for it.

243. A rigid system of mental discipline, of diet, of gentle exercises, of attention to the alvine and the urinary secretions, and early hours, must be combined with these and any other remedies that may be deemed proper.

244. One of these, from which I think I have seen benefit, is the acetate of strychnia. The important question to determine is—what is the *tonic* dose of this remedy? I believe it has been generally given in a dose which is *stimulant*, and therefore injurious. From many trials, I am led to propose the fiftieth part of a grain, given thrice a day, as the proper dose as a tonic, and in cases of nervous exhaustion and susceptibility, and to propose the following *formula:*

℞ Strychniæ Acetatis, gr. i.
Acidi Acetosi, m. xx.
Alcoholis, f ʒii.
Aquæ distillatæ, f ʒvi. Misce.

Ten drops of this solution contain the medium dose of the remedy.

245. In all cases of what may be justly designated nervous exhaustion,—the effect of mental harass, of physical fatigue, of sexual excesses,—this remedy appears to me to be of great promise. And the susceptibility to paroxysmal seizures, at once their effect and their cause, is of this nature.

3.—*On Spasmo-Paralysis, and its Diagnosis.*

246. The *attack* of apoplexy or hemiplegia is sometimes complicated with convulsion or spasm;

247. The attack of epilepsy or convulsion sometimes leaves paralysis.

248. These two cases of spasmo-paralysis require to be accurately distinguished from those of pure spasm and pure paralysis. The former of these is, of course, spinal; the latter may be either purely cerebral or purely spinal; but spasmo-paralysis may be either spinal or cerebro-spinal. When the spasmo-paralysis is distinctly *hemiplegic*, I think it always involves both the cerebrum and the spinal centre.

249. When hemiplegia is complicated with convulsion or spasm, either in the attack or afterwards,

the cause of the hemiplegia—generally softening or rupture of the opposite hemisphere—is either complicated with such *tumefaction* as to affect the medulla oblongata by pressure or counter-pressure, or with arachnitis, with effusion at the base of the brain, affecting the medulla oblongata. In one deeply interesting case of this kind, the hemiplegia presented an exception to the general rule of augmented irritability in the paralytic limb. Whether this fact will be found in other cases of this kind, I do not yet know. But if it should, it will at once indicate a peculiarity in the pathology; for the paralysis must be more or less spinal, and suggest the diagnosis.

250. This last question applies to the case of paralysis left by the convulsive or epileptic seizure. Is it *spinal?* Is it attended by diminished irritability of the muscular fibre?

251. The attacks of paralysis which we so frequently observe in children, and refer to dental, or gastric, or enteric irritation, require special investigation in this respect.

252. The hemiplegia observed after the epileptic or convulsive seizure is sometimes entirely dissipated. In one case this event occurred after repeated seizures, the hemiplegia being rapidly evanescent in each. In another case, the hemiplegia, after severe epileptic or convulsive seizures, seemed, like those seizures themselves, of the most hopeless kind; yet it disappeared so entirely, that the patient, a seal-engraver, has recovered the perfect use of his fingers, as of the arm and leg.

253. The questions are—whether there be mere irritation or organic change;—whether there be mere intra-vascular, or extra-vascular derangement.

254. These two forms of spasmo-paralysis are strictly connected with the subject of these Lectures—paroxysmal seizures. But there are others which belong to a more extensive view of the subject, to which I can, of course, only advert in a few words.

255. First, chronic hemiplegia is apt to become complicated with spasm, the effect of *tone*, the acts of volition being suspended. This is generally seen in the closed and rigid hand, and in the arms.

256. Secondly, spasmo-paralysis is apt to supervene in chronic cases of paralysis agitans; and, in this case, strange to say, I think it is the effect of a sustained act of volition, of which the patient is unconscious. It ceases, on certain occasions, when the attention is drawn to another object.

4.—*Spinal Syncope.*

257. Sometimes, instead of the usual apoplectic or epileptic attack, there are sudden pallor, perhaps with sickness, faintishness, a clammy perspiration, &c.

258. This state of things may be the result of irregular circulation in the medulla oblongata, or the

effect of alarm; in the latter case, with or without previous flushing.

259. I have already compared this kind of apoplectic or epileptic affection to the state of things induced by the motion of a swing or of the sea, or by a blow or fall on the head.

260. The recumbent position and cordials are required. Otherwise, the treatment is the same as in the more ordinary apoplectic or epileptoid affections.

5.—*Hidden Seizures.*

261. This subject will be best illustrated by the following most interesting case:

262. At the close of 1848, I was summoned to see Mr. ——, of ——, aged about fifty, a merchant. I found him in a state of delusion in regard to his affairs. The other symptoms involved a bilious tinge of the eye and complexion, and the urine loaded with lithates, which led me, at that time, to the opinion that the condition of the brain and intellect might be the effect of disarrangement or defect of the secretion of the liver and kidney. I prescribed alterative doses of the mercurial pill and mild antacid aperients, and my patient soon recovered.

263. This amendment was not destined to be of long duration. Mr. —— suddenly relapsed, and became the subject of a violent maniacal paroxysm, of

considerable duration, and requiring a keeper. What was now the *precise* nature of the disease?—an anxious and difficult question in every case of mania. There was, on this occasion, no remarkable tinge of the eye or skin,—nothing very wrong in the secretions,—to account for the symptoms. Was the case arachnitis? This opinion seemed probable. It was treated with more decided mercurials and antacid aperients, with a spirit lotion applied to the head, and fomentations to the feet; whilst opium, in large doses, was given, at the suggestion of another, for the violence of the delirium, and apparently with good effect. The patient again recovered, less speedily, however, than before.

264. We were again doomed to be disappointed. The patient again suddenly relapsed; but now, instead of delirium, the principal symptom was a sort of amentia, or dulness of intellect; so that, as I had before suspected arachnitis, I now suspected effusion. We pushed our former remedies, the opium excepted, and the patient again recovered; and indeed, so little tardily, as to compel us to relinquish the idea of effusion.

265. It was after this event—after this third attack, in which, for a time, I suspected *effusion*, but which passed off too soon for effusion—that a new idea occurred to me, involving a new question; and on reconsideration of the whole case, I asked—Had there been a seizure, or rather seizures, of an epileptoid character, unobserved, in the night, or when the patient was from home? In a word, was it a case of hidden

seizures?—a question now, I believe, occurring in the practice of medicine for the first time; and of how great importance will, I think, shortly appear,—a question agitated most anxiously, not only by the physician, but by the most devoted of wives.

266. Indeed, it is to extracts from this lady's letters that I now beg your especial attention, as to an account of events, free from bias, and full of the deepest interest:

267. "The sad experience of the last two months (during which time I have witnessed several distinct convulsive attacks) has convinced me that Mr. —— has been subject to many seizures entirely *unknown* and unobserved, except in their effects. During the last week of February last he was in a state of great mental excitement—quite distressing to those around him. On the 1st of March, about noon, a sort of stupor came over him, to me quite unaccountable. We were walking at the time, and he had remained *perfectly silent* for at least a quarter of an hour before my attention was drawn to the altered expression of his countenance. This stupor lasted only a few—perhaps three or four—hours, but it was followed by great nervous excitement or mental agitation, almost bordering on delirium. I did not *suspect*, of course, the *real* cause of this—indeed, I looked upon it as another phase of his distressing illness. On the night of Saturday, March 3, Mr. —— retired to his room in a state of the greatest mental agitation. At one

o'clock he fell into an apparently sound sleep. At about half-past seven o'clock on Sunday morning, he arose from his bed, and began, as usual, to dress himself, or rather, he *tried* to dress himself. I was greatly surprised and alarmed to observe that a great change had come over him. His hand was feeble, his step was unsteady, his intelligent countenance had a vacant expression, and to my anxious and repeated enquiries he only answered by a movement of the head, to which I could attach no meaning. During that and the following day he remained in a deep stupor, only occasionally giving imperfect and indistinct replies to questions put to him. On Monday morning Dr. Marshall Hall saw him. He thought there must have been some attack of an epileptoid character; but nothing had been observed—nothing could be told. On Tuesday morning there was decided delirium, which lasted three or four hours. The same evening, in walking to and fro in the drawing-room, his hand, in which he held mine, was nervously contracted several distinct times, and his head gradually drooped till it almost rested on the shoulder. Shortly afterwards he was seized with a sort of shudder, which I thought arose from fear—a noise having been heard, which he said was 'loud thunder.' This attack, slight as it was, enfeebled yet more the hands and feet, and increased the stupor, but no delirium followed. This was all that could be detailed then to Dr. Marshall Hall, who made most anxious and minute inquiries on the subject.

268. "About the end of the month of March, Mr. ——, while sitting in his chair, fell asleep, no very unusual occurrence. I left the room to arrange some domestic matters, and Miss —— remained alone with him. On my return, she described what we both ignorantly believed to be the effect of a troubled dream, or an uneasy position, or both combined. Miss ——'s attention was first called to her brother by a slight gurgling in the throat. The lower lip had fallen greatly; the tongue, she said, moved 'most curiously from side to side,' and the eyeball was drawn upward; but in a few minutes all this passed away; the features resumed their former expression; and all this took place without any apparent interruption to the sleep.

269. "The first week in May we removed to ——. Within the short space of ten days after our going thither, I was distressed and perplexed to observe, that on two distinct occasions the articulation suddenly became slow and imperfect, the voice low and feeble, and on each occasion there was a loss of power, mental and bodily. But I had observed no seizure, neither did I suspect any. On the 19th of May I was standing talking with Mr. ——, and while he was in the very act of speaking, the mouth was suddenly drawn to the right side, the tongue became paralyzed, and the right hand was drawn inward. In great alarm (for this was the first *unequivocal* seizure I had ever witnessed), I took the hand and rubbed it, as I would have done for cramp, four or five minutes. While I

was doing this, all appearance of a seizure passed away, only the effects remained. For several hours afterwards the articulation continued to be slightly imperfect, the voice low, and the step feeble and unsteady.

270. "Within a week after this, just as we were finishing a game at Backgammon, Mr. —— had a similar attack, equally short in duration, but rather different in its effects. On this occasion, slight delirium followed, but the articulation was afterwards perfect.

271. "Both these seizures would have been entirely unknown, unnoticed, save in their effects, had my attention at the time been directed to any other object.

272. "In a few days after this, followed the severe and most alarming attack, which lasted four hours. Then succeeded another, and another, equally distressing, the effects after each attack varying very considerably. Thursday, July 26."

273. On one of these occasions this lady writes, "This morning my dear husband has unhappily had another of those dreaded seizures, which, though slighter than some of the previous attacks, has taken away the power of speech; and the right side is also paralyzed." On another she writes—

274. "I think I have in conversation once, or more than once, referred to the peculiar feeling, or rather absence of all feeling, in the right arm, which Mr. —— often felt on first awakening from sleep. It is about three years since he first complained of this; observing that his right arm must either be 'paralyzed

or benumbed.' Sometimes he complained of this on awaking in the morning, but I think more frequently when he awoke from the hour's sleep which he usually took every evening after dinner, when he had no guests at his table."

275. My *conjecture* must indeed have appeared extraordinary to every unbiassed mind, for it was soon —too soon, alas!—converted into *fact*, by the occurrence of seizures of no dubious or equivocal character.

276. The fourth serious attack was one of distinct epilepsy, leaving defective articulation, paralytic weakness of the hand, and imbecility of intellect, for a time, and then gradually but imperfectly receding.

277. Other seizures followed, open and unequivocal: these it is unnecessary to detail. My conjecture had become a sort of prediction fulfilled. My patient died, and a post-mortem examination was made, of which the following is the brief and imperfect detail:—

278. "The arachnoid membrane presented the appearance of opacity, with effusion of lymph beneath its surface. The brain, immediately beneath the arachnoid membrane, was remarkably firm, and contained an unusual quantity of blood. Three or four tablespoonfuls of serum were found in the lateral ventricles. No other morbid change was observed in the brain. No other organ was examined. September 23, 1849."

279. It now becomes an interesting question—

What are the probable effects of repeated seizures of the kind described on the delicate tissues of the brain and its membranes? May they be such as are described in this post-mortem examination?

280. The *first* effect is, doubtless, congestion. This may subside after the first and second attacks. But does it entirely subside after the third or fourth? May it leave lesion of tissue? And if so, of what kind? In the delicate tissue of the encephalon, may it have the appearance of arachnitis or of encephalitis? — effusion of serum or of lymph?—or softening or induration?

281. When, in cases of paroxysmal disease, such effects are found, who shall say, without years of special study and observation, whether, in fact, they be *causes* or *effects?*

282. But that in all such cases a most careful inquiry should be made, in regard to past 'hidden seizures,' there can be no doubt.

283. Nor does this question cease here. It may become a *legal* question; and, in another and terrible sense, a question of life and death.

284. A seizure—perhaps a hidden seizure—may take place, and leave a monomaniacal tendency to suicide or homicide. *Crime* may be committed, and no proof of previous insanity exist. Of such a case, the Law, hitherto, equally with Medicine, has taken no cognizance. This crime may be one involving loss of property, honour, life.

285. Such a case occurred recently at Greenwich. A nurse-maid rose from her bed, went into the kitchen, seized a carving knife, partially severed the head of her little charge from its body, and all this without detectible motive. She had been subject to some kind of seizure, supposed to be hysterical, but far more probably epileptic.

286. How fearful the consequences of such a state of things might be, I need not say; but certainly every means should be employed to detect such a hidden seizure in such a case; and especially the temples should be examined for ecchymosis; the tongue, for a bitten wound; the pillow, for marks of foaming at the mouth; and the linen, for the stains left by some evacuation; whilst the patient should be carefully interrogated, to detect the slightest incoherence or aberration of ideas, or confusion or defect of memory.

287. Under all circumstances of sudden crime, the possibility of the occurrence of a seizure should be present to the mind; how much more, if the patient have been epileptic, or if the case be *puerperal!*

288. But, to return to the medical view of this subject, and the case before us: let us bear in mind that the diagnosis is every thing in the practice of medicine; and that we have, in diseases of the head, sometimes to trace the affection to deranged function of remote viscera; sometimes to detect an original organic disease of the encephalon; and sometimes to

trace the symptoms to a previous, but unobserved, and therefore hidden, paroxysmal seizure.

289. The observant Heberden remarks: "Qui semel occupatus est gravi paralysi, sæpe experitur leviores morbi accessiones, quæ, cum noctu, vel per quietem invadant, facile *latent* eos, qui ægrotis famulantur. Harum vero justissima erit suspicio, ubi ea mala, quæ secuta sunt accessiones priores, denuo intra paucas horas plurimum ingravescunt*."

6.—*Paroxysmal Mania.*

290. I have known a maniacal paroxysm to follow an epilectic attack. I have just described a case in which a violent maniacal paroxysm followed what afterwards appeared to have been a *hidden seizure.* I have had occasion to watch a case in which a paroxysm of mania came on at uncertain intervals, after a prolonged and perfect 'lucid interval,' and was superseded by the well-timed administration of emetics.

291. May we not infer from these facts that mania is frequently a paroxysmal disease, holding the place, in regard to other cases of mania, which paroxysmal apoplexy does to organic apoplexy? And does not this view suggest the propriety of the repeated administration of emetics in such cases of mania?

* Op. cit. p. 296.

292. The paroxysm may be excited, like that of paroxysmal apoplexy and inorganic epilepsy, by emotion or gastric irritation. Some source of exasperation may have occurred, or some improper article or quantity of food may have been taken, or gradual load of the stomach or bowels may have taken place—may have proved the source of trachelismus, and this of a hidden seizure, and this, in its turn, of mania. The mind must be kept tranquil, the diet must be of the most digestible kind, and the bowels must be kept well moved daily; in addition to which, an emetic should be given at stated intervals, or on the occurrence of any symptoms threatening an attack.

293. Some cases of mania assume decidedly the paroxysmal form, subsiding entirely in their 'lucid intervals.' Others continue without absolute intermission, but experience paroxysmal exacerbations.

294. In some cases these paroxysms have been distinctly traced to intemperance in diet. In the case to which I have alluded, the attack, which had usually returned after the space of four or six weeks, has been warded off by weekly emetics for sixteen weeks! These emetics consisted of two grains of the tartrate of antimony, mingled with the patient's tea, unknown to him, when he had been observed to commit an error in his diet.

295. It will be remembered that mania is apt to follow an apoplectic, paralytic, epileptic, or convulsive affection; and I need scarcely again advert to the

case of hidden seizure just detailed. Mania forms one of the *Class* of paroxysmal cerebral and spinal diseases. It may arise from mere vascular distension, the effect of such a seizure.

296. In paroxysmal mania, as in paroxysmal epilepsy, I am persuaded that there is the same pathology in trachelismus, and the same hope of successful treatment from emetics, or from emetics, antacid aperients, and mild alterative mercurials combined.

297. I am persuaded too that this form of mania, at least, admits of remedy more frequently than is supposed; and we have still to discover the rationale of other forms of insanity.

298. These cursory remarks must be viewed as merely suggestive. The subject must be carefully investigated. But I have long meditated the institution of an Asylum appropriated to cases of short duration, the stay of which within its walls should be duly limited. Each of these limited periods might be *one year*.

CONCLUSION.

299. Whatever may be the exciting cause or causes of paroxysmal cerebral and spinal seizures and their mode of operation, the following events must be involved in them:

1. They must be capable of inducing and ex-

plaining flushing of the countenance, ecchymosis, epistaxis, &c.

2. They must be capable of inducing and explaining the *venous hue and turgidity* both of the *face* and of the *neck;*

3. They must be capable of inducing and explaining both *cerebral* and *spinal* symptoms;

4. They must be such especially as will explain the ready transition of the *cerebral* into the *spinal* epileptic seizure; *see especially* § 232;

5. They must admit of accession and recession in a moment of time;

6. They must admit of assimilating the latent with the evident spasmodic conditions of the muscles and veins of the neck, with their ulterior effects;

7. They must involve the cause and influence of *Sleep*, the influence of the *Emotions* and of the *Irritations;*

8. They must admit of inducing and explaining the morbid anatomy, and especially the transition of mere cerebral congestion into effusion, rupture, and softening; &c. *See* § 188.

300. It is no unusual occurrence to meet with cases in which the slight attack with cerebral symptoms only, and the severer attack with spinal symptoms, take place variously in the same patient, the former sometimes passing into the latter,—convulsion, torticollis, laryngismus, and the bitten tongue, being superadded to unconsciousness, with a flushed countenance, dilated pupils, &c. These are obviously different degrees and phases of the *same* affection. But in the severer case, the trachelismus is *obvious*. Can it be doubted that it exists equally in the milder, although *latent?*

301. But, in other cases, the milder form of threatening or seizure consists in giddiness, loss of consciousness, the fear of falling, or a momentary loss of power of the articulation or of the hand; whilst the severer seizure is decidedly apoplectic and hemiplegic.

302. In a third class of cases, the epileptic seizures themselves gradually assume more and more of the apoplectic and hemiplegic forms.

303. All tend to impair the memory or intellect; the first attacks may be followed by mania, and repeated attacks, by amentia and general paralysis, in various degrees.

304. A momentary trachelismus and phlebismus, with *congestion*, explain the transitory and milder seizure; a severer congestion, with greater intra-vascular distension, explains the severer seizure, from which recovery still takes place speedily and without any permanent effects; or which, if fatal, leaves no lesion, except congestion, detectible on a post-mortem examination; when, to intra-vascular congestion, ecchymosis, or extravasation of blood, or the effusion of serum, supervenes, we witness the sad and permanent effects of the same trachelismus and phlebismus, either partial or general.

305. Thus the chain of cause and effect, or effects, appears to me complete.

306. All this, and much more, is accomplished by the doctrine of *Trachelismus*. I think, therefore, I am justified by bringing it before you, and commending it to your notice.

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307. And here, Gentlemen, I must bring these Lectures to a close. It has been my anxious wish to lay before you, in facts, and in the words of others, as much as possible, the argument for the institution of a *Class of Cerebral and Spinal Seizures*, with their rationale, diagnosis, prevention, and treatment. How imperfectly I have done this, I am well aware. But I trust that the attempt will be received by you with candour and generosity.

308. Allow me to thank you, Sir, once more for the opportunity you have kindly afforded me of bringing the subject before this College; and you, Gentlemen, for your kind attention during the course of my imperfect observations.

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NOTES.

Note to § 20—29.—The same remarks which are here applied by Abercrombie to apoplexy and paralysis, may be applied with equal truth to epilepsy, to the various forms of convulsion, and to mania.

The morbid appearances may be of the same negative character, or consist merely in distended vessels, or effused lymph or serum.

Note to § 108.—Mr. L——, of S——, aged 50, consulted me for the following affection:—He was liable to be taken with loss of speech, and loss of power of the right hand, and, on riding in his chaise, with loss of power of the side. At these times he felt the sensation of '*strings*' drawn tightly along each side of the neck, with a '*rush of blood*' up the neck and cheeks, with dimness of vision, deafness, vertigo, &c.

Mr. R——, aged 50. In this gentleman the whole face was of the deepest red, the everted under eye-lid presented the appearance of a deep venous congestion, and the veins of the forehead were largely distended. He too described a sensation of '*dragging*' on each side of the neck.

In August last, on walking across a court yard, he was seized with giddiness, and was in danger of falling. In October, he lost the power of the right hand, and did not regain it in a fortnight, nor even afterwards perfectly. Once, on awaking, he felt 'as if he was going to have a fit.'

By cupping, daily antacid aperients, abstinence from all stimulants, a simple diet, a raised position in bed, an alcoholic lotion applied to the head, and attention to preserve the feet warm and dry, Mr. R—— was effectually relieved.

Of this case Mr. Prescott Hewett kindly took the following note:

"On examining the patient, Mr. ——, whom I saw with you, I found that the whole skin of the face was minutely injected, and of a scarlet colour. The conjunctivæ of both eye-lids were also intensely injected throughout, and of a deep red colour. The right hand, the power of which had been, at times, partially lost, was weaker than the left; but the corresponding leg was not affected.

"The following was the history given by the patient. Frequent swimming in the head, especially when stooping; extreme heat of the face upon first lying down; also headache in the recumbent posture, which frequently disappears on rising. Studying, or application of any kind, also causes swimming in the head, and, on one occasion, sickness. At night, when in bed, the hand frequently becomes weaker than usual. At times, strange sensations on both sides of the neck, as if of strings passing upwards on both sides towards the head. Last week, frequent pain down the thigh and leg, like cramp flying about, but for a very short time. Two or three days before I saw him, he had suddenly awoke in the night with the idea that he was about to have a fit. The swimming in the head was at the time very distressing, and the strings, as he called them, on the sides of the neck were very painful."

Note to § 168.—It is important to observe, that, whilst in paroxysmal apoplexy the trachelismus is latent, in severe epilepsy it is first latent and then evident.

In cerebral epilepsy, the trachelismus is, as in paroxysmal apoplexy, latent. The *head* and eyes are fixed, the face flushed, and the pupils dilated, and nothing more. To this state evident trachelismus may, or may not, supervene. In the former case, it is manifested in the form of laryngismus, odaxismus, torticollis, &c.

The effect of a ligature applied round the neck is, according to its degree and duration, that of the latent or that of the evident trachelismus. How fearful is the following short account of the poor girl, Jael Dennys, by the eye-witness, Elizabeth Hammond, given in *The Times* for March 8th, 1851:—"I helped to undress her. I observed that her face was very black and swollen, that her mouth was bubbling with blood, and her tongue protruded from it and clenched very tightly by her teeth. Blood was also oozing from the nose, the eyes, and the ears. When I took off her clothes, I saw that her body, from the waist to the shoulders, was very black indeed, and her neck was quite lacerated by the cord through the skin."

The first effect of a cord tightened round the neck, is the same as that of trachelismus, or the apoplectic state; and Mr. Williams well observed, in the case from which I have made the foregoing extract, that "the effect of the pressure by the first coil of the rope upon the trachea" (the neck rather) "must have been immediate insensibility, and that it was impossible for her to have made two other coils of the rope round the neck afterwards."

The epileptoid state is a subsequent effect—an effect of a severer application of the cord (which, I think, could scarcely be induced by the mere force of the hands of a suicide), or of a later stage. It is in this manner that, to the apoplexy, convulsive phenomena, the protrusion of the tongue, and the closure of the maxillæ, are superadded.

In the same manner convulsion is occasionally superadded to apoplexy, and spinal supervenes on cerebral epilepsy. They are different phases or degrees of one and the same morbid affection.

Mania may supervene on both, or either; and amentia, if the seizures be repeated, or the induced condition be severe and long continued.

Note to § 169.—I omitted to state, in its proper place, my views respecting the morbid anatomy in paroxysmal diseases. The morbid

appearances found on a post-mortem examination are apt to be viewed as the disease or as the cause of the disease. They are, in reality, its *Effects*. Fulness of the veins and of the intervening blood-channels placed between these and the arteries, red points, or points of ecchymosis, the effusion of a clot of blood, the consequent softening, the effusion of serum, the presence of fibrine in the arteries, are *all* the effects of repeated congestion,—the effects and not the causes of the original malady, though the causes, in their turn, of subsequent symptoms. Of these symptoms, I may observe that the local softening is the cause of partial paralysis, whilst the general effusion of serum is frequently the cause of amentia and of general paralysis.

The effusion of serum is seen in the ventricles and under the arachnoid of the surface, and at the base of the brain. In some instances the arachnoid is raised by the serum into the form of a vesicle. In others, the plexus choroides is affected in a similar manner, and a vesicle or cyst is seen to occupy one part of it.

These views, in regard to the morbid anatomy of paroxysmal diseases of the cerebral and spinal systems, are of the utmost moment. We have too long been in the habit of concluding that such morbid anatomy is the disease; and in this manner even the most positive department of medicine has led us into error. These very appearances must be *interpreted*, and that—by the *physiology*.

It will be interesting to ascertain whether there be any difference between the post-mortem appearances in paroxysmal apoplexy and epilepsy. I believe there is none,—a further proof of the nature and identity of the causes and rationale of these diseases.

Note to § 188.—It becomes a most interesting question—What are the precise conditions of the arteries and veins after repeated paroxysmal seizures? I imagine the deposit of fibrine, frequently found in the arteries, not unfrequently the *effect*, rather than the *cause*, of softening of the brain, and perhaps of other tissues, organs or limbs; and even, in some cases, of gangrene.

The difference between Apoplexy and Epilepsy is the difference between trachelismus and laryngismus, jugular and vertebral vein, cerebrum and medulla oblongata.

Heberden observes—"Paralysis et apoplexia sunt tantum diversi gradus ejusdem morbi*." This is true in a certain limited sense, especially in the *paroxysmal* forms of these diseases. But, in the same sense, not only apoplexy and paralysis, but these and epilepsy, and mania, are one and the same disease, differing in degree. But whilst apoplexy affects the cerebrum, and paralysis a hemisphere,—epilepsy affects the medulla oblongata, and mania again the cerebrum.

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1. *Apoplexy,*
2. *Paralysis,*
3. *Epilepsy,*
4. *Convulsion,*
5. *Mania,—may each be arranged into—*

Class I.
1. *Of Inorganic Origin;*
2. *Of Recurrent Paroxysmal Form;*
3. *Of short Duration, terminating in perfect recovery, or Fatal,*
4. *Without post-mortem appearances, or*
5. *With such as are Effects only.*

Class II.
1. *Of Organic Origin;*
2. *Of Permanent Form.*

Of the whole doctrine of *Trachelismus*, there is not the shadow or the possibility of a doubt, as far as *Inorganic Epilepsy* is concerned. But the paroxysm or paroxysms of inorganic epilepsy lead

* Op. cit. p. 285.

to apoplexy, to paralysis, or to mania. Nay, the milder form (*'le petit mal'*) of epilepsy *is* cerebral or apoplexy, the trachelismus being *latent*, however it may become evident enough when this passes into the severer or spinal form, or *'le haut mal.'*

Apoplexy and paralysis have been paroxysmal and recurrent, receding entirely in the intervals, for years,—in one case, for twelve years. The peculiar form of mania of which I am treating is characterized by its 'lucid intervals.'

Every fact conduces to the view that these cases should be separated from such as are of *organic origin*, arranged together, and connected together. 'Le petit mal' itself is sometimes, as Heberden beautifully states, '*oblivium*' or apoplexy, and sometimes '*delirium*' or mania; *see* § 6; the next stage being convulsive epilepsy.

And what a ray of light is thrown upon the post-mortem morbid appearances in some cases, and their absence in others!

In speaking of the subjects of these Lectures, we may now speak, not of apoplexy, paralysis, epilepsy, mania, but of *nervous seizures*, assuming an *apoplectic*, *paralytic*, *epileptoid*, or *maniacal* form; and thus our diagnosis will be implied in one designation.

AN

INAUGURAL ESSAY

ON

ZOO-ADYNAMIA,

PRESENTED FOR THE DEGREE OF DOCTOR OF MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA.

BY

GEO. J. ZIEGLER, M. D.

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PREFACE.

In presenting this essay to the profession, the writer is influenced by various considerations, the most important of which is the desire to be useful. He hopes to excite upon the subjects discussed more direct attention and deeper reflection than they have hitherto received, more with a view to the establishment of correct pathological principles and treatment than a desire for hypothetical or theoretical speculation. He has endeavored to arrange and classify a great number of morbid conditions, and to exhibit the fact, which appears to be very much overlooked, that there are many diseases induced by or dependent upon different pathological conditions, and thus do away with the prevalent idea of the existence of specifics for such diseases, as, for instance, epilepsy, chorea, &c.

In examining this essay, the reader will find that frequently the same disease is placed in two or more and sometimes all of the divisions therein made, as being dependent upon different pathological changes, and hence require a diversified and varied, and, in many cases, totally opposite course of treatment.

The subject affords an opportunity to the writer to call attention to a much-neglected remedial agent, which has not received that favorable consideration in the treatment of disease to which he believes it to be entitled, viz., *Nitrous Oxide Gas;* and he has pointed out some of the deranged states of the organism to which it appears to be well adapted, and in which he believes it will be found highly useful.

In consequence of the necessarily limited extent of an inaugural essay, the writer can merely point out examples of the conditions to which he refers, leaving further illustrations to be supplied by the suggestions of the mind of the reader.

It is not pretended that all the suggestions or opinions which are advanced in the following pages are supported by the test of experience, opportunity not having yet been afforded to substantiate them; still it

is believed that the more closely the views are examined and made the subject of practical inquiry, the more manifest will be their correctness.

The writer has been induced to attempt an arrangement and classification of the abnormal conditions which are presented, from the confusion and obscurity in which they are found in most authors; and, if this attempt is at all successful, the main objects of this essay will be attained, believing that, as much error has prevailed in reference to the pathology of the diseased actions in question, much benefit must accrue from a removal of such error, and thus a more correct knowledge will be afforded of the method of subverting them.

PHILADELPHIA.

INTRODUCTION.

In selecting this extensive, interesting, and highly important subject, viz., Zoo-adynamia, for an essay, I have done so, not so much with the expectation of suggesting or adding anything new as for the purpose of drawing the attention of the profession more particularly to its importance, and thus induce them to undertake a full and complete exposition of it, so far as a knowledge of physiology, pathology, and therapeutics will admit; and, in this manner, not directly, but indirectly, I hope that my humble effort may result beneficially to "suffering humanity."

REASONS FOR MODIFYING THE TERM "ADYNAMIA."

This term, "adynamia," is employed to designate the deficiency or privation of vital or animal power; but, if the etymology of it is examined, it will be found to be too general in its application, and may be understood as being applied to the deficiency or privation of either mechanical, physical, chemical, or vital power. In adding a prefix, therefore, I do so from the conviction of the inadequacy of the word alone to express the idea really desired and intended in its employment in medicine; and, to make it more specific in its signification, I would, therefore, suggest the prefix zoo from zoon, thus making it zoo-adynamia, signifying privation or deficiency of animal or living power.

ZOO-ADYNAMIA.

Zoo-adynamia, a ζωον, anima or life, α, privation; and δυναμις, force or power.

Zoo-adynamia may be divided into psychico-adynamia, and neuro-adynamia; the first from ψυχη, soul or mind, α, and δυναμις, thus meaning a deficiency or privation of mental power; the second, from νευρον, nerve, α, and δυναμις, deficiency or privation of nerve power.

Psychico-adynamia and neuro-adynamia may be local or general, partial or complete, temporary or permanent in their character, and may result from, 1st. Modification of structure; 2d. Interference with function without modification of structure; 3d. Inanition; and, 4th. Sympathy. All of these may be very sudden or very gradual in their occurrence.

1st. PSYCHICO-ADYNAMIA.

As this division for a proper elucidation will require more time, space, and knowledge than I possess, I will pass it, and confine my attention more particularly to that division which I have denominated neuro-adynamia, in which there is more or less connection with the first division; for there may be derangement of the mind acting upon and deranging the nervous system, or the mind may become implicated with or be acted upon by the nervous system.

2d. NEURO-ADYNAMIA.

This may be subdivided into sensory and motor adynamia; the first, still further, into general sensory-adynamia and special sensory-adynamia; the second, into voluntary motor-adynamia, and involuntary motor-adynamia, thus—

NEURO-ADYNAMIA.

A. *Sensory-adynamia.*
B. *Motor-adynamia.*

A. SENSORY-ADYNAMIA

a. *General Sensory-adynamia.*
b. *Special Sensory-adynamia.*

B. MOTOR-ADYNAMIA.

a. *Voluntary Motor-adynamia.*
b. *Involuntary Motor-adynamia.*

For greater conciseness and convenience of description, and to prevent too much repetition, and also on account of the comparatively unfrequent occurrence of one of these forms of adynamia, uncomplicated with any other, I will place these divisions in the two groups of general and local neuro-adynamia, in which the different sensory and motor derangements will be readily recognized; comprising under the first head both sensory and motor-adynamia of the whole system, whilst under the second will be included the various forms of local sensory and motor-adynamia.

GENERAL NEURO-ADYNAMIA.

With general neuro-adynamia, we have often complications of local neuro-adynamia, some forms of which most generally precede the general attack, unless it should be very sudden, when all the nervous functions fail simultaneously; and, if the general attack does not prove fatal, it will be very apt to terminate in some variety of local adynamia. Thus, frequently the sequela of general neuro-adynamia, when not fatal, is local neuro-adynamia, except in those cases of simple syncope, &c., where the prostration is very temporary, and does not materially affect either the functions of the brain or spinal marrow, unless to debilitate them, and in this way produce a more perfect state of general inanition.

1. MODIFICATION OF STRUCTURE.

This may consist of, first, actual change of structure, as softening or hardening; second, increased or deficient development, or diminution of nervous matter by wasting or absorption after development, as in hypertrophy, atrophy, &c.; and, third, solution of continuity from mechanical causes. Softening as the result of inflammation is of very common occurrence, and is often seen in the inflammations of the different parts of the brain and spinal marrow and their membranes, denominated according to the part affected. There is also a species of softening or ramollissement not dependent at all upon inflammation, viz., that in which there is a deficiency of nutritive materials, or in which the materials for the support and development of the nervous tissue are appropriated to supply an excessive secretion or drain. The effects of the first may sometimes be found in the brain and spinal marrow; the second is frequently the result of masturbation or excessive venery, and may also be found in the spinal cord, &c. This latter would tend to indicate a similarity between the particles of the spermatic secretion and neurine, or that the constant impressions transmitted to spinal centres cause an inflammation or inanition of the centres impressed, which terminates in softening.

Induration is also the frequent result of inflammation from the excessive deposit of plasma or coagulable lymph, which is sometimes effused, and by which the inflammatory action is frequently arrested. It may also be caused by an interstitial deposit of tuberculous or other morbid matter, or from hypertrophy, giving rise to various derangements, such as epilepsy, chorea, convulsions, &c. It is also the result of the consolidation of old age.

Atrophy may be congenital, or the development of the brain and spinal marrow may be impeded or arrested after birth, or there may be diminution of nervous substance from absorption or consumption; as in pressure from tumors, hydrocephalus, excessive mental and physical labor, &c.—the latter frequently producing the incurable forms of epilepsy and other convulsive diseases, either from atrophy or some modification of nervous tissue.

Hypertrophy is the result of increased development from increased nutrition, induration or greater consistence being the consequence.

Solution of continuity of nervous substance may be produced by effusions of fluids, as blood, serum, &c., as seen in apoplexy, dropsies (active and passive), &c.; by blows and injuries of various kinds, either by a crushing operation, such as is exhibited in compression of the brain and spinal cord, or by a more direct separation of nervous matter from a sabre cut or similar instrument or means.

Treatment of Modification of Structure.—This will depend upon the cause, extent, and position of organic change, and the effects or symptoms resulting from the same, with the length of time of continuation of these symptoms. But it must be admitted that there is not much probability, even in the most favorable cases, except where a limited quantity of cerebral matter is removed or discharged, of effecting a permanent cure; death, preceded by convulsions and coma, generally resulting sooner or later; and in many cases it is immediate—as in apoplexy, compression of the brain, &c.—or the patient may remain in an adynamic condition for a long period, and ultimately die of some other disease.

The treatment of softening will mainly consist in supporting the vital powers; but before this change takes place it may be prevented in many cases; as, for instance, in inflammation by antiphlogistics; in deficiency of nutrition by increasing nutritive materials; and from excessive drainage of spermatic secretion, &c., by arresting and preventing that drain, this being obviously dependent in a great measure upon the will of the patient.

In those cases of induration from effusions or hypertrophy, exciting absorbents by means of depletion, mercury, iodine, low diet, &c., may cause a removal of the deposited matter, and thus relieve or cure the condition dependent upon that deposit.

That from interstitial deposit of tuberculous and other morbid matter will probably be most benefited by alteratives and tonics, such as cod-liver oil, preparations of iodine, iron, &c., and most of the mineral and vegetable tonics, nutritious diet, fresh air—in fact, strict and prolonged attention to the hygienic laws.

In congenital atrophy, it is obvious that not much can be done, although by increasing the vital powers the deficiency may possibly be ultimately supplied to a certain extent. In that dependent on pressure of tumors or liquids, the treatment is limited to the removal of the offending substance, which can only be effected, if at all, through the general system, and by means of absorption induced by discutients, alteratives, and at the same time corroborant treatment, blisters, cathartics, diuretics, &c., although the liquid in many cases might be removed by the mechanical expedient of tapping.

In most cases of solution of continuity there is no time for treatment, death being instantaneous; and in those cases which do survive, success depends, in a great measure, upon the extent and position of organic change; although death is frequently the consequence of injury of a very small extent of surface in apparently a most favorable position. The proper plan to pursue would be to ward off the tendency to inflammation by strict antiphlogistic treatment, and the avoidance of all things which might tend to excite the brain or spinal marrow. Many cases are recorded in which part of the brain has been removed, either by the injury or the hands of the surgeon, and yet the persons have recovered, in consequence, most generally, of a strict adherence to the above-mentioned plan of treatment, to the truth of which in one case at least the writer can testify, which was in the Pennsylvania Hospital, of a young man who was kicked by a mule over the eye on the supra-orbital ridge, crushing the bone and causing the escape of cerebral matter; and who, from the enforcement of this plan by Dr. Fox, completely recovered.

2. INTERFERENCE WITH FUNCTION WITHOUT MODIFICATION OF STRUCTURE.

This is generally mechanical in its nature, arising from a great number of disturbing substances or causes, and is exhibited in a great variety of derangements; consisting in compression of the brain and spinal marrow, or upon different and limited parts of the same, from simple congestion, effusions of blood, serum (active or passive), coagulable lymph, purulent matter, tumors of various kinds, hydatids, compressed bone, as in fracture of the skull, spiculæ of bone, &c.; with the consequent production of apoplexy, epilepsy, catalepsy, chorea, trismus nascentium, paralysis, convulsions of different kinds or degrees, stupor, coma, and death.

Treatment of Interference with Function without Modification of Structure.—In many of these conditions, the indications for treatment are evident, but not, therefore, by any means, always successful. These indications are for the removal of the compressing fluid or body, the correcting of the tendency to a return, by changing the action, and the improving and strengthening the tone of the tissues and general system.

This may sometimes be effected, in compression from congestions and effusions, by bleeding, general and local, cathartics, diuretics, diaphoretics, vesicants, counter-irritation, absorption by means of mercury, when there are no contra-indications, as in the passive dropsies, viz., hydrocephalus, &c., these depending frequently upon a condition of system which scarcely permits of bleeding or mercury at all; and it is only in the active or acute forms, frequently preceding and often terminating in this condition, that they are obliged to be resorted to as the lesser evils; hence another class of remedies are employed, medicinal and mechanical, viz., the preparations of iodine, with tonics and vesicants, cathartics, &c., and the mechanical evacuation of the fluid, by means of compression and tapping, and trephining if the effused fluid be blood. Where tumors are suspected, either one of these plans, except the mechanical, are employed, according to the indications. If they are of an inflammatory character, antiphlogistics, although with caution, omitting mercury; if of scrofulous character, and the patient anæmic, iodine and its preparations, the chalybeates and other tonics, with corresponding hygienic measures.

In those cases dependent upon compressed or fractured bone or spicula of bone, elevation or removal of the bone, by means of the trephine, forceps, elevator, saw, &c., with a subsequent enforcement of the antiphlogistic treatment to its fullest extent, except where there is so much

debility that it cannot be carried into effect, is the course most favorable to recovery.

3. INANITION.

The conditions of system included in this division are very numerous, and present an almost infinite variety of modifications and complications, which may be comprised under the two heads of *Lesions of Nutrition* and *Lesions of Function*. These are frequently so conjoined that it is difficult to determine which is the primary derangement, as in most cases the one accompanies or speedily follows the other.

1st. *Lesions of Nutrition.*—Instances of these may be seen in the sudden drainage which takes place in epistaxis, hemoptysis, hematemesis, menorrhagia, uterine hemorrhage, hematuria, bursting of heart or aneurism, severing of vessels; in fact, hemorrhage from any part and from whatever cause; drains from excessive or perverted secretion and diseased action, as in diuresis, diabetes, diaphoresis, colliquative sweats, seminal evacuations, as in masturbation, excessive venery, nocturnal emissions; blennorrhœa, in its most extensive signification, cholera Asiatica, cholera morbus, cholera infantum, dysentery, diarrhœa, leucorrhœa, gonorrhœa, pus from suppurating surfaces and abscesses, serous evacuations, as in the dropsies (active and passive); bad organization and malformation, either congenital or acquired, as is seen in the scrofulous and tuberculous diathesis, anencephalous and other monstrous productions, insufficient nourishment, either from incapacity of digestion, as is seen in the different forms of dyspepsia, or a partial or entire privation of food, as in starvation, or of certain kinds of food, with the consequent production of certain diseases, such as scurvy, purpura, &c.; inflammatory affections, in the synocha variety, being most marked in convalescence, in the synochus, before and during that period; poisons which produce, as a primary effect, inflammatory action with subsequent prostration, as some of the mineral preparations, of arsenic, mercury, antimony, silver, copper, iron, zinc, lead, &c.

2d. *Lesions of Function.*—These supervene upon mental emotions, excitement, anxiety, &c., affecting communities and individuals, as is shown particularly during and after periods of great excitement or distress, as in sieges and epidemics; upon great mental or physical labor, or both combined, and in degree from the mere fatigue necessarily dependent on an amount of exertion requisite to healthful vigor and repose to that of complete exhaustion and death, or by disturbing the vital harmony give rise to various disorders, which, immediately or ultimately, rapidly

or gradually, terminate in dissolution, such as apoplexy, epilepsy, catalepsy, hysteria, chorea, convulsions, debility, temporary or prolonged, syncope, stupor, and coma, and sometimes sudden death—non-arterialization of blood thus preventing the nervous system from receiving its stimulus, oxygen, instances of which are seen in apnœa from suffocation, drowning; impurity and vitiation of air, as in mines, sinks, wells, &c.; from the burning of coal or other fuel in close places; from large assemblages or numbers of persons in confined or badly-ventilated apartments, halls, cells, or other places (a striking historical example of which is the celebrated "Black Hole of Calcutta"), or few persons, or only one person, in a close cell or other place. The asphyxiated condition of newly-born infants, and the imperfect oxygenation of blood of infants and children, and sometimes older persons, termed "cyanosis." Upon poisons, with the production of the adynamic fevers, such as the intermittent, remittent, yellow, typhus, congestive, &c.; poisoning from bites of rabid and venomous animals, as hydrophobia from rabid dogs and other animals; from serpents, insects, &c.; dissection and other poisonous wounds; narcotics, including under this head, for brevity, all of the cerebral and nervous stimulants and sedatives, such as alcohol, opium, aconite, belladonna, conium, digitalis, tobacco, chloroform, hydrocyanic acid, oxalic acid, strychnia, &c. The preparations of lead, arsenic, mercury, &c., in the production of tremors, or palsy. The habitual use of alcohol, opium, tobacco, &c., producing a species of delirium or tremors, somewhat analogous, as delirium tremens, simple tremors, &c., with sometimes great prostration of both mind and body. The retention in the circulation of excrementitious matter, such as bile, urea, carbonic acid, &c. The remarkable prostration attending coup de soleil, influenza, peritonitis, simple or complicated, erysipelas, &c.; prostration, in fact, preceding all fevers or inflammatory affections, there being a primary adynamic condition, the resulting fever seeming to be an excessive reaction, on the principle *ubi irritatio ibi afflurus*. Injuries—compression and concussion, in every degree, of the brain and spinal marrow, from injuries and wounds of different parts of the body, such as from falls, blows, bruises, &c., of various kinds; operations, as amputations, excisions, &c.; from gunshot wounds, &c., with the production of that remarkable condition termed the shock, analogous to that from electricity, in the form of lightning, &c., blow on epigastrium, draughts of cold water, burns, &c. The removal of the mechanical support or pressure from the nervous system, as seen in syncope, from the sudden arrest of the heart's action; by the removal of large quantities of fluid and solid from off the vessels and nervous system, as seen in the with-

drawal of fluid from abdomen by paracentesis abdominis; sudden extrusion of child from uterus, and by the sudden escape of fluid from sac of spina bifida, either by bursting or tapping; from cranium in hydrocephalus; and from bursting of an aneurism, or in sudden hemorrhage or liquid evacuation from any part of system, though of course the greater effect is produced, in such cases, by the withdrawal of the nutritive and stimulating materials. The exposure to changes of temperature, as cold or heat, in the first producing a state analogous to the stupor from alcohol, and resulting generally in a state of mild or great sedation; but if continued or prolonged, even terminating in permanent stupor, coma and death. The exhaustion, or rather non-production or development of nervous or vital force or power, from want of action or exercise, with the production of that ennui or lassitude so often exhibited in persons of indolent habits or wealth. The debility of old age from the wearing out of the machinery of life, and the consumption of the materials for the production of vital force, &c.

Treatment of Inanition.—The treatment of this condition or conditions will be as various and changeable as the diseases included under this head, and modified according to the type and peculiarities of the morbid condition and patient, although there are some general principles to guide us in our treatment and efforts to ameliorate and modify the abnormal actions and tendencies. Thus, under the head of "Lesions of Nutrition," in drainage from the system, of whatever kind and character, the first indication is to arrest that drain, or rather, in many cases, allay or cure the condition upon which it is dependent, except in some cases where that drainage is a succedaneum, or has become so, as in vicarious menstruation, old suppurating surfaces, &c.;—and thus within itself relieves or cures the deranged action.

In the hemorrhages, the treatment must be varied according to their active or passive character, as in fact it must in all diseases: in the former, it may be necessary to deplete the general system; whilst in the latter, unless the patient is plethoric, which is not generally the case, depletion will, by weakening and relaxing the tissues, assist the hemorrhage; hence it is necessary to resort to styptics, and, at the same time, very frequently to stimulants and tonics, and the class of remedies indicated are those which will produce thickening and coagulation of the albumen and fibrin of the tissues and blood; others to produce contraction of, and give tonicity to, the tissues; and others to diminish or prevent arterial and nervous excitement. Also quiet, rest, position, and temperature must be included as auxiliaries. The remedies thus

indicated are the empyreumatic oils, as creosote, naphtha, turpentine; the preparations of iron, lead (acetate of lead preferred), silver, and sometimes the actual cautery where the hemorrhage is local, ipecacuanha, ergot, matico, alum, tannin, and astringents generally; also cold and warm applications, arterial sedatives, and anodynes.

But it will generally be better to combine several of these remedies to fulfil the various indications; thus, in hemorrhage from any part or organ of the system, in debilitated patients, or persons of feeble vital powers and lax tissues, a combination of an astringent or styptic with an anodyne, and an agent to arrest and hold in check the excessive action of the vital pump (viz., the heart), will be found in most cases to answer the desired purpose of arresting the effusion without debilitating the patient. In hemoptysis, occurring in the above-described persons, I have seen no formulæ more philosophical, or, as far as my experience goes, more successful, than one by my preceptor, Dr. George W. Patterson, of this city (Philadelphia), viz :—

℞.—Plumbi acetat. gr. xij;
Pulv. opii,
Pulv. digitalis, āā gr. ij;
Misce ft. pil. No. viij.
Signa.—One every two hours till the hemorrhage ceases.

The proportions may be increased or diminished to suit cases. The acetate of lead, comprising the three properties of a styptic, astringent, and sedative, thickens and coagulates the blood, contracts the tissues, and allays excitability; the opium quiets the general nervous disturbance, and also assists in stimulating the tissues to contraction and supporting them in that condition; and the digitalis arrests and holds in check the heart's action, thus preventing it from throwing or forcing as much blood to the part, and in this way causing the congestion to subside, and the pressure to be removed from the bleeding surface. For the suppression of this and also other hemorrhages, the ergot is highly recommended by Professor Wood.

In the active hemorrhages, and particularly when the patient is plethoric, it may be necessary to deplete both generally and locally, first to reduce the general plethora; second, the local; also cathartics, revulsives, &c., may be employed as adjuncts. However, this course will not do in all cases of active hemorrhage; as, for instance, in uterine hemorrhage, or hemorrhage from a torn or severed vessel. In the first, we must endeavor to produce contraction of the uterine tissues, and thus close the patulous orifices of the uterine vessels or sinuses. For this purpose, we must resort to ergot alone, or combined with digitalis,

and probably also some remedy to act on the blood, as creosote, turpentine, &c.; frictions, pressure, cold applications to uterine regions, &c.; and it is even highly recommended to pour a stream of cold water from a height of several feet upon the hypogastrium; elevation of the lower part of the body, either by pillows, or by raising the foot of the bed upon which the patient is reclining will also be found useful; tampons, cold injections into rectum, are also employed. In the second, it would, if the vessel should be of any size, be necessary to resort to the ligature. In menorrhagia, active form, the treatment would be similar to that just mentioned in uterine hemorrhage; in the chronic variety, ergot, creosote, turpentine, counter-irritation, tampon, revulsives, cold and astringent injections per vaginam and rectum. Strychnia and electricity also might be employed, as the drain frequently depends upon a debilitated and relaxed condition of the tissues of the uterus; or it may be an accompaniment or dependent upon general debility, in which case the tone of the system must be improved by the use of the above tonics and corroborants, with other measures tending to assist and promote that end.

In bursting of the heart, or aneurism, death is generally instantaneous; but in the latter, by immediate pressure upon the vessel, if it is in a favorable position, and easily reached until assistance can be procured, and the vessel secured and ligated, the patient may be saved, with of course the appropriate supporting treatment subsequently.

Drains from excessive or perverted secretion and diseased action.—In diuresis, the treatment would be modified by the variety; thus, in diabetes, limiting this term to that form in which sugar is developed and evacuated, there is perverted secretion, whilst in diuresis insipidis there is an excessive secretion of the watery parts of the urine, being an increase in quantity; the first being an entirely new product thrown upon the kidneys to be eliminated: other varieties may have other abnormal products or an increase of the solid constituents of the urine, such as albumen, urates, phosphates, &c. In simple diuresis, it will be necessary to stimulate the other secretions, that of the skin and alimentary canal particularly, to improve and give tone to the system, to quiet any nervous disturbance, and endeavor to alter the action of the system generally, according to the indications, temperament, habits, condition of the system, &c. In cases of perverted secretion, we must first ascertain the condition or circumstances upon which the abnormal product or formation is dependent, and on account of the difficulty of so doing, is the unsuccessful treatment of such diseases in some measure attributable. Thus, in diabetes, the digestive organs seem to be in fault, although prominent symptoms

point to the kidneys; but if we look a little further, we might be induced to suspect that it arose from a deficiency of certain materials in the blood, which were necessary to the action of the stomach, and in consequence of the deprivation of which the stomach could not work up the materials of the food to that natural state or condition necessary for nutritive purposes. This view is supported in some measure by the formation of sugar when all materials generally supposed to be convertible into that substance are prohibited. And the writer would here direct attention to the salts of the blood, one of which, chloride of sodium, most probably by its containing chlorine, assists in the formation of the acids in the stomach. This, by being decomposed, forms chlorohydric acid, which assists materially in the digestive function; hence, by exhibiting this and other salts, as the phosphates, or their acids, chlorohydric particularly, in the form of dilute solution or water acidulated with it as a drink, with the other treatment generally adopted, it might probably prove beneficial. This view is still further supported to a certain extent by the constitution of a diabetic bread which has recently been used "with decided advantage" by Dr. James Johnstone and others. It is the receipt of Mr. Palmer, of Birmingham, viz—"Take the ligneous matter of sixteen pounds of potatoes, washed free from starch; three-quarters of a pound of mutton suet; half a pound of fresh butter; twelve eggs; half an ounce of *carbonate of soda;* and two fluid ounces of dilute *hydrochloric acid*. This quantity to be divided into eight cakes, and in a quick oven baken until nicely browned." (Braithwaite's *Retrospect of Practical Medicine and Surgery*, No. xix. p. 113.) The treatment recommended, however, is absolute abstinence from all amylaceous or farinaceous food, in fact, all vegetable substances convertible into those principles or forms, and from them into sugar, except those which are principally composed of ligneous matter, such as cabbages, spinach, &c.; and a strict adherence to animal food, warm clothing, tonics, alteratives, such as the preparations of iron, iodine, and the alkalies; and, indeed, everything to improve the general condition of the system, and stimulate the secretions of the body, those of the skin and liver particularly. Most writers also recommend occasional bleeding to equalize the components of the circulating fluid, but there seem to be objections to this, viz., it debilitates the powers of life, and, of course, the stomach, though appearing at the time to afford relief; and it also prostrates the nervous system by the removal of the mechanical support afforded to it by the fluid, which is necessary to a certain extent to the nervous system, instances of which have been given under the head of "Lesions of Function." I would also suggest the occasional use of injections of nutritive materials per rectum; they would

relieve the stomach in some measure, and at the same time afford an opportunity of ascertaining whether the whole alimentary canal was implicated in this abnormal elaboration of materies morbi.

In diaphoresis, such as colliquative sweats, &c., it is frequently dependent on a depressed state of the system, but of itself assists in debilitating still further; therefore, it will be necessary to improve the general strength, and at the same time prevent the undue action of the skin, for which purpose sulphuric acid in the form of dilute or aromatic sulphuric acid, tannic acid, and compound infusion of roses, are employed with advantage; and, for the general condition, sulphate of quinia is one of the best tonics; also infusion of wild cherry bark, and appropriate hygienic and medical measures, according to the modifications and complications.

In seminal evacuations, if they are voluntary on the part of the patient, the cure will depend upon himself to a great extent. In the first place, the absolute refraining from anything which might excite the discharge, such as exciting thoughts, conversation or reading, coition, masturbation, &c., and the mind and body must be occupied with active employment. If the patient has not moral power to do this, marriage is advisable, thus giving the organs their natural stimulus, which is the only "permanent remedy," according to Professor Jackson. But if married, and the system suffers from the excessive drain or evacuation, he must refrain also, as in the other case, from those thoughts, &c., which excite the passions; and if the mind cannot be diverted otherwise, it would be proper to separate from the wife temporarily by going on a journey; at the same time, to assist in the suppression of those feelings and excitements of the genital organs, lupulin, as recommended so highly by Dr. B. Page, of this city (Philadelphia), might be given. According to his account, it is almost a specific in the suppression of the venereal excitement. The infusion of hops might be substituted in many cases, as it contains the same principle, and, in addition, tonic properties highly advantageous to the condition of the general system. Dulcamara, camphor, conium, and even sugar, are also recommended as antiaphrodisiacs. The treatment recommended by Professor Jackson is, however, very judicious and appropriate, viz., as it is a local affection to some extent, to treat it locally as well as generally, and for this purpose he uses aconite internally, and by injections of the concentrated tincture of aconite, suspended in the mucilage of the pith of sassafras, thrown into the urethra and then pressed down against the neck of the bladder, and held there a short time; but, previously, before resorting to this remedy, he introduces a bougie into the urethra to quiet the irritation, and allows it to remain there about a quarter of an hour; if these should fail to cure, he

cauterizes the urethra by means of nitrate of silver, at the same time, in conjunction with these, employing corroborants, and general improving and strengthening measures. And for this and all similar conditions of the nervous system, he employs and recommends the phosphate of iron; phosphorus being an essential ingredient of nervous matter or neurine.

Although the organic life may not appear in some rare cases to suffer much, yet there may be a state of general inanition of the cerebral and nervous system; a remarkable instance of which came under my notice about a year since, of a young man who was then under the care of one of our best physicians. He was remarkably muscular and well developed, and to look at him one would suppose he was in the enjoyment of most perfect health, and would be very apt to point him out as an instance of superior physical development; but I was informed, to my astonishment, that he was so weak as to be incapable of performing an ordinary amount of either physical or mental labor, and that he had unfortunately contracted the habit of masturbation, and had continued it for some time, though when I saw him he had refrained from the practice for about one year. This case I have seen recently, and from appearances it has greatly improved under the judicious treatment of his physician.

It may well be supposed that I was much interested in this case, particularly as it afforded a fine example of several somewhat analogous cases mentioned by Professor Jackson a short time before in his lectures, as illustrations of the adynamic condition of the nervous system, while at the same time the organic life and muscular system was in a good state of health and development. One case was that of a young lady in whom a little too much exertion or exercise would produce an extraordinary degree of inanition, requiring active stimulation to keep her alive; but by prolonged rest, &c., she would recover, and by limiting the expenditure within the production of vital force, would enjoy apparently good health, and even improve very much in flesh, although the tendency to a similar condition still existed, from a supposed disposition to softening of the nervous tissue, and only required the exciting cause to put it in force. The subject of another case was a young man in whom the organic life was not so perfect, the adynamia being produced by disease, and, therefore, not so strikingly illustrative of the condition referred to, yet interesting on account of its sudden and fatal termination; and although he was so weak as to be scarcely able to walk any distance, and notwithstanding the advice and expostulations of Dr. Jackson, yet the father of the young man could not be persuaded but that he was feigning, and, in consequence, forced him to get on a spirited horse and take a ride, which, of course, exhausted so much of the remainder of his vital forces that there were not sufficient left to continue and support

life action, and, in consequence, a short time after his return from the ride, on the same evening, he died, to the dismay and sorrow of his then repentant parent.

Also after a complete state of paralysis of a part and even almost all of the body, the organic life may be adequate to the support of life for a short time, and occasionally for many years, and the patient may even increase in flesh. An exceedingly interesting and remarkable case is mentioned by Dr. Watson (*Watson's Practice*, pages 350–1), which came under the notice of Dr. Abercrombie, viz., "A servant girl, about twenty years old, sprained her back in lifting some heavy article of furniture. She felt no great inconvenience at the time; but some little while after, weakness of the legs came on, and gradually increased to complete paraplegia. After an interval, the affection extended to her arms, and she then had not a vestige of motion of any of the parts below the head, except a very slight movement of one of the fingers; but the internal functions were all perfect, and her speech was distinct, except that in speaking she was sometimes seized with spasmodic twitches of the lips and lower jaw. She lived in that state, without any change of the symptoms, and her general health continuing good, for about twenty years. In the morning she was taken out of bed and placed in a chair, so contrived as to support her in a sitting posture. Her arms rested on a cross board which passed before her; and if by any accident one of them slipped from this support, she had no resource but to call for the assistance of another person to replace it. In the same manner, if her head fell forward upon the thorax, it remained in that position until raised by an attendant. Her mind was entire. She died after four days' illness with symptoms of low typhus fever." Dr. Abercrombie looked with the greatest interest for the cause of these most remarkable symptoms. "I examined the body with the utmost care," says he, "along with Dr. Pitcairn, who had been in the habit of seeing her for several years; and we could not discover any disease either in the brain or in the spinal cord."

Leucorrhœa and gonorrhœa must be treated by local and general treatment, and modified according to the active or passive form; the former, generally, antiphlogistically, alteratives, &c.; the latter by stimulants, tonics, alteratives, injections, and hygienic measures.

In the choleras, such as Asiatica or epidemic, morbus, infantum, dysentery, and diarrhœa, there are the same general indications for treatment, modified, however, in some measure, by the stage, type, &c. In all of them, the most prominent symptom is the constant discharges per rectum, and in some per oris also, and seeming in

most of them to be the debilitating cause, although it is but a mere effect of the cause, yet does undoubtedly assist that cause in prostrating the living powers and forces. In cholera Asiatica, however, there is a different condition of the circulation from that of the others. Thus, there seems to be a tendency to a modification of the blood, or, as believed by some, a mere separation of the serum or liquor sanguinis from the red globules or hematin, with an exosmotic action through the intestines, excited by a peculiar poisonous principle, or the privation of certain principles in the atmosphere, destroying, at the same time, the endosmotic action and tendency of fluids, &c., to the circulation or interior of the body. Now, it must be evident that, if this is the case, the throwing of remedies and fluids into the stomach must be useless, except those which act through nervous communication; and, therefore, we must resort to some other method of preventing this tendency of the components of the blood to separate, and also to keep up and revive the endosmotic action and power of absorption and imbibition; and as it is notorious that all the usual and tried remedies are incompetent to this, and to the prevention and cure of this disease, I have been forced to the conclusion that we ought to direct our treatment, and the introduction of our remedies into the circulation, through another channel, viz., *the lungs*—and also for this purpose I beg leave to introduce and suggest a therapeutical agent, which, it is believed, will be found to be a more effectual one than any now employed, viz., *nitrous oxide*. I am constrained to the belief, from an examination of the constitution and properties of this substance, that it will not only be found highly useful in this disease, but also in many other abnormal conditions of the system, which will, and the applications thereto, be pointed out hereafter.

This gas appears to have been strangely overlooked and neglected by the profession as a remedial agent. It is well known that it is a powerful, rapid, and permanent arterial and nervous stimulant, exciting an ecstatic feeling, as if we were elevated many degrees above this life to a higher and more refined degree of organization or existence, divested of all the gross accompaniments of this, and this feeling not being followed by that state of sedation or depression which results from oxygen and other stimulants, having properties much more analogous, and therefore more appropriate, to the atmospheric air than any other compound of nitrogen and oxygen, or even pure oxygen, or any other known substance. To prove the permanently stimulating effects of this agent, I have only to quote Sir Humphrey Davy's experiments with it. He, in his own vivid and graphic language, thus describes the effect of the inhalation of this gas upon himself:—"To ascertain with certainty whether the most extensive action of nitrous oxide compatible with life

was capable of producing debility, I resolved to breathe the gas in such quantities as to produce excitement equal in duration and superior in intensity to that occasioned by high intoxication from opium or alcohol.

"To habituate myself to the excitement, and to carry it on gradually, I was inclosed in an air-tight breathing-box; twenty quarts of nitrous oxide were then thrown into the box. For three minutes I experienced no alteration in my sensations; in four minutes I began to feel a slight glow in the cheeks, and a generally diffused warmth over the chest. At this period, twenty quarts more of nitrous oxide were thrown into the box, and well mingled with the mass of air by agitation. In twenty-five minutes the animal heat was 100°, pulse 124. In thirty minutes, twenty quarts more of gas were introduced. My sensations were now pleasant; I had a generally diffused warmth without the slightest moisture of the skin, a sense of exhilaration similar to that produced by a small dose of wine, and a disposition to muscular motion and merriment. In three quarters of an hour the pulse was 104, and animal heat not quite 99.5°, the temperature of the chamber was 64°. The pleasurable feelings continued to increase, the pulse became fuller and slower, till in about an hour it was 88, when the animal heat was 99°. Twenty quarts more were admitted. I had now a great disposition to laugh; luminous points seemed frequently to pass before my eyes; my hearing was certainly more acute, and I felt a pleasant lightness and power of exertion in my muscles. In a short time the symptoms became stationary; breathing was rather oppressed, and, on account of the great desire of action, rest was painful." In a note, it is stated that, in the commencement of the experiment, the gas was too much diluted ("being mingled with near twenty-two times its bulk of atmospheric air,") to have much effect. He remained in the box one hour and a quarter; and he continues—"The moment after I came out I began to respire twenty quarts of unmingled nitrous oxide. A thrilling, extending from the chest to the extremities, was almost immediately produced. I felt a sense of tangible extension, highly pleasurable, in every limb; my visible impressions were dazzling, and apparently magnified. I heard distinctly every sound in the room, and was perfectly aware of my situation. By degrees, as the pleasurable sensations increased, I lost all connection with external things; trains of vivid, visible images rapidly passed through my mind, and were connected with words in such a manner as to produce perceptions perfectly novel. I existed in a world of newly-connected and newly-modified ideas. I theorized—I imagined that I made discoveries. When I was awakened from this semi-delirious trance by Dr. Kinglake, who took the bag from my mouth, indignation

and pride were the first feelings produced by the sight of the persons about me. My emotions were enthusiastic and sublime; and for a minute I walked round the room, perfectly regardless of what was said to me. As I recovered my former state of mind, I felt an inclination to communicate the discoveries I had made during the experiment. I endeavored to recall the ideas: they were feeble and indistinct: one collection of terms, however, presented itself; and with the most intense belief and prophetic manner I exclaimed to Dr. Kinglake, *Nothing exists but thoughts!—the universe is composed of impressions, ideas, pleasures and pains!* Not more than half of the nitrous oxide was consumed. After a minute, before the thrilling of the extremities had disappeared, I breathed the remainder. Similar sensations were again produced; I was quickly thrown into the pleasurable trance, and continued in it longer than before. For many minutes after the experiment, I experienced the thrilling in the extremities; the exhilaration continued nearly two hours. For a much longer time I experienced the mild enjoyment, before described, connected with indolence. *No depression or feebleness followed.* I ate my dinner with great appetite, and found myself lively and disposed to action immediately after. I passed the evening in executing experiments. At night, I found myself unusually cheerful and active; and the hours between eleven and two were spent in copying the foregoing detail from the common-place book, and in arranging the experiments. In bed, I enjoyed profound repose. When I awoke in the morning, it was with consciousness of pleasurable existence, and this consciousness more or less continued through the day." Afterwards, he says: "My susceptibility to its power is rather increased than diminished. I find six quarts a full dose, and I am rarely able to respire it in any quantity for more than two minutes and a half."

And also, "whenever I have breathed the gas after *excitement from moral or physical causes, the delight has often been intense and sublime.*" And again, "the pleasurable sensation" in the middle of another experiment "*was for a moment so intense and pure as to absorb existence.*" Davy also speaks of the thrilling sensation being felt in his teeth, as also do others who inhaled it during his experiments.

Dr. Kinglake says, "Among the circumstances most worthy of regard in considering the properties and administration of this powerful aërial agent, may be ranked the fact of its being both highly respirable and salutary; that it impresses the brain and system at large with a more or less strong and durable degree of pleasurable sensation; *that, unlike the effect of other violently exciting agents, no sensible exhaustion nor diminution of vital power accrues from the exertions of its stimulant properties; that its most excessive operation even is neither permanently*

nor transiently debilitating; and, finally, that it fairly promises, under judicious application, to prove an extremely efficient remedy as well in the vast tribe of diseases originating from deficient irritability and sensibility, as in those proceeding from morbid associations and modifications of those vital principles."

Mr. Wedgewood states that before he breathed the air he felt a good deal fatigued from a long ride he had had the day before, but *after breathing lost all sense of the fatigue.*

Mr. M. M. Coates says: "During the rest of the day," of that in which he had respired the gas, "I experienced a degree of hilarity altogether new to me. For *six or seven days afterwards I seemed to feel most exquisitely at every nerve, and was much indisposed to my sedentary pursuits.*"—(*Researches on Nitrous Oxide*, by Sir Humphrey Davy.)

Further proofs might be adduced of the permanently exciting character of this gas, if space would permit; but for further details I would refer to his work on that subject.

In cholera Asiatica, the poison seems to act primarily on the nervous system to depress it, and at the same time to arrest the chemical action going on in the blood, both by its impression upon the nervous system, and also most probably by its destroying the affinities between the components of the blood by catalysis, or by its greater affinity for different parts of the blood; thus probably forming a compound of the poison and serum, or liquor sanguinis, which would be the whitish or rice-water discharges from the stomach and bowels: this fluid or compound having a great affinity for the membranes of the alimentary canal, or from the great vascularity of the abdominal viscera, the fluids having a greater tendency to that part, and when exosmosed through into the canal, by its presence causing an action of emesis or purgation to get rid of it.

In our treatment of this disease, however, without regard to our views of its pathology, we must endeavor to prevent or correct this nervous depression, and also the separation of the blood; and as in other diseases we resort to the setting up of another action to get rid of the diseased, we on the same grounds would resort to an analogous mode in this disease; therefore, the nitrous oxide, by its nervous and arterial stimulation through its chemical and vital action, would, I believe, subvert both the tendency to a separation of the components of the blood and the nervous depression, and consequently prevent or arrest the progress of the disease after it has commenced; that is, before the stage of collapse, provided that collapse is dependent in some measure on the excessive drain; if there has not been any drain, or to a small extent, from the circulation, and the temperature has not been reduced too low for chemical action, even in collapse the nitrous oxide would probably

assist materially in reviving the depressed vital forces, and thus preserve the life of the patient.

In the exhibition of nitrous oxide, however, it should not be used ad libitum or indiscriminately, as it is capable of doing much harm, and particularly where the movements of the living machinery are impeded, or become sluggish, upon mere mechanical principles. Thus, if a body is at rest, or in slow motion, and it is attempted to set that body in rapid motion suddenly, there will in all probability be a rupture or separation of the particles; hence, in the prostrated condition of the system in cholera or any other similar condition, by exciting a sudden or rapid action of the heart and nervous system, death may ensue from actual rupture or solution of continuity of the heart itself, the vessels leading from it, or the parts to which the blood is sent, as the brain, &c.; and also, by exciting vital action too rapidly, all of the free force of vital power may be exhausted before the latent (if the expression may be allowed) is developed and eliminated, and thus again produce a fatal result. Therefore, in using this agent—and the principle is applicable to other stimulants in a similar condition of system, from whatever cause produced—it should be given in small quantities, and at first very gradually; on the principle of slowly introducing steam into the cylinder and gradually increasing it to move the piston and drive the machine.

In this way, in a short time, a considerable quantity of nitrous oxide might be introduced into the system, exciting active and permanent chemical and vital action, and thus overpowering or subverting the action of the poison, and supporting arterial and nervous power until the poison has been eliminated or its influence exhausted, analogous to the treatment in other poisons, such as typhus, opium, &c. During our attempts, or after we have thus arrested the abnormal action, small doses of calomel and opium, with acetate of lead as recommended by Professor Wood, with the addition of strychnia, being one of the most powerful nervous stimulants known, by acting on, exciting and increasing the secretion from the liver and other organs, contracting intestinal and other tissues, and stimulating and supporting still further the nervous system, would no doubt prove highly useful. Also, at the same time, in addition, "electrical insulation," as recommended by Mr. Pallas, might be employed with probably great advantage.

In dysentery, diarrhœa, cholera infantum, &c., if dependent upon irritable ingesta, they should first be removed by castor oil, conjoined or not with opium or its preparations, or, as many prefer, blue mass or calomel, followed by castor oil, opium, &c. If there should be any inflammatory excitement, as is sometimes the case in dysentery, it may be

necessary to bleed from the arm, or by cups or leeches from the abdomen, anus, &c. Also counter-irritation, vesicants, the astringents, opiates, turpentine, Hope's mixture, sulphate of copper, nitrate of silver, &c. In cholera infantum, Dr. Hodge prefers small and continued doses of mercurials. The chalk mixture, or mercury with chalk, particularly if complicated with acidity, and sometimes Dover's powder, are also used. Fresh and pure air, with a removal or occasional journey in the country, is one of the most useful adjuncts.

In all of these diseases, however, the most prominent and primary indication is to arrest the drain as soon as possible, and thus reserve the vital forces; and for this purpose, in diarrhœa, I have found no formula more effectual than a combination of plumbi acetas, pulv. camphoræ, āā gr. j vel. ij; pulv. opii ½ gr.; made into one pill, and given every two hours, with, of course, in all of these diseases, rest in a recumbent position, and other corresponding measures, one of which is of particular importance, viz., proper clothing, to keep the surface of the body at a proper and more uniform temperature, and to excite and continue the cutaneous secretion.

Another great point in getting and preserving the cutaneous surface in a normal condition is, not only to assist in the cure of the above-mentioned diseases, but to prevent them; and I am convinced that if persons would wear suitable underclothing so as to prevent the temperature, and probably electrical condition of the surface of the body from being readily changed, there would be much less of these diseases. This view was more and more confirmed during the prevalence of the epidemic cholera last summer. I found that those persons who were properly protected by suitable clothing were less liable to any intestinal irritation or evacuations than those who were not; and almost if not quite all persons with whom I came in contact, and who were suffering, were regardless of this point. I recollect hearing a gentleman, towards the decline of the epidemic, remark, "It is strange everybody during the summer has been complaining of diarrhœa and looseness of the bowels. Now it has been the reverse with me, for I have been suffering from constipation the whole summer." He was immediately asked if he did not wear underclothes, and his answer was in the affirmative. This has also been my own personal experience, except for a few days when there was a disposition to increased intestinal action. I have been thus led to consider that by an excess of clothing we may, by increasing the action of the surface of the body, produce to a certain extent constipation, and therefore it should be modified to suit different persons. Dr. H. Hartshorne, in his essay, entitled "*Water* versus *Hydropathy*," suggests also the use of silk, or a combination of wool and silk, as a protection and a non-conductor, to preserve the electrical equilibrium between the earth and objects around us and our bodies, he considering that many diseases and debilitated conditions are caused by, or are connected with, in some measure, the excessive loss of electricity from the body. This idea or opinion is also entertained by many authors of celebrity.

Mr. Pallas, physician in chief to the Military Hospital at Oran, entertains an exalted opinion of "electrical insulation as a curative and preservative means in many diseases," and particularly those of warm climates. He says, "I feel convinced that the great electric currents which exist, either in the atmosphere or on the earth, take an active part in the production of diseases in general, and especially in those of hot climates; and that by modifying the activity of these currents, which are always penetrating the human body, we shall be enabled to diminish, if not actually to destroy, the causes of the endemo-epidemic diseases of Algeria." And if of Algeria, certainly of all other parts of the world. In accordance with his views, "he had two beds constructed whose legs rested on glass nearly a foot in thickness, so that they were completely insulated. To one of the bedposts there was attached a chain, with a glass handle at the free extremity, so that the bed might be completely insulated, or placed in communication with the earth at will." In these, on his first trial, he placed two patients "suffering very severely from an aggravated form of dysentery, and in three hours time there was a marked amelioration of their symptoms." "One of them, who had had four bloody evacuations, with violent colic, shortly before noon," the time of insulation, "did not pass another stool till 8 P. M.; and he improved so rapidly from the time of insulation that in five days he was convalescent." The other recovered, though not so rapidly. "The pulse became considerably slower from the moment of insulation." Two other cases of a severe attack of sporadic cholera and quotidian intermittent fever were cured in three days, by the same means. These were followed by 23 others, with a very happy result, embracing "cases of acute and chronic dysentery, choleriform diarrhœa, intermittent fever, periodic epilepsy, acute articular rheumatism, visceral neuralgia, bronchitis, and meningitis." "It always exerted a favorable influence on the number of the evacuations, and on the heart's action." In consideration of these facts, it would be proper to consider in what other conditions of the system or forms of disease this would be applicable; and as electricity seems to have a close connection with the nervous system and other parts of the body—though having no confidence in the assertion that electricity and nervous force are identical—yet there seem to be good reasons for considering that there are great effects produced upon our bodies by the electrical changes and phenomena in the

air and earth, as shown probably to a certain extent in damp weather—it would appear, by the insulation and consequent retention of that which is constantly passing off, analogous to the passing off in excess of the other materials of the body, that it would be found useful in all adynamic conditions, and particularly in those persons of low vital powers; and in consequence I would recommend the *experiment of such persons sleeping* altogether *on insulated beds;* and in *all forms* of *prostrating diseases* it would also be *advisable* to *try* the *same*, to ascertain whether there is any influence exercised, and if so, to what extent, and in what diseases and conditions most applicable, not neglecting at the same time the long-tried and useful remedies.

In discharges of pus from suppurating surfaces and abscesses, the treatment must be stimulating and supporting, with nutritious and full diet.

In serous evacuations, as in the dropsies, the treatment depends in some measure upon the active or passive form. In the active, it may be the consequence of inflammation and the cure of that condition, and if the system is good and healthy, depletion by bleeding, general or local, carthartics, diuretics, and blisters. But when effused on or in the brain, as a result of inflammation, as is seen in hydrocephalus, it is, generally, immediately fatal, and affords no time for treatment. In the passive form, however, it is generally dependent on mechanical obstruction of the veins, or mere passive effusion from the vessels, the tissues not possessing sufficient vital contractility to retain the fluids. The most obvious course then is, if the effused fluid does not interfere with the continuation of life, to strengthen the vital powers, and through them the tissues, and at the same time to withdraw gradually the excess of fluids from the system by inducing gentle catharsis, diuresis, &c. Where the fluid presses upon some vital organ, as the lungs, heart, or brain, paracentesis is sometimes resorted to, as in hydrothorax, chronic hydrocephalus, abdominal dropsy, &c.; as it is also in an analogous condition, viz., empyema, in which the mechanical effects are the same.

In many cases, however, we find an excess of the serous part of the blood, with deficient plasma and red globules; and although the patients may look round and plump, they are very weak, and incapable of active or prolonged exertion, having occasional œdema of the face and extremities, and incipient indications of serous apoplexy of the brain or other parts of the system. In such a condition, the patient must diminish the usual amount of fluid taken into the system, and modify, strengthen, and consolidate their tissues by using the more solid and nutritious parts of food, assisted by occasional and gentle hydragogue catharsis, if necessary, by tonics, the chalybeates particularly, and other corroborant and hygienic measures.

In bad organization, medicine is not of much service, the improvement of the organism being entirely dependent on the patient, or if a child on its protectors; and in proportion as their knowledge of, and attention to, the hygienic laws, so will be their success in modifying, building up, and strengthening the system; this course, it is obvious, must be continued during life. In malformation, medicine is still less able to perfect the machine, although in some cases, such as spina bifida, &c., surgery, by relieving the condition and placing the parts in apposition, may cause nature to finish the operation.

In cases of disease from insufficient nourishment, as in the various forms of dyspepsia, they must be treated according to their character; thus, if of an inflammatory type, antiphlogistic; if from loss of tone of the stomach, local stimulation or rest, &c.; if of a nervous character, nervous stimulation of the whole system, tonics, &c. When the system has been reduced very much by an insufficiency or entire privation of food, as is frequently the case at sea from prolonged voyages, disasters, &c., it must be given in small quantities at first, and very gradually increased, and in many cases stimulants and tonics will also be indicated as auxiliaries. In purpura, the blood must be enriched by nutritious diet, stimulants, tonics, and attention generally to those hygienic measures which improve the blood and living forces. In scurvy, very frequently, the lemon juice, or juices of other succulent fruits, and potatoes, are adequate to the cure; but it may be also necessary sometimes to assist by improving and strengthening remedies.

In inflammatory affections during the inanition of convalescence, the remedies indicated are of general tonic and mildly stimulating character; during the synochus, it may be necessary to resort to active stimulating and supporting treatment, such as brandy, wine, or wine whey, quinia, nutritious diet, &c. In convalescence, the more permanent tonics, vegetable and mineral. However, in all cases of debility or inanition, from whatever cause produced, in which tonics, and iron particularly, are indicated, the best chalybeate, according to Prof. Jackson, is the following, viz:—

R.—Horseradish, grated, ℥j;
Mustard seed, ℥ij;
Cider, Oj;
Rusty nails, j or ij;
Oxide of manganese, ℥j;
M. Signa.—Teaspoonful twice or thrice a-day.

Also, in connection, the body must be well clothed and protected

from the changes of temperature, with prolonged rest, so as to allow of the re-collection, re-acquisition, or accumulation of life force, &c.

Prof. Jackson illustrates this condition of system in his own ingenious and beautiful manner, by an analogy, which can be understood by the most unlearned. He compares the organism to so much capital in money; this capital is producing a certain amount of interest, and as long as the owner is satisfied to live on or within that interest, his principal is not affected, or accumulates and increases in quantity; but if he expends all of his interest and commences on his capital, according to the expenditure so will be the rapidity of the decrease, until he becomes bankrupt. Thus Dr. Jackson thinks it is with our bodies:—each person has a certain amount of vital capital or material; this produces or develops a certain amount of interest or force; if we live upon or within that power or force developed, we will continue in good condition or even improve that condition, but when, by injudicious exhausting efforts, by sickness, or by the privation of food, this capital is diminished, it becomes necessary to relax those efforts, to correct morbid derangements, and to strengthen and support the system, and build it up by nutrient and stimulating materials, and if this cannot be effected, vital bankruptcy and of course death will ensue. When a person is reduced to this nearly vital bankrupt condition, and even long before, instead of continuing the expenditure, as is unwisely recommended by many persons, and even many in the profession, he (Dr. Jackson) advises that if the person take exercise, the expenditure caused by that exercise should be limited within the generation of force; thus permitting of the re-accumulation of material and power, and ultimately of that quantity necessary to the existence of life and good health. In connection with rest, he also recommends the use of tonics, particularly the formula above mentioned, nervous stimulants, warm clothing, nutritious diet, and attention generally to the organic laws.

The treatment of poisons, producing primarily an inflammatory action, must be first to apply the antidote; second, to produce emesis and prevent inflammation; and third, to correct the resulting inflammation. Thus from corrosive sublimate, preparations of copper, sulphate particularly, give albumen or white of eggs, with the occasional production of emesis, as a large quantity of albumen is said to redissolve the compounds between the antidote and poison; sulphate of copper is also incompatible with a great number of substances, as the alkaline salts, astringent vegetable infusions, &c.; from arsenic, hydrated peroxide of iron, if this cannot be obtained, sesquioxide or protocarbonate of iron. Also magnesia has been recommended; from antimony, or tartar emetic, tannic acid;

from nitrate of silver, chloride of sodium; from preparations of lead, sulphate of magnesia, tannic acid, &c.

One of the best emetics in such cases is the sulphate of zinc, in doses of from gr. x to gr. xxx, although it may of itself prove poisonous in large doses, therefore it should not be given in more than one or two doses; and if it does not operate, substitute some of the vegetable emetics, as ipecacuanha, &c.; the antidotes are, however, alkalies, such as magnesia, &c. Diluents and demulcent drinks must also, at the same time, in all cases, be used to dilute the poisons and protect the mucous surfaces, and also to assist in emesis; this latter process is all important, and it should not be delayed for the antidote. The subsequent inflammation must be treated by the appropriate remedies.

Treatment of Lesions of Function.—In the treatment of lesions of function from mental emotions, anxiety, excitement, great mental and physical labor, &c., the exciting cause or causes must first be removed; and the difficulty of doing this in cases of mental disturbances, arising from the circumstances, position, and trials of life, render it in many cases almost impossible. In such cases, the treatment must be moral to a great extent, assisted by those remedies which soothe and quiet the brain and nervous system, and improve the vital energies. The further exhaustion must be arrested; by a relaxation or complete cessation from labor, to give an opportunity for the system to recover from its debilitated state, assisted in many cases by proper remedial and hygienic measures.

The predisposition to or an attack of apoplexy would evidently be more of the passive kind, there being a predisposition to it from a too great excess of serum, or from a relaxed condition of the tissues, connected with the general state of system. The treatment would be to correct this tendency; and in the event of an attack, active purgation or local depletion by cups, or leeches, blisters, diuretics, &c., exciting the absorbent action in this more mild manner, mercury and general depletion being more or less incompatible with this condition of system, yet might be employed as the lesser evils. Epilepsy from mental emotions, intense study, &c., is of the centric, and, as Professor Jackson believes, of the incurable variety, probably from some "modification of structure," and if so, would be more appropriate under that head. Treatment can, therefore, be only palliative, although efforts ought to be made to cure it; and if all other means should fail, the most favorable course, according to Dr. Jackson, would be for the patient to withdraw completely from the world, and to live a mere vegetative existence, living on the plainest diet, and that

of vegetable matter, refraining from mental or physical labor or excitement of any kind or degree; taking at the same time, to reduce nervous and arterial excitement, aconite and digitalis, with the hope that in the course of time the brain and nervous system might be so modified as to get rid of the tendency to spasmodic action.

Professor Jackson, in his lectures, mentioned an interesting case of a young and highly talented lawyer, who, by intense study, brought on epilepsy. After consulting many eminent medical men both in this country and in Europe, going there expressly for this object, without having derived any benefit from their advice and treatment, he finally called upon Dr. Jackson, who informed him there was but one plan that afforded any hope of success, which was the novel one just mentioned. The young man adopted it, and secluded himself for about two years, by which time he had improved so much, that, contrary to the advice of Dr. J., he violated the prescribed course by an attention to some legal matters, and was in consequence again seized with a paroxysm at night, in which he threw his neck over the side of the bed, and was suffocated. Diet, in epilepsy, must be nutritious and exclusively vegetable. Dr. Jackson says that he has never known a case to get well during the use of animal food. Also the head must be kept cool, and the feet warm.

All of the nervous derangements, such as epilepsy, catalepsy, chorea, convulsions, syncope, stupor, coma, &c., dependent on, or predisposed to, inanition, should be treated with tonics, the mineral particularly, such as the preparations of iron, zinc, copper, bismuth, silver, &c., not forgetting or neglecting the vegetable, as strychnia, quinia, cimicifuga, &c.; also stimulants, antispasmodics, alteratives, corroborants, good and nutritious diet, warm clothing, and all other appropriate remedial and hygienic means for improving the tone of the system, and correcting and removing abnormal tendencies and action.

Those cases of adynamia resulting from non-arterialization of blood, by inhalation of impure or vitiated air, or complete, or partial privation of air, may most generally be relieved by the mere exposure of the patient or patients to fresh and pure air, by ventilation, &c.

In many cases, however, it becomes necessary to resort to stimulants, artificial respiration, electricity, &c.; but in numerous instances, all of these remedies fail on account of the impossibility, by them, of introducing sufficient oxygen in the limited quantity of air taken into the lungs, to excite or continue chemical action or arterialization sufficiently rapid to sustain life; consequently, under such circumstances, it becomes absolutely necessary to have some means by which the vital actions may be sustained until the carbonic acid can be eliminated from the system; and, as this can only be effected by respiration, with the introduction of

oxygen, which by uniting with carbon forms carbonic acid, and in this form is evolved, we should resort to the use of some agent with properties analogous to the atmospheric air, containing more oxygen, and hence more immediately stimulating and more peculiarly appropriate to this condition. Fortunately, such an agent is easily accessible in the form of nitrous oxide, sufficient evidence of the permanently arterial and nervous stimulant effects of which have been previously given. Sir Humphrey Davy supposed, from the quickness and rapidity of its operation, that it would probably become "useful in cases of extreme debility produced by deficiency of common exciting powers." He also remarks, "Perhaps it may be advantageously applied, mingled with oxygen or common air, to the recovery of persons apparently dead from suffocation by drowning or hanging." (*Researches on Nitrous Oxide*, p. 328.)

From the analogy of nitrous oxide, in constitution, to atmospheric air, and its superior stimulating power, I was first led to think of its exhibition in the asphyxia produced by carbonic acid, and in fact contemplated instituting some experiments before I became acquainted with Sir Humphrey Davy's researches; and thus being induced to examine the subject more particularly, my attention was directed to his experiments, which are very extensive and very satisfactory, proving by them that nitrous oxide differs from all other stimulants, to a certain extent, being permanent, and not followed, except in certain tendencies mentioned, by that state of depression which is always the result of stimulation; hence there are many conditions in which it might be usefully employed.

In drowning, particularly, as recommended by Davy, and also in the apnœa or asphyxia induced by inhaling gases from burning coal and other substances, and in suffocation from other causes, as in mines, sinks, wells, &c., where the vital machinery has been impeded or apparently arrested, the ready introduction of this gas into the lungs would most probably excite the respiratory, circulatory, and ultimately nervous functions, and thus rescue the patient from an otherwise inevitable death. In consequence of its valuable properties, and its easy elimination, it would be advisable for the "Humane Societies for the Rescue of Persons from Drowning" to keep an apparatus, with nitrate of ammonia, for its generation and exhibition; and also in the working of mines, in the cleaning of sinks, wells, and other places where accidents calling for its use are likely to happen, the workmen should always have with them an apparatus and material for its generation, having at the same time a small quantity of it ready for use, which could be obtained easily at a trifling expense, so that it could be at hand in cases of necessity, and thus probably save many valuable lives which would otherwise be sacrificed.

There are other cases to which it might be applicable. Thus, in the

asphyxia of newly born infants, there is not sufficient oxygen in the air which enters the lungs to put or keep the machinery of life in motion—like a deficiency of steam for an engine, yet by connecting the cylinder with another boiler containing sufficient, the machinery may be started without loss of time; so in the case of the child, by resorting to an analogous compound to the air, containing more oxygen, and being also a permanent and more active stimulant, hence may in such cases require dilution with common air, life action might be readily excited to that degree to which atmospheric air would be sufficient to continue it.

In cyanosis, also, it might be used with great advantage; but, as in the above case, may require dilution. Adopting Dr. Hodge's view of this disease, viz., that there is deficient respiratory action, and hence not sufficient oxygen introduced into the lungs to perfectly arterialize the blood, by the use of nitrous oxide this deficiency would be supplied, and proper respiratory efforts probably excited. In all cases of children, however, it would require dilution with atmospheric air, and also in many cases of adults; and the rules already mentioned respecting the mode of its exhibition would require more particular observance to prevent injurious effects from rapid stimulation. Its use would also be indicated in all cases of debility or adynamia dependent on deficiency of oxygen in the blood, and also in those cases in which there was a tendency to a degeneration or separation of the components of the blood, and in those diseases or conditions in which by exciting active chemical, arterial, or other action, would subvert abnormal tendencies or actions. To sum up, it may be used, first, to supply oxygen to the blood, where there is a deficiency or privation; second, as an arterial, cerebral, and nervous stimulant; and third, as an alterative, and would be applicable in all cases calling for these indications, there being no complications contraindicating its use.

In the adynamic fevers from poisonous exhalation, either of vegetable or animal origin, one of the most important indications is to support the strength until the poison has exhausted itself or been eliminated from the system, or the cessation of its influence upon the economy, except in cases where the attack is comparatively mild, as it is very frequently in intermittent, remittent, &c.; the supporting and curative means being the same, and acting at the same time, the paroxysms being prevented or arrested in a short time by nervous stimulants, tonics, antiperiodics, &c., as cinchona, quinia, strychnia, opium, &c. In the early stages of intermittent, before the system has become much reduced, nitrous oxide, by its action on the blood and nervous system, would most probably arrest or entirely prevent the paroxysm. In intermittent, Mr. Pallas speaks very highly of "electrical insulation." He says that "cases of intermittent fever—whether quotidian, tertian or irregular, if not

complicated with bronchial irritation—yielded to the sole influence of insulation, without the necessity of having recourse to quinine or any other medicine." In this disease, it is well known that powerful mental impressions, or a consciousness on the part of the patient that he will not have another paroxysm, will frequently prevent it; therefore, further experiments will be necessary before it is received as an established fact, although not wishing in the least to impugn the veracity of Mr. Pallas's experiments and statements, the attempt to, or the addition of any new facts to the mass of human knowledge being highly commendable; and if it is useful in this disease, it will, in all probability, be also in all adynamic conditions, particularly the adynamic fevers.

In typhus, yellow, and congestive fevers, and all similar conditions, the disease runs its course sometimes very rapidly, and, if not arrested, will speedily prove fatal. In all of these diseases there appears to be a tendency to a lesion of the blood, which would account, in some measure, for their intractability to treatment and fatality. Considering that in all such diseases, including cholera, hydrophobia, &c., death is not always the consequence directly of the nervous prostration, but from the permanent change in the blood, from the arrestation or perversion of chemical or other action in it necessary for its perfection, and the consequent prevention or suspension of the process for the formation of plasma, &c., and the production and conveyance of nutriment and stimuli to the nervous system.

The poison appearing to act in an analogous manner to that of the supposed choleraic, either by catalysis or by affinity, but having more tendency to the red and more solid parts of the blood; and the question might be propounded, whether by the action of the poison or disturbing agent there was not a reversion of the fibrin to albumen? as, according to Prof. Chapman, in continued fevers the blood "has *lost much* of its *fibrin, salts* and *solids*," this substance, albumen, not being spontaneously coagulable, and easily putrefied; or, as in sudden death from nervous prostration, as stroke of lightning, blow on epigastrium, &c., there being a similar fluid condition of the blood. Hence it might be, and I believe is, the prevalent opinion that the poison or disturbing cause acts in a similar manner upon the brain and nervous system, thus preventing nervous influence from being generated and transmitted to the circulation, and in this way arresting or interfering with arterialization, &c., of the blood, which in turn becomes incapable of stimulating the nervous system, and thus still more assists in destroying life action.

The treatment is, in the early stages, emetics, cathartics of calomel, &c., and subsequently to support strength by stimulants and tonics, such as brandy, wine, wine whey, carbonate of ammonia, turpentine, sulph. of

quinia, &c.; along with the mild diaphoretics, such as acetate of ammonia, and in the complications, opium, Dover's powder, ipecacuanha, &c. At the same time, endeavor to alter the action and stimulate secretions by mercury, ipecacuanha, and opium, or by combining the three, as calomel and Dover's powder, or calomel, opium, and ipecacuanha. The treatment most general is by emetics, cathartics; sometimes depletion, alteratives, stimulants, antispasmodics, diaphoretics, anodynes, &c., according to the stage, type, complications, and modifications.

In those stages or conditions calling for stimulation, uncomplicated with local lesions or inflammation, I would suggest the use of nitrous oxide; there being a failure of the nervous powers and a degeneration of the blood, it would stimulate the nervous system, and thus act analogous to quinia and the other stimulants, being superior to them in the permanency and character of the stimulation, and at the same time, by acting on the blood and producing the same chemical changes as the natural by atmospheric air, except in a superior degree, would, probably by its alterative effects, thus turn the barque of life from the mælstrom of death towards the haven of life and health—stimulating the secretions and supporting the system at the same time by means of mercury, quinia, strychnia, &c., and assisting with the other appropriate treatment.

Influenza is sometimes accompanied by a remarkable state of nervous prostration, a case of which was mentioned by Professor Jackson, of a lady who was unable, for one or two years, to use the slightest exertion without being so much exhausted as to require the use of active stimulants to keep her alive; but, by being at last obliged to follow the judicious advice of Dr. Jackson, with regard to rest, &c., in a recumbent position, she finally recovered her health and strength, after being confined about three years to her room.

For the cure of hydrophobia there is an almost infinite variety of remedies suggested, and many believe in the possibility of there being a specific for the poison; but as this antidote has not as yet been discovered, if it ever will be, we must fall back upon general principles, and first, the pathology necessary to elucidate the principles for treatment. In all writers that I have examined, there are several conditions pointed out as constantly existing, viz., the peculiarly spasmodic action of the throat; the condition of the blood, being analogous to that of the adynamic fevers; the congestion of the medulla oblongata and upper part of the spinal marrow, the poison seeming to have a peculiar or specific tendency to that part of the nervous system; and the tendency to death by asthenia.

The prominent indications would be, therefore, to prevent this change in the blood; to relieve the congestion of the medulla oblongata and spinalis, on which the spasmodic action of the pharynx, œsophagus, &c.,

appears to be dependent; to reserve and support the vital forces until the poisonous action should be exhausted or the poison eliminated from the system; and, to counteract the tendency to death by asthenia.

To do this it would be necessary to excite an action in the blood or system, counteracting that of the poison, as is sometimes done by stimulating the secretions and altering the action of the system by calomel, &c., in other abnormal conditions. To prevent or subvert the change in the blood, therefore, and to support the vital energies, nitrous oxide would be indicated; and if there were no contra-indicating circumstances, might be employed, at the same time exhibiting as an alterative and stimulant to the secretions, calomel, with or without ipecacuanha and opium, or Dover's powder, promoting diaphoresis by the hot-air bath; to relieve the local congestion of the medulla oblongata, &c., by cupping along the back of the neck, and if necessary along the spine, counter-irritation afterwards in the same place. Give aconite or opium internally to quiet restlessness. To keep the patient perfectly quiet, administer everything in solid form, and avoid by all means the excitement produced from offering fluids, passage of air, &c.; supporting at the same time the strength by means of quinia, strychnia, and other stimulants and tonics.

Blood-letting ad deliquium animi is by many highly recommended; but as bleeding is contra-indicated in all cases, except sometimes in the first stages, and then only to a very limited extent, where the tendency to death is by asthenia, it would appear to be also on that account contra-indicated in this disease.

Recently, chloroform has been highly spoken of to quiet the spasmodic action; but as this appears to depend on the changed or changing blood or local congestion, and this changed blood and congestion are assisting in destroying and exhausting nervous power, by exciting these spasms, it would seem to be objectionable, though there is no doubt of its capability in quieting spasmodic action. It would also do so in congestion and inflammation of the brain and spinal marrow, yet it would not cure or relieve that condition, but by its prostrating effects upon the nervous system would rather assist the disease, by taking away the power to resist the abnormal condition and to support life action under the additional pressure.

In a case, however, reported by Professor Jackson, simulating rabies canina, he used it with success; but notwithstanding there were almost all of the hydrophobic symptoms, still he is inclined to the opinion that the case was one of hysteria with the symptom hydrophobia (meaning of the term being *dread* or *abhorrence of water*), and that this symptom may be included in those of hysteria, or other nervous diseases; therefore, he is not prepared to say that this was a case of true rabies canina.

If chloroform should, however, prove beneficial in this opprobria medicorum, it will be a great blessing, and all hypothetical views must give way.

In poisoning from bites of venomous animals, from dissection, and in other poisonous wounds, there is also a great diversity of views with regard to the treatment; but the most general course is—and this will apply also to the prevention of hydrophobia—to apply, if on an extremity, a ligature round that extremity, and immediately a cupping-glass over the wound, or by suction with the mouth—except in hydrophobia, or in other cases if the mucous membrane of the mouth be abraded—to endeavor to draw out the poison, or by producing congestion of the part, to confine the blood and the poison in the blood to the wounded region; then to excise the part completely, re-application of the cup, followed by the application of caustics, such as lunar caustics, white arsenic and sulphur, mineral acids, actual and potential cautery, &c., to produce suppuration and sloughing; at the same time, for internal exhibition, salt and water, sweet oil, alcohol, ammonia, and other alkalies, and arsenic are highly recommended; also in some cases, as from stings of bees, &c., over a large part of the body, antiphlogistics, such as bleeding, calomel, opium, antimony, saline remedies, &c.

Others prefer the stimulant plan of plenty of wine, porter, &c.; for local applications, the caustics, opium, aconite, acetate of lead, emollient applications, &c. But as there appears to be an analogy between the effects of the various poisons, that is of certain kinds, both animal and vegetable, upon the human system, viz., to produce a change in, and a deterioration of the blood, with a tendency to death by asthenia, it would seem that the same general treatment would be indicated, with modifications according to the indications and modifications of the diseased action, viz., alteratives, stimulants, tonics, diaphoretics, anodynes, &c., with the antidote if there should be any known, and an agent to prevent this change or degeneration of the blood; and for this purpose, as well as for its stimulant properties, it would be advisable to use the nitrous oxide as an auxiliary to the other remedies mentioned.

For the stings of bees and wasps, and bites of musquitoes, spiders, flies, and other insects, local applications are generally sufficient, such as salt and water, aqua ammonia. Professor Gibson says that he has often known this latter, applied to a part stung by bees, "act like a charm." Also lead water, rose water, cold water, opium, aconite, &c., will often prove highly beneficial in allaying the pain and inflammation.

From the vegetable poisons, as the narcotics, &c., the first indication is to remove the poison from the stomach, which may often be effected by the stomach-pump or emetics, and large quantities of fluids, particularly if impregnated with the antidote, before the poison has been absorbed into the circulation, and the introduction of the antidote at the same time. A very good plan would be in all cases to use fluids, impregnated with the antidote, for washing out the stomach. For these vegetable poisons, which are principally, if not entirely, composed of vegetable alkalies, nature has, fortunately, in the same kingdom, kindly provided an antidote in the form of an acid, viz., tannic acid, which forms insoluble compounds with the most of them; hence, by using an infusion or decoction of tannin, or almost any of the vegetable astringents, such as oak bark, galls, &c., or even common tea or coffee, then emetics, diluents, and the stomach-pump, with stimulants, such as brandy, wine, ammonia, and sometimes opium, quinia, strychnia, &c., the patient may, if not left too long, be speedily relieved and placed out of danger. But when the poison has been absorbed into the circulation, in addition to these remedies, dashing cold water on the head and down the spine to remove congestion and thus relieve nerve centres, switching with cloths wet with a saturated solution of salt in water, with sticks, &c., around the buttocks and legs of the patient, keeping the patient in motion all the time to prevent sleep and to keep up nervous power, constant motion alone being sometimes sufficient. Bleeding is also sometimes employed, particularly in poisoning from opium, but great care is necessary not to deplete too much, as the patient may die in the subsequent prostration, which always follow the action of these poisons; and if the prostration from bleeding is superadded, there is still less chance for recovery. The subsequent prostration or inflammation should be treated on general principles according to the indications.

In some of the conditions from the effects of these poisons, opium particularly, there seems to be, from the impression upon the nervous system, an arrestation of the chemical action of the blood, viz., arterialization; hence it would appear to call for the use of nitrous oxide, notwithstanding the congestion of the brain, this appearing to depend upon the non-oxygenation of the blood, and if this should be induced, the congestion would probably subside. This idea is supported to a great extent by the beneficial effects of artificial respiration, and also of electricity, which are generally considered as the only remedies to be resorted to and depended upon after all others fail; and it would no doubt prove useful in all cases of poisoning from this class of substances, not only by its continuing or increasing chemical action in the blood, and thus revivifying it, but by its superadded stimulus to the nervous system, thus fulfilling the indications which are endeavored to be fulfilled by our other treatment.

A very striking case, showing the utility of electricity in poisoning

from opium, is reported by Dr. Martin Barry. A woman gave her child, aged nine months, twenty-five minims of laudanum, to procure sleep. The mother's attention was soon after attracted to it by "its loud breathing;" it was given the breast, and afterwards fell asleep, and for six hours remained in this state. After the expiration of another hour, "the breathing was more oppressed, and the child insensible to all impressions: at eight;" another hour, "its countenance was pale, with an expression of deep and placid repose; eyelids closed;" and the infant appeared to be in a slumbering state, with a tendency to relapse into sleep if roused, "breathing laborious, at times stertorous, or accompanied with a distinct stridor," also "bronchial irritation and expectoration. An emetic of gr. j. of tartar emetic, with gr. v. of ipecacuanha, in solution, was then given alternately until vomiting occurred; at half past eight, another emetic of gr. iij of antimony, and gr. v. of ipecacuanha was given; and flagellations and repeated agitations, together with cold affusions, were employed. The tartar emetic solution was repeated in half grain doses every ten or fifteen minutes, to relieve the chest," vomiting always proving beneficial. "This treatment was continued for two hours; but the restored consciousness was always imperfect, and succeeded by coma when the stimulating influence was withdrawn. At one o'clock P. M., congestion of the brain had increased; all means failed to rouse the patient; the coma was augmented, and the vital energy decreasing rapidly." The electro-magnetic machine "was now had recourse to," by Dr. Barry, "and it was not till the greatest degree of power was used that signs of perfect recovery ensued. The application was continued for five hours. We believe there is no other case on record in which a child under one year of age recovered from so large a dose." (*British and Foreign Medico-Chirurgical Review*, April, 1849, page 387.)

For hydrocyanic and oxalic acids there is not much time for treatment, the action of these poisons on the nervous system being rapid and fatal; yet, in poisoning from the former, it has been found that by the free use of water dashed upon the head and upper part of the spine the poisonous action is somewhat retarded, and is sometimes entirely prevented; and, in some cases, is adequate to the cure. But it is absolutely necessary to resort, also, immediately to the antidotes; for hydrocyanic acid, viz., chlorinated water, dilute solutions of "chlorinated lime, or soda, internally or externally," aqua ammonia very much diluted, and the cautious inhalation of its vapor; and a "mixture of the sulphates of the protoxide and sesquioxide of iron, with carbonate of potassa." When they come into contact with the poison, double elective

affinity takes place, resulting in the formation of sulphate of potassa and Prussian blue. If these antidotes fail or their action is too tardy, artificial respiration, nitrous oxide, electricity, &c. The treatment for poisoning from oxalic acid is similar to that formerly mentioned, consisting in promoting the removal of the poison by emetics, stomach-pump, &c., with the use of diluents and demulcent drinks, administering, at the same time, the antidote—magnesia, chalk, lime water; or, if neither is at hand, a common plan recommended is, to take the plastering from the wall, powder it and give it in water, if lime cannot be obtained in any other way. The resulting prostration and inflammation will require, of course, the appropriate treatment.

The palsy resulting from the mineral preparations of lead, arsenic, mercury, &c. From the first, the palsy should be treated with warm baths, sulphur baths, tonics, aperients, &c.; with mercury, sulphuric acid, strychnia, electricity, &c. That from arsenic with iron, warm baths, aperients, tonics, mercury, strychnia, electricity, &c. From mercury, a similar course, tending to improve, stimulate and strengthen the system, and eliminate the poison; also, lead might be used, these two substances seeming to be antagonistic to each other.

Recently, another remedy has been proposed by MM. Guillot and Melsens, in a memoir to the Académie des Sciences, "the object" of which "is to render soluble the metallic compounds which have entered the economy, by associating them with a body of very easy elimination." This body is iodine in the form of iodide of potassium. "All of the insoluble compounds formed by the salts of mercury with the matters met with in the economy are soluble in iodide of potassium, which substance is easily and rapidly got rid of by the economy. The compounds of lead are also very probably dissolved and eliminated in the same manner; and, in the memoir, cases of saturnine affections so cured are given." It requires to be given in "small and gradually increased doses, as a large dose of the iodide to a dog already suffering from disease from lead poisoning is speedily killed;" exhibited in the former mode, however, "the animal gets rapidly well."—(*Brit. and Foreign Medico-Chirurgical Review*, April, 1849, p. 543.) Probably the iodide will also be found to have a similar effect upon the compounds of arsenic in the system, and it would, therefore, be advisable to resort to it in cases of palsy from this metal, with the same or even greater precautions in regard to the dose.

The adynamia arising from the use of alcohol, tobacco, opium, &c., before there is any great debility or disturbance of system, may be relieved by refraining from the use of such articles; but when the habit

and consequences become too firmly fixed, they may be corrected by substituting other stimulants, given as medicine. These substitutes should be gradually abandoned as the system becomes quieted, and returns to its normal or healthy state. When delirium tremens is developed, it requires more active treatment; and for this purpose there are two plans proposed—one by stimulation with brandy, &c., the other with opiates. Either of these is very useful, but by the combination of both of them, it is most probable that the greater benefit would result.

The suppression of bile, urea, &c.; the first is not comparatively dangerous, and it may generally be eliminated by exciting the secretion of the liver with mercury, taraxacum, nitro-muriatic acid, &c. In the suppression of urea, however, unless the secretion and elaboration is speedily obtained, the patient sinks into a fatal coma from which he cannot be roused, and death shortly results. The treatment consists in endeavoring to produce the secretion of the urinary fluid by the action of remedies on the kidneys, &c., which may sometimes be effected by the different diuretics and corollary measures, such as spirits of nitre, Hoffmann's anodyne, turpentine, and infusion of diuretic herbs in combination, compound powders of calomel, digitalis and nitre, camphor and calomel, warm drinks of chamomile tea, cathartics, &c.; externally, fomentations about the pelvis, counter-irritation in the lumbar region, blisters, &c.; and at the same time it may be necessary to bleed, both generally and locally, if complicated with inflammation. Another instance in which a secretion, differing, however, in its being ordinarily highly nutritious, becomes poisonous, is that of the milk, it being converted, from mental impressions of the parent or nurse, into a virulent and active poison, producing in the child immediate and rapidly fatal effects, affording, generally, no time for treatment.

The prostration attending influenza, peritonitis, &c., is generally complicated with inflammatory action, indicating antiphlogistic treatment; yet it will not do always to resort to this too actively, on account of the tendency to asthenia, and it may even be necessary to support the system at the same time we are depleting; but, in many cases, direct stimulation is necessary to support the failing vital forces. Also, local depletion, blisters, sinapisms, cold or warm applications, iodine, nitrate of silver, lead water, opium, aconite, ice, &c., may be required.

In the shock from compression and concussion of the brain and spinal marrow, coup de soleil, injuries of various kinds, gunshot, operations, &c., the primary condition frequently calls for active stimulation; but this should not be attempted unless, as the least of evils, to support life, because the reactive tendency is to excitement and inflammation, particularly of the brain and spinal marrow, which requires, most generally,

antiphlogistic treatment, although, in some cases, such as from stroke of lightning, blow on epigastrium, &c., the system requires to be supported by immediate and active stimulation, &c.

The disposition to syncope or prostration, from the removal of fluids or solids from the body, may be prevented, to a certain extent, by the substitution of pressure from some other agent, external or internal. Thus the injection of water into the veins of persons who are almost moribund in cholera, seems to have an astonishing effect in reviving the powers of life, and, in some cases, actually to such an extent that the persons have subsequently recovered; and it is very probable that transfusion would prove still more effectual in reviving and preserving life. In view of this fact, it would be advisable by experiment to ascertain whether the nitrous oxide would not continue life action after this partial and generally temporary recovery from venous injection. The value of refilling the emptied vessels has also been successfully demonstrated in cases of hemorrhage; the lives of many persons having been preserved by tranfusion, cases of which are on record, although, in these cases, the transfused fluid being blood must have assisted materially in the recovery.

In paracentesis, parturition, &c., pressure is made by means of the bandage; in hydrocephalus and spina bifida, by the adhesive plaster in addition, although, in the latter, acupuncturation is preferably employed to the trocar, as sudden death will result from the evacuation of the whole of the fluid at one time, as it will often also in hydrocephalus, thus allowing only a small quantity of fluid to exude at one time. The wound is then closed by adhesive plaster, and additional pressure should be made on the tumor by covering it with collodion or with adhesive plaster, the latter preferable, as in hydrocele; taking care, however, that the pressure is not sufficiently great to cause inflammation; and in this way the combined advantages of the evacuating and compressing treatment may be obtained.

In bursting of an aneurism, or hemorrhage from any part which can be reached, pressure may arrest the hemorrhage until the vessels can be ligated or styptics applied; at the same time, the general system may require to be supported by stimulating drinks, &c.

The stupor from exposure to cold will generally pass off without recourse to medical treatment, by a return gradually to a more equable temperature, at the same time continuing the motions of the body; but sometimes it is necessary to resort to frictions, &c., to the surface of the body. Stimulants may also, in some cases, be required, as is well known from the history of the St. Bernard dogs on the Alp mountains.

4th. Sympathy.

For sympathetic action, it is obvious that there must be a predisposition, from excessive excitability and irritability; or a predisposition may be formed from the constant action of the ultimately exciting cause, as in tetanus, and, most probably, also in epilepsy and other convulsive diseases of the eccentric form.

The disturbing causes may be located in almost any part or organ of the body, the part most active, probably, being the alimentary canal, extending over such a vast distance, and affording a nidus for almost every sort of matter, among the most common of which are ingesta, indigestable and fæcal matters, worms, the secretions, &c. Another very prominent apparatus in the production of this state is the generative, particularly of the female, one organ of which, from its constant offending, has given rise to the name of a disease, viz., hysteria, from ὑστέρα, which springs, however, from a great variety of causes acting in any part of system—brain particularly.

The derangements are exhibited in the form of hysteria, epilepsy, chorea, catalepsy, tetanus, trismus nascentium, and the various forms of spasms and convulsions, as seen particularly in children; the frequency of their occurrence in them depending upon the predominance of the ganglionic or excito-motory over the cerebral centres, the controlling influence of the latter appearing to be inversely as the age, although there are periods of peculiar susceptibility, particularly in the female, as puberty, &c.

Treatment of Sympathy.—In all cases of nervous aberration and derangement, the attention of the physician should be particularly directed to the condition of the different surfaces, organs, and apparatuses of the body, to ascertain whether there is any irritation or disturbance in them, which by connection or reflection would excite a similar or exaggerated condition of other parts of the body, and from them implicating the whole system; and of no diseases is this more likely to take place than in those which have been just mentioned; hence the absolute necessity for a careful and accurate examination of the whole organism, before attempting to prescribe for any such derangement, except probably traumatic tetanus, where the cause and condition are evident.

The treatment of these diseases, and in fact all diseases, varies according to the stage of the disease, condition of the system, &c. Thus, for those under consideration, it may be divided into that applicable to, or during the paroxysm, and that during the interval.

In the first, there will generally be found considerable difficulty, the

system being in such a disturbed condition--shown particularly during the paroxysm of hysteria or epilepsy—as to prevent very active interference, in many cases it being better to wait until its cessation, on account of the difficulty of getting any information respecting the disease or patient from persons present, and the impossibility of getting it from the patient; and, in such cases, as much or more harm might be done by the officious interference of the physician as by the disease.

Where, however, the sympathetic condition is evident, and not connected with congestion, or inflammation of the brain and spinal cord, it would be advisable, first, to endeavor to allay spasmodic action and quiet the system by means of the inhalation of the anæsthetic agents, viz., chloroform, sulphuric ether, &c.; but their use must be conducted judiciously, as they are capable of doing much harm; the former might be preferably employed in all cases dependent upon an excess of action requiring direct sedation, it being a direct and powerful sedative; the latter, in those cases of derangement attendant upon, or occurring in, debility, it being a stimulant, would bring the system up to that point in which the nervous actions would be equalized and, consequently, morbid action allayed, analogous to the quieting of the nervous excitement of delirium tremens by opium, alcohol, &c. At the same time, if any disturbing causes are suspected in the alimentary canal, remove such by means of cathartic injections, following their operation by enemata of antispasmodics, anodynes, &c.

The treatment for tetanus is generally more active, and requires more perseverance to correct and hold in check the paroxysms than the other diseases of this class; and in many cases, to our mortification, they cannot be arrested even by the most powerful medicines of the materia medica, the system seeming to have lost its susceptibility to the action of those remedies to which it ordinarily succumbs.

The treatment most generally adopted is to give opiates profusely, even ad saturandum, as recommended by Professor Gibson. The stimulant plan as practiced by the late Dr. Hosack, consisting of the exhibition of brandy and wine in large quantities, has also been found successful in many cases. More recently, the inhalation of the anæsthetic agents has been highly recommended, and has undoubtedly proved very useful.

Other remedies which have been recommended are bleeding, purgatives, sudorifics, caustics, and the potential cautery along the spine, as practiced by Dr. Hartshorne; iron, prussic acid, tobacco, digitalis, strychnia, electricity, and many others of minor importance.

As, however, none of these individually have been very successful, would it not in all cases be better to use a combination of the most

powerful sedatives—and one of which particularly, viz., aconite, its action being somewhat analogous to that of opium, yet more directly sedative, and more specifically directed to the excito-motory system, without so much action on the brain, which is not required, as the cerebral functions are generally unaffected in this disease.

The internal exhibition of aconite, the occasional inhalation of chloroform, and the external application of either of them along the spine, and also to the wounded part, the latter particularly, immediately on the appearance of the incipient indications of the disease, conjoined with the internal exhibition of digitalis in case of much arterial action, would most probably afford more favorable hopes of success than any other plan. If these should however prove abortive, and the declining stage appeared, when the living powers were about failing from exhaustion, it would be proper to resort to stimulants, such as brandy, quinia, strychnia, or electricity, and thus by rousing and supporting the vital forces life might be prolonged and probably ultimately saved.

In this class of diseases, in all cases, during the interval, a very good plan is to correct and modify the actions of the alimentary canal, by removing all offending substances by the use of emetics, antacids, anthelmintics, cathartics, &c., assisted by anodynes, &c., at the same time removing or quieting the disturbances in other parts of the system. If the uterus or other parts of the generative apparatus are in fault, the appropriate remedies must be resorted to according to the indications; also the same with any other portion of the system; and, for this purpose and the general improvement and regulation of the system, it may be necessary to use a great variety of remedies, such as bleeding, general or local, counter-irritation, purgatives, emetics, diaphoretics, diuretics, anodynes, antispasmodics, tonics, and particularly the mineral tonics, such as the sulphates and oxides of iron, copper and zinc, nitrate of silver, subnitrate of bismuth, and the other preparations of mineral and vegetable tonics and alteratives, &c., or a combination of some of these. Great attention must also be paid to clothing, to preserve and regulate the proper temperature of the body, exercise, and diet; and in fact to all hygienic rules.

The diet is so important in epilepsy that Professor Jackson says he has cured twelve or fifteen patients by attention to this alone, confining them however strictly to a vegetable diet, he never having seen a case of this disease get well during the use of animal food—and in the above-mentioned cases the patients recovered without medication, under the use of nutritious vegetable food. The question arises whether these cases were not of the sympathetic variety, and whether, in most cases of sympathetic epilepsy proceeding from irritation or disturbance

in the alimentary canal, &c., the cure could not be perfected by a similar dietetic course exclusive of medicinal treatment?

In chorea, in addition to the above treatment, the vegetable tonics, as, for example, cimicifuga, &c., appear to be very applicable.

In each one of these diseases, in fact, there is some modification which renders necessary the exhibition of one remedy preferable to another, although the same general principles are required to be acted upon.

There is, however, another point to which I wish to draw attention particularly, viz., the local treatment, both as prophylactive and curative. In tetanus, and in many cases of epilepsy, it is well known that the impression or irritation is conveyed from the periphery, or some internal organ or part of the body, to the ganglionic centres, hence called eccentric; and most probably in all cases of epilepsy having the aura epileptica, the origin of the impression will be found to be from some disturbance of the sentient expansions of the nerves, which, from its sympathetic transmission to the nerve centres, breaks or disturbs the chain or connection for voluntary motion, and excites the peculiar involuntary spasmodic action, just as the breaking or detaching the mainspring of a watch, and thus removing the check, will excite or permit a rapid action of the machinery until it runs down—although of course there must have been a predisposition of the ganglionic centres produced either by the constant transmission of the morbid impression from the part or surface affected to the centres, till the impression became the exciting cause, or by some other cause.

In such cases, it would appear obvious that, by preventing the transmission of this morbid impression, the paroxysm would be prevented or entirely cured; and this has actually been done in epilepsy and tetanus by a ligature or tourniquet around the limb, thus compressing the nerve or nerves; by separation of the nerve or nerves proceeding from the part; and by amputation of the extremity from which the aura or impression proceeded.

An interesting case of "Epilepsy Periphera," recorded by Dr. Stumke (*British and Foreign Medico-Chirurgical Review*, January, 1849, p. 265), confirmatory of the same, was produced by a clavus and cured by its removal.

In this case, the "paroxysms came on every week or fortnight. An unpleasant feeling in the toes, proceeding upward along the leg, always preceded the attack. After awhile, the muscles of the entire limb became convulsively attacked; she then fell down, lost her consciousness, and the ordinary symptoms of an epileptic attack manifested themselves, a deep sleep terminating the fit. The *peculiar* kind of *aura* having directed the author's attention to the foot, he found there a *clavus* in an

inflamed and *painful condition*. The patient was confined to her bed, and by various applications the *corn* was in a few days softened and *removed;* and from that period to the present (more than three years) the *attacks* which had latterly occurred nearly daily, *have never returned*."

This case and the facts before mentioned would tend to prove that eccentric epilepsy, tetanus, and the other forms of sympathetic derangements arise from a primary disturbance or disease of the nervous expansions, or, probably, any part of the afferent nerves, and by the constant transmission of the morbid impression to the spinal marrow and brain, acting first as a predisposing cause, producing a predisposition, thus deranging their functions; and second, as an exciting cause, setting up spasmodic action.

And in this case, the steps or changes from the cause to the ultimate effects, and the cessation of morbid action on the removal of the cause, are very well exhibited; first, by the unpleasant feelings in the toes, which proceeded up the leg to the lower part of the spinal marrow, the functions of which after a time becoming deranged, resulted in spasmodic action in the limb; next it implicated the rest of the spinal marrow and the brain, and general convulsions took place, attended with loss of consciousness; but on the removal of the cause (the clavus), all of the effects ceased, on account most probably of there being only a functional disturbance, the disease not having existed long enough to produce modification of the nervous structure.

In consideration of these facts, therefore, the writer wishes to suggest a plan of treatment which he believes will be found more effectual than either pressure upon or severing the nerves, or excision or amputation of the part or extremity, and not so barbarous as the latter, although acting on the same principle, viz., to narcotize, by means of the local application of aconite, opium, chloroform, &c., the nerves of the part or extremity from which the impression is being transmitted, and keep them thus narcotized till the morbid tendency may be corrected. It is obvious, however, that it will require some short time to get them under the influence of narcotics, and the ligature or tourniquet might be resorted to in the event of an attack in the meantime. If, as in neuralgia, we can allay the sensibility, by local narcotism, we can also most certainly, in the same way, prevent the transmission of impressions as effectually, the action being similar in both cases, differing probably only in degree, and thus arrest or prevent the development or production of spasmodic action, and particularly those formidable forms of it designated by the names of "epilepsy" and "tetanus."

This treatment would also be applicable to all surfaces of the body, internal or external. Thus, if the irritation was in the alimentary canal or

uterus, &c., the internal exhibition, or local application of these same remedies, by injections, poultices, unguents, lotions, &c., would no doubt prove as effectual as the former; and, in fact, the narcotics have been for centuries, and are being constantly given to allay pain, irritation, and spasmodic action, they acting in the same way either by preventing the nervous centres from receiving or sending impressions, or the ultimate nervous expansions from transmitting or responding to those sent from the centres.

The foregoing treatment must, of course, except when used as prophylactive, and even in some cases as curative, be most generally associated with the other general treatment before mentioned, and in this way would most probably be applicable to the cure of all forms of sympathetic nervous aberration, where there had not been any modification of nervous tissue.

LOCAL NEURO-ADYNAMIA.

Local neuro-adynamia, generally, is not fatal, its utmost danger being mostly the loss of the use of the part affected, except it be some vital organ, or connected with the functions of such organ as the heart, lungs, and their immediate appendages, &c.; or it may be a prodroma of a general attack, which is very often the case, and in no disease, probably, more so than in apoplexy, preceding which there may be paralysis of almost any nerve of the body, but particularly the cerebral nerves, which may be so slight and transient as scarcely to attract any attention, or permanent and fixed, and continuing so until the general paroxysm supervenes; but it often exists independently, and separate from any general derangement, so much so that there may not appear the slightest disturbance of the functions of any of the other organs or parts of the body.

In local neuro-adynamia, the active or exciting cause may be at the ganglionic centres, or origin of the nerve or nerves, in some part or in the whole of their course, at their ultimate expansion or ramifications; or it may arise from disturbance in some other part of the system, or from debility or death of nervous mass and tubes.

1st. MODIFICATION OF STRUCTURE.

This may consist of the same as in general neuro-adynamia, and may be produced by and result from the same causes.

Induration and hypertrophy in the nervous tubes in addition may be excited by a ligature or contracting of the tissues, excited by irritation,

inflammation, &c., around them, thus causing a swelling of the nerve, as if it were an attempt at the formation of a new ganglion, producing, most generally, violent neuralgic pains, and also sometimes paralysis, as is occasionally seen in or on pericranium, and different parts of the body, extremities particularly. An interesting case of this form of neuroma is mentioned by Dr. Gibson (Gibson's *Surgery*, vol. ii. p. 432), which came under his own immediate care, viz., one of these tumors, "the size and shape of a goose egg, seated on the inner edge of the biceps muscle, near the middle of the right arm, firm and solid to the touch, movable, but not particularly painful." There may also be swelling or enlargement of the nerves by interstitial deposit of a sarcomatous, tuberculous, or gelatinous character.

From these various forms of "modification of structure," we may have any or every kind of local nervous disturbance, from an excessive neuralgia to a complete paralysis of the parts which the affected nerve or nerves supply, as exhibited in hemiplegia, amaurosis, palsy of one side of the face, or any part supplied by the cranial nerves, asthma, pains in the chest, cough and dyspnœa, difficulty or privation of deglutition, aphonia, paraplegia, sciatica, palsy of one extremity, of one muscle, or a finger, or toe, &c., or paralysis of the sensory (anæsthesia) and not motor functions, or vice versâ; the first, the most rare, as generally, where there is general sensory adynamia, motor power also fails, but the reverse not so much so. And what is also very singular, adynamia of the special sensory functions results on the loss of the general sensibility connected with them, by division or otherwise of the nerve of general sensation of the face and head, &c., viz., the fifth pair or trigeminus; and also if the branch of this nerve, accompanying the nerve of special sensation, have its function destroyed, the function of special sensation also fails, as in the case of the optic nerve, &c. And what is still more singular, if the supra-orbitary branch of the fifth pair, out of the orbit, at or about the supra-orbitary ridge, be severed, loss of vision will result. My preceptor, Dr. George W. Patterson, having seen one such case, suggests that probably "the same result might also follow a violent contusion or inflammation of that nerve." One curious case of the local paralysis (anæsthesia) of the sensory, and not of the motor nerves, is that of a woman who had lost all sensation in her hand and fingers; and as long as she directed her sight to them she could grasp or carry any object, but the moment her attention was directed to some other point, they would begin to relax, and the object would fall, on account of the sensory or afferent nerves not being able to transmit impressions of the condition of the part to the brain. Other cases are mentioned, in one of which this form of adynamia was limited to one arm and one side of the body (Watson's *Practice*, p. 350).

Adynamia of any of the organs may also take place, as of the heart, stomach, uterus, bladder, &c.

And it is probable that angina pectoris is sometimes dependent on neuroma in or about the cardiac and neighboring plexuses and nerves connected with them; Professor Jackson, however, believes that it is dependent on the derangement of the eighth pair, or par vagum, complicated with the brachial plexus; and, according to the degree of change, or the amount of deposition, so would, most probably, be the extent of diseased action, as from a slight difficulty or attack to violent pains, dyspnœa, palsy, and necessarily death, its progress being analogous to neurosis of other parts, viz., alternation of exacerbation, and of apparent health, or feelings of that kind, until the disease is so far advanced that there is no complete cessation of the effects, and death ensues. Dr. Chapman's opinion may thus, in some measure, be found to be true, as he thinks that it is connected with the gouty and rheumatic diathesis.

Treatment of Modification of Structure.—This would be similar in many cases to that of general neuro-adynamia of the same class, the condition of system being the same and the disease being internal.

In softening of the brain, spinal marrow, or nerves, it is obvious that nothing could be done, except, probably, if dependent on inflammation, to endeavor to prevent its extension and thus limit the destruction, although in these cases, unless it were in a single ganglion, nerve, or branch of a nerve, it generally extends, and thus runs into general neuro-adynamia, and consequently death, as is often seen in the extension of hemiplegia, paraplegia, &c.

In hardening or induration by interstitial deposit of foreign matter, by hypertrophy or otherwise, absorption may probably remove the superabundant deposit. This may be effected by depletion, internal exhibition or external application, or both, of mercury, iodine, &c.—the general treatment, however, depending, in a great measure, upon the diathesis, temperament, and condition of the patient. Thus, in debility, &c., the preparations of iodine, with tonics, corroborants, &c.; in plethoric and active constitutions, bleeding, mercury, and other evacuant and reducing treatment. But in those cases of neuroma, or enlargement of a nerve near the surface of the body, as mentioned by Professor Gibson, the treatment as indicated and practiced by him would be most appropriate, viz., the excision or extirpation of the tumor, by severing the nerve, on either side, connected with it, or the application of the actual cautery. In the case quoted, he (Dr. Gibson) removed the tumor; and although "numbness of the arm, forearm, and fingers, amounting almost to paralysis, followed, the wound healed kindly, and in a short time the general numb-

ness disappeared, though it remained in the fingers, which were cold and almost useless for nearly three years; but at the end of that time was entirely removed, and the use of the fingers restored." (Gibson's *Surgery*, vol. ii. p. 433.)

In cases arising from a ligature, it must be removed immediately, and if it has not been on too long, the narcotizing of the nerve, by the local application of aconite, chloroform, opium, &c., may be sufficient to cure it; but it may sometimes be necessary, as in a case mentioned by Dr. Gibson (Gibson's *Surgery*, vol. ii. p. 435), to apply the actual cautery to the wounded nerve.

In those cases in which the nerve is confined and tied down by the tissues, as is seen sometimes on the pericranium, the cutting down upon, severing the tissues, and thus liberating the nerve, will completely relieve the affected part and any abnormal action dependent upon it.

In atrophy, stimulation will give the most favorable indications for a cure, and if there are no contra-indications, strychnia or the occasional use of electricity would most probably excite an increased nutrition and development, improving if necessary the general system with tonics, as phosphate of iron, &c.

In solution of continuity, the only thing is to trust to nature for a cure, taking care to bring and keep the severed parts as near in contact as possible, correcting at the same time any tendency to inflammation.

2d. Interference of function without modification of structure.

This, as in general neuro-adynamia, is generally mechanical in its character, and may arise also from compression of the brain or spinal marrow, or limited parts of the same, and from the same causes, viz., congestion, effusion of blood, serum, coagulable lymph (active or passive), purulent matter, pressure of tumors, bone, &c., with the production of hemiplegia, chorea, derangement of the functions of the thoracic viscera, as heart, lungs, &c., resulting in palpitations or other irregular action of the heart, angina pectoris, syncope, asthma, dyspnœa, difficulty or privation of deglutition, aphonia, &c.; adynamia of the abdominal and pelvic viscera, as stomach, intestines, bladder, uterus, &c.; also, anæsthesia, neuralgia, sciatica, paraplegia, &c.

The "interference" may, however, be limited to a single ganglion or plexus, or even to a single nerve or branch of a nerve, producing either anæsthesia, neuralgic pains, spasms, or complete or partial palsy of the part to which the nerves are distributed, examples of which may be seen in almost any part of the body;—thus from the cranial nerves, may have spasmodic action, paralysis, anæsthesia, or neuralgia, from pressure

within or without the cranium upon the motor or sensory ganglia or nerves; for example, loss of smell, from "interference with function" of olfactory nerves; amaurosis, from the optic nerve; movements and expression of the eye, from abducentes, motor-oculi, and patheticus; loss of sensibility and disturbance, even to paralysis of the special sensory nerves from the fifth pair; paralysis of the masticatory muscles, independent of any disturbance of the facial muscles of expression, from the motor branch of the fifth pair; deafness, from portio-mollis; paralysis of the muscles of the face, from portio-dura; disturbance of the respiratory, circulatory, digestive, and other functions, from the eighth pair or par vagum; paralysis of the tongue, from the hypoglossal, &c.; and thus we might pass in review the whole body, and specify the adynamia of the sensory and motor functions of the different parts of the system.

Treatment of Interference with Function without Modification of Structure.—The treatment will be similar to that of the same class in general neuro-adynamia, and must be modified according to the diathesis, age, temperament, condition of system, locality, and cause of the disturbance, &c.; and hence, in many cases, although the disease may be limited and circumscribed, yet it may be necessary to treat the system generally, whilst local treatment is employed at the same time.

In other cases, local treatment may answer all the indications, and be adequate to the removal of the offending substance, and in this way cure the condition depending upon that interference. Thus, local congestions and effusions may frequently be removed by revulsion and absorption from the application of a sinapism, or more active and permanent irritants, as pitch and cantharides plaster, tartar emetic, croton oil, &c., blisters, cups, leeches, mercury, iodine, &c.

In many cases, however, such as the active effusion upon portio-dura, which most generally occurs from a draught of cold air upon aural region, thus exciting inflammation and effusion of coagulable lymph upon the nerve, it will be necessary sometimes to deplete actively, by the lancet, from the arm, by cups or leeches from the part, and by cathartics from the general system, also employing at the same time mercury to reduce the amount of fibrin in the blood, and to prevent organization in, and to promote absorption of the effused plasma. In numerous cases, although general depletion is indicated, yet it will not do to bleed or give mercury; as, for instance, where there is local adynamia depending on plethora, but that plethora is of a serous character, and may threaten an attack of general adynamia, which may be, and is, generally, first signified by some form of local adynamia. Thus, for example, I was requested by

a gentleman to look at the eye of his wife, who was occasionally attacked with partial and temporary blindness. On examination, I could find nothing the matter with the structure of the eye; but, on inquiry, was informed that she was troubled with headache, particularly about and at the period of the attack. On examining the condition of her system generally, I found her rather embonpoint, yet her skin was pale and waxy, her muscles, though apparently full, were neither hard nor firm. She was incapable of much exertion, being easily exhausted; her vessels were loaded with serum, it being, as might be supposed, in great excess, with a great tendency to passive effusions, from the debility of the tissues. I concluded that it was a case of threatened serous apoplexy, showing itself in the form of amaurosis, caused by the pressure upon the optic nerve; and, in consequence, I prescribed a hydragogue cathartic—not an active one, however, but sufficiently so to produce a moderate amount of drainage, to relieve present symptoms—and, as I found that she was in the habit of taking large quantities of fluids, in the forms of coffee, tea, soup, &c., I requested her to diminish the quantity to the smallest amount necessary for her wants, leaving the daily secretions and excretions to remove and equalize the remaining quantity; and at the same time to endeavor to strengthen and consolidate her tissues and enrich her blood by more solid and nutritious diet, which course, it is obvious, in such a system, must be continued during life, to correct and prevent the tendency to a return. Tonics were also indicated, and particularly the chalybeates; but as she was averse to taking medicine, I did not order any further, and she is now, after the lapse of several months, comparatively well, the amaurosis and oppression of the brain having entirely disappeared. She subsequently informed me that she had been bled at a former attack, which relieved the symptoms, but debilitated the system generally, as might *à priori* be supposed; and she felt much worse after than before the bleeding, the extracted fluid being almost all serum, with very little crassamentum.

In cases of tumors, they must, if they cannot be removed by local application of discutients, or mechanically, be treated on the same general principles as those before mentioned in the same class under the same head in general neuro-adynamia.

Bone, splinters, &c., may be generally removed mechanically, by means of the trephine, saw, knife, forceps, &c.

3d. Inanition.

This, as in general neuro-adynamia, may be from want of nutrition, or stimuli, or from loss of function, rendering nervous matter incapable

of receiving nutrition or responding to stimuli. The first, viz., *Lesions of Nutrition*, may be exemplified by the Fakirs of the East and other devotees, who, from false and misguided views, lose the use of their limbs or other parts of the body by maintaining them in one position, generally an upright or extended one, for a long time, thus draining the vessels and keeping them empty, or comparatively so, until the parts become atrophied or debilitated to such an extent as to become incapable of responding to impressions, or the dictates of the will, &c.

A similar condition is often temporarily produced by accidentally arresting the circulation to or in a part; as, for instance, to the arm or leg, when it gets in that condition commonly called "asleep." An analogous condition may result in any organ or part of the body, if the blood should be prevented from going to that part or organ.

In anæmia, although there may be an abundance of fluid, yet that fluid does not contain sufficient nutritive plasma, or stimuli; and in consequence, there may be an adynamic condition of almost any organ or part of the system, as well as the whole organism, exhibited in the form of headache, hemicrania, neuralgia, blindness, deafness, perverted sensations, or complete anæsthesia, or mere debility of the different parts or organs, as heart, stomach, uterus, bladder, &c., or local spasmodic or other perverted action, as chorea, dyspnœa, asthma, difficulty or privation of deglutition, &c., or mere tremor, as of the head or some other part, or partial or complete paralysis, as paraplegia, &c.

The same condition may be produced by drains, as hemorrhages, excessive lactation, seminal evacuations, or profuse secretion, or evacuation of any kind—all or any of these giving rise very frequently to amaurosis particularly, and in fact adynamia of any part, organ or function of the body. And in none is it probably more often exemplified than in the one which has just been mentioned, viz., the adynamia of the optic nerve, it seeming to be sooner affected than almost any other part of the system, as if it was intended by its prominence to report to, and warn us of some violation of the organic laws, the effects of which are thus forced upon our attention.

2*d. Lesions of Function.*—These, as in the general division, may result from mental disturbances of any kind, either of an exhilarating or depressing character, and also from great mental or physical labor, or prostration from any cause, with the production of derangement of any part or organ of the body, as well as of the whole body, thus causing an adynamia o the actions of the heart, with syncope, or a more permanent debility o its powers; of the uterus, os uteri and sphincter of vagina, causing abortions, which are very frequent during and after periods of great excite-

ment, as from epidemics, sieges, &c., or from the ordinary accidents of life. Also the relaxation of the other sphincters, as of the cardiac orifice with vomiting; of the rectum and bladder, with the consequent expulsion of the contents of the bowels and bladder, which are frequently the effects upon new recruits in a first battle; and the same effects also result in children and even adults from the ordinary excitements and impressions of life. Also local spasmodic action, as chorea, &c., confined to the greater portion of the body, limb, or even a single muscle, or partial or complete paralysis of the same. But there is also another condition which is not so often exemplified in local as in general adynamia, viz., in which there is debility without any perceptible cause, constituting that condition generally called "inanition," as of the heart, uterus, bladder, rectum, intestines, &c.; of the functions influenced by the cerebral nerves, as partial or entire privation of sight from debility of the optic nerve, or motor oculi, the latter allowing the iris to expand and thus admitting too many rays of light; deafness from debility of portio-mollis, &c.; and also the other functions depending upon these nerves, as the movements of the tongue, eyes, facial and masticatory muscles, &c.; paralysis agitans of old age, limited to the head, hands, legs, or any other part of the system. Also anæsthesia, and many forms of neuralgia where the general health appears to be good, as hemicrania, cephalalgia, sciatica, pains in the chest and in various other parts of the body, &c.

From electricity, as shown particularly in the effects of lightning, or from any other depressive cause, as concussion from falls, blows, &c., with the production of almost any form and degree, and of any part, of local spasm, anæsthesia, palsy, &c., thus hemiplegia, paraplegia, chorea, blindness, deafness, aphonia, dyspnœa, dysphagia, debility of heart, uterus, bladder, bowels, &c.

From local application and general exhibition of narcotics. This state is, however, most generally induced for medicinal purposes.

From poisonous action of the metals with paralysis and anæsthesia, as from lead, mercury, &c., often confined to the wrists and hands, but may also take place in the lower extremities, or any other part of the body, and be complicated with spasmodic action, of the intestines particularly, from lead, constituting the disease termed colica pictonum.

Treatment of Inanition.—In many cases of local inanition, although the diseased action or debilitated state be limited, yet it will require general treatment on account, in many cases, of the difficulty of affecting the parts by local remedies; in numerous cases, however, the local affection or condition may be remedied by local treatment.

Treatment of Lesions of Nutrition.—In Lesions of Nutrition, where the adynamia depends upon an arrestation of blood, if from any mechanical cause, the obvious indication is to place the parts in a proper position, or remove obstructions and thus permit the reflow of the blood, when all the symptoms will generally soon disappear, if the cause has not been continued too long, accompanied, however, by a peculiar tingling or prickling sensation as the circulation returns, being dependent most probably upon the stimulus of the blood, or oxygen in the blood, as it steals through the capillaries, and impinges on the sentient extremities of the nerves, analogous somewhat to the sensation from tickling; but if in the latter case the part be grasped more rudely, or in the former the blood be forced upon the nerves in a larger quantity and with greater impetus, by concussion or otherwise, its pressure removes that peculiar almost unbearable feeling. This same feeling seems to be experienced to some extent in the return to sensibility from paralysis or anæsthesia.

As this state of anæsthesia is so easily produced in the extremities by pressure on the vessels, the question arises *whether it could not be applicable for some practical purposes in the performance of operations*, and thus supersede in some measure the necessity for the general anæsthesia artificially induced?

In cases, for example, of required amputation of an arm or leg, or any part of them, by the preceding use of the tourniquet around the limb compressing the vessels of that limb, in a few minutes would arrest the circulation and thus produce a palsy and loss of sensation in it, and the operation might be performed with comparatively little shock to the system, and probably little or no sensation to the patient.

In local adynamia, as a consequence of anæmia, the first indication is to improve the blood and through it the system, and if there is an excess of serum to equalize the circulation. This may in the former be affected by stimulants, tonics, mineral and vegetable—the former particularly—alteratives, and those remedies which seem to have a more specific tendency to the different parts or organs affected; also diet, dress, and other hygienic measures. But in many cases after the blood is enriched and the system improved the local affection is not cured or relieved; it may then be necessary to resort to strychnia or electricity, to disturb or remove the inertia, or in similar cases combine the use of these with the other remedies above mentioned and at the same time.

In those cases in which there is an abundance or preponderance of serum, it might be supposed that removing the excess by bleeding or active purgation would relieve the condition and assist in the more rapid recovery, but on experiment this would be found to prove fallacious, except there were local congestion, &c., there being to some extent an

advantage in this condition of general plethora from serum, as by the mechanical pressure of the fluid upon the brain, &c., it probably prevents frequently the disposition to syncope, which is often experienced by persons in the anæmic state; and it may be from the want of the due amount of pressure or support from the fluids or blood-vessels upon the brain and nervous system, &c., as, in cases of spurious and in some of chronic hydrocephalus, pressure relieves and cures the disease; they differing, however—in the former requiring pressure from within, by refilling the vessels and by stimulation and improvement of the system; the latter from without, judiciously graduated, to support and at the same time to cause absorption of the accumulated fluid; and in anæmia, the brain and nervous centres acquire strength as the blood becomes richer and more abundant, thus filling the vessels, and by its pressure supporting and steadying the nervous system, at the same time affording nutrition and stimuli, which of course is the most beneficial. Other examples have been given in the preceding pages, as the injection of fluid in the veins in cholera, and the transfusion of blood, &c. This condition is also shown by the fact of some persons being able to hear distinctly when their heads are filled with blood, either from excitement or by a recumbent position, whilst in a standing position they are deaf; also in the fact of placing persons in a state of syncope in a recumbent position to cause a greater flow of blood to the brain and thus revive them.

In those cases resulting from drains from the system, they must first be corrected, and the same general course, as before mentioned, pursued to improve the system, at the same time avoiding all causes which produce such drains. Thus, in lactation, the child must be weaned or provided with a nurse; in seminal evacuations, either from excessive venery or masturbation, they must be avoided; and in excessive secretions, hemorrhages or drains of any kind, except as succedanii, must be arrested by the appropriate treatment, according to the character of the discharge, &c.

Treatment of Lesions of Function.—In these cases, where there are complications with general debility, or bad state of system, the treatment must be similar to that of "lesions of nutrition."

Where there is merely functional debility, without any general complication or local inflammation, &c. (except probably in the chronic variety of the latter with loss of tone, as in diarrhœa, dysentery, &c.), or after the effects of inflammation or concussion, &c., have been entirely removed, yet the adynamia remains, there are no remedies which are probably superior to those of strychnia and electricity (including in this latter term, for brevity, all the analogous agents); and it is only in these cases in which they appear to be at all applicable, although

they have been used indiscriminately in almost all forms of adynamia, both general and local, and in many cases with injury, which must necessarily result when the adynamia is dependent on some "modification of structure," or physical disturbance; hence by many they have been thrown aside as useless or injurious; but, on account of our deficient or limited knowledge of physiology, pathology and therapeutics, the fault is probably rather in the application of than in the remedy. The same might be and is said of opium, mercury, the lancet, &c., but it does not prevent the judicious practitioner from resorting to them when the indications call for their use. In general terms, these remedies are applicable in all cases of local and also general adynamia, where there is no modification of tissue, inflammation, &c., or physical impediments or disturbances, or where these have been removed and the inertia still remains, particularly in those cases in which the connection between the voluntary and involuntary movements are not entirely broken. Where the connection is severed, they seem to be particularly injurious, as their tendency is directed more especially to the involuntary actions; thus, if strychnia is given in complete paraplegia, &c., it appears to increase the disease and separate still further the voluntary from the involuntary movements, and hence prevents the reunion; but in a case of functional debility, where the two have not been separated, it is particularly applicable, as its action is directed primarily to the weaker part, or the debilitated nerve centres appreciate or respond to its stimulus the sooner.

In those cases, however, of adynamia of the involuntary organs, as of the heart, intestines (as in chronic diarrhœa, dysentery, &c.), bladder, &c., strychnia seems to be peculiarly appropriate, and acts more specifically than any other remedy in giving tone and strength. In many cases in which general treatment is also indicated, it would no doubt be usefully combined with the other tonics, &c.

Electricity is also applicable to many of these local forms of adynamia, and has been used with success in amaurosis, chorea, uterine inertia or paralysis, and almost every form of local prostration or inanition, where this condition was merely functional, and the general health was not deranged or had been improved. This latter appears to be a point of some importance, and the neglect of it will probably explain the reason of the failure in many cases of treatment and remedies, and of these two more especially, viz., strychnia and electricity, the latter particularly; the former at the same time that it is acting on the adynamic part is also improving to a certain extent the general condition, hence the advantage, in many cases, of its combination with the other tonic preparations and stimulant treatment, in both general and local adynamia.

In all cases therefore in which such remedies are indicated, it will in the first place be necessary to ascertain the condition of the general system; if this be good, the use of them will no doubt prove immediately beneficial, if not curative; but if the condition of the system is bad or below the natural healthy point, the general health should first be corrected, and by the subsequent use of such remedies, the local affection or inertia may most generally be removed or corrected, if it has not been, as it often is, remedied by the general treatment, the local affection being in numerous instances dependent upon the general derangement or debility, and disappearing upon its correction.

Ergot is also highly beneficial in the inertia of the uterus and bladder, acting almost specifically in the former, particularly in the debility frequently attendant upon and resulting from parturition, in the retention of the placenta, and in the arrest or prevention of uterine hemorrhage; and from analogy it might be supposed that it would also prove useful in local adynamia of other organs, as the heart, &c.

For the prevention of abortions, mental quietude, anodynes, antispasmodics, tonics, &c., and other treatment with general hygienic measures, modified by the condition of system, temperament, habit, &c., of the patient.

Those cases resulting from the metals, such as palsy, lead colic, &c., must be treated on the same general plan as described in the general division, viz., avoidance of the exciting cause, bathing, exercise, tonics, strychnia, electricity, &c.; the iodide of potassium, as recommended by Messrs. Guillot and Melsens, cathartics, &c. In lead palsy and colic, in addition, the sulphates of magnesia, of alumina and potassa or common alum, with the use at the same time of the preparations of mercury, &c.

The adynamia resulting from narcotism will generally disappear if the exciting cause is removed; but it is applicable to a great variety of useful purposes both in surgery and medicine; thus for instance to the relaxation of the sphincters, &c., and for this purpose the class most appropriate is that of which belladonna is the type, and may be used for the dilatation of the iris, of the air-cells of the lungs, of the intestinal fibres and rectal sphincters, of the os uteri, of the perineum in labor when the child's head presses too hard upon it, thus permitting its easier passage, and removing the greater tendency to the rupture of that part; of the abdominal ring in the reduction of hernia, &c. From the rapidity and certainty of its action, atropia in solution would be most appropriate in the majority of these cases.

The application of atropia, &c., externally in hernia, to allay the irritation and relax the constricting tissues, and the exhibition by the mouth, and by injection per rectum, of astringents and stimulants, as tannin,

acetate of lead, opium, &c., to contract the intestinal tissues, and thus withdraw the incarcerated intestine through the stricture, assisted by position, and, if necessary, the taxis, would most probably afford a safe and effectual mode of reducing hernia, without, in many cases, the necessity of resorting to other means, or an operation.

In other cases where local anæsthesia only is desired, another class would be more appropriate, the type of which is aconite, chloroform, or even opium; in such cases as neuralgia, &c.; the application to tumors and surfaces before excising or cauterizing them, such as carbuncles, &c.; abscesses before opening them; to mammary and other glands before excision; to piles before strangulation, &c.; and it is probable that local anæsthesia might be substituted in most cases of minor surgical operations, instead of resorting to the, in many cases, unnecessary and frequently dangerous general anæsthesia so indiscriminately induced at the present time, and occasionally with such injurious and even fatal effects.

There is another application of local anæsthesia, viz., as a prophylactive, before operations, particularly those involving the serous membrane, as the peritoneum in paracenteses, hernia, &c.; wounds or injuries, where not too extensive, to prevent the supervention of tetanus, irritative fever, inflammation, &c., which result as a consequence of the above.

Where the wound, &c., is very extensive, the substitution of water impregnated with aconite or other sedative, for the more concentrated narcotics, &c., would prove no doubt highly advantageous, or water alone, as used so extensively and beneficially at the present time as a dressing, probably owes its virtues to its acting to a great extent in a similar manner, by producing a state of sedation of the nerves, at the same time contracting the tissues, thus regulating the quantity of blood in the part, and in this way preventing too extensive irritation or inflammation. An eminent surgeon, Sir A. Cooper, speaks very highly of it as a prophylactic against tetanus. He states that "he has never seen tetanus come on when wounds, however severe and likely to produce it, were healed under water dressing." (*Water* versus *Hydropathy*, by Dr. H. Hartshorne, p. 115.)

4th. Sympathy.

The predisposition in this may be induced in the same way as in general sympathetic adynamia. The disturbing or exciting causes may also be located in any part or organ of the body, and the irritation thence reflected to any other organ or part of the system, thus producing local derangement of any or of every kind, exhibited in the forms of cho-

rea, which may be confined to an arm or leg, fingers, single muscle, or any other portion of the body, or almost all of it, local spasms, amaurosis, deafness, or disturbance of any of the cerebral nerves, thus deranging the actions of the parts to which they are distributed, producing either excessive or deficient or entire privation of motion or sensation. Also adynamia of some of the organs, as the heart, with arrest of or impeded action, from sickness of stomach, or from action of remedies, as digitalis, antimony, &c.; stomach, with vomiting from pregnancy, sea-sickness, &c.; uterus, with the production of abortion from the relaxation of the sphincter of os uteri, from cerebral, spinal, and intestinal impressions, &c.

Another class which does not strictly come under the head of adynamia, yet is often indicative of that state, as proved by the juvantia and lœdentia, viz., neuralgia, in its various forms of hemicrania, sciatica, cephalalgia, &c.

In many cases, there may be a complication of symptoms, particularly about the head and thorax, &c., with impeded action of the muscles of respiration, &c., such as dyspnœa, asthma, neuralgia, &c., or convulsive action or paralysis, with or without anæsthesia of the face or some parts of it, or any parts of the body.

Treatment of Sympathy.—This must be in many respects the same as that mentioned under the same head in the general division. The cause and its location must first be ascertained and removed, if possible, and if it cannot be removed, as sometimes in sickness of stomach from pregnancy, must be palliated, till the condition upon which it depends has terminated, by, occasionally in the case of pregnancy, expulsion of the contents of uterus. This palliation or quieting may be effected in many instances in pregnancy by stimuli, as champagne wine, tonics, &c., but in the majority of cases, which would, however, seem to be cases of hyperdynamia, by anodynes, which, by coming into direct contact with the nerves of the stomach, narcotize them, and thus prevent their responding to those transmitted impressions, or by entering the circulation, act upon the whole nervous system, and in this way allays, temporarily, the disturbance; and in all such cases of sympathetic derangement, where the cause appears to be internal, the internal exhibition of anodynes would be indicated, except where the disturbance arose from irritable ingesta, secretions, worms, &c., in alimentary canal, when they must be preceded by emetics, cathartics, antacids, vermifuges, &c. In cases of chorea and analogous affections, this treatment should be preceded or accompanied, premising always that the offending substance or condition has been removed, which is of itself often adequate to the cure, by tonics, both mineral and vegetable, chalybeates particularly, alteratives and general hygienic

measures, unless resulting from inflammation, congestion, &c., when it must be treated antiphlogistically.

In all those cases, however, in which the exciting cause is external to the part to which the sensations or impressions are reflected, it may be quieted on the same general principle by the local application of the sedative to the punctum irritatio, thus by the induced narcotism preventing the nerves of the part from transmitting impressions. The anodynes most appropriate and useful in these conditions are aconite, opium, chloroform, camphor, &c.

In all those cases of paralysis, spasmodic action, &c., as amaurosis anæsthesia, twitching, &c., it may be necessary to stimulate by means of nervous stimulants, tonics, &c., as musk, particularly in that spasmodic contraction of the diaphragm, the effect of which is known as "hiccough," valerian, quinia, strychnia, electricity, &c.; always removing previously of course the exciting cause, and particularly before using either strychnia or electricity.

Many of the sympathetic disturbances depend on anæmia or debility, and by curing this condition will disappear, such as sciatica, hemicrania, cephalalgia, spasmodic and other irregular actions, &c., hence tonics, particularly the chalybeates, nervous stimulants, corroborants, proper diet, dress and hygienic measures generally. For some cases of hemicrania, however, Dr. Watson (Watson's *Practice*, p. 444) highly recommends the muriate of ammonia, in "ʒss doses, dissolved in water, three or four times a-day," to the beneficial and speedily curative effects of which the writer can testify in one case which came under his care. Dr. Watson states that "if the pain does not yield after four doses, you may cease to expect any benefit from it."

Many cases of neuralgia, of the chest particularly, have been cured by Professor Jackson by the simple addition of clothing; thus, according to his directions, having a jacket quilted with lamb's wool or cotton, and wearing it next the skin, will often relieve the affection like a charm, causing the pain to subside when everything else had failed. Probably silk clothing, or silk and woolen combined, and worn next the skin, would also have the same beneficial effects.

With respect to "electrical insulation" in neuralgia, Mr. Pallas remarks that "its most marked and speedy effects were exhibited in neuralgia." (*British and Foreign Medico-Chirurgical Review*, No. vi. p. 386.)

Those cases depending on inflammation or congestion, antiphlogistics, counter-irritation, astringent and anodyne applications, &c., will often relieve. These latter cases may and often do cause a complication of symptoms:—thus, a very interesting one was afforded me by the condi-

tion of a medical friend, who, after convalescence from an adynamic fever induced by the inhalation of chloroform, and in fact during his illness, but not to such an extent, was troubled with pain in the chest, coughs, dyspnœa, &c., so much so that his friends imagined him to be phthisical. As he had, however, soreness along the spine, and particularly about the origin of the phrenic nerves, I was induced to believe that those symptoms were sympathetic, and resulted from a passive congestion with a subacute inflammation of the spinal cord, and in consequence recommended him to apply a large pitch and cantharides plaster along the spine, which he did, and with the happiest effect. All of the pulmonary symptoms began gradually to subside as the plaster acted, and ultimately, under its use, entirely disappeared. Since then, I have had another opportunity of observing a similar state of things, in the person of the same gentleman, who was attacked with neuralgic pains down his arms and through his chest, and other unpleasant feelings throughout the system, with a general malaise, which he believed would be all removed by a cathartic dose of calomel and jalap, which operated, and notwithstanding ptyalism supervened unintentionally, yet those pains and unpleasant feelings, &c., did not disappear until counter-irritation along the spine was employed.

ON

BENGAL DYSENTERY

AND

ITS STATISTICS,

WITH

A NOTICE OF THE USE OF LARGE ENEMATA IN THAT DISEASE,

AND OF

QUININE IN REMITTENT FEVER.

By
JOHN MACPHERSON, M.D.,
1st ASSISTANT PRESIDENCY GENERAL HOSPITAL.

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CONTENTS.

To SIMON NICOLSON, Esq., *F.R.C.S.*,

Surgeon of the Presidency General Hospital.

My Dear Sir,—To whom could I with greater propriety present these gleanings from the records of the Institution, with which your name has been so honorably associated for more than a quarter of a century, than to you, even did I not lie under a weight of personal obligation to yourself, which I am proud to acknowledge?

Had the Medical and Physical Society been in existence, or were there now any local Medical Journal, these pages would probably not have appeared in a separate form. Bengal Dysentery has been admirably described by former officers of this Hospital. The present is a slight attempt, but one of the first of its kind, to apply the numerical method to the subject. It is not an exposition of opinions, but a statement of facts, (perhaps too copious in detail,) from which the reader may draw his own conclusions. I have not thought it desirable to expand these materials, as they might easily have been expanded, into a regular treatise.

In the numerical statements it has been found impossible to obtain absolute accuracy, but I have had the advantage of comparing them with some carefully constructed tables of Mr. Hare's, and they are accurate enough for all practical purposes. You will be surprised at the high rate of mortality

that has been found to prevail, but if a rigid analysis of cases were made, and if those which were evidently the terminations of other maladies, and those which died within twenty-four hours after admission, were excluded, the mortality would be considerably less.

I should hope, that the short historical notice of the use of enemata in dysentery, and of quinine in remittents, may, at the present time, be of some interest to you and to the profession in this country.

Believe me,
My dear Sir,
Your's very sincerely,
JOHN MACPHERSON.

General Hospital:
May 20th, 1850.

ON

BENGAL DYSENTERY.

INTRODUCTION—TREATMENT.

I propose—1st, to give some returns of the mortality from dysentery, and of the diffusion of the disease throughout the year, from the books of the General Hospital, and to make a few comparisons with what is found to occur elsewhere.

2nd, to give an abstract of the pathological appearances presented in 160 cases of acute and in 55 of chronic dysentery, which have occurred during the last nine years.

The symptoms and history of Bengal dysentery have been so fully described by Twining and Raleigh, that it is unnecessary on the present occasion to add any thing to what they have said on those heads: nor do I intend to say more than a few words on the management of the disease. Practitioners seem to have been gradually losing faith in the mercurial treatment, which dates back from the latter half of the last century, and has often been exclusively followed. The virtues of ipecacuanha, also a very old remedy, though they were brought prominently forward by Twining, are not so much trusted to now as formerly. Opium again, which since the days of the liquid laudanum of Syden-

ham down to the present time, has been more or less used, but which had latterly got the character of masking the disease, seems of late to have regained its rights. In Bengal, Drs. Mackinnon and Goodeve and others have suggested the propriety of returning to a more free use of this drug, and that most sound observer, Dr. Morehead of Bombay, has borne testimony to its value. My own belief is, that the return to a milder and more soothing treatment has been attended with great advantage—though it may be difficult to prove this by reference to any Hospital records within reach. One obvious difficulty in arriving at safe conclusions is, that the disease itself varies so much in intensity in different years, as well as in different periods of the same year.

Thus, H. M. 55th at Secunderabad, lost in 1837.. 1 in 4½
1838.. 1 in 7½
1839.. 1 in 10

yet the treatment was the same, and by the same Medical Officer throughout.

The only data bearing on this point in my possession are afforded by the records of the Seaman's Hospital. Such as they are, they are subjoined, though they must be taken with great reservation—1st, because the number of facts is small—2nd, because it is possible to support by statistical returns almost any pre-conceived notion.

Treatment by bleeding and calomel for five years, ...	227 cases,	48 deaths, or 21 per cent
Treatment by leeching, opium and astringents for three years, ..	80 „	10 „ or 12 per cent.

Still I would not be understood as advocating any exclusive treatment. In an account of the latest Irish epidemic, a most intelligent physician, Dr. Mayne, states—"opium most certainly aggravated the disease." "Mercury must be considered the principal remedy." As the character of dysentery varies, so no doubt should our practice.

I may allude here, as being quite a curiosity in its way, to the most singularly successful mode of treatment that I have seen on record. Mr. Marbot tells us, that in a French vessel on the coast of Zanzibar, he has treated 300 cases of dysentery with aconite and ipecacuanha without a single casualty. Two things seem very plain: first, that Mr. Marbot deceived himself: second, that the disease he treated is something very different from Bengal dysentery.

GENERAL STATISTICS.

MORTALITY FROM DYSENTERY.

1. *Out of Bengal.* It is difficult to procure any accurate statistical data regarding the proportion of deaths to cases treated in Northern Europe, and dysentery does not occur in Major Tulloch's tables of the diseases of troops in Great Britain. Dr. Williams of St. Thomas', however, states, that more than 25 per cent. of cases of chronic dysentery die in the London Hospitals, and says, "in candour it must be allowed, that no class of diseases in them offers so few chances of recovery." In Dublin, Dr. Mayne lost 32 per cent., or omitting cases in old men after the age of 60, as much as 22 per cent.; children under 10 years died at the rate of 65 per cent. At Gemünden, in Southern Germany, where dysentery is often epidemic at the fall of the year, the mortality during the great epidemic of 1834 was 11 per cent. In the Peninsular war, scarcely more than 2 out of 3 recovered. Further South in Malta, which can scarcely be said to have an European climate, the mortality among H. M.'s troops is about 8 per cent.

The accompanying table, compiled from Major Tulloch's reports, shows the percentage of deaths to cases among H. M.'s troops in various parts of the world:

Bermuda, ...	3.2	Mauritius, ...	6.0
Nova Scotia, ...	7.4	Jamaica, ...	4.3
Canada, ...	5.0	Windward and Leeward Islands, ..	7.1
Mediterranean, ...	4.0		
St. Helena, ...	10.6	Ceylon, ...	13.0
Sierra Leone, ...	18.0	Tenasserim, ...	8.5
Cape of Good Hope, ..	4.7	Three Presidencies, ..	8.3

2. *In Bengal* Major Tulloch gives the mortality of H. M.'s troops at 8 per cent., and Dr. Mackinnon finds, that among the European troops for 7 years at Cawnpore, it was 6.16. In Calcutta and its neighbourhood, the percentage among European troops appears to be rather more than 9 per cent.; but it varies much, for some regiments have suffered much more heavily. Thus, H. M.'s 21st Fusiliers lost in Fort William and Chinsurah, at the rate of 17 per cent.; and H. M.'s 70th, in crowded barracks at Dum-Dum, suffered at the rate of 35 per cent. during the first six months after its arrival from England.

3. In an Institution like the General Hospital, to which patients are often sent in the last stage of disease, and which receives all sick soldiers left behind by their regiments, as well as all sick invalids on their way home, one would naturally expect to find a high rate of mortality. Raleigh gives it as from 10 to 14 per cent., and Martin says, it is 10.27.

But the following table of admissions and deaths during the last 20 years will shew that they have greatly underrated it. The mortality from all "bowel complaints" is about 19 per cent., much higher than the rate assigned by them, but not equal to the reality. Of course the mortality in returns can be made to vary much according as cases are classed under the heads of diarrhœa or dysentery, a point often requiring nice discrimination.

This will be abundantly evident if we contrast the proportion of deaths from bowel complaints, with that from dysentery during the last 16 years, in periods of four years.*

	1834—38.	1838—42	1842—46.	1846—50.
Bowel complaints, ..	68.6	71.8	70.8	69.
Dysentery, ...	24.3	21.2	25.6	26.3

Thus, while the mortality from dysentery has, on the whole, been increasing, that from all bowel complaints has varied to but a trifling extent.

The following have been the admissions and deaths from dysentery in the General Hospital, from 1830 to 1850:

Year.	Admission.	Deaths.	Year.	Admission.	Deaths.
1830	144	22	1840	68	11
31	128	20	41	172	42
32	124	20	42	147	41
33	128	19	43	88	19
34	147	39	44	141	28
35	71	20	45	91	31
36	55	10	46	87	27
37	54	15	47	78	17
38	52	13	48	87	25
39	78	15	49	94	24

Total admissions 2044, total deaths 457, or 22.3 per cent.

The extremes of mortality have been 14.8 in 1833, and 34. in 1845.

The average is higher than that of the Bombay General Hospital, which, for a period of 5 years, was 18.3, and lower than that of the Madras one, in which, for a period of 10 years, it was 30 among Civilians, though only 5.3 for the Military. The average mortality in a series of years appears to have been almost the same in the Calcutta General and Medical College Hospitals, and the extremes in the latter have been 14.1 and 33.

The mortality in the year 1840 in the General Hospital was 16, in 1849, 25.5, and in the Medical College Hospital at the same periods 25, and 27.

* I have taken a period of 16 years, because in the year 1834 there was a sudden increase of mortality in bowel complaints, which has ever since continued. This was the year of the great Continental epidemics of dysentery, and what is perhaps more to the point, the port of Calcutta began to be crowded with ships, many of an inferior class.

In the table of admissions and deaths, acute and chronic dysentery are classed together: indeed, they cannot be separated with advantage, and their severance in Major Tulloch's tables gives rise to some very strange results. In them chronic dysentery (which is returned as infinitely more fatal than acute) is made to kill in Malta 1 in 4, while in the Mauritius it kills only 1 in 14½, and deaths by acute dysentery are made to vary from 1 in 57 in the Bermudas, to 1 in 2½ in Sierra Leone. There is manifestly some error in such statements.

On analysing the classes, among whom the mortality occured in the General Hospital during the only two years, 1847 and 1849, in which that analysis can be easily made, we find that the mortality of the Military in those years was 10 and 21 per cent., giving a mean of 15½ per cent., which is considerably less than that of the non-military.

It might be expected, that some statement should be given of the average period after the first invasion of the disease, or after admission into Hospital in which death occurs, and it may be stated, that death within a week from the first attack is extremely rare. But patients are so seldom brought to the General Hospital at the commencement of the attack, and the attack itself varies so much according to the prevailing character of the disease, that no such statement would be satisfactory. In like manner, almost all the dysenteric patients being soldiers and sailors, are between the ages of 18 and 40, and the chief mortality is of course between those two periods of life. Most admissions also occur between the ages of 18 and 30, and consequently most deaths.

The number of fatal cases among women is extremely small: for in the following tables of the appearances in 215 dissections, the names of only 5 females are found. I have imagined, that Bengal acute dysentery is peculiarly fatal in boys from 14 to 20 and in middle aged men: boys suffer less from the chronic form. I have been repeatedly astonished to discover after death an immense extent of structural

change in boys whose illness could not be ascertained to have exceeded 8 or 10 days.

It may be finally remarked, that about 1-10th of all admissions into the General Hospital are cases of dysentery, and that while 10 to 11.5 per cent. is the average mortality on all admissions, that on dysentery is about 22, cholera and hepatitis alone proving more fatal.

PREVALENCE OF THE DISEASE AND MORTALITY FROM IT, ACCORDING TO SEASON.

I. 1. *Out of India.*—The following are a few notices of the prevalence of the disease, according to season, in Europe. In the Infirmary for children in London, Dr. West found the rate of prevalence of dysentery to be, spring 8.3: summer 13: autumn 24.4: winter 7.2. In the last Dublin epidemic, in the year 1847, the admissions in the Work-house were, 1st quarter 136: 2nd quarter 159: 3rd quarter 206: 4th quarter 157:—and at Grätz, where dysentery is an autumnal disease, the distribution was—

Spring 8 cases: Summer 67: Autumn 163: Winter 11.

2. *In India,* Annesley found on the large scale that out of 13,900 cases which occurred in 5 years in Bengal, 2,400 were in the cold, 4,500 in the hot and dry, and 7,000 in the hot and moist season. This accords with the results in Ceylon among Queen's troops, who have most cases of dysentery in the second and third quarters of the year. In like manner Dr. Mackinnon found at Cawnpore, in a series of 7 years, that dysentery among Europeans was most frequent in the rains, next in the hot weather, least frequent in the cold season, and far most frequent in the months of August and September. The results in the General, the Seaman's, and the Medical College Hospitals, will be found generally similar, although the disease is in Calcutta somewhat more prevalent

in the cold than in the hot weather. These points will be illustrated by the following tables. The prevalence of the disease, according to season, is modified by the climate of the particular place, and we thus find, that in the General Hospital at Bombay, it is said to be most common in the cold season.

Table of Admissions and Deaths from Dysentery in the General Hospital, for 10 *years.*

Months.	1840.		1841.		1842.		1843.		1844.		1845.		1846.		1847.		1848.		1849.		Total Admission.	Total Deaths.	Percentage of Deaths.
	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.			
Jan...	6	1	14	2	7	1	8	3	10	0	18	3	10	2	6	1	21	4	7	1	107	18	16
Feb...	5	2	7	2	6	2	19	0	5	2	5	0	3	2	6	2	5	1	4	1	61	14	23
Mar. .	6	1	13	3	7	2	8	2	5	2	1	1	1	2	4	2	3	0	4	1	52	17	32.6
April,.	6	0	4	1	4	1	6	1	6	2	2	1	7	1	7	0	7	2	7	1	56	10	14.2
May, .	4	0	13	3	8	3	2	2	5	1	4	0	9	3	8	3	7	1	8	5	66	21	31.8
June, .	1	2	6	3	10	2	6	1	10	7	9	3	3	1	3	1	6	2	5	0	59	22	37
July, .	4	1	21	2	15	3	7	3	13	7	10	5	11	3	8	3	5	2	12	3	106	32	30
Aug. .	6	0	17	7	18	7	6	3	10	1	11	5	7	2	5	0	9	2	10	1	98	28	28
Sept. .	5	1	19	5	25	8	6	0	3	2	4	2	7	3	5	1	9	3	7	4	96	29	30
Oct...	11	0	21	7	10	7	5	0	5	2	5	1	3	1	7	3	8	4	12	3	87	28	32.1
Nov. .	7	1	4	4	25	2	8	2	21	1	8	2	16	2	10	1	4	3	9	2	112	20	17.8
Dec...	7	2	33	3	12	3	7	2	48	1	14	7	10	5	9	0	3	1	9	2	152	26	17
Total,	68	11	172	42	147	41	83	19	141	28	91	29	87	27	78	17	87	25	94	24			
Percentage of Deaths.	16		24.4		27		21.5		20		31.8		30		21.8		28.7		25.5				

The average prevalence of the disease in the different months is fairly enough represented in the foregoing table: if we make a few corrections to allow for the increase of cases caused by the arrival of the invalids of the season in

the end of November, and in December and January, the number of cases would stand thus—

January, ...	75	July, ...	106
February, ...	60	August, ...	98
March,..	52	September, ...	96
April, ...	56	October, ...	87
May, ...	66	November, ...	82
June, ...	59	December, ...	80
1st half of the year,	368	2nd half of the year,	549

or, according to season, in the following proportions: four cold months 74, three hot ones 68, five of rains 88.

The results are nearly the same in the College Hospital.

3. Regarding the degree of prevalence of the disease in different years, nothing of distinct value can be gathered from the Hospital records, because the number of admissions is in great measure dependant on the number and strength of detachments of troops arriving at the Presidency and on the number of shipping lying in the river. Thus the year 1842, in which there were most admissions from bowel complaints, was that of the return of the invalids from Chusan.

II. Regarding the rate of mortality according to season, out of India I possess no data, but if we proceed to investigate the rate of mortality in the General Hospital, we find it to be in this proportion, almost equal in the first and second halves of the year; but according to season, cold weather 18.4: hot weather 26.2: rains 31.4.

The results in the College Hospital are nearly the same. The most fatal months in the General Hospital have been March, May, June, and October, and in the College Hospital May, June, August, September and December. In Ceylon the disease was most fatal in April, May, and June, while at Cawnpore, October and November were the worst months. This comes nearest to Bombay, where the cold season is said to produce most deaths, and the monsoon, or July, August and September, next most.

*

The information on this point is not very full or satisfactory, but probably for India generally, August, September and October will be found to be the most fatal months, as the fall of the year is in Europe, though perhaps from different causes.

The unusually high mortality in the General Hospital in the month of March, when the prevalence of the disease is smallest, cannot be very readily explained.

Nor can any satisfactory information be given as to the rate of mortality in different years. It was remarked above, that the average mortality in a *series of years* was the same in the General and in the Medical College Hospitals, yet in *individual* years the mortality in the two institutions differed most widely.

Through the kindness of Dr. Mouat, I am enabled to give the following tables of admission and deaths among Europeans and Natives in the Medical College Hospital, during the last 10 years.

Table of Admissions and Deaths of Europeans from Dysentery, in Medical College Hospital.

Months.	1840.		1841.		1842.		1843.		1844.		1845.		1846.		1847.		1848.		1849.		Total Admission.	Total Deaths.	Percentage of Deaths.
	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.			
January,	8	0	2	0	10	1	3	0	8	0	9	2	9	1	12	4	8	0	12	1	82	12	14.6
February,	8	1	1	0	12	2	4	0	10	2	8	1	13	1	11	1	10	2	8	2	81	13	16
March,	6	1	5	0	8	2	7	2	10	2	10	0	15	1	10	1	13	0	11	2	93	11	11.8
April,	5	0	3	0	9	3	8	1	7	0	14	3	6	2	11	1	10	3	11	1	99	16	17.7
May,	4	2	7	2	12	5	9	2	1	1	11	4	9	2	10	1	15	4	13	7	91	30	33
June,	6	1	10	2	6	3	11	3	9	0	9	4	6	1	10	0	16	1	13	7	83	24	28
July,	4	0	8	3	7	3	13	3	2	0	21	3	18	4	15	2	18	6	16	3	124	27	21.7
August,	6	3	9	3	13	4	16	4	5	2	17	4	18	3	12	4	23	4	12	3	129	34	26.3
September,	6	2	17	7	6	2	17	4	10	2	16	3	18	2	10	2	14	6	13	3	125	33	26.4
October,	7	2	13	3	10	3	14	2	9	1	12	1	16	2	13	3	10	2	11	4	116	23	19.8
November,	5	2	7	2	7	3	13	3	11	2	18	2	14	0	11	3	19	1	10	2	127	21	16.5
December,	2	1	6	1	12	6	11	2	16	4	24	2	14	2	10	1	17	3	4	1	117	31	26.5
Total,	55	14	87	25	112	37	127	31	99	17	169	32	156	22	136	23	179	32	134	36			
Percentage of Deaths ..		25.5		28.7		33.0		24.4		19		19		14.1		17		17 8		27			

Table of Admissions and Deaths of Natives from Dysentery, in the Medical College Hospital.

Months.	1840.		1841.		1842.		1843.		1844.		1845.		1846.		1847.		1848.		1849.		Total Admission.	Total Deaths.	Percentage of Deaths.
	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.			
Jany.	5	1	8	1	5	2	5	2	6	1	8	2	8	1	11	1	7	0	6	0	68	11	16
Feby.	5	0	3	2	4	2	3	0	6	1	7	0	6	1	7	1	8	1	9	2	58	10	17
March	3	3	4	0	3	1	5	1	6	2	9	0	8	0	7	1	6	1	5	0	56	9	16
April,	4	1	3	0	2	1	6	2	2	0	8	3	7	1	6	0	7	1	7	1	52	10	19
May,	8	0	5	0	3	1	9	4	4	1	9	0	8	0	7	0	7	0	9	1	69	7	10.1
June,	5	4	6	1	6	0	5	1	5	0	11	0	2	0	4	0	5	0	7	2	56	8	14
July,	6	2	3	1	5	1	3	1	6	2	7	2	7	2	8	0	7	0	7	0	59	11	18.6
Augt.	7	2	6	0	6	1	8	2	10	1	8	1	4	2	12	4	6	1	6	2	73	16	21.9
Septr.	5	2	4	1	5	1	4	2	7	2	8	0	6	0	4	0	11	0	9	0	63	8	12.6
Octr.	4	0	3	0	3	1	6	1	9	1	5	1	9	2	9	2	7	0	5	0	60	8	13.3
Novr.	4	3	4	1	5	1	7	2	7	1	8	1	4	0	10	0	8	2	3	1	60	12	20
Decr.	4	2	2	0	3	0	9	0	10	2	8	2	8	1	7	1	8	2	2	0	61	10	16.2
Total,	60	20	51	7	50	12	70	18	78	14	96	12	77	10	92	10	87	8	75	9			
Percentage of Deaths.	33		13.7		24		25.6		18		12.4		13		10.8		9.2		12				

Though it is foreign to the object of these pages to treat of dysentery among Natives, it is worthy of remark, how uniform the rate of admission among them seems to be throughout the year. Though they do not vary much, the admissions are most numerous in August and in May, while the mortality is highest in August, and next highest in November. The general results are much the same as with Europeans.

The average mortality among Natives has been 16.9, that among Europeans 22.5. This accords with general experience, which has shown the disease in Natives to be more amenable to treatment than in Europeans.

PATHOLOGICAL FACTS.

PRELIMINARY REMARKS ON TABLES.

The following tables exhibit a true representation of the structural changes commonly effected by fatal Bengal dysentery. Cases complicated with pthisis, syphilis, or any other constitutional taint, have been, as far as possible, excluded,—(and here I cannot help remarking, how strange it seems that Rokitansky should have asserted the antagonism of phthisis and dysentery, which in this place so commonly occur together.) An abstract is given of the pathological appearances in all cases which are at all fully recorded in the Hospital books, so that the reader may draw his own conclusions from them. A few results only have been noted, but every opinion of a theoretical nature is studiously avoided. On going over the records of these cases it is impossible not to be struck with the fact, that no two observers saw with the same eyes. Thus one gentleman has invariably found the mesenteric glands enlarged, while another describes the liver as dry in one half of the cases in which he mentions its condition. Where there has been no notice of the state of a particular organ, the space is left blank. In such cases its condition may be presumed not to have differed widely from the normal one. In the column of remarks any striking variation from the usual symptoms is noted.

ACUTE DYSENTERY.

No.	*State of large Intestines.*	*State of small Intestines.*	*State of Liver and Gall-bladder.*	*State of Mesentery, Mesenteric glands or other organs.*	Remarks.
1.	Cœcum and colon in a sloughing state throughout.	.. ..	Cicatrix of ulcer on lower surface of right lobe: bands of adhesion.	.. ..	Seaman—died in 3 weeks.
2.	Cœcum ulcerated and sloughing, firmly tied to omentum: ulcers in transverse arch and sigmoid flexure of colon.	Stomach natural.	Liver natural.	.. ..	Seaman, ætat 42—died in 20 days.
3.	Cœcum slightly injected, one or two small points of ulceration: these increased in the transverse arch and sigmoid flexure. Rectum one mass of ulceration.	Small intestine healthy.	Liver healthy. Gall-bladder with healthy bile.	.. ..	Soldier caught dysentery from sleeping on deck—died in 20 days.
6.	Spotted ulceration of large intestine, increasing as it went downwards: internal ulceration, not perceptible outside.	Small intestine distended.	Liver large, pale and soft. Gall-bladder flaccid: a little yellow pale fluid.	Effusion into mesentery.	Engineer—died in 10 days.
7.	Slight partial ulceration of cœcum. General ulceration of lower portion of large intestine.	Healthy.	Liver large and pale. Gall-bladder distended.	.. ..	Townsman—died in 15 days after 1st attack.
8.	Ulceration of large intestines.	.. ..	A hepatic abscess.	.. ..	European Seaman—whole illness 12 days.

No.	*State of large Intestines.*	*State of small Intestines.*	*State of Liver and Gall-bladder.*	*State of Mesentery, Mesenteric glands or other organs.*	Remarks.
9.	Much ulceration of large intestines.	.. ..	Liver enormously enlarged.	.. ..	European from house of correction—died in 31 days.
10.	Cœcum and transverse colon healthy: mucous coat of the descending down to rectum, ulcerated and sloughing.	.. ..	.. ..	.. ..	Seaman—died in 21 days.
11.	Much ulceration at cœcum and sigmoid flexure, coats thickened and easily torn.	.. ..	Liver much enlarged: abscess in right lobe. Gall-bladder half full of greenish thin bile, ducts pervious.	.. ..	Seaman—died in 13 days.
12.	Cœcum in state of mortification, transverse coat and sigmoid flexure covered with minute ulcers.	.. ..	Liver large. Gall-bladder full of dark bile.	.. ..	Seaman—died in 22 days.
13.	Large intestine congested, mucous membrane sloughing: large coagulum in colon.	.. ..	Liver large and gorged.	Mesenteric glands enlarged and indurated.	Seaman—ill a few days: doing well—suddenly passed a quantity of blood and died.
14.	Mucous surface of whole large intestines sloughing.	.. ..	Liver large and mottled. Gall-bladder full.	.. ..	Boy of 14—ill three weeks.
15.	Cœcum in sloughy state, distended with large coagulum of blood: colon inflamed and ulcerated.	.. ..	Liver enlarged, pale—abscess in posterior portion of right lobe.	.. ..	Seaman from house of correction—ill say a fortnight—died suddenly after passing a quantity of blood.

No.	*State of large Intestines.*	*State of small Intestines.*	*State of Liver and Gall-bladder.*	*State of Mesentery, Mesenteric glands or other organs.*	Remarks.
16.	Extensive ulceration of cœcum and throughout colon.	Distended.	.. ..	.. ..	Seaman—died in a fortnight.
17.	Ditto.	.. ..	Small abscess, size of a walnut, on lower surface of liver.	.. ..	Seaman—died in 3 weeks.
18.	Ditto, and cœcum perforated.	.. ..	.. ..	.. ..	Seaman, intemperate—died in 8 days.
19.	General ulceration of cœcum and colon.	.. ..	.. ..	.. ..	Sailor—ill nearly 6 weeks.
20.	Old disease of cœcum—ulceration of whole colon and rectum.	Healthy.	Healthy. ..	Healthy. ..	Old Seaman: only 14 days' illness.
21.	Whole large intestine, especially cœcum, in sloughing state.	.. ..	.. ..	.. ..	Cook—ill some weeks.
22.	Do. do., with adhesions to peritoneum.	.. ..	.. ..	.. ..	African Seaman—died in 18 days.
23.	Large intestine inflamed and ulcerated throughout. Cœcum perforated in various places, containing pus.	Healthy.	Liver much enlarged.	.. ..	Sailor, got dysentery in hospital—died in a fortnight.
24.	Sloughing of valve of cœcum. Colon thickened, ulcerated and cartilaginous.	Inflammatory blush on internal surface of stomach: small intestines little affected.	.. ..	.. ..	Seaman—had recovered from cholera—ill 10 days.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
25.	Ulceration of cœcum and transverse colon.	.. ..	Small abscess.	.. ..	Old Seaman—10 days in hospital.
26.	Cœcum and colon ulcerated throughout.	Small intestines here and there ulcerated.	Abscess, size of walnut, in right lobe.	.. ..	Seaman—27 days ill.
27.	Colon inflamed and ulcerated throughout: in some places perforated.	Healthy.	Liver rather pale.	.. ..	Had been treated for same complaint in previous month—died in 9 days: passed large quantity of blood.
28.	Whole intestine in right iliac region matted together. Cœcum ruptured.	.. ..	Liver much enlarged: 3 abscesses, one in right lobe containing 3ij of pus.	.. ..	African—ill 5 weeks.
29.	Cœcum thickened and sloughing: lower intestines ulcerated but less so.	Healthy.	Healthy. Gall-bladder distended.	Inflammation of peritoneum and tying down of omentum.	Seaman—died in 17 days.
30.	Intestines dark outside; internally covered with dark grumous blood: when wiped off, mucous membrane pale and bloodless. Cœcum thickened and ulcerated.	White and bloodless.	Liver enlarged and pale.	.. ..	Seaman—died in 28 days: stools bloody.
31.	Cœcum disorganized and ulceration of colon.	.. ..	.. ..	.. ..	Seaman—died in 20 days.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
32.	Large intestine ulcerated and sloughy throughout.	Small intestines in several places inflamed.	Liver somewhat large.	.. ..	Seaman—died in 22 days: had diarrhœa before.
33.	Ulceration of large intestines, especially towards rectum.	.. ..	Congested. ..	.. ..	Seaman—died in 10 days.
34.	Cœcum semi-cartilaginous, perforated and full of pus, colon dark and ulcerated throughout.	.. ..	.. ..	.. ..	Seaman—died in 9 days: lately had cholera.
35.	General ulceration throughout large intestine.	.. ..	.. ..	.. ..	Seaman, ætat 36—died in 10 days.
36.	Cœcum and colon thickened, ulcerated, sloughing.	.. ..	An immense abscess of liver.	.. ..	Young Seaman—died in 26 days.
37.	Large intestines thickened and ulcerated throughout.	Healthy.	Large and soft.	.. ..	Ætat 54—died in 19 days.
38.	Large intestine ulcerated throughout, chiefly in arch of colon.	.. ..	.. ..	Abscess of right kidney.	Recruit—died in 17 days.
39.	Great thickening of large intestine, with occasional spots of ecchymosis.	.. ..	Liver large, pale and hard.	Kidneys pale and soft: some effusion at neck of bladder.	Seaman, age 18: ill about a month: passed bloody urine.
40.	Ulceration and sloughing state of cœcum extending along the colon.	.. ..	.. ..	.. ..	Midshipman, ætat 14—died in 20 days.
41.	Cœcum and colon thickened and ulcerated throughout.	Blush on small intestines.	.. ..	Mesentery injected, mesenteric glands enlarged.	Seaman, ætat 23—died in 20 days.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
42.	Cœcum much ulcerated.	.. ..	.. ..	.. ..	Young European woman, much retching of coffee ground substance, and blood passed by stool.
43.	Large intestine, especially cœcum, a mass of ulceration. Ulcers varying from size of half crown to sixpence.	.. ..	Liver large: gall-bladder empty.	Spleen large.	Recruit—died in 13 days.
44.	Large intestine thickened, ulcerated, and indurated throughout.	Stomach little affected.	Liver large, gall-bladder distended, with tarry bile.	Glands enlarged: spleen much enlarged.	Drummer: ætat 15—died in 22 days.
45.	Large intestines ulcerated in parts throughout.	.. ..	Five small abscesses in liver.	.. ..	Young Seaman—died in 34 days.
46.	Cœcum and colon greatly ulcerated—arch and descending colon little so.	.. ..	.. ..	.. ..	Seaman, ætat 22—died in 29 days.
47.	Ulceration throughout the large intestines, and perforation.	.. ..	Gall-bladder adhering to large intestine.	.. ..	Seaman—died in 3 weeks.
48.	Large intestines thickened, ulcerated and covered with curdy matter.	Lower half of ileum livid externally, vascular internally.	Liver pale and enlarged.	.. ..	West Indian—died in 12 days.
49.	Cœcum and neighbouring parts one mass of corruption: perforated.	Healthy.	Healthy. ..	.. ..	Seaman—died in 3 months.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
50.	Cœcum and the rest of large intestine one mass of disease.	.. ..	Healthy. ..	.. ..	Young man: ill 15 days.
51.	Large intestines immensely thickened, ulcerated and perforated.	Healthy.	Healthy. ..	.. ..	Recruit—died in 13 days.
52.	Cœcum and colon ulcerated throughout, and a pint of muddy fluid in abdomen from perforation of cœcum.	.. ..	Liver very large, pushing up the diaphragm.	Glands hard and matted together.	Ætat 34—ill nearly two months.
53.	Large intestine ulcerated throughout.	.. ..	.. ..	.. ..	Seaman—died in a fortnight.
54.	Large intestines ulcerated with fungous excrescences and lined with grumous matter throughout.	Small intestines discolored and vascular.	.. ..	Mesentery discolored and vascular.	Female—died in 8 days: had phrenitis and vomiting: stools tarry.
55.	Scirrhous thickening of transverse and descending colon and rectum. Calibre of rectum would scarcely admit a finger: whole surface studded with fungous growths.	.. ..	.. ..	.. ..	Old Soldier—died in 21 days.
56.	Large intestines ulcerated throughout in patches of irregular shape: contained bloody fluid and mucus.	Healthy.	Liver pale. Gall-bladder distended.	.. ..	Recruit: ill about 40 days.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
57.	The whole large intestines ulcerated and sloughing, contracted in parts.	.. ..	Liver natural in size, with cicatrices on surface : gall-bladder with healthy bile.	Glands enlarged.	Young stout man—died in 2 months.
58.	Whole large intestines ulcerated and sloughing : ileo-colic valve ulcerated.	.. ..	Liver healthy. Gall-bladder full.	.. ..	Ill 15 days.
59.	Large intestines one mass of disease.	.. ..	Liver large, reddish flush on convex surface : gall bladder not distended.	.. ..	Ætat 26—died in 10 days.
60.	Whole disease of intestines slight, except at cœcum.	.. ..	Liver large, soft. Gall-bladder distended : duct pervious.	Mesentery injected.	Ætat38—died in 12 days.
61.	Large intestines greatly altered, in some places very thin : chief seat of disease sigmoid flexure. Folds of transverse colon ulcerated.	Small intestines healthy.	A large and a small abscess of left lobe : small abscess of right lobe.	Meso-colon injected.	Pauper, ætat 30—died in 21 days.
62.	Cœcum and large intestines ulcerated throughout.	.. ..	.. ..	.. ..	Ætat14—died in 36 days.
63.	Large intestines one mass of disease.	.. ..	.. ..	.. ..	Ætat20—died in 4 weeks.
64.	Do. cœcum perforated.	.. ..	.. ..	.. ..	Ætat 20,—ill 1 month.
65.	Large intestines diseased throughout, especially cœcum.	Small intestines generally healthy.	Liver, soft : a large and a small abscess in left lobe : gall-bladder half full.	Glands enlarged.	Seaman, ætat 32—died in a month.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
66.	Effusion of coagulable lymph on upper and outer surface : ulceration of ileo-colic valve. Large intestines diseased throughout : appearance here and there of a deposit of degenerated tubercular matter in the submucous tissue.	Omentum tied down to small intestines.	Liver healthy : effusion of coagulable lymph on lower surface.	.. ..	Old Seaman—died in 21 days.
67.	Generally ulcerated, especially the rectum.	.. ..	Liver large with marks of old disease.	Glands enlarged.	Seaman, ætat 35—died in a month.
68.	Large intestines ulcerated throughout.	.. ..	.. ..	.. ..	Seaman, ætat 27—ill a fortnight.
69.	Ditto.	.. ..	.. ..	.. ..	Seaman, ætat 25—ill 1 month.
70.	Ditto. Sigmoid flexure perforated.	.. ..	Liver healthy.	Glands enlarged.	Chief officer—died in 3 weeks.
71.	Highly diseased state : bowels breaking down under the fingers.	.. ..	.. ..	.. ..	Seaman—died in 6 days : motions nearly pure blood.
72.	Generally ulcerated.	Small intestines healthy.	Liver large, hard and mottled.	Glands very large and hard.	Boy—died in 5 weeks : tubercles in lungs. Was a hard liver.
73.	Villous coat of large intestine red, and getting of deeper colour downwards : small ulcers at rectum of irregularly circular form, and rectum greatly thickened. Disease almost confined to rectum.	Healthy.	Healthy. ..	Healthy. ..	Seaman, ætat 33—died in 20 days.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
74.	Thickened, indurated and sloughing.	.. ..	Pale and soft, slightly granular.	.. ..	Pensioner, æt. 54—died in 18 days.
75.	Diseased throughout, with much ulceration, but chiefly at rectum, where much thickened: ulceration least in cœcum: villous coat destroyed and surface rough.	1½ foot of ileum had villous coat injected.	Liver healthy, adhesion to colon. Gall-bladder small, lined outside with rough false membrane.	.. ..	Ill 14 days.
76.	Thickened and ulcerated, also covered with sloughs and coagula: latter chiefly in cœcum.	Pale and bloodless.	Liver pale and enlarged. Gall-bladder inordinately full of dark bile.	.. ..	Seaman, ætat 37—died in 4 or 5 days: passed much blood.
77.	Irregularly shaped ulcers throughout, especially at the sigmoid flexure: intestine much thickened, contained a great quantity of bloody fluid, and at the cœcum was friable.	Healthy.	Healthy. ..	.. ..	Seaman, in hospital 3 days; ill 3 weeks on board ship. Immense hemorrhage.
78.	Ulceration of rectum and also of cœcum, rest comparatively free from it: colon of dull purple colour.	For a few inches dull vascularity.	Liver somewhat larger than usual: five abscesses, one had about 3 ozs. of curdy pus.	.. ..	Seaman, ill 3 weeks, in hospital 2 days: immense stools of blood.
79.	Ulcers occupying nearly whole calibre of intestine: worst at cœcum, right and left turns of intestine and sigmoid flexure, entirely destroying the villous coat, and in some places, the muscular, and leaving only the peritoneal, which gave way on being handled.	Healthy.	Healthy. ..	Omentum of dull pink colour, and adhering to cœcum.	Stout man, ill a fortnight, 8 days in hospital. Bloody motions.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
80.	Large intestines ulcerated and thickened throughout, chiefly in transverse colon.	.. ..	Small abscess in left lobe.	Spleen enlarged.	Seaman, ætat 25—died in 10 days.
81.	Ulcerated throughout.	.. ..	Liver large, soft and pale.	Omentum inflamed.	Seaman, ætat 18—died in 20 days. Bloody stools.
82.	Suppurating ulcers chiefly at arch of colon.	.. ..	Liver pale and granular.	.. ..	Seaman, ætat 16—died in 3 weeks.
83.	Cœcum and descending colon thickened and ulcerated: rectum in state of slough.	Slight adhesions between the convolutions of small intestine.	Liver pale. ..	Omentum vascular.	Seaman—died in a fortnight.
84.	Large intestine gangrenous and ulcerated throughout: whole one sloughy mass: every where covered with ash coloured sloughs.	Healthy.	.. ..	.. ..	Attack succeeded jungle fever—died in 5 weeks.
85.	Cœcum and colon, as far as sigmoid, thickened and cartilaginous—internal surface one continuous sloughing ulcer.	.. ..	Liver very large and mottled: rather pale.	.. ..	8 days in hospital—ill some weeks.
86.	Colon dilated to a diameter of 4 or 5 inches: cœcum a mass of ulceration and thickening, but no ulceration of rectum.	.. ..	Liver small, pale, yellow, dry, and granular.	.. ..	Old Seaman, ill 10 days: 3 in hospital.
87.	Cœcum sloughing, intestines, ulcerated and cartilaginous throughout.	.. ..	Healthy. ..	.. ..	Seaman, ætat 29—died in 5 or 6 days.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
88.	Large intestines in state of slough throughout.	.. ..	Surface of liver shewed recent cicatrices: extensive abscess of right lobe.	.. ..	Seaman, ætat 26—ill a month.
89.	Large intestines lined with ulcers, some of them apparently healing: coats perforated in several places.	.. ..	Liver hard and granular, with some tubercles. Gall-duct obstructed and gall-bladder full.	Lungs tubercular: some effusion into chest.	Old man,—ill for one month before death.
90.	Intestines at most depending point in state of slough: rectum thickened and ulcerated.	.. ..	Liver small and turgid.	Spleen slightly enlarged.	Ætat 67—died in 12 days: had immense hernia.
91.	Large intestines thickened, ulcerated and lined with sloughs throughout.	.. ..	Liver small and softened: small abscess.	.. ..	Seaman, ætat 30—died in 17 days.
92.	Large intestines lined with fungous growth, thickened, especially near rectum.	.. ..	Liver pale, granular and soft.	.. ..	Soldier, ill about 10 days.
93.	Colon, purple spots outside—inside mass of ulceration and thickening with lichenoid granulations.	Healthy.	Healthy. ..	.. ..	Seaman—died in 8 days, but had just been discharged from hospital cured of dysentery.
94.	Mass of ulceration and thickening of cœcum, gave way on being handled—in sigmoid flexure and rectum less disease.	Healthy.	Liver one small abscess, size of nutmeg, otherwise healthy.	.. ..	Ill 20 days—ætat 54.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
95.	Universal ulceration of large intestine, which broke down under fingers.	Healthy.	Liver enlarged: nutmeg.	.. ..	Old pensioner: ætat 46, ill for 32 days.
96.	Cœcum, ascending and transverse colon thickened, cartilaginous and ulcerated.	.. ..	Liver somewhat soft.	.. ..	Townsman, ætat 33, ill 3 weeks.
97.	Cœcum and colon thickened: full of suppurating ulcers.	Intestines floating in yellow serum.	Liver granular and dark: considerable abscess in left lobe.	.. ..	Pauper—ill 12 days.
98.	Ditto..	.. ..	Liver large and soft: left lobe pale.	.. ..	Seaman, ætat 33, ill for 18 days.
99.	Cœcum and colon tough, and ulcerated throughout, rectum on the point of sloughing en masse.	.. ..	Liver hard, granular, and almost white.	.. ..	Pensioner, ætat 50—died in a fortnight.
100.	Large intestine mass of phagedœnic ulcers.	.. ..	Liver large and pale.	.. ..	Townsman, ætat 24—ill say 3 weeks.
101.	Large intestine ulcerated throughout: colon almost in state of decomposition.	.. ..	Healthy. ..	.. ..	Boatswain, ætat 45—ill 15 days.
102.	Ditto ditto, lower intestines less affected: no coagula in bowels.	.. ..	Healthy. ..	.. ..	Seaman, ætat 31—died in 15 days. Immense quantity of blood in stools.
103.	Ditto ditto, ulcerated throughout.	.. ..	Liver pale and small.	Spleen large.	Stout: ætat 42—ill 17 days.
104.	Cœcum and ascending colon a mass of sloughing ulceration.	.. ..	Ditto.. ..	.. ..	Ætat 31—ill 12 days.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
105.	Colon and whole of the large intestine full of cicatrised ulcers: very few in a state of active ulceration.	Small intestines in many places injected and red: slight abrasion of mucous surface.	Healthy. ..	Spleen and pancreas healthy.	Seaman, ætat 17—died in 31 days.
106.	A good deal of detached ulceration along the colon, but no great extent of it: coats of intestines not thickened.	Healthy.	Mottled. ..	.. ..	Seaman, ætat 42, ill 3 weeks—stools chiefly of bloody washings.
107.	Cœcum much thickened and ulcerated, with imperfect granulations: colon, few superficial ulcers.	Small intestines and stomach blanched: patches of red on inner side of stomach.	Liver a little enlarged and full of abscesses, from size of pea to that of nutmeg, especially on upper surface of right lobe.	.. ..	Recruit, ætat 23, ill at Dum-Dum—died 5 days after admission.
108.	Large intestines enormously ulcerated throughout and filled with coagula.	.. ..	Liver dark, mottled and granular.	.. ..	Invalid Artillery man—ætat 21, ill for 11 days.
109.	Extensive and deep ulcers throughout cœcum and colon—transverse and descending colon somewhat contracted.	Healthy.	Healthy. ..	Many glands enlarged.	Soldier, ætat 39: ill six weeks—stools bloody.
110.	Large intestines thickened and ulcerated throughout—sloughy.	Several patches of inflammation in small intestines.	Liver large and scirrhous, and adhering to diaphragm.	Spleen and kidney healthy.	Ætat 27, ill six weeks.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
111.	No perceptible lesion.	Intestine near the cœcum a mere web, and destitute of mucous membrane.	Liver, contained an immense abscess.	.. ..	Ætat 18, ill 45 days: passed 12 or 14 inches of thickened mucous membrane 3 days before death.
112.	Arch of colon a mass of ulceration.	.. ..	.. ..	.. ..	Seaman, ætat 24—ill 15 days: stools dark putrid blood.
113.	Patches of inflammation in colon and incipient ulceration.	Healthy.	Healthy. ..	2 pints serum in abdominal cavity.	Pensioner, ætat 40—died in 12 days: pulse hard.
114.	Large intestines much attenuated: cœcum and ascending colon exhibited large black patches ulcerating.	.. ..	Small, soft, flabby and pale.	Mesentery very vascular: spleen healthy.	H. C. M. ætat 19—57 days ill: recovered but got relapse after fever chronic.
115.	Superficial ulceration throughout whole extent, not much thickening, ulcers like abrasions.	Healthy.	Healthy. ..	.. ..	Ætat 28—struck by lightning: ill a fortnight: doing well: got attack of cholera—recovered. Dysentery returned—died in 40 days.
116.	Externally congested: internally of reddish hue throughout, thickly studded with small red spots: no ulceration or thickening but sort of superficial abrasion.	Small intestines vascular, inner surface in many places congested.	Healthy. ..	.. ..	Midshipman, ætat 17—died in 7 days.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
117.	Mass of corruption breaking down under the fingers.	.. ..	.. ..	.. ..	Attacked in hospital—died in 7 days—ætat 45.
118.	Large intestines a good deal thickened and ulcerated, especially inferiorly, but to no very great extent.	Healthy.	Liver small. ..	.. ..	Seaman, ætat 30—died in 35 days.
119.	Intestine thickened and ulcerated throughout, lined with mucous and bloody coagula.	.. ..	Liver rather large and pale.	.. ..	Seaman, ætat 18—died in 34 days.
120.	Rectum and sigmoid flexure in state of irritable ulceration.	.. ..	.. ..	Some fluid effused in pelvis.	Soldier, ætat 21—died in 5 days, complicated with scorbutus and fever.
121.	Cœcum and sigmoid flexure thickened, ulcerated and breaking under fingers.	Small intestines slightly glued together, lower portion filled with dark brown fluid.	Liver rather large and soft.	Spleen large.	Soldier, ætat 25—died in 17 days: stools pure blood, mixed with mucus.
122.	Upper part of colon and cœcum in state of sphacelus.	Lower part of ileum in state of sphacelus.	Liver enlarged, granular, grey degeneration.	.. ..	Pensioner, ætat 44—ill 40 days.
123.	Cœcum distended with clots of blood, much effused blood along the whole course of colon.	Rather blanched but quite healthy internally.	Nutmeg liver.	.. ..	French Sailor—ætat 24: 11 days in hospital.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
124.	Ulcerated and thickenedthroughout caput coli: cœcum studded with large unorganized projections from ulcerated base: ulcers in every stage: whole rectum raw and abraded. Appendix vermiformis very long, full of sanious fluid.	.. ..	.. ..	.. ..	Seaman, ætat 24—died in 3 weeks.
125.	Large sloughy ulcers in cœcum, less in transverse arch, but bowel in sloughy state throughout.	Small intestines inflamed and partially agglutinated together.	Liver granular, more like lung than liver. Gall-bladder small, with inspissated bile.	Mesentery much inflamed.	Seaman, ætat 24...died in 14 days: 2 days in hospital.
126.	Cœcum and transverse colon ulcerated and sloughy.	Ileum inflamed.	Liver gorged.	Mesentery and meso-colon inflamed: right iliac fossa a bath of blood and inflammation.	Seaman, ætat 27—died in 11 days.
127.	Large intestines mass of ulceration and sloughing.	Stomach healthy: small intestines in places discoloured.	Liver nutmeg.	Mesentery healthy: spleen healthy.	Ætat 24—died in 20 days.
128.	Externally dark, internally purple, with more or less abrasion, scarcely amounting to ulceration: transverse colon and rectum much affected: no clots of blood.	Small intestines dark and congested: some superficial abrasion.	.. ..	Veins of mesentery turgid, with dark blood.	Seaman, ætat 19—died in 18 days: motions bloody.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
129.	Colon much thickened and ulcerated throughout.	Mucous coat of stomach red and abraded.	Healthy. ..	Healthy. ..	Seaman, ætat 41—ill 8 days—in hospital 6 hours.
130.	Large intestines purple, with ulcerations throughout, breaking down under the fingers.	Healthy.	Healthy. ..	Healthy. ..	Ætat 31—died in 11 days.
131.	Extensive ulceration of large intestine, especially cœcum.	Red blush externally, but internal coat healthy.	Healthy. ..	.. ..	Seaman, ætat 22—died in 26 days.
132.	Thickened and ulcerated throughout with black spots as from hæmorrhage.	Healthy.	Healthy. ..	.. ..	Ætat 38—died in 3 weeks: passed much blood.
133.	Large intestines ulcerated throughout, towards rectum frequent bloody patches, and the last part of the gut sloughing.	Healthy.	Healthy. ..	.. ..	Soldier, ætat 24—died in 3 weeks: rectum especially affected.
134.	Externally dark purple, ulcerated throughout.	Small intestines dark outside: mucous membrane dark red, inclined to ulcerate.	Healthy. ..	.. ..	Seaman, ætat 22—died in 16 days: small intestines more than usually affected.
135.	Cœcum and rectum ulcerated, former lined with coagula.	.. ..	.. ..	Spleen large.	Ætat 26—died in a month.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
136.	Cœcum distended.	.. ..	.. ..	.. ..	Old man—died in three weeks.
137.	Large intestines in sloughy state.	.. ..	Granular. ..	.. ..	Ill about a month, ætat 32.
138.	Cœcum and colon lined with slough.	.. ..	Small, yellow and granular.	.. ..	Pensioner, ætat 44, ill 18 days.
139.	In cœcum much ulceration, less in ascending colon: transverse free from ulcers, a few in sigmoid: ulcers looked indolent: villous coat puckered round them as if going to cicatrise.	Nothing abnormal.	Liver enlarged, adhering throughout to diaphragm: structure loose and friable: abscess containing 4 ℥ of sanious pus. Gall-bladder full of bile.	.. ..	Ætat 36: dysentery came on 5 weeks before, after fever.
140.	Large intestine one continued ulcer.	.. ..	Liver large, pale and dry.	.. ..	Ætat 26—died in 7 weeks.
141.	Large intestines ditto.	Small intestines in parts of dark purple colour.	Rather large but healthy.	.. ..	Æt. 27, about 40 days. Odd nervous symptoms.
142.	Cœcum thickened, cartilaginous and studded with small perforating ulcers.	6 inches of small intestines thickened, &c.	Pale, yellow, large, hard and granular.	.. ..	Invalid: ill 16 days.
143.	Ulceration throughout large intestines.	.. ..	Liver pale, yellow and granular.	.. ..	Died in three weeks.
144.	Large intestine ulcerated, but to no great extent.	Healthy.	Slightly hardened: right lobe had 2 small abscesses: one healthy, one of sanious pus.	.. ..	Pensioner, ætat 41—died in a month.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
145.	Ulceration of large intestines throughout.	Healthy	Healthy. ..	.. ..	Engineer, ætat 28—ill a month.
146.	Ditto.	Ditto. ..	Ditto. ..	.. ..	Pauper, ætat 28, ditto.
147.	Large intestines thickened and ulcerated throughout.	Healthy.	Healthy. ..	.. ..	Seaman, ætat 26—died in 11 days: stools like coffee grounds.
148.	Considerable ulceration of large intestine: mucous membrane in places destroyed.	Ditto. ..	Ditto. ..	.. ..	Ætat 24, ill about 14 days.
149.	Thickening and ulceration of large intestine, and especially rectum, which broke down on handling.	Ditto. ..	Ditto. ..	Vascularity and enlargment of glands.	Midshipman, ætat 16, ill 12 days.
150.	Ulceration throughout whole extent: whole surface rough and covered with whitish-yellow lichenoid substance, which could scarcely be detached.	.. ..	Ditto. ..	.. ..	Pauper, ætat 43, recovered from jungle fever: ill a month.
151.	Extensive ulceration: coats thickened and friable.	.. ..	.. ..	.. ..	Soldier, ætat 26—died in 3 weeks.
152.	Thickened throughout with numerous small ulcers.	Inner coat of stomach injected.	.. ..	.. ..	Soldier, ætat 20—died in 10 days.

No.	State of large Intestines.	State of small Intestines.	State of Liver and Gall-bladder.	State of Mesentery, Mesenteric glands or other organs.	Remarks.
153.	General oozing of blood from mucous surface throughout its whole extent: collections of blood in rectum and cœcum, latter had a few deep ulcers, no general ulceration.	Healthy.	Healthy. ..	.. ..	Ætat 25—died in 16 days from active hæmorrhage: bled from nose and lungs.
154.	Ulcerated throughout: cœcum in state of sphacelus.	Ditto. ..	Ditto. ..	.. ..	Soldier, ætat 28, ill 20 days.
155.	Generally ulcerated.	.. ..	Pale. ..	.. ..	Woman, ætat 38—died in 5 weeks.
156.	Ulcerations chiefly of cœcum and rectum: ulcers distinct, separate and large, not much thickening.	.. ..	Healthy: one or two yellow discoloration on upper surface.	.. ..	Seaman, ætat 22—died in 15 days.
157.	Much ulcerated and thickened throughout.	.. ..	Liver yellow.	.. ..	Midshipman, ætat 18, ill 3 weeks.
158.	One mass of thickening and ulceration throughout.	Rather pale.	.. ..	.. ..	Ætat 15, ill 30 to 40 days.
159.	Large intestines one mass of ulceration.	Small intestines injected, especially cœcum.	Liver with many cicatrices.	.. ..	Seaman, ætat 40—died in 17 days.
160.	One mass of disease: mucous membrane swollen and gangrenous, with black patches of slough.	Healthy.	Healthy. ..	.. ..	Soldier, ætat 22, ill 18 days.

SUMMARY OF TABLE OF ACUTE DYSENTERY.

Thus in 160 cases of Acute Dysentery—

The liver is found to be altered in	84
to contain abscess in	21
is enlarged in	40
is gorged or turgid in	4
is small in	7
is pale in	26
is granular or nutmeg in ..	22
is soft in	12
is hard in	5
contains cicatrices* in.. ..	3

The gall-bladder appears to be almost always full, and to contain healthy or somewhat inspissated bile.

The ileum is noted as over-vascular or congested in	21
Slight ulceration and abrasion are seen in	3
It is in a state of sphacelus in	1
The stomach has its mucous coat over-vascular or somewhat softened in	4
and ulcerated in	1

The large intestine is ulcerated in all, and chiefly at the cœcum, sigmoid flexure, and rectum.

The cœcum, transverse and descending colon were free from ulceration in	3
It was perforated (generally in the cœcum) in ..	8
There was ulcerative destruction of ileo-colic valve in	3
Suppuration of appendix vermiformis (though not uncommon) is only recorded in	1
There is thickening and stricture of the intestine in	4
and dilatation in	1
The mesenteric glands are enlarged or inflamed in..	17
The spleen is enlarged in	6
The kidneys are diseased in	2

* These cicatrices do not appear to be the sequelæ of abscesses.

CHRONIC DYSENTERY RUNNING INTO DIARRHŒA.

No.	*Large Intestines.*	*Stomach and small Intestines.*	*Liver and Gall-bladder.*		Remarks.
1.	Extensive ulceration of sigmoid flexure.	.. ..	Large abscess of liver.	.. ..	Ill 2½ months.
2.	Colon in a sloughy state: coats gave way on handling	.. ..	.. ..	.. ..	Long suffered from dysentery.
3.	Lining of colon ulcerated throughout, greatest lesion at sigmod flexure and rectum, where ulcers were almost continuous.	.. ..	Liver enlarged, weighing 6½ lbs. attached to spleen by false membrane, substance soft: colour pale. Gall-bladder distended with green bile.	Spleen enlarged, indurated: mesenteric glands enlarged.	Invalid—died in 10 days of acute dysentery: supervening on chronic.
4.	Extensive ulceration of colon, orifice in sigmoid flexure, through which contents of bowels extravasated.	Slight attempt at inflammation of peritoneal surface.	.. ..	.. ..	Woman of 55—ill for six months.
5.	Intestines attenuated, several ulcers in cœcum and along arch of colon to sigmoid flexure. Ulcers in an atonic state: no raised edges, no increased vascularity.	.. ..	Liver small and unhealthy.	Congestion of mesentery: some enlargement of mesenteric glands.	Soldier, ill for 7 weeks out of and 115 days in hospital.
6.	Large intestines ulceratedthroughout; transverse colon thinnerthan natural: mucous membrane, had honeycomb appearance.	Stomach distended with air.	Liver dark, granular, with some congestion. Peritoneal coat puckered in various places: gall-bladder distended: ducts pervious.	.. ..	Soldier, admitted bedridden—died in 18 days.

No.	*Large Intestines.*	*Stomach and small Intestines.*	*Liver and Gall-bladder.*		Remarks.
7.	Large intestines thickened and ulcerated, lined with pus.	Healthy.	3 abscesses in right lobe full of curdy matter.	.. ..	Pauper, ill several months—died in six days.
8.	Contraction of colon, general superficial ulceration of bowel: deeper of rectum.	Mucous surface of stomach abraded towards pylorus and an inch down the duodenum.	.. ..	Mesenteric glands enlarged.	Invalided for chronic dysentery—died after two months' treatment.
9.	Large intestine thickened and ulcerated throughout, cœcum nearly obliterated.	.. ..	Cicatrized fissure on upper surface, from 4 to 5 inches in length 4½ an inch deep. A large hydatid firmly attached to left lobe.	.. ..	Old Soldier, from Cabul.
10.	Large intestines contracted in several parts, ulcerated in patches of circular and irregular form from cœcum to rectum, which was in a state of slough.	.. ..	Liver healthy.	Abscess in cellular tissue between cœcum and psoas, which was dissected by it.	Portugese Doctor, ill 8 months.
11.	Large intestines ulcerated throughout.	.. ..	Pale and enlarged.	.. ..	Invalided for bowel complaint.
12.	Scirrhous thickening and ulceration throughout, but chiefly of rectum.	.. ..	Liver pale and granular.	.. ..	Pauper—ill 6 or 8 months.

No.	*Large Intestines.*	*Stomach and small Intestines.*	*Liver and Gall-bladder.*		Remarks.
13.	Whole cœcum and descending colon in sloughy state, transverse arch not much affected: rectum cartilaginous.	.. ..	Liver healthy. Gall-bladder empty.	Glands enlarged.	Pensioner, a long resident in India, had been in China: ill many months.
14.	Large intestine in state of slough throughout.	.. ..	.. ..	.. ..	Mate of Floating Light, æt. 24, ill 1 year.
15.	Cœcum rotten and perforated, transverse colon ulcerated, sigmoid flexure healthy.	.. ..	Abscess of liver communicating with ascending colon.	.. ..	Invalid, had been several weeks in hospital; discharged, but returned with fresh attack.
16.	Large intestines ulcerated and sloughing throughout.	.. ..	.. ..	Spleen greatly enlarged.	Young man, ill about 9 months.
17.	Cœcum thickened with villous coat, destroyed and like tanned leather: colon and rectum both much diseased.	.. ..	Gall-bladder gorged.	Effusion in abdomen.	Ætat 26, ill one year.
18.	Large intestine contracted, thickened and cartilaginous with fungous granulations.	.. ..	Liver pale and soft.	.. ..	Recruit, ill for one year.
19.	Colon adhering to stomach.	Small intestines glued to abdominal parietes & torn into shreds on separation.	Pale and small.	.. ..	Pensioner, ætat 40, ill 4½ months.
20.	Large intestines one mass of ulceration.	Healthy.	Pale and gritty.	.. ..	Ætat 62, ill some months—female.

No.	Large Intestines.	Stomach and small Intestines.	Liver and Gall-bladder.		Remarks.
21.	Large intestines lined with fungous granulations, and calibre of gut in places contracted to size of finger.	Much attenuated.	Yellow, hard and granular.		Seaman, ætat 40, ill three months.
22.	Hæmorrhage external to bowels, which were purple internally, with little ulceration.	.. ..	Liver cirrhosed.	Mesentery and meso-colon full of effused blood: spleen greatly enlarged.	Ætat 44, broken down—drunkard, died suddenly on close stool.
23.	Large intestine ulcerated throughout.	Healthy.	Pale. ..	.. ..	Soldier, ætat 25. Ill many months.
24.	Large intestines slightly ulcerated, some thickening of cœcum.	Healthy.	Liver double its natural size, like a coagulum of blood: nine small abscesses containing 3 or 4 ozs. of pus.	.. ..	Invalid, ætat 31—many months ill.
25.	Large intestines with patches of rather superficial ulceration, and of dark red discolouration.	Healthy.	Healthy. ..	.. ..	Ætat 27: 75 days ill—complicated with secondary syphilis.
26.	Colon not much thickened, extensive patches of ulceration and spurious granulations.	Pale. ..	Pale. ..	.. ..	Seaman, ætat 27—ill 5 months.
27.	Large intestine ulcerated at cœcum, effusion of grumous matter on its surface, and dotted with spots of ulceration throughout.	.. ..	Healthy. ..	.. ..	Ætat 34, ill 5 months.

No.	Large Intestines.	Stomach and small Intestines.	Liver and Gall-bladder.		Remarks.
28.	Internal surface of colon ulcerated and abraded throughout: not much thickening of coats.	Healthy.	Healthy. ..	.. ..	Ætat 33—ill about 3 months.
29.	Large intestines purple outside, inside quite gangrenous, with those lichenoid excrescences or abortive cicatrization?	Healthy.	Healthy. ..	.. ..	Soldier, ætat 34, ill many months.
30.	Coats of large intestine extensively ulcerated, especially cœcum and rectum.	.. ..	.. ..	Mesentery vascular, glands enlarged.	French Sailor, ill 3 months.
31.	Extensive ulceration, coats black, and in state of sphacelus.	.. ..	.. ..	.. ..	Ætat 26, ill 2 months.
32.	Much thickened, inner coat livid and ulcerated.	Coats of stomach vascular, and abrasion of coat of small intestines.	.. ..	Mesentery injected, glands swollen, with some hydatids in them.	Pensioner, ætat 46, ill 8 months.
33.	Purple inside, with superficial ulceration throughout.	.. ..	Immensely enlarged, granular, with white patches on upper surface: cicatrices.	.. ..	Soldier, ætat 23, ill some months on voyage out.
34.	Thickened, with a few ulcers—and general vascularity of mucous coat.	.. ..	Healthy. ..	.. ..	Ætat 31, ill 3 months.
35.	Coats thin and friable, extensive ulceration throughout.	.. ..	Enlarged, large abscess on lower surface of right lobe.	Glands swollen.	Ætat 22, ill 7 weeks.

No.	*Large Intestines.*	*Stomach and small Intestines.*	*Liver and Gall-bladder.*		Remarks.
36.	General ulceration and thickening.	Coats of stomach vascular.	Liver full of abscesses, large one in left lobe. Gall-bladder unusually large.	Mesentery vascular, glands enlarged.	Ætat 30, ill 3 months.
37.	General ulceration and thickening, especially of cœcum and rectum.	.. ..	.. ..	.. ..	Ætat 28, ill about 3 months.
38.	Large intestines lined with bloody tenacious matter.	.. ..	Liver small and hard.	Pancreas small and hard, omentum and mesentery discoloured: latter nearly black: glands were rather enlarged.	Ætat 63, ill 2 or 3 months: passed 2 or 3 pints of blood every day.
39.	A good deal of ulceration of large intestine.	Stomach healthy, ulcer at the pyloric orfice, where there was cancerous ulceration.	Healthy. ..	.. ..	Sailor, æt. 40, says he had been ill 3 months—died in 16 days.
40.	Patches of purple discolouration on inner coat.	Pale. ..	Early stage of nutmeg degeneration.	.. ..	Seaman, ætat 40, ill some months—died with odd head symptoms.
41.	Attenuated, not ulcerated.	Mucous membrane of stomach softened throughout: towards pylorus abraded and rough.	Pale. ..	.. ..	Has secondary syphilis.

No.	*Large Intestines.*	*Stomach and small intestines.*	*Liver and Gall-bladder.*		Remarks.
42.	Internal surface of cœcum covered with a red efflorescence, some spots of ulceration in the colon. Descending colon and rectum contracted, thickened and cartilaginous.	Intestines generally glued together.	Liver natural, contents of gall-bladder pale.	Glands enlarged and livid.	Invalid, ill long time.
43.	Towards sigmoid flexure a few patches of ulceration.	Congested ecchymosed state of pyloric orifice, coat partially abraded—this extending to duodenum—congestion of ileum; bowels on whole softened, œdematous, and tearing readily.	Small particles like tubercular matter, on surface of liver.	Glands enlarged.	Ætat 36—ill some five weeks.
44.	Rectum in scirrhous state, with ulceration extending up to sigmoid; patches of ulceration in transverse colon and ileo-colic valve.	Ulceration extending some inches up the ileum.	Liver hard and nutmeg.	Glands enlarged and diseased.	Old Seaman, 3 months ill.
45.	Spots of ecchymosis on descending colon.	Small intestine thickened, in lower portion some ecchymosis.	Liver small: flaccid gall-bladder.	Mesenteric glands enlarged and injected.	Diarrhœa after cholera—died in five weeks.

No.	*Large Intestines.*	*Stomach and small Intestines.*	*Liver and Gall-bladder.*		Remarks.
46.	Large intestine pale externally, slight ulceration inside.	Pale. ..	Liver rather pale.	Mesenteric glands enlarged.	Invalid, ill many months.
47	Throughout thickened and cartilaginous, at the arch and sigmoid flexure surface irritable.	Stomach distended with bilious fluid: small intestine doughy.	Liver small and pale. Gall-bladder full of dark bile.	Mesenteric glands enlarged, spleen large.	Invalid: long ill.
48.	Large intestine pale, and so much contracted as scarcely to admit of the introduction of the blades of a pair of scissors: destitute of lubricating mucus, with red efflorescences towards rectum.	Small intestines extremely attenuated and transparent.	Dark. ..	.. ..	Soldier, from China. Ætat 20—ill four and a half months.
49.	Considerable chronic inflammation of mucous coat of large intestine. Coats a good deal thickened: no deep ulceration.	Chronic inflammation of mucous coat of stomach and small intestines.	Healthy. Gall-bladder full.	Mesenteric glands atrophied: spleen and pancreas small.	Ætat 30—about six months ill.
50.	Some ulceration of large intestines.	Healthy.	Small dark surface, studded with spots of organized lymph.	.. ..	Ætat 36—2 years' standing. Complicated with lungs.
51.	Colon and rectum contracted, dry and cartilaginous, external surface superficially ulcerated.	.. ..	.. ..	.. ..	Soldier, ætat 43—ill 7 months.

No.	*Large Intestines.*	*Stomach and small Intestines.*	*Liver and Gall-bladder.*		Remarks.
52.	Large intestines pale and blanched, otherwise healthy.	Coats of stomach bloodless: small intestines pale.	Liver yellow, mottled or nutmeg.	.. ..	Invalid, ætat 53, had been drinking—much bilious vomiting: ill 3 or 4 months.
53.	Large intestines congested and abraded throughout—no deep ulceration.	Small intestines congested: and mucous surface abraded partially.	Healthy. ..	.. ..	Seaman, ætat 18—ill 3 months.
54.	Pale and bloodless.	Pale and bloodless.	Light yellow colour, slightly enlarged.	.. ..	Half starved Seaman—ætat 43, ill 2 months.
55.	Pretty general ulceration of superficial kind.	.. ..	Small and contracted from former inflammation.	.. ..	East Indian, ætat 59, ill many months.

SUMMARY OF TABLE OF CHRONIC DYSENTERY.

In 55 cases of chronic dysentery—

the liver was altered in ..	31
abscess was found in ..	6
hydatids in	1
cirrhosis in	1
enlargement in	5
diminution of bulk in ..	8
nutmeg alteration in ..	6
it was pale in	11
it was hard in	4
soft in	1
contained cicatrices? in	1

The gall-bladder and its contents seem generally to have been healthy: the bile occasionally rather pale coloured, but at other times the reverse.

The large intestines were ulcerated in	50
the colon contracted in	3
cœcum nearly closed in	1
colon perforated in	1
The stomach is noted as unhealthy in	6
There was chronic inflammation and softening in ..	2
increased vascularity in	2
abrasion of pylorus in	3
cancer of pylorus in	1
The small intestines are noted as unhealthy in ..	12
Ulceration or abrasion of ileum in	3
Mesenteric glands enlarged in	16
The spleen was enlarged in	4

As no distinction can be drawn between acute and chronic dysentery, which is not, to a great degree, arbitrary, so chronic dysentery and diarrhœa are divided by no marked line. The last 15 cases belong rather to diarrhœa than dysentery, and it will be observed, that in them the large intestine is paler than natural, and in several instances not ulcerated, and that in no instance was there abscess, though the liver was found altered in 11 out of 15 cases.

CHUSAN DYSENTERY.

Although a great many patients died in the General Hospital from Chusan dysentery, these cases are excluded from the preceding tables,—1st, because they are instances of a form of malarious dysentery, which is very different from the common disease of Bengal,—And 2nd, because ulceration of the large intestine, of a true dysenteric character, though sometimes met with in China, seems chiefly to have supervened after the patients arrived in Bengal. But the mortality from it was so great, and it was altogether so unmanageable a form of disease, that the accompanying abstract of the pathological appearances in those who died soon after their arrival in Bengal is worthy of being preserved.

Large Intestine.	*Stomach and small Intestine.*	*Liver and Gall-bladder.*	*Mesentery, &c.*
Generally attenuated, now and then inflammatory blush: in further advanced stage some ulceration.	Attenuated: rosy hue of pyloric orifice, some roughness at commencement of duodenum: frequent abrasion and softening of mucous coat of jejunum and ileum, ulceration of glands of Peyer.	Liver healthy or pale: gall-bladder full of fluid bile, often flaccid: bile pale and limpid.	Mesenteric glands always enlarged, containing curdy, scrofulous like matter: spleen sometimes enlarged.

Chusan differed from Bengal dysentery chiefly in these respects: in its setting in with much fever, which here is very unusual; in the slight degree in which the liver and large intestine were affected; and the great amount of mesenteric disease and affection of the small intestines. In many respects it resembled Cabul dysentery, which the men called "the white flux," especially in the general absence of blood in the motions.

COMPARISON OF ACUTE AND CHRONIC DYSENTERY.

On a general review of the appearances presented in cases of acute and chronic dysentery, it would appear, that the liver is most frequently altered in the latter: that abscess is about equally frequent in either form—that in acute dysentery the liver is frequently enlarged and soft, while in chronic it is more generally small and indurated.

The stomach and small intestines also suffer more frequently in the chronic form, and the mesenteric glands are more frequently altered in it.

The extent of disease in the colon is least in the cases bordering on diarrhœa, and whereas in 160 cases of acute dysentery the large intestine is perforated 8 times, in chronic it was only once in 55.

Nothing very distinct can be said about the gall-bladder or its contents, but in the acute form it appears to be generally healthy, although often loaded with inspissated bile; while in the most chronic form bordering on diarrhœa the gall-bladder is often flaccid, and its contents pale and fluid.

The proportion of hæmorrhagic cases is smaller in the chronic than in the acute form.

Enlarged spleen is perhaps more common in the chronic form, but this is to be looked on as an accidental complication. No occasional mention of the state of the pancreas or kidney is of value, as they are certainly not implicated in the disease.

TRUE DESCRIPTION OF THE CHANGES PRODUCED BY BENGAL DYSENTERY.

It is not my intention to enter into any minute description of the state of the intestines, which has been faithfully described by both Twining and Raleigh, nor am I able to throw any fresh light on the nature of the dysenteric process. It has been compared to erysipelas by Siebert, and to the corrosion of mineral acids by Cruveilhier and Rokitansky. The mechanical theory of the irritation of scybala or accumulation of fæces acting on an inflamed surface, though generally abandoned, still finds some supporters. It has by many been attributed to the irritation of altered biliary secretion, or to its absence. Parkes considers it to be a process of ulceration universally commencing in the solitary glands of the large intestine. Others, with Raleigh, consider it to be a simple inflammation of the mucous coat of the large intestine, (if it were simple it would be more amenable to treatment). Whatever of truth or error there may be in these opinions, the appearance presented to us in simple Bengal dysentery is, that of an inflammation of the large intestine, which may be diffusive, ulcerative, purulent, hæmorrhagic, or gangrenous, according to circumstances. The disease in Europe and in India is essentially the same, and the best scientific descriptions of Bengal dysentery are those given by Dr. Baly of London and Rokitansky of Vienna, although the latter has not met with the amount of ulceration, which is common here. As seen here, the process is very generally one of mortification and sloughing, not of simple ulceration, i. e. the ulceration is often secondary, and occurs only after the sloughs are thrown

off. Inflammation and ulceration of the solitary glands is very unusual, or has been very carelessly observed, and I believe it may be stated generally, that in Bengal dysentery, they are not peculiarly or primarily diseased. It should be borne in mind, that the state of the solitary glands, as observed by Murray and Parkes, exactly corresponds with their usual appearance in cholera, and that all Murray's and most of Parkes's cases occurred in dysenteric patients suddenly carried off by that disease.

FREQUENCY OF COMPLICATION WITH DISEASE OF LIVER.

Perhaps I may as well offer a few remarks on the frequency of the occurrence of disease of the liver in connexion with dysentery. Our information on this subject is very meagre, as few observers have recorded any change of structure in the liver except that of the formation of abscess.

On the continent of Europe, Broussais, Gueretin, Thomas, Siebert, and Rokitansky, some of whom have written at great length on the pathology of dysentery, do not, as far as I can learn from abstracts of their writings, allude to hepatic complications. In the miasmatic form of dysentery, abscess is very unusual: it was rarely observed in the Continental wars of the 18th century* and in the Peninsula—not at all in various Austrian epidemics or in the Penitentiary. In the Burmese war, Waddell says he never saw any structural lesion of the liver. In China, Dr. Wilson found abscess only twice in 61 fatal cases. The only exception to this general rule seems to be Ireland, where Dr. Cheyne found it four times in 30 cases; but again Dr. Mayne never met with it.

* Pringle however found abscess in 2 out of 5 cases, of which he gives dissections, but elsewhere says that the liver was generally healthy.

The following data are sufficient to confirm the opinion, that abscess of the liver is a much less frequent complication of dysentery in Calcutta, than in Bombay or Madras. The reason I am not prepared to explain: but assuming, what there is every reason to believe, that abscess co-existing with dysentery is rare in the miasmatic form, and common in the other—that it is more common in the other presidencies than in Bengal, (where dysentery itself prevails so extensively in its worst shape,) and that it may be common one season and rare the next, it naturally follows, that some far more general cause must determine the formation of abscess than the limited one, of the absorption of pus from the ulcerated surface of the colon, or from suppurating mesenteric glands, assigned by Dr. Budd.

In New Orleans Dr. Robertson found hepatic abscess the common cause of death in dysentery.

In Madras, Annesley ..	.. in	51	cases finds	26 abscesses.
Dr. Shanks	.. in	96	..	36 „
Parker and Innes	.. in	61	..	13 „
Ballingall ..	.. in	35	..	4 „
At Bombay, Dr. Morehead	.. in	30	..	12 „
At Calcutta, Seaman's Hospital*	in	24	..	5 „
Medl. College Hospital*	.. in	54	..	14 „
Genl. Hospital ..	.. in	215	..	27 „

It is however worthy of remark, that the liver has been found in the General Hospital to have been altered in 118 out of 215 cases: in the Medical College Hospital in 13 out of 30 cases, while Sir James MacGrigor found it in India altered 16 times in 22 cases, "and in Egypt as in India found it diseased." In the Peninsula the liver was found generally healthy, but sometimes indurated and softened and sometimes the seat of abscess. In Dublin Dr. Mayne says, it was always healthy, but sometimes congested.

* If these cases were minutely examined, I think that the number of cases of abscess would be somewhat less.

While on this subject, I may add, though not prepared to enter on the question of the connexion between liver disease and dysentery, that, on analysing 46 cases in the General Hospital, returned as hepatitis and terminating in abscess, the large intestine was found ulcerated in 15, and the small intestine in parts over-vascular, or with its mucous surface abraded in 4:—14 patients had been suffering for sometime from dysentery, and 41 had been ailing for a week or two before admission.

Simple acute inflammation terminating in abscess would therefore seem to be comparatively unusual in this part of India, though I am unacquainted with what is termed duodenal dyspepsia by Dr. Parkes, and which he considers to be the usual precursor of abscess.

I would also venture here to intimate a doubt, whether the occlusion of the duct from enlarged glands of the capsule of Glisson, or inflammation of the gall-bladder in new arrivals, as described by Twining, be at all of common occurrence.

ON CERTAIN APPEARANCES MENTIONED BY AUTHORS.

Ere concluding, I would offer an observation or two on various appearances which have been described by authors. Scybala or accumulations of fæces appear to have been scarcely ever observed, during life, and never after death.

No sloughs of complete portions of the intestinal canal have been remarked, but during life large, tough, tubular portions of mucous membrane and effused lymph have come away frequently, and no doubt they are occasionally found in situ.

Ulceration of the ileo-colic valve is probably more frequent than it is represented in these tables to be, but no such thing as intus-susception of the small into the large intestine has been witnessed.

The appearance described by MacGrigor, as fungoid or lichenoid excrescence of the colon, is common enough.

No suppuration of the mesenteric glands or occurrence of pus in the vena cava, have been met with.

Displacements of the large intestine are frequent, but have not been noticed with sufficient accuracy; however they are of slight practical importance.

The same must be said of adhesions and matting of the omentum.

The *lumbricus teres* is often found, especially in patients who have come off a long voyage.

Cicatrization of ulcers, which Raleigh considers so unusual, is far from infrequent.

The pouring out of blood in immense quantities from the commencement of the disease, as described by Twining, and stated to be very rare at Bombay by Morehead, occurred in 19 out of 160 acute cases.

On the subject of spasm of the intestine, post mortem examination throws no light.

CONCLUSION.

Were it not my object to make these remarks as short as possible, I should like much, to append the history of some of the most interesting cases, for instance of two or three cases of what has been clumsily termed perityphlitis, or abscess in the cellular tissue round the cœcum, of a case carried off by cholera, just after the process of cicatrization, marked by a dark coloured deposit, had been completed, of various hæmorrhagic ones, especially an instance in which hæmorrhage from the bowels was preceded by epistaxis and hæmoptysis, and most of all, a fatal case complicated with odd tetanic symptoms, the only one I have met with, which corresponds at all with the nervous symptoms described as occurring in some of the cases at the Penitentiary.

NOTES.

1. Having alluded only very cursorily to the subject of treatment, I may be allowed to add that, after greatly increased experience of the disease, the opinions expressed by me in former years in the following extracts, still appear to me in the main just. Possibly, the value of the use of sugar of lead in practice among Europeans may be over-rated, and others do not speak so well of it, but English authors have never placed so much confidence in it as American and German ones. After all, our practice in bad dysentery is very unsatisfactory, and quite a contrast to that in fever:—would that other diseases were as manageable as the latter! The approximate percentage of deaths in the General Hospital will shew, that except in bad seasons, and when cases are brought in late, the mortality from it is small.

1841.	1842.	1843.	1844.	1845.	1846.	1847.	1848.	1849.
6.09	5.2	3.8	2.1	4.	10.4	6.	6.2	2.9

"It cannot admit of doubt, that calomel and drastic purgatives have been injudiciously used, and that a return to a milder mode of treatment will be attended with the most beneficial results.

There is no difference of opinion as to the propriety of free depletion in the earlier stages of this disease, followed up by the use of mild purgatives, among which castor oil is quite invaluable. The combination of blue pill, ipecacuanha, gentian and hyoscyamus, so commonly employed, is a most useful preparation, and opium is also a very important remedy, although the belief that it merely masks the disease is very prevalent. An opiate enema, or Dover's powder, may, in most stages of the disease, be most usefully administered. In the dysentery of children, no medicine exceeds in value the Hydrarg. c Cretâ, combined with other remedies."—*Med. Gazette, June* 25, 1841.

"As the incipient stage is usually past before men are sent to Hospital, general depletion and the means commonly applied to check the onset of the attack, are often inapplicable. Indeed, as dysentery is essentially an inflammation of a mucous not of a serous surface, it may be doubtful whether local be not often as effectual as general depletion. As to the use of calomel, which is so commonly employed at its onset, the general feeling of the profession seems to be against its employment at a later stage, and indeed it is difficult to conceive what beneficial influence it can exert on an ulcerated surface.

The usual practice in the Seaman's Hospital is the free exhibition of sugar of lead and opium, and it appears to answer well, and has never produced those disagreeable constitutional effects which are attributed to the use of the preparations of lead. As much as from nine to fifteen grains of sugar of lead, combined with small quantities of opium, (one-half or a whole grain of opium to three of sugar of lead) are given within the twenty-four hours, and

this treatment is continued for several days, along with the free use of leeches and opiate and sugar of lead enemata, with purgatives every other morning and milk diet. This mode of treatment has been tried very extensively among natives, and appeared to be very efficacious and convenient, as avoiding the chance of salivation. It would seem to be peculiarly adapted to that insidious form of hæmorrhagic dysentery, supervening on chronic disease, of which several cases have of late occurred in middle aged men, characterised by the pouring out of immense quantities of blood from the mucous surfaces, indeed compared by some to uterine hæmorrhage. But we do not advocate any exclusive mode of treatment, or assert the superiority of sugar of lead over various other remedies, and of course, after the dysenteric symptoms have begun to subside, various alteratives are useful in aiding the bowels to regain their healthy tone."—*Seaman's Hospital Report for* 1844.

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2. As the number of autopsies in cases of dysentery, occurring in children at the General Hospital, is extremely small, and as no such case is included in the preceding tables, I add the post mortem appearances, in a case lately treated by my colleague Dr. Cantor, and obligingly communicated by him to me. It will be seen that the case was one of simple ulceration, without any sloughing; also that the ulcers were uniformly diffused along the mucous membrane, and that the solitary glands of the cœcum were not specially implicated; the process of ulceration was still going on, and that of reparation had not commenced at any point.

Child ætat. 4—ill for 3 weeks, death preceded by convulsions.

Large intestine studded throughout with equally diffused ulcers. The earliest stage of the ulcer was a raised white point, with greyish contents, (enlarged mucous follicle?) which enlarged and ulcerated, destroying the mucous membrane. The ulcers varied in size from a pin's head to a sixpence, and had raised margins of a white finely fringed appearance. The cœcum and rectum were somewhat thickened: appendix vermiformis studded with numerous minute grey points. There were nine lumbrici in the large intestine.

The small intestines were healthy, slightly injected here and there.

Liver somewhat small. Spleen healthy.

There were clusters of enlarged mesenteric glands, and some single ones enlarged, while others were natural.

APPENDIX.

Use of large Enemata in Dysentery.

1. *Out of India.*—With the use of enemata in dysentery, the profession has been familiar, at least since the days of Celsus. That author recommends various soothing and oleaginous injections. And to come to more modern times, Sydenham used not merely small opiate clysters, but enemata of ½ lb. of milk. In the systematic use of these three or four times daily, he had the utmost confidence, although he remarks, that really bad dysentery, with much structural change, will not yield to clysters, whether purgative, astringent, or emollient. Böerhave exhibited emollient clysters three or four times daily.

It would be tedious to enumerate the different authors who have recommended particular enemata in this disease. As fashion has varied, they have been in high repute, or fallen into comparative disuse. The French especially have been fond of their lavements and demi-lavements, and have thought them peculiarly efficacious in the commencement of the disease. And of late years, in the dysentery of children, Trousseau strongly urges the employment of enemata of nitrate of silver of 8 or 10 oz., to be thrown up in a child of two years old with the long tube, after the bowels have been washed out with a common warm water lavement. All these enemata were meant to act on the ulcerated surface of the

colon, and all authors seem to have believed that with a syringe used in the common way, injections may be made to pass up as far as the ileo-colic valve,—and recently, Boudin states, that he has had positive evidence of such enemata passing beyond it.

Some twenty years ago, O'Beirne proposed the introduction of a long tube above the sigmoid flexure, (on the idea of its often being the seat of spasm,) to clear out the intestinal canal more effectually, and published two cases of dysentery which he had cured by it, and at a later period a third one. Whether his plan has been acted on in England, where the disease is rare, I am unable to say, but in 1840, Dr. Symonds (Library of Medicine) calls attention to the practice, and in Ireland, where epidemics of it are still not unusual as in former centuries, and where there must have been free scope for its use, it seems not to have been employed.

The chief novelty of late years appears to be the use of injections of water and the albumen of eggs by Mondière. He injected this mixture thrice daily, and by his own account, with wonderful success. All writers, however, with the exception perhaps of O'Beirne and Mondière, seem to have regarded enemata only in the light of most useful adjuvants.

2. *In India.*—To turn to tropical dysentery, it would be difficult to find an author who does not recommend enemata in one shape or another. In 1639, Bontius our first writer is found using them,—in 1783, Mathews advises their employment "for sheathing the bowels and obtunding the fluids." He gives clysters of bark to support the system, or of tobacco to clear out the bowels, and these he administers through a hookah snake. With so formidable an instrument it seems probable that his enemata were large. Since his day, I imagine that few practitioners have failed to have recourse to clysters, small when opiate, and of about one pint in bulk, when meant to act generally on the surface of the bowel.

Sir James MacGrigor, for instance, in the beginning of the century, records the employment of almost every possible variety of them.

In 1837 Dr. Jackson, of H. M. 6th Regt., (a great advocate for the employment of clysters in ordinary constipation, and who recommended six pints to be usually thrown up), mentions having passed up the long tube 15 inches beyond the anus in a fatal case of dysentery.* Large enemata of tepid water, and the injection three times daily of a pint of water with nitro-muriatic acid, are frequently recommended by Madras writers some ten years ago; and in 1840, the officiating editor of the Madras Journal mentions, that he has often, in dysenteric cases, pursued O'Beirne's plan, and given wonderful relief with large emollient enemata.†

Enemata of various sorts have been constantly used in Bengal. Acetate of lead seems to have been always a favourite. Twining used it often; and up to 1840, Mr. Raleigh employed it largely in the General Hospital, and believed that with a pint and a half of fluid he covered the surface of the large intestines: these injections he repeated every 2 or 3 hours in hæmorrhagic cases.

In the end of the year 1847, Mr. Hare again invited attention to O'Beirne's method, and to the systematic use of large enemata. He thinks that they have not been hitherto used in sufficient bulk or with sufficient frequency, and he endeavours to assign to them a position of primary importance in the treatment of dysentery. He seems with Jackson to have arrived at a maximum of six pints. He believes "that he may save the lives of hundreds who die under the present system," which he considers to be commonly salivation, and hopes to produce quite a revolution in practice. Various medical officers have since published cases of the successful employment of large enemata, chiefly in chronic dysentery.

* "No one has thought of making use of this method."—Mr. Hare on Dysentery, p. 14.

† This mode of treatment ought to have been noticed and applied in India. —Hare, p. 7.

To raise them into this new position of independent value, it appears to be necessary that we should have a series of bad cases of dysentery treated successfully by the long tube, unaided by the various other modes of internal treatment.

Whether Mr. Hare's zeal and energy will be able to elevate them to this new rank, or whether the long tube will share the fate of the long-forgotten hookah snake of Mathews, it is for the experience of future years to determine, for like other remedies, injections vary in efficacy in different seasons and in different forms of the disease. Of great value as injections are, they certainly produced no diminution of mortality in the years, in which they were employed in the General Hospital most frequently and in largest quantity, namely, in 1848 and 1849: and the consideration of their past history does not encourage the hope that they will ever permanently retain a position higher than that of most useful adjuvants. The large ones can be used systematically only in Hospitals, and are never likely to become favourites in private practice.

Use of Quinine in Remittent Fever.

An apology may be deemed necessary for collecting at this time of day, evidence in favour of the free use in remittents, of one of the very few specifics we possess in the whole circle of the materia medica. Yet it is possible, that some may not have had the opportunity of carrying their investigations on the subject further than my friend Mr. Hare, who thus expresses himself: "I have searched every where indeed, and all that I can find in books and Magazines since Johnson's time till now is, bleed and give large calomel pur-

gatives, but be very careful not to give quinine too soon." *Hare on Fever*, p. 13. To such readers the following information may be interesting from its novelty, and to others perhaps, as being a connected historical sketch of the use of quinine in remittents in India. After all, the use of quinine in remittents is merely an extension of its use in intermittents, and in either case, only a substitute for the older treatment by bark; but to trace all this would lead us into still further details, while I fear that even the following brief recital of facts may be tiresome to many.

1. *Out of India.*—In the year 1835, M. Maillot published a work on the fevers of Africa, and states that, by the exhibition of quinine he reduced the mortality in bad remittents from 1 in 3½ to 1 in 20. He gave it without any reference to the stage of the fever, and in bad cases he gave from 1 to 2 scruples by the mouth, and a drachm as an enema. In this manner he in several instances gave as much as 148 grs. in 24 hours.

The following extract from Bouchardat's Materia Medica, published in 1839, will show the state of European opinion and practice at that period. "In bad remittents the salts of cinchona save the patient from certain death. In these cases we must act much more rapidly, and augment the doses. We must then, according to Torti, administer the bark on the first sign of remission, for there is often no intermission. Bretonneau goes still further: he commences the exhibition of bark in the middle of the paroxysm, and is not afraid of increasing the intensity of the access during which it is given; for experience shows, that the medicine does not commence to act till several hours after it has been administered, i. e. during the remission. Bretonneau used bark, but we now greatly prefer quinine, which is more quickly absorbed, and acts more rapidly. We prescribe 30 grs. of it with 1 gr. of opium, and give it in 3 doses. From the moment that the access has been prevented or moderated, it is no longer necessary to give it in these large doses, but we may continue to give smaller ones for some days."

Dr. Hille in Casper's Wochenschrift in 1839 mentions, that quinine is the only medicine for the intermittents and remittents of Surinam; and in 1842, writing in more detail, says, it must be given in 12 to 40 gr. doses in all stages.

The American physicians seem to have been somewhat slow in following their Continental brethren, and although quinine was given in yellow fever at New Orleans in 1839 in scruple and scruple and a half doses,* I believe that Dr. Byrne (Boston Medical and Surgical Journal, July 1845) is the first who talks strongly in favour of quinine in remittents. "He found that quinine might be used with the greatest advantage even in the hot stage. As regards the dose, he observes, that in ordinary remittents it is a matter of indifference what dose is administered, provided 10 or 20 grains are introduced into the system a certain number of hours before the paroxysm. In severe cases, there is no longer any choice; but the quantity above specified should be given in 1 or 2 doses at most. We thus find, that by the middle of the year 1845, the use of quinine in large doses, without much reference to the stage of the remittent, was fairly established, and the year 1846 produced papers by Drs. Tuck, Upsher, and Professor Van Buren, and the year 1847, by Holmes,† all recommending the practice. It is unnecessary to allude to writers of later date.

In India.—The free use of quinine in remittents appears to have been slowly and cautiously adopted, and this invaluable medicine seems on the whole to have been more extensively used in Madras than in the sister presidencies. Some of the earliest notices on the subject are the following: Dr.

* Mr. Hare alludes to this, p. 15.

† Mr. Hare mentions Mr. Holmes, page 14.

Geddes in 1828 mentions giving quinine with the pulse at 108, and after extensive use of it, he in after years announced, that he preferred giving it in every instance to affecting the mouth with mercury. In 1833, Corbyn treated a bad remittent fever in Calcutta with most gratifying success, and says, that his routine practice was, six or seven hours after the administration a purgative to exhibit 7 or 8 grs. of quinine, and to continue this steadily every four hours without reference to paroxysms. He seems to have produced giddiness and deafness frequently. In 1834 Dr. Wright, in the Goomsur country, gave the sepoys 20 grs. daily, but used to watch for remissions: in 1835, Mr. Eyre gave it in the same country often in scruple and a half doses, of which the Superintending Surgeon disapproved; and, the supply of quinine running short, Lord Auckland sent down a supply of 20lbs. at his private expense,* when every one who could get quinine, employed it with an unsparing hand, and I believe with very little regard to remissions.

In 1835, Twining, in the second edition of his book, strongly recommends it in remissions; and Goodeve in 1837 says, that it has produced quite a revolution in practice, having almost brought back the days of bark: still he waits for remissions. In 1840 Mr. Green recommends its free use in remissions, and the Madras Journal of the same year calls attention to Maillot's large doses. In this year also, Dr. J. Murray, writing of the Malwa sweating sickness, says, there were few conditions that would prevent his giving quinine; that he was not deterred by headache, but if it were violent, he would use other measures previously. In 1841, Mr. Martin writes, "when the remissions are well marked, quinine should be given without waiting for every thing; if we wait for every thing, we shall often wait till it is too long or too late." In 1842, Dr. Bell in Persia gave quinine in small doses without any

* For this information I am indebted to Mr. Surgeon General Stiven.

reference to the stage of the fever, and in the same year the recorded practice in the General Hospital at Madras was as follows: "Quinine is given in 5 gr. doses every hour on the least tendency to remission, or change, however slight. No bad consequences, nor any aggravation of the symptoms, have been observed, even after it has been continued throughout the greater part of the exacerbation."

In 1843, Dr. Morehead expressed himself thus clearly regarding the use of quinine in remittents at the General Hospital of Bombay: "I think that even in cases where the remission is very imperfect, quinine should be tried, and repeated or not according to the effect." "Should the remission be distinct, dryness and brownness of the tongue offer no drawback to the use of quinine." "I do not think the presence of the phlogistic diathesis or the presence of fixed congestions of necessity contra-indications to the use of quinine." "We are frequently, it is feared, induced to intermit the remedy, because a febrile exacerbation may have followed after its use in the first or second remission, under the apprehension, that the febrile excitement has been produced by the remedy," "but we have no sufficient grounds," &c. In this year also, though he waits for a remission, MacGrigor talks of the paramount importance of keeping off the next paroxysm. About the same time the present writer published a case in which quinine was successfully administered during grave cerebral congestion, notwithstanding the presence of a black tongue and sordes about the teeth; and in 1844, in the report of the Howrah Seaman's Hospital, he attributes the great reduction of mortality from the 8 per cent. of preceding years to 1.7 in one year, and to 0 per cent. in the next, or no death in 127 cases, to abstinence from free venesection and the early use of quinine, always making allowance for what the Germans call the "Genius epidemicus morborum." In the same year, Eveleigh in the Calcutta Journal mentions his having

employed quinine in severe cases, in which the head and the liver were affected.*

Meanwhile, quinine was becoming more extensively used, and especially in parts of Madras. In the bad remittents in Goomsur it was given in very large doses, and the Government supply of quinine did not equal the demand. It used even to be reported facetiously, that the Madras Board had recommended the appointment of a Special Superintending Surgeon to controul the extravagant expenditure of quinine. In 1845, the Bengal Medical Board was applied to, and liberally sanctioned a small extra supply for the Hill Agency, but very many lbs. were procured from private sources at the expense of the Agent. Mr. Cadenhead regularly and constantly used it in the hot stage of fever, from the commencement of the year; and at the request of the Editor of McClelland's Journal, began to write a paper on the subject, which, owing to the distraction of political duties, was never finished. There was a regular schism among the practitioners in the Northern division regarding the stage at which it should be given; in short, the most impartial account of the state of the question at this time is given by Dr. Williams, of St. Thomas, writing in the same year. After alluding to the use of large doses of quinine in India and in various parts of the world in remittents, he remarks: "The battle still rages between those who would treat this disease symptomatically, and those who prefer the specific remedy."

Two years after this, or towards the close of 1847, Mr. Hare published a pamphlet, in which he most strongly advocated the use of quinine from the commencement in all miasmatic fevers. He advises its administration without any reference to the stage of the fever, and his specific directions are chiefly the same as Mr. Corbyn's treatment of the fever

* "This is the only approach to the truth I can find."—Hare, p. 12.

of 1833. Since then, Dr. Macrae of Howrah has published an account of many cases of fever treated similarly: this mode of treatment he has pursued since the middle of 1847, as shewn by the records of the Seaman's Hospital; and Dr. Ford of Madras states, that he has been following a similar practice since the year 1845, as testified by his reports to the Medical Board.

By this time pretty abundant evidence had accumulated on the subject, and I do not know that any more recent has been submitted to the profession in Bengal. A paper by Dr. Cameron of Ceylon, advocating the same views, has since appeared, but I have not seen it.

To the enquiry, what has been established by all these facts regarding the free use of quinine in tropical remittents during the last twenty years, the following would seem to be the natural reply.

It admits of no question, that the free and early employment of quinine in remittent fever is a great step in advance in practical medicine, and it is also important to know that there is no necessity for extreme caution in its use, but the late Dr. Williams, as already quoted, describes the state of the case correctly. It remains for practitioners to adopt the symptomatic or the specific mode of treatment: or a mixture of both, which may be termed the ecclectic. Much must of course depend on the intelligence and experience of the individual, on the circumstances in which he may be placed, and on the character of the fever which he may be called on to treat. No practice should be indiscriminate, for such treatment is never scientific, and is not always safe.

BISHOP'S COLLEGE PRESS.

MÉMOIRE

SUR

UNE DYSENTERIE ÉPIDÉMIQUE

QUI A RÉGNÉ A L'HÔPITAL MILITAIRE DE NAMUR, PENDANT LE DERNIER TRIMESTRE 1831;

PAR LE DOCTEUR **FALLOT**,

MÉDECIN PRINCIPAL A NAMUR, MEMBRE CORRESPONDANT DE LA SOCIÉTÉ MÉDICALE D'ÉMULATION, etc.

MÉMOIRE PRÉSENTÉ EN JANVIER DERNIER A LA SOCIÉTÉ MÉDICALE D'ÉMULATION, ET PUBLIÉ PAR ELLE.

(*Extrait des Archives générales de Médecine.*)

MÉMOIRE

SUR

UNE DYSENTERIE ÉPIDÉMIQUE

QUI A RÉGNÉ A L'HÔPITAL MILITAIRE DE NAMUR, PENDANT L'HIVER 1831 A 1832.

J'avais eu l'occasion de voir deux fois des épidémies de dysenterie : la première en Espagne, en 1810; elle était aussi soudaine dans ses attaques, que prompte dans sa propagation et fatale dans son issue; elle régnait en automne; on la jugeait contagieuse; elle se présentait dès son début avec tous les symptômes propres aux fièvres dites alors adynamiques; et, traitée en conséquence par le quinquina, le camphre, l'éther, et d'autres médicamens de même nature, elle exerçait les plus grands ravages.

J'en revis une seconde en Belgique, en 1815; elle s'étendait le long du littoral de la mer, attaquant un grand nombre d'individus à-la-fois, mais tout particulièrement les militaires; je ne recueillis aucun fait démonstratif de

sa propriété contagieuse ; rebelle aux méthodes excitante, évacuante et astrictive, elle cédait promptement aux féculens unis à l'opium. Cette médication obtenait des succès tellement prompts et constans, que je la jugeais alors comme un véritable spécifique anti-dysentérique.

Seize ans se sont écoulés depuis, et je n'avais plus eu occasion d'en rencontrer ; ce n'est pas que, de temps à autre, et surtout pendant les automnes, des entéro-colites aiguës plus ou moins graves ne se fussent présentées à mon observation, mais elles étaient sporadiques ; des émissions sanguines locales faites à l'anus et sur le trajet du colon, tant par les sangsues que par les ventouses scarifiées, conjuraient le mal assez promptement, et prévenaient une issue fatale.

Dès le mois de septembre de l'année précédente (1831), mon attention avait été saisie par l'irritabilité extraordinaire du gros intestin. Depuis quatorze ans que je suis les principes de la médecine physiologique, jamais je n'avais vu, sans les juger, survenir autant de diarrhées dans les phlegmasies aiguës des organes respiratoires et digestifs ; ailleurs, quand ces flux de ventre arrivaient, elles étaient accompagnées de la rémission ou de la cessation des symptômes principaux de la maladie ; à présent ils ne faisaient qu'y ajouter, n'opéraient pas une révulsion critique, mais une extension du travail phlegmasique, une altération organique de plus que celles qui existaient, et, ajoutant au trouble morbide en raison composée de l'irritation colique et de l'étendue des pertes causées par la multitude des déjections et la précipitation des substances alimentaires, ils compliquaient la position du malade, et rendaient la médication plus difficile et plus incertaine. Dans la très-grande majorité des cas, cependant, l'abstinence de tout aliment, la persévérance dans l'usage

des boissons gommées, les lavemens féculens, et, après l'appaisement de la phlogose gastrique, l'administration de quelques gouttes de laudanum dans la décoction de salep, mettaient bientôt un terme à la maladie.

Il est à remarquer qu'à cette même époque, l'irritabilité de toute la muqueuse intestinale était telle, que les acides végétaux, généralement si avidement appelés, si facilement supportés, si utilement administrés dans les gastro-entérites aiguës, excitaient des nausées, des pesanteurs et la diarrhée. C'est une observation qui fut faite par tous les médecins de l'hôpital militaire.

C'est ainsi que les choses allèrent jusqu'au 19 octobre, qu'une première évacuation dans laquelle se trouvaient plusieurs dysentériques, nous arriva de l'hôpital de Louvain (1). Une seconde, expédiée de là le 3 novembre, succéda, qui en comprenait plusieurs autres. Voilà

(1) Nous avons rencontré, il est vrai, dans les derniers jours d'août, un choléra-morbus. J'en joins ici l'histoire en note.

Un volontaire, de trente ans, d'un constitution sèche, en traitement à l'hôpital pour un bubon ouvert, et n'ayant pas pris de mercure, fut saisi dans la nuit du 28 au 29 août, tout d'un coup et sans autre avertissement que de l'inappétence et de la tension dans le ventre, d'un trousse-galant des plus graves. Les déjections alvines et les vomissemens sont incessans. A la visite du 29, face tirée et retombée, yeux caves et cernés, facultés intellectuelles lentes et embarrassées, paroles traînantes et indistinctes, peau froide, crispée, peau filiforme, brisement de forces tel que le malade ne peut se redresser ; langue pâle, muqueuse ; soif insatiable, ventre plat, indolore. Les vomissemens étaient d'abord porracés, d'une amertume pénétrante. A la visite, ils ont l'aspect et la couleur d'une eau de riz saturée. (Un gros de teinture d'opium dans quatre onces d'eau sucrée, à prendre une cuillerée de quart-d'heure en quart-d'heure, des sinapismes volans.) Peu-à-peu les déjections s'arrêtent, la peau s'échauffe, la face se colore, une grande pesanteur avec sentiment de suffocation se manifeste à la région précordiale. (Suppression de la potion opiacée, décoction de salep sucrée.) Le soir, éruption de plusieurs milliers de boutons rouges, accompagnée d'un insuppor-

le fait. Je ne décide pas si, entre lui et celui de l'apparition de la dysenterie à l'hôpital, chez des malades qui n'en avaient jamais offert aucun symptôme, il existe des relations de filiation, de causalité, ou si c'est une simple coïncidence, je narre et ne conclus pas; le lecteur se chargera des rapprochemens.

Les malades arrivans furent placés dans plusieurs salles; ils avaient beaucoup souffert en route. Les journées pendant lesquelles on les avait transférés avaient été froides et humides; le trajet était de neuf lieues du pays: ils n'étaient arrivés chez nous qu'à dix heures du soir.

Le lendemain l'entéro-colite fut signalée chez plusieurs d'entr'eux, tantôt simple, tantôt compliquée de pneumonite et de péritonite.

Et, à cet égard, un mot sur la manière dont les histoires de maladies sont faites dans mon hôpital. — Jour par jour je dicte à haute voix des notes au lit des malades mêmes; elles sont recueillies par tous ceux qui se trouvant à la visite veulent s'en donner la peine. Je porte ensuite mon diagnostic, tel que je le conçois; il est de même inscrit en marge: arrive-t-il quelque nouveau symptôme, il est consigné et interprété. Après la mort de l'individu qui a fait le sujet de l'histoire, ces notes sont relues avant l'ouverture du cadavre; les jugemens portés pendant la vie sur la nature du processus morbide, sont rappelés. Ensuite on passe à l'autopsie, et c'est elle qui confirme, casse, modifie le jugement, et prononce l'arrêt définitif.

table prurit sur toute l'étendue du corps. Le lendemain, désir de manger. Le 1.er septembre, il n'y paraissait plus.

Le 5 septembre, après quelques écarts de régime, réapparition de la diarrhée, mais cette fois avec ténesme et sans vomissement, douleurs coliques. (20 sangsues sur le ventre et à l'anus, lavemens amidonnés, cataplasmes émolliens.) Le 6, tous les symptômes ont cédé. La convalescence a été longue, mais ne s'est plus démentie.

Bornée d'abord aux seuls malades évacués de l'hôpital de Louvain, elle gagna bientôt plusieurs de ceux qui se trouvaient en traitement chez nous. Chaque jour ajoutait de nouveaux dysentériques à ceux que nous avions visités la veille. Le 5 novembre, j'en comptais 48 dans mon service; le 9, 53, quoiqu'il en fût mort deux pendant la nuit; le 12, 62; le 16, 70; le 17, 72; le 19, 79.

Ce n'était pas par des arrivés de la caserne que le nombre grossissait, mais par l'extension de la maladie aux autres malades, et par des rechutes chez ceux qui déjà l'avaient éprouvée.

Jusqu'au 25 novembre, l'épidémie alla toujours en augmentant d'intensité, alors elle commença à perdre de sa violence, et devint moins rebelle à l'action des modificateurs.

C'est de cette épidémie de dysenterie renfermée dans l'enceinte de l'hôpital et n'en franchissant pas les limites (1), que je vais essayer de tracer l'histoire. Elle ne sera pas sans intérêt, peut-être.

Je ne me flatte pas de rien dire de nouveau; mais on peut être sûr que, lorsqu'il s'agira de faits, je ne dirai rien que de vrai, rien qui ne résulte des notes prises au lit même des malades, et dans les discussions auxquelles je me suis livré, rien que de conforme à ma conviction.

Mais avant d'y procéder, jetons un rapide coup-d'œil sur la constitution atmosphérique de l'année.

L'hiver avait été doux, mais humide; le printemps fut froid, pluvieux; l'été et l'automne superbes et tels qu'on les voit rarement dans nos latitudes. La chaleur constante

(1) Je ne sache pas qu'aucun de mes confrères de l'ordre civil en ait eu à traiter en ville; s'il s'en est trouvé, c'est sporadiquement et avec des formes très-douces.

et modérée, les orages rares, les pluies peu abondantes, douces, rafraîchissantes. Il est aussi quelques circonstances au milieu desquelles nos soldats se sont long-temps trouvés, et qui méritent une attention spéciale.

Depuis la révolution d'octobre, ils ont toujours été tenus sur le qui-vive; placés aux avant-postes, cantonnés dans des hameaux et des chaumières, ou bivouaqués dans les polders de la Flandre, le long du littoral de l'Escaut ou dans les marais de la Campine. L'aggression improviste et vive de l'ennemi, au mois d'août, la retraite un peu précipitée qui en avait été la conséquence sur plusieurs points, avaient produit sur leur moral une fâcheuse impression et semé un grand découragement. Recomposée à la hâte, immédiatement après la fin de la courte campagne, notre armée fut réunie dans un camp au mois de septembre, et soumise à une sévère discipline, et à l'accomplissement de tous les actes qui pussent former son éducation et hâter son instruction militaire. Ce camp ne fut levé que fort avant dans le mois d'octobre, lorsque déjà plusieurs mauvais jours avaient annoncé l'approche de l'hiver.

Avant de passer maintenant à l'exposition de l'histoire générale de la maladie, citons quelques observations particulières; elles ont toutes été prises jour par jour au lit des malades mêmes. Si je les ai en général privés de leur forme de journal, c'est parce que celles-ci obligeant à reproduire chaque jour les mêmes symptômes, la lecture en est presque toujours fatigante. Les bornes que je dois me prescrire dans ce travail m'ont forcé à n'en produire qu'un petit nombre. Je les ai choisies parmi celles qui m'ont paru les plus propres à mettre en saillie les principaux caractères de l'épidémie; j'ai commencé par les plus aiguës et les plus simples. Cette manière de procéder m'a semblé la plus rationnelle comme aussi la plus facile pour le lecteur.

Obs. I.re — *Colite aiguë traitée et guérie par les sangsues.* — Rossau, de Bruxelles, long, mince, d'une pâleur excessive qu'il dit lui être naturelle, cheveux châtains, yeux noirs, peau blanche et transparente, poitrine plate, était sorti de notre hôpital le 1.er novembre, après y avoir séjourné un mois pour fièvre tierce. Il y rentra le 18, 4.e jour de la maladie: abattement moral, tranchées, sensibilité et rénitence de l'S iliaque du colon, selles sanglantes, avec ténesmes, langue pâle, peau chaude, pouls animé, fréquent. (20 sangsues, 16 à l'endroit douloureux, 4 à l'anus.

Le 19, quoique le malade ait peu dormi, la nuit a été tranquille; une seule selle facile et stercorale. A la visite, apyrexie, langue nette, pouls égal, ventre mou, insensible. (Diète absolue, eau d'orge édulcorée.)

Le 22, flux de ventre complètement arrêté; le sommeil est tranquille, la peau souple, le pouls régulier et l'appétit vif; le teint seul reste mauvais. (Diète, bouillie.)

Le 14, la face s'épanouit et perd de sa pâleur, l'appétit est vif, les déjections faciles, le sommeil paisible, les évacuations régulières..... Il est sorti guéri le 1.er décembre.

Voilà la colite dans la forme la plus simple où nous l'ayons observée; une seule application de sangsues faite à l'endroit douloureux a suffi pour la calmer. L'abattement moral observé chez le malade était motivé par la crainte de subir le sort de tant d'autres atteints du flux de ventre à l'hôpital; car quoique la mortalité fût grande en effet, la rumeur publique l'exagérait considérablement.

Obs. II.e — *Entéro-colite aiguë guérie par les sangsues.* — Vendenbosch, lancier, servant depuis sept mois, petit, mais bien proportionné et médiocrement musclé, était en traitement à l'hôpital depuis quelques jours,

pour perte d'appétit et courbature, quand survint dans la nuit du 12 au 13 novembre, un flux de ventre sanglant avec ténesme et prostration. A quatre heures de l'après-midi, le 13, nous le trouvons dans l'état suivant : face colorée, yeux injectés, peau brûlante, langue rouge et sèche, soif vive, pouls dur, vibrant, à 90; région iliaque gauche rénitente, douloureuse; épreintes continuelles avec déjections pelliculeuses nageant dans un liquide couleur de lavures de boyaux. (30 sangsues à l'endroit douloureux; eau de gomme édulcorée et acidulée.)

Le 14, pendant la nuit, sueurs générales abondantes, selles non sanglantes. A la visite : face bonne, apyrexie complète, ventre souple, insensible; langue rouge au limbe, pointillée sur le corps, soif calmée, pouls moins fréquent. (Diète, continuation de boissons.)

Le 15, selles naturelles, sommeil tranquille, face bonne, pouls développé, appétit. Sorti guéri le 26 novembre.

Ici la portion supérieure du canal alimentaire a été évidemment affectée; c'est dans sa modification morbide qu'il faut chercher la cause de l'inappétence qui précéda de plusieurs jours l'explosion de la colite. Les phénomènes sympathiques organiques ont été plus prononcés que dans le cas précédent; les sécrétions morbides ont été plus viciées, mais le centre circulatoire a conservé toute son énergie; le sang était lancé avec vigueur dans la surface tégumentaire, et dès qu'une application de sangsues a fait taire l'inflammation du gros intestin et la contraction spasmodique des capillaires cutanés, qui en était en effet sympathique, des sueurs abondantes sont survenues, et le rétablissement d'un émonctoire naturel a détourné le travail morbide de sa direction.

Obs. III.e — *Entéro-colite aigué, guérie par les sangsues.* — Lebas, âgé de vingt ans, wallon, taille moyenne,

squelette régulier, cheveux blonds, yeux gris, convalescent depuis plusieurs jours d'une tuméfaction avec saignement des gencives, fut atteint d'un flux de ventre sanglant et envoyé dans ma division le 7 novembre.

Le 7 au soir, face grippée, peau terreuse avec incrustation d'ocre, céphalalgie sus-orbitaire, langue rouge au limbe, muqueuse sur le corps; peau chaude, pouls fréquent, soif vive, envies continuelles d'aller, déjections sanglantes avec dépôt argilleux. (20 sangsues le long du colon et à l'anus; cataplasmes émolliens.)

Le 8, rémission des symptômes pendant la nuit; le malade n'a eu que huit selles sans ténesme, mais toujours précédées de coliques. A la visite : découragement, peau toujours terne, respiration égale, soif calmée, pouls sans fréquence, égal, développé; langue plate, molle, muqueuse au centre; tranchées à l'approche des selles, douleurs hypogastriques, ténesme. (18 sangsues sur l'S du colon, 4 à l'anus; décoction de riz.)

Le 9, amélioration marquée; les selles sont moins fréquentes, plus abondantes et plus consistantes; urines copieuses; la langue est humide, le pouls souple, sans fréquence.

Le 10, cinq selles liquides, mais sans mélange de sang dans les vingt-quatre heures; le malade s'assied et conserve sans effort cette attitude; la peau est souple, le pouls égal et lent, la soif calmée, la langue nette, mais pas encore d'appétit. (Eau de riz.)

Le 11, les selles s'épaississent et deviennent plus rares; le sommeil est paisible, la peau et le pouls naturels; l'appétit revient après être resté long temps languissant.

Sorti guéri le 21 novembre.

Ici l'inflammation colique a été plus opiniâtre, une seule émission sanguine n'a pas suffi pour la calmer. Ce-

pendant la maladie était récente, attaquait un sujet qui n'en avait pas éprouvé d'autres. On remarque ici cette décoloration particulière du teint, cette pâleur jaune avec mélange d'un rouge ocré. La violence du travail phlegmasique avait mis en jeu des irritations sympathiques tant de relation que de nutrition, mais qui disparurent avec l'inflammation qui leur avait donné naissance.

Obs. IV.e — *Colite aigue enlevée par une application répétée de sangsues.* — Marmart, flamand, blond, long, mince, squelette régulier, arriva de Louvain dans ma division le 21 octobre; convalescent d'une fièvre tierce. Son teint est jaune et son amaigrissement très-sensible; soumis à un régime proportionné à son état, il recouvre progressivement ses forces et ses couleurs, et quitte l'hôpital, guéri le 5 novembre. Reçu à l'établissement des convalescens formé à la caserne, il obéit aux sollicitations de son estomac et mange et boit immodérément. Dans la soirée du 8 novembre, il est reporté à l'hôpital dans l'état suivant : face altérée; traits affaissés; douleurs ventrales atroces; épreintes continuelles avec déjections de quelques gouttes de sang et de glaires; ventre ballonné, très-douloureux; pouls serré, vif; peau chaude et sèche; par intervalles envies de vomir et quelques crampes dans les mollets. (Vingt sangsues, seize sur le trajet du colon, quatre à l'anus; bain de corps; tisane gommée, administrée par cuillerées à café.)

Les coliques s'appaisent, les épreintes sont moins fréquentes, la prostration moins profonde; le liquide administré pour boisson a été conservé. (Seize sangsues; bain de corps; cataplasme émollient; continuation de boissons.)

Vers le soir, retour des douleurs hypogastriques; langue jaune, pointue, sèche; pouls à 80; le malade se sent prostré; la température est basse. (Vingt sangsues

à l'hypogastre, deux lavemens à la graine de lin).

Le 10, selles peu nombreuses, semblable à de l'eau de riz épaissie; la peau est réchauffée; le pouls descendu à 70 a repris de la force et de la rondeur; le malade a goûté quelques instans de sommeil; il est moins découragé; la pression sur le ventre n'y excite plus de douleur; l'eau de gomme acidulée, qui avait été appétée jusqu'alors, répugne maintenant, et des boissons chaudes sont désirées. (Deux lavemens de graine de lin; décoction de salep avec un demi-gros de teinture d'opium.) Depuis ce jour, les symptômes d'inflammation abdominale allèrent toujours en diminuant; les déjections se régularisèrent, et le malade fut évacué le 20 novembre aux convalescens; il sortit guéri le 6 décembre.

Déjà le tableau se complique : le sujet de cette observation avait été en proie pendant long-temps à une fièvre intermittente, ce qui, en langue physiologique, signifie une congestion viscérale non-continue; en sortant de l'hôpital, il mange immodérément et stimule avec excès les organes digestifs; il s'expose au froid, et la sédation produite par cet agent sur la surface tégumentaire, en suspend les fonctions dépuratives et sollicite les organes internes à y suppléer; aussi voit-on tout le tube digestif s'en ressentir, l'estomac se révolter, le gros intestin se contracter convulsivement : transmise à la moelle épinière, cette modification sollicite des crampes dans les plans musculeux postérieurs des jambes. Cependant la somme de vitalité est encore assez grande pour que la réaction s'opère; la peau s'échauffe; le cœur se contracte avec fréquence et avec énergie; des efforts sont faits pour exciter des sécrétions dont l'établissement puisse contre-balancer celle dont le gros intestin est le siége. Pour que cet heureux résultat s'obtienne, il faut qu'au préalable l'inflammation colique soit vaincue, et c'est à quoi l'on parvient

par les sangsues, les bains, les cataplasmes, le repos et la diète.

Obs. V.e — *Entéro-colite récidive guérie par les émissions sanguines et l'opium.* — Bélinse, lancier, taille moyenne, squelette bien conformé, cheveux châtains, yeux bleus, revenait de l'hôpital d'Anvers, où, à son retour de la captivité, il était entré pour fièvre intermittente quotidienne.

Quant à son arrivée ici, ayant été trouvé trop faible pour faire son service, il nous fut envoyé : son teint alors est pâle; les paupières tuméfiées; son moral découragé; il n'a plus de fièvre, mais ses forces lui semblent défaillir plutôt que d'augmenter; nous en trouvons la cause dans un flux de ventre avec ténesmes et sanglant qu'il a gagné en route, et dont il est encore actuellement attaqué, mais qui cède à un traitement gommeux et opiacé continué pendant trois jours.

Le 20, il est évacué aux convalescens, mais les selles étant redevenues liquides, il est renvoyé dans ma division; quelques cataplasmes et le salep édulcoré arrêtent le flux, et le malade est remis à la demi-portion. La peau reste toujours pâle; la figure triste, les paupières infiltrées.

Dans la nuit du 5 au 6 décembre : déjections terminenses, sanglantes, petites, fréquentes, rapprochées.

A la visite du 6, moral très-affecté; tranchées aiguës; région hypogastrique soulevée, dure, sensible; peau chaude; pouls vif; soif ardente; ténesme. (Vingt sangsues à la région hypogastrique; décoction de salep édulcorée et opiacée.

Au soir, disparution de la douleur; diminution du nombre des selles et de la soif; chute de la réaction vasculaire. Le 7 pendant la nuit, retour des tranchées fixées à présent à la fosse iliaque gauche qui est soulevée et très-rénitente, ce qui donne au ventre une forme bos-

selée : au milieu des épreintes les plus fatigantes; expression d'un peu de sang mêlé de pellicules; cependant le moral est beaucoup meilleur; la langue est humide; le pouls égal et régulier; la peau ouverte et souple; les irritations sympathiques paraissent calmées. (Quinze sangsues sur l'S iliaque du colon; salep édulcoré avec trente gouttes de laudanum; cataplasme.

Le 8, après l'application des sangsues, disparution immédiate des douleurs, mais continuation des épreintes et des déjections de petites pellicules nageant dans un liquide rougeâtre; soif nulle; pouls développé, égal; peau souple.

Le 9, nuit tranquille; déjections mêlées d'excrémens et de sang, accompagnées de ténesme; la face est bonne; la langue molle et nette; la soif nulle; le pouls égal et régulier. (Un petit lavement avec six gouttes de laudanum; décoction de salep avec un demi gros de laudanum.)

Le 10, appaisement complet du ténesme; selles stercorales, moulées, très-abondantes; appétit prononcé. (Deux bouillies; salep édulcoré.)

Le 11, selles stercorales, indolores; peau moite; urines abondantes; ventre souple, insensible; pouls régulier. (Continuation du régime.)

Le 18, la bouffissure de la face est dissipée; la peau est souple; la face se colore; les traits s'épanouissent; évacué aux convalescens.

Sorti guéri le 26 décembre. Dans cette circonstance encore les sangsues nous ont rendu service; mais seules elles n'auraient pu suffire pour obtenir la guérison; il a fallu l'intervention de l'opium non-seulement par la bouche, mais aussi par lavemens; la raison en est que la phlogose à laquelle nous avons eu à faire, quoique aiguë quand nous l'avons prise au traitement existait, depuis un

certain temps, déjà sous forme chronique. La liquidité des selles, la décoloration de la face, l'œdème des paupières, l'amaigrissement, la chute des forces en étaient autant d'accidens.

Nous attribuons une grande part dans la guérison à la sévère diète à laquelle le malade a été soumis après la cessation du flux de ventre, et dont nous ne nous sommes relâchés que lorsque la bouffissure de la face a disparu et que la couleur de la peau est devenue plus naturelle.

Obs. VI. — *Entéro-colite aiguë compliquée de péritonite devenue mortelle au bout de 23 jours.* — Mulders, flamand, taille élancée, mince, poitrine étroite, peau blanche et transparente, constitution molle et chétive, était en traitement à l'hôpital, pour un ulcère scrofuleux à la jambe, lorsqu'il fut atteint le 4 novembre, (toutes les salles de l'hôpital étant remplies, on fut forcé d'admettre dans la division des blessés beaucoup de malades arrivés par évacuation de Louvain, le 3 novembre), d'une entéro-colite, dont l'invasion fut accompagnée d'une profonde prostration, abaissement de température, rareté des urines, inertie de la circulation et des selles extrêmement nombreuses et rapprochées.

Après un séjour d'une heure dans un bain chaud, la réaction s'étant rétablie, on applique 25 sangsues sur l'hypogastre et on administre un grain d'extrait d'opium gommeux; les déjections n'ayant pas diminué de nombre et les tranchées étant aussi violentes, on revint le lendemain aux émissions sanguines faites à l'hypogastre et à l'anus, et on continua l'emploi de l'opium, des ventouses scarifiées, des bains de corps; la diète absolue et les mucilagineux joints aux opiacés appaisèrent les douleurs et diminuèrent le nombre des selles, au point que dans la nuit du 10 au 11 il n'en rendit plus que quatre, liées, sans mélange de sang, et qu'il goûta un instant de sommeil.

Dans la nuit du 12 au 13, retour des douleurs, du flux et du tenesme; le ventre est extrêmement sensible à la pression, les urines sont de nouveau supprimées; les selles semblables à de la raclure de boyaux nageant dans de la bierre. (Ventouses scarifiées; deux grains d'extrait d'opium dans six onces de véhicule.)

La diarrhée et la douleur continuant et le pouls s'enfonçant tous les jours davantage, on fit prendre des bains chauds, on posa deux vésicatoires aux mollets, et le 18 un large vésicatoire sur le ventre.

Le 20, la peau était anémique et froide; le vésicatoire n'a presque pas rougi la peau; la langue est pointue, pâle, sèche et raide; l'haleine est glacée; vomissement de tout ce qui est ingéré: vive appétence pour les boissons chaudes ou spiritueuses; pouls imperceptible, ventre rétracté, raison intacte; quelques cuillerées de vin sucré, qui d'abord plaisent au malade, mais provoquent bientôt la plus cruelle cardialgie; le ventre est excessivement douloureux et rétracté; des matières vertes, noires, fétides, coulent incessamment de l'anus; l'introduction de la canule, essayée pour passer des lavemens opiacés, excite de vives douleurs; c'est dans cet état qu'il reste depuis le 22 jusqu'au 27.

Dans les dernières 36 heures, des douleurs atroces qu'aucune embrocation ni fomentation ne peut calmer, occupent le ventre; le malade boudit dans son lit, au milieu des plus affreuses convulsions et des distorsions des membres les plus effrayantes, et ne cesse de vivre que le 27.

Autopsie 24 heures après la mort. — *Habitude extérieure.* — Marasme très-avancé.

Appareil digestif. — L'estomac présente des traces évidentes de phlogose; dans son grand cul-de-sac est une grande plaque oblongue, d'un vert foncé; sa membrane

muqueuse, recouverte par un mucus verdâtre et filant, est ramollie dans une grande étendue; le duodénum est teint en jaune safrané, et l'on remarque d'espace en espace des taches de la largeur d'une pièce de cent sols, d'une couleur brun foncé, au-dessus desquelles la muqueuse s'enlève facilement en râclant avec le dos du scalpel.

L'intestin grêle est phlogosé dans presque toute son étendue; sa rougeur est très-foncée dans certains endroits et plus claire dans d'autres.

Le gros intestin, entièrement désorganisé et ramolli dans toute son épaisseur à un tel point que par la seule traction avec les doigts et sans l'intervention de l'instrument tranchant, on le divise en autant de morceaux qu'on veut, ne présente qu'une longue traînée d'ulcères; il est tout recouvert d'une substance qui exhale une odeur pénétrante de putréfaction semblable à celle de stockfich pourri.

La rate, quoiqu'augmentée deux fois de volume, n'a cependant pas perdu de sa force de cohésion; sa tunique séreuse offre des traces évidentes d'inflammation ancienne.

Le foie est très-friable; sa vésicule est remplie d'une matière noire, poisseuse; les circonvolutions intestinales sont unies ensemble par de fausses membranes denses, résistantes et évidemment d'ancienne formation; le petit bassin contient un peu de liquide couleur de chocolat; le péritoine n'offre aucune résistance.

Voici une entéro-colite nouvelle; car si nous nous en rapportons à la déclaration du malade, jamais avant l'invasion de la présente maladie il n'avait eu de cours de ventre; cependant les émissions sanguines ont été impuissantes pour en arrêter les progrès: soutenue par les mucilagineux, les opiacés, les bains de corps, cette médication en modère bien pour quelques jours la violence, mais bientôt elle envahit toute l'épaisseur de l'intestin et

gagne le péritoine. On sait que le propre du processus phlogistique est de ramollir les tissus où il opère; ce ramollissement se trouve partout et toujours après l'inflammation, mais ici, il a été porté à un degré extrême tel qu'aucun autre cadavre n'en a offert d'exemple. Est-ce à sa violence seule qu'il faut l'attribuer, ou la constitution molle et lâche de l'individu y a-t-elle contribué? L'inflammation gastro-entéritique rivalisait de force avec la colique; les taches vertes et brunes observées dans l'estomac et le duodénum étaient de véritables ecchymoses. C'est la seule maladie où l'agonie ait été si douloureuse et agitée de convulsions; ceux dont la portion inférieure du tube digestif a été seule trouvée malade n'ont dans aucun cas présenté ces symptômes. Il est permis d'en conclure que la modification morbide des centres nerveux supérieurs dépendait de la gastro-duodénite. Nous avons trouvé une série d'ulcères dans les gros intestins. Cette espèce de désorganisation n'a été remarquée en général par les anatomo-pathologistes que dans les cas chroniques. Cependant, d'après Frank, ils s'observent, quoique rarement, dans la dysenterie aiguë. Il faut faire attention, d'ailleurs, qu'ici l'inflammation avait sévi pendant 25 jours avec une intensité variable.

Obs. VII. — *Gastro-entéro-colite aiguë compliquée de pneumonite.* — *Mort.* — Beklots, flamand, âgé de 22 ans, d'une bonne constitution, squelette régulier, n'ayant jamais été malade, était depuis trois semaines en proie à une fièvre quarte, avec tuméfaction de la rate, gagnée dans les polders, lorsqu'il fut évacué sur nous de Louvain, le 21 octobre. La diète, des boissons adoucissantes suffirent pour prévenir les accès du 25 et du 26.

Le 27. Langue rouge, sèche, recroquevillée, peau chaude, pouls fréquent, abattement moral extraordinaire. La face, qui était jaune déjà à l'entrée,

est actuellement bistre, déjections alvines comme des chairs lavées avec lambeaux membraneux et dépôt argileux, très-multipliées, avec ténesme; la région colique est soulevée et douloureuse, et la fosse iliaque gauche remplie en haut par une tumeur large et arrondie qui se termine inférieurement par une espèce de saucisson. (16 sangsues aux régions épigastro-coliques; cataplasmes émolliens, eau d'orge édulcorée). les douleurs ventrales s'appaisent par l'application des sangsues; le moral du malade s'améliore, mais les déjections alvines ne changent ni de quantité, ni de nature. L'acétate de morphine est employé à la dose d'un quart de grain, matin et soir; mais il s'ensuit des envies de vomir qui ne permettent pas d'en continuer l'usage et nous forcent d'en revenir aux boissons adoucissantes. A ces différens symptômes se mêlèrent bientôt ceux d'une véritable phlogose pulmonaire : tussitation, respiration dyspnéique, crépitation dans le poumon droit; crachats visqueux et sanglans. (Ventouses sèches à la poitrine).

Le 5 au soir, reparurent des symptômes de gastrite aiguë : peau chaude et sèche, langue rouge, pointue, raide, envies de vomir; épigastre chaud et tendu, inquiétude extrême; le malade ne reste pas un seul instant dans la même attitude, et pousse de longs et douloureux gémissemens; le teint devient cadavéreux, la face hypocratique, la figure s'hébète, le pouls reste fréquent, mais s'affaiblit de plus en plus. D'innombrables taches lenticulaires violettes apparaissent sur le tronc et les extrémités, la peau se refroidit, le pouls s'efface; tous les symptômes de gangrène intestinale apparaissent, et après une lente agonie, le malade expire sans convulsions le 7 novembre à sept heures du matin.

Autopsie 14 heures après la mort. — Habitude externe. —Odeur pénétrante de putréfaction; demi-marasme.

Appareil respiratoire. — Adhérence du poumon droit sur toutes ses faces par des membranes serrées, engorgement péripneumonique dans son lobe inférieur; le poumon gauche est libre dans toute sa cavité; la muqueuse bronchique est vivement enflammée; du mucus écumeux, sanguinolent sort des poumons par la pression.

Appareil digestif. — Foie très-volumineux, ardoisé, gorgé de sang poisseux, la vésicule du fiel est pleine. La rate, d'un quarré alongé, arrondi par les deux bouts, a plus du double de sa grosseur; son parenchyme est privé de toute cohésion; le grand cul-de-sac de l'estomac est vivement enflammé, il est couvert d'une couche gélatineuse d'un vert safrané. Cette même couleur de safran se remarque par plaques dans l'intestin grêle, et interrompt la couleur rouge et arborisée de la membrane interne de l'intestin dans le cœcum et le colon. On remarque surtout au bord libre de l'intestin, une injection rouge et un développement extraordinaire des cryptes muqueux; la membrane interne est ramollie mais sans être ulcérée. Depuis la région splénique du colon jusqu'au rectum, l'intérieur de l'intestin est complètement sphacélé et répand une odeur de gangrène des plus pénétrantes; il présente une surface bosselée, verdâtre, interrompue par des points blancs; on dirait des épinards hachés avec du pain. L'intestin a, pour le moins, le triple de son épaisseur ordinaire; en coupant les nombreuses fongosités qui donnent à son intérieur l'aspect décrit plus haut, on tombe dans une matière d'un blanc grisâtre nacré, dont la section est parfaitement nette et qui ressemble à du blanc d'œuf, durci par une coction prolongée; en d'autres points, c'est de la substance tuberculeuse déjà ramollie; les glandes mésocoliques sont tuméfiées.

Appareil sensitif interne. — Sérosité limpide dans le tissu cellulaire sous-arachnoidien et dans les ventricules cérébraux.

Voici un entéro-colite contractée à l'hôpital; rien ne nous autorise à dire qu'elle existait avant l'entrée du malade. Il est permis de croire que son habitation prolongée dans les polders et son séjour à l'hôpital de Louvain lui en avaient implanté le germe. L'épaississement des tuniques intestinales et leur métamorphose en substance fongueuse indiquent d'ailleurs un travail phlegmasique lent et ancien dans le gros intestin; dans le grêle ainsi que dans l'estomac existent des traces certaines de phlegmasie aigüe.

Obs. VIII.e — *Entéro-colite chronique repassée à l'état aigu. Mort.* — Keubels, flamand, revenait de la captivité holandaise, lorsqu'en mer il fut saisi de fièvre intermittente endémique; traité et débarrassé de sa fièvre à Anvers, il fut dirigé sur nous, et nous arriva le 21 octobre. Le type était alors tierce, la face du malade était jaune paille, sa maigreur très-avancée, les déjections alvines liquides et fréquentes; trois grains de sulfate de quinine administrés une heure avant l'invasion de l'accès coupèrent la fièvre; des cataplasmes émolliens et du salep à l'intérieur arrêtèrent la diarrhée, et le 25 le malade était debout devant son lit et demandait à manger. Le teint se colore, les chairs se renourrissent, et tout annonçait une convalescence établie, quand dans la nuit du 9 au 10 novembre il lui survint un cours de ventre sanglant avec ténesme; envies continuelles d'aller, expression de quelques lambeaux membraneux et d'un peu de sérum rougeâtre, toutes les demi-heures, collapsus de la face; refroidissement de la peau, suppression des urines, enfoncement du pouls, dessèchement et recroquevillement de la langue, soif vive, tranchées, sensibilité du ventre, froid glacial des pieds; tussitation, (Cataplasmes très-chauds autour des pieds et

sur le ventre, deux demi-lavemens, eau de riz édulcorée, cinq sangsues à l'anus).

Le 11 diminution de la diarrhée, appaisement du tenesme; mais abusant de l'amélioration, le malade s'expose au froid et la diarrhée sanglante revient avec une nouvelle intensité. Le 12 depuis cette époque jusqu'au 17, jour de sa mort, les évacuations sanglantes avec ténesme continuèrent sans relâche. L'opium en extrait donné à la dose de deux grains en solution, dans les 24 heures, provoqua des vomissemens et alluma une soif ardente, le malade exhalait une odeur ammoniacale, pareille à celle de raie pourrie: de petits grumeaux verdâtres, friables, s'échappent incessemment de l'anus, et de petits lavemens d'amidon opiacés, injectés dans l'espoir de modérer la fréquence de ses déjections, sont incontinent repoussés; la peau est froide, le pouls imperceptible, la face livide, cadavéreuse, la langue pâle, raide, pointillée, l'haleine insoutenable, et les facultés intellectuelles s'exercent dans toute leur plénitude. Le malade s'éteint lentement et expire le 17 sans convulsions, sans râle.

Nécropsie 24 heures après la mort. — *Habitude extérieure.* — Squelette bien conformé, appareil musculaire bien développé, mais coloré et maigri.

Appareil digestif. — Inflammation par zônes distinctes du grand épiploon et du péritoine pariétal, aucun épanchement dans le ventre, muqueuse stomachale rosée, plus foncée en couleur le long de la grande courbure; plaques enflammées, séparées d'abord, ensuite continuées dans l'intestin grêle. Les follicules agminés sont tuméfiés, saillans, ulcérés à leur sommet et tranchans. Par ce pointillement blanchâtre sur la muqueuse rouge pourpre et violette, les parois du gros intestin sont considérablement épaissies, son aspect intérieur est bosselé; partout où la muqueuse existe encore, elle est d'un rouge ardent, mais

de longues traînées d'ulcères grisâtres la détruisent dans le sens de la longueur de l'intestin. Dans le rectum, la membrane interne est détruite, et remplacée par un putrilage verdâtre très-fétide. La rate a le double de sa grandeur ordinaire; son parenchyme est d'un brun noirâtre et réduit en bouillie, et festonné sur ses bords.

L'existence de l'entéro-colite chronique se déduit de la liquidité des déjections alvines continuées pendant trop long-temps, mais les selles cependant ne sont pas très-fréquentes; l'impression du froid paraît avoir joué un grand rôle dans l'avivement de l'inflammation; la désorganisation du gros intestin est déjà avancée, moins cependant que nous n'allons la rencontrer dans d'autres malades où la phlogose a sévi encore plus long-temps.

Obs. IX.e — *Entéro-colite chronique repassée à l'état aigu. Mort.* — Déprez, grand et mince, d'un tempérament lymphatique, ayant gagné la diarrhée au camp de Diest, à une époque qu'il ne sait pas préciser, a toujours eu depuis des selles liquides. Admis à l'hôpital de Louvain pour y être traité d'une fièvre tierce, il est évacué sur nous le 19 octobre, et déposé dans mon service le 21.

Le 21, face pâle, jaune, maigreur, peau sèche, appétit vif. L'accès de fièvre qui vient tous les jours à 10 heures du matin céda promptement à l'administration du sulfate donné une demi-heure avant l'invasion de l'accès.

Depuis le 22 jusqu'au 27, état fort satisfaisant, les selles sont liquides, mais peu nombreuses. Placé entre deux malades qui contractent successivement la diarrhée, elle augmente aussi chez lui : les déjections sont fréquentes, rapprochées, peu abondantes, séreuses, le ventre est mou et peu sensible, mais la peau est aride, les urines nulles; sa translation dans une autre salle, une diète absolue et la décoction de salep conjurèrent cette nouvelle atteinte. Cependant le teint reste jaune avec une nuance

ocrée, la langue sèche et rugueuse; la tête lourde et la soif vive. (Eau d'orge édulcorée, bouillie à la farine.)

Dans la nuit du 2 au 3 novembre, rechute sans cause appréciable, déjections fréquentes avec ténesmes, peu abondantes, formées de sérosité rougeâtre et d'un fond pareil à de la terre glaise; avivement de la soif, prostration extrême des forces, retrait du pouls, sensibilité pour le froid, mollesse et sensibilité du ventre. (Eau d'orge édulcorée, deux fois; deux vésicatoires aux cuisses.)

Le 4, face cadavéreuse, langue sèche et noire, paroles traînantes, peau sèche, chaude, pouls filiforme, ventre douloureux, soubresauts des tendons; les déjections alvines continuent sans relâche. Depuis ce jour jusqu'à celui de sa mort qui arriva le 8 novembre, les selles continuèrent à couler. Une atmosphère fétide entourait le malade; l'opium ne fut pas supporté, il excita de suite des nausées et sécha la langue; le quinquina combiné avec l'acide sulfurique, administré par cuillerée, provoqua après l'ingestion de la première dose une cardialgie étouffante et des efforts pour vomir; on fut réduit pour toute indication aux eaux de gomme et de riz à l'intérieur et aux révulsifs. Tout au contraire de ce que nous avons observé chez les autres dysentériques, la jactitation fut extrême, l'agonie longue, accompagnée de vomituritions et de hoquets.

Mort le 8 novembre.

Nécropsie 24 heures après la mort. — *Habitude extérieure.* — Amaigrissement très-avancé, taches gangréneuses étendues aux endroits où les vésicatoires ont été appliqués.

Appareil digestif. — Sérosité rougeâtre peu abondante dans l'abdomen; concrétion gélatineuse rougeâtre sur les deux feuillets du péritoine; phlogose intense de cette séreuse dans la fosse iliaque gauche. Foie très-volumineux, de couleur ardoisée; vésicule du fiel gorgée de bile verte.

La rate, d'un volume double de celui de l'état ordinaire, est d'une forme inégale, marquée sur son bord stomachal par des étranglemens au nombre de quatre; la tunique fibreuse est dans cet endroit blanche et crie sous le scalpel. La muqueuse stomachale est d'un bleu d'ardoise, pointillée en rouge et ramollie jusqu'à consistance de gélatine dans le grand cul-de-sac. L'intestin grêle offre partout des traces d'inflammation et de nombreuses ulcérations, petites, à bords perpendiculaires vers la terminaison. La muqueuse du gros intestin est généralement d'un bleu d'ardoise, avec des entrecoupures violettes. L'S iliaque du colon et le rectum, d'un rouge très-vif, criblés de petites ulcérations à fond grisâtre, ont leurs parois considérablement épaissies.

Appareil sensitif interne. — Dans le tissu cellulaire sous-arachnoïdien existe beaucoup de sérosité. La substance blanche est ponctuée et comme sablée en rouge.

Appareil respiratoire. Les poumons adhèrent sur toutes leurs surfaces.

Appareil circulatoire.—Le cœur, à la grosseur, est une fois et demie plus gros que le poignet du malade; cet aggrandissement est dû à l'hypertrophie du ventricule gauche.

Cette phlegmasie est d'une forme plus décidément chronique, elle s'étend à un espace de temps assez long, n'ayant pour symptômes que la sursécrétion intestinale et le défaut de nutrition. La couleur ardoisée du tube digestif est bien une des phlegmasies chroniques de sa membrane interne; le pointillement rouge est plus particulièrement dû à l'appel fait au sang par un surcroît d'inflammation. Ici de nouveau ce processus a envahi toute l'étendue du cylindre et s'est étendu au péritoine, dans cette partie de la séreuse qui enveloppe l'S romaine de l'iléon, qui était, avec le rectum, le siége principal de la phlegmasie dans cette circonstance.

Il y a eu beaucoup de jactitation et de malaise; et on

trouva une infiltration du tissu cellulaire sous-arachnoïdien et injection des vaisseaux de l'encéphale.

Obs. X.e — *Entero-colite chronique repassée à l'état aigu et compliquée de péritonite. Mort.* — Marlar, 20 ans, bien constitué, d'une constitution très-irritable, blanc-blond, fut évacué sur nous de Louvain le 21 octobre, porteur d'une diarrhée dont il avait été atteint au camp de Diest, et pour laquelle il était en traitement depuis 20 jours. La diète, le repos et les boissons adoucissantes ayant rendu les selles moins fréquentes et appaisé le ténesme, l'appétit étant devenu très-vif et ne se contentant pas de la portion de l'hôpital, le malade se dit guéri et le 6 novembre il quitta l'hôpital.

Il y rentra le 8, dans la journée, dans l'état suivant: traits retombés, face pâle, peau crispée et froide, pouls filiforme, fréquent, déjections alvines incessantes de sang pur mêlé de grumeaux blanchâtres; ces déjections continuaient déjà depuis vingt-quatre heures; urines nulles, efforts pour vomir, tranchées violentes, ventre douloureux à la pression, respiration entrecoupée, courte, haletante, suspirieuse. Appétence pour les boissons chaudes, nuit extrêmement mauvaise, pas une goutte de liquide n'est ingéré sans qu'il ne s'ensuive des vomissemens. (Émulsion huileuse opiacée; cataplasmes émolliens, frictions sur la colonne vertébrale avec un liniment ammoniacal camphré.)

A l'heure de la visite, violens frissons, peau et muqueuse buccale froide. Le malade demande instamment de la boisson chaude. Pouls filiforme; les déjections sanglantes continuent, mais l'envie de vomir et les fortes tranchées sont appaisées. (Cataplasme de moutarde aux pieds.)

Le 10, la nuit s'est passée entièrement sans sommeil à cause de fréquentes déjections dont une grande partie a

coulé sous le malade. Le sang est moins pur, les nausées, les vomituritions et la soif sont calmées.

A la visite, face pâle, retombée, dents et lèvres noires, langue sèche, brunâtre, gercée; peau froide, pouls faible autour de 96. Tranchées fréquentes, ventre sensible. (Cataplasmes émolliens et anodins sur le ventre, décoction de racine de salep ℥ viij, sirop d'althéa ℥ j.)

Au soir, depuis deux heures, les selles sont moins fréquentes, et ne sont plus teintes de sang, il n'y a pas d'amélioration du reste, la jactitation continue, la face mauvaise, la langue sèche, le pouls fréquent, faible; le cataplasme n'a pas été supporté.

Le 11, déjections incessantes, la nuit; à la visite, affaissement profond, voix éteinte, pupilles dilatées, langue pâle, froide, douleurs ventrales atroces; quand elles surviennent, elles paraissent couper la respiration qui devient haute et sonore; le pouls s'accélère en faiblissant, la température reste froide, le ventre est plat, sensible au toucher. (Large vésicatoire sur le ventre).

Le 12, les douleurs produites par le vésicatoire ne permettent pas de le laisser plus d'un quart d'heure en place. La journée et la nuit sont extrêmement fâcheuses; le malade reste la tête renversée en arrière, les paupières affaissées, la voix éteinte, la peau, l'haleine glacées, hoquet fréquent, petites selles d'expression continuelles, vertes, noires, putrides.

A la visite, teint livide, nez pointu, regard éteint, lèvres et dents noires, langue pâle et sèche, atmosphère fétide; le malade réclame instamment du bouillon chaud; ventre rétracté, tranchées fréquentes. (Fomentations de graine de lin tièdes, un grain d'extrait d'opium.) Quand on apporte le bouillon le malade se relève avec précipitation. Il meurt.

Autopsie dix heures après la mort. — *Habitude externe.* — Demi-marasme.

Appareil sensitif interne. — Congestion sanguine des vaisseaux et sinus du cerveau, sérosité abondante dans les ventricules.

Appareil digestif. — Mucosités épaisses et filantes dans l'estomac, teinte rosée interrompue par des vergetures bleuâtres de la muqueuse ventriculaire; péritoine vivement enflammé, ainsi que tout l'intestin grêle. Il n'y a pas une seule partie qui ait la nuance naturelle; de distance en distance, on remarque, dans l'étendue de plusieurs pouces, une teinte pourprée. A trois pieds environ de la terminaison de l'intestin grêle, cette teinte devient générale, les follicules muqueux tuméfiés et saillans sont ulcérés à leur sommet; le gros intestin, dont les parois ont plus de cinq lignes d'épaisseur, est couvert de végétations verruqueuses lichénoïdes, vertes, rugueuses, tellement pressées dans l'S iliaque du colon et le rectum, qu'elle donne à l'intestin l'aspect de l'écorce d'un vieux chêne; les crevasses qui les séparent sont d'un rouge obscur et hérissées de granulations; ces végétations sont formées par un tissu squirrheux dont la section est nette, grisâtre, et pareille à du blanc d'œuf durci par la chaleur.

La promptitude de la mort doit être attribuée à l'excès de la douleur. En effet, depuis son entrée jusqu'à son décès, le malade a toujours été en proie aux plus grandes souffrances. Si elles ont perdu pendant quelques instans de leur atrocité, elles n'ont pas pour cela cessé d'être intolérables. L'abaissement de la température, le tremblottement du pouls qu'on observa pendant tout le cours de la maladie et qui accompagna son début, m'avaient fait penser à une affection morbide du prolongement rachidien, mais l'autopsie ne nous a rien révélé à cet égard.

Obs. XI.e — *Entéro-colite chronique repassée à l'état aigu, méningo-céphalite, péritonite. Mort.* — Bous, flamand, taille haute, mince, cheveux blonds, peau blanche, est évacué de Louvain le 19, et déposé dans mon service le 25 octobre. Attaqué de la diarrhée au camp, il en a été traité pendant quinze jours à l'hôpital de Louvain, et en était à peu près guéri, quand il a été désigné pour l'évacuation; mais en route, elle est revenue avec une nouvelle violence. Depuis quatre jours qu'il est arrivé à Namur, il a été traité par le salep. J'observe : amaigrissement avancé, peau pâle et crispée, plutôt froide que chaude, langue pâle, molle, forte altération, pouls petit et profond. Ventre indolore à la pression; tension et proéminence de tout le trajet du colon; les déjections sont continuelles, sanglantes, mêlées de lambeaux membraneux. Pendant la nuit, le malade ne quitte pas un instant la chaise percée. (Décoction de salep avec teinture d'opium.) Cette médication a pour premier résultat la diminution des selles, le réchauffement de la peau, l'élargissement du pouls; on y persévère.

Le 3 octobre, retour de la diarrhée avec une nouvelle force, sans qu'on puisse présumer à quelle cause la récrudescence est due. Idées confuses, regard fixe, paroles paresseuses, tendance au sommeil, la langue est couverte d'un enduit verdâtre. La soif est très-vive quoique la langue soit pâle.

Le 2 novembre, vomissement érugineux pendant la nuit. A la visite, somnolence, regard hébété, pupilles très-contractées, langue sèche, couverte d'un enduit vert-pré; hoquet par intervalles, pouls presqu'imperceptible.

Les pulsations se confondent; le malade laisse aller ses selles sous lui, et est entouré d'une atmosphère des plus fétides, ventre retombé sensible. Le plus léger attouchement de cette partie réveille le malade de sa stupeur, respiration lente et profonde. (Deux larges vésicatoires aux mollets.)

Au soir, les envies de vomir ont été fréquentes, et quelques gorgées porracées rejetées après les plus violens efforts. A la visite, face fatiguée, paupières pesantes, sentiment profond d'anéantissement; facultés intellectuelles confuses, relâchement musculaire excessif. Les membres retombent par leur propre poids, langue sèche, verte; pouls filiforme, atmosphère ammoniacale, ventre plat, tendu, sensible. (Vin sucré.)

Le 3 novembre, face cadavéreuse, peau froide, pouls imperceptible, respiration lente, agonie; les vésicatoires ont à peine rougi la peau.

Mort à cinq heures du soir, le 3 novembre.

Autopsie le lendemain à une heure. — A l'ouverture de l'abdomen, il se répand dans l'appartement la puanteur la plus pénétrante.

Organes abdominaux. — Péritoine épaissi, opaque, estomac rempli d'un liquide verdâtre, la membrane muqueuse est recouverte d'une couche épaisse de mucus vert et collant. Toutes les valvules conniventes de l'intestin grêle sont enduites d'une matière semblable, mais plus foncée en couleur. Le fond de l'intestin est d'un rouge bleuâtre qui devient violet aux approches de la valvule iléo-cœcale. Le colon ascendant, le descendant jusqu'à l'anus, sont sphacélés, d'un vert noirâtre; le transverse est d'un rouge vif, carnifié et parsemé d'innombrables ulcères, les tuniques intestinales sont doublées d'épaisseur.

Appareil sensitif interne. — Les sinus de la dure-mère et le système veineux de l'encéphale sont gorgés de sang noir; infiltration séreuse très-abondante du tissu cellulaire sous-arachnoïdien; adhérences multipliées des deux feuil-

lets de l'arachnoïde, tantôt par des brides assez résistantes, tantôt par des membranes à l'état de gélatine, à la hauteur du pressoir d'Hérophile, la méninginе est considérablement épaissie et d'une dureté cartilagineuse. Le corps calleux est tellement ramolli que, par la séparation spontanée des deux hémisphères, il se déchire par le milieu et laisse le quatrième ventricule à nu. Sérosité abondante dans les ventricules cérébraux.

Appareil circulatoire. — Le péricarde est rempli de sérosité, le sang est fort ténu; quelques petites concrétions blanches, mollasses, très-faibles, se montrent dans les cavités droites du cœur.

On trouve ici des désordres nombreux tous dus au processus de l'inflammation. Le point de départ cependant est le tube digestif; la phlogose dont il était le siége au camp, a perdu fréquemment son caractère d'acuité par une médication appropriée, mais ne me paraît pas avoir jamais été entièrement guérie : existant toujours sous forme chronique, latente, elle est remontée à l'état aigu, dès qu'un surcroît de stimulation a retenti soit directement, soit indirectement dans les organes surexcités. Aussi voit-on le malade faire de fréquentes rechutes. On sait que la répétition des phlogoses dans les organes leur donne une grande prédisposition à les contracter. Le chagrin dont le malade était atteint et que nourrissait sans aucun doute l'entéro-colite chronique, entretenait dans l'encéphale une irritation dont l'existence a été reconnue pendant la vie et les traces retrouvées après la mort. L'épaississement du péritoine paraît indiquer que là aussi la phlegmasie avait longtemps existé, ce n'est cependant que dans les derniers instans de la vie qu'elle s'est fait reconnaître à nous.

Obs. XII.e — *Entéro-colite chronique, repassée à l'état aigu, compliquée de pneumonite et péritonite*

chroniques. — *Mort.* — Débale, flamand, âgé de 19 ans, petit, fluet, blanc, est évacué de Louvain sur nous, le 21 octobre. Il était atteint de diarrhée et de toux : peau sale, terreuse; urines rares; maigreur très-décidée, langue plate, molle; ventre indolore; voracité extrême. La diète et la décoction de salep, ensuite l'opium continué pendant quelques jours, arrêtent la diarrhée ou la modèrent au moins de manière à la rendre supportable. Mais la toux continue; le malade importune sans cesse pour avoir des alimens, ce nonobstant on ne lui accorde que du riz et d'autres féculens : atteint de la gale, il est transféré dans la salle particulièrement destinée au traitement de ce genre de maladie; mais avant la guérison, qui cependant n'a été poursuivie que par des bains et des lotions hydro-sulfuriques, l'inflammation entéro-colique se réveille avec violence, et plus de deux cents déjections très petites, formées par une sérosité rougeâtre, écumeuse, sont rejetées pendant les vingt-quatre heures; suppression des urines. La figure s'enfonce et la peau est d'un froid glacial. L'immersion dans un bain chaud rappelle la chaleur et un peu de coloration des lèvres.

Le nombre des déjections se réduit, et bientôt ce n'est plus qu'un ténesme pendant lequel sont exprimées quelques gouttes de sang ténu, fétide; la langue est pointue, rouge et sèche; tussitation fréquente. (Deux lavemens de décoction de pavot; décoction de salep à l'intérieur.)

Le 13, le nombre des selles est notablement diminué, mais la face reste sinistre; la peau est chaude, sèche, âpre au toucher; le pouls vif, bat plus de 100; la langue est rouge, pointue. (Décoction de salep deux fois; bain de corps.) Les déjections recommencent à être fréquentes, rapprochées, avec ténesmes, sanglantes ou noires; la toux est sèche, très-fatiguante, surtout pendant la nuit; la langue reste pointue, rouge, sèche. Le marasme

fait des progrès rapides ; la face se creuse ; les yeux se cavent ; le pouls s'efface, et le malade s'éteint le 17 novembre, après une très-courte et tranquille agonie.

Autopsie vingt heures après la mort. — Habitude extérieure. — Dernier degré de marasme, œdème autour des malléoles.

Escharre profonde au grand trochanter droit, taches bleuâtres autour du cou et sur les extrémités. L'abdomen est concave, son intérieur contient environ deux pintes d'un liquide citrin, collant aux doigts ; le tube digestif est diminué d'ampleur dans toute son étendue ; à l'extérieur il s'est enveloppé d'une fausse membrane comme gélatineuse, contenant dans ses vacuoles un liquide semblable à celui qui est libre dans la cavité péritonéale. Les glandes du mésentère sont tuméfiées, tuberculeuses ; la muqueuse stomachale est vivement injectée et rosée dans toute son étendue. Celle de l'intestin grêle ardoisée et recouverte de mucosités épaisses et collantes ; près de la valvule iléo-cœcale, existe un seul ulcère rond à bords perpendiculaires, à fond grisâtre, de la largeur d'une pièce de vingt sols. Les parois du gros intestin sont très-irrégulièrement épaissies et altérées. L'aspect général est bosselé, d'une couleur de bœuf lavé ; de distance en distance, et notamment dans la région splénique et l'S iliaque du colon, les bosselures sont plus élevées ; leur sommet est criblé d'ulcères et leur surface est d'un vert grisâtre ; le rectum est également criblé de petits ulcères innombrables, et présente un aspect rugueux. Le foie est énorme, mais sans altération. La rate fort petite, d'un bleu verdâtre ; sa tunique externe offre mille rides. La muqueuse trachéo-bronchique est vivement injectée ; le parenchyme pulmonaire est compacte, dense, mais privé de cohésion. Le poumon droit adhère sur toutes ses faces, des milliers de tubercules miliaires se remarquent dans son parenchyme ; il y en a d'innombrables à la surface,

immédiatement sous la plèvre, qu'ils soulèvent de manière à rendre le toucher rugueux. Dans le poumon gauche, qui n'est pas adhérent, il existe de même un grand nombre de tubercules miliaires, mais on n'en rencontre pas immédiatement sous la plèvre.

Il ne paraît guère possible de méconnaître ici la transition répétée de l'entéro-colite chronique à l'acuité et la compression de celle-ci par la diète, le repos, les féculens et l'opium. C'est tout particulièrement le gros intestin qui souffre ; aussi l'estomac étant libre d'irritation, la faim est-elle dévorante et cette sensation fait-elle taire toutes les autres. Quand le malade est arrivé sous mon traitement, la profondeur de l'atteinte portée aux actions nutritives, et dont l'aspect terreux de la peau, la faiblesse, la maigreur et l'abaissement de la température étaient les principaux symptômes, ne me peignaient que trop celle de l'altération organique dont les appareils gastro-pulmonaires étaient le théâtre, et ne me laissaient pas beaucoup d'espoir de guérison. Il ne serait pas impossible que l'appaisement de l'irritation psorique qui, en vertu des rapports d'antagonisme bien connus de la peau et de la muqueuse colique, peut avoir agi comme révulsif, ait contribué à faire prédominer de rechef l'irritation dans le gros intestin ; il est à remarquer que dans ce cas, le bain chaud, en stimulant la surface tégumentaire et y rappelant de la chaleur, a diminué le nombre des selles. Dans les derniers jours de la vie, la peau s'est échauffée ; elle est devenue sèche, âcre ; la langue a rougi, s'est effilée et séchée, et l'estomac a offert des caractères évidens d'inflammation aiguë. La complication de péritonite et de pleuro-pneumonite ne peut être contestée. Elle n'a pas décidé la mort sans doute, mais en a probablement hâté le terme.

Obs. XIII. *Entéro-colite chronique avec inflamma-*

tion du péritoine et de l'arachnoide; Désorganisation du foie et nombreuses ecchymoses.—Mort. — Au nombre des évacués de l'hôpital de Louvain, le 19 octobre, se trouvait le nommé Keclar, flamand, long, mince, blanc; poitrine très-plate et très-étroite; il est porteur d'un abcès à la main gauche; déposé d'abord dans la division des blessés, il est transféré, le 23, dans celle des fiévreux. A ma visite, j'appris de lui qu'il traînait depuis long-temps dans les hôpitaux, ne pouvant se débarrasser de la diarrhée; je remarque qu'il est fort maigre; que son teint est d'un jaune pâle, mêlé d'une couleur de rouille ferrugineuse. La prostration musculaire est telle que, malgré l'intégrité des facultés intellectuelles, le malade ne peut supporter, même pendant un instant, aucune station.

Peau sèche et crispée; autour de l'ombilic existent plusieurs ecchymoses de la largeur d'une pièce de 40 sous, chaudes, molles, conservant l'impression du doigt; langue rouge, rugueuse, très-sèche; hoquets par intervalles, toux fréquente, excitant de la douleur tout le long des attaches du diaphragme; épigastre soulevé, tendu, dur, chaud, très-sensible à la pression; pouls très petit, très-fréquent; les selles ne laissent aucun relâche, elles sont vertes, accompagnées de ténesme; l'atmosphère du malade est d'une odeur douceâtre, très-pénétrante, et excessivement nauséeuse. (Lavement d'amidon opiacé; décoction de salep avec un gros de teinture d'opium sur 8 onces).

Je suis forcé de renoncer sur le champ à l'opium qui excite des envies de vomir et dessèche la langue, sans diminuer le nombre des déjections.

Le 24, nuit très-mauvaise; gémissemens sourds, les douleurs sont spécialement fixées dans la fosse iliaque gauche; décubitus en supination; cuisses rétractées; de la plaie placée au dos de la main s'échappe un pus sanieux, noirâtre, à odeur de gangrène.

Le 25, escharres gangréneuses entourées d'une aréole rosée sur l'index et le dos de la main gauche.

Le 26, maculatures violettes, dures, élevées au-dessus de la peau sur tout le tronc, existant en quantité innombrable sur la tête de la verge et le scrotum. Elles envahissent successivement les extrémités, le cou, la face et la cavité buccale. On essaie l'opium sous diverses formes; mais à l'intérieur il n'est jamais supporté. Les boissons acidulées, tant végétales que minérales, provoquent de l'angoisse et du vomissement; la seule solution de jus de réglisse est supportée.

Le 28, la jactitation est telle que le malade ne peut demeurer un seul instant en place. La respiration est courte, dyspnéique, ce qui rend l'exercice de la parole très difficile.

Gémissemens plaintifs, petite toux sèche, dysphagie; le malade demande à manger, à boire du vin, et cependant quelques cuillerées de vin doux excitent la plus cruelle gêne. Le malaise devient extrême; le pouls tremblote et s'efface par la plus légère pression. Il n'existe pas de délire; le malade apprécie toute l'horreur de sa position, se plaint de douleurs intolérables, mais sans pouvoir désigner aucun endroit précis où elles sont fixées ou prédominantes.

Dans la soirée du 29, il commence à délirer, perd peu à peu connaissance et meurt vers deux heures du matin.

Nécropsie 24 heures après la mort. — *Habitude externe.* — Toutes les maculatures signalées pendant la vie sont actuellement des élevures blanches de forme inégale, dures, portant au centre une croûte brunâtre, déprimée. L'abcès placé sur le dos de la main est rempli d'une matière noire, putrilagineuse, gangrénée; les ecchymoses circa-ombilicales existent dans le tissu cellulaire sous-dermoïde injecté, et se laissent écraser sous les doigts.

Appareil sensitif interne. — Infiltration séreuse du tissu cellulaire sous-arachnoïdien; épanchement de même nature dans les ventricules ; engorgement sanguin des vaisseaux encéphaliques.

Appareil respiratoire.—Les deux poumons sont tuberculeux. C'est dans les lobes supérieurs surtout que l'infiltration de la matière tuberculeuse et sa réunion en masse s'observent. Les poumons sont adhérens et les plèvres soulevées à leur surface costale par de petits dépôts de matières blanches comme plâtrées. La muqueuse bronchique est violette et présente de nombreuses ulcérations.

Appareil digestif. — Le foie est d'un volume énorme, l'hypertrophie s'étend aux trois lobes à la fois; il occupe les deux hypochondres, la région épigastro-ombilicale et toute la fosse iliaque droite, et refoule en bas et à gauche tout le paquet intestinal ; il adhère fortement par une membrane serrée, courte et étendue, au diaphragme et aux parois ventrales; sa force de cohésion est tellement diminuée qu'il se déchire pendant l'opération de l'extraction ; son parenchyme est décoloré, granuleux ; et, pressé entre les doigts, il s'écrase et se réduit en pulpe avec la plus grande facilité.

Le tube digestif offre dans toute son étendue des traces évidentes de phlogose. La muqueuse buccale et pharyngienne est semée de boutons durs, varioliformes et déprimés au centre. Autour du cardia la rougeur est intense; l'estomac est criblé de tachetures rouges vers le centre desquelles rayonnent des vaisseaux sanguins.

Les valvules conniventes sont saillantes, teintes en jaune safrané; la muqueuse est injectée, tantôt par arborisation, tantôt par suffusion, comme si la couleur avait été étendue avec un pinceau. Les plaques des glandes agminées se présentent comme des lentilles blanches et molasses sur un fond bleuâtre.

A un pied et demi environ au-dessus de la valvule de *Bauhin* on remarque les premières ulcérations, comme si elles avaient été faites avec un emporte-pièce, à fond grisâtre, et formées aux dépens de la membrane muqueuse. Près et autour de la valvule existe une ulcération de plus d'un pouce de hauteur, grise, rugueuse, à bords découpés et durs. Les tuniques du gros intestin sont considérablement épaissies. Dans le cœcum, le fond est grisâtre, semé d'une innombrable quantité de granulations rougeâtres et abreuvées par une bouillie purulente; détritus de la membrane muqueuse. Plus loin, l'intestin est bosselé, d'un rouge vif; la masse folliculeuse s'offre sous forme de mamelons rouges, carnifiés, déprimés en godet au centre, dont les intervalles sont remplis par la même bouillie purulente.

Dans l'S iliaque du colon, cette métamorphose est surtout remarquable; ce n'est qu'un amas de fongosités ou de porreaux d'un rouge foncé, mollasses et saignans. Le rectum est verdâtre, rugueux.

Ici la muqueuse de tout le tube digestif, depuis la bouche jusqu'à l'anus, était malade, et sous ce rapport la dénomination d'entérocolite placée en tête de l'observation est sans doute inexacte ou du moins incomplète; mais outre que nous ne connaissons pas en langue médicale de mot pour désigner la phlogose de toutes les divisions du canal alimentaire, il me paraîtrait peu rationnel de contester à la muqueuse du gros intestin et de la fin de l'iléon d'avoir été le point de départ d'une phlegmasie, qui, par son extension successive à d'autres organes, a imprimé plus tard à la maladie la forme compliquée sous laquelle on vient de la voir figurer.

Il n'entre pas dans notre plan de faire l'analyse des symptômes et de les rattacher chacun à la lésion organique dont ils étaient les représentans. Je ne ferai qu'une seule

remarque; elle a trait à l'état cacochyme du malade, état auquel s'attribuent les escarres gangréneuses, les maculations, les ecchymoses dont il était porteur tant en dedans qu'en dehors, et cette tendance à la décomposition, dont pendant la vie on a pu constater l'existence.

Ce n'est pas que j'attribue cette cacochymie à l'asthénie générale, et ces maculations et ces ecchymoses à l'atonie des vaisseaux qui, distendus par le sang, le laissent échapper par l'écartement en quelque sorte mécanique de leurs molécules. Je les crois dues, au contraire, à une véritable exhalation active, opérée sous l'empire d'un travail irritatif.

Voyez vers les tachetures rouges de l'estomac rayonner des vaisseaux sanguins injectés; remarquez les aréoles dont sont entourées les escharres, la congestion du tissu cellulaire sous-cutané où sont déposées les ecchymoses.

La disparition, après la mort, de la couleur rouge des maculatures ainsi que des pustules cutanées, est un indice irrécusable pour moi que cette couleur était due à un mouvement fluxionnaire, à un appel fait au sang par l'irritation, car celle-ci étant une modification de l'état de vie, ses effets ont dû nécessairement cesser avec son extinction; mais, tout en admettant pour cause de la congestion des parties un surcroît de vitalité, je n'en pense pas moins que si la crâse des humeurs n'avait pas été imparfaite et leur force de plasticité diminuée, il n'y aurait pas eu dans les tissus phlogosés cette tendance à une rapide décomposition. Toutefois cette dyscrasie des humeurs, cette dépravation du jeu de leurs affinités vitales n'est pas un fait primitif, spontané, indépendant: il est subordonné à la longue phlogose dont le tube digestif et l'appareil pulmonaire avaient été le siége.

On sait combien, en général, dans les inflammations aiguës de l'appareil digestif, le travail de la chylose est

entravé. On conçoit qu'il doit en être plus particulièrement ainsi quand elles ont leur siége dans le colon, puisqu'un des caractères des colites aiguës est la précipitation brusque et soudaine des alimens aussitôt après leur ingestion et au milieu d'une abondante sécrétion muqueuse ou sanglante, et qu'il doit être tout-à fait nul, ou du moins extrêmement incomplet. Quand tout le canal alimentaire est enflammé, comme nous en avons ici un exemple, du trouble de la chylose, combiné avec la perturbation que produit dans l'économie toute inflammation violente ou étendue, résulte inévitablement la langueur de la nutrition générale, l'imperfection de la réparation des solides, et par suite leur impuissance à résister aux effets du processus phlogistique, dont il est de l'essence de tendre à la désorganisation.

La dégénérescence si avancée du foie, dont l'importance dans le travail de la chylose a été si bien appréciée par MM. Tiedemann et Gmelin, a sans doute puissamment contribué à l'altération humorale. Il faut en dire autant de la désorganisation du poumon qui a dû rendre l'aération du sang très-incomplète.

Voilà du moins comme j'entends les maladies des humeurs. Très-rarement idiopathiques, et cela dans les seuls cas où ces humeurs servent de véhicule à un principe miasmatique ou vénéneux, elles surviennent toujours secondairement à une affection morbide des solides. Altérées alors dans leur mixture ou dans leur cohésion, ces humeurs perdent la faculté de stimuler convenablement les solides et d'exciter en eux les mouvemens nécessaires à l'accomplissement régulier de leurs fonctions. Mal élaborées par suite d'une irritation dont les solides étaient le siége, elles deviennent à leur tour causes de nouvelles irritations.

Voilà le cercle dans lequel tournent les actions pathologiques. *Consensus unus, consentientia omnia.*

Obs. XIV^e^ — *Entero-colite chronique, anasarque. Mort.* — Devoder, flamand, mince, élancé, blond, lymphatique, arriva de Louvain le 19 octobre, en proie depuis seize jours à la diarrhée; face pâle, jaune, mélangée d'ôcre, peau sèche, rude, ponctuée sur le corps; langue pâle, pointue. Sept à huit selles liquides en partie stercorales, en partie glaireuses dans les vingt-quatre heures; urines rares, tumeur molle, volumineuse dans l'hypocondre gauche, appétit très-énergique.

Pendant sept semaines qu'il vécut encore à l'hôpital, il ne quitta jamais son lit que pour satisfaire à ses besoins; fort sensible au froid, il se tenait tous les jours ramassé sur lui-même, les cuisses fléchies sur le tronc, et enfoncé sous ses couvertures. Quand les selles devenaient plus nombreuses, on y opposait quelques gouttes de teinture d'opium : c'est le seul des remèdes essayés qui ait eu des effets constans.

Le ratanhia, le columbo, l'arnica et la thériaque, semblaient plutôt augmenter le flux de ventre que de le comprimer.

Pendant tout le temps de son séjour parmi nous, la peau fut toujours aride et rugueuse, les urines rares; il s'infiltra peu à peu, et le pouls allant toujours en s'affaiblissant, il mourut le 24 décembre.

Nécropsie, le 25, 26 heures après la mort. — *Appareil sensitif interne.* — Sérosité limpide dans les mailles de la pie-mère, des cavités arachnoïdiennes du cerveau et de son prolongement, substance nerveuse saine.

Appareil respiratoire. — Sérosité dans les deux plèvres sans altération des séreuses; innombrables tubercules miliaires dans les deux poumons.

Appareil circulatoire. — Péricarde distendu par de la sérosité limpide, cœur flasque, décoloré.

Appareil digestif. — Sérosité limpide, citrine, dans

l'abdomen, enveloppe gélatineuse autour du tube digestif, surtout dans sa portion grosse. Parois intestinales épaissies, celles du gros intestin ont plus de quatre lignes; muqueuse stomacale pâle, semée de points grisâtres; celle de l'iléon est ardoisée. A sa terminaison elle rougit et présente de nombreux ulcères. Colon hérissé de distance en distance de porreaux ulcérés à leur sommet, grisâtres, nacrés à leur section.

L'S romaine du colon et le rectum sont rugueux et enduits d'une sanie sanguinolente; le foie est comme cendré, grumeux; la bile cystique décolorée, semblable à de l'eau de laitue; la rate a plus de trois fois son volume; elle descend jusqu'au niveau de l'ombilic. Sa membrane externe est ridée; son parenchyme semblable à du pain trempé dans du gros vin.

Cette maladie m'a semblé remarquable par la profonde langueur dont étaient frappés les actes de la vie de relation. Sans cesse enterré sous ses couvertures, indifférent à tout ce qui se passait autour de lui, insensible aux paroles de consolation qu'on lui adressait et aux soins empressés dont il était l'objet, il traînait une vie végétative. Peut-être la compression exercée sur le cerveau par la congestion séreuse a-t-elle contribué à émousser les sensations. Celle du besoin de l'alimentation était cependant vivement sentie pendant tout le temps du séjour du malade à l'hôpital; les contractions du cœur furent lentes, molles, à peine sensibles au stéthoscope.

Pendant que la prostration musculaire, la lenteur et l'inertie des mouvemens du cœur, et l'affaiblissement des opérations intellectuelles pouvaient dépendre d'un défaut d'excitation gastrique, j'employai quelques amers, le vin, la thériaque; mais je n'eus pas lieu de m'en applaudir. Il est possible que, glissant inaltérées au travers d'un estomac débilité, ces substances venaient en contact avec

la muqueuse *entéro-colique phlegmasiée*, et que de là ait dépendu l'augmentation des selles observées après leur ingestion. J'attribue l'hydropisie générale à la cessation des actes dépurateurs confiés à la peau et aux reins. La péritonite lente et obscure, dont l'extravasation abdominale et les formations gélatineuses péri-entériques on fait reconnaître l'existence, me paraît moins avoir été cause de la suppression de ces travaux excréteurs, que l'effet de l'action supplétive à laquelle a été forcée la séreuse abdominale déjà surexcitée par l'extension de la phlogose muqueuse. Remarquez bien que la couche pseudo-membraneuse était plus développée autour du gros intestin qu'ailleurs.

Obs. XV^e^ — *Entero-colite chronique, excavation tuberculeuse des poumons; dégénérescence squireuse du gros intestin.*

Herings, flamand, taille moyenne, mince, blond, lymphatique, arriva de Louvain par évacuation le 19 octobre. Étant au vingt-septième jour d'une diarrhée gagnée au camp, qui, sanglante d'abord et tormineuse, était actuellement indolore et paraissait peu inquiéter le malade. Il n'a en effet que quatre à cinq selles liquides la nuit; le jour il n'en ressent aucune incommodité. Cependant sa maigreur est extrême, sa figure a une expression marquée de souffrance, son teint paille, mêlé de rouille, sa peau sèche, muqueuse, rude, l'appétit fort vif.

J'emploie les moyens recommandés par les auteurs contre la diarrhée chronique, mais sans succès. Le salep et l'opium diminuent bien le nombre des selles, mais n'en épaississent pas la consistance. Avec cela il tousse, mais crache peu; cependant le stéthoscope fait reconnaître une pectoriloquie retentissante dans la fosse susépineuse gauche, et peu de temps après, une autre avec râle bouillonnant sous la clavicule du même côté. Le flux

de ventre est tantôt plus, tantôt moins abondant, tantôt indolore, tantôt avec ténesmes. L'opium gommeux en solution et un lavement, la décoction de salep avec la teinture thébaïque, sont les seuls moyens par lesquels nous parvenons à adoucir un peu ses souffrances: encore faut-il souvent en suspendre l'emploi à cause des nausées qu'ils excitent. Le malade succombe dans le dernier degré de marasme, le 24 novembre.

Nécropsie 27 heures après la mort. — *Appareil respiratoire.* — Adhérence intime et très-ancienne du poumon gauche, sur toutes ses faces; plusieurs excavations tapissées intérieurement d'une membrane fibro-cartilagineuse, sèche et luisante dans son parenchyme, qui est du reste criblée de tubercules et privée de toute cohésion; le droit en contient également beaucoup, mais crépite par la pression.

Appareil digestif. — Coarctation notable et épaississement peu commun du tube alimentaire: l'estomac n'est pas plus gros que d'ordinaire le cœcum; sa membrane interne est comme gélatineuse. Des milliers de petits ulcères dévorent la muqueuse de l'iléon; autour de la valvule iléo-cœcale de larges ulcérations. Le gros intestin est dans ses deux portions verticales d'un vert foncé, ponctué en blanc et en rouge, on dirait d'épinards hâchés avec du blanc d'œuf et de la viande; au milieu on remarque de larges ulcères placés au sommet d'espèces de verrues à base large et à coupe nette et nacrée. L'arc du colon est d'un rouge vif, on y reconnaît facilement l'existence de la membrane muqueuse, mais elle est, comme un crible, percée d'innombrables ulcères à fond lardacé. La vessie est contractée et vide.

Obs. XVI^e^. — *Entero-colite chronique, dégénérescence cancéreuse de l'intestin.* — Braf faisait partie de l'évacuation du 19 octobre; il succomba à la dysenterie

chronique le 29 novembre, après avoir souffert horriblement de douleurs ventrales et de vomissemens que l'opium, loin de calmer, exaspérait, et conservant jusqu'au dernier moment sa présence d'esprit, alors que tout mouvement du cœur était imperceptible, même au stéthoscope, que la langue, la peau, l'haleine étaient glacées. Il offrit à la section, des signes évidens de péritonite aiguë, et, avec cette même couleur granitique que je viens de décrire ci-dessus, qui, cette fois, occupait toute l'étendue du gros intestin, une dégénérescence telle des parois intestinales, qu'il m'a été impossible d'y reconnaître aucune couche, aucune tunique. Le tout aurait assez bien représenté une couenne, si de nombreux petits amas de matière encéphaloïde, tuberculeuse, n'avaient été déposés dans son intérieur.

Je n'ai que peu de mots à dire sur ces deux derniers cas; ils ont cela de commun que, dans l'un comme dans l'autre, l'entéro-colite avait existé long-temps à l'état chronique et, en entretenant une nutrition anormale dans les parois intestinales, les avait considérablement épaissies et déformées, au point de les rendre méconnaissables. Dans le premier cas cependant, la muqueuse colique n'était pas entièrement détruite, dans le second elle était convertie en entier en putrilage. Cette dernière désorganisation, je l'attribue à l'exacerbation de l'état inflammatoire, à sa réascension à l'état aigu dans un tissu déjà ramolli et privé d'avance de sa force d'association organique par la persistance de la phlogose. C'est encore, suivant moi, une inflammation chronique remontée à l'acuité. Elle n'a duré que peu d'instans, parce que, déjà l'excitabilité était tellement épuisée, la somme des forces réduite à un tel point, qu'une courte récrudescence a suffi pour l'anéantir totalement. Chez d'autres, où la mort arriva plus lentement, nous rencontrâmes des

espèces de bourrelets ou anneaux comme fibreux, placés de distance en distance dans le gros intestin dont ils rétrécissaient considérablement l'aire; ils étaient formés par une matière d'aspect squirrheux, sécrétée dans le tissu cellulaire sous-muqueux qu'ils soutenaient : au sommet de la proéminence circulaire à laquelle cette désorganisation donnait lieu, la muqueuse était détruite par les ulcérations.

Obs. XVII^e^ — *Entero-colite chronique repassée à l'état aigu, exhalation de sang très-abondante dans le canal intestinal. Mort.* — Verkleven fut évacué sur nous de Louvain le 3 novembre; il était petit, maigre, pâle et infiltré, en proie à la diarrhée, à son dire, depuis plusieurs semaines. A son arrivée il n'a guère que quatre à six selles dans les vingt-quatre heures, mais toujours liquides et accompagnées de ténesme. Il est d'une inconcevable apathie, et sans l'active surveillance des infirmiers et leurs incessantes exhortations pour qu'il ne s'oubliât pas, il laissait aller toutes ses déjections sous lui. Sa peau est sèche, rapeuse, ses urines rares, son pouls lent, faible, petit, son appétit peu prononcé. Le salep opiacé réduit le nombre des selles et en augmente la consistance; le pouls se développe un peu, mais le moral reste mauvais, et la peau et les reins sans action. J'emploie les frictions sèches, l'acétate d'ammoniaque en combinaison avec l'extrait de chiendent. La peau semble un peu s'assouplir et la sécrétion urinaire augmenter. Mais cette amélioration est de courte durée. Le 4 décembre, colique violente, ténesme, flux de ventre séreux, sanglant, prostration musculaire et intellectuelle extrême (salep opiacé, cataplasme); diminution des douleurs et de la diarrhée, mais persistance du ténesme; le pourtour de l'anus est rouge, excorié et extrêmement douloureux. Le 9 évacuation copieuse de sang par l'anus, après de violentes coli-

ques, profond anéantissement, froideur glacée du corps, imperceptibilité du pouls. Sinapismes, vésicatoires aux mollets, vin sucré, réaction légère et courte, douleurs de ventre atroces, gémissemens sourds, peau marbrée, nez pointu, regard terne, paroles lentes, confuses, odeur cadavéreuse très-prononcée. C'est ainsi que le malade reste jusqu'au 12 qu'il meurt.

Nécropsie 19 heures après la mort. — Toute la partie postérieure du corps est d'un bleu noirâtre dû à l'infiltration du sang dans le tissu cellulaire sous-cutané. La peau du reste du corps est marbrée, veinée, vergetée en bleu.

Appareils respiratoire et circulatoire : poumons flasques, peu crépitans, engorgés de sang noir, liquide; caillots jaunâtres, gélatineux, emplissant l'oreillette et le ventricule droit; cœur décoloré et mou; sérosité copieuse dans le péricarde.

Appareil digestif et annexes : Estomac petit, rétréci, muqueuse pâle, intestin grêle inaltéré jusqu'à quatre pouces au-dessus de la valvule iléo-cœcale. Là, brusquement et sans gradation, la muqueuse prend dans tout son pourtour une teinte rouge qui se continue jusqu'au rectum, et qui est due à une exhalation de sang épais, visqueux. Cette teinte est tellement uniforme, qu'on dirait que c'est avec un pinceau qu'on l'a étendue. Dans le rectum elle se fonce davantage et passe au noir. La surface iléo-cœcale est lisse, celle du rectum est grumeleuse; le simple lavage n'enlèverait pas la couleur rouge; on a mis macérer les parties qui en étaient enduites. Après un séjour de quelques heures dans l'eau, qui a profondément rougi, le tronçon intestinal a présenté les phénomènes suivans : parois considérablement épaissies, muqueuse gonflée et injectée, bosselée, criblée de petits ulcères qui, par leur union, constituaient dans le colon

des traînées dirigées dans tous les sens et circonscrivant des mamelons rougeâtres de forme et d'étendue inégales qui lui donnent l'aspect bosselé.

Le tissu cellulaire sous-muqueux est gonflé, ramolli; on sépare aisément, et par larges lambeaux, la tunique interne de la musculeuse; dans le colon descendant, le tissu sous-muqueux est épaissi, condensé, transformé en matière semblable à l'albumine cuite. Dans le rectum la muqueuse est détruite; les granulations rouges qu'on y remarque semblent être les follicules enflammés qui n'ont pas encore subi la destruction.

Voilà une maladie observée par nous dès le commencement à l'état chronique, remontée ensuite à l'acuité, et terminée par une hémorrhagie en disproportion avec les forces du malade, et qui a été promptement suivie de la mort. L'excrétion de sang ne ressemblait pas du tout à ce qu'on remarquait chez d'autres dysentériques : ici le sang était rendu abondamment à demi-pot de nuit à la fois et pur, sans mélange de mucus, de pus ou de matière stercorales. Je ne sais si je me trompe, mais le sang qui, chez les dysentériques, est mêlé aux selles, provient souvent d'une sécrétion opérée par les follicules irrités, comme on voit chez les nourrices une succion trop prolongée des mamelons donner du sang au lieu de lait, tandis qu'ici il était fourni directement par les exhalans.

J'ai déjà, il y a plusieurs années, conçu et émis, (*Journal complémentaire*, décembre 1829, page 142), l'opinion que les hémorrhagies autres que les traumatiques, et les inflammations que rapprochent, de l'aveu de presque tous les physiologistes de nos jours, l'identité des causes, la similitude des phénomènes et la propension au remplacement mutuel, étaient dues à la même modification organique, et que la variété des formes sous lesquelles elles se présentaient à nos sens ne dépendait

que de la différence de texture et des fonctions des systèmes organiques qu'occupait cette modification. Je pensais que, fixée sur l'ordre des vaisseaux chargés de l'apport des matériaux nécessaires à la recomposition ou nutrition des tissus, l'irritation revêtait la forme inflammatoire, et se présentait sous l'hémorhagique quand elle intéressait vivement ceux auxquels sont confiées les exhalations autres que nutritives.

Je sais qu'à l'appui de mon opinion je ne puis fournir aucune preuve; si pourtant on veut considérer que l'organisation et la vitalité qui s'ensuit doivent nécessairement différer dans ces deux ordres de vaisseaux, puisqu'ils accomplissent des fonctions différentes, il faut, par contre, admettre qu'ils peuvent être séparément et même isolément irrités. Le principe une fois admis, calculez quels doivent être les effets de l'irritation sanguine dans les capillaires nutritifs, et vous verrez que naturellement et rigoureusement vous serez conduits, par la seule argumentation *à priori*, aux quatre phénomènes fondamentaux de l'inflammation, tumeur, rougeur, chaleur, et douleur; ce dernier phénomène variant de caractère d'après mille circonstances que tout physiologiste peut prévoir et apprécier. Transportez maintenant cette même congestion sur les capillaires exhalans, sur ceux qui dans l'état ordinaire de leurs fonctions élaborent et sécrètent ce liquide vaporeux qui favorise le glissement des membranes séreuses et contribue à humecter nos surfaces de rapports, et vous verrez se produire les phénomènes des hémorrhagies. Partant de cette hypothèse, vous concevrez sans peine ce qu'aucune autre théorie n'a pu expliquer, comment les inflammations suppriment les hémorrhagies, comment celles-ci remplacent, préviennent souvent et jugent fréquemment les inflammations, à savoir par l'effet d'une révulsion opérée par un ordre de vaisseaux sur un autre; elle

rendra compte rationnellement des bons effets de la vésication cutanée dans les hémorrhagies, et dira pourquoi elle y réussit mieux et plus promptement et plus sûrement que dans les inflammations; elle expliquera, en s'appuyant sur la comparaison de plusieurs autres faits analogues observés dans d'autres tissus, pourquoi, antagonistes par nature, ces deux formes irritatives s'allient quelquefois et portent alors une atteinte prompte et inévitable à la vie qu'elles attaquent; pourquoi les sédatifs de l'appareil périphérique sanguin, froid, acides et autres médicamens astringens, tant végétaux que minéraux, arrêtent si énergiquement les hémorrhagies et échouent contre les inflammations intérieures.

Je le redis de conviction, je ne puis administrer à l'appui de mon opinion aucune vivisection, injection ni expérience qui pourrait servir de preuve; mais je crois ma théorie conforme à la saine physiologie, propre à rendre un compte satisfaisant de tous les faits pathologiques qui se rapportent aux inflammations et aux hémorrhagies, et propre à justifier les indications thérapeutiques dont jusqu'ici la pratique s'est bien trouvée et en faire naître de nouvelles.

Histoire générale.

§ I^er^. *Symptomatologie.*—Voici les symptômes principaux avec lesquels la maladie s'est offerte à notre observation : chez les uns, elle est précédée d'un sentiment de malaise et de plénitude gastrique, constipation, inappétence. Cependant le malade continue à manger et à boire, et la faiblesse qu'il éprouve est une raison de plus pour ingérer des alimens. Diarrhée, selles liquides avec soulagement du poids stomacal, plus fréquentes la nuit que le jour; douleurs ventrales, tantôt vagues, mais le plus souvent iliaques ou hypogastriques : les infirmiers ne s'en

aperçoivent pas et les malades s'applaudissent de nous en avoir dérobé la connaissance, et s'efforcent de manger; augmentation de la diarrhée, diminution des urines, pâlissement et étirement de la face, ce qui fait naître la suspicion de l'existence du flux de ventre; dénégation absolue, assurance qu'on se porte bien; quelques malades cependant conviennent de leur état, et alors une diète rigoureuse, des boissons gommées préviennent les progrès du mal et remédient à celui qui déjà est fait. Chez d'autres, l'invasion est brusque, elle a presque toujours lieu pendant la nuit; elle est accompagnée d'un sentiment général de prostration et de froid, d'évacuations fréquentes, tormineuses, qui, liquides et stercorales d'abord, deviennent bientôt sanglantes; douleurs abdominales déchirantes ressenties le long du colon, quelquefois à son origine, mais presque toujours à l'S romaine ou à l'hypogastre, qui est souvent soulevé, rénitent. Le besoin d'aller se multiplie de plus en plus et devient bientôt incessant; le malade ne quitte plus le bassin, cependant il n'exprime qu'avec peine et au milieu des plus douloureuses épreintes quelques gouttes de sang ou quelques grumeaux sanglans. La sécrétion urinaire est suspendue; la peau se plisse; celle de la face est d'un blanc terne et sale, mêlé de rouille ferrugineuse; les traits s'affaiblissent et se grippent, les yeux se cernent et se cavent, la température baisse; les extrémités, surtout les pieds, sont glacés; le pouls s'enfonce et s'efface sans s'accélérer et souvent en se ralentissant; la langue est pâle, devient bleuâtre, se sèche et se recouvre d'un enduit ponctué blanc ou jaune; la soif est quelquefois nulle, d'autres fois forte, et dans ce cas le malade appète les boissons chaudes, repousse celles qui sont froides et acidulées parce qu'elles excitent des nausées et des coliques. Si le vomissement survient, il se compose d'un liquide porracé, âcre, fétide; souvent il se

manifeste dès l'invasion et s'accompagne alors de constriction précordiale, et chez quelques-uns de crampes dans les mollets.

La maladie prend-elle une tendance funeste, tous ces différens symptômes s'aggravent, la figure se creuse de plus en plus, le refroidissement va sans cesse croissant; le pouls devient tout-à fait insensible; le hoquet se déclare et les déjections continuent sans relâche; le malade ne prend plus aucun soin de les retenir et les laisse couler; elles deviennent vertes, noires, répandent une odeur particulière, douceâtre, pénétrante, nauséabonde, dont s'imprégne l'atmosphère du malade; la langue et l'haleine sont froides, sa peau livide, sa face cadavéreuse, son nez pointu, ses dents et lèvres noires, son corps glacé comme un marbre; et c'est dans cet état qu'il languit pendant plusieurs jours, jouissant de toute sa raison, sans que cataplasmes chauds, irritans de toute espèce, sinapismes, frictions spiritueuses, camphrées, puissent rappeler chez lui quelque chaleur; quelquefois nous les avons entendus, dans les derniers jours de leur vie, accuser un froid intérieur sans que la nécropsie ait pu, par quelque circonstance particulière, expliquer cette sensation. Les déjections ne sont pas toujours du même aspect; quelquefois c'est du sang pur que le malade rejette, d'autres fois ce sont des pellicules blanchâtres nageant sur un liquide brun, ou bien du mucus tremblant strié de sang, ou des lambeaux membraneux, lisses sur une face, tomenteux et hérissés de prolongemens chevelus sur l'autre; ailleurs c'est un liquide homogène à la sortie du corps, mais qui se sépare bientôt en deux parties; celle qui surnage est rouge, semblable à de la lavure de chair; celle qui se précipite est blanchâtre, pareille à de la terre glaise; elle colle au fond du vase; soumise à la chaleur, elle se congule d'abord, se boursouffle et répand une

odeur ammoniacale. Peut-être ces déjections ne sont-elles que du sang décomposé par le travail phlegmasique, dont la matière colorante, n'existant que dans une petite proportion, douée d'une faible cohésion, est tenue en dissolution par le sérum au lieu d'être combiné à la fibrine; peut-être aussi n'est-ce que la partie albumineuse du sang seule qui, sécrétée à la surface de la muqueuse enflammée, est momentanément dissoute par la sérosité intestinale, alcalinisée par le travail phlogistique, et qui se dégage de sa combinaison et se précipite par le refroidissement. Ce sont au reste là des hypothèses qui ne peuvent apporter aucune modification au traitement, et sont par conséquent plus curieuses qu'utiles.

Un fait seul domine toute la question thérapeutique, c'est que la maladie est de nature phlegmasique, et c'est ce que concourent à démontrer et ses symptômes et ses effets sur le cadavre. Mais avant de nous occuper de ces derniers, disons que la maladie montra une prédilection toute particulière pour les individus d'une constitution faible et délicate, ou ceux qui étaient épuisés par une longue maladie, et tout spécialement ceux qui avaient déjà offert des symptômes de diminution dans la force de cohésion ou d'altération dans la mixture des élémens du sang. Ceci n'est encore qu'un fait que j'articule; je suis moins qu'un autre partisan de la médecine dite humorale; j'ai souvent déjà eu l'occasion de m'expliquer sur ce que je pense de l'altération des humeurs; mais j'ai vu ce que je rapporte et je laisse aux autres à en déduire des conséquences.

Depuis le mois d'août je rencontrai plusieurs fois des tuméfactions des gencives avec ramollissement, suintement de sang, fétidité de l'haleine, quelquefois avec des ulcérations étendues de la muqueuse de la langue ou des joues en arrière des dernières molaires, ou ébranlement et même chute des dents.

Le sulfate d'alumine en solution pour gargarisme, l'attouchement avec l'acide hydro-chlorique dans du miel rosat ou la cautérisation avec l'acide pur, remédiaient à ce stomacace qui souvent se montra rebelle et dont la guérison se fit long-temps attendre; mais ce furent particulièrement ceux qui en étaient ou qui en avaient été atteints qui contractèrent la dysenterie les premiers, parmi les malades en traitement à l'hôpital, et avec le plus de violence.

Voici ce que j'observai entre autres: un jeune homme, qui avait eu un écoulement sanguin aux gencives, et qui en était guéri, contracta la dysenterie le lendemain de cette entéro-colite, qui était accompagnée de déjections très-nombreuses de sang ténu; des milliers de taches bleuâtres, lenticulaires ou losangées, circonscrites, couvrirent tout son corps, particulièrement les plians des membres.

A l'ouverture du cadavre, nous trouvâmes, indépendamment des désordres communs à tous, la membrane interne de la fin de l'intestin grêle, et ce qui restait de celle du gros intestin, enduite d'une couche sanglante; au-dessus de la zône intestinale, siége de cette exhalation, on rencontrait dans le tissu cellulaire sous-muqueux d'innombrables ecchymoses de grosseur et de forme variables, ce qui donnait à la partie affectée un aspect tigré.

La température, qui s'élevait lorsque la réaction vasculaire s'établissait, et que le sang était poussé avec quelque force vers la périphérie, baissait au fur et à mesure que celle-ci perdait de son énergie, et cet abaissement était d'un funeste augure. Le pouls suivait la même échelle; fréquent et vif au moment de la réaction, il se ralentissait et s'enfonçait lorsque les progrès de la phlogose enrayaient les mouvemens du cœur. Autant l'élargissement et le développement du pouls étaient un signe fa-

vorable, autant son affaiblissement, alors même que le ralentissement l'accompagnait, m'a paru funeste. La langue rougissait quelquefois vers la fin de la vie, mais rarement, parce que chez ces individus, épuisés par une longue maladie, le sang était trop appauvri; sa quantité, trop petite, peut-être aussi l'appel fait à l'intérieur trop impérieux pour qu'il s'en trouvât assez pour rougir les tissus sympathiquement affectés. Voilà ce que nous trouvâmes en général : la langue plate, raide, rouge au limbe, sèche, jaune, grenue sur le corps : pâlissait-elle, devenait-elle verte, ces phénomènes s'accompagnaient-ils de vomiturition et d'abaissement de la température, on pouvait annoncer une mort très-prochaine avec sphacèle des intestins. Il est digne de remarque, qu'au rebours de ce qui a lieu dans les phlegmasies de l'estomac et du commencement de l'intestin grêle, les centres nerveux qui président aux actes de l'intelligence conservaient toute leur intégrité d'action. Le délire était un phénomène rare, tandis qu'il est presque inséparable des gastro-entérites aiguës; cela vient parfaitement à l'appui des expériences et des observations de M. Scoutetten sur l'intensité des liaisons sympathiques entre l'estomac et la méningine cérébrale. Dans aucune des morts survenues à la suite de l'entéro-colite, où nous n'avions vu ni convulsions, ni contractions spasmodiques, ni paralysie, aucune altération n'a été trouvée dans les organes intra-céphaliques.

Dans les cas où du narcotisme et quelques mouvemens musculaires avec du trouble intellectuel ont été observés durant la vie, des lésions intra-crâniennes ont été reconnues. Nous ne notons cette circonstance que parce qu'elle fait ressortir cette immortelle vérité, découverte et proclamée la première par la médecine physiologique, que toutes les fois qu'il existe des troubles fonctionnels, des

altérations doivent se présumer dans les organes chargés de remplir ces fonctions, et par contre, que la régularité de la fonction pendant la vie doit faire conclure à l'intégrité de la portion de matière organisée chargée de son accomplissement. Si des exceptions qui, de l'aveu de tous les médecins, deviennent au reste tous les jours plus rares, étaient invoquées contre cette règle générale, il faudrait les expliquer ou par le peu d'exactitude avec laquelle on procède souvent aux ouvertures des cadavres, ou au peu de connaissances anatomico-pathologiques que possède celui qui rend compte des ouvertures, ou enfin par le peu de traces qu'en ne faisant que passer les processus morbides laissent souvent dans les organes.

Jusqu'ici je n'ai donné les signes que des seules phlegmasies aiguës, soit récentes, soit entées sur un état chronique : ceux de la chronicité sont un peu différens, mais non moins caractéristiques.

Les déjections sont peu nombreuses; souvent il n'y en a plus qu'une dans les vingt-quatre heures, mais elles sont toujours liquides. Dans la plupart des cas, le malade reste long-temps, souvent un jour entier, sans sentir le besoin d'aller, mais alors il fait cinq, six selles de suite, petites, avec ténesmes, mêlées d'excrémens et de glaires. Cependant les forces reviennent, le courage renaît, il se lève de son lit, parcourt les salles; l'appetit est dévorant, le malade rebat sans cesse les oreilles du médecin de ses plaintes sur l'insuffisance de sa nourriture. Les urines passent avec plus d'abondance, mais le teint reste blafard, la peau sèche; au bout de quelques jours d'alimentation, récidive, selles nombreuses, suppression des urines, chute des forces, peau aride, rude au toucher; face d'un jaune sale, paupières œdématiées, lèvres décolorées, progrès du marasme; il se fait souvent encore un ou plusieurs arrêts dans le flux de ventre, mais il revient toujours; en-

fin les malades s'affaiblissent au point de ne plus pouvoir quitter le lit, ils deviennent extrêmement impressionnables au froid; pour s'en garantir, les uns se blottissent sous leurs couvertures qu'ils retirent sur leur tête; les autres demandent à occuper des places dans le voisinage du poële; dans cet état de choses survient promptement l'infiltration générale; les urines sont complètement supprimées, il n'en passe même pas avec les selles, mais comme celles-ci sont très-liquides, nous en avons vu qui croyaient pisser par l'anus. C'est ainsi qu'ils languissent pendant plusieurs semaines; affaiblis au point de ne pouvoir se redresser sans aide, n'ayant plus assez de souffle pour articuler distinctement leurs paroles, j'en ai rencontrés qui demandaient encore à manger. La mort est douce, elle n'est précédée ni de râle, ni de convulsions.

§ II. *Siége et nature.*—Le siége de l'inflammation est à la fois le gros intestin et le grêle. Nous n'avons ouvert aucun cadavre, (et malheureusement le nombre en a été fort grand) qui n'ait présenté de grands désordres dans ces deux divisions du tube digestif; il ne s'est pas offert un seul cas où la muqueuse du gros intestin n'ait offert de l'injection sanguine, de l'épaississement, des ulcérations nombreuses, profondes, étendues. Je ne parle pas du sphacèle dont nous avons constaté la présence dans un grand nombre de cas. Or, on sait que des anatomo-pathologistes du premier ordre ont si rarement rencontré des ulcères dans les colites aiguës, qu'alors que de semblables altérations se sont présentées sur les cadavres de ceux qui avaient succombé à la dysenterie aiguë, ils les ont considérées comme accidentelles, tandis qu'ils les ont constamment remarquées et annotées dans les dysenteries chroniques.

Nous avons également remarqué dans la grande majorité des cas la nuance rouge, violette, pourprée, ardoisée

de la muqueuse de l'iléon, ramollissement et tuméfaction, saillie, ulcérations des follicules muqueux. Est-il un médecin de nos jours qui, à la vue de semblables désordres, hésitât à proclamer l'existence de l'inflammation?

§ III. *Anatomie pathologique.* — Les détails d'anatomie pathologique se rapportent naturellement à deux chefs : l'examen des organes directement intéressés et ce lui de leurs annexes. — 1° Pour ce qui est du tube digestif, les altérations constantes sont : la désorganisation de la terminaison de l'intestin iléon; sa tunique muqueuse est toujours partout sensiblement altérée; tantôt la phlogose est rouge, tantôt ardoisée avec ramollissement ou épaississement de son tissu et souvent de toute la paroi intestinale. Dans le gros intestin la muqueuse est dénaturée dans sa presque totalité, ou du moins dans une grande étendue; les follicules muqueux sont tuméfiés et saillans, ayant quelquefois la grosseur d'un grain de millet, d'autres fois celle d'un poil; l'intérieur de l'intestin est tantôt rugueux, tantôt bosselé; sa surface est inégale, grisâtre, verdâtre, bleuâtre, hérissée de granulations diversicolores, ce qui lui donne un aspect granitique : ailleurs elle est couleur de chair, mamelonnée, bourgeonnée, saignante, sillonnée de gouttières anfractueuses, grisâtres; ailleurs encore, le gros intestin est sphacélé en tout ou en partie, et alors ce sont plutôt les deux portions verticales, mais plus particulièrement la portion descendante du colon et le rectum que la transversale, où la mortification existe; ces parties ainsi mortifiées répandent une odeur pénétrante; un pus sanieux et noirâtre s'en écoule, la couleur de l'intestin est d'un vert foncé, pointillé de blanc et de rouge, mâché, broyé, on dirait d'un mélange de choux verts écrasés avec du pain et de la viande.

Quand le malade succombe à la suite de l'état chronique, toutes les tuniques intestinales sont très-épaisses; les

deux internes sont dévorées par de larges ulcères à bords durs et renversés, à fond bleuâtre ; l'externe, épaissie, est le seul empêchement à la communication de la cavité intestinale avec l'abdomen ; le gros intestin contient des matières noires, pareilles à du marc de café, son intérieur est hérissé de pustules, de larges porreaux, de fongosités étendues : quand on coupe ces productions morbides, on pénètre dans une matière d'un blanc bleuâtre, nette à la coupure et semblable à du blanc d'œuf durci par la chaleur : elle est formée par les tuniques celluleuses devenues squirrheuses et n'offrant, en d'autres cas, qu'une masse ulcérée, cancéreuse, et dans laquelle on chercherait en vain quelques traces de l'organisation primitive. Une seule fois l'intestin était si ramolli dans toute son épaisseur, que la traction avec les mains seules suffisait pour en faire autant de tronçons qu'on le voulait. C'est à la suite de la phlogose chronique et lente que le tissu cellulaire sous-cutané et les cavités séreuses, surtout celle de la poitrine et de l'abdomen contiennent beaucoup de liquide citrin, limpide, collant.

L'appareil cérébro-rachidien n'a presque jamais présenté d'altération ; l'étonnante inertie de la circulation et le refroidissement constant de la périphérie, m'avaient fait soupçonner l'existence d'une lésion de la moelle, mais toutes les recherches à cet égard ont été sans succès.

L'intestin grêle contient des vers morts; dans aucun cadavre on n'en a trouvé plus de trois : ils étaient enveloppés de mucosités épaisses. Le foie est presque toujours altéré, tantôt de couleur ardoisée, et rempli alors de sang poisseux, noir, tantôt jaune et gras ou cendré, grumeux, de couleur de chair lavée et ramollie; la vésicule est ordinairement fort pleine de bile épaisse, foncée en couleur et incrustant sa couleur aux doigts qui l'avaient touchée ou décomposée, séparée en liquide séreux, limpide et en

fond blanchâtre, comme du plâtre mouillé. Nous avons vu une fois la vésicule épaissie criant sous le scalpel et noircie en dedans. La rate a été trouvée en général très-volumineuse et ramollie; on en a vu qui portaient de nombreuses cicatrices à leur surface, traces incontestables d'inflammations antécédentes guéries.

Les diverses duplicatures de la tunique péritonéale ont très-souvent offert des indices d'inflammation; l'injection de ses vaisseaux, le ramollissement de sa texture, les granulations couvrant sa surface, la sérosité au fond de laquelle on trouvait des flocons albumineux remplissant sa cavité, n'ont pu laisser de doute à cet égard.

L'appareil sanguin du mésentère a presque toujours été rencontré rempli de sang. Les glandes mésentériques et mésocoliques, ont subi de prodigieuses métamorphoses, tant sous le rapport de la grandeur que sous celui de la consistance.

§4. *Pronostic.* — Le pronostic était toujours fâcheux, la sur-sécrétion dont le canal intestinal est le siége et qui détermine un accroissement effrayant de l'absorption interstitielle; les mouvemens désordonnés et convulsifs du plan musculeux que sollicitent les évacuations, appellent sur l'organe affecté toute l'innervation qu'il y dépense rapidement. L'arrêt brusque introduit dans les autres actions vitales, atteint surtout le muscle central de la circulation, dont les mouvemens incertains et incomplets augmentent la langueur et l'inertie de toutes les autres fonctions; la nutrition générale est nulle, ainsi que l'accuse le rapide amaigrissement, le collapsus de la face, la flétrissure de la peau, l'abaissement de la température, je dis *nutrition générale*, parce que l'hypertrophie de l'intestin démontre que là, quoique vicieuse et dénaturée, la nutrition locale a lieu ; mais cette surnutrition même est un obstacle irréparable à la conservation de la vie et

au retour de la santé. Est-il possible de croire que des désordres pareils à ceux que, sans une seule exception, tous les cadavres nous ont présentés, soient susceptibles de redressement? qu'une aussi profonde désorganisation puisse rentrer dans l'état normal. Voici, au reste, d'après quels faits nous nous sommes guidés pour établir notre pronostic. Dans les cas favorables, la réaction s'établit au bout de quelques heures, la peau se réchauffe, le pouls se ranime et s'élargit, la figure s'épanouit, et dans ce cas, alors même que les selles restent aussi rapprochées et aussi fréquentes, il est permis de concevoir de l'espérance.

La face me paraît un indicateur plus précis, plus sûr, plus fidèle de l'issue que dans aucune autre phlegmasie; il m'a suffi dans le plus grand nombre des cas de dévisager le malade pour connaître exactement le degré du danger. On a vu les selles diminuer de nombre, augmenter de consistance, mais la face restée affaissée et livide, et tous les malades où cela s'observait ont succombé!

Mais il s'en faut que le fait de la réaction seul suffise pour fonder un pronostic heureux: cette réaction n'est très-souvent que passagère et remplacée par le plus profond collapsus. Chez quelques malades, nous avons eu la certitude que la rechute était due à quelques imprudences, en tête desquelles il faut placer le refroidissement (car il s'est montré plus funeste encore que l'ingestion d'alimens); dans d'autres la cause de la rechute nous est restée inconnue. Et ici il faut signaler combien il est difficile dans les hôpitaux militaires et particulièrement dans des momens de foule, de presse et d'encombrement, de suffire avec un infirmier, même instruit, exercé, actif, zélé, (le nombre de ceux-là est infiniment limité), au service de quinze malades, comme le prescrit le règlement, et à

cheval sur le texte, l'administration refuse d'excéder cette proportion, ou si elle n'ose pas refuser ouvertement, elle n'a qu'à employer la force d'inertie (1). Pour parvenir à ces fins, combien, dis je, il est difficile d'empêcher les malades de commettre des imprudences; nous avons rencontré des dysentériques au mois de novembre, égarés, le soir, dans les corridors, n'ayant pour tout vêtement, que leur capotte jetée sur leurs épaules: ils allaient aux latrines ou en revenaient.

Quand avec l'élargissement du pouls et le réchauffement de la peau, coïncidaient la diminution du nombre et l'épaississement des selles, surtout quand en même temps les urines reprenaient leurs cours, il était permis d'annoncer une heureuse issue.

La suspension de la sécrétion urinaire a été dans la dysenterie que je décris un symptôme aussi constant que

(1) Quoique le service sanitaire belge ne soit pas comme en France une fraction de l'administration militaire, qu'il ne se fonde et ne s'engloutisse pas sans nom, sans existence personnelle dans la machine dite administrative, qu'il constitue un corps dans l'armée, qui a son individualité, son chef et son représentant direct près du ministre de la guerre; que, sous ce rapport là, il ait fait un pas immense vers l'émancipation, il n'est pas encore entièrement libre dans ses mouvemens, et par là même ne peut pas faire tout le bien que l'État doit attendre de ses lumières et de son zèle.

Au nombre des améliorations qu'exige la raison et que le temps prépare, se trouve la subordination exclusive du personnel des infirmiers au personnel sanitaire; en effet, les médecins ne sont pas seulement plus intéressés que tous les autres officiers hospitaliers, à ce que le service d'infirmier se fasse de la manière la plus parfaite possible, mais eux seuls sont juges compétens dans la question de savoir combien il faut d'infirmiers, quels individus conviennent à cet emploi, et de quelle manière il faut les distribuer ou il faut les placer.

Dans une société neuve il suffirait de ce simple énoncé pour que tout le monde se rendît à son évidence; mais chez nous la tradition s'y oppose, et il est si difficile en général de faire sortir les hommes de l'ornière dans laquelle depuis long-temps ils se sont traînés!

la fréquence des évacuations. L'humectation et l'assouplissement de la peau étaient de même un excellent signe : assez commune dans les *entéro-colites aiguës*, elle devenait de plus en plus rare à mesure qu'on se rapprochait plus de la chronicité. Nous n'avons pas, malgré l'assertion contraire de Quarin, remarqué plus de danger dans les maladies débutant par des déjections de sang pur que dans celles où elles étaient glaireuses. Celles, au contraire, qui se partageaient en deux parties, une plâtreuse solide, l'autre liquide, semblable à de la lavure de chair, se sont toujours montrées de fort mauvais augure ; nous ne pouvons pas souscrire davantage à cette allégation des classiques, que les selles purulentes indiquent une mort prochaine : nous avons recueilli plusieurs exemples du contraire. En voici un : un jeune militaire atteint, à la suite d'écart de régime et d'exposition au froid, d'une rechute d'*entéro-colite aiguë*, rendit plusieurs fois pendant trente jours qu'il resta en traitement de copieuses selles, blanches à la sortie, au fond desquelles se réunissait au bout de quelques heures, un pus crémeux, homogène, lié. Il était réduit à un grand état de maigreur, mais avec des ménagemens, la diète et le salep opiacé, il s'est parfaitement guéri.

Un mauvais signe, c'est la persistance de la liquidité des selles ; elles ont beau diminuer de fréquence, si elles continuent à la longue à être claires, elles indiquent toujours l'altération de la muqueuse intestinale et présagent le marasme ou l'hydropisie. Si à cette liquidité se joint la répétition rapprochée des déjections, après une suspension plus ou moins longue, il est à prévoir qu'une portion de la terminaison de l'intestin est profondément désorganisée ; nous avons fréquemment remarqué cette circonstance chez ceux dont les cadavres présentaient dans l'S romaine du colon ces bourrelets circulaires ulcérés que

nous avons décrits. Le pronostic se règle spécialement encore d'après les complications qui surviennent pendant le cours de la phlegmasie entéro-colite. Disons quelques mots de celles que nous avons le plus communément observées.

§ V. *Complications et théorie de leur enchaînement.* Le péritonite en est une, elle m'a paru résulter le plus généralement de la propagation de l'inflammation de la tunique interne à celles qui la recouvrent de manière à intéresser toute l'épaisseur du tube digestif ; quand elle se déclarait d'une manière prompte, elle s'accompagnait de dureté, de tension et de douleur du ventre. Nous l'avons vu souvent circonscrite, ne s'étendant pas au-dessus de l'ombilic ; d'autres fois elle envahissait toute la surface péritonéale, elle était alors accompagnée d'une altération toute particulière des traits de la face, d'angoisse extrême et de vomissement. Dans les premiers cas, elle se laissait maîtriser ; dans le second elle menaçait promptement la vie, et la mort n'arrivait qu'après d'intolérables douleurs. L'accumulation des liquides, soit dans les cavités séreuses, soit dans le tissu cellulaire sous-cutané a compliqué presque toutes les dysenteries longues et qui duraient longtemps sous forme chronique. Toutes ne m'ont cependant pas paru devoir être attribuées à la même altération organique, et comme la reconnaissance exacte des causes me paraît donner lieu à différentes indications thérapeutiques, j'y ai porté une attention toute particulière. Voici sommairement quels ont été les résultats de mes études et de mes recherches.

Elles peuvent être attribuées à cinq ordres de causes qui agissent tantôt simultanément, tantôt isolément. 1° La plus constante me paraît être la diminution de l'action sécrétoire de la peau. En effet la surface tégumentaire soutient, avec la muqueuse du gros intestin, des rapports

tels que l'augmentation d'un côté entraîne une diminution relative de l'autre. — Sans avoir été expliqué par le père de la médecine, ce fait avait déjà été reconnu par lui et généralisé dans cet aphorisme : *Alvus liquida, cutis sicca.* Or, dans la dysenterie, la peau se sèche, se refroidit, se flétrit; les excrétions sudorales s'arrêtent, et la matière excrémentitielle qui devait être éliminée du corps par cet émonctoire demeure retenue. L'arrêt qu'éprouve cette fonction donne une activité plus grande aux exhalations du tissu cellulaire sous-cutané, les absorbans ne peuvent reprendre au fur et à mesure du dépôt, la vapeur séreuse se condense, se liquéfie, et l'œdème est produit. Dans d'autres cas, la brusque suspension des sécrétions à accomplir par la périphérie externe fait prédominer l'action exhalante dans les séreuses et donne naissance aux collections aqueuses dans leurs cavités.

2.° Une autre cause, moins constante peut-être, mais quand elle existe, non moins puissante, d'hydropisie, c'est l'obstacle qu'apporte au retour du sang et de la lymphe l'engorgement du système vasculaire de l'abdomen, suite nécessaire de la phlogose chronique des parois intestinales. En effet, comment la veine-porte pourrait-elle suffire à ramener dans le torrent circulatoire les liquides que lui apportent différens ordres de vaisseaux absorbans, quand celui que les tissus phlegmasiés lui versent la tient dans un état de congestion permanente?

3.° L'affaiblissement de la contractilité de l'appareil locomoteur, dont on sait que le concours contribue puissamment à la circulation centripète, et qui accompagne toujours les phlegoses viscérales chroniques, favorise aussi le ralentissement des progrès du sang veineux et de la lymphe, par conséquent l'apparition des hydropisies.

4.° Il en est de même de l'inertie des mouvemens du muscle central de la circulation, car si la progression des

liquides dans le système vasculaire centripète n'est pas entièrement et immédiatement dépendante de l'impulsion communiquée à ce liquide par le ventricule gauche du cœur, elle l'est au moins indirectement, et par une action médiate. D'ailleurs le retour du sang vers le cœur s'exerce plus spécialement encore par la force aspirante des cavités droites. — Or, dès que, frappé d'atonie, le ventricule gauche n'imprime plus au sang qu'une faible propulsion, qui diminue encore à mesure qu'on l'observe à une distance plus éloignée de son moteur, et que le droit n'agit de même qu'avec langueur sur le sang envoyé dans l'artère pulmonaire, de manière que l'oreillette ne se vidant pas complètement ne peut plus faire office de ventouse, il s'ensuit que la double action, dont le cœur est chargé dans sa fonction circulatoire, savoir celle de fouler et d'aspirer, n'est plus remplie que fort imparfaitement, d'où résulte indubitablement la stase du sang veineux et de la lymphe, et l'accumulation de celui-ci dans le tissu aréolaire et les cavités séreuses.

5.° Une cinquième cause d'hydropisie à la suite des inflammations de la muqueuse des intestins, c'est l'extension de ce travail phlegmasique aux séreuses de l'abdomen. Les épanchemens qui en résultent sont actifs, opérés par l'augmentation de l'action des exhalans; quand ces phlogoses sont aiguës, il est facile de les reconnaître. Se déclarent-elles sous forme chronique, le diagnostic est plus obscur, il ne se tire alors que de cette circonstance, que, dans les péritonites, il y a toujours un peu de douleur et de ténitence avant l'épanchement, et que celui-ci précède l'infiltration des extrémités inférieures, tandis que dans les accumulations passives le contraire a lieu. — Les collections liquides provenant des phlegmasies séreuses accidentelles contractées, par exemple, à la suite de l'impression du froid, ne compliquaient pas l'état du malade d'une

manière aussi fâcheuse que celles qui provenaient de la propagation du travail phlogistique ou d'un obstacle à la circulation. Plus ces dernières étaient multipliées, plus aussi le danger augmentait. Nous avons vu des cadavres où toutes les cavités viscérales contenaient de l'eau. L'hydropisie du péricarde a été reconnue et annoncée pendant la vie; la rareté, la faiblesse et l'irrégularité des battemens, et la mollesse de la percussion éprouvée par les parois thorachiques, étaient les symptômes sur lesquels ce diagnostic était appuyé.

Les sub-inflammations pulmonaires sont venues souvent se surajouter à l'entéro-colite chronique, et l'issue de cette complication a toujours été mortelle. Les signes de cet état morbide sont trop connus des médecins pour qu'il ne soit pas superflu de s'y arrêter. Je suis persuadé que l'exposition répétée au froid, nécessitée surtout pendant la nuit par la fréquence des déjections, a souvent été la cause des catarrhes bronchiques dont tant de dysentériques ont été atteints. J'ai souvent insisté près de l'administration de la guerre sur l'opportunité d'augmenter les fournitures de corps pour les malades dans les hôpitaux, de camisoles de laine. En faisant cette proposition, je n'avais pas, à la vérité, des dysentériques en vue : je ne pensais qu'à ceux dont les organes respiratoires sont morbidement surexcités; mais j'en sens plus que jamais la nécessité depuis que l'épidémie dont je viens d'être témoin m'a démontré à quel point l'impression du froid est nuisible à ceux qui sont atteints d'une entéro-colite.

L'apparition de phlegmasies sur différentes parties du corps ou de douleurs musculo-articulaires a été une circonstance rare dans les faits soumis à notre observation, ce qu'il faut attribuer en grande partie à ce que nous avons vu peu de dysenteries nouvelles, et que, par la durée de la maladie, les sympathies avaient beaucoup

perdu de leur activité. Mais lorsque ces métastases se sont présentées, elles ont été, en général, critiques, et ont amené une heureuse solution.—Il n'en est pas de même des ecchymoses; partout où nous les avons vues, elles ont été du plus mauvais augure, et si elles n'ont pas toujours annoncé la mort, elles ont au moins présagé une longue et opiniâtre phlogose.

On a très-fréquemment observé aussi la coexistence de l'entéro-colite chronique et des fièvres d'accès sous plusieurs types, mais le plus ordinairement quartes. Les sujets chez lesquels cette complication se rencontrait avaient une tendance toute particulière à s'infiltrer, et c'est dans l'abondance et l'opiniâtreté de ces collections, dans la rapidité de leur accumulation et la nature des troubles organiques qui en étaient la conséquence que le pronostic se puisait, plutôt que dans la présence de la fièvre intermittente.

Une dernière complication qu'il me faut signaler, c'est celle de la nostalgie. Il est peu de sujets atteints de dysenterie chronique chez lesquels elle ne soit survenue à un degré plus ou moins élevé. La longueur de leur séjour à l'hôpital, la sévérité des privations auxquelles ils y étaient soumis, le décès de plusieurs de leurs camarades avec lesquels ils y étaient entrés, répandaient dans leur âme un profond découragement et réveillait incessamment en eux le souvenir de leurs parens et le désir de retourner près d'eux. Cette complication était d'autant plus affligeante pour le médecin qu'elle prenait sa source dans une situation qu'il savait d'avance être désespérée, et dont par conséquent la satisfaction des désirs du malade ne pouvait tout au plus que reculer l'issue fatale; y accéder, c'était l'envoyer mourir dans ses foyers, où il n'avait peut-être ni lit, ni moyens pécuniaires pour se procurer une nourriture saine, ni médecin, ni médicamens; les retenir à l'hô-

pital, c'est ajouter à leurs douleurs physiques celles qui proviennent d'une imagination tourmentée de terreurs et de désirs.

Dans les cas désespérés j'ai cru devoir cependant opter, dans l'intérêt du malade, pour l'hôpital où je savais que tous les soins réclamés par son état lui seraient pleinement rendus, en même temps que mes promesses calmeraient ses peines morales. J'ai eu lieu d'être satisfait de ma résolution, car d'une part j'ai enlevé à la malveillance, toujours active, jusqu'au prétexte de répandre qu'on éloignait de l'hôpital militaire des individus contagiés, au risque de porter dans les campagnes la désolation et la mort; de l'autre, j'ai vu les malades reconnaissans de nos soins, et, confians à leur avenir, conserver jusqu'à leur dernière heure le contentement et l'espoir.

§ VI. *Causes.*—Il est facile d'attribuer la cause de toutes les maladies à une modification inconnue de l'atmosphère, à des altérations dans la composition chimique de l'air, qu'aucun eudiomètre n'a pu constater, et dont aucun signe sensible n'a du reste démontré l'existence; mais indépendamment de ce qu'une pareille allégation est purement gratuite, qu'on ne peut administrer à son appui la moindre preuve, elle a d'ailleurs l'immense inconvénient de ne fournir aucune indication ni prophylactique ni thérapeutique. En effet, comment se dérober à l'influence des causes qu'on reconnaît agir incessamment sur nous d'une manière inappréciable; quelle indication puiser dans l'admission d'un modificateur dont la nature et par conséquent le mode d'action sur l'économie sont un secret pour nous (1).

(1) Je suis loin de contester la puissance des variations de l'atmosphère sur les êtres organisés soumis à son action. Ce n'est pas à un médecin militaire qui, depuis vingt-quatre ans, exerce dans les hôpitaux et a parcouru à la suite de nombreuses armées le nord et le midi de l'Europe, que le pouvoir de l'influence des diverses températures et des climats peut être resté inconnu.

L'effet du froid et de la chaleur, et le plus ou moins d'énergie que communique à ces agens la présence ou l'absence de l'humidité, sont des faits faciles à constater, et tous les livres de physiologie s'accordent sur ce point. Le rôle qu'ils jouent dans la production des maladies a été de même étudié et connu; à cet égard aussi il n'y a pas de dissentiment dans les Écoles. Nous possédons des instrumens à l'aide desquels différentes conditions physiques de l'atmosphère sont rendues sensibles; leur existence n'a rien de problématique, leurs effets rien de douteux, rien de contesté.

Il n'en est déjà plus de même des influences électriques ou magnétiques; nous savons bien que l'électricité favorise quelques opérations organiques, la germination, par exemple, parce que les temps d'orage font promptement germer les semences; la physique possède un instrument à l'aide duquel on reconnaît la quantité, la nature, le degré de tension du fluide électrique dans l'atmosphère; mais cet instrument ne peut jamais devenir d'un usage commun, et ses résultats sont restés jusqu'à ce jour sans aucune application à la physiologie animale.

Que dire maintenant des altérations des propriétés chimiques de l'air, quand nous ne savons même pas à quel état de combinaison s'y rencontrent les différens gaz qui les constituent, quoique ces élémens proportionnels soient connus? Quel sens attacher à ces phrases sonores, mais malheureusement vides, de *modification de l'air, altération de l'atmosphère*, quand elles ne se rapportent plus à ces variations physiques, dont, ainsi que nous le disons plus haut, les effets sont appréciables et le mode d'action connu?

Je ne blâme pas les médecins qui en invoquent l'intercession. Ils agissent, j'en suis sûr, par des motifs réfléchis et désintéressés; ils peuvent s'appuyer, je le sais, sur l'autorité des anciens qui, dans chaque épidémie, admettaient l'existence d'une divinité, un *quid divinum*, τὸ θεῖον, qui devait se charger de l'explication de tout ce que les phénomènes de la maladie laissaient d'obscur pour l'observateur, tant dans leur origine que dans leur forme.

Je ne pense pas cependant que l'épidémie dysentérique que je viens d'être appelé à traiter, présente des circon-

stances telles qu'on ne puisse en rendre compte sans recourir aux *Dieux inconnus*.

Elle est née dans un camp formé au mois d'octobre, en grande partie de jeunes soldats nouveaux dans la vie militaire, servant à contre-cœur, et poursuivis sans cesse par la crainte d'une guerre acharnée et meurtrière. Pour celui qui connaît la vie des camps, qui sait combien sont étroites les baraques comparativement à leur population; combien est actif le service partagé entre les gardes, les exercices, les manœuvres, les corvées; combien peu peuvent être observés, en pareilles circonstances, les soins de propreté si nécessaires au maintien de la santé du soldat, et à quel point il est difficile de lui faire délivrer des alimens de premier choix; qui ajoute à ces conditions celle de la succession des nuits longues et froides à des jours encore chauds, de nombreux écarts de régime, les affections tristes de l'âme, les souvenirs de famille, les inquiétudes pour l'avenir, toutes circonstances dont l'expérience a constaté l'influence directe sur le tube digestif, et tout particulièrement sur sa portion inférieure; il n'y a pas à chercher ailleurs et dans des agens insaisissables surtout, la cause de la dysenterie qui a régné au camp de Diest, et qui s'est répandue de là dans les hôpitaux de Louvain, de Namur, et peut-être ailleurs.

Je reconnais que pour moi il y a déjà une réunion de plus de causes qu'il n'en faut à ma conviction pour en expliquer naturellement l'étiologie. De quelque puissance cependant que je les croie douées, je pense qu'il a fallu le concours de l'accumulation subite de beaucoup de malades dans les hôpitaux et de l'encombrement de ceux-ci par des arrivées soudaines, disproportionnées à leur étendue, pour les douer du degré d'activité nécessaire à la propagation de la maladie, et lui donner, par l'envahissement simultané d'un grand nombre d'individus, le caractère épidémique.

J'ignore ce qui depuis quelques années a pu jeter tant de défaveur sur la notion de contagion. Je sais que de prétendus philanthropes, plus riches, à mon sens, de zèle que de lumières, ont sollicité, supplié les médecins de ne pas articuler le mot de contagion, de peur de répandre la terreur parmi les populations, de semer partout la défiance et de frapper de stupeur l'activité publique. Mais qu'a-t-il donc de si épouvantable le mot de contagion? Sur quoi est fondé l'effroi qu'on prétend qu'il inspire? Posons les faits : Une maladie englobe un grand nombre d'individus, passe de ville en ville, de maison en maison; un médecin en infère que la maladie est contagieuse. Sur le champ il est mis au ban de la société, parce que, dit-on, il en trouble la tranquillité et en détend les ressorts. Un autre arrive et déclare que la maladie est épidémique, qu'elle tient à des altérations inconnues de l'atmosphère, et celui-là trouve faveur. Il n'est ni alarmiste ni perturbateur du repos public. Etrange et douloureux effet des préoccupations. Quoi! celui qui vient dire au peuple : la maladie qui règne chez vos voisins et y exerce d'affreux ravages, ne pénétrera chez vous qu'autant que vous le voudrez, qu'autant que par une coupable négligence, une criminelle collusion, vous permettrez une communication entre vous et les endroits infectés. Il ne tient qu'à vous, à votre volonté seule de vous en garantir à tout jamais. Cette détermination ne pourra s'accomplir, j'en conviens, sans quelques sacrifices; les intérêts de plusieurs en pâtiront; votre commerce, votre industrie en souffriront; mais ces pertes, que sont-elles en comparaison de celles que vous réserve l'introduction d'une maladie qui dévaste les contrées limitrophes et dont, à l'aide de la séquestration dont je vous prêche la nécessité, il ne dépend que de vous de préserver vos foyers. Quoi! dis-je, celui-là sera considéré comme alarmiste et mauvais citoyen, tandis que celui qui

représente la maladie comme dérivant d'une altération inconnue de l'air, à laquelle personne ne peut se soustraire, dont il faut subir l'action, et endurer les effets comme d'une aveugle, rigoureuse et inévitable nécessité, qu'il faut bien, par conséquent, laisser arriver, agir, s'étendre et s'éteindre quand et comme bon lui semblera, jouira de la faveur publique, sera applaudi par l'opinion, rémunéré par l'administration, recommandé par sa résolution et son courage! Certes, je me trompe fort, ou ce qu'il est de mode depuis quelques jours d'exalter et de prôner si haut est tout bonnement le fatalisme, qu'on blâme chez les Turcs.

J'ai foi à la médecine, et beaucoup, parce que chaque jour j'en reconnais la puissance; mais celle qui prévient les maladies m'est plus chère encore que celle qui les guérit.

Quoi qu'il en soit, la question qui se présente ici n'est pas *la dysenterie est-elle contagieuse?* car, proposée ainsi d'une manière générale, je la crois insoluble. Des faits pertinens constatent d'une part, que souvent la dysenterie se transmet; d'autres établissent qu'en d'autres circonstances elle ne s'est pas communiquée; il n'y a qu'un moyen de concilier cette apparente contradiction; heureusement il est simple, et c'est de définir le mot *contagion*. En effet, et cette remarque est ici à sa place, la véritable *contagion*, quoique dans la bouche de tous les médecins, n'a pas encore en médecine une acception convenue pour tous. Il semblerait toutefois qu'il fût grand temps de mettre fin à cette incertitude; si par *contagion* on ne veut indiquer que les seules maladies transmissibles par un attouchement *direct* de l'individu sain avec l'individu malade, ou avec des vêtemens qu'il a portés ou d'autres objets qui ont servi à son usage, je pense qu'il serait prématuré encore, d'après les faits connus, de ranger la dysenterie dans leur nombre; mais si la notion *contagion* doit s'étendre également à celles qui se propagent à l'aide des émanations morbides fournies par le corps malade et les miasmes ou effluves qui s'en élèvent, alors je ne crois pas qu'il puisse y avoir de doute sur sa propriété contagieuse.

On a, je le sais, introduit dernièrement dans le langage médical en France une épithète spéciale pour ces dernières; mais cette innovation me semble peu heureuse, non seulement parce que n'ayant pas été adoptée par tous les médecins, et spécialement ceux des autres pays, il doit laisser du vague et de la confusion dans les discussions relatives à cette matière, mais encore parce qu'il introduit une distraction plutôt formelle que réelle, et dont je ne vois pas l'utilité pour la prophylaxie ni pour la thérapeutique. Je me suis déjà expliqué sur ce point (voyez *Recherches sur l'Ophthalmie*, par Fallot et Varlez, Bruxelles, 1829); et dans tout ce que j'ai vu et lu depuis cette époque, je ne trouve aucun motif de changer d'opinion. Quoi qu'il en soit, si le mot *d'infection* doit remplacer celui de *contagion miasmatique*, le dysentérique constitue selon moi un foyer d'infection. Si les rayons qui s'en échappent ne produisent pas toujours chez ceux qui en sont frappés une maladie semblable à celle dont ils émanent, c'est que, dans ces cas, ils ne sont pas suffisamment concentrés, qu'ils ne pénètrent pas assez avant ou qu'ils ne sont pas absorbés faute de rencontrer une organisation prédisposée; car c'est une loi qui règle la transmission de toutes les maladies susceptibles de se communiquer d'un individu à un autre, *concentration suffisante du principe morbifère, disposition propre à en subir l'influence chez celui qui est exposé à son action.* Tout ce qui tend à disperser sa concentration ou à amoindrir la capacité d'en ressentir l'influence, énerve son action et

entrave ses progrès. Voilà pourquoi la multiplication des foyers d'infection est si dangereuse en répandant de toutes parts et dans tous les sens des miasmes qui, par leur accumulation se condensant et se fortifiant, font éprouver à la fin leurs effets délétères à ceux qui avaient d'abord échappé à leur action, et frappent bientôt tout ce qui se rencontre dans leur sphère d'activité. Ne pouvant pas s'opposer à l'élaboration de ces miasmes inséparables de la maladie dont ils sont un produit nécessaire, la tâche du médecin est d'en diminuer la puissance, d'en disperser les forces afin d'en garantir ceux qu'elle menace de ses coups, et c'est ce qui constitue la prophylaxie. Nous allons consacrer quelques lignes à l'exposition de celle dont nous avons fait usage.

§ VII. *Prophylaxie.* — Les moyens prophylactiques ont consisté à espacer le plus possible les malades, à répartir les dysentériques dans toutes les salles de l'hôpital, persuadé que la ressource la plus puissante que possède la médecine contre l'extension de la contagion appelée miasmatique par les écoles, c'est la dispersion de ces miasmes; à placer en évaporation constante du chlorure de chaux en pâte dans des tinettes à large surface, à aérer et ventiler les chambres autant que c'était conciliable avec le danger qui serait résulté pour les dysentériques d'un abaissement brusque de température, à éloigner soudain des salles toutes les matières excrémentitielles. Ce n'est pas que je pense qu'elles sont le véhicule de la contagion, c'est tout gratuitement et en désespoir de cause que cette opinion a été émise par les anti-contagionistes; mais parce que les particules putrides, odorantes, qui s'en exhalent, se répandent dans l'air qu'ils vicient et rendent impropre à l'acte de la respiration. Le lavage des vases de nuit avec le chlorure liquide, et, aussitôt que la diminution des malades l'a permis, le rebattage des matelats et le blanchissage de leur toile et de celle des paillasses avec le chlorure ont complété ces mesures. Voilà pour les moyens physiques. Les moraux n'ont pas été, peut-être, moins utiles. Je persuadais aisément aux infirmiers, aussi bien qu'aux malades qu'épouvantaient les nombreuses morts dont ils étaient les témoins, que le contact des dysentériques n'avait rien de dangereux, en restant long-temps près du lit de chacun, explorant avec un soin minutieux tous leurs organes, inspectant leurs excrétions, et dictant sur le champ les notes relatives à leur état. A l'appréhension qu'aurait pu faire naître la perte d'un de leurs camarades, j'opposais l'espoir dont ils avaient droit de se nourrir en voyant tant d'autres échapper à des dangers plus grands que ceux qu'ils courraient eux-mêmes. Je faisais valoir hautement les fautes de régime, les imprudences que les victimes avaient commises, et m'en servais comme d'un motif d'agir avec plus de précaution et de docilité. Dans les phlogoses aiguës des autres viscères, le trouble de la raison dispense en général les médecins de pareils soins; mais ici elle restait intacte jusqu'à la fin; et quand tout espoir était perdu chez nous, encore fallait-il en faire luire aux yeux de ces infortunés, et faute de pouvoir prolonger leur existence, leur en rendre les derniers momens moins douloureux et moins pénibles. C'est à quoi nous parvenions en écoutant toutes leurs doléances, en nous montrant affectueux, empressés, infatigables près d'eux, et ayant jusque pour leurs caprices tous les égards conciliables avec l'intérêt de leur situation.

§ 8. *Traitement.* — En commençant ce chapitre nous sommes forcés de reconnaître que chez plusieurs de nos malades, aucun moyen ne nous a réussi. Ce furent surtout les premières dysenteries arrivées de Louvain, qui se jouèrent avec la plus désespérante pertinacité de tous nos efforts pour arrêter leurs progrès et prévenir leur

issue funeste. Les mêmes remèdes, qui plus tard nous rendirent de si importans services, administrés de la même manière, se montrèrent impuissans, et j'en fus réduit à dire avec Stoll : *Dysenterias quasdam nullum huc usquecognitum remedium sanabat*, et à rester pour ainsi dire spectateur oisif des ravages de la maladie. A l'intérieur, aucun médicament n'était supporté. L'opium excitait de prompts et douloureux vomissemens et une soif ardente. L'ingestion des boissons, quelqu'elles fussent, était incontinent suivie de contractions convulsives du gros intestin avec épreintes douloureuses et l'expression de quelques gouttes d'un liquide sanglant. J'avais recours aux boissons chaudes et aux ventouses, aux sinapismes, aux vésicatoires, aux frictions cutanées ammoniacales, camphrées, sur la colonne vertébrale sans pouvoir rétablir la circulation expirante, ramener ni réaction, ni chaleur dans des corps passés déjà, si j'ose m'exprimer ainsi, à l'état de cadavre pendant la vie, ni calmer des souffrances que l'état d'intégrité des facultés sensoriales et intellectuelles ne servait qu'à rendre plus intolérables.

Heureusement cet état des choses ne fut pas de longue durée : on a remarqué souvent qu'à l'invasion les épidémies sont plus meurtrières que lorsqu'elles ont déjà régné quelque temps. Ce fait, signalé à plusieurs reprises, a été diversement expliqué. Les uns l'ont attribué à ce qu'à son apparition, la nature de l'épidémie n'était pas bien connue des médecins, qu'il fallait un certain temps à ceux-ci pour se familiariser avec elle et démêler par quel ordre de moyens elle pouvait être le plus efficacement combattue.

Cette explication, toutefois, était bien loin d'être satisfaisante pour tous les cas, puisqu'on voyait des maladies épidémiques céder aisément pendant leur cours aux mêmes

moyens à l'action desquels elle s'était montrée réfractaire pendant leur origine. Il existe une autre explication plus conforme à l'expérience et découlant naturellement de la saine théorie : c'est que les maladies épidémiques faisant brusquement irruption chez un grand nombre d'hommes, en attaquent tout d'abord, et de préférence, beaucoup dont la santé est altérée ou par les excès ou par de longues maladies, ou qui, soumis depuis long-temps à l'influence des causes débilitantes, ne peuvent tenir tête à la violence du processus morbide et en tombent les premières et inévitables victimes. Ceux, au contraire, dont la constitution est meilleure, qui sont doués d'une résistance vitale plus puissante, ne subissent pas aussi promptement son action, et atteints plus tard, luttent contre elle avec plus d'avantage.

La première et la plus importante indication à remplir dans le traitement des entéro-colites aiguës, celle dont la négligence rendrait l'accomplissement de toutes les autres infructueuses, c'est de soustraire autant que possible l'organe malade à toute espèce d'excitation, de soumettre par conséquent le malade à la plus rigoureuse diète. Cette indication, si précise à la fois et si simple, n'est pas aussi facile à remplir dans toute son étendue qu'on le croirait facilement : en effet, les besoins incessans que le malade éprouve et qui partent d'un estomac libre d'inflammation et appellant la stimulation alimentaire dont il a contracté une si longue et si douce habitude, le sentiment de défaillance qui en est la suite et qu'il semble au malade qu'un peu d'alimens ferait incontinent cesser, ne permettent pas toujours au médecin de persévérer dans une rigoureuse abstinence. On a recours alors aux féculens, aux gommeux; mais la stimulation exercée par ces substances sur la muqueuse stomacale n'est souvent pas suffisante pour que s'effec-

tue l'acte de leur digestion; alors renvois nidoreux et aigres, pesanteur d'estomac, profond mécontentement; peu ou point assimilés, ces alimens franchissent le pylore, descendent le tube digestif, et arrivés en contact avec la zône phlegmasiée, ils sont tumultueusement éliminés du corps au milieu des épreintes les plus fatigantes et de nombreuses déjections.

J'ai essayé souvent alors quelques cuillerées de vin, mais avec des effets tellement variables, que je ne puis en recommander généralement l'emploi. On conçoit du reste aisément que l'excitation produite en pareil cas par les alcoholiques sur la surface sensible de l'estomac, réfléchie vers le cerveau et irradiée par lui dans les muscles de la locomotion, doive faire cesser, au moins pendant un temps, le besoin d'alimens, et faire renaître le sentiment de la force; mais la sympathie entre l'estomac et le colon est si étroite et si active, que cette stimulation, quoique ne donnant aucun résidu stercoral, produit souvent les plus douloureuses contractions du gros intestin.

Dans le peu d'entéro-colites nouvelles qu'il nous a été donné de traiter, la force vitale n'a pas été aussi brusquement enrayée, l'irritation du cœur aussi complètement détruite que dans celles qui nous sont arrivées d'ailleurs; aussi, quoique plus d'une d'entr'elles ait débuté par l'affaissement des traits de la face, l'enfoncement des yeux, la lividité de la peau, le refroidissement des extrémités, la disparition du pouls, la réaction vasculaire momentanément comprimée n'a pas tardé à se rétablir et les signes de l'inflammation à se dessiner franchement. C'est dans cette circonstance que les émissions sanguines locales, même répétées, ont produit de bons effets. Les premières observations relatées plus haut, peuvent être consultées à cet égard. C'est bien à tort qu'on fait un précepte général de ne pas soustraire du sang dans les phlegmasies an-

ciennes; il en est souvent, parmi celles qu'on appelle chroniques, certaines dont les progrès désorganisateurs ne peuvent être arrêtés que par des émisions sanguines. Ce n'est pas d'après le temps de leur existence, mais selon le plus ou moins de sympathies qu'elles provoquent qu'on peut juger de leur nature, conclure à leur degré de chronicité et déterminer la médication la plus rationnelle.

Les cataplasmes émolliens, les fomentations tièdes, les bains de corps répétés plusieurs fois par jour et continués pendant plusieurs heures à la température de 28°, employés après les saignées ou de concert avec elles, convenaient dans les mêmes cas; nous les avons prescrits aussi dans les dysenteries chroniques avec l'intention de rappeler la chaleur à la peau et exciter la circulation, mais sans succès constans ou durables. Quand les lavemens étaient supportés, qu'ils n'irritaient pas le pourtour de l'anus, et n'étaient pas rejetés immédiatement après leur injection (circonstance qui n'avait lieu que trop fréquemment), on en administrait des demi plusieurs fois dans la journée.

Ces moyens se montraient-ils impuissans, nous avions tout de suite recours à l'opium, et c'est de tous les agens thérapeutiques le seul qui nous ait rendu des secours prompts et fidèles; aussi l'avons-nous de conviction proclamé avec Frank le *summum remedium*.

Il n'a pas pu parvenir à guérir tous les malades, mais il y en a peu qui ne lui aient été redevables de soulagement à leurs souffrances. Mais que de précautions à prendre dans son administration! quelle différence dans les doses d'après le plus ou moins d'irritabilité des organes digestifs! Rarement nous lui avons vu produire le narcotisme; mais chez un grand nombre il a excité des nausées et des cardialgies, des vomissemens. Si dans quelques-uns de ces cas le rougissement de la langue a pu faire

présumer l'échauffement de l'estomac, dans d'autres la langue reste pâle, décolorée. Il est une remarque intéressante à faire, c'est que tous les auteurs qui ont écrit sur la dysenterie s'accordent à donner des éloges à l'opium. C'est le seul remède qui réunisse tous les suffrages, au milieu de cette foule d'autres qui ne jouissent que d'une confiance isolée. C'est à tort qu'on prétendait, comme je l'ai entendu faire par des ultra-sceptiques en médecine, que ce n'est pas plus à lui qu'aux gommeux ou aqueux, qu'on lui donne comme véhicule ou auxiliaires, qu'il faut attribuer le succès; j'ai vu un grand nombre de fois (les occasions étaient journalières et trop belles pour ne pas en tirer parti) les déjections alvines continues, fréquentes et liquides, malgré l'emploi du salep ou toute autre décoction mucilagineuse, et revenir à leur nombre et consistance normaux après l'addition de quelques gouttes de teinture; la suspension de l'emploi de l'opium être marquée par le retour et la liquidité des selles, et ces symptômes se dissiper après un nouveau recours à son emploi.

Nous l'avons administré en mixture, en lavement, en cataplasmes, en frictions; des nombreuses préparations, sous lesquelles l'opium existe dans les pharmacies, c'est l'extrait aqueux et la teinture simple qui nous ont le mieux réussi. Du premier nous donnions d'un à trois grains dans les 24 heures, en solution dans de l'eau de gomme édulcorée; le dernier était prescrit depuis 10 gouttes jusqu'à ʒi dans ʒviij de décoction de salep.

Comme les déjections étaient toujours plus fréquentes pendant la nuit, c'était à son approche que nous en augmentions la dose, que par contre on diminuait pendant le jour. Les sels de morphine, la décoction des têtes de pavots indigènes se sont montrées inférieures en vertu aux préparations indiquées ci-dessus. Nos lavemens se composaient de ʒ iij à iv de mucilage ou de fécule avec

addition de 5 à 10 gouttes de teinture. Nous formions nos cataplasmes avec parties égales de farine de graine de lin et de têtes de pavots, ou nous arrosions les cataplasmes émolliens de teinture d'opium. Nos frictions étaient composées d'huile ou d'onguent mercuriel en combinaison avec cette même teinture. Ces frictions mercurielles nous ont fréquemment été utiles dans les péritonites partielles.

A mesure que l'inflammation baissait, que des déjections devenaient moins douloureuses et moins fréquentes, nous diminuions proportionnellement la dose du narcotique.

Nous n'avons pas à nous applaudir, dans l'entéro-colite, de la poudre de Dower, quoique nous l'ayons beaucoup employée surtout dans les commencemens. L'ipécacuanha à dose vomitive ne nous a été utile qu'une seule fois. Il s'agissait d'un homme d'un tempérament mou et lymphatique, tourmenté de nausées continuelles, vomissemens de matière jaune et amère, et se sentant soulagé après chaque vomissement. Après l'administration de l'ipécacuanha, les tranchées, les ténesmes et les déjections diminuèrent beaucoup; cependant il fallut recourir à l'opium pour les appaiser complètement; et la convelescence qui s'ensuivit fut longue et interrompue par de fréquentes rechutes.

Si quelquefois nous avons dépassé les doses énoncées, c'est en désespoir de cause, quand nous voyions continuer la diarrhée et les autres accidens. Nous nous soumettions alors à cet ancien adage : *Satius est experiri anceps remedium quam nullum*, adage pour lequel, malgré son antiquité et sa respectable origine, nous n'avons plus la même vénération qu'autrefois. Souvent au lit du malade, quand le cercle des médications rationnelles a été parcouru sans succès, et qu'on recommande l'essai de moyens empiriques, que nous jugeons téméraires par cela seul que

leurs effets ne peuvent être calculés, nous remplaçons l'adage précédent par cette autre maxime que nous croyons plus sage, plus conforme aux vœux de la raison et de l'humanité : *Il vaut mieux laisser mourir le malade que de le faire mourir.*

Les astringens, dans l'acception qu'y donnent les pharmacologistes, ne se sont jamais montrés utiles qu'en combinaison avec l'opium, et encore dans les cas les plus décidément chroniques ; la poudre de la racine d'arnica, tant vantée par les auteurs allemands contre les anciennes diarrhées, n'a de même exercé quelqu'influence que lorsque l'opium y était mêlé. J'ai mis en usage le columbo, le ratanhia, le sulfate d'alumine ; j'y ai quelquefois eu recours quand tout symptôme d'inflammation gastrique était dissipé, et je n'en n'ai retiré aucun avantage ; souvent j'en ai vu résulter de la cardialgie, de la sécheresse à la bouche et l'augmentation du flux du ventre.

Il n'en n'a pas été de même des féculens ; le riz, le salep, le pain, l'orge, la gomme arabique : dans beaucoup de cas où les préparations opiacées n'étaient pas supportées, leurs décoctions seules modéraient la douleur, tempéraient le cours du ventre et préparaient l'estomac à recevoir l'opium.

En un mot, partout où la méthode dite antiphlogistique n'a pu être employée ou n'a pas suffi à la guérison, les fécules et les opiacés ont seuls fait les frais du traitement ; et là où ils n'ont pas eu de succès, tous les autres médicamens prônés par les auteurs se sont montrés inefficaces ou nuisibles.

Dans celles des dysenteries chroniques dont nous avons été assez heureux pour atteindre la guérison, nous avons insisté long-temps sur l'opium administré à doses progressivement décroissantes, et nous ne l'avons abandonné complètement qu'après que la régularisation des évacuations, le retour de la coloration, des forces et de l'embonpoint nous avait attesté le rétablissement des actions digestives.

Les complications ont exigé des modifications dans le traitement. Voici en quoi elles ont principalement consisté. Dans les cas de péritonite, on a fait les émissions sanguines ou par des sangsues, ou plus souvent par une ou plusieurs ventouses scarifiées. On a ensuite appliqué des cataplasmes et frictionné le ventre avec un liniment mercuriel opiacé. Cette modification a eu plus d'une fois les suites les plus promptes et les plus heureuses. Elle a de même réussi dans les ascites aiguës, en même temps qu'après l'épaississement du flux de ventre, on administrait à l'intérieur le sur-tartrate de potasse soluble. Aux hydropisies passives résultant de la suppression des sécrétions cutanées, nous avons opposé avec un succès remarquable par sa constance, un mélange, par parties égales, d'extrait de chiendent ou de sureau et d'acétate d'ammoniaque (une once de chaque dans six onces d'eau pour les 24 heures). Des observations faites avec soin m'autorisent à conclure que c'est à cette dernière substance (l'acétate d'ammoniaque) surtout que l'effet salutaire du mélange était dû. L'augmentation des urines influait de la manière la plus marquée sur la quantité et la nature des selles. Plus la sécrétion de l'urine était active, moins par contre les déjections étaient nombreuses et plus leurs matières étaient épaisses. Pour exciter l'action des reins, j'ai fait quelquefois usage du vin et du vinaigre scilliticques en frictions ; mais je ne les ai pas trouvés d'une grande efficacité. Les frictions sèches aromatiques employées seules n'ont déployé aucune vertu. Le bandage expulsif appliqué sur les extrémités inférieures a beaucoup abrégé le traitement des œdèmes, quand d'ailleurs les urines coulaient.

Quand après l'évacuation des eaux le teint restait blême

et l'hématose languissante, sans que l'existence d'une phlogose chronique rendit compte de cet état, nous nous sommes bien trouvés du sulfate de fer combiné à une faible quantité d'opium (un grain du premier avec un quart de grain d'extrait du second, de deux à quatre fois par jour). Quand une fièvre intermittente venait compliquer l'entéro-colite chronique, nous la traitions comme si elle avait existé seule; trois à quatre grains de sulfate de quinine étaient donnés une demi-heure avant l'invasion de l'accès, et dans la très-grande majorité des cas cet accès était prévenu; cela n'empêchait pas la continuation des féculens unis aux opiacés. La plupart des dysentériques qui déjà avaient été attaqués auparavant des fièvres intermittentes en ont éprouvé des rechutes.

§ IX. *Convalescence.* — Dans les dysenteries nouvelles et aiguës, promptement enlevées par les émissions sanguines et autres moyens antiphlogistiques, les convalescences ont été courtes, solides; deux à trois jours suffisaient pour les rétablir.

Il n'en a pas été de même dans les dysenteries anciennes récidivées ou passées à l'état chronique. Là les convalescences ont été longues, incertaines, troublées par des accidens nombreux et traversées du jour au lendemain par des rechutes imprévues. Elles étaient d'autant plus difficiles à conduire que les malades étaient moins dociles aux bons conseils, ne se doutaient pas du péril auquel ils s'exposaient en mangeant à leur appétit, se permettant des mets indigestes ou affrontant le froid et l'humidité. Il n'est pas de maladie où les écarts de régime, et en général la violation des lois de l'hygiène, aient des effets plus funestes pour les convalescences que celle-ci, et il n'en est pas où le besoin de l'alimentation, l'appétence pour une nourriture stimulante soient plus impérieux. On persuade difficilement à des gens que la faim dévore, qu'ils

doivent, pour se guérir, s'abstenir de manger : ventre affamé n'a pas d'oreilles. — Cependant, pour le médecin qui ouvre les cadavres et apprécie l'étendue et la profondeur des lésions dont ils offrent le tableau, la nécessité d'une sévère diète, long-temps et persévéramment suivie, doit être abondamment prouvée, et son observance placée en tête des indications. Les temps sont loin de nous où une maladie était une entité, un être hostile seulement à la vitalité et étranger à l'organisation, pouvant être éloigné, dompté, anéanti par des moyens spécifiques : aujourd'hui une maladie est un *organe malade*, c'est-à-dire une partie du corps tellement altérée dans sa texture qu'elle ne peut plus remplir convenablement ses fonctions, car les fonctions dérivent aussi directement de la texture ou arrangement des molécules du corps vivant, que les mouvemens d'une machine de sa construction. On sait combien les dysenteries chroniques sont funestes. Les estimables auteurs de l'art. *Dysenterie* du grand Dictionnaire des sciences médicales l'évaluent à 90 pour 100 dans les hôpitaux.

D'après ce que je viens de voir, la proportion ne me paraît pas exagérée. Je le redis encore, un désordre pareil à celui dont les cadavres des dysentériques à l'état chronique ont présenté le spectacle ne me semble pas susceptible de redressement ou de réparation.

Pour guérir la maladie parvenue à ce degré, l'art du médecin (il faut l'avouer) est impuissant; aussi tous ses efforts doivent tendre à empêcher qu'elle n'y parvienne.

ÉTAT des dysentériques traités dans la première division des fiévreux à l'hôpital militaire de Namur, depuis le 19 octobre 1831 jusqu'au 10 janvier 1832.

ENTRÉE EN TRAITEMENT.		SORTIS guéris.	SORTIS convalescens.	SORTIS morts.	Restans.	Observations.
Evacués de Louvain le 19 octobre.....	36	16	4	16	3	
Evacués de Louvain le 3 novembre.....	52	40	4	5		
Venus de la garnison.	9	7	1	1		
Ayant contracté la dysenterie étant entrai-tement à l'hôpital pour d'autres maladies.....	10	5	1	4	3	
TOTAL.....	107	68	10	26	3	
		107				

FIN.

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SOME ACCOUNT

OF THE

LIFE, WRITINGS, AND CHARACTER

OF THE LATE

JAMES COWLES PRICHARD,

M.D., F.R.S., M.R.I.A.,

CORRESPONDING MEMBER OF THE NATIONAL INSTITUTE OF FRANCE; ETC., ETC.,

(BEING THE SUBSTANCE OF A MEMOIR READ AT THE MEETING OF THE BATH AND BRISTOL BRANCH OF THE PROVINCIAL MEDICAL AND SURGICAL ASSOCIATION, IN MARCH, 1849,)

BY

JOHN ADDINGTON SYMONDS, M.D.,

CONSULTING PHYSICIAN TO THE BRISTOL GENERAL HOSPITAL.

1849.

INDEX.

SOME ACCOUNT

OF THE

LIFE, WRITINGS, AND CHARACTER

OF THE LATE

J. C. PRICHARD, M.D., F.R.S.,

ETC., ETC.

SINCE the last Meeting of our Society, the profession, and not only the profession, but, indeed, the whole world of literature and science, has suffered a severe loss in the death of Dr. PRICHARD. A tribute to his memory would come appropriately from almost any Society devoted to science; much more so then from one that can boast of having directly derived lustre from his name; for at its first meeting, and in this very room, it was honoured by the presidency of that illustrious man. However far short I may fall of the proper execution of the pleasing task which I have imposed upon myself, I am sure that I shall have the sympathy and the interested attention of my fellow members, while I endeavour to give some account of his life and labours. In one respect the duty might seem superfluous: it might seem that all that Dr. Prichard accomplished for science is fresh in our recollection, and needs not to be retouched. This,

however, is in reality no reason. The vivid memory of benefits, so far from being a reason for refraining from the enumeration of them, naturally gives an impulse to the utterance of our feelings; and the tongue fondly dwells on what is near the heart, no matter how familiar may be the subject. In all ages, and among all civilized nations, it has been the custom to recount the virtues and great deeds of the departed while fresh in the recollection of the survivors; nay, among some people, almost before the ashes were cold the funereal eulogium was pronounced. If the men of by-gone ages were so ready to acknowledge and commemorate those actions and conquests of their heroes which might have been achieved under their very eyes, we should not be slow to record those triumphs of mind which we have witnessed, and which had something better for their object than mere personal or even national glory; for they gained possessions for the whole human race, redeeming, as it were, from the domain of darkness which surrounds us, large territories of light and knowledge.

Dr. Prichard was born at Ross, Herefordshire, in the year 1786. His education was altogether private. His father, a man of a highly cultivated and refined mind, superintended it with the help of different masters or tutors. A strong inclination to study very soon manifested itself. It was often requisite to compel him to leave his books in order that he might have needful recreation and exercise; yet when he joined his companions in the play-ground he entered into their sports with as much animation as the idlest

and gayest. Some of his early friends even avow that their most vivid recollections of the young Prichard have reference to his love of fun. The studies to which he most eagerly addicted himself were History and Languages. For acquiring the latter he had a remarkable aptitude. It was a great pleasure to him when he visited Bristol to talk with foreigners, who arrived at that port, in their own tongues. On one occasion he accosted a Greek sailor in Romaic, and the man was so delighted that he caught the boy-linguist in his arms and kissed him heartily.

When the choice of a profession became necessary he selected that of Medicine, not from any bias towards it, but because it presented no difficulties to him as a member of the Society of Friends, and at the same time admitted of his pursuing his favourite studies. He was first placed with Dr. Pole, of Bristol, who had a considerable reputation for skill in anatomical preparations.

From Bristol he went to Staines, in order to learn Medical Pharmacy under Dr. Pope and Mr. Tothill.

In due time he repaired to London, and devoted himself to the study of Anatomy, in the school attached to St. Thomas's Hospital. He afterwards removed to Edinburgh, where he spent three years of hard study. Among his fellow-students the most distinguished were Arnould, Estlin, and Hancock, and they continued to be his intimate friends for the remainder of his life. After his graduation in Edinburgh (1809), he spent a few terms in Cambridge, having become a member of Trinity College. In the

following year he joined the Communion of the Church of England, and having determined to pass some time at Oxford, he entered at St. John's College; but, not finding the society congenial, he took his name off the books and entered as a Gentleman Commoner, at Trinity. The time that he remained at Oxford must have been very short, for in 1810 he began his career in Bristol. He was appointed Physician to St. Peter's Hospital, about the year 1812, an appointment more memorable than any other that he subsequently held, because this Institution contained a class of patients whose maladies gave an impulse to his prosecution of a particular department of Pathology with which his name will ever be associated. His work on Nervous Diseases, as well as a later one on Insanity, was founded on the experience which he had gained in the wards devoted to insane patients in St. Peter's Hospital. In 1813 he published the first edition of his "Researches into the Physical History of Man." In 1816 he was elected Physician to the Bristol Infirmary. To his duties in that magnificent institution he devoted himself with a zeal worthy of the office, and reaped from its fertile field a vast amount of practical knowledge. He took an active part in the foundation of the Bristol Literary and Philosophical Institution; he frequently delivered lectures, and read papers at the meetings of the Philosophical Society, and was appointed one of its Pro-Directors.

It was wonderful how much he contrived to accomplish, even while engaged in his large private

practice. This was in part owing to his power and habit of employing small fragments of his time. His knowledge was so completely under his command, and his faculties were in such constant exercise, that he could immediately return to an argument or a train of thought, undistracted by any recent interruption. He made time also by his habit of early rising, which gave him three or four hours before the business of the day commenced. Whatever he undertook, he devoted the whole energy of his mind to its completion. He used to say that he experienced what John Wesley used to feel, when a student at Oxford, "the lust of finishing."

In 1845 he retired to Town, having been appointed Her Majesty's Commissioner in Lunacy; an honourable and comparatively lucrative appointment; at least, lucrative in comparison with most medical appointments, for no profession is so destitute as our own of offices of high emolument. No one better deserved a public reward, not only for his exertions in behalf of science in general, but also and especially for his contributions to the science and practice of that particular department of medicine.

Honours, such as belong to men of science, fell thick upon him. He became a Fellow of the Royal Society. He was elected Corresponding Member of the National Institute of France, and of the French Academy of Medicine. Besides these distinctions he received diplomas of honorary membership from all the chief learned societies on the continent and in

America. His work on Egyptian Mythology, and that on Nervous Diseases, had the honour of being translated into German. The people who speak that language were, I am afraid, more early alive to the great merit of his works, and even more interested in them, than his own countrymen. In 1835 the University of Oxford determined on conferring upon Dr. Prichard the degree of Doctor of Medicine by diploma,—the very highest honour which she has the power of bestowing, and which has been given at very long intervals only, and only to pre-eminent merit. In that year the Provincial Medical and Surgical Association held its anniversary in Oxford, under the presidency of the accomplished Regius Professor of Medicine, Dr. Kidd. Dr. Prichard had been appointed to deliver the Annual Address, and the day of the meeting was happily selected for the presentation of the diploma, the University deputing the President to hand it to him whom she thus delighted to honour. Those who know, as I do, the natural eloquence and classical refinement of Dr. Kidd, will imagine how wisely the University had chosen her representative. The scene was one that could not be easily forgotten by those who witnessed it. Under the august dome of the Library, built by the munificence of a physician of other days (Dr. Radcliffe), some of the most eminent members of the profession, from the metropolis and the provinces, were assembled. Dr. Prichard appeared rather pained than elated by all the flattering notice that

fell upon him, and was obviously relieved to turn attention from topics so personal to him by reading his Retrospective Address.

In a life like Prichard's the most remarkable events are his works. These I shall presently enumerate. It only remains for me in the present department of my subject to relate that he was in full mental vigour when overtaken by his last illness. This was of comparatively short duration. It was apparently occasioned by fatigue and exposure during the performance of his public duties. He fell ill at Salisbury, but he was removed to his home, Woburn Place, Russell Square, London. The disease baffled all the efforts of his medical friends, and, after great suffering, he died on the 23rd of December, 1848.

The work by which Dr. Prichard's name is best known to the world is that with which he commenced his scientific career, and which, ever improving under the continued consideration which he gave it, and ever deriving augmentations from the additions which he was perpetually making to his stores of knowledge, was the companion of the rest of his life. Works which derive their subject-matter from the world of thought only, when once completed are rarely added to. Any subsequent processes they undergo are those of finish and elaboration. But those which take their theme from the book of nature are not easily ended. Farther study of that book only brings more and more matter for extract and interpretation.

The Physical History of Mankind, when born into

the world, was an Inaugural Dissertation of 150 pages, which was a very unusual length for an Edinburgh Thesis, the average of such compositions varying from 20 to 30 pages. It was entitled "De Humani Generis Varietate." In 1831 it was expanded into a goodly octavo volume, and appeared in an English garb under the title, "Researches into the Physical History of Man." A second edition in 1826 appeared in two volumes, illustrated with plates. The first volume of a third edition was published in 1836. This edition extended over eleven years, the fifth and last volume having been published in 1847. While it is highly instructive to survey the gradual development of this production, growing with the growth of the author's mind and knowledge, it is no less interesting to trace the germinal nucleus, the generative idea in the original Thesis.

When Dr. Prichard entered upon the study of the Natural History of Man, it was an almost uncultivated field. Camper had made an attempt at classifying the human races according to the facial angle, having found that in the European it averaged 80°, in the Kalmuck 75°, and in the Negro 70° only. But his views were founded on a very narrow induction, for his collection of skulls was very small. Their inaccuracy in other respects, and especially the disregard of the difference between the infantine and adult skull, has been particularly pointed out by Professor Owen.

That Blumenbach was the real founder of Ethnology Dr. Prichard repeatedly announced; although

his own researches had commenced before the work of the illustrious German had come into his hands. Blumenbach, having examined a very large number of skulls, divided the prevalent forms of the human head into five departments, which he designated, not according to the form, but by the names of the races to which they belonged, or of the regions of the world whence these races were supposed to have originated. They were the Caucasian, Mongolian, American, Ethiopian, and Malayan; a distribution pronounced by Doctor Prichard to have been complete at that period of Ethnographical knowledge. This principle of classification, if now adopted, would require us to enumerate many additional varieties in the shape of the cranium, and to constitute correspondingly additional human races.

If we except, then, what had been done so slightly by Camper, and more elaborately and scientifically by Blumenbach; and if we also pass over, as we may very easily do, the vague *a priori* speculations of Sir W. Jones and Lord Kaimes (the former arguing for one species because one pair could by calculation be proved more than sufficient for peopling the earth, the latter presuming that Providence would not allow so many fair and fertile regions to wait for inhabitants by the slow process of dispersion, but that autochthones must have been *ab origine* assigned to them); if we except these, the ground which under Dr. Prichard's labours became so fruitful of interesting observation and inference, was when he entered upon it, unknown and sterile.

Dr. Prichard first set himself to inquire whether the *genus Man* contains more than one species. He carefully examined the characteristics of different tribes as to colour—the albino, the yellow, the tawney, the red, and the black: as to diversity of form, whether as to physiognomy, cranial configuration, or peculiarities in other parts of the skeleton; diversities of stature, as in Patagonians and Greenlanders; and having compared their diversities with known tendencies to variation in the inferior species of animals, he arrived at the conclusion that they are strictly analogous phenomena, "depending on a principle of natural deviation, and, as such, furnishing no specific distinction." The diversities of figure, considered by some to be an insuperable argument in favour of distinctness of specific origin, were found to be rather less permanent in mankind than those of colour, "and none of them so general in any race of men that it is not in many examples wanting." (1st Edition, page 85.) But though this conclusion was arrived at, it might still be argued that original stocks of the same species might have arisen in different parts of the world. To meet this view he inquired into the laws which govern the distribution of some of the inferior species (Mammalia), and found that every existing species may be traced with probability to a certain point originally its own abode, and that few or no species have been found in countries separated from their primary seats by barriers which their locomotive powers and peculiar structure do not enable them to surmount.

"On the whole, it appears that it has not been the scheme of nature to cover distant parts of the earth with many animals of every kind at once; but that a single stock of each species was first produced, which was left to extend itself according as facilities of migration lay open to it, or to find a passage by various accidents into countries removed at greater or less distances from the original point of propagation."—(1st Edition, page 145.)

He then proceeds to consider the migrations of man, and whether the facts prevent our applying the general inference drawn above to the particular instance of our own species, and he finds in them nothing irreconcilable with such a view.

The next inquiry he made was into the causes of the diversities in the human race. Climate has some influence, but civilization more. Varieties spring up more readily in temperate climates. One conclusion at which Dr. Prichard arrived in connection with this subject, and which has been the subject of more discussion among the uninformed than any other, is the transmutation from the Negro to the European; together with the announcement of his opinion that the original human stock probably belonged to the former race. The arguments adduced in support of this idea were as follows:—(1.) The analogy of lower species in which changes of colour are from dark to lighter hues. The lighter colours of domestic animals are the effects of cultivation. (2.) We have examples of light va-

rieties appearing among the negro races, but not of the reverse. (3.) The dark races appear by their organization better adapted to the wild or natural state of life. Witness the easy parturition in the female, and the high development of the senses of smell, taste, and hearing. (4.) All nations that have never emerged from the savage state are negroes, or very similar to negroes.

The next department of the inquiry carries him deeply into the physical history of the most remarkable races, which I cannot, of course, follow; but I may notice that with wonderful extent and minuteness of erudition he endeavours to prove a common origin of the ancient Indians and Egyptians from their mythologies, theogonies, and the physical character of the people respectively, and thus to support the previous inference that the most ancient nations of which any record exists were negroes. An investigation of the origin of the European races, conducted with no less learning and sagacity, led him to the recognition of an eastern origin, or connection by affiliation with the Asiatics.

Such is a faint outline of the original form of the great work by which Dr. Prichard's name will go down to posterity. Many were the modifications which it underwent, not only by expansion and addition, but also by withdrawal and absolute mutation. Topics which formed rather prominent members of the original organism were in the process of development dwarfed down to a proportion which anatomists call

rudimentary. Such is the opinion once so strongly and broadly stated as to the derivation of races from an original negro stock.

The second edition appeared in 1826, that is, after the lapse of thirteen years. It was enlarged to fully double the limits of the first, and entirely re-written. A more ample space was given at the beginning to the preliminary inquiry, as to the laws which govern the distribution of organized beings in general. This investigation, in the first edition, had been limited to the Mammalia. It now included the whole range of organic nature, beginning with the species of plants and extending to the whole of zoology. The conclusion arrived at in his previous more limited investigations were abundantly strengthened, and thus expressed :—

"The inference to be collected from the facts at present known seems to be as follows :—the various tribes of organized beings were originally placed by the Creator in certain regions for which they are by their nature peculiarly adapted. Each species had only one beginning in a single stock: probably a single pair, as Linnæus supposed, was first called into being in some particular spot, and their progeny left to disperse themselves to as great a distance from the original centre of their existence, as the locomotive powers bestowed on each species, or its capability of bearing changes of climate and other physical circumstances, may have enabled it to wander."

A new element in this edition was a discussion of the criteria of identity or diversity of species, by re-

ference to the principal laws of the animal economy; *e.g.*, (1.) As to duration of life, times and frequency of breeding, periods of utero-gestation, and number of progeny; liability to the same diseases; and possession of like faculties, instincts, and habits. (2.) To the laws of propagation of mixed breeds. (3.) To analogy to known variations.

The application of these tests to the human races was attended with the same results as before. In the course of the inquiry into analogous variations we meet with some new terminology, which was an unquestionable improvement. (Indeed, I may remark, in passing, that Dr. Prichard was particularly happy in his coinage of new names.) Thus the various black-haired races of man constitute the Melanic variety. The Xanthous comprises brown, auburn, yellow, flaxen, or red. The Albino is distinguished by white hair and red eyes. Again, in considering the varieties in the form of skulls, he classifies them according to the form of the vertex, as Meso-bregmate, Steno-bregmate, and Platy-bregmate; the type of the first being the Caucasian, of the second the Negro, of the third the Mongole.

The bulk of the work consists of the Physical History of particular races, evidencing most remarkably the continued labour that had been spent on the investigation since the first edition. The Races are considered under six divisions,—1st. The African Races. 2nd. Those of the Great Southern Ocean. 3rd. The Indo-European Nations. 4th. The Western Asiatics, including the Syrian or Semitic nations, Georgians, and Caucasians. 5th. The North and Eastern Asiatics, including the Finnish or Tschudish Nations, the Samoiedes, the Mongoles, the Tartar or Turkish Races, the Tungusians, and the Chinese. 6th. The Native Races of America.

The last book is devoted to a survey of the Causes which have produced Varieties in the Human Species. In the course of it appears an interesting discussion of facts relating to Hereditary Transmission of peculiarities of structure, the bearings of which on the chief question are obvious; and he shows as a general law how none but connate peculiarities descend to the offspring. "Whatever varieties are produced in the race have their beginning in the original structure of some particular ovum or germ, and not in any qualities superinduced by external causes in the progress of its developement. Yet the influence of climate and modes of life, domestication, &c., is unquestionable, and therefore according to this view it must be on the ovum that this influence is exerted."

The argument in this part of the work appears to me less satisfactory than in the other parts. For while it is strongly insisted on, that acquired peculiarities are never transmitted to the offspring, yet abundant proofs are given that great variations arise in races under the influence of external circumstances of climate, and in adaptation to them. No more striking instances can be adduced than those which belong to the Indo-European family, which were originally of one stock, yet which now present the black

Hindoos of the Deccan and the tribes of the Northmen of Europe. Dr. Prichard does not profess to explain how it is that the children of parents who have been exposed to changes of climate display peculiarities of structure corresponding with the climate, but he is satisfied that it is not by any change produced in the parents but by some qualities which they impress on the progeny. When a peculiarity has once been generated, that is, when it shows itself in an individual from birth, there is no difficulty in understanding its propagation. Thus many varieties may occur casually, as in the six-fingered family, the porcupine family, and the like. But the origination of varieties after transplantation to new localities is too extensive and uniform, both in the human and in the inferior species, to be explained in this manner. In the third edition the same line of reasoning is not pursued: but both in that edition and in the volume on the Natural History of Man, facts are adduced proving the transmission of acquired properties from parents to offspring, more especially those of a psychical nature, as in the acquired instincts of dogs. After some consideration of the whole subject, the following appear to me to be the most probable conclusions. In all healthy individuals of a species the elements of the varieties of that species exist; some actually developed, others only potentially present. External circumstances are adequate or even necessary to their development, but they can operate only through successive generations. The principal facts adduced against the hereditary transmission of ac-

quired peculiarities are those having reference to mutilations, losses of members, &c. These cases are altogether different from those in which a change has taken place in the colour of the skin under the influence of climate; for this change is not effected by subtraction of parts, but by increased action in a particular portion of the cutaneous organism. Now the offspring represents the properties and tendencies in the organization of the parents at the time of conception. Abundant instances in proof of this remark might be derived from pathology. The progeny of parents embrowned during a tropical residence, it is true, may be born quite fair, and yet with a liability in the skin to be influenced by climate in like manner with the parents, and to a greater degree. The next generation will inherit a yet stronger liability; but many centuries may need to pass before the structural change becomes so great as to be obvious at the time of birth. When the structural variety has been produced, it may require at least an equal length of time for external alterations to produce a return to the original type.

The work concludes with the consideration of the diversity and origin of Languages, an investigation which proves highly favourable to the inference drawn from other lines of argument, that the races of men have descended from a single pair.

The scientific reputation of Dr. Prichard, which had been gradually increasing from the time of the first edition of this work, as well as from his book on the Egyptian Mythology, may be said to have now be-

come universal. Among the learned of France and Germany he took the highest rank.

The last edition, as I have said, commenced in 1836, and was issued in single volumes, which appeared at intervals during eleven years. The actual amount of matter was treble what had constituted the second edition, and the whole was again re-cast and re-written.

The first volume is entirely devoted to the consideration of the two questions;—1st, Whether each species in the animal and vegetable world exists only as the progeny of one race, or has sprung originally from several different sources. 2nd, Whether the various races of men are of one or several species. In pursuance of this inquiry, analogically conducted, that is, by comparing different tribes as to their anatomical and physical characters, the author introduced matter of a highly interesting nature under the head of Psychological Characters.

He showed that no characters are more primordial and none more permanently transmitted than instincts, feelings, propensities, and habitudes of action. In trying the different races of man by this criterion, he found that there were none in which the characters belonging to the species are wanting. However degraded the castes, whether Bushmen of Africa, Australian savages, or Lappes of northern Europe, still we find in them the moral and social attributes which distinguish humanity. Not only is there no tribe wanting in the use of speech, and none in which we do not find traces of those necessary arts of life

which consist in the use of fire, of artificial clothing, of arms, and the art of domesticating animals; but also it has been ascertained that all tribes give evidence of the possession of sentiments, feelings, sympathies, and internal consciousness, with resulting habitudes of life and actions, which, more than any outward or physical character, whether of skull or of skeleton, of complexion or of hair, give the stamp of human likeness.

The following passage affords a striking view of the community of character in different races as to one most important law of thought and feeling, and is at the same time a specimen of the author's masterly style of writing.

"If we could divest ourselves of all previous impressions respecting our nature and social state, and look at mankind and human actions with the eyes of a natural historian, or as a zoologist observes the life and manners of beavers or of termites, we should remark nothing more striking in the habitudes of mankind, and in their manner of existence in various parts of the world, than a reference which is everywhere more or less distinctly perceptible to a state of existence after death, and to the influence believed both by barbarous and civilized nations to be exercised over their present condition and future destiny by invisible agents, differing in attributes according to the sentiments of different nations, but universally believed to exist. The rites everywhere performed for the dead, the various ceremonies of cremation, sepulture, embalming, mummifying, funereal processions, and pomps

following the deceased, during thousands of successive years, in every part of the earth,—innumerable tumuli scattered over all the northern regions of the world, which are perhaps the only memorials of races long extinct—the morais, pyramids, and houses of the dead, and the gigantic monuments of the Polynesians,—the magnificent pyramids of Egypt, and of Anahuac,—the prayers and litanies set up in behalf of the dead as well as of the living in the churches of Christendom, in the mosques and pagodas of the East, as heretofore in pagan temples,—the power of sacerdotal or consecrated orders, who have caused themselves to be looked upon as the interpreters of destiny, and as mediators between the gods and men,—sacred wars desolating empires through zeal for some metaphysical dogma,—toilsome pilgrimages performed every year by thousands of white and black men, through various regions of the earth, seeking atonement for guilt at the tombs of prophets and holy persons,—all these, and a number of similar phenomena in the history of all nations, barbarous and civilized, would lead us to suppose that all mankind sympathize in deeply impressed feelings and sentiments, which are as mysterious in their nature as in their origin. These are among the most striking and remarkable of the psychical phenomena, if we may so apply the expression, which are peculiar to man; and if they are to be traced among races of men which differ physically from each other, it will follow that all mankind partake of a common moral nature, and are, therefore, if we take into account the law of

diversity in psychical properties allotted to particular species, proved, by an extensive observation of analogies in nature, to constitute a single tribe."—(Vol. 1, p. 175-6.)

The Ethnography or Physical History of each of the different races is prosecuted in the four succeeding volumes. The prodigious amount of information is not more surprising than the skill with which the vast mass of facts is made to bear on the solution of the great question. In this department one is struck by the great accession of strength derived from the comparison of languages.*

But while the "Researches" were undergoing their fullest and, alas! their final development, Dr. Prichard found time to produce a volume on the Natural History of Man, containing an account of the different tribes, their peculiarities, and the causes of those peculiarities, but in a more summary way than in the large work, to which he refers for evidence of the positions which he lays down. In the preface he adverts to two opposite classes of critics,—those who accuse him of hesitation and reserve, or over caution, in his assertion of the great principle of the unity of the human species, and those who, on the other hand, allege against him an obstinate and intolerant ad-

* As I have noticed the change of terminology, as to the forms of the cranium, in the 2nd edition, I ought to have stated that in the 3rd edition the names were again changed to,—1. The Oval or Oöidal, which is the skull of the European and western Asiatic nations. 2. The Prognathous, so called from the prominence of the upper jaw, as in the negro of the Gold Coast. 3. The Pyramidal, or broad-faced skull, of which form the Mongoles present a good specimen, and the Esquimaux an exaggerated one.

herence to this view: and he was justified in laying claim to the probability that he had pursued a just, middle, and philosophical course, from the very opposite nature of those charges.

After surveying this work, one might say that it would have been no mean result, had it been the single product of Dr. Prichard's life and labours. But we shall see that he found time for many others, some more or less cognate to it, others of a remote nature.*

In 1819 he published his treatise on Egyptian Mythology, the main object of which, in a historical point of view, was to disprove the opinion entertained by Professor Murray, "that the religion and philosophy, as well as the language and all the other possessions of the Egyptian people, were peculiar to themselves, and entirely unconnected with those which belong to other nations of antiquity;" and, consequently, that the Egyptians were a race peculiar to Africa. He endeavoured to prove the early connection between the Hindoos and Egyptians, by their similarity of religious institutions, social castes, &c.

* Those who wish to obtain a clear view of the present state of Ethnographical Science will do well to read an excellent article in the 88th volume of the Edinburgh Review. I cannot let this opportunity pass without expressing my surprise that, in the text which accompanies the Ethnographical Maps in the Physical Atlas of Keith Johnson, so very slight a reference is made to Dr. Prichard's services. He is merely quoted for the illustration of a small matter of detail, and in the bibliographical list at the end of the paper, his work is not even mentioned,—a work which, at home and abroad, is allowed to be *facile princeps*. The omission can only have arisen from oversight. But such a blemish should be removed as speedily as possible from a publication so truly eminent.

Whether this connection was by colonization or by origin from the same stock he has discussed in the "Researches."

Against the former supposition the historical and other difficulties appear insuperable. And the latter conclusion, at first sight, seemed almost impossible to be maintained, from the extreme diversity of the Indian and Egyptian languages. Yet, on reading the discussion of this subject, in the second volume of the "Researches," we find the force of the difficulty breaking down under the powerful reasoning brought to bear upon it from the profound philological resources of the author's learning. He shows how much greater was the tendency to diversification in the structure of languages in the earlier ages of the world. He instances the diversity which had taken place in those sister-languages,—the Sanskrit, Greek, Latin, and Mæso-gothic, though sprung from a common stock, and which diversity had taken place as far back as fifteen centuries before the Christian era,—and he argues that "the diversifying process, within nearly an equal period of time, may have given rise to differences even so great as those which exist between the Semitic and Indian languages. That such was the fact we have the historical proof above cited. But if so great a diversity in language as this was really brought about, no difference of human idioms will afford proof of original diversity of race, and the Egyptians and Hindoos may have had common ancestors, from whom they derived their characteristic traits of resemblance." After this statement, it is

very interesting to find that Dr. Prichard's sagacious reasonings have been confirmed by the latest researches; and, as Dr. Hodgkin has remarked, "from a quarter the least expected. Recent investigations into the structure of the old Egyptian language, revealed to us by the successful interpretation of the hiero-grammatic writing, have demonstrated an early original connection between the language of Egypt and the old Asiatic tongues. By this discovery the Semitic barrier interposed between the Egyptian and the Asiatic races is broken down, and a community of origin established which requires the hypothesis neither of the immigration of sacerdotal colonies nor the doubtful navigation of the Erythræan Sea."*

A remarkable part of the work was the analysis of the remains of Egyptian Chronology. He showed that Manetho's Chronicle was constructed, perhaps by mistake, from the combination into one whole of many different records or tables of kings, which, though apparently successive, can be shown by internal evidence to contain repetitions of the same series.

The Chevalier Bunsen, in his great work on Egypt, has done justice to the value of Doctor Prichard's labours in this field of inquiry, when he says that "simultaneously with the first steps in the progress of modern hieroglyphical discovery (in 1823), Dr. Prichard, one of the most acute and learned investigators of his time, had once more vindicated the

* Abstract of a Memoir of Dr. Prichard, by Dr. Hodgkin, in the British and Foreign Medico-Chirurgical Review, April, 1849.

claims of Egypt to a primeval chronology, and suggested a collation of the lists of Eratosthenes and Manetho, as the true method of elucidating the earliest period. In the work on Egyptian Chronology and Mythology he shows that the continually recurring coincidences which they offer must represent a chronological canon."*

Another work, bearing on the great question, was entitled, "The Eastern Origin of the Celtic Nations, proved by a comparison of their dialects with the Sanskrit, Greek, Latin, and Teutonic Languages, forming a supplement to Researches into the Physical History of Mankind." Languages display four kinds of relations:— 1. As to vocabularies. If the communication between the nations was one of close commercial intercourse or of conquest, the words in common will be found to have reference to the new stock of ideas thus introduced. Such is the influence of the Arabic on the idioms of the Persians and Turks, and of the Latin upon some of the dialects of Europe. But if the connection was of a more ancient and intimate nature, the correspondence in the vocabularies will be found to involve words of the most simple and apparently primitive class, expressive of simple ideas, and universal objects. 2. There are languages with few words in common, but having a remarkable analogy in grammatical construction. Such are the polysynthetic idioms of the American tribes, and the monosyllabic languages of the Chinese and Indo-Chinese. 3. Some languages present both

* Egypt's Place in Universal History.—(Vol. 1, p. 242.)

these characters of affinity, and are denominated by Dr. Prichard, *cognate*. 4. There are languages in which neither of these connections can be found. Such languages are not of the same family, and generally belong to nations remote from each other in descent, and often in physical character. Dr. Prichard proved that the Celtic nations spring from a common stock with the Indo-European group from an elaborate comparison both of primitive words and of grammatical structure.

The last work that I have to notice, of a purely scientific character, is the "Review of the Doctrine of a Vital Principle." It is an admirable specimen of physiological reasoning, and had it been duly studied by many writers who have since treated of the subject-matter of it, much needless writing, both in support and in refutation of a hypothesis that had been already demolished, might have been saved.

The object of the work was to review the Hunterian doctrine of a vital principle; that is, of a subtile agent, somewhat analogous in its nature to electricity, invisible, impalpable, and imponderable, manifesting itself only by its effects, controling and modifying mechanical and chemical properties in a manner peculiar to itself, altering affinities, disposing to new combinations, so as to effect the separation of a variety of substances from the blood, evolving animal heat, presiding over chymifaction, exciting processes of development, nutrition, and reparation, and preserving the fluidity of the blood. He first points out that this doctrine is not a theory, because the actual

existence of the principle in question has never been proved; for a theory requires the alleged cause to be proved to be a fact in itself, before it is shown to stand in that relation to the phenomena assigned to it as effects. The doctrine in question is only a hypothesis, inventing the principle as a complete and the only means of interpreting certain phenomena. In the examination to which Dr. Prichard subjects it, he considers first the analogical arguments in its favour. (In this place I shall take the liberty of making use of a review which I wrote many years ago.) The hypothesis of a vital principle is allowed by its advocates not to admit of direct evidence; but they consider that collateral probabilities are in its favour, and that it is adequate to all the explanation required of it. An examination of the evidence put forth in support of these positions occupies the principal portion of the author's dissertation.

"Among the analogies," says Dr. Prichard, "adduced in favour of this doctrine, one has been already adverted to; I mean, that of electricity, or the operation of the electric or galvanic influence. It must be confessed that this analogy is so vague and indefinite as to afford scarcely a shadow of probable evidence. There is nothing in it on which the mind can lay hold with a clear and distinct apprehension."

Another analogy, and even more remote than the former, has been derived from the immaterial soul. The existence of this principle has been conceded on inferential grounds only, and the believers in a vital principle claim a similar allowance for their doctrine.

They urge that if a soul or immaterial entity is allowed, because it is necessary to explain mental phenomena, the existence of a vital principle ought to be conceded, because it is no less essential to the production of organic phenomena. Dr. Prichard, however, shows that the two doctrines are founded on premises that have no analogy whatever. Thus the immateriality of the soul is argued from the utter diversity of mental from material phenomena, from their being contemplated by internal consciousness instead of external sensation, from their indivisibility as contrasted with the infinite divisibility of matter, and from the impossibility of resolving them into the component qualities of matter, a process which may be executed on every physical substance. But this kind of reasoning is perfectly inapplicable to the functions of an organized body. We are never made acquainted with these phenomena as with those of mind, by consciousness, but by the same means as reveal to us other physical objects.

"The whole sphere of agency ascribed to the vital principle is, therefore, within the region of matter and its attributes; and if its existence is capable of proof, it must be on grounds totally different from those on which we have proceeded with respect to the existence and properties of a soul or immaterial being."

In the above very brief abstract of this part of the author's argument, we have passed over a very masterly discussion of the question of materialism, in the fifth section. We beg particularly to direct attention

to his disposal of Dr. Priestley's well-known argument; viz., that the phenomena of mind, and those of matter, belong to the same substance, because the former are never seen but in conjuction with the latter. Dr. Prichard's reply is as follows:—

"The whole universe displays the most striking marks of the existence and operation of mind or intellect, in a state separate from organization, and under conditions which preclude all reference to organization. 'The universal mind,' says a distinguished philosopher (Dugald Stewart), 'though everywhere present, where matter exists, though everywhere moving and arranging the parts of matter, appears to do so without being united with matter as is the case with visible created beings. There is, therefore, at least one being or substance of that nature which we call mind, separate from organized body.'"

The answer is very ingenious, but does not appear to us to be completely conclusive. The manifestations of intelligence in the two instances are of different kinds. Dr. Priestley seems to refer to the actual manifestation of thinking and feeling properties, not of their effects merely; and to the non-appearance of such properties in action, excepting when they are connected with organized matter. But the manifestations of mind and intelligence in the works of creation are such as are afforded by the *results* of the operation of mind on matter; and although it is highly improbable that the mind which acted upon it was connected with organization, yet there is no

evidence to the contrary derivable from these signs; indeed they do not seem to us to indicate either the one view or the other. By the same reasoning, if, on a desert island, a tool or a piece of machinery were discovered, which furnished evident marks of the operation of human contrivance, there would be no intimation from this source alone, that the designing mind was, or was not, connected with a brain and nerves; the knowledge that the human mind acts in concert with an appropriate organization, would be the result of other kinds of experience. The evidence, then, of the Divine mind, is contained in the effects of its operations; and we are ignorant whether any organization is, or has been, made use of by this exalted principle. The evidence of human or animal mind is also contained in its effects; but we likewise know that it never produces these results, except in co-operation with the nervous system.

We may be wrong in this view, and it is suggested with diffidence; but even if it be correct, and the objection, founded on a different view of it, to Dr. Priestley's argument, be consequently weakened, there still appear to us to be sufficient reasons for rejecting the conclusion of the materialist. A certain collection of properties which we call mind, is never presented to our observation, except in connection with a collection of properties utterly dissimilar, which we designate organic matter; but it is not a legitimate inference from these premises, that the connection is one of *dependence*, not of *alliance* only. It is true, that when the organic phenomena are dissipated the

others also disappear; but if the existence of the latter in other beings than ourselves, can only be made known to us through the medium of the former, as by motion, speech, action, &c., how can we presume to say that the thinking principle was dependent upon that medium, merely because the latter was destroyed? A man suddenly struck blind might, with equally good logic, argue that, because he had always recognized the existence of the sun in connection with his eyes, and because an impairment of his visual organs had destroyed the perception of that luminary, the existence of the latter was, therefore, dependent on the former. This is not precisely Dr. Priestley's position; but supposing that we allow, that in consequence of mental properties being never manifested, except in connection with those of organization, they must, therefore, belong to the same entity or substance, what possible use can be made of such a conclusion? For what is an entity abstracted from its properties? Nothing: for nothing is the absence of properties. If materialists are satisfied with the possession of this conclusion, we are well satisfied, for our own parts, to concede it to them; and do not care to prove that the two classes of properties belong to separate entities, or nothings. This view will appear satisfactory only to those who can discard from their minds the notion of there being necessarily a substratum of properties. We consider this substratum only as a term expressing the *collection* of certain properties, and have elsewhere endeavoured to illustrate the subject by saying that

"the prismatic rays, incapable of independent existence, belong to the substance light, which, in its turn, cannot exist without them; and thus properties are attached to substance, which is itself made up of those properties." The difficulty in receiving this opinion is produced, in a great measure, by the term *property*, which expresses relation to something else. But the analysis of properties shows them to be only expressions of various kinds of experience, which are grouped in various relations, and divided into two great classes, the former of which, we are told by instinctive belief, are the result of a causation external to our own identity, while the latter have their origin within ourselves; the one constituting what is called matter, the other what is called mind. The two are thus felt or experienced to be independent of each other, and no evidence can go higher.

After disputing the analogical evidence set up by the advocates of a vital principle, the author proceeds to examine the other argument adduced in its favour, to wit, that the functions of living beings can be explained only by the hypothesis in question. We cannot follow the refutation, as it would lead us into too many details. The result is, that the doctrine is not only inadequate to the interpretation of the facts, but also injurious to a philosophical inquiry into them, by allowing us to stop short of an ultimate analysis of complex phenomena, in the same manner as the old physiologists ceased to inquire further into the process of digestion when they had stumbled upon a *vis concoctrix*.

The work concludes with an interesting dissertation on the mental faculties. An attempt is made to distinguish those which require the instrumentality of nervous structure for their operation from those which are independent of it. But we do not think the attempt at this distinction a successful one.

I now proceed to notice the more strictly professional writings of Dr. Prichard. Of this class the earliest was (1822) "A Treatise on Diseases of the Nervous System," founded on cases observed in his practice at Saint Peter's Hospital. The main object of this work was to assist the discrimination and classification of those secondary forms of nervous disorder which spring from remote organs, and which, in the language of Dr. Marshall Hall, comprise the nervous diseases produced by eccentric irritation. The diseases particularly described were Epilepsy and Mania. And he distinguished their forms, as arising,—(1.) From irregularity of the functions of the uterine system. (2.) From disorder of the alimentary canal. (3.) From hepatic disorders. (4.) The idiopathic or cerebral form. These forms were happily described and were illustrated by a large number of instructive cases. Although the author took no credit to himself for originality in ascribing many cases of nervous disorder to faults in the organic functions, yet it was plain that no one before him had so well discriminated the different kinds, and referred them to their appropriate causes. The work added greatly to Prichard's reputation, and it had the honour of being translated into German.

The next professional writings were the articles in the "Cyclopedia of Practical Medicine," comprising, Delirium, Hypochondriasis, Insanity, Somnambulism and Animal Magnetism, Soundness and Unsoundness of Mind, and Temperament. Of these the largest and most important was the article Insanity. It was afterwards expanded into a separate treatise, which will always be a classic in this department of medical literature. Its most striking feature was the discrimination of that form of mental derangement which is now known as Moral Insanity. M. Pinel had described mania without delirium, consisting of ungovernable fury without any delusion;* but he had not pursued the subject farther. Dr. Prichard had the great merit of proving the existence of insanity without marked intellectual aberration.

I shall never forget the satisfaction I derived from the study of the article Insanity, in the Cyclopedia; and the light which I then derived from it has repeatedly been a help and a guide to me in the investigation of cases of derangement in which no lesion of judgment was discoverable. On looking over the work on Nervous Diseases lately, I was surprised to find that on this subject Dr. Prichard had quite changed his views; for in this treatise, when noticing Pinel's "Mania sine delirio," he threw doubts on the existence of such a morbid condition of mind, and intimated the probability that there might be latent delusion giving origin to the disordered feelings. Subsequent inquiry and observation led him to

* He termed it "Emportement Maniaque sans délire."

alter his views, and, as I have said, to extend the morbid condition far beyond the limits sketched by Pinel. I shall beg permission of the Society to dwell somewhat on this point, as it is one of high importance to us as medical practitioners, as well as being connected more than any other practical subject with the name of Dr. Prichard. It seems to me strange that when we reflect on the large share which the emotions and sentiments and passions bear in the mental constitution of man (a fact conceded by all who have speculated upon this branch of philosophy), and when we consider that there has been no disinclination to attribute susceptibility of separate and independent derangement to another part of our constitution, I mean the purely intellectual; and moreover that the most strenuous asserters of the doctrine, that insanity, in all cases, involves a perversion of judgment, do not attempt to conceal that the propensities, tastes, and emotions, are often, or indeed in most cases, morbidly affected; I say it seems strange that the question should not have presented itself before, as to whether there are not actual cases in which mental derangement is confined to the moral feelings and the emotions, just as in other cases the perceptive and reasoning powers are the sole subjects of disorder; and stranger still, that, whether such *a priori* suspicions ever arose or not, the real existence of such cases should not have attracted observation. That they have been so entirely overlooked can only be explained on the ground that the sentiments and passions of man have been generally con-

sidered subservient to the will and reason, and that any undue excitement of the former (the passions) has been consequently supposed to arise either from a criminal want of controul on the part of the will, or from a deficiency of rational power; so that, according to this view, a man of violent passions or eccentric conduct, unless proved to entertain some delusion or hallucination, must be either wilfully perverse, or chargeable with moral delinquency.

Now, as to the slighter forms of moral insanity, as distinguished from intellectual, the subjects of them may perhaps have passed through life without producing a conviction that they were actually mad, and yet they have exhibited such eccentricities of demeanour, such waywardness of conduct, and peculiarity of temper, as to have occasioned no little concern on the part of their friends. Such persons have often inherited a tendency to insanity, have at former periods of their lives been unquestionably insane, or have suffered inflammatory affections of the brain. The characteristic distinction of such cases is that, notwithstanding the strangeness of their habits and conduct, they never betray any delusion; any belief, for instance, in things morally or physically impossible, or at variance with the general opinion and common sense of mankind; nor do they manifest any deficiency of reasoning power; they will even display great ingenuity in accounting for the eccentricities of their conduct, and in explaining and justifying the state of moral feeling under which they appear to exist. Sometimes the derangement is

manifested not so much in peculiarity of conduct as in a preter-natural excitement or depression of the spirits. The latter is one of the most frequent forms of the complaint. A person is overwhelmed with despondency, and though possessed of every requisite for happiness, can take no pleasure in any thing under the sun. In other cases there is a preter-natural elevation of the spirits, an uncontrolable vivacity, an incessant restlessness, a desire to undertake great enterprises, and an everlasting disposition to talk loudly and boisterously, without proper regard to place or time or person. Upon the tendency which the morbid dejection manifests to become involved in religious subjects, Dr. Prichard makes the following observations:—"In examples of a different description, the mental excitement which constitutes this disease is connected with religious feelings, and this is often the case when the period of excitement has been preceded by one of melancholy, during which the individual affected has laboured under depression and gloom, mixed with apprehensions as to his religious state. A person, who has long suffered under a sense of condemnation and abandonment, when all the springs of hope and comfort have appeared to be dried up, and nothing has been for a long time felt to mitigate the gloom and sorrow of the present time, and the dark and fearful anticipations of futurity, has passed all at once from one extreme to another; his feelings have become of a sudden entirely changed; he has a sense of lively joy in contemplating the designs of Providence towards him, amounting some-

times to rapture and extacy. Such a change has been hailed by the relatives of the individual thus affected, when they have happened to be pious and devout persons, as a happy transition from a state of religious destitution to one of acceptance and mental peace; but the strain of excitement is too high, the expressions of happiness too extatic to be long mistaken; signs of pride and haughtiness are betrayed, and of a violent and boisterous deportment, which are quite unlike the effects of religious influence, and soon unfold the real nature of the case; or it is clearly displayed by the selfishness, the want of natural affection, the variableness of spirits, the irregular mental habits of the individual. In the cases to which I have now referred there has been no erroneous fact impressed upon the understanding; no illusion or belief of a particular message or sentence of condemnation or acceptance specifically revealed; a disorder so characterised would not fall under the head *moral insanity*. The morbid phenomena in the cases of disease which I am now attempting to describe extend only to the state of the feelings and spirits, the temper, the preternaturally excited sentiments of hope, and fear, and the results which these influences are calculated to produce in the mental constitution."

Moral Insanity often presents violent anger as its most prominent phenomenon, at other times an inclination to theft, arson, or even homicide. Sometimes the most striking characteristic is a sudden change of disposition. There are many instances which show a transition from moral insanity to monomania.

On the whole, I cannot help viewing the subject as one of the most interesting in the whole range of morbid psychology. And it is impossible to think of it without having the mind filled with very melancholy reflections. The deprivation of reason, in the ordinary and most acknowledged forms of moody melancholy or of raving mania, has abundantly served the purposes of moralizers on the imperfection of human nature, or of such as have wished to exhibit the most startling pictures of human misery; and, in truth, no subject is more productive of horror, or more humiliating to pride. Yet the consideration of that perversion of the natural feelings, tastes, and habits, which constitutes moral insanity, introduces us to a wide world of human suffering, which, though it may not be peopled with such appalling apparitions as have risen before the imagination of poets, and been embodied into the undying forms of Orestes, Ajax, and Lear, yet swarms with unhappy beings; sufferers whom we view not in those throes of anguish which by their novelty throw an air of elevation or sublime indistinctness over their subjects, but in the ordinary habit of the mind, in the quiet paths of life, in the domestic chamber, and by the friendly hearth. The maniac, and the melancholic, before their maladies have been recognised, may have inflicted severe pangs on the minds of affectionate friends and relatives—for few ears are impassive to the mournful discord of "sweet bells jangled out of tune"—and their removal from society may have left blanks which can never be so well filled; but in their retirement they are fol-

lowed by feelings of tenderest compassion and regret, as those who have been visited with the sorest chastisement of heaven. Alas! how different the fate of those whom it has pleased Providence to afflict, not with aberrations of judgment, which are detected by even the simplest of sound-headed observers, but with marked obliquities of feeling, which are so easily confounded with bad passions wilfully indulged, and with evil habits wilfully pursued. In childhood, to suffer a constraining, torturing discipline, intended to controul a waywardness, the root of which is beyond the reach of the most anxious parent, or the most persevering educationist; in youth, to be marked for incorrigible vice, or for a perverseness which incapacitates for any important occupation:—in manhood, to be despised and hated for singularities of manner and conduct; to scatter confusion and dismay over a once happy household by the development of unworthy passions, and intolerable irregularities of temper; to distract an affectionate and honourable wife by strange suspicions, and unfounded jealousies; to harass the timid child by irritability, violence, and tyranny, which no tender submission can appease, no fond attentions can mitigate; to plunge helpless dependents into ruin and beggary; and in all these several conditions to be considered a person fully responsible for his actions, and as capable of subduing evil tendencies as are other people:—these are but a few of the miseries incident to the victims of the malady in question, and however inferior they may appear in the picturesque to maniacal and melancholic visitations, they are productive of far more sorrow to the individual, and of far more lasting and wide-spread distress to those around him.

Dr. Prichard published, in 1842, a small volume, "On the Different Forms of Insanity in relation to Jurisprudence, designed for the use of persons concerned in legal questions regarding unsoundness of mind." It is an extremely useful manual for the purpose, conveying the distinctions laid down in the larger work in a more popular form, mixed with rules for the guidance of the medico-legal practitioner.

In 1831 a very interesting practical paper, from his pen, appeared in the Medical Gazette, giving an account of a new mode of applying counter-irritation in diseases of the brain. It consisted in making an incision of the scalp along the sagittal suture, and keeping the wound open by means of peas, as in an ordinary issue. It was entitled, "On the Treatment of Hemiplegia, and particularly on an important remedy in some diseases of the Brain." The subject was renewed in a paper read before the British Association of Science at the meeting held in Bristol, in 1836. I had the honour of reading it for the author, and I well remember the very great interest it excited among the members of the medical section, among whom were some of the most distinguished physicians and surgeons of the United Kingdom.

Were I to enumerate all his smaller compositions, both on professional and general topics, the list would be a very long one, for he contributed largely to many periodical journals and reviews. Enough has

been said to show the extent and variety of his learning; yet I cannot refrain from recording that, in 1815, he translated, jointly with Mr. Tothill, Muller's Universal History; that he rendered the Birds of Aristophanes into English verse; that he studied Biblical criticism profoundly, and made many translations from the Hebrew Scriptures.

Perhaps it would be more prudent were I now to content myself with having related the principal events and achievements of Dr. Prichard's life. The hand of a more experienced artist would be requisite even to sketch such a mind and character; much more to attempt, by a skilful adjustment of light and shade and gradation of colour, to give a faithful portrait of the eminent subject of this memoir. Yet it would be hardly respectful to leave my task without endeavouring to give some idea of the original, though it may prove to be only a rude likeness, drawn by the hand of a friend.

In Dr. Prichard were recognised, of course, all those attributes which belong more or less to men who are distinguished among their fellows by intellectual power. The mere fact of his having been able to produce such works as bear his name, tells what endowments he possessed; but were I to endeavour to present what was most characteristic of his intellect, I should say it was largeness of capacity, united with readiness of command over his resources. All men of powerful minds have strong memories, for memory is the feeder of the other faculties: even if

originally robust, these must pine and languish unless maintained by the nutriment which the former supplies. But Dr. Prichard's memory was above the average, even for one of his general mental caliber. His perceptions were by no means defective in acuteness, yet it was not by acute observation that he was particularly distinguished; nor though his judgment was sound and accurate, should I say that this faculty was so prominent as to be singled from the rest as one of his characteristics. Had he been engaged in the legal profession, I think he would have shone particularly in collecting and methodically arranging, and in luminously and eloquently stating an immense mass of evidence bearing upon a particular point; not, however, in the spirit of a mere advocate or partisan, but as one whose mind, magnetised by a particular idea, attracted and assimilated to itself every thing that could give support to that idea. It was not a mind to produce a mere agglomeration of facts and notions, but one that impregnated, informed, and organized them all into one living whole. Yet, had he been placed on the bench, I think he would not have been remarkable for mere judicial qualities, such as made Tenterden and Eldon so eminent. Comprehensiveness, rather than subtlety, was the character of his understanding. In conversation he showed his preference to broad decided views rather than to the fine-drawn distinctions, the hair-splittings of metaphysical analysis. Yet in his writings it will not appear that his mind was warped by a foregone conclusion. Few

compositions give one a stronger impression of fairness and equity in weighing evidence.

Fancy and imagination were not prominent faculties in Dr. Prichard. He was never at a loss for a suitable illustration to enrich his style, which was affluent as well as terse and vigorous. Yet there was not that conscious enjoyment in the pursuit of analogies and likenesses, which belongs to men in whom the faculties I have adverted to are strongly marked. And, correspondently with this, I think that he had no decided æsthetical tendency, no such sensibility to the beautiful as would lead him to dwell on the enjoyments of poetry and the fine arts; though he was too much of a scholar, and in every way too well informed, not to be able to converse on these subjects. A powerful memory, and a strong philosophical bias, by which I mean the disposition to trace events to their causes, and to classify phenomena under general laws, together with an astonishing capability of undergoing mental labour, will, I think, be found to have been the most distinguishing traits of Dr. Prichard's understanding.

In the moral department of his character, high—nay, highest integrity and honour, and an utter abhorrence of whatever even bordered on the mean and truckling, were united with general benevolence and with strong domestic affections. He was by no means prone to suspicion of motives, and was, perhaps, too easy in the admission of testimony, so that his ears were sometimes open to the first informant

on any subject, and he thus might receive impressions which afterwards had to be corrected. The freedom from assumption in his ordinary life and demeanour was very remarkable. The simplicity, and all but diffidence of manner displayed in company, where his intellect far overtowered that of others, could not fail to strike observation. He would converse with persons infinitely his inferiors in mind and attainments, as if they were on the same level with him, asking their opinions in connection with subjects upon which he might have dictated to the whole republic of science.

Persons familiar with his works would not be surprised to hear of the prodigious amount of erudition which would come out in conversation. It was no matter how remote the subject might seem to be from the pursuits of a physician; he would unroll such stores of information upon it, as might be expected of a man who had devoted his whole time and attention to it. He was fond of discussion, and would sometimes, for the sake of amusement, support views that were paradoxical, or maintainable solely for the sake of argument; yet he was quite free from dogmatism, or anything like an overbearing tone. If a person of more assurance than knowledge were discoursing or arguing in an unbecoming manner, Dr. Prichard, instead of vehemently assailing him, might ask one or two questions, *more Socratico*, which sufficed to deprive the pretender both of his false position and of his presence of mind; but he would be the first to

try to help the defeated disputant out of his disgrace and confusion. Every one left his society impressed, as much by the modesty of the great man, as by the marvellous extent of his knowledge.

As a physician he was distinguished, not only by his extraordinary natural powers, and by the extent of his professional attainments, both scientific and practical, but also by the earnestness with which he devoted himself to his duties, and by his kind and considerate condnct towards his patients. He weighed their symptons anxiously, and was most conscientious in carrying out the appropriate treatment. He was particularly successful with cases that required a decided uncompromising line of action; and his boldness, consistency, and fearlessness met with their best rewards. Of the little matters of detail that must have their share of attention in many cases, he was rather impatient. He liked in practice, as in other matters, broad views rather than a fine analysis of symptoms and minutiæ of treatment. Many of my present hearers had the privilege of knowing Dr. Prichard, as an associate by the bedside. And I confidently assert, in their presence, that there never was a man whose conduct towards his professional brethren was more strictly upright, honourable, kind, and considerate. "*Quid dicam de moribus facillimis? bonitate in suos? justitid in omnes? hæc nota sunt vobis.*"

In his moral constitution, reverence was very prominent. It showed itself in the value which he attached to the opinions and authority of really great

men, and more especially in his sentiments towards the great First Cause. Those who had but very slight communication with him must have felt assured that nothing could ever have proceeded from him disparaging to the interests of religion; and no one knew him intimately, without being aware of the strong influence which piety maintained over his mind, and how it actuated all his conduct. His opinions, during the greater part of his life, were in strict conformity with the doctrines embodied in the book of Common Prayer.

Dr. Prichard was in stature rather below the middle height, and of rather slight make. He had light hair, and grey eyes, which, though somewhat small, were of singularly intelligent expression. The form of his head was very fine; broad and prominent in the forehead, lofty and capacious in the crown. The countenance, to the most superficial observer, betokened deep thoughtfulness, with something of reserve and shyness, but blended with true kindliness. His voice was rather weak and low, but very distinct in articulation. His manners and deportment, as I have already remarked, were simple and unaffected;—and in general company he evidently spoke with effort or even reluctance, unless upon subjects of business or of scientific and literary interest.

His last illness was one of great suffering. A few days before its termination he became conscious that his earthly career was drawing towards its close, and he awaited the event with the resignation and calmness that befitted a Christian philosopher. Though

he had not ceased from his labours, nay, the sickle was in his hand when it drooped, few could so well have said, though he would have been the last to say it, "I have not lived in vain." If one could venture in imagination to follow the musings of that departing spirit, one might conceive the satisfaction with which he looked back on his well-spent life. He had not to regret the consumption of precious hours in the pursuit of sensual gratification, nor yet in more refined enjoyment; neither in "lordly ease," nor in "learned leisure." Youth had found him assiduous in acquiring truth and knowledge; manhood and advancing age had witnessed untiring exertions in a profession, which, whatever it may produce to the practitioner, is, if grounded on adequate knowledge, an employment pre-eminently useful to his fellow creatures. And the intervals in those avocations, instead of having been set apart, as they might innocently have been, for recreation and amusement, had been filled up with labours, which, had he done nothing else, would have enabled him to bequeath honour to his family, as the inheritors of his renown, and lasting benefits to mankind of the highest order; for I know not what gifts can surpass those of truth and wisdom. As the death-shadows began to gather over the spirit, which till it was extinguished could not but be still "looking before and after," the memories of his noble and useful labours might have loomed large before his dimming vision, mingled with recollections of happy hours passed in that loving domestic circle, over which his benign and gentle disposition shed peace and con-

tentment. And one fancies that with such remembrances he might well say, *Nunc dimittis*. But his mind, originally so humble, and so chastened and purified by religious principle, was far more likely to have spent its last moments, not in contemplating what he had done, but what he had left undone; thinking whether he could render a good account of his stewardship of those remarkable talents with which his Maker had endowed him; reposing on infinite goodness; and aspiring to a blessed state of being for which this mingled life of joy and sorrow, hope and disappointment, is but the preparation and discipline. I doubt not that the deeds of his life, which to us look large and brilliant, before *his* failing sight shrank small and dim, and that his soul, which no earthly vision could content, much less the contemplation of his own doings, turned towards that Parent Source from which all its light had been drawn, and longed to be absorbed into its divine and immortal essence. But though, with that true modesty which belongs to the most gifted, because they are the most capable of measuring real virtue and greatness; which led Newton to liken himself to a little child picking up pebbles on the shore of an unexplored ocean; and which modesty, as I have said, was so remarkable in my lost friend, that I cannot choose but dwell upon it;—though he would have depreciated rather than magnified himself, we who look at him from without, and estimate him by the standards that enable men not only to recognize moral excellence, but to mete out

the degrees of their approval, cannot refrain from declaring that no spirit could pass more blameless and unstained from its mortal trial, none more fitted for the communion of the great and good, none more ready to appear

> " Before the Judge ; who thenceforth bade him rest,
> And drink his fill of pure immortal streams."

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PAMPHLETS

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