

lung is opposed by three forces: (1) the weight of the fluid, (2) the action of the diaphragm and (3) the weight of the liver. If the patient is in the sitting position these three all act in the same direction—that is, downwards. When the effusion is on the left side, the lung has only two of these forces to support, the weight of the liver being absent. It follows that the elastic force of the lung will be balanced by these negative forces more quickly on the right side than on the left, and any subsequent increase of fluid will be manifested by a sinking downwards of the diaphragm and liver, whilst with the same amount of effusion on the left side there is no displacement. If, then, in a case of right-sided pleural effusion you find on percussion and palpation that the liver is depressed you will know that the intra-pleural pressure is positive and that the amount of exudation is therefore great.

Now, what information is to be gathered as to a left-sided effusion by observing the effect of lung elasticity on the diaphragm? The position of the diaphragm is here ascertained clinically by percussion of the stomach, the upper border of which follows the movements of the diaphragm. In the normal condition, as you are aware, a portion of the stomach lies behind the lower and anterior part of the chest wall on the left side and can be mapped out as a concave area, the concavity of which is directed downwards. This is known as the semilunar space of Traube, who first called attention to its importance in the diagnosis of pleuritic effusion. In the early stage the action of the diaphragm is diminished, whilst the elasticity of the lung is still strong. The result is that the diaphragm is drawn up into the chest even higher than it is during normal expiration, and this fact is learned clinically by an extension upwards of the tympanic resonance which is heard on percussion over the semilunar space. This is a sign of great importance in distinguishing between pneumonia and pleural effusion. It is not found in the former or in any acute pulmonary disease save fluid effusion in the pleura. It is, of course, a well-marked sign in advanced contraction of the lung due to pleuritic adhesions or to fibroid changes of long duration, and these must be carefully excluded before any conclusions are drawn. You must also see that no increased intra-abdominal pressure is present, which would push the stomach upwards. As the fluid increases in amount, it passes to the front and the semilunar space is diminished in extent, until finally, when the exudation is very great, the diaphragm and stomach are depressed and the tympanic note of the semilunar space is entirely obliterated.

The alterations in the physical signs over the sound lung are not directly of great value. The measurement of the unaffected side of the chest wall usually shows a slight increase, which may be appreciable if quiet respiration is present, and which will be greater according to the amount of dyspnoea. The breath sounds are of the puerile type—that is to say, harsh, vesicular, with prolonged expiration. Of more importance are the results of the unopposed action of this lung on the mediastinum and its contents. Instead of the equally balanced traction on the two sides of the mediastinum by the expansion of the lungs and the consequent filling of the heart and large veins with blood, there now exists a single force tending to draw the mediastinum as a whole towards it—namely, the unopposed traction of the sound lung. The heart must therefore depend on its own action for emptying the veins as well as for filling the arteries, and this leads frequently to considerable cardiac embarrassment, as shown by a weak and irregular pulse. I do not wish to ignore another factor here, on which much stress is often laid—namely, the impeded circulation through the collapsed lung; but I think this is balanced to a certain extent as regards the heart by the freer movement of blood through the sound lung. In considering this question of circulatory disturbance a third factor of great importance comes in—namely, the displacement of the heart itself. This is usually ascribed in the text-books to the pressure of the effused fluid; but I wish to call your attention to the fact that there may be cardiac displacement when the effusion is still moderate in amount—at a time when the fluid is not in contact with the heart but separated from it by collapsible pulmonary tissue. It is difficult to see how any actual pressure can be exerted under these circumstances. We have already seen that the lung on the sound side gains in elasticity by the loss on the affected side, and that consequently the mediastinum is drawn towards the sound side. This action is directly proportional to the amount of fluid effused, and therefore the cardiac displacement is dependent on (1) the quantity of fluid present and (2) the amount of elasticity in the sound lung. As the elasticity of

the lung in early life is greater than in later years you will expect to find the cardiac displacement manifest at an earlier period in the case of children, and this is what actually occurs. We have, unfortunately, no very exact clinical means at our disposal for estimating accurately a small amount of cardiac displacement. By palpation we ascertain the position of the apex beat and by percussion we determine the lateral boundaries of the heart. We then note whether these correspond with the normal conditions, and, what is of more importance, we observe, as the illness proceeds, whether there is any change in the position of the heart on repeated examination. The amount of pulmonary tissue covering the heart varies to such an extent that accurate measurements are extremely difficult and in the case of adults they are almost impossible. Deviations of less than half an inch are not, therefore, of much clinical value. Another source of fallacy lies in the fact that the right side of the heart may be overfilled and the percussion dulness therefore increased to the right of the sternum. It is well known that a left-sided effusion produces more cardiac displacement than a right-sided one of equal extent. On the pressure theory this is difficult to explain, and one distinguished physician<sup>4</sup> has solved the difficulty by denying the fact and has maintained that it is really over-distension of the right ventricle which is mistaken for cardiac displacement. This explanation has not been generally accepted, but the clinical difficulty is easily understood when we regard the elastic traction of the sound lung as the cause of cardiac displacement. The movement of the heart which is of importance clinically is in the lateral direction. Now the heart is situated normally to the left of the middle line. If, then, we compare the amount of elastic traction which can be brought to bear on the heart, we find that this is greater on the right side than on the left, because there is more lung tissue between the heart and the right side of the chest wall than between the heart and the left side of the chest wall. Consequently in the case of a moderate effusion on the left side the cardiac displacement will be manifest sooner than if the effusion is on the right side, and even when the fluid increases and positive pressure is brought to bear on the heart the effect of the lung elasticity in the early stages will still be apparent.

I have not hitherto stated definitely the signs which mark the period of effusion when the intra-pleural pressure becomes positive. Dr. Douglas Powell<sup>5</sup> considers that this stage is reached if, when the patient is in the sitting position, the absolute dulness extends above the third rib anteriorly and the Skodaic resonance is lost. This, as you will perceive, indicates the presence of a very large amount of fluid. Direct evidence of positive intra-pleural pressure is to be found, as already stated, in the depression of the liver on the right side and in the obliteration of the semilunar space of Traube on the left, and in a rush of fluid from the pleural cavity on the introduction of a trocar. The first stage, however, passes so gradually into the second that it is extremely difficult clinically to diagnose the exact period, but a marked amount of positive pressure is quickly followed by an increase in the symptoms present—such as breathlessness, faintness, and weak cardiac action.

## WHAT CONSTITUTES THE ACHING STRUCTURE IN HEADACHE?

BY HARRY CAMPBELL, M.D. LOND. &c.,  
ASSISTANT PHYSICIAN TO THE NORTH-WEST LONDON HOSPITAL.

THE purpose of this paper is to give a historical sketch of the views which have been held regarding the seat of the pain in headache. Before doing so it will be well to define exactly what is meant when it is said of any part that it is the seat of pain. It is now known that, as a matter of fact, all sensations arise in connexion with the brain and are only mentally referred to various parts of the body. When, therefore, a part is spoken of as being the seat of pain, it is meant that the pain, which arises in consequence of certain cerebral processes, appears to the eye to proceed from that part. This is the *mental* criterion of what constitutes the seat of pain. It will be seen that the aching part is not, in

<sup>4</sup> Dr. Moxon: THE LANCET, Jan. 12th, 1884.

<sup>5</sup> Loc. cit.

the strict sense of the term, painful—i.e., full of pain—since pain, so far as it is possible to speak of it as having any position in space, would be most accurately placed in the brain. Seeing, however, that mental phenomena have no relation whatever to space, one cannot rightly speak of a sensation occupying any part of space. To the ancients no such difficulties presented themselves. They did not, as we do now, regard mind as absolutely distinct from matter, but rather as an attenuated form of it—a view of which traces still survive in such words as “spirit,” “soul,” “ghost,” “mind,” all of which are related to the word “wind,” the wind not unnaturally being regarded by them as a refined, attenuated form of matter, to which what they conceived to be the subtle material essence of mind bore, to their imagination, some resemblance. When, therefore, the ancients spoke of a part as being painful they regarded it as pervaded by a sentient substance—spirit; but the mental criterion by which, as I have said, the seat of a pain is determined is by no means adequate in all cases, and in no case less so than in headache. Defining this latter as pain felt anywhere above the base of the skull, it is obvious that the pain of headache may be seated (I use the expression in the sense indicated above) in many different structures. The aching structure may be (1) the brain itself; (2) its membranes—the pia mater, arachnoid and dura mater—these including the tentorium cerebelli, the falx cerebri and cerebelli and the large fold of pia mater forming the velum interpositum; (3) the skull-bones and mucous membranes lining the frontal, ethmoid, sphenoid and mastoid sinuses and the middle ear; and, finally, (4) the structures covering the cranial vault, including the scalp, the skin of the forehead and temples and all the tissues lying between these and the bone—viz., the pericranium and certain aponeuroses and muscles. Now, it is very seldom that a person suffering from headache can definitely refer the pain to any one of these structures, and therefore the mental criterion is practically useless in determining the seat of the pain in headache. Wherefore one has to fall back upon another test, which may be termed the “physiological.” Where any part of the body capable of feeling pain is irritated so as to cause pain, certain nervous arrangements in the cortex are agitated and the concomitant pain is referred, with more or less definiteness, to the part irritated. All the structures above the base of the skull which are capable, when irritated, of feeling pain have such nervous arrangements, or, to use a more convenient term, algenic centres, allotted to them, and when without any peripheral irritation the algenic centres of any portion of these structures are being agitated that portion constitutes the seat of the pain. It is clear that headache may result from irritation of the aching part (as may happen in the pain of migraine, which some attribute to colic of the temporal or other artery) or that it may arise independently of such irritation. In the former case what is meant by the “seat of the pain” is at once manifest, but in the latter case the notions on the subject would be obscure were not the physiological criterion applied. Suppose, for instance, that the pia mater is free from irritation of any sort, but that the algenic centres belonging to it are being agitated: the pain not being definitely referred by the patient to the pia mater, it would be impossible without this second criterion to speak of it as having any “seat” at all. It is thought by some that the brain itself is incapable of feeling pain—that no kind of irritation of its own substance will provoke pain; and if such is the case then it must be supposed that the brain has no special sensorial nervous system, no sensory nerves distributed throughout its substance and connected with special centres in the cortex. At first sight it seems strange that the brain, consisting, as it does, of the largest and most complex mass of nervous matter in the body, should not be exquisitely sensitive to pain. On a little reflection, however, it will be seen that a part is sensitive only by virtue of sensory end-organs distributed throughout its substance and that it is impossible for it to be sensitive without these. If a sensory fibre is stimulated in its continuity, the resulting sensation—if, indeed, any sensation result—is referred, not to the point of the fibre irritated, but to its peripheral distribution; whereas if a nerve containing sensory fibres—say, the ulnar—is stimulated a sensation is felt both at the spot stimulated and at the peripheral distribution of its fibres. The former sensation is due to the irritation of special sensory end-organs belonging to the nervi-nervorum distributed to the nerve-trunk and on which the pain of neuralgia depends; the latter to the irritation of the sensory fibres of the ulna in their course. A sensory nerve-fibre is in its continuity far less responsive to irritation than its end-organ—the retina, e.g., is far more

sensitive to stimuli than the optic nerve. Were this otherwise, the results would be disastrous—the slightest irritation in the posterior portion of the hind limb of the internal capsule, for instance, would give rise to sensations all over the body and to the various special sensations of sight, hearing, taste and smell.

Having cleared up these preliminary points I may address myself to the proper subject of this paper. It is remarkable that the views of the ancients respecting the structures involved in the pain of headache were very much the same as those of to-day. They for the most part excluded the brain and fixed the seat of the pain in extra cerebral structures, such as the meninges, pericranium and scalp. This view has been maintained by all writers of importance, with one or two exceptions, down to the present time. Hippocrates does not seem to have expressed any very definite opinion on the subject. Galen, whilst not altogether excluding the brain,<sup>1</sup> seeing that he regarded the arteries and veins as capable of sensibility, certainly looked upon the meninges and pericranium as the chief seats of headache. The following passage from Bonetus sets forth his views<sup>2</sup>: “The pain may be spread throughout the entire head or affect only some one part of it, and it may be internal or external. By internal pain I mean pain in the membranes or the substance of the brain or the veins, arteries and nerves, of which some are more, others less, sensitive.” There can be no doubt that the meninges and the nerves are the most sensitive, and it is thus that they perceive the pangs of pain the more vehemently; but it would rather appear that the arteries, the veins and the brain substance suffer from that dull and heavy pain which is wont to attack parts endowed with weak sensibility. We are not justified in assuming that these parts are altogether devoid of sensibility, for Galen<sup>3</sup> mentions the veins and arteries amongst the structures capable of perceiving pain, speaking very plainly regarding the arteries<sup>4</sup>. He has pronounced the brain to be “devoid of sensibility”—a dogma which Bonetus goes on to question—and he gives reason for believing that the brain is sensitive. He regards heaviness in the pain as indicating that it is situated in the substance of the brain rather than in the meninges. In the latter case it is “pungent and acute,” whilst when the osteal membranes are involved it is “bruising.” Finally, he gives special ways of distinguishing pain in the arteries from pain in the nerves. Galen gives a method of distinguishing between intra- and extra-cranial pain. He contends that external headache may be distinguished from “the internal pain by the fact that the internal pain spreads to the roots of the eyes [i.e., optic nerves], whilst the external one never does,”<sup>5</sup> and he explains this supposed peculiarity on the ground that the tunics of the eyeballs are continuous with the cerebral meninges and that this continuity facilitates the propagation of the pain. This view was taken up and propagated by Paulus Aegineta,<sup>6</sup> Etius and Avicenna,<sup>7</sup> and is repeated, almost in Galen’s own words, by many writers of the sixteenth, seventeenth, and eighteenth centuries.<sup>8</sup> Fernelius was one of the first, if not the first, to confute it. “It is altogether wrong to suppose that pains of internal origin only involve the roots of the eyes,” he writes concerning it, for he argues that owing to the continuity of the pericranium with the lining membrane of the orbit external as well as internal pains may spread to the roots of the eyes. Rondeletius did not regard this objection as valid, arguing that pain of the pericranium most frequently arises from cold, which cannot reach the orbit because the latter is protected “by the warmth of the eyes, by the spirits and the blood,” whilst if external headache were excited by heat or a similar cause it would rather be the skin than the more deeply-placed pericranium that would suffer. Bonetus, however, rightly dissents from these remarks of Rondeletius, observing that if the external headache arose from any organic disease of the pericranium, such as tumour, “nulla ratio erit cur dolor ad oculi cavum non feratur.”<sup>9</sup> I might quote from several other writers to show how much they exercised themselves on this question.<sup>10</sup> One can, I think, well understand how Galen came by his view. The eyes

<sup>1</sup> Labarraque: *Essai de la Céphalalgie*, p. 78.

<sup>2</sup> J. Boneti Sepulchretum, tome i., p. 35 (1700).

<sup>3</sup> Lib. X., *Tóras*.

<sup>4</sup> Lib. de Tumor., v., cap. 3, de Loc. Aff.

<sup>5</sup> Kühne’s edition, vol. xii., p. 563.

<sup>6</sup> Commentaries, vol. i., p. 355.

<sup>7</sup> Archipathologia, p. 73. Lutetiae, P. E. Montalto (1614).

<sup>8</sup> Joannes F. Stuckens: *De Dolore Capitis* (Brux., 1787).

<sup>9</sup> J. Boneti Sepulchretum, tome i., pp. 19 and 20 (Lond., 1700).

<sup>10</sup> See also Archipathologia &c., p. 73 (P. E. Montalto, 1614), and De Urinis et Pulsibus, p. 576 (Leipzig, 1698).

often ache in headache, and bearing in mind the close relation between the sclerotic and the dura mater he not unnaturally concluded that pain in the one would readily involve the other. As a matter of fact the eyes are apt to be involved in pain in all cases of frontal headache, and although it is impossible to exclude the meninges there can be little doubt that the pain is generally located chiefly in the more superficial structures. The forehead and the eyes each receive their sensory nerves from the superior division of the fifth nerve and it is therefore no wonder that the two are often involved in pain together; but seeing that the extra-cranial part of the frontal regions has far more numerous branches from it than the intra-cranial structures one would expect the former to be chiefly involved in the pain of frontal headache. Galen regarded tenderness of the head as another means of diagnosing external from internal headache, for he writes: "Sometimes the pain of hemicrania is so great that contact with the hand can scarcely be borne, in which case it is clear that the pericranium and overlying skin are affected."<sup>11</sup>

C. Aurelianus, who probably lived shortly after Galen,<sup>12</sup> is most explicit in regard to the structures supposed by the various authorities he had consulted to be involved in the pain of headache. "Some have said that the membrane (of the brain) suffers in headache; others, that which surrounds the skull; others, the whole skin of the head; others, the temporal and buccal muscles."<sup>13</sup> He himself, however, regards one or another as affected according to circumstances. The passage shows how very carefully the question had been thought over before his time. It is very remarkable that whilst he does not name the brain as a possible source of the pain he mentions that certain muscles had been so regarded. A similar view, as will be seen, has since been advanced by Willis, Whytt and other writers. I ought also to mention that Galen placed the pain in some cases of hemicrania in the temporal muscles.<sup>14</sup>

I pass over the Arabian writers since they do not appear to have added anything new to the views of Galen on the subject and come to the mediæval writers. Fernelius has a passage to this effect: "By membranes I mean the meninges and pericranium, which alone are capable of feeling the exquisite pain of cephalalgia, cephalæa and hemicrania."<sup>15</sup> The views of Willis are also expressed in the most unmistakable terms. The parts affected by the pain are, he declares, "the two meninges and their various processes" [this appears to me an important qualification], "the coats of the nerves, the pericranium (or skin compassing the skull) and other thin skinny membranes, the fleshy panicle of the muscle, and, lastly, the skin itself," whilst the brain, medulla and skull, he thought, lacked "sensible fibres, apt to be wrinkled and distended."<sup>16</sup> He regarded the pain of headache as internal or external, the former as rarer and less severe than the latter "because the parts above the skull are not so sensible as the interior meninges; nor are they watered with so plentiful a flood of blood, that by its sudden and vehement incursion they may be easily distended or inflamed above measure." The internal headache "is more frequent and much more 'cruel' because the membranes clothing the brain are very sensible, and the blood is poured upon them by a manifold passage and by many and greater arteries."<sup>17</sup> The "circle of Willis" is, it will be recollected, named after this physician, who must very frequently have dissected the cerebral bloodvessels, and we can well understand how he came to be impressed by the abundance of the arteries and other "passages" (sinuses?) between the brain and the skull. Bartholinus recalls a case of hemicrania which he attributed to acid lymph mixed with bile attacking the frontal muscle. He also refers to other muscles of the head

being the possible seat of pain in headache.<sup>19</sup> Montalto says "the pain of hemicrania sometimes involves the cerebral membranes and at other times the membranes surrounding the skull, the pain in the latter case frequently involving the temporal muscles."<sup>20</sup> Sydenham has no special article on headache, but makes some interesting remarks on the subject of "Clavus," the pain of which he localises in the pericranium; he quaintly supposes that the "spirits" of the body at large are concentrated upon a limited part of this latter, much as the sun's rays may be focussed by a lens, and he compares the pain of the one to the scorching action of the other.<sup>21</sup> The early pathologists were not content merely to differentiate between internal and external headache; they even sought to discover the particular structure involved in the pain, in which attempt they were guided by definite rules, handed down for the most part from Galen. I quote the following from amongst many similar passages in Wepfer. After minutely describing the symptoms of a case of headache he argues that the affected part does not appear to be the pericranium, "because the pain is not increased by external contact, but the dura mater, because it becomes more severe after coughing, screaming &c., and because it is diminished by mental exercise. It is not the pia mater, because there is no vertigo or sleepiness, for the patient does not sleep beyond six or seven hours, and then quietly; nor is the pain superficially situated or in the interior of the brain, because the sharper pain is felt chiefly in the forehead."<sup>22</sup> Physicians of this and of the next century were even more minute in diagnosing the nature of the pathological process—to what extent it depended upon this, that or the other humour; and, as usual, they were largely influenced by the writings of Galen, whose highly speculative mind had loved to dwell in the airy regions of hypothesis.<sup>23</sup>

Coming now to the eighteenth century, one finds Van Swieten, in his "Commentaries of Boerhaave," adopting the opinion of Louis Duret, a French writer of the same period, to the effect that "cephalalgiam calvariae ejusque integumentis proprium esse malum."<sup>24</sup> He regarded the brain as being quite devoid of sensation. Hoffmann expresses his views on the subject in the following passage: "The seat of pain in the head is most frequently the pericranium or the membrane which immediately covers the skull; sometimes the cutis itself, particularly its interior surface contiguous to the pericranium; sometimes also the dura mater, which communicates with the pericranium through the sutures; and sometimes the ..... pituitary tunic—i.e., the mucous membrane lining the frontal and other accessory nasal chambers. The other membranes immediately investing the brain, as the pia mater and arachnoids, seem little adapted to be the seat of the pain, as no elastic fibres or nervous branches are distinguished in them."<sup>25</sup> Hoffman regarded the brain as incapable of feeling pain because the cortical substance may be severely injured without pain being felt; but Colin points out, in reference to this fact, that many organs which in the healthy state are apparently devoid of sensibility may become painful when diseased;<sup>26</sup> and a similar remark has been made by a number of other writers. Not only did the older authorities ascribe to the dura mater great sensibility but the power of "oscillation" also. It was reserved for Whytt, whose writings on the nervous system will still repay perusal, to confute both these views. He declared that the membranes of the brain are altogether destitute of motion and that in the healthy state they are endowed with only a very obtuse kind of feeling, and he refers to Haller's experiments in favour of this view;<sup>27</sup> but in another passage he speaks of the cerebral membranes and pericranium being the seat of violent pain.<sup>28</sup> J. Fordyce alludes to the subject in a passage which does not lend itself readily to translation, but in which he apparently suggests that the pain of hemicrania may be situated in the falx cerebri and in the temporal

<sup>11</sup> See also Johannis Fernelii, Ambiani, *Universa Medica*, p. 59, *Trag. ad Rhen.*, 1656; and Rhondeletius, referred to in the *Sepulchretum*, op. cit., p. 35.

<sup>12</sup> The date is uncertain, but as no mention is made of him in the writings of Galen it is probable that Galen lived first. The fact that Aurelianus wrote in Latin perhaps favours the same view.

<sup>13</sup> *De Capitis Passione*.

<sup>14</sup> Kühn's edition, vol. xii., p. 591.

<sup>15</sup> *De Morbis Universalibus*, p. 91 (1645).

<sup>16</sup> Willis, one of the fathers of nerve physiology, believed, it must be remembered, that pain depended upon a "convulsion or corrugation of the nerves."

<sup>17</sup> "This term still survives amongst the poorer classes in the sense here used; I frequently hear it. The older French writers also speak of 'la douleur cruelle' See, for instance, *Journal de Médecine et Chirurgie*, Paris, July, 1781, pp. 241, 242."

<sup>18</sup> The above quotations from Willis are from the English translation of his work by 'S. P.', London, 1684, *Of the Soul of Brutes*, p. 105

<sup>19</sup> J. Bartholini *Acta II.*, vol. iii. and iv. Hafniae, 1677.

<sup>20</sup> Montalto, op. cit., p. 157.

<sup>21</sup> *Opera Omnia*, p. 373, by G. A. Greenhall. London, 1844.

<sup>22</sup> B. et G. M. Wepfer, *Observationes de Affectibus Capitis*, p. 76. Scaph., 1727.

<sup>23</sup> See, for instance, Kühn's edition of Galen, vol. xii., p. 499 et seq.

<sup>24</sup> Quoted by H. E. Colin in *De la Céphalalgie*, pp. 5, 6. Paris, 1847.

<sup>25</sup> F. Hoffmann: *A System of the Practice of Medicine*, vol. I, pp. 459-477. An Abridged English Translation by W. Lewis, London, 1783.

<sup>26</sup> Colin, loc. cit.

<sup>27</sup> *Observations on the Nature &c. of those Disorders &c. called Nervous, Hypochondriac or Hysterical*, p. 41. By Robert Whytt, M.D., F.R.S. Edin., 1765.

<sup>28</sup> *Ibid.*, p. 15. See also *Works of R. Whytt*, p. 621; Edinburgh, 1783.

muscles.<sup>29</sup> Heberden apparently ignored what others have said on the subject, merely writing that headaches "appear to be seated in the brain itself."<sup>30</sup> Many eighteenth-century writers—e.g., Stüeckens, Sauvages, Devilliers, Deschamps ( fils)—place the seat of pain in the membrane lining the frontal sinuses in cases of headache involving the lower frontal region and root of the nose.<sup>31</sup> Thus Stüeckens asserts that the parts affected in "external headache" are the pericranium and neighbouring parts and the pituitary membrane lining the frontal and sphenoidal sinuses; he then goes on to say that in the latter case the pain affects the lower frontal and the supra-orbital regions, whence it may extend to the nose.<sup>32</sup> Sauvages, who wrote somewhat laboriously on the subject of headaches, thought that the pain might be located in the brain, for he regarded congestion of the cerebral convolutions as a potent cause of headache.<sup>33</sup> The last writer I shall quote from the eighteenth century is Gilbert Blane, who wrote: "Ordinary headache.....seems to be seated in the integuments of the head. The seat of that kind called hemicrania is certainly in these parts, for there is great tenderness on external pressure; and as there is sometimes an obscure redness of the skin and a suffusion of the eye it would appear that the proximate cause is a too great fulness or *error loci* in the circulating vessels."<sup>34</sup>

This rapid survey brings me down to the present age, in which one finds the views regarding the seat of headache remaining substantially the same as those held during the previous centuries. The tendency to locate the pain within the brain is as small as ever, though some few writers yield to it. Thus Romberg termed megrim "neuralgia cerebralis," and Lenbuscher held the same view.<sup>35</sup> Bennet contended that all headaches except "the organic and neuralgic" depend upon a greater or less degree of congestion of the vessels of the brain, being thus apparently influenced by Sauvages,<sup>36</sup> from which it appears that he too located the pain in the brain, though we are not thereby justified in concluding that he regarded the vessels as aching. Here a circumstance may be mentioned which to some may seem to support the view that the cerebral vessels may feel pain. Gowers<sup>37</sup> has established the interesting clinical fact that hemiplegia due to syphilitic disease of the arteries (thrombosis) is very frequently (in one half of his cases) preceded, for a period varying from a few days to a few months, by headache, and such a case has recently come under my own observation. He cautiously remarks that the pain in these instances is "apparently in some way due to the arterial disease itself"; but he does not commit himself to the conclusion that it is actually located in the arteries.<sup>38</sup> Wilks, whilst pointing out that the pain of headache often seems to proceed from the very depths of the brain itself, expressly states that he does not believe that such is actually the case;<sup>39</sup> but to the list of those who do must be added the names of Seller and Symonds, since each (apparently independently) has adopted the same explanation. Seller regarded the pain in those cases where there is no manifest disease of the meninges, skull and scalp as due to involvement of the nerves accompanying the cerebral bloodvessels;<sup>40</sup> and Symonds, in answer to the question, "In what tissues are the aching nerves distributed?" alludes to the difficulty experienced, especially by children, in mentally localising the pain, observing that there may be a dim idea that the pain is internal, whilst external tenderness leads to the conclusion that it is external. He then goes on to remark that most of the nerves which enter the cranium are derived from the sympathetic and expresses his belief that they are sensory as well as motor, instancing in support of this view the fact that

the visceral sensations are felt through the sympathetic.<sup>41</sup> Handfield-Jones is of opinion that in certain varieties of headache, at least, the brain itself is chiefly, if not exclusively, affected, "because its faculties are often notably impaired."<sup>42</sup> These views are, however, exceptional, and the majority of writers at the present day place the pain of headache not in the brain itself but in the extra-cranial tissues. W. King writes: "For years I have expressed distrust of all the supposed seats of headache within the dura mater,"<sup>43</sup> and he argues that, as with all other viscera, so with the brain, pain does not occur until the enveloping membrane is involved; he also points out that most severe headache may occur without any cerebral affection, whilst cerebral softening and hæmorrhage may be unaccompanied by the slightest headache. It has been seen that some early writers (including Galen, C. Aurelianus and in later days Willis, Montalto, Whytt and Fordyce) were careful to include certain extra-cranial muscles amongst the structures involved in the pain of headache. Briquet in recent times has again urged this view, apparently without any knowledge that it had been held long ago. From a number of observations on headache in "hysterical" patients he concludes that the seat of pain in nine-tenths of them was in the fleshy portions of the muscle. No writer has so ably defended this position as Léon Colin, who regards the pain of headache as almost entirely extra-cranial. He argues that the aponeurosis of the occipito-frontalis is peculiarly liable to painful affections owing to its structure, to its position and to the constant changes in temperature to which it is subjected. "The fashion of having the head alternately covered and uncovered, of having the hair cut at long intervals, the baldness often premature, explain the frequency in these regions of rheumatic pains, gouty pains and pains from lead poisoning, which occur pre-eminently in white fibrous tissue,"<sup>44</sup> and he asserts that the anatomical characters of this aponeurosis, notably its inextensibility, render its nerves very liable to be compressed by the hyperæmia which he supposes to result from exposure and other causes. Amongst the latter he gives diminution of atmospheric pressure, which he asserts to be a common cause of headaches and also of fevers. That the majority of intense febrile headaches have an extra-cranial origin is, he contends, proved by the fact that in most of them compression of the temporal arteries diminishes the pain, a point proved by Guyon in different fevers, notably yellow fever. Colin's article is, however, chiefly remarkable for the importance which it shows he attaches to the cranial muscles as being a seat and source of the pain in headache. Like Briquet, he seems to be unaware that others had previously held similar views. "Of all the muscles of the economy," he writes, "those which are inserted into the head are precisely those whose action is most frequent and most continuous, the only ones which during sedentary occupation do not rest. They play a considerable part in causing headache." Thus not only are they liable to the pain of fatigue, but they are apt to set up headache from their close connexion with the aponeurosis of the occipito-frontalis. More recently Norström has expressed the opinion that certain cases of headache are due to inflammatory thickening at the insertions of particular muscles in consequence of contracting cold, no pain being usually felt at the indurated spots.<sup>45</sup>

Perhaps no variety of headache affords better opportunities for determining the seat of the pain than megrim. There is now a general consensus of opinion that in this headache it is wholly extra-cerebral and chiefly extra-cranial. Pinel regarded megrim as a facial neuralgia and Chaussier localised it in the supra-orbital nerves.<sup>46</sup> Laycock considered it as "rather a neuralgia of the cranium or dura mater or the scalp than of the encephalon."<sup>47</sup> Anstie<sup>48</sup> is not disposed to believe that pain is felt in the structure of the brain; he regards megrim as a neuralgia of the fifth nerve, but does not look upon the meninges as exempt from the suffering of headache as the pain is in some cases deep-seated. Some have thought

<sup>29</sup> *Dissertatio de Hemicrania*; London, 1758. The writer here refers the reader to Spiegel's *Anatomy*, lib. x., c. 3.

<sup>30</sup> W. Heberden: *Commentaries*, third edition, p. 92. London, 1806.

<sup>31</sup> Labarraque: *De la Céphalalgie*, p. 28.

<sup>32</sup> *De Dolore Capitis*. Brussels, 1787.

<sup>33</sup> See an *Essay on Headache*, by Dr. Vaughan, p. 10; London, 1828.

<sup>34</sup> *Transactions of the Society for the Imparting of Medical and Chirurgial Knowledge*, vol. ii., p. 209, by Gilbert Blane. London, 1800.

<sup>35</sup> Ziemssen's *Encyclopædia*, vol. xiv., p. 1.

<sup>36</sup> *Library of Medicine*, vol. vii., p. 155. Quoted by Symonds in the *Gulstonian Lectures* of 1858.

<sup>37</sup> THE LANCET, Feb. 2nd, 1889.

<sup>38</sup> Heubner thinks that the headache which precedes the distinct appearance of cerebral syphilis is due to extra-cranial causes (Ziemssen's *Encyclopædia*, vol. xii., p. 318).

<sup>39</sup> Handfield-Jones: *On Functional Diseases of the Nervous System*, 2nd edition, p. 552.

<sup>40</sup> *Monthly Journal of Medical Science*, September, 1848, p. 137. Seller alludes to a view which regarded the encephalon as devoid of sensation during health, but capable of becoming sensitive under certain morbid conditions, the sensibility being independent of nerves and altogether sui generis.

<sup>41</sup> *Gulstonian Lectures*, No. I., *Medical Times and Gazette*, vol. i. 1858, p. 285. Sir W. Gull, in criticising Romberg's view that hemicrania is cerebral neuralgia, says he regards it as originating in the sympathetic nerves, but it is not quite clear whether he locates the pain within these nerves (*British and Foreign Medical and Chirurgial Review*, July, 1854, p. 103).

<sup>42</sup> *Functional Disorders of the Nervous System*, p. 420.

<sup>43</sup> *London Medical Gazette*, Dec. 27th, 1844.

<sup>44</sup> *Dictionnaire Encyclopédique des Sciences Médicales, Article Céphalalgie*. Paris, 1873.

<sup>45</sup> THE LANCET, vol. ii. 1890, p. 683.

<sup>46</sup> Labarraque, *op. cit.*, p. 37.

<sup>47</sup> *Medical Times and Gazette* vol. i. 1865, p. 488.

<sup>48</sup> *Neuralgia and its Counterfeits*, p. 85.



that the pain of megrim is felt in the extra-cerebral arteries—that it is an arterial colic. Such is the view of Nothnagel and Lauder Brunton. Nothnagel refers to Traube's discovery of sensory nerves in arteries and he assumes that when, as in Du Bois Reymond's case, the pain is attended by arterial constriction of the affected side it is due to contraction of the arterial wall and consequent pressure on the sensory nerves, in the same way as in intestinal colic,<sup>49</sup> adding: "We must look for the pain not in the brain, or dura mater, or the bony skull, but in the sensory nerves of the meninges." Romberg does not attempt to explain the origin of the pain in those cases of megrim in which there is dilatation instead of contraction of the vessels on the affected side, whilst Brunton is inclined to dispute the assumption that simple dilatation or simple constriction ever occurs. He believes that affected arteries are tightly contracted peripherally, but are dilated on the proximal side of the constriction, the result being that the blood is driven with great impact against the constricted part, thus causing the pain.<sup>50</sup> In connexion with these views a passage from Whytt is worth quoting. After pointing out that sympathy with the stomach is one of the commonest causes of headache, he states his belief that by this sympathy "the nerves chiefly of the forepart of the head suffer; and the small vessels to which they are distributed (vaso-motor nerves?) are either affected with a continuous spasm or agitated with uncommon alternate contractions and relaxations; in consequence of which the patient feels a pain, straightness, fulness and pulsation about the forehead and temples."<sup>51</sup> And again he says that headache may be caused by "a viscid or acrid humour obstructing or irritating the vessels of the pericranium, cranial muscles or dura mater, as in gout or scurvy." If for "viscid or acrid humour" one substitutes "uric acid," a remarkable likeness is seen between Whytt's view and that more recent one of Haig which attributes to this substance great powers of contracting the bloodvessels.

Devonshire-street, W.

## THE TREATMENT OF PERICARDITIS.

By D. B. LEES, M.A., M.D. CANTAB., F.R.C.P. LOND.,  
PHYSICIAN TO ST. MARY'S HOSPITAL AND TO THE HOSPITAL FOR  
SICK CHILDREN; EXAMINER IN MEDICINE, JOINT  
BOARD FOR ENGLAND.

THE majority of cases of pericarditis are due to rheumatism. It is a frequent manifestation of this disease, especially in children, and the possibility of its occurrence ought to be constantly kept in mind. Not seldom the associated affection of the joints is exceedingly slight and thus there is good reason for thinking that many of the cases of pericarditis which have been described as "idiopathic," coming on after exposure to cold and wet without any arthritis at all, are really of rheumatic nature. And the cases associated with chorea must be classed under the same heading, for chorea is certainly in most instances a manifestation of rheumatism. Now if we group together all the cases of pericarditis due to these three conditions—obvious rheumatism, latent rheumatism and chorea—we shall find that a large number of cases are accounted for. In rheumatic pericarditis it is common to find also a dry pleurisy, especially on the left side, and occasionally some pneumonia, and this leads to the consideration of a second but much smaller group of cases of pericarditis in which, without any evidence of rheumatism, there is inflammation of the pericardium along with a similar condition of one or both pleuræ, or of one or both lungs. In such cases the pericarditis is sometimes said to have arisen by "extension"; but I very much doubt whether this is the correct explanation, except in rare cases of empyema. It is unlikely that a simple inflammation spreads from the one serous membrane to the other; in most cases of pleurisy the seat of greatest inflammation is far away from the heart and its covering, and a pericardial rub seems to be heard quite as frequently when pneumonia is on the right side as when it is on the left. In addition to this the simultaneousness of the outbreak of the pericarditis and the pleurisy often indicates clearly enough that they are independent results of the same cause. I wish to draw attention specially to cases in which the pericardium and both pleuræ

are simultaneously attacked, sometimes along with pneumonia, at other times without any implication of the lung. Such cases are apt to be very rapidly fatal, indeed they are often admitted into hospital in a moribund condition. I have not seen the peritoneum affected with the other serous membranes in this group.<sup>1</sup> Granular kidney is sometimes asserted to be a common cause of acute pericarditis, but the coincidence of these two pathological states is rare. Very few of the patients with pericarditis have granular kidneys, and few patients with granular kidney are found post mortem to have suffered from pericarditis. In 406 necropsies of chronic Bright's disease collected by Sir William Roberts pericarditis was found not more than thirty times, or in only 7 per cent. Probably in many even of these it was not discoverable during life. Tuberculosis is another occasional cause of pericarditis; in my experience it is not nearly so common as has recently been stated by Dr. Osler. Malignant growths involving the pericardium are a rare cause of pericarditis.

Of 28 cases of pericarditis under my care during the last six years 16 were rheumatic in origin. In 8 others the pericarditis accompanied pneumonia, pleurisy or empyema, without distinct evidence of rheumatism, though the illness had often followed an exposure to cold and wet. In 5 of these 8 cases pneumonia was present, in 6 pleurisy and in 3 empyema. The pleurisy was often on both sides or there was empyema on one side and simple pleurisy on the other. Only one case was due to chronic Bright's disease, one to tuberculosis, one to sarcoma of the left lung which had invaded the pericardium and one apparently to the presence of an aneurysm of the root of the aorta. Thus of a total of 28 cases, 16 were due to rheumatism, 8 accompanied pneumonia or pleurisy, 1 was due to granular kidney, 1 to tubercle, 1 to sarcoma of pericardium and 1 to aneurysm of aorta. Hence 16 cases out of 28 were due to rheumatism, which gives a proportion of 57 per cent. If adherent pericardium were included and the origin considered of all cases of pericarditis and its consequences during life which come under clinical observation, the rheumatic percentage would be much increased, for, as a rule, pericarditis from other causes is fatal.

I need not discuss the ordinary symptoms and physical signs of pericarditis, but there are one or two special points to which I wish to draw attention. The first is this—that a double pericardial rub may at first be so soft in character as to simulate the blowing murmurs of valvular disease, and as it may be audible only over the base of the heart it may imitate almost exactly a double aortic murmur. Thus pericarditis may be mistaken for commencing disease of the aortic valves. This mistake is a very serious one, for of the two lesions pericarditis is much the more serious, both for its immediate and its later results, and it is also the lesion which is the more open to treatment. Especially in children should the possibility of this mistake be kept in mind, and it should be remembered that whilst mitral disease is very common in children aortic regurgitation is rare, so that a fresh double murmur at the base of the heart occurring during an attack of rheumatism in a child is much more likely to be due to commencing pericarditis. It is hardly necessary, perhaps, to refer to the triple rhythm without definite rub, which may sometimes be the first sign of the onset of pericarditis, but I should like to draw special attention to the fact that pericarditis may sometimes exist and run its course, apparently from the very beginning to the end, without any rub at all. I have seen two or three instances of this. They have all been cases belonging to the second of the above groups, in which the pericarditis has accompanied pleurisy or pneumonia. In such instances the pleural condition is usually diagnosed, whilst the similar condition of the pericardium

<sup>1</sup> Since this paper was written a case has been under my care at St. Mary's Hospital in which the peritoneum was implicated, along with the pericardium, both pleuræ, and one lung. The patient was a woman aged fifty-three whose illness had commenced a few days before with shivering and vomiting, followed by diarrhoea. On admission she showed remains of herpes on her right cheek and on the nostrils. There was some dulness at both posterior bases, especially the left, marked pleural rub on both sides, loss of breath sounds at the left base and no bronchial breathing anywhere. The heart's dulness extended to the left nipple line but not beyond the mid-sternum, and no pericardial rub could be heard, though it was repeatedly sought for. The abdomen was tumid, resonant, nearly motionless, and a little tender. The temperature was only 100° to 101°, but the pulse was very rapid (130) and feeble. The necropsy revealed large patches of thick, yellow lymph on each pleura, some consolidation of the base of the right lung, some fluid in the left pleural cavity, a coating of lymph over nearly the whole surface of the heart, and purulent peritonitis, the intestines being matted together with recent adhesions. Her kidneys were not granular.

<sup>49</sup> Medical Press and Circular, vol. i. 1889, p. 29.

<sup>50</sup> Disorders of Digestion, p. 103.

<sup>51</sup> Observations &c., pp. 315, 316.