THUCYDIDES AND THE PLAGUE OF ATHENS

1. INTRODUCTION

Two problems involving Thucydides and medicine have attracted intense treatment by classical scholars and medical men working separately or in combination. They are, first, the nature of the Athenian Plague which Thucydides describes and, second, the possibility of his having been influenced by the doctrines and outlook of Hippocrates and his followers. It is the purpose of the present paper to reconsider both these problems, to indicate some false assumptions made in the methodology of previous attempts to identify the Plague, and to suggest a somewhat radical revaluation of Thucydides' approach to medical matters compared with that of Hippocrates (if, indeed, the surviving evidence about Hippocrates' method has any validity).

Before any attempt at identification of the Plague can be made it is necessary to establish certain basic scientific points which have been neglected in previous discussion. The first of these concerns the concept of a 'disease'.

2. THE CONCEPT OF A DISEASE

If a number of sick people are similarly affected in respect of certain identifiable characteristics of their illness, they may be said to be suffering from the same disease. The name of a disease is the label attached to a category of sick persons. The criteria available for classifying sick people depend on the state of medical knowledge of a particular community at a particular time. As knowledge advances, the classification of illness changes in a variety of ways. Categories may split: typhus and typhoid fevers were differentiated at the beginning of the nineteenth century; haemophilia and Christmas disease in the early 1950s. Several categories may be united into a new one: consumption of the lungs, scrofula, tabes mesenterica, and lupus vulgaris were joined in the new category called tuberculosis when it became clear that all were manifestations of infection by the same bacterial species; general paralysis of the insane, tabes dorsalis, and certain kinds of aortic aneurysm turned out to be late stages of syphilis, a name previously used to describe what we now regard as the early stages of the disease. The boundary between two disease categories may be moved: this has happened in the case of pernicious anaemia and steatorrhoea with the result that some patients who would have been diagnosed as having pernicious anaemia in the past would now be said to have steatorrhoea.

When an advance in knowledge necessitates reclassification a new term may be coined or an old one adapted. In the examples given above, haemophilia now refers to a sub-category of cases previously called haemophilia, while syphilis is a word whose meaning has enlarged. Such redefinition of the same word can cause confusion, but the naming of diseases is a matter of convenience rather than consistency. This leads us to the real purpose of names of diseases: they are convenient short expressions whereby one medical practitioner can convey to another a lengthy but standard message. In one or two words he can convey what could in principle be expanded to hundreds or thousands of words, and it is assumed that the recipient of the message has the knowledge to do so. (If not, as is the case for most patients, the professional code-words may be uninformative

or positively misleading: does pernicious anaemia sound like a condition that is easily and completely treatable?)

Since the name of a disease is a code-word for a lengthy message whose detailed content is changing continuously, it follows that such a name is of limited applicability outside the time and place to which it belongs. Guy de Maupassant is said to have died of general paralysis of the insane, and indeed he did, as the disease was then defined. But it can now be defined much more precisely. If a modern physician could examine him, would he reach the same conclusion? Probably, but we cannot be sure. Certain observations which would now be regarded as crucial were not made at the time, and of course they cannot be made now. But we know enough to make it likely that he died of the disease as we now define it.

Maupassant died less than a century ago. When we consider the case of the Athenian Plague we are dealing with a very different time-scale, and a completely different problem arises. When an ancient historian asks a physician to diagnose the Athenian Plague what he is really asking is: 'Guess what you would call it if you could travel in a time-machine to Athens in 430 B.C., and carry out any observations you would like to make.' When the problem is formulated in this way it at once becomes clear that there is a concealed assumption. It is supposed that the hypothetical well-equipped time-traveller could in principle answer the question. Unfortunately this is not the case for reasons we must next examine.

3. EVOLUTION OF HOST–PARASITE RELATIONSHIPS

A parasite is a living organism that exists in or on another living organism (its host) from which it derives its nourishment. Pathogenic (disease-producing) bacteria and viruses are by definition parasites. It should be noted, however, that a parasite does not necessarily harm its host to any serious degree. Evolution by mutation and natural selection can over a period of time cause marked changes in the effect of parasites on their hosts, and evolution of both host and parasite may contribute to this changed relationship, as must next be explained.

This is not the place to consider the development of ideas about evolution and genetics in any detail, but certain important points must be made clear. When Darwin[1] published his book on the origin of species he produced such a wealth of evidence in favour of his main contention that it has never been seriously contested on rational grounds. Nevertheless (as he was well aware) he left something very important unexplained. To Darwin and his contemporaries inheritance was a blending of characters derived from both parents. But if this were really true then surely members of a species could only become more and more similar as generation succeeded generation. How could the exceptional individuals arise that were fitter to survive than their kindred? The beginnings of a solution to this difficulty came a few years later with the work of Mendel[2] who showed that inheritance was particulate and that what was inherited was not characters but elements which determine characters (genes as we now call them). Genes are transmitted from generation to generation, but characters may be

[1] C. Darwin, *The origin of species by means of natural selection or the preservation of favoured races in the struggle for life* (London, 1859).

[2] G. Mendel, 'Versuche über Pflanzen-Hybriden', *Verhandlung des Naturforschenden Vereines in Brünn,* iv (1865), 1 ff.. An easily accessible English version can be found in *Br. med. J.* (1965) 1. 370 ff.

submerged completely. For example, Mendel crossed purple-flowered peas with white-flowered peas. The progeny plants all had purple flowers. But the offspring of these progeny plants included both purple-flowered and white-flowered plants in a ratio of three to one. Unfortunately Mendel's work did not become widely known until it was confirmed in 1900 by Correns in Tübingen, Tschermak in Vienna, and de Vries in Leyden. Shortly afterwards Bateson in Cambridge demonstrated Mendelian inheritance in animals. At first sight these results seem to make the mechanism of evolution even harder to understand. If finite particles are inherited there can only be a finite number of them and the number of ways in which they can combine, though large, must be limited, so that in the end evolution would come to a stop when the most favourable combination had been selected. In fact this was never a serious difficulty (except, apparently, to Lysenko). Almost at the same time as the rediscovery of Mendel's work, de Vries discovered the phenomenon of mutation: from time to time a gene becomes altered and is then inherited in its altered form, so that fresh combinations become possible.

The fact that bacteria and viruses can evolve by mutation and selection has been established only comparatively recently. These simple organisms are so very different in their structure and modes of multiplication from more complex forms of life that it could not be assumed without direct evidence that they were capable of evolving in anything like the same way. Only with the experiments of Luria and Delbrück[3] and of Newcombe[4] in the 1940s and of the Lederbergs[5] in 1952 did it finally become clear that bacterial evolution by mutation and selection can and does occur. The wider implications of these discoveries are still being worked out. In the past many people (including writers on the Athenian Plague) have been under the impression that bacteria and viruses are stable in their properties (for example, in the types of disease they can cause) over long periods. As we must now explain, this is unlikely in theory and has not happened in practice.

It has long been clear that many infectious diseases have tended to become less severe over a long period. In some cases improved living conditions and advances in medical knowledge have no doubt been at least partly responsible. In other cases it is difficult to see how this can be the whole story. For example, the very steady decline in tuberculosis mortality during the half-century preceding the First World War and the spectacular disappearance of scarlet fever seem to require some other explanation. When syphilis first arose in Europe at the end of the fifteenth century it seems to have caused many deaths in the early stages. This has certainly not been so in the past century. As early as 1546 Fracastoro noticed that it had become less severe in the half-century since its introduction. It now seems likely that evolutionary changes in both host and parasite contribute to declining severity of microbial infections. Within a given species of pathogenic microbes it is usually possible to demonstrate that there are strains of very variable virulence for the host. Clearly a strain which manages to survive in its host for a longer period will usually have a better chance of spreading to other hosts than one that kills its host quickly (and thereby commits suicide). Natural selection should operate to favour the development of strains of steadily decreasing

[3] S. E. Luria and M. Delbrück, 'Mutations of bacteria from virus sensitivity to virus resistence', *Genetics*, 28 (1943), 491 ff.

[4] H. B. Newcombe, 'Origin of bacterial variants', *Nature*, 164 (1949), 150 ff.

[5] J. Lederberg and E. M. Lederberg, 'Replica plating and indirect selection of bacterial mutants', *J. Bacteriol.* 63 (1952), 599 ff.

virulence. Natural selection can also operate on the host species in such a way that its members become gradually more resistant to the infection. For this to happen, there must be variation in susceptibility to infection among different individuals of the host species, and this variation must be inherited. Natural selection will then tend to reduce gradually the proportion of more susceptible individuals. Such selection will be most effective if a large proportion of deaths from the disease occur among children and young adults, i.e. those who have not yet had the opportunity to transmit their genes determining susceptibility to the next generation. Genetic variations in susceptibility to pathogenic microbes have been extensively studied in experimental animals and there can be little doubt that similar variations occur in man. Definite evidence for this is not so far very extensive but there are some well-authenticated examples, such as genetic variation in susceptibility to tuberculosis. Probably the decline in deaths from tuberculosis is partly explained by the slow elimination of genetically susceptible hosts.

The most thoroughly studied example of the kind of process we have been considering comes not from a human disease but from rabbit myxomatosis. This disease killed most of the rabbits in several parts of the world in the decade following 1950. The outbreak of the disease in Australia was carefully studied by Fenner.[6] Over a ten-year period he isolated strains of the virus, tested them against stock laboratory rabbits, and showed that the virus was becoming steadily less virulent. He also captured groups of wild rabbits and showed that they became more and more resistant to a standard culture of the virus. The host–parasite relationship had evolved in a direction beneficial to both rabbit and virus in the struggle for existence. Because of the short generation time of the rabbit a change took place over ten years that might take centuries in man.

It will at once be clear that a terminological difficulty arises. Is rabbit myxomatosis of 1950 'the same disease' as rabbit myxomatosis of 1960? The virus is different; the rabbits are different; the effect of the one on the other is to produce a much milder disease. And the virus originally came from a South American species of rabbit in which the disease caused is known as rabbit fibroma.

In short, it is not strictly accurate to talk about 'the same disease' when discussing events separated by more than a short time. If the disease changes slowly, no harm is done, even though it is clear, for example, that tuberculosis is not the dreaded killer of a century ago. Moreover some diseases probably change so slowly that it might seem needlessly pedantic to object to talking about 'the same disease'. Thus the bacterium which is now known as *Yersinia pestis* was isolated in an outbreak of bubonic plague in Hong Kong. The disease was very similar to the Black Death (well described by Boccaccio[7]) and to a sixth-century outbreak described by Procopius.[8] Many other outbreaks were described in between. Here we seem to feel the lack of a suitable term to describe the situation. No doubt there has been some small change in the effect of *Yersinia pestis* on *Homo sapiens* and it is not quite 'the same disease'. One would like to be able to talk about a 'disease descent line' or a 'pathological evolutionary continuum'. The reason why no such expression is in common use is that an insufficient number of examples exists to warrant the coining of a new term.

Names of diseases, we must repeat, are code words used to help communication

[6] F. Fenner, *The Biology of Animal Viruses* (New York, 1968), ii. 762–9.

[7] G. Boccaccio, Proem to *Il Decamerone*.

[8] Procopius, *Bell. Pers.* 2. 22.

between medical practitioners. Historians who wish to use these names for an entirely different purpose are misusing them and there is a limit to how much misuse they will stand before absurdity is reached. In respect of bacterial and viral infections of man it is not possible to talk about 'the same disease' when discussing events separated in time by twenty-four centuries. To do so is at best seriously inaccurate and at worst meaningless.

4. SOME PREVIOUS ATTEMPTS TO IDENTIFY THE PLAGUE OF ATHENS

We must now review briefly the principal diagnoses that have been made in the past. We have, we hope, made it sufficiently clear that there would be little point in attempting a comprehensive review, a task that in any case would be virtually impossible in view of the enormous amount that has been written on the subject. Quite apart from articles dealing specifically with the Plague of Athens, innumerable medical and historical textbooks and monographs refer to it briefly and so do practically all annotated editions of Thucydides that have ever been published. Anyone who has made a serious attempt to read the relevant literature has reached the point at which it is as difficult to know where to look next as to know when to stop.

Some general points must be made. The diversity of opinions expressed by able physicians might and indeed should lead an unprejudiced person to conclude that Thucydides' description does not exactly correspond to any disease of the present day. We can go further. If it were certain that Thucydides' account was accurate in all respects it could be said with equal certainty that the Plague of Athens was no known modern disease. Naturally Thucydides' description can be made to fit a number of modern diseases if it is assumed that one can leave out any part of the account that conflicts with the diagnosis being proposed, but this does not seem a very useful intellectual exercise. In the case of each of the diagnoses to be considered below, some awkward discrepancies have been swept under the carpet. We shall lift the edge of the carpet and retrieve some of them.

(i) *Smallpox.*[9] This seems to be the suggestion that has been made most often, but psephology has little to contribute to the solution of this problem. Superficially, the suggestion has much to commend it. We are looking for a highly contagious, febrile illness in which there is a skin rash; which has a high case mortality,[10] but good acquired immunity among the survivors. So far, so good.

[9] R. Kobert, 'Zur Geschichte des Mutterkorns', in: *Historische Studien aus dem Pharmakologischen Institute der Kaiserlichen Universität Dorpat* (Halle, 1889), i. 1–47; 'Ueber die Pest des Thucydides', *Janus*, 4 (1899), 240–51, 289–99; S. Widmann, *Thucydides für den Schulenbrauch erklärt von Gottfried Boehme*, 6th edn. (Leipzig, 1894), i. 197 f.; A. Panayotatou, 'La peste de Thucydide—la peste d'Athènes', *Comptes rendus du Deuxième Congrès International d'Histoire de la Médecine*, ed. Laignel-Lavastine and Fosseyeux (Évreux, 1922), pp. 533–6. In the discussion following Dr. Panayotatou's paper, she was strongly supported by Professor Jeanselme (pp.536 f.); H. Zinsser, *Rats, Lice and History* (London, 1935), pp. 119–27; B. von Hagen, 'Die sogenannte Pest des Thukydides', *Gymnasium*, 49 (1938), 120 ff.; R. J. Littman and M. L. Littman, 'The Athenian Plague: Smallpox', *TAPA* 100 (1969), 261–75.

[10] 'Case mortality' means the proportion of people contracting a disease who die from it. It must not be confused with the death-rate' from a disease, which is the proportion of individuals dying from that disease in a defined population considered as a whole. For example, in this country about 4 per cent of all women develop carcinoma of the breast, and about half of these die from the disease. Hence the case mortality is about 50 per cent, while the death-rate among all women in the U.K. is about 2 per cent.

Opponents of the idea have stressed the fact that smallpox survivors are frequently pock-marked; Thucydides has nothing to say about this. Undoubtedly this is a difficulty but it does not necessarily dispose of the idea.

Another difficulty about the smallpox theory is that there is one glaring discrepancy in the description of the symptoms and signs: gangrene of the extremities seems to have been a regular occurrence but does not often, if ever, happen with smallpox. Kobert[11] was impressed by this difficulty and fell back on the suggestion that the population was simultaneously suffering from ergotism. It was because of the occurrence of gangrene of the extremities that the diagnosis of smallpox was rejected out of hand by Sir William MacArthur.[12] Littman and Littman[13] have argued that Thucydides may not have meant that the patients had gangrene of the extremities, and alternatively plead that this can in fact occur in outbreaks of smallpox. One cannot really have it both ways and they do not seem to have disposed of this difficulty satisfactorily.

There are some stronger objections than these to accepting the idea that the Athenian Plague was smallpox. The smallpox virus has man as its only host: no other animal species ever becomes infected. Now Thucydides tells us (2.50. 1–2) that carnivorous birds and quadrupeds were probably, and domestic dogs certainly, liable to the disease. He emphasizes that this was a particular point of difference from other diseases. It is difficult to understand why he should say so if it did not in fact occur or if he felt uncertain.

Then there is the fact that second, non-fatal attacks could occur (2.51.6). Recovery from smallpox leads to a particularly solid immunity and there is no evidence that second attacks can occur. (The immunity following vaccination is less complete and less long-lasting, but we are concerned with the consequences of a natural infection.)

Finally there is the question of the case mortality. Thucydides has provided one precise piece of information about the death-rate: out of 4,000 hoplites in the expeditionary force led by Hagnon to Potidaea, 1,050 died of the Plague (2.58.3). Thus the case mortality would have been about 26 per cent if all 4,000 were infected or even higher if some were not. Now a figure of 26 per cent (or higher) would be in line with the case mortality rates in several nineteenth-century smallpox outbreaks for which reliable figures are available, *if we are considering the population as a whole.* But in these same nineteenth-century outbreaks it was found that 85 to 90 per cent of deaths occurred in children under the age of five. For smallpox, a case mortality rate of 26 per cent (which is a minimum figure) among men of military age would be impossibly high.

Any one of these last three objections is fatal to the smallpox theory. Each is based on unambiguous statements by Thucydides. Is it likely that he has misled us about all three?

(ii) *Bubonic plague.* This suggestion was made in the early nineteenth century by Ozanam.[14] More recently it has been revived by Williams[15] and

[11] Op. cit. (above, n. 9).

[12] W. P. MacArthur, 'The Plague of Athens', *Bull. Hist. Med.* 32 (1958), 242–6.

[13] Op. cit. (above, n. 9).

[14] J. A. F. Ozanam, *Histoire médicale générale et particulière des maladies épidemiques, contagieuses et épizoötiques,* 2nd edn. (Paris, 1835), iv. 6 ff.

[15] E. W. Williams, 'The sickness at Athens', *G & R,* 26 (1957), 98–103. 'The end of an epoch', *G & R,* 2nd ser. 9 (1962), 109–25.

Hooker.[16] The same suggestion was made more tentatively by Barger,[17] while Major[18] wrote: 'probably it was both bubonic plague and typhus fever'. Many other writers have considered and dismissed the possibility. There are several powerful objections to the idea.

First, Thucydides' description of the symptoms and signs has little resemblance to bubonic plague. In particular, there is no mention of the most obvious feature, the buboes, which are large swellings, occurring most often in the groins but sometimes in the armpits or elsewhere. This striking manifestation of the disease was clearly described, for example, by Procopius[19] and by Boccaccio,[20] neither of whom had any claim to extensive medical knowledge and both of whom provided much shorter and less detailed accounts of the illness they were describing than did Thucydides.

It was suggested by Hooker[21] that Thucydides' Plague was bubonic and that he used the word ἕλκος to mean *bubo* because it was a suitable word and she claims that other writers used ἕλκος in this sense. This is the crucial point, since if ἕλκος can be shown to be a normal word to use for a *bubo* even when βουβῶν was in regular use, then the demonstration that βουβῶν was already in use in Thucydides' time would be irrelevant. But, in fact, there is no ground for believing that ἕλκος was so used by other writers except on the assumption that all 'plagues' were bubonic—which is precisely the question at issue.

The word βουβῶν was in common use in the fifth century, as Hooker herself admits, and it means a swelling as opposed to a wound, which is the normal meaning of ἕλκος. There is a noteworthy passage in Menander (*Georgos* 50–2) where the farmer complains that a βουβῶν has arisen on his groin as the result of a wound (ἕλκος) inflicted on his foot by a spade. The two words are here used by a layman in their distinct and correct meanings and there is no reason why Thucydides should have confused them.

One objection to the idea that the Plague of Athens was bubonic plague cannot be sustained and must be disposed of. In most outbreaks of bubonic plague the disease is spread from rat to man by the rat flea, which can crawl for short distances from the moribund rat; hence the bacteria will usually enter the human body via the feet or legs and hence the usual localization of buboes in the groin. Now the Greeks in classical times seem to have had no word for rat. Page[22] concludes that there were therefore no rats in Greece at the time and consequently that a diagnosis of bubonic plague cannot be considered. Others believe that μῦς referred to rodents in general, including both the mouse and the rat, although Page will have nothing of this idea. Sir William MacArthur[23] has persuasively argued that it is almost impossible to believe that there were no rats in Greece in the fifth century. But all this is irrelevant. About 200 species of mammals have been shown to harbour *Yersinia pestis* and many of these, ranging from mice to camels, have been implicated in transmitting bubonic plague to man. It would therefore be quite unreasonable to argue that there was no possible animal

[16] E. M. Hooker, 'Buboes in Thucydides?', *JHS* 78 (1958), 78–83.

[17] G. Barger, *Ergot and Ergotism* (London, 1931), p. 42.

[18] R. H. Major, *War and Disease* (London, 1942), p. 17.

[19] Op. cit. (above, n. 8).

[20] Op. cit. (above, n. 7).

[21] Op. cit. (above, n. 16).

[22] D. L. Page, 'Thucydides' description of the great plague at Athens', *CQ* N.S. 3 (1953), 97–119.

[23] W. P. MacArthur, 'The Athenian Plague: a medical note', *CQ* N. S. 4 (1954), 171–4.

reservoir of infection. But to dispose of this particular objection to the bubonic-plague theory does not dispose of the serious objections considered above.

A further difficulty arises from the fact that bubonic plague in the strict sense is not really a contagious disease: it is not spread directly from person to person, but is derived from infected rats or other mammals (see above). In some outbreaks, however, a variant form of the disease known as pneumonic plague is common. The bacteria in a particular patient may spread to the lungs causing a rapidly fatal pneumonia. This can then be spread to other people by close contact with a patient. Pneumonic plague was probably common with the Black Death, but must have been rare in the epidemic described by Procopius (see below, p. 299, and n. 57. It is usually fatal in two to four days from the onset of symptoms, which are those of a very severe pneumonia. Nothing in Thucydides' account sounds remotely like pneumonic plague.

There is really only one good point to be made in favour of bubonic plague: this is the only major epidemic disease of man which affects other animal species as well. This point has been stressed by Williams.[24]

If the Plague of Athens was bubonic plague, Thucydides was wildly wrong in his detailed account of the symptoms and mode of transmission. It seems, in short, a most improbable suggestion.

(iii) *Scarlet fever.* Rolleston[25] stated that:

> Dr. Charles Collier, a distinguished Fellow of this College [the Royal College of Physicians], in 1857 boldly asserted but without a shred of evidence, that the pestilence of Athens in 430 B.C., described by Thucydides, was malignant scarlet fever and this interpretation was also held by the late Sir Benjamin Ward Richardson and received some measure of approval from Sir Thomas Clifford Allbutt[26]. . . . The same groundless interpretation has been given by Malfatti at the beginning of the nineteenth century.

The coolness of Rolleston's appraisal of the merits of this suggestion is very understandable. Scarlet fever is a disease of man alone and has a very low mortality at the present day (though, of course, the mortality may well have been higher in the past). We must beware of over-interpreting Thucydides' account of the skin rash in view of the difficulty of determining with sufficient precision the meanings of the words he used. But we can surely make enough of Thucydides' description to exclude a disease with such a very different kind of rash as has scarlet fever. This suggestion has very little merit and has aroused very little interest among recent writers on the Plague of Athens. It will therefore not be considered further.

(iv) *Measles.* This diagnosis has been proposed by Shrewsbury[27] and Page.[28] It is quite unacceptable at its face value but far from unreasonable in a modified form. In European countries in the present century, even before effective treatment for the serious complication of bronchopneumonia became available, the mortality in outbreaks was usually under 5 per cent. However, bearing in mind the tendency for contagious diseases to evolve to milder forms for reasons considered above, it is quite likely that if the pathogenic micro-organisms responsible for the Athenian Plague have descendants that are with us at the present day they would now produce a much milder disease. If this has happened, measles is a plausible candidate as a remote descendant of the Plague of Athens.

[24] Op. cit. (above, n. 15).

[25] J. D. Rolleston, *The History of the Acute Exanthemata* (London, 1937), p. 49.

[26] T. C. Allbutt, *Greek Medicine in Rome* (London, 1921), p. 341. Allbutt thought that both scarlet fever and typhus were possibilities.

[27] J. F. D. Shrewsbury, 'The Plague of Athens', *Bull. Hist. Med.* 24 (1950), 1–25.

[28] Op. cit. (above, n. 22).

The measles virus attacks man only, but it may well be descended from a virus with a much wider host range. Indeed, the similarity in structure and chemical composition between the measles virus, the dog-distemper virus, and the rinderpest virus, is sufficiently close to suggest that all three have a common evolutionary origin. If this is so, the hypothetical ancestral virus might well have been capable of attacking not only man but also domestic dogs, the jackals from which many breeds of dog are derived, and perhaps even certain birds as well. It might, indeed, have produced in man a disease much more severe than present-day measles, but not wholly unlike it. Furthermore, measles cannot be a disease of great antiquity. Nineteenth-century American studies showed that it could be propagated continuously only in cities or conurbations of 300,000 inhabitants or more. In smaller communities, it died out until reintroduced from outside. At the time of the Peloponnesian War there were few centres of population of such a size. By an intriguing coincidence, 300,000 would not be an impossible estimate of the number of people gathered within the Long Walls of Athens at the time the Plague broke out. But all this is sheer speculation. Even if it could be shown (which it cannot) that an ancestor of the measles virus, with the appropriate properties, existed at that time, it would be unacceptable to call the Plague of Athens 'measles'. To do so would be to stretch the meaning of the word to an intolerable degree.

There is one plank in the platform of Shrewsbury and Page on which they have placed far more weight than it will bear. In 1875 there was an outbreak in the Fiji islands of a highly contagious disease with a high mortality. It was observed that sufferers often immersed themselves in the sea in a manner reminiscent of the Athenians plunging into water tanks. The idea that this Fijian epidemic was one of measles seems first to have been put forward by B. G. Corney,[29] a colonial surgeon appointed to the islands. Corney's account was published nine years after the event. In it he said that he had 'been able to throw together from memory a few observations'. He had been unable to consult his notes as he had left them in the Fiji islands. He had not himself observed the outbreak since he had arrived 'after its termination'. In fact, the main first-hand accounts were those of two Methodist missionaries, the Revd. J. Waterhouse and the Revd. A. J. Webb. Now, 1875 was long before the measles virus (or, indeed, any virus) had been isolated and characterized, so that there could in any case have been no certainty about the nature of the epidemic in Fiji. For what it is worth, the Revd. Mr. Waterhouse expressed the opinion that the disease was not measles. The fact that the sick Fijians took to the sea is not very helpful: such was the practice of the islanders when they felt feverish for any reason (see MacArthur[30]). All in all, there seems to be far too much doubt about what happened in Fiji in 1875 for it to be profitable to compare the outbreak with the Plague of Athens.

If the Athenian Plague has evolved into something much milder (a plausible, but, of course, unprovable assumption) there are other candidates than measles for the claim to be the modern, milder form. In the course of the supposed evolution, the symptoms and signs of the disease might well have changed substantially so that resemblances could be fortuitous and large differences would

[29] B. G. Corney. 'The behaviour of certain epidemic diseases in natives of Polynesia with especial reference to the Fiji islands' *Trans. Epidem. Soc., London*, N. S. 3 (1884), 76–95.

[30] Op. cit. (above, n. 23).

not exclude an evolutionary link. So why choose measles? Why not chicken-pox or rubella or something even less like the Plague of Athens?

(v) *Typhus fever.* This is the diagnosis preferred by Crawfurd,[31] Keil,[32] MacArthur,[33] and Ferguson.[34] It needs particularly careful examination. The case in its favour has been argued with great thoroughness, particularly by Sir Raymond Crawfurd and by Sir William MacArthur. Gomme[35] accepted this diagnosis and his conclusion is widely accepted as authoritative among classical scholars.

There are two distinct types of typhus fever: epidemic typhus, caused by *Rickettsia prowazeki*, and endemic typhus, caused by *Rickettsia mooseri.* These two organisms were not at first recognized as being distinct, but evidence for the existence of two separate species gradually accumulated during the 1920s largely through the work of Mooser, Zinsser, and their collaborators. After a group of experimental studies published in 1931 there could be no further doubt that there are two species and not one. *Rickettsia prowazeki* is spread from man to man by the body louse. Man and the body louse are its only hosts. It causes a grave disease in man and a disastrous one for the lice among whom both the infection rate and the case mortality approach 100 per cent. *Rickettsia mooseri* is spread from rat to rat by the rat flea, causing little harm to either. It is occasionally transmitted by the rat flea to people who come in contact with rats. In the individual human patient, the symptoms and signs of epidemic typhus and endemic typhus are very similar, hence the earlier confusion. Before these two species of *Rickettsia* had been distinguished, it was thought that an infection acquired by contact with rats could be propagated further through the man–louse–man cycle, but it now appears that this does not happen. Both Shrewsbury and Page based their cases against typhus in part on their belief that there were no rats in ancient Greece, a controversial question of some interest in its own right but wholly irrelevant to the question as to whether or not the Plague of Athens was typhus.

If we dismiss the rat as a red herring, a number of serious difficulties remain.

First, there is a problem about the nature of the rash. Thucydides states that the skin broke out with φλυκταίναις μικραῖς καὶ ἕλκεσιν. This is usually taken to mean 'small blisters and sores' or words to that effect. If this is what Thucydides meant, his description would fit the rash of smallpox much better than that of typhus (as Littman and Littman[36] have rightly pointed out). In typhus there are macules (red spots not raised above the skin surface) and papules (red spots slightly raised above the surface). In an attempt to get round this difficulty, MacArthur[37] observes that Thucydides 'noticed even small blisters (common in some outbreaks especially in summer)'. Commenting on MacArthur's statement, Littman and Littman wrote: 'If this is true it is most unusual. In all our reading we have never found the typhus rash associated with blisters.' Here we must agree with Littman and Littman. If the case for typhus rests in part on the assumption that we are dealing with an unusual form of the disease, it becomes very much less convincing.

[31] R. Crawfurd, *Plague and Pestilence in Literature and Art* (Oxford, 1914), pp. 23–41, 212–22.

[32] H. Keil, 'The louse in Greek antiquity', *Bull. Hist. Med.* 25 (1951), 305 ff.

[33] Op. cit. (above, n. 12).

[34] T. Ferguson, in a letter to A. W. Gomme. This letter is quoted extensively in *HCT* ii. 151–3.

[35] A. W. Gomme in *HCT* ii. 153.

[36] Op. cit. (above, n. 9).

[37] Op. cit. (above, n. 23).

Second, if the Plague of Athens was typhus there is a striking and serious omission in Thucydides' account. He says nothing of the mental symptoms which are such a prominent feature of typhus. Indeed, it is the clouding of the mind that gives the disease its name. MacArthur, challenged by Page on this point concedes that Thucydides 'does not, in so many words' refer to mental derangement. But MacArthur goes on to claim that: '"Violent convulsions" (whatever their cause) cannot occur if the brain is normal.' But does σπασμὸς ἰσχυρός mean 'violent convulsions'? Page,[38] in a rejoinder to MacArthur, having taken other medical advice, pointed out that the words 'naturally signify nothing more than "the strong muscular reaction which occurs in vomiting, rather than a subsequent convulsion"'. We agree with Page and can find nothing else in Thucydides' account that suggests that mental derangement was a general feature of the Athenian Plague. There is his statement that some suffered from a total loss of memory immediately after recovery (2.49.8), but this is not the same thing as mental disturbance at the height of the disease; indeed, MacArthur did not even mention this point.

Third, there is the serious difficulty that *Rickettsia prowazeki* can infect only man and the body louse and certainly does not attack carnivorous birds or quadrupeds or the domestic dog. Of the four authors we have cited, only Crawfurd referred to this discrepancy, and he was unable to explain it away.

Gomme quoted MacArthur as stating that 'if Thucydides had said no more than that the disease was a severe fever with gangrene of the extremities as a character, that alone would be a diagnostic of typhus.' This is an extraordinary claim. If that were *all* that Thucydides had told us, we would be able to say that there was no reason why the Plague could not have been typhus. But Thucydides told us a great deal more. And some of what he told us makes the case for typhus seem much weaker than its proponents would have their readers believe.

(vi) *Typhoid fever.* This has been mentioned as a possibility by several writers but seems to be no one's favourite apart from Grote[39] who provided no more than a few dogmatic words in a subordinate clause. Since typhus and typhoid fevers were distinguished only in the early part of the nineteenth century, it would appear on the face of it that if typhus is a possibility so is typhoid. There is, however, one overwhelming objection to the idea. Typhoid (which is caused by a bacterial species in no way related to the causal organism of typhus) is spread in a variety of ways. There is, however, only one way in which an outbreak of typhoid could have spread as extensively and rapidly as did the Plague of Athens and that is from the public water supply. Such outbreaks of typhoid can and do occur when a large population draws its water supply from a central source. The Croydon typhoid outbreak of 1937 was the most recent serious event of this kind to take place in this country. Now the water supply of Athens at that time was from many individual water tanks fed by separate springs. Consequently no major outbreak of a water-borne infection could have occurred rapidly. This seems an insuperable objection to the suggestion that the Plague of Athens was typhoid fever.

(vii) *Ergotism.* The extraordinary suggestion that the Athenian Plague was

[38] D. L. Page, 'The Plague: a lay comment on a medical note', *CQ* N. S. 4 (1954), 174.

[39] G. Grote, *A History of Greece,* new edition in 10 volumes (1888), v. 78. Grote quoted Littré in support of his statement (E. Littré, *Oeuvres complètes d'Hippocrate* (Paris, 1839), i. 122). It seems that Grote misunderstood Littré (see below, n. 50).

an outbreak of ergotism has been taken seriously by some distinguished classical scholars and for this reason only must be examined further. Finley,[40] Page,[41] and Gomme[42] were all under the mistaken impression that Kobert[43] had suggested that the Plague was a simple outbreak of ergotism.[44] So too was Williams in his 1962 paper.[45] As Page and Gomme both pointed out, it would be very difficult to believe that the Plague was not a contagious disease when Thucydides seems to make it so very plain that it was. It would be particularly hard to account for the events following Hagnon's expedition to Potidaea on any hypothesis that excluded the idea of contagion. Moreover it would be necessary to postulate a series of imports of contaminated grain to various places at various times for which there is no collateral evidence.

A paper by Salway and Dell[46] suggesting that the Plague of Athens was ergotism was published in 1955. Early on in their paper Salway and Dell considered 'the possibility that the disease may now be either extinct or so altered as to be unrecognisable' and commented: 'This is no more than distantly possible without the intervention of medicine developed to a degree that is only now being attained.' This argument cannot be accepted. Evolution of host-parasite relationships as discussed above can occur, and, indeed, has occurred, when no medical treatment of any kind has been involved. The rabbits of Australia in the 1950s certainly did not enjoy the benefits of modern medicine, and yet myxomatosis changed greatly in a decade. Indeed, modern medicine is more likely to slow down the evolutionary process by saving the lives of children and young adults who would otherwise have died, with the result that they have the opportunity to transmit to their children genes determining high susceptibility to a particular infection. Modern medicine can certainly contribute to a disease becoming extinct. For example, skerljevo, a spirochaetal disease once common in parts of Yugoslavia has been effectively exterminated since the Second World War. But infectious diseases can become extinct simply because there is an insufficient supply of susceptible hosts to allow the parasite to propagate itself. At the present day, there are small island communities where the common cold regularly dies out until reintroduced by the arrival of the next supply ship. If the world's population came to consist entirely of small, isolated communities, the common-cold viruses would rapidly become extinct. In the ancient world, with a much smaller total population, and with most people living in small,

[40] J. H. Finley, Jr. *Thucydides* (Cambridge, Massachusetts, 1942), pp. 158 f.

[41] Op. cit. (above, n. 22).

[42] A. W. Gomme in *HCT* ii. 150 f.

[43] Op. cit. (above, n. 9).

[44] The concluding sentence of the relevant section of Kobert's earlier paper is as follows: 'Ich kehre damit zu meinem Ausgangspunkte zurück, nämlich zur Deutung der im dritten Buche der Epidemieen des Hippokratischen Schriftencorpus beschriebenen Masserkrankung, und glaube durch die Analogie mit der Pest des Thucydides nachgewiesen zu haben, dass wohl beide als Blatternepidemieen bei einer an latentem Ergotismus leidenden Bevölkerung aufgefasst werden können.' Widmann (op. cit., p. 000) wrote: 'Es war eine Blatternepidemie einer an latenten Ergotismus leidenden d. h. infolge des Genusses von mutterkornhaltigem Brode (μᾶζα) vergifteten Bevolkerung . . . Kurz alle Symptome lassen sich nur durch die von Kobert gegebene Erklärung verstehen.' In 1899 Kobert published another paper in which he forcefully reiterated his previous claim, and argued his case at formidable length. Poor Kobert! Among writers on the Plague he is one of the ones most often mentioned by others, but clearly the least often read.

[45] Op. cit. (above, n. 15).

[46] P. Salway and W. Dell, 'Plague at Athens'. *G & R* 24 (1955), 62–70.

isolated groups, there would obviously have been a much greater likelihood of diseases becoming extinct in this way.

Later on, after a brief and incomplete review of previous identifications of the Plague, Salway and Dell concluded that 'the outstanding difficulty in the infectious disease theory is the fact already demonstrated that the known infectious diseases will not fit the symptoms.' Having for these reasons decided that the Plague was not an infectious disease at all, they then proceeded to argue the case for ergotism. Now it emerges with unambiguous clarity from Thucydides' account that the Plague *was* contagious and that it conferred immunity. It must therefore have been a microbial infection of some kind. No other recent writers dispute this obvious fact. If it was not a disease that we know today (and here we must agree with Salway and Dell) then the chances of its now being extinct, or so much changed as to be unrecognizable as the disease described by Thucydides are far from being 'no more than distantly possible'. They are manifestly probable.

(viii) We must also consider the possibility that the Plague was a simultaneous outbreak of two or more modern diseases. We have already mentioned concrete suggestions to this effect by Kobert and by Major. The idea has recently been revived by Longrigg.[47] The crowded conditions in Athens would certainly have favoured the spread of any highly contagious disease. We must of course remember that similar crowded conditions existed in 431 and nothing serious occurred. In 430 there had been a period of exceptional freedom from disease immediately before the Plague broke out (2.49.1). The fact that the first cases occurred at Piraeus while later cases arose in Athens itself (2.48.2) suggests a single infection in the first instance, though on the face of it there would seem to be no compelling reason why other diseases might not have supervened later.

However, no plausible combination of diseases could be convincingly reconciled with Thucydides' description taken as a whole. Bubonic plague could hardly have been one of them for reasons given above and none of the other candidates we have examined will account for the infection of other animal species. Furthermore, the multiple-infection theory has its own special difficulty which does not apply to any single-infection hypothesis. If one supposed that the Plague was really a mixture of, say, smallpox, typhus, and a severe form of measles, then recovery from one of them would have conferred no protection against the others, and second attacks of 'the Plague' would have been common and often fatal. Had this happened, Thucydides could never have concluded that there was a highly effective and specific acquired immunity (2.51.6). Indeed he would probably not have realized that any such thing as immunity occurred. The fact that he did makes it virtually certain that he was describing a single infection. Kobert's suggestion of smallpox plus ergotism is free from this objection, but is a very unlikely solution of the problem for reasons already considered.

Kobert in his 1899 paper[48] stated that it had also been suggested that the Athenian Plague was cerebro-spinal fever, and that it was influenza. These proposals do not have sufficient merit to deserve discussion. According to Keil,[49] the further

[47] We are most grateful to Mr. J. Longrigg for kindly allowing us to read a draft of his as yet unpublished article on the Plague.

[48] Op. cit. (above, n. 9).

[49] Op. cit. (above, n. 32).

suggestions that it was dengue and that it was syphilis have been made. If possible, these ideas are even less worth considering. We emphasize the qualification.

5. THUCYDIDES' CONTRIBUTIONS TO SCIENTIFIC MEDICINE

Our argument so far may be summarized as follows. Almost everything written in the past about the Plague of Athens has been dominated by the thought that it must have been some disease that we know today. Not only is this not necessarily so, it is not even particularly likely. There appear to be four formal possibilities:

(i) The Plague of Athens was indeed some disease that exists today, or a mixture of two or more such diseases. If so, on any view, Thucydides made a number of bad mistakes in his account and which modern disease (or diseases) it was cannot ever be known because of his seriously inaccurate account.

(ii) The Plague of Athens still exists in some remote part of the world, unknown to the medical profession. This is no more than a formal possibility. One cannot imagine how something so contagious and so lethal could remain either isolated or unknown under modern conditions.

(iii) The Plague of Athens is now extinct. This is quite likely. This possibility was suggested long ago by Littré[50] and recently by McNeill.[51] However, McNeill cannot be correct in suggesting further that it 'burnt itself out within a single season.' Thucydides made it quite clear (3.87.2–3) that it lasted for at least three years with a recrudescence in 427 B.C. It might well have become extinct shortly after that, because by that time almost everyone living in Attica at the outbreak of the war must have been dead or immune. Or, of course, it might have spread more widely and become extinct at some later date.

(iv) The Plague of Athens has so changed in its clinical manifestations during the past twenty-four centuries that the modern descendant cannot be recognized in Thucydides' account.

The truth, we suggest, almost certainly lies in possibility (iii) or (iv). But we can see no way of choosing between them. On either view the question: 'What was the Athenian Plague?' is in principle unanswerable if the questioner is wanting to attach to the Plague the name of some modern disease or diseases.

Although it may seem frustrating that no simple answer is available to the problem of the Plague it will be seen that there are important points to be derived from Thucydides' account which have not always been properly valued and which show his powers of observation to have been outstanding: first, the process of contagion, and second, the phenomenon of acquired immunity.[52] He further

[50] Op. cit., (above, n. 39). Littré wrote: 'C'était une fièvre éruptive, différente de la variole, et éteinte aujourd'hui.'

[51] W. H. McNeill, *Plagues and Peoples* (Oxford, 1977), p. 105.

[52] Thucydides' contributions to knowledge about each of these important general properties of infectious diseases have occasionally been noted by medical writers but do not seem to be at all well known. Thus Sir Raymond Crawfurd (op. cit. in n. 31 above) pointed out that 'Thucydides was the first of extant writers to enunciate clearly the doctrine of contagion', while of Hippocrates he rightly observed: 'Of contagion from man to man he had not the vaguest conception.' William Bulloch, in his book *The History of Bacteriology* (London, 1938) p. 6, also gives Thucydides full credit for his observations about contagion. For a recent acknowledgement of Thucydides' understanding of acquired immunity, see B. D. Davis, R. Dulbecco, H. N. Eisen, H. S. Ginsburg, and W. B. Wood, *Microbiology* (New York, 1967), p. 358.

appreciated that acquired immunity is specific, i.e. recovery from an attack of the Plague prevented (or at least reduced the severity of) further attacks of the Plague but did not protect against other diseases. That Thucydides was able to arrive at such important conclusions about the Plague in the climate of opinion prevailing during his lifetime is indeed remarkable. We must elaborate.

Nowadays the idea of contagion (that one person catches a disease from another) seems so obvious that it is hard to realize that the matter was controversial until the nineteenth century. The schools of Hippocrates and Galen attributed plagues to miasmata, i.e. poisonous air, and their authority was so powerful in the ancient and medieval worlds that as far as specifically medical writers are concerned the concept of contagion dates only from the work of Fracastoro[53] in the sixteenth century—almost 2,000 years after the time of Thucydides; and Fracastoro's ideas were not generally accepted for another four centuries. Then in the last three decades of the nineteenth century, Pasteur, Koch, and many others demonstrated that a large number of human diseases were microbial infections spread from person to person in a variety of ways, close contact with a patient being a common one. The idea of contagion rapidly became public property. Hence recent commentators on Thucydides have understandably failed to appreciate that he was saying something original.

Those who have not studied the history of medicine are apt to think that contagion is too obvious a fact of life to have been overlooked even in the most primitive cultures. But is this attitude justified? If we try to look at the problem from the standpoint of one who knows nothing of the scientific discoveries of the last century it becomes clear that the idea of contagion is by no means as obvious as it now seems. What has to be accounted for is that a number of people in a restricted area and within a restricted period of time develop the same disease. To explain how this might happen, two other theories (with many variations) have been widely supported in the past:

(i) That God is angry with the inhabitants of a particular place because they have been collectively guilty of some offence, e.g. worshipping idols (Psalm 106: 29).

(ii) That in the area concerned there are evil exhalations from the earth. As late as 1666, William Boghurst, an apothecary and one of the heroes of the great plague of London, explained its occurrence along these lines. This theory differs from the Hippocratic idea of aerial miasmata only in the supposed source of the evil influence. Akin to such theories are various ideas that outbreaks of disease are caused by astronomical events such as eclipses, planetary conjunctions, and the appearance of comets.

The divine-displeasure theory of disease now commands as little support from theologians as from medical scientists. The obvious objection to the second group of theories is that there is not and never has been any substantial body of evidence in their support. But this is not quite fair. Isolated observations must have seemed to favour such ideas. Thus, the lethal effects of the death valleys of Java are indeed due to carbon dioxide exuding from the earth. Miasmata may be figments of the Hippocratic imagination, but mosquitoes carrying malaria parasites are real enough. Simple goitre in the Peak District of Derbyshire is due to iodine deficiency in the water supply. And it is perfectly true that the Black Death followed a rare planetary conjunction. To be sure, two of the four examples just

[53] G. Fracastoro, *De Contagione* (1536).

given are not microbial infections at all, but the distinction between microbial and non-microbial diseases only began to be made a century ago and is not yet complete. It is understandable that theorists of bygone centuries should have sought a unifying theory to explain every event where a number of people became ill in a similar way in a restricted area during a limited period of time, and contagion is indeed not the explanation of every such occurrence. Moreover, until the germ theory of disease rested on a substantial body of sound experimental evidence (say eighty or ninety years ago) champions of the idea of contagion left one important matter unexplained. Something, they correctly supposed, was transmitted from one person to another. But Fracastoro's seminaria were no more objectively verifiable than Hippocrates' miasmata.

The real reason for rejecting these theories is that once it has become clear in a particular outbreak of a disease that one person is catching the illness from another, divine displeasure, evil emanations, eclipses, and the like become simply irrelevant. If *A* infects *B* who infects *C* who infects *D*, then it is pointless to look for some external influence that attacks *A*, *B*, *C*, and *D* independently.[54]

There is probably only one way in which the phenomenon of contagion could ever have been discovered: if an unprejudiced person made sufficiently extensive observations during the course of a major outbreak of a contagious disease, he could deduce the process with confidence. Thucydides did just this. He observed that those who nursed the sick and physicians attending them often contracted the disease themselves. He noticed that those who felt that the ties of friendship obliged them to visit sick friends likewise tended to get the disease. And he recorded that at the siege of Potidaea the soldiers already present caught the disease from Hagnon's forces, a most important observation in that it indicated that the Plague could be transported from place to place by people and could not be supposed to be due to a malign influence confined to Athens and its immediate neighbourhood. It seems likely that Thucydides made many more observations of this kind than he recorded, but he recorded enough to show that his ideas about how the disease spread were not a lucky guess at the truth but a sound deduction from observed facts.

Thucydides' ideas may well have developed from discussions with his contemporaries and his information about Potidaea was clearly not based on first-hand observation. But contagion was certainly not a well-known idea to Greeks at that time. One searches the Hippocratic corpus in vain for any suggestion that the authors understood contagion. Indeed, their whole attitude towards the causes of disease more or less excluded any such idea. Pericles clearly had no idea of the appalling risk involved in crowding so many people into a small space. Indeed, he disclaimed any responsibility for the Plague (cf. Thuc. 2.60.1, 61.3, 64.1–2).

Thucydides described acquired immunity and its specificity in two terse sentences in 2.51.6. Those who had the disease and recovered knew themselves to be safe; for it never attacked the same person twice, at least not with fatal results; some foolish people in this position thought that they were protected

[54] A slight qualification is needed here. *A*, *B*, *C*, and *D* may all be undernourished and living in damp, cold, and overcrowded slums, and thus the spread of infection may be facilitated. But these are complicating factors of variable importance, not prime causes of the kind of disease we are discussing. Where there is no smallpox virus there can be no smallpox, and where there are no tubercle bacilli there can be no tuberculosis, however bad the environmental conditions may be.

against other diseases as well. This last point is important since it implies that the specificity of acquired immunity was not then a matter of common knowledge. Again, this may at first sight seem surprising. Nowadays everyone knows that a child who recovers from an attack of measles will not get measles again but this will have no bearing on its liability to catch chicken-pox. Yet for most people such knowledge is accepted on authority. Few outside the ranks of the medical profession even have the opportunity to draw such conclusions from their own observations. If the medical profession does not understand acquired immunity and its specificity no one else is likely to know about these matters. In the absence of informed authority they would be only too likely to make the same mistake as Thucydides' less gifted Athenian contemporaries.

Unfortunately Thucydides' conclusions seem to have had next to no influence on his contemporaries or on those who came after. Indeed, the history of the ideas of contagion and infection in the ancient world is somewhat puzzling. Thucydides' allusion to these ideas seems to be the first clear literary testimony, since Leviticus 13–15 is probably concerned with cultic pollution, not medical. After Thucydides there are references to epidemics by various historians, for example Diodorus 14.70 (of 396 B.C.) and Livy 25.26 (of 211 B.C.). Diodorus attributed his epidemic to bad weather and miasma, as did Livy. Thus both conformed with orthodox medical theory, but both went on to say that subsequently the disease was spread by attending upon or coming in contact with the sick. So it is clear that common observation noted the occurrence of contagion. But the odd thing is that the idea of contagion made little appearance in medical literature. The pseudo-Aristotelian *Problemata* 1.7 refers to the contagiousness of the plague, but somewhat spoils the effect elsewhere (7.8) by saying that fevers are not contagious and listing three other diseases as contagious on grounds which in some cases are philosophical rather than empirical. However, this performance is superior to that of the Hippocratic corpus which, as mentioned above, does not seem to contain any reference to the possibility of contagion. A recent assertion to the contrary by Hans Diller[55] cannot be accepted. Diller wrote: 'Da [sc. in the Hippocratic corpus] ist das Phänomenon der Infektion festgehalten worden.' This assertion is based on a statement by Sigerist[56] which in turn depends on a single passage, viz. *Epidemics* 1.1. It describes a disease in Thasos which produced large swellings, particularly below the ears, and pain in the testes. This sounds very like mumps or perhaps something from which mumps is descended. The writer noted that it affected particularly youths, young men, and men in their prime, usually those who frequented the wrestling school or gymnasium. He observed that few women were affected (they would, of course, have had somewhat different symptoms). Sigerist assumed that the writer understood the significance of these circumstances, i.e. that the men who attended public gatherings were picking up an infection from other men, while the women, who did not do so, were less likely to be affected. But there is no evidence that the Hippocratic writer comprehended this. He merely reported any circumstances known to him in case they might be relevant and helpful in identifying any future occurrence. He may have thought that young and active males were particularly prone to the disease, as he did in another disease, reported in

[55] H. Diller in *Antike Medizin*, ed. H. Flashar (Darmstadt, 1971), p. 49.

[56] E. Sigerist *History of Medicine* ii (New York, 1961), 330–1.

Epidemics 1.15–16, where 'the great majority had haemorrhage, especially youths and those in the prime of life. Older people had jaundice or disordered bowels, fewer women fell ill and they died less frequently.' The places in which people fell ill are also often reported even though they can now often be said to be irrelevant, as are details of prevailing winds etc.

It almost seems as if professional medical men were the victims of their own *a priori* theorizing about the causes of illnesses and epidemics, which assigned overriding importance to climate, air, breath, and miasma, so that both Hippocrates in the *Nature of Man* 9 and Celsus in *de Medicina* 1.10 recommended that in time of pestilence one should seek a different climate and, if this cannot be done, rest as much as possible so as to breathe in less miasma. Lucretius in copying Thucydides' account of the Plague of Athens (including contagion) added a passage on the influence of climate in the Hippocratic manner (*de Rerum Natura* 6. 1296 ff.) as did Livy and Diodorus in the passages mentioned above. It seems as if the attempt to fit the observed facts into the medical picture of the causes of disease proved too much for the medical men so that they tended to bypass them.

It is to Procopius' credit that he recorded the 'unexpected' fact that his plague (a bubonic one) did *not* attack the attendants of the sick or those who buried them, as it is also to the credit of the other historians we have mentioned that they recorded the facts about contagion even though they gave way to orthodox medical opinion of the day to the extent of adding a weather report.

Eighteen centuries after the time of Thucydides the concept of contagion was hammered into the heads of physicians of the day by the grim realities of the Black Death.[57] Or at any rate into the heads of some of them, whose traditions survived long enough to be incorporated into Fracastoro's *De Contagione.* Too much should not be made of Fracastoro's influence which was largely local and waned in the following century. It is disappointing to find the great William Harvey disagreeing with him a hundred years later.[58] But if the lesson of the Athenian Plague had long been forgotten, the lesson of the Black Death precariously survived.

If the principal fruit of previous work on Thucydides' account of the Plague of Athens seems to be a confusing mass of doubt and contradiction, no one should suppose that the inability of the medical profession in the past to reach a consensus of opinion on the identity of the Plague casts doubt on Thucydides' capabilities as an observer and interpreter of passing events. On the contrary, his understanding of contagion and immunity showed remarkable insight, and even if his ideas were not entirely his own, they certainly owed nothing to contemporary

[57] We have stressed above that bubonic plague is not a contagious disease *sensu stricto* being spread to man from the rat (usually) by the rat flea. But in some outbreaks the variant form of the infection called pneumonic plague is common. This is a rapidly fatal pneumonia which readily spreads from person to person. This probably happened extensively during the Black Death. Boccaccio is very explicit in saying that person-to-person transmission occurred.

[58] W. Harvey. Letter to John Nardi of Florence, dated 30 Nov. 1653, in *The Works of William Harvey, M. D., translated from the Latin with a life of the author by Robert Willis, M. D.* (London: The Sydenham Society, 1847), p. 610. In this letter Harvey does not mention Fracastoro by name but it is clear that he was referring either to Fracastoro or to someone whose ideas were derivative from those of Fracastoro.

medical thinking. It is most unfortunate that the Hippocratic writers and their successors took no notice of Thucydides. Had they done so the subsequent history of medicine might have been very different.[59]

Trinity College, Oxford A. J. HOLLADAY

Sir William Dunn School of Pathology, Oxford J. C. F. POOLE

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