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Suicide and Euthanasia

SIR,—Dr. S. L. Henderson Smith in his letter (10 April, p. 111) commented on the B.M.A. report¹ and made the suggestion that the question of voluntary euthanasia would be considerably eased if the Suicide Act (1961) were amended to allow a doctor to help a patient, if he so desired, to take his own life. I am sorry that Dr. Henderson Smith did not amplify this suggestion when he and other doctors who support voluntary euthanasia gave evidence before the panel who were drawing up the B.M.A. report. As chairman I think I did not give as much attention to this suggestion as the matter deserves.

While it is true that in England and Wales there are penalties up to 14 years imprisonment for anyone who abets or counsels suicide, nevertheless while suicide and attempted suicide were once a crime in Scotland neither is recognized as such today. Since there is no principal crime of suicide in Scotland, presumably there is no art and part guilt for the individual facilitating the other's suicide.² In Scotland there appears to exist a liberty to facilitate suicide which is broader in scope, since it includes the laity as well as the medical profession, than that proposed by Dr. Henderson Smith for England and Wales.

What evidence is there that doctors, friends, and relatives avail themselves in Scotland of their liberty to assist suicide? There have been several communications concerning suicide in Scotland in recent years, but I have traced no reference to assisted suicides, by either doctors or friends. Has anyone information on this point? I am endeavouring to write a book about vol-

untary euthanasia and information on this point would be received gratefully.

There is one practical point if doctors or friends assisted suicide. Someone would have to watch by the comatose body until death lest another caller rushed the person off to hospital where modern methods of resuscitation save almost everyone who arrives in time. I have always felt that anyone desiring euthanasia should discuss matters with the nearest of kin, possibly a few friends, and obtain if possible their agreement. If this does not occur they will be deeply hurt by the unexpected death. This practical point might ensure this agreement.

Dr. Henderson Smith advocated that a doctor should be allowed to help his patient yield up his life if he so desires. Does this mean that a doctor could assist suicide only if there was a painful incurable physical disease or would it also allow him to assist a person considered to be sane who had a determined wish to take his own life because of mental distress, say from domestic unhappiness? The latter proposal was apparently actually made recently by the head of the Social Welfare Board of Denmark.³ I find this suggestion alarming to say the least of it, but I do not know if it would be supported by any doctors who are members of the Voluntary Euthanasia Society.—I am, etc.,

HUGH TROWELL

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¹ Special Panel of the Board of Science and Education of the British Medical Association, *The Problem of Euthanasia*. London, British Medical Association, 1971, and leading article, *B.M.J.*, 23 January, p. 187.

² D. W. Meyers, *The Human Body and the Law*. Edinburgh, University Press, 1970.

³ *Daily Telegraph*, 20 January 1971.

Pathogenesis of Myasthenia Gravis

SIR,—The wider concept of myasthenia gravis (leading article, 3 April, p. 1) may be usefully extended. Goldstein and his associates¹ produced experimental thymitis by using Freund's complete adjuvant, and spontaneous myasthenia in man may well arise in a similar way. The associated disorders (immune thyroiditis, Addison's disease, and other endocrinopathies) are occasionally complications of sarcoidosis.²

Both Freund's adjuvant (killed mycobacteria) and the sarcoid agent³ can induce local defensive antibodies. The latter eventually may also be shown to contain mycobacterial debris. It is therefore likely that myasthenia with, for instance, Hashimoto's thyroiditis sometimes results from sarcoid inflammation. Usually sarcoidosis heals in a year or two, leaving at most some lymphocytes and perhaps a few giant cells in fibrotic scars.

Rarely, these secondary changes progress, and immune disease will then become evident. The syndrome of giant-cell myocarditis with myasthenia (of which less than 30 cases are reported) may be an example. Not only does the histology of this condition include Langhans giant cells, asteroid bodies and lymphorrhages in the heart,⁴ but also chronic lymphadenitis and giant-cell granulomas in the lungs and liver.⁵ Mononuclear and giant cells are also found in the two organs in Addison's disease with hypoparathyroidism.⁶

A case showing chronic inflammation and giant-cells in the heart, with interstitial fibrosis of the lungs, had in life evidence of renal tubular acidosis and thrombocytopenia.⁷ The illness resembled "hyperglobulinaemic

fibrosing alveolitis"⁸ with the addition of giant cells. In immune disease stimulation and later failure of affected endocrines may be a by-product of the antibody response.

Preliminary studies by me of three thyroid patients showed reversal of hyperthyroidism on 15 mg daily of prednisone. The first case, with sarcoid hyperthyroidism, remains euthyroid three months after prednisone was withdrawn. Massive doses of steroids are not always necessary to "block" the thyroid overaction. Hyperthyroidism complicated by temporary myasthenia is rare, but early investigation is important because it may be caused by sarcoidosis. Steroid treatment of myasthenia gravis may be more effective if started sooner.—I am, etc.,

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- ¹ Goldstein, G., *Lancet*, 1962, 2, 119.
- ² Karlish, A. J., and MacGregor, G. A., *Lancet*, 1970, 2, 330.
- ³ Mitchell, D. N., and Rees, R. J. W., *Lancet*, 1969, 2, 81.
- ⁴ Palmer, H. P., and Michael, I. E., *Archives of Internal Medicine*, 1965, 116, 444.
- ⁵ Burke, J. S., Medline, N. M., and Katz, A., *Archives of Pathology*, 1969, 88, 359.
- ⁶ MacGregor, G. A., *Lancet*, 1970, 2, 1257.
- ⁷ Miller, W. V., Senhauser, D. A., and Martt, J. M., *Missouri Medicine*, 1966, 63, 811.
- ⁸ Mason, A. M. S., McIlmurray, M. B., Golding, P. L., and Hughes, D. T. D., *British Medical Journal*, 1970, 4, 596.

SIR,—As your unsigned leading articles tend to acquire the authority of holy writ I hope you will permit me to comment on your first leading article (3 April, p. 1) to correct some misunderstandings which have appeared elsewhere. You kindly refer to my paper¹ giving the first fully formulated autoimmune hypothesis but infer that this was confirmation of earlier work by demonstrating clinical relationships with other autoimmune diseases. In 1960 this was hardly possible (and Miller's paper on the thymus was still a year off). It showed that myasthenia gravis was probably a multisystem disease with some resemblances to the natural history of systemic lupus erythematosus. An autoimmune hypothesis was presented to account for this. In the next decade many of the associated conditions have subsequently been recognized as autoimmune.²

Reference to the original paper will show that it reported the conclusions of five years of clinical and experimental work, freely discussed in teaching and research seminars. The passing reference of Smithers,³ a few months before final publication, to the possible implication of autoimmunity in the thymic changes of myasthenia gravis was referred to, but that paper offered no concept of the nature of the neuromuscular disorder.

It is not possible to decide whether Smithers considered that the thymic pathology was the cause or the result of autoimmune disease. This is a critical point in current thinking about myasthenia. It is unfortunate that your leading article ignored the valuable symposium at the New York Academy of Sciences in December 1970. It quotes the only group of workers supporting Goldstein's experimental work. To the longer list of negative findings⁴ will be added a paper (in press) from an Australian group which includes Goldstein's original collaborator. My original hypothesis was that a breakdown of immunological tolerance resulted from a thymic disorder (genetic or

acquired). The Goldstein hypothesis considers that the thymus is damaged immunologically without indicating where the primary immunological disorder may occur and the theory requires a neuromuscular blocking substance, which is against all the evidence.⁵ The theory lacks the heuristic value of the original hypothesis, for which no incompatible evidence has yet been produced, as it does not predict involvement of other tissues—the very reason for conceiving an autoimmune theory.—I am, etc.,

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- ¹ Simpson, J. A., *Scottish Medical Journal*, 1960, 5, 419.
- ² Simpson, J. A., In *Muscle Diseases*, Ed. J. N. Walton, N. Canal, and G. Scarlato, Amsterdam, Excerpta Medica, 1970, p. 14.
- ³ Smithers, D. W., *Journal of the Faculty of Radiologists*, 1959, 10, 3.
- ⁴ Vettors, J. M., Simpson, J. A., and Folkard, A., *Lancet*, 1969, 2, 28.
- ⁵ Simpson, J. A., In *The Biological Basis of Medicine*, Vol. 3, Ed. E. E. Bittar, and N. Bittar, New York, Academic Press, 1969, p. 345.

Carcinoma in Pleomorphic Adenomas

SIR,—Pleomorphic adenomas (mixed tumours) sometimes undergo carcinomatous change; in a recent study¹ roughly half of the more malignant types of carcinoma had arisen in this way. The only known predisposing factor is a long history, the average time before the development of the carcinoma in the above series being 19 years. The risk in the individual case is impossible to determine from these figures, since the size of the population of pleomorphic adenomas from which the carcinomas developed is unknown. Nor, so far as I am aware, are there any other pertinent figures. Under these circumstances the following experience may be worthy of reporting.

I have operated on altogether eight cases of the rare dumb-bell pleomorphic adenoma of the parotid, which because of its origin deep in the gland remains latent until it attains a large size, and therefore presumably for many years. In two of these eight cases carcinoma, from which the patients subsequently died, had developed. The figures are obviously too small to serve as anything more than the roughest index of the risk of carcinomatous change in a pleomorphic adenoma left indefinitely. They may however indicate the way in which this question might be solved.—I am, etc.,

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- ¹ Patey, D. H., Thackray, A. C., and Keeling, D. H., *British Journal of Cancer*, 1965, 19, 712.

Air Filtration for Asthma

SIR,—Some preliminary results of an investigation into the prevention and treatment of asthma are reported. This treatment consists of the removal from the inhaled air of pollens and mould spores to which the individual is sensitive by the simple process of filtration.

The sizes of individual fungal spores are generally in the range of 2-20 μ m, although some are much larger, and pollen grains usually range from 10-50 μ m. Air-conditioning has been tried in the past to treat asthma, but has usually been ineffective because the degree of filtration achieved has been inadequate. However, an effective,

simple air filtration unit has now been made in Adelaide (Pureair filter). An efficacy of 99% or greater in excluding particles of 2 μ m and above is claimed for this filter, and the unit is easily installed in a bedroom window. This means that while in the bedroom the patient can breathe air from which nearly all the offending allergens are removed.

This filter was originally used in 1967 by a man for his 7-year-old son, who was a chronic invalid from asthma. An improvement in the child was noticed immediately and within six months he was able to lead a normal life. Following this more filtration units were made which were then advertised commercially. Favourable reports of the use of this filter for asthma prompted me to send a questionnaire to all the 75 individuals who had bought the filter for asthma, and used it for longer than six months. Fifty-four replies—from 29 men and 25 women—were received. The majority considered that their asthma was markedly improved by using the filter, and that they were now able to lead a normal life after many years of ill-health. The patients had had chronic severe asthma for periods from three to 41 years (mean 14.5 years).

After using the filter for periods from six months to three years three patients considered that they were cured, 36 were markedly improved, 10 were slightly improved, four were unchanged, none was worse, and one did not know whether the filter had helped or not. Twelve had stopped all medication, 21 had reduced their intake of drugs, 15 were on the same therapy, and six were taking more drugs than before. Twenty-two had noticed immediate improvement in their asthma when the air filter was installed, but in others the improvement was not obvious until a varying period of anything up to six months had elapsed.

The answers to this questionnaire strongly suggest that the Pureair filter had produced considerable relief from asthma in the majority of individuals who have used it. These observations are being extended by a double-blind controlled trial of air filtration which is now in progress. It is interesting that although the improvement is often noticed immediately the air filter is installed, there is sometimes a delay before its beneficial effect is evident. This may be due to the chronically over-reactive bronchial mucosa still responding to the small amount of antigen which is not removed by filtration.

It is also interesting that although the most relief from asthma by filtration is noticed by individuals while they are in the filtered room, once improvement has started many individuals have noticed that their asthma becomes progressively less severe when away from the filtered environment. Perhaps the continuing exposure to a very much reduced dose of the relevant antigens enables the patients to develop tolerance to these antigens by the same mechanisms which apply in normal individuals.—I am, etc.,

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Serum Enzymes and Propranolol

SIR,—Most studies on the use of propranolol report no effect on the levels of transaminase or alkaline phosphatase.¹⁻⁴ An