

The Other Skate Drops: The NHL Concussion Lawsuit

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Introduction

“Getting your bell rung,” “hits to the melon,” and basic bare-knuckles fighting have a long history at all levels of hockey including, most prominently, the National Hockey League (NHL), the premier professional hockey league in North America, comprised of 30 franchises located throughout the United States and Canada. Hockey, like football, is an extreme contact sport: body-checking¹ and body contact² are not only legal within the rules of the game, but are two of the primary means for impeding offensive movement and causing turnovers of the puck. Fighting, while a violation of the rules at all levels of hockey, has long been at least tacitly accepted for many years at the NHL level and in major junior hockey in Canada,³ teams have long employed “enforcers” known more for their pugilism than hockey skills. In recent years, the NHL has shown more motivation to limit fighting⁴ and eliminate head contact from the game,⁵ but as of the 2013-14 season, both remain represented in the sport; however, often penalties are called.

Due to the violent nature of the sport, concussions and subconcussive impacts (known collectively as mild traumatic brain injury, or MTBI) are common. Occasionally, worse occurs: In 1968, Bill Masterton, a player for the Minnesota North Stars, died due to a brain hemorrhage after being knocked down by a check and hitting his head on the ice.⁶ While Masterton remains the only NHL player to have died due to an on-ice incident, many more players over the years have been forced to retire due to the lingering impacts of MTBI.⁷

2011 was a particularly bad year for the NHL when it came to evidence of on-ice incidents causing lasting impact to brain functioning. While its marquee player, Sidney Crosby, was in the midst of a year-long battle with post-concussion syndrome (his initial concussion occurred on January 1, 2011; he did not return to full-time play until March 2012), Bob Probert’s family announced that researchers at Boston University’s Center for the Study of Traumatic Encephalopathy (BUCSTE), to whom the family had donated enforcer Probert’s brain after his heart-failure-related death at the age of 45, found that Probert’s brain displayed evidence of chronic traumatic encephalopathy (CTE), which had previously been diagnosed in over 20 deceased NFL players and two former NHL players.⁸ Probert became the first retired NHL player from the mandatory helmet era (which began with the NHL draft class of 1979, over the objections of the NHL Players’ Association (NHLPA)⁹), to be diagnosed with CTE.

Then, in the summer of that same year, three young players, each of whom was an enforcer—Wade Belak (35 years old and recently retired), Derek Boogaard (28 years old), and Rick Rypien (27 years old)—died in unusual circumstances. Belak and Rypien’s deaths were labeled suicides; both had struggled with depression. Boogaard’s death was attributed to an accidental overdose of prescription painkillers. Boogaard’s family donated his brain to BUCSTE and, again, the researchers discovered evidence of CTE.¹⁰ Furthermore, on July 19th of that year, 75 former National Football League (NFL) players brought suit against the NFL and NFL Properties,¹¹ alleging that the defendants were aware of, and intentionally withheld from NFL players, evidence of a link between MTBI and continuing disability, including early-onset dementia and post-concussion syndromes of indefinite duration.¹²

The filing of this suit and later consolidation of multiple lawsuits brought by former NFL players into one master case set the stage for action against the NHL. On November 25, 2013, after the NFL and its former players settled at \$765 million (via mediation),¹³ 10 former NHL players, on behalf of the class of all former NHL players who had retired by February 14, 2013, filed suit, in *Leeman v. NHL*, against the NHL and the NHL Board of Governors. While as of this writing the NHL has not yet delivered its response, the retired players have thrown down the gauntlet, as at last report over 200 former players had joined the class action.¹⁴

A Brief Look at the Medical Aspects of MTBI

Subconcussive Impacts

Although the classic example of a subconcussive impact is that of “heading” the ball in soccer, subconcussive impacts occur in all contact and collision sports. In hockey, subconcussive impacts are most likely to occur through body contact (e.g., a check into the boards, a fight), contact with the ice (e.g., a fall), or stick contact to the head (e.g., high-sticking). Although subconcussive impacts were rarely studied before 2000, mounting evidence questions whether repeated impacts can lead to neurological dysfunction later in life.¹⁵

In a subconcussive impact, the clinical evidence necessary to diagnose a concussion cannot be found; there are no diagnostic signs of neurological dysfunction.¹⁶ As a result, players continue to play through subconcussive impacts, which are most often caused by cranial impacts but may also be caused by a rapid acceleration or deceleration of the torso, allowing the “sloshing” of the brain

within the cranium.¹⁷ It is hypothesized that the effect of subconcussive impacts is cumulative; that the more subconcussive impacts an athlete suffers, the more likely such experiences contribute to later neurological deficits, including depression, postconcussive syndrome, post-traumatic stress disorder, mild cognitive impairment, CTE and dementia pugilistica.¹⁸

Subconcussive impacts, like all MTBI, involve at least a primary and secondary injury. The primary injury occurs at the moment of impact, while the secondary injury results from the pathophysiological processes—immediate and delayed cellular events and subsequent attempts by the body to reestablish homeostasis—involved in the injury.¹⁹ Researchers now posit that MTBI may also have a tertiary phase, which may become both chronic and compounded if multiple low-impact insults to the brain occur.²⁰ The likelihood of an athlete to suffer tertiary effects, especially later in life, cannot be predicted at this time, but is likely caused by a variety of factors, including: “age at exposure, type and magnitude of exposure, recovery periods, differential rates of recovery, genotype, individual vulnerability, and others.”²¹

Concussion

The definition of, and diagnostic procedures related to, concussion have evolved significantly over the years. In 2012, the fourth International Conference on Concussion in Sport (CIS Group) defined concussion as follows:

Concussion is a brain injury and is defined as a complex pathophysiological process affecting the brain, induced by biomechanical forces. Several common features that incorporate clinical, pathologic, and biomechanical injury constructs that may be utilized in defining the nature of a concussive head injury include:

(1) Concussion may be caused either by a direct blow to the head, face, neck, or elsewhere on the body with an “impulsive” force transmitted to the head.

(2) Concussion typically results in the rapid onset of short-lived impairments of neurologic function that resolves spontaneously. However in some cases, symptoms and signs may evolve over a number of minutes to hours.

(3) Concussion may result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance rather than a structural injury and, as such, no ab-

normality is seen on standard structural neuroimaging studies.

(4) Concussion results in a graded set of clinical symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course. However, it is important to note that in some cases symptoms may be prolonged.²²

This was a revision of the definition of concussion the third International Conference of the CIS Group promulgated in 2008, as that definition (1) did not include recognition of symptoms and signs of a concussion developing over hours and (2) stated that only *in a small percentage of cases* could symptoms be prolonged.²³

Concussion is diagnosed through assessment of “clinical symptoms, physical signs, cognitive impairment, neurobehavioral features, and sleep disturbance.”²⁴ The most common symptom of concussion is a headache, with dizziness, visual disturbance, temporary loss of consciousness or mental acuity, nausea, and fatigue also being common.²⁵ A report of any one or more of these symptoms should cause the medical support and training staff to suspect a concussion and commence evaluation.

The CIS Group recently set out a comprehensive set of guidelines, called the Sport Concussion Assessment Tool—3rd Edition (SCAT-3), for the use of athletic trainers and doctors involved in the diagnosis of concussions in athletes. Designed to update the widely-used SCAT-2,²⁶ the SCAT-3 contains guidelines for both medical assessment and sideline assessment, warning all training and medical staff that: “Any athlete with a suspected concussion should be REMOVED FROM PLAY, medically assessed, monitored for deterioration (i.e., should not be left alone), and should not drive a motor vehicle until cleared to do so by a medical professional. No athlete diagnosed with a concussion should be returned to sports participation on the day of the injury.”²⁷

Most concussions resolve within a week to 10 days.²⁸ However, a medical staff must evaluate each case on its own merits before issuing “return-to-play” (RTP) clearance. Over the past 30 years, several different RTP guidelines have been promulgated, with the CIS Group’s Graduated Return to Play Protocol (GRTPP) now being used as the touchstone. These guidelines provide that athletes should proceed to each subsequent step of the GRTPP if they remain asymptomatic at the previous step. The steps are: (1) No activity; (2) light aerobic exercise; (3) sport-specific exercise; (4) non-contact training drills; (5) full contact practice; (6) return-to-play. If symptoms appear at any step, the athlete is to drop back to the last step at which the athlete was asymptomatic, remain there for

at least 24 hours, and then try to progress to the next step again.²⁹ In addition to the GRTPP, the NHL utilizes neuropsychological (NP) assessments, meaning that a player who has been diagnosed with a concussion must return to his baseline neuropsychological functioning prior to being permitted to return to play, as "cognitive recovery may precede or more commonly follow clinical symptom resolution."³⁰ However, studies question the value of NP baseline testing,³¹ and the CIS Group does not recommend its widespread use, due to insufficient evidence of its efficacy.³²

Post-Concussion Syndrome

Post-concussion syndrome is defined as "symptoms and signs of the concussion that persist for weeks to months after the incident."³³ It is not a well-understood syndrome, as:

[S]ymptoms of a postconcussion syndrome can be subjective or objective and are often vague and non-specific making the diagnosis difficult. Although any symptom of concussion can be involved, commonly reported symptoms include: headache; dizziness; insomnia; exercise intolerance; cognitive intolerance; psychological symptoms such as depressed mood, irritability and anxiety; cognitive problems involving memory loss, poor concentration and problem solving; fatigue; or noise and light sensitivity.³⁴

Nor has the medical community agreed on a cause of post-concussion syndrome. Currently, researchers have been unable to prove a correlation among the severity of the concussion, post-concussion syndrome, structural brain damage, and psychological deficits.³⁵ However, there is little question that the syndrome is quite real, and that passage of time is the most important factor in recovery from post-concussion syndrome.

Chronic Traumatic Encephalopathy

CTE was first diagnosed in boxers in the 1920s.³⁶ However, in recent years, evidence of CTE has also been found in the brain tissue of deceased NHL and NFL players and, at the time of this writing, had been discovered in the brain of a deceased Major League Baseball player.³⁷ Diagnosis of CTE has proven difficult, as it can only be diagnosed post-mortem through histopathology.³⁸ Despite the limited sample available for study, it appears that a statistically significant number of professional athletes in collision and contact sports may develop CTE at a fairly young age. (Derek Boogaard, who died at 28, is the youngest professional athlete whose brain contained evidence of CTE).

CTE results from the aggregation of a certain protein, called tau, in specific regions of the brain.³⁹ As such, CTE

is a "tauopathy," like Alzheimer's Disease. The symptoms of CTE include "executive dysfunction, memory impairment, depression, and poor impulse control"⁴⁰ or, more specifically, "memory loss, confusion, impaired judgment, impulse control problems, aggression, depression, suicidality, parkinsonianism, and eventual progressive dementia."⁴¹

The current hypothesis is that CTE is caused by repeated MTBI (perhaps including repeated subconcussive impacts) wherein initially undetectable damage is done at the cellular level, thus causing a disease process which results in the manifestation of symptoms many years later.⁴² However, thousands of athletes have been subject to MTBI, and most have not proven symptomatic for CTE. Therefore, it is posited that other factors, such as a genetic predisposition, may increase the likelihood of developing CTE.⁴³

In 2013, UCLA neuroscientists released the results of a pilot study that may signal potential for diagnosing CTE in premorbid patients.⁴⁴ The UCLA researchers performed enhanced PET⁴⁵ scans on five retired NFL players, all of whom had a positive history of mood disturbance and cognitive impairment. Results of the study showed that all five players displayed enhanced signals in brain areas producing tau proteins after trauma (subcortical regions and the amygdala) when compared with members of the control group.⁴⁶ Although this study was a preliminary investigation, it does provide hope that there will soon be a way to diagnose CTE in living patients and thus determine how widespread it may be among athletes retired from contact and collision sports.

The Prevalence of MTBI in the NHL

Throughout the history of professional sports, scant records have been kept on injuries which do not cause a player to be removed from a game. In this respect, the NHL is no different from other professional sports. Therefore, evidence of subconcussive impacts (and, in the years before the mid-1980s, concussions) is largely anecdotal, coming from player and trainer recollections. Former player Bob Bourne, who played with the New York Islanders and Los Angeles Kings from 1974 through 1988 (and who has joined the players suing the NHL), described the situation to reporter Daniel Friedman as follows:

...All I can tell you is that I know for a fact that I felt like I was playing with the flu five times a year. It just felt weird, and we were sick a lot. We shouldn't have been, because we were very well taken care of; we had great doctors and everything like that and there's just no way that we should've been that sick all the time.

Now when I look back, I know there were certain times in my career where I must've been having concussions, because I was throwing up on the bench and throwing up on the ice and in practice. We threw up all the time. Now, a lot of that is because of the work we did, but it just felt weird a lot in those days and there had to have been a reason for it.

...When we went down on the ice, we got right back up; there was no laying on the ice. It was a peer-pressure thing—you came back from anything as soon as possible. Now, there's certain things like knees and shoulders where we couldn't just come out and play if it's not healed yet. But there was an unwritten rule that you played sick. It didn't matter how sick you were, you played. That's just an honor code that we had in those days, and I think the players would today too, if concussions weren't out on the forefront.⁴⁷

There is, however, statistical evidence of concussion numbers and rates in the NHL commencing in the 1980s. As Richard Wennberg and Charles Tator, a neurologist and neurosurgeon at the University of Toronto, carried out two retrospective studies on concussion rates in the NHL from the 1986-87 season through the 2007-08 season, utilizing injury reports made public by *The Hockey News*, *The Sporting News Hockey Register*, and *STATS LLC*.⁴⁸ Although their research was subject to the vagaries of team self-reporting of concussions (and NHL teams are loath to describe injuries to players with specificity), their results showed the following:

- Between 1986-87 and 1996-97, while the number of teams increased from 21 to 26, the reported number of concussions was fairly stable between seven and 17 concussions per season;
- Between 1997-98⁴⁹ and 2001-02, as the NHL increased from 26 to 30 teams, the number of concussions shot up as follows:
 - 1997-98: 62 concussions
 - 1998-99: 80 concussions
 - 1999-00: 75 concussions
 - 2000-01: 84 concussions
 - 2001-02: 67 concussions
- After 2002-03, in which there were 79 concussions, the number of concussions dropped steadily to 58 in 2006-07 (while the number of teams remained stable);

- Player average height and weight increased by one inch and nearly 10 pounds between 1986-87 and 2001-02 (no evidence is presented on size increases through 2007-08);
- There was a general downward trend in concussions suffered between 1998-99 and 2007-08, but a gradual increase in the number of games missed due to concussion.⁵⁰

Wennberg and Tator hypothesized that the increase in games missed per concussion was either due to increased severity of concussions or increased following of RTP guidelines; without evidence they were unable to conclude which hypothesis was correct. They also determined that changes made to the rules of the game and implemented in 2005-06 in order to “open up” the game (e.g., the elimination of the two-line pass, the stricter calling of obstruction penalties) resulted in the biggest one-season drop in concussions over the course of the last 10 years of the study (perhaps because body contact is reduced when the ice surface is more open, as in power plays or when players may “dangle” on the offensive side of center ice while the puck remains behind their defensive blue line).⁵¹

In 1997, the NHL and the NHLPA became the first major professional league to institute a formal study of concussion in their sport, in the form of the NHL-NHLPA Concussion Program (Concussion Program). A study approved by this program and carried out by the University of Calgary produced similar results to the Wennberg and Tator study, finding that over the years between 1997 and 2004, “the mean number of concussions per year was 80, with an overall rate of 5.8 concussions per 100 players per season.”⁵² Of these concussions: 362 were first concussive events (resulting in a median loss of six days); 116 were second concussive events (resulting in a median loss of eight days); 32 were third concussive events (resulting in a median loss of nine days); 12 were fourth concussive events (resulting in a median loss of seven days); and four were fifth concussive events (resulting in a median loss of 31 days).⁵³

Oddly, this study also reported that in 8% (44/529) of concussive instances, players returned to play in the game in which they were concussed, and later 14% of those players (6/529) went on to miss more than 10 days due to those concussions. This study, however, is prior to the CIS Group development of and NHL's adoption of the SCAT-2 criteria and also prior to the general acceptance of “when in doubt, sit them out.” The results of this study were reported to the NHL and NHLPA, with the researchers suggesting that:

...more should be done to educate all involved with the sport about the potential adverse effects associated with

continuing to play while symptomatic, failing to report symptoms to medical staff and failure to recognize or evaluate any suspected concussion. Our findings also suggest that more conservative or precautionary measures should be taken in the immediate postconcussion period, particularly when an athlete reports or experiences a post-concussion headache, low energy or fatigue, recurrent concussion, or many different postconcussion symptoms, or when the athlete has an abnormal neurologic examination.⁵⁴

In 2013, a study on the effects of repeated concussions on retired NHL players was published. This study, while qualitative and not quantitative (as it was based on a social-science study of player interviews), and using very small sample size of five retired players who had suffered multiple concussions, found that all five players' post-hockey lives (both professional and personal) were significantly impacted by the effects of their concussions.⁵⁵ Headaches, visual disturbances, and forgetfulness were common among the players.⁵⁶ Interestingly, and perhaps harmful to the current lawsuit, the players reported hiding their concussion symptoms from coaches and medical staff, and reported that they were removed from play only when the symptoms were discovered.⁵⁷

Leeman v. NHL: The Players' Complaint

In the *Leeman* complaint, the plaintiffs contend, repeatedly, that the NHL has been aware of—or should have been aware of—medical evidence of links between sports and brain injury for up to 85 years, and that, in the intervening years, a vast wealth of medical evidence has been developed linking sports injuries, and hockey injuries in particular, with the risk of MTBI and long-term neurocognitive damage. As the NHL is the world's premier professional hockey league,⁵⁸ because the NHL has promoted "a culture of violence"⁵⁹ and because the NHL has voluntarily assumed the duty to become the "arbiter of safety,"⁶⁰ the suit contends that the NHL failed in its duty to the plaintiff players.

This allegation that the NHL serves as the safety arbiter for the players, and has failed in that duty, deserves further explication. The complaint contends that:

- The NHL, since its earliest days, has engaged medical consultants to advise on hockey health risks;⁶¹
- The NHL has and had unilateral, monopolistic power to determine the rules and policies of the game;⁶²
- The NHL's voluntary assumption of this duty is exemplified by the helmet requirement; the NHL required all players to wear helmets starting in

1979, but grandfathered in all current players, who did not need to begin wearing a helmet;⁶³

- The NHL failed to act regarding concussive and sub-concussive injuries until 1997, although, since fighting was always accepted as an adjunct to the game, the NHL knew or should have known that the dangers inherent in the sport of boxing applied to the NHL;⁶⁴
- The NHL actively concealed concussive and sub-concussive risks from the players;⁶⁵
- In 1997, the NHL funded and engaged in a Concussion Program to study head impacts, which proves that the NHL assumed a duty of care regarding head injuries, but the NHL then fraudulently and negligently failed to make any statement of substance on the issue of concussions, despite the existence of the Concussion Program⁶⁶ and that the NHL, while engaging in the Concussion Program, made changes to the sport which increased risks to the players (including changing the glass in all arenas from flexible to rigid and speeding up the game by penalizing obstruction);⁶⁷
- It was not until 2010 that the NHL caught up with other hockey leagues to make head hits a distinct penalty;⁶⁸
- It was not until 2011 that the NHL required a doctor (instead of a trainer) to examine for a concussion, and not until 2011 that the doctor's exam had to take place in a quiet room, as opposed to on the bench. The doctor at present need not be a neurosurgeon;⁶⁹ and
- It was not until 2013 that the NHL mandated visors, but "veteran players" were grandfathered such that they need not wear visors.⁷⁰

All of this, according to the complaint, added up to a case of "too little, too late." The rules are alleged to be ineffective, the Department of Player Safety (established in 2011) to be ineffectual, and the NHL, although it knew or should have known about these risks for decades, complacent at best and engaged in the commission of fraud at worst.

Therefore, states the complaint, the players brought seven causes of action against the NHL. The first Count seeks declaratory relief as to NHL liability, stating that (a) the NHL knew or should have known that repeated head impacts to NHL players would expose them to brain damage and disability; (b) that the NHL, by virtue of its position vis-à-vis the players and its voluntary engagement in the Concussion Program, assumed a duty to warn players of the risks; (c) that the NHL "willfully and intentionally concealed from and misled" players con-

cerning the risk; and (d) that the NHL “recklessly endangered” its players.⁷¹

The second count demands that, because of the NHL’s tortious conduct in failing to disclose truthful information to the players after it voluntarily assumed a duty to them, the court enjoin the NHL to set up a medical monitoring program and provide a medical monitoring fund for the plaintiffs. The monitoring is to consist of diagnostic exams, not generally given to the public, to diagnose long-term effects from hockey-related MTBI in order to reduce the possibility of long-term harm.⁷² Such a medical monitoring fund was recently set up as part of the NFL lawsuit settlement.

The third count alleges fraudulent misrepresentation by concealment on the part of the NHL. The plaintiffs here argue that the NHL had been aware of short-term and long-term brain injury from repetitive head impacts since the 1920s and fraudulently concealed that risk from the players through 2010 (including the years of the Concussion Program). The players, the suit argues, would reasonably rely on the NHL’s statements or silence regarding MTBI, and, since the NHL was silent (although aware of the material facts), the plaintiffs relied on such silence to their detriment. As a direct and proximate result of this fraudulent concealment and willful misconduct, the plaintiffs allege that they have suffered injury and are entitled to damages.⁷³

The fourth count alleges fraudulent misrepresentation by non-disclosure. Much like in the third count, the plaintiffs argue that the NHL knew the facts about MTBI throughout the years at issue, that current and former NHL players did NOT know the facts, that the NHL *knew* that current and former players did not know the facts, and that by not disclosing these facts to them, the NHL could induce the players to continue to expose themselves to head injury. This, the complaint alleges, amounts to an intent to deceive and defraud, which fraudulent non-disclosure was the direct and proximate cause of the plaintiffs’ injuries, for which they are entitled to damages.⁷⁴

The fifth count sounds in fraud. Arguing that the NHL knew that MTBI risks would be diminished by the use of flexible glass; active monitoring of MTBI signs and symptoms; sideline neurologists; acceptable RTP rules; requiring doctors (instead of trainers) to evaluate potential MTBI in quiet rooms (instead of on the bench); and banning of fighting and body-checking, the plaintiffs posit that the NHL deliberately delayed the implementation of these changes and even now continues to “allow and market violence” because doing otherwise would be costly and decrease NHL profitability. As the NHL had superior experience and knowledge in these areas (as the “arbiter of player safety”), the players looked to the NHL for guidance. Therefore, as the NHL was withholding information and ignoring risks, all while knowing and fraudulently concealing such risks from the players, the players were

justifiably and reasonably relying on the NHL’s conduct. As a result, they assert, they suffered damages.⁷⁵

The sixth count claims negligent misrepresentation. The plaintiffs allege that a special relationship exists between the NHL and its players, thereby imposing a duty on the NHL to disclose accurate information to the plaintiffs. The NHL, although aware of and understanding the medical literature regarding short-term and long-term consequences of MTBI, continually insisted (and continues to insist) that more data is needed to establish a proven link between repetitive MTBI in hockey and neurocognitive impairment. This representation, per the plaintiffs, amounts to misrepresentation and concealment as it doesn’t comport with current medical knowledge, and, due to the special relationship between the NHL and its players, the NHL had a duty to disclose the consequences of MTBI in the medical literature.⁷⁶

The final count argues that the NHL has been negligent in failing to adequately address health issues caused by MTBI. The NHL, argue the plaintiffs, voluntarily assumed a duty to provide reasonable safety. That duty was breached by the NHL’s (1) failure to publicize to current players, retired players, and the public the evidence of neurocognitive problems arising from hockey-related MTBI and (2) perpetuation of the tough-guy culture, in which one plays through injury. These breached duties were the proximate causes of the plaintiffs’ injuries, thereby entitling the plaintiffs to damages.⁷⁷

Do the Players Have a Case?

As in the NFL concussion lawsuits, the *Leeman* plaintiffs have several large hurdles to overcome, both prior to and if this case finds its way to court. These hurdles include:

Pre-emption by Labor Law

The NHLPA was founded in 1967, with Alan Eagle-son as Executive Director and Bob Pulford as president. Since that time, the NHL and the NHLPA have negotiated multiple collective bargaining agreements between them (CBAs). Article 34 of the current CBA contains an extremely detailed set of terms agreed to by the parties regarding player health and safety.⁷⁸ Article 34.9 tasks a joint advisory committee of the NHL and NHLPA, the Joint Health and Safety Committee, to make advisory opinions to the NHL and the NHLPA regarding player health and safety issues, although final decisions regarding health and safety issues (provided that such decisions do not contravene CBA terms) are reserved to the NHL. Further, pursuant to the CBA, all disputes relating to the application and interpretation of, and compliance with, the CBA are to be resolved through the NHL-NHLPA Grievance Committee, and if a resolution cannot be reached there, through arbitration by an impartial arbitrator.⁷⁹

Pursuant to Section 301 of the Labor-Management Relations Act (LMRA), state law claims are completely pre-empted when the resolution of those claims depends upon the interpretation of a CBA.⁸⁰ The claims brought in *Leeman* appear to be just such claims. As the NHL and NHLPA have negotiated medical care, authority for player health and safety, and grievance arbitration procedures relating thereto, it is very possible that LMRA pre-emption will apply, and the *Leeman* case will then face dismissal.

Causation

Even if the *Leeman* plaintiffs leap the pre-emption hurdle, there is the question of causation: can any player actually prove that the injuries causing damage were sustained at the NHL level and not at another level of hockey (e.g., peewee, minor league, junior league)? Even if it can somehow be proven that a player's neurocognitive injury was caused by a hockey injury (in itself a difficult proof, given the current state of medical science on MTBI), how can that injury be proven to have occurred in the NHL?

Given the relatively small number of games some named members of the putative class in *Leeman* played in the NHL (Brad Aitken played 14,⁸¹ Morris Titanic played 19,⁸² Darren Banks played 20,⁸³ Warren Holmes played 45⁸⁴), the problem of causation becomes even more clear: Warren Holmes played 45 games in the NHL and over 500 in minor league hockey. Brad Aitken played 14 games in the NHL and over 350 in minor league hockey. The question then becomes: Where is it most likely that the damage occurred: the NHL or elsewhere in another league?

Even Morris Titanic, a named member of the putative class, does not profess to know: "...only playing 19 games for Buffalo didn't much matter. You're playing somewhere. Whether things happened while you were in Buffalo, in the American League, junior, who knows? There's really nothing I guess from what I've read about this CTE and things of that nature, there's really not a specific injury that you can put a finger on that, 'Yeah, well, it happened on that date and that's why he's all messed up.'"⁸⁵

When even the class members are legitimately unsure of causation, it seems a good bet that the fact-finders will be as well.

NHLPA Responsibility

The NHLPA—as exclusive bargaining representative for the players, equal partner in the Joint Health and Safety Committee, and co-founder of the Concussion Program—is mentioned nowhere in the *Leeman* complaint. Why, however, would the NHL not turn around and lay any blame there is to be had at the feet of the NHLPA? Should not the duty to inform the players of health risks they face be an obligation of the very union that represents them? Furthermore, if blame is to be placed, there

is historical evidence of NHLPA foot-dragging on safety-minded modifications the NHL desired to make, such as in adoption of mandatory helmets and visors.

Lack of Evidence of NHL Fraud and Malfeasance

Unlike the NFL complaint, in which there were allegations that NFL actively meddled with the science, no such allegations are found in the *Leeman* complaint. Instead, some see the *Leeman* case as trying to promote an ethical, but not legal, claim. Michael McCann, Professor of Sports Law at the University of New Hampshire, states the following:

I don't know if I saw in this complaint as much as we saw in the complaint against the NFL, in terms of allegations of misconduct. Much of this complaint focused on how the NHL could've made the game safer at various points in time and how the league knew of information and didn't allegedly share it. In the NFL, there was the allegation that the league went out of its way to cloud the science. I didn't see any of that in this complaint. I saw that NHL could've done more and was interested in making money. Maybe there are ethical issues, but I don't see how that's necessarily a strong legal argument.⁸⁶

Furthermore, there is evidence that the NHL has been the most proactive of all the major leagues when it comes to concussion. For example:

- The NHL was the first league to institute baseline testing;
- The NHL was the first league to create a Concussion Program; and
- The NHL was the first league to create a quiet room for the use of the SCAT-2.

Therefore, the moral argument that the NHL did not do enough is clearly on the table, but the legal argument that the NHL was engaged in fraudulent misrepresentation through concealment and non-disclosure may rapidly fail.

Issues Regarding Availability and Conclusive Nature of Data

The complaint repeatedly argues that the NHL had the ability to—and did—synthesize and understand data pertaining to MTBI which it thereafter did not make available to the players. Yet this data comes from generally available medical research. Again, Professor McCann argues: "Those studies are publicly available. So it's hard to call that any kind of fraud. It seems as if the players and their own union could've availed themselves of that information."⁸⁷ While it may be unrealistic to expect a

20-year-old hockey player to pay attention to this information, it may be equally unrealistic to lay the obligation to serve as clearinghouse for all health-related information upon the NHL.

Furthermore, since this information was publicly available,

[t]he league will also take the position that it didn't conceal any information. Rather, they will argue that there wasn't any conclusive science at the time and they had the same information the players had. Basically, they will say, "we knew what you knew." That being the case, the league will maintain that the players were aware of the risk associated with playing hockey based on the science at that time, and agreed to those risks each time they stepped onto the ice. This legal principle is called informed consent.⁸⁸

In reviewing the studies cited in this article, it became apparent that to a large extent, conclusions regarding MTBI still cannot be drawn with confidence, thus supporting any argument by the NHL that the data necessary to make a final determination regarding the causation and effects of MTBI is not conclusive. For example:

- "At present there is insufficient evidence to recommend the widespread routine use of baseline NP testing."⁸⁹
- "The exact structural and chemical changes that produce these changes are not clear entirely."⁹⁰
- "At this time the number or type of hits to the head needed to trigger degenerative changes to the brain is unknown."⁹¹
- "The exact role and impact on concussion management of baseline testing remains unclear, as no study has shown that the use of these tests provides better short-term or long-term outcomes for athletes with concussions."⁹²

Given inconclusive statements such as the above, it will likely be difficult to pin the NHL to an obligation to reach a conclusion regarding the short- and long-term risks of MBTI to its players based on the data provided.

Conclusion

In recent years, it has become clear that a significant number of professional athletes retired from contact and collision sports like football and hockey are struggling with long-term post-concussion syndrome and neurocognitive impairment. Furthermore, studies have found evidence of CTE in several dozen deceased players. A tragic situation for each player involved, certainly, but the

question remains: how much liability should and do the professional leagues have for these outcomes when causation is uncertain and the science is developing? As yet, we have no judicial answer, and the hurdles that must be overcome by the plaintiffs in the lawsuits currently being brought are high.

One thing is certain: the NHL is not as wealthy as the NFL. While the NFL's 2012 revenues were \$9.5 billion,⁹³ the NHL's revenues in that same year were \$3.3 billion.⁹⁴ The NFL can manage a settlement in the amount of \$765 million or more, while such a settlement in the NHL would gobble up one-quarter of its revenues. It also appears that the NHL has not engaged in any of the nefarious activities of which the NFL was accused, and also made efforts to implement concussion monitoring and protection at an earlier stage in the development of the science of MTBI than did the NFL. A protracted lawsuit seems to serve no one—not players suffering from neurocognitive impairment, and not the leagues. However, a quick, large settlement to avoid the years and expense invested in a lawsuit could be very harmful to the NHL.

Perhaps the NHL should once again play the role of the bellweather in MBTI management and prevention. A medical monitoring program formed, managed, and funded jointly by the NHL, the NHLPA, and the NHL Alumni Association would be just such a step. If these three parties can see the value in collaborating, players who need help will receive it, players at risk of MBTI will be aware, and the three entities which together represent or employ nearly every current or retired player will prove that they consider the issue of MBTI prevention and management to be a serious problem worthy of a medicolegal solution, instead of rapid and expensive settlement to avoid public relations issues or drawn-out litigation to prove liability.

Endnotes

1. Body-checking is defined as: "an individual defensive tactic designed to legally separate the puck carrier from the puck. This tactic is the result of a defensive player applying physical extension of the body toward the puck carrier moving in an opposite or parallel direction. The action of the defensive player is deliberate and forceful in an opposite direction to which the offensive player is moving and is not solely determined by the movement of the puck carrier." Hockey Canada, *Teaching Checking Skills*, available at <http://www.hockeycanada.ca/en-ca/news/2003-gn-009-en>.
2. Body contact is defined as "an individual defensive tactic designed to legally block or impede the progress of an offensive puck carrier. This tactic is a result of movement of the defensive player to restrict movement of the puck carrier anywhere on the ice through skating, angling and positioning. The defensive player may not hit the offensive player by going in opposite direction to that player or by extending toward the offensive player in an effort to initiate contact. There must be no action where the puck carrier is pushed, hit or shoved into the boards." Hockey Canada, *Teaching Checking Skills*, available at <http://www.hockeycanada.ca/en-ca/news/2003-gn-009-en>.

3. "Major junior hockey" consists of the Quebec Major Junior Hockey League, the Ontario Hockey League ("OHL") and the Western Hockey League.
4. The NHL's fighting rules may be found at <http://www.nhl.com/ice/page.htm?id=26336> (Rule 46).
5. The NHL's rules regarding checks to the head carry with them suspensions for repeat offenders, <http://www.nhl.com/ice/page.htm?id=64063> (Rule 48).
6. Masterton played in the pre-mandatory helmet era. He was not wearing a helmet at the time of his injury.
7. See, e.g., *Thirteen NHL Careers Cut Short by Concussions*, TOTALPROSPORTS.COM (Sept. 1, 2011), <http://www.totalprosports.com/2011/09/01/13-nhl-careers-cut-short-by-concussions/>.
8. Reggie Fleming and Rick Martin, both of whom played prior to the mandatory helmet era, were the two former NHL players whose brains showed evidence of CTE prior to the Probert revelation.
9. Mark Ritter, *NHLPA to Blame for Safety Issues, Not the NHL*, BLEACHERREPORT.COM, <http://bleacherreport.com/articles/285645-nhlpa-to-blame-for-safety-issues-not-the-nhl>.
10. Boogaard's family filed a wrongful death suit against the NHL in May 2013. John Branch, *In Suit Over Death, Boogaard's Family Blames the N.H.L.*, N.Y. TIMES, May 12, 2013.
11. *Maxwell v. National Football League*, No. BC 465 842 (Cal. Super. Ct. July 19, 2011).
12. Carrie Anderer, *The NFL Concussion Settlement: A Win for Both Sides?* 24 ENTERTAINMENT, ARTS, & SPORTS 3 (2013).
13. *Id.*
14. Greg Wyshinski, *NHL Concussion Lawsuit Stumbles into the Spotlight*, SPORTS.YAHOO.COM (Nov. 29, 2013), <http://sports.yahoo.com/blogs/nhl-puck-daddy/nhl-concussion-lawsuit-stumbles-spotlight-175433418--nhl.html>.
15. Julian E. Bailes, MD et al., *Role of Subconcussion in Repetitive Mild Traumatic Brain Injury*, 119 J. NEUROSURG. 1235 (November 2013). It is of relevance to the NHL concussion suit that this study also reveals that, "it is now often stated that the MTBI research produced during the past decade supersedes the volume and content of all previous information."
16. *Id.*
17. *Id.*
18. *Id.*
19. *Id.*
20. *Id.*
21. *Id.* Dr. Bailes, the lead author of the study cited in note 15, also wrote an op-ed for the New York Daily News, arguing that, with regard to football, "The sum of these 'subconcussive' blows may cause serious harm. Organized football, at all levels, needs to go beyond its admirable work in trying to prevent helmet-to-helmet hits and concussions and address the additional issue of repetitive head blows. We must work to reduce and remove head impacts from the game as much as possible." Julian Bailes, MD, *This is your brain on the gridiron: Are subconcussive hits the most dangerous of all?* N. Y. DAILY NEWS, March 9, 2011, www.nydailynews.com/opinion/brain-gridiron-sub-concussive-hits-dangerous-article-1.120538.
22. Paul McCrory, MBBS, Ph.D. et al., *Consensus Statement on Concussion in Sport—the 4th International Conference on Concussion in Sport held in Zurich, November 2012* (hereinafter "CIS 4"), 23 CLIN. J. SPORT MED. 2 (March 2013).
23. Paul McCrory, MBBS, Ph.D. et al., *Consensus Statement on Concussion in Sport: The 3rd International Conference on Concussion in Sport Held in Zurich, November 2008*, 44 J. ATHLETIC TRAINING 4 (August 2009).
24. CIS 4, *supra* note 22.
25. Mark Lovell, Ph.D. *The Management of Sports-Related Concussion: Current Status and Future Trends*, 28 CLIN. SPORTS MED. 95 (2009).
26. SCAT-2 is used by the NHL. See Jeff Z. Klein, *N.H.L. Urges Uniformity on New Concussion Protocol*, N.Y. TIMES, March 20, 2011.
27. A copy of the SCAT-3 is available at <http://bjsm.bmj.com/content/47/5/259.full.pdf>.
28. CIS 4, *supra* note 22.
29. CIS 4, *supra* note 22, at Table I.
30. CIS 4, *supra* note 22.
31. *Id.* See also Christopher Randolph, *Is Neuropsychological Testing Useful in the Management of Sports-Related Concussion*, 20 J. ATH. TRAIN. 139 (Jul-Sept 2005).
32. CIS 4, *supra* note 22.
33. Kimberly G. Harmon et al., *American Medical Society for Sports Medicine Position Statement: Concussion in Sport*, 47 BR. J. SPORTS. MED 15 (2013).
34. *Id.*
35. *Id.*
36. *What is CTE?* Boston University Center for the Study of Traumatic Encephalopathy (hereinafter "BUCSTE"), www.bu.edu/cste/about/what-is-cte/.
37. Elliott Hannon, *Report Finds MLB's First Case of Degenerative Brain Disease in Former Player's Suicide*, SLATE.COM, Dec. 16 2013, http://www.slate.com/blogs/the_slatest/2013/12/16/former_mlb_player_ryan_freel_had_cte_when_he_committed_suicide_a_report.html.
38. Histopathology is the study of the microscopic anatomical changes in diseased tissue. THEFREECTIONARY.COM. <http://www.thefreedictionary.com/histopathology>.
39. BUCSTE, *supra* note 36.
40. Harmon, *supra* note 33.
41. FAQ, Boston University Center for the Study of Traumatic Encephalopathy (hereinafter "BUCSTE FAQ"), www.bu.edu/cste/about/faq/.
42. In this respect, Derek Boogaard would be an extreme outlier or his injuries must have been caused at an early age.
43. BUCSTE FAQ, *supra* note 41.
44. Gary W. Small, MD et al., *PET Scanning of Brain Tau in Retired National Football League Players; Preliminary Findings*, 21 AM. J. GERIATR. PSYCHIATRY (Feb. 2013).
45. PET stands for Positron Emission Tomography; the enhancing agent that was intravenously injected with a chemical marker called FDDNP.
46. Small, *supra* note 44.
47. Daniel Friedman. *Former Islander Bob Bourne Opens Up on Concussions*, www.newyork.cbslocal.com/2013/11/27/friedman-former-islander-bob-bourne-opens-up-on-concussions/.
48. Richard A. Wennberg and Charles H. Tator, *Concussion Incidence and Time Lost from Play in the NHL During the Past Ten Years*, 35 CAN. J. OF NEUROL. SCI. 647 (2008) (hereinafter Wennberg 2008); Richard A. Wennberg and Charles H. Tator, *National Hockey League Reported Concussions, 1986-87 to 2001-02*, 30 CAN. J. NEUROL. SCI. 206 (2003) (hereinafter Wennberg 2003).
49. In this year, the NHL and the NHL Players' Association formed the NHL-NHLPA Concussion Program.
50. Wennberg 2008, *supra* note 48; Wennberg 2003, *supra* note 48.
51. *Id.*

52. Brian W. Benson, MD, PhD, *A Prospective Study of Concussions Among National Hockey League Players during regular season games: the NHL-NHLPA Concussion Program*, 183 CMAJ 905 (May 2011).
53. *Id.*
54. *Id.*
55. Jeffrey G. Caron et al., *Effects of Multiple Concussions on Retired National Hockey League Players*, 35 J. SPORTS & EXERCISE PHYSIOLOGY 168 (2013).
56. *Id.*
57. *Id.*
58. Complaint for Damages and Demand for Jury Trial, *Leeman v. National Hockey League*, Civil Case No. 13-CV-1856 (U.S. Dist. Ct. D.C., Nov. 25, 2013) at ¶¶ 78-84.
59. *Id.* at ¶¶ 85-90.
60. *Id.* at ¶¶ 91-97.
61. *Id.* at ¶ 92.
62. *Id.*
63. *Id.* at ¶ 95. The Complaint fails to discuss the NHLPA's role in the helmet controversy.
64. *Id.* at ¶ 99.
65. *Id.* at ¶ 100.
66. *Id.* at ¶ 102. The Complaint nowhere mentions that this is a joint program of the NHL and NHLPA.
67. *Id.* at ¶ 110.
68. *Id.* at ¶ 112.
69. *Id.* at ¶ 116.
70. *Id.* at ¶ 121. Again, the role of the NHLPA in this issue is not disclosed in the complaint. In March 2013, the NHLPA rejected the NHL's request to make visors mandatory. It was not until the NHLPA polled its members that the NHLPA agreed to the visor rule. See Mike Halford, *NHL Makes Visors Mandatory for New Players*, NBCSPORTS.COM, June 4, 2013, <http://prohockeytalk.nbcports.com/2013/06/04/nhl-makes-visors-mandatory-for-new-players/>.
71. *Leeman v. National Hockey League*, *supra* note 58, at ¶¶ 148-150.
72. *Id.* at ¶¶ 151-168.
73. *Id.* at ¶¶ 169-182.
74. *Id.* at ¶¶ 183-196.
75. *Id.* at ¶¶ 197-210.
76. *Id.* at ¶¶ 21-225.
77. *Id.* at ¶¶ 226-233.
78. Collective Bargaining Agreement Between National Hockey League and National Hockey League Players' Association, September 16, 2012-September 15, 2022, Article 34, available at http://cdn.agilitycms.com/nhlpacom/PDF/NHL_NHLPA_2013_CBA.pdf (last visited Dec. 24, 2013).
79. *Id.* at Article 17.
80. Labor Management Relations Act (LMRA), 29 U.S.C. § 185.
81. Statistics of Brad Aitken, <http://www.hockeydb.com/ihdb/stats/pdisplay.php?pid=5952>.
82. Statistics of Morris Titanic, <http://www.hockeydb.com/ihdb/stats/pdisplay.php?pid=6759>.
83. Statistics of Darren Banks, <http://www.hockeydb.com/ihdb/stats/pdisplay.php?pid=193>.
84. Statistics of Warren Holmes, <http://www.hockeydb.com/ihdb/stats/pdisplay.php?pid=6541>.
85. John Vogl, *Ex-Sabre Titanic states his case against NHL*, BUF. NEWS, Dec. 15, 2013.
86. *Not All Players Ready to Take League To Task in NHL Concussion Lawsuit*, THE ASSOCIATED PRESS, Nov. 26, 2013, <http://www.usatoday.com/story/sports/nhl/2013/11/26/nfl-nhl-concussion-lawsuit-not-same/3761489/>.
87. *Id.*
88. Eric Macramella, *Legal Look: The Concussion Lawsuit Against the NHL*, TSN.CA, Nov. 26, 2013, <http://www.tsn.ca/nhl/story/?id=437555>.
89. *CIS 4*, *supra* note 22.
90. Steven P. Broglio, *Cognitive Decline and Aging: The Role of Concussive and Subconcussive Impacts*, 40 EXERC. SPORT SCI. REV. 138 (July 2012).
91. BUCSTE FAQ, *supra* note 41.
92. Harmon, *supra* note 33.
93. Daniel Kaplan, *The Road to \$25 Billion*, SPORTS BUS. J., January 28, 2013, <http://www.sportsbusinessdaily.com/Journal/Issues/2013/01/28/In-Depth/NFL-revenue-streams.aspx>.
94. *Impact of Revenue Growth on NHL and NHLPA Proposals*, SB NATION, Sept. 14, 2012, <http://www.silversevensens.com/2012/9/14/3329508/impact-of-revenue-growth-on-nhl-and-nhlpa-proposals>.

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