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THERAPEUTIC EXPLANATION AND THE EDINBURGH BLOODLETTING CONTROVERSY: TWO PERSPECTIVES ON THE MEDICAL MEANING OF SCIENCE IN THE MID-NINETEENTH CENTURY

by

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JOHN HUGHES BENNETT (1812-1875), the Edinburgh pathologist, microscopist, and professor of the institutes of medicine, remarked in a lecture delivered before a class of medical students in 1849:

> Whilst pathology has marched forward with great swiftness, therapeutics has followed at a slower pace. What we have gained by our rapid progress in the *science* of disease has not been followed up with an equally flattering success in an improved method of treatment. The science and art of medicine have not progressed hand in hand.[1]

Bennett's comment reflects the striking paradox of medicine in the first half of the nineteenth century: medical science was progressing at an unprecedentedly rapid rate, yet therapeutics – what the physician actually did to the patient – was in a troubling state of confusion. Moreover, the meaning of physiology and pathology for the treatment of disease was not at all obvious. Medical men disagreed widely on whether the application of the products of laboratory science, or of empirical observation, represented the best approach to advancing medical practice.

The disparity of physicians' perceptions of the proper relationships among scientific knowledge, medical theory, and therapeutic practice in the mid-nineteenth century emerged with striking clarity from the debate on bloodletting in Edinburgh.[2] The principal foci of the discourse on bloodletting that flourished in the mid-1850s were two species of therapeutic theory. One type of theory was designed to explain, justify, or guide therapeutic management; the second type of theory sought to explain why therapeutic practice had changed. The arguments physicians put forward in defending their therapeutic theories in the case of bloodletting reveal the sources of authority to which they turned in formulating and validating therapeutic theory and illustrate clearly their conceptions of the medical role of laboratory science.

Two distinct perceptions of the proper nature of medical theory and of the medical relevance of scientific knowledge are clearly discernible in the discussion of bloodletting. Most Edinburgh physicians looked to the bedside as the locus of both generation and validation of therapeutic theory. Justification for therapeutic management derived principally from observations of the effects of therapeutic

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[1] John Hughes Bennett Papers, Gen 2007/, Edinburgh University Library, 1848. This collection, containing materials dated from 1841 to 1872, includes Bennett's manuscript lecture notes on the institutes of medicine, manuscript drafts of many of his published works, other lecture notes, and some correspondence.

[2] Lester S. King discusses the course of this debate in his valuable study, 'The blood-letting controversy: a study in the scientific method', *Bull. Hist. Med.*, 1961, **35**: 1-13.

operations on the clinically perceived symptomatology. Therapeutic change and progress, they further maintained, stemmed from attentive clinical observation. A quite different posture was proselytized by the clinician and teacher John Hughes Bennett. In contrast to most of his medical colleagues, Bennett looked to the physiology and pathology laboratory as one locus of authority for both constructing and testing therapeutic theory and the practice it defined. Bennett was intellectually and emotionally willing to ascribe some measure of therapeutic authority to laboratory science. Theory informed by pathophysiological knowledge acquired in the laboratory could explain and even guide action at the bedside, Bennett believed, while prior theory could be affirmed or invalidated by criteria generated by laboratory research. Similarly, advances in scientific knowledge about disease could generate therapeutic change and progress. The discussion of bloodletting in Edinburgh, evincing the disparities between these two perspectives on the relationship between scientific knowledge and medical theory and practice, provides a useful context for evaluating the cognitive and social construction of therapeutic explanation in the mid-nineteenth century.

EPIDEMIOLOGICAL CHANGE AND THERAPEUTIC CHANGE

Bloodletting had declined markedly in the practice of Edinburgh physicians since the early 1830s. Nevertheless, the 1850s witnessed an animated debate in which most members of the profession intransigently defended the theoretical propriety and the principle of bleeding patients. The course of bloodletting's clinical decline and the relationship of this to epidemiological changes in Scotland provide a solid backdrop for evaluating the ways physicians fashioned their explanations for changes in practice and identifying the sources of authority to which they turned in constructing and testing their theories. The relationships between theory and practice exemplified by bloodletting's career with respect to epidemic fevers in the first half of the nineteenth century makes it clear why discussion of a relatively infrequently used therapy commanded such a prominent position in medical attention in the mid-1850s.

Following widespread epidemics of typhus during the first three years of the nineteenth century, the British Isles enjoyed a relatively epidemic-free period from 1803 to 1817. Endemic cases of "typhus" were characterized by debility, a slow gradual pulse, low temperature, and an enfeebled constitution. Physicians perceived endemic typhus as an asthenic (*sthénos* = strength) or enfeebled condition that indicated support with nourishment or stimulation with alcohol, cold water affusions, and diaphoretics.[3]

This epidemic-free period ended abruptly in the autumn of 1817. A widespread epidemic of what was termed "relapsing" or "famine" fever followed a period of severe crop failure, unemployment, Irish immigration, and famine. Unlike the enfeebled or mild symptomatology of typhus, this new fever was characterized by overexcitement of the patient's system, a hard fast pulse, high temperature, and delirium. Relapsing fever

[3] W. Alison, 'Types of fever and blood-letting', *Br. med. J.*, 1865, i: 624-625, p. 624; Charles Creighton, *A history of epidemics in Britain*, 2 vols., [2nd ed., London, 1894], reprinted London, Frank Cass, 1965, vol. 2, pp. 162-163; Charles Murchison, *A treatise on the continued fevers of Great Britain*, 2nd ed., London, Longmans, Green, 1873, p. 31.

was regarded as a highly inflammatory, phlogistic fever, which was classified under the genus *synocha* of William Cullen's nosology. Significantly, physicians conceived typhus and relapsing fever to be merely different forms of the same fever, but whereas the former was clinically perceived to be asthenic, the latter was clearly sthenic.[4]

The change in fevers from an asthenic to a sthenic constitution also signalled a shift in therapeutic practice. Early in the relapsing fever epidemic physicians employed the fever therapies they had used for typhus during the past decade – wine, cold water affusions, and sweating – but apparently these were of no avail. "A fever with such a vivid reaction", an Edinburgh practitioner later wrote, "demanded some sedation. Bloodletting, the most powerful and certain of all, was resorted to."[5] Bloodletting, which was called for in such sthenic conditions in which the patient was highly compromised, slowed the pulse, lowered the temperature, alleviated delirium by lowering the temperature, and relaxed the patient so that he could sleep. Physicians, indoctrinated in their medical training to beware the "terror of debility", at first were reluctant to embrace bloodletting,[6] but within a few months after the start of the epidemic copious bloodletting had come to be used in treating almost all cases of fever.[7]

This epidemic of relapsing fever lasted for four years; the next epidemic of relapsing fever occurred between 1826 and 1829, and again bloodletting was the treatment of choice. Patients were bled until they fainted, generally losing between twenty and twenty-four ounces of blood in each therapeutic encounter.[8] One physician who had been a medical student during this period later recalled that "there was hardly a morning that some twenty or thirty unfortunate creatures were not phlebotomized largely. The floor was running with blood; it was difficult to cross the prescribing hall for fear of slipping. Patients were seen wallowing in their own blood, like leeches after a salt emetic."[9] "Never", one medical man later summed up, "has more blood flowed from the veins and arteries of mankind, under the authority of medicine."[10]

But when the next major fever epidemic broke out in 1831, typhus rather than relapsing fever prevailed. The epidemic constitution had reverted to an asthenic type.

[4] Robert Christison, 'On the changes which have taken place in the constitution of fevers and inflammations in Edinburgh during the last forty years', *Edinb. med. J.*, 1858, **3**: 577-595, and 1858, **4**: 38-58, pp. 580-587; Murchison, op. cit., note 3 above, p. 32.

[5] Christison, op. cit., note 4 above, p. 580.

[6] 'Critical analysis. Thomas Mills, *An Essay on the Utility of Blood-letting in Fever* (Dublin, 1816)', *Edinb. med. J.*, 1817, **13**: 363-374; Murchison, op. cit., note 3 above, p. 42; Peter H. Niebyl, 'The English bloodletting revolution, or modern medicine before 1850', *Bull. Hist. Med.*, 1977, **51**: 464-483.

[7] One Edinburgh practitioner later recalled that "within a few months afterward blood-letting, which had been brought into vogue by the prelections and example of Dr. Gregory, then Professor of the Practice of Physic in our own university, attained its highest reputation and widest range in the care of diseases at large" (Christison, op. cit., note 4 above, p. 580).

[8] Ibid., pp. 587-588; William Pulteney Alison, 'Observations on the epidemic fever now prevalent among the lower orders in Edinburgh', *Edinb. med. J.*, 1827, **28**: 233-263; *The life of Sir Robert Christison, Bart.*, edited by his sons, 2 vols., Edinburgh, Blackwood, 1885, vol. 1, pp. 149-156; Murchison, op. cit., note 3 above, p. 45.

[9] William Stokes, quoted in William Orlando Markham, *Bleeding and change of type of disease*, London, J. Churchill, 1866, p. 18. Another Edinburgh practitioner recalled that when he began practice in 1822, "the lancet was in great vigor, and a well-employed medical man almost lived in a stream of blood. *'Vigorous practice'* was the order of the day" (Andrew Combe, 'On the observation of nature in the treatment of disease', *Br. for. med. chir. Rev.*, 1846, **21**: 505-524, p. 514).

[10] Markham, op. cit., note 9 above, p. 18.

At the beginning of the epidemic physicians employed copious bloodletting. However, they found that their patients could not sustain free venesection and fainted after losing two or three ounces of blood, and therefore physicians turned to supportive and stimulative therapeutics like alcohol.[11] Epidemics of typhus continued to afflict Britain for the next few decades, and depletion by bloodletting was seldom practised.[12] An attending physician at the Royal Infirmary of Edinburgh later wrote that in the fever epidemic of 1831-33, "I altogether gave up blood-letting in fever. Nor has it ever been revived since .... There has never been again that high state of inflammatory reaction of the circulation which formerly made the treatment of blood-letting useful and practicable."[13] Thus these changing epidemiological patterns were closely linked to changes in therapeutic practice, and the clinically perceived shift of the epidemic constitution in the early 1830s from a sthenic to an asthenic type was accompanied by a decline of bloodletting for contagious fevers.

By the mid-1850s the use of bloodletting had declined substantially in treating not only epidemic fevers but also internal inflammations like pneumonia. One prominent physician later commented: "We can hardly conceive of a revolution in practice more complete. Venesection is now, from being the most frequent, the rarest of operations".[14] Yet this "revolution" in *practice* was not accompanied by a decline in the value of bloodletting in *theory*. Reigning medical theory upheld the principle of bloodletting, and thus the same physicians who rarely employed the lancet at the bedside could still maintain that bloodletting was in principle "one of the grandest agents in the practice of medicine".[15] Contemporary observers noted that leading physicians continued to advocate bloodletting in their published writings long after they had rejected it from their own practices.[16] The theory-practice connexion for bloodletting apparently was severed as a discrepancy emerged between the dictates of medical theory and actual clinical practice.

THE CHANGE-OF-TYPE THEORY: A DEFENCE OF THE PRINCIPLE OF BLOODLETTING

Both intellectual and social factors impeded the rejection of bloodletting in theory. Existing theory remained unchallenged largely because of the absence of a competing theoretical schema capable of drawing the existing paradigm into question. Moreover, few physicians were anxious to announce to the public or admit to themselves that they and their predecessors had for years followed a theory that dictated an incorrect, worthless, and possibly injurious practice. Most of the leaders of the profession in the 1850s had practised medicine during the sanguinary days when bleeding was in

[11] Christison, op. cit., note 4 above, p. 589; Creighton, op. cit., note 3 above, vol. 2, pp. 189-190. On the subsequent rise of alcoholic therapeutics to vogue and the controversy this mode of therapeutic management endangered, see John Harley Warner, 'Physiological theory and therapeutic explanation in the 1860s: the British debate on the medical use of alcohol', *Bull. Hist. Med.*, [forthcoming].

[12] Creighton, op. cit., note 11 above, pp. 192-203; Murchison, op. cit., note 3 above, pp. 48-50.

[13] *Life of Sir Robert Christison*, op. cit., note 8 above, vol. 1, pp. 150-151.

[14] William Stokes, 'The address in medicine', *Br. med. J.*, 1865, ii: 133-142, p. 134.

[15] J. A. Hingeston, 'The neglect of the use of bleeding in the treatment of some of the milder ailments', *Ass. med. J.*, 1854: 266-267, p. 266. See Robert Hardey, 'On the general disuse of venesection in the treatment of acute disease', *Br. med. J.*, 1863, i: 461-464, p. 462.

[16] William Orlando Markham, 'Remarks on the uses of bloodletting in disease', ibid., 1850: 284-287, 307-308, p. 285.

therapeutic fashion before the 1830s. Furthermore, Edinburgh physicians in the mid-nineteenth century, practising at a time when Edinburgh's pre-eminence as a medical centre had declined sharply, enjoyed an intellectual and emotional identification with the Edinburgh tradition; yet plainly many of the leading figures in that tradition had extensively bled their patients. "The thinking man", remarked one physician, "finds it hard to believe that the fathers of British medicine were always in error, and that they were bad observers and mistaken practitioners."[17] Rejection of bloodletting's theoretical value – its decline in practice notwithstanding – entailed a challenge to the principle of bloodletting which most Edinburgh medical men perceived as a threat to the profession's status, jeopardizing the profession's public confidence, respect, and sanction of authority.

The coupling of status anxieties with intellectual constraints forced Edinburgh physicians in the 1850s into the ambiguous and disconcerting position of claiming that the paradox between their theory and practice *vis-à-vis* bloodletting presented no genuine contradiction. Defence of this position demanded an explanation for the change in practice that would affirm the principle and theoretical validity of bloodletting, support the notion that the extensive bleeding of patients in prior practice had been prudent and correct, and maintain the practical and theoretical propriety of the contemporary disuse of the lancet. Moreover, a satisfactory theory explaining the change in practice had to be grounded upon a stable source of therapeutic authority that the profession would widely acknowledge, that is, empirical clinical observation. Physicians found an explanation for the change in practice that met these criteria in the change-of-type theory of disease.

The change-of-type theory was not wholly new in the mid-nineteenth century.[18] However, William Pulteney Alison (1790-1859), the elder of Edinburgh medicine, gave the theory its canonical form in the early 1850s.[19] Alison argued that since the early 1830s all inflammatory diseases, including epidemic fevers and sporadic internal inflammations like pneumonia, had changed from a sthenic type to an asthenic type. The sthenic type of internal inflammations that predominated before the 1830s exhibited a "phlogistic diathesis" which demanded depletive therapies like bloodletting, whereas the asthenic internal inflammations that prevailed after the early 1830s were of an enfeebled or "typhoid character" that would not sustain bloodletting, requiring in its stead supportive or stimulative treatment. Inflammatory disease had changed in type, and therapeutic practice had changing accordingly. Thus, the theory's advocates argued, the belief that bloodletting was the best remedy for sthenic disease was correct, but because disease had changed to an asthenic type, bloodletting seldom was called for after the early 1830s. This theory had the singular advantage of explaining the decline of bloodletting in practice while at the same time maintaining

[17] Stokes, op. cit., note 14 above, p. 135.

[18] Christopher Johnson, 'On change of type', *Edinb. med. J.*, 1870, **15**: 1054-1055. For an eighteenth-century example of this species of explanation for therapeutic change, see John Hunter, *A treatise on the blood, inflammation, and gun-shot wounds*, London, George Nicol, 1794, p. 227.

[19] Alison developed the theory in *Cases illustrating the asthenic form of internal inflammations now common in this country*, Edinburgh, Sutherland & Knox, 1852; 'Notice of cases of pleurisy and pneumonia in the clinical wards of the Royal Infirmary in summer 1850, – being the substance of two clinical lectures delivered on 7th May and 18th June', *Mthly J. med. Sci.*, 1850, **11**: 157-173; and 'Observations made on some [c]ases in the clinical wards of the Royal Infirmary, Edinburgh, November 1850', ibid., 1851, **12**: 71-78.

that it was in principle a most valuable therapy. British physicians widely took up the change-of-type theory in the 1850s and 1860s – especially following a serious theoretical challenge to bloodletting posed in 1855 – elaborating it and using it as their principal explanation for the change in therapeutic practice.

Alison claimed that three sorts of evidence had brought about his allegiance to the change-of-type theory. First, he proposed that since physicians generally acknowledged that a shift in epidemic fevers from sthenic relapsing fever to asthenic typhus had taken place in the early 1830s, it was reasonable to conclude that sporadic diseases like pneumonia had also changed in type.[20] Second, he argued that his own empirical observation of clinical patterns gradually had convinced him that internal inflammations had assumed a more adynamic, asthenic type than formerly.[21] And third, Alison asserted that the unexpected success of contemporary homeopathic treatment of pneumonia, constituting a completely venesection-free therapeutic trial, indicated that internal inflammations must have changed to a form that responded favourably to treatment by regimen alone.[22]

Other physicians affirmed Alison's formulation of the change-of-type theory by bringing forward their own recollections of having observed the postulated shift in disease in their own practices. They further argued that such former leaders of Edinburgh medicine as James Gregory were astute clinicians who could not have erred in bleeding patients for pneumonia before the 1830s.[23] Both the considerations that conducted Alison to the change-of-type theory and the arguments other physicians put forward to confirm his position drew upon observation at the bedside as their source of authority.

A second version of the change-of-type theory proposed that not disease itself but rather the constitution of the human body had undergone a change since the early 1830s to an asthenic or enfeebled type that could no longer withstand bloodletting. "At different epochs", wrote one physician, "and under the potency of some intangible and mysterious agency, human bodies assume different conditions of sthenia and asthenia – different states of par and impar."[24] Another practitioner suggested that perhaps the potato blight or the increasing social use of alcohol may have brought about the change. "But", he added, "I am more inclined to believe that the too frequent use of obnoxious tobacco, is [the] one great cause of enfeeblement of the system; for men and women smoke, and when women do not smoke how frequently do they inhale the fumes from their husbands' pipes in their own apartments."[25] An Edinburgh physician attributed the change-of-type to "the influence on the human frame of a town life – of

[20] Alison, 'Notice of cases of pleurisy', op. cit., note 19 above, p. 165. Although most physicians recognized that the change-of-type stemmed from about the time of cholera's first appearance in Britain in 1832, few suggested that there was any connexion between cholera and the shift of disease to an asthenic type. They perceived cholera as something external or foreign that had been superimposed upon Scottish disease patterns.

[21] Alison, 'Cases illustrating the asthenic form', op. cit., note 19 above, p. 495.

[22] Ibid., p. 507; *idem*, 'Notice of cases of pleurisy', op. cit., note 19 above, p. 162.

[23] See, for example, Henry Kennedy, 'On the change of type theory of disease', *Edinb. med. J.*, 1859, **4**: 624-640, p. 627; J. A. Symonds, 'On change of type in disease', *Med. Times Gaz.*, 1866, **ii**: 368-370, p. 368; 'Transactions of the Medico-Chirurgical Society of Edinburgh', *Edinb. med. J.*, 1856, **1**: 947-953, p. 950.

[24] Markham, op. cit., note 9 above, p.iv.

[25] William Norris, 'Is there a change of type in disease?', *Br. med. J.*, 1867, **i**: 81-82, p. 81.

an indoor existence – of a tea and coffee diet – of preambulators, and the travel-made-easy rail – and of head-work instead of limb-work."[26] Most medical men ascribed the change-of-type of the body to the destructive effects of urbanization on the human constitution.[27] Both versions of the change-of-type theory – suggesting a change in disease or in the human constitution – explained the decline of bloodletting in practice while preserving the theoretical value of bleeding for sthenic diseases. This theory resolved the apparent paradox between theory and practice while supporting the correctness of both current and prior therapies.

The fact that there was a shift in the prevailing type of epidemic fever in Edinburgh in the early 1830s from sthenic relapsing fever to asthenic typhus, and the close association of this epidemiological shift with a change in reigning fever therapies, indicates clearly that there was an existential basis for some of the arguments postulated in the change-of-type theory. But in formalizing this perceived epidemiological shift, the change-of-type theory as it took form in the 1850s also generalized from epidemic fevers to all inflammatory diseases. Indeed, the change-of-type theory's principal explanatory function in the 1850s was to rationalize the permutation in therapeutic management of pneumonia – not of epidemic fevers. Physicians drew upon the empirical observational evidence available to them and upon the authority of medical tradition in formulating and supporting the change-of-type theory. Nevertheless, Edinburgh medical men openly embraced this theory less for the intrinsic merit of its evidential foundation than for the way the theory satisfied certain social and intellectual needs. The change-of-type theory and its dissemination plainly were largely products of the Edinburgh medical profession's status anxieties.

JOHN HUGHES BENNETT AND THE MOLECULAR THEORY OF ORGANIZATION

An alternative explanation for the change in practice coupled with a quite different evaluation of bloodletting was put forward by the Edinburgh physician John Hughes Bennett. Bennett's particular posture *vis-à-vis* bloodletting, moreover, was rooted in a fundamentally different perspective on therapeutics. However, the distinctiveness of this perspective is discernible not in the specific form of clinical activity it informed – which deviated only in degree from the practice advocated by Alison and most other Edinburgh practitioners – but rather in the sources of authority upon which Bennett's criteria for therapeutic evaluation were grounded. Bennett's particular conceptions of the relationship between laboratory-derived medical theory and clinical practice and of the proper sources for the construction and validation of therapeutic theory were the distinguishing features of his philosophy of therapeutics.

For Bennett, the relationships among scientific knowledge, medical theory, and clinical practice were clear. He believed that scientific knowledge and theory elaborated from it were useful guides to the improvement of medical practice. Theory generated in the laboratory played more than merely legitimizing and explanatory bedside roles: science was a fruitful participant in the construction of therapeutic models and an admissible test of therapeutic practices. Bennett maintained that all

[26] W. Turnbull, 'On bloodletting in internal inflammations', *Edinb. med. J.*, 1857, **3**: 187-188, p. 187.
[27] See James Stephens, 'Letter to the editor. On blood-letting in internal inflammations', ibid., 1857, **3**: 184-187, pp. 185-186.

organic processes – healthy and morbid – must conform to the same fundamental principles; pathology was simply the "physiology of disease". Consequently, biological theories that explained physiological phenomena must also explain pathophysiological processes and constitute fruitful guides to therapeutic measures that would restore these processes to normal. Thus for Bennett, the laboratory joined the bedside as a locus of authority for therapeutic theory and a useful source of guidance for clinical practice.

Bennett's position was not an open avowal of faith in the sovereignty of science over experience as a source of clinical authority; rather, it was an attempt to vindicate the therapeutic relevance of scientifically-grounded theory. "There can be no doubt that a too exclusive attention either to practice or theory tend to circumscribe the usefulness of the physician", Bennett annually cautioned his students, "whilst on the other hand it is the proper cultivation of both which constructs rational medicine."[28] Bennett was a practising clinician, and no sober practitioner would deny the importance of bedside observation in testing the validity of a theory; yet he was also a laboratory scientist who was deeply committed to a belief in the clinical value of experimental science.

Bennett's dual commitment to the relevance of medical theory for clinical practice and the value of laboratory science in guiding the construction of therapeutic theory found its clearest expression in his theory of elemental organic organization and his application of this theory to clinical evaluation. Through his molecular theory of organization, Bennett sought to heal the rifts between medical theory and therapeutic practice and between scientific knowledge and clinical activity: the objective of his molecular theory of oganization was "to blend into a harmonious whole the kindred sciences of physiology, pathology, and therapeutics".[29] From his system of theoretical physiology Bennett developed a molecular pathology that, in turn, led to a system of rational molecular therapeutics. He employed the tenets of his molecular theory of organization in constructing explanatory models for tissue formation, nutrition, pathogenesis, and the actions of therapeutic agents. Bennett's highly visible challenge to the principle of bloodletting in the mid-1850s and the position he defended in the ensuing bloodletting controversy were fostered by the therapeutic posture that his conception of molecular pathology informed.

After receiving his M.D. degree from Edinburgh in 1837, Bennett spent four years studying clinical medicine, experimental physiology, and medical microscopy in the medical centres of France and Germany, where he developed a particular interest in microscopic pathology. Returning to Edinburgh in 1841, Bennett offered clinical instruction modelled after his experience in the German polyclinics and gave private courses in pathological histology and the use of the microscope. Through his

[28] Bennett Papers, op. cit., note 1 above, 1849. See also John Hughes Bennett, *Lectures on clinical medicine*, Edinburgh, Adam & Charles Black, 1856, p. 5.

[29] John Hughes Bennett, 'Lectures on molecular physiology, pathology, and therapeutics, and their application to the treatment of disease. Lecture 12. The present state of therapeutics', *Lancet*, 1863, **ii**: 671-675. For accounts of Bennett's molecular theory of organization, see J. K. Crellin, 'The dawn of the germ theory: particles, infection and biology', in F. N. L. Poynter (editor), *Medicine and science in the 1860s*, London, Wellcome Institute for the History of Medicine, 1968, pp. 57-76, on pp. 59 and 70; L. J. Rather, *Addison and the white corpuscles: an aspect of nineteenth-century biology*, London, Wellcome Institute for the History of Medicine; Berkeley and Los Angeles, The University of California Press, 1972, pp. 218-220; William Turner, 'The cell theory, past and present', *Nature, Lond.*, 1890, **43**: 10-15, 31-37, p. 14.

appointment as the director of the pathological department at the Royal Infirmary of Edinburgh in 1844 Bennett gained ready access to abundant clinical resources, greatly facilitating his clinical and pathological researches. Bennett's institutional recognition as a leader of Edinburgh medicine was secured in 1848 when he was unanimously elected to the chair of the institutes of medicine at the University of Edinburgh, a post he held until a year before his death in 1875.[30]

By the time he returned from Europe in 1841, Bennett had taken up Theodor Schwann's model of cell formation, calling it "the most beautiful and harmonious [doctrine] of the whole range of physiology".[31] Schwann, who maintained in his cell theory that the cell was the fundamental structural and functional unit of organic tissue, had put forward in his more speculative theory of cells a model of cell development by a sort of organic crystallization from a fluid blastema. The blastema, which Schwann described as "homogeneous substance" that could become "minutely granulous", gave rise to cells through the coalescence of blastemic granules or "molecules", forming in turn a nucleolus, nucleus, and cell membrane.[32] Bennett adopted this blastemic model of cytogenesis, hoping that it would provide a common referent for understanding physiological processes and "a key which may ultimately enable us to open the secrets of organization".[33] However, Bennett proposed that the dynamic relations of the blastema and the process of cytogenesis should be the objects of greater physiological scrutiny than the formed cell. During the 1840s Bennett's attention progressively shifted from the cell to the particulate constituents of the blastema from which it arose; by the late 1840s he had become convinced that the "molecule", rather than the cell, was the true elemental unit of organic organization. "The molecule", he wrote, "is the real basis of all tissues."[34]

Bennett's "molecules" were microscopically distinct particles – what he called the "visible molecules of the histologist". Structurally, a molecule was a minute globular body having a diameter of from 1/4000 to 1/20,000 of an inch. Chemically, molecules varied widely, but could be broadly classified as albuminous, fatty or oily, and mineral. Functionally, molecules were distinguished according to their proximate origins and immediate functions in nutrition. "Histogenetic molecules" formed by precipitation in the blastema through the union of two simple organic fluids, such as oil and albumen, or the aggregation of mineral matter; these molecules contributed to histogenesis. "Histolytic molecules" were the products of tissue disintegration. This distinction

[30] There is no satisfactory biography of Bennett. The most informative sketches of his life are John G. M'Kendrick, 'Obituary, John Hughes Bennett', *Edinb. med. J.*, 1875, **21:** 466-474; *idem*, 'Obituary, John Hughes Bennett', *Br. med. J.*, 1875, **ii:** 473-478; W. W. Johnston, 'John Hughes Bennett – his services to medicine', *Trans. med. chir. Fac. Md.*, 1899: 139-150. Much useful information on Bennett's European experience is contained in *Testimonials in favour of John Hughes Bennett, candidate for the chair of the institutes of medicine in the University of Edinburgh*, Edinburgh, Murray & Gible, 1848.

[31] Bennett Papers, op. cit., note 1 above, n. d. See John Hughes Bennett, 'On the structural relation of oil and albumen in the animal economy, and on certain physical laws connected with the origin and development of cells', *Mthly J. med. Sci.*, 1847, **8:** 166-176, p. 170.

[32] Theodor Schwann, *Microscopical researches into the accordance in the structure and growth of animals and plants*, trans. by Henry Smith, London, The Sydenham Society, 1847, pp. 168-180. On Schwann's theory of cells, see Russell C. Maulitz, 'Schwann's way: cells and crystals', *J. Hist. Med.*, 1971, **26:** 422-437; Everett Mendelsohn, 'Schwann's mistake', *International Congress of the History of Science. Actes*, 1964, **10:** 967-970.

[33] Bennett Papers, op. cit., note 1 above, [n.d.].

[34] John Hughes Bennett, *On cancerous and cancroid growths*, Edinburgh, Sutherland & Knox, 1849, p. 142.

between histogenetic and histolytic molecules was physiological rather than morphological.[35]

Bennett's schema of molecular physiology hinged upon his conception of the participation of molecules in growth and nutrition, which he summarized succinctly in the "molecular law of growth" or "Bennett's Law": "Growth and transformation in organic tissue is due to the successive formation of Histogenetic and Histolytic molecules".[36] Bennett acknowledged that most tissue was formed by cellular development, yet he opposed an exclusive cellular theory. He maintained that some organic structures – certain fibres, filaments, and membranes – arose directly from the blastema by molecular aggregation, independently of cell formation. However, all tissue – cellular or acellular – originated from the molecular blastema and ultimately broke down, returning molecules to the blastema. "Inasmuch as the molecular element is the first as well as the last form which organized matter assumes", Bennett wrote, "it must constitute the principal foundation of organization itself".[37]

Bennett viewed elemental organic organization through the eyes of a pathologist and clinician as well as of a physiologist. "It is daily becoming more and more apparent that the results of *post mortem* examination have ceased to furnish us with facts sufficiently novel and important to advance the study of pathology", Bennett commented as early as 1843;[38] microscopical pathological anatomy and physiology, on the other hand, were fruitful fields for further inquiry. Bennett had returned from his studies in Europe firmly committed to a belief in the clinical relevance of pathological histology, and was largely responsible for introducing the use of the microscope into British medical education.[39] Moreover, Bennett's manuscripts show that he used the microscope for clinical diagnosis in the wards of the Royal Infirmary of Edinburgh from the time of his appointment in 1844.[40] As early as 1849 he recommended that to identify neoplastic tissue in cancer surgery, "The microscope ought to be a necessary instrument in the operating theatre, and every suspected tissue

[35] John Hughes Bennett, 'Lectures on molecular physiology, pathology, and therapeutics, and their application to the treatment of disease. Lecture 1. Introduction', *Lancet*, 1863, **i**: 1-4; *idem*, *Treatise on the oleum jecoris aselli or cod liver oil, as a therapeutic agent in certain forms of gout, rheumatism, and scrofula; with cases*, London, S. Highley, 1841, pp. 53-60.

[36] William Stirling, student notebook from the physiology lectures given by John Hughes Bennett, November 1868 to March 1869, MS. 9165, National Library of Scotland, Edinburgh. See also John Hughes Bennett, 'On the law of molecular elaboration in organized bodies', *Rep. Br. Ass. Advmt Sci.*, 1855: 119-120.

[37] John Hughes Bennett, 'On the molecular theory of organization', *Lancet*, 1861, **i**: 504-507, p. 507. See *idem*, 'On the molecular origin of the tissues', *Mthly J. med. Sci.*, 1852, **14**: 476-477; *idem*, 'On the structural relation of oil to albumen in the animal economy', *Proc. roy. Soc. Edinb.*, 1847, **2**: 136-138; *idem*, 'Lectures on molecular physiology, pathology, and therapeutics, and their application to the treatment of disease. Lecture 3. The fibrous elements of the tissue', *Lancet*, 1863, **i**: 139-141, p. 139.

[38] John Hughes Bennett, 'On inflammation as a process of abnormal nutrition, being the substance of six lectures delivered before the Royal College of Physicians, Edinburgh, during the summer of 1843', *Edinb. med. J.*, 1844, **62**: 24-52, and 1844, **63**: 351-387, pp. 23-24. See *idem*, 'Contributions to pathology and rational medicine. No. 1. Introduction. How should medicine be advanced? With a few remarks in reply to the suggestions of Dr. Forbes', *Mthly J. med. Sci.*, 1846, **7**: 20-27, p. 26.

[39] See John Hughes Bennett, *On the employment of the microscope in medical studies: a lecture introductory to a course in histology*, Edinburgh, Machlachlan, Steward, 1841; *Testimonials in favour of John Hughes Bennett*, op. cit., note 30 above, pp. iii-iv.

[40] Bennett Papers, op. cit., note 1 above, various dates. See John Hughes Bennett, 'The microscope as a means of diagnosis', *Mthly J. med. Sci.*, 1850, **11**: 548-552.

examined on the spot, before the lips of the wound are closed."[41] Microscopy and investigation of the levels of organization it revealed, Bennett believed, should be integral parts of the physician's clinical world.

The molecular theory of organization, to Bennett's mind, was a conceptual tool that would order the translation of microscopic investigation into pathological knowledge and guide clinical activity. In the early 1840s he stated that the application of Schwann's model of cytogenesis to pathological explanation constituted the most promising means of advancing rational medicine. He asserted that a genuine understanding of physiological phenomena – both healthy and morbid – derived from study of the composition of the molecular blastema. Whereas normal function depended on a healthy blastema, pathogenesis involved its morbid derangement. In the final analysis, pathogenesis could be referred to alterations in the blastema or "molecular lesions".[42]

BENNETT'S MOLECULAR THERAPEUTICS AND A REJECTION OF BLOODLETTING IN PRINCIPLE

The confluence of Bennett's pathological theories and his definition of the relationship between medical theory and practice led him to develop a system of molecular therapeutics. "The molecule", Bennett wrote, "offers us *not only* the most correct idea of organization, it constitutes in my opinion the only basis for a rational therapeutics."[43] Since his molecular pathology defined disease as a morbid alteration in the molecular blastema, his molecular therapeutics sought therapeutically to modify blastemic composition to restore normalcy. Bennett conceived of proper clinical management as a sort of molecular engineering of pathophysiological processes that rectified morbid conditions: "If we could add to, or subtract from the particular molecular elements which are essential to [a pathological] process, we could accelerate or retard it: and this is within the reach of the medical practitioner."[44] The objective of molecular therapy was to restore the deranged processes to normal and to repair the lesions they had produced.

The pivot upon which Bennett's system of molecular pathology revolved was inflammation. "Any doctrine capable of explaining the various phenomena which usher in, constitute, and follow this morbid phenomenon [inflammation]", Bennett wrote, "cannot but furnish those principles on which medicine, as a science and as an art, must ultimately rest."[45] Bennett's evaluation of the treatment of inflammation – and particularly internal inflammation like pneumonia – on the basis of the molecular theory of organization placed him in direct conflict with reigning medical belief and drew him into the most visible controversy of his career. In this instance, Bennett used laboratory-derived theory to vindicate his rejection of prior anti-inflammatory therapies, notably bloodletting, which his theoretical schema judged to be worthless or

[41] Bennett, op. cit., note 34 above, p. 248.

[42] Bennett, 'Contributions to pathology', op. cit., note 38 above, p. 25; *idem*, 'On inflammation', op. cit., note 38 above, p. 25.

[43] Bennett Papers, op. cit., note 1 above, 1848.

[44] Bennett, 'Lectures on molecular physiology', op. cit., note 35 above, p. 4; ibid., 'Lecture 2. The cell elements of the tissues', pp. 55-57.

[45] Ibid., 'Lecture 8. Inflammation', pp. 597-600, on p. 597. Cf. Bennett, 'On inflammation', op. cit., note 38 above, p. 23.

harmful, and to give theoretical legitimacy to new therapeutic models.

Bennett held that the essential phenomenon of inflammation was exudation of the *liquor sanguinis* or blood-plasma, and that this, and this alone, should be taken as pathognomonic of the inflammatory process. The exudate could be removed or assimilated to the economy in one of two ways. If, on the one hand, the exudate lost its formative power, it disintegrated giving ulceration or gangrene. On the other hand, if the exudation retained its formative power it constituted a molecular blastema which was transformed into various tissue structures according to the laws of cytogenesis. As the inflammatory exudate passed into organization, the inflammation disappeared. Permanent structures formed in this manner could be either normal tissue elements or morbid products, depending on the organism's nutrition and the composition of the blastema. Alternatively, temporary structures or pus cells could arise from the blastema, subsequently breaking down forming histolytic molecules. These, in turn, either became histogenetic molecules that contributed to normal tissue growth or were reabsorbed by the circulation and excreted from the system. The inflammatory exudate, constituting a molecular blastema, thus was removed by forming tissue elements – normal or morbid, temporary or permanent, cellular or acellular – according to the same doctrine of growth by molecular coalescence that Bennett applied to all tissue formation.[46]

The therapeutic indications of this model of inflammatory pathology seemed clear to Bennett. The principal objective of treatment was to eliminate the inflammatory exudate by furthering its transformation into cells and by facilitating the absorption and excretion of "effete matter". Proper therapy should support the nutrition of the molecular blastema, maintaining its formative power, and thereby promoting the absorption or normal resolution of the exudate.[47] "An inflammation having occurred", Bennett wrote, "the great work now to be accomplished is an increased growth by cell formation, whereby that exudation is to be broken up, the pressure it exerts on the nerves and blood vessels removed, and the whole rendered capable of being eliminated from the economy."[48] Supported by this model of inflammatory pathology, in the late 1840s Bennett advocated his "restorative treatment" for internal inflammation, that is, pneumonia, putting it into practice in the clinical wards of the Royal Infirmary of Edinburgh. "Good beef tea and nutriments", he maintained, promoted transformation of the exudate into cells, while diuretics, nitric aether, and colchicum wine favoured excretion of breakdown products.[49]

[46] Ibid., pp. 34, 40, 42, 49, 252-256, 373-384. See John Hughes Bennett, 'Contributions to pathology and rational medicine. No. 4. On exudation, part 2. Its development', *Mthly J. med. Sci.*, 1846, **7**: 583-598, p. 592; *idem*, 'On the formation of pus, in reference to the doctrine of cell pathology', *Rep. Br. Ass. Advmt Sci.*, 1865: 101-102.

[47] John Hughes Bennett, 'Contributions to pathology and rational medicine. No. 3. On abnormal nutrition and diseases of the blood', *Mthly J. med. Sci.*, 1846, **7**: 326-333, p. 333.

[48] John Hughes Bennett, 'Observations on the results of advanced diagnosis and pathology applied to the management of internal inflammations, compared with the effects of a former antiphlogistic treatment, and especially bloodletting', *Edinb. med. J.*, 1857, **2**: 769-796, p. 779.

[49] Ibid., pp. 782-783, 792. See John Hughes Bennett, *On the restorative treatment of pneumonia*, 3rd ed., Edinburgh, Adam & Charles Black, 1866; *idem*, 'Reports of cases of pulmonary diseases treated in the clinical wards of the Royal Infirmary during the latter half of the summer session, 1851', *Mthly J. med. Sci.*, 1851, **12**: 546-559.

Bennett's plan of restorative treatment was strikingly akin to the doctrines put forward by the devotees of the healing power of nature in their prescriptions for bedside management. The therapeutic philosophy erected around an allegiance to the healing power of nature, which claimed considerable medical support in the mid-nineteenth century, proposed that the diseased patient should be sustained with nourishment while nature provided a cure. Both Bennett and the advocates of the healing power of nature rejected interventionist therapeutic strategies and advocated in their stead a reliance on the system's normal curative powers. One American practitioner even linked Bennett with the physician John Forbes, a British spokesman for the nature-trusting impulse, calling them "the two high priests of Nature".[50] However, this clinical resemblance contrasted sharply with the irreconcilable rift between their theoretical stances and conceptions of the proper sources of medical authority. Forbes and his fellows embraced an extreme therapeutic empiricism that carried with it a strong bias against the clinical relevance of laboratory science. Bennett, on the other hand, looked to the laboratory as one important source of clinical justification, defining the principles of his restorative treatment by his own theory of the molecular pathology of inflammation.[51]

More consequential (both for Bennett's own reputation and for the therapeutic thought of other physicians) than his restorative treatment was his evaluation of existing therapies for pneumonia, particularly bloodletting, by the criteria for rational therapy that his conception of inflammatory pathology informed. Bleeding, Bennett reasoned, reduced the supply of nutriments to the exudative blastema, thereby rendering it deficient in the molecular elements required for healthy cytogenesis and hindering normal resolution of the inflammation.[52] He wrote that cell formation from an inflammatory exudate

> should be looked upon as a kind of growth which enables the exuded and coagulated blood-plasma to be rapidly broken up and eliminated from the economy. If so, instead of being checked, it should be encouraged as much as possible – a very different doctrine from what has hitherto prevailed. Again, everything that lowers the vital strength and weakens the economy must impede the nutritive processes of growth . . . . Blood-letting, especially has this tendency, and must therefore be wholly opposed to the rapid disappearance of inflammation.[53]

Bennett claimed that on the basis of scientific knowledge interpreted through the molecular theory of organization, the treatment of inflammation by bloodletting was inert or injurious and therefore should be banished from medical practice. He seconded this laboratory-derived invalidation of the theoretical value of bloodletting with clinically-gathered statistics, which compared different treatments for pneumonia and purported to show that mortality rates declined with diminished use of the lancet.[54]

[50] Bennett Dowler, 'Speculative and practical researches on the supposed duality, unity and antagonism of nature and art in the cure of diseases', *New Orl. med. surg. J.*, 1858, **15**: 787-805, p. 802.

[51] For a discussion of the differences between Bennett's therapeutic posture and the philosophy of therapeutics to which the advocates of nature's healing power subscribed, and of the impact of Bennett's thought on the development of this philosophy, see John Harley Warner, '"The nature-trusting heresy": American physicians and the concept of the healing power of nature in the 1850's and 1860's', *Perspect. Am. Hist.*, 1977-1978, **11**: 291-324, pp. 301-303, 310-313. Bennett rationalized the action of the healing power of nature by the precepts of his molecular theory. See John Hughes Bennett, 'Contributions to pathology and rational medicine. No. 8. On exudation', op. cit., note 38 above, pp. 368-369.

[52] Bennett, op. cit., note 47 above, p. 333.

[53] Bennett, op. cit., note 48 above, pp. 783-784.

[54] Ibid., pp. 787-794.

On the face of it, in rejecting bloodletting Bennett appeared to be naïvely neglecting the concerns that were so prominent in the thought of Alison and other leaders of the profession. Bennett, however, was younger than most of the other leaders of Edinburgh medicine and had not practised before the 1830s when venesection was used extensively. Nor did he identify as fully with the Edinburgh tradition, associating himself rather with the emerging Continental tradition of scientific medicine. Furthermore, Bennett's interpretation of the pathophysiology of inflammation within the framework defined by his molecular theory of organization provided the basis for posing a theoretical challenge to bloodletting. The coupling of Bennett's professional identification with his particular conception of the pathophysiology of inflammation thus freed him from both the social and cognitive constraints that prevented other physicians from questioning and repudiating the value of bloodletting in principle.

TWO JUDGMENTS UPON THE THERAPEUTIC RELEVANCE OF SCIENTIFIC KNOWLEDGE

Bennett put forward his theoretical challenge to the principle of bloodletting most bluntly in a lecture he delivered before a class of medical students in 1855 on 'The present state of the theory and practice of medicine'. Scientific knowledge, Bennett told his students, was "silently revolutionizing the study of medicine" and transforming clinical practice.[55] The pathophysiological doctrines embodied in his molecular theory of organization indicated that bleeding was opposed to proper therapy and had always been incorrect practice. In pneumonia,

> lowering the vital strength and vital power of the individual is directly opposed to the necessary vital changes which the exudation must undergo in order to be removed by cell growth and disintegration. Hence it is, in my opinion, that the mortality from pneumonia has diminished since large bleedings have been abandoned, and not because, as has been suggested by an eminent authority, inflammations, like fevers, have changed their types since the days of Cullen and Gregory.[56]

Improved scientific knowledge of disease had led to the change in practice, Bennett maintained, and Alison's formulation of the change-of-type theory was untenable. Addressing his remarks to students, Bennett placed his challenge in a highly visible forum which the profession could not comfortably ignore.

Debate between Bennett and the other members of the Edinburgh Medico-Chirurgical Society, which represented the medical élite, culminated in the "bloodletting controversy of 1857".[57] But the use of bloodletting was only superficially the theme of this controversy. The real questions at issue were the principle of bloodletting in pneumonia, the discrepancy between theory and practice, and the reasons therapeutic practice had changed. One Edinburgh physician cogently observed in that year: "The revolution in practice has been a silent one, and . . . we are only now

[55] John Hughes Bennett, *The present state of the theory and practice of medicine. An introductory lecture to the class of the institutes of medicine in the University of Edinburgh. Delivered November 6, 1855*, Edinburgh, Sutherland & Knox, 1855, pp. 16-17.

[56] Ibid., pp. 18-19.

[57] See King, op. cit., note 2 above. An informative contemporary account of the controversy is 'Analytical and critical reviews. Review I. The blood-letting controversy', *Br. for. med. chir. Rev.*, 1858, **22**: 1-40. The capacity of the content of Bennett's challenge to stimulate controversy was exacerbated by his acerbic, often sarcastic style. Bennett was known within the profession for his fondness of polemics and his "ram-stam way" of doing business (R. Christison to "My Dear Doctor", 12 December, Hannay Papers, MS. GD214/68/29, Scottish Record Office, Edinburgh).

beginning to inquire into its cause."[58]

Alison and most other leaders of the Edinburgh medical profession supported bloodletting in principle, claiming that empirical observation of the change-of-type had led to the change in practice. Bennett, on the other hand, rejected both the principle of bloodletting and the change-of-type theory, asserting that venesection was and always had been improper practice. "If we hold with Professor Bennett", one practitioner later lamented, "that the doctrine of change-of-type is untenable – we must believe one of two things, either that . . . [Cullen, Gregory, and the other former leaders of Edinburgh medicine] were themselves deceived, or themselves deceivers."[59] Bennett openly embraced the implications of his arguments: "We cannot suppose for a moment", he asserted, "that inflammation has ever undergone any change whatever among mankind, [and] it necessarily follows, if modern practice in this matter be correct, that former bleedings must have been inert or injurious."[60]

Bound up with these arguments were questions about what constituted proper sources of authority for therapeutics and for therapeutic change. Alison and his supporters asserted that scientific knowledge had not led to the decline of bloodletting and could not properly influence therapeutic practice. Medical practice and changes in it, Alison claimed, should be grounded "on empirical observation only".[61] Bennett maintained that an improved knowledge of pathology – not empirical observation of a change-of-type – had led to the abolition of prior, improper practice and to the institution of sounder clinical management. "I sincerely regret that modern pathology has no better exponent of its views than myself", Bennett wrote, "but supported by the conviction that it is to the advancement in medical science that we must look for its improvement as an art, I venture to enter upon a controversy."[62] Scientific knowledge generated in the laboratory, he believed, was one valid source of authority for therapeutic change.

Bennett's explanation for the change in practice and his theoretical assessment of bloodletting enabled him to go beyond his Edinburgh colleagues in calling for the abolition of bloodletting's clinical use. Although venesection in practice had declined considerably after the early 1830s, no one could call for its *complete* rejection from practice until after first having challenged bloodletting in theory and thereby in principle. The most that an advocate of the change-of-type theory could claim without compromising his theoretical beliefs was that bloodletting should be abandoned for *all but* sthenic inflammations. However, by turning to the authority of the laboratory for a

[58] W. T. Gairdner, 'Remarks on Dr. Bennett's paper on blood-letting and antiphlogistic treatment, in the Edinburgh Medical Journal for March 1857', *Edinb. med. J.*, 1857, **3**: 197-229, p. 198.

[59] Stokes op. cit., note 14 above, p. 135. See W. P. Alison, 'Reply to Dr. Bennett's observations on the results of advanced diagnosis and pathology in the management of internal inflammations'. ibid., 1857, **2**: 971-995, p. 976; 'Transactions', op. cit., note 23 above, pp. 947-953; 'Transactions of the Medico-Chirurgical Society of Edinburgh', ibid., 1857, **2**: 1034-1040; Thomas Watson, 'Note to the lecture on blood-letting; formerly published', ibid., 1857, **2**: 1084-1088.

[60] Bennett, op. cit., note 42 above, p. 787. Bennett also argued against the notion that the constitution of mankind had changed (ibid., p. 775). See also John Hughes Bennett, 'Reply to the previous note of Dr. Watson', *Edinb. med. J.*, 1857, **2**: 1088-1091.

[61] William Pulteney Alison, 'Reflections on the results of experience as to the symptoms of internal inflammations, and the effects of bloodletting, during the last forty years', ibid., 1856, **1**: 769-788, pp. 772, 774. See also Gairdner, op. cit., note 58 above, pp. 197-229.

[62] Bennett, op cit., note 48 above, p. 770.

pathological definition of inflammation, Bennett found scientific criteria for considering internal inflammation and the effects of therapeutic agents upon it in a way that did not necessarily admit a distinction between sthenic and asthenic conditions. The tenets of Bennett's molecular theory of organization, extended to molecular pathology and therapeutics, dictated that bloodletting was opposed to correct practice in treating all cases of inflammation – semiologically sthenic or asthenic. Bennett's repudiation of bloodletting in principle on the basis of laboratory-derived theory permitted him to call for the total rejection of bloodletting from therapeutic practice.

The differing judgments that Bennett and the other leaders of Edinburgh medicine passed upon bloodletting reflected an underlying rift in their conceptions of what constituted proper and fruitful sources of authority for validating as well as formulating therapeutic theory. Bennett grounded his arguments with respect to the theoretical value of bloodletting upon a pathological definition of inflammation and of inflammatory diseases like pneumonia; his opponents argued from a clinical definition of inflammation and of disease. Bennett maintained that clinical management could be tested by both scientific criteria generated in the laboratory and by statistics gathered in the clinic, whereas Alison and his fellows maintained that the bedside was the sole locus for evaluating a therapeutic operation.

This differential selection of sources of authority in the instances of therapeutic justification and evaluation also appeared in the explanations physicians formulated to rationalize why practice had changed. Bennett looked to the laboratory's products to explain therapeutic change, asserting that the transformation in practice had been the consequence of scientific enlightenment and that improved scientific knowledge of disease should guide future therapeutic advancement. His opponents argued that bedside observation was the sole valid source of therapeutic change and progress, drawing upon the perceived epidemiological shift, extended to the full complement of arguments embodied in the change-of-type theory, to explain the shift in the clinical perception of bloodletting's therapeutic efficacy and the ensuing change in practice. Therapeutic change, they asserted, was the consequence of a change in the phenomena – that is, internal inflammations – not in physicians' knowledge of them.

The reluctance of most physicians to embrace Bennett's explanatory models for therapeutic change and evaluatory criteria for clinical management clearly was bound up with professional status insecurities and fear of indicting former clinical practices. More fundamentally, however, the conflict Bennett's position stimulated reflected a difference between Bennett and his Edinburgh colleagues in their perceptions of the therapeutic relevance of scientific knowledge. Most Edinburgh medical men maintained that the proper clinical role of science was to explain and legitimize – but not to test or guide – medical practice; they further held that science should be consistently subordinated to bedside observation even in formulating theories to explain therapeutic actions. Bennett, however, believed that scientific knowledge could both guide the construction of therapeutic theory and provide one test of its clinical validity.

In large measure, the difference between Bennett's therapeutic outlook and that of the other leaders of the profession stemmed from educational differences. Alison and most other elders of Edinburgh medicine had received their medical training entirely in

Scotland and derived both their therapeutic philosophy and professional identity from the Edinburgh tradition. On the other hand, Bennett's orientation to laboratory investigation and scientific knowledge stemmed principally from the four years he spent in France and Germany. His initiation into the tradition of laboratory science also gave him a different professional identity grounded upon an allegiance to science. Education thus changed Bennett's conception of the relationship between experimental science and medical practice, altering in turn his emphasis upon what generated and validated medical theory.

The arguments that emerged from the bloodletting controversy and the commitments that animated these arguments defined clearly two distinct perceptions of therapeutics. Alison and the other defenders of the principle of bloodletting drew support for constructing and defending their therapeutic postures from bedside observation and the clinical authority of medical tradition. Bennett, representing a newer tradition of laboratory science, believed that scientific knowledge constituted one source of weighty authority both for generating and for validating therapeutic theory. He affirmed this belief in the clinical relevance of scientific knowledge by erecting his own molecular theory of organization on the basis of laboratory investigation and, in turn, by using this scientific theory to guide the construction of new therapeutic theory and to test existing clinical theory and principle. The inherent tensions between these two perspectives on therapeutics and the intra-professional conflict they engendered in the mid-nineteenth century represent one stage in the emergence of the concepts that laboratory science is a proper foundation for therapeutic action and that scientific knowledge can confer authority upon clinical medicine.

SUMMARY

Two distinct perspectives on the therapeutic relevance of scientific knowledge are clearly discernible in the controversy over bloodletting that flourished in Edinburgh in the mid-1850s. Bloodletting had declined in the practices of most Edinburgh physicians since the early 1830s, yet it was fully retained in theory through the 1850s. Most physicians explained this apparent paradox by postulating that either disease or the human constitution had changed since the early 1830s from a sthenic type, which demanded depletive therapies like bloodletting, to an asthenic type, which could not withstand bloodletting. They asserted that clinical observation, not scientific knowledge, had engendered therapeutic change. An examination of epidemiological changes and their therapeutic correlates during this period reveals that there was some existential basis for the change-of-type theory; however, in large measure this theory was a product of the Edinburgh medical profession's status anxieties.

John Hughes Bennett put forward an alternative explanation for the change in therapeutic practice and a quite different evaluation of bloodletting. Bennett's medical studies in Europe had conferred upon him a professional identity grounded upon experimental science and a commitment to the medical relevance of laboratory science. After returning to Edinburgh to take up the chair in the institutes of medicine, Bennett developed his molecular theory of organization, which he used to support his theoretical rejection of bloodletting. The coupling of Bennett's professional

identification with his particular conception of the pathophysiology of inflammation freed him from the social and cognitive constraints that prevented other physicians from repudiating bloodletting in principle. The arguments that emerged from the ensuing bloodletting controversy and the commitments that animated these arguments defined clearly two distinct perceptions of the proper sources of authority for clinical practice; the ways therapeutic theory should be generated and validated; and the dynamics of desirable therapeutic change.

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