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Viscoelastic changes in the blood and vascular wall in a pulsating circular flow

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Altered flow conditions, such as separation and recirculationg zones, low and oscillatory shear stress, play an important role in the development of arterial disease. Endothelial denudation by the blood flow is the first step in atherosclerosis. The description of blood flow in vivo is complicated due to the viscoelasticity of vessel walls. However, conventional researches of the effect of the blood vessel viscoelasticity on the blood pressure wave propagation using non-linear one-dimensional models do not take into account the viscoelasticity, despite it being importance in the analysis of pulse wave propagation in arteries.

The purpose of this paper is to study the impact of the arterial pulse wave on the viscoelastic blood flow and initial factors of atherosclerosis.

In 12 healthy men (25-39 years of age) peak velocity, mean velocity, mean flow and net flow in the aorta have been investigated by MR angiography.

Initial velocity was registered after 43msec of the ECG-R wave, and it differed from zero at all sites of the aorta, although net flow was equal to zero. Womersley's number from the ascending to the thoracic aorta decreased from 12.5 ± 1.5 to 7.3 ± 1.2; flow modified from inertio-elastic to viscous. Pulse pressure wave move on artery walls fifteen or more times more rapidly than the blood flow. In the aortic arch in protodiastole blood flow separated into the opposite directed streams resulting in wave superposition with the high net flow. At the isthmus area separated waves interferences and reflects to anterograde direction.

Pulse oscillation increases strain rate to the contiguous vessel wall flow layers. At the sites with the flow wave negative interference vessel pulse oscillation attenuates and at the boundary reflection flow wave can shift the vessel wall.

Key words: arterial pulse, blood flow, wave propagation, blood viscoelasticity, endothelial denudation, magnetic resonance imaging, atherosclerosis.

Introduction

Atherosclerosis is a focal chronic inflammatory fibroproliferative disease of the arterial intima caused by the retention of modified low density lipoprotein and hemodynamic stress. It can be present throughout a person's lifetime. In fact, the earliest type of lesion, the so-called fatty streak, which is common in infants and young children, is a pure inflammatory lesion [9].

Local hemodynamic temporal pressure and wall shear stress are important for understanding the mechanisms leading to various complications in cardiovascular function [7].

At present blood motion has been studied as a continuum with the steady flow and has been described by the Navier-Stokes equations. These equations arise from applying Newton's second law to fluid motion and are nonlinear partial differential equations [10]. In a real situation blood flow in the large arteries is unsteady, with the flow separation and waveform propagation of the thyxotropic mass.

Polymer solutions are convenient for experimental studies of viscoelastic flows. The Couette-Taylor flow is often chosen because of its geometrical simplicity and its diversity of instability modes and turbulent states. The most striking elastic property of the polymer solutions is, probably, the dependence of mechanical stresses in flow on the history of the flow. Equations with the expression of time derivative of the polymer stress constitute the Oldroyd-B model of polymer solution rheology [1].

Up today it is impossible to describe blood flow only by the fluid mechanics. For the complicacy of the problem we are discussing the theoretical basis for the waveform propagation of the viscoelastic substances and the ways of its correlation with our dates.

Subjects and methods

We have investigated 12 healthy men (25-39 years of age) with a 1.5-T MR imager with the breath hold (18 sec.) and ECG triggering in different sites of the aorta. Pulse rate 72

to 78 beats per minute. Kinematic viscosity of the blood $3.8 \cdot 10^{-6}$ m^2/s. Volunteers were preliminarily examined by the cardiologist, angiologist and hematologists. Flow quantification (mean velocity, peak velocity, mean flow, net flow) was carried out in every 43 sec. at different sites of the aortic arch and thoracic aorta in 1mm slices of 7 cm^2 area. All data are given below as graphs (Figures 1-4).

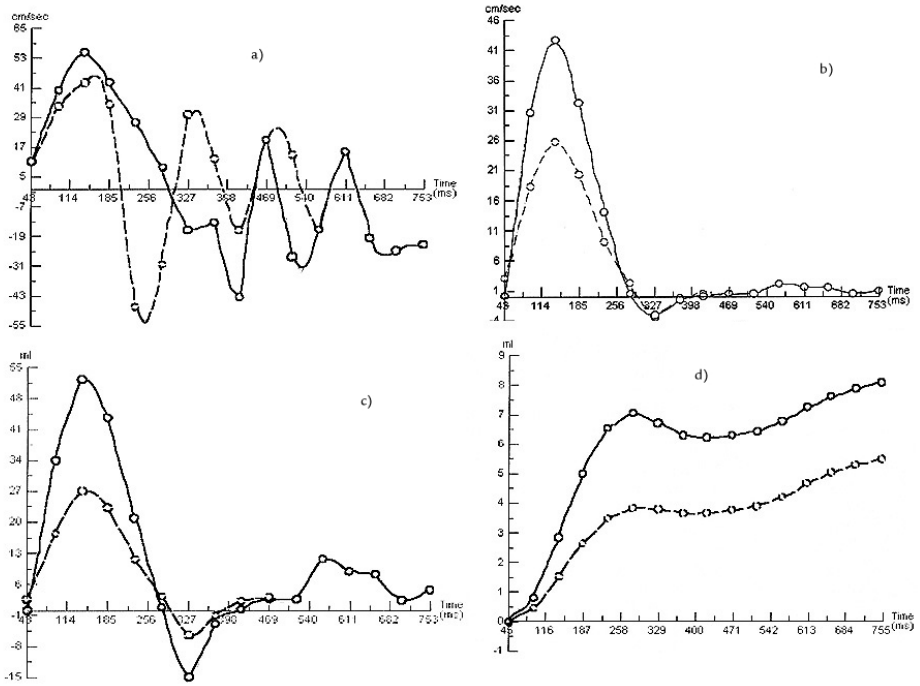

Figure 1. Peak velocity (a), mean velocity (b), mean flow(c) and net flow (d) graphs at the aortic arch. Dotted line – flow at the external wall. At peak velocity graph separated stream flows in opposite direction. Net flow increases at the end diastole.

Results

In our cases the initial flow velocity was registries after 43 msec. of the ECG-R wave, and it was different from zero at all sites of the aorta, although net flow was equal to zero. During the heart cycle blood systolic velocity varies in sinusoid shape, whereas net flow increases gradually at the diastole when blood velocity was low. Initial flow acceleration was lower than of the next one, although gradient pressure much higher initially and gradually decreased at the flow. Womersley's number from ascending to thoracic aorta decreased from 12.5 ± 1.5 to 7.3 ± 1.2 and flow modified from the inertio-elastic to viscous. Specificity, sensitivity and accuracy of the MRA to the explored area were 95%, 97%, and 96%. (Figures 1-4).

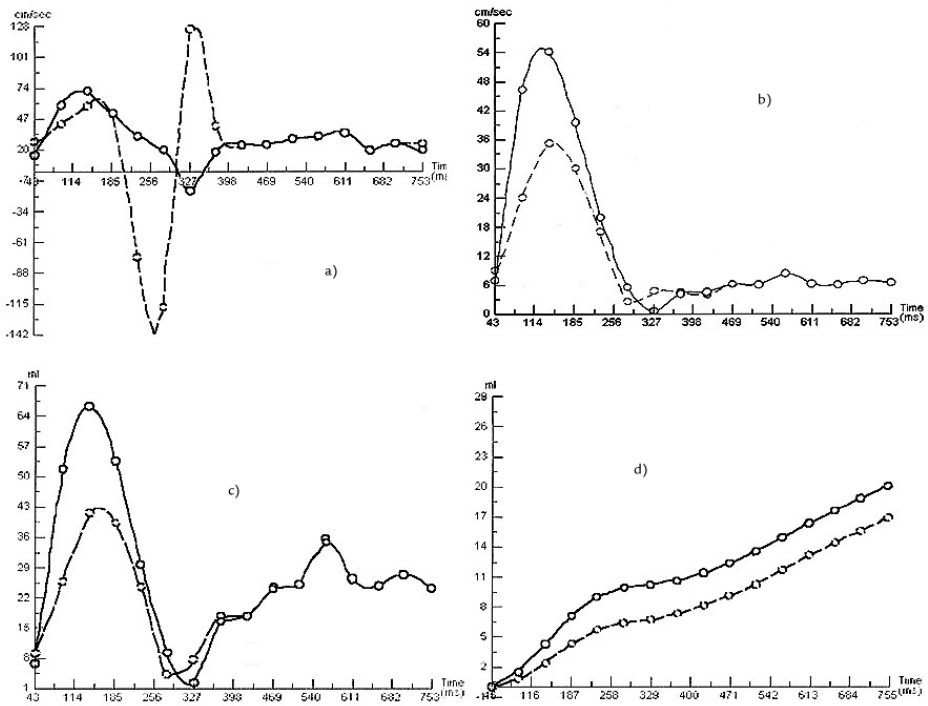

Figure 2. Peak velocity (a), mean velocity (b), mean flow (c) and net flow (d) graphs at the isthmus of the aorta. Dotted line – flow at the external wall. Peak velocity at the 300 msec. is zero (negative interference) and then sharply increases to downstream. Here acceleration is 6 times higher than that in systole. Net flow increases at the end diastole.

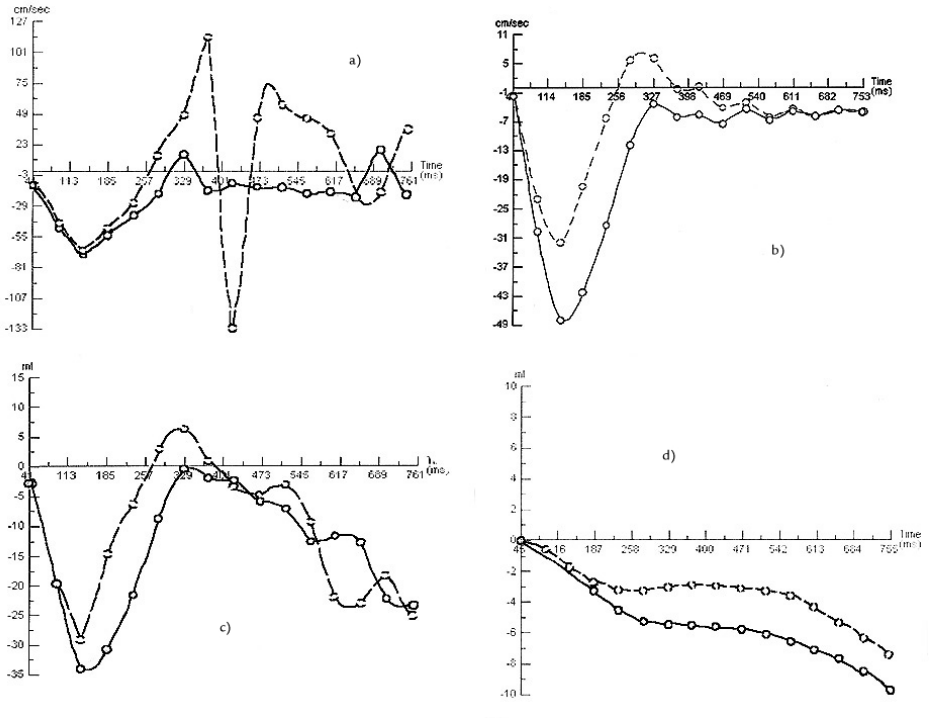

Figure 3. Peak velocity (a), mean velocity (b), mean flow (c) and net flow (d) graphs at the end of aortic arch (a). Dotted line – flow at the internal wall. Flow direction at the graphs inverted due to slice position at MRI. Wave oscillation is transmits from external to internal wall. Incident (Figure 3a) and reflected (Figure 4a) wave has the same polarity (no phase change). Peak velocity formation delays to the same at the isthmus area. Net flow increases at the end diastole.

Discussion

Systolic pulse pressure in the vessel wall and inside the blood propagates as a wave. Wave is the disturbance that runs through the space and time, transferring energy, little/no associated with the mass transport; instead there are oscillations around almost fixed positions. Within wave, phase of the oscillation is different for adjacent points in space.

Arterial pulse pressure evaluating in the initial systole, move on arteries and it is not caused by the forward movement of the blood: blood stroke volume forming distension of the medium and pulse pressure propagates through the aorta in waveform by elastic

deformation of the vessel wall and the blood. Further to elastic, sliding deformation of the blood is accompanying, preventing excessive fall in arterial pressure [2].

Pulse oscillation in arterial wall increases strain rate to the contiguous wall flow layers: pulse wave velocity from aorta to small arterial wall is ≈ 7-15-35 m/sec. Pressure wave velocity in blood is ≈ 6-8 m/sec. Peak flow velocity in aorta is ≈ 1.2-1.4 m/sec. [20]. At the identical frequency, wave length to the vessel wall and adjoining flow blood slice must be different. Initiation of the strain rate and blood flow is shown on the stream surface and gradually involves the whole blood mass; so, phase delayed creep flow becomes simpler. These correlations are more expressed at the proximal aorta. Here wave oscillation amplitude in vessel wall is high, blood in systole reveals inertio-elastic

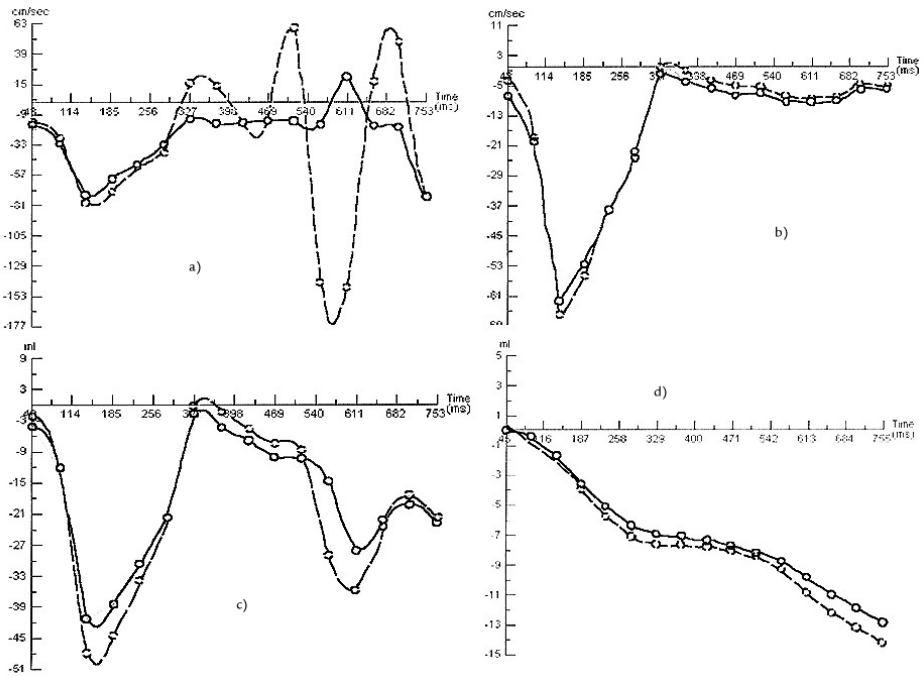

Figure 4. Peak velocity (a), mean velocity (b), mean flow(c) and net flow (d) graphs at the thoracic aorta. Dotted line – flow at the internal wall. Flow direction at the graphs inverted due to slice position at MRI. There is no flow separation at the protodiastole. Net flow increases at the end diastole.

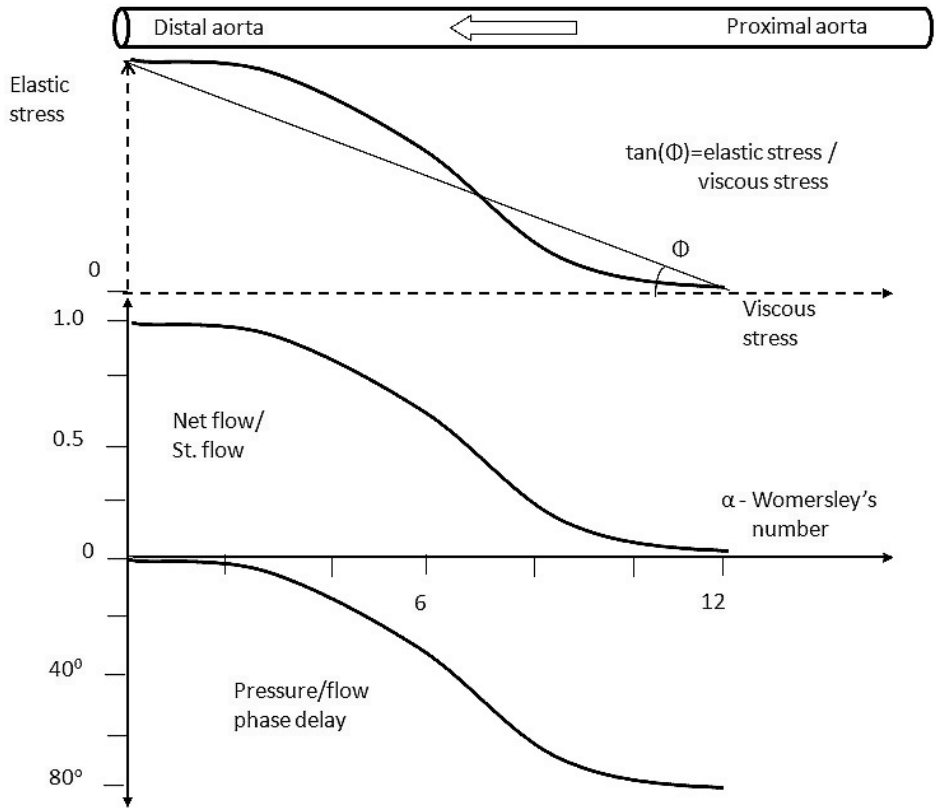

Figure 5. Pressure and flow phase delay in viscoelastic flow at the different sites of aorta. Elastic/viscous stress correlation in the blood (above), Net flow/st. flow correlation (middle; St. flow – rate which would be established at the maximum gradient pressure at the Poiseuille's flow) and pressure/flow phase delay (below) at the sinusoidal varying pressure with the increasing Womersley's number-α.

properties. To the distal wall pulse wave attenuates and boundary layer enlarges, flow becomes viscous.

Inertial flow is characteristic for the Newtonian fluids. Blood is non Newtonian [4], thyxotropic and as a viscoelastic substance must be expressed in some phenomena:

1. If the stress is held constant, the strain increases with the time (creep).
2. If the strain is held constant, the stress decreases with the time (relaxation).
3. Effective stiffness depends on the rate of application of the load.

4. If cyclic loading is applied, hysteresis (phase lag) occurs, leading to a dissipation of mechanical energy.
5. Wave experience attenuation.
6. Rebound of the object following an impact is less than 100% [8].

Blood flow (creep/relaxation) delays to the pressure wave oscillation by the phase. Pressure and flow phase difference in viscoelastic flow is shown in Womersley's number-α.

$$\alpha = \frac{d}{2}\sqrt{2\pi f}\,\frac{\rho}{\mu}$$

where: d – diameter, f – fluctuation frequency, ρ – density, μ – viscosity.

It defines relationship between elastic and viscous stress during the pressure oscillation and shows which part of the pipe is occupies by the boundary layer's viscous flow. In the large arteries, boundary layer is less than the blood volume provided by the pressure. At the 43 msec. distances propagating by the pressure wave is about 28-60 cm and covers all of the imaged places.

At the ascending aorta flow is inertio-elasatic. Elasticity is promoted by the blood and vessel wall structure. Womerslay's number is 12.5 ± 1.5. In the elastic material phase angle between causative force-pulse pressure and strain rate (blood oscillation) is low. Blood viscous stress exceeds to elastic and pulse energy stored mostly in the substance. Flow phase delay to pressure is about 85°; Net flow is low (Figure 5). This is main reason for the low initial systolic flow acceleration.

Blood decreases in the apparent viscosity under shearing, followed by a gradual recovery when the shear is removed. The effect is time dependent and more expressed at the distal aorta. At the distal aorta pressure dissipates in blood structural rearrangement. Womerslay's number is 7.3 ± 1.2. In the viscous material phase angle between the causative force-pulse pressure and strain rate (creep flow) is high. Flow is more viscous/less inertial. Flow phase delay to pulse pressure decreases (about 40°). Net flow is high (Figure 5).

In diastole pressure is less pulsating. Pressure exposition time for the viscoelastic creep flow is higher than that for the systole and net flow increases.

In line of our date at the aortic arch blood circular movement facilitates flow velocity dispersion. In initial protodiastole blood flow is separated into the opposite flowing streams. Retrograde and anterograde directed flow waves initially have different frequencies and at the superposition do not resist to each other. Wave superposition facilitates to increase strain rate: blood particles at the same time participating in different oscillations. It causes to the high net flow at the diastole as shown in graphs and can simplify blood outflow in the different arterial branches (Figures 1d-4d).

At the isthmus area (end/entrance of the circle flow) separating flow waves at 300-400 msec. have identical frequencies and phase, so they can interfere (Figure 2a). Here at the destructive interference flow velocity is zero and further sharply increases.

Flow anterograde acceleration in protodiastole is 6 times higher than in systole. At zero velocity systolic kinetic energy of the blood passes in to potential energy of the vessel wall. Wall elastic stress by the stagnation pressure increases. and systolic pulse wave can be attenuates. Here phase difference between the high stagnation pressure and flow is 90°.

At the isthmus area flow wave with the high acceleration changes position from external to internal wall. Circumstances can be promoted by the presence of the recirculation zones. Wave propagation on the graphs is similar to the wave reflection at the boundaries (Figure 6).

By our dates, in protodiastole blood peak velocity at the external wall is much higher than that of systolic and peak shear stress is about 25 N/m^2. But just before the flow local pressure is much higher than at the flow and is equal to shear stress, it exceeds thresholds of the elastic deformation and as the outer slice of the blood is adhesive to the wall, forming endothelial denudation [6].

When the wave encounters a boundary which is neither rigid (hard) nor free (soft) but instead somewhere in between, part of the wave is reflected from the boundary and part of the wave is transmitted across the boundary. The exact behavior of reflection and transmission depends on the material properties on both sides of the boundary. The frequency of the incident pulse is the same as the frequency of the transmitted pulse. At the soft boundary, the restoring force is zero and the reflected wave has the same polarity (no phase change) as the incident wave. Correlation of the transmitted/reflected energy depends on the incidence stream angle to the boundary surface. At the isthmus area incidence angle is low and transmitted energy to the vessel wall is high [3].

So damage of the vessel wall at the end of the circle site can be confirmed by the pulse wave propagation and viscous flow.

The ground substance, non-cellular components of extracellular matrix fibers in the human body, is thyxotropic and acts as a support for the cells. It seems that, if the high energy uploads to the vessel wall, to the viscoelastic transformation undergo not only blood, but endothelial extracellular matrix too.

The more so as cholesterol in experimental membranes can modify lateral tension and surface viscosity of the lipid bilayers [5]. In atherosclerosis it can be compensatory reaction of the vessel wall to the increased stress.

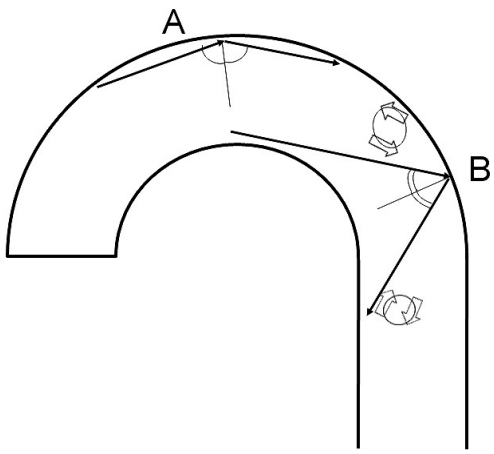

Figure 6. Flow wave reflection and recirculation sites at the different sites of the aortic arch. Incidence and reflected wave at the different sites of aortic arch. At the isthmus area (B) incidence and reflected angle is lower than that for the aortic arch (A).

It is significant that red blood cell aggregation affects blood viscoelasticity, but shear stress threshold to destruction of erythrocyte membrane is 6 times higher than for the endothelial sheet and erythrocyte membrane can move around the cell.

Circular movement at the arterial bifurcation can be facilitated by the same changes as at the aortic arch. It seems that at the vessel branching sites wave superposition increases net flow, but is fraught with the endothelial denudation.

Conclusion

Systolic pulse oscillation in arterial wall increases strain rate to the contiguous to the wall blood flow layers.

At the circular sites flow separation and wave superposition promotes to the blood viscoelastic modification and high net flow in diastole.

At the isthmus area flow wave superposition forms destructive interference. Here phase difference between the high stagnation pressure and flow is 900.

At the high tension wall elastic deformation exceeds the threshold value and protodiastole anterograde directed flow wave, reflects and shifts (creep) endothelial layer.

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