

COMMENTS ON THE ROUND TABLE *THE HISTORY OF DISEASE**

Lars-Göran Tedebrand

Professor of Historical Demography
Department of Historical Studies, Umeå University

For long, let us say up to the middle of the twentieth century, historic human disease was discussed as an objective biological phenomenon mainly analyzed by scientific physicians. During the past two decades new paradigms have renewed the history of disease. Disease in a historical context is seen not only as a pathological reality but also as a social construction affecting everyday life, mental habits, and social relations. Public health, the role of local and central authorities in the provision of health care, the professionalization of medicine, the interrelation between disease and economic, social, ideological and political change in early modern and modern Western societies have come into focus. Also the interrelation between disease and imperialism and colonialism has been discussed revealing the role played by bio-politics in economic exploitation and military oppression. Comparisons between the history of disease in Western societies with present third world conditions have been fruitful.

This round table reflects only to some extent these new tendencies. The main theme is the possibility of performing retrospective studies based on prelaboratory cause of death classifications. The papers presented, despite methodological differences, provide no doubt an important contribution to an ongoing discussion. Before going on I must add that I did not receive Marie Christine Pouchelles paper before the congress and only a short summary of Dr. Cunninghams presentation.

The physician, often a retired elderly gentleman, as a detective is well known to all of us. Did Wolfgang Amadeus Mozart die on the 5th of December 1791 out of a heart attack due to rheumatic fever? The official cause of death was «severe fever». Or did he die out of tb or from problems with his liver and kidneys? Perhaps he was murdered by Antonio Salieri?

As an historian, specialized on historical demography, I do not posses the necessary expertise to discuss retrospective cause-of-death-classifications at a theoretical level that a trained philosopher or a historian of science would consider acceptable.

* Round table organized by José Luis Peset at the 19th Internacional Congress of Historical Sciences, Oslo 6-13 August, 2000.

However it is, according to my opinion, a pity if the discussions between historical epidemiologists and historical demographers on the one side and philosophers and historians of science on the other side concerning this fundamental topic turn out to become a dialogue of the deaf. We can no doubt narrow the gap between positivist historians of disease and social constructionists or cultural relativists. I am convinced that historical demographers have a lot to learn from what representatives from these directions tell them and, perhaps, vice versa. We historians are in favour of historical reconstructions. We support ourselves from making reconstructions. Our tools, methods and results have been disputed lately. My own standpoint is quite clear. Let me quote the radical British historian Raphael Samuel, progenitor of History Workshop. In one of his last publications he warned «that the deconstructive turn in contemporary thought invited everyone to see history not as a record of the past, more or less faithful to the facts, but as an invention, or fiction, of historians themselves». Like Samuel I find this view unacceptable.

Disease in history is not only a pathological reality but also a social construction rooted in mental habits and social relations. I totally agree with Dr. Cunningham that there is a line between pre-germ-theory diagnosis and post-germ-theory diagnosis. Comparisons with the pretransitional period are extremely dangerous. In my ongoing study of leprosy in Sweden I of course have problems to compare my late 19th and early 20th century patients suffering from Hansens disease, which is a spectrum of types ranging from highly lepromatous leprosy to tuberculoid leprosy, with lepra-notations in medieval and early modern sources. Like Dr. Arrizabalago, in his paper and in his and Hendersons and Frenchs recent book, I can see problems in seeing the French disease in Renaissance Europe as roughly equivalent to syphilis.

But historical epidemiologists can not generally be accused of performing misguided scholarly enterprises when carefully using cause of death information from the late transitional and early transitional period. Let me add that the basic problem of diagnosis and certifications still exists. For instance when reviewing complete case histories against death certificates.

Having said this I agree with Dr. Cunningham that identifying diseases, in any society, is very complex. Biological criteria must be combined with social aspects. But for me the former still have primacy. I also agree that we have to apply a hermeneutic approach when discussing pre-laboratory classifications but my standpoint is not as radical as Cunninghams.

A history of infectious diseases which exclude the period before the 1880's is an incomplete history to me although I can see the points in the logical arguments raised by Cunningham and Dr. Hordern for this chronological restriction.

According to my mind we have to periodicize the pre-laboratory classifications.

Natural criteria are: who made the classifications and what types of diseases are we studying. Dr. Arrizabalago rightly states that «The specific nature of disease at

issue must be taken into consideration». We must, according to my mind, be careful not to throw the baby out with the bathwater.

The task of selecting the principle cause of death is a problem that has followed cause-of-death statistics since its beginning. The difficulties become very obvious when a combination of concurrent and non-interdependent causes of death has been identified. In Sweden during the earliest compilations of cause-of-death statistics, 1749-1830, the selection of the principal cause of death was left entirely to the certifier, usually a clergyman. They were given training in pastoral medicine for this purpose. The training, advocated by Carl von Linné for instance, was of a relative high standard and was provided in the faculties of theology. Moreover, the clergy acquired considerable empirical knowledge by meeting their parishioners regularly. Pulmonary tuberculosis is a disease with relatively clear symptoms in its final stages. Its «timetable» was well known, and the clergy could distinguish between its different phases. The same can be said about some other major infectious diseases.

There is also an important difference in prelaboratory cause-of-death classifications made by priests and those made by physicians. In urban Sweden this line can be dated back to 1860. On the certificates, designed on the model developed in Great Britain, a distinction was made between primary and secondary cause of death. In 1892 it was changed to «principle cause of death» and «contributing causes of death».

A number of studies during the late twenty years have given sharper contours and even modified Abdel Omran's well-known transition theory from 1971 stating among other things that pandemics of infection gradually were displaced by degenerative diseases as the chief form of morbidity and primary cause of death.

By using the rich Swedish data Peter Sköld can study the decline and disappearance of smallpox in Sweden at a level of analysis not possible in other countries. The role of the strong state for the introduction of the compulsory vaccination is evident. Vaccination became compulsory much earlier than in the homeland of the inventor of the method. The overall restriction of smallpox to children, except among the Saami population in northern Sweden, is surprising. Peter also shows that the decline in smallpox mortality during the latter part of the eighteenth century was not paralleled by a decline in the virulence of the disease.

The role of inoculation, often discussed but in earlier research never quantified due to source deficiency, played obviously only a marginal role for the reduction of smallpox mortality. The method was monopolized by physicians and seem to have been most popular in remote areas. But it is a pity that the otherwise rich Swedish sources do not permit a definite estimation of inoculation practices.

Sköld's most interesting results derive from his use of parish registers for studying cultural, mental, demographic and social consequences of the disease, consequences seldom examined in any detail in previous literature. Some of the findings are still somewhat tentative and need to be tested on larger parish samples. By com-

paring the inoculated, the vaccinated, those previously infected, and those who had escaped the disease, Peter achieves results which have significant implications for historical demographic research. Is it in a broader European perspective possible to reach more firm conclusions about the interrelation between smallpox and the drop of marriage age?

CONCLUSION

The history of disease must be studied in a sensitive and multidisciplinary way. The recent breakthrough within genetics can lead to simplifications as far as the relation between man and biology is concerned. We must be on our guard against such a backlash. But it can also lead to fascinating research about the structure of gene pools as well as the origins, levels and basic characteristics of hereditary disorders solving the problems of many diseases.