the application of methods of statistical analysis, and it is clear that as a result much more light is likely to be thrown on the movements of populations in the past. In some cases, it is interesting to note, sufficient remains of ancient communities have been found to permit conclusions regarding the sex and age composition, mortality rate, the expectation of life, and the relation of physique to social grade.

In the field of human and primate palaeontology the past fifteen years have been marked by most remarkable discoveries in many parts of the world—in Europe, South Africa, East Africa, and the Far East. In Europe the discovery of well-authenticated skulls of pre-Mousterian date raises anew the problem of the position in human evolution of Neanderthal man; in South Africa the great quantity of Australopithecine material found in limestone deposits near Johannesburg and elsewhere now provides an opportunity for a correct assessment of the affinities of these extinct ape-like creatures; in East Africa the discovery of an astonishing variety of Early Miocene apes of a generalized type (including a skull and a number of limb bones) has added very considerably to our knowledge of the earlier stages of hominoid evolution; and in the Far East the further discovery in Java of many more remains of *Pithecanthropus* and allied forms of early man (much of it still in course of publication) appears to indicate that the evolutionary radiations of primitive hominids showed a much greater diversity than had previously been suspected.

Considering their novelty, it is not surprising, of course, that such remarkable discoveries should have given rise to a certain amount of controversy, but in general this controversy has been concerned with details of interpretation rather than with main issues. I confess to a considerable amount of sympathy for those energetic field workers who, in remote parts of the world, have shown such initiative in their search for the remains of early man and other fossil primates. For, having by their own strenuous efforts made available for scientific study all this valuable material, they sometimes have patiently to look on while laboratory scientists (like myself) dispute among themselves on the precise value of specimens which they themselves may never even have seen. It is rather like throwing bones into a den of lions and watching the inevitable squabbles which ensue—an entertaining spectacle in some cases, no doubt, but also one which may tend after a time to become a little irksome. As a matter of fact, if the history of palaeo-anthropology is reviewed it is astonishing to find how often the original interpretations of obscure fossil remains turn out to be broadly correct. No anthropologist to-day, I think, gives serious consideration to the criticism that *Pithecanthropus* was nothing more than a giant gibbon, or that the remains of Neanderthal man found at Düsseldorf were only those of an idiot or an acromegalic. Even the original interpretation of Piltdown man has now received unexpectedly strong support from the fluorine analysis of the jaw and skull fragments. Special mention should be made of this method of estimating the comparative antiquity of fossil remains found together in the same deposit. It has already been applied with success to some of the fossils of uncertain date which have been the subject of much debate in the past, and it will certainly be applied as a routine test to similar problems as they arise in the future.

Conclusion

This short review of the progress and trends evident in anatomical science to-day, I should like again to emphasize, is no more than a personal impression. It is not, of course, based on a detailed study of all the relevant litera-

ture; it is simply the casual survey of one individual who, preoccupied with his own particular field of studies, gleans scattered pieces of information here and there from discussions with his colleagues or from all-too-hurried glances at the contents of papers and journals. For this reason, also, it has perhaps tended to make too much use of illustrative examples near at hand. You will therefore find many gaps in the review—some of which you may feel to be rather glaring. But at least it may serve to show how very active are the numerous branches of our science and how many problems of paramount importance are now engaging the attention of anatomists. And, even if I have not made this fully apparent, the programme of communications at this international congress in Oxford can hardly fail to do so.

AMOEBIASIS IN GREAT BRITAIN WITH SPECIAL REFERENCE TO CARRIERS*

BY

CECIL A. HOARE, D.Sc., F.R.S.

(From the Wellcome Laboratories of Tropical Medicine,
London)

The recent war has left an aftermath of medical problems arising directly from the fact that large bodies of men returning to Great Britain have been exposed to exotic diseases in tropical and subtropical regions. As a result the medical authorities are concerned about the possibility of the introduction and spread of protozoal diseases among the local inhabitants.

I propose to restrict myself to a consideration of infections due to *Entamoeba histolytica* among the population of Great Britain. The problem of amoebiasis in this country is more complicated than that of other protozoal diseases owing to the cosmopolitan distribution of *E. histolytica*, on account of which infection with this parasite can hardly be described as a merely tropical disease.

The medical practitioner is apt to encounter cases of amoebic infection of different types—some with typical clinical symptoms, others with non-characteristic manifestations, and others again without any symptoms whatever. While in cases of overt amoebiasis there can be no doubt about the necessity for specific treatment, opinions regarding the policy to be adopted towards apparently healthy "cyst-passers" are divided. This is due to conflicting views on the aetiology, host-parasite relations, and pathogenesis of amoebiasis. Although much research has been devoted to these questions, the results are inconclusive, and most physicians are influenced in their choice of a practical policy either by personal preference for some particular hypothesis or by expediency. The problem of amoebiasis in this country can be best appreciated from a review of the present state of our knowledge of the subject.

Incidence of Infection

Investigations carried out in the last 35 years have demonstrated that *E. histolytica* has a world-wide distribution and that the incidence of infection with this parasite may be as high in countries with a temperate climate as in those with a tropical climate. It has also been established that the majority of infected persons everywhere are carriers

*Read in opening a discussion in the Section of Tropical Medicine at the Annual Meeting of the British Medical Association, Liverpool, 1950.

without any symptoms of disease. As regards clinical cases (amoebic dysentery, etc.), these are common in warm countries lying between latitudes 40° N. and 30° S., but are exceptional among the indigenous populations of temperate regions. Cases of amoebiasis in Great Britain will therefore comprise, on the one hand, persons whose infection is of local origin, and, on the other hand, those who have acquired their infection abroad.

During and shortly after the 1914-18 war single stool examinations of more than 3,000 persons, who had never been abroad, resulted in the detection of *E. histolytica* in about 3.4%. Since one faecal specimen reveals less than one-third of the infections, it has been estimated that the actual incidence of infection was from 7 to 10% of the general population (Dobell, 1921; Dobell and Harvey, 1923). In a similar examination made after the 1939-45 war by Morton *et al.* (1950) an examination of three stool specimens of 1,000 young R.A.F. apprentices, who had never been abroad, revealed the presence of *E. histolytica* in 1.6%. Though the sample—representing a total of 4,000 persons—is small in relation to the population as a whole, it is fairly representative, and indicates that probably between one and four million of the inhabitants of these islands are infected with the “dysentery amoeba.”

The most important feature of these indigenous cases of amoebic infection is that they are to all intents and purposes normal and healthy persons who are symptomless carriers of the parasite, the presence of which is usually revealed only when their cysts are passed in the stools, hence the designation “cyst-passers.” It is now generally accepted that most persons infected with *E. histolytica* throughout the world are carriers, and the incidence of infection in various countries and different climates is comparable to the figures reported for Great Britain. A Russian worker, Gnezdilov (1947), reckons that the average global incidence of symptomless carriers of amoebiasis is about 20%.

In considering the occurrence of amoebiasis in Great Britain we must also take into account cases introduced from abroad. While in a maritime country like ours opportunities for the introduction of individual cases are always present, they multiply considerably as the result of wars. The available figures for imported cases are all based on the examination of troops returning from overseas. Thus during the 1914-18 war the incidence of amoebic infection among men invalided for dysenteric complaints was from 18 to 25%, while among the non-dysenteric cases it was from 12 to 15% (Dobell and Harvey, 1923). Later, in the course of routine examination of R.A.F. personnel, Whittingham (1923) found 10% harbouring *E. histolytica*. A similar investigation was carried out recently by Rickards (1949) on 2,000 men of the R.A.F. after their disembarkation from the Mediterranean and Indian areas. Single stool examinations revealed *E. histolytica* in 3.47% and 7.33% respectively, but it was estimated that the true incidence of infection was from 10.4 to 36.6%.

Although the majority of cases of clinical amoebiasis among the troops underwent a course of treatment, many carriers returning to civil life must have escaped detection and contributed to the contamination of the resident population, about 10% of whom are already carriers of *E. histolytica*. Among the carriers in Britain one is therefore likely to encounter the following three types, which—in the absence of clinical signs—may be indistinguishable: chronic cases in the latent phase, convalescents, and contacts. However, chronic cases and convalescent carriers are liable to relapse, and can then be dealt with by appropriate treatment. But contact carriers—who are the great

majority—are in a different category, and the attitude adopted in their case depends on the view held regarding their host-parasite relations.

Host-Parasite Relations

Though at present nobody will deny the existence of symptomless carriers of *E. histolytica*, their danger—as a source of disease in others—has not been fully elucidated, but it is well known that, in spite of the presence in this country of upwards of a million individuals harbouring this parasite, the number of clinical cases of amoebiasis is negligible. The significance of carriers can be properly appreciated only from a knowledge of the available facts and their interpretation, which can now be considered.

Most clinicians and many parasitologists in this country and in America (for example; Dobell, 1919; Faust, 1941; Craig and Faust, 1945) regard *E. histolytica* as an obligatory tissue-parasite which invariably invades the gut wall, producing the characteristic gross or minute lesions and feeding on red blood corpuscles. While admitting the occurrence of a large proportion of carriers, they deny the very existence of healthy symptomless cases among them, believing that lesions, however minute, are present in all carriers. However, many other observers—chiefly on the Continent—believe that in carriers, especially of the contact type, amoebic infection is not necessarily accompanied by pathological changes, the parasites in most cases living as commensals in the lumen of the intestine, where they feed on bacteria and other faecal contents. During the last quarter of a century this conception—first enunciated in Holland by Kuenen and Swellengrebel in 1913—has been steadily gaining support until it now rests on a solid foundation (cf. Hoare, 1949b).

The commensal habits of *E. histolytica*, which have been established beyond all doubt *in vitro* since the discovery of suitable culture media in 1925, were later fully confirmed *in vivo* in experimental infections of monkeys (Dobell, 1931) and rats (Neal, 1948). The mode of life of this amoeba in human carriers does not readily lend itself to direct observation, but in the past a number of observers have described and depicted the ingestion of micro-organisms by *E. histolytica* recovered from human cases. I have personally studied material from symptomless carriers and patients suffering from chronic amoebiasis, and have satisfied myself—and demonstrated to others—that the amoebae from such persons contain food-vacuoles enclosing bacteria, which had obviously been ingested from matter in the lumen of the gut (Hoare, 1947, 1949a). Further proof of the commensal existence of *E. histolytica* in the lumen is provided by direct examination of the intestinal wall in symptomless carriers. Thus Morton *et al.* (1950), who performed sigmoidoscopy on such cases, found that the mucosa was perfectly normal, while Acton (1933) made careful post-mortem examinations of several cases without detecting any signs of ulceration.

There is also a considerable amount of circumstantial evidence that *E. histolytica* may live as a commensal. Thus the number of non-haematophagous amoebae and cysts discharged by carriers may reach 350,000 per gramme of faeces, or 35 million daily, and it is inconceivable that such enormous masses of amoebae could be living in the gut-wall without producing any intestinal disorder. Another clue is provided by the administration of purgatives: this is followed by the discharge of numerous amoebae, but on succeeding days there is a marked fall in the number of cysts found in the stools. This diminution would not have taken place if the amoebae were derived from ulcers,

unless it is assumed that the purgative causes them to emerge into the lumen, which is highly improbable (Reichenow, 1931).

An interesting investigation on this question was carried out by Andrews and Atchley (1932), who argued that if all the amoebae resided in tissues haemorrhage would occur in a high proportion of cases, but if they were lumen-dwellers bleeding would not necessarily result. Using the benzidine test for occult blood, they examined the stools of symptomless carriers without detecting any traces of blood in them, and concluded that *E. histolytica* is not an obligatory tissue-parasite, but that in the great majority of carriers it lives in the lumen of the gut. They also remarked that if the vast numbers of cysts evacuated daily in these cases originated from amoebae in the tissues it would be necessary to assume that millions of parasites can be expelled daily from the bowel without loss of detectable amounts of blood.

From these observations it is evident that the recognition of a commensal phase in the life-history of *E. histolytica* is fully justified. However, the bias against this view is still very strong. Thus, Faust (1941) examined the intestines of 202 individuals who had died of accidents in New Orleans, and found that 13 harboured *E. histolytica*. Though only seven of them showed superficial lesions of the mucosa, Faust assumed that undetected lesions must have been present in the other six as well. However, the only logical conclusion from these findings is that in about 50% of the carriers observed by him the amoebae were actually living in the lumen in the absence of any gut lesions (Hoare, 1947). As regards the minute lesions detected in the other cases, it is highly probable that they represented superficial necrotic changes of the mucous membrane which commonly develop after death (Reichenow, 1931; Napier, 1947).

In the light of our present knowledge the host-parasite relations in amoebiasis can be summarized as follows: although *E. histolytica* is a pathogenic parasite, capable of invading the tissues of its host with the production of lesions and clinical symptoms of disease, in most cases it does not manifest any evidence of virulence, for in up to 90% of persons harbouring this parasite the infection is symptomless and the host is a carrier. In most carriers the amoebae live in the lumen of the gut as harmless commensals and represent the only stage capable of producing cysts, which serve to propagate the infection to new hosts. But under certain conditions the lumen-dwelling amoebae may reveal their virulence and invade the intestinal wall, giving rise to the typical symptoms of amoebiasis (Hoare, 1949b).

Pathogenesis

The factors which determine the virulence of *E. histolytica* are not clearly understood. Though some workers (Kuenen and Swellengrebel, 1913; Adams, 1945; Wenyon, 1947; Murgatroyd, 1947) believe that a carrier may at any time develop clinical symptoms, it is generally admitted that this rarely occurs in countries with a temperate climate. To account for this, it has been suggested that there are races or strains of *E. histolytica* differing in the degree of their pathogenicity. Among such races there are two which differ in size (Gnezdilov, 1934; Sapero *et al.*, 1942; Hoare, 1945): a large one with cysts having a mean diameter of 11–12 μ , and a small race with cysts measuring 7–8 μ in diameter. In practice a diameter of 10 μ can be taken as a dividing-line between the two races.

It has been found that in the United States (Sapero *et al.*, 1942) and in Great Britain (Morton *et al.*, 1950) at least

50% of the symptomless carriers harboured the small race. The large race corresponds to the conventional *E. histolytica*, which is the pathogenic form causing amoebic dysentery and other symptoms of amoebiasis. As regards the small race, which is sometimes described as *E. hartmanni*, from the available evidence it would seem that the small amoebae do not ingest erythrocytes and do not produce dysenteric symptoms in experimentally infected cats, while Morton *et al.* (1950) found that rats were refractory to infection with this race. There is also a general consensus of opinion that in human infections clinical symptoms are never associated with the presence of amoebae of the small race, which is therefore not pathogenic.

While most workers now agree that the small race of *E. histolytica* is quite harmless to man, opinions regarding the existence of physiologically distinct strains within the large race are divided. An extreme view is that held during the last 25 years by Brumpt (1949), who recognizes two morphologically indistinguishable strains of the large race. One (which he separates as a distinct species, *E. dispar*) is regarded as being non-pathogenic and responsible for infections in symptomless carriers throughout the world; the other (*E. dysenteriae* or *E. histolytica sensu stricto*), which is said to be restricted to warm countries, is the pathogenic form causing amoebic dysentery primarily in endemic areas and secondarily in countries with a temperate climate, when introduced by carriers returning from the Tropics.

Apart from the unnecessary splitting of *E. histolytica* into two species, it is difficult to admit the existence of a non-pathogenic race of this amoeba, when it is known to be potentially pathogenic even while its virulence for man is not manifested. Thus it has been demonstrated that strains from diverse sources—including carriers in countries with a temperate climate—are capable of producing ulceration of the gut in experimentally infected animals (Matevossian *et al.*, 1941; Frye and Shaffer, 1948; and others), but it is noteworthy that some observers (Morton *et al.*, 1950; Neal—personal communication) have found that British strains from indigenous carriers were less virulent for rats than strains from overseas.

From these observations it is evident that Brumpt's views cannot be accepted unreservedly, but it is conceivable, as already suggested by Dobell (1921), that in different parts of the world there are various physiological strains of *E. histolytica*. These strains not only are probably specially adapted to the local inhabitants but also differ in the degree of their pathogenicity. The strains in countries with a temperate climate, like England, would seem to be much less virulent than strains in warm countries, and are usually harmless to their hosts, who are symptomless carriers. It is therefore possible that clinical cases of amoebiasis in this country are almost if not entirely due to imported virulent strains of *E. histolytica*.

Similar views have been expressed by some foreign workers. Thus, Westphal (1937b) found that in Germany cases of amoebic dysentery were restricted to the Baltic ports, whereas in the interior of the country only symptomless carriers occurred; and Sluiter (1948) believes that all cases of amoebic dysentery in Holland are of Indonesian origin. The number of cases of clinical amoebiasis reported in Great Britain among persons who had never been abroad has always been insignificant, the total not exceeding three dozen. Among those recorded recently, in some instances (Morton *et al.*, 1950) there was direct proof of contact with patients suffering from amoebiasis contracted overseas, while in others, reported from Liverpool (Adams and Seaton, 1949), there was strong circumstantial evidence of contact with extraneous sources of infection.

However, we are still in ignorance regarding all the factors governing the pathogenesis of *E. histolytica*, though a number of hypotheses have been advanced. According to some investigators (Walker and Sellards, 1913) the course of infection depends mainly upon variations in the susceptibility of the human host. From this point of view the greater severity of the disease among Europeans in hot countries is attributable to lower resistance due to an unfavourable climate, unsuitable diet, and similar causes (Hoare, 1949b). Other observers (Westphal, 1937a; Deschiens, 1938) believe that the development of amoebic dysentery is due to the association of the parasite with pathogenic bacteria—especially dysentery bacilli (Westphal, 1948)—which are the first to damage the intestinal wall, thereby exposing it to invasion by the amoebae. Recent observations have shown that bacterial infections are responsible for at least some complicating symptoms in amoebic dysentery (Hargreaves, 1945). It is possible that any or all of the factors mentioned (races differing in virulence, host-resistance, climate, synergic action of bacteria) may influence the course of amoebic infection.

Conclusions

From the data at our disposal it is evident that the incidence of amoebic infection in the native inhabitants of Britain has remained relatively stationary during the last 35 years and has not been significantly influenced by the two intervening world wars. This is also the conclusion arrived at by the Amoebiasis Panel in the United States (Wright, 1950; Lincicome *et al.*, 1950). Practically all the cases encountered in this country are symptomless carriers. One-half of them harbour the non-pathogenic small race of *E. histolytica*, while the other half are infected with the potentially pathogenic large race, the local strain of which seems to be comparatively harmless to its hosts. It is conceivable that the isolated cases of amoebic dysentery occurring from time to time among the native residents may be due to contamination with more virulent imported strains of this parasite. It would seem, therefore, that on the whole the local inhabitants have a high degree of tolerance to infection with *E. histolytica*, which is only occasionally broken down when virulent exotic strains are introduced into the country. But even these are apparently unable to establish themselves permanently in the community, probably owing to the absence in the host-environment of conditions favouring the maintenance of the parasite's virulence.

It must be admitted, however, that there is no absolute proof that *E. histolytica* in this country is invariably innocuous, though the experience of many years indicates that the prevalence among the indigenous population of amoebic carriers need not be regarded with alarm. Nevertheless, as there is an element of doubt, it would be rational, as some clinicians advocate (Adams, 1945; Murgatroyd, 1947; Napier, 1947), to treat all cases in which infection with *E. histolytica* has been diagnosed, though infections with the small race of this amoeba can be safely ignored. However, since it is impossible to detect and treat the many thousands of carriers present in this country, in actual practice they are usually left alone and only cases which develop clinical symptoms undergo treatment. As past experience has shown that amoebic carriers in a temperate country are of no danger to the community, some workers (Dobell, 1921; Gnezdilov, 1947; Bach, 1949) even prefer to adopt this course as a practical policy. The problem of carriers thus resolves itself into a question of expediency.

Whatever views may be held regarding the measures to be taken against them, the presence in our midst of

thousands of amoebic carriers has an important bearing on public health, which the late Clifford Dobell (1921)—who was noted for plain speaking—expressed as follows: "When we find persons infected with intestinal protozoa we must conclude that they have previously swallowed particles of the fairly fresh and damp excrement of other persons; and when we find many people presenting such evidence of pollution we can only conclude that the disposal of human faeces has not yet been brought generally to that pitch of perfection which seems desirable. . . . It is clear that . . . we still have much to learn about individual and communal hygiene."

I wish to express my gratitude to Air Vice-Marshal T. C. Morton and Dr. R. A. Neal for permission to consult and quote from their papers while these were still in the press.

REFERENCES

- Acton, H. W. (1933). *Trans. R. Soc. trop. Med. Hyg.*, **27**, 119.
 Adams, A. R. D. (1945). *Ibid.*, **38**, 237.
 — and Seaton, D. R. (1949). *British Medical Journal*, **2**, 136.
 Andrews, J., and Atchley, F. (1932). *J. Amer. med. Ass.*, **99**, 1340.
 Bach, F. W. (1949). *Zbl. Bakt. (I, Orig.)*, **153**, 4.
 Brumpt, E. (1949). *Précis de Parasitologie*, 6th ed. Paris.
 Craig, C. F., and Faust, E. C. (1945). *Clinical Parasitology*, 4th ed. London and Philadelphia.
 Deschiens, R. (1938). *Ann. Inst. Pasteur*, **61**, 5.
 Dobell, C. (1919). *The Amoebae Living in Man*. London.
 — (1921). *Spec. Rep. Ser. Med. Res. Comm.*, No. 59. London.
 — (1931). *Parasitology*, **23**, 1.
 — and Harvey, D. (1923). *History of the Great War. Medical Services: Pathology*, p. 277. H.M.S.O., London.
 Faust, E. C. (1941). *Amer. J. trop. Med.*, **21**, 35.
 Frye, W. W., and Shaffer, J. G. (1948). *Proc. 4th Int. Congr. Trop. Med. Malaria*, Washington, **2**, 1075.
 Gnezdilov, V. G. (1934). *Rev. Microbiol., Saratov*, **13**, 137.
 — (1947). *Med. Parazitol., Moscow*, **16** (5), 13. (In Russian.)
 Hargreaves, W. H. (1945). *Trans. R. Soc. trop. Med. Hyg.*, **38**, 244.
 Hoare, C. A. (1945). *Ibid.*, **38**, 255.
 — (1947). *Ibid.*, **41**, 87.
 — (1949a). *Ibid.*, **43**, 7.
 — (1949b). *Handbook of Medical Protozoology*. London.
 Kuenen, W. A., and Swellengrebel, N. H. (1913). *Zbl. Bakt. (I, Orig.)*, **71**, 378.
 Lincicome, D. R., Thiede, W. H., and Carpenter, E. (1950). *Amer. J. trop. Med.*, **30**, 171.
 Matevossian, S., Sarkissian, M., and Markaryan, A. (1941). *Med. Parazitol., Moscow*, **10**, 264 (In Russian). Review in *Trop. Dis. Bull.*, 1943, **40**, 310.
 Morton, T. C., Neal, R. A., and Sage, M. (1950). *Lancet*. In press.
 Murgatroyd, F. (1947). *Trans. R. Soc. trop. Med. Hyg.*, **41**, 63.
 Napier, L. E. (1947). *J. trop. Med.*, **50**, 169.
 Neal, R. A. (1948). *J. Hyg., Camb.*, **46**, 90.
 — (). *Trans. R. Soc. trop. Med. Hyg.* In press.
 Reichnow, E. (1931). *Zbl. Bakt. (I, Orig.)*, **122**, Bericht, p. 195.
 Rickards, A. G. (1949). *J. trop. Med.*, **52**, 2.
 Saper, J. J., Hakansson, E. G., and Louttit, C. M. (1942). *Amer. J. trop. Med.*, **22**, 191.
 Sluiter, C. P. (1948). *De Dierlijke Parasieten van den Mensch*, 5th ed. Amsterdam.
 Walker, E. L., and Sellards, A. W. (1913). *Philipp. J. Sci. (B)*, **8**, 253.
 Wenyon, C. M. (1947). *Trans. R. Soc. trop. Med. Hyg.*, **41**, 55.
 Westphal, A. (1937a). *Arch. Schiffs- u. Tropenhyg.*, **41**, 262.
 — (1937b). *Festschrift Nocht*, p. 662. Hamburg.
 — (1948). *Z. Hyg. InfekKr.*, **128**, 73.
 Whittingham, H. E. (1923). *British Medical Journal*, **1**, 799.
 Wright, W. H. (1950). *Amer. J. trop. Med.*, **30**, 123.

Among administrative changes made in the provision of medical and surgical appliances the following is announced by the Ministry of Health. When a hospital specialist recommends the provision or renewal of surgical footwear he may, if satisfied that the case has reached stability, advise that the supply of such footwear in accordance with the prescription may continue up to five years without further medical examination. This should be stated on the patient's hospital record, and the Appliance Order Form No. 2 should be endorsed "stabilized for (so many) years." In these cases the Ministry of Pensions will renew the footwear provided that the patient is satisfied with the previous pair. If a change of contractor is necessary, the Ministry of Pensions will refer the patient back to the hospital specialist. At the end of the specified period the Ministry of Pensions will refer these cases to the hospital specialist, who may again advise renewal.