

Author John Barry talks about the 1918 flu pandemic and COVID-19

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In today's COVID-19 Update, the author of the #1 New York Times best-seller, "The Great Influenza: The Story of the Deadliest Pandemic in History" is a sought-after expert on influenza preparedness and response. He talks about lessons learned from the 1918 pandemic and how it compares to COVID-19.

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Speakers

- John M. Barry, author, "The Great Influenza: The Story of the Deadliest Pandemic in History"

Transcript

Unger: Hello, this is the American Medical Association's COVID-19 update. I'm your host, Todd Unger, AMA's chief experience officer. Today, we have a special guest, John M. Barry, author of multiple award-winning books, including the number one New York Times bestseller, "The Great Influenza: The Story of the Deadliest Pandemic in History," which was also named 2004's Outstanding Book on Science and Medicine by The National Academies of Sciences. Since writing it, Mr. Barry has become a sought-after expert on influenza preparedness and response. Mr. Barry, in the book, you say that you wanted to see how American society dealt with simultaneous challenges of two wars, one of nature against humans, on top of a war of humans against each other, to see how people that had the power to do something about it, whether they were politicians or scientists, how they reacted and the lessons we could learn.

And that's pretty much the direction of the discussion that I'd like to have today with you. For starters, I would love it if you could give some quick context on the 1918 pandemic, and how it differed from what we're seeing with COVID-19 now in terms of severity, and contagion, and cases, and deaths and things like that.

Barry: Well, very briefly, of course, it was much more virulent. It killed between 50 and 100 million people. Adjusting for population, that would be 225 to 450 million people today. It was probably less transmissible. An estimated one-third of the world's population was infected, and the U.S. actually seemed to be a little less than that. It looks like about 28%. It was much, much faster. This is one of the biggest differences. Influenza, in general, everything about it is quicker, the incubation period, how long you're sick, how long you shed virus and so forth. So in 1918, probably two thirds of the deaths were in a incredibly compressed timeframe of 14 or 15 weeks, in the fall of 1918. And in any particular city, it was faster than that. Generally six to 10 weeks, herd immunity was established, and then it looked like the disease was gone.

Even if we had not intervened to interrupt transmission, this would have lasted much, much longer than just a serial infection because everything takes longer. So we did, of course, intervene, which I entirely support. I think the herd immunity idea has been pretty much disproved. Even Sweden has all but formally abandoned their effort. And in Manaus, a city of 2 million people in Brazil, then, where 76% of the population was infected, we now see a variant that is surging again. So the natural infection, whether it was pursued as policy. And in Sweden, though they don't use the term, or just happened in Brazil, then it seemed very effective. Yeah-

Unger: I was talking to one of my colleagues about this and said, "Be glad you weren't around in 1918. This was something that could come on in a period of 12 hours, you're turning dark blue and dying."

Barry: Right, right. Most of the deaths were probably bacterial pneumonia, secondary infections, which is still today 6 to 8% case mortality, but a very, very sizable number. We don't know. The exact number was probably more directly due to the virus or the immune cytokine storms, so forth. There clearly were deaths in 12 to 24 hours. The observers who noted them or just too good, reputable, to dismiss them. So it was a much more intense and horrific experience. By the same token, what we are going through today in terms of the duration and the economic damage, it's greater today.

Unger: One of the key differences that you noted in the book with the 1918 influenza was that it was most deadly among younger people. It's almost the opposite about COVID-19. What explains that difference, and how did that affect our response?

Barry: Well, and incidentally, that was as mild as 2009's pandemic was for the people. It was almost, 2009 was almost two entirely different experiences. The vast majority got something even more mild than normal seasonal flu, but for that tiny minority, it was like 1918, including the age of the death. I

think the median age in 2009 for deaths was 30, 31. And in 1918, it was in the mid-to-late twenties. My hypothesis about 1918 is that people's immune systems, of course, stronger when you're younger and overreacted cytokine storm, and so forth. The irony today is older people like myself, our immune systems aren't strong enough to fight the disease off, but apparently they're strong enough to amount enough of an immune response once the virus gets established to create very serious problems. Another similarity today, of course, we have lung COVID. Something very akin to that, although they didn't name, it was clearly the case in 1918. Now, whether that was, or is today, directly because of the virus, which would, of course, be a little bit different because they're different viruses. But to the extent that that is a result of the immune system's response to the virus, that would probably be pretty similar today as it was in 1918.

Unger: Yeah, you noted there was some ... those after effects, including impacts on the brain.

Barry: I think there's very well established neurological impact in 1918. And the other thing, seasonal influenza viruses just about never bind to cells in the lung, that was not the case in 1918. Just as with SARS-CoV-2, the 1918 virus very clearly could bind to cells deep in the lung. So you're starting out with a pretty serious condition if that happened to be the case. And of course, it also could bind to cells in the upper respiratory tract, which made it easily transmissible.

Unger: You wrote that in the other pandemics, the major ones across the century, that you'd seen a pattern of waves where there was a first one that maybe a bit more mild, and a second one that was much more deadly. Are we seeing that pattern now? Because it feels like we're just being in one wave.

Barry: Yeah. I think it's different today. This is all hypothesis and speculation on my part, and I'm not a virologist, I'm an historian. But the first wave in 1918, number one, it was mild, so mild that you saw medical journal articles saying this looks and smells like influenza, but it's not killing enough people, so it can't be influenza. The other thing about the first wave of 1918 is it was very hit or miss. A lot of cities, we don't really know in detail, my guess is probably a majority of cities, at least in the United States, didn't even see it first wave. Los Angeles didn't record a single influenza death in the spring of 1918. There were clearly well-established first wave experiences in New York and in Chicago, although they didn't, they weren't really noticed at the time. Whether the virus wasn't as great as it later became at infecting people, it's hard to say.

In Western Europe, a little bit later in the spring, in fact, those waves in New York and Chicago I referred to tended to be earlier, January, February. In Western Europe, it did get more widespread, but it was still very mild. The lethal second wave, which was what spread around the world very rapidly, even without air travel, that was extraordinarily widespread. It got everywhere, villages in the middle of the African jungle, Inuit villages, pretty deep into Alaska. Today, in fact, I wrote an op-ed back in April for The New York Times in which I predicted, based actually on 1918, I predicted that the summer would not provide relief. And the reason was that I thought susceptibility was more important than seasonality. It does seem to be true, clearly in influenza, and we think in SARS-CoV-2, that

temperature and humidity do affect the ability of the virus to transmit.

But in the summer this time around, we had 95% of the population susceptible, highly transmissible virus. So I didn't expect much relief. What influences the spread of the virus, certainly in the U.S., And probably most of the world, was our control measures. Obviously, public health measures, the non-pharmaceutical interventions, they work. They have worked in some countries better than, I think, anybody in public health would possibly have imagined that you can completely control, if you do it right, a virus as transmissible as this strictly with public health measures. That's astounding. I was part of the initial teams in the Bush administration that recommended MPIs. I don't think anybody on those teams thought they could be more successful. Obviously, we didn't do a great job with that in the United States, but we did use them, and they did tamp down the virus. We never got the baseline low enough, but I think that was really, as you said in your question, that we've seen more continuity. And I think that's more a function of releasing the various measures, or people get tired of complying with them, and so forth and so on. And that has really determined the spread.

Unger: You said in that same op-ed that the most important lesson to be learned from the 1918 pandemic is to tell the truth. Can you elaborate on that? Because I learned a lot in reading about the situation then. How would you on that?

Barry: Well, it's not too much to elaborate on. Another way of putting it is you don't manage the truth, you tell the truth. If you are going to rely on people complying with public health measures to have any impact on the spread of anything, then they better pay attention to what you're saying. And if you have a mixed message, that's not going to work. If you lie to them, they will find that out fairly quickly. And once you lose credibility, people aren't going to pay any attention to you. So clearly, the best strategy, both short and long term, in any crisis situation, and particularly again when you're looking for public compliance with your advice, is to tell the truth and then keep telling it. And then you—

Unger: Yeah, one of the things that I was unaware of, or I hadn't remembered was, basically there was a morale directive in 1918, it was no bad news. And so it was really a withholding of information about the pandemic, itself, as the primary narrative there, no bad news. So I guess—

Barry: Right. Very much so, the motivation was quite different. We were at war, so they were trying to keep morale up and they thought, as you say, any bad news would hurt morale, and therefore, hurt the war effort. That's a lot different from a political decision to advance an individual's political interests, which seemed to have been the case this time around. But the result was the same. They're not exactly, for one thing in 1918, national public health leaders were saying things like, "This is ordinary influenza by another name." However, nobody in the public believed that. This time around, people in the public did believe efforts to minimize the impact of this disease, and still believe it. But in 1918, that did not happen, because the variance of the virus pretty much demonstrated within a matter of hours that this was something to be taken seriously. You had, for example, [in] Philadelphia, I wrote in the book, you literally have priests driving horse-drawn carts down the street, calling upon

people to bring out their dead.

You had bodies staying in houses for 48, 72 hours. You had, as you said yourself earlier, people dying in 12 hours, horrific symptoms. The book quotes a physician writing a colleague that people with cyanosis was so intense that he had difficulty distinguishing African American troops from Caucasian troops. Some of the most frightening symptoms where we have good data in some army camps, you had 15% of the troops had a nosebleed. You had a smaller minority, but it still clearly happened. People would bleed from their eyes and ears. That's pretty terrifying, and particularly to a lay person. So when you have symptoms like this, when you have deaths piling up, when every city in the country is running out of the coffins, nobody is taking you seriously if you're saying this is ordinary influenza by another name. All that did was create an alienation in the population.

And it basically said, "You're not getting any help from us, from people in power. So you are on your own." Trump left it to the states, but in this instance, the government basically left it to the population. It was very alienating. And I quoted in the book, Victor Vaughn, who was dean of the University of Michigan Medical School before the war and during the war, he was head of communicable diseases for the army. And right at the peak of the pandemic, he said, "If the current rate of acceleration continues for a few more weeks, civilization could easily disappear from the face of the earth." So that's how bad it got at the very peak in the worst circumstances. Obviously with COVID-19, we're not facing anything like that, thank God.

Unger: Yeah, thankfully. It's interesting that even in 1918, you wrote that the modelers concluded that layering interventions like social distancing, which you were talking about in this book almost 20 years ago were required to flatten the curve. That's not a term they use, but basically to relieve the pressure on the health care system. You started talking about MPIs, were there other learnings about interventions that worked or didn't work that we've been able to learn from and apply?

Barry: Well, a lot of our ideas, and number one, they come from common sense, would probably come up with them whether 1918 had happened or not. And of course, quarantine and so forth, that goes back way, way, way before 1918. But the social distancing, closing bars, those places, churches even, things like that, they did seem to work in 1918. Modelers looked at it, Richard Hatchett and Carter Mecher. Richard now runs CP and CPI in London. Carter Mecher's at the VA now. They were both in the National Security Council, and they did it. Marty Setrone at CDC, with a lot of data from Howard Markel at the University of Michigan did an even more comprehensive study. And to be perfectly candid, although I always supported MPIs, I thought that the inclusion the modelers came up with, well again, I supported the conclusions.

But I was much less enthusiastic about the impact that MPIs would have than most of them were. And clearly, I was mistaken on that. They've been remarkable in how effective they've been when people comply with them. Probably, I became somewhat skeptical based on army data. There were 120 army camps, 99 of them imposed quarantine and things like our other MPIs, 21 didn't do anything.

There was no statistical difference between the two of them, the camps that did, and those that did not. Not just wasn't statistically significant, there was no difference. But the person who did that study was George Stopper, a very good, great even, pioneer epidemiologist who later did the first epidemiological studies of cancer, and was the first head of the American Cancer Society. He not only looked at...he not only did the quantifying analysis, quantification analysis, but he did a qualitative analysis, as well.

And he discovered that in the very few camps that rigidly adhered to the various MPIs, they did benefit, but there were so few of them that it didn't register statistically in the larger universe of 120 camps and probably about 2 million people. So my thinking was, if you couldn't successfully sustain compliance over a period of weeks, and in an army camp in the middle of the war, you weren't going to be very successful in a civilian community at peace time. We have been much more successful or some countries have been incredibly successful, far more than I ever would have imagined, as I said earlier. Other places, we haven't done so well, but a lot of that is bad leadership, frankly.

Unger: Well, thank you so much, Mr. Barry, for being here today. It's fascinating to talk to you and reflect on the book and how much that I learned from that. Really appreciate you being here. That's it for today's COVID-19 update and we'll be back with another segment tomorrow. In the meantime, for resources on COVID-19, visit ama-assn.org/covid-19. Thanks for joining us. Please take care.

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