Hemodynamic Simulation with Lattice Boltzman

Harvard IACS AC 290R - Group 1
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1 Problem Statement and Motivation

AC 290R is a course on extreme computing with a focus on the application domain of fluid dynamics. In our first module we attempted a prototypical problem using the continuum description of fluids governed by the Navier-Stokes equation: Rayleigh-Bénard Convection. In this module, we shift from the continuum to the mesoscale description and simulate fluids using Lattice Boltzmann methods. These methods are based on the Boltzmann Equation of thermodynamics and statistical physics.

The particular task we we undertook was a hemodynamic simulation. Hemodynamics is the study of the dynamics flow blood. It is a rich field at the intersection of anatomy and physics with a storied history. Pioneers in the field have included Leonardo Da Vinci, Leonhard Euler, Thomas Young, and Jean L.M. Poieseuille.

The particular problem was as follows. We aim to model the dispersion of a therapeutic drug that is injected with a catheter to treat a stenotic artery. Stenosis is a disease of the arteries in which an artery becomes narrowed. It is frequently caused by atherosclerosis, a build-up of fatty deposits (cholesterol) on the walls of the artery. Stenotic arteries can cause serious medical problems including heart attacks and strokes. One approach to treating stenoses is to introduce a therapeutic drug with a catheter, a small tube inserted surgically into the patient that can be threaded through the circular system. The goal of our simulation was to understand how the drug molecules dispersed and to see how many of them passed through the stenotic region, and at what times.

This is a worthwhile object of study for both pedagogical and practical reasons. Pedagogically, this topic rounds out our survey of both techniques and domain knowledge in the course. We are aiming to master the techniques of extreme computing and their application to fluid dynamics. The first topic covered the continuum approach, and this topic covers the mesoscale approach. The first topic used traditional CPU-centric computations, and this topic introduces us to GPU computing.

On a practical level, heart disease has been for many years a leading cause of death in Americans alongside cancer. It is a complex disease with many treatment options and a need for accurate diagnosis. Clinical practice still often relies on human judgment about which arterial blockages look risky, and there is reason for optimism that advances in biologically realistic computer simulations could lead to materially faster and more accurate diagnosis and improve treatement selections.

2 Overview of Numerical Methods Used

The main numerical method used in this simulation is the Lattice Boltzmann Method (LBM). LBM is based on the Boltzmann Equation, which describes the statistical behavior of a thermodynamic system and dates to 1872. The idea behind the Boltzmann Equation is that particles in the system each have 6 degrees of freedom, 3 positions and 3 momenta along the 3 coordinate directions x, y and z. Molecules of a given type are physically indistuinguishable from each other, so the system can be described completely by the populations of particles as a function of time and these six dimensions in the phase space. The Boltzmann Equation describes the evolution of such a system. The populations of particles change due to three terms: forces applied to the system, diffusion, and collisions.

LBM is a technique in computational fluid dynamics that uses the Boltzmann Equation to devise a numerical simulation of a fluid that can accurately capture mesoscale dynamics when it is tuned properly. The physical system is discretized, typically on a rectilinear grid and most commonly one with cubic spacing. Particle velocities are also discretized, with particles allowed to jump from from one grid point only to nearby grid points over one simulation step. The most common discretization scheme for 3D fluid simulations, which we used for this problem, is called D3Q19. The label can be parsed as referring to 3 dimensions and 19 discrete velocities. The 19 discrete velocities have the following structure:

- 1 0^{th} neighbor; displacement $(0,0,0)$; weight $\frac{1}{3}$
- 6 1^{st} neighbors; displacement one of 3 permutations of $(\pm 1,0,0)$; weight $\frac{1}{18}$
- 12 2^{nd} neighbors; displacement one of 3 permutations of $\pm 1, \pm 1, 0)$; weight $\frac{1}{36}$

The 6 first neighbors have displacements $(1,0,0), (-1,0,0), (0,1,0), (0,-1,0), (0,0,1), (0,0,-1)$. The 12 second neighbors follow a similar pattern; there are $\binom{3}{2} = 3$ permutations of indices i, j, and each index has 2 choices in ± 1, leaving $4 \cdot 3 = 12$ second neighbors. The total weight of the 6 first neighbors is $6 \cdot \frac{1}{18} = \frac{1}{3}$. The total weight of the 12 second neighbors is $12 \cdot \frac{1}{36} = \frac{1}{3}$.

The state of the simulation at a time step is given the *population* of particles, $f_p(x,t)$, where the suffix p refers to the discrete velocities above. In a system with more than one type of particle, each populations of each particle must be maintained separately. The movement of populations can be described by the Btatnagar-Gross-Krook update rule:

$$f_p(x + hc_p, t + h) = f_p(x,t) + \omega(x,t)h\left[f_p^{eq}(\rho, \mathbf{u} - f_p)(x,t) + w_p \frac{c_p \cdot \mathbf{g}}{c_s^2}\right]$$

Here is a brief description of all the terms appearing in this equation from left to right:

- f_p is the actual population of the particle introduced above.
- h is the time step, often taken to be 1 in simplified notation
- c_p is the displacement vector corresponding a given discrete velocity, e.g. $c_0 = (0,0,0)$, $c_1 = (1,0,0)$, etc.
- ω is the relaxation frequency which is related to the kinematic viscosity ν (described below)
- $\rho(x,t)$ is the density of the fluid in this cell, a macroscopic quantity; essentially the 0th moment of the velocity
- $\mathbf{u}(x,t)$ is the velocity of the fluid in this cell, a macroscopic quantity; essentially the first moment of the velocity
- w_p is the weight of particles with each velocity in the D3Q19 scheme; scalars that do not change over the simulation

- c_p is the discrete velocity
- $\mathbf{g}$ is the acceleration applied to the body by external forces; the letter g evokes gravity but it can be any external force
- c_s is the speed of sound in dimensionless units on this lattice. (The speed of sound of the physical medium depends on the gradient of pressure with respect to density).

The equilibrium populations can be approximated with a Taylor expansion that accounts for the 0th, 1st, and 2nd moments of the populations.

$$f_p^{eq}(\rho, \mathbf{u}) = w_p \rho \left[1 + \frac{\mathbf{u} \cdot c_p}{c_2^2} + \frac{(\mathbf{u} \cdot c_p)^2 - c_s^2 u^2}{2c_2^4} \right]$$

The resulting f_p^{eq} will match the first two moments (density ρ and velocity $\mathbf{u}$), but in general it will **not** match the second moment (energy density) unelss the system is at equilibrium. This is how energy dissipation in a viscous fluid away from equilibrium is modeled. This approximation is valid as long as the system is sufficiently close to equilibrium. If the parameters are not set properly, the populations can depart from equilibrium by too much and the simulation will break down.

The macroscopic quantity density ρ is simply the sum of the fluid populations in a cell.

$$\rho(\mathbf{x}, t) = \sum_p f_p(\mathbf{x}, t)$$

The momentum density is the first moment of the particle velocities. This is equal to the density times the velocity in a cell, giving us the formula for $\mathbf{u}(\mathbf{x}, t)$:

$$\mathbf{J}(\mathbf{x}, t) = \rho(\mathbf{x}, t)\mathbf{u}(\mathbf{x}, t) = \sum_p c_p f_p(\mathbf{x}, t)$$

$$\mathbf{u}(\mathbf{x}, t) = \frac{1}{\rho(\mathbf{x}, t)} \sum_p c_p f_p(\mathbf{x}, t)$$

The relaxation frequency is related to the kinematic viscosity ν by the following relationship:

$$\nu = c_s^2 \left(\frac{1}{\omega} - \frac{1}{2} \right)$$

A Lattice Boltzmann fluid simulation can be organized into two logical phases, which are sometimes called *collision* and *streaming*. The collision step describes how particles populations in the same cell interact and how their weights move toward their equilibrium values as a result of collisions. The intuition is that when particles hit each other, momentum is conserved, but some kinetic energy is dissipated as heat or otherwise. The equation for the collision step can be written

$$f_p^* = (1 - \omega) f_p + \omega f_p^{eq}$$

where f_p^* are called the temporary post-collisional populations. We can think of these as the new populations one "instant" after the previous streaming step.

The streaming step describes how particles of the post-collisional population move forward into the next time step. This is very straightforward, the particles just move at their discrete velocities according to

$$f_p(x + c_p, t + 1) = f_p^*(x, t)$$

A key fact is that streaming is a *local* operation. This makes it ideally suited to GPU computations, which excel at simple, highly parallel tasks with memory locality.

Stability is an important concept in the numerical solution of differential equations generally. One of the best known stability criteria is the Courant-Friedrichs-Lewy (CFL) Condition. This relates the range of time steps Δt for which a numerical method is stable to the spatial discretication δx, the speed of movement u, and a dimensionless constant that is a property of the numerical method. A rule of thumb for LBM is that optimal results are achieved when

$$|u| < \sqrt{\frac{2}{3}}\frac{\Delta x}{\Delta t}$$

A representative value of u is 0.1, leading to a guideline that stable results can be found when the kinematic viscosity ν is selected in the range $0.05 < \nu < 1$.

Like any differential equation solution method, LBM must also cope with initial conditions and boundary conditions. Common initial conditions are prescribed values for the pressure (equivalent to a prescribed density) and velocity, i.e.

$$\rho(x, t = 0) = \rho_0(x)$$
$$\mathbf{u}(x, t = 0) = \mathbf{u}_0(x)$$

A common choice is to set the initial density to be uniform and the initial velocity to be zero. Once ρ and $\mathbf{u}$ are initialized, a common modeling choice is to initialize the populations to the equilibrium implied by these macroscopic variables, i.e.

$$f_p(x, 0) = f_p^{eq}(\rho_0(x), \mathbf{u}_0(x))$$

Boundary conditions are a bit trickier. An astute reader will quickly point out that with these equations as written, any finite domain will have particles "falling off the edge" and without sensible treatment of boundary conditions, the simulation would just report that there were no particles left. That would be sad. In general, a boundary condition in an LBM simulation can be expressed as a linear combination of a constraint on the flux in a direction normal to a wall, and on the density itself. Taking ϕ to denote the quantity of interest (can be density or velocity) and n the normal direction,

$$b_1\frac{\partial \phi}{\partial n}(x_b, t) + b_2\phi(x_b, t) = b_3$$

A few special cases are the most common. When $\phi = \mathbf{u} = 0$, we have the **no slip** boundary condition. This is common and physically realistic. When $b1 = 0$, we have a Dirichlet boundary condition, in which the value is imposed. When $b2 = 0$, we have a Neumann boundary condition, in which the flux is imposed. When $b1 \neq 0$ and $2 \neq 0$, the boundary condition is mixed (linear relationship between field value and flux) and is called a Robin boundary condition. In addition to no-slip boundary conditions, another common choice is for velocity to be fixed at an *inlet* or *outlet*. Finally, larger sytems can often be simulated using a *periodic* boundary condition. For the simulation we ran of an artery, we imposed a non-slip boundary condition on the side walls of the artery, and a periodic boundary condition on the longitudinal direction, effectively modeling a longer artery with periodic stenoses.

3 Description of Code

The workhorse of this simulation is a Lattice Boltzmann fliud simulator called MUPHY. MUPHY is about 10 years and designed for multi-physics simulations. We also used a newer software package called MOEBIUS. MUPHY is an open source project on which our guest instructor Simone Melchionna was one of the lead developers MOEBIUS is a commercial package developed by a private company, Lexma, founded by Dr. Melchionna.

MUPHY and MOEBIUS (hereafter referred to simply as MUPHY to save space) follow a similar strategy to Drekar. They are object oriented written primarily in a mix of C / C++ and Fortran, with GPU acceleration written in Cuda. The back end is implemented in lower level languages like C / C++ to meet the requirements for high performance. Another similarity to Drekar is that parallelization is proved using OpenMP. There is also a front end interface for managing jobs. This is written in Python. To run our simulations, we needed to install the MUPHY package and write scripts in Python. We did not need to write or compile and C++ or Cuda.

MUPHY was written with a particular focus on applications in life sciences. It can handle the complex geometries arising in biological systems, ranging in scales from folding proteins and cell membranes up to highly branched arteries. We saw demonstrations in class run on MUPHY of a DNA molecule passing through a cell membrane, and of a flow simulation in arteries whose geometry was based on a patient.

The first code we wrote is in the file `ShapePainter.py`. This code can be found in a repository named BUFFY our team created on Odyssey. We needed to fork the MUPHY repository so we could make edits and push changes. We named it BUFFY in homage to the hit TV show Buffy the Vampire Slayer. Access to this repository can be provided to the teaching staff on request. We took snapshot of the most relevant Python scripts and saved them in our team repository (where we submit our coursework) in the directory `/project2/BUFFY`. `ShapePainter.py` creates a geometry for the system using the `vtk` library. The artery is modeled with a simplified geometry as a cylinder extending down the z axis longitudinally. It has a radius R on the healthy region and $G = R/2$ in the stenotic region. The length of the entire artery is L and the length of the stenotic region is S. This image was provided with the problem statement and demonstrates the basic layout and parameter names:

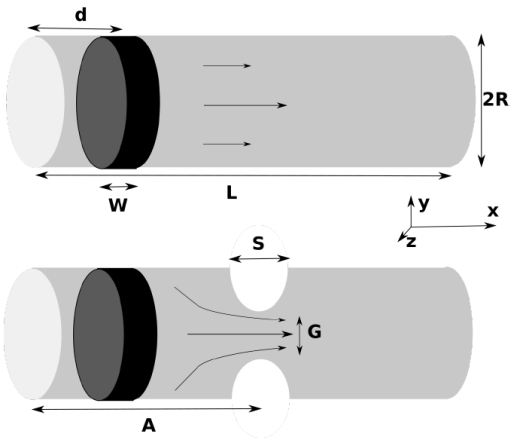


Figure 1: Artery Geometry.

IMAGE OF ACTUAL GEOMETRY WE GENERATED

YUE TO SAY MORE ABOUT SHAPE PAINTING SCRIPT AND RUNS

The next code we wrote is the Python script that runs the simulation on MUPHY. Each simulation run had a file, conventionally named `run2.py`, that kicks off the simulation. The script that ran our baseline simulation

is located in `scratchlfs/ac290r/project2/blood_cells/BUFFY/RBC_0_Re10` (We tried to follow advice given to use organized directory names. While this is a bit long and slightly cryptic, we did at least follow a consistent scheme this time organized everything hierarchically!) We've included a copy of this file in project repository as well in `project2/BUFFY/RBC_0_Re10` for convenience in grading our submission.

The first part of this file sets the physical parameters for the simulation, e.g. the geometry of the cylinder and the viscosity. The Python module `MagicUniverse` is the fancifully named interface to the BUFFY simulation engine. The script initializes simulation objects including:

- Universe
- Scale
- Mesh
- Fluid (for blood)
- Fluid (for drug)
- Tracker (for diagnostics)

These are all class instances from the `MagicUniverse` module. Parameter values are set to the appropriate class instances. We set the boundary conditions to be periodic on the z axis, and no-slip on the x and y axes. The alternative configuration would have been to create inlets and outlets at the start and end values of z.

YUE TO SAY MORE IF NECESSARY

To run this code on Odyssey (Harvard's supercomputing cluster in Western Massachusetts) we also wrote shell scripts that were submitted to the Odyssey job manager `slurm`. The script to run the baseline simulation is called `runrbc_0.sh` and is located adjacent to `run2.py`. The key lines in this script set the job options and load the required modules. Important flags include:

- `-p shared` - run the job on the shared partition
- `--reservation=ac290r` use the reservation so we don't have to wait in the queue to get 1024 CPUs
- `-t 1200` hold the job open for up 1200 minutes = 20 hours
- `-n 512` run on a total of 512 CPU cores
- `N 16` run on 16 nodes; we are therefore requesting 16 nodes with 32 cores each
- `mem=64000` request 64,000 MB = 64 GB per node; that is a lot of memory, 1024 GB = 1 TB total
- `--job-name=RBC0RE10` the descriptive job name refers to 0 red blood cells and Reynolds Number 10
- `--output=RBC0RE10.out` the directory where output is written; matches the job name

The rest of the script loads the required modules and sets environment variables. We need modules for `gcc` and `openmpi`, provided by `gcc/7.1.0-fasrc01` and `openmpi/3.1.1-fasrc01` respectively. The environment variables to set are `MOEBIUS_PATH` and `PYTHONPATH`. These allow Python to find the MAGIC modules. In order to invoke the job as a Python 2 script with MPI, the line of the script that kicks off the actual job is

```
srun -n $SLURM_NTASKS --mpi=pmi2 python2 run2.py
```

The last major batch of code we wrote performs calculations at the post-processing stage. This is in two Python scripts `vtk2np.py` and `rbc.py` located in the folder `project2/src`. The MUPHY simulation generates output in the form of VTK files with the extensions .vtu and .pvtu. These files are well suited to visualization, especially intensive visualizations like movies. For computations and 2D plots of one time instant, we find it more convenient to use `numpy` and `matplotlib`.

We made a strategic decision to do the computationally heavy graphics rendering remotely on Odyssey but to do the computations and 2D ploting locally on a few selected time frames. After our baseline simulation ran, we downloaded snapshots every 100,000 frames from 100,000 to 1,000,000. Each time step corresponds to 1 microsecond, so these frames were 100 milliseconds apart for the first 1.0 second of the simulation. These files weren't too large, with a total size of about 9 GB each for the blood and drug for a total of 18 GB. Downloading these files was a bit painful though because SFTP connections to Odyssey are considerably slower than data downloads from large commercial websites for whatever reason.

The script `vtk2np.py` converts the VTK files to numpy arrays using the library `vtki`. `vtki` has a nice feature making it possible to read a .pvtu file. A .pvtu file is essentially a wrapper around a number of .vtu files, each with one part of a larger mesh. In our case, we ran on 512 nodes and each time step generated 512 .vtu files that were summarized by 1 .pvtu file. These outputs were generated for both blood and the drug. The VTK files include data that is keyed both by *points* and *cells* on the mesh. These are not the same! Our mesh for $Re = 10$ had 7,022,647 cells and 8,578,503 points. One important optimization is to realize that the geometric layout of the mesh does not change between frames. While the files contain the whole grid every time, it is only necessary to save the point and cell position data once. Only the density, velocity, and shear stress change over frames.

`vtk2np.py` extracts and saves the following data from the VTK files as Numpy arrays:

- `point_pos.npy` - the (x, y, z) position of each point on the mesh
- `cell_pos.npy` - the (x, y, z) position of each cell center of the mesh
- `cell_vol.npy` - the volume of each cell in the mesh
- `drug_framenum.npy` - the volume of the drug in each cell of the mesh at this frame
- `drug_point_framenum.npy` - the volume of the drug at each point on the mesh at this frame
- `vel_framenum.npy` - the blood velocity at each point on the mesh at this frame
- `vel_cell_framenum.npy` - the blood velocity at each cell on the mesh at this frame
- `rho_framenum.npy` - the blood density at each cell on the mesh; proportional to pressure

This program was run once locally to create numpy arrays, allowing the next Python program to run without worrking about VTK data structures.

`rbc.py` performs all the requested calculations and generates the 2D plots presented below that were not generated remotely using Paraview.
`drug_delivery` answers the headline question of how much drug is in the stenotic region as a function of time. We estimated this by summing up the density of drug in the cells in the stenotic region. We extracted the velocity of each cell and found that they were all equal to 1. (We knew that the interior cells should have a volume of 1 because they were cubes with side length 1, but didn't know how MUPHY and VTK handle cubes on the boundary.) The total amount of drug present in a region Ω is $\int_\Omega \rho dV$ i.e. the density integrated with respect to infinitesimal volume slices. Therefore discrete analog would be to sum up the density of each cell in the stenotic region multiplied by its volume. Since all the cell volumes are equal to 1, we omitted the multiplication from the calculation to improve performance. We also computed the total quantity of drug in the system as a quality check, expecting it not to change.
`plot_speed_contour` creates a contour plot of the speed on a cross sectional slize $z = z_{plot}$. While we computed drug quantities using cells, we chose to plot speed using points. The main programming challenge is generating a contour for data that doesn't cover an entire rectangular grid. This was done by creating an augmented grid with the full square cross section containing the circular cross section of the artery. Grid points outside of the artery were marked with a speed of 0, which was exlcuded from the range of values in the contour.

`plot_streamlines` plots streamlines of the velocity in the xy plane (i.e. velocity components u and v) on a cross-sectional slice $z = z_{plot}$. Like the speed contour, this plot uses points rather than cells. It uses the same technique as `plot_speed` for padding the circular region with dummy entries that don't appear on the plot. Streamlines are plotted with a width proportional to their speed in the xy plane.
`plot_drug_conc` plots the concentration of the drug on a cross sectional slice $z = z_plot$. This plot uses cell data. A mask is created using the relationship that z at the center of a cell is equal to z at the start of the cell plus $\frac{1}{2}$. This was preserved exactly by VTK so we could create a logical mask efficiently. The conentration of drug at each (x, y) point is then read right off the masked array.
`plot_drug_profile` plots the "profile" of the drug along the z axis for a given time. This calculation is also based on cells. A mask is created for each value of z as before, and the total amount of drug at this longitude is estimated as the sum of the drug concentration array on the mask. An initial attempt at plotting this profile showed some obvious artifacts where a handful of z values had "holes" where the dropped well below their neighbors. We appplied a smoothing operation before plotting the profile $B(z)$ in which we set each b_z equal to the max of itself and its two neighbors b_{z-1} and b_{z+1}.
Plots of each type are displayed below in the Results section.

4 Parameters of the Simulation

These are the parameter values that we set at the start of the Python script for the baseline simulation with 0 red blood cells and a Reynold Number of 10.

- ν = 0.1 - this is the kinematic viscosity
- ρ = 1 - the blood density wsa set to 1 by convention
- $\bar{u}$ = 0.01 - this is the mean speed of the blood
- Re = 10.0 - the Reynolds number is dimensionless and characterize the turbulence / regularity of the flow
- Pe = 10.0 - the Pectet number is dimensionless and characterizes the importance of radial to axial diffusion
- R = $Re*\nu/2\bar{u}$ - the radius is implied by Reynolds number and velocity
- DIFFUSIVITY = $\frac{\bar{u}*R}{Pe}$ SAY SOMETHING HERE
- C0 = 0.01 - the baseline drug concentration away from the bolus
- C1 = 1.0 - the high drug concentration on the bolus at $t = 0$
- NSTEP = 1000000 - total number of simulation steps; each step is one microsecond
- NDIAG = 100 - interval between diagnostics
- NVTKFREQ = 1000 - interval between VTK output frames
- UNFREEZE_TIME = 100000 - number of time stepss for the system to equilibrate before drug release

Our goal was to also run a big simulation. This was to be similar to the baseline, but would also include a large number of red blood cells. These red blood cells would be simulated as rigid particles with a position and orientation. Including the red blood cells makes the simulation more physiologically realistic, with increasing importance the narrower a blood vessel is. Our initial attempt was a 30% hematocrit with otherwise identical parameters to the baseline case. Unfortunately multiple attempts to simulate the system with large numbers of red blood cells failed. We will explain in detail the various failures and their causes in the section below. Fortunately, we did complete a successful run of the baseline case without

red blood cells. We have made the best of a difficult situation by perofrming a complete analysis on the baseline case.

5 Results

5.1 Failed Runs on Odyssey with Red Blood Cells

YUE TO ENUMERATE VARIOUS FAILURES HERE USING EMAIL LOGS

5.2 Drug Delivery Over Time

As described in greater detail in the Description of Code, we computed the quantity of drug in the stenotic region and the whole system by summing the density over cells. This was a simple sum of the density because each cell has a volume of 1.0. In general, we would need to sum over concencration times cell volume. We started by computing the total amount of drug simulated to be in the system, because it should remain constant after the simulation starts. Our frist attempt at this calculation incorrectly used points rather cells, and we noticed a marked increase in drug over the first 1.0 second, greater then 10%. This was an indication something was wrong and we realized we needed to use cells. Here is the plot of drug volume in the system. The axis is scaled to start at zero so our eyes can read off the magnitude of the fluctuation in this ostensisibly invariant quantity. The simulation has the total quantity increasing by 3.59%.

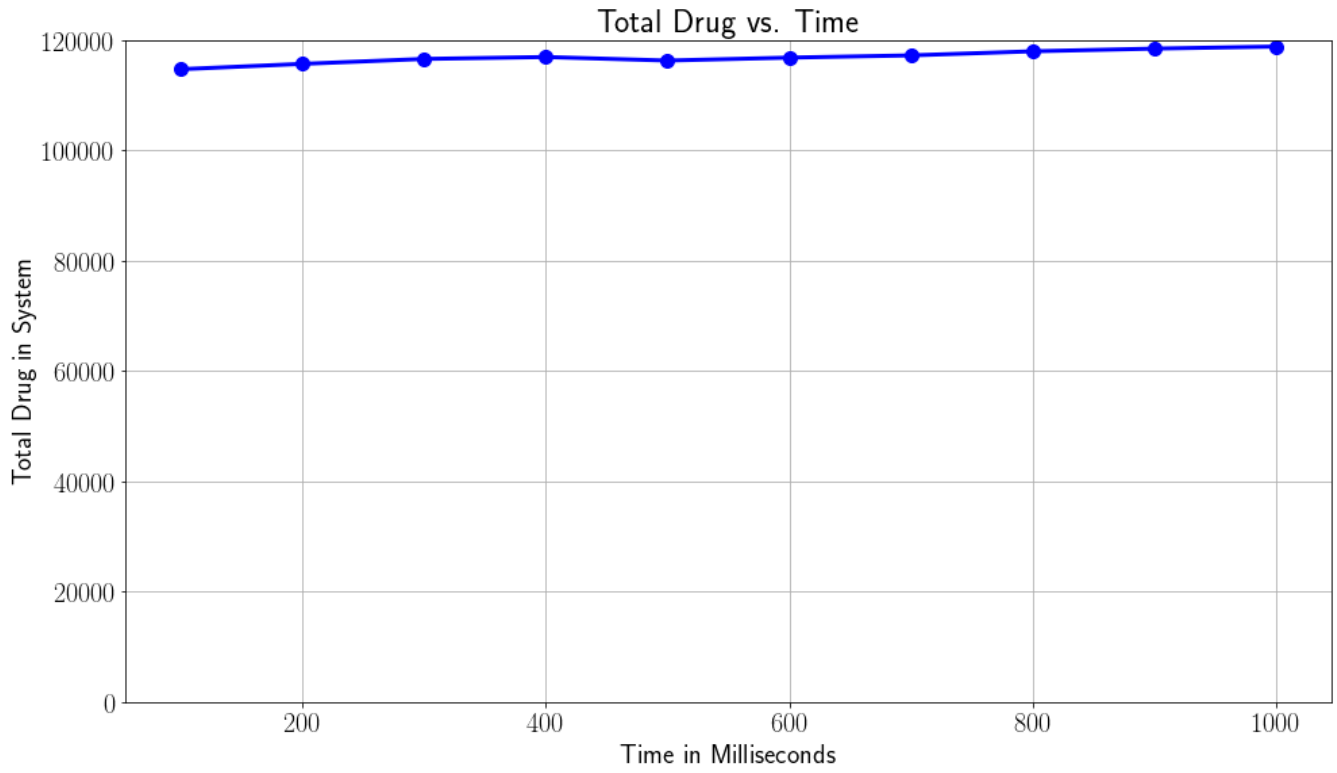


Figure 2: Total Drug Quantity in the System Over Time

Here is the requested plot showing the quantity of drug in the stenotic region. Because the absolute units in this problem are somewhat arbitrary, we are presenting the amount of drug in the system as a fraction of the total. We show the fraction in the stenotic region over time as the blue series. We compare this to a flat line in red, which is the fraction of the total volume occupied by the stenotic region. This was computed by counting the cells in the stenotic region using the same mask we used for the drug quantity. (The sum of volume is a simple cell count since each cell has volume 1.0). When the system reaches equilibrium, we would expect the drug to be completely diffused and at a uniform concentration. In that case, the fraction of drug in the stenotic region would be equal to the volume fraction. By comparing these two quantities,

we can develop an intuition as to how much the drug has been delivered vs. its equilibrium. We can see

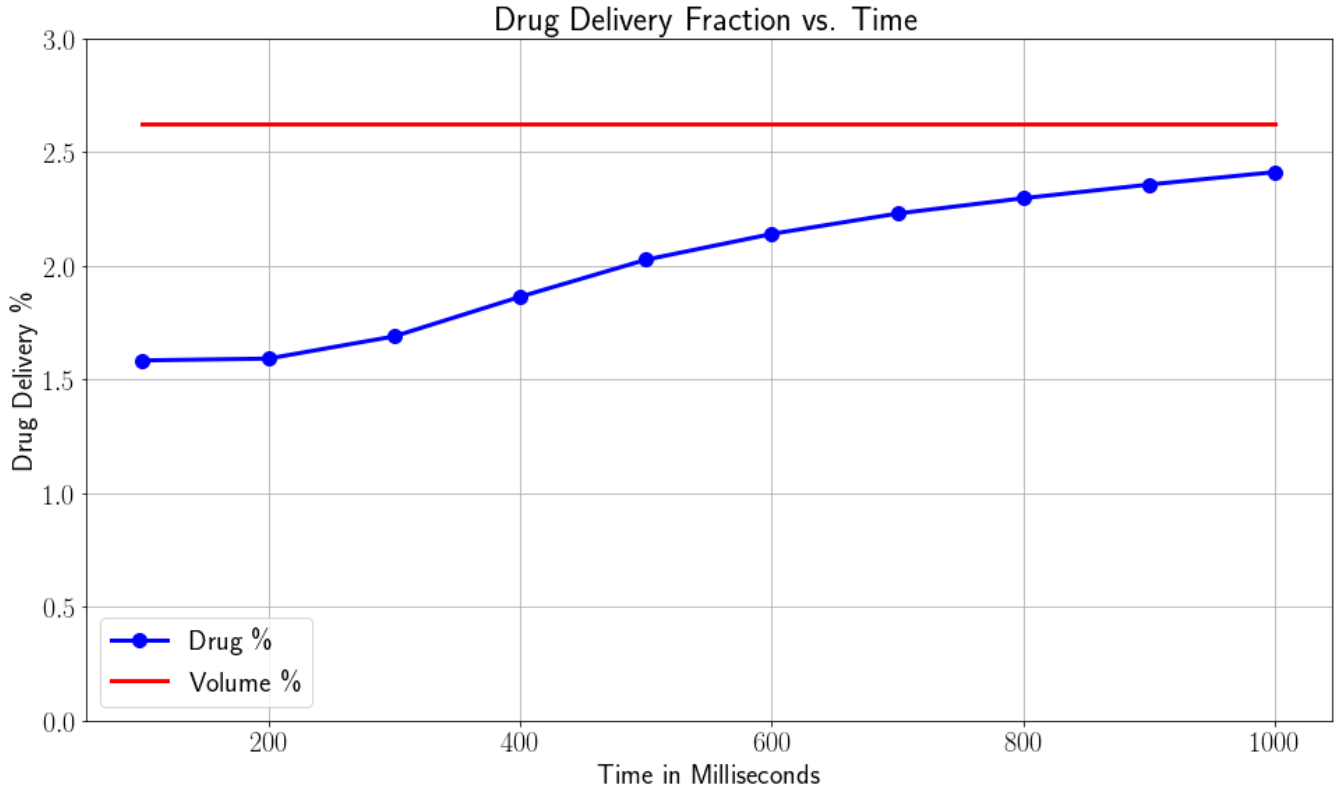


Figure 3: Relative Drug Delivery to Stenotic Region Over Time

that by the end of the first second, the drug delivery is approaching its equilibrium level, showing that it disperses quite rapidly.

5.3 Velocity Field - Contours of Speed and Streamlines

Here are two plots showing the contours of flow speed at time t=800 milliseconds, when the flow is fairly well established. We plot cross sections at two locations: the center of the stenosis, $z = 500$, and to the right of the stenosis at $z = 800$. At the center of the stenosis, we can see that the flow is fastest in the center, and slowest close to the walls, as we would expect. The difference in speed is less pronounced but still present away from the stenosis. There is also an interesting effect where the speed is greatest not at the center, but to one side. This is not a permanent feature of the flow; the "hot spot" moves around.

Here are two analogous plots for the same two time instants and z cross sections showing streamlines in the XY plane instead. In looking at these plots, we are becoming suspicious that there may have been a conceptual error in the boundary conditions. Dr. Melchionne suggested that we set the periodicity flag to '111' indicating that all three components x, y and z have periodic boundaries. We had initially planned on setting this parameter of '001' indicating that the z axis was periodic, but x and y were not.

In looking at the last streamline plot above, it seems as if a strong flow has developed in the XY plane that is wrapping around from the southeast corner to the northwest corner. We are writing this remark too late in the development process to re-run our simulation and post-processing pipeline. As scientists we prefer to be honest and express some reservations about one element of this computation than to try to sweep difficulties under the rug. For the purposes of a course project under these time constraints, we believe this is a solid effort that meets the requested requirements. If this were a paper to be submitted to peer review or an analysis that would be used in treating patients, we would need to do additional work to get

a definitive answer about whether the periodic boundary conditions on the x and y axes were incorrect.

5.4 Drug Concentration - Contours

Here are two contour plots for the concentration of the drug at the center of the stenosis at $z = 500$. They are run at times of 200 and 800 milliseconds. The scale on these charts is tricky. There is only a very small variation in concentration between the lowest and highest end of the scales. Still the visual pattern is clear, and we can see that the drug concentration is highest in the center and lower on the edges.

5.5 Longitudinal Drug Profile vs. Time

We would like to reiterate that we needed to apply a small degree of smoothing to generate interpretable plots for the drug profile. We computed the concentration as a function of z and t using the finest resolution possible, $z = 1$. Mostly the results were good, with just a few artifacts. We applied a simple filter with a width of $z = 1$ to get a picture for what is really happening in the system. All the plots below are scaled with the y-axis starting at zero. This allows us to compare profiles at different time and develop an intuition about how the bolus migrates left to right and diffuses outward over time. Without a common scaling, all the charts look very similar, and it is much harder understand the interplay between advection and diffusion. We plot the profile at times of 200, 300, 400, 600, 800 and 1000 milliseconds. The profile at

6 Conclusions and Future Work

References

[1] Succi, Sauro: The Lattice Boltmann Equation for Fluid Dynamics and Beyond

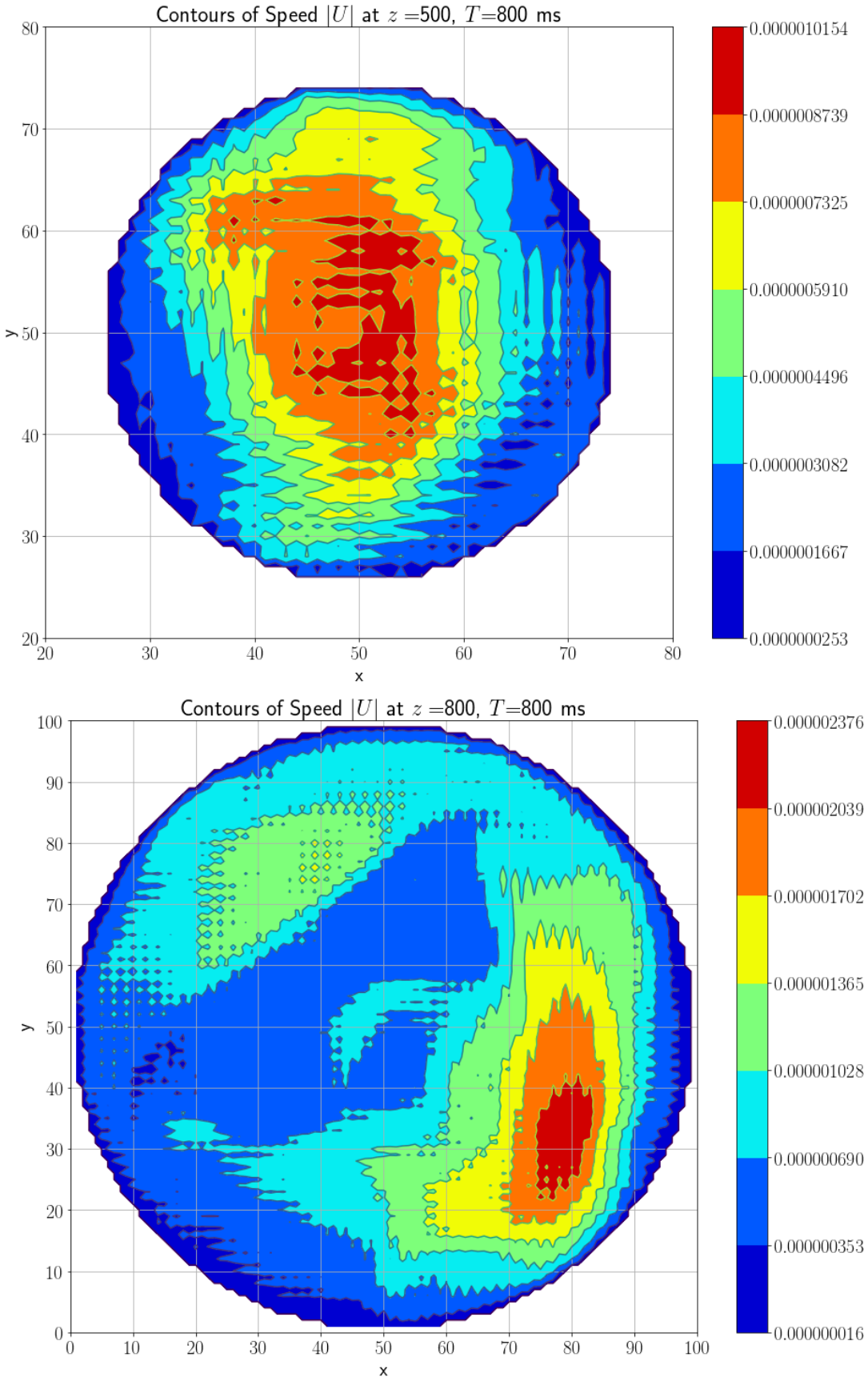


Figure 4: Contours of Speed at time t=800 ms; at two locations, center and right of stenosis

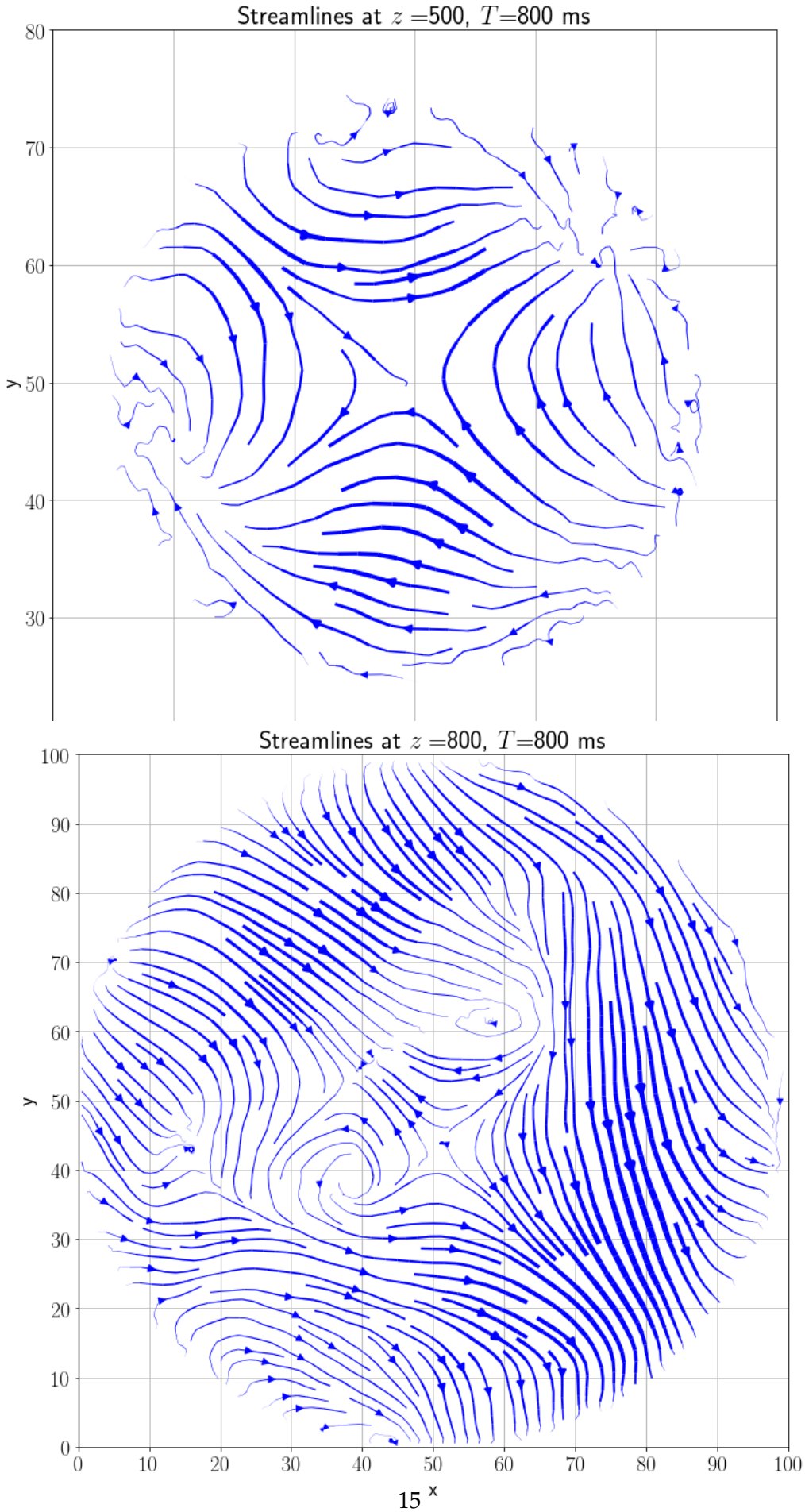


Figure 5: Streamlines at time t=800 ms; at two locations, center and right of stenosis

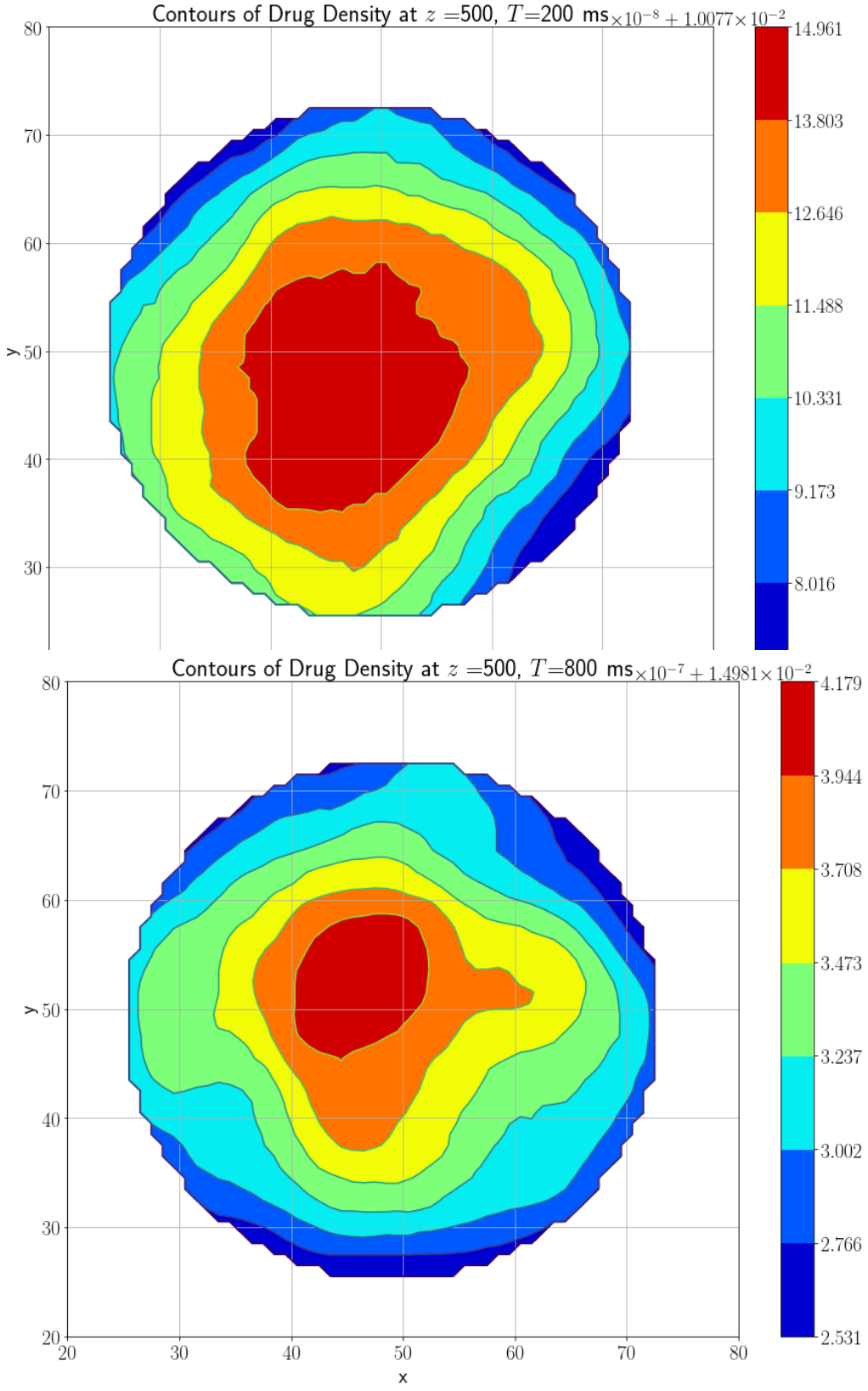
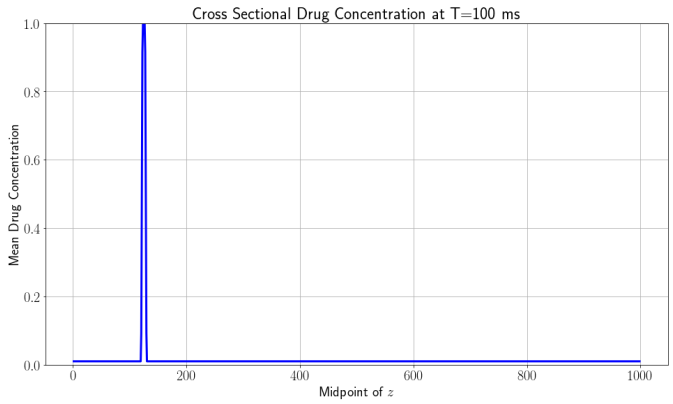
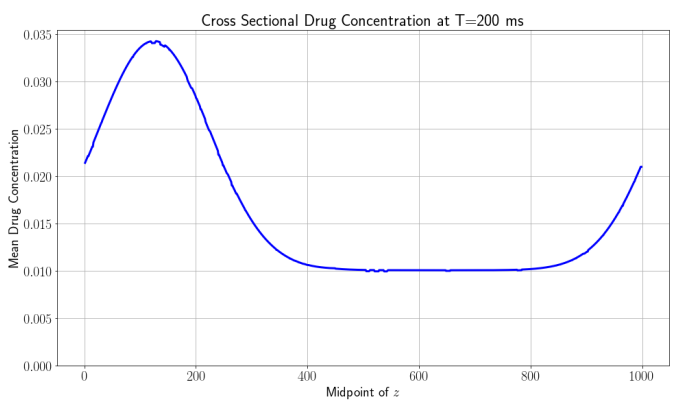
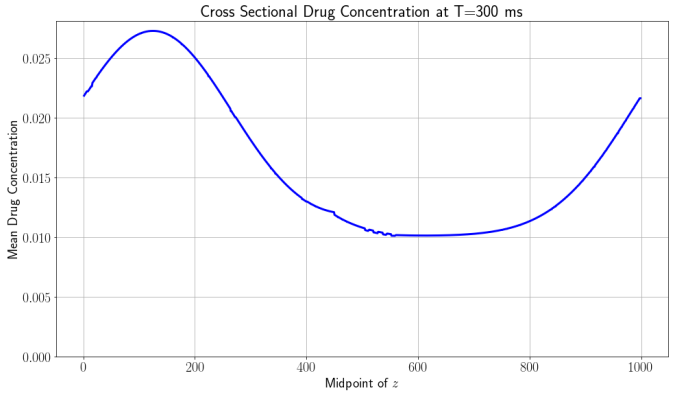
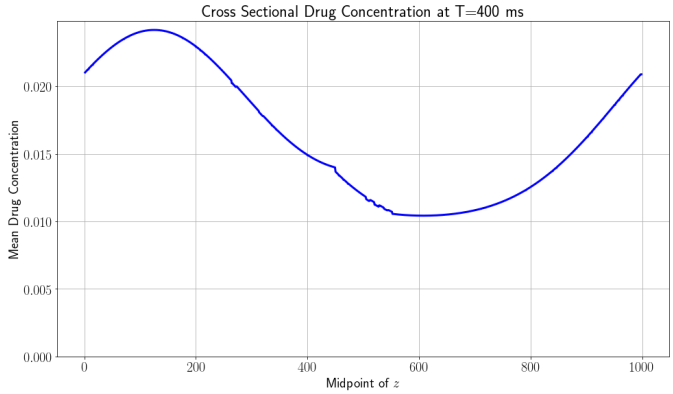


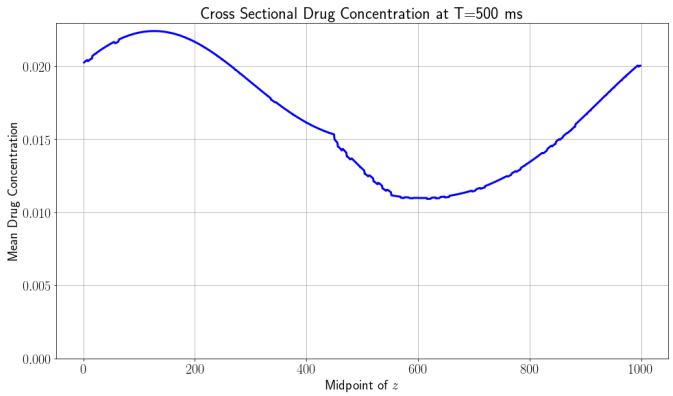
Figure 6: Drug concentration at center of stenosis at two times, 200 and 800 ms

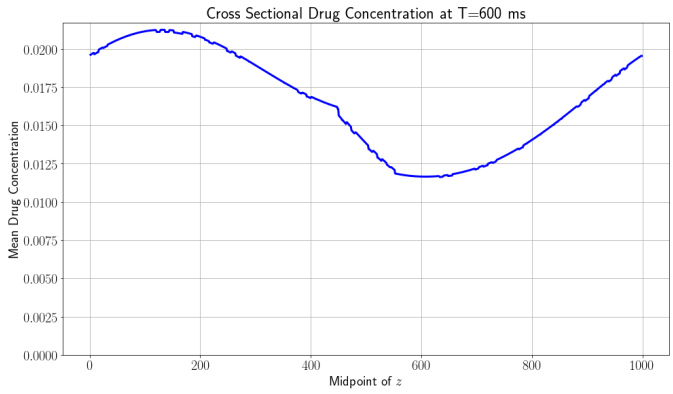

Cross Sectional Drug Concentration at T=100 ms
Mean Drug Concentration
Midpoint of z

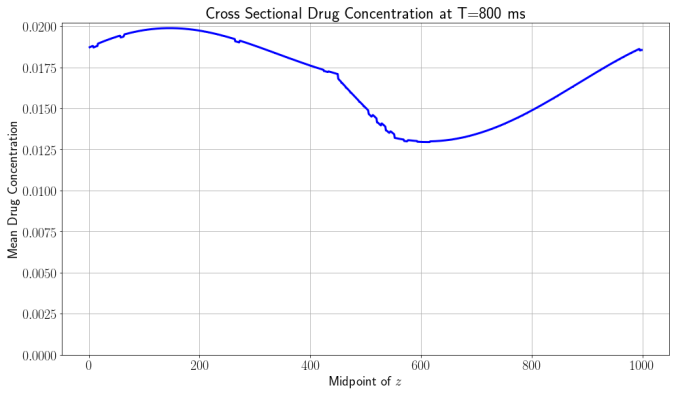

Cross Sectional Drug Concentration at T=200 ms
Mean Drug Concentration
Midpoint of z

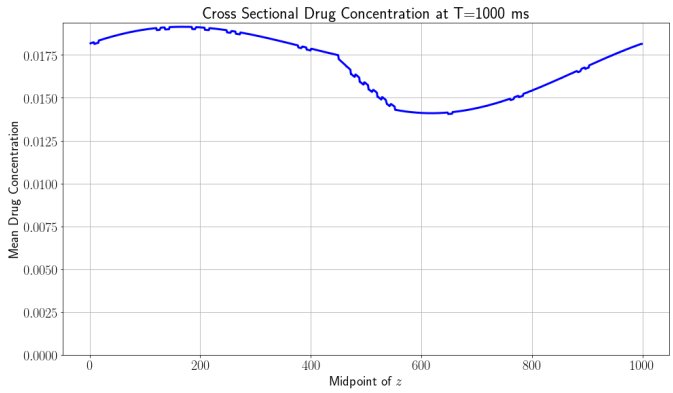

Cross Sectional Drug Concentration at T=300 ms
Mean Drug Concentration
Midpoint of z


Cross Sectional Drug Concentration at T=400 ms
Mean Drug Concentration
Midpoint of z


Cross Sectional Drug Concentration at T=500 ms
Mean Drug Concentration
Midpoint of z


Cross Sectional Drug Concentration at T=600 ms
Mean Drug Concentration
Midpoint of z


Cross Sectional Drug Concentration at T=800 ms
Mean Drug Concentration
Midpoint of z


Cross Sectional Drug Concentration at T=1000 ms
Mean Drug Concentration
Midpoint of z