An Address

ON

THE UNCERTAINTY OF POST-MORTEM EVIDENCE IN SUSPECTED LEAD POISONING.

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THE scope of this paper is not so wide as the title might imply. Cases of industrial lead poisoning only are under consideration, in which access to the poison and the possibility of its absorption are undisputed. It is, however, precisely in these industrial cases that conclusions are often reached on, as it seems to me, totally insufficient grounds. Dangerous occupations do not exclude the possibility of death from other causes, and, although such a statement may sound a truism, I am convinced that its applications are frequently neglected in the "lightning" diagnosis of industrial diseases. To quote Tanquerel des Planches, "Not every trouble in a lead worker is due to lead poisoning, just as not every trouble is cancerous in a cancer patient or gout in the gouty."

Nothing should stimulate accuracy and caution in judgement more than the prospect of having to enunciate one's views before a jury, and this stimulus is supplied in cases of fatal plumbism. Since the early months of 1904 compensation is payable to the relatives of workers in china and earthenware whose deaths are certified, after due inquiry, to be due to lead poisoning. Inquests are held on every suspected case, and the verdict of the jury largely guides the insurance companies in their payment of claim.

In many of the cases, little or no room for doubt exists as to the propriety of the verdict, since typical clinical histories are forthcoming. It is a reasonable assumption, for instance, that death has resulted from lead poisoning when the deceased had been for some time under suspension on account of plumbism (receiving weekly certificates to that effect) and was presumably under attendance for the original and final symptoms.

Workers are suspended for various reasons, and I append a rough classification of their value, in diminishing order of likelihood that death, if it follows, was due to plumbism.

- (a) Headache and optic neuritis.
- (b) Paralysis, especially when in combination with anaemia.
- (c) Hysterical manifestations, in previously healthy subjects.
- (d) Grave anaemia.
- (e) Colic.
- (f) Anaemia.

When death succeeds (a) and (b), the verdict must necessarily attribute it to lead poisoning, unless other grave intercurrent diseases were known to be present. In (c), (d), (e) and (f), where these symptoms, although justifying the suspension, had then rather a warning than a diagnostic significance, the presumption is that death was due to the supervention of the severer complications of plumbism, and probably a clinical history to that effect would be afforded by the practitioner in attendance.

In a second group, we may place those cases in which death has taken place, although the worker had presented no symptoms at the previous monthly examination which called for his suspension. Such interim fatalities, when attributable to lead, are usually due to sudden encephalopathic troubles, in which the whole interval between the sudden seizure and death may be only a few days. If a suggestive uncomplicated clinical history is given on these lines, it calls for a verdict in correspondence therewith.

There remains a third group of cases, in which death overtakes an apparently healthy worker suddenly and unexpectedly, or terminates an illness of an entirely anomalous character, *qua* lead. It is to this group that I wish particularly to direct attention, for there is danger here that an unwarrantable verdict may be given by a jury prone to *a priori* reasoning, or liable to be influenced unduly by dogmatic but unsound statements made by medical witnesses.

It is very natural that a jury should presume *post-mortem* evidence to be decisive and irrefutable, and the insignia of

lead poisoning to be as visible and tangible as the bullet found in the brain of the suicide. But, as a matter of fact, the pathologist is confronted by an aggravating and perplexing uncertainty; at most, the indications found are not combined in inevitable and pathognomonic association, and, on the other hand, a decisive clinical history may have to stand destitute of any support that morbid anatomy could give.

This third group, in which obscure illnesses give ground for so much speculation and argument, embraces several morbid conditions. Some of the possibilities may be indicated here with a suggestion of the alternatives in diagnosis which should be borne in mind (apart from the direct evidence of other causes of death which a *post-mortem* examination may reveal).

(a) Death from Colic.

The rarity of the recorded instances is much against this diagnosis—only 1 in 6,000 cases being known to Tanquerel des Planches. Death following attacks of quasi-colic is more probably due to such causes as acute gastritis, appendicitis, complications of renal or hepatic colic, perforation of ulcers, rupture of ectopic gestation, etc. In those cases where the patient has died before the doctor could be called, the difficulty of deciding the true cause of death naturally becomes infinitely greater. In one such case in my experience death was assigned to colic on the *post hoc* principle so common in industrial maladies, although the previous history pointed clearly to an acute gastritis of alcoholism, resulting in vomiting and syncope.

(b) Very Rapid Paralysis.

Assuming this to be of the extensor type, the extreme rapidity of an attack contraindicates lead as its cause. Sir W. Gowers says that from lead there is never immediate considerable paralysis, complete development being always spread over some days. There should be no difficulty in calling to mind the more reasonable causes of such conditions, according to the region affected, and it is unnecessary to enumerate them here. In hemiplegia due to cerebral haemorrhage, occurring at a comparatively early age, the influence of lead in the production of granular kidney must be remembered. The possibility of alcohol as an equally important contributor must, however, be estimated, while the degenerative processes of syphilis, purpura, etc., may be responsible in some cases.

(c) Convulsions, Delirium, and Coma.

These symptoms, in true lead-encephalopathies, may exist separately or in combination. Other notable causes of such phenomena suggest themselves at once, and should be excluded or reckoned in the diagnosis. Those most frequently met with include uraemia, traumatism, true epilepsy, otitis interna, alcohol, belladonna, and other poisons.

(d) Abortions due to Lead, followed by Fatal Sepsis.

It is notorious that the abortifacient in many of these cases is not the lead absorbed during occupation, but a lead compound, such as diachylon, taken for a specific purpose. I need hardly say that it is extremely difficult to obtain evidence of this, as one is met by an impenetrable conspiracy of silence.

(e) Accidental Ingestion of Lead Compounds.

I may cite here a case in which very severe and extensive paralysis followed the consumption of the contents of a tin of sardines which, after being opened, was filled up with vinegar and allowed to stand for some hours. The resulting acetate of lead, derived from the dissolved solder, was of course responsible for the illness.

When death has resulted from one or other of these conditions, and a provisional diagnosis of suspected lead poisoning has been made, a *post-mortem* examination is invoked to decide the question. We can better realize the untrustworthiness of the pathological data which will be available as material for deduction if we review the morbid anatomy of a lead worker who has died in classical fashion—that is, with symptoms of encephalopathy after previous attacks of colic and paralysis.

The body is probably pale and thin, with obvious muscular wasting. The gums show the well-known blue line. Although, clinically, its appearance under various conditions—which cannot be discussed here—may have given ground for action, recognition of this line does not help us in an industrial *post-mortem* examination, because the opportunities for absorption consequent on occupation are not in dispute.

The contents of the thorax present no abnormalities, except such as are associated with general malnutrition.

In the abdomen interest centres on the liver and kidneys. The liver is probably the seat of a cirrhosis—an inflammatory hyperplasia round the biliary tubules; but the microscope is necessary for the appreciation of this, and I question whether lead, unassisted by alcohol, ever gives rise to marked naked-eye cirrhotic appearances. Certainly, the yellow liver of typical cirrhosis is never seen.

The change in the kidneys, if there is any disease at all, will probably be of the red granular type—the organs diminished in size and weight, the cortex shrunken, and the capsule adherent, with the microscopical appearances characteristic of interstitial nephritis. In this relation, however, it is only fair to quote the late Dr. Arlidge, who says, in his *Diseases of Occupations*:—"My long and extensive experience among the victims of lead poisoning in the Potteries has convinced me that the general assumption of the connexion between plumbism and kidney disease does not hold good, at least not in the case of potters exposed to lead. Among hundreds of cases of wrist-drop and other varieties of paralysis, as well as of colic and constipation from lead, the existence of albuminuria will be looked for in vain, and when found its ratio among workers in lead is not greater than it is among the general population."

Other observers in the Midlands and the North of England have pointed out the scanty connexion between gout and lead poisoning in their districts, although the two diseases are so frequently associated in London and the South. The explanation is not clear, but alcohol seems mainly responsible for this difference in geographical pathology.

In cases of acute encephalopathy, after a short exposure to lead, Dr. Oliver has noted a true parenchymatous nephritis. I have not seen this, possibly because the few fatal cases of encephalopathy in my district have occurred in girls who have worked for some considerable time in lead, in circumstances which did not expose them to the large, immediately toxic doses associated with employment in white lead.

Dr. Dixon Mann pointed out some years ago, as the result of most careful analyses, how very small is the amount of lead found in the urine compared with that found in the faeces. Even that complement of the metal which has been absorbed into the circulation is mainly eliminated by the bowel. If, therefore, the bulk of the excretory duty falls on the intestines, the comparative immunity of the kidneys becomes comprehensible. A combination of lead and alcohol, however, is very active for evil, and enormously increases the liability of the kidneys to disease.

Assuming for a moment the possibility of death from uncomplicated colic, we should expect to find evidence of this in the intestines. In guinea-pigs experimentally poisoned the gut has been found tightly contracted at intervals, but this condition has not been seen in man. The picture of painful tetanic spasm, which we form from clinical observations, would lead us to expect areas of contraction, with practical obliteration of the calibre, but no such *post-mortem* condition has been reported. In one case at which I was present, a medical witness considered that importance was to be attached to slight contractions of the intestine at irregular intervals, somewhat like the divisions in a series of sausages. Their small length—about $\frac{1}{4}$ in.—and the ease of extensibility of the little rings, combined with the fact that the whole intestine was empty, seemed to me to negative their pathological importance entirely.

Sir W. Gowers considers that the colon is the seat of the pains of lead colic, but does not give reasons for his opinion, which, to help us, should be based on morbid appearances. It must be remembered that no diagnostic significance can be attached to contractions of the intestines, seen occasionally after illnesses whose mode of termination would suggest that the condition was a mere "agony" phenomenon.

The brain may not, and probably will not, show any abnormal appearances whatever. Occasionally its substance has been reported as appearing firm, resistant, and pale, as if compressed, with some obliteration of the convolutions. But such appearances at best are indecisive, and, moreover, are certainly not found in the majority of cases. Indeed, the absence of any visible changes becomes reasonable when we reflect how insignificant is the apparent anatomical disturbance after years of true epileptic attacks. That changes have taken place, and those of the gravest character, it is impossible to doubt; but they are concerned with bio-chemical molecular phases, of which we know practically nothing with our present methods of investigation.

The muscles and nerves, apart from the obvious naked-eye wasting, show the ordinary degenerative changes under the microscope, characterized by increase of the nuclei, fatty and granular degeneration, and, in the case of the nerves, a breaking up of the medullary sheaths and axis cylinders.

Chemical analysis of the organs, especially the brain, liver, and kidneys, discloses the presence of lead, generally in quantities estimated by milligrammes. The presence of lead in the tissues is to be expected; indeed, its absence would be a matter for surprise. It is manifestly impossible for any lead-worker to ply his occupation without constantly absorbing lead by the mouth and nasal passages. It is equally certain that there is as constant an elimination by the bowel and kidneys. Elimination, however, is slow, and cannot be considered to correspond with the daily ingestion, so that the tissues must retain a certain amount in store. We know little or nothing, however, of the conditions of storage, or of the changes which make for its solubility and the production of toxic effects. Analysis will reveal simply the presence of so much lead—a grain or two, perhaps—in an organ, which amount may lie dormant and harmless for years. And yet, in ill-comprehended conditions, a fractional quantity of this, inappreciable by the scales, may suddenly provoke a fatal toxæmia. In one recorded case of encephalopathy, actually no lead at all was found in the brain. To complicate matters, some authors (Gautier, Devergie, etc.), assert that lead is found normally in healthy tissues, as a result of the fact that each of us takes daily half a milligramme of the metal into the body. The actual amount found on analysis cannot therefore justify any conclusion as to its active toxicity, since analysis cannot distinguish between what we may call *plumbum mortuum* and *plumbum vivum*. It is more reasonable to believe that lead causes disease, not by its direct action, but rather by so reducing the excretory powers (mainly of the bowels and kidneys) that toxic products are accumulated, and a true toxæmia results. It is interesting, in this connexion, to call to mind Sir Andrew Clark's theory of the dependence of anaemia on constipation. The anaemia associated with lead may, after all, be due to its constipating effect, and not to its direct interference with hæmatosis.

If an undoubted case of lead poisoning can offer no better *post-mortem* evidence than this just summarized, how trivial and untrustworthy must be the appearances which are put forward as proofs of death from plumbism in workers who die suddenly of mysterious maladies. The *post-mortem* examination may, of course, disclose the existence of aneurysm, cerebral tumour, apoplexy, phthisis, etc.; but, on the other hand, an obvious cause of death may be sought in vain. Is it fair, in the absence of a clinical history suggestive of plumbism, coupled with completely negative *post-mortem* evidence, to ask a jury for a verdict of death from lead poisoning? Nothing can be more unreasonable, in my opinion.

The attempt is sometimes made to construe the statement that a certain condition of an organ was compatible with lead poisoning into an admission that the condition was caused by, and actually indicated, the disease. Without commenting on the impropriety of these ingenious perversions on the part of a legal advocate, I should like to point out how careful a medical witness should be not to allow any such unjustifiable extension of his statements. The average jurymen is not likely to appreciate the exact differences between compatibility and causal connexion when specious inferences are dangled before him. To give an example *ad absurdum*, the existence of ingrowing toe-nails and a bald head is compatible with lead poisoning, but neither condition has the slightest diagnostic value in reference to it.

Again, it often happens that at certain inquests "bogus" clinical evidence is advanced by the deceased's friends, who give details of attacks of colic, headache, etc., complaints of which the worker, it is said, had suppressed during his lifetime "for fear of being stopped." When we consider that compensation is given to any worker whose suspension is deemed advisable on account of his report of suspicious symptoms, it seems hardly possible that such silly reticence should be maintained. The whole system of monthly examinations is rendered futile unless the worker co-operates by reporting honestly his subjective troubles, since these, rightly interpreted, are of more value as indications for suspension than the physical signs of plumbism which develop later on.

On the other hand, it is extremely difficult to elicit from witnesses who are related to the deceased worker a distinct history of alcoholism, even although the ravages of drink have been manifest at the monthly inspections for some time

before death. The guarded and scanty admissions of relatives at the inquest contrast forcibly with the authentic reminiscences of disinterested friends whose evidence has not been called. But, humanly speaking, it is too much to expect a really honest biography when claims for compensation are in the wind; the expectant heirs must naturally be to the "virtues very kind," to the "faults a little blind."

To sum up: In those cases where death has been preceded by well-marked signs of plumbism, with previous suspension (and it is only fair to say that such cases form a majority), the clinical history is sufficiently decisive to warrant a verdict of death from lead poisoning. A *post-mortem* examination supports the clinical evidence to the limited extent indicated above, presuming that it does not disclose another more reasonable, but previously unsuspected, cause of death.

To a minor degree the same conclusion holds for those cases of fatal illness of rapid intermense development, with no prodromal troubles demanding suspension, but with clinical characters entirely compatible with acute lead poisoning. The *post-mortem* evidence in such instances will most probably be of an absolutely negative character, and valueless as a confirmatory contribution to diagnosis, but a verdict of death from lead poisoning will follow legitimately on the recital of an adequate clinical history.

But the vague, anomalous illnesses of which I have spoken, irreconcilable with any known form of lead poisoning, can never be delimited and defined by an appeal to the negative indications of morbid anatomy. A *post-mortem* examination will be valueless unless it happens to demonstrate the existence of independent organic lesions, discovery of which serves to exculpate the suspected lead.

If a coroner and jury expect mathematically-exact statements in these matters, where the limitations of pathological research make absolute certainty impossible, they must remain disappointed; and I hold it unworthy conduct on the part of a medical witness to maintain a dogmatic attitude, when his statements are based mainly on untested prejudices, and but slightly on observed truths.

ON OPERATION FOR CLOSURE OF CLEFT PALATE IN INFANTS.*

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OPERATIONS for the closure of clefts in the hard palate have not undergone any striking developments in late years, except in one particular, and that is the introduction by Mr. Brophy, of Chicago, of a new method of closing the cleft soon after birth by approximation of the superior maxillae and palate bones. The development of the surgical treatment of cleft hard palate divides itself readily into three periods:—

1. That commencing with the practice of closing the cleft by operation, introduced by Warren, of Boston, about 1843.

2. That dating from developments of methods during the years immediately preceding 1868.

3. That inaugurated by the operation of Mr. Brophy, described in his paper in *The Dental Cosmos* of April, 1901.

1. Dr. Warren must be regarded as the pioneer of the operations for cleft hard palate. Up to the time when his paper appeared in the *American Journal of Medical Science*, in 1843, no method existed of dealing with this deformity, except by the application of obturators, which are never satisfactory, and are only in these days resorted to where special measures have failed.

2. The work of Sir Thomas Smith has probably lent itself more to the perfection of the operation on the hard palate than that of any English surgeon. His paper, published in the *Med. Chir. Trans.* for 1868, inaugurated the second period in the history of uranoplasty. He advocated the operation in patients of tender years, recommended the use of chloroform, and devised his well-known gag to assist the surgeon; and this when Fergusson two years later could only say that he had resorted to the proceeding with partial success, and recommended it for further trial. It is true that in the list of cases published in 1868, Smith in all operated on the soft palate only, and left those in which a cleft hard palate existed, to be dealt with later on. This will be admitted by those who

have practised Brophy's operation to be inadmissible to-day, when it will be found better to defer the closure of the cleft in the soft palate until the defect in the hard structures has been corrected.

Since the years preceding 1868, the age selected for closing the hard palate has steadily become earlier. At first it was usually done after puberty. In 1870 we find Mr. Pollock advising that it should be delayed for two or three years after birth, and now it is open to operate by Brophy's method at any time during the three months after birth.

3. The operation of Mr. Brophy has been done by him at from ten days to three months of age. It constitutes a new departure, not only on account of the early period of life at which it is done, but because it is novel both in principle and method. It consists essentially in drawing the upper maxillae and palate bones of opposite sides together by wire sutures, generally two in number, passed transversely through the alveolar processes above the level of the palate processes of the maxillae and palate bones. There are details of the operation which will be best understood by following Mr. Brophy's own description. The method cannot be successfully carried out after the child is three months old, as the bones are then too fully ossified to be displaced by any force that could be safely applied. I have had the opportunity of using this treatment in three cases, and may at once say that I shall use it in future when suitable subjects offer, reserving, however, a few minor points of the procedure about which to offer an independent opinion.

I may epitomize Mr. Brophy's plea for his method as follows. He advocates its employment during early infancy for the following reasons:—

1. "The surgical shock is less, because the nervous system of a child is not well developed, and it is not, therefore, capable of receiving the same impressions that it would later in life, for young children usually react better. Moreover, all mental apprehension is eliminated, and we know that alarm and dread are among the most powerful factors in producing shock."

2. "Before the bones are fully calcified they may be bent or moved without fracture, etc."

3. "If the muscles are very early brought into action they develop instead of atrophy, and hence a good return is secured, with plenty of tissue; whereas if the operation is undertaken later in life, after the parts are shrunken through non-use, they can rarely be made to subserve the same purposes as organs which develop through natural employment, etc."

4. "When the palatal processes of the maxillae are united, it will be observed that the development of the bones of the alveolar processes of the upper jaw assume a form nearly or quite normal, and when the teeth are erupted they will properly occlude with the lower ones, or nearly so. . . . It was predicted by my surgical friends that the upper jaw would be much narrower than the lower one, and that it must always remain contracted." Mr. Brophy then proceeds to show that this has not been the case, and that no such deformity is likely to occur beyond what may be corrected by orthodontal methods.

5. "Following early operations there is much less deformity, for all the tissues, bony as well as soft, develop naturally and according to accepted types."

6. "When the operation is made in early infancy, the parts are sufficiently advanced to give possibility for normal speech when the time comes for learning to articulate. If the operation is not made until faulty habits are acquired it is with difficulty that they can be overcome, even supposing the muscular parts could be made sufficient."

These arguments are too sound to require much comment. With reference to the first I may say I have been struck with the absence of shock or other grave symptoms. In none of the cases I have done has there been any cause for alarm. Even when the anaesthesia was prolonged for nearly three-quarters of an hour, or when a good deal of blood for a tender infant to lose was lost, I have seen far less disturbance than is frequent in shorter operations, with less severe bleeding, at a later period of life. One of the matters, however, requiring attention when operating on very young children is the avoidance of bleeding, and some suggestions later on in alteration of Mr. Brophy's technique are worthy of attention.

The second observation, with reference to the absence of tendency to fracture, requires a remark. In the last case I operated on the child was six weeks old, and the gap in the hard palate of great width. The sides could not be brought

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