Musculoskeletal decompression sickness and risk of dysbaric osteonecrosis in recreational divers

Emmanuel Gempp, Jean-Eric Blatteau, Olivier Simon and Eric Stephant

Key words
Diving, decompression sickness, dysbaric osteonecrosis, magnetic resonance imaging (MRI)

Abstract
(Gempp E, Blatteau J-E, Simon O, Stephant E. Musculoskeletal decompression sickness and risk of dysbaric osteonecrosis in recreational divers. *Diving and Hyperbaric Medicine*. 2009;39(4):200-4.)
Introduction: Dysbaric osteonecrosis (DON) is a complication that usually occurs in professional divers or compressed-air workers. Its correlation with a previous musculoskeletal decompression injury (i.e., 'limb bend') remains a controversial subject. There is little information about the prevalence of DON and its relationship to decompression sickness (DCS) in recreational divers.
Methods: We undertook an observational, retrospective study of recreational divers treated for musculoskeletal DCS between 2004 and 2008 in three hyperbaric centres in the south of France using magnetic resonance imaging (MRI) following hyperbaric treatment.
Results: Twenty-five (11.5%) musculoskeletal DCS cases were identified amongst 288 diving accidents treated during this period. Average age was 38 years with a mean body mass index of 26 $kg.m^{-2}$. Joint pains were located in the shoulder area in 21 divers, mainly in experienced male divers after performing repetitive long, deep dives with adequate decompression using dive computers. Twenty-one of 25 injured divers were examined by MRI of the affected area shortly after the accident. Six had initial humeral lesions compatible with ischaemic necrosis, but in two repeat MRI examinations at three months did not reveal bone abnormalities. Increasing pain during hyperbaric treatment appeared to be the only factor associated with DON occurrence.
Conclusions: Musculoskeletal DCS in recreational diving is particularly seen in provocative dive profiles considered to carry a high risk for bubble production during decompression. The occurrence of this insult appears also to be related to other factors needing further study. The risk of early development of DON should not be ignored.

Introduction

The reported prevalence of musculoskeletal decompression sickness (DCS) or 'limb bends' (previously, type 1 DCS) in scuba divers varies from 3 to 31%,[1–3] mainly because of the different diving populations examined (recreational, commercial or military divers) or the decompression procedure used (computer-generated versus decompression table). Dysbaric osteonecrosis (DON) is a potentially disabling condition resulting in osteoarthritic changes when bone necrosis is juxta-articular. This pathological event usually occurs in professional divers or compressed-air workers exposed to iterative high ambient pressure, but has also been reported to appear in recreational scuba divers.[4,5] It has been reported that diving could lead to deterioration in pre-existing DON lesions, thus requiring that divers who have DON should be followed up frequently or excluded from diving.[6] In certain circumstances, DON may be considered as a late manifestation of a previous musculoskeletal DCS injury. However, despite the observed link between the two illnesses, their correlation remains controversial.[7–11] There is a general consensus that gas bubble formation during decompression is the primary cause of DCS and DON. Most hypotheses focus on an autochthonous bubble mechanism but there is no agreement on the actual site as far as DCS development is concerned, and it is unclear where, or how, bubbles form in the bone marrow cavity causing DON.

Magnetic resonance imaging (MRI) is a highly sensitive technique to detect early signs of DON.[12,13] However, there are no pathognomonic MRI findings specific to DON compared to osteonecrosis by other mechanisms. This technique can be used to demonstrate bone marrow oedema on fat-suppressed T2-weighted images and, subsequently, the classic necrotic area delineated by a hypointense signal line on T1- and T2-weighted sequences as described by Mitchell et al.[14] These imaging examinations have great prognostic value in determining whether the spherical shape will collapse or not.

To date, there are no data on the prevalence of DON after development of musculoskeletal DCS in recreational divers. This study was designed to determine the main predisposing factors of bends occurrence in this population and the proportion of DON after hyperbaric treatment by use of MRI of the affected site.

Materials and methods

We reviewed the clinical and diving data on scuba divers presenting between November 2004 and October 2008 with symptoms indicative of musculoskeletal DCS in three hyperbaric centres in the south of France (two in Toulon and one in Nice). Information obtained from the medical records included anthropometric data, history of previous

musculoskeletal DCS, diving experience (number of dives), parameters of diving exposure (maximum depth, total dive time and decompression schedule), delay from surfacing to first symptom occurrence, time to recompression and resolution or worsening of pain during hyperbaric treatment. A questionnaire was also used to define past medical history, alcohol and drug consumption. The study was approved by the hospital ethics committee.

The clinical diagnosis of musculoskeletal DCS was made when the criteria of joint pain, accompanied by myalgia and numbness, were recognized after the diver surfaced. Divers with symptoms suggesting neurological DCS (e.g., paraesthesia, motor impairment) were excluded after careful examination by the duty diving physician.

The follow up of injured divers was routinely performed by initial MRI of the affected site between one and 30 days after the insult, except in five divers who were not investigated until between four weeks and three months after the insult. Repeat MRI was performed between three and four months in divers in whom abnormalities were detected on the first MRI sequences. MRI examinations were performed on 1.5-Tesla MR units and consisted of T1- and T2-weighted images in the coronal and sagittal planes. MR images were evaluated initially by several radiologists from different imaging departments but subsequently reviewed by one of the authors (ES) trained in reading bone MRI. Imaging criteria to identify DON development at different stages were based on the staging system described by Mitchell et al.[14]

Although the data analysis planned was mainly descriptive, a stepwise multiple regression analysis was used to identify potential predictors of DON. Additional analysis to compare time to treatment between injured divers with and without bone lesions was performed using the Mann Whitney U test. A value of $P < 0.05$ was considered significant. Calculations were computed using Sigmastat 3.0 software program (SYSTAT Inc., Richmond, CA). All parametric data are presented as mean ± SD and non-parametric data as median and range.

Results

Twenty-five (11.5%) cases of musculoskeletal DCS were reported amongst 288 injured divers (58% neurological DCI, 21.2% inner ear DCS and 13.2% miscellaneous presentations) treated during the study period. However, only 21 divers (20 men and one woman) were retained for analysis after MRI examination (four patients missed). Age was 38 ± 8.4 years and BMI 26 ± 3 kg.m^2. Five of these divers had a history of previous limb bends. Close questioning did not reveal other identified causes of aseptic bone necrosis (e.g., trauma, coagulopathy, corticosteroids, alcoholism).

Diving profiles were as follows: maximum depth 45 ± 18 metres' sea water and bottom time 40 ± 16 min. A repetitive dive was recorded in 10 cases and no diver performed an inadequate decompression procedure (i.e., fast ascent or omitted decompression stops according to their dive computers). Physical exercise with excessive use of limbs was observed in only two divers. The breathing mixture was air except for one case where trimix (nitrogen 41%, helium 41% and oxygen 18%) was used.

The most frequently affected site was the shoulder (18 out of 21 divers) and both sides were equally affected (ten left versus eight right). Two limb bends were located in the elbow and one case involved the ankle. The median time from surfacing to the onset of initial symptoms was 10 min (range 5–600 min), and the median delay to recompression was seven hours (range 2–40 h). All patients underwent a single hyperbaric oxygen treatment (100% oxygen breathing at 253 or 283 kPa for 70–150 min), and no extensions or repeat treatments were given. Pain was usually fully relieved at the end of the treatment, but in seven divers the symptoms increased during recompression and continued to exacerbate while at depth, requiring intravenous therapy with non-steroidal anti-inflammatories. It is important to state that each of them was recompressed with a therapeutic table using 100% oxygen at 283 kPa for 150 min, equivalent to USN table 5, according to the general recommendations.[15]

MRI evaluation revealed a total of six out of 21 divers with juxta-articular humeral lesions consistent with DON in the same area as joint pain had occurred. Of these, two divers

Figure 1
T1-weighted coronal image from MRI at three months shows humeral diaphysal serpiginous lines specific to ischaemic necrosis

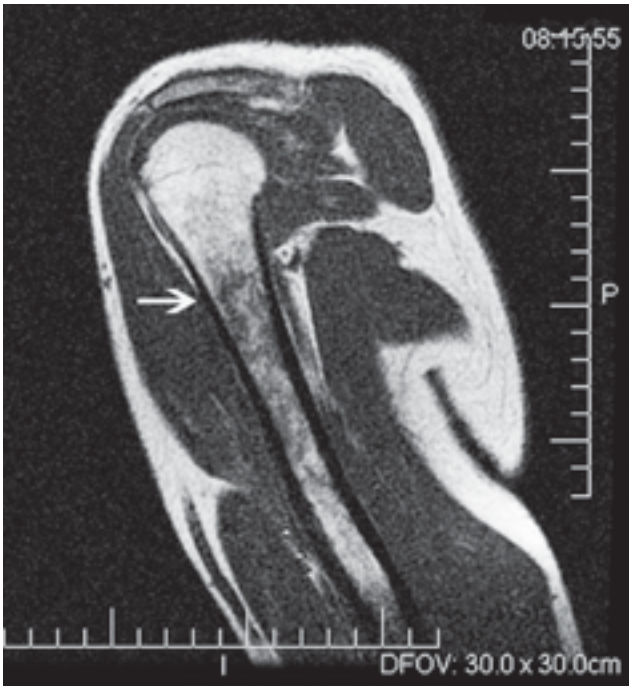
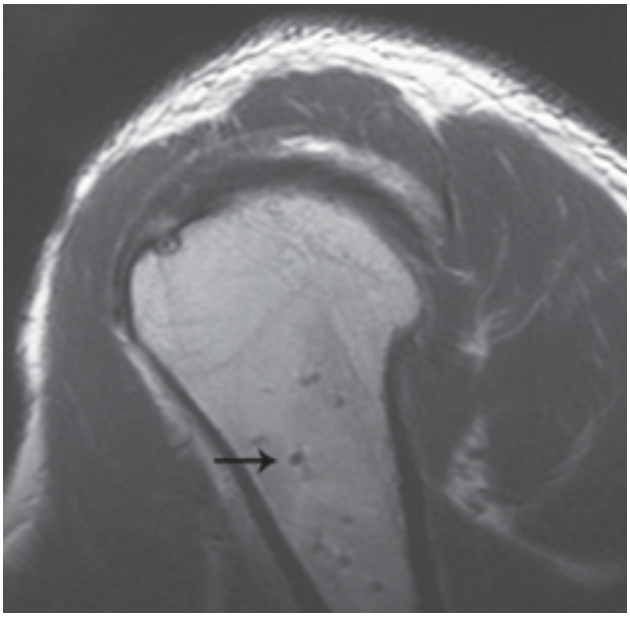


Table 1
Analysis of MRI outcome in 21 divers with musculoskeletal DCS according to diving data, clinical characteristics and time to recompression; MRI + indicates the presence of dysbaric osteonecrosis lesions; OR (95% CI) – odds ratio and 95% confidence intervals

Variable	MRI +	MRI -	*P* value	OR (95% CI)
Age (yr)				
≤ 40	1	9	0.15	7.5
> 40	5	6		(0.7,81.2)
BMI (kg.m^{-2})				
≤ 27	4	12	0.60	2
> 27	2	3		(0.2,16.6)
Diving experience (no of dives)				
≤ 200	1	2	1	1.3
> 200	5	13		(0.1,17.3)
History of DCS				
yes	2	3	0.60	2
no	4	12		(0.2,16.6)
Dive time (min)				
≤ 40	2	9	0.36	3
> 40	4	6		(0.4,21.8)
Depth (msw)				
≤ 45	4	8	0.66	1.7
> 45	2	7		(0.2,12.6)
Repetitive dive				
yes	3	7	1	1.1
no	3	8		(0.1,7.6)
Delay to onset of symptoms (min)				
≤ 30	4	9	1	1.3
> 30	2	6		(0.2,9.7)
Delay to treatment (h)				
≤ 6	1	9	0.15	7.5
> 6	5	6		(0.7,81.2)
Paradoxical pain				
yes	6	1	< 0.001	NA
no	0	14		

had MRI features of advanced metaphysal and diaphysal ischaemic necrosis (two and three months after the insult, respectively) while the four other cases presented findings suggesting bone marrow oedema on MR images initially performed between 24 h and 3 days following the accident. In the latter cases, when re-examined 3–4 months thereafter, the MRI scans showed significant metaphysal and diaphysal bone infarction concordant with definite lesions in two divers while the initial abnormalities detected in the remaining two divers had disappeared. Two example MRI slices with diaphyseal anomalies are shown in Figures 1 and 2.

Figure 2
T2-weighted sagittal image revealing multiple unexpected hypo-intense spots in the humeral marrow strongly evocative of bubbles (MRI examination 24 hrs following DCS)



Results of univariate analysis are presented in Table 1. Paradoxical pain, which continues to increase while at depth, was found to be the only significant variable associated with the development of ischaemic lesions seen on MRI ($P < 0.001$), and remained the only independent variable on multivariate analysis ($P < 0.001$). Moreover, the delay between onset of symptoms and hyperbaric treatment was not statistically different in divers without DON (median, 4.5 hrs) when compared with divers with DON (median, 8.0 hrs) ($P = 0.13$).

Discussion

The 11.5% prevalence of limb bends in divers presenting with DCS in the south of France is lower than epidemiological data from DAN reports.[2] The main reason is that DAN findings are not drawn from the treating diving physician but are completed by the patient or a health care professional after the DCS event, thus limiting the accuracy of recorded manifestations in this database.

Our results show that musculoskeletal DCS affected mainly experienced, male divers after performing repetitive, long, deep dives with adequate decompression schedule using dive computers. Occurrence did not appear to be related to some individual factors such as excess weight. However, it is noteworthy that the intensity of physical exercise on the bottom, that was previously thought to be a risk factor for DCS development, was uncommon in this study.[16] Similar

results were also demonstrated in a report of 58 recreational divers with DCS.[17]

The distribution of pain indicates that, in almost all cases, the shoulder was the predominant site, as already observed in bounce diving.[17] This difference from compressed-air workers or saturation divers, who experience a higher proportion of musculoskeletal DCS in the lower limbs,[18] has no obvious explanation. One possibility is the gravitational force between the upper and lower extremities, which causes pooling of blood at the bottom (and consequently alters blood circulation and nitrogen elimination), in the case of dry dives or when workers spend longer hours working in a standing position. The symptom latency after dive completion shows that bends presented soon after surfacing (60% within 30 min), but with onset being reported 12 hours after the dive in three cases, supporting findings from previous reports.[16,17]

In the present study, the 28% proportion of early DON lesions detected with MRI and the 19% prevalence of definite ischaemic necrosis in our cohort of musculoskeletal DCS divers is higher than expected since recreational divers are supposed to perform dives with conservative exposure. Unfortunately, the increase in number of scuba divers during the last decade who go deeper, for longer, and use gas mixtures containing helium implies that this population will probably be at greater risk to develop DON in the future, on a level similar to professional divers and caisson workers. The reported prevalence of DON ranges from 0–4% in military divers to 50% in native diving fishermen, and even 70% in Turkish sponge divers.[7,8,12,19,20] These varying rates can be explained by different and often poor decompression practices, the lack of recruitment standards and periodic medical examination and the presence of predisposing factors for avascular necrosis (e.g., alcohol intake, hyperlipidaemia) in the latter groups. Nearly 30% of professional divers who have had a history of limb DCS have been reported to have subsequent bone lesions.[10,11] The present study is the first to analyse the association between musculoskeletal DCS, early bone marrow damage and DON development in recreational divers. Recently, we have described two divers in whom MRI examination performed 24 hours after HBO treatment for a painful shoulder following scuba air dives showed multiple micro-cavities in the fatty marrow cavity highly consistent with bubble formation (Figure 2). Bone scintigraphy obtained the day after confirmed the hypovascularization of the affected area and, six months later, control MRI revealed extensive DON in both divers.[21]

The proposed mechanism linking DCS and DON is based on the hypothesis of elevated intramedullary pressure resulting from bubble formation in the marrow cavity during decompression. Expansion of bubbles in the fatty tissue of bone may be responsible for pain by irritating nerve endings located in marrow sinusoids or near the periosteum, but may also contribute to the reduction of blood flow, with resultant vascular stasis, ischaemia and 'compartment syndrome' of bone. If high intramedullary pressure is sufficiently prolonged death of both marrow and calcified bone may occur.[22] However, it is thought that bubble formation is not sufficient to cause DON and that there may be some other predisposing factors for ischaemic bone necrosis, such as fat embolism, hyperoxia and hypoxia, coagulation abnormalities and rapid rates of compression.[23–26]

Interestingly, we have noticed that divers who presented with bone infarction often complained of increased pain during hyperbaric treatment.[21] Statistical analysis confirmed this impression and this was the only predictor of ischaemic abnormalities detected with MRI in this series of divers. We propose that the rapid compression induced a rise in intra-osseous pressure in the rigid marrow cavity previously altered by intramedullary bubbles, thus producing subsequent ischaemic pain in the affected area. Expansion of gas bubbles during the initial phase of oxygen recompression might also contribute, as demonstrated experimentally.[27] This raises the question of whether prompt hyperbaric treatment is beneficial or deleterious.

The average time to recompression was 8.0 h in the DON group versus 4.5 h in the non-DON group. Although not statistically different in this small study population, the increased delay could be another contributory factor; prompt recompression has been reported to prevent DON occurrence after musculoskeletal DCS using a sheep model.[28,29] However, in those studies, hyperbaric exposure was prolonged, similar to saturation dive with provocative decompression, excluding a possible comparison with our data.

Our results are limited by the lack of statistical power of this preliminary cross-sectional study due to the small sample size of divers with musculoskeletal DCS. Another limitation is a possible selection bias with respect to including more serious cases of bends only (i.e., that divers with mild symptoms did not present for treatment) and with respect to MRI examinations (prevalence of DON lesions in musculoskeletal DCS under reported due to the loss of four patients to MRI follow up). Further work including more data from additional hyperbaric facilities is needed to confirm the present findings.

Conclusion

Musculoskeletal DCS in recreational diving was seen following dives considered to carry a high risk for bubble production. The prevalence of definite DON was 19% in our cohort, suggesting that MRI for routine screening is justified in recreational divers treated for musculoskeletal DCS before they return to diving. Increasing pain during hyperbaric treatment should be considered as the intra-osseous manifestation of limb bends, requiring early MRI

examination of the affected area post treatment. However, the benefit of MRI in detecting initial bone marrow lesions before conversion into subsequent osteonecrosis has not been determined. Iterative sessions of hyperbaric oxygen could limit ischaemic necrosis as suggested in a recent pilot study dealing with early-stage avascular necrosis of the femoral head,[30] but the possibility that prompt recompression could worsen the initial damage remains debatable.

References

1 Méliet JL. [Study of 47 cases of osteo-arthro-muscular decompression sickness.] *Bulletin Medecine Subaquatique Hyperbare*. 1983;2:1724. French.

2 Divers Alert Network. *Report on decompression illness and diving fatalities*. Durham, NC: Divers Alert Network; 2005.

3 Decompression Sickness Central Registry and Radiological Panel. Aseptic bone necrosis in divers. *Lancet*. 1981;2:384-8.

4 Laden GD, Grout P. Aseptic bone necrosis in an amateur scuba diver. *Br J Sports Med*. 2004;38(5):E19.

5 Wilmshurst P, Kenneth R. Dysbaric osteonecrosis of the shoulder in a sport scuba diver. *Br J Sports Med*. 1998;32:344-5.

6 Toklu SA, Cimsit M, Kolat A. Does diving deteriorate preexisting dysbaric osteonecrosis lesions? *European Journal of Underwater and Hyperbaric Medicine*. 2007;8:36.

7 Otah Y, Matsunaga H. Bone lesions in divers. *J Bone Jt Surg*. 1974;56B:3-16.

8 Toklu AS, Cimsit M. Dysbaric osteonecrosis in Turkish sponge divers. *Undersea Hyperb Med*. 2001;28:83-8.

9 Cimsit M, Ilgezdi S, Cimsit C, Uzun G. Dysbaric osteonecrosis in experienced dive masters and instructors. *Aviat Space Environ Med*. 2007;78:1150-4.

10 Yangsheng T, Anquan Y, Weimin L, Jingxi Q. Investigation and analysis of dysbaric osteonecrosis in 171 divers. *Journal of Hyperbaric Medicine*. 1991;7:123-6.

11 Zhang H, Li JS, Han CY, Gou J. Dysbaric osteonecrosis in divers. *Journal of Hyperbaric Medicine*. 1991;6:183-8.

12 Bolte H, Koch A, Tetzlaff K, Bettinghausen E, Heller M, Reuter M. Detection of dysbaric osteonecrosis in military divers using magnetic resonance imaging. *Eur Radiol*. 2005;15:368-75.

13 Shinoda S, Hasegawa Y, Kawasaki S, Tagawa N, Iwata H. Magnetic resonance imaging of osteonecrosis in divers: comparison with plain radiographs. *Skeletal Radiol*. 1997;26:354-9.

14 Mitchell DG, Rao VM, Dalinka MK. Radiology femoral head avascular necrosis: correlation of MR imaging, radiographic staging, radionuclide imaging, and clinical findings. *Radiology*. 1987;162:709-15.

15 *US Navy diving manual*. Naval Sea Systems Command; 2005, Vol 5, chap 20. p. 13-14.

16 Rivera JC. Decompression sickness among divers: an analysis of 935 cases. *Milit Med*. 1964;129:134-334.

17 [Kauert A, Wolkiewiez J. Time of appearance and localisation of arthrological bends. From 58 diving decompression sickness. *Bulletin Medecine Subaquatique Hyperbare*. 1992; 2:129-36.] French

18 Francis TJR, Dutka AJ, Hallenbeck JM. Pathophysiology of decompression sickness. In: Bove AA, Davis JC, editors. *Diving medicine*. London: WB Saunders;1990. p.170-86.

19 Uzun G, Toklu AS, Yildiz S, Sonmez G, Aktas S, Sezer H, et al. Dysbaric osteonecrosis screening in Turkish navy divers. *Aviat Space Environ Med*. 2008;79:44-6.

20 Hunter WL, Biersner RJ, Sphar RL, Harvey CA. Aseptic bone necrosis among US Navy divers: survey of 934 nonrandomly selected personnel. *Undersea Biomed Res*. 1978;5:25-36.

21 Stephant E, Gempp E, Blatteau JE. Role of MRI in the detection of marrow bubbles after musculoskeletal decompression sickness predictive of subsequent dysbaric osteonecrosis. *Clin Radiol*. 2008;63:1380-3.

22 Lanphier EH, Lehner CE, Lemos SE. The role of intramedullary pressure in hyperbaric osteonecrosis. In: Jardine FM, Mc Callum RI, editors. *Engineering and health in compressed air work*. London: E & FN Spon; 1994. p. 148-55.

23 Jones JP. Fat embolism, intravascular coagulation and osteonecrosis. *Clin Orthop*. 1993;292:294-308.

24 Gregg PJ, Walder DN. Caisson disease of bone. *Clin Orthop*. 1986;210:43-54.

25 Miyanishi K, Kamo Y, Ihara H, Naka T, Hirakawa M, Sugioka Y. Risk factors for dysbaric osteonecrosis. *Rheumatology*. 2006;45:855-8.

26 Hutter CD. Dysbaric osteonecrosis: a reassessment and hypothesis. *Med Hypotheses*. 2000;54:585-90.

27 Hyldegaard O, Madsen J. Effect of air, heliox, and oxygen breathing on air bubbles in aqueous tissues in the rat. *Undersea Hyperb Med*. 1994;21:413-24.

28 Lehner CE, Sobakin AS, Abraham JL, Dueland RT, Wilson MA, Gendron-Fitzpatrick AP, et al. Investigation of delayed recompression treatment for limiting the induction of dysbaric osteonecrosis: sheep model of the diver [abstract]. *Undersea Hyperb Med*. 2007;34(4):267.

29 Lin TF, Lehner CE, Lanphier EH, Wilson MA, Markel MD, Dueland R. Prompt recompression of sheep with limb bends prevents the development of dysbaric osteonecrosis [abstract]. *Undersea Hyperb Med*. 1993;20(4):E6.

30 Reis ND, Schwartz O, Militianu D, Ramon Y, Levin D, Norman D, et al. Hyperbaric oxygen therapy as a treatment for stage-I avascular necrosis of the femoral head. *J Bone Surg Br*. 2003;85:3715.

Submitted: 02 July 2009
Accepted: 03 August 2009

Emmanuel Gempp, MD, and Jean-Eric Blatteau, MD, PhD, are consultants in the Department of Diving and Hyperbaric Medicine, St Anne's Military Hospital, Toulon.
Olivier Simon, MD, works in the Department of Hyperbaric Medicine, University Hospital, Nice, and
Eric Stephant, MD, is a radiologist in the Department of Radiology, St Anne's Military Hospital, Toulon.

Address for correspondence:
Emmanuel Gempp
Department of Diving and Hyperbaric Medicine, Ste Anne's Military Hospital,
BP 20545, 83041 Toulon cedex 9, France
E-mail: *<gempp@voila.fr>*