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Zinc deficiency

Shuttleworth, in this paper entitled 'Zinc in perspective', gave an historical overview of the discovery of the role of zinc in plants, animals and man. It is remarkable that, before 1960, deficiency in man was unrecognised and considered unlikely; in spite of known deficiency syndromes in citrus fruit and other crops, and markedly in pigs. The author described the pioneering work of Prasad in the early 1960s with peasants in Iran and Egypt, rounded off a decade later by Halsted, also in Iran. He commented that mild deficiency is prevalent throughout the world, even in infants in the USA when fed with infant foods lacking in zinc. He referred to the steady evolution of knowledge of deficiency in special situations, citing eight examples, including alcoholism, chronic enteritis, susceptibility to infection, and burns and wounds. His reference to the difficulty of laboratory diagnosis might explain the late appreciation of zinc deficiency in man. Serum levels are frequently normal in mild deficiency, and are not a measure of bioavailability.

The author then discussed the value of homeopathy in this condition, pointing out that zinc does not have a well defined homeopathic picture; apart from 'fidgety legs', with emphasis on the compulsive, repetitive nature of the symptom. However, it is interesting that the old homeopaths referred to 'depressed, exhausted and irritable condition of the nervous system such as may arise from a variety of causes principle among which are injuries, sexual excesses, etc', while it is now known that some of the tissues richest in zinc are the prostate, testis and spermatozoa (1 mg or more in an ejaculate); and to 'weak memory—difficult conception, incoherent ideas, etc', while the parts of the brain richest in zinc are the pineal gland and hippocampus. Shuttleworth commented that 'in modern nutritional

literature there is little reference to the mental state and feelings of zinc deficient patients', and in conclusion he considered that 'Medical homoeopaths generally take a detailed comprehensive multi-angled history from patients, and are therefore in a position to evolve a coherent *Zincum* picture correlated to our modern nutritional knowledge. Zinc obviously has therapeutic potential'.¹

A model for drug tests with statistical analysis

In this paper, G Bayr described a model which had been used in both Germany and Austria since 1980 to carry out drug tests (provings). He stated that drug registration laws now require a different kind of data including proving data. It has to be shown that the symptoms recorded by subjects taking the drug under investigation are not due to expectation or occurring at random but are on the whole drug specific. The aim was to find a method of statistical analysis which would give confirmation of drug specificity; the author outlined the methodology and reasons why statistical analysis based on individual symptoms, as used previously, is not possible. He stated 'It is therefore necessary to compare the totality of symptoms produced while the drug was exhibited in a test with the totality of symptoms recorded whilst placebo was taken, adequate placebo phases being recorded in the model. This will make statistical analysis at least admissible.'

In this present model there is established a new system of evaluation. Bayr described how comparison is now made, not of symptoms or groups of symptoms elicited on exhibition of drug or placebo, but *the totality of symptoms* recorded during exhibition of the drug or placebo is compared with the totality of placebo symptoms. The single parameter or frequency or incidence has been replaced by *a number of new*

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parameters. The provings continue for 3 weeks, using low potency and high potency of drug, and placebo, each for a week. The author now detailed three cleverly worked out features. The first feature is the elimination of habitual symptoms; those noted more or less frequently during the preceding 6 months. They are ignored, unless there is a change, either aggravation or improvement. The second involves the drawing up of symptom lists for each week, whereby only those symptoms first recorded in any 1 week are included, while the same one in subsequent weeks is discarded. This deals both with the problem of continuing drug action into a placebo week, and the possibility of a placebo symptom causing distortion of the subsequent drug week. The third feature, a new element in the evaluation scheme, was the finding of as many potential weightings as possible, which would permit quantitative assessment of aspects peculiar to symptoms, for the purposes of statistical analysis. The parameters used for this are; duration of symptom; objectivity (evident not only to the subject); intensity; periodical nature; other striking features (eg 'as if sensation'); and remarkable improvement. Duration yields a range of figures, while the others (with yes/no decisions) yields only 0 or 1.

On decoding, the symptom lists for individual weeks are combined in lists for the whole proving; and lists for high potency and low potency, these also cover the weighting given to individual symptoms. It was hoped to present the results of the first drug test on this model in due course. This paper presented the model as such for comment and discussion.²

The miasms in contact thermography

The first two pages of this paper by Arno and Jutta Rost describe the use of contact thermography and its value in early diagnosis of both local and general pathological processes; heat being the essential product of intermediary metabolism, and therefore reflecting the quality of the life processes. They refer to the two previous methods of thermography; the bolometer measuring infra-red radiation developed by Ernst Schwamm over 30 years previously; electronic thermovision; and plate thermography with liquid crystals, producing colour zones.

The authors found that these three methods were not adequate to meet their aims, but the most recent, contact thermography, did meet their requirements. It is not clear why, and what their aims actually were (were they seeking confirmation of miasmatic theory?); but they introduced their findings with the statement 'From the beginning of our work with thermography, it struck us that there were three highly characteristic response patterns shown by chronic patients, three extremes of pathological reaction'. Then later, they stated that Hahnemann's and Ortega's doctrine of miasms *finally* offered an explanation that helped them

to understand the chronic conditions presented by their patients. The regulating change of skin temperature before and after stress (exposure of body at room temperature) should be no less than 0.5°C and no more than 1.0 in the periphery. The first response pattern showed little or no response, with temperatures of less than 0.5; the second response was excessive, with decreases of more than 1. The third response had no general trend as in the other two. In this case, for the 60 readings of the thermogram, excessively high and low temperatures occurred within a few centimetres of each other, and made diagnosis impossible.

These three thermal reactions correspond to the three types of pathological reaction shown by Hahnemann's patients who did not respond to his medicinal stimuli in the manner expected. He used the term 'miasm' (Psoric, Sycotic, and Syphilitic); the greek word *miasma* means 'pollution'. However, the connections which *appeared* to exist had to be clearly shown to correspond to the thermogram. This the authors did by looking for the most extreme examples and comparing them with the relevant patients' case notes (taken at earlier dates, when miasmatic aspects were not even considered). Two pages give examples of the three groups. They showed a close agreement for which the authors 'had hardly dared hope'. A further two pages show examples of thermograms. The authors comment that therapeutic measures will be followed by changes in regulatory capacity. This would seem to give much scope for future research, but the authors make little comment on this. In conclusion they state 'Contact thermograms therefore permit us to assess the patient's reactive potential at any given stage and consequently the miasmatic taint. They provide this information at a very early stage, often years before the patient becomes aware of the subjective symptoms'.³

Hahnemann and nutrition

'Nutrition—its relationship to homoeopathic practice and Hahnemannian theory' is the title of this paper by Alan Stewart. Hahnemann wrote his *Organon* at a time when little was known of nutrition; 'yet Hahnemann makes a significant number of references to the use of diet as part of the broader homoeopathic approach'. Throughout the volume... he makes several direct references to the role of diet and food (10 or so out of 294 sub-sections in the fifth edition).

The concept of prevention occurs early in Hahnemann's text 'He (the physician) is likewise a preserver of health if he knows the things that derange health and cause disease, and how to remove them from persons in health' (sub-section 4). The dietary exhortations made in the NACNE report are a modern version of this. He was aware of the concept of predisposition. In reference to 'morbific noxious agents', he stated 'we are made ill by them only when our organism is sufficiently disposed and susceptible...they do not

produce disease in everyone nor at all times' (sub-section 31). A good modern example is carcinoma of lung; and this may be more common where there are high serum levels of vitamin A.

The author then refers to the theory of miasms. Hahnemann writes that 'all chronic affections' have three possible causes; 'persistent unhealthy mode of living'; 'pernicious treatment of diseases' (iatrogenic conditions); and the 'chronic miasms', which 'are inevitably destined by mighty Nature sooner or later to burst forth' (paragraph 204). Stewart suggested that we can now appreciate that he was possibly referring to genetic disease, which is subject to change by such factors as radiation, drugs and nutrition. He referred to two recent elegant experiments by Hurley and others, demonstrating that experimental zinc deficiency in mice could have an effect for several generations. The author thought that perhaps this, as well as the more obvious genetic diseases, is what Hahnemann was referring to when he spoke of miasms. In the 1920s, there was further support for the theory from Garrod's concept of inborn predisposition to metabolic disorders, which were likely to be clinically silent until the individual was placed under an appropriate stress.

In paragraph 73, Hahnemann writes about acute disease thus; 'Excesses in foods, or an insufficient supply of it... are exciting causes of such febrile affections; in reality, however, they are generally only a transient explosion of latent psora'. In 1950, Willams put forward the concept of genetotropic disease. This was defined as 'one in which the genetic pattern of the afflicted individual called for an augmented supply of a particular nutrient (or nutrients) for which there develops as a result a nutritional deficiency'. Examples of this condition are vitamin B6 dependence syndromes; variations in speed of metabolism of alcohol; vitamin B1 deficiency or Wernicke-Korsakoff's syndrome. Recent evidence suggests a predisposition to this, probably because of genetic variations in the structure and functions of the thiamine-dependent enzymes. At the same time there are several nutritional examples which present the homeopathic principle of similars: deficiency or excess of vitamin A both result in dryness of the skin; deficiency and excess of vitamin B6 results in nerve damage, etc.

Hahnemann placed great importance on dietary factors, particularly in the early stages of disease. 'If a

patient complains of one or more trivial symptoms that have only been observed a short time previously A slight alteration in the diet and regimen will usually suffice to dispel such an indisposition' (paragraph 150). Stewart states 'Here is the medicine of minimal intervention and prevention, one that is only borne out of experience. It is noteworthy that Hahnemann refers specifically to the areas of 'diet' and 'regimen'. Perhaps these are the two most important cost-effective treatment modalities in medicine today, just as they were 150 or more years ago'.

In dealing with the problem of dietary factors that may interfere with effectiveness of a medicine, Hahnemann writes '... during the treatment everything must be removed from the diet and regimen which can have any medicinal action ...' (sub-section 259). Modern examples are the effect of calcium-rich foods on tetracycline absorption and the effect of vitamin B6 reducing the effectiveness of L-Dopa. He stressed the importance of looking for dietary and other obstacles to cure 'Hence the careful investigation into such obstacles to cure is so much the more necessary in the case of patients effected by chronic diseases, as their diseases are usually aggravated by such noxious influences and other disease-causing errors in the diet and regimen, which often pass unnoticed' (sub-section 260). Modern examples which fit this description are: sub-clinical nutritional deficiencies, as often found in the elderly; environmental toxins such as lead, affecting childrens' performance; food allergy, etc.

The author concludes 'If Hahnemann were alive today, he undoubtedly would be a holist, and I think, from this interpretation of the *Organon*, he would be a nutritionist and biochemist, as well as having a number of complementary therapies at his finger-tips'.⁴

References

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