A comprehensive discussion of the ethics of caring for persons with the syndrome of dementia must be attentive to key issues that arise following the course of the underlying causative disease, which is most likely to be Alzheimer disease (AD). This analysis will therefore focus upon the progressive course of AD, although what is discussed pertains to dementia generally. Ethical issues beginning with prevention and diagnosis and concluding with a good dying are considered. A chronologically ordered discussion of these issues over the course of AD provides a practically useful framework.

In the tradition of Aristotle’s practical reasoning, I will present practical perspectives that admit of some exceptions based upon specific cases and cultural specifics. While ethics must be sensitive to both the One and the Many in a pluralistic society, there are issues that afford an ethically objective analysis, often based upon clear empirical data indicating significant burdens to the patient and few benefits, or vise versa. Other issues remain more subjective or culturally relative, such as the continuum between patient self-determination (autonomous decision making) and entrustment to family decision makers. Principles of clinical ethics include nonmaleficence (“do no harm” or, more realistically, minimize harms as far as possible and justify them by appeal to benefits), beneficence (act for the person’s good), respect for autonomy (honor competent decision makers, or their wishes as indicated while formerly competent, including wishes to entrust others with decision making), truthtelling, confidentiality, and distributive justice (fairness) in allocating resources.

AD PREVENTION

The prevention or delay of AD onset is an urgent priority. Medical science in this area is advancing. Biological susceptibility markers, coupled with preventive compounds, provide one major strategy. Ethically speaking, the benefits of primary prevention or delay of onset are obvious to all, and far superior to efforts to protract lives of the most severe dysfunction in the advanced stage of the disease. While the
ethical debate over therapeutic goals and pharmacology is complex, there must be a
general moral maxim to delay, prevent, stabilize, or cure dementia, but not to
protract life’s severe morbidity artificially in the late stages of disease when it if
necessary to speak of AD as a terminal condition (Post 2000).

In pursuing preventive or delaying strategies, it is important that clinical
usefulness of scientific advances not be exaggerated. There is, for example, no
clearly predictive test for late-onset AD. An apolipoprotein E ε4 allele on
chromosome 19 [apoE=protein; APOE=gene] was discovered in 1993 to be
associated with susceptibility to late-onset AD (after 60 years). A single ε4 gene
(found in about one third of the general population) is not predictive of AD in
asymptomatic individuals – i.e., it does not foretell disease. Among the 1% of
people with two of the ε4 genes, AD still may not occur. While this is the group
most at risk, no one can indicate whether AD is more likely to manifest in the
seventh, eight, or ninth decade of life, so that even those with two susceptibility
genes (4/4/) may not live long enough to become demented. All the major consensus
groups have recommended against susceptibility testing in asymptomatic individuals
because the data are not very useful, even if they hold some statistical significance
(Post et al. 1997). If additional susceptibility genes are found, this prevailing view
may change. At the moment, however, the only serious debate concerns the use of
APOE genotyping as an adjunct diagnostic test for those patients already presenting
with dementia. Robert N. Butler, editor of Geriatrics, urged clinicians to be cautious
about requests for susceptibility testing (Butler 1994). He emphasized that APOE
testing was not yet established as a diagnostic or predictive marker, that people
should avoid the emotional toll of thinking that the APOE genotype means they are
doomed after forgetting the car keys, and that discrimination in employment and
insurance was likely.

Would people want APOE susceptibility testing? Based upon information
obtained from Alzheimer’s Association focus groups, it is clear that very few
caregivers would elect to undergo susceptibility testing unless it were much more
predictively accurate than is currently the case, and unless it provided medically
useful information linked to effective preventive or delaying treatments. Where there
is interest in genetic testing for purely information reasons, family history already
provides adequate general data on susceptibility above that of the general
population. In the future, if genuinely predictive biological susceptibility markers
are found, and if preventive medications are established, presymptomatic testing
may become a standard of care.

However, it is not clear that treatment efficacy will ever be closely linked to
genotype, and it is unlikely that any effective preventive medication with no major
adverse side effects would be denied to anyone, regardless of genotype. Of course,
there is much debate about how to define therapeutic effectiveness in this area.

Genetic testing does provide precise predictive information in the context of the
three determinative or causal (disease) genes (located upon chromosomes 21, 14,
and 1) that were discovered over the last decade. These are autosomal-dominant
genes and pertain to early-onset forms of AD (i.e., usually manifesting between the