point of passage through the pia mater at the periphery of the trunk of the posterior roots. It is these fine fibers that go to make up the marginal zone of Lissauer, which is ordinarily the first attacked in incipient tabes. Furthermore, he finds that there are two or three regions of the cord where this constriction of the posterior roots occurs in the most marked degree, viz., the cervical enlargement, and the lower dorsal and upper lumbar regions. These are the regions first affected in locomotor ataxia. At the point where the pia mater normally constricts the posterior nerve root there is ordinarily a vessel more or less circulatory embracing to a greater or less degree the nerve root. Now, if there is any chronic change produced by syphilis which is more constant than another it is arteriosclerosis, a thickening of the vessels generally throughout the system, or localized more here or more there. Redlich concludes that at this point of normal constriction a very slight meningitis will induce a very marked change in the nerve root and in the next place, an affection of this vessel at this place will also produce relatively marked changes in the nerve fibers without very extreme changes taking place in the vessel. There is localization which coincides with the ordinary beginning place of the lesions of locomotor ataxia. Again, it has been shown very conclusively that the lesions in the cord in locomotor ataxia are practically and substantially those of ascending degeneration, which may be produced experimentally or by pathologic processes destroying the posterior roots. I would simply state this, that in considering the so-called complication of syphilitic meningitis with locomotor ataxia, we ought to consider at the same time whether the locomotor ataxia is not really the sequence of the syphilitic meningitis instead of the latter being a fortuitous complication of the former. While that is not conclusive, I think it is to be considered, for the peripheral origin of locomotor ataxia has very strong arguments to support it.

Dr. J. G. KIERNAN said there was one phase of the subject which deserved a little more attention than has been paid to it, namely, whether the luetic lesions were not often the result of lues developing secondarily to the tabes rather than a primary condition. Miner, of Moscow, asserted that the cognate disease, parietic dementia, never occurred except in the victim of inherited or acquired syphilis and asked for cases in which syphilis had occurred in parietic dementals after the development of the parietic dementia. Rippler and a number of others had previously cited cases. Dr. Kiernan had seen eleven such cases, in which the parietic dement in consequence of the disease had contracted lues which ran its course *pari passu* with the parietic dementia. What is true of parietic dementia is very apt to be true of the cognate condition, locomotor ataxia. Pournier places these two conditions, parietic dementia and locomotor ataxia, among the parasyphiloses.

With regard to the cases cited, Dr. Kiernan recalled cases of parietic dementia in which there were tabetic symptoms, and in which cerebral gummata were found. There were also luetic changes in the spinal cord as well as meningeal gumma. Probably they were cases in which, if the lues coëxisted with the locomotor ataxia, it produced the ordinary pathologic changes of the latter disease, and later the gummata.

With reference to one of the points raised by Dr. Patrick, Dr. Kiernan remembers making autopsies in cases of ataxic mental trouble which had led to ataxics dying in an insane hospital, in which a complete examination of the spinal cord, meninges and brain was made and the findings were absolutely nil. There was nothing truly pathologic found either microscopically or macroscopically. Colloid spheres had been found and artefact of that character produced by alcohol. But these artefacts excluded, no other lesions were present. The question here certainly could not be raised whether the meningeal condition was not the primary one and the other changes secondary.

Dr. HAROLD N. MOYER—I am impressed with the progress of this discussion, from the fact that my neurologic friends do not understand all there is to be known about the pathologic anatomy of the nervous system. Indeed, I think it is still in a most chaotic state. We have to unlearn the subject almost as rapidly as it is acquired, and we are getting down, as Dr. Kuh has admirably done in his article, to a thorough, careful study of a few cases, though taking in the whole range of the nervous system; in this way we shall have an accumulation of observations which will perhaps place the pathologic anatomy of nervous diseases on a more substantial basis. When one stops to think of the extent and variety of function and the possibility of change in this extended nervous system, it is no wonder that the pathology of other organs of the body should be far better elucidated. I rise to commend Dr. Kuh's paper, not to discuss it or to differ with him. On a former occasion he drew

extended clinical deductions from pathologic observations with which I did not wholly agree, but I concur heartily in what he has said to-night, and I wish simply to commend this masterly observation in neuro-pathologic anatomy.

Dr. KUH (closing the discussion)—The discussion has opened up an immense field, but I shall not detain you by dealing with all of the points which have been touched upon. I wish to reply briefly to one or two questions which have been raised. First, as to the Redlich theory of the constriction of the posterior nerve roots. It is a very seductive theory. There is only one fault to find about it. There are any number of cases of locomotor ataxia on record examined during the first stage of the disease in which there is not the slightest trace of any changes in the meninges, and I can show Dr. Patrick some specimens which will corroborate this statement. This being true, it does away with Redlich's theory entirely. Beside, I need but call Dr. Patrick's attention to the fact that a very large number of cases of locomotor ataxia begin with cerebral symptoms; how they can be due to constriction of the posterior nerve roots in the lumbar region is something I fail to see.

Dr. Kiernan's theory, that the syphilis in my case might possibly be the consequence of tabes, is very improbable. We can all understand how a patient suffering from parietic dementia is liable to expose himself to the danger of contracting syphilis on account of loss of control over himself, his tendency to indulge in excesses, from lack of judgment in selecting proper victims, etc., but the same thing does not hold good in locomotor ataxia. I mentioned in my paper that one of the first symptoms was impotence, which is more frequent than satyriasis. Besides, we know positively that in this case syphilis did exist; that the patient had contracted a chancre when 20 years old. I have not claimed that the meningitis in this case was the cause of the locomotor ataxia; in fact, my impression is that the meningitis was of much more recent date than the changes in the posterior columns, and it was not by any means more marked around the posterior half of the spinal cord than the anterior. The only gumma found was in the anterior portion of the cord.

Another point which may be of interest is this: We know that a number of cases of locomotor ataxia are improved very materially by anti-syphilitic treatment. Dinkler's statistics show that improvement took place in 75 per cent. of his cases. If specific meningitis is as common a complication of locomotor ataxia as I am inclined to believe, then we have in its existence the explanation for the effects of anti-syphilitic treatment. We all agree that neither mercury nor iodid of potassium can restore the destroyed fibers in the posterior part of the spinal cord; but we can understand how other symptoms might be added and existing symptoms aggravated by meningitis, and that when the latter is improved there results an improvement in the condition of the patient.

SPECIFIC TREATMENT OF NECROSIS OF THE ALVEOLI AND MAXILLÆ WITH AROMATIC SULPHURIC ACID.

Read in the Section on Dental and Oral Surgery, at the Forty-sixth Annual Meeting of the American Medical Association, at Baltimore, Md., May 7-10, 1895.

BY W. A. MILLS, D.D.S.

BALTIMORE, MD.

I desire to bring before your attention the non-operative treatment of necrosis of the alveoli and maxillæ with aromatic sulphuric acid. I shall cite only two cases in office practice, entering as little as possible into any minute detail of their history or etiology.

Case 1.—A lady of nervo-bilious temperament, aged 30, called to consult me about a fistulous opening situated at the right side of the superior central incisor which was discharging freely a dark-colored pus. On examination, I found the superior right central and lateral incisors and cuspid teeth dead. Patient informed me they had been so for seven years or more, and had never been treated. I diagnosed the case as necrosis superinduced by chronic abscesses. I prescribed the following:

R Acid sulphuric aromatic ʒ ij. 60
Aqua. f ʒ x. 300
Misce.

To be injected by the patient into the fistulous opening five or six times daily.¹ The patient was also instructed to visit the office every other day for examination.

¹ Bicarbonate of soda and water, used as alkalin mouth wash after every injection.

At the expiration of two weeks all discharge had ceased, the soft tissues had fallen into the cavity, made by the chemic action of the aromatic sulphuric acid upon the necrosed tissue. An incision was made extending from the fistulous opening to the right first bicuspid. The cavity was then packed with absorbent cotton saturated with the following:

R	Acid carbolie	
	Tinct. iodine	5 ss. 2
	Aqua	f3 xij. 360
	Misce.	

Patient was then dismissed to return the next day, when cotton pledget was removed. The soft tissues having been pushed aside, I was able to see to what extent the bony structures had been diseased. I found the line of necrosis had involved the right facial surface of the superior maxilla from the left central to the right bicuspid teeth and upward from the alveolar ridge to the anterior nasal spine, a part of which with bony structure around the apices of the dead teeth had been destroyed; only sufficient alveolar septum remained to hold them in position.² I opened the nerve canals of the dead teeth and cleaned, disinfected and filled them at once. The filling material was forced through the apical foramina, from the posterior surface, and dressed in the usual manner. The patient was then dismissed with instructions to syringe out the cavity twice a day with the carbolie acid and iodine wash, as long as syringe could be used; afterward the medicament was to be used as a mouth wash. In five weeks new bone-tissue had filled the void, when no suspicion of the parts having been diseased remained, the outline being quite perfect, and all the tissues in a normal condition.

Case 2.—Mr. T., a lawyer, aged 40, of sanguino-bilious temperament, presented the following conditions: A fistulous opening situated to the right of the left inferior cuspid tooth, another to the left of the right inferior cuspid; both openings discharging pus copiously. This condition had continued for over a year, and Mr. T. failing to get any relief from many medications, he consulted a surgeon, who diagnosed his case as necrosis, caused, the surgeon said, by the toxic effect of mercury or syphilis. To either being the cause Mr. T. protested most vehemently. Mr. T. was informed that he would have to be operated upon. The first thing the surgeon suggested was to have all the incisor teeth extracted in the hope that nature would have a better chance to throw off the sequestrum. Mr. T. did not object to having the operation performed, but he did object most strenuously against having his teeth extracted, as he possessed a beautiful set, being perfect in form and arrangement. He consulted me to find out if I could in some way manage to save his teeth. I suggested the sulphuric acid treatment, which I thought would not only save his teeth, but save him from having to undergo any operation. After consulting with the surgeon it was agreed that I should take charge of the case, with the understanding that I was to consult with the surgeon as the case progressed, and not, under any circumstances, change treatment without his knowledge. The treatment in this case was the same as in case of number one. After the first day's treatment Mr. T. came to the office early in the morning with a very distressed countenance, and said he believed I had aggravated the case, because the flow of pus had been so great during the night that he could scarcely sleep. I assured him it was a good sign and that the remedy was doing its work well. The first week the teeth became movable and tender to the touch. A vulcanite splint plate was made to hold the teeth in place and to protect them from shock during the process of mastication. At the expiration of two weeks all discharge had ceased. Then an incision was made from the left fistulous opening to the right, the cavity packed with absorbent cotton saturated with the carbolie acid and iodine mixture (as in first case) and left to remain until next day. When pledget of cotton was removed the following con-

ditions were presented: Line of necrosis found to have involved the facial surface of the inferior maxilla, from cuspid to cuspid, slightly exposing the nerves at the apices of two central incisors. The septa were nearly destroyed, the teeth were only held in position by their attachment to the external or posterior plate of the alveolar process and gum tissue. Same instructions for injection and mouth wash were given as in former case. In six weeks Mr. T. was pronounced cured—teeth all living and firm, and the continuity of maxilla outline fully restored.

I not only received the congratulation of the surgeon upon the successful treatment of the case, but the most grateful thanks of Mr. T. I take no credit for the successful treatment of these cases, but will give all honor and praise to Dr. Gross, of Philadelphia, who, I believe, was the first to suggest the sulphuric acid treatment for necrosis. Some one may ask the question: How does the sulphuric acid act? In reply I will say, that the bony structures of the human organism contains 33 per cent. organic matter, 4 per cent. water and 63 per cent. mineral matter, 60 per cent. of which is carbonate of lime. The action of the sulphuric acid is to seize chemically upon the carbonate of lime, break it down or dissolve it, throwing off carbon dioxid gas; and subsequently the animal tissues, etc., break up through a process of fatty degeneration and flow out in form of pus, etc. The acid in the strength prescribed has no effect upon healthy tissues, except to stimulate them. The advantage of this treatment in the oral cavity over that of operative is: The periosteum is in no way injured, thereby not causing a loss of the continuity of outline of any structures, nor the loss of teeth through the necessary extraction in removal of the sequestra.

Miss G., the first case described, told me not many weeks ago she had not fully appreciated what I had done for her until after she had made a visit to Germany, some three years ago. She informed me of a cousin living in Germany who had a similar case with her own, age the same, conditions about the same, but the treatment and final result quite different. In her cousin's case the teeth involved had been extracted by a surgeon prior to the removal, by operation, of the sequestrum. During the operation the periosteum had been injured, causing a loss of a great part of the maxilla; in consequence her face was disfigured for life. Moreover, she had never been able to have the continuity of outline restored by any artificial means. Now, suppose the two cases I have cited had been treated by operation, in like manner, what would have been the consequences?

In conclusion, I would call the attention of the surgeon, no matter how prone he may be to operate in the oral cavity, not to do so before he has failed in the sulphuric acid treatment, or use more conservative surgery than is usually practiced in similar cases.

THE PROGRESS OF MODERN METHODS IN DENTAL PRACTICE.

Read in the Section on Dental and Oral Surgery at the Forty-sixth Annual Meeting of the American Medical Association, at Baltimore, Md., May 7-10, 1895.

BY ALTON HOWARD THOMPSON, D.D.S.

TOPEKA, KANSAS.

The marvelous success of modern dental practice is founded upon two main elements, viz., *mechanical skill* and *medicinal precision*. In regard to the first named element, mechanical skill, the profession has been noted for that for many years. The best practitioners of fifty years ago were famed for artistic taste

² A similar case is noted by Dr. Black in American System of Dentistry, vol. 1, p. 950.