Mechanism of Arterial Infection by 
Chlamydia pneumoniae

To the Editor:

The article by Gieffers et al1 in the January 23, 2001, issue of 
Circulation reiterates the general belief that lungs harbor Chla-
mydia pneumoniae organisms that are continuously released into 
the bloodstream. It is suggested that these organisms are carried 
by lung macrophages, which repeatedly infiltrate and infect 
atherosclerotic lesions.

There are certain pathological features of human atheroscle-
rotic lesions that suggest that this mechanism may not be entirely 
correct. Chlamydia pneumoniae usually causes mild upper respira-
atory tract infections. If the organisms do indeed enter the lungs, 
then there is some difficulty in explaining how macrophages are 
dispersed into the bloodstream to infiltrate arteries. Lung mac-
rophages drain to the lymphoid tissue of the lung and to the hilar 
glands, not into the circulation. It should also be borne in mind 
that blood monocytes, not macrophages,2,3 infiltrate atheroscle-
rotic lesions. These blood monocytes are derived from spleen, 
lymphoid tissue, and bone marrow rather than from lung tissue. 
In fact, these cells cannot be derived from lung macrophages 
because lung macrophages contain anthracotic pigment, and no 
such pigment is noted in the cells that infiltrate the artery.

The pathology of the arterial lesion indicates that only 1 
infecetive episode is necessary. The infection usually occurs in 
adolescence or earlier. The organisms possibly may enter arteries 
via the vaso-vasorum, inasmuch as early lesions are seen to occur 
in the lower intima. In this region, C pneumoniae infects intimal 
smooth muscle cells, causing rupture of the cells with dispersion 
of organisms and infection of adjacent intimal tissue. A florid 
monocyte and T-lymphocyte infiltrate occurs in relation to this 
damage. Monocytes enter the subendothelial space, are trans-
formed into a phagocytic state,2,3 and are seen to ingest C 
pneumoniae organisms.4

With progression, necrosis and ulceration of the lesions occur. 
This is especially prominent in the aorta, where the surface is 
usually covered with open atherosclerotic ulcers. These open 
ulcerating lesions release necrotic tissue, together with cells and 
organisms, into the circulation. It is not necessary to look further 
than this for the source of infection or for the C pneumoniae–
containing macrophages in the circulation.

There are some unclear aspects of the mechanism by which C 
pneumoniae reaches the arteries from the respiratory tract,1 but 
because the infection occurs in childhood, the benefit to be 
derived from treating this aspect of the disease in later years is 
questionable. In adults, the central focus of treatment should be 
directed at the lesions in the arterial vasculature.5

Allan Shor, MB, ChB
Department of Pathology
National Center for Health
University of Witwatersrand
Johannesburg, South Africa

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Allan Shor

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