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ALEXANDER NAUMANN MEMORIAL SYMPOSION ON "RHEOLOGY AND ATHEROGENESIS"

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Alexander Naumann * 1905 — 1983

OPENING REMARKS: Holger Schmid-Schönbein

Alexander Naumann, Dr.phil, Dr.med.h.c., Emeritus Professor of Aerodynamics of the RWTH Aachen, died unexpectedly on March 3, 1983. The biofluid-dynamicists and hemorheologists in Germany mourn the death of the "grand old man" of their science in the post-war period. Alexander Naumann was not only an outstanding scientist and researcher, but an inspiring teacher of students, physicians and fluid-dynamicists, and a farsighted organizer. In no small part he inspired and structured research in the field of bioengineering in our country.

It is but a small consolation to dedicate this symposion to his memory. Aged 78, he was in full command of his mental power and crativity when he was

suddenly struck by a fatal disease which hit him while working on a manuscript of a biomedical paper that he anticipated to deliver to this meeting.

Alexander Naumann's devotion to problems of biomedical engineering came late in his career; exactly 20 years ago, when he was elected head of the Aerodynamic Institute and professor of fluid-dynamics at the RWTH Aachen, he was considered the leading experimentalist and an internationally respected authority in the field of instationary gas dynamics, i.e. pulsatile gas flow and developing turbulence. Born in 1905, he had received his early training in mathematics and physics at the University of Leipzig, where he earned his doctoral degree in philosophy. His academic career in Leipzig and Aachen was interrupted by his work for the French Government in the immediate post-war period when he was assigned to construct a large wind channel as head of the "Bureau d'Études" in Emmendingen. Later he headed various institutions for aerodynamic research and was one of the directors of the "Deutsche Forschungs- und Versuchsanstalt für Luft- und Raumfahrt" (DFVLR) in Wahn near Cologne that later developed into the German "Space Center". He pioneered in the design of appropriate wind channels and measuring devices and was considered a leading experimentalist in the field of aerodynamics and kinetics of instationary gas flow.

His interest in biomedical research, namely in blood flow problems and hemorheology was initiated in the early phase of cardiac surgery and implantation of artificial heart valves in the 1960s. The observation of flow dependent hemolysis and thrombotic processes that occurred in patients supplied with the newly invented artificial heart valves brought the surgeons and physicians to the fluid-dynamicist. This occurred when Alexander Naumann had just been elected chairman of the Department of Aerodynamics in Aachen.

His first publication on this topic dates from 1969, i.e. in his 64th year(1). The majority of biomedical publications were published after his retirement in 1973 (e.g. 2,3). As professor emeritus he continued to supervise and initiate experimental research, and he was a catalyzer for interdisciplinary research in biomedical engineering in our country.

Alexander Naumann was a very successful man in university politics. He was deeply involved in the foundation of a medical faculty in Aachen, i.e. in the attempt to extend an existing, large school of engineering. Moreover, in no small part he helped in the establishment of the Helmholtz-Institute for Biomedical Engineering in Aachen. His authority helped in convincing the federal and states governments to funnel money into biomedical research. The hemolysis laboratory that he had installed in the Aerodynamic Institute was and is a model for interdisciplinary biomedical research.
In 1979, the Medical Faculty of the RWTH Aachen awarded to him the degree of "Dr. med. honoris causae"; he was the first and to date the only person selected for this honour.

In the context of the present symposion his detailed knowledge about the physics of secondary flow in tubes deserve to be emphasized especially. As already mentioned, he entered the field as one of the leading authorities in instationary gas dynamics. He was familiar with many details of the rapidly forming and vanishing secondary flow phenomena as they occur at l o w Reynold's numbers in pulsatile flow and irregularly shaped conduits. He had shown previously that such flow irregularities, while often preceeding full turbulence, are by no menas identical to turbulence in instationary flow, but represent highly complex yet regular secondary motions of fluid elements at very low Reynold's numbers. As a gas dynamicists he knew that these fluid-dynamic peculiarities were highly effective in eliciting specific physicochemical processes: deposition processes, ignition of gases, local pressure fluctuations.

This leads me to his most important and potentially fruitful contribution: the idea of the interactions between chemical and fluid-dynamic processes. Starting from his observation on flow dependence of hemolysis he suggested that in flowing blood specific deviations from laminar flow were capable of initiating specific biological processes. He opened our eyes for the following fundamental combination of phenomena that occur in areas of secondary flow.

1. Locally, there are high shear stresses, even though they act very shortly,
2. There are recirculation zones where particles may remain for extended periods of times and may react chemically,
3. There are reattachment points, i.e. areas where flow vectors point towards the tube wall and carry reaction pastures towards the wall at a much higher rate than it would occur by mere diffusion. When flow is pulsatile, all 3 phenomena oscillate in localization or extension in magnitude and in direction - nevertheless they follow strictly established fluid-dynamic laws.

In praising Naumann's contributions, however, it is important to stress that he introduced his knowledge about gas dynamics only after he had seen the work of GOLDSMITH and KARINO (4), of MÜLLER-MOHNSSEN's group (5, 6) and of LIEPSCH (7) and of the work of his own students (8, 9, 10) and coworkers. The experiments of these authors had shown that flow instabilities can occur at very low Reynolds's numbers, such as they can be assumed to occur in arterial systems in man in pulsatile flow. He was extremely interested in the progress of the work of these authors and he often underlined their significance of our understanding of biofluid-dynamic problems. Seeing these details of flow in the models of arteries studied by the above mentioned authors, it occurred to him that in his own published work or in that from other fluid-dynamicists many details about instationary flow in complex conduits had already been solved experimentally.

Personally, I was priviledged to work with Alexander Naumann for almost a decade, and on a quite regular basis during the last three years. I would like to close this eulogy by showing two slides that we constructed together in the attempt to understand the localization of atherosclerotic lesions. They depict a theory we have recently published (11) in which we explain the localization of early and progressed lesions of the arterial wall on the basis of evolving and devolving secondary flow and migrating stagnation point due to flow pulstions (Fig. 2).
This theory takes into account that in the arteries the pulsatility of flow can be far more pronounced that the pulsatility of pressure, esp. in the aging vasculature.

He proposed that the fluid-dynamic, cellular and biochemical events associated with stagnation point flow act as primary injury to the normal arterial vessel wall as well as the perpetual and complicating stimulus to the developing and complicated lesions. Inherent in this theory is an assumption about the localization of the lesions: it was proposed that the area of endothelial damage and the deposition of platelets and of platelet derived mediators coincides with the migrating stagnation point, the extent of the lesion therefore being an indication of the domain swept over by the migrating reattachment point as the secondary flows pulsate in systole (see Fig. 2, the legend of which contains the pertinent fluid-dynamic considerations.

Fig. 3 contains an additional hypothesis which Naumann proposed; namely a possible protective effect of cuboidal endothelial cells. After having discussed at great length the so-called "arterial cushions" at the vortex of the flow dividers, and after studying again and again the striking morphological diffe-

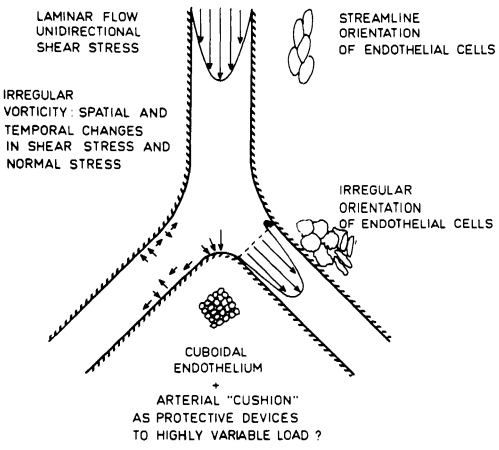

FIG. 2

Schematic representation of the fluid dynamics of pulsatile flow in an arterial bifurcation. Left half: during diastole low flow velocity and thence low Reynolds numbers provide pure laminar flow (which can be oscillating in direction); there is a stagnation point flow directed towards the vertex of the flow divider with low normal stresses and shear stresses. In systole, the pulsating flow with an increment and subsequent decrement in Reynolds number leads to an evolution and subsequent devolution of a vortex or recirculation zone. At the reattachment point, there is a flow vector directed against the wall, which migrates peripherally during the acceleration phase and centripetally during the deceleration phase of flow pulse. Depending on flow velocity, there is a more or less pronounced stagnation point flow directed against the divider (open arrows).

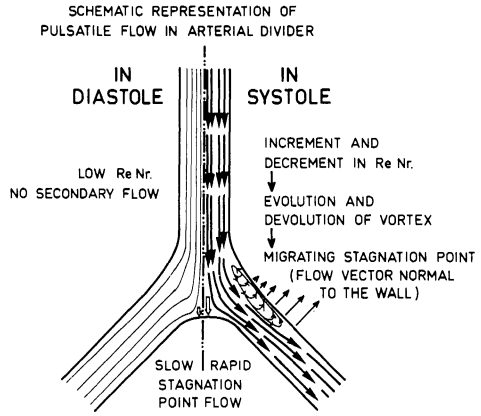

FIG. 3

Schematic representation of the morphogenetic effects of arterial flow on the structure of the intima. Pulsatile flow with phasic acceleration and deceleration leads to periodic oscillations in pressure, velocity, wall shear rate and local and general Reynolds number. In arterial segments with laminar flow, shear stresses acts unidirectionally in diastole and systole, they lead to streamline orientation of endothelial cells, which have a large surface area. The irregular vorticity near the flow dividers results in complicates spacial and temporal changes in the flow forces acting tangentially and normal to the wall. Correspondingly, shape, size and orientation of endothelial cells are irregular. In the area exposed to stagnation points both diastole and systole small cuboidal endothelial cells and subintimal transformations develop during life and may serve as a protective device.

rence in the elongated endothelial cells in the unbranched arterial segments contrasting the irregular cuboidal endothelial cells with microvilly, Naumann proposed they might be more deformable than the flattened endothelium. Naumann proposed that cuboidal cells might be suited to comply to rapidly changing shear- and normal forces, which "pulse" in magnitude and direction at the arterial flow dividers. It was his striking idea that the wall at the divider, where very pronounced stagnation flow occurs, would be even more prone to fluid-dynamic damage unless it were not "protected" by a specialized endothelium with small, deformable cells adapted to intense forces. Flaccid cells

might be capable of viscous compliance and a damping reaction, thus capable of withstanding short acting and rapidly changing flow forces as they occur during each systole in the arteries.

Obviously, compared to KARINO's data, the concepts outlined in Fig. 2 and 3 represent a crude simplified approximation of the true complexities of the flow near bifurcations. This notwithstanding, Naumann's concepts provide a plausible explanation for the localization of atherosclerotic lesions that might lead beyond the dead-lock between the proponents of the "high shear" and the "low shear" theory of atherogenesis (12,13).

In short: 1. Lesions are assumed to occur in areas where stagnation point flow impinges upon "non-protected" endothelial areas.
2. Stagnation point flow occurs physiologically at the divider in any bifurcation; the wall at the flow dividers, however, adapts to stagnation flow by a host of structural alterations including the transformation of endothelial cells.
3. Whenever socondary flow occurs in pulsatile flow, a migrating stagnation point occurs where flow reattaches. In strongly pulsatile flow, the reattachment point and therefore the stagnation point "sweeps" or "migrates" across extended segments of the vessel wall in each systole.
4. It is obvious that the areas swept over in systole coincide locally with the so-called "low shear areas" as identified by CARO et al. The divider areas coincide with the high shear areas;since they are protected by specialized endothelium, "early lesions" occur here but not classical atherosclerotic plaques.

CONCLUSION

Alexander Naumann was endowed with unusual spiritual clarity and spontaneous inquisitiveness: He was innovative and scientifically productive to his very last days. Most of all, however, he was a superb teacher not just of students but of colleagues from other disciplines. He has bestowed on us a conceptional estate that must now be conquered in order to inherit it. I hope that the spirit of today's symposion will live up to the standards set by Alexander Naumann, the great exponent of interdisciplinary research in biofluid-dynamics and hemorheology. My sincere thanks to Dr. Robert Nerem and Dr. Dieter Liepsch for organizing and moderating this symposion.

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