is a prerequisite for setting priorities. Without such data it may be impossible to persuade governments, whose attention may be diverted by other demands, to direct limited resources to blindness. In some poor countries trained ophthalmologists cannot be integrated into the national health service because of lack of money—both India and Senegal have ophthalmologists working as taxi drivers and waiters.

Perhaps the greatest advance in tropical ophthalmology was the introduction of ivermectin (donated by Merck Sharpe and Dohme) to treat onchocerciasis. One dose of ivermectin a year reduces transmission by three quarters. As it kills only microfilaria, however, it must be taken for at least 10-15 years, which is the lifespan of the adult female in the human body. Research is currently focusing on the logistics of the massive distribution programme required.

The tragedy of childhood blindness may be easily overlooked. Vitamin A deficiency accounts for 70% of cases, and as this is related to concomitant measles and malnutrition more than half of those affected die within a year. Distributing high dose vitamin A capsules (donated by Hoffmann-La Roche, Switzerland, under its task force "Sight and Life") is part of the solution. (So is improved sanitation, female literacy, vaccination against childhood diseases, improved diet, education, and eliminating poverty.)

Another disease linked to ignorance and poverty is trachoma, a leading cause of blindness in several African countries. The single most effective means of control is regular face washing. Providing clean water to all communities and use of tetracycline eye ointment by those who are infected might eradicate this scourge.

With national wealth dwindling in developing countries the extensive skill and experience of international agencies working to prevent blindness makes them valuable allies. Among the larger agencies, Christoffel Blindenmission works through mission hospitals, the Royal Commonwealth Society for the Blind (Sight Savers) prefers to work through host governments, Helen Keller International has particular skills in technical cooperation and planning the distribution of vitamin A, and the International Eye Foundation emphasises primary eye care. Each year they have millions of dollars to spend.

Surprisingly, given the burden of blindness and visual handicap, many existing facilities and staff are underused. Several factors account for this. Blindness tends to affect older people who either consider it a normal part of being old or cannot persuade their children to use their limited money to travel long distances for treatment. They may be left in the villages, where their blindness reduces their life expectancy. Many do not know that inexpensive surgery is available, something that health educators must remedy.

Several challenges therefore face the ophthalmological community. National governments need to be made aware of the burden of blindness in their countries. Access to eye care should be made more widely available, especially to rural communities. Programmes of primary eye care, closely related to vaccination, health education programmes, and improved availability of water and sanitation, should be strengthened. Better use of existing facilities and trained staff and closer cooperation among non-governmental organisations and government health services are also necessary.

Unless things change the number of people who are blind will double by the year 2025. As Primo Levi wrote, "Once we know how to reduce torment, and do not do it, we become the tormentors."

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Helen Keller International, 15 West 16th Street, New York, New York 10011, United States.

International Eye Foundation, 7801 Norfolk Avenue, Bethesda, Maryland 20814, United States.

Operation Eyesight International, PO Box 123, Station M, Calgary, Alberta T1P 2H6, Canada.

Royal Commonwealth Society for the Blind (Sight Savers), Commonwealth House, Haywards Heath, West Sussex RH16 3AZ, England.

Thyroid dysfunction and affective illness

Check the hypothalamic-pituitary-thyroid axis in patients resistant to treatment

Mental disorder resulting from gross thyroid dysfunction has long been recognised. Delirium in hyperthyroidism and coma in myxoedema mark out the two extremes with less severe thyroid dysfunction being associated with proportionately less severe symptoms and signs.[1,2] With recent refinements of endocrinological and biochemical techniques has come the concept of subclinical thyroid dysfunction (exaggerated release of thyroid stimulating hormone in response to thyroid releasing hormone in the presence of normal or raised concentrations of circulating thyroid stimulating hormone and normal concentrations of circulating thyroxine and triiodothyronine[3]). This raises two linked questions for psychiatry. Does subclinical dysfunction of the thyroid play any part in affective illnesses, and if so do thyroid hormones have a place in their treatment?

Overt hypothyroidism is much more common in patients suffering from bipolar (manic-depressive) affective illness treated with lithium carbonate than in unselected psychiatric inpatients.[4-9] Even higher rates of overt hypothyroidism have been reported in a subgroup of manic-depressive patients who have the "rapid cycling" form of the illness.[10-12] (These patients have at least four episodes of illness a year, which is refractory to treatment.[11-16] Antidepressants may make them worse.)

Subclinical forms of hypothyroidism are also common in bipolar affective illness[4,7-11]; more so in those with the rapid cycling form of the disease.[11,12] Some of these findings may be explained by most of these patients being treated with lithium, which has antithyroid effects.[4,8,13] The fact that overt hypothyroidism was found to be more common in those with the rapid cycling disease than in the unselected patients with bipolar disease,[13] however, is unlikely to be due solely to lithium as lithium treatment was a common feature of both groups. Hypothyroidism and rapid cycling are both more

common in women,[10 11 14 15 17] who are more vulnerable to antithyroid agents, including lithium.[12] Rapid cycling may follow the development of hypothyroidism, which in turn may be precipitated by treatment with lithium, in patients with bipolar illness.

Confusingly, some depressed patients seem to have changes in the function of their hypothalamic-pituitary-thyroid axis that are more consistent with hyperthyroidism. Some patients with major (endogenous) depression have a low 24 hour output of thyroid stimulating hormone and a reduced nocturnal rise of thyroid stimulating hormone.[2] A blunted response of thyroid stimulating hormone to thyroid releasing hormone (characteristic of hyperthyroidism[2 18 19]) has been found in about one in four depressed patients, although only in those with unipolar affective disorder. Patients with a bipolar disorder may show an exaggerated response of thyroid stimulating hormone to thyroid releasing hormone, consistent with the high prevalence of subclinical hypothyroidism in this condition. This difference between patients with unipolar and bipolar disease, however, remains controversial.[19]

Many attempts have been made to use hormones of the hypothalamic-pituitary-thyroid axis to treat depression, and all of them have been claimed to work. There have been reports that thyroid releasing hormone has an acute, short lived, mood raising effect, and clinical improvements have followed injections of thyroid stimulating hormone given before the start of treatment with imipramine and thyroxine and triiodothyronine given with tricyclic antidepressants.[2 20] More recently, thyroxine given in doses high enough to produce supranormal plasma concentrations of the hormone (100-400 μg/day) has been found to stabilise the mood of patients with the rapid cycling form of bipolar affective illness.[16 21 22] This was found not only in patients with overt or subclinical hypothyroidism but also in patients with apparently normal hypothalamic-pituitary-thyroid axes. This has led to the hypothesis that those with rapid cycling disease suffer from a localised deficiency of thyroid hormone in their brain that may be corrected by high doses of thyroxine. Drugs such as lithium and tricyclic antidepressants, which interfere with the use of thyroid hormones by the brain, may initiate or aggravate this deficiency.[12 22]

What are the clinical implications of these findings? Firstly, in every patient suffering from a form of chronic or recurrent affective illness that is resistant to treatment, the function of the hypothalamic-pituitary-thyroid axis should be thoroughly investigated and any abnormality should be corrected. Secondly, in the incapacitating disorder of rapid cycling bipolar affective illness, if it proves to be refractory to other therapeutic measures (for example, stopping any antidepressant or a trial of carbamazepine), cautiously administering a small dose (100-400 μg/day) of thyroxine, even in the absence of endocrinological evidence of thyroid dysfunction may be worth while. This should be done with proper clinical and laboratory monitoring. It should be emphasised, however, that the evidence for the clinical effectiveness of thyroid hormones in this condition is based on few patients and the theoretical underpinning of this regimen is still tenuous.

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