

CHANGING DISEASE IDENTITIES: CRETINISM, POLITICS AND SURGERY (1844–1892)

by

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In 1883 the Swiss surgeon and later Nobel laureate Theodor Kocher (1841–1917) made a journey to the Valais and Bernese Oberland. The purpose of the expedition was to see cases of cretinism which occurred in this area. Earlier in the same year Kocher had noticed that he had inadvertently created “artificial cretins” by total ablation of the thyroid gland. His objective now was to see whether there were any pathological changes of the thyroid gland in “naturally” occurring cretinism too. Kocher made the local authorities show him all known cases of cretinism at the localities he visited. He was not, however, very happy with what they presented to him as cretinism. Out of thirty-two alleged cretins he accepted only six as being genuine cases. Kocher was confronted with the same problem when he checked the literature: admittedly many of his predecessors had been excellent observers, but they all had mixed up cretinism with other, different diseases. Therefore, there was a need to redefine the disorder. The criteria for this redefinition must be the true cause of the disease discovered by Kocher: the affection of the thyroid gland which is the *conditio sine qua non* of the development of genuine cretinism, its specific cause.¹

So if we follow this account, in 1883 Kocher found out the cause of cretinism, a discovery officially sealed by his Nobel Prize of 1909. But, as we have seen, cretinism was a different disease after its cause had been discovered. So what did Kocher mean when he called a disease “cretinism”? What had his predecessors meant when they used the term? And what do we today mean when we speak about the history of cretinism?

Writers on the history of medicine often assume a simple continuity of the identity of diseases, conceiving them as unproblematic natural entities which have a manifestly obvious character. Thus the historian’s task is to indicate how diseases were described wrongly or partially until a significant individual discovered the truth about them and

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¹ Theodor Kocher, ‘Zur Verhütung des Cretinismus und cretinoider Zustände nach neuen Forschungen’, *Deutsche Zeitschrift für Chirurgie*, 1892, 34: 556–626, pp. 556–8, 560–71, 595–9. On Kocher see Ulrich Tröhler, *Der Nobelpreisträger Theodor Kocher. Auf dem Weg zur physiologischen Chirurgie*, Basel, Birkhäuser, 1984.

provided us with correct and definitive descriptions.² Part of this approach is the desire to name past diseases by reconstructing historical accounts of them in terms of contemporary biomedical knowledge. This entails a claim about identity, namely, as Andrew Cunningham has put it, “that disease X in the past *was the same as* (or was identical to) disease Y in the present”.³ The identity of a disease is above all constituted by the attribution of ill persons, “cases”, to a disease entity, thus giving a name to the state of ill health.⁴ Looking at the problem in this way has general implications on how we conceive the reality of diseases. In the words of Charles Rosenberg, it is only by perceiving, naming, and responding to a disease that we agree that it exists.⁵ Dealing with the identity of diseases is not merely of academical interest: putting a name to the patient’s pain and discomfort is an essential aspect of the healer’s social role. It makes a disease more manageable, emotionally and socially, than a mysterious and unpredictable affliction. Naming and classifying individual diseases inevitably includes an explanatory component and ultimately guides both the physician’s treatment and the expectations of all who are involved.⁶ The legitimacy of access to health care as well as therapeutics are organized around diagnostic decisions; “disease concepts” (which, as we will see, are inextricably connected with disease identity) “imply, constrain, and legitimate individual behaviours and public policy”.⁷

Obviously the identities of diseases are a crucial problem for anyone looking into the mechanisms that underlie change in mortality and morbidity in history.⁸ Social historians who are interested in the relations between knowledge, the professions, and social power have looked upon disease definitions and explanations as tools of social control, as labels for deviance, and as a rationale for the legitimation of status relationships.⁹

The majority of those who have challenged the view of continuous and self-evident disease identities have largely focused on instances that are untypical of modern biomedicine: either they have looked at disease entities that are wrong by the prevailing standards of medical science, or at “soft” disease entities, diagnoses which are conspicuously resonant with culture and in which a biopathological mechanism is either

² See Andrew Cunningham, ‘Transforming plague. The laboratory and the identity of infectious disease’, in Andrew Cunningham and Perry Williams (eds), *The laboratory revolution in medicine*, Cambridge University Press, 1992, pp. 209–43; and Christopher Lawrence, ‘“Definite and material”: coronary thrombosis and cardiologists in the 1920s’, in Charles E. Rosenberg and Janet Golden (eds), *Framing disease. Studies in cultural history*, New Brunswick, NJ, Rutgers University Press, 1992, especially p. 52. Peter Wright and Andrew Treacher, ‘Introduction’, in *idem* (eds), *The problem of medical knowledge. Examining the social construction of medicine*, Edinburgh University Press, 1982, especially pp. 8–9.

³ Cunningham, op. cit., note 2 above p. 210.

⁴ This corresponds to Andrew Cunningham’s “operational definition”, *ibid.*, p. 213. In this paper the terms “disease identity” and “disease entity” are used according to this notion.

⁵ Charles E. Rosenberg, ‘Introduction’, in Rosenberg and Golden, op. cit., note 2 above, p. iii.

⁶ *Ibid.*, pp. xvii–xviii.

⁷ *Ibid.*, p. xiv.

⁸ *Ibid.*, p. xv. Some historical epidemiologists have acknowledged changes in disease perception as a challenge, see e.g.: Jan Brügelmann, ‘Der Blick des Arztes auf die Krankheit im Alltag 1779–1850. Medizinische Topographien als Quelle für die Sozialgeschichte des Gesundheitswesens’, PhD thesis, Free University Berlin, 1982, especially pp. 65–71, 242–85; Öivind Larsen, ‘Die Krankheitsauffassungen und ihre historische Interpretation’, in Arthur E. Imhof (ed.), *Mensch und Gesundheit in der Geschichte*, Husum, Matthiesen, 1980, pp. 45–58. See also Jens Lachmund and Gunnar Stollberg, ‘Introduction’, in *idem* (eds), *The social construction of illness*, Beiheft 1, Medizin, Gesellschaft und Geschichte, Stuttgart, Franz Steiner, 1992, p. 12.

⁹ Rosenberg, op. cit., note 5 above, pp. xv–xvi. See, e.g., Wright and Treacher, op. cit., note 2 above.

unproven or unprovable, such as psychiatric diseases, psychosomatic disorders, or such “medicalized” issues as homosexuality and gender identity.¹⁰ “Hard” disease entities, such as infectious disease or endocrinological disorders (to which modern cretinism belongs), are only rarely looked upon as a product of human agency. Usually they are conceived as discovered, not constructed, based on nature not on culture.¹¹

In this paper I will contrast two identities of cretinism.¹² I will look at the process of disease definition, its preconditions and consequences.¹³ One of these disease identities is obsolete by our present standards, whereas the other is still valid today. We will see that if we apply the same criteria to both notions, neither of them can be conceived as being more independent of its social and cultural setting, more “natural”, than the other. The first section will be about the definition of cretinism as an “endemic” disease, the dominant concept up to Kocher’s time. The term “endemic” is used not in the modern sense of spatial distribution of disease, but according to the usage of the first half of the nineteenth century as a classificatory and causal category. The second identity of cretinism considered is our modern notion of cretinism as a failure of thyroid function. Of special interest for the redefinition process is an article Kocher wrote in 1892. Here he explicitly redefined cretinism, contrasting his notion of the disease with the preceding one. This section (II) includes a brief outline of the development from 1883 to 1892, when in a complex process of negotiation involving different medical and scientific communities in different countries a consensus on the notion of cretinism was achieved. Since the main subject of this paper is the identity of disease, space prohibits a more detailed investigation of this process. In the concluding section I will elucidate some of the further implications of the identity of disease and its discontinuity, finally suggesting a general way of dealing with disease identities in historiography.

Apart from these considerations, the redefinition of cretinism as an organ disease is of particular interest for the history of two other issues. Kocher’s work on the thyroid gland contributed much to the acceptance of the concept of internal secretion and was thus a crucial step in the development of modern endocrinology.¹⁴ Redefining cretinism as an organ disease was part of a process that led to a more localistic, organ-centred view in medicine, a view that made organ transplantation conceivable and desirable: on the basis of his new concept, Kocher was the first to transplant an organ, the thyroid gland, in order to cure a complex internal disease¹⁵ and thus invented organ transplantation in the modern sense.

¹⁰ Rosenberg, *op. cit.*, note 5 above, p. xv. For some of the classics of this genre see *ibid.*, p. xxv, note 9. Cf. Cunningham, *op. cit.*, note 2 above, pp. 212–13; for examples see *ibid.*, p. 213, note 5.

¹¹ Lachmund and Stollberg, *op. cit.*, note 8 above, p. 13.

¹² The changing concepts on goitre are closely related to those of cretinism and could be investigated along the same lines, as Bernardino Fantini has kindly pointed out to me. I will, however, focus on cretinism, as the conceptualization of goitre differed from that of cretinism in some aspects.

¹³ Cf. Rosenberg’s proposal to look at the actual process of disease definition, which, as he regrets, has often been lost sight of, and at “the consequences of such definitions in the lives of individuals, in the making and discussion of public policy, and the structuring of medical care”, Rosenberg, *op. cit.*, note 5 above, p. xvi.

¹⁴ See, e.g., Merrileys Borell, ‘Origins of the hormone concept: internal secretions and physiological research, 1889–1905’, PhD thesis, Yale University, 1976, pp. 41–6, 58–60, 68–81, 95–9.

¹⁵ Kocher’s priority in thyroid transplantation has been established by Tröhler, *op. cit.*, note 1 above, pp. 132–3. The relationship to organ transplantation in general is the subject of work in progress.

I. CRETINISM AS AN ENDEMIC DISEASE

Approach and Disease Definition

As a typical example of the approach to cretinism prior to Kocher's work I have chosen that proposed by Carl Rösch (1808–1866) and Johann Jacob Maffei in 1844.¹⁶ Kocher referred to them as important predecessors and they were frequently quoted in the pertinent literature of the period. Rösch and Maffei wrote two treatises but presented them as parts of one project—they were published as two volumes of one work—and despite some minor differences, their approach and overall conception were the same.¹⁷

For Rösch and Maffei, as physicians who practised in areas where cretinism occurred, the obvious thing to do was to go out, look at the cretins, speak with the local people, write down, collect, compare and select the data obtained—a procedure reminiscent of the expeditions of contemporary explorers and naturalists. To these practical options we must add intellectual options: Maffei, at the beginning of his treatise, claimed that in order to find out the causes of cretinism “calm and continuous experience” of “many truthful physicians” was necessary, “without”, as he stressed, “any mania for systematizing”.¹⁸ Generalizations must be made very cautiously. Rösch's and Maffei's pronounced empiricism was part of the empirical turn of the medical and scientific discourse in German speaking countries which had followed on a period of more theoretical interest, usually referred to as “Romanticism” or “Naturphilosophie”.¹⁹ Though the empiricists reacted to what they felt to be an excess of speculative theorizing on the part of the romanticists, they nevertheless took their concepts as the basis for constructing their own, new ideas. Similarly Rösch and Maffei built their concepts upon the ideas and methods which their predecessors had put at their disposal. That cretinism was perceived as a medical problem at all, and, moreover, as a problem that could be solved, was a legacy of their predecessors, especially the Swiss romanticist physician Ignaz Paul Vital Troxler (1780–1866). Though Maffei had an intense dispute with Troxler, Rösch and Maffei nevertheless quoted him on the title page of their book and in the general introduction as a positive example. With regard to the identity of disease this means that only their inheritance from others of a named disease entity, “cretinism”, enabled Rösch and Maffei

¹⁶ This choice does not preclude any claim of priority for Rösch and Maffei. Rösch and Maffei referred to similar concepts by earlier investigators and their own concept represents a typical way of approaching the problem during that period. For good surveys see Sigmund Bornhauser, *Geschichte der Schilddrüsen- und Kropfforschung im 19. Jahrhundert*, vol. 14, Veröffentlichungen der Schweizerischen Gesellschaft für Geschichte der Medizin und der Naturwissenschaften, Aarau, H. R. Sauerländer & Co., 1951; and Heinrich Mauri, ‘Die Entwicklung des Kretinenproblems. Die Kretinismusforschung in der 1. Hälfte des 19. Jahrhunderts und ihre Auswirkungen auf die Schwachsinnigenfürsorge’, MD thesis, University of Freiburg im Breisgau, 1976.

¹⁷ Johann Jacob Maffei and Carl Rösch (eds), *Neue Untersuchungen über den Kretinismus*, Erlangen, Ferdinand Enke, 1844; vol. 1: Carl Rösch, *Untersuchungen über den Kretinismus in Württemberg*; vol. 2: Johann Jacob Maffei, *Der Kretinismus in den Norischen Alpen*.

¹⁸ Maffei, op. cit., note 17 above, p. 1.

¹⁹ See *ibid.*, pp. 136–41. On “romantic” science and medicine, see Brigitte Lohff, *Die Suche nach der Wissenschaftlichkeit der Physiologie in der Zeit der Romantik*, vol. 17, *Medizin in Geschichte und Kultur*, Stuttgart, New York, Gustav Fischer Verlag, 1990; see also Andrew Cunningham and Nicholas Jardine (eds), *Romanticism and the sciences*, Cambridge University Press, 1990.

to redefine it according to their own criteria.²⁰ Rösch and Maffei's conception shared many characteristics with what has been described as Johann Lukas Schönlein's (1793–1864) "school of natural history" in medicine. The programmatic aim of those who shared this approach was to define a disease entity that brought together aetiology, disease process, pathophysiological mechanisms, observable symptoms, and therapy in a rational way.²¹ To achieve this, disease, like a natural object, could be empirically investigated, employing a whole range of methods of gaining knowledge. These included diligent post-mortem examinations, the results of which, like any other observation, might be used to find out more about the pathological processes occurring in the sick body.²² But pathological anatomy and physiology have no particular explanative power concerning the cause of disease. According to Maffei, products of the disease, such as anatomical changes, or, more pathophysiological, conditions like a lack of vital force or an imbalance of the humours must not be taken as causes. For causal explanations one has to look outside the cretinous subject.²³ In the hierarchy of causes traditionally applied to medical problems—a hierarchy that ranged from *predisposing*, over *external* and *antecedent* to *immediate* causes—the emphasis was laid on the first two categories.²⁴

A way to arrive at disease entities that reflect the "nature" of disease²⁵ is, by analogy with the comparison of specimens in botany or zoology, the comparison of clinical pictures. Rösch and Maffei did this with cretinism, rickets, and scrofula, explicitly drawing the analogy with species of animals or plants.²⁶ Though acting as if cretinism was a botanical species, Maffei did not fail to emphasize that this was *only an abstraction*. Each cretin is an individual person with an individual disease, he stated. Of these individual diseases the typical, the "pathognomonical" signs must be determined and distinguished from other, coincidental symptoms occurring in the same patient. This must be done without bias, without having a "prototype" of cretinism in mind.²⁷

In terms of our modern understanding, Rösch and Maffei described a number of different diseases, including Down's Syndrome and epilepsy, among them cases which look like modern cretinism. The disease entities of both authors are thus broader than modern cretinism, although Maffei's were narrower than Rösch's. Rösch included different kinds of imbecility, goitre (as a possible preliminary stage), stunted physical

²⁰ Rösch and Maffei dealt extensively with the concepts of their predecessors. For a survey of these see Bornhauser, op. cit., note 16 above, pp. 7–39.

²¹ See, e.g., Maffei, op. cit., note 17 above, pp. 175–6. The "natural historical school" in the medicine of the first half of the nineteenth century must not be confused with natural history of the preceding century. On Schönlein's method of "natural history" in medicine see Johanna Bleker, *Die Naturhistorische Schule 1825–1845. Ein Beitrag zur Geschichte der klinischen Medizin in Deutschland*, vol. 13, *Medizin in Geschichte und Kultur*, Stuttgart, New York, Gustav Fischer Verlag, 1981, especially pp. 43–80; from a more general perspective see Volker Hess, 'Von der semiotischen zur diagnostischen Medizin. Die Entstehung der klinischen Methode zwischen 1750 und 1850', MD thesis, Free University of Berlin, 1992.

²² See, e.g., Rösch, op. cit., note 17 above, pp. 148–9, 164–6, 188–9. Cf. Hess, op. cit., note 21 above, pp. 203–5.

²³ Maffei, op. cit., note 17 above, pp. 135–41, 196–9. Cf. the same attitude in Schönlein's approach, Bleker, op. cit., note 21 above, pp. 56–7.

²⁴ Cf. Cunningham, op. cit., note 2 above, p. 221. On different kinds of causality in medicine see also Lester S. King, *Medical thinking. A historical preface*, Princeton University Press, 1982, chs 9 and 10.

²⁵ This was the unanimous aim of many investigators at that time, cf. Hess, op. cit., note 21 above, p. 187.

²⁶ Rösch, op. cit., note 17 above, pp. 184–91; Maffei, op. cit., note 17 above, p. 175. For the use of analogy to botanical and zoological species in the "school of natural history", see Bleker, op. cit., note 21 above, pp. 53–7.

²⁷ Maffei, op. cit., note 17 above, pp. 194–5.

growth, blunting of the senses, albinism, cataract and other ophthalmic disorders, and certain kinds of deafness.²⁸ Similarly, a considerable proportion of Maffei's cases would not be diagnosed as cretinism today.²⁹

The broadness of the disease entity was justified as a consequence of the empirical programme: only a complete registration of all forms of the object allowed a truly natural classification of cretinism and its sub-species. The basis for this was not one single arbitrary symptom, as in the "artificial systems" of the eighteenth century, but the totality of symptoms. Completeness was the basis on which the characteristic symptoms were to be determined.³⁰ Any phenomenon observed must be registered, even if the causal connection to the disease was not obvious as yet. The descriptions of the cases were detailed and extensive.³¹ They included not only physical details but also those of behaviour or clothing. Case histories here seem to have had an analogous function to specimens in botany. Maffei wrote that after having seen several hundreds of cretins he was not in a position to assume a constant "prototype" of cretinism. Instead of this, one could find a continuum of a greater or lesser degree of stupidity and degeneration of the body. In the end there were only a few constant pathognomonic symptoms left: a more or less pronounced lack of common sense and reason plus a sluggish posture and a shuffling clumsy way of walking, which, however, was not necessarily always present.³²

But how to decide then whether a case belonged to cretinism or not? The result of the abstraction from the described symptoms could not provide an answer because, by definition, the characteristic symptoms were not yet known. So in practice Rösch and Maffei used another criterion to decide. Proceeding from the supposition that the terrain moulds the people living in it, and that it has a strong influence on the physical constitution and the illnesses of its inhabitants, the place where the disease occurred was regarded as crucial.³³ The criterion of locality was applied in several instances: Rösch included albinism in cretinism, provided it occurred in the same areas as cretinism did. Also certain cases of deaf-muteness and imbecility were included solely on this basis.³⁴ Concerning reports about cretins in the plains—i.e. outside the endemic areas—Maffei conceded that these cases might resemble cretinism, but should not be classified as such, they "have their roots in a completely different soil".³⁵ So in contrast to the programme, the place, not the clinical picture, was the basis for constituting the disease entity. This is also reflected in the definition Maffei gave in his treatise: cretinism was "a distinct, chronic, inborn or acquired disease of the whole body, engendered by endemic influences, occurring exclusively in the Alpine chain of mountains, characterized by a lack of general common

²⁸ Rösch, *op. cit.*, note 17 above, pp. 1–2, for details see pp. 145–78.

²⁹ Maffei, *op. cit.*, note 17 above, pp. 5–9. He explicitly excludes some of Rösch's categories from cretinism, see p. 139. See also Maffei's list of differential diagnoses, *ibid.*, pp. 126–34. This broadness is typical of that time, cf. Mauri, *op. cit.*, note 16 above, pp. 74–84.

³⁰ Maffei, *op. cit.*, note 17 above, pp. 5–9. For parallels to Schönlein's school, see Bleker, *op. cit.*, note 21 above, pp. 24–6, 53–7, 72–80.

³¹ Rösch, *op. cit.*, note 17 above, pp. 131–89. Maffei, *op. cit.*, note 17 above, pp. 9–61.

³² *Ibid.*, pp. 91, 118, 134–5.

³³ *Ibid.*, pp. 5–9; Rösch, *op. cit.*, note 17 above, p. 131. Individual diagnosis was not only a problem for Rösch and Maffei, cf. Mauri, *op. cit.*, note 16 above, p. 76.

³⁴ Rösch, *op. cit.*, note 17 above, p. 142, 147.

³⁵ Maffei, *op. cit.*, note 17 above, p. 156.

sense, a lack of articulate human language, and by a certain expression of stupidity or brutality in the faces of those affected”.³⁶

Causes of Cretinism

K. Codell Carter has claimed that the concept of specific causes is a prominent characteristic of modern western medical thought. To be specific for a certain phenomenon, a cause must be sufficient, that is, it must be able to bring about the phenomenon, and it must be necessary; that is, without it, the phenomenon does not occur. Although the historical actors almost never used these terms themselves, analysing the structure of argument along these lines can help in distinguishing different approaches.³⁷

Rösch and Maffei collected numerous different causes. The point that linked them all was the place of occurrence, the so-called endemic condition (“Endemie”), which was inextricably connected with certain places. Rösch and Maffei presented the endemic condition as a factor without which cretinism did not occur. This looks very much like a necessary cause.

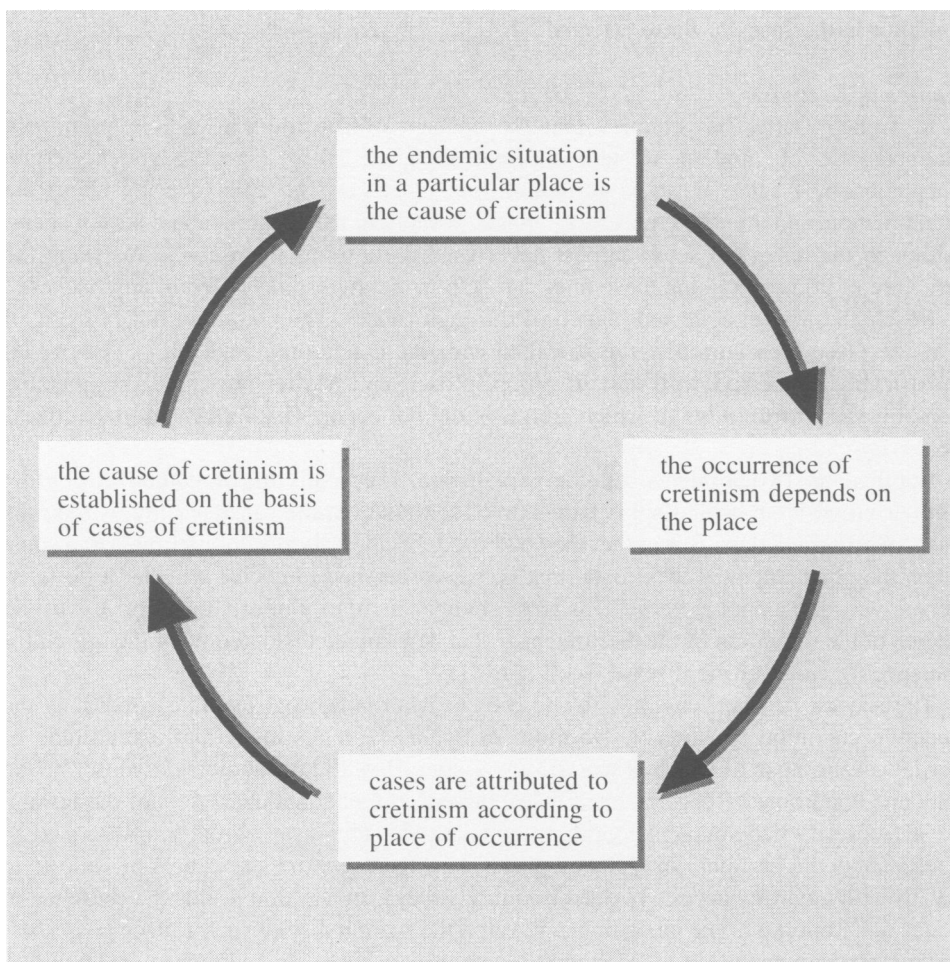
But there are two problems with this explanation. The first is one of method. According to their programme, the authors claimed to have found out the cause of cretinism on the basis of empirical data. But in fact they had used the alleged cause of cretinism at a stage when they had collected these data, that is, when they had to decide whether a diseased person was or was not a cretin. This is an obvious circularity of argument. So it was the choice of the definition of the disease entity that determined the outcome of the search for the specific cause of the disease (see Figure 1).

The second problem emerges if one tries to determine what tangible conditions the endemic condition consists of. On close examination, it dissolves into a multitude of partial causes, none of which is specific any longer. How did the authors circumscribe the endemic conditions? Rösch claimed that it depended on certain characters of the terrain, its altitude, its geographical longitude and latitude, the form of the landscape etc.³⁸ Cretinism could be found in secluded places, situated in narrow curvatures of valleys, in basins with a narrow access, in deep tributary valleys. In addition to this the presence of water was important. The more stagnant water, the more moisture such a place possessed, the more “conjunctures” for the cretinoid nature were given. In addition to air humidity, frequent sudden changes of temperature were typical in places where cretinism occurred. This description of the preconditions of cretinism implies that a specific cause had been determined—an impression that is quickly dissolved by a closer look: what Rösch described was only a “relative” necessity. There were cases in which only some of the conditions described led to cretinism. The factors did not need to be present in their maximum degree, their effect depended on how they combined with each other and with diverse non-endemic, “occasional” causes. The higher the number of causal influences present, the higher the incidence, and vice versa. So in the extreme—in the case of “sporadic cretinism”—the necessary cause was not present at all, and the occasional

³⁶ *Ibid.*, p. 202.

³⁷ K. Codell Carter, ‘The development of Pasteur’s concept of disease causation and the emergence of specific causes in nineteenth-century medicine’, *Bull. Hist. Méd.*, 1991, 65: 528–48, on pp. 528–9.

³⁸ Rösch, *op. cit.*, note 17 above, pp. 192–221.



causes alone brought about cretinism. So the conditions described by Rösch were only more or less necessary, and thus not necessary at all.

Sufficiency too was only very weak: secluded, moist air, subjected to frequent and considerable changes of temperature, was only one causative factor. The problem is that—owing to his rich erudition and experience—Rösch knew valleys where all these conditions existed—without cretinism occurring in them. So this partial cause was not strictly sufficient. It had to be supplemented with an additional cause, e.g., social conditions, poverty, lack of education, bad housing, bad clothing, bad feeding, diminished electricity of the air. In the course of his arguments Rösch constantly added factors, without excluding any of them: thus he wrote that people said the mere sight of a cretin to a pregnant woman made the unborn child cretinous as well. For Rösch this was bizarre

and superstitious. On the other hand, he could not exclude the possibility that under certain circumstances it might actually be true.³⁹

So what at first sight seemed to be a specific cause turns out to be neither really necessary, nor strictly sufficient. The difficulties involved in grasping the endemic situation show even more clearly when we look at Maffei's account.⁴⁰ Maffei claimed that cretinism occurred only at altitudes between 1,300 and 3,400 feet, where a typical physiognomy of the inhabitants prevails. The fact that cretinism did not occur in all mountainous areas he could, like Rösch, explain by adding to the predisposing causes of the terrain, occasional causes which must join the others to convert the ailment from "possibility to reality",⁴¹ as he put it.

What, therefore, did the special character of the endemic area consist of? Unlike Rösch, Maffei did not focus on one necessary cause. His experiences did not permit this. Although he stated that one influence was crucial, he could always bring forward another example of cretinism having occurred without this factor. To explain this, a new factor had to be found, and was then added to the list.⁴² To explain cretinism's basic cause Maffei enumerated all the circumstances he had observed in the endemic areas without being able to evaluate them or to exclude some of them as being not pertinent to the problem.⁴³ All the differences between the mountains and the plains were included: plants, animals, diet, clothing, housing, occupation, climate, etc., etc. No one single factor was strictly necessary (apart from the altitude which was, however, the common factor he set out to explain in the first place).⁴⁴ As the list grew longer and longer certainty about the cause diminished. One could never know beforehand which detail might be important in an individual case. So the better the observation—the less certain the knowledge.⁴⁵

This is how Rösch and Maffei came to collect the multitude of facts that amaze the modern reader. Taking up the empiricist tradition of medical topography⁴⁶ they gave a detailed account of the places where cretinism was prevalent.⁴⁷ These included geographical characteristics, geology, vegetation, agriculture, population density, climate, drinking water, type of buildings, roads, bridges, housing, and clothing. The inhabitants were characterized according to their descent, occupation, social conditions, diet, cleanliness, morals, customs, religion, physical and psychological constitution and prevailing diseases. The aim was a complete description; any detail might be important.

Based on all his empirical data, Maffei's conclusion was that only the totality of influences from the endemic area could be a necessary cause, the influences themselves could not be analysed into single causes (see Figure 2). Cretinism is a product of a "bundle

³⁹ Ibid., pp. 204–6. This theme occurred frequently in treatises on cretinism at that time, cf. Mauri, op. cit., note 16 above, pp. 52–4.

⁴⁰ On the causes of cretinism, see Maffei, op. cit., note 17 above, pp. 135–201.

⁴¹ Ibid., p. 8.

⁴² See, e.g., ibid., pp. 152–4.

⁴³ See ibid., e.g., pp. 120–2. On p. 122 he claims anything that exerts an influence on the child as a possible occasional cause.

⁴⁴ Ibid., pp. 177–8.

⁴⁵ Ibid., p. 133. On similar problems of the French hygienists before Pasteur, see Bruno Latour, *The Pasteurization of France*, Cambridge, Mass., Harvard University Press, 1988, pp. 20–1.

⁴⁶ On this genre with its connection with medical science and public health, see Brügelmann, op. cit., note 8 above. For connections to Schönlein's approach see ibid., pp. 161–79.

⁴⁷ Rösch, op. cit., note 17 above, pp. 35–130. Maffei's topographical section is shorter (op. cit., note 17 above, pp. 1–9), he gives most of the details with his case histories.

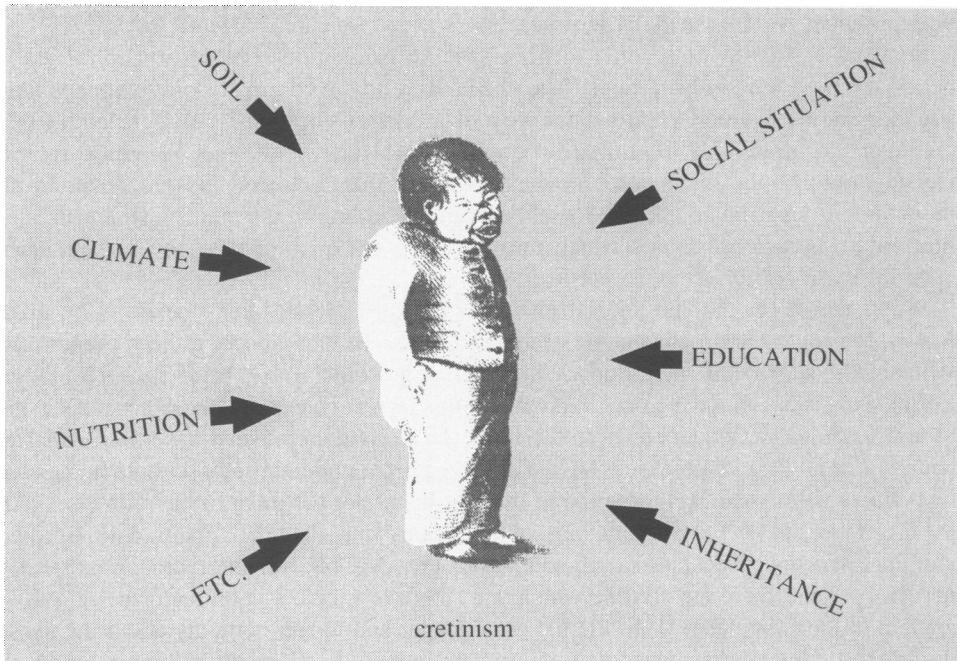


Figure 2: Causes of cretinism as an “endemic” disease. (The little cretin is part of an illustration in Rudolph Virchow, ‘Über die Physiognomie der Kretinen’, *Verhandlungen der physikalisch-medizinischen Gesellschaft in Würzburg*, 1957, 7: 199–213, after: F. Merke, *Geschichte und Ikonographie des endemischen Kropfes und Kretinismus*, Bern, Hans Huber, 1971, p. 243.)

of causes laid down at a certain place”.⁴⁸ Maffei conceded that the results of his detailed and laborious empirical investigations were not very satisfying. No cause could be singled out, the single endemic influences were almost innumerable, it was absolutely impossible to know all the endemic influences and combinations among them, so nobody could explain with certainty how cretinism came about.⁴⁹

Therapy and prevention—politics

The pronounced aim of all the descriptions was to increase knowledge about ways to prevent and cure cretinism.⁵⁰ What was written on therapy, however, is exceedingly brief. Treatment and prevention of cretinism did not fall within the scope of Maffei’s treatise at all (which was designed to be the first volume of a larger work). Rösch dedicated a mere twelve pages out of 234 to this point.⁵¹

The most radical prophylaxis⁵² would have been for the patients to leave the places where the endemic condition prevailed. As this was not practicable, elements of the

⁴⁸ Maffei, op. cit., note 17 above, pp. 119–20.

⁴⁹ Ibid., pp. 194–6.

⁵⁰ See, e.g., ibid., p. 8.

⁵¹ Rösch, op. cit., note 17 above, pp 222–34.

⁵² On prophylaxis see ibid., pp. 222–9.

endemic condition had to be tackled individually, as well as the occasional causes which acted together with the endemic condition to bring about the ailment. This left a host of partial causes, none of which was necessary or strictly sufficient. As far as the improvement of the areas affected by cretinism was concerned, Rösch appealed for a number of public measures: draining of the soil, regulation of the course of rivers and streams, maintenance of clean streets in the towns, building of new and better roads. Detailed building regulations should be enacted: houses should be built on higher terrain, not in basins and low places, if necessary on a high terrace, not beside water, sleeping rooms must not be situated above stables, etc. The old city walls and gates should be demolished. High trees with thick foliage must not be allowed near residential buildings; in places where there were forests situated near settlements, the trees must be cut down. Drinking water of high quality had to be provided. The consumption of alcohol must be strictly limited. As Rösch ascribed a role to inheritance as well, he pleaded for strict regulations concerning marriage. And there was also a social dimension to the fight against cretinism: as Rösch noted, large parts of the population could not afford proper nutrition. The peasants, for instance, had not enough land to support themselves. In order to stop cretinism the state must do something to remedy this.⁵³ The kind of empirical medicine propagated in the mid-nineteenth century was especially suitable for a perception of the social conditions of illness,⁵⁴ which in turn could result in political demands. It is indicative of his political sympathies that Rösch mentioned a place where cretinism had been frequent “before the revolution”, but once the poor had their own land and were much better off, cretinism decreased dramatically.⁵⁵ Rösch not only hinted at the social and political implications of this problem, he also suggested what the state should do: the authorities must organize and run programmes of emigration from poor and populous regions; taxes, tithes, tributes to landlords must be abolished; new roads and bridges must be built by the state to stimulate trade and the economy; improvement of agriculture must be initiated and supported; new industries must be established.

Rösch's demands were detailed, far-reaching, expensive, and they were political. He was not only a philanthropist and the president of a temperance society (hence his point of limiting alcohol consumption),⁵⁶ but also a revolutionary who took part in the 1848 upheavals in Germany. After the revolution had been suppressed, the authorities removed him from his professional positions and he fled to North America, where he died in 1866.⁵⁷ So Rösch's work in medicine cannot be separated from his political programme; both aspects of his person were inextricably connected. His medical explanation, which was

⁵³ *Ibid.*, pp. 226–7.

⁵⁴ Johanna Bleker, ‘Biedermeiermedizin—Medizin der Biedermeier? Tendenzen, Probleme, Widersprüche 1830–1850’, *Medizinhistorisches Journal*, 1988, 23: 5–22, see p. 18. On the sensitivity of the authors of “medical topographies” to the social conditions of illness see Brügelmann, *op. cit.*, note 8 above, e.g., pp. 158–9, 180–218. Concerning cretinism see Mauri, *op. cit.*, note 16 above, p. 45–51.

⁵⁵ Rösch, *op. cit.*, note 17 above, p. 13.

⁵⁶ In the public measures against cretinism, Rösch included the list of demands his temperance society put forward to the king, see *ibid.*, pp. 225–6.

⁵⁷ Rudolph Virchow, ‘Ueber den Cretinismus’, in *idem, Gesammelte Abhandlungen zur wissenschaftlichen Medicin*, Frankfurt on Main, Verlag von Medinger Sohn & Comp., 1856, pp. 891–939, see p. 935, footnote 6; August Hirsch (ed.), *Biographisches Lexikon der hervorragenden Ärzte aller Zeiten und Völker*, Munich and Berlin, Urban und Schwarzenberg, 1962 (reprint of the 1932 original), vol. 4, p. 849.

based on his particular definition of cretinism, was a reflection of his political attitude too. Let us now look at the other part of Rösch's therapeutic programme, the cure of the individual cretin.

"For a long time cretinism was thought to be incurable",⁵⁸ with these words Rösch started his chapter on individual therapy. As evidence that cretinism was curable now, he reported on Johann Jakob Guggenbühl's (1816–1863) Swiss institute on the Abendberg for the cure of cretins. The Abendberg was the model for a veritable wave of foundations of this kind. Guggenbühl claimed to cure cretins by separating them from the detrimental influences of their environment. The cretinous subjects were brought to his institution (opened in 1841), which was built in a place where there were no such influences. The change of environment had to be supported by other measures which were to counteract the "occasional" causes of cretinism. Guggenbühl tried a number of different non-related activities on his patients: mental and physical training, emotional care, supplemented by pharmaceutical, surgical or other medical means. Based on the notion of cretinism he had expounded in the preceding chapters, Rösch suggested the establishment of a similar institute in Württemberg. Obviously this project was the central point of his book on cretinism. And Rösch was successful: three years after its publication, the king of Württemberg, William I, having visited the celebrated Abendberg, commissioned Rösch to organize an institution "modelled on the Abendberg". Mariaberg, a former monastery in the Swabian Alps, was turned over to him as the first German imitation of Guggenbühl's concept.⁵⁹

So Rösch's way of defining and explaining cretinism was part of his political project and his professional ambitions. This does not mean that he promoted his own interests at the cost of the cretins, for he certainly did what he thought to be the best for them. His political and professional projects were also a result of how he experienced and conceived cretinism, given the background of his practical and intellectual options.

The demands by Rösch and others were put into practice in only a very rudimentary form.⁶⁰ He had proposed a multitude of public measures, each of which was equally important, and the neglect of any one might prevent a successful result, which was not in any case assured. This made it difficult to convince the authorities that investment in such a project was worthwhile.⁶¹

On the level of individual therapy, Rösch's success was short-lived. For Rösch personally it was over when he was dismissed by the authorities. Eventually, the whole concept of curing cretinism according to Guggenbühl's model failed spectacularly. After a few years of wild enthusiasm in many countries, accompanied by a flood of donations, acclamation, fame and respect for the founder, attitudes shifted.⁶² Apparently the public felt that Guggenbühl could not live up to his promises, and the ensuing frustration changed to animosity. The authorities closed down his institution on the Abendberg in 1858. For two decades his enterprise was never mentioned except in a derogatory way, and

⁵⁸ Rösch, op. cit., note 17 above, p. 229, on individual therapy and care see pp. 229–34.

⁵⁹ Leo Kanner, 'Johann Jakob Guggenbühl and the Abendberg', *Bull. Hist. Med.*, 1959, 33: 489–502, see p. 494. On the concept and similar enterprises at the same time see Mauri, op. cit., note 16 above, pp. 116–26; for additional details of Rösch's initiative see *ibid.*, pp. 106–7.

⁶⁰ *Ibid.*, pp. 113–18.

⁶¹ Cf. the difficulties of the French hygienists as seen by Latour, op. cit., note 45 above, pp. 20–1, 33.

⁶² See Kanner, op. cit., note 59 above, pp. 496–500.

when the animosity finally cooled down, what was acknowledged as his achievement was not the cure of cretins but that he had directed public attention to the care of feeble-minded children in general. Accordingly, the institutes modelled on the Abendberg, including Rösch's were converted to serve this more general purpose. Rösch himself was later celebrated as a pioneer of psychiatry; his connection with Guggenbühl was not mentioned at all.⁶³

Though subsequently attacked by the protagonists of “physiological” and “rational” medicine, the “method of natural history” was in fact not abandoned but incorporated into scientific medicine,⁶⁴ especially for diseases, such as cretinism, in which a physiological explanation could not be found. In survey articles on cretinism we find Rösch and Maffei as important references, and, variations of details apart, we find the same broadness of the disease entity, the collection of numerous examples and counter-examples, the same multitude of causes that are neither necessary nor strictly sufficient, the same emphasis on the significance of place. As a consequence of all this the same uncertainty of explanation and medical action prevailed.⁶⁵ In 1883, the year when a new approach to cretinism was introduced, August Hirsch (1817–1894) in his eminent work on historical-geographical pathology, noted that with increasing empirical knowledge contradictions had increased too, and that one must concede that “the cause of the endemics of goitre and cretinism is still shrouded in mystery”.⁶⁶

II. CRETINISM AS AN ORGAN DISEASE

The occasion of Kocher's discovery of the cause of cretinism was surgical practice. Paradoxically, it was a surgical success that turned out to be a catastrophe for the patients concerned. While the removal of goitre had previously been a difficult and dangerous operation, Kocher became so proficient that he could remove the whole of the thyroid gland without the patient dying. He had, however, not reckoned with the serious consequences of the ablation of the gland. Some time after the thyroidectomy a remarkable change occurred in the patients.⁶⁷ They lost their physical strength and their intelligence, showed symptoms like swollen hands and feet, a puffy face, and anaemia.

⁶³ See Wildermuth, ‘Die Fürsorge für Idioten und Epileptische in Württemberg’, *Medicinisches Correspondenz-Blatt des Württembergischen ärztlichen Vereins*, 1902, 72: 760–5, pp. 761, 763–4, and Theodor Schön, ‘Die Entwicklung des Krankenhauswesens und der Krankenpflege in Württemberg’, *ibid.*, 1903, 73: 917–22, see pp. 921. Mauri, *op. cit.*, note 16 above, pp. 99–103. Wilhelm Griesinger, *Die Pathologie und Therapie der psychischen Krankheiten*, 2nd ed., 1861, pp. 352–99.

⁶⁴ Bleker, *op. cit.*, note 21 above, pp. 103–41.

⁶⁵ See the surveys on the state of the art: Oberamtsarzt Dr Vötsch, ‘Zur Orientierung in der Cretinenfrage’, *Medicinisches Correspondenz-Blatt des Württembergischen ärztlichen Vereins*, 1867, 37: 327–33, 335–41, 343–9, 351–4; and August Hirsch, *Handbuch der historisch-geographischen Pathologie*, Stuttgart, Verlag von Ferdinand Enke, 2nd ed., 1883, vol. 2, pp. 83–140, with a compilation of all the pertinent literature; see also Bornhauser, *op. cit.*, note 16 above, pp. 34–43, and Mauri, *op. cit.*, note 16 above, pp. 11–54.

⁶⁶ Hirsch, *op. cit.*, note 65 above, p. 123. Hirsch with his “historical-geographical pathology” was heir to the school of natural history, see Johanna Bleker, ‘Die historische Pathologie, Nosologie und Epidemiologie im 19. Jahrhundert’, *Medizinhistorisches Journal*, 1984, 19: 33–52, p. 46.

⁶⁷ T. Kocher, ‘Über Kropfexstirpation und ihre Folgen’, *Verhandlungen der Deutschen Gesellschaft für Chirurgie*, 1883, 12: 1–84; At the same time Kocher's Geneva colleague J.-L. Reverdin made the same observation, see J.-L. Reverdin, A. Reverdin, ‘Note sur vingt-deux opérations de goitre’, *Revue médicale de la Suisse romande*, 1883, 3: 170–98, 234–78, 309–64. See also Bornhauser, *op. cit.*, note 16 above, pp. 74–112, and Tröhler, *op. cit.*, note 1 above, pp. 130–2. It may well be that Kocher only became aware of the effects of total thyroidectomy after J.-L. Reverdin had told him about his observations. In my account I will concentrate on

The changes in children were even more marked: their physical growth as well as their intellectual development were seriously stunted. These children reminded Kocher of cretins. He collected all the evidence available and presented it to the congress of the German Surgical Association in April 1883, warning other surgeons of the detrimental consequences of the total ablation of the gland. As early as July 1883 he tried to reverse the damage by re-implanting a thyroid gland in order to replace the removed organ. He was, however, not content with the results of thyroid transplantation and abandoned the method for the time being. Subsequently another method of organ replacement, the parenteral or oral administration of a thyroid preparation, was developed and later became the standard treatment for thyroid deficiency.⁶⁸

When presenting his arguments Kocher claimed that it was the removal of the thyroid that had induced the side-effects he described, and that there was a close relationship between cretinism and the thyroid gland. Though Kocher's suggestions were not accepted unanimously,⁶⁹ his findings were eventually taken up by many other practitioners and scientists, and spread beyond geographical and disciplinary boundaries.⁷⁰ The organ concept provided a new focus for research and was seen to offer potential for practical and theoretical success. The new idea, which, as we will see, included the reclassification of patients, was the result of a process of negotiation and persuasion over a period of time. Building up the new concept entailed a restructuring of clinical and experimental experience⁷¹—a process that allowed scope for competing claims: one theory maintained that an infection by living agents was the cause of cretinism and goitre,⁷² another thesis blamed changes in the nervous system.⁷³ This process and the different groups and partial interests involved cannot be described in any detail here. We will just look at those aspects that will help us to understand Kocher's argument when he redefined cretinism.

Perhaps the most important collaboration was with physiology.⁷⁴ Kocher's thesis was welcomed by many physiologists. The subject provided a field where physiology could demonstrate its importance for medicine.⁷⁵ If the removal of the organ was to be established as the specific cause of the effects observed, then these had to be distinguished

Kocher not because he was the first to note the phenomenon but because he was more important for subsequent developments than Reverdin.

⁶⁸ In 1891, throughout Europe, a number of patients were being treated for myxoedema and cachexia strumipriva with injections of thyroid extract. Priority is most often given to B. R. Murray. See Borell, op. cit., note 14 above, p. 45; Tröhler, op. cit., note 1 above, pp. 126–39.

⁶⁹ Tröhler, *ibid.*, pp. 128–30.

⁷⁰ Tröhler, *ibid.*, pp. 134–56. On internationalism and transdisciplinary co-operation in this and other related subjects see W. F. Bynum, "'C'est un malade"—animal models and concepts of human diseases', *J. Hist. Med.*, 1990, 45: 397–413.

⁷¹ Cf. Lawrence, op. cit., note 2 above, p. 53–4 on coronary thrombosis.

⁷² Bernardino Fantini, 'La révolution pastoriennne et les théories sur l'étiologie du goitre et du cretinisme', *Gesnerus*, 1992, 49: pp. 21–38.

⁷³ See, e.g., Hermann Munk, 'Zur Lehre von der Schilddrüse', *Virchows Archiv*, 1897, 150: pp. 271–305. Pathological changes of the nervous system were a common alternative explanation for diseases which later became endocrine disorders, see, e.g., Thomas Schlich, 'Making mistakes in science—Eduard Pflüger, his scientific and professional concept of physiology and his unsuccessful theory of diabetes (1903–1910)', *Stud. Hist. Phil. Sci.*, 1993, 24: 411–41.

⁷⁴ As it is "questionable how far we can separate experimental physiology and experimental pathology in the nineteenth century" (Bynum, op. cit., note 70 above, p. 400), I will subsume both under the term "physiology".

⁷⁵ The first physiologist to become interested in Kocher's findings was Moritz Schiff who noticed the relevance of the experiments on thyroidectomy he had performed decades before, see M. Schiff, 'Bericht über die

from other, non-specific side-effects of the operation. To tackle this problem, animal experiments, especially ablation and transplantation, were the most appropriate method. Thus, the internal secretion of the thyroid gland became a popular subject for experimental research in physiology.⁷⁶

Kocher was one of the surgeons of the time whose approach to surgery can be called physiological. In the course of his career he strove to base his medical action on physiological knowledge and did not shrink from using physiological methods, such as animal experiments, himself.⁷⁷ The case of the thyroid gland illustrates the close affinity of surgery and experimental physiology. Like surgery, experimental physiology aimed at controlling the processes of life, not just at observing or supporting them.⁷⁸ In terms of sufficient and necessary causality, this means that in the ideal case an intervention by the experimenter results in a specific reaction of the organism, a reaction for which the intervention is sufficient as well as necessary.⁷⁹ So if the removal of the thyroid gland in animals caused a certain combination of phenomena which could not be provoked by any other manipulation, then the absence of thyroid tissue was a necessary precondition of this state. In the case of complete control these phenomena could also be reversed at will, namely by reinserting the gland. The ability to provoke and reverse cretinous symptoms independent of place was also the crucial argument against the still-prevailing view that cretinism was endemically caused. Kocher's view had at first been rejected because his observations were interpreted in accordance with the old notion as simply another stage of the endemically caused process which had advanced in spite of the removal of the goitre.⁸⁰ Only after the effects of thyroidectomy had been observed in different places could this argument be refuted.

Another important factor in establishing the new theory was collaboration with British physicians, surgeons, and experimenters. They provided a crucial constituent for the equation of Kocher's "cachexia strumipriva" with cretinism. In Britain, Kocher's thesis was applied to another disease, myxoedema. By 1883 myxoedema had been conceived as a discrete disease entity for a decade and had already been regarded as being somehow related to cretinism. On the basis of clinical and, more important, experimental evidence collected by a special committee that had been founded after Kocher's findings became

Versuchsreihe betreffend die Wirkungen der Exstirpation der Schilddrüse', *Archiv für experimentelle Pathologie und Pharmakologie*, 1884, 18: 25–34, p. 25; on the problematic relationship between physiologists and clinicians see, e.g., Schlich, op. cit., note 73 above.

⁷⁶ See Bynum, op. cit., note 70 above, concerning experiments on thyroid function pp. 409–11. On the role of ablation and transplantation see also Thomas Schlich, 'Vom physiologischen Experiment zur Therapie: Die Pankreastretransplantation', *Medizinhistorisches Journal*, 1993, 28: 29–54.

⁷⁷ See Tröhler, op. cit., note 1 above, and *idem*, 'Die Wechselwirkung von Anatomie, Physiologie und Chirurgie im Werk Theodor Kochers und einiger Zeitgenossen', in Urs Boschung (ed.), *Theodor Kocher 1841–1917*, Bern, Hans Huber, 1991, pp. 53–71.

⁷⁸ Claude Bernard, *An introduction to the study of experimental medicine* (English translation of the 1865 original), New York, Dover Publications, 1957, pp. 65–74; on the close relationship between experimental physiology as practised by Bernard, and surgery, see John E. Lesch, *Science and medicine in France. The emergence of experimental physiology*, Cambridge, Mass., Harvard University Press, 1984.

⁷⁹ For the aim of experimental physiology to find out the conditions which are sufficient and necessary for a certain phenomenon so that the experimenter can promote or prevent it at will, see Bernard, op. cit., note 78 above, pp. 66–8.

⁸⁰ Cf. 'Report of a committee of the Clinical Society of London to investigate the subject of myxoedema', *Transactions of the Clinical Society of London, Suppl.*, 1888, 21: pp. 172–3; Tröhler, op. cit., note 1 above, pp. 128–9.

public, a destructive change of the thyroid gland was declared to be the cause of myxoedema. This definition provided an important link between “cachexia strumipriva”, a disease of adults, and cretinism: myxoedema was also a disease of adults, its symptomatology being very similar to that of cachexia strumipriva. By contrast, cretinism, a child’s disease, resembled more the consequences of thyroid ablation in children. Now, for all three diseases—cretinism, myxoedema and cachexia strumipriva—thyroid deficiency was claimed to be the necessary and strictly sufficient cause. This connection altered Kocher’s original theory and at the same time became one of the strongest points in favour of it.⁸¹

So the new concept was spread beyond geographical and disciplinary boundaries. It was strengthened and compared with concurring concepts. As a result the original concept was considerably changed.⁸² Though Kocher’s personal role in all this was limited, he was given the credit for it. In 1909 he was awarded the Nobel prize for the discovery of thyroid function.⁸³ By the time Kocher’s account of 1892 was published, cretinism was generally assumed to be a special form of thyroid deficiency, being caused by the lack of the internal secretion of the gland during childhood.⁸⁴

Kocher’s account of 1892

When Kocher wrote his 1892 article there was still a certain extent of disagreement with the new view; it was, however, already sufficiently accepted for Kocher to propose a new policy on cretinism on that basis. Written nine years after he had first noticed the side effects of total thyroidectomy, it was the same article in which he mentioned his journey described in the introductory passage of this paper.⁸⁵ Here he made the change of identity of the disease one of his central themes. He explicitly rejected the old notion of it, restricting the term to only some of those cases that had until then been regarded as cretinism. As “building material” for his own concept, Kocher—like Rösch and Maffei before him—drew on his predecessors’ works. Kocher picked out from their descriptions what he could use to support his own account. Concerning disease identity, he excluded the majority of the “cretins” shown to him on his excursion, as well as many of the categories and cases of cretinism he found in the literature, among them Rösch’s and Maffei’s treatises. For Kocher, Rösch’s and Maffei’s accounts were based on wrong disease identity as a consequence of his redefinition.

The crucial criterion for the attribution of cases to cretinism was now the resemblance of their clinical picture with the effects Kocher had observed after total thyroidectomy, his “cachexia strumipriva”. All those cretins who did not look like Kocher’s artificial cretins were no longer cretins, for example, some deaf-mutes. With regard to thyroid deficiency

⁸¹ On the British connection and the impact of Kocher’s finding’s on British investigation as well as vice versa see Report, op. cit., note 80 above; Kocher op. cit., note 1 above, pp. 588–94. Tröhler, op. cit., note 1 above, pp. 130–56; Bynum, op. cit., note 70 above, p. 410. The Report was not the first publication in which myxoedema, cretinism, and cachexia strumipriva were likened. The Reverdins had done this already in 1883 and in the same year Felix Semon had pointed out to his British colleagues the observation made on the Continent. But because of its quasi-official character the Report was an important step in establishing the new concept.

⁸² For this see Tröhler, op. cit., note 1 above, pp. 126–39, also Report, op. cit., note 80 above.

⁸³ This is similar to what can be seen in the case of Pasteur, see Latour, op. cit., note 45 above.

⁸⁴ See a survey by Victor Horsley, ‘Remarks on the function of the thyroid gland: a critical and historical review’, *Br. med. J.*, 1892, i: 215–19, 265–8.

⁸⁵ Kocher, op. cit., note 1 above, pp. 596–601.

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as the cause of cretinism, he stated that among the six persons selected as genuine cretins from thirty-two shown to him, three had a goitre and two had “not a trace of a thyroid gland”. This was, according to Kocher, the proof that endemic cretinism, like cachexia strumipriva, was caused by a lack of thyroid function.⁸⁶ We note here the same kind of circularity of argument Rösch and Maffei had been using. Rösch and Maffei had chosen their collection of cases according to the same criterion which they later declared to be the cause—the place of occurrence. Kocher selected his collection according to resemblance to cachexia strumipriva, only to find later that those selected really had signs of a lack of working thyroid tissue. This he then—in circularity—defined as the cause of cretinism (see Figure 3). What Kocher presented here was the cause of a new cretinism, a cretinism that was defined according to the effects of surgery.

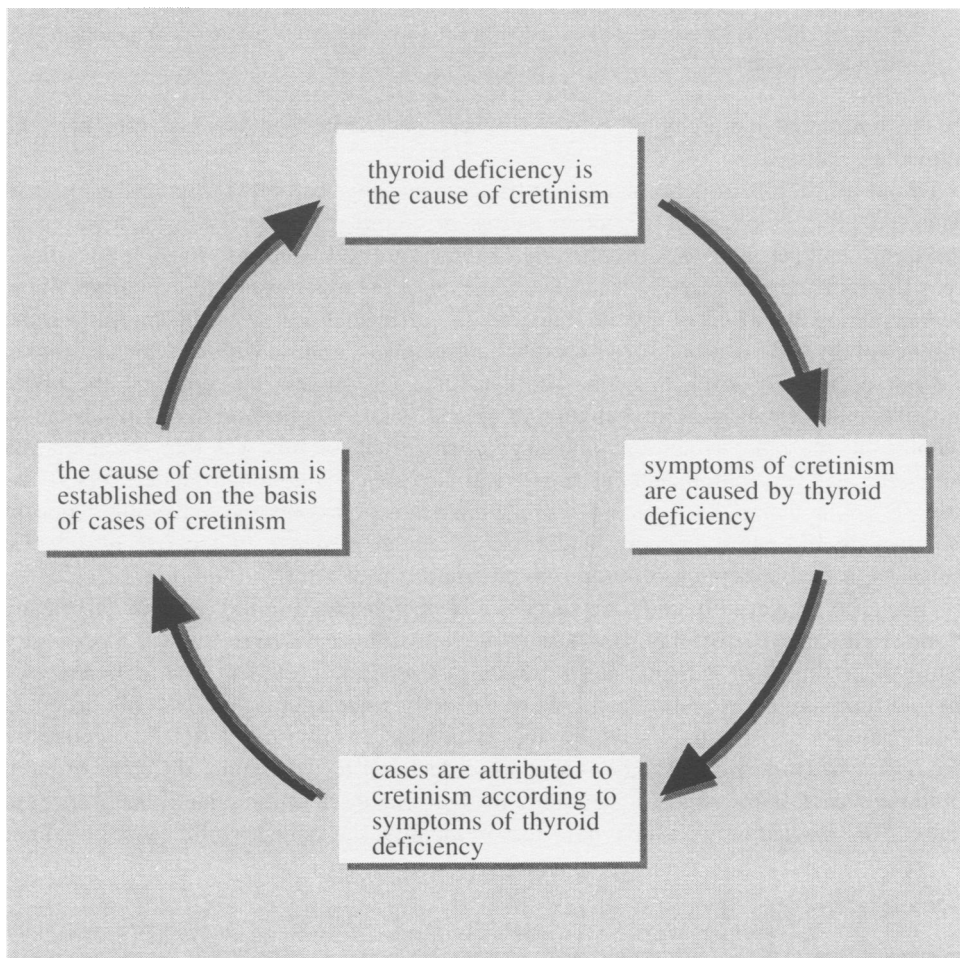


Figure 3: Circularity of argument 1892: cretinism as organ failure.

⁸⁶ *Ibid.*, pp. 596–9.

In contrast to his predecessors, Kocher displayed a high degree of certainty about the attribution of individual cases to cretinism. The new disease entity was so objective, that “any student of medicine can with certainty make the diagnosis”, and distinguish a cretin from any other ill person, such as deaf-mutes, imbeciles and idiots.⁸⁷ Kocher based his certitude on the knowledge of the necessary cause—the *conditio sine qua non*—of cretinism: “We know now that the pathological disturbance affects a particular organ, that the affection of the same and of only this organ is the *conditio sine qua non* of the development of genuine cretinism. We can as well say *that at present we know the cause of cretinism*”.⁸⁸ Contrasting his concept with the previous one he wrote:

Rather than a large number of causes acting in combination, the effects of which were even thought to continue over long periods of time to bring about such a terrible degeneration of human beings, it became obvious that the failure of a very small gland, which until then had been taken to be insignificant, can bring about the picture of cretinism within a few years . . .⁸⁹

In the traditional hierarchy of causes Kocher concentrated on the last category, the immediate cause.

To put it in terms of necessary and sufficient causality: before 1883 investigators had collected large numbers of sufficient causes of cretinism, thus creating a network of conditions and partial causes. Kocher, by contrast, directed interest to a single specific—i.e. sufficient and necessary—cause. The removal of the organ was a test for sufficiency, demonstrating that a lack of thyroid function was sufficient to cause cretinism. But was the absence of thyroid function also a necessary cause, that is, a cause without which cretinism did not occur? To check this, one could reverse the process by replacing the organ function, either by surgical implantation of organ tissue or application of organ substance, keeping all the other conditions unchanged. The result of this was that when thyroid deficiency had been eliminated the disease was also gone: without thyroid deficiency—no disease. So the therapeutic concept of organ replacement, as opposed to the older concept of changing the environmental conditions, is another instance of the rise of specific causality in the conception of disease as expounded by Carter.⁹⁰

Thus specific causality could be successfully demonstrated in the cases of “artificial” cretinism after thyroidectomy. But how to prove that thyroid deficiency was a necessary cause of cretinism in general? Easily done: just redefine cretinism as a deficiency of thyroid function. As we have seen, this was exactly what Kocher did. He only accepted those patients as cretins in whom the symptoms of thyroid deficiency could be demonstrated (Figure 3). All the others were declared to have some different disease. Defining a disease by a specific aetiology made it true by definition that it had a specific cause. This worked only with the new disease identity: the same specific cause could not

⁸⁷ Ibid., p. 566.

⁸⁸ Ibid., p. 570–1. Kocher’s emphasis. The sentence continues: “as far as one can speak of a cause if the pathological-anatomical basis of a malady can be demonstrated.”

⁸⁹ Ibid., p. 594.

⁹⁰ Carter, op. cit., note 37 above. Carter names germ theory, deficiency diseases, and psychopathology (p. 528). Note that this concentration on specific causes of disease in bacteriology came up at the same time as in the case of cretinism.

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be attributed to the old, endemic characterization of cretinism.⁹¹ In contrast to Rösch and Maffei, Kocher claimed to work from the cause to the symptoms: first establish the cause, then define the disease entity according to it.⁹² This is, however, only the structure of the argument. What Kocher actually did was to use symptoms, namely those of the artificial cretins, to decide which cases “really” belonged to cretinism.

As a result of all this, disease and organ were firmly linked. Any environmental influence that might lead to cretinism in the end, always operated via a disturbance of thyroid function.⁹³ All further considerations concerning aetiology, diagnosis and control of cretinism had to take the thyroid gland into account (see Figure 4).

So the new disease entity was restricted on one side—many of the old cases of cretinism were excluded. It was, however, extended on the other side: myxoedema and other mild forms of what was now called “thyroid deficiency” were included. The inclusion required new criteria and special skills and knowledge. Spreading this know-how was deemed so important by Kocher that he dedicated his Nobel lecture to the subject.⁹⁴ As the equation

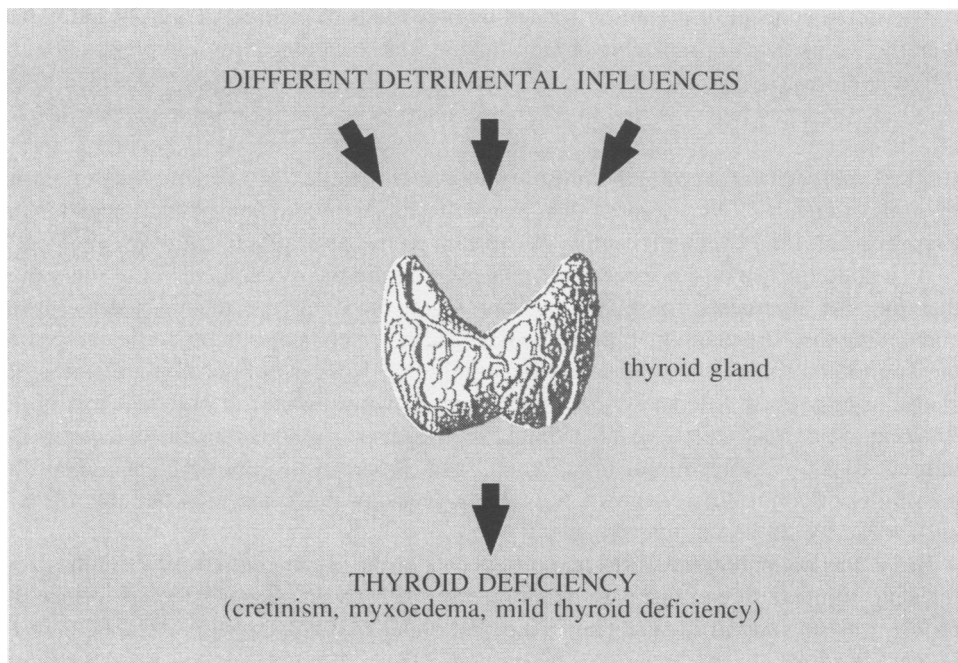


Figure 4: Causes of cretinism as organ failure.

⁹¹ On this relation between aetiological characterization and necessary causality of disease, see K. Codell Carter, ‘Ignaz Semmelweis, Carl Mayrhofer, and the rise of germ theory’, *Med. Hist.*, 1985, **29**: 33–53, pp. 33–4.

⁹² Cf. Cunningham, *op. cit.*, note 2 above, pp. 217–18.

⁹³ Kocher, *op. cit.*, note 1 above, pp. 578–9.

⁹⁴ Theodor Kocher, ‘Concerning pathological manifestations in low-grade thyroid diseases. Nobel lecture, December 11, 1909’, in *Nobel lectures. Physiology or medicine 1901–1921*, Amsterdam, London, New York, Elsevier Publishing Company, 1967, pp. 330–83. See also Kocher, *op. cit.*, note 1 above, pp. 599–601.

of cachexia strumipriva, myxoedema, and cretinism was the basis of the new disease entity, the criteria for the diagnosis of mild “cachexia thyreopriva” consisted of a variety of symptoms taken from these diseases. Significantly, in cases of doubt the diagnosis was to be made *ex juvantibus*: if the application of a thyroid preparation improved the condition of the patient, the diagnosis was thyroid deficiency, if not, it was something else—so again, the same points that were later used to explain the disease were also used as criteria for the inclusion of individual cases in the disease entity. Again, the new disease entity was not determined by nature.

Therapy and prevention—surgery

According to Kocher, not only were all the old statistics wrong because they were based on the wrong disease identity,⁹⁵ therapy too had failed, because the true cause of cretinism was not known. In his 1892 article Kocher referred to the problems the old approach entailed: the multitude of partial causes had made control of the disease impossible. As the most famous example of therapeutic failure he cited Guggenbühl's institution (the one emulated by Rösch), which, as Kocher wrote, had turned out to be a “fiasco”.⁹⁶

The organ concept of cretinism opened up new roads to medical action. In individual therapy two methods of replacing organ function were developed: thyroid preparations in different forms, and transplantation of thyroid tissue or whole organs. As mentioned above, Kocher had been the first to try transplantation in order to reverse the detrimental effects of organ removal but had subsequently given up the method. Others took up the surgical approach and eventually transplantation was regarded as a feasible way of curing thyroid deficiency.⁹⁷ As a result of that, Kocher resumed thyroid transplantation and in the first decades of the twentieth century became an enthusiastic proponent of the method.⁹⁸

As a consequence of the successful application of the theory on the level of individual therapy, the arguments for public measures supported by the theory became more convincing too. The claim that the immediate cause, which is also the specific cause, was the key to controlling the disease did not imply that Kocher did not know of the other causal categories. In Kocher's view some yet unknown substance or organic agent in the drinking water was the cause of endemic cretinism and goitre, though, of course, the indirect cause, via affection of the thyroid gland. Kocher was very optimistic about the possibility of controlling cretinism, now that its cause was known, provided the relevant knowledge would be put into practice.⁹⁹

But it was not public measures that proponents of the organ-centred view emphasized: focusing on the failure of one particular organ of the body enabled the doctors to treat the problem as an individual one that could and should be solved within the domain of

⁹⁵ Ibid., p. 557, 608.

⁹⁶ Ibid., p. 558, 574.

⁹⁷ See as one of many examples, E. Payr, ‘Transplantation von Schilddrüsengewebe in die Milz; experimentelle und klinische Beiträge’, *Verhandlungen der Deutschen Gesellschaft für Chirurgie*, 1906, 35: 503–99.

⁹⁸ Theodor Kocher, ‘Über Schilddrüsentransplantation’, *Verhandlungen der Deutschen Gesellschaft für Chirurgie*, 1908, 37: 231–7. *Idem*, Nobel Lecture, op. cit., note 94 above, p. 358. *Idem*, ‘Ueber die Bedingungen erfolgreicher Schilddrüsentransplantation beim Menschen’, *Verhandlungen der Deutschen Gesellschaft für Chirurgie*, 1914, 43: 484–566. Tröhler, op. cit., note 1 above, pp. 149–52.

⁹⁹ Kocher, op. cit., note 1 above, pp. 607–26. On Kocher's contributions to the epidemiology of endemic cretinism and goitre see Tröhler, op. cit., note 1 above, pp. 154–5.

medical competence. There was no more need for social and political measures; it was sufficient to trust in the doctor. The organ concept allowed doctors to distance themselves from moral, political, and social questions, avoiding the frustrations of their meddling in these fields.

In order to understand Kocher's particular choice of definition, it is useful, as with Rösch, to look at what projects he was engaged in. Kocher was not so much interested in general politics, rather he was committed to securing surgery a respected position within medicine. With the redefinition of the disease, the balance of power of those who dealt with it had been changed. If, as before the organ concept, local lesions are understood as the results of a pathological change of the whole organism, the specialist for local treatment—the surgeon—will be subordinated to the physician, the physician will be the one who can overlook the consequences of a local intervention for the body as a whole. Cure can be achieved only by a therapy for the whole body.¹⁰⁰ This relationship will be reversed if the mechanism of causation has been reversed. If the organism becomes ill because of a localized pathological change, say in the thyroid gland, then local therapy becomes crucial. Now the surgeon, not the physician, is the one who can cure the disease.

Throughout his long career, Kocher was intent on expanding the field of competence of surgery. With many of his surgical colleagues he shared an optimistic outlook on surgery's future role, as well as a predisposition to fight for the position of his speciality against the claims of competing disciplines, especially internal medicine. This is evident by his perennial quarrels with his local physician colleagues at Berne on whether surgery or internal medicine provided the best therapeutic options, as well as by his frequent arguments in defence of surgery against the predominance of internal medicine in general.¹⁰¹ When he was awarded the Nobel Prize, the first surgeon ever to receive it, he exulted that "a surgical method of treatment, which was crowned by the most brilliant cures, was made possible in the great majority of the so-called internal diseases."¹⁰² That Kocher also supported a non-surgical preventive approach alongside the surgical treatment by transplantation did not diminish the importance of surgery. Rather, it stressed that as a discipline, surgery had competence in both, providing ideas and solving practical problems in medicine.

Like Rösch, Kocher certainly did what he thought best for the cretins. His concept of cretinism was not a simple reflection of his professional interests, because his professional project was at the same time informed by how he experienced and perceived medical problems such as cretinism.

To sum up, the success of the organ-oriented approach depended on the redefinition of the disease entity. It was not Rösch's and Maffei's cretinism for which Kocher and those who took up his approach could provide a convincing concept and impressive practical successes. So Kocher's success was made possible by a new context, a context which he himself had actively created.

¹⁰⁰ Cf., e.g., Malcolm Nicolson, 'The metastatic theory of pathogenesis and the professional interests of the eighteenth-century physician', *Med. Hist.*, 1988, 32: 277–300.

¹⁰¹ Tröhler, op. cit., note 1 above, pp. 97–120, 184–9.

¹⁰² Kocher, Nobel Lecture, op. cit., note 94 above, p. 330.

III. CONCLUSION

So we can clearly see that cretinism encompassed different patients before and after its redefinition in terms of organ failure (see Figure 5). We can also see that the disease identity was not simply determined by nature but set up by historical actors. Of course, cretinism could not be construed arbitrarily. Only certain selections and classifications were possible. But neither of the notions of cretinism could be derived from nature alone: both were based on a circular argument in which the crucial decisions were made by the investigators themselves, not by nature. And in both cases the actors did not stick to their programme. Rösch and Maffei did not (and could not) construct their cretinism on the basis of the symptoms alone, they had to take the cause (which they claimed to be the result, not the presupposition of their investigation) into account. Nor did Kocher construct his cretinism according to a specific cause given by nature. From the beginning he had to take into account the symptoms of his would-be cretins, though in theory he claimed that symptomatology could only be the outcome of his investigations. Both authors, however, claimed to have acted according to their programme and presented their respective disease entities as being derived from nature.

In both cases the explanation of cretinism was consistent only in connection with a particular disease identity. The explanation of the disease then led to a certain type of action against it, so in the end it was the disease identity that made a certain kind of medicine convincing. The kind of action which had followed from a definition of the disease entity was part of the more general projects of those who set up the definition: Rösch could justify his political demands and raise funds for the institution he planned to build. Kocher could keep politics out of medicine, propagate individual and specific

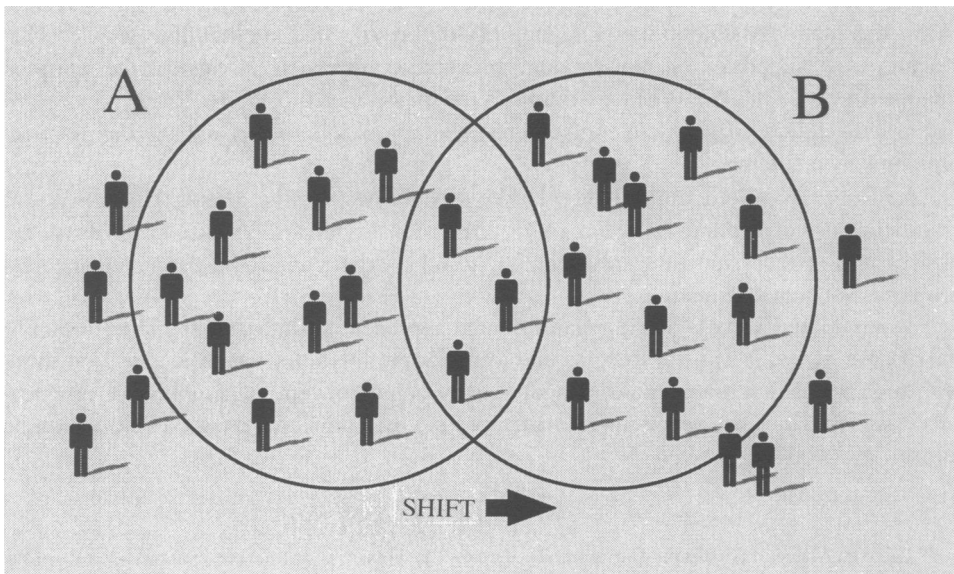


Figure 5: A. Cretinism as an “endemic” disease; B. Thyroid deficiency (including cretinism).

therapy, and make surgery more important. The relation between the professional or political projects of the actors and their respective conception of cretinism, however, was not a one-way street. The concepts of disease helped them to achieve their professional and political goals, but these were also part of the fight against disease.

Thus, on a general level, an historical account of a disease identity is not a more or less successful approximation to a natural disease identity that exists independently of those who write about it. Rather, the disease identities we encounter in historical texts have been written down by a particular author in order to stabilize a particular position within a process of continuous negotiation about reality among historical actors.