Pete Hesser's children, *la gourme,* and new information concerning the 1918 flu

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"The horses growing better, a cough and sore throat seized mankind". This was the news from Dublin toward the end of 1727 reported in Charles Creighton's monumental *History of Epidemics in Britain - Volume II - From the Extinction of the Plague to the present time,* Creighton's "present time" being 1894.

Matters had been much the same in 1688 as in 1727. A "short time before the general fever, a slight disease, but very universal, seized the horses too: in them it showed itself by a great defluxion of rheum from their noses". Creighton's source "was assured by a judicious man, an officer in the army of Ireland … there were not ten horses in the regiment that had not the disease". In Dublin "not one [man] in fifteen escaped".

Then in Huxham in 1732 some months prior to an influenza outbreak, the horses were affected by "the strangles". This seriously unpleasant term seems to be the most common synonym for horse flu, though in World War I, *la gourme,* a term used by the French military's veterinarian staff also crept into English. (You can look it up but Google will first ask if you didn't mean "*la gourmet*".) *Gourme,* horse flu, or the strangles, this was a nasty disease, a *gourme* that the veterinarians described as considerably worse than *"la gourme habituelle*". By April 1918, civilian horses in France had also been affected. The April timing is intriguing because the first reported cases of the "Spanish Flu" among humans had been in March. But that had been among soldiers at a cavalry training site in Kansas.

At a professional meeting in 1924, a Dr. Reece, about whom I know nothing more, spoke of the "remarkably large number of observations" of coincidences between influenza epidemics and "epizootics of the same character", starting in horses, then "dogs, cats and the like". It seemed that aside from the disease itself and coincidences in timing, however, the only common element among dogs, horses and humans was crowding. Sir William Hammer added that "epizootics may precede or follow epidemics", and they may accompany it too. Horse flu and human flu had both been troublesome in 1657-1659, 1727, 1737, 1743, 1760-1762, 1775, 1788 and 1889-1890 but a recurring question for modern epidemiologists is whether the diseases, equine or human, had been correctly identified. In general, I suspect they have and I am happy to follow the cautious Creighton whose learned opinions and judgements are clearly the work of a first-rate medical mind. At times Crieghton is barely committal as when he discusses London in the spring of 1658 and perhaps in the following year "if Whitmore has made no mistake in his dates" in reporting the sudden and

> universal coughs and catarrhs,
> as if a blast from the stars.

Although the sequence of events in not entirely clear, this explosion of an influenza-like illness was apparently followed by "a great death of coach-horses almost in every place"… "and it has [now] come into our fields".

From daily experience, Creighton and all his sources had been familiar with cities in which large numbers of horses had been kept. In times past, people would have had detailed knowledge about horses in cities, knowledge that has been universally forgotten in the generations brought up in the age of the automobile. But as recently as the 1920s, annual statistics for horses were cited for places such as Omaha, Nashville, Atlanta and Chicago, in some instances tabulated along with cause of death, somewhat as they are for humans. The problems of horses in cities had even given rise to a tongue-in-cheek warning that by such and such future date, the streets of London would be filled to the second story by horse manure.

Horses and mules - which are less susceptible to equine flu - had also been ubiquitous in all the world's armies. Pigeons too had been a common accompaniment of the military in World War I and in the Paris Flea Market in the 1980s I stopped at a stall with a stock of World War I pigeon-carriers. Pointing out details of their design and construction, the dealer assured me they were from the American army, not French, "the perfect thing for an American in Paris".

In addition there were historic flu epidemics that were not associated with reports of a corresponding disease in horses or mules, and episodes of equine flu with no flu-like illnesses among humans. But the association of the two appears to have been well known. Yet while insisting on this matter, I have avoided mentioning a key observation: people do not catch the flu from horses. Creighton's compilation of 1894 seems to contain nothing to indicate the contrary nor, really, does A.J. Williams' "Analogies between Influenza of Horses and Influenza of Man" in the *Proceedings of the Royal Society of Medicine* for February 24, 1924. Williams had perhaps not been sufficiently unambiguous in his original oral presentation, however, so a Professor Hobday rose to insist that there were no reports of transmission from horses to men or *vice versa.* This is how things then stood and pretty much as they now stand. About the best we can do is quote a statement made in 1919 by George A. Soper, a major in the United States Sanitary Corps whose conclusions were cited and emphasized by Alfred Crosby in his *America's forgotten Pandemic* (first published in 1976 under the title *Epidemic and Peace: 1918*). In a contribution to *The New York Medical Journal,* Major Soper noted that despite many epidemiological and clinical similarities, horse flu and human flu were not identical, nor were they transmissible from the one species to the other, but "it would seem probable that a more thorough knowledge of the disease in horses would yield facts of great value". As concerned horses, Soper mentioned transmission via the respiratory tract, sudden onset, fever, cough, indications of muscle and joint pain, and explosive spread over whole continents on occasion, features that characterize human influenza as well. Soper also included an element I have not seen elsewhere, that horse flu was thought to be transmissible via feces and stable dust.

Dogs can catch influenza directly from horses, and humans can get it directly from pigs and *vice versa*. Humans can also get the flu from diverse avian species. Direct species-to-species transmission is relatively rare, however. Usually a mixing-bowl species or individual is needed, with a reassortment of viral genes taking place in a creature - human or not - which has become co-host for viruses originating in different species.

The "Spanish Flu" wasn't Spanish at all. But at the time the flu raged through Europe, many countries were at war. Spain was neutral and did not impose press censorship so journalists there could print the demoralizing truth, namely, that the country was going through a horrific epidemic. Elsewhere people whispered, muttered or screamed that it was an Allied plot or that the Hun had once again let loose a poisonous gas. And what censor would pass an article saying that the enemy had been so terribly successful? In truth, the whisperings, mutterings and screams, whether from the Allies or the Axis, seemed to have had some logic to them for those most targeted by the 1918 flu were in the prime of their lives, military age, essentially those between the ages of 20 and 40.

Influenza is also an endemic seasonal disease that is always with us. In the U.S., some 35,000 flu deaths occur in non-epidemic years these days, primarily among children, older people, and others who lack fully functioning immune systems. In 1918, by contrast, those in the 15-34 year age bracket who came down with influenza or pneumonia (the most common and deadly complication of the flu) were about twenty times more likely to die than had been the case in 1917, a non-epidemic year. And, of course, many, many more were catching the flu in 1918. Estimates vary. Twenty million died worldwide. Or else it was fifty million. Or 100 million. Or some other large figure. In India, with its youthful population, mortality reached an estimated 50 per thousand. As in the U.S., the disease in India followed the railroads and when military mounts shipped from Australia to Calcutta arrived with a nasty strain of equine flu, it too then spread across India with the rail transport of cavalry.

The flu of 1918 was exceedingly contagious and is estimated to have affected over one fourth of the U.S. population and one fifth of the population worldwide. These estimates are not necessarily at all accurate. It is true that doctors everywhere were badly overworked and that quite a few medical personnel were dead before they could fill out and sign death certificates or other forms. But the real problem in trying to deduce the numbers, however approximately, is simply that influenza was not a universally reportable disease in those days. Until 1918, it had not been considered sufficiently serious. But in 1918, the flu virus became "unlike any strain ever seen".

It was not just deadly. It was quick. According to a young woman working at the military laundry at Camp Funston, Kansas: "We'd be working with someone one day, and they'd go home because they didn't feel good, and by the next day they were gone" (Barry, 2004). Within some months following the outset of the epidemic, gauze face-masks were being widely used and vaccines and various treatments were available. None of them actually worked, but they did much to calm nerves. In the medical profession itself, or perhaps it was only in the health administration units of cities, states, hospitals, and army camps, many eventually came to terms with the fact that doctors could not do much. What was really needed was nursing care and calls went out to anyone with the least bit of training in nursing. Many of the best were overseas with the troops, but older women and student nurses came forth. Young and old, many of these courageous volunteers caught the flu and among the older women, some surely died. We might also think to say that groups of student nurses - young women in the prime of life - had been decimated, but such a statement would betray the original sense of the word "decimate" for in places

considerably more than one in ten student-nurse volunteers would die. Another group particularly susceptible to the 1918 flu were pregnant women; one study showed a hard-to-believe 71% death rate among pregnant women who had been hospitalized with the flu.

It was an awful disease which, due to the war and the accompanying censorship and the jolly peace that followed, was half forgotten until Crosby's book in 1976. It was also a peculiar disease. Although it eventually struck a broad portion of the population, it had seemingly started among the young and most fit and from beginning to end it would be most efficient at killing individuals whose immune systems were the best *H. sapiens* can produce. There are no usable statistical measures but here and there doctors in hospitals, nurses in wards, sailors on ships, and soldiers in barracks reported that it was the most robust, strongest, most fit, disease-free athletic sorts who suffered the worst. *Post mortem* studies on such victims of the 1918 epidemic often showed enormous damage to the lungs, deadly damage that it was not then possible to explain. The explanation came years later: such victims had literally drowned in the waste products of their own powerful immune reactions to the virus. As put by Crosby, "a springtide of fluids overwhelms the lungs". Thus in 1918 - in contrast to other flu epidemics - many robust young people died of the influenza itself rather than from secondary infections of pneumonia-causing bacteria, the standard cause of death among those with weaker immune systems. They died so rapidly that pneumonia-causing bacteria had had no time to establish secondary infections.

Despite the name "Spanish Flu", the endless accusations aimed at the Hun, and unfounded quasi-scholarly speculations that the epidemic had started in China, the first clinically demonstrated cases were at Camp Funston, Kansas, a site established to train troops in World War I. (Funston still exists, incorporated into Fort Riley.)

A recurrent complaint at Funston in those days arose from a twofold unpleasantness. First, in common with much of the American Midwest, the camp was subject to severe dust storms. The experience at Funston was doubly unpleasant, however, due to the great concentrations of horses and mules on the base and the manure they produced. I have not seen any figures for the actual number of animals but they were sufficiently numerous to produce nine tons of manure a week.

On March 9, 1918, despite an impending dust storm, standard practice at Camp Funston was followed and the manure was burnt. The result was a stinging yellow haze with the sun going "dead black". Or so it is said. There had been similar days at Funston in the past and presumably elsewhere, but this event (if it actually occurred as related) was remembered as particularly severe and the clean-up that night supposedly involved a hundred men and many hours of raking and sweeping.

According to some websites, it was two days later, "shortly before breakfast March 11th", that Company cook Albert Gitchell reported to the infirmary with a "bad cold". The March 11th date is most probably incorrect, however, and is perhaps a story-teller's concoction intended to tie in with the account of the manure-burning. Better sources give March 4th as the date of Gitchell's illness. In any case, Corporal Lee W. Drake was right behind Gitchell with similar symptoms and by noon the Camp Surgeon had a 107 flu patients on his hands. Within three weeks the number of sick

and dead at Funston was above a thousand. In the next two months, over 500 prisoners at San Quentin penitentiary also came down with the illness, followed by comparable outbreaks at Camps Hancock, Lewis, Sherman and Fremont. It is said that there were few incidences of the flu within the general population during these months and although self-censorship at the newspapers has to be taken into account, later investigators have concluded that there had been nothing special to report. Civilians had been healthy that summer.

In September the disease reached Boston, first affecting sailors and shipyard workers, then soldiers, and then moving into the general population and overseas. Some of the most awful sounding accounts are those of outbreaks on the badly overcrowded troop transports on their way across the Atlantic. An often-cited sailor's diary reads "October 5 – fifteen more bodies have just been buried from the *President Grant*". During the last two months of the war, over four thousand American servicemen died at sea or after being put ashore for hospitalization at Halifax. There were also those who died the first few days after arriving in Europe. Troopships disembarking large numbers of sick and dying men in French ports hindered the Allied effort, but such things were only written about after the war.

Some people have associated the flu outbreak at Camp Funston with episodes of manure burning, whatever the exact dates. But in his *The Great Influenza: the epic story of the deadliest plague in history* (2004), John M. Barry suggests that the ultimate origin of the 1918 pandemic is to be found elsewhere. Barry had gone back to original sources, always the best procedure if time is available, and in doing so, he identified a Dr. Loring Miner who in 1918 had had a decades-old medical practice in the sparsely populated Haskell County, Kansas, some three hundred miles west of Camp Funston. Doing his rounds from town to town in "late January and early February 1918", Miner encountered a new ailment that he diagnosed as influenza, signaling the U.S. Public Health Service to warn of "influenza of severe type".

Newspapers across the U.S. in 1918, whether large or small, had been reluctant to publish items that might hurt morale. Nevertheless, on February 14, 1918, the *Santa Fe Monitor* in Haskell County (as cited by Barry in 2004) reported: "Mrs. Eva Van Alstine is sick with pneumonia. Her little son Roy is now able to get up... Ralph Lindeman is still quite sick... Goldie Wolgehagen is working at the Beeman store during her sister Eva's sickness... Homer Moody has been reported quite sick... Mertin, the young son of Ernest Elliot, is sick with pneumonia... Pete Hesser's children are recovering nicely... Ralph McConnell has been quite sick this week." A week later, the same paper reported "Most everybody over the country is having lagrippe or pneumonia".

In 1918 the population of Haskell County was 1,720, with its fair proportion composed of fit military-age men, all of whom trained at Camp Funston along with some fifty thousand others before being shipped overseas or being buried as flu victims. The issue of the *Santa Fe Monitor* with the news that "most everybody over the country is having lagrippe or pneumonia" also reported that "Dean Nilson surprised his friends by arriving at home from Camp Funston on a five days furlough" and that Ernest Elliot left "to visit his brother at Funston just as his child fell ill" (Barry, 2004). The February 28th issue recorded the departure of John Bottom for Funston, and it is clear that there must have been many other comings and goings between

the camp and parts of rural Kansas, of suppliers of fodder, for example. It is also clear that there must have been comings and goings of horses.

The incubation period for influenza is very rapid, just 1 - 3 days, which partially explains how and why the flu is able to spread so rapidly. Another reason is that individuals about to come down with the flu may spread the virus before they themselves develop symptoms. (These same properties characterize most epidemic diseases.) Influenza subsides once it runs out of susceptible people to infect but after an estimated 10 - 30 passages through humans, the virus may change and adapt to a different sub-population.

The flu virus in the fall of 1918 had not been identical to that of the spring disease and toward the very end of 1918 additional mutations gave rise to a third wave. This was less deadly than the second but still a truly terrible disease to which individuals who had survived infection during one of the earlier waves had only partial immunity. The flu was still present through 1919, and in 1920 the reported death rate by influenza among young adults was still well above normal. "Just Married" couples were mourned throughout the epidemic and the parents of young children could die together within days. During the second wave, feverish newly-orphaned children were found dazedly wandering city streets.

With a difference of one week in dating from the web sources mentioned above, Crosby and Barry gave March 4th as the day the first soldier at Camp Funston reported ill with the symptoms of influenza. This fits Barry's claim that the epidemic may have originated in Haskell County in January or early February 1918. If so, as Barry writes, it might have readily died out for lack of susceptible human hosts in sparsely populated Haskell where the epidemic was so short lived that school had reopened with healthy children by mid-March. But, Barry writes, the war then brought the flu to Funston.

There is nothing obviously wrong with Barry's belief that the epidemic started in Haskell County but it is of limited value because it leaves so many important questions unanswered. Why Haskell County? Why Funston? Why the virulence of the disease? Why was it especially severe among individuals who were fit? Do we get the real poop when we read of the disposal of the horse manure? Valid answers would also have to be consistent with the very recent news that the human virus of 1918 had had an avian source.

The *Santa Fe Monitor* had mentioned two-way traffic between Haskell County and Camp Funston but as best I can tell there was nothing very special about Haskell County, in fact, nothing special at all, except for the presence of the flu-like disease somewhat before March 1918. But Camp Funston *was* special. First of all, there was the crowding. Then there were the horses and mules and their weekly output of nine tons of manure. And then there was the select presence of fit young men and women *to the near exclusion of anyone else.* (There were enough young women at Funston for weekend dances but, I would guess, few babies, children, or older people other than some senior officers and their wives.) So if a weak or "standard strength" flu virus had been brought to Funston, perhaps by birds feeding on undigested seeds in the manure, it would have found itself in proximity to an unreceptive human population whose members all possessed well-functioning immune systems. A soldier who caught this flu prior to March 1918 would have presumably been subject

to a few coughs and perhaps some aches in his joints. He might well have avoided a visit to the camp hospital for in the wartime society of the United States during this period, the very worst thing to be, or to be called, was a "slacker". (Following a disappointment in love, my great-uncle Jake, my grandfather's brother, left home as a very young man and joined the U.S. Customs Service in the Philippines. There, around the turn of the century, he caught some tropical disease and had to be repatriated. He was later exempted from military duty. When the draft was again activated in the 1940s, Uncle Jake was at pains to explain to anyone who would listen that he had not been "a slacker" during The Great War.)

Weak or standard-strength versions of avian or avian-equine or equine-avian influenza may have passed from soldier to soldier at Funston without necessarily exhibiting consequences severe enough to be preserved in military records or memories. And those who carried the virus might have included men belonging to the prime-of-life cohort such as Dean Nilson who had made a round trip between Funston and Haskell County while on furlough. In Haskell they would have encountered people whose immune systems were far weaker, some whose identities are known to us, "Pete Hesser's children", "Mrs. Eva Van Alstine's little son Roy", and "Mertin, the young son of Ernest Elliot" who had been "sick with pneumonia".

In these children and anyone who may have picked up the virus from them, the particular viral strain would have undergone changes that could have then been brought back to Camp Funston. While this is hypothetical and will perhaps forever remain so, we know that at the time the children of Haskell County were recovering and heading back to school, a highly contagious version of the flu began to run its course through the soldiers in training at Camp Funston. At this stage the illness gave few signs of becoming the horrific thing Barry calls "the deadliest plague in history". According to Crosby, the flu at this stage usually meant "two or three days of misery" and in any case the epidemic at Funston waned rapidly, "bobbing up only now and then as new lots of draftees arrived". It was much the same story throughout March and April 1918 as the disease spread through Camps Oglethorpe, Gordon, Grant, Lewis, Sherman, Doniphan, Fremont, Hancock, Kearney, Logan, McClellan, Sevier and Shelby, an unpleasant countrywide epidemic in the military which at this stage had still not spread into the civilian population. Some unfortunate soldiers at Funston and other camps had indeed died but the cause of their deaths generally appears to have been pneumonia and other secondary complications. For although it was highly contagious, the Funston-style version of the 1918 flu - "the first wave" - was not itself a great killer. The second wave, which was the dreadful pandemic itself, would not come until the autumn when it would break out from the military camps on the East Coast.

This sequence of events depended on *mutations* of the 1918 flu virus. These produced a pattern of happenings of a sort that could not and cannot be predicted in advance. Simultaneously another pattern was being traced out within particular populations, military camps, towns and cities (and perhaps ships though, if so, perhaps incompletely). This depended on the number of local person-to-person *passages* of the virus and the pattern produced by the sequential passages was always the same: those struck in the first ten days were far more likely to become severely ill than those affected after the virus had undergone a number of person-to-person passages. By October 1918 mutational changes to the virus had rendered it especially deadly. It then weakened everywhere with passages from person to

person and by late November, conditions were briefly very much better. But additional mutations then set off a third wave in December. In common with the second, it was worldwide. Crosby, who touched on such matters at many points in his study, concluded that a patient's outcome depended primarily on the virus rather than the care he received.

It looks as though the human version of the "1918" epidemic had started at Camp Funston at the end of 1917 or toward the beginning of 1918 with birds, horses, manure and the burning of manure all perhaps implicated. It was not then able to cause human illness sufficiently severe to be recorded or remembered. Yet it had an enormous potential for harm because it was by then already adapted to people with first class immune systems, passing among them and perhaps producing the occasional cough or touch of fever, or perhaps not even that. Its ecological niche was strictly among the healthy. It knew no other type of host environment. In February and perhaps January 1918, carriers of this flu variant had mixed with the general population in Haskell County where, as is typical, the virus passed from person to person, undergoing unknown changes. When it returned to Funston in late February or very early March, it spread, causing considerable short-term miseries, but not as an especially deadly disease. The virus at this stage was active, contagious, and disease-causing but it was confined to an inhabitual ecological niche characterized by host-individuals with strong immune systems, army nurses, men in military training camps, troops moving about on trains, dockers, miners, and men in prisons. And as luck and the Kaiser would have it, these individuals were frequently forced into crowded circumstances, tightly packed among others with similar immune profiles and then shipped from one camp to another and then overseas.

As a consequence of its passage through the general population in Haskell County, some of the early strains of the 1918 influenza virus were capable of surviving and multiplying outside their preferred niche and eventually more or less thriving in other environments. Still, it would always remain a disease of the robust, one Swiss doctor recording that he had never seen a severe case in anyone over 50.

The virus might be compared to a Wall Street employee who might readily thrive if transferred to a high-stress environment in Washington or London but might not be able to adapt to a low-stress job as a beachboy. In general, it is only the individual creature itself - human, animal or microbial - who knows whether a new environment is a good one. That's why zoo-keepers have so much trouble keeping certain animals. It is much the same with medical researchers who can do little better than trial and error in selecting a suitable medium on which to try to culture unfamiliar bacteria.

Some potentially useful lessons emerged while researching this material. One involved the immense danger of crowding and insufficient ventilation. Men in wind-blown trenches had far less flu - or perhaps it was less flu with pneumonia - than those living in crowded barracks behind the lines. The risk of the flu was far higher in the spic-and-span military camps on the East Coast of North America than it Europe's muddy trenches and this was not something it took a statistician to detect. French soldiers who left the crowded barracks for the front reduced their chances of getting the flu by a factor as high as twelve (Crosby, 2003, p.153). The dangers of crowding were apparently double, a greatly increased risk of catching the flu and, it

seems, a similarly greatly increased risk of dying from it. Sailors on ships came down with the flu just as frequently as the tightly packed troops they were transporting. But their onboard living conditions and daily routines - which presumably excluded playing cards for long hours below decks - provided them with a systematically lower mortality rate, lower by a factor of three or four or five.

Another potentially useful lesson relates to what has inexactly been called "relapses". Deaths among recovered patients were not necessarily because of the flu itself, nor due to its secondary complications. The risk for newly recovered patients was that their immune systems were so exhausted that exposure to most any infectious agent at all in the four to six weeks following recovery from the flu could be fatal. The cause of death could be a tertiary factor, the sniffles, for example.

There are other lessons too, though it is hard to see how they might be broadly applied in today's world. It is clear, for example, that the flu could be avoided by total isolation. Eskimos were so severely affected by the epidemic that there had been talk of the possible disappearance of the entire people. Yet at least one Eskimo family living by itself with no contact with others had had no incidences of disease.

One symptom of the pandemic of 1918-1920 may not have been given sufficient attention. In diverse countries around the world, flu patients complained of diarrhea. (See Crosby, 2003, Ch.9.) Diarrhea, however, is not "supposed" to accompany human flu. Yet when stool tests for the usual bacterial culprits and for amoebas were carried out on U.S. military personnel, the results were systematically negative. The regular incidence of diarrhea without a known cause, this "loose end" so to speak, constitutes a decidedly inconvenient category of information. But as many scientists have discovered, ignoring data that does not fit current theory is a short and sure road to failure. Whether or not there is a useful lesson here, this seems a good place to state that in birds the flu virus normally infects the intestinal tract.

Other issues raised by the events in 1918 and 1919 are of interest for different reasons. To start with, we have Crosby's thought-provoking observation that during an epidemic, "democracy can be a very dangerous form of government" (Crosby, 2003, p.236). Martial law may provide an acceptable escape exit from democracy during such times and it might be a good idea for those in power today to publicly reexamine the conditions under which martial law can be imposed and how to supply convincing guarantees that it will be relaxed when the time is right. It is odd then that Barry's account of American's war hysteria, the censorship, the search for spies, and the pursuit of "slackers", gives the impression that we did not in any case have a full-fledged democracy in the United States at the time of the epidemic. Barry, who is harsh in his judgement of Woodrow Wilson, essentially claims that from Wilson down, the non-democratic choice imposed on Americans was to favor the war effort to the exclusion of even admitting that the flu was a problem. Barry gives the example of the actions of Dr. Wilmer Krusen, "a political appointee" who directed Philadelphia's Department of Public Health and Charities. A Liberty Loan parade had been scheduled for September 28, 1918 but on the 27th, local hospitals had admitted two hundred new flu patients. Krusen, who was fully aware of the risk of contagion by crowding could have cancelled the parade but did not. Before the end of the month - i.e. two days later - the city was in deep trouble and in short order the health question

of the day was not whether or not to allow parades, but what to do about the shortage of coffins and the refusal of grave diggers to come to work.

Another highly troubling issue was the impaired judgement and mental stability of many after recovering from severe cases of influenza - a matter that may not be specifically limited to the flu of 1918-1920. "Toxic involvement of the nervous system" was one term and " slowed cerebration" was another. Individuals who were touched appear have included Wilson who, according to one aide, had "manifested peculiarities" after coming down with typical symptoms of influenza plus severe diarrhea in early April 1919.

This is a good-news bad-news article. The bad news has two sources. One is familiar, the fact that the world has become more crowded and interconnected since 1918. The other bad news comes from Creighton's survey of historic flu outbreaks, some of them very unpleasant indeed, though none with consequences comparable to "the Great Influenza". The good news is that the conditions in 1918 were a product of the times and are unlikely ever to be repeated. A strain of influenza will probably never again be offered such a highly specific and frightfully dangerous niche within the human population. Barring bad luck, the 1918 epidemic will permanently retain its title as "The Deadliest Plague in History".

Paris, 26 October 2005 (Revised 17 Jan. 2006)