The Physiology and Pathology of Formula One Grand Prix Motor Racing

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INTRODUCTION

In 1978, Professor Watkins was appointed as Surgical Consultant to the Formula One Constructors Association, the body representing Grand Prix racing in the World Championship Formula One races of the Federation du Sport de L'automobile (FIA), the World Governing body for International Motor Sport. His presence was required at all Grand Prix events from June 1978 until November 2004, a total of 423 races. His role was to raise the standards of medical response at the circuits and to provide specialist medical advice in the case of injury.

In May 1994, after a disastrous weekend at Imola, Italy, in which two drivers were killed, the President of the FIA, Max Mosley, appointed Professor Watkins to chair a commission of experts to examine all aspects of car construction, circuit design, and safety features, with the ultimate goal of zero mortality in Formula One Grand Prix Racing. Much research emanated from this important decision. Research extended into other forms of motor racing and led to the formation of the Institute of Motor Sport Safety in 2004, which encompasses now all Open Cockpit car racing, Closed car racing (Rally, Saloon, Grand Touring) and recently Go-kart racing.

THE PHYSIOLOGY OF MOTOR RACING

The emotional and physical stresses experienced by a Formula One driver are summarized in *Figure 14.1*, published by Bertrand et al. in 1983.[1] These include the muscular exercise involved in the physical effort of driving the car, and withstanding the G forces during acceleration and cornering, the latter up to 4G. Thus, the neck and shoulder muscles in a 4G corner would need to be controlling a head and helmet mass of approximately 6.5 kg, which would produce a load at 4G of 26 kg. Building up the shoulder and neck muscles by heavy exercise is an important part of training for race drivers. However, the most dramatic effects of the stresses are on the cardiovascular system.[1–3] In addition to muscular physical effort and G forces, there are added burdens on the heart rate from vibration, thermal loads, and emotion, leading to pulse rates of up to 200 beats per minute (bpm). *Figure 14.2* shows the effect of increasing speed on the pulse rate of two drivers (Villeneuve and Pironi) and also demonstrates individual differences arising from personality and training—Villeneuve being the cooler and more composed of the two. *Figures 14.3* and *14.4* show the stress of these two drivers in practice in Monaco, there being a drive on the heart on entering the circuit and relief on leaving it. Clearly, in Villeneuve's case, an incident between laps in the motor home accelerated his pulse!

The effect of G forces and cornering on the heart rate are shown in *Figures 14.5* and *14.6*, where, at Le Mans, Pironi's pulse rate was at its lowest (155–135 bpm) on the fastest section of the circuit, the Mulsanne straight. Figure 14.6, at Fiorano, the acceleration of the pulse is clearly shown during cornering, although the G forces at the corners there did not exceed 2.9G. The effect of anxiety on heart rate when entering the circuit and at the start of a session at Le Mans is shown in *Figure 14.7*.

The effect of thermal load and increase in body temperature provides a further factor. In 1956, Ladell and Watkins[4] showed that for a rise of each degree, the heart rate increases 25 beats during working. The need for heat acclimatization for races in hot climates is paramount, despite which, Senna, who ran in the heat of Brazil to acclimatize (8 km, 16 km, and 24 km on successive days) reached a pulse rate of 190 bpm.[5]

Acclimatization helps performance by body cooling that is more efficient because of increased sweat rate response at lower body temperatures and a reduction of salt loss in sweat conserving sodium. Salt depletion in muscles produces cramp, and sweat gland fatigue because of raised body temperature and dehydration will cause collapse. Dehydration can occur because of sweating at maximal rates of 2 L/h. Richalet and Bertrand[1,2] found fluid loss in Formula One drivers to be 1 L/h and, at Le Mans, endurance driving led to an increase of blood cellular volume from 46 to 56% in 24 hours, indicating a 20% reduction of circulating fluid volume. Naccarella et al.[6,7] confirmed a 10 to 15% increase in hemat-

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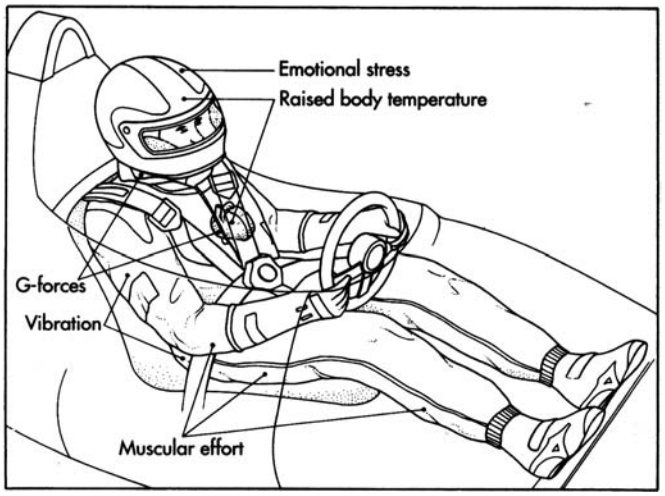


FIGURE 14.1 Emotional and physical stresses experienced by a Formula One driver (from Bertrand C, Keromes A, Lemeunier BF, Meistelmann C, Prieur C, Richalet JP. *Physiologie des Sports Mécaniques*. 1st International Congress of Sport Automobile, Marseilles, 1983[1]).

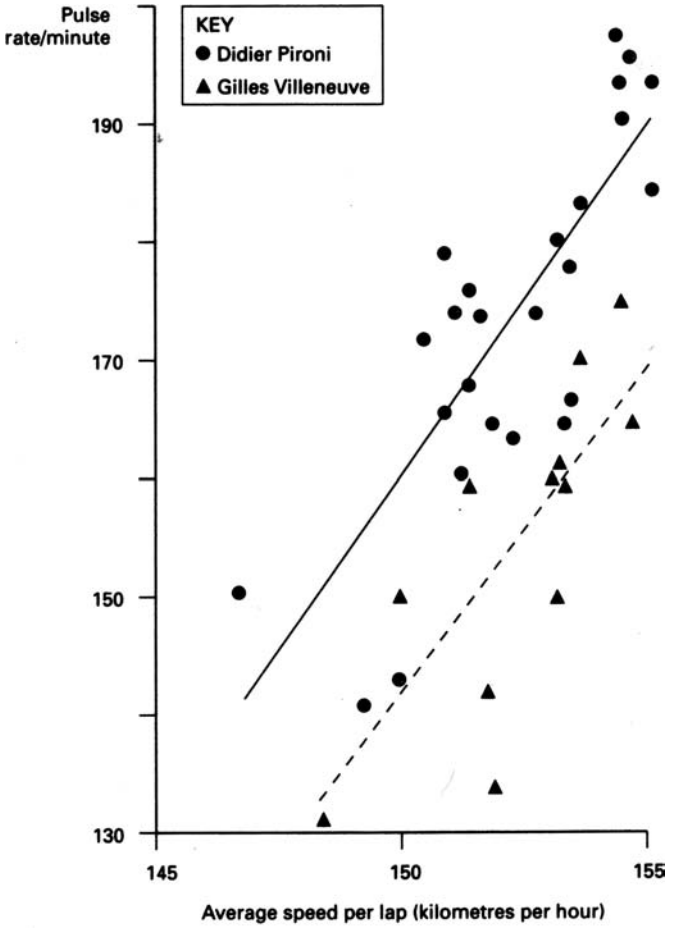


FIGURE 14.2 Formula One trials at Fiorano. Each point represents a lap. Notice how Pironi's pulse rate reached nearly 200 bpm at the highest speeds, whereas Villeneuve's pulse rate barely exceeded 175 bpm.

ocrit, with reductions of sodium, potassium, and magnesium ions and an increased production of catecholamine, cortisol, and aldosterone as an adrenal biological response to stress. Former Grand Prix driver, Dr. Jonathan Palmer, together with neurosurgeon Brian Simpson, measured cortisol and testosterone levels in saliva during testing and racing and found significantly increased levels (personal communication). These increases in the stress hormones also increased in the investigators saliva during a frightening helicopter flight in bad weather. FIA fluid intake guidelines[8] advise taking 1 L before racing, 1 to 2 L during, and 2 L after a race. Vibration in the cockpit caused by rigid suspension systems produces vertical loading in the spine in the vertebral bodies and intervertebral discs with the spinal muscles working to maintain posture, counteracting G forces, and working constantly to minimize vertical stretch and compression. Burton[9,10] showed changes in suspension from 1982 (virtually no suspension) introduced by the FIA with a reduction of down force and of 50% in suspension stiffness, significantly reduced back strain and spinal pain in the 15 Grand Prix drivers studied.

Because of the vertical forces, disc herniation is more likely to be vertical (intraosseous) rather than horizontal, as observed in the lumbar and thoracic spine, and the constant neck movement is likely to lead to early cervical arthritis changes—as in boxers, footballers, and jockeys.

Damage to peripheral nerves caused by vibration and contact with the cockpit sides may occur. The tight constraint of the cockpit opening has produced malfunctions of the radial nerves in the radial groove of the humerus, with the result of dropped wrists at the end of the race (so called Saturday night palsy). Repeated concussion of the lateral popliteal nerves against the inside of the cockpit produced extensor palsy in one driver that took months to recover.

All of these factors outlined can affect performance. The influence of climate on psychomotor skills and physical performance is of great importance. During World War II and, thereafter, much research was conducted by military and defense scientists to define the adverse factors producing a decrement in performance in air pilots, naval technicians, tank and truck drivers, and submariners. Blockley et al.[11–13] at the Wright Patterson Air Force Base showed that the competent performance of aircraft pilots could be maintained for only 20 to 30 minutes in temperatures of 160 to 200°F before psychomotor and physical collapse occurred. In 1950, Mackworth[14] and his colleagues at the Cambridge Psychological

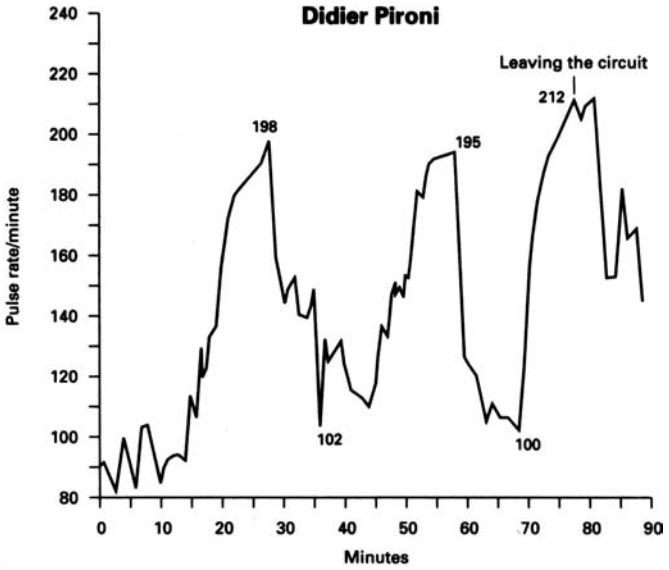


FIGURE 14.3 Monaco: Formula One practice. Didier Pironi's pulse rate reflects the stress of driving on the circuit.

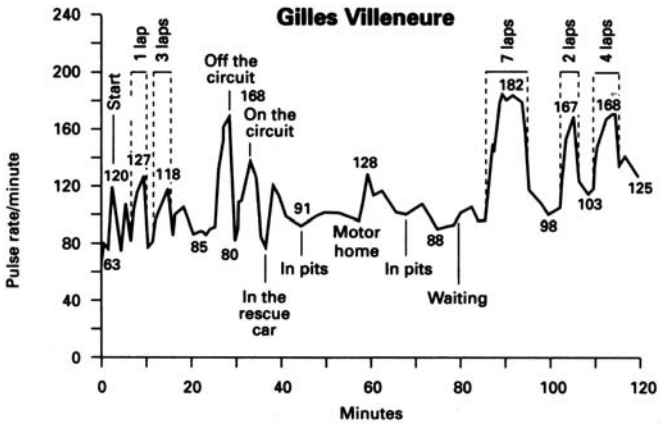


FIGURE 14.4 During the second series of nonqualifying practice, Villeneuve's cardiac response reached a maximum of 182 bpm.ñ

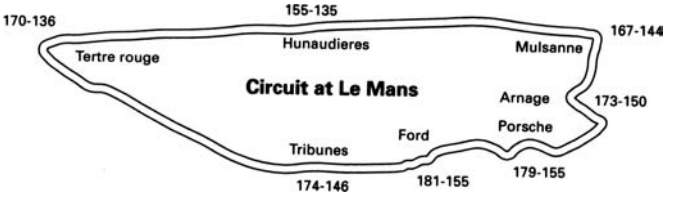


FIGURE 14.5 Variations in Pironi's pulse rate, mean of eight laps. The first figure relates to the first driving session and the second to the fourth and final driving session. The pulse rate is at its lowest on the Mulsanne Straight, despite the speed being at its highest.

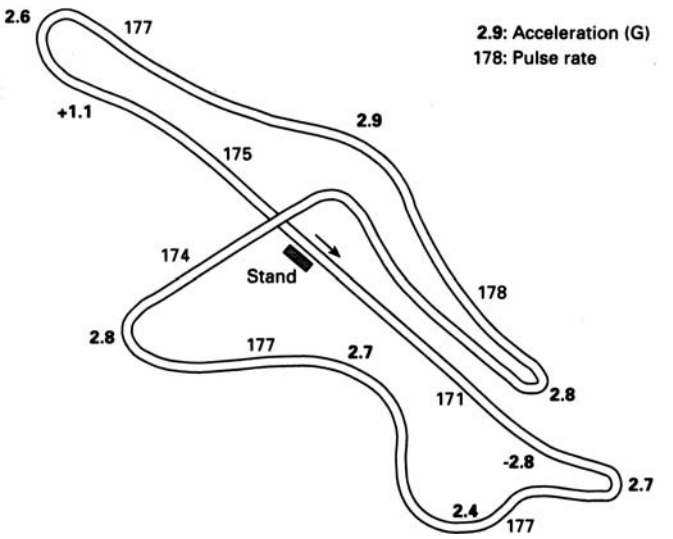


FIGURE 14.6 Pulse rate and G forces under cornering, breaking, and accelerating at Fiorano. The heart rate goes up to 178 bpm during cornering; the G force on the corners reaches 2.9G.

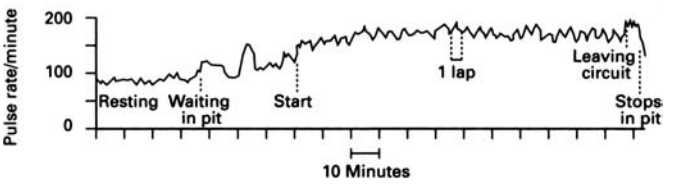


FIGURE 14.7 Variations in Pironi's pulse rate during 24 hours at Le Mans against time (2 hours). First session.

Unit tested unacclimatized service personnel in five differing climates of dry bulb and wet bulb temperatures, respectively 85°F and 75°F; 90°F and 80°F; 95°F and 85°F; 100°F and 90°F; and, the hottest, 105°F and 95°F in an air movement speed of 100 ft/min.

Three tasks were used: a simple manual dexterity test, a task requiring concentration and physical effort tracking a marker through a complex tortuous pattern (pursuitmeter test, driving simulation) by moving a lever clamped by a heavy weight, and, finally, a mental task of receiving wireless telegraphy messages in Morse Code. The results of the mental task are summarized in *Figures 14.8* and *14.9*, together with the results obtained in tropically acclimatized men in work repeated by myself[15] using the same tasks and the same climatic conditions.

In both series, climate made no statistically significant difference to the performance of the manual dexterity test. In the pursuitmeter test, severe decrement occurred in both series in temperatures exceeding Dry bulb 95°F, Wet bulb 85°F—an effective temperature of 86°F.

In the wireless telegraphy test in both series, decrement in performance occurred in climates hotter than 95°F Dry

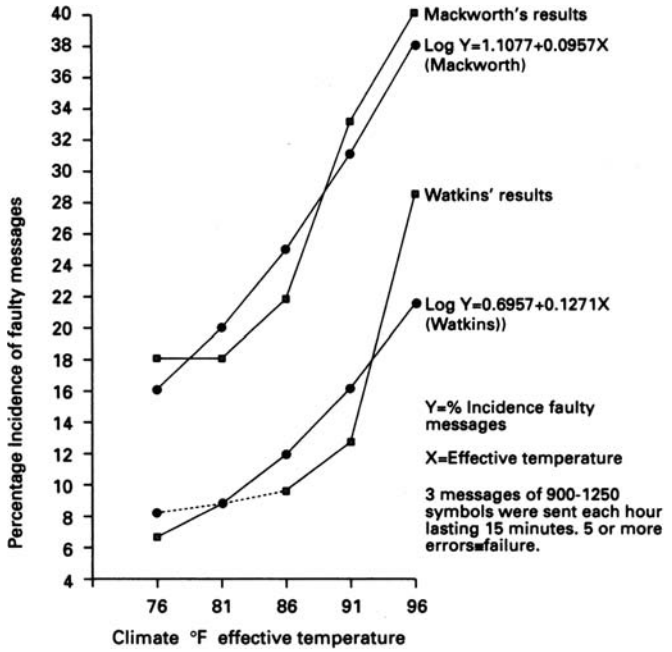


FIGURE 14.8 Percentage incidence of faulty messages with climate and logarithmic curves of performance compared on the same scale.

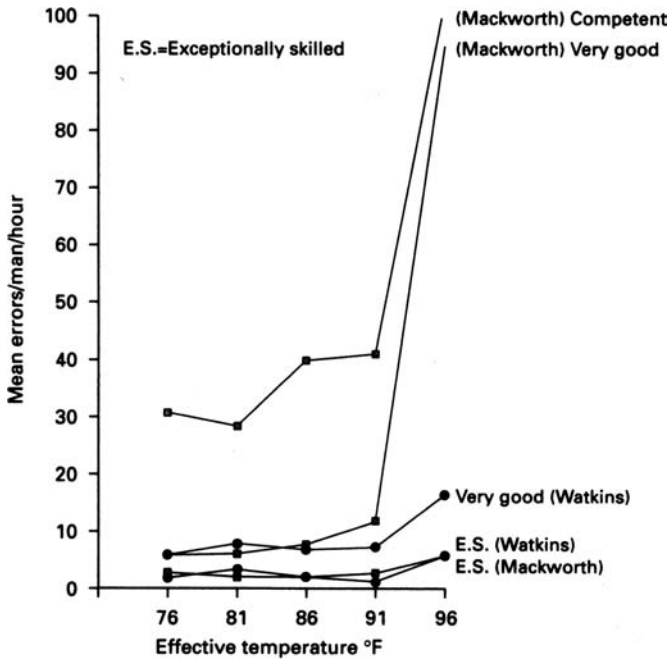


FIGURE 14.9 Errors per man per hour classed by ability.

bulb, 85°F Wet bulb. In both series, there was a logarithmic relationship to ambient temperature of remarkable similarity, but the failure rate in tropically acclimatized man was lower. The skill of the subjects is of importance and those of greatest

TABLE 14.1

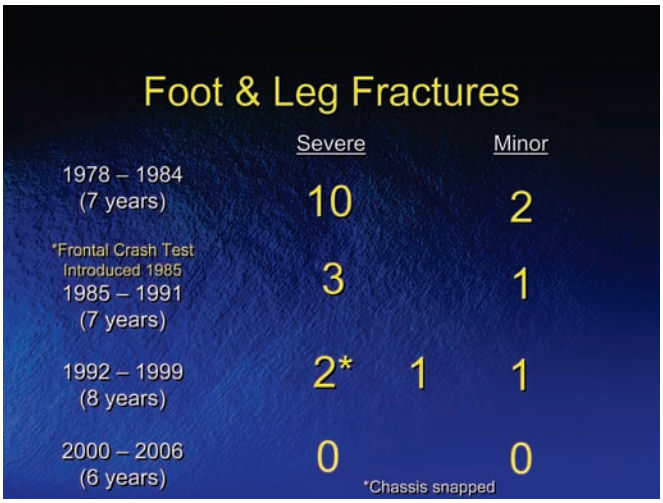
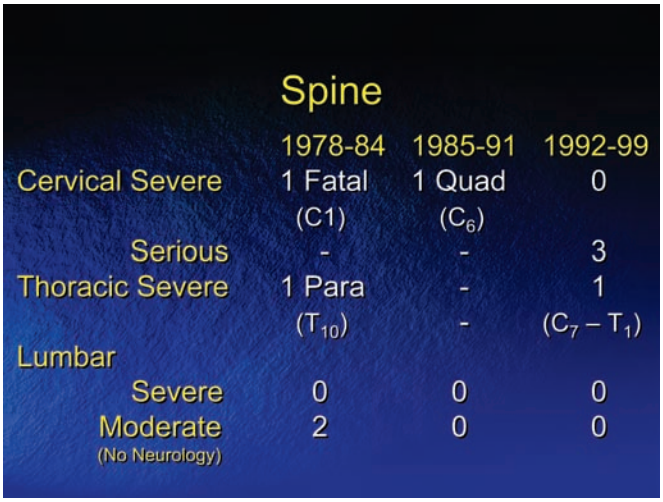


TABLE 14.2



competence suffer the least decrement—perhaps indicating why racing drivers of the highest calibre, Clark, Stewart, Lauda, Prost, Senna, and Michael Schumacher perform so well in adverse circumstance and climates hot or wet.

THE PATHOLOGY OF GRAND PRIX RACING

The following statistics relate to the incidence of injury and mortality rate between May 1978 and June 2006—during which time 450 races have been run.

In the years up to 1970, fire after a crash was a serious hazard. In 1970, safety bladder fuel tanks were introduced; in 1972, safety foam in the fuel tanks; and, in 1974, self-seal breakaway coupling for fuel lines became mandatory. Since May 1978, there has been only one moderate burn injury to

TABLE 14.3a.

TABLE 14.4

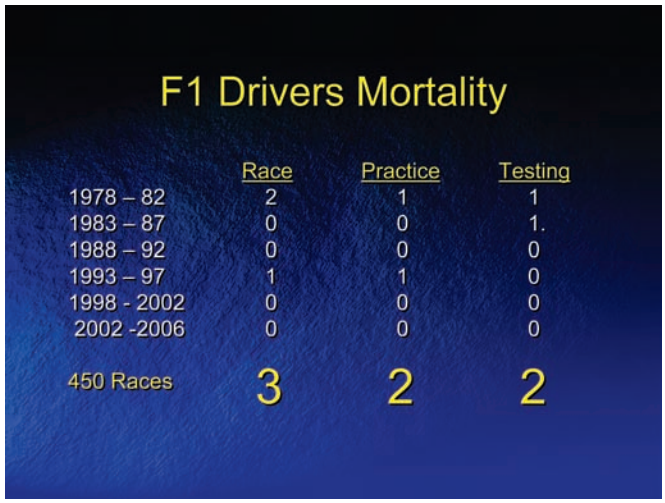
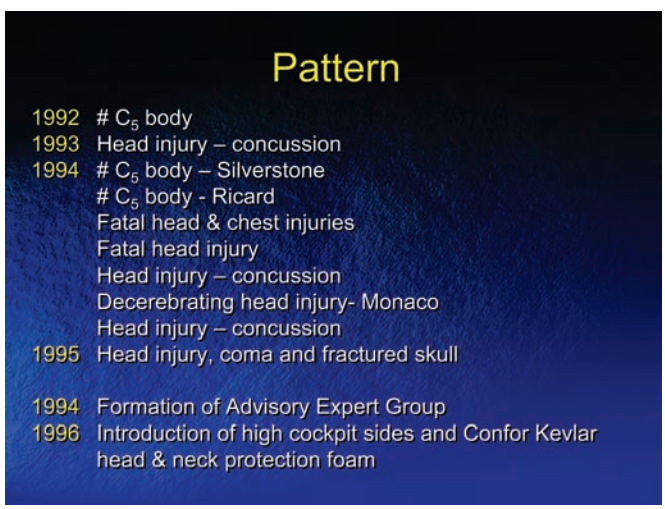


TABLE 14.3b

TABLE 14.5



the torso and two minor burns—to the hands in one case because of defective gloves and to the posterior neck skin in another driver who took his helmet off—with the rear placed engine on fire! Similarly injury to upper limbs has been rare, one case of compound metacarpal fracture and one of fracture of the radius and ulna. In the years 1978 to 1984, however, there were frequent injuries to the legs.

Table 14.1 shows the incidence through the years with a significant reduction in severe leg fractures after the introduction of the carbon fibre chassis, reinforcement of the survival cell to the feet in 1981 and a mandatory frontal crash test in 1985. Injury to the spine (*Table 14.2*) has dropped markedly with better body and neck restraint systems and the introduction of foam head and neck protection to cap the high cockpit sides introduced in 1996. These measures have protected against severe head injury and reduced the incidence of concussion (*Table 14.3*). Table 14.4 shows the mortality rates—there being no fatalities since May 1994 when, as was noted earlier, two drivers were lost in one weekend.

During the 28 years and 450 races and much official practice (in recent years, 4 hours each race) and in addition, private testing, there have been three deaths in races, two in official practice and two in private testing. This compares favorably with the mortality in Rugby—14 deaths in 25 years and approximately 150 spinal cord injuries with paralysis.

Reference has been made earlier to the loss of two drivers in one weekend in May 1994. Before this loss, since June 1982, there had been no fatal accident in virtually 12 years. However, in 1992, one driver had a compression fracture of the 5th cervical vertebra, and early in 1994, two

TABLE 14.6

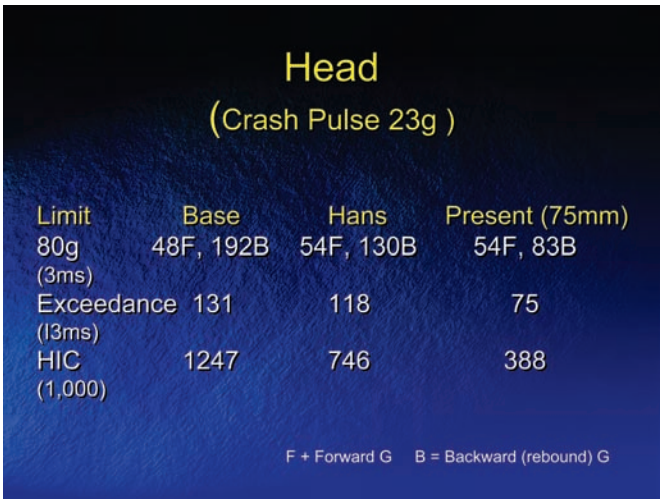


TABLE 14.8

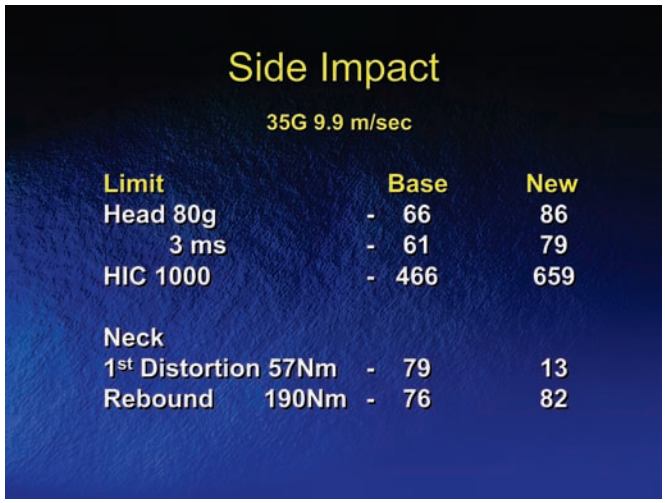


TABLE 14.7.

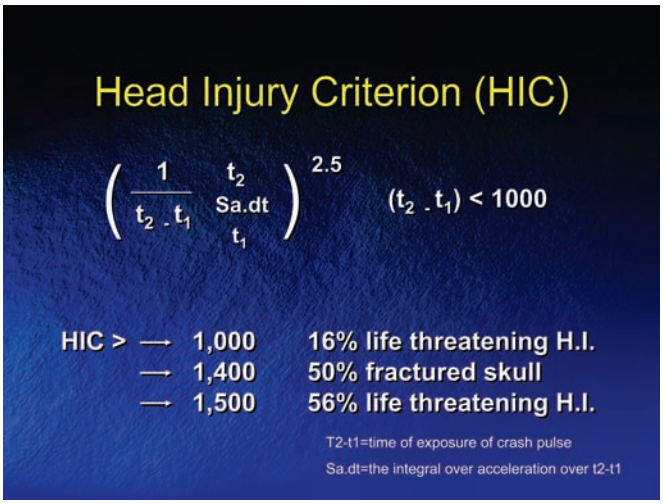
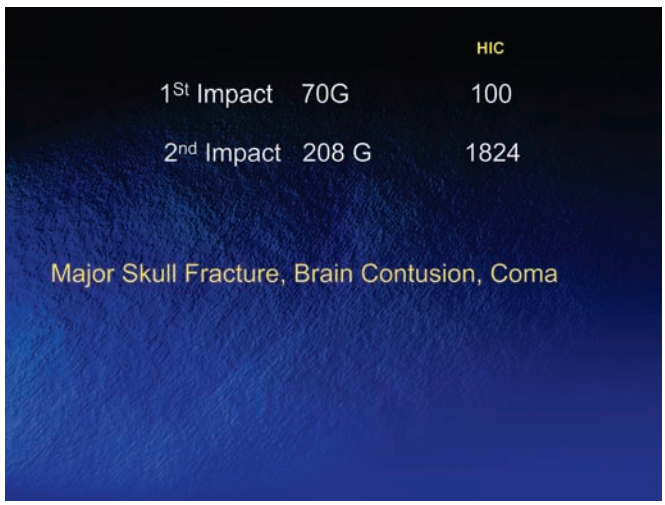


TABLE 14.9



drivers had compression fractures of again the 5th cervical vertebra. Subsequently, in May 1994, the two drivers killed died from head injury, one of whom also had a C7–T1 spinal fracture and chest injuries. At the following race in May, in practice, a driver sustained a decerebrating brain injury from striking his head against a protective barrier, but made a good recovery eventually. In the same period, two other drivers had concussion from striking their heads on the steering wheel. These events are summarized in *Table 14.5*. In response to the May fatalities and the decerebrating head injury, Max Mosley, President of the FIA, appointed an Expert Advisory Group to examine all aspects of safety—car and circuit design and barrier protection. Regulations were also introduced to reduce speed on the circuits, particularly at corners. The group consisted of expert engineers and designers, the FIA safety delegates and race directors, an eminent Formula One driver, and, ultimately, expert bioengineers from the Transport Research Laboratory (UK) and Daimler Mercedes Benz in Stuttgart. Research was immediately instituted with the Motor Industry Research Association at Nuneaton and crash testing with high-G sled tests started with instrumented sophisticated dummies (Hybrid III) using a McLaren Formula One chassis. In frontal, rear, side impact, and angled frontal and rear impacts, the response of the facsimile human form was cinephotographed at high speed, videotaped, and multiple accelerometers provided G force information sustained in these simulations in the head, neck, chest, and pelvis of the dummies. The results were fascinating, showing that the head and neck movement were remarkably excessive. In frontal impacts, the helmet of the dummy struck the steering wheel

TABLE 14.10a

Confor Headrest 2:

	G force	HIC	Rotation rads/sec/sec		Outcome AIS
2002	80	454	3724	1	1
2003	77	694	2538	2	2
2003	89	449	2430	2	2

TABLE 14.10b

Table 10

Confor Headrest 1 :

	G force	HIC	Rotation rads/sec/sec		Outcome AIS
1996	180	1980	5700	1	1
1997	105	693	4173	1	1
1998	126	578	2147	0	0
1998	116	694	2538	1	1
1999	122	1070	4021	0	0
1999	124	855	5276	1	1
1999	203	3343	6626	2	2
2001	243	4182	11570	2	2

TABLE 14.11

Correlation

Protection	Speed (km/hr.)	G >80-3ms	HIC 1,000	Rot^N 10,000	Result
Crash Helmet Only					
F_{3000} Wall	40	850	24,000	37,000	Fatal
F_1 Wall	160	500	8,500	34,000	Fatal
Porsche Rollcage (2 Impacts)	50	800 234	15,500 1,613	14,500	Vegetative state
F_1 Safety Barrier	80	360	5,000	34,000	Decerebrate Recovery
F_3 Cockpit	20	295	2,815	13,016	Coma Recovery
F_1 Cockpit 2 Impacts		70 208	100 1,824	6,000	Coma Recovery

TABLE 14.12

Abbreviated Injury Scale

Coding Injury Potential Against Peak G Value

AIS 0 = < 50g

AIS 1 = 50 – 100g

AIS 2 = 100-150 g

AIS 3= 150-200 g

AIS 4= 200-250 g

AIS 5 =250-300 g

AIS 6 = >300G

or the cockpit edge as the safety belts stretched, and then the head was thrown back on rebound to impact the chassis at the back of the cockpit. *Table 14.6* shows with a crash pulse of only 23G (speed 11.4 m/s), the head injury tolerance limit (80 g during a 3-ms duration) was exceeded on rebound by approximately 250%, reaching 192G. Correlations with previous experience in road accidents using the Head Injury Criterion (HIC) (*Table 14.7*) indicated at 192G a coefficient of 1247. As may be seen in *Table 14.8*, this could be expected to result in a life-threatening head injury in 16% of cases. The use of the Head and Neck Restraint alone (HANS) invented by Professor Hubbard of Michigan State University, would reduce the G force on rebound to 130G (HIC746) and, together with the Confor foam (75-mm thick) head and neck cockpit protection, to 83G (HIC388)—levels well below the injury tolerance level of HIC 1000. Early experiments increasing the width of the seat belts from 50 mm to 75 mm to spread the load on the chest immediately reduced the chest loads to 43G—below the injury tolerance level of 60G during 3 ms.

Cinephotography of the side impact tests with a crash pulse of 35G (speed 9.9 m/s) showed frightening lateral distortion of the neck. *Table 14.8* shows the head and neck loads, the primary neck distortion at 79 Nm exceeded the injury tolerance level of 57 Nm. The rebound level 76 Nm was well within the tolerance at 190 Nm. Introduction of high sides with Confor foam head and neck rest brought the primary distortion down to 13 Nm and the rebound still well within the tolerance level. The head G was increased somewhat, but with a HIC of only 659, safe.

This work was performed in the summer of 1994 and in 1995. In the last event of 1995, and before the high cockpit sides and foam protection was made mandatory at the start of 1996, a major and important accident occurred, and a driver sustained major skull fractures across the base of the cranium from mastoid process to mastoid process and through the pituitary fossa. He was immediately unconscious with bilateral facial palsies, and magnetic resonance imaging scan showed multiple brain contusions. The accident was replicated at the TRL by Chinn and Mellor, and this showed that two impacts had occurred. The first impact was with the side of the cockpit, at 70G with a HIC of 100, and a second impact fractured part of the steering wheel with an impact of 208G and a HIC of 1824 (*Table 14.9*). The driver was in a coma for 48 hours but made a slow but complete recovery after surgery to relieve compression of both facial nerves and to replace the ossicles that had dislocated. Since the high cockpit sides and foam were introduced, there have been no serious head injuries (*Table 14.10*), despite major accidents. However, as can be seen, only minor concussions occurred, even in high-speed accidents, which have been replicated by Mellor.[16] The replicated accidents with major head injuries in which the driver was protected only with a crash helmet are summarized in *Table 14.11*, together with the neurological outcome. The enormous improvement since the introduction of the Confor foam in 1996, and, more recently, the HANS in 2000, is obvious.

In 1997, Accident Data recorders (accelerometers x, y, z) were made compulsory to be fitted to the car to provide data for accident reconstruction. *Figure 14.10* shows such a recording, with the peak acceleration reaching 16G.

Using coding injury potential (Abbreviated Injury Scale [AIS]) against peak G values (*Table 14.12*) and the data obtained by accident replication, Mellor has produced correlations between linear and rotational acceleration and the AIS. These correlations are shown in *Figs. 14.11*, *14.12*, and *14.13*, and previous criteria for serious or fatal injuries by Newman,[17] Schuller,[18] Lowenheilm,[19] and Ryan et al.[20] are superimposed on the data obtained by the motor sport accident replication. From these studies, it would seem that the threshold for injury in linear accidents would be in these protected circumstances between 200G and 300G. In rotational accidents, the threshold seems to be 5000 rad/s/s. These results correlate well with the earlier studies.[17–20]

REFERENCES

1. Bertrand C, Keromes A, Lemeunier BF, Meistelmann C, Prieur C, Richalet JP: *Physiologie des Sports Mécaniques*. 1st International Congress of Sport Automobile, Marseilles, 1983.
2. Richalet JP, Bertrand C: *Evaluation and Perspectives of Medical Study on Race Car Drivers*. Créteil, Paris, 1983.
3. Richalet JP, Bertrand C: *Aspect Physiologique de la Compétition Automobile*. Créteil, Paris, 1983.
4. Ladell WS, Watkins ES: Prediction of body temperature from heart rate. **J Physiol** 135:51, 1956.
5. Dallaire J, Marisi DQ, Fagnan R: *Reports of Motor Sport Research Group*. McGill University, Montreal, 1986.
6. Naccarella F, Nanni G, Savoia M, Barbato G, Accorsi A, Caldarelli P, Bertacinni P, Martuzzi M, Brachetti D: *Cardiovascular and Holter Monitoring and Laboristic Evaluation of Fluid, Electrolytes and Stress Hormone Response to Prolonged Rally Road Racing*. Bologna, 1983.
7. Naccarella F, Nanni G, Savoia M, Accorsi A, Caldarelli P, Venturoli MG, Bertacinni P, Bertacinni B, Martuzzi M, Brachetti D: *Cardiovascular and Holter Monitoring and Assessment of Fluid, Electrolytes and Hormones Response Elicited by Psycho-physical Stress of Prolonged Rally Road Racing*. Bologna, 1987.
8. Communiqué issued by Isserman JJ. FISA Medical Commission, 1982.
9. Burton AK: Back pain in Grand Prix drivers. **Brit J Sports Medicine** 17:150, 1983.
10. Burton AK, Sandover J: Back pain in Grand Prix drivers; a "found" experiment. **Applied Ergonomics** 18:1, 1987.
11. Blockley WV, McCutcham JW, Craig LT: *Prediction of Human Tolerance for Heat in Aircraft*. Wright Air Development Centre Technical Report, 53:346, 1954.
12. Blockley WV, Lyman J: *Psychomotor Performance of Pilots as indicated by a Task Simulating Aircraft Instrument Flight*. Air Force Technical Report, 6521, Wright Patterson Air Force Base, 1951.
13. Blockley WV, Lyman J: *Mental Performance under Heat Stress as indicated by Addition and Number Checking Tests*. Air Force Technical Report 6022, Wright Patterson Air Force Base, 1950.
14. Mackworth NH: *Researches on the Measurement of Human Performance*. Medical Research Council Special Report, no.268, 1950.
15. Watkins ES: The effect of heat on psychomotor efficiency with particular reference to tropical man. Liverpool, University of Liverpool, and reports to Medical Research Council, West Africa, 1956 (dissertation).
16. Mellor A: F1 Accident Investigation SAE00MSV-37, 2000.
17. Newman J: Biomechanical Engineering, USA, 1999.
18. Schuller E: University of Munich, Germany, 2000.
19. Lowenheilm P: University of Lundt, Sweden, 1998.
20. Ryan GA: RAR Unit Adelaide, 1999.