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CLINICAL MEETING, 10th FEBRUARY, 1983

A Clinical Meeting of members of the Association was held at the Rand Mutual Hospital, Johannesburg, on Thursday, 10th February, 1983, at 14h00, with Dr. O. Martiny in the Chair.

Adventures with Nitrous Oxide

by Dr. Mark Gillman

The title of this lecture, "Adventures with Nitrous Oxide", may appear flippant, but this work has been an adventure for me and will, I hope, be so for you after today.

What prompted my interest in N_2O? As a dentist, I use N_2O in analgesic doses and noticed that some patients became euphoric, lost their anxiety and enjoyed a measure of analgesia following this procedure. I enrolled as a post-graduate student in Pharmacology at Potchefstroom in order further to investigate these effects. Here we carried out experiments based on Berkowitz's hypothesis that naloxene reversed the analgesic effect of N_2O.

Painful stimuli were applied to patients given N_2O and naloxene and to our surprise the analgesic effect was reversed. This indicates to us that there must be two systems, the first affected by N_2O and the second by naloxene. Having decided that one system suppressed pain and the other increased pain, we believe that there must be a line between pain and pleasure.

We then consulted a neurosurgeon who, following a morphine saturation test, performs operations for the relief of pain by inserting brain implants. The experiment which followed, using morphine followed by narcan, showed an increased response, which is paradoxical and these results have been published.

We then looked at the pleasure response to see whether this could be manipulated with N_2O and naloxene. N_2O was used to stimulate sexual fantasies and the narcan was given in small doses to potentiate orgasm. This in fact happened, and these results have been published in the *American Journal of Sex Research*.

We are at present experimenting on yogic states as well as addiction, and preliminary results, by independent observers, indicate that 100 per cent oxygen relieves the symptoms and signs associated with alcohol withdrawal in addicts. N_2O is also effective, improving mainly the affective symptoms. We postulate that the withdrawal of alcohol results in an O_2 debt as well as overacting on the cholinergic and adrenergic systems.

We now believe that N_2O in analgesic doses stimulates the opiate system and can safely be used to investigate psychiatric illnesses and we have, in fact, been doing this for eighteen months, working particularly with depressive states.

Recently, we have shown that patients given N_2O and narcan developed a flat EEG tracing (technical errors have been excluded) while continuing to breathe normally. We hypothesise that brain death is over-activity of the opiate system, and these results have been published in *The Lancet*.

Finally, nitrous oxide used correctly is extremely safe, and myeloneuropathy following N_2O occurs only in addicts. Reversible megaloblastic anaemia may also occur following long operations, but this is a clinical curiosity.

This lecture has been summarised by the Hon. Editor.

CLINICAL MEETING, 14th APRIL, 1983

A Clinical Meeting of members of the Association was held at the Rand Mutual Hospital, Johannesburg, on Thursday, 14th April, 1983, at 14h00, with Dr. O. Martiny in the Chair. The meeting took the form of a discussion on the theme "Problems Arising from the Re-employment of Treated Silicotics and Tuberculotics", introduced by Drs. F. Wiles and R.L. Cowie.

What is Silicosis?

by Dr. R.L. Cowie

The new regulations[1] concerning silicosis require us to take a more thoughtful look at this disease. In the past, a suspicion of silicosis set us automatically upon a statutory, inflexible course which required neither thought, discretion nor knowledge beyond crude recognition on the part of the Mine Medical Officer.

We are now called upon to use our pro-

fessional judgement in our patient population with silicosis. With this new dispensation it behoves us to know about silicosis, its likely effect upon our patient and the risks of further exposure to silica-containing dust, and with that knowledge to take the best decision for each affected employee in our care.

WHAT IS SILICOSIS?

Silicosis as we see the disease in the gold-mining industry in South Africa is synonymous with *chronic silicosis* which is also referred to as *simple silicosis*. This is usually recognised by a typical chest radiograph with predominantly upper zone nodularity which is usually of gradual onset and slowly progressive with increasing profusion and size of nodule. The nodules do not exceed 10 mm in diameter. Hilar nodes are often prominent and occasionally show the typical egg-shell calcification. Such a chest radiograph can be considered to represent chronic silicosis if the man has been exposed to silica-containing dust for a period exceeding 15 years.

The term "silicosis" also encompasses other forms of the disease which are not usually seen in our employees but which need to be distinguished from chronic silicosis for different management. In *accelerated silicosis* a similar nodularity is often less obviously of upper zone predominance and is readily distinguished by the history of too short an exposure to silica-containing dust for the definition of chronic silicosis. The history is usually of exposure for less than 10 years and may be as little as 3 years.

An even more rapidly developing and lethal form of silicosis which, for practical purposes, does not occur in the gold-mining industry, is known as *acute silicosis* or *silicoproteinosis*. In this disease, which results from massive exposure over periods of between 6 months to 2 years, nodule formation is not apparent and symptoms of dyspnoea, wasting and weakness are striking, with death following within a matter of months rather than years.

It must be clear, therefore, that the disease to which this paper refers is chronic or simple silicosis. It occurs commonly in our South African gold-miners and is characterised by a long history of dust exposure (not less than 15 and usually more than 20 years), and by a readily discernible nodular opacification of the lungs which is seen to develop and progress over years.

NATURAL HISTORY OF CHRONIC SILICOSIS

It is remarkable but true that the natural history of chronic silicosis is not really known. The disease in miners has been known for centuries, with literature dating back to 1556 and first descriptions from South Africa in 1902. It is probable that the lack of data results from failure to separate chronic silicosis from the accelerated and acute forms of the disease. The much higher incidence of complicating tuberculosis which was untreatable until the last 30 years has also made a separate outcome for chronic silicosis difficult to identify.

However, through the uncertainty, several authoritative reports[2, 3] reflect the view which is steadily gaining support that chronic silicosis is a disease which results in little or no pulmonary disability. Having said this, most authorities hasten to add that additional work needs to be done to substantiate this benign view of the disease and at least two studies are under way in our industry.

For the present, we depend on random samplings which suggest that silicosis, as we see it, is seldom complicated by lung dysfunction, that there is a steady progression of the disease after diagnosis and that continued exposure to silica-containing dust has little effect upon the rate of progression.

COMPLICATIONS

All the complications of silicosis are far more common in the accelerated and acute forms than in the chronic disease.

Pulmonary tuberculosis remains the commonest and probably the only relevant complication in our patient population. The excess of pulmonary tuberculosis which can be attributed to silicosis is not known but is steadily falling.[3]

A recent study[4] presents evidence that the complication of tuberculosis in chronic silicosis can be successfully treated using a 100-dose, four-drug regimen[5] similar to that which is currently standard in our industry.

Prompt diagnosis and treatment of tuberculosis in men with silicosis should prevent any

adverse effect of this complication on the natural progression of their silicosis. This view is, however, implicitly rejected by the official interpretation of the new regulations[1] and cannot be tested for lack of data. Nevertheless, there is no reason to believe that the natural history of tuberculosis, the success of its treatment nor the relapse rate after treatment are in any way affected by continued exposure to silica-containing dust. It would seem, therefore, that a mineworker with pulmonary tuberculosis whose general clinical state is satisfactory, should be allowed to resume his previous occupation irrespective of his stage of treatment, his sputum bacteriology or the presence or absence of underlying chronic silicosis. It is to be hoped that the official view will eventually make such practice permissible.

CRITERIA FOR EMPLOYMENT OF CHRONIC SILICOTICS

The fear of pulmonary disability has motivated previous restrictions for the re-employment of employees with silicosis. For this reason, a measurement of lung function must be central to any decision concerning a man with chronic silicosis. As it is known that silica-dust exposure leads to lung dysfunction of an obstructive type[6] quite independently from silicosis, the vital capacity which is the simplest of the tests for a restrictive lung process such as silicosis, has been chosen to assess the employee with silicosis. To allow for the normal variation about the predicted value for vital capacity, a cut-off level of 75 per cent of the predicted vital capacity is recommended: those whose vital capacities are 75 per cent or less than predicted should not be allowed further exposure to silica-containing dust. It should be emphasised that chronic silicosis will only exceptionally cause a reduction of vital capacity and that such a finding should raise suspicions that the diagnosis of chronic silicosis has been wrongly applied. This leads on to the next criterion for re-employment of an employee in a "risk" occupation; we must be certain that the disease which we have detected through our regular chest radiographs really is chronic silicosis. Doubt must arise if the pulmonary opacification has developed rapidly, if the duration of service is less than 15 years, if the distribution of the nodules does not emphasise the upper zones and, as noted above, if there is a reduction of the vital capacity. Doubt on any one of these counts should result in a more complete investigation with a transbronchial lung biopsy often proving to be the most definitive as well as the quickest and easiest of the available investigations. In my experience, doubt about the diagnosis of chronic silicosis has resulted in diagnoses of sarcoidosis, of disseminated tuberculosis and of accelerated silicosis.

Further criteria for re-employment of silicotic employees in "risk" work relate to their length of previous dust exposure which, in turn, reflects the chronicity of the process, as does their age, with the older man likely to have had longer exposure. Age is also relevant as the older man will have less future exposure, thus the decision to allow a 55-year-old to return to, at most, another 5 years of dust exposure is far less critical than a similar decision in a 40-year-old with a possible 20 years of further exposure. In other words, the vital capacity and duration of exposure criteria will be less strictly enforced in an older subject than they would in a younger man. In general a history of less than 15 years of dust exposure in association with chronic silicosis implies a more aggressive process and would contraindicate further exposure.

Lastly, it is an essential requirement that the silicotic employee be allowed to choose further employment in a dusty environment and that he does so with the knowledge that there are dust-related lesions in his lung. This criterion, with a confident diagnosis of chronic silicosis, and a normal vital capacity, must be seen as the definitive criteria. Age and duration of exposure are the negotiable criteria for allowing a man with silicosis to continue "risk" work (Table I).

TABLE I

CRITERIA FOR CONTINUED EXPOSURE TO SILICA-CONTAINING DUST IN A MINEWORKER WITH SILICOSIS

1. Has vital capacity >75 per cent predicted
2. Has chronic (simple) silicosis
3. Is 40 or more years old
4. Has 15 or more years' exposure
5. Makes an informed request for continued exposure

CONCLUSION

With the implementation of the new regulations which permit the continued employment of men with silicosis in "risk" work, exposed to silica-containing dust, it has become necessary for the Mine Medical Officer to redevelop clinical skills which the previous legislation rendered atrophic and rudimentary. The true story of chronic silicosis has yet to emerge, but it is very different from that which prevails in mining mythology and in many of the standard medical texts. In this short paper I have attempted to show that chronic silicosis is a mild disease associated with little or no pulmonary disability, and that it is no longer reasonable to cripple those who develop it by excluding them from their only means of earning a living.

I have suggested criteria which may be used to determine which silicotic miners may safely continue with "risk" work.

The prevalence of accelerated silicosis is very low but, as is the case with other lung diseases such as sarcoidosis and disseminated tuberculosis, it may mimic chronic silicosis radiologically. An alternative diagnosis should always be sought if any feature seems to be inconsistent with chronic silicosis. It is my practice to include a vital capacity of 75 per cent or less of the predicted capacity, as an indication for further investigation, which usually includes a transbronchial lung biopsy.

Pulmonary tuberculosis is the only complication of chronic silicosis with which we need to concern ourselves in the South African gold-mining industry. My view of pulmonary tuberculosis with silicosis differs from the official view. I believe that with proper surveillance the disease can be diagnosed before it has destroyed lung and before it has accelerated the silicosis. With a complete course of modern, short-course, rifampicin-containing therapy, I believe that the chronic silicotic can again be judged as such and not as a silico-tuberculotic.

REFERENCES

1. Occupational Diseases in Mines and Works Act, 1973. *Government Gazette*. Amendment of Regulations, 1982. No. 8482 pp. 15-16

2. Morgan, W.K.C., and A. Seaton. Occupational Lung Diseases. (W.B. Saunders Co., Philadelphia. 1975. p.80)

3. Ziskind, M., R.N. Jones and H. Weill. Silicosis. *Am. Rev. Respir. Dis.* 1976. **113**, 643-666

4. Escreet, B.C., M.E. Langton and R.L. Cowie. Short-course Chemotherapy for Silico-tuberculosis. *Unpublished Report.* 1982

5. Escreet, B.C. and R.L. Cowie. Short-course Chemotherapy for Pulmonary Tuberculosis. *S. Afr. Med. J.* 1981. **60**, 951-3

6. Wiles, F.J. and M.H. Faure. Chronic Obstructive Lung Disease in Gold Miners. In: Inhaled Particles IV. (Pergamon, Oxford, 1977. pp. 727-734)

CLINICAL MEETING, 14th JULY, 1983

A Clinical Meeting of members of the Association was held at the Stilfontein Golf Club on Thursday, 14th July, 1983, at 10h00, with Dr. D.J. Heydenrych, President, in the Chair.

Epidemiology of Cardiovascular Disease in S.A. with Emphasis on the Treatment of Chest Pains with Sudden Onset

Dr. H.S. Viljoen

South African White males have for some time run a close second to their counterparts in Northern America in deaths due to Coronary Artery Disease (CAD). The Federal Government embarked, a decade and a half ago, on a vigorous programme to arouse the consciousness of the nation in combating CAD : known causative factors such as overweight, high blood cholesterol, smoking, lack of exercise and a high-flying, competitive, stress-inducing environment, were more clearly defined. There is now enough evidence to show that the reduction or elimination of some of these causative factors results in a significant drop in deaths due to CAD and to a remarkable improvement in mental health, thus saving the country in the region of 470 billion dollars