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Against Normal Function

*Ron Amundson**

The concept of normality has been the target of criticism in recent years. Social critics claim that the term carries ideological baggage. Describing individuals or groups as 'abnormal' is seen as marginalizing them by use of a falsely objective criterion. This paper will continue that tradition. It will examine the concept of *normal function*, said by many philosophers to be objectively grounded in the practice of biological and biomedical science. This concept is used in discussions of health care policy, quality-of-life assessments, and even radical 'treatments' such as assisted suicide. The core of this paper will be an examination of the biological legitimacy of the concept of functional normality. Social concerns aside, does current biology imply a concept of functional normality, and a distinction between normal and abnormal function? I will argue that it does not. In the last sections of the paper I will introduce the social context of this issue, emphasizing the disadvantages experienced by people whose function is assessed as abnormal. I will distinguish between the *level* of an individual's functional performance and the *mode* or style by which that performance is achieved. This distinction will help reveal that the doctrine of biological normality is itself one aspect of a social prejudice against certain functional modes or styles. The disadvantages experienced by people who are assessed as 'abnormal' derive not from biology, but from implicit social judgments about the acceptability of certain kinds of biological variation.

1. Normality as Race

We humans have innumerable ways of categorizing ourselves, of managing the variation among us. Some but not all of these categories are taken to reflect a biological reality. Differences between men and women are believed to be biologically real in a way that differences between Lutherans and Catholics are not. Until

* University of Hawaii at Hilo, Hilo, HI 96720, U.S.A. E-mail: ronald@hawaii.edu.
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quite recently the category of *race* was taken to reflect biological reality. There were scientific debates about the number of human races, the characteristics that typify each race, and whether races were incipient species. Racial traits were invoked to explain the differences in accomplishment among groups of people, and the dominance of some groups over others.

During this century the assumption that races are biologically real has been called into question. Biologists no longer see race as a biological category (Lewontin, 1995; Marks, 1995; AAPA, 1996). There is more variation within the traditionally named races (Caucasian, Negroid, etc.) than between them. The tradition of naming races, assigning individuals to them, and then treating individuals differently depending on their racial assignment had no factual basis in biology. Today the study of 'race' is a study of the far-reaching social consequences of that old, biologically confused tradition. We were not carving nature at its joints when we partitioned human variability into races.

I consider the concept of *normal function* to be similar to the traditional concept of race. Like the concept of race, the concept of biological normality is invoked to explain certain socially significant differences, such as unemployment and segregation. Like the concept of race, the concept of normality is a biological error. The partitioning of human variation into the normal versus the abnormal has no firmer biological footing than the partitioning into races. Diversity of function is a fact of biology.

2. Functional Determinism and Naturalism About Disease

The topic of biological normality is related to a philosophical debate on the concept of disease. Naturalists consider disease to be a straightforward, non-evaluative, theoretical concept within the sciences of medicine and physiology. Normativists consider disease concepts to embody evaluative judgments of the conditions designated as diseases. Much of the present paper is an argument for the normativity of the concept of functional normality, at least as the concept is currently used. Naturalists and normativists agree that certain disease concepts in the past ('diseases' such as homosexuality and masturbation) were ideologically tainted. The difference is that naturalists believe that such taint can be avoided by careful science, and normativists do not. My purpose is to show that the normative taint is not avoided in current discussions of biological normality.

Christopher Boorse's Biostatistical Theory (BST) of disease is the most influential naturalistic account (Boorse, 1975, 1997). It provides a foundation in the philosophy of science for most of the writers on health care ethics discussed in Section 7 and Section 8 (including the author of Amundson, 1992; but see Wendell, 1996, pp. 16*ff*., for a corrective). Boorse uses a technical definition of disease that covers such conditions as blindness, paralysis, and limb loss. My interest is in these permanent and stable conditions, commonly called disabilities, rather than in the more episodic or life-threatening conditions commonly called diseases (e.g. measles and

cancer). Boorse's account of disease is founded on the concept of biological normality. Normativists have challenged the biological foundation of Boorse's theory, but they mostly question his definition of disease *in terms of* biological normality. They do not challenge the concept of biological normality itself (Boorse, 1997, p. 41). If my critique of normality is forceful, it will presumably have implications for Boorse's account of disease, but I will not explore those implications here. Boorse's naturalism about normality can be seen in the background assumptions behind two definitions:

(1) The *reference class* is a natural class of organisms of uniform functional design; specifically, an age group of a sex of a species.
(2) A *normal function* of a part or process within members of the reference class is a statistically typical contribution by it to their individual survival and reproduction (Boorse, 1997, p. 7).

Points 3 and 4 in this list go on to define 'disease' as reduction in normal function, and 'health' as the absence of disease. The expressions 'typical contribution to . . . survival and reproduction' and 'species typical function' are used by Boorse and his followers as synonyms for *normal function*. In this paper I will use the term 'typical' not as a synonym for 'normal', but in the colloquial sense of common, usual, or frequent. On my usage a 'typical' trait may be merely the least unusual, and an atypical trait need not be abnormal. This convention allows the discussion of typical and atypical traits without assuming that they are respectively normal and abnormal. It should also be noted that, as with other quasi-statistical uses of the concept of normality, abnormality is usually to be read as subnormality. Better-than-average function is not usually labeled as abnormal even though it is statistically atypical.

Boorse's two definitions imply that natural species have a certain statistical characteristic: the variations of function among their members is sufficiently narrow to justify a dichotomy between normality and abnormality based on the distribution alone. Obviously not all species members function in exactly the same way. We can treat them as if they do by labeling as abnormal any non-conforming species members. This labeling is statistically justified only if the bell curve of functional design is very steep, i.e. there are many uniformly designed individuals and only a few scattered individuals with novel functional design. I will refer to this statistical claim about functional diversity within species as *functional determinism*. I will challenge the claim, and argue that the facts of functional variation do not support functional determinism.

Boorse and other functional determinists recognize the existence of at least some 'normal' variation. '[T]he BST can accommodate normal polymorphisms, and, of course, admits normal statistical variation . . .' (Boorse, 1997, p. 39). Eye color and blood type are customarily cited as examples of normal variation (*ibid.*, p. 32; Wachbroit, 1994a, p. 590). Boorse gives no account of how normal variation is differentiated from abnormal variation. One suspects that normal variation simply

means *functionally equivalent* variation, like eye color and blood type. No examples are given of functionally distinct but still normal variation.

To set the stage, let us dissect functional determinism a bit. It is useful to distinguish between the *level of performance* of a function and the *mode* of its performance (Silvers, 1998, p. 101). Functional mode is the manner in which a functional outcome or performance is achieved. Performance level is the quantitative degree of the functional performance, such as the speed or the strength of a motion. In addition, we can identify functions at different organizational levels of the biological hierarchy (Wachbroit, 1994b, p. 237). Functions can be seen as occurring at genetic and physiological levels of the hierarchy, at the level of limb movements, and even in ecological interactions (e.g. 'obtain food'). Whatever the hierarchical level, functional determinism states that functions take place in a uniform mode at a relatively uniform performance level by a statistically distinctive portion of the members of a species. These are the normals.

Discussion of functional determinism will consider information from evolutionary biology, developmental biology, physiology, and anatomy. First, evolution.

3. Darwin

Variation among individuals in a species can be seen as arising from two sources. One is the genetic variation that exists in all natural species, and on which natural selection operates. The other is developmental plasticity, the variability of the traits that an organism actually develops during its lifetime due to influences other than its genome. From an evolutionary perspective, this distinction is rather artificial. It is a version of the infamous nature/nurture contrast. Developmental plasticity itself evolves by natural selection, and genomes only determine phenotypic traits within the context of developmental plasticity. But for present purposes I will treat nature and nurture as distinct. This section will deal with heritable variation in natural populations, and what modern evolutionary theory has to say about it. The following section will discuss developmental plasticity.

Current evolutionary theory considers natural species to contain very large amounts of heritable variation. This contradicts certain earlier doctrines about 'pure lines' and 'the wild type', that considered natural species to be relatively genetically homogenous. The first influential proponent of the modern view was Theodosius Dobzhansky (Mayr, 1980, p. 128). Based on his beliefs in high degrees of genetic variation, Dobzhansky had this to say about normality: 'The use of the word "normal" poses a semantic problem. No end of misconceptions and lax thinking is caused by the belief in something called "normal man" or "normal human nature"' (Dobzhansky, 1962, p. 126). Is the Darwinian view of rich ranges of variation consistent with the notion of a determinate species design?

Boorse and Robert Wachbroit recognize and comment on the contrast between the Darwinian doctrine of variability and their own doctrines of a determinate species design. Both acknowledge that functional determinism might be seen as a

typological or essentialistic theory, and so as antithetical to Darwinian population thinking.[1] Wachbroit argues that biological normality is theoretically grounded in biomedical science. He appears simply to accept the contrast with Darwinian evolution in his comment that 'tensions between established scientific doctrines are not uncommon' (Wachbroit, 1994a, p. 590). He does seem to attempt a reconciliation, however: 'Of course, some variations will be abnormal from the perspective of physiology, while others, understood in evolutionary terms, will be ascribed to speciation, where variation constitutes a different, not abnormal, physiology' (*ibid*.). If this is intended as a reconciliation, it is unsuccessful. Variation is not confined to speciation events in evolutionary biology. Variation is ubiquitous. It is always 'different, not abnormal' simply because there is no Darwinian interpretation of abnormality.

Boorse makes more detailed comments on the tension. One response is similar to Wachbroit's; the BST analysis is based on contemporary physiology, and any typology comes from the present state of the science (Boorse, 1997, p. 33). A second is the assertion that evolution typically drives traits to fixation in a species, and the traits thereafter are kept from varying by normalizing selection (a very aptly named phenomenon if this is what it accomplishes) (*ibid.*, p. 32). A third is that essentialism usually involves a claim about the causal powers of the essential traits, and the BST has no such implications (*ibid*., p. 38).

I will briefly comment on these defenses in turn. First, current physiology may not be as typological as the determinists believe. And if it is, it might well be wrong in its typology. These points will be argued in Section 6. Second, I am willing to consider it an open empirical question whether evolution results in the kind of functional uniformity that would license normality definitions. Frankly I doubt it, but theoretical considerations do not suffice to answer the question. Third, I agree that the concept of normality invokes no essentialist causal powers, in that the functional type does not explain biological form. I am concerned, however, that once the concept is introduced and reified, it is *itself* used in causal explanations of social phenomena. It is used to explain and rationalize the social disadvantages of people labeled abnormal.

My own opinions about the tension between evolution theory and functional determinism tend towards those of David Hull. Hull argues that no set of traits can be constructed so as to characterize all and only members of a natural species, that species are rife with variation, and that this is an unavoidable outcome of Darwinian biology. '. . . [A]ttempts to argue away this state of affairs by reference to "potentiality" and "normality" have little if any foundation in biology' (Hull,

[1]As one of the very few defenders of typological thinking in evolutionary biology I should state my position carefully. The kind of thinking I defend in 'Typology Reconsidered' (Amundson, 1998) is exemplified by the nineteenth-century school of Unity of Type thought, with types associated with taxonomic groups above the species level. I am an orthodox Darwinian on the nature of species. There are no types within species (contrary to racist biologists); species themselves are not types; and the typological appearance of functional determinism is a problem to be reckoned with.

1986, p. 4). I doubt, however, that the refutation of functional determinism can be achieved on the basis of evolutionary *theory* alone. Evolution is a process that gave rise to tapeworms and elephants. It could surely give rise to species members as functionally alike as paper clips, and to species members as functionally diverse as . . . well, as human beings.

If we base our estimate of functional diversity on genetic diversity, there seems to be plenty available in the human species. Studies of a group of over four hundred distinct species of cichlid fishes in Lake Victoria have shown that there is less genetic variation among the cichlid species than within the single species *Homo sapiens*. The four hundred cichlid species show wide variations in functional organization (Stiassny and Meyer, 1999). So one cannot argue that human beings share a single functional design based on the lack of genetic variance. High genetic variance creates at least a potential, if not a proof, of functional variability. Evidence from developmental biology gives further evidence of variability.

4. Developmental Plasticity and Integration

Boorse recently explicated his naturalistic concept of health by connecting it with the concept of the goal-directedness of life processes (Boorse, 1997, pp. 9*ff*.). He takes goal-directedness to be expressed in the notion of *species design*. I agree with the goal-directedness of life, but I consider Boorse's version of it unnecessarily narrow. Functional determinism does not follow from life's goal-directedness. In fact, certain goal-directed biological processes make the notion of a determinate design seem presumptuous.

Two pre-evolutionary concepts of teleology illustrate the contrast between my preferred notion of goal-directedness and Boorse's. Functional determinism was anticipated in the tradition of British natural theology, with William Paley as the traditional spokesperson. Body parts of an organism are specifically designed to adapt the organism to its environment, and each member of a species is functionally identical. A contrasting pre-evolutionary sense of teleology existed in the Continental tradition of developmental morphology. Teleology was seen not in the external fit of the organism into its environment, but in the internal directedness of the processes of embryological development. Lenoir identifies Kant's *Critique of Judgment* as an early representative statement of this emphasis on development (Lenoir, 1982).

The Kantian concept of biological directedness focuses on the processes of embryological, ontogenetic development, which are directed towards the development of functioning adults. These processes are remarkably plastic and resilient to perturbation. If the genome actually were a set of blueprints or instructions for building a body, as some modern metaphors have it, the slightest perturbation would throw off the end result. Any embryo that could not be built to fit the determinate design would be non-viable. But in fact functioning adults can develop in an indefinitely large number of ways. The goal-directedness seen in developmen-

tal plasticity renders the concept of *species design* highly suspect. Development yields adults that *function*, but not adults that *function identically*. Functional diversity is a product of developmental plasticity.

The processes of ontogeny bring about the functional integration of the organism. As various body parts and systems develop, they adjust to each other. This integration occurs during the development of every organism, whether the organism is destined to be statistically typical or atypical of its species. The lens of the eye is not determined to develop in the location it does by its position on some genetic blueprint. Rather, the already-formed optic vesicle induces the ectoderm that overlays it to differentiate into the lens (after an earlier and more complex series of tissue interactions) (Gilbert, 1997, pp. 665*ff*.). If some trauma happened to relocate an optic vesicle to an unusual position on the head, lens induction would still proceed and result in a functioning eye. A more familiar aspect of developmental plasticity is the ontogenetic adaptation of an organism to its external environment. Development of use-enlarged muscles and protective calluses are customary examples of this kind of phenomenon.

These facts of developmental biology do not conclusively refute functional determinism. But they do make it seem unnecessary. A non-typical but viable phenotype is not *broken* by its failure to comply with some imagined blueprint for its species. It will function anyhow, in spite of its atypicality. It will owe its function to the same developmental processes of integration and adaptation responsible for the function of typical organisms of its species. Section 5 will list several examples of developmental plasticity that challenge functional determinism. I will later argue that the kind of functional diversity that follows from developmental plasticity is also an ordinary part of everyday life.

5. Examples of Developmental Plasticity

5.1. Slipjer's Goat

In the 1940s the biologist E. J. Slipjer studied a goat that was born without forelegs (Maynard Smith, 1975, p. 317; Rachootin and Thomson, 1981, p. 184). The goat learned to walk bipedally, showing that individuals of the same species can perform a function like walking using different means. But this is not the whole significance of the example. Slipjer's goat had many other deformities (relative to the statistical norm) in its skeletal and muscular anatomy. It had an S-shaped spine, an atypically broad neck, many atypically shaped bones and atypically positioned muscles. Its thorax was oval shaped, unlike the V-shaped cross section of the typical goat. By this census of 'abnormalities' it was a radical departure from its species design, and each abnormality pulls it further from the norm. By the species design criterion of goal-directedness, Slipjer's goat was a notable failure. By the developmental criterion it was a roaring success. The goat's skeletal and muscular abnor-

malities were, each of them, adaptively suited for life as a biped. They mimic the body conformation of kangaroos and humans.

Slipjer's goat illustrates the inadequacy of the metaphor of the genetic blueprint. Ontogenetic processes are epigenetic. The genome provides developmental resources for ontogeny; it is not a preformed image of the adult body (Nijhout, 1990). 'No one would maintain that goats have genes for developing an S-shaped spine, "just in case". What we see here is a basic mammalian potential emerging from the self-righting properties of the skeleto-muscular systems of all mammals, and the sort exploited by our hominid ancestors' (Rachootin and Thomson, 1981, p. 184). Many of the mechanisms that gave rise to the bipedal goat are well understood. 'These secondary modifications occurred because muscles which are used grow bigger, tendons grow along lines of tension, bone grows along lines of compression, and so on. The relevance of such developmental flexibility is that a single major change—for example the loss of the forelegs—instead of being a disaster may be compatible with life' (Maynard Smith, 1975, p. 317).

It is important to recognize that the self-righting properties of the mammalian developmental system are not emergency measures that only kick in when pathology is present. They are exactly the same processes involved in the generation of more typical quadrapedal goats and bipedal humans under different circumstances. Without these morphogenetic processes, well-functioning mammals would never develop at all. Biological 'types' are unified not by the functional identity of their eventual phenotypes, or the common blueprint from which they were built. Rather they are unified by their shared developmental processes. These processes generate phenotypes that are functionally diverse, both between and within species.

5.2. Is Your Brain Really Necessary?

Hydrocephaly can lead to profound physical and mental disabilities. A backup of cerebrospinal fluid causes the ventricles of the brain to balloon to many times their usual size. The resulting pressure leads to enlargement of the cranium and/or reduction in the volume of brain tissue. In the most severe category, ventricle expansion fills 95% of the cranium. This category includes some profoundly disabled people. But half of this severely affected group has IQs over 100 (Lewin, 1980, p. 1232). Usually associated with spina bifida, hydrocephaly can also occur subclinically in people who show no signs of abnormal function. The people in the subclinical category have heads of average or slightly above average size. In an article entitled 'Is Your Brain Really Necessary?' Roger Lewin describes a University student in the UK who has an IQ measured at 126, a normal social life, and 'virtually no brain'. He was tested only because his professor was familiar with a colleague's ongoing study of subclinical hydrocephaly, and the student had a large head. The student was functionally indistinguishable from his colleagues, but had no more than 10% of the average person's brain tissue. Accounts of similar phenomena are common in medical literature. '[A] substantial proportion of

patients appear to escape functional impairment in spite of grossly abnormal brain structure' (*ibid*.). The mode of function of these persons is statistically 'abnormal', even though their level of performance is statistically average.

5.3. Nerve Crossing

The two prior cases show the extreme plasticity of early development. As ontogeny progresses, alternative pathways are reduced and flexibility decreases. But it never disappears. Learning and healing are ontogenetic processes that occur throughout the lifetime of many organisms. There is evidence that primates, and especially humans, have a greater degree of some kinds of developmental plasticity than other mammals. Experiments have shown that non-primate mammals adapt poorly to the experimental surgical reversal of nerve attachments to muscles. But monkeys are able to adapt to the surgical reversal of flexor and extensor muscles in their forelimbs (Brinkman and Porter, 1983). Humans adapt extremely well to the surgical transposition of nerves between muscles. This is a common procedure in cases of disability and injury. The result is often a high level of performance based on a very atypical mode of innervation.

5.4. How to Handicap a Basketball Player

The sport of wheelchair basketball began during World War II and has steadily increased in popularity and competitiveness. Variation in physical ability among players is extensive, with large differences in arm and abdomen musculature, and in upper body balance in two planes. In order to allow fair competition among players with different physical abilities, a system of ranking of players was devised. Rankings go from 1 to 3 points, with more points assigned to players with more upper body control. Teams are allowed to have no more than a specified number of ranking points on the court at one time. In this way a skilled but more disabled player can be of more value to a team than a less disabled player. (This is 'handicapping' in the original sporting sense of the term. Less disabled players have to carry the handicap of a higher point ranking.) A medical committee originally administered the ranking system, with medical specialists assigning ranks based on physical examinations of each athlete. Athletes were ranked by their usable musculature, based on the assumption that athletes with identical musculature would function identically. The results were unsatisfactory. Medical assessments of the athletes did not match their performance on the court. Athletes that were judged equal in physical ability by the doctors were seen to differ significantly by the players and coaches. With much resistance from the medical committee, the sport switched over to a system of assessment based on observation of actual on-court performance (Craven, 1990). The assumption that *mode* of function determined *level* of performance was falsified. Different athletes achieved different performances with the same musculature. For example, some athletes with a given abdominal muscular loss achieved balance in the lateral plane, and some did not. Conversely, athletes who achieved the same function did so by different modes.

Athletes who possessed the typical musculature for achieving lateral balance did so in a different way from those with atypical musculatures. Clearly the athletes with high function but low musculature were achieving their performance via a different mode from their similarly-ranked but differently-muscled competitors.

5.5. Signed Languages

Since about 1960 two major innovations have modified our understanding of human language. The first, begun by Noam Chomsky (1966), is the notion that human natural languages are extremely highly structured in hierarchical levels of organization, and are acquired by means unlike the learning of other human skills and abilities. Chomsky and others believe that major aspects of language learning are hardwired into our cognitive equipment. The second innovation, begun by William Stokoe (1960), is the realization that the sign languages used within many deaf communities are themselves natural human languages. They are not mere pidgins, or signal systems, or substitutes for 'real' (spoken) language. They have the full structural complexity, and the cognitive and expressive powers of spoken languages. It has even been shown that brain injuries that cause certain kinds of aphasias in spoken languages have similar affects on signed languages (Poizner *et al*., 1987).

It has not been widely recognized what an incongruous pair of doctrines these two are. The ability to learn language is as innate to, and as distinctive of the human species as any biological trait. Modern linguistic analyses of spoken languages are strongly tied to phonology, the perceptual analysis of spoken sounds. Nevertheless, it turns out that human language can be manifested in a completely distinct sensory and performance modality, namely manual gesture. Unless modern linguistics is grossly mistaken, there exists *some* human capacity specific to the learning of language. In statistically typical humans (those of average hearing abilities growing up in typical linguistic environments) this capacity gives rise to a spoken language. But the capacity cannot be purely the capacity for *spoken* language, because the same capacity also gives rise to signed languages in a minority of people (those growing up in signing environments). It is as if we were to discover a population of honeybees that were unable to secrete wax, but built fully functional honeycombs out of clay they dredged from river bottoms.

There are two ways of interpreting this anomaly. First, the language capacity might actually be innately and evolutionarily tied to vocal sound, as has usually been assumed. If this is so, then humans have an astonishing flexibility in applying an innate capacity to a domain that is foreign to it. Second, the language capacity might not be innately tied to vocal sound at all, but be abstract enough to apply indiscriminately to signed or spoken language. This has very intriguing evolutionary implications (Armstrong *et al*., 1994). Neither case gives any succor to functional determinists. In the first case, developmental plasticity greatly dilutes any

claim of the privilege of normality for spoken language. In the second, both language modalities are equally 'normal' insofar as biology is concerned.

Ian Hacking dates the origin of the concept of normality to the rise of statistics in the nineteenth century. He says that normality '. . . uses a power as old as Aristotle to bridge the fact/value distinction, whispering in your ear that what is normal is also right' (Hacking, 1990, p. 160). There was no time lost in exploiting this shortcut between facts and values. Many nineteenth-century educators of deaf people used the new concept of statistical normality to suppress a highly functional minority adaptation (Baynton, 1996, Ch. 6). In certain schools deaf children were forbidden to use sign. They were trained according to a doctrine called 'oralism'. They were taught to lip-read and to speak aloud, two skills that are extremely difficult to learn and of marginal value for most profoundly deaf individuals. Biology, then as now, gives no legitimacy to this practice. Lip-reading and speaking may be useful to a deaf signer in a crowd of English speakers, but no more so than sign would be useful to a non-signing English speaker in a crowd of signers.

In summary, the goal-directed processes of biological development are not finely tuned towards the production of functionally identical species members. Their inherent flexibility can be expected to generate a rich diversity of functional modes.

6. Physiology and Anatomy

Recall that Boorse and Wachbroit claim that contemporary medicine and physiology imply functional determinism, even if it is not implied by evolutionary biology. We will now consider dissenting opinions, two criticisms of the concept of normality taken from within the sciences of physiology and anatomy. Each concludes that the range of functional variability is too wide to justify a concept of biological normality.

Jiří Vácha has written a series of papers on the notion of normality of physiological function (Vácha, 1978, 1982, 1985). Like other authors (e.g. Davis and Bradley, 1996) Vácha emphasizes the multiplicity of meanings for 'normality' in medicine, and especially the 'intermingling of normality in the statistical and value sense, which is typical of current practice' (Vácha, 1978, p. 823). He considers the common use of 'normality' to be typological and idealistic in that it assumes that 'the frequent [is] the [normal] and, besides that, the healthy' (Vácha, 1982, p. 730). A part of this idealism is the unstated assumption that health and illness are distinct alternative body states. In fact there is a multidimensional continuum of states of health. The health/illness and normal/abnormal dichotomies are illusions. A high degree of variability exists among individuals on any physiological measurement, with even the most extreme values found within healthy individuals. Extreme values of physiological parameters, associated with disease in some individuals, are compensated for in others. Indeed, the constellation of other parameters in an individual may directly require the extremeness of a particular character for good health. 'Immense variability has been found in the manner in which individuals in

the population attain health' (Vácha, 1985 p. 339). Functional integration at the physiological level gives rise to a range of differently functioning, but comparably successful physiological systems. Physiological analogs to Slipjer's goat are walking among us.

Vácha, following the German medical theorist L. R. Grote, suggests that the concept of species normality be replaced by a concept of individual normality or 'responsiveness'. Rather than testing a patient's statistical conformity, medical judgments should assess 'that congruence between physiological performance of the individual and the performance necessary for him. . . . An individual may be healthy—responsive—without regard to the quality or quantity of the morphology and function which the statistical norm would wish to prescribe for him' (Vácha, 1978 p. 826). Notice that the concept of responsiveness (individual normality) abandons the statistical and comparative basis of normality, replacing it with an assessment of the relation between individual performance and needs. There is no need for a species design.

Variation in human anatomy is the subject of a remarkable Internet document-in-progress entitled *Illustrated Encyclopedia of Human Anatomic Variation* (Bergman *et al*., 1992-1998). Part I, 'The Muscular System', is complete at this writing. Additional sections on the skeletal, cardiovascular, nervous, and organ systems are in progress. The document summarizes medical reports since antiquity on observed variations in human musculature, categorized by specific muscle and muscle group. There is no easy way to summarize the richness of variation; one must browse the reports on individual muscle groups. These authors show no patience with labels of normality. Any variation consistent with viability is accepted as 'normal' '. . . however imperfect or monstrous by Galen's and Vesalius' definition'.

> Many or most variations are totally benign. . . . Some of these variations may seriously compromise parts of the muscular, vascular, nervous, skeletal and/or other organ systems. . . . What we are trying to convey to interested readers is that the things we describe here are 'normal' even though they may differ from the mean or usual. They are found in 'normal' long-lived individuals, and they are statistically (for the most part) predictable. Man is not machine-made but rather more subjectively fashioned with many developmental and environmental factors intervening in the process. (Bergman *et al*., 1992-1998)

The *Encyclopedia* gives no information on functional differences among the variants, although such variation surely exists. One would expect the large variability of muscle and tendon positioning in the hand, for example, to correlate with level of strength or dexterity in certain kinds of manual tasks. Do better violin players tend to have a common configuration of hand musculature, or one that is unusual? Do people with the best and worst penmanship tend to have certain configurations? These questions seem meaningful, but they do not draw us towards a robust concept of 'normality'. We always knew that people varied in their manual abilities, and now we know that they differ in musculature as well. Perhaps muscular variation maps onto the variation in manual skills. If so, so what? Skills in penmanship and

musicianship are so various that no one seriously thinks there are 'normal' ranges here. There is no reason that discovering a biological explanation for variations in functional performance should cause us to declare certain performances abnormal.

The views of Vácha and the *Encyclopedia* are a challenge to Boorse and Wachbroit's claims that functional determinism is implied by contemporary biomedical science. If medical textbooks emphasize average or typical cases, there may well be pragmatic reasons to do so. It would be a mistake to infer from this that diversity constitutes abnormality. Nevertheless, functional determinism remains an underlying assumption of certain discussions of health care ethics, to which we now turn.

7. The Reification of Abnormality in Health Care Ethics

Human beings are distinctive among species in their extensive use of tools and in the degree to which they modify their environment. A weak person using an atlatl can throw a spear farther than a strong person without one. A weak person can walk faster on pavement than a strong person can walk on a sandy beach. Such improvements are entirely typical of human beings, in the statistical sense that everyone does them. Tool use and environmental design change the modes and levels of human function. From a broad biological perspective these changes can be seen as an extension of the principle of functional integration. Richard Dawkins has suggested that tools and environmental modifications could be seen as an organism's *extended phenotype* (Dawkins, 1982). We saw in Section 4 that an individual does not possess in its genome a preformed determinate design, but rather develops its adult phenotype (and its functional potential) through ontogenetic growth processes that include functional integration and adaptation. The present point is that even if we assume a fixed bodily phenotype, the functional potential of an individual human being is not fixed. The speed at which a given human walks and the distance she can throw a spear depend on the surfaces and tools available to her, her 'extended phenotype'. Nevertheless, the notion of a fixed species design with determinate limits on functional potential still plays a dominant role in discussions of health care ethics.

Norman Daniels argues that the preservation and restoration of normal function is a primary goal of health care. '[T]he kinds of [health care] needs picked out by reference to normal species functioning are objectively important because they meet this high-order interest persons have in maintaining a normal range of opportunities' (Daniels, 1987, p. 301). The goal of normality is seen as especially legitimate, because it is fixed by nature rather than by human convention; '. . . we can take as fixed, primarily by nature, a generally uncontroversial baseline of species-typical [i.e. normal] functioning' (*ibid*., p. 303). Daniels proposes three levels of health care provision. The first is preventive health care. The second is curative and rehabilitative—returning people to species-normal functioning. The third level is services for the people who cannot be normalized, 'extended medical and social support services for the (moderately) chronically ill and disabled and the frail eld-

erly' (Daniels, 1985, p. 48). Silvers points out that Daniels's schema implies that mode of function has a higher priority than level of performance, apparently because mode of function receives its objective validation from nature itself (Silvers, 1998, p. 101).

Daniels does not actually argue for the reality of species-normal functioning. He cites Boorse, and accepts it as an obvious fact. He goes beyond Boorse in one important respect: the linkage between normality and opportunity. Abnormals have reduced opportunity, and so maintenance of normality is maintenance of opportunity. Health care sustains normality, and normality sustains opportunity. Normality is the crucial objective link between health care and opportunity. And since normality is determined by objective science, judgments based on it carry a high authority.

The link between normality and opportunity may help us recognize the hierarchical level at which biological normality is conceived to operate. A person with unusually low blood pressure, or an unusual muscle configuration in the hand, may experience no direct loss of opportunity. So a socially oriented functional determinist like Daniels might not be concerned about the variability documented in Section 6. But people who are blind or paraplegic do experience a reduction of opportunity. It is probably this level, the level of 'basic personal abilities' that draws the functional determinist's attention (Amundson, 1992, p. 107). All *normal* humans can see and walk. Those whose opportunities are diminished by their inability to see and walk have their own abnormality to blame. Their status as abnormals is a fact of nature; the associated opportunity loss seems likewise to be entirely natural.

The tight linkage between opportunity and normality reappears in Dan Brock's analysis of the concept of quality of life. '[Q]uality of life must always be measured against normal, primary functional capacities for humans . . .' (Brock, 1993, p. 308). This is taken to follow from Daniels's position that the 'normal opportunity range' is only available to functionally normal humans. One might think that quality of life would be measured by the satisfaction and fulfillment actually experienced by those living those lives. This would allow an empirical test of the identification of quality of life with functional normality. If the linkage is empirically correct, then functionally atypical people would report low qualities of life. Unfortunately, the data do not support this identification. Atypical people typically report a high quality of life. There is a great deal of empirical evidence that people with even serious disabilities report a quality of life averaging only slightly lower than that reported by non-disabled people. Physicians in particular estimate the quality of the lives of their disabled patients to be much lower than do the patients themselves (Bach and Tilton, 1994).

Brock is aware of the mismatch between biological normality and the reported quality of people's lives. If we were discussing a genuinely empirical hypothesis, such a mismatch would be taken as evidence that one's biological normality is irrelevant to the quality of one's life. After all, if happiness doesn't correlate with

normality, then normality doesn't measure quality of life. But Brock argues exactly the reverse. Since normality doesn't correlate with happiness, happiness itself does *not* measure quality of life! In order to protect from refutation the link between normality and quality of life, Brock distinguishes *ad hoc* between the *subjective* and *objective* aspects of quality of life (Brock, 1993, p. 306). Subjective aspects are the degree of happiness and satisfaction that a person experiences. Objective aspects include the person's own objective abnormality and the opportunity associated with it. Abnormal people who report a high quality of life are simply mistaken about the quality of their own lives. Their quality of life is merely *subjectively* high. Objectively, it is low.

How does Brock account for the mismatch between high subjective quality and low normality-defined ('objective') quality? He offers only one explanation. Functionally abnormal people who report a high quality of life have lower expectations than functionally normal people. Lowered expectations are more easily satisfied, and the easy satisfaction of low expectations yields a high subjective quality of life. This, to Brock, is not *real* quality of life. 'To be satisfied or happy with getting much less from life, because one has come to expect much less, is still to get *less* from life or to have a less good life' (Brock, 1993, p. 309). One is reminded of John Stuart Mill's account of how higher pleasures are qualitatively and not just quantitatively better than lower pleasures; '. . . better to be Socrates dissatisfied than a fool satisfied' (Mill, 1996, p. 526).

I do not deny that people labeled as abnormal have a reduced range of opportunity. And I agree that equality of opportunity is an important moral value. But the discussion of opportunity takes a very different form in the context of supposed biological abnormality than in other contexts. Racism and sexism, for example, cause very serious reductions of opportunity. Moral discussion of these problems centers on how opportunity should be restored to the disadvantaged groups, by changing social institutions if necessary. We are well past the time when academic discussion of race and sex was centered on rationalizations of how the disadvantages experienced by certain races and genders were caused by nature itself. But the normality discussions do just that. The abnormals are said to be disadvantaged by nature itself. If a black woman today considered herself to have a fulfilling life, would a moral philosopher be likely to suggest that her happiness only results from lowered expectations, and she is really getting *less* from life than a white male? I doubt it. But the abnormals can still receive this patronizing treatment.

The present unequal distribution of opportunities among people with varying biological traits can only appear to be fixed by nature if we ignore the fact that *all* human beings use tools and live in built environments, and that the design of tools and environments is an outcome of human choices. Given the appropriate technology and environment, blind people can read and paralyzed people can be mobile. The disadvantage that attaches to blindness and paralysis derives not from the atypicality of one's biology, but from the absence of appropriate tools and

environments. This simple fact goes unnoticed by philosophical commentators on normality. We consider the social prohibition of hiring based on race or sex to be a remedy for the disadvantages caused by racism and sexism. But we do not consider the social provision of appropriate tools and environments to be a remedy for the disadvantages of abnormal people. Why not? In Section 8 I will argue that it is because the tools and environments that enable atypical people to function at a high level are *themselves* stigmatized by social prejudices against the conditions they ameliorate.

The concept of normality, and not the concept of function, controls current thought about the disadvantages caused by biological atypicality. If we thought merely about *level* of functional performance, rather than the mode, fashion, or style of function, the disadvantages of disability would not seem so natural and inevitable. High levels of function are possible for very atypical people when they use atypical modes of functioning. A concern with functional normality is less a concern with the level of performance than with cosmetic aspects of functional mode. The widespread fascination with normality of functional mode is itself a hindrance to functional performance.

8. Unfashionable Function

During the past three decades the concept of disability has undergone critical evaluation and reconceptualization. The customary way of thinking about disability is based on what is now called the Medical Model. Disability is thought of as a biomedical condition of an individual, an abnormality that is naturally associated with disadvantages. Disability activists began in the 1970s to think of disabled people as an oppressed minority, and to demand civil rights parallel to the rights earlier won by 'racial' minorities and women (Eisenberg *et al*., 1982). As a part of this movement, the Medical Model is being replaced by the Social Model of disability (Oliver, 1990; Shakespeare *et al*., 1996). It was long recognized that the disadvantages experienced by people with disabilities were at least partly caused by the social context in which they lived, if only by the widespread negative stereotypes of disabled people. The Social Model makes disability *entirely* an issue of social context, arising as it does from the disabling ways in which certain kinds of human variation are dealt with in society.

Section 7 asserted that high levels of function were possible for atypical people using atypical modes. It intimated that functional deterministsw were more concerned with the cosmetic issue of the mode of function than the pragmatic issue of the level of function. If this is so, the aversion to atypical modes of function is a simple prejudice, and not an objective scientific assessment. What evidence is there that functional mode is favored over level of performance?

There is abundant social evidence that atypical modes of function are stigmatized. Many disabled people attempt to hide their disability. Some refuse to use tools that would make their disability more apparent, even though the tools would

greatly enhance their level of function. A large population of survivors of the polio epidemics of the 1940s and 1950s are now experiencing Post-Polio Syndrome, a condition that causes increased weakness and discomfort with exertion. Many were *passers* for most of their life (a term significantly borrowed from the racial context to mean a disabled person who passes for non-disabled). It is common wisdom in this group that most passers will resist the use of new assistive devices (canes, crutches, wheelchairs, or ventilators, depending on the nature and extent of the paralysis) even though their waning strength would make the devices extremely useful. These people voluntarily suffer increasing pain and limitations on their activities just to avoid acknowledging their muscular weakness. Exclamations of joy can be seen on Internet lists as individuals finally give in and discover how much the adaptive equipment liberates them and increases their level of function. The same is true of other gradually acquired disabilities, such as resistance to the use of a hearing aid or a white cane. Publicly acknowledging one's own disability is often a personally momentous 'coming out' similar to acknowledging one's homosexuality, or one's unacknowledged ethnic background.

The fact that individuals try to hide their disability has usually been interpreted patronizingly, as evidence of the failure to accept one's own limitations. It should instead be seen as a recognition on the part of disabled people of a deep social prejudice against them. Cosmetic normality at the cost of functional performance has been an acknowledged goal of many rehabilitation programs. As cited above, many schools for deaf children forbade the use of sign among their students, just as government schools for Native Americans forbade the use of indigenous languages. Oralism produced a lower level but more cosmetically normal performance. (It also allowed somewhat more integration into hearing society. But integration could equally well have been achieved by requiring hearing people to learn sign language!) A second example is the rehabilitation program in Canada in the 1960s for the babies affected by thalidomide. Many of them had no legs, or legs that would not function in walking. The children were strapped onto specially designed upright platforms that looked vaguely like legs. With great effort they could teeter the platforms back and forth and slowly 'walk' forward. The children were forbidden to use wheelchairs. Only when they were old enough to refuse to cooperate were they were allowed to use the devices that maximized their performance (WBGH, 1989, p. 8*ff.*). Cosmetic normality dominated their 'rehabilitation' while their functional performance was artificially suppressed. Now that they are free to function atypically, many have become high functioning adults.[2]

A third example is the education often given to children with autism. Much of the education is oriented towards getting the child to *appear* normal. Many people

[2]Brock comments on these adults, saying that their high level of function makes it 'problematic even to characterize those affected as disabled' (Brock, 1993, p. 307). He notices no conflict with the doctrine of biological normality, or its supposed linkage with opportunity and quality of life, even though the mode of function of the thalidomide adults is extremely atypical of the human species.

with autism engage in 'stimming', small repetitive self-stimulations like rocking while sitting, tapping one's face, or flipping fingers in front of one's eyes. These visibly abnormal behaviors are strongly discouraged by most educators. But it turns out that stimming is often functionally beneficial to people with autism. It reduces the chaos they experience, chaos created by their heightened sensory sensitivity in 'normal' environments, and allows them to concentrate on particular features of the environment (e.g. the voice they are listening to). Environments 'normal' to non-autistic people can create sensory overloads in autistic people. A non-stimming autistic person may be more cosmetically normal, but able to function only at a lower level. Stimming is now becoming a civil rights issue to some autistic activists, just as sign language is to deaf activists and curb ramps to mobility-impaired activists (Sinclair, 1997). Atypical people can function at their highest level using atypical modes of function. Mainstream concerns with normality are directed at typicality of functional *mode*, and are antagonistic to the functional performance of atypical people.

Wheelchairs are another example of the stigmatization of an unfashionable performance mode. As in the thalidomide case, many people with mobility impairments are taught not to use wheelchairs if there is any way to avoid it. This is true even if avoiding the wheelchair means walking with difficulty, pain, and very low efficiency. Depending on the environment and the task at hand, a wheelchair user can function at or above the level of a person with bipedal mobility. The world's record for a marathon race is 45 minutes faster for a wheelchair user than for a runner. Nevertheless, the phrases 'wheelchair-bound' and 'confined to a wheelchair' are used as synonyms for paralysis. The irony is that wheelchairs are tools of mobility, not confinement devices. The people who are genuinely confined are paraplegic people who do not *have* a wheelchair, or who have one but live in an environment filled with barriers to its use. The stigmatization of wheelchairs is another example of a higher level of performance sacrificed to cosmetic normality. Upright walking is socially approved over wheelchair use, no matter how painful and inefficient the walking.

This is not a mere popular prejudice from which academics are immune. Brock discusses three assessment instruments designed to measure what he describes as 'functions of the "whole person"' (Brock, 1993, p. 298). They actually measure something quite different. One such instrument has a scale for 'mobility' and a scale for 'physical activity'. These scales illustrate the bias towards fashionable normality of mode over level of functional performance. The 'physical activity' scale scores 4 points for walking freely, 3 points for walking with limitations (using a cane or crutches), and 2 points for moving independently in a wheelchair (Brock, p. 303). A walking person scores higher in physical activity than a person who uses a wheelchair, even if the walker manages only slow and painful steps and the wheelchair user is a marathon racer. Cosmetic normality wins over functional performance. Recall that Brock refers to these very measures as the 'objective'

components of genuine quality of life. The post-polio population is filled with people who can attest that wheelchair use improves not only their level of physical activity, but also their quality of life. The wheelchair is a stigmatized tool, and the stigma is reinforced by the doctrine of biological normality.

The 'mobility' scale of this instrument awards 5 points for using public transportation alone, 4 points for requiring assistance to use public transportation, and 3 points for needing assistance to go outside (*ibid*.). Consider how a physically fit paraplegic wheelchair user would score on this assessment. If there were barriers between his living quarters and the street (e.g. stairways without elevators), he would score 3. If there were no such barriers but his city's public transportation was inaccessible to wheelchairs, he would score 4. If his living quarters and his public transportation were both wheelchair-accessible, he would score 5. The differences in score depend not on the biological traits of the person, but on the environment he is living in. Does this scale measure 'functions of the whole person'? Not in the least. It measures the accessibility of the person's environment. To conceive of these criteria as measuring the functional traits *of a person* is the crudest of prejudices. The design of the environment is the cause of the disadvantage. The doctrine of biological normality obscures this cause.

9. Conclusion

Causal attribution is a complicated thing. We pick out one antecedent event or condition and baptize it as *the cause* of a phenomenon. Different perspectives, different theoretical orientations, or different prejudices can lead to the baptism of different antecedent events or conditions as *the cause*. The Social Model of disability never identifies the biomedical condition of a person as *the cause* of that person's disadvantages. The causes of disadvantage are always identified in the environment and the social context. A critic might dismiss this approach as politically motivated and therefore not scientifically objective.

But consider the alternative. Functional determinism, the doctrine that biological normality is a part of the real natural world, is presented as an objective scientific claim. Philosophers and medical practitioners alike have used the category to conclude that the disadvantages of disabled people result from their own abnormality; they have only themselves (and nature) to blame. Is *this* assessment scientific and objective? Or does it merely reflect a preference for 'ways of doing things that are preferred by the dominant classes and to which we have therefore become accustomed' (Silvers, 1998, p. 108). If the latter, then 'policies of normalizing threaten not to equalize but to preserve existing patterns of functional dominance and privilege' (*ibid*.).

When an inaccessible environment causes the confinement of a wheelchair user, the abnormality of the wheelchair user is identified as the cause of the confinement. The doctrine of biological normality (Boorse and Wachbroit), the linkage of normality to opportunity (Daniels) and thence to quality of life (Brock) rationalizes

this assessment. The opportunity losses of abnormal people are theorized to be not only natural and obvious, but morally innocuous.

In past years, versions of biological determinism have buttressed racist and sexist doctrines. Celebrated for their scientific objectivity, they had little objective biological foundation. Their plausibility was enhanced by their congruence with the social prejudices of their time. Functional determinism, the reificiation of functional normality and abnormality, is typical of this genre. The ideology it supports and is supported by has been labeled 'ablism', the chauvinism of the non-disabled. It has little else to recommend it.

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References

American Association of Physical Anthropologists (1996) 'Statement on Biological Aspects of Race', *American Journal of Physical Anthropology* **101,** 569–570.

Amundson, R. (1992) 'Disability, Handicap, and the Environment', *Journal of Social Philosophy* **23,** 105–118.

Amundson, R. (1998) 'Typology Reconsidered: Two Doctrines on the History of Evolutionary Biology', *Biology and Philosophy* **13,** 153–177.

Armstrong, D. F., Stokoe, W. C., and Wilcox, S. E. (1994) 'Signs of the Origin of Syntax', *Current Anthropology* **35,** 349–358.

Bach, J. R. and Tilton, M. C. (1994) 'Life Satisfaction and Well-Being Measures in Ventilator Assisted Individuals with Traumatic Tetraplegia', *Archives of Physical Medicine and Rehabilitation* **75,** 626–634.

Baynton, D. C. (1996) *Forbidden Signs: American Culture and the Campaign against Sign Language* (Chicago: University of Chicago Press).

Bergman, R. A., Afifi, A. K., and Miyauchi, R. (1992-1998) *Illustrated Encyclopedia of Human Anatomic Variation* (Iowa City: University of Iowa Health Care, http://www.vh.org/Providers/Textbooks/AnatomicVariants/AnatomyHP.html).

Boorse, C. (1975) 'On the Distinction between Disease and Illness', *Philosophy and Public Affairs* **5,** 49–68.

Boorse, C. (1997) 'A Rebuttal on Health', in J. M. Humber and R. F. Almeder (eds), *What is Disease* (Totowa, NJ: Humana Press), pp. 3–134.

Brinkman, C. and Porter, R. (1983) 'Plasticity of Motor Behavior in Monkeys with Crossed Forelimb Nerves', *Science* **220,** 438–440.

Brock, D. W. (1993) *Life and Death* (Cambridge: Cambridge University Press).

Chomsky, N. (1966) *Cartesian Linguistics: A Chapter in the History of Rationalist Thought* (New York: Harper and Row).

Craven, P. L. (1990) 'The Development from a Medical Classification to a Player Classification in Wheelchair Basketball', in *Adapted Physical Activity: An Interdisciplinary Approach* (Berlin: Springer-Verlag), pp. 81–86.

Daniels, Norman (1985) *Just Health Care* (Cambridge University Press: Cambridge).

Daniels, N. (1987) 'Justice and Health Care', in D. Van deVeer and T. Regan (eds), *Health Care Ethics* (Philadelphia: Temple University Press), pp. 290–325.

Davis, P. V. and Bradley, J. G. (1996) 'The Meaning of Normal', *Perspectives in Biology and Medicine* **40,** 68–77.

Dawkins, R. (1982) *The Extended Phenotype* (Oxford: Oxford University Press).

Dobzhansky, T. (1962) *Mankind Evolving* (New Haven: Yale University Press).

Eisenberg, M. G., Griggins, C., and Duval, R. J. (eds) (1982) *Disabled People as Second-Class Citizens* (New York: Springer).
Gilbert, S. F. (1997) *Developmental Biology, Fifth Edition* (Sunderland, MA: Sinauer Associates, Inc).
Hacking, I. (1990) *The Taming of Chance* (Cambridge: Cambridge University Press).
Hull, D. L. (1986) 'On Human Nature', *PSA 1986, Vol. 2* (East Lansing, MI, Philosophy of Science Association), pp. 3–13.
Lenoir, T.(1982) *The Strategy of Life* (Chicago: University of Chicago Press).
Lewin, R. (1980) 'Is Your Brain Really Necessary?', *Science* **210,** 1232–1234.
Lewontin, R. C. (1995) *Human Diversity* (New York: W.H. Freeman).
Marks, J. B. (1995) *Human Biodiversity: Genes, Race, and History* (New York: Aldine de Gruyter).
Maynard Smith, J. (1975) *The Theory of Evolution, Third Edition* (Cambridge: Cambridge University Press).
Mayr, E. (1980) 'The Role of Systematics in the Evolutionary Synthesis', in M. Ernst and W. Provine (eds), *The Evolutionary Synthesis* (Cambridge: Harvard University Press), pp. 123–136.
Mill, J. S. (1996) 'Utilitarianism', in Joel Feinberg (ed.), *Reason and Responsibility*, 9th edition (Belmont, CA: Wadsworth), pp. 522–534.
Nijhout, H. F. (1990) 'Metaphors and the Role of Genes in Development', *BioEssays* **12,** 441–445.
Oliver, M. (1990) *The Politics of Disablement* (Basingstoke: Macmillan).
Poizner, H., Klima, E., and Bellugi, U. (1987) *What the Hands Reveal about the Brain* (Cambridge, MA: MIT Press).
Rachootin, S. P. and Thomson, K. S. (1981) 'Epigenetics, Paleontology, and Evolution', in G. G. E. Scudder and J. L. Reveal (eds), *Evolution Today* (Pittsburg: Hunt Institute).
Silvers, A. (1998) 'A Fatal Attraction to Normalizing', in Erik Parens (ed.), *Enhancing Human Traits: Ethical and Social Implications* (Washington, DC: Georgetown University Press), pp. 95–123.
Shakespeare, T., Gillespie-Sells, K., and Davies, D. (1996) *The Sexual Politics of Disability: Untold Desires* (London: Cassell).
Sinclair, J. (1997), personal e-mail, September 13, 1997.
Stiassny, M. L. J. and Meyer, A. (1999) 'Cichlids of the Rift Lakes', *Scientific American* **280,** 64–69.
Stokoe, W. C. (1960) *Sign Language Structure: An Outline of the Communication Systems of the American Deaf*: Studies in Linguistics Occasional Papers 8.
Vácha, J. (1978) 'Biology and the Problem of Normality', *Scientia* **113,** 823–846.
Vácha, J. (1982) 'The Problem of So-called Normality in Anthropological Sciences', *Anthropos* **22,** 73–85.
Vácha, J. (1985) 'German Constitutional Doctrine in the 1920s and 1930s and Pitfalls of the Contemporary Conception of Normality in Biology and Medicine', *Journal of Medicine and Philosophy* **10,** 339–367.
Wachbroit, R. (1994a) 'Normality as a Biological Concept', *Philosophy of Science* **61,** 579–591.
Wachbroit, R. (1994b) 'Distinguishing Genetic Disease and Genetic Susceptibility', *American Journal of Medical Genetics* **53,** 236–240.
WBGH, (1989) WBGH Educational Foundation, 'Extraordinary People', transcript of Frontline television program originally broadcast on May 2, 1989.
Wendell, S. (1996) *The Rejected Body: Feminist Philosophical Reflections on Disability* (New York: Routledge).