Sounds and murmurs have long been employed to qualitatively diagnose cardiovascular disease. However, quantitative diagnosis has been hindered by the lack of understanding of the sound generation and transmission mechanisms. Clinical phonoangiographic studies have shown that simple assumptions about low frequency sound transmission through tissue surrounding an artery are inadequate for obtaining meaningful quantitative diagnosis. Therefore, a theory is developed which relates internal turbulent flow in constricted peripheral arteries to the sound observed at the surface of the skin by means of assumptions of similarity and local axial homogeneity of the internal turbulence. It is found that the spectrum of pressure at the wall of the artery is related to the spectrum of the pressure at the surface of the skin by a filtering factor approximately proportional to $\omega^{-2}$. This arises not because of frequency dependent volumetric absorption in the surrounding medium, as with ultrasound, but because of the manner in which stochastic signals add when observed.

Introduction. Arteriosclerosis is a term applied to a number of conditions in which there is a general hardening and thickening of the arteries. Atherosclerosis is a form of arteriosclerosis in which there are localized accumulations of material within or beneath the intimal, or inner surface of the arteries. Over a period of years these deposits become calcified atherosclerotic plaques which can severely compromise the arterial diameter. Substantial evidence also exists that the plaques are susceptible to hemorrhage and local thrombosis.

Atherosclerotic deposits are found somewhat randomly throughout the human arterial tree, and although the mechanisms by which these deposits are
formed remain unidentified, it is known that there are particular regions of the arterial tree which are predisposed to becoming sclerotic, including the coronary arteries, the bifurcation of the aorta at the iliace arteries, the carotids, and the femorals to mention a few.

Most commonly this condition is diagnosed by a method known as X-ray angiography, which involves insertion of a catheter into the artery of interest. X-ray opaque dye is released through the catheter and X-ray cinematography is used to map the arterial lumen and the perfusion of the subject vessel. However, this procedure has a significant risk of morbidity and is non-trivial to perform (Willcutt, 1968). It is therefore of substantial interest to develop a non-invasive method of diagnosing the disease both clinically and subclinically. (A complete diagnosis is taken to be the prediction of flow rate through the artery as well as the degree to which the lumen is occluded.)

![Figure 1. Idealized representation of a peripherical stenosed artery below the surface of the skin](image)

The stenosis can be thought of as a converging–diverging nozzle, or an orifice (Figure 1). During systole, the blood immediately proximal to the stenosis undergoes a rapid convective acceleration as it passes from the unobstructed portion of the artery through the converging section of stenosis. At the point of smallest cross-sectional area the mean flow velocity is a maximum, and the hydrostatic pressure is a minimum. As the flow passes through the diverging section of the stenosis the flow separates from the walls due to its inability to overcome the adverse pressure gradient. At the boundary between the high velocity separated jet and the slower moving fluid in the recirculating separation zone a shear layer is created which is susceptible to shear instabilities. This shear layer provides a source from which these instabilities can extract energy from the mean flow.

This energy extraction process proceeds at a sufficiently rapid rate that before systole has ended the instabilities break down into fully turbulent motion (provided the jet Reynolds number is high enough, \( \text{Re}_j = u_j d / \nu \), where \( u_j \) = mean jet velocity, \( d \) = jet diameter, \( \nu \) = kinematic viscosity of the fluid. Smith (1972) has observed turbulence in blood distal to stenosis at jet Reynolds numbers as low as 450). The turbulence continues to extract energy from the