Dietary Roulette:
Human Poisoning from Ruffed Grouse (*Bonasa umbellus*)

Human poisoning after eating plants and animals has been recorded from antiquity through the modern era. Toxic plants and molds have been responsible for a broad range of medical-related conditions, among them ergotism, favism, and lathyrism. Toxic animals and animal products include poisonous honey produced by bees pollinating rhododendron, ciguatera fish poisoning, and puffer fish-related intoxications associated with consumption of the Japanese dietary specialty *fugu*.

It is less well recognized that four avian taxa are poisonous. Best described of the four is the European migratory quail (*Coturnix coturnix coturnix*), a sub-species sometimes poisonous to humans at different seasons of the year at widely separated geographical areas of southern Europe, northern Africa, the eastern Mediterranean, and portions of the former Soviet Union. First documentation of toxic migratory quail is Biblical (*Numbers*: 11: 31-34), with subsequent commentary by Greek(Aristotle; Didymus; and Galen), Roman (Lucretius; Pliny; and Sextus Empiricus), Jewish (Moses ben Maimon and Philo) and Muslim authors (al-Demiri; al- Qazwiny; and Ibn Sina). Toxic quail also have been a research interest of scientists in the modern era as well especially from the 1950s through early 1980s. Sergeant, the prominent French 20th century microbiologist, suggested that common hemlock (*Conium maculatum*) was the mechanism for quail toxicity, but Sergeant's hemlock thesis was disproved nearly 20 years ago by research conducted by my graduate student, Bruce Kennedy. How quail become toxic at different seasons at discontinuous geographical regions of the Old World remains poorly understood.

The second group of toxic birds is endemic to New Guinea. Included are at least three representatives of the genus *Pitohui*: hooded pitohuis (*P. dichrous*), variable pitohuis (*P. kirhocephalus*), and rusty pitohuis (*P. ferrugineus*). Researchers from the University of Chicago, the Smithsonian Institute, and National Institutes of Health, Bethesda, discovered that both feathers and muscle tissues of *Pitohui* spp. contained a homobatrachotoxin that acted as a defensive mechanism.

A third toxic avian species is the bronze wing pigeon (*Phapes chalcoptera*), a southern hemisphere passerine, first identified poisonous by 19th century immigrants to Western Australia. Reports of bronze wing pigeon toxicity suggested that domesticated house cats died after eating entrails or bones of this species, and observers reported that heart leaf poison bush (*Gastrolobium bilobum*), a component of bronze wing pigeon diet, was the responsible agent. Bronze wing pigeon meat, however, reportedly, remained safe for human consumption.

The fourth toxic bird and subject of the present essay is ruffed grouse (*Bonasa umbellus*).

The geographical distribution of ruffed grouse extends throughout North America and regional subspecies are identified. Ruffed grouse, also called partridge or pheasant in local New England vernacular, have been widely hunted throughout New England and the American Midwest from pioneer times into the modern era. There is a substantial body of medical and ornithological literature from the 18th and 19th century, however, that documents human poisonings after eating

ruffed grouse poisoning. These ornithological and medical case histories have defined a range of dramatic, variable toxic symptoms and an unusual seasonal association with late winter. Curiously, no accounts or case histories of human poisoning from ruffed grouse have been reported in the United States since 1886. Even passing mention of grouse-related intoxications apparently has disappeared from standard medical and toxicological textbooks of recent decades. Indeed, the last general comment on such cases known to me was published more than 50 years ago.

The question logically may be posed: why did cases disappear and grouse-related intoxications become an essentially forgotten medical problem? This disappearance is all the more curious, given that episodes were widely recognized throughout the northeastern United States, especially in urban centers such as Philadelphia and Boston, where cases were frequent causes of food poisoning from animal sources.

The purpose of our essay is to examine the ruffed grouse dietary-toxicological mystery. We will identify the earliest medical accounts present and comment upon selected case histories; review and evaluate the several hypotheses advanced during the 18th and 19th centuries to explain these intoxications; and will present a logical explanation why cases of ruffed grouse poisoning ceased.

Given different susceptibilities of animal species to various toxins, it is plausible that a lower animal may feed on a particular plant with no injury, then serve as a vector for a poisonous element in that plant to the human who in turn feeds upon it. While primitive human ancestors may have identified flesh-foods that could be dangerous this way, such lore faded from memory after domestication of human terrestrial food resources. In historical times, the plant-animal vector problem sometimes has appeared when humans adopted or selected unfamiliar foods, or occupied unfamiliar environments.

Escargot, for example, took hold as an article of French diet in the 19th century. Perusal of the French literature revealed that snails gathered carelessly from fields sometimes caused serious illnesses in consumers, and the sickness, in turn, was attributed to various toxic plants upon which the mollusks fed. Modern producers in France, aware of this problem, fatten their snails on standard “clean” rations before offering them for the table. As a result toxic escargot have remained a distant memory.

Milk sickness a prominent toxicological problem in frontier America presents a second example. Human illness after consuming milk was a common malady of the frontier era, whose most notable victim was Nancy Hanks, mother of Abraham Lincoln. Pioneers accurately observed that the plague of milk sickness tended to disappear with the “civilization” of former rural districts, an indirect recognition that cattle no longer free-ranged in the half-tamed woods where animals could graze on the poisonous white snakeroot, but were confined on improved pastures or stall-fed clean hay.

POISONING FROM RUFFED GROUSE: A HISTORICAL INQUIRY

Eating ruffed grouse during the late 18th and mid-19th century was a risky proposition, and might almost be categorized as "dietary roulette." Culinary delight/pleasure usually was the "winning" outcome but on other occasions consumers experienced a range of unexpected, miserable consequences, even death.

Within 30 minutes to four hours after dining on ruffed grouse, some but not all consumers grew dizzy, nauseated, vomited, and experienced visual disturbances to the point of blindness. Patients told attending physicians that their limbs became cold and numb, while violent pains racked their necks, backs, and stomach. As the intoxication deepened, pulse and respiratory rates slowed, eyes glazed and pupils dilated. Some patients experienced severe muscle weakness, graduating to paralysis and physical collapse. One frequent symptom was a terrible sense of dread, a feeling of impending doom or approaching death. Males and females were affected in relatively equal numbers; most cases were concentrated in late winter. February was the most common month for the intoxications.

Victims of ruffed grouse poisoning were treated using emetics (usually mustard or ipecac), purgatives, or were plied with brandy, laudanum, smelling salts, or wine. Patients commonly were blistered or bled in medical attempts to stimulate the body and to restore humoral balance. In most instances symptoms diminished within 24 hours, although some patients complained of generalized weakness for several days and spoke of residual tingling or soreness in the extremities. Full recovery was achieved in some patients within 24 hours, while others took several weeks. In four cases the patients died.

James Mease, a prominent Philadelphia physician, presented the first documented case when he described a dinner held in Philadelphia during February, 1791. He published his account seven years later in 1798:

> *Several persons ... were seized in about three hours after dining upon pheasants [i.e. ruffed grouse], with giddiness, violent flushing of heat and cold in the face and head, sickness at stomach, and repeated vomiting. These symptoms were soon succeeded by delirium, weak pulse, and the greatest debility, which threatened approaching death. Some cases were marked by the preservation of the senses, but a total inability to articulate. By plentiful draughts of warm water, or chamomile tea, the vomiting was promoted and the offensive matters discharged. Gentle stimulants and cordials, as wine, whey, volatile salts, and liquid laudanum, were afterwards exhibited, and most commonly with success. In some cases, however, the event was not so fortunate; from the co-operation of other causes, the attack was more violent, and either death, or a protracted recovery, ensued.*

Mease, in his retrospective account, concluded that the intoxications were caused by a presumed component of ruffed grouse diet:

> *[I observed] that in the winter, and early in the spring, these birds live chiefly upon buds of trees, and particularly upon those of the laurel which is one of the few shrubs that preserves its verdure throughout the winter, and from many melancholy proofs, is known to be highly poisonous. It is not improbable, therefore, that the noxious quality of the laurel may have been communicated sufficiently strong to the pheasants [i.e. ruffed grouse] to produce the effects described.*

The laurel Dr. Mease identified was mountain laurel (*Kalmia latifolia*), an evergreen shrub of the forest edge and understory, common along the eastern seaboard and mountain areas from Georgia northward to Quebec. Farmers of the region had long known that mountain laurel and the related species, sheep laurel (*Kalmia angustifolia*), were dangerous stock-poisoning plants. Modern 20th and 21st century toxicologists have determined that both species contain andromedotoxin (grayanotoxin I: acetylandromedol), one of the plant kingdom's more potent poisons

While Mease argued that the poisonings ensued after the birds had fed on mountain laurel during winter, he remarked that he regularly dined on ruffed grouse during winter but had not suffered any ill effects. Given his familiarity with poison cases, and his own dining habits, Mease advanced four reasons why the poisonings were not more widespread geographically:

1. ***Dilution:*** ruffed grouse were meal components so any toxin contained in the meat would be "diluted" by other foods consumed;

2. ***Toxin location:*** not all portions of a ruffed grouse carcass were eaten and the portions consumed usually were distant from the bird's digestive tract;

3. ***Alcohol as an antidote*:** wine or other stimulating liquors commonly taken during dinner could counteract and weaken the effects of any potential toxin;

4. ***Toxin strength:*** toxic properties of mountain laurel leaves would vary seasonally and would be weakest during late winter when sap and plant fluids were concentrated in the plant's vegetative root system.

Mease's theory on the relationship of *Kalmia* to ruffed grouse poisoning, however, was not original. First credit for this theory may be attributed to Benjamin Barton who noted in 1794 that the association between the two was founded in popular belief and folklore. The concept that ruffed grouse were sometimes rendered toxic by feeding on laurel buds during winter, however, took hold widely after Mease's publication and was not seriously challenged by subsequent writers.

Reports of toxic, dietary misadventures involving ruffed grouse continued into the 19th century. These reports confirmed the symptoms outlined by Mease, and added complementary details to the picture. Case histories with symptoms and patient outcomes were reported beyond Boston

and appeared in New York City and Philadelphia, where the malady was well known to physicians:

> **Case 1**: vertigo, dim vision, nausea-vomiting, languor-exhaustion; cool, moist skin; low pulse; excruciating pain and heat in chest, violent headache, convulsions; and
>
> **Case 2**: retching, vomiting, blindness, pain on top of head extending to neck and back, extremities cold, low pulse.

The eminent Dr. Jacob Bigelow, Professor at Harvard University, published his influential book, *Nature in Disease* (1854) that contained particulars of ten cases gleaned from the files of various Boston physicians. Bigelow noted inconsistency in symptoms among the cases but summarized the common signs seen in the patient's respective circulatory, digestive, neurological, and respiratory systems:

> **Circulatory**: feeble, irregular heartbeat; weak, slow, almost imperceptible pulse; sense of intense cold; skin pale, alternatively, livid;
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> **Digestive**: nausea, vomiting, accompanied by abdominal pain;
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> **Neurological**: vertigo, blindness, tinnitus, loss of the sense of touch, and lower limb paralysis;
>
> **Respiratory**: breathing slow.

Bigelow conceded a widespread medical and public suspicion that mountain laurel in ruffed grouse diet caused the poisonings. He remained unconvinced, however, and presented a strong counter-argument:

> *I have observed in experiments made purposely, that the leaves of the Kalmia are not particularly poisonous, when taken into the human stomach in any quantity which the bird would be likely to devour; and the [bird's] crops, when examined in winter, are found to contain leaves and fragments of most of the wild evergreen plants which are in verdure at that time. I have found among other things portions of leaves of Pyrola, Gaultheria, Smilax, Coptis, Mitchella – also buds of Azalea, alder and apple tree, which latter appears to be a favorite food with the partridge.*

Bigelow rejected the mountain laurel hypothesis and advanced three alternative suggestions to explain why humans were poisoned after eating ruffed grouse:

> **Disease**: ruffed grouse were diseased at the time they were shot and passed their disease and symptoms to humans;

> **Cellular change**: poisonings were caused by an undefined, slow "chemical change" that occurred when recently killed grouse were kept for long periods in cold weather; and
>
> **Idiosyncratic**: specific consumers were either tolerant or intolerant of ruffed grouse meat.

Of the three hypotheses advanced Bigelow was attracted most to the idiosyncratic explanation and argued:

> *The same person has sometimes been affected twice, that a majority of persons, partaking of the same partridge, escape unharmed, when others are poisoned, and that [other] individuals are [known] who cannot eat lobster, mackerel and certain other kinds of food without suffering symptoms approaching in character to those [poisoning symptoms] already described.*

Despite Bigelow's prominence as a Harvard Professor, his idiosyncratic theory was not accepted within the medical community. In fact, his supposition was widely rejected and the laurel hypotheses continued to be supported by less scholarly, less well-trained physicians.

By the mid 19th century human poisoning after eating ruffed grouse eventually was recognized in neighboring parts of Canada and elsewhere. In 1866 a surgeon stationed with the Royal Army Garrison, Halifax, described the discomfiture of one of his colleagues after breakfasting on grilled partridge. In 1882, a professor at the Halifax Medical College published his personal experience and account of the effects of partridge poisoning. Across the Atlantic in Europe several cases were documented in Great Britain, where consignments of American and Canadian ruffed grouse were imported when local British game became scarce. A first-hand memoir of ruffed grouse poisoning, penned by the British author Henry Stephens, was especially vivid.

In this instance the patient (adult male) had dined on imported ruffed grouse and experienced initial uneasiness, followed by the sense that a "severe weight" had been applied to the back of the head, shoulders, arms, chest, and abdomen, accompanied by a "glowing heat" in the same regions. Shortly thereafter, he suddenly became blind [!] and a sense of intense cold seized his hands and feet. Subsequent symptoms included nausea, vomiting (while blind!), a sense of choking and continued chills, whereupon the patient's sight suddenly returned. Although treatment included administration of brandy and being plied with warm blankets, the patient experienced residual soreness in the chest and abdominal muscles with associated tingling of the hands and arms. Eventually, the patient recovered.

Burt's account includes other intriguing data as well. His patient had two house cats. The housekeeper had given one of the cats a piece of ruffed grouse breast, and then disposed of the bird's skin, crop, and bones on a nearby dunghill, where the second cat fed upon the discarded components. The first cat that ate a portion of ruffed grouse breast – the same part consumed by the patient who was poisoned – exhibited no symptoms. The second cat that fed on the bird's skin, crop, and bones exhibited a curious, staggered gate, vomited, but subsequently recovered.

Dr. Burt apparently knew of Bigelow's three hypotheses published several years earlier, but rejected each. Burt wrote that it was unlikely that the bird was diseased, since it did not differ in plumage or plumpness from others purchased at the same time. He considered it unlikely that putrefaction was the culprit, since other hunters consumed the remaining ruffed grouse for sale without experiencing disagreeable symptoms. He also argued against Bigelow's idiosyncratic hypothesis, since the patient regularly ate ruffed grouse but never before had experienced toxic effects.

Burt supported the mountain laurel dietary hypothesis initially proposed by Barton and Mease, with a subtle difference, a two-part theory, one that ultimately would be known as the "diffusion hypothesis." He buttressed his dietary-diffusion argument by citing Alexander Wilson's 1808 text that stated human poisonings from ruffed grouse were due to hunters who had not cleared the bird's crops and such oversights allowed potentially toxic products to "diffuse" into the meat:

> *During the deep snows of winter, [ruffed grouse] have recourse to the buds of alder, and the tender buds of the laurel. I have frequently found their crops distended with a large handful of these latter alone; and it has been confidently asserted, that, after having fed for some time on the laurel buds, their flesh becomes highly dangerous to eat of, partaking of the poisonous qualities of the plant ... Though I have myself ate freely of the flesh of the Pheasant [ruffed grouse], after emptying it of large quantities of laurel buds, without experiencing any bad consequences.*

Six years after publication of Burt's "diffusion" proposition a physician known only as Dr. Parks published an account of ruffed grouse poisoning where a young women, had dined with several guests. After dinner she alone exhibited a sequence of toxic symptoms that began with chills and tinnitus, followed by temporary loss of hearing. Subsequently, the patient became blind and experienced severe epigastric oppression. After vomiting, she lost consciousness but was revived and treated by Dr. Parks. Although her serious symptoms abated, her pupils remained dilated; she exhibited weak pulse, cold skin, and tremors, but ultimately recovered.

The importance of Parks's paper lies in three key observations.

1. Three persons had on the same bird but only one exhibited symptoms;
2. The only portions consumed during the meal were the grouse's legs and backbone, rib attachments with presumed breast-meat;
3. During the preparation process, the cook had tasted and eaten a portion of the grouse meat, pronounced it good, and suffered no ill consequences.

The same year as Parks's account, the Editor of the *Medical Times and Gazette* [London] reprinted a note by Dr. F. Taylor that had first appeared in early September in the *Times of London.* Dr. Taylor had been called to treat a woman thought to be dying; her body was cold and she appeared insensible, without measurable pulse. He plied her with brandy to stimulate return of warmth and circulation, whereupon she recovered. The following day she complained of "acute thrilling," described as tingling sensations throughout her facial muscles. The woman

related to Dr. Taylor that she had dined the previous evening upon ruffed grouse imported from Canada.

Dr. Taylor wrote that at the time he attended the woman he noted a young cat in the house that exhibited hind leg paralysis and was unable to move. The patient related that she had fed her cat "some bits" of the ruffed grouse (quantity and portion not defined) and as both doctor and patient looked on, the animal vomited, but remained in misery. Taylor wrote that the cat recovered after several days.

In a follow-up editorial to the Taylor account, the Editor of the *Medical Times and Gazette* [London] accepted the general thesis that "certain berries" rendered ruffed grouse toxic in Canada, but that specifics remained unknown. He then discussed how toxic plant elements might retain their properties after being digested by animals and incorporated into muscle tissue. He restated the mountain laurel hypothesis and drew upon two complementary lines of reasoning, whereby meat of hares sometimes became toxic to humans after browsing on *Rhododendron chrysanthemum,* and that Frenchmen sometimes became poisoned after eating snails fattened on *Coriaria myrtifolia*.

Other accounts appeared in England during the middle decades of the 19th century. Three females, two women and the daughter of one (age unknown), dined one evening on a meal that contained ruffed grouse. Both the adults were poisoned; the daughter was not affected. Patients stated that the intoxications began with a sense of extreme cold, followed by exhaustion, depression, and feeling of imminent death. Upon arrival of a physician, the pulse of the adult women was noted as feeble and both women complained of intense cold that started with the extremities and ascended. The mental capacity of both women was not impaired during the intoxication, but the eyesight of one faltered and she became temporarily blind. As the poisoning progressed, both women experienced a sense of choking and ultimately collapsed due to lower limb paralysis.

Each of the three females had eaten portions of the same ruffed grouse. One of the adult women had consumed a wing, part of the breast, and a leg; the second woman had eaten only breast meat. The daughter, who did not exhibit symptoms, had eaten a portion of the breast meat from her mother's plate, but disliked the flavor and refused to eat the remainder. Newbigging concluded that contents in the bird's crop posed a danger to consumers, and restated the "diffusion" hypothesis:

> *[Especially if] allowed to remain for some time in contact with the flesh of the bird, and that thus the subtle poison, whatever that is, pervades the flesh of the whole animal, and becomes capable of affecting, in a most serious and dangerous manner, persons partaking of it as food.*

The physician James Somers, President of the Nova Scotia Institute of Science, provided a personalized account of the effects of ruffed grouse poisoning in 1886. This account – the last published case – is remarkable for two reasons. First, Somers experimented upon himself and his family, and placed all at potential risk. Second, his account contained more detail regarding

timing and onset of symptoms than previously published cases. In his preface he wrote that the flesh of ruffed grouse was often poisonous during winter and that the symptoms resembled an "arterial sedative." In order to conduct his experiment he shot four ruffed grouse, then identified their crop contents. One of the four birds killed had eaten leaves of the following genera/species: *Aspidium* sp. (fern), *Betula* sp. (birch), and *Kalmia angustifolia* (sheep laurel). Somers cooked this bird and served it to his family. He wrote that only he ate the dark meat, strongly flavored portions, and gave only white meat portions to his children ages 2, 5, and 10 respectively. Somers kept a log and reported that his children experienced no ill effects from eating the meat. He, however, was poisoned and his symptoms were extensive and noted in detail in his log. Onset of his intoxication began approximately one hour after consumption and he noted the following in his diary:

> *My first feeling was one of fullness in the brain, a well marked sense of numbness around the mouth and lips ... occasioned nausea ... was seized with dizziness and inability to maintain [an] erect position ... dizziness subsided but the nausea increased until it terminated in copious and prolonged emesis ... accompanying the vomiting there was pain in [my] back at the root of the neck and between the shoulders ... [of] a dull aching character ... [that] passed down both arms but changed from that of pain to one of numbness and tingling ... and terminating in the fingers; there existed also a feeling of coldness well marked in both extremities ... [my] skin was colorless, [my] face had a ghastly look, perspiration [was] not increased but rather diminished, urine [was] not increased in quantity ... no action of the bowels ... pupils normal and intellect not disturbed ... the action of [my] heart [was] closed, as evidenced by the fall of [my] pulse ... took small doses of brandy and my pulse then began to rise in volume .. I got up and made an attempt to go to the supper table, but was immediately seized with giddiness, and fell on the floor in a state of insensibility, which lasted for a few second only ... [I took some tea then felt] ... languor, some trembling of the muscles, and chillness of [my] whole body ... [I retired and slept well] ... it took three or four days for [my] system to recover its usual tone.*

Somers ended his account with the statement that he regularly consumed ruffed grouse but never before had experience any "evil results." He discussed his "self-poisoning experiment" in a matter-of-fact manner and concluded that individual susceptibility, or the idiosyncratic theory, could not explain the cause. Somers rejected putrefaction as an explanation since he had hunted the birds, himself, and cooked them while they were fresh. He contended that ruffed grouse absorbed toxins from the plants contained in their crop and concluded that a "man in health, with good digestion, [could] dispose of flesh in a state of putrefaction with perfect ease," and concluded that the offending toxin had to be from *Kalmia angustifolia* or sheep laurel.

There are many passing references to ruffed grouse poisoning that do not provide details. Most important, therefore, are the 20 thorough clinical descriptions of poisoning by ruffed grouse that span a 95-year period between 1791-1886. These accounts documented ruffed grouse toxicity at several widely separated places, Boston, New York City, Philadelphia, Halifax in Canada, and at

several localities in England. The quantity and quality of detail in these accounts permits identification of a fairly consistent complex of symptoms.

Onset began with nausea, pallor, a remarkable slowing, light and feeble pulse, coupled with vertigo in all cases. Accompanying these symptoms were obscured vision, sometimes led to temporary blindness, and sensation of intense cold in nearly all. Great weakness, sometimes exhibited by abject prostration, was another frequent feature, while loss of consciousness was also recorded frequently. In other cases, however, the victim's state was described as "trance-like," alternatively "somewhat delirious," but usually the victim remained alert enough throughout the intoxication that a detailed recollection of the ordeal could be supplied to the attending physician.

Pain or numbness in the head, neck, and back were common signs, as were epigastric discomfort to a greater or lesser severity. Feelings of numbness, sometimes heaviness in the extremities was typical, a pattern that sometimes graduated to virtual paralysis of the limbs and collapse in a minority of cases. Respiratory distress, variously described as shortness of breath or as a sensation of choking, was a frequent symptom occasionally accompanied by partial or complete loss of speech. Partial deafness and ringing in the ears (tinnitus) was reported in some cases, as were "twitching," "thrilling," or tremors in a minority of cases, and rarely "fearful" convulsions and "frothing."

How much of the variation expressed by these symptoms can be ascribed to dose effect, and how much to individual sensitivity cannot be determined from an early 21st century vantage point. Further, it is not possible to determine from this distance in time whether or not alcoholic beverages or medicinal drugs taken by the victims prior to or during dinner where ruffed grouse was served could have had any coactive roles. What emerges, however, is that the onset of illness was relatively sudden, usually within an hour or two after ingestion of ruffed grouse, and that most of the acute symptoms subsided within four or five hours. For most of those affected the subsequent symptoms were short-lived, and for the most part were not serious, although in at least four cases the result was death.

It is significant that all documented cases of human poisoning from ruffed grouse documented occurred in late winter, when heavy snows blanketed the eastern United States and adjacent portions of Canada. February was an especially dangerous month. Authors of the old accounts assumed correctly that deep snow forced the ruffed grouse to abandon their ground habitat and to forage on fresh-looking buds and leaves in trees and tall shrubs. Under such conditions, the evergreen laurels would be especially inviting. It is also significant that all published cases of ruffed grouse poisoning occurred in urban settings where the birds had been obtained, second-hand, from professional fowlers. We cannot determine from this distance in time, however, whether this urban pattern was genuine or merely reflected under-reporting from rural districts.

Physicians of the era deemed the inferred connection between ruffed grouse ingestion of *Kalmia* and cases of poisoning to be sound and sure. In more than one 19th century *materia medica*, authors cited cases of ruffed grouse/partridge poisoning to prove the pharmacological effects of *Kalmia* leaf.

As the 19th century progressed, the problem of human poisoning from ruffed grouse merited passing reference in most medical botany, medical jurisprudence, and ornithological texts. While these secondary sources are numerous, they offered few additional facts. Still, several contributed observations worthy of consideration. Most commentators were satisfied with the *Kalmia* theory initially proposed by Barton and by Mease, that the toxic juice/resin of laurel was digested by ruffed grouse, assimilated, thereby rendering the flesh toxic. Indeed, the Editor of the *Medical Times and Gazette* wrote:

> *It has long been known that the poisonous principles of certain plants retain their properties after having passed through the digestive laboratory and become incorporated in the tissues or secretions.*

Other authors, however, argued for the "diffusion" whereby meat was contaminated by slow seepage of the poison from the dead bird's viscera, given that a week or two might pass before a shipment of grouse reached market in a distant city and additional days could pass before the birds were purchased for the table. It was the fowlers' custom of this age to leave the birds un-plucked and un-gutted in order to preserve their plump appearance and thereby attain higher prices. In fact, a prominent early 19th century ornithologist had ventured the suggestion that bagged grouse became poisonous only if the birds had been kept un-drawn for long periods of time.

Wilson was followed in his opinion by James Audubon, who claimed to have feasted on freshly shot grouse many times over campfires out in the wilds, even after extracting large plugs of laurel from the bird's craws, and yet:

> *[I] never felt the least inconvenience from eating them, nor ever perceived any difference of taste in their flesh.*

Other physicians also questioned whether of not *Kalmia* was the culprit. Their protests, however, failed to take hold, even though the author of one popular 19th century formulary maintained that the poison evolved from the "putrid and decomposed condition" of the birds, and that the cause of ruffed grouse poisonings was really a question of rotten meat

Most physicians, including those who reported cases of the sickness, recognized ruffed grouse poisoning as distinctive from maladies usually identified and associated with meat spoilage. Further, the possibility of inadvertent or deliberate contamination of the meat with some "unnatural compound" was also considered. Following a rash of poisonings from imported grouse in a London suburb in 1862, a disgusted Londoner speculated in the *London Times* that the birds had been taken by unprincipled hunters who used poison or that the birds had been defiled by toxic preservatives by crooked shippers, since "knavery was an ingrained Yankee trait."

But if such suspicions had been widely accepted or generalized, it would be reasonable to assume that public outcry would have been far greater in North America. In fact, there is no evidence for widespread public concern over ruffed grouse poisoning or that this issue played any role in

contemporary agitation for better food safety standards, or was influential in state or municipal legislation to that end. The evidence suggests that despite the occasional toxic tragedy, Americans of the era regarded ruffed grouse consumption as no more than an agreeable form of "culinary roulette," and accepted the risk as small or incidental, given the usual gustatory reward, much the way that 20th and 21st century Japanese gastronomes have relished their puffer-fish.

Another theory that fell on deaf ears was proposed by the Editor of the *Boston Medical and Surgical Journal* who suggested that an unknown "spontaneous change" in the flesh of the grouse caused by onset of the mating season was responsible for the toxic meat. He theorized that fluids originating in the "spermatic apparatus" of the male grouse permeated its meat, making the flesh "intolerable to the human stomach." This novel hypothesis, however, had no subsequent supporters.

Jacob Bigelow, despite his influence as a scholar and physician, wrote emphatically that the laurel hypothesis was in error, and doubted that grouse could store enough laurel toxin in the flesh to adversely affect a human. His proposition that a specific "idiosyncrasy of individuals renders some persons intolerant of this species of food," also was not accepted during his time, although such a view from the 20th and early 21st centuries would receive strong attention and collectively be associated with a food allergy. His proposal, however, was discounted – in part – because many people stricken by toxic grouse relished the birds on other occasions, and consumed the meat with impunity while suffering no ill effects. In view of such contrary experiences and opinions, other researchers argued that most consumers might be unbothered by small amounts of toxin sequestered in the meat of a given grouse, while a certain few – the occasional "hyper-reactor" – might be extremely susceptible.

Despite the ongoing controversies of the 19th century no one attempted to test or rigorously question the problem of grouse toxicity through controlled experimentation. The botanist Victor Chestnut made a gesture in this direction towards the end of the century, and claimed to have produced "nearly fatal results" in a cat using meat from a chicken that had been fed for some time [not specified] on an extract of *Kalmia*, although the quantity/dose was not specified. The only other scientific attempt known to us stemmed from the 20th century experiment performed by Gardiner Bump of the New York State Conservation Department.

Bump, ever the scholar found Alexander Wilson's 1812 reference to poisonous grouse and decided to put the matter to a test. He obtained eight ruffed grouse: four were provided a normal ration, heavily supplemented with fresh laurel leaves; the remaining four were fed a diet only of laurel leaves. Six of the birds, three in each group, were killed; portions of the meat were fed to experimental animals (mice and kittens) with no indication of toxicity. Bump then dined on the remaining two grouse (one from each lot) and experienced no ill effects. His only adverse comment during this "personal trial," was that the taste of the grouse flesh was distinctive. He concluded there was no indication that "laurel was poisonous to grouse, or through them to humans."

Wilson's 19th century theory that the poison did not collect in the flesh of the birds but diffused postmortem from their entrails when left un-gutted for long periods was not tested. Further,

blood and tissues in the Bump grouse experiments were not assayed for andromedotoxin or its metabolites. Therefore, the question whether or not the *Kalmia* toxins circulate or accumulate in grouse at levels sufficient to evoke a reaction in some consumers remained unanswered.

The *Kalima*-associated dietary theory, originally proposed by Barton and Mease just before the turn of the 19th century, has attracted most supporters through the years. Examination of ruffed grouse diet, however, shows that its foods are highly variable throughout the species' geographical range, and that diet differs widely by season. Data from New York State, for example, document more than 400 different plants and 580 different animals present in ruffed grouse diet, with the greatest contribution coming from plants (98.9%) and the highly diversified animal component (1.1%) accounting for a minority of foods consumed. Laurel, however, remains a relatively minor dietary component, present in only 30 of more than 1,000 birds examined, and only 1.5% of the total plant material consumed.

Ruffed grouse eat several plants known to cause toxic symptoms in humans, and some of these species exhibit distinctive seasonality while others remain available throughout the year. When examining a dietary rationale to explain the toxicity of ruffed grouse, it is logical to focus attention onto late fall/winter plant candidates since no cases of ruffed grouse poisoning have been documented during spring, summer, or early fall.

Late 18th century through mid-19th century ornithologists and physicians suggested that mountain laurel (*Kalmia latifolia*) was an especially inviting evergreen winter food, known to be tolerated by ruffed grouse but toxic to humans. Numerous 20th century authors, as well, also supported a mountain laurel explanation. Despite the large body of *Kalmia*-related literature that suggests this genus to be the toxic mechanism in cases of ruffed grouse poisoning, at least 22 potential toxic plant candidates meet the condition of winter availability: *Arctium* spp. (burdock), *Arisaema triphyllum* (jack-in-the-pulpit), *Celastrus scandens* (bittersweet), *Equisetum* spp. (horsetail rush), *Hypericum* spp. (St. John's wort), *Kalmia angustifolia* (sheep laurel), *Kalmia latifolia* (mountain laurel), *Nepeta hederacea* (ground ivy), *Phytolacca americana* (pokeweed), *Polygala paucifolia* (fringed milkwort), *Polygonum hydropiper* (water pepper), *Polygonum* spp. (smartweed), *Prunus pennsylvanica* (bird cherry), *Prunus serotina* (black cherry), *Prunus virginiana* (choke cherry), *Ranunculus abortivus* (small-flowered buttercup), *Ranunculus acris* (tall buttercup), *Rhus toxicodendron* (poison ivy), *Rumex acetosella* (sheep sorrel), *Sambucus canadensis* (elderberry), *Solanum dulcamara* (bittersweet nightshade), and *Symplocarpus foetidus* (skunk cabbage).

Six of these 22 species meet the second requirement that the toxic component produce a suite of symptoms in humans similar to those expressed by patients poisoned after eating ruffed grouse:

Celastrus scandens (bittersweet)
Distribution: Quebec to Manitoba, from Kansas throughout the central portion of the United States to New Mexico; throughout eastern North America through the Carolinas.
Symptoms: neuro-gastric: vomiting, diarrhea, mental changes, and loss of consciousness.

Equisetum spp. (horsetail)
Distribution: Newfoundland to Virginia; throughout the United States and southern Canada.
Symptoms: neuro-muscular: weakness in legs, ataxia, trembling, muscular exhaustion, prostration, weak pulse, and cold extremities.

Kalmia angustifolia (sheep laurel)
Distribution: eastern Canada to Newfoundland; from Hudson Bay south to Georgia and Michigan; eastern United States.
Symptoms: neuro-gastric, cardio-vascular, and respiratory: nausea, vomiting, vertigo, blindness, tinnitus, loss of speaking ability; lower limb paralysis; feeble, imperceptible pulse, sense of intense cold.

Kalmia latifolia (mountain laurel)
Distribution: Canada to Maine, through the Allegheny Mountains, West Florida, Ohio, Kentucky, and Tennessee; eastern North America, primarily Appalachia.
Symptoms: neuro-gastric, cardio-vascular, and respiratory: nausea, vomiting, vertigo, blindness, tinnitus, and loss of speaking ability; lower limb paralysis; feeble, imperceptible pulse, sense of intense cold.

Rumex acetosella (dock; sheep sorrel)
Distribution: throughout the United States and southern Canada.
Symptoms: neuro-gastro: upper respiratory complications, ataxia, and prostration.

Solanum dulcamara (bittersweet or climbing nightshade)
Distribution: New Brunswick to New Jersey; Pennsylvania west to Minnesota and Kansas; eastern and north-central United States.
Symptoms: neuro-gastro: pupils dilated, apathy, drowsiness, trembling, exhaustion-prostration, stupefaction, progressive weakness, and paralysis.

Of these six species, only the two laurel species have been investigated in any detail as the toxic mechanism for ruffed grouse poisonings in humans. Considering the two laurels, sheep laurel appears as a ruffed grouse dietary component only during winter in the American northeast, whereas mountain laurel appears in ruffed grouse diet throughout the calendar year and especially throughout Appalachia where the poisonings have never been recorded. Research conducted by the New York State Conservation Department ultimately determined that mountain laurel was not poisonous to ruffed grouse; that a diet of only *Kalmia* leaves caused malnutrition in these birds; and that *Kalmia*-dosed birds were not toxic to humans. Before either *Kalmia* species can be established as the toxic plant that causes ruffed grouse poisoning, additional experiments must be conducted to characterize the toxins in the other non-laurel species of winter plants eaten by ruffed grouse. Further, it must be established whether or not ruffed grouse consume, metabolize, and store enough toxin from *Kalmia* – or any other plant source – to elicit symptoms in humans.

But if diet was not the causal mechanism, could it have been bacillary contamination and associated putrefaction, a hypothesis initially proposed by Burt in 1856? Putrefaction or decomposition of organic matter, however, was a process viewed quite differently by researchers and physicians before the 20th century. Since remote antiquity it had been observed that organic matter decomposed and produced foul-smelling products and that maggots sometimes grew in spoiled meat, a process thought due to "spontaneous generation." These views changed during the late 19th century after Louis Pasteur published his classic research on anthrax, which established a bacillary origin for this disease, explained its transmission, and gave rise to the concept of germ theory. As a result of Pasteur's research, germ theory began to replace humoral balance as the accepted medical dogma of the era.

Accounts of ruffed grouse poisonings, therefore, were published during a time when germ theory was unknown and principles associated with the concept of humoral balance dictated treatment and understanding of diseases. The essence of humoral balance or allopathic medicine was the system of opposites: all diseases and all available foods were classified as hot or cold, wet or dry. The dietary system of opposites held that hot diseases were treated using a diet based upon cold or neutral foods; cold diseases were cured by hot or neutral foods; wet illnesses by dry or neutral foods; and dry diseases by wet or neutral dietary items.

The foul smells and odors of putrefied organic matter were not enough to discourage consumers from eating putrid meats, since smell and stench are culturally relative. Before the 20th century most people throughout the world consumed meats and prepared dishes in various stages of spoilage or putrescence. It was the global search and quest for spices during the 15th-17th century era of exploration that reflected the human endeavor to mask off-odors so frequently encountered in poorly preserved foods.

Organisms associated with putrescence produce classical food poisoning and express a range of symptoms. The major bacillary organisms associated with food poisoning include: *Bacillus cereus*, *Campylobacter* spp., *Clostridium botulinum*, *Clostridium perfringens*, *Salmonella* spp., *Shigella sonnei*, and *Staphylococcus aureus*. Human intoxications from five of these (*Bacillus cereus*, *Campylobacter* spp., *Salmonella* spp., *Shigella sonnei*, and *Staphylococcus aureus*) produce mild to serious symptoms usually expressed as nausea, vomiting, abdominal cramps, and diarrhea but occasionally accompanied by dizziness, headache, and fever. As a result, these organisms may be eliminated as potential contributors to ruffed grouse poisoning since the symptoms do not meet the second requirement that they also be characteristic of such intoxications.

This is not the case, however, with *Clostridium botulinum*. Botulism, a serious, life-threatening illness, expresses as symptoms that include neurological, respiratory, and muscular effects that parallel some of those seen in ruffed grouse poisoning. The symptoms of botulism are variable but include: blurred vision, dilated pupils, difficulty in talking and swallowing, severe abdominal pain, diarrhea, nausea, vomiting, confusion, dizziness-vertigo, ataxia, respiratory impairment, and paralysis. The principle difference, however, is that botulism commonly is lethal in 5-44% of cases. In contrast, deaths from ruffed grouse poisoning – although reported – have been rare.

So what does all this mean?

As early as 1847 toxicologists were attracted to the physiological proposition whether or not a plant poison could be transmitted to human beings through consumption of a bird that fed harmlessly on plants that presumably contained toxins. From a late 20th and early 21st century vantage point, circumstantial evidence based upon comparative symptoms seems to favor the original dietary *Kalmia*-related hypothesis proposed by Barton and Meese. Ruffed grouse are widely distributed throughout much of North America and the only reports of toxicity have been from the geographical region where the bird's range overlaps with that of one *Kalmia* species (sheep laurel) and is restricted to a narrower geographical range where hard winters with deep snowfall are the rule. It would appear that ruffed grouse are tolerant of the toxic principle of *Kalmia*, andromedotoxin, and that the symptoms described in accounts of human poisoning from grouse appear consistent with those suffering exposure to this compound. The minimal effective dose of andromedotoxin in mammals is low, only 2 micrograms per kilogram body weight and the notion that the poison might be vectored to a sensitive consumer in the meat of a rather small bird is plausible. The metabolic fate of andromedotoxin in ruffed grouse after eating the leaves of laurel, remains to be elucidated. It is not necessary here to postulate a complex process whereby andromedotoxin is bound or stored in grouse tissue, only that a significant level be present in the bloodstream at the moment the birds are shot by hunters, since wild game birds are not thoroughly exsanguinated [i.e. bled out].

At the same time, the merits of competing theories cannot be ignored. Colleagues in the Department of Food Science and Technology at the University of California, Davis, drew our attention to parallels between some human symptoms produced by toxic grouse and intoxications caused by bacterial toxins. Botulism, however, could be ruled out given its high mortality rate among unfortunate consumers. The possibility that an obscure fungal or other bacterial toxin could be at play, however, should not be dismissed. The handling, shipping, and storage practices for 19th century wild game would have been abominable from the perspective of modern food sanitation standards. Still, if a microbe was the root cause, then logic holds that occurrences of grouse poisonings would have increased and peaked during warm months of spring, summer, and fall, and not the dead of winter. Further, why should only certain ruffed grouse be the vectors? What would have made the flesh of ruffed grouse a unique substrate for a given organism? Similar toxic problems were not identified with domestic poultry or with other upland game birds also brought to market in vast numbers during the 19th century and presumably stored and transported under similar less-than-savory conditions? In fact, evidence from the self-experiment conducted by Bump suggests the possibility that a toxic plant other than *Kalmia* was the cause.

The decline and disappearance of ruffed grouse poisoning toward the end of the 19th century coincided with two important changes in the American scene. Perhaps the interplay between the two was responsible? First, there was a philosophical change and shift in attitudes of the general public and hunters towards conservation and protection of animals. With this rise was an associated promulgation of hunting bans, especially in winter when grouse were most vulnerable. The Massachusetts enactment of 1886 was the first in this regard, followed by statutes in other states that imposed bag limits and other protective laws. Winter was the season when ruffed grouse were toxic, and the potential impact of hunting restriction on incidence of human

poisoning by ruffed grouse would have been serendipitous. By the turn of the century commercial hunting of ruffed grouse and other wild game birds had been banned in most eastern jurisdictions: Pennsylvania in 1897, Massachusetts in 1900, and New York in 1902. There is no evidence that grouse poisoning led to the passage of these laws. But after passage, the phenomenon swiftly faded as a cognizable medical and public health concern. Indeed, writing from the perspective of 1927, a famed American ornithologist briefly alluded to the problem, as if to a legend from olden times:

> *Such poisoning ... seems to have been caused long ago and only by birds taken in winter. It is illegal now to kill them at that season.*

An esteemed Harvard Professor, one of the subsequent editors of Osler's *Principles and Practice*, saw fit to mention poisonings caused by ruffed grouse as late as 1944, but his was the exception and other medical writers long since ceased to note the problem.

The second change that occurred throughout America resulted from scientific advances in the fledgling field of microbiology that followed the research of Louis Pasteur. As a result, laws were enacted that focused on food handling and marketing of food changed dramatically during the late 19th and early 20th centuries. The unseen world of microorganisms changed medical thinking as germ theory displaced the paradigm associated with humoral balance as the key to health and transmission of disease. This change in medical explanation for illness and disease transmission gave rise to what was known as the "sanitarian movement," an approach to understanding microbiology that challenged both consumers and governmental officials alike: if Pasteur was correct, then humans were surrounded by "microbes" in the air, ground, and in/on the food supply! As a result, consumers and scientists alike both pressured legislators at the local, state, and national levels to improve food-related sanitation and handling, especially how foods were presented for sale.

While academicians, physicians, and other scientists were attracted to germ theory, the general public did not take easily to this explanation for illness and disease transmission. Doubts were raised in the public sector regarding food safety: foods should be covered since even fresh items sold at markets were potentially dangerous. Such public fear over food safety continued into the 1880s when specific pathogenic organisms were discovered that caused cholera, diphtheria, pneumonia, tetanus, tuberculosis, and typhoid. As a result scientific training changed as well. By 1890 microscopes had become standard university equipment as more scientists taught their students the new germ theory basis for disease transmission.

Conservation measures directed at preserving ruffed grouse had two results. The first was expected, and grouse ultimately thrived. The second result was serendipitous. Because of conservation measures taken, ruffed grouse essentially disappeared from American and Canadian dining tables during winter especially throughout northeastern North America, when the birds occasionally were toxic. Complementing the conservation measures, other laws regulated food preparation, handling, marketing, and sale of food and these, too, had the effect of improving American public health standards, reduced food spoilage and putrefaction, and increased the quality of foods that otherwise might be sold to unsuspecting consumers.

In conclusion, the anomalous sickness called partridge or ruffed grouse poisoning was a notorious form of food poisoning in portions of the eastern United States and Canada in the 19th century. Such poisonings stopped almost spontaneously before its etiology could be properly analyzed. The circumstances of its departure are perhaps the most vexing part of the mystery. It is easy to conclude that the poisonings ended with passage of bans on winter hunting, which restricted harvesting of grouse at the time of year the birds could feed on *Kalmia*. The answer, however, might not be so simple and the question must be posed here: is partridge-grouse poisoning entirely a problem of the past?

In preparing this essay 18th, 19th, and 20th century medical and toxicological accounts were reviewed. Internet connections also were utilized and Poison Control Centers throughout the United States were contacted to obtain information whether or not the Centers had records of human poisoning after consuming mountain laurel or sheep laurel, or records of suspected or diagnosed cases of human poisoning after eating ruffed grouse. All replies were negative.

We also identified bird hunting/outfitting web-sites and solicited public assistance from ruffed grouse hunters to answer two basic questions: have you or your grouse hunting friends in the United States or Canada ever experienced illness after consuming ruffed grouse? If so, what were the symptoms? Our request for information remained on e-mail "bulletin boards" for more than six months. Responses were uniformly negative – except for one astonishing reply:

> *I saw your query on the Internet and am compelled to respond with an experience that I had many years ago. I consumed a bird that I believed was a Cornish game hen and the following morning was overwhelmed by extreme vertigo and tinnitus. I was unable to move my head or stand. This lasted for several days, diminishing in severity each day. As soon as I was able I went to the University of Minnesota where I was a student and was examined by the Ear, Nose and Throat service where the diagnosis was not clearly determined. A virus attacking the eighth cranial nerve was postulated as a possible cause but not confirmed. The dizziness gradually went away, however I was left with some tinnitus in my right ear and a slight loss of high frequency hearing in that ear. I had always had some suspicion that consuming the liver of the bird was a causal effect of my illness but had no substantiation and had never heard of such a thing happening. I had never eaten the livers before and could not get it out of my mind that there was a relationship between the incidents.*
>
> *The reason for my answer to your message is that I am an avid grouse hunter and there is the remote possibility that the bird may have been a grouse. The incident occurred in the fall when grouse would have been in my household and available. It is also possible that the two birds may have been prepared and served together. It is unlikely that I ate the liver of a grouse because it is not my practice to save the livers.*
>
> *This happened in approximately 26 years ago [1971] and I have always associated my illness with the livers of that bird without proof. Another factor is*

that I was a Minnesota resident at the time and the bird would not have had access to laurel or buds associated with the southeast. I was born in Western North Carolina and hunted the birds there for many years. It was not a practice to eat the livers and I have never heard of anyone becoming ill from eating the birds or the livers from them. I still believe the bird that I ate was a game hen but the circumstances are too closely related to ignore. I graduated from the University of [omitted here] School of Pharmacy in 1974 and I am aware that toxins may become concentrated and detoxified in the liver of animals. I have recovered from the incident with sufficient balance and hearing to make my living as an [omitted here], however, I no longer ride the " Tilt-a- Whirl" at the Minnesota State Fair because of an increased need for visual orientation from this illness. I thought you might be interested in this (Received October 18th, 1997).

This account, vivid and rich in detail, clearly documented a case of avian-related food poisoning in 1971, during the Fall season, in the state of Minnesota where laurel species and ruffed grouse are *not* contiguous. While the respondent could not recall with certainty whether or not the bird in question was a ruffed grouse or Cornish game hen, the symptoms expressed were similar to those tabulated by 19th century physicians and naturalists from ruffed grouse poisoning.

This report raises the tantalizing possibility: has human poisoning from ruffed grouse vanished as a medical problem, or are cases disguised and expressed under different names due to variable symptoms and differential diagnoses?

Henry Christian, the eminent Harvard Professor, professed to have seen sporadic cases of ruffed grouse poisoning in the vicinity of Boston in the early 20th century. As Dr. Christian pointed out, it would be reasonable to expect that cases would appear from time to time among pot-hunting farmers, back-road poachers, and other out-of-season consumers. More than often – with manifestation of light symptoms – the passing illness would be shrugged off as too much bourbon, perhaps a touch of the influenza, and would go unrecognized, perhaps misdiagnosed, even if a patient required physician assistance.

If, too, just one or several birds in many score were potentially toxic, and if human poisoning depended upon an uncommon sensitivity in the individual consumer, logic would militate against the illness appearing in American farms, villages, or cities. Instead, odds would favor the encounter of such a rare toxic bird and rare idiosyncratic consumer only if large numbers of grouse were available at numerous markets. Only then would grouse/partridge poisoning appear frequently enough to be recognized.

In the course of this investigation of human poisoning by ruffed grouse, numerous conflicting accounts and statements in the scientific-medical literature were encountered: leaves and buds of *Kalmia* are poisonous/not toxic to animals; leaves of *Kalmia* are poisonous/not toxic to humans; ruffed grouse eat *Kalmia* and are not poisoned; since ruffed grouse eat *Kalmia* and some humans eat grouse and become poisoned and express *Kalmia*-like symptoms, the toxic agent must be *Kalmia*. Alternatively, *Kalmia* has no relationship to ruffed grouse toxicity. Other factors further

confound an explanation including differences in consumer's age, body size, gender, portion of the grouse carcass consumed, and quantity of meat/dose level ingested.

A careful inspection of the literature also reveals intriguing questions that need further consideration: why were ruffed grouse toxic only during winter, and are they potentially so today? What is the explanation for the several cases where two persons dined on different portions of the same ruffed grouse and experienced different outcomes: one poisoned – the other not? What explanations may be advanced for cases where two persons ate from the same portion of the same grouse: one being poisoned and the other not? Why would persons poisoned at the same meal exhibit different symptoms? All the more interesting is the broad array of symptoms suffered by individuals poisoned after eating ruffed grouse: the vertigo, blindness, tinnitus, loss of sense of touch, and lower limb paralysis; the slow respiration; the feeble, irregular heart beat; the sense of intense cold, coupled with nausea and vomiting. Such symptoms, while typical of *Kalmia* poisoning, also are exhibited after ingestion of other plant species, and the plant symptoms differs substantially from typical bacillary cases of food poisoning.

Finally, to what extent might ecological change and environmental modification also have been a factor in the geographical distribution of the poisonings? How has the abundance of ruffed grouse in specific localities changed during the past 150 years? How has the local abundance of laurel been affected by land clearing, pasture improvement, and weed eradication activities? In addition to passing hunting laws and the corresponding rise in acceptance of germ theory and better ways to handle food, how might the obscure interplay of landscape and habitat alteration have conspired to break the chain of contingencies that once resulted in this human illness? A historical precedent may be suggestive here. While the ancient Greeks and Romans, grappled for centuries with the question of toxic quail and "dietary roulette," this malady still visited today in contemporary Greece remains unknown in modern Italy.

Human poisoning from ruffed grouse, therefore, can be compared to a Gordian knot of tangled questions for scientists to address before this unresolved problem in food safety is solved. And perhaps the most interesting, intriguing riddle stems not from the 18th-19th century accounts but from an electronic mail message received in 1997: what bird caused the food-related poisoning of the respondent in Minnesota, during Fall, 1971? While his symptoms match those published for poisoning from ruffed grouse, the geographical location and season do not fit. The puzzle continues.

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