

# Spotlight on Learning Touch(ed)



Pioneer Theatre Company's Student Matinee Program is made possible, in part, through the support of Salt Lake County's Zoo, Arts and Parks Program, The Simmons Family Foundation, The Meldrum Foundation Endowment Fund, and R. Harold Burton Foundation.

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**GEORGE Q. MORRIS  
FOUNDATION**

**Approximate running time:** Two hours and 20 minutes. This show has one fifteen-minute intermission.

**Student Talk-Back:** There will be a Student Talk-Back directly after the performance.

## SPOTLIGHT ON LEARNING JANUARY 2010

A Pioneer Theatre  
Company Classroom  
Companion

Directed by  
**Charles Morey**

# Touch(ed)

Jan. 8 thru Jan. 23, 2010

A World Premiere written by Bess Wohl



"...IN ANCIENT TIMES..THEY THOUGHT IT WAS GOD WHO HAD TOUCHED THEM. AND WHEN HE DID, HIS HAND WAS SO HOT THAT IT BURNED RIGHT THROUGH."

—BILLY IN *TOUCH(ED)*

Pioneer Theatre Company was very fortunate to have playwright Bess Wohl in house during the weeks leading up to the production of the world premiere of her third play, *Touch(ed)*. Ms. Wohl was asked to share a few words about the development of this play.

**N**ormally I start writing a play because something is bugging me—either in my own life or out in the world—and I have to write to figure it out. If it's something that makes me too irate, the play ends up being a screed, and if it's something I don't really care about, the play dies at page twenty-five. But if it's bugging me consistently, like a pebble in my shoe, and I start writing to try to get rid of it, I may very well end up with a play.

**I**began working on *Touch(ed)* several years ago on a lonely and strange trip I took to the Berkshires. I was a Brooklyn girl, stranded in the "wilds" of Massachusetts, and even though it was idyllic Norman Rockwell country, it seemed to me surreal and frightening. I felt totally alienated and convinced that at any moment I would be swallowed up by a bear. And so, of course, I began to write.

**T**he pebbles that were bugging me at the time involved questions about what family members owe one another, the effect of a weakened family member on the surrounding members, the way we all balance taking care of each other and insisting on our own freedom—and what the cost of that freedom is.

**S**o that was what I intended to write about—but the most exciting moment for me is always when new themes start emerging and the play becomes about something unexpected. After all, if a play winds up being about exactly what you thought it was, you might as well have written an essay. What excites me is the force of the unconscious, and the strange way that creative works start to take on a life of their own.

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For example, I had started out thinking it was obvious who the most “crazy” character in the play was, but now I’m not so sure. In fact, I’m not even sure I know exactly what it means to be “crazy” anymore, and what it means to be sane. As I wrote and rewrote the play, my sympathies began to shift from character to character, and I started to see the world from their points of view—much more interesting to me than my own.

And then, of course, there was the delicious moment of watching the characters “become flesh, literally” (to borrow a phrase from Billy) as this incredible company of actors began to embody them in rehearsal, and [Director] Chuck [Morey] began to uncover moments and nuances I never knew existed in my own work. (I also realized, for the first time, how many props I had written into this play! Zester? Melon baller? Breadmaker? What was I thinking?)

By now the pebble in my shoe, far from disappearing, has become more like a mountain, one that never lets you reach the top. But that’s what’s exciting to me about this process—setting out to find answers and ending up with a mystery.

—Touch(ed) Playwright Bess Wohl

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June 13, 2008

EXPERT Q & A

## Visualizing Schizophrenia

By IRENE WIELAWSKI, New York Times

Paul Thompson is professor of neurology at the University of California, Los Angeles, and leads the research group at the school’s Laboratory of Neuro Imaging. He uses imaging technology to map disease processes involving the human brain, carried out in collaboration with the National Institutes of Health and more than 40 laboratories around the world. A goal is to create disease-specific atlases of the brain that can aid in the diagnosis, treatment and possible prevention of illnesses like schizophrenia.

**Q:** Your team has found evidence of significant and progressive brain damage in people with schizophrenia. What areas of the brain are affected, and how does this account for symptoms?

**A:** The damage in schizophrenia appears specific to two basic areas: the parietal cortex and the frontal lobe. The parietal cortex is located just above the temple area by the ears; it’s the part of the brain that makes sense of what we hear, see, taste or touch — essentially, our sensory experience. We know about differences in function between a normal parietal cortex and a damaged one from people who have suffered brain trauma. They can’t make sense of what something is. They may be given an apple or an orange, and they can see it and touch it, but they can’t name it or understand its purpose.

The frontal lobe helps us organize our lives, go to work, analyze information and make decisions. This area of the brain is where teenagers have the most developmental changes — a process of pruning excess cells and streamlining brain function until it reaches its adult form around age 25. This reshaping process seems to go profoundly awry in young people with schizophrenia. Instead of healthy pruning, you see massive loss of brain tissue. Because the frontal cortex is also the part of the brain that prevents you from doing things that are rash, a result of this damage is that people with schizophrenia may behave in a bizarre way; they may shout in public or react in an exaggerated way to minor upsets. Ten percent of schizophrenia patients die by suicide.

**Q:** What causes the damage, and over what period of time does it take place?

**A:** Mapping this timeline was one of the things we wanted to accomplish through our imaging studies of young people with schizophrenia. From images taken at regular intervals of literally hundreds of patients and control subjects, we created an aggregate image of the disease process — basically, time-lapse movies of what happens when and at what rate. In the movies, you see this traveling wave of tissue loss, starting with the parietal cortex and then relentlessly sweeping forward into the frontal lobe. We’ve calculated the tissue loss at over 5 percent a year, which is comparable to Alzheimer’s disease — brain cells are actually dying as a result of schizophrenia.

**Q:** Clinically, there’s great variation in schizophrenia patients. Some are able to hold jobs and sustain relationships while others are severely disabled. How does this variation show up in the brain?

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A: It appears that the amount of tissue loss depends upon the age at which you develop the illness. If it comes on in your early teens, up to 25 percent of your brain tissue can be lost over a period of about five years. That is very severe — comparable to Alzheimer's in the degree of damage, but different in that schizophrenia does not attack every area of the brain. If you develop schizophrenia later, with your first psychotic episode in your latter 20s, brain tissue loss appears to be no more than 1 percent a year. Because it is a much slower process, the opportunities to intervene with drugs are greater. In brain scans of people who developed schizophrenia later and have lived with the illness for a long time, we see maybe only 10 percent to 15 percent of tissue loss over all.

Q: For centuries, mental illness could be described only by its external symptoms, with causal theories that ranged far outside the boundaries of science, including devil-possession and witchcraft. How did scientists come to see schizophrenia as a brain-damaging disease?

A: The earliest sign of structural differences in the brains of people with schizophrenia came in the 1970s. Eve Johnstone, a scientist in Scotland, used 3-D X-ray and found that the fluid-filled spaces in the brain, called ventricles, were abnormally large in people with schizophrenia. There was huge controversy when she reported it. A lot of people didn't believe it, partly because you couldn't see the pathology on autopsy the way you can with neurologic diseases like Alzheimer's, where the amyloid plaques so toxic to brain cells are clearly visible. It led to a huge flurry among scientists to identify which parts of the brain are damaged in schizophrenia, why you don't see pathology on autopsy when you can see it on imaging, and so on.

The next big step was having the tool of M.R.I. in the mid-1980s, which greatly aided these investigations. But because no two brains are exactly alike structurally, it's difficult to identify a subtle disease process or make a diagnosis simply with one image — as you can with a single X-ray of a broken leg. It required a lot of mathematics, a lot of computer science and really a lot of ingenuity to figure out what were the collective differences between people with healthy brains and people with schizophrenia. We needed to establish scientifically consistent patterns of difference.

Q: How did you achieve this?

A: Judith Rapoport at the N.I.H. proposed imaging the brains of children with schizophrenia every two years in order to assemble a scan database to see if there were changes over time. Similar studies were under way in Scotland. Basically, by the year 2000, we had hundreds and hundreds of scans from schizophrenia patients and from controls, collected every two years over six years.

What we then did was to merge them into a time-lapse movie of brain changes in people with schizophrenia. It's comparable to what you would get from time-lapse photography of the weather in your backyard if you took a photo every hour. You would see clouds moving around and wind and maybe some rain, but if you were simply standing outside you wouldn't necessarily focus on these changes or notice how they came about. By using mathematics, we were able to string these images into a movie; the random variations go away if you have enough scans.

Q: Were the movies surprising?

A: We were absolutely staggered by the amount of tissue loss in the subjects with schizophrenia compared to controls. We had expected, of course, to see some loss of tissue, because we already knew from earlier findings that there were excessive fluid-filled spaces in the brains of people with schizophrenia, which suggests tissue loss. But the degree of loss that we saw in our scans was shocking.

We were also surprised to find that this destruction has a shifting pattern. Most neurological illnesses affect one part of the brain. If you have epilepsy, for example, typically the seizures have a focus. But it is quite different in schizophrenia. You have this progressively spreading wave of grey matter loss — brain cells that can never be replaced.

The first sign of schizophrenia is usually a psychotic break, with hallucinations and sensory distortions. The patient may think someone is talking to them who is not really there, for example. Our brain scans in this very early stage of illness showed structural changes in the parietal cortex — damage that would be consistent with these psychotic symptoms. But then the tissue loss progressed to the frontal lobe, which controls our ability to regulate behavior and make sense of sensory perceptions. These images support a theory of schizophrenia that there's some trigger that causes the normal pruning of brain cells that goes on throughout the teenage years to be accelerated, leaving the patient with insufficient brain tissue to function normally.

Q: These movies are quite stark in their depiction of schizophrenia's impact on brain tissue, yet you refer only to "theories" of how and why.

A: That's absolutely correct. Schizophrenia is so horrendously complicated from a scientific point of view that everything one says has to be qualified. This is true for all the mental illnesses. There's simply no agreed-upon physical marker in the brain for what causes them. There are various theories, but even the most basic information is a matter of debate.

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This makes mental illness very tricky to treat, in contrast to many neurological illnesses. In Alzheimer's disease and epilepsy, for example, we know exactly what is happening in the brain. We may not be able to prevent it in all cases, but we know what the structural and functional change is, which greatly helps us to develop treatments to intervene somehow in the disease process and stop or lessen the damage. In mental illness, we're not so far along.

Q: What cause-and-effect theories are scientists pursuing?

A: There are three basic theories, all of which rest on a genetic base, since schizophrenia runs in families. We've already discussed one — that some unknown trigger causes exaggerated pruning of brain cells, leaving the patient with insufficient tissue to function normally.

The second theory has to do with inadequate myelin coating. Myelin is a taffy-like substance that insulates your brain cells and enables communication among them — as much as 100 times faster than if the cells had no myelin. We know that some of the drugs that are effective in treating schizophrenia promote myelin growth. So if you put the drug findings together with the cell damage findings, it makes sense that even with drastic loss of brain tissue, improved myelin growth could ameliorate symptoms.

The third theory has to do with chemical imbalance, specifically excessive amounts of the brain chemical dopamine. Some schizophrenia cases are environmentally triggered; there may be a genetic predisposition, but the activating trigger is external — stress, possibly, or trauma or, in a significant number of cases, drug abuse. Schizophrenia-like symptoms have been observed in people who use methamphetamine, and we know the effect of this drug is to stimulate the release of a huge amount of dopamine into the brain. At the same time, we know that some medicines for schizophrenia act to limit dopamine. This makes a very powerful case for schizophrenia being caused by dopamine imbalance.

Q: Even if the specific mechanism of schizophrenia remains elusive, how can better knowledge of its impact on brain structure contribute to treatment?

A: With any illness, it is extremely important to know if it is progressing. If you are a patient or the doctor treating a patient, you need information on the degree of change not only in clinical symptoms — how the patient feels, say — but also with regard to what's going on inside the body. In cancer, for example, we can measure tumor size to know if the chemotherapy is working. This is just as important for mental illnesses.

There are many questions which better knowledge of brain changes could help answer. How far along is the disease? Is a particular medication effective in slowing or preventing progression — actually saving brain tissue? If so, when is the best time to start a patient on this medication? Is the maximum effect achieved in the first year, or should they continue taking it, bearing in mind that many of the drugs have side effects? And for people with a family history who are worried about developing schizophrenia, can you reassure them that their brain is O.K.? All of these questions require a means to see into the brain, understand the difference between normal and damaged structures, and measure progression in the context of treatment.

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## UNDERSTUDIES IN THE THEATRE

Touch(ed) uses four “understudies.” These are performers who learn the lines and blocking (or choreography) of additional roles should those actors be unable to appear on stage because of illness or emergencies. Theatres may or may not employ understudies for most roles, and when they do, they do so for different reasons.

When an understudy takes over, the theater will make an announcement prior to the start of the performance. In fact, AEA actors (members of Actors' Equity Association, the professional union for actors) require that an announcement be made in two of the following three ways: an announcement from the stage before curtain, a sign in the lobby, and/or an insert in the playbill.

In some productions, especially musicals, understudies may also be a part of the ensemble or chorus when not used to fill in for a role, but in other productions, like Touch(ed), the understudy may not appear on stage at all unless the actor they have understudied cannot.

Questions:

1. Think about what is required of an understudy, especially if they also have a part in the play. What challenges might that actor have?
2. Look at the bios for the actors who understudy the four roles in Touch(ed). Do you notice some things they all have in common? PTC, while we are a professional theatre that primarily uses Equity actors, is closely associated with the University; what might that tell you about how and why these particular understudies were selected?

# Director and Cast for *Touch(ed)*

**CHARLES MOREY** (Director) has been the Artistic Director for PTC since 1984 and has directed over 70 productions. He is pleased to present the world premiere of the first play produced as a result of PTC's New Plays Initiative.

**KELLY HUTCHINSON\*** (Emma) Films include *Catch Me If You Can*, *Slippery Slope*, and *Hysterical Psycho*. On television, Kelly has been seen on *Law & Order*, *Law & Order: SVU*, *Hack*, *The Jury*, as well as a recurring role on *Strangers with Candy*. Broadway credits include *Desire Under the Elms*, *Major Barbara*, and *Macbeth*. Off-Broadway, she has been seen in *Or* (Women's Project), *The Voyage of the Carcass* (Soho Playhouse), Tony Kushner's *Homebody/Kabul* at New York Theatre Workshop, and *Romola* and *Nijinsky* at Primary Stages.

**DENNIS PARLATO\*** (Doctor) most recently appeared at PTC in *Paint Your Wagon* and *The Vertical Hour*. His film credits include Tom DiCillo's *Johnny Suede* and *Delirious*, Eddie Muller's *Dillinger's Dead*, Greg De Felice's *Bury the Evidence*, *Rick*, *First Born*, *Starting Out in the Evening* and *Bride Wars*. Broadway credits include: Lawrence in *Dirty Rotten Scoundrels*, Mr. Robinson in *The Graduate*, John the Baptist in *Salome*, Captain Von Trapp in *The Sound of Music*, CIA agent Walter Anderson in *Chess*, and Rooster in *Annie*.

**ALEX PODULKE\*** (Billy) is very happy to return to PTC after appearing here in *The Heiress* and *Enchanted April*. His film and TV credits include *Welcome to the Cosmos* and *Guiding Light*. His recent New York credits include *Corleone*, *the Shakespearean Godfathers* at the NY Fringe Festival, *Flesh and the Desert* at Theatre Row, *Romeo and Juliet* in Central Park, and *Cyrano de Bergerac* at the Metropolitan Opera. He has numerous regional credits including six seasons at the Guthrie Theater. Alex's one-man show *DNA* and *the Dancing Fool* has been produced in MN and CA. Alex is a graduate of the London Academy of Music and Dramatic Art.

**JENNIFER JOAN THOMPSON\*** (Kay) is making her Pioneer Theatre debut. She has appeared on television in *The Good Wife*, *All My Children*, and *Guiding Light*. Her Broadway credits include *Dividing the Estate* (Lincoln Center Theater). Regionally she has appeared in *Bloodline: Children of Argos* and *Julius Caesar* (both at the Hangar Theatre). Her New York credits include *Gallathea* (HERE), *Measure for Measure*, *House of Blue Leaves*, and *Holiday* (NYU Grad Acting). Jennifer holds a BA from Yale University and an MFA from NYU/TISCH.

## UNDERSTUDIES:

**NATALIE BLACKMAN** (u/s, Emma) recently appeared as Sophie in *Master Class* at Salt Lake Acting Company, and has performed many roles with University of Utah Theatre. Favorites include Little Sally in *Urinetown*, Mamma in *The Cripple of Inishmaan* and Maria in *Treasure*. She attended Circle in the Square Theatre School and will graduate of the University of Utah's Actor Training Program in 2010.

**STACEY HULL** (u/s, Kay) is a native of Glendale, CA and a senior in the U of U's Actor Training Program. She has appeared in several Babcock Theatre productions, including *The Seagull*, *Blood Wedding*, *Medea*, and *Helen*. She was also seen in Studio 115's *Bus Stop* as Cherie and is a recipient of the Victor Jory Scholarship. Stacey is very thankful for the opportunity to understudy this wonderful production.

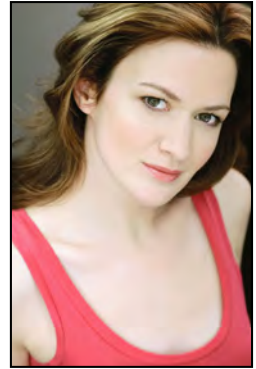
**JAKE MILO KOEPL** (u/s, Doctor) is excited to be working on his first production at Pioneer Theatre. He recently played Eugene in *Biloxi Blues*, and E.J. Lofgren in *Harvey*, both at the Neil Simon Festival, and Alan in *Time and the Conways* at the Babcock Theatre. He just completed his BFA in the Actor Training Program at the University of Utah and is very pleased to be using his education.

**ANDY RINDLISBACH** (u/s, Billy) has appeared with the U of U Theatre as Billy in *The Cripple of Inishmaan*, Katurian in *The Pillowman*, and recently as Dionysus in *The Bakkhai* for the department's Classical Greek Theatre Festival. Other favorite roles include Jimmy in *The Rainmaker* at Hale Centre Theatre and Jason in *Rabbit Hole* at SLAC. He will graduate from the University of Utah's Actor Training Program in 2010.

\* Represents member of Actors' Equity Association.

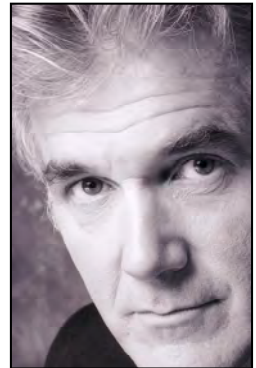
**KELLY HUTCHINSON**

Emma



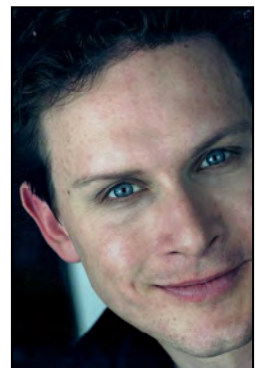
**DENNIS PARLATO**

Doctor



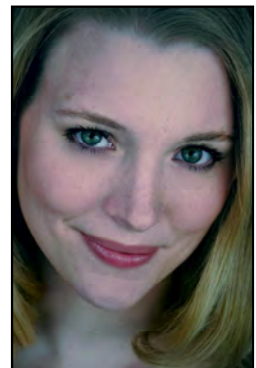
**ALEX PODULKE**

Billy



**JENNIFER JOAN THOMPSON**

Kay



More cast information at:  
[www.pioneertheatre.org/  
2009-2010-season/touched](http://www.pioneertheatre.org/2009-2010-season/touched)