they are now designated young disabled units and serve the needs of those who are grossly handicapped, they are often unable to grapple with the problems of the aggressive and the apathetic at the same time and in the same place. The needs of these various groups are being brought to light, and many would agree that most of these needs should be satisfied "in the community" with such persons adequately supported in their own homes wherever possible.

Thus many disparate groups have been identified, and all districts are faced with the need for planning resources to meet them in the most effective way. To provide for such a range of requirements requires a widespread awareness of patients' needs in many medical spheres. Since most handicapped people should remain for most of their lives at home, much attention must be given to training of general practitioners. Yet a recent survey suggests that, perhaps because of the pressure of their work, few general practitioners are aware of the usefulness of their local aids centre.[23] Wright and colleagues showed that few undergraduates and just as few postgraduates had any training in rehabilitation.[24] Yet—as Gloag states—primary carers must be made aware of this need. Questions on rehabilitation should feature in medical students' final examinations, and relevant rehabilitation training must become mandatory in vocational training for general practice.

Whatever solution is found for the most effective organisation of district and regional resources change will occur only in those districts with at least one doctor committed to ensuring the best use of therapists and therapies, and colleagues who understand and support him. Every district should, therefore, have one medical coordinator of services. Traditionally rheumatologists have performed this function well, but there is every reason to hope that neurologists, general physicians, and others will increasingly enter the discipline. In addition, several other consultants will be needed to practise their own long term care of their own patients, as learnt in their training programmes. For highly complex multiple disabilities multidistrict or regional services will be required (such as is currently found with spinal injuries).

Finally, though many research projects have been undertaken (Gloag's series is a valuable guide to these and other initiatives) many basic questions remain unanswered. We need to know more about recovery from brain damage; there is an urgent practical need to define the best ways of retraining those with memory, attention, and cognitive impairments. Occupational therapy departments are experimenting with the use of microcomputers for patients which promise to have potential for both assessment and training. Further relevant research is urgently required.

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1 Wendell-Holmes O. In: Kottke, Stillwell, Lehmann, eds. *Krusen's handbook of physical medicine and rehabilitation*. 3rd ed. Philadelphia: W B Saunders, 1982:xvi.
2 Gloag D. Introduction and a look at some short term rehabilitation. *Br Med J* 1985;**290**:43-6.
3 Gloag D. Rehabilitation in rheumatic diseases. *Br Med J* 1985;**290**:132-6.
3a Gloag D. Aids and the environment. *Br Med J* 1985;**290**:220-3.
4 Gloag D. Severe disability: 1—Tasks of rehabilitation. *Br Med J* 1985;**290**:301-3.
5 Gloag D. Severe disability: 2—Residential care and living in the community. *Br Med J* 1985;**290**:368-72.
6 Gloag D. Rehabilitation of the elderly: 1—Settings and services. *Br Med J* 1985;**290**:455-7.
7 Gloag D. Rehabilitation of the elderly: 2—Mind and body. *Br Med J* 1985;**290**:542-4.
8 Gloag D. Rehabilitation of patients with cardiac conditions. *Br Med J* 1985;**290**:617-20.
9 Gloag D. Rehabilitation after stroke: 1—What is the potential? *Br Med J* 1985;**290**:699-701.
10 Gloag D. Rehabilitation after stroke: 2—Language and memory training and the requirements of rehabilitation services. *Br Med J* 1985;**290**:768-71.
11 Gloag D. Rehabilitation after head injury—1: Cognitive problems. *Br Med J* 1985;**290**:834-7.
12 Gloag D. Rehabilitation after head injury: 2—Behaviour and emotional problems, long term needs, and the requirements for services. *Br Med J* 1985;**290**:913-6.
13 Gloag D. Rehabilitation in psychiatric conditions: 1—Community and residential care. *Br Med J* 1985;**290**:981-4.
14 Gloag D. Rehabilitation in psychiatric conditions: 2—Psychological approaches and support, the family, and activities. *Br Med J* 1985;**290**:1059-62.
15 Gloag D. Occupational rehabilitation and return to work: 1—General services. *Br Med J* 1985;**290**:1135-8.
16 Gloag D. Occupational rehabilitation and return to work: 2—Psychiatric disability. *Br Med J* 1985;**290**:1201-3.
17 Gloag D. The time is now: education, and organisation of services. *Br Med J* 1985;**290**:1333-6.
18 Almont L. *Antecedents of the National Health Service, socialized medicine in England and Wales: the National Health Service 1948-1961*. 21.
19 Office of Population Censuses and Surveys. *Registrar General's statistical review of England and Wales for the year 1949—supplement on general morbidity, cancer, and mental health*. London: HMSO, 1953.
20 Harris A. *Handicapped and impaired in Great Britain*. London: HMSO, 1971.
21 Kottke, Stillwell, Lehmann, eds. *Krusen's handbook of physical medicine and rehabilitation*. 3rd ed. Philadelphia: W B Saunders, 1982.
22 Scott CJ. Hospitals for the disabled. *Int Rehabil Med* 1984;**6**:166.
23 Stowe JA. *Investigation into the use of the Leeds Aids and Information Centre by GPs*. Leeds: Rheumatism Research Unit, School of Medicine, University of Leeds, 1985.
24 Wright V, Hopkins R, Burton KE. What shall we teach undergraduates? *Br Med J* 1979;i:805-7.

Onchocerciasis now

Onchocerciasis, "river blindness," is caused by *Onchocerca volvulus*, a nematode worm transmitted by a small biting fly of the genus *Simulium*. Victims suffer from a chronic itchy dermatosis and in the most severely affected from blindness. Of the 20-50 million people infected, 95% live in Africa, mainly in the great western African savannas. There is no reservoir of the infection in animals.

When an infected *Simulium* fly bites, infective larvae enter the wound and develop in about a year to become adult worms in the subcutaneous tissues. The worms are motile, slender creatures up to 70 cm long, living either free or imprisoned in fibrous nodules over bony prominences. After mating, the fertilised females produce larvae called microfilariae, which migrate into the skin. When taken up by a *Simulium* fly during feeding the microfilariae develop in the flight muscles before migrating to the mouthparts, when the fly becomes infective. Adult worms may live up to 20 years, the females producing microfilariae throughout their lives. The normal life span of a microfilaria is about one year. The nodules themselves cause only a minor cosmetic blemish, and the serious pathological process caused by the disease is a sequel to the death of microfilariae. When a microfilaria dies it becomes the centre of an inflammatory focus containing eosinophils, lymphocytes, and macrophages. This subsides when the microfilaria has been totally destroyed and its remnants disposed of in the regional lymph nodes. Unidentified toxins destroy elastic tissue in the skin, leading to premature aging called presbydermia. In black skinned people a characteristic depigmentation called "leopard skin" may occur.

Microfilariae in the eyes cause the most serious effects. They include sclerosing keratitis (in which the cornea is invaded by connective tissue), chronic iridocyclitis, and the posterior segment lesions choroidoretinitis and optic atrophy. Secondary effects such as cataract and glaucoma may occur.

In some parts of Africa up to a tenth of the population is blind because of onchocerciasis. This has led to a great effort by the World Health Organisation to control the disease in Africa, but efforts have been greatly hampered by a lack of acceptably non-toxic drugs to kill the parasites and their young. So the World Health Organisation has been compelled to undertake long term vector control by applying insecticides to the rivers in which the larval stages of the

parasite develop. This has been done by helicopters with non-persistent insecticides such as temephos. The strategy assumes that if transmission can be arrested for longer than the life span of the parasites, then when spraying stops and the flies return there will be no reservoir of infection to reinfect them.

Unfortunately, the insecticide programme has not completely arrested transmission, and the target area is bordered by infected people living in untreated forest areas. The forest strain of *Onchocerca* has been assumed to be less pathogenic than the savanna strain, but there are doubts. So the fear is that when spraying stops the disease will return.

Any antiparasitic chemotherapy that could be used for mass treatment would enormously enhance the effectiveness of the control programme by reducing the reservoir of infection immediately, but neither of the drugs in widespread use is suitable for this purpose. Diethylcarbamazine is a piperazine compound with great activity against microfilariae, but treatment with it is often associated with a severe allergic reaction to the death of the baby worms—the Mazzotti reaction—with fever, pruritus, adenitis, iritis, and hypotension. Furthermore, since it has no effect on the adult worms the microfilariae soon return as a result of continued production by the females. A course of intravenous injections of suramin (a complex urea derivative) will kill the adult worms and accelerate the death of microfilariae, but it may cause a fatal generalised disease to which there is no antidote. So there is a desperate need for more effective antiparasitic chemotherapy in onchocerciasis both for individual and for mass treatment. Carefully conducted trials at the Onchocerciasis Chemotherapy Research Centre in Tamale, northern Ghana, have shown that metriphonate, mebendazole, and levamisole do not provide the answer needed,[1,2] and control of the Mazzotti reaction produced by diethylcarbamazine has proved pharmacologically intractable.[3,4]

It has been encouraging, therefore, that the new drug ivermectin can dramatically reduce the numbers of microfilariae in the skin without causing the severe Mazzotti reactions caused by diethylcarbamazine. The initial reports were in lightly infected patients,[5] but more recently the drug has been shown to be effective in patients with heavy infections—and only a single oral dose is needed.[6] Ivermectin is a compound produced when the macrocyclic lactone avermectin is subject to fungal metabolism. It has been recognised as an important veterinary broad spectrum antiparasitic drug since 1980.[7] As has so often been the case, the veterinary surgeons have shown the physicians the way.

The surprising effect of ivermectin on onchocerciasis is its prolonged microfilaricidal activity combined with the low incidence and mild severity of the Mazzotti reaction which follows its administration. It seems to lack effect on the adult worms. Nevertheless, ivermectin seems to provide us with a very useful tool which, when combined with vector control measures, will greatly enhance the prospects of eradication of this horrid disease. Periodic mass treatment alone would probably eliminate serious ocular lesions, and the drug should be welcomed as the first major advance in onchocerciasis for several decades.

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1 Awadzi K, Gilles HM. The chemotherapy of onchocerciasis III. A comparative study of diethylcarbamazine (DEC) and metrifonate. *Ann Trop Med Parasitol* 1980;74:199-210.
2 Rivas-Alcala AR, Greene BM, Taylor HR, *et al.* Chemotherapy of onchocerciasis: a controlled comparison of mebendazole, levamisole, and diethylcarbamazine. *Lancet* 1981;ii:485-90.
3 Awadzi K, Orme ML'E, Breckenridge AM, Gilles HM. The chemotherapy of onchocerciasis VI. The effect of indomethacin and cyproheptadine on the Mazzotti reaction. *Ann Trop Med Parasitol* 1982;76:323-30.
4 Awadzi K, Orme ML'E, Breckenridge AM, Gilles HM. The chemotherapy of onchocerciasis VII. The effect of prednisone on the Mazzotti reaction. *Ann Trop Med Parasitol* 1982;76:331-8.
5 Aziz MSH, Diallo S, Diop IM, *et al.* Efficacy and tolerance of ivermectin in human onchocerciasis. *Lancet* 1982;ii:171-3.
6 Awadzi K, Dadzie KY, Shulz-Key H, Haddock DRW, Gilles HM, Aziz MA. Ivermectin in onchocerciasis. *Lancet* 1984;ii:921.
7 Egerton JR, Birnbaum J, Blair LS, *et al.* 22,23 Dihydroavermectin BI, a new broad spectrum antiparasitic agent. *Br Vet J* 1980;136:88-97.

Hypertension in children

The United States National Heart, Lung, and Blood Institute recommended in 1977 that all chidren over the age of 3 years should have their blood pressure recorded annually.[1] The intention was to encourage the discovery of those with secondary (and potentially reversible) hypertension and possibly also those likely to develop essential hypertension in adult life. In the years since that recommendation was made interest in the early clinical course of essential hypertension has increased in parallel with new clinical and epidemiological data. We may now reasonably re-examine whether children ought to be screened for hypertension, how the condition is to be defined, its relations to future adult hypertension and hence impaired health, and the criteria for treatment—all topics which were discussed at a recent workshop convened by the National Heart, Lung, and Blood Institute.[2]

Blood pressure in children increases progressively with age, the increase being more considerable for systolic pressure, so that any definition of normal and abnormal blood pressure must take age into account. Furthermore, the method of measurement (with respect to such factors as posture and cuff width) should be standardised and identical with that used for the reference population. National Heart, Lung, and Blood Institute charts give 95th percentile levels of seated blood pressure in 3, 6, 9, and 12 year olds of 112/78, 116/79, 125/82, and 135/87 mm Hg, respectively. Since blood pressure at all ages is normally distributed and the ultimate fate of children whose blood pressure is consistently above these levels is unknown "hypertension" defined in this way is arbitrary and may be a misnomer. Indeed, the institute has cautioned against labelling a child as hypertensive, preferring instead the more euphemistic term "high normal" blood pressure.

What, then, is known of the epidemiology of blood pressure in childhood, and what inferences may be made regarding the possible progression of high normal blood pressure to established hypertension? The data come from various studies in the United States (notably those from Muscatine, Ohio,[3] and Bogalusa, Louisiana,[4] the Netherlands,[5] and New Zealand.[6] Blood pressure in children correlates predominantly with indices of body mass and obesity.[3,4,7,8] Other associations are with parental blood pressure, resting heart rate, salt intake, sex, and sexual maturity. "Tracking" of blood pressure—that is, maintenance of rank order within the distribution over time—is present during childhood but its predictive power is low. In the Muscatine study raised blood pressure was found initially in 13% of 6622 schoolchildren but persisted in fewer than 1%, many of whom were obese.[9] Similar findings have been reported from Dallas.[10] In a more recent study only 45% of children remained within the highest