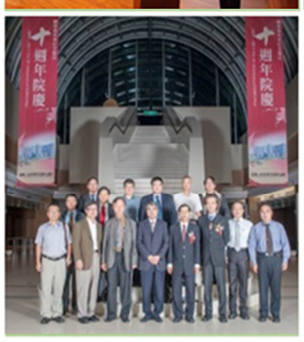
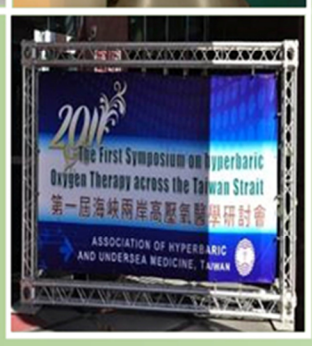
中華民國高壓暨海底醫學會

2021

電子壁報論文展示

活動名稱:110 年年會暨學術研討會

展示時間:中華民國 110 年 11 月 14 日

主辦單位:中華民國高壓暨海底醫學會

中國醫藥大學附設醫院 高壓氧中心

國軍高雄總醫院
Kaohsiung Armed Forces General Hospital

電子壁報-P01

急性一氧化碳中毒所引起之突發性聽力喪失：病例報告及文獻回顧

Sudden hearing loss following acute carbon monoxide poisoning: A case report and literature review

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Case presentation

A 42-year-old woman came to our emergency department with symptoms of **hearing loss along with dizziness, nausea, and chest tightness for few hours**. She complained of **hearing loss in the left ear and tinnitus in the right ear**. She had a history of smoking over 10 years with daily consumption of 10 cigarettes. She was apparently healthy with no history of hearing loss in the past. Initially, **Meniere's disease was diagnosed**. **However, after a thorough history session, she reported that her husband was found unconscious from carbon monoxide poisoning associated with home fuel-burning heating equipment when she came home in the morning**. She started to feel discomfort after she had completed the admission program for her husband.

Her vital signs was within normal range. Her Glasgow Coma Scale was 15. The neurological examination was unremarkable. We also checked the level of cardiac enzyme and carboxyhemoglobin (COHb) due to chest pain and exposure to CO. **Elevated levels of troponin I of 0.5 μg/L (normal range, 0~0.3μg/L)**, creatine kinase-MB fraction of 60 μg/L (nor- mal range, 0~20 μg/L), **and COHb of 12%** were noted after termination of exposure without oxygen supplement for about four hours. Chest X-ray imaging showed a small focal consolidation on the lower right lobe.

Due to the elevated COHb level and sudden hearing loss on the admission day, the patient was **referred to the hyperbaric therapeutic unit and underwent a session of oxygen therapy at 3 atmosphere absolutes (ATA) and 90-min duration**. Thereafter, an audiometric test was performed, which showed bilateral asymmetrical mid- to high-frequency hearing loss. Bone conduction thresholds inter- weaved with air conduction thresholds indicating the impairment to be sensorineural in origin bilaterally. **A tapered course of intravenous corticoid treatment was prescribed (methylprednisolone at a dose of 1 mg/kg/day) for 2 weeks**. And the patient was subjected **to a total of 10 sessions of hyperbaric oxygen (HBO) therapy at 3 ATA of 90 min duration each**. About 2 weeks later, when the HBO therapy was finished, the patient reported improvement in hearing loss and subsiding of tinnitus. We repeated the audiometric test, which revealed **a partial recovery of hearing loss.**

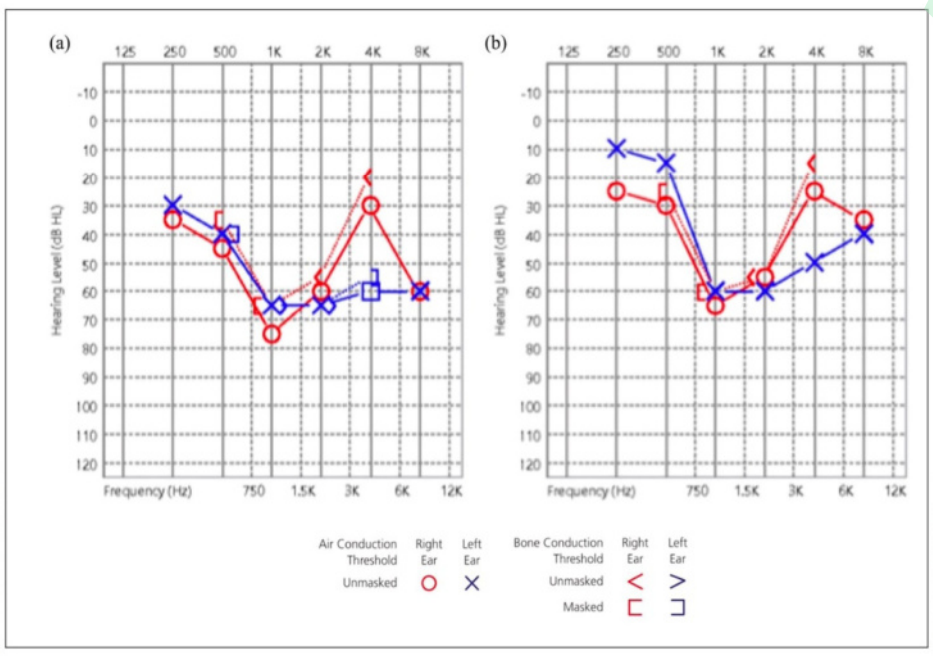

Figure 1. Patient's audiometric test (a) on the day of admission and (b) after 10 sessions of hyperbaric oxygen (HBO) therapy.

Table 1. Summary of case reports of sudden hearing loss following acute CO poisoning.

Authors	Age (years)/ sex	Symptoms at presentation	COHb level (%)	Affected frequencies of Pure-tone audiometric test	Treatment	Outcome
Morris[7]	22/M	Unconscious	25	125–8000 Hz bilaterally	NR	Partial recovery after 4 weeks
Baker[8]	16/F	Unconscious	NR	500–4000 Hz bilaterally	Hypothermia, 100% oxygen, IV steroids	Partial recovery after 8 months
Hassan et al.[4]	30/M	Unconscious	6.6	1000–8000 Hz bilaterally	100% oxygen	Partial recovery after 2 months
Razzaq et al.[9]	53/M	Sudden deafness, disoriented, dull bifrontal headache, ringing in the ears	NR	250–8000 Hz bilaterally	NR	Good recovery after 3 months
Pillion[10]	17/M	Unconscious	NR	1500–8000 Hz bilaterally	One HBO session on admission day with unknown pressure and duration	No significant changes after 1 month
Mehrparvar et al.[11]	22/M	Unconscious	NR	1000–8000 Hz bilaterally	100% oxygen	No significant changes after 3 months
Bonfiglioli et al.[5]	37/F	Malaise, visual impairment, dizziness, lost consciousness	6.9	250–8000 Hz bilaterally	Methylprednisolone for 1 week, 25 HBO sessions (2.5 ATA, 90 min)	Good recovery after 5 months
Costa et al.[3]	49/M	Psychomotor restlessness	NR	500–8000 Hz in the right ear	IV methylprednisolone (1 mg/kg/day) for 10 days, 30 HBO sessions (unknown pressure, 60 min)	Partial recovery after 3 months
Marcano et al.[6]	16/F	Unconscious	42.4	125–8000 Hz bilaterally	100% oxygen, IV methylprednisolone (1 mg/kg/day) for 14 days, 10 HBO sessions (2.4ATA, 90 min), oral steroid for 1 month	Good recovery after 1 month
Present case	42/F	Hearing loss, tinnitus, dizziness, nausea, chest tightness	12	500–8000 Hz bilaterally	100% oxygen, IV methylprednisolone (1 mg/kg/day) for 14 days, 10 HBO sessions (3.0ATA, 90 min)	Partial recovery after 2 weeks

CO: carbon monoxide; COHb: carboxyhemoglobin; M: male; NR: not reported; F: female; HBO: hyperbaric oxygen; ATA: atmosphere absolute; IV: intravenous.

Discussion

CO poisoning is ideally diagnosed by a clinical triad: symptoms consistent with CO poisoning, history of recent CO exposure, and elevated COHb levels. However, these criteria are not strict and the most important one is the exposure history.

The characteristics of CO-induced hearing loss can be concluded in three points: **(1)high-frequency deficit (due to hypoxic damage to the base of the cochlea)**, **(2)bilateral, and (3)reversible** as presentation of our case.

Nevertheless, although rare, a low-frequency deficit, unilateral hearing loss, and an irreversible condition have also been reported, **audiometry of which may present a "U-shaped" curve, also called a "cookie-bite**."

If a patient is diagnosed with hearing loss caused by acute CO poisoning, the subsequent management we suggested should be done as below: **(1) termination of exposure of CO**; **(2) monitoring for any cardiac, neurological, and pulmonary complications**; **(3) corticosteroid therapy**; and **(4) HBO therapy**. Chronic exposure to CO could lead to a poor prognosis.

Conclusion

If there is no common etiology in patients with sudden hearing loss, ototoxic substances, especially CO poisoning in the working/home environment should be considered as a diagnostic possibility. As evident from the summary of current literature available , it is difficult to establish associations between outcome, age, symptoms, COHb level, and treatment. Therefore, more studies are needed to determine the best clinical guidelines for CO poisoning. HBO and corticosteroid therapy are treatment options for sudden hearing loss following acute CO poisoning. Nevertheless, the treatment efficacy was not proven by controlled studies. Our report highlights that early recognition and subsequent management may mitigate the morbidity and help in boosting the recovery process in hearing loss.

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編號 02

使用輔助性高壓氧治療換肝病患合併高膽紅素之病例報告
Hyperbaric Oxygen Therapy as an Adjuvant Treatment in Post Liver Transplant Related Hyperbilirubinemia ---A Case Report

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前言:

肝臟移植(liver transplantation)是治療晚期肝病、急性肝衰竭與肝臟腫瘤的一種治療選擇。肝臟移植後併發功能性膽汁淤積(cholestasis)原因包括缺血與再灌注損傷(ischemia and reperfusion injury)、感染、藥物與排斥等，而其診斷與治療，對於臨床醫師而言，是一個重大的挑戰。

個案報告:

本個案為67歲女性，本身為慢性C型肝炎合併肝硬化。2010年12月發現肝癌，曾接受射頻燒灼術(radiofrequency ablation)，於2011年8月接接肝臟移植，之後接受於2011.10.3接受干擾素(interferon)和抗病毒藥物(ribavirin)治療。2012年10月開始發現全身發黃，解灰白便和茶色尿，食慾差和易喘而入院。住院期間曾因高膽紅素血症(hyperbilirubinemia)接受分子吸附再循環系統（Molecular Adsorbents Recirculating System, MARS）治療。2012/12接受肝切片檢查，病理報告顯示缺血性膽管病和膽道阻塞，故以高壓氧做為輔助治療。

治療措施:

高壓氧做為輔助治療，每天一次，一周5次，一次2.5 ATA 120分鐘，共26次。

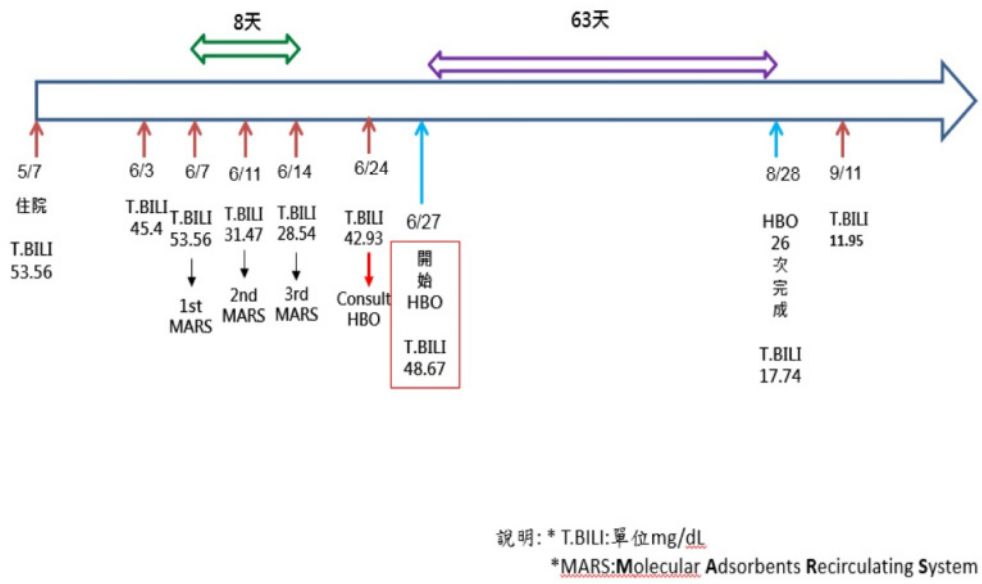

圖1. 治療流程圖

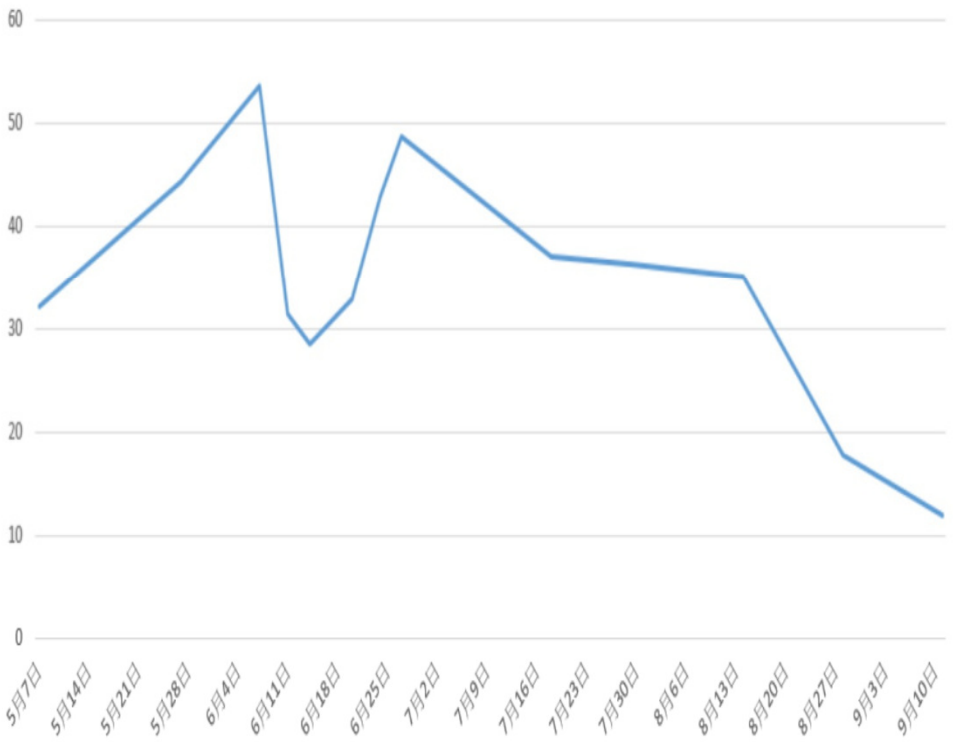

圖2. Serum total bilirubin 變化圖

結果:

本個案經由併用高壓氧輔助治療26次後，於門診追蹤，膽紅素由48.67mg/dl降到11.95mg/dl。

討論:

在Silva等人(2006)個案報告中指出，高壓氧治療可以增加血漿中氧氣之溶解，增加門脈血氧含量和增強肝動脈的側枝循環，降低肝臟組織移植後產生之缺血與缺氧的影響。由此個案得知使用高壓氧輔助治療對於肝臟移植病患發生高膽紅素血症是有助益的。

參考文獻：

1. Lv H, Han C, Sun X, Liu W. Application of hyperbaric oxygen in liver transplantation. Med Gas Res. 2016;6:212-18.
2. Zarrinpar A, Busuttil RW. Liver transplantation: past, present and future. Nat Rev Gastroenterol Hepatol.2013;10:434-40.
3. Castro-Silva O, Sankarankutty AK, Martinelli AL, Souza FF, Teixeira AC, Feres O, Mente ED, Oliveira GR, Akita R, Muglia V, Elias JJr, Ramalho LN, Zucoloto S. Therapeutic effect of hyperbaric oxygen in hepatic artery thrombosis and functional cholestasis after orthotopic liver transplantation. Transplant Proc. 2006;38:1913-17.
4. Sun Y, Wen Y, Shen C, Zhu Y, You W, Meng Y, Chen L, Feng Y, Yang X, Chen ZB. Hyperbaric Oxygen Therapy in Liver Diseases : International Journal of Medical Sciences.2018;15:782-87.

關鍵字:

肝臟移植(liver transplantation)，高膽紅素血症(hyperbilirubinemia)，高壓氧治療(hyperbaric oxygen therapy，HBOT)

編號 03

子宮內膜癌手術後下肢淋巴水腫病人使用高壓氧治療之照護經驗-病例報告

Experiences in Hyperbaric Oxygen Therapy for Patients with Lower Limb Lymphedema after Endometrial Cancer Surgery - A Case Report

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臺北榮民總醫院胸腔部呼吸治療科[1]、國立陽明大學[2]

【個案情況】

◆**子宮內膜癌**(Endometrial Cancer)是一種常見的疾病且在生活中時有所聞，普遍好發於年齡超過五十歲的婦女，隨著近年飲食、生活作息的改變，病例有增加的趨勢。其主要是指子宮最內層所長出的惡性腫瘤，常見治療手段為**淋巴結清掃術**(Lymphadenectomy)和**放射線治療**(Radiation therapy)，但在2014年Kathleen J. Yost的文章有提到這些治療可能會增加罹患**下肢淋巴水腫**的風險。

◆本個案為60多歲女性，過去病史為Endometrial cancer ，因子宮內膜癌手術後致使下肢淋巴水腫，故於2020/03曾在本院進行治療。住院期間有給予藥物及建議病人可抬高右腳等相關的治療準則。但因病人右下肢還是持續有水腫的情況，因此在2020/09/01至高壓氧門診尋求可能的幫助。因病人有進行手術及放射線治療，故有可能會造成淋巴管狹窄、閉塞以及纖維化，導致下肢皮膚、皮下組織的淋巴液及組織間液滯留，進而產生淋巴水腫。因此，建議病人可運用高壓氧讓受損組織缺血缺氧的狀況得到改善，藉以促進血液流動，生成新血管，促進傷口癒合並減少纖維化，改變患部水腫的現象。病人於2020/09/02開始治療，期間共計完成30次高壓氧治療(療程為120分鐘/2.5ATA)。治療期間病人主述其下肢水腫有明顯的改善，此外本院技術員也有發現患部治療前和治療後有明顯的差異。故由此可知，此次高壓氧治療對於病人來說是有助益的。

【討論】

◆淋巴水腫是指淋巴系統受傷或失去功能造成淋巴液阻塞腫脹的現象，尤其癌症病人進行淋巴結清掃術，幾乎都會造成不同程度的淋巴水腫。就女性而言，**乳癌的治療可能會引起上肢淋巴水腫**，而**子宮頸的治療則可能造成下肢淋巴水腫**，本個案屬於後者。

◆在2014年Kathleen J. Yost的文獻中探討接受子宮內膜癌手術患者下肢淋巴水腫的患病率，並評估淋巴水腫對生活品質的影響。結果發現如病人有肥胖、充血性心衰竭、使用淋巴結清掃術或放射線療法，會與淋巴水腫的患病率有關，而這也顯示本個案為何會有下肢水腫的情形。另外，在2020年Jung Hoi Koo的文章中，作者將族群分成兩組{第一組為單純的整合性去腫治療(complex decongestive therapy)，第二組為高壓氧＋整合性去腫治療}，藉以探討高壓氧對於乳腺癌造成的淋巴水腫的功效，其結果是建議高壓氧可以做為現有療法的輔助治療，原因是**高壓氧可以透過刺激血管內皮生長因子使纖維化減少進而改善淋巴水腫**，因此，由這篇文章可知高壓氧治療對於這類型的病人是有幫助的。

◆或許在未來，可以多加利用高壓氧來治療此類型的病人，使病人有更多的治療方式選擇及更好的治療結果。

2014 年 Kathleen J. Yost

Table 4

Multivariable Analysis of Predictors of Lower-Extremity Lymphedema

Characteristic	Adjusted Odds Ratio (95% CI)	*P*
BMI at the time of surgery (kg/m^2)		<0.001
Underweight or normal (BMI<24.9)	Reference	
Overweight (BMI 25.0–29.9)	1.11 (0.66, 1.87)	
WHO class I/II (BMI 30.0–39.9)	1.45 (0.90, 2.34)	
WHO class III/super obese (BMI 40.0–49.9/50+)	4.69 (2.71, 8.13)	
Congestive heart failure	2.58 (1.23, 5.40)	0.01
Lymphadenectomy	2.04 (1.39, 2.99)	<0.001
External beam irradiation therapy	3.00 (1.46, 6.16)	0.003

Note: Odds ratios shown are adjusted for each of the other characteristics listed in this table.

BMI, body mass index; WHO, World Health Organization.

2020 年 Jung Hoi Koo

Table 3

Changes in BIS parameters before and after treatment.

	HBOT+CDT group (n=5)	CDT only group (n=5)	*P*-value
SFBIA 1 Hz (pre)	1.5±0.2	1.4±0.6	.222
SFBIA 1 Hz (post)	1.4±0.1	1.3±0.4	.151
P-value	0.043*	0.686	
SFBIA 5 Hz (pre)	1.5±0.2	1.4±0.6	.222
SFBIA 5 Hz (post)	1.4±0.1	1.3±0.4	.310
P-value	0.043*	0.686	
ECF ratio (pre)	1.5±0.2	1.4±0.6	.151
ECF ratio (post)	1.3±0.1	1.3±0.4	.151
P-value	0.043*	0.465	

Values are mean±SD. BIS = bioelectrical impedance spectroscopy, CDT = complex decongestive therapy, ECF = extracellular fluid, HBOT = hyperbaric oxygen therapy, SFBIA = single frequency bioimpedance analysis.

* Statistically significant difference ($P < .05$).

潛水旅館內自由潛水產生潛水黑視症—病例報告
Hypoxic Blackout Occurrence During Breath-hold Diving (Freediving) in a Diving Hotel Pool – A case report

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【Introduction】

With the vigorous development of freediving occurring worldwide, there have been more and more reports surrounding concomitant complications due to this activity. Hypoxic blackout is one of the life-threatening complications caused by cerebral hypoxia during freediving.

【Case report】

Mr. Chang, a 29-year-old male had no significant systemic disease. He performed freediving with 2 diving buddies at the Divecube Hotel, Asia's first hotel built with a pool dedicated to scuba diving and freediving in Taichung. The pool has five various depth levels, with the deepest one being measured at 21 meters (Figure 1). All three divers were certified freedivers so it was not necessary for an instructor to accompany them. They did however follow the buddy system while undergoing this activity. The three began diving at 8 am, during which time no obvious discomfort was complained of by Mr. Chang. At 12 noon, Mr. Chang performed another freedive lasting 2 to 3 minutes but lost consciousness while underwater at a level of 11 meters. He subsequently sank down to 21-meters below the surface before being immediately rescued by lifeguards. There was no voluntary breathing from Mr. Chang after being pulled up from the pool so artificial respiration was performed on him. When the EMTs arrived at the pool, the patient was still unconsciousness but had regained his own respiration. A non-rebreathing mask was applied.

At the emergency center, desaturation was noted and intubation was performed. Respiratory and metabolic acidosis with elevated lactate and ammonia levels were found. A CXR revealed a bilateral alveolar pattern with upper lobe predominant and central distribution, which indicated near drowning pulmonary edema. (Figure 2A) He was admitted to the intensive care unit and therapeutic hypothermia was performed for a total 72 of hours. He recovered consciousness after the discontinuation of his sedative medication. An subsequent CXR revealed improvement in bilateral infiltration (Figure 2B) and extubation was performed 3 days later. The patient was discharged 8 days after admission with fully recovered consciousness (E4V5M6).

【Discussion】

Rather than gradual downregulation of brain function, functions within the human brain decline rapidly upon hypoxia and humans experience an abrupt loss of consciousness. Hypoxia induced loss of consciousness is labeled hypoxic blackout, which typically occurs at arterial PaO2 below 30mmHg during static apnea. A blackout usually occurs during the ascent to shallower water because one's PaO2 drops rapidly during the process.

Apnea is terminated when the breakpoint is reached. Breakpoint corresponds with low oxygen and high carbon dioxide levels reflecting chemoreceptor sensitivity and urge to breathe. Excessive hyperventilation lowers the initial CO2 level, prolonging time to the breakpoint, allowing critical hypoxia to be reached underwater, which may induce the occurrence of a blackout (Figure 3).

This case raised our concerns that freedivers can experience blackouts from hypoxia due to a drop in oxygen partial pressure during ascent. We should also be aware of the danger of the hyperventilation before attempting long breath-holds because it may induce hypoxic blackouts with virtually no warning.

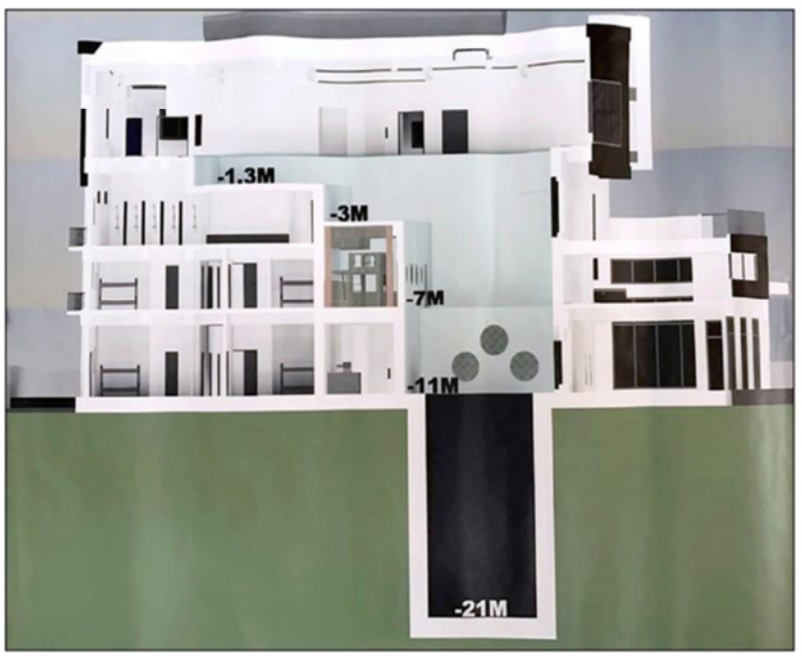

Figure 1
The building section of Divecube Hotel. The pool has five various depth levels, with the deepest one being measured at 21 meters

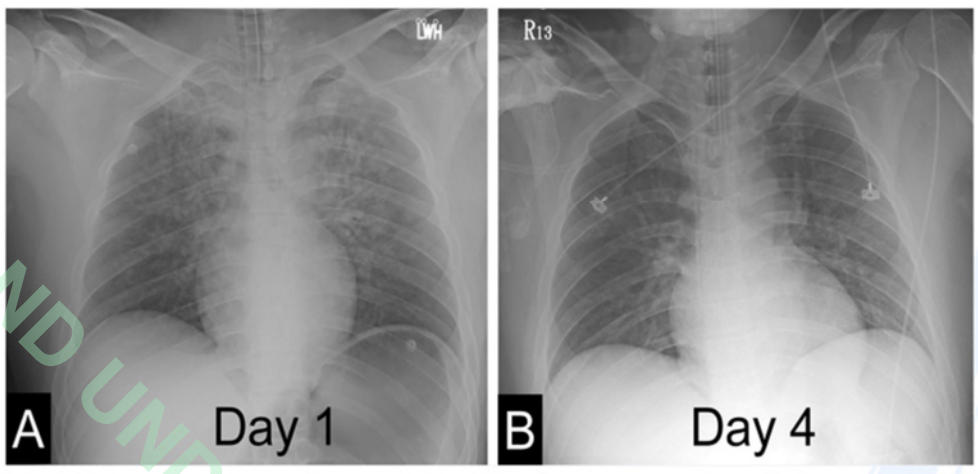

Figure 2
(A) The CXR revealed a bilateral alveolar pattern with upper lobe predominant and central distribution on day 1. (B) An subsequent CXR revealed improvement in bilateral infiltration on day 4.

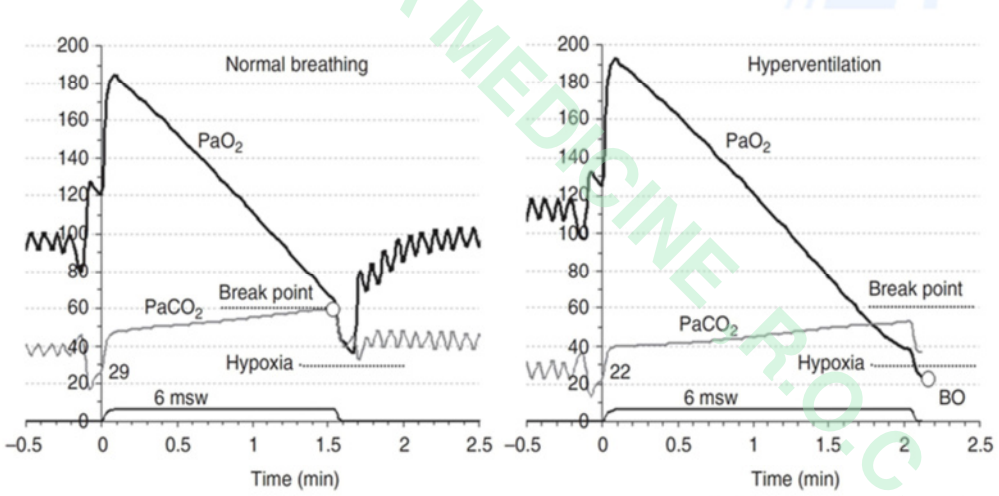

Figure 3
Computer-simulated gas levels during a swimming breath-hold dive to 6 msw indicated at the bottom. Initial arterial PaCO2 is 29 mmHg (left) after normal breathing and the breakpoint is reached after 1.5 min when PaCO2 reaches 60 mmHg (circle). Ascent avoids critical hypoxia, and the dive is safe. In contrast, predive hyperventilation (right) lowers initial PaCO2 to 22 mmHg and allows a longer dive without the urge to breathe. PaO2 drops below 30 mmHg on ascent and hypoxic blackout occurs near the surface.
Reference: American Physiological Society. Compr Physiol 8:585-630, 2018.

編號 05

高壓氧治療產生中耳氣壓傷合併症之探討

The complication of Middle ear barotrauma with hyperbaric oxygen therapy

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Purpose: Middle ear barotrauma (MEB) is a common complication of hyperbaric oxygen therapy HBOT). The purpose of the present study was to determine the overall incidence of MEB and evaluate the incidence of MEB in different disease entities. The study also sought to identify the potential risk factors for the development of MEB.

Methods:Prospective study including all the patients treated in a multiplace Hbo2 chamber between January 2017 and December2020. Scoring of MEB before and after Hbo2 by otoscopy was performed using the Haines and Harris classification.

All intubated patients received bilateral tympanostomy before HBOT.

Results: We included 1336 patients: 591 males,745 females, 57.2 ± 17.3 years old; 13.4% were treated for co poisoning, 10.6% for CVA, 16.8% for problem wound, 9.1% for chronic osteomyelitis, 9.4% for crush injury, 25% for sudden hearing loss, 6.9% for radiation damage, 8.7% for others (decompression sickness, necrotizing soft tissue infection etc). 2.8% (37 patients) were intubated. MEB occurred in 5.0% (65 patients) of the patients. 30 males, 35 females, 53.9 ± 15.1 years old; 6.2% were treated for CO poisoning, 7.7% for CVA, 20% for problem wound, 7.7% for chronic osteomyelitis, 6.2% for crush injury, 32.3% for sudden hearing loss, 12.3% for radiation damage, 7.7% for others. The severity of MEB according to the Haines and Harris classification was grade1 35.4%, grade, grade 2 55.4%, grade 3 9.2%. There was no influence of age, sex on the occurrence of MEB. Multivariate logistic analysis identified only CO intoxication (adjusted odds ration: 3.16, p=0.038) as an independent risk factors for MEB.

Conclusion: MEB induced by HBOT occurred in 5.0% of the patients. CO intoxication is the only independent risk factor found in present study.

Keywords: Hyperbaric oxygen therapy (HBOT), Middle ear barotrauma (MEB), CO intoxication, Cerebrovascular accident (CVA)

類別變數編碼

		次數	參數編碼 (1)
診斷8	0	1220	1.000
	others	116	.000
診斷2	0	1194	1.000
	CVA+REHALBATION	142	.000
診斷3	0	1111	1.000
	DM foot+problem wound	225	.000
診斷4	0	1244	1.000
	osteomylitis	92	.000
診斷5	0	1214	1.000
	radiation damage	122	.000
診斷7	0	823	1.000
	sudden ear loss	513	.000
診斷6	0	1210	1.000
	crash +fx	126	.000
診斷1	0	1157	1.000
	co intoxication	179	.000

變數不在方程式中[a]

			分數	df	顯著性
步驟 0	變數	性別	2.558	1	.110
		診斷1(1)	3.090	1	.079
		診斷2(1)	.620	1	.431
		診斷3(1)	.487	1	.485
		診斷4(1)	3.132	1	.077
		診斷5(1)	.171	1	.680
		診斷6(1)	.859	1	.354
		診斷7(1)	.000	1	.991
		診斷8(1)	.085	1	.771
		年齡60歲	.375	1	.540

a. 因為重複的關係，所以無法計算殘差卡方統計量。

亞東紀念醫院
FAR EASTERN MEMORIAL HOSPITAL

編號 06

高壓氧療法成功治療視網膜中央動脈阻塞（CRAO）

Successful treatment with hyperbaric oxygen therapy for Central retinal artery occlusion (CRAO)

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Purpose: Central retinal artery occlusion (CRAO) is a relatively rare emergency and potentially devastating condition of the eye. Patients present with sudden, painless monocular visual loss. The retina has the highest oxygen consumption rate of any organ in the body at 13 mL/100 gm per min.Hyperbaric oxygen therapy (HBOT) has been used to treat patients with CRAO and most of treated cases have shown improvement.

Methods : We collected data which reported sudden visual loss due to CRAO that was treated with HBOT in Far Eastern Memorial Hospital from 2017. to 2020 Dec. VA was recorded using Snellen optotypes, to compare the means, compared t test, chi-square test were performed using SPSS Statistics version 19.0(IBM,Armonk,New York,USA)

Results: Totally 19 patients were included (age 60.95±12.4 years, sex:10male/9 female, the days of CRAO onset until to hospital 4.11± 3.46,the days of CRAO onset until to HBOT 12.37±8.06,the times of HBOT:8.79±4.97)we corrected the result about visual acuity. Other variables (e.g. age, duration of symptoms) also did not differ significantly at baseline. Post the treatment of HBOT , in the lesion side group improved by a mean of lines:1.16±1.19 to 2.05±1.268 to ($p<0.015$). In the health vision group, mean improvement was line:4.16±1.21to 4.42±1.07($p=0.135$). we separated two group: the 4 days boundary of CRAO onset until to HBOT(3/16). The group of less 4 days(age 68.95±18 years, sex:1male/2 female), The group of more than 4 days(age59.63±11 years, sex:9male/7 female). the lesion side group of less 4 days have improvement more than 4 days : mean of lines:0.895±0.309 to 2.63±0.162 to ($p<0.011$). The 3 month follow-up visit yielded similar data.

Conclusion : HBOT provides oxygen at leves of atmospheric pressure. The purpose of hyperbaric oxygen is to preserve the retina in an oxygen state until recanalization and reperfusion occur, typically at 24-96 hours. Some reseracher describe that there are important key factors to determine the HBOT success: time at HBOT is started. The improvement may be related to the early timing of the treatment of our patients of CRAO. .

Keywords: Central retinal artery occlusion (CRAO), Hyperbaric oxygen therapy (HBOT), Count fingers (CF)

敘述統計

	個數	最小值	最大值	平均數	標準差
年齡	19	36	81	60.95	12.389
發生日至求醫天數	19	1	15	4.11	3.462
發生日至高壓氧天數	19	2	30	12.37	8.064
HBO天數	19	1	33	13.95	8.997
HBO次數	19	1	19	8.79	4.973
HTN	19	0	1	.47	.513
高血脂	19	0	1	.21	.419
DM	19	0	1	.32	.478
腎衰竭	19	0	1	.11	.315
CVA	19	0	1	.05	.229
CAD	19	0	1	.05	.229
有效的 N (完全排除)	19				

敘述統計

	個數	最小值	最大值	平均數	標準差
HBO次數	19	1	19	8.79	4.973
發生日至求醫天數	19	1	15	4.11	3.462
發生日至高壓氧天數	19	2	30	12.37	8.064
HBO天數	19	1	33	13.95	8.997
VA(患側)	19	0	4	1.16	1.119
VA(患側)	19	1	5	2.05	1.268
有效的 N (完全排除)	19				

成對樣本檢定

		成對變數差異					t	自由度	顯著性 (雙尾)
		平均數	標準差	平均數的標準誤	差異的 95% 信賴區間 下界	上界			
成對 1	VA(患側) - VA(患側)	-.895	1.449	.332	-1.593	-.196	-2.692	18	.015
成對 2	VA(健側) - VA(健側)	-.263	.733	.168	-.617	.090	-1.564	18	.135

亞東紀念醫院
FAR EASTERN MEMORIAL HOSPITAL

編號 07

Early intervention of hyperbaric oxygen therapy on crush injury：A Case Report

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秀傳醫療財團法人彰濱秀傳紀念醫院前瞻傷口醫學部高壓氧中心[1]
神經內科部[2]

個案簡介：

25歲女性，無特殊疾病史，2020年12月29日工作遭機器壓傷，致右手第三到五指碾壓傷及合併撕裂傷，急入清創與骨內固定，2020年12月30日，患肢手掌腫脹及發紺，於12日31介入高壓氧治療。

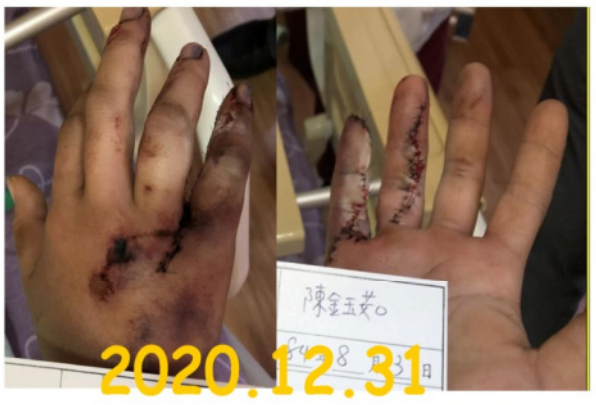
討論：

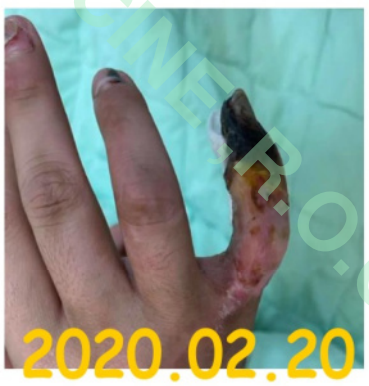
肢體的碾壓傷是一種複雜性的復合傷害，其合併骨折、肌肉、認袋撕裂、神經血管斷裂等狀況。引發的傷害不單患肢一開始的組織損傷，依傷害的嚴重程度，甚至會引發更多的合併症，最常見的是肢體的腫脹引發腔室症候群。組織中微血管通透壓為30-15毫米汞柱，受傷後組織腫脹超過此壓力，即造成肌肉組織缺血，超過四小時以上，將產生不可逆的組織壞死，最終截肢。藉由高壓氧氣治療的輔助治療，有效達到：

1.緩解組織缺氧，增加組織含氧量。2.減少組織水腫。3.增加白血球防禦功能，減緩感染。4.加速骨骼、膠原蛋白合成、血管新生成，達促進傷口的癒合。一般建議能再在受傷黃金的72小時內就實施高壓氧治療是最好，能減輕組織的傷害程度。

結果：

個案自2020年12月31日開始高壓氧治療，壓力2.5ATA，每次90分鐘，一天一次，每週五次，共計12次，於治療第六次時，傷口發紺範圍縮小，12次治療結束後，第五指末梢成乾性壞疽，餘傷口結痂，於同年2月22日行末梢壞疽截除手術，傷口癒後良好。





結論：

碾壓傷是一種併發血管、神經、組織傷害的急性損傷，且容易造成周圍組織的腫脹而引發組織缺氧壞死，如能提早介入治療，將有效減損傷範圍，個案雖在黃金期介入治療，但如能在手術後提早介入治療，將有機會避免截指之機會。

編號 08

真心關懷、擁抱健康
成為在地民眾

新北市立聯合醫院
NEW TAIPEI CITY HOSPITAL

高壓氧治療視網膜中心動脈阻塞-個案報告及文獻回顧

Application of Hyperbaric Oxygen Therapy in Central Retinal Artery Occlusion (CRAO)-A case report and review of literature

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個案情況：

個案為66歲男性，本身具糖尿病和高血壓病史，且長期處方用藥服用，吸菸史超過50年。於109年12月2日因左側上下肢乏力入本院急診求治。意識清楚，GCS=E4V5M6，生命徵象穩定，NIHSS：9分，四肢肌肉強度：RUL(5)、LUL(1) 、RLL(4) 、LLL(3) ，經腦部及頸椎核磁共振確診：右側丘腦梗塞收治神內。個案同月10日主訴清晨七點多睡醒，突然感覺右眼一片漆黑看不到物品。緊急照會眼科行視力和眼底鏡檢查，報告為右眼最佳矯正視力(BCVA) 0.3、眼內壓(IOP) 16/17和發現右眼視網膜中心動脈阻塞(CRAO)，即俗稱的「眼中風」。除增加抗血栓藥物和抗凝血劑劑量，同時緊急照會高壓氧專科醫師安排高壓氧治療，予以個案每日一次2.5ATA治療100分鐘，總共10次。個案於治療第三天視力恢復BCVA至0.5，逐漸看到景物輪廓和分辨顏色，治療第十天視力恢復BCVA達0.9。

討論：

視網膜為一特異化的神經構造，發生血管阻塞、中毒或發炎，因而缺氧導致神經無法傳導視覺訊號造成突發性失明。視網膜中心動脈阻塞(CRAO)是眼科急症之一，以單側、突發性且無痛的嚴重視力喪失表現。其動脈阻塞種類有四：血栓、栓塞、動脈炎和血管痙攣。前兩者為視網膜中心動脈阻(CRAO)常見原因，危險因子包含糖尿病、高血壓、高血脂、動脈粥狀硬化和心臟瓣膜病變等。根據文獻說明若視網膜超過105分鐘缺乏血液和氧氣供應將造成永久性損害。

高壓氧治療益處：(1)提高細胞氧分壓和血氧含量(2改善患部水腫(3)抑制發炎反應和氧化壓力等。根據Textbook of Hyperbaric medicine收集1965-2015年37篇文獻，共758位視網膜中心動脈阻塞(CRAO)，經高壓氧治療後共482位視力(BCVA)顯著改善，改善率高達64%。故目前高壓氧治療已被認可成為視網膜中心動脈阻塞(CRAO)急性期一線治療方式之一，且應於病發後6-8小時內或至多24小時內介入，及早治療以增加視網膜甚至脈絡膜的氧氣張力，此攸關視力能否大幅改善的幅度，正所謂"Time is vision in CRAO"。本個案因腦梗塞入院且具糖尿病、高血壓和長年吸菸史，符合視網膜中心動脈阻(CRAO)高風險。除按照Textbook of Hyperbaric medicine第一時間採用USN Table 9或Table 6介入高壓樣治療外，仍須鼓勵和協助個案戒菸、血糖和血壓得良好監測和服藥控制，追求健康促進態度，捍衛自己健康積極作為。

※Textbook of Hyperbaric medicine適用於突發性失明的緊急高壓氧治療條件
Age>40
Presentation within 24h of vision loss
Corrected visual acuity 20/200 or worse
Visual acuity still 20/200 or worse with pin hole testing
No history of recent eye trauma
No pain associated with the vision loss
No history of acute onset of flashes or floaters prior to vision loss

編號 09

一氧化碳中毒合併腔室症候群接受高壓氧治療之經驗分享

Experience of using hyperbaric oxygen therapy (HBOT) in carbon monoxide poisoning with compartment syndrome

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2：大千綜合醫院　復健科

個 案 資 料

個案為42歲的男性，在自家轎車內燒炭自殺，自行甦醒後返家，因感到右手腫脹無知覺、全身無力就醫，經醫生診斷一氧化碳中毒，需做高壓氧治療而轉至本院，住院期間因右手指無法彎曲、右手掌明顯腫脹無力，會診復健科後確診為腔室症候群，接受高壓氧治療共50次，並積極接受相關的復健訓練治療，目前右手腫脹改善，且已經可以握筆寫字。

討 論

腔室症候群(Compartment syndromes)是由於各種原因導致肢體腔室內腫脹引起壓力增加，造成循環受阻而缺血。嚴重時若不及時處理，則會引起缺氧反應而造成肌肉組織壞死、神經缺損、骨折及傷口癒合不良等情況。急性腔室症候群一般建議接受手術，進行筋膜切開術(fasciotomy)，盡快降低腔室內壓力，減少組織受到壓迫壞死的程度。亞急性腔室症候群同樣通常仍建議接受手術治療。慢性腔室症候群可以接受保守性的治療

高壓氧治療可提高血液中的含氧量，減少因局部壓力上升，造成血液灌流量不足引起的組織缺氧症狀，且因血液含高濃度氧氣，可以維持血管收縮。綜合上述的生理機制，高壓氧治療不僅可以減少組織缺氧壞死，同時還可以避免為了提高供氧量而提高血液灌流量，造成腔室症候群區域壓力上升引起惡化。因此透過高壓氧治療，不僅可以降低造成腔室症候群的壓力，同時間仍能保持高濃度的血液含氧量，避免組織因灌流量下降而引發的組織缺氧壞死反應。氧氣濃度的上升，可促進纖維母細胞(fibroblast)分化成膠原蛋白(collagen)，促進血管新生，並往較嚴重的傷口區域生長，加速組織癒合。

根據本個案的治療成果顯示，對於腔室症候群的個案，高壓氧治療機轉能降低受傷區域的壓力，並改善組織缺氧壞死狀況，同時促進纖維母細胞(fibroblast)促進膠原(collagen)蛋白合成，促進血管的新生並加速傷口癒合。但目前針對這方面的醫學研究佐證仍不足，建議未來如有更多個案，可持續追蹤分析治療成效。

編號 10

突發性耳聾接受高壓氧治療之經驗分享

Experience of using hyperbaric oxygen therapy (HBOT) in sudden sensorineural hearing loss (SSHL)

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2：大千綜合醫院　復健科

個 案 資 料

個案為27歲男性，五個月前突然左耳聽力受損，前往耳鼻喉科診所就診，口服類固醇藥物治療但聽力持續未獲得改善，經醫師建議接受高壓氧治療。此個案透過單人式高壓氧艙，每次治療劑量為2.0 ATA，治療時間60分鐘，共計12次治療。治療後聽力從80db進步至71db，患者自我感覺聽力功能有明顯進步，生活品質提升。

討 論

突發性耳聾在醫學上的定義為突然於72小時內聽力診斷圖上三個相鄰的音頻，發生大於30分貝閾值的感音性聽力減退。發病年齡多介於40歲至60歲，男女比例相當，多數病例出現單耳症狀，較少雙耳同時發生。目前真正原因不明，可能的原因包括感染、耳毒性藥物、腫瘤、創傷、自體免疫、微血管疾病或其他因素，但造成突發性耳聾的最常見的原因可能是血管因素導致之組織缺血或病毒感染導致之神經炎。

突發性耳聾在治療上多數皆採合併式療法，目前確認最有療效的是類固醇藥物施用；其他治療的主要原則是改善血液循環及組織灌流為原則。目前醫學研究中，已有許多文獻支持高壓氧治療用於突發性耳聾之輔助療法，對於突發性耳聾患者，有不錯的治療成效。研究指出接受高壓氧治療後，可提昇動脈氧分壓使供應內耳組織的血液含氧量增加，緩解內耳受傷後水腫及缺氧，並促進耳蝸及半規管內微循環和淋巴液的流動，增加新陳代謝修復加速聽力恢復。其中，研究結果顯示年齡小於50歲，且合併中重度聽力損失(平均聽力>60分貝)之患者，在傳統治療下建議輔以高壓氧治療。

雖然目前引發突發性耳聾的原因仍不明確，但目前推估多數都跟缺血性引起的缺氧的神經炎相關，高壓氧治療可以盡速改善缺血及缺氧的狀況，雖醫學研究上仍未找到較確定的致病原因，但已有許多臨床實例佐證，高壓氧治療可以有效改善突發性耳聾之狀況。

編號 11

高壓氧治療運用於肌腱斷裂撕裂傷之經驗

Experience of using hyperbaric oxygen therapy (HBOT) in tendon rupture

侯詩臻[1]、陳泰安[1]、邱智弘[1]、林威宇[2*]

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2：大千綜合醫院　復健科

個 案 資 料

個案為40歲男性，因為交通事故導致右小腿有一處長達20公分的撕裂傷，傷口深及肌腱、肌肉層，經骨科醫師進行縫接神經及肌肉組織的修復手術後，建議再接受每天一次的高壓氧輔助治療，個案的組織修復狀況良好，且因傷及神經及肌肉組織，持續接受復健治療。

討 論

肌肉、神經、血管及骨骼直接的破壞，會造成組織缺氧及水腫，且因為缺氧而造成血液流量增加，再次造成水腫壓迫，如此惡性循環的破壞，會造成組織缺氧過久而壞死無法修復。若傷口較嚴重且引起肌腱肌肉組織破壞，會產生更多毒素，引起造血及免疫功能下降，並造成全身微血管通透性增加，促使血管中血漿和電解質流失到週邊組織，引發血管內脫水性休克及肺、腦全身部位之水腫。如果受傷時造成血管斷裂，並重新接合血管，血管接合處容易有大量白血球堆積，同時釋放許多有害的氧游離基，對該處的血管內皮細胞和已經受傷之組織進行更嚴重的破壞，並進一步引起血小板聚集和血液凝固，最後導致缺血再灌流傷害，引起更大面積的組織缺血性破壞。

高壓氧治療可以提升微血管擴散的能力，包含提高擴散速率及範圍，且因為處於高壓力環境下，血液中的氧分壓提高，可以提供組織細胞足夠的氧氣，可以阻斷水腫及缺氧的惡性循環，恢復正常的有氧代謝及循環。同時因為氧分壓提高，可促進纖維母細胞(fibroblast)分化成膠原蛋白(collagen)，促進血管新生，並往較嚴重的傷口區域生長，加速組織癒合。此外，因為高壓氧治療提供了足夠的氧氣，促使白血球能夠有效工作，避免傷口受到細菌的感染，且因為高壓氧治療提供組織良好環境，使相關藥物在執行作用時，也能達到事半功倍的效果。

高壓氧治療可以促進消腫、傷口癒合及降低感染的機率，因此嚴重且範圍較大之傷口，影響程面也更廣，治療時效性特別重要，能越早接受高壓氧治療成效越好，後續所造成的傷害越少。

編號 12

Hyperbaric Oxygen Therapy in the Management of Hand Crush Injuries
高壓氧於手部壓砸傷的治療

張惇皓[1,2] 張哲瑋[1,2] 謝綺瀅[1,2] 王旭輝[1] 王家弘[1] 張厚台[1]
亞東紀念醫院 [1]高壓氧中心及 [2]整形外科

Purpose

Hyperbaric Oxygen therapy (HBOT) can improve tissue hypoxia and stimulate acute wound healing and has been an adjuvant treatment for crush injury. However, previous studies were mostly case series without a control group and the actual role of HBOT in crush hand injury is still not clear. This study aims to evaluate the efficacy of HBOT as an adjunctive therapy to the surgery in patients with a crush hand injury.

Methods

We retrospectively reviewed the chart and photos of crush hand injury patients between January 2018 and May 2020. The patients were classified as HBOT and a control group. The wound photos of the injured hand were reviewed by a therapist who didn't know the patient's group. The skin necrosis area, skin graft or flap survival area were evaluated by percentage. Excluding the patients undergoing abdominal or groin flap, the time for wound healing, hospital stay, total operation times and complications were recorded and analyzed.

Results

Sixty patients with a crush hand injury were enrolled. The patients consisted of 50 males and 10 females with a mean age of 39.5±13.6 years. 27 patients received the HBOT while 33 patients were the control group.(**Table1**) The average session of HBOT was 17.1 (2-32) per patient. The usual protocol was 120 minutes with 100% oxygen at 2.5 atm. The demographic data and the characteristics of injury were similar between the two groups.

Table 1. Patient Demographics and Injury Characteristics

	HBO(n=27)	Control(n=33)	P value
Age (years)	38.0 ±13.3	40.7 ±13.6	0.45
Sex			0.32
Male	21(77.8%)	29(87.9%)	
Female	6(22.2%)	4(12.1%)	
Smoking	10(37.0%)	9(27.3%)	0.42
HTN	2(8.3%)	4(12.5%)	0.69
DM	0	4(12.5%)	0.13
Finger amputation	9(33.3%)	16 (48.5%)	0.24
Total injured area (cm^2)	85.2 ±113.2	52.3±47.8	0.15
Reconstruction method			
STSG	10(37.0)	15(45.5)	0.51
Groin flap	5(18.5)	4(12.1)	0.72

When excluding the patient undergoing groin flap, there's no significant difference in time for wound healing (35.1.±22.6 VS 42.1±22.2 days, p=0.27), hospital stay (12.1±10.1 VS 10.7±9.2, p=0.61), operation times (2.48±1.89 VS 2.52±1.52, p=0.94) and wound injury area (62.4±43.2cm2 VS 49.2±48.1cm2, p=0.32), but the HBOT group seemed to have more graft/ flap survival area (98.6±3.2% VS 89.4±21.6%, p=0.1).(**Table2**)

Table 2. Outcome of the treatment group (Excluded the patient underwent groin flaps)

	HBO(n=22)	Control(n=27)	P value
Total injured area (cm^2)	62.4± 43.2	49.2 ±48.1	0.32
Wound infection	7(25.9)	8(24.2)	0.88
Secondary amputation	5(18.5)	6(18.2)	0.97
Skin graft area (cm^2)	24.3±19.0	14.9±9.2	0.14
Necrosis percentage (%)	20.5±24.6	19.9±12.6	0.92
Skin graft take rate (%)	98.6±3.2	89.4±21.6	0.10
Hospital stay (Days)	12.1±10.1	10.7±9.2	0.61
Time to wound heal (Days)	35.1±22.6	42.1±22.2	0.27
Total Operation	2.48±1.89	2.52±1.52	0.94

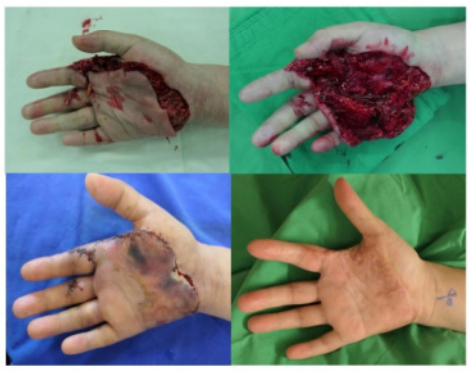


Figure 1. A 37-year-old woman suffered from right hand crush injury with avulsion flap, and underwent primary closure with HBOT 15m, 90 mins for 23 sessions.

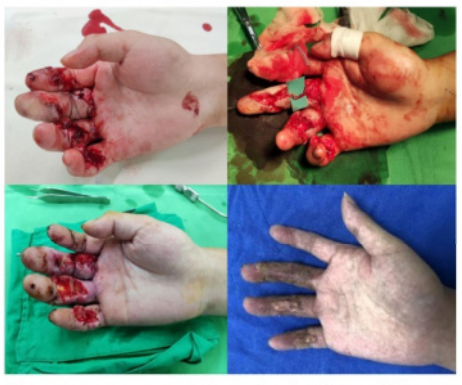
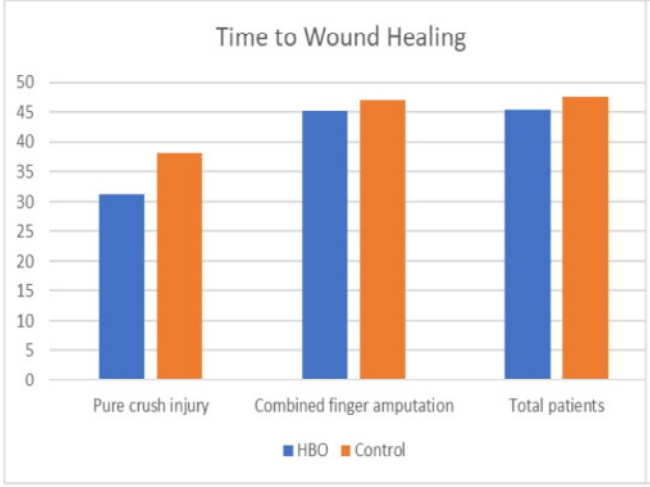
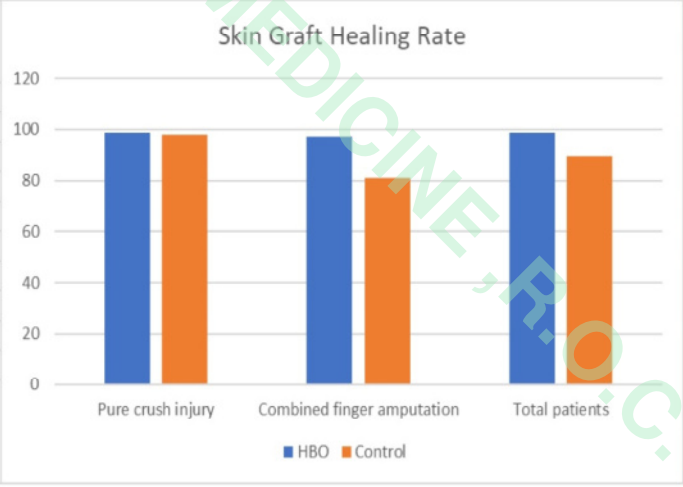


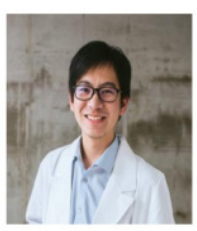
Figure 2. A 18-year-old man had right middle, ring and little finger crush injury with middle finger devascularization. After restoring circulation, artificial dermis was used for wound coverage. After HBOT 10m, 60 mins for 20 sessions, granulation tissue grew well with subsequent good result of skin graft.





Conclusion

The patients' injury profiles were diverse in this study, however, HBOT may be effective in improving wound healing and skin graft or flap survival. We believe that HBOT is a useful adjunct in the management of crush hand injury. Further study is required.



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編號 13

照顧一位慢性傷口接受高壓氧治療之經驗

Hyperbaric Oxygen Therapy for chronic Wounds Care-Case Report

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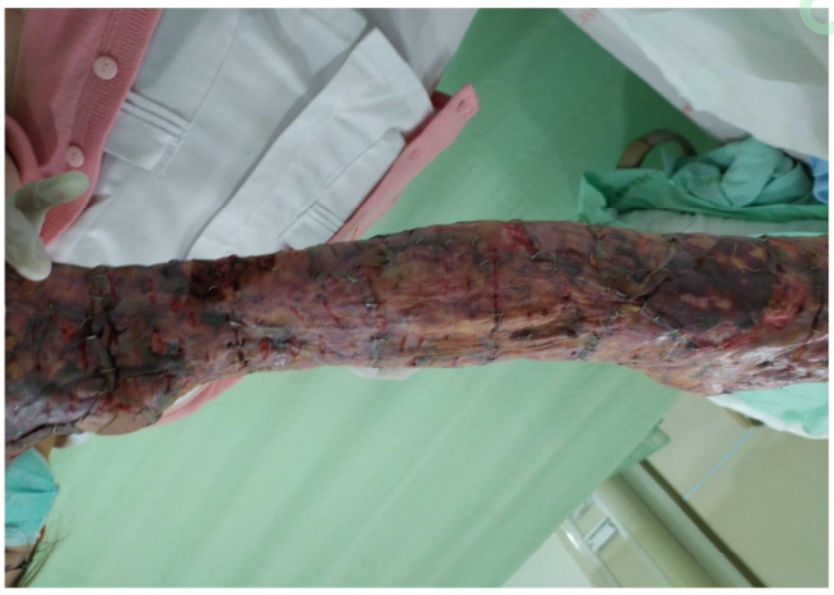
衛生福利部南投醫院高壓氧中心[1]

個 案 報 告 目 的

慢性傷口主要包括有靜脈性潰瘍、動脈性潰瘍、糖尿病潰瘍以及壓力性潰瘍，因受到局部血液灌流差、患者本身營養代謝低下、傷口感染或壞死範圍或局部高壓力、缺氧等多因素影響，甚至引發系統性及骨骼感染，耗費醫療資源，造成病患及家屬生活品質影響甚遠，已成為健康照護上不可忽視的忽視的議題。高壓氧的治療可提高組織氧分壓及氧含量，改善組織缺氧情形，可增加白血球抑菌或弒菌能力、控制感染、增加纖維母細胞形成及促進膠原蛋白合成，加速傷口癒合。

個 案 介 紹

67歲女性109.07因不明原因導致左下肢反覆蜂窩性組織炎，109年09、110年01月分別至他院行淋巴切開術，術後左大腿至足跟大面積傷口。患者共執行15 次2.3 ATA並配合每天一次換藥，傷口狀態從發黑、滲液多至外觀乾淨、滲液少明顯改善。



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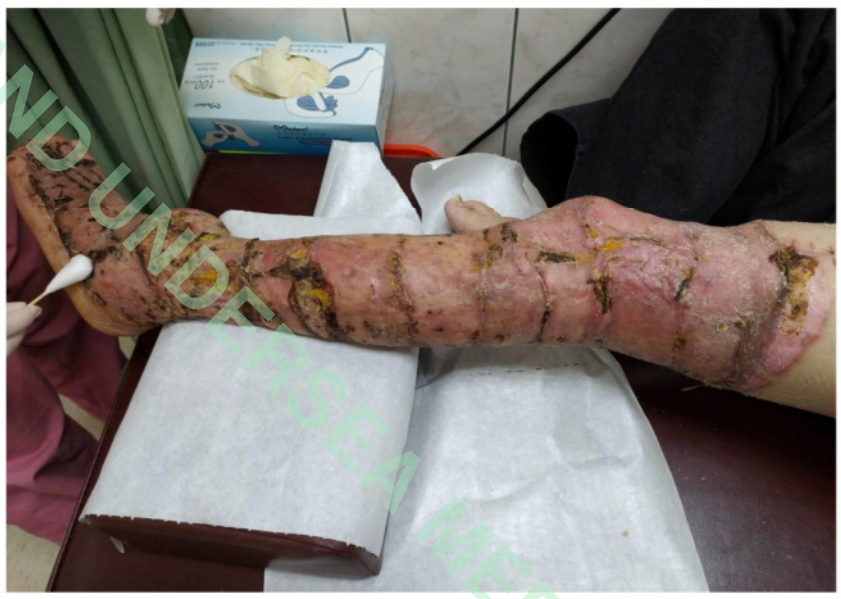

110-03

結 果 與 評 值

本案例無糖尿病史，其傷口癒合不良的原因有很多，多次開刀使得血流供應不佳、缺氧區域容易藏污納垢導致細菌感染反覆發生。綜論之，傷口重建需要內科藥物控制、外科血管重建或清創植皮手術治療、以及多次的高壓氧治療，才能獲得較滿意的療效。 高壓氧治療雖是輔助治療的角色，只要適當的使用，不但可以強化傷口感染控制，加速傷口癒合。所以，高壓氧氣做為慢性傷口的輔助治療應是值得繼續關注的。

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Nantou Hospital,Ministry of Health and Welfare

專業●品質●當責●創新

編號 14

2011至2020年最常被引用的100篇高壓氧治療研究分析

Top-100 cited Hyperbaric Oxygen Therapy Related Papers from 2011 to 2020

葉忻慈[1]、李景行[2,3]

[1]林口長庚紀念醫院 急診醫學部；

基隆長庚紀念醫院 [2]高壓氧治療中心 [3]急診醫學部

目的

高壓氧治療應用於臨床醫學和基礎實驗逐年增加，本研究以論文被引用次數(times cited)為參考，回顧近10年來最常被引用的高壓氧研究，評估高壓氧於各領域的重要研究主題、成果與趨勢，一方面做為了解10年來高壓氧醫學研究進展的變化，也能做為高壓氧臨床研究設計的參考。

方法

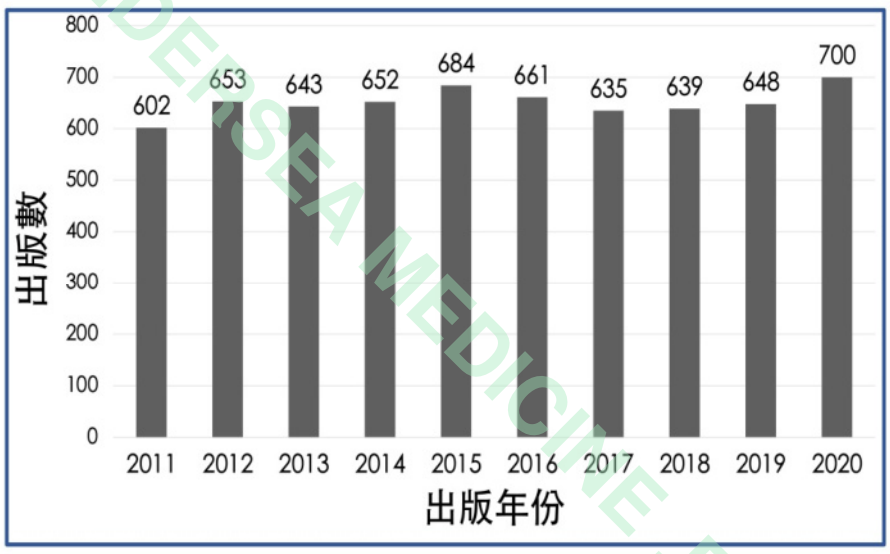
從Scopus資料庫以hyperbaric oxygen or hyperbaric oxygenation or HBO or HBOT為關鍵字搜尋2011至2020年相關論文，排除非以高壓氧為主要研究主題的研究，選出前100篇引用次數最高的論文，分析出刊年份、第一作者、國家、收錄期刊、期刊主題領域、研究對象、論文體裁(原著、回顧)。

結果

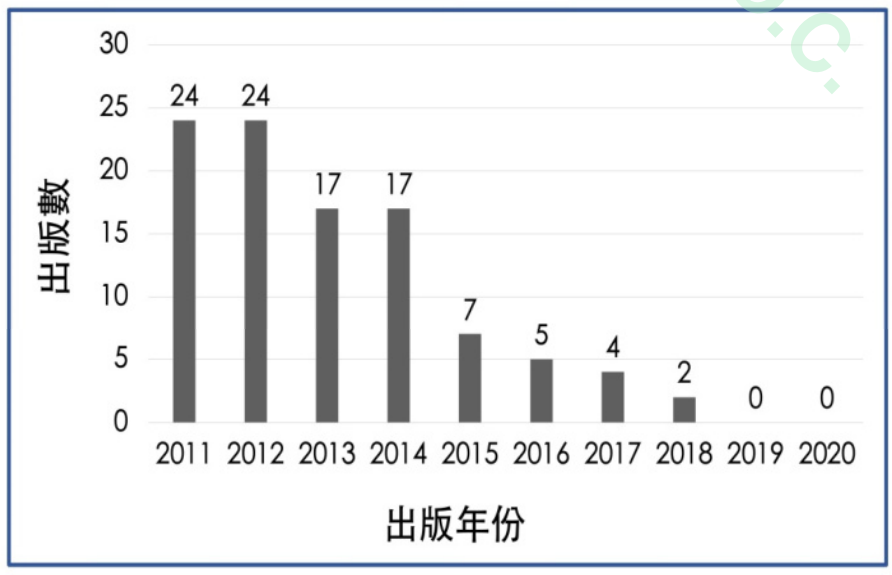
2011至2020年共有7562篇高壓氧相關論文(圖一)，以高壓氧為研究主題、被引用次數最多的100篇論文中，依刊出年份分析，2011年和2012年各24篇最多(圖二)，逐年遞減；依國家分析，包含16個國家，美國31篇最多，中國(16)次之，澳洲(11)第三；依作者分析，總共有82位作者，Bennett M.H. 6篇最多；依期刊分析，有65本期刊，Cochrane Database of Systematic Reviews和Undersea and Hyperbaric Medicine各6篇最多；依期刊領域分析，包含36個不同的領域，神經科學方面13篇最多，外科10篇居次，臨床神經學8篇第三；文章體裁方面，article 72篇最多，review 26篇第二。被引用最多次的論文為Thom S.R. 於2011年發表於Plastic and Reconstructive Surgery的Hyperbaric oxygen: Its mechanisms and efficacy，共被引用289次。

結論

近10年高壓氧相關研究論文，以臨床醫學領域佔大宗(56%)，其他還包含工程、化學、藥學、護理照護等，2011年開始每年有650-750篇論文發表，2018年後開始逐年上升，顯示最近三年高壓氧研究進入持續成長的階段。外傷性腦傷、腦部缺氧與糖尿病足為主題的研究，占最常被引用論文的37%，為近10年最熱門的研究方向，相較2000年到2010年以缺血性中風、一氧化碳中毒和放射線治療傷害相關為主有很大的不同，代表高壓氧在臨床上的應用開始有更廣的發展；最常被引用的論文國家前3名與2000-2010年前3名美國(52)，德國 (11)，加拿大(5) 相比，可以看出高壓氧研究雖仍由美國主導，但其他國家的研究正逐漸上升中。台灣有5篇進入名單，分別由高雄長庚、成功大學、奇美醫院、三軍總醫院發表，與2000-2010年2篇相比有明顯進步。2019至2020年並無論文成為前100篇最常被引用的研究，此情形可能因論文出刊後到引用該論文的研究出刊都存在時間上的延遲情形，故此期間發表論文的重要性可能被低估，熱門研究方向需要時間追蹤。



圖一：2011至2020年高壓氧相關論文的年出版量



圖二：2011至2020年引用次數前100的論文數量分布

長庚醫療財團法人
Chang Gung Medical Foundation
基隆長庚紀念醫院
Chang Gung Memorial Hospital,Keelung

由Undersea and Hyperbaric Medicine期刊1974至2020年論文看高壓氧醫學研究的全球化與國際合作趨勢

Globalization and International Collaboration of Hyperbaric Oxygen Therapy: Perspectives from Publications in Undersea and Hyperbaric Medicine, 1974 to 2020

李景行[1,2]

基隆長庚紀念醫院 高壓氧治療中心[1] 急診醫學部[2]

目的

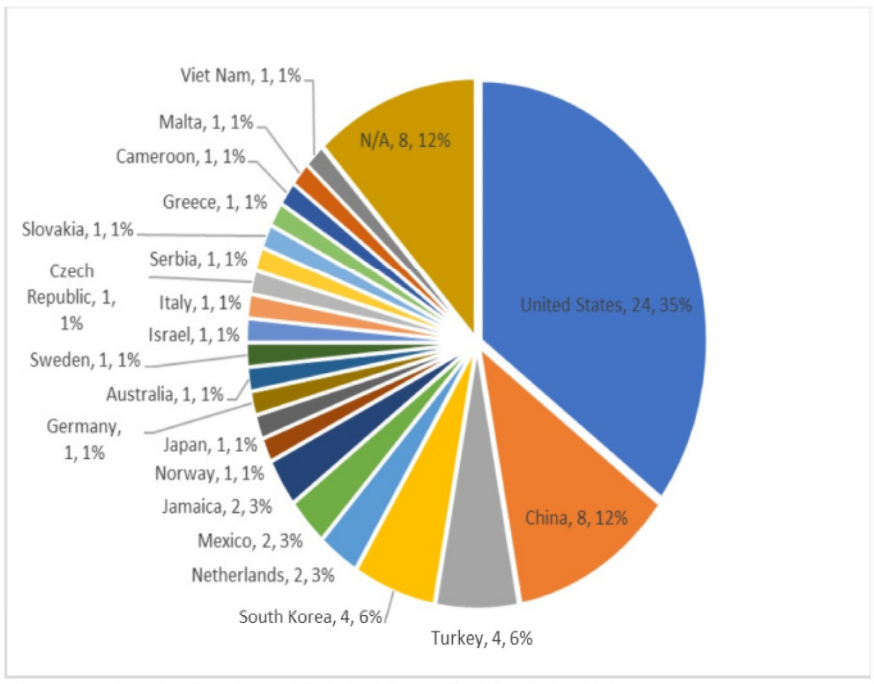
Undersea and Hyperbaric Medicine (*UHM*)為歷史最悠久的高壓氧醫學期刊，自1974年刊行至今47年，發表的論文反映過去40多年來全球高壓氧研究的變化趨勢。本研究分析*UHM*創刊以來論文作者國籍變化趨勢，做為未來高壓氧醫學研究國際合作的參考。

方法

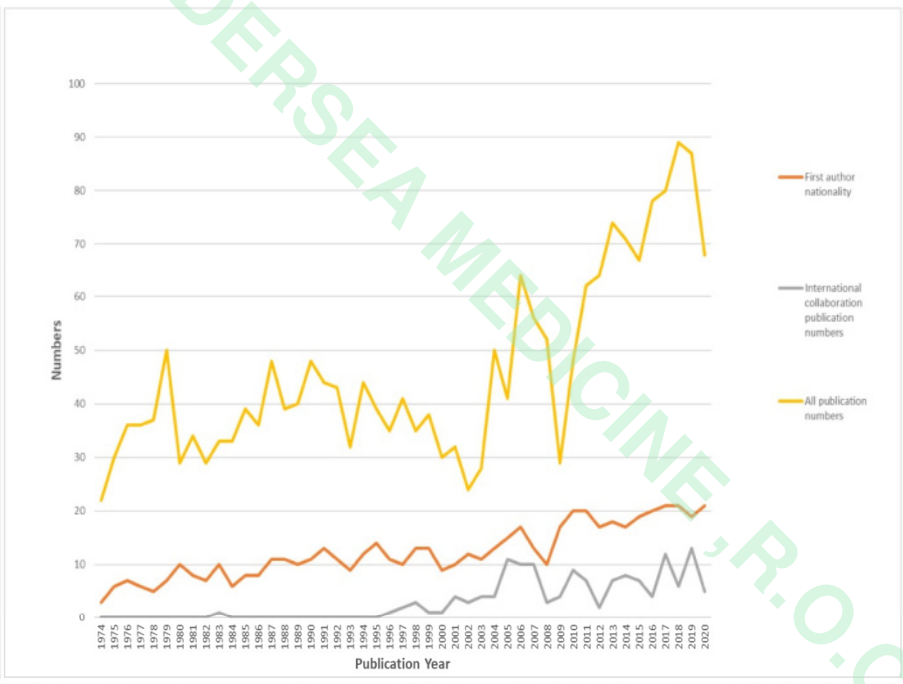
以 Scopus搜尋*UHM* 1974至2020年間發表的所有論文，擷取論文發表年份、作者國家、論文類別、引用次數進行分析。以第一作者國家定義為論文發表國家；作者均為同一國家定義為單一國家論文，作者來自兩國家以上定義為跨國合作論文。趨勢分析以線性迴歸的斜率代表增加趨勢。類別分析以chi-square statistic檢定差異。組別間論文被引用次數中位數差異，以Wilcoxon Rank Sum Test進行檢定。

結果

1974至2020年間，*UHM*共刊出2164篇文章，其中142篇(6.6%)為跨國合作論文，第一篇跨國合作論文發表於1983年。1974年的22篇論文，發表國家為美國(19, 86.4%)、瑞典、加拿大；2020年的68篇論文，由21個國家發表(圖一)，美國最多(24, 35.3%)，其中5篇(7.4%)為跨國合作論文。年度發表論文數、發表國家、跨國合作論文數(圖二)增加趨勢分別為0.927、0.323、0.219 (all p<0.001)。跨國合作論文分析方面，單一國家與跨國合作論文Article (1639, 112)與非Article (383, 30)類別比例無差異(chi-square statistic: 0.4102, p=0.521)。因2000年之前每年未穩定有跨國合作論文發表，故單一國家與跨國合作論文引用次數分析期間為2001至2020年，論文數分別為987、133，平均引用次數為5.90、9.68，引用次數中位數為2、4 (p<0.001)。



圖一：2020年UHM論文第一作者國家數量



圖二：1974至2020年UHM論文、發表國家、跨國合作論文趨勢

結論

47年來*UHM*高壓氧論文發表數量、各國高壓氧研究與跨國合作逐年正成長，顯示在高壓氧醫學研究為全球化且持續成長的領域，跨國合作研究也逐年增加，美國持續為高壓氧醫學研究的領導國。單一國家與跨國合作論文種類上並無差異；引用次數方面，跨國合作論文高於單一國家論文，可能原因為具備跨國合作研究能力的作者群擁有較多研究經驗與資源，能針對具跨國重要性主題進行研究發表，故研究結果影響力較大，被引用次數較多。未來高壓氧醫學研究，應從主題選取、方法設計、研究資源等面向，以取得國際合作機會為目標，提高研究結果影響力。

編號 16

高壓氧治療BK病毒相關的出血性膀胱炎-案例報告

Hyperbaric Oxygen Treatment of BK Virus-related Hemorrhagic Cystitis-Case Report

劉家妤[1]、廖怡秋[1]、黃馨卉[1]、陳良媛[1]、賴妍廷[1]、劉姿萱[1]、林任家[1,2]
[1]中山醫學大學附設醫院高壓氧醫學科 [2]中山醫學大學附設醫院骨科部

前言

出血性膀胱炎(hemorrhagic cystitis; HC)的定義是由於感染性或非感染性病因導致膀胱粘膜出血而引起的膀胱瀰漫性炎症。是造血幹細胞移植(hematopoietic stem cell transplantation, HSCT)術後常見的併發症。臨床表現從微小的血尿到嚴重的出血，並伴有血塊形成和尿路阻塞，甚至在慢性和反復發作時導致腎積水和急性腎衰竭。

早發性HC發生在移植後的第一週，通常與高劑量化學治療或全身放射治療引起的尿道上皮損傷有關，通常是短暫和局限的。相反，遲發性HC可能是危險的並且可能危及生命的疾病，並且經常與不同病毒感染有關，尤其以BK病毒最多。

BK病毒是多瘤病毒家族(polyomavirus family)的成員，為不具莢膜的雙股DNA病毒。多數人在幼兒時期就曾感染過，病毒會潛伏在泌尿道系統作為伺機性的感染，當免疫力功能受到抑制時再活化。HSCT術前必須使用特定的高劑量化學治療或全身放射治療，引起尿道上皮細胞的損害，術後需服用免疫抑制劑預防移植體抗宿主疾病(GVHD)，因此容易使潛伏的BK病毒再活化，引起出血性膀胱炎(BK virus-associated hemorrhagic cystitis, BKHC)，嚴重將可能威脅生命或導致移植失敗。

個案簡介

個案為6歲女童，患有急性淋巴芽球性白血病ALL (Acute lymphoblastic leukemia)，接受化學療法及幹細胞移植一個多月後，併發出血性膀胱炎，血清中檢驗出BK病毒，除投予抗病毒藥物及症狀治療外，另外接受2.5ATA/60分鐘高壓氧輔助治療共30次。由原來的血尿中存有肉眼可見血塊，療程中進步為肉眼可見血尿，治療結束後尿液呈淡黃色，腹部超音波檢查顯示膀胱無明顯血凝塊。

討論

目前國際尚未建立BKHC的治療準則，臨床處置以免疫抑制劑減量和給予抑制BK病毒複製的藥物為主，其他包括膀胱沖洗，輸血和症狀緩解治療等。高壓氧治療可有效應用在各種原因造成的組織損傷和受傷，如燒傷、糖尿病引起的細胞損傷、放射性損傷等。將其應用於輔助治療BKHC的機轉為，使氧氣作用在組織癒合不良的區域，提高動脈血氧分壓，促進纖維母細胞增殖，使毛細血管新生，減輕水腫，使血管內皮生長因子上升，一氧化氮降低導致血管收縮，細胞增殖和遷移，修復受損尿路上皮細胞，臨床上有越來越多的病例報告證明高壓氧應用於輔助治療BKHC具有良好的成效。

編號 17

中山醫學大學附設醫院
Chung Shan Medical University Hospital

高壓氧治療毒蛇咬傷引發毒性多發神經病變後遺症之個案報告

A case report of hyperbaric oxygen assisted treatment of sequelae of toxic multiple neuropathy caused by snake bite

廖怡秋[1]、劉家妤[1]、黃馨卉[1]、陳良媛[1]、賴妍廷[1]、劉姿萱[1]、林任家[1,2]
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前言

全世界蛇類有3500種以上，毒蛇約佔400種。臺灣氣候溫暖潮溼，毒蛇約有23種，其中較常見的有6種。健保資料顯示每年約有1000名以上的民眾遭毒蛇咬傷。毒蛇咬傷症狀又分為出血性、神經性和混合性。出血性症狀，咬傷部位會有疼痛、局部瘀血、腫脹、水泡等症狀若未經治療，則腫脹會迅速由咬傷處向上擴展，肌肉壞死、血小板降低與各器官出血，甚至血壓下降、昏迷等嚴重狀況；神經性症狀以神經肌肉麻痺為主要症狀，如複視、眼瞼下垂、吞嚥困難、肌肉無力、瞳孔擴大、呼吸衰竭等；混合性則會產生兼具出血性及神經性的中毒症狀。

個案簡介

54歲王先生有肝硬化和第二型糖尿病病史，3年前右手不慎被毒蛇咬傷，隨後產生右手掌腫脹、頭暈和嘴唇麻木等症狀，被送至本院急診，一小時後開始呼吸急促，血氧濃度降低，因此進行了氣管插管及收入加護病房治療。出院後仍然有嘴唇麻木、四肢麻痺且異常性疼痛等症狀，深感困擾。2年前開始配合高壓氧輔助治療，頻率為一週兩次，半年後主訴四肢麻木症狀改善，治療頻率改為一週一次，持續治療共86次。

討論

國際疼痛研究協會(International Association for the Study of Pain，IASP)將神經性疼痛(Neuropathic pain，NP)定義為中樞或周圍神經系統直接受傷或功能障礙引起的疼痛。NP是一種慢性疼痛，其特徵包括自發性疼痛、痛覺敏感和異常性疼痛等。引發發病原因和機制複雜，包括代謝性(例如糖尿病性神經病變），傳染性(例如帶狀皰疹後神經痛)，自身免疫性(例如多發性硬化症），血管性疾病(例如中風)，神經性(例如三叉神經痛)和癌症在內的所有因素均可導致。高壓氧可以透過增加一氧化氮(NO)的產生來改善血液循環及抑制星形膠質細胞活化和炎症反應，從而減輕疼痛。對於使用傳統的治療例如藥物或經皮電神經刺激(TENS)沒有明顯的效果，又不想做進一步做侵入性治療例如和脊髓刺激(SCS)的患者而言，高壓氧可能為神經性疼痛的治療提供新的目標。

醫人 醫病 醫心
To treat patients, cure diseases, most of all, to care holistically.

中山醫學大學附設醫院
Chung Shan Medical University Hospital

編號 18

Early Intervention of Hyperbaric Oxygen in the Treatment of Burns Injury：A Case Report

莊力錡1、許婷喻1、陳思潔1、蔡佩娟1、魏誠佑2、李宗勳1

秀傳醫療財團法人彰濱秀傳紀念醫院前瞻傷口醫學部高壓氧中心 1
神經內科部2

個案簡介：

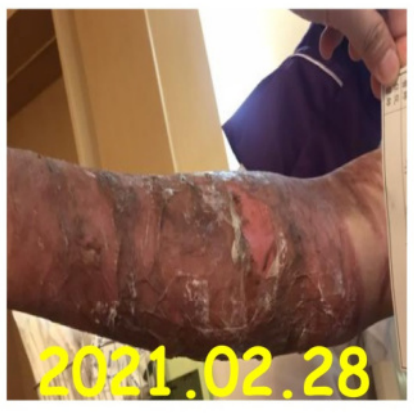
59歲男性，有右膝人工關節置換術病史，2021年2月24日工作被高壓蒸氣鍋熱氣燙傷，至本院急診就醫，診斷臉、頸、前胸、雙前臂、雙大腿內側有2-3度燒燙傷，總體表面積大於15%，轉介至本中心，進行傷口護理及高壓氧輔助治療。

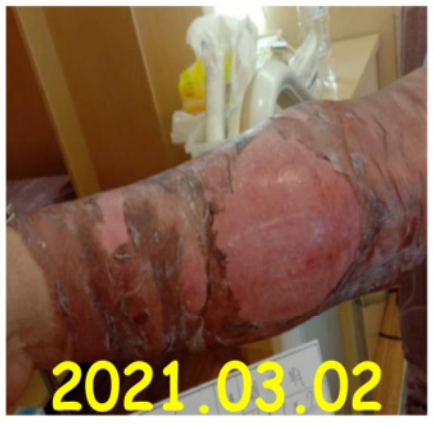
討論：

高壓蒸氣鍋一般溫度設定在121~132℃，皮膚一旦接觸40~44℃以上的熱源，導致皮下組織蛋白質結構受到破壞；而常見以燒燙傷總體表面積(TBSA)評估燒燙傷嚴重度，臨床上常用九分法則計算法來估算，而皮膚受傷深度分為：（1）一度：表皮層（2）淺二度：部份真皮表層（3）深二度：真皮深層（4）三度：全層皮膚（5）四度：全皮層、皮下組織、肌肉、骨骼。常規治療有體液補充、抗生素治療、傷口護理，再配合高壓氧輔助治療，有助迅速消除水腫，減少體液、電解質和血漿之流失及預防感染。

結果：

個案自2021年2月25日至2021年3月4日行高壓氧治療，壓力2.3ATA，共5次。個案於48小時內緊急高壓氧治療，觀察其皮膚變化由發紅逐漸轉為多重性水腫(水泡)，在第5次治療後，臉、頸部、前胸、雙大腿內側及左手前臂受損皮辦已完全角化脫屑，外觀為新生之表皮，但右手前臂仍須傷口護理，於2021年3月8日癒合。





結論：

皮膚為人體第一道免疫防線，燒燙傷患者最主要死亡原因為感染，個案若能早期介入高壓氧輔助治療，則可降低傷口感染風險及敗血症機會，增加復原成效，由此個案治療過程中顯示高壓氧治療對燒燙傷是有幫助的。

編號 19

秀傳醫療財團法人彰濱秀傳紀念醫院
Chang Bing Show Chwan Memorial Hospital

運用多媒體數位影音介入高壓氧治療前衛教的成果探討

Use Multimedia Digital Audio and Video Discussion on the Achievement of Interventional Hyperbaric Oxygen Treatment

陳思潔[1,2]、蔡佩娟[1,2]、黃琪雯[1,2]、莊力錡[1,2]、魏誠佑[1,3]、李宗勳[1,2]

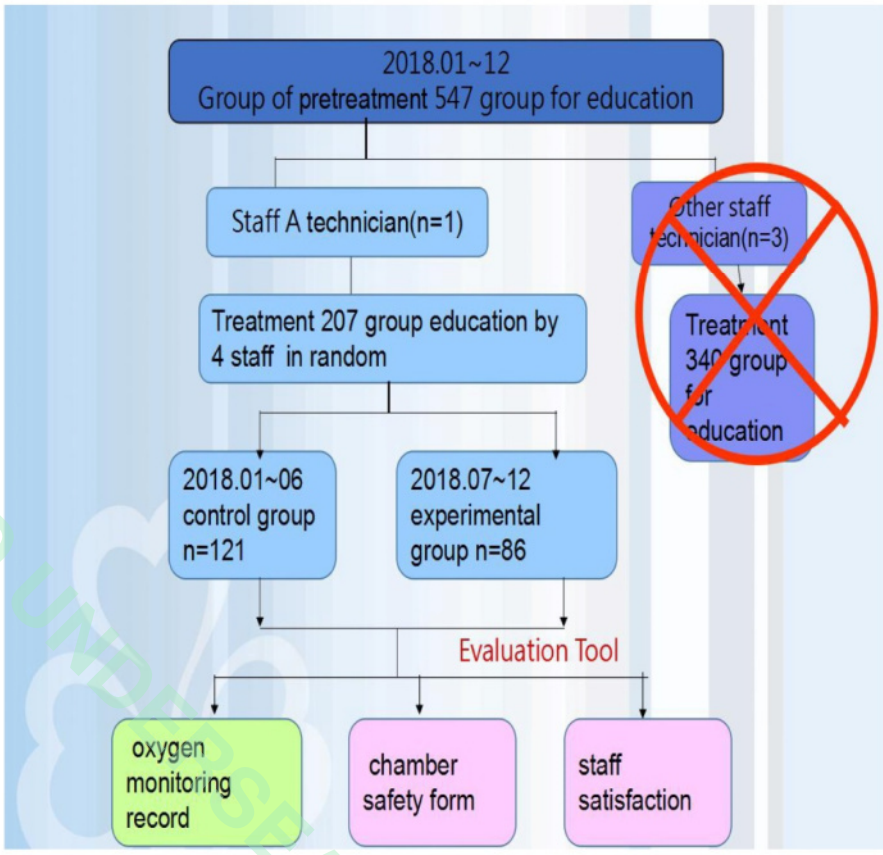
秀傳醫療財團法人彰濱秀傳紀念醫院 [1]高壓氧中心 [2]前瞻傷口中心 [3]神經內科部

目的：

高壓氧治療不僅有促進疾病改善的功效外，並且不會對人體造成任何的神經後遺症，是一項安全的輔助醫療行為；但其治療前的準備工作相當繁瑣，不僅需教導病患使用治療面罩及加壓、減壓期的注意事項外，技術人員與家屬仍會擔心患者進艙後執行動作不確實，導致治療失敗或引起其它爆炸安全等問題。因此，為了讓高壓氧治療的衛教方式更淺顯易懂，本中心將探討介入多媒體數位影音的衛教方式，是否能改善治療期間常見的相關問題。

方法：

採回朔性統計彰濱秀傳紀念醫院2018年1月至12月的HAUX-2000高壓氧艙治療艙數，共開547艙次，總共1636人次，為使統計數據更有價值且確保艙操方式一致，僅採用同一位技術員的操艙記錄共207艙次，而對照組僅以口頭紙本方式衛教為121艙次，實驗組介入多媒體影音衛教為86艙次，並以5分法設計病患滿意度、技術員對於病患治療執行的滿意度量表，其中使用氧氣分析儀監測艙內氧氣濃度漏氧情形統計其平均標準差來評估治療的安全性，再依據治療中造成耳擠壓疼痛患者的數量百分比來瞭解治療品質是否提升。



結果：

病患滿意度由3.52分提升至4.65分，技術員對於病患治療執行的滿意度由3.33分提升至5.25分，艙內氧氣濃度監測記錄漏氧情形由21.5%(±0.25)下降至21.3%(±0.16)，治療中造成耳擠壓疼痛患者數量由0.86%下降至0.61%。

結論：

高壓氧治療常規以療程為主，治療過程不僅冗長且無趣，且病患因為疾病關係，需以高壓氧合併輔助其它治療，以縮短疾病病程，然而瑣碎的衛教內容常讓身體不適的人們無法專心聆聽，但為了確保治療安全，醫療人員仍須事倍功半的仔細講解說明與指導。因此，藉由本篇研究統計結果得知，透過多媒體數位影音的方式介入衛教，不僅增加了病患及技術員的滿意度以外，還提高了治療過程的安全性，降低病患因環境壓力造成的耳痛情形，亦幫助患者不因耳痛而中斷治療，大大改善治療品質及反覆衛教的困擾，進而達到事半功倍的成效！

高壓氧治療導致中樞神經氧中毒之發生率及風險因子分析

Incidence and the Risk Factors of Central Nervous System Oxygen Toxicity during Hyperbaric Oxygen Therapy

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三軍總醫院　胸腔內科　高壓氧中心

Purpose

Oxygen-toxic seizure is a rare but serious side effect of hyperbaric oxygen therapy (HBOT), and the real-world data is absent in Taiwan. The objective of this study was to analyze the incidence and the risk factors of HBOT-associated central nervous system (CNS) oxygen toxicity in a medical center of Taiwan.

Materials and Methods

This is a retrospective observational study of consecutive patients receiving HBOT in Tri-service General Hospital from January 2012 to December 2020. All patients with seizure during HBOT were recorded. We excluded subjects with seizure due to hypoglycemia. Demographic and clinical data were extracted for analysis.

Results

A total of 52753 chamber sessions of HBOT (5936 patients) were done during this period, and 23 episodes (22 patients) of oxygen-toxic seizure were recorded. The incidence of CNS oxygen toxicity was 0.417‰ (Figure 1, Table 2).

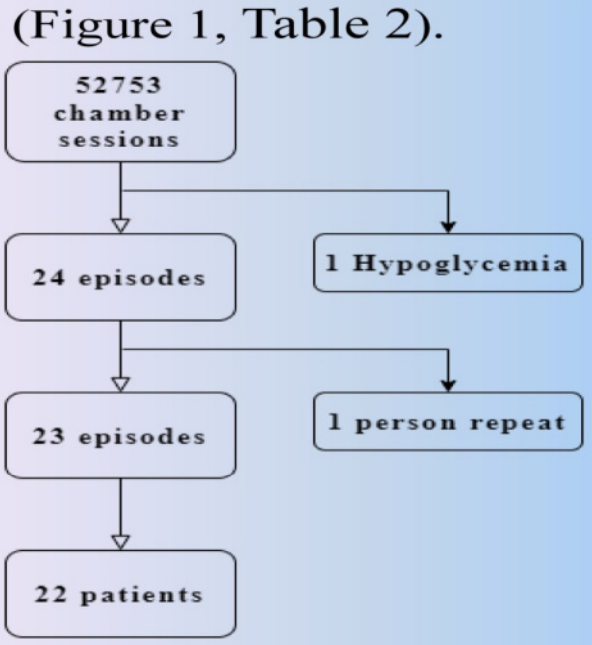

Figure 1

The maximal compressing pressure > 2.0 ATA and the diagnosis of acute carbon monoxide poisoning are the risk factors associated with CNS oxygen toxicity in patients receiving HBOT (Table 1).

Table 1

Gender	patients	percent	Time	patients	percent
Male	9	41%	Descending	2	9%
Female	13	59%	Bottom	18	82%
Age			Ascending	2	9%
<30	5	23%	**Depth(FT/SW)**		
30-40	4	18%	33	2	9%
40-50	4	18%	50	13	59%
50-60	6	27%	60	2	9%
>60	3	14%	66	5	23%

Among these episodes, 12 (55%) occurred during the treatment for acute carbon monoxide poisoning (Table 2). A subgroup analysis showed that 83% (10/12) oxygen-toxic seizure occurred during the first 3 chamber sessions in patients with the diagnosis of acute carbon monoxide poisoning (Figure 2).

Table 2	Number of treatments	Patients	Incidence (‰)
CO	5270	12	0.227
NF	1563	2	0.038
OM	4860	2	0.038
SD	5729	2	0.038
Radiation cystitis	6724	1	0.019
Hypoxic	3869	1	0.019
DCS	1636	1	0.019
Pressure and Oxygen resistance	765	1	0.019
Chamber sessions	52753	22	0.417

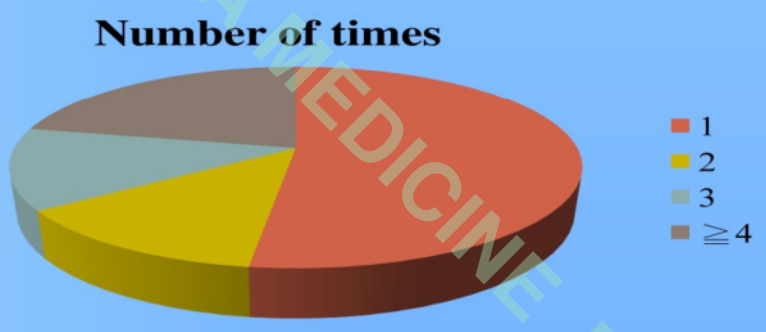

Figure 2

Conclusions

The incidence of HBOT-associated CNS oxygen toxicity in our patient population is greater than historical reports, which may be associated with the higher prevalence of acute carbon monoxide poisoning in Taiwan. Oxygen-toxic seizure may occur during the first 3 chamber sessions of HBOT for these patients.

編號 21

中華民國高壓暨海底醫學會110年會員大會暨學術研討會壁報論文
ASSOCIATION OF HYPERBARIC AND UNDERSEA MEDICINE OF REPUBLIC OF CHINA

突發性耳聾成人病患進行高壓氧治療之回朔研究

王旭輝 王家弘 張厚台
亞東紀念醫院高壓氧中心

Retrospective Study of Adult Patients with Idiopathic Sudden Sensorineural Hearing Loss Treated with Hyperbaric Oxygen Therapy

Hsu Hui Wang Jia-Hung Wang Hou-Tai Chang
[1]Hyperbaric Oxygen Treatment Center, Far Eastern Memorial Hospital, Taiwan

Objective

This retrospective study examined the adult patients with idiopathic sudden sensorineural hearing loss (ISSNHL) treated with hyperbaric oxygen therapy (HBOT), and if there is prognostic factor predicting better hearing recovery chance .

Material and Method

From 2017 to 2019 ,patients receiving HBOT in Far Eastern Memorial Hospital due to ISSNHL were included in study. Patients without follow up audiogram, not meeting criteria of ISSNHL, receiving HBOT ≤2 times, age under 20 years old , interval from symptoms to HBOT >180 days, or known etiology were excluded. Hearing improvement was analyzed using average hearing level of 250Hz, 500Hz, 1KHz,2KHz,4KHz in pure tone audiogram. Hearing recovery is defined as hearing level improvement ≥10dB, and further classified into complete recovery (All 5 frequencies within20dB),good recovery(≥30dB),fair recovery(10-29dB), and no recovery(<10dB). Age, grade of initial hearing loss, interval from symptoms to HBOT, and if receiving intratympanic steroid injection(ITSI) was compared to see if significant difference of hearing recovery exist.

Result

79 patients were included in analysis. 78 of 79 patients received systemic steroid before or during HBOT and 46 patients received ITSI. Total hearing recovery rate was 58.23%. 27(34%) patients had complete or good hearing recovery. If the patients received HBOT within 14 days from symptoms onset , the hearing recovery rate increased to 83.33%. There were no significant difference of hearing recovery when compared age, grade of initial hearing loss, and if receiving ITSI.

TABLE I
PATIENTS' DEMOGRAPHIC AND CLINICAL CHARACTERISTICS

Characteristic	Value
TOTAL	79
Age (Years)	
- Range (Mean)	
- >=60	27
- < 60	52
Sex	
- Male	42
- Female	37
Side	
-Right	37
-Left	42
Grade (averaged hearing level)	
-1 (<=40dB)	1
-2 (41-60dB)	9
-3 (61-90dB)	36
-4 (>90dB)	33

Data represent numbers of patients unless indicated otherwise.

DISTRIBUTION OF HEARING IMPROVEMENT AFTER TREATMENT

Characteritics	Hearing improvement ? (n) Yes	No	Improvement rate (%)
Oveall improvement rate			
-Complete Recovery	6		7.59
-Good Recovery	21		26.58
-Fair Recovery	19		24.05
-Total Recovery	33		58.23
-No Recovery		33	41.77
Total n= 79	79		
Age (years)			
>=60 yrs	16	11	59.26
< 60 yrs	30	21	58.82
Grade (averaged hearing level)			
-1(30-40dB)	1	0	100
-2(41-60dB)	3	6	33.33
-3(61-90dB)	23	13	63.89
-4(>90dB)	19	14	57.58
Duration from symptoms to HBOT			
0-7 days	15	1	93.75
8-14 days	20	6	76.92
0-14 days	35	7	83.33
15-30 days	8	12	40
31-180 days	3	17	17.65
Intratympanic steroid injection			
-Yes	28	21	57.14
-No	18	12	60

Conclusion

In our experience, adult patients with ISSNHL received HBOT and systemic steroid within 2 weeks from onset of symptom had better hearing recovery chance. We look forward further prospective trial with multi-centers and multidisciplinary team approach for further proof.

Reference :

1 Hughes GB, Freedman MA, Haberkamp TJ, Guay ME. Sudden sensorineural hearing loss. Otolaryngol Clin North Am 1996;29: 393–405

2 Goto F, Fujita T, Kitani Y, Kanno M, Kamei T, Ishii H. Hyperbaric oxygen and stellate ganglion blocks for idiopathic sudden hearing loss. Acta Otolaryngol 1979;88:335–42

3 Fujimura T, Suzuki H, Shiomori T, Udaka T, Mori T. Hyperbaric oxygen and steroid therapy for idiopathic sudden sensorineural hearing loss. Eur Arch Otorhinolaryngol 2007;264:861–6

亞東紀念醫院 FAR EASTERN MEMORIAL HOSPITAL

一氧化碳中毒患者高壓氧療法的成功治療

Successful treatment with hyperbaric oxygen therapy for patients with Carbon Monoxide

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Purpose: ntoxication by carbon monoxide (CO) is a very serious problem, CO binds to hemoglobin with an affinity known to be 200-300 times greater than oxygen, generating carboxyhemoglobin (HbCO). This reduced oxygen delivery to cells is the explanation for limitations in the oxygen consumption of cell . Hyperbaric oxygen therapy (HBOT) has been used to treat patients with CO intoxication and most of treated cases have shown improvement.

Methods : We collected data which reported CO intoxication that was treated with HBOT in Far Eastern Memorial Hospital from 2017 Jan. to 2020 Dec. The data collected about 162 patients into the HBOT. Data collect about age. sex and so on. to compare the means, compared t test, chi-square test were performed using SPSS Statistics version 19.0(IBM,Armonk,New York,USA).

Results: Totally 162 patients were included (age :36.7±14.9 years, sex: 77male/85 female, the hours of CO intoxication until to hospital 19± 95.6,the days of CO intoxication until to HBOT 2.19±4.48.the mechanical ventilation).the percentage of mechanical ventilation:27/135(16.7%),the successful weaning rate of mechanical ventilation :25/27(92.5%),the rate of delayed neurological sequelaie:4/162(2.4%), After HBOT , The leve of COHb :78.9%±to 3.5:0.4%±0.16 ($p<0.01$), MMSE:26.64±2.7 to 29.0±1.59 ($p<0.01$),the GCS scale:13.25±3.48 to 14.43±1.82 ($p<0.01$). There was no influence of age, sex on the occurrence of CO intoxication. Generalized estimating equations identified only COHb(adjusted odds ration: 257131, p=0.014).,GCS(adjusted odds ration: 0.275, p=0.001), troponine T(adjusted odds ration: 0.957, p=0.001)as an depedent variables of MMSE

Conclusion : The timing of the HBO2 is important and derives the most benefit if done within the first 6 hours after exposure. HBO2 helps at reducing the risk of DNS by helping with the ischemia-reperfusion injury in the central nervous system. prevention of morbidity from delayed neurological sequelae (DNS)represents a substantial cost savings to the health care system

Keywords: carbon monoxide (CO), Carboxy Hemoglobin (HbCO), Hyperbaric oxygen therapy (HBOT), delayed neurological sequelae (DNS)

連續變數資訊

		個數	最小值	最大值	平均數	標準離差
依變數	GCS	324	3	15	13.84	2.817
共變量	性別	324	.0	1.0	.475	.5002
	年紀	324	4.0	74.0	36.790	14.9129
	使用呼吸器	324	0	1	.17	.373

參數估計值

參數	B 之估計值	標準誤差	95% Wald 信賴區間		假設檢定			Exp(B)	Exp(B) 的 95% Wald 信賴區間	
			下界	上界	Wald 卡方	df	顯著性		下界	上界
(截距)	14.536	.1215	14.298	14.775	14309.781	1	.000	2056222.610	1620443.949	2609193.254
[指標1=1]	-1.179	.2023	-1.575	-.783	33.973	1	.000	.308	.207	.457
[指標1=2]	0[a]	.	.	.	.	.	.	1	.	.
脫離呼吸器	-8.947	1.4251	-11.740	-6.154	39.415	1	.000	.000	7.969E-6	.002
(尺度)	6.646									

依變數：GCSGCS

模式：(截距), 指標1, 脫離呼吸器

a. 設定為零，因為這個參數是冗餘的。

病例報告：高壓氧輔助治療瀰漫性軸索損傷

Case Report：Adjunctive hyperbaric oxygen therapy for Traumatic Diffuse Axonal Injury

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彰化基督教醫院[1]高壓氧中心、[2]骨科部及[3]胸腔外科

個案報告：

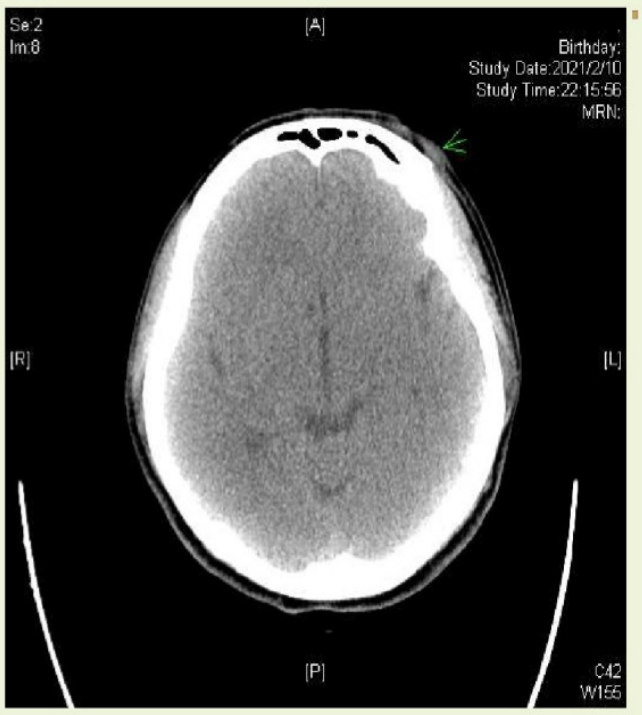
瀰漫性軸索損傷(Diffuse axonal injury, DAI)為頭部外傷後造成意識喪失及持續性植物人狀態的重要原因。臨床表現嚴重神經失能但影像檢查無法解釋時，應考慮DAI；其成因起源於外力使頭部發生加速-減速的甩鞭作用，導致神經細胞體與軸突交接處有剪力作用(shearing force)而造成損傷與出血[1]。症狀可能包括：頭痛、頭暈、噁心、嘔吐、意識喪失、記憶嚴重衰退、注意力渙散、判斷力薄弱或平衡困難、視力模糊、聽力障礙、嗅覺改變、行為人格異常、睡眠障礙和心情變化等。

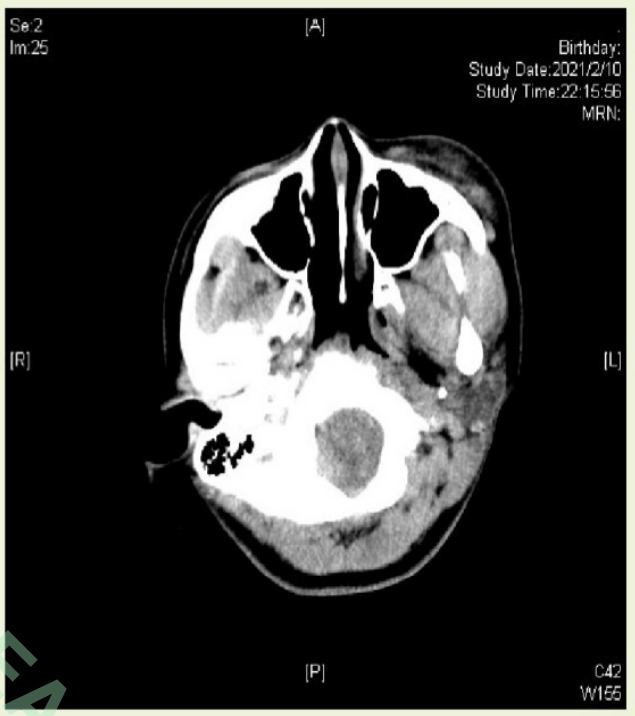
個案19歲女性，於2021年2月10日乘坐機車後座，發生車禍被拋出，當下導致意識喪失情形，左手、左眉骨上方撕裂傷，左膝擦傷，由119送至彰基急診就醫(GCS：E2M5V2)；腦部電腦斷層顯示無急性腦出血、X ray無骨折情形，外傷腹部超音波無肋膜積液，入加護病房觀察頭部外傷引起瀰漫性軸突損傷相關症狀，外傷嚴重度分數75分。患者有頭脹痛、眩暈、血壓不穩定，伴隨噁心、嘔吐；陸續出現專注力渙散、語言邏輯思考能力及記憶力下降、行為人格退化，需將床頭抬高30度臥床休息；此外，有嚴重的視力模糊失明情況。安排相關神經學檢查、眼科檢查有小部分組織水腫但無發現異常神經損傷情況；10天後，考慮是否能介入高壓氧輔助治療，由高壓氧專科醫師評估後，給予安排治療。

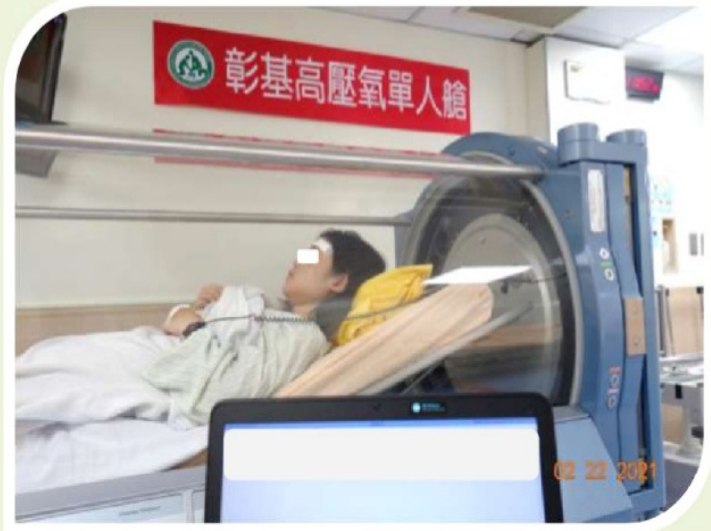
疾病急性期，每天給予兩大氣壓，每次60分鐘，共20次的高壓氧治療；完成第1次的高壓氧治療之後，頭暈頭痛的症狀就大幅減輕，連續5次的治療視力模糊的情況就完全改善。

治療期間觀察到的特殊狀況：(1)在第8次治療後的夜間，病人因頭痛劇烈，緩解後又出現視力模糊行為退化症狀，在止痛藥劑量調整後，予連續高壓氧輔助治療之下，出院前視力恢復正常。(2)在第14次治療後，病人因為爬樓梯的動作，出現頭痛、頭暈、視力模糊症狀，在臥床2小時休息後，視力恢復正常。建議未來在出院衛教時，要特別加強：如何避免導致顱內壓升高的動作及相關處理。

Chelly等人研究顯示DAI佔頭部創傷住加護病房病比例13.9%，以交通事故92%最多，外傷性病人神經損傷失能，若未能在電腦斷層上看到病灶或病灶無法解釋臨床症狀時，宜加做MRI，確認病灶深度以預期相關預後[2]。治療方面，尚無明顯突破；提供積極綜合性治療可改善患者的不良預後，包括：一般治療、控制顱內壓、手術治療、低溫治療、早期高壓氧治療、神經康復治療、預防併發症等。







➢ **CT scan：**
Soft tissue swelling and hematoma at the left frontal and orbital region is seen, favors due to post trauma.

使用單人艙治療，相關經驗分享：

1. 顏面損傷患者：不須使用氧氣面罩吸氧，沒有傷口壓迫，或因面罩密合度不良，導致漏氣造成艙壓提升或是多人艙內氧氣濃度增加的問題。
2. 腦部受傷患者：治療壓力的微調、加壓速度的客製化調整，提高患者安心舒適感；此外，如果合併暈眩無法久坐情況，臥位治療是另外考量的重點，且床頭抬高並保持頭頸一直線可促進靜脈回流及CSF引流，進而降低ICP。
3. 失明的患者：複雜簡單化，提高患者的理解力與治療的配合意願，對於疾病的連續治療是相當重要的。

聖經 箴言17:22『喜樂的心乃是良藥，憂傷的靈使骨枯乾。』

References：

1. Graham DI, Clark JC, Adams JH, Gennarelli TA. Diffuse axonal injury caused by assault. J Clin Pathol. 1992;45(9):840-841.
2. Chelly H, Chaari A, Daoud E, et al. Diffuse axonal injury in patients with head injuries: an epidemiologic and prognosis study of 124 cases. J Trauma. 2011;71(4):838-846.
3. 鄧允武(Yun-Wu Deng), 刁偉民(Wei-Ming Tiao), 謝盛林(Cheng-Lin Hsieh), 張亮霖(Liang-Lin Chang). 瀰漫性軸突損傷. 台灣急診專科醫師期刊. 2012;4(4):109-110.
4. 余佳倫(Jia-Luen Yu). 照顧一位瀰漫性軸突損傷病人之護理經驗. 志為護理-慈濟護理雜誌. 2019;18(5):111-121.
5. 溫恪瑩(Ko-Ying Wen), 張源驛(Yuan-Yi Chang), 侯毓昌(Yu-Chang Hou). 中西合治外傷性瀰漫軸突損傷－病例報告並文獻探討. 中醫藥雜誌. 2018;29(1):75-87.
6. Figueira Rodrigues Vieira G, Guedes Correa JF. Early computed tomography for acute post-traumatic diffuse axonal injury: a systematic review. Neuroradiology. 2020;62(6):653-660.
7. Humble SS, Wilson LD, Wang L, et al. Prognosis of diffuse axonal injury with traumatic brain injury. J Trauma Acute Care Surg. 2018;85(1):155-159.
8. van Eijck MM, Schoonman GG, van der Naalt J, de Vries J, Roks G. Diffuse axonal injury after traumatic brain injury is a prognostic factor for functional outcome: a systematic review and meta-analysis. Brain Inj. 2018;32(4):395-402.

編號 24

高壓氧處理退化性關節炎關節軟骨細胞微小核醣核酸及相關訊號傳遞的調控

Hyperbaric oxygen intervention regulated microRNAs related signaling in human osteoarthritic chondrocytes

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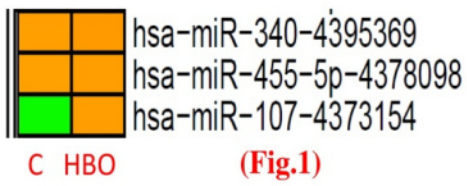
[1]林口長庚醫院骨科部；[2]林口長庚醫院骨骼關節研究中心；[3]長庚大學醫技系；[4]長庚科大護理系;[5]義大醫院骨科

Purpose: MicroRNA (miRNA) are a class of noncoding small RNAs that act as regulators of gene expression. This study was to determine miRNA changes in OA chondrocytes after hyperbaric oxygen (HBO) treatment.

Materials & Methods: OA chondrocytes were separated from the knees of OA patients. The hyperoxic cells were exposed to 100% O_2 for 25 min and then to 5% CO2/95% air for 5 min at 2.5 ATA in a hyperbaric chamber. Control cells were maintained in 5% CO_2 / 95% air throughout the experiment. MicroRNA expression profiling was performed using TaqMan MicroRNA Cards and ABI 7900 Real Time PCR System. MiRNA targets were identified using bioinformatics and a luciferase reporter assay. MiR-107 mimic was transfected and the HMGB-1 was analyzed in OA chondrocytes. After HBO treatment, the mRNA or protein levels of HMGB-1, RAGE, TLR2, TLR4, and inducible nitric oxide (NO) synthase (iNOS) and phosphorylation of mitogen-activated protein kinase (MAPK) were evaluated. The secreted HMGB-1and matrix metalloproteases (MMPs) levels were quantified. Finally, we detected the HMGB-1 and iNOS expression in rabbit cartilage defects.

Results: Expression levels of 51 microRNAs were down-regulated (ex. miR-455) while 127 microRNAs (ex. miR-140, and mir-107) were up-regulated in OA chondrocytes after HBO treatment (Fig.1). The 3′UTR of HMGB-1 mRNA contained a 'seed-matched-sequence' for miR-107 (Fig.2). MiR-107 was induced by HBO and a marked suppression of HMGB-1 was observed simultaneously in OA chondrocytes. Knockdown of miR-107 upregulated HMGB-1 expression in hyperoxic cells (Fig.3). HBO downregulated the mRNA and protein expression of HMGB-1, RAGE, TLR2, TLR4, and iNOS, and the secretion of HMGB-1 (Fig.4). HBO decreased the nuclear translocation of nuclear factor (NF)-kB (Fig.5), downregulated the phosphorylation of MAPK (Fig.6), and significantly decreased the secretion of MMPs (Fig.7). Morphological and immunohistochemical observation demonstrated that HBO markedly enhanced cartilage repair and the area stained positive for HMGB-1and iNOS tended to be lower in the HBO group (Fig.8).

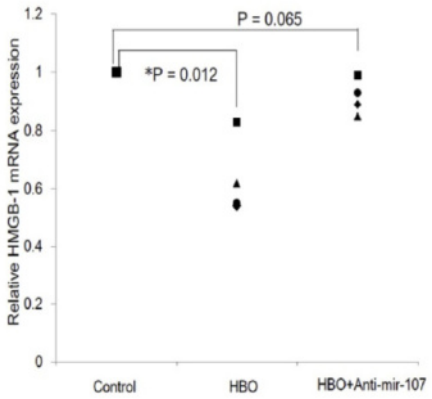
Conclusions: HBO inhibits HMGB-1/RAGE signaling related pathways by upregulating miR-107 expression in human OA chondrocytes.

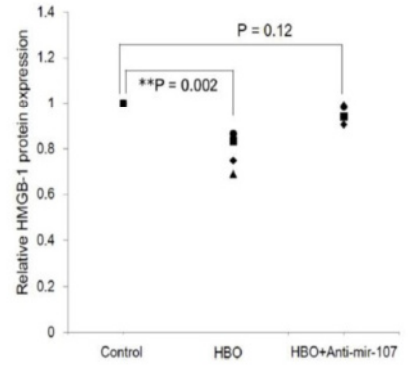


(Fig.1)

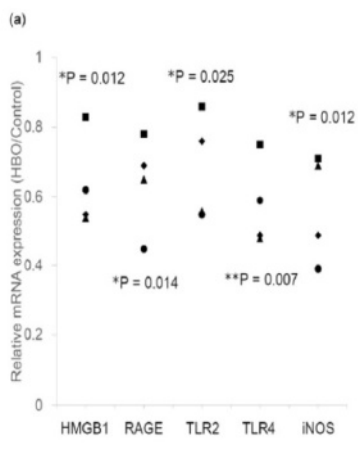
	Predicted consequential pairing of target region (top) and miRNA (bottom)
Position 50-56 of HMGB1 3' UTR	5' ...UGAUAUUUUGGAUAUUGCUGCAU...
hsa-miR-107	3' ACUAUCGGGACAUGUUACGACGA

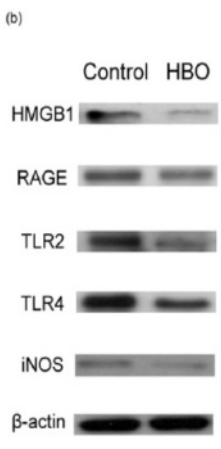
(Fig.2)



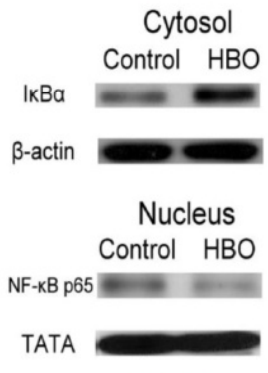


(Fig.3)

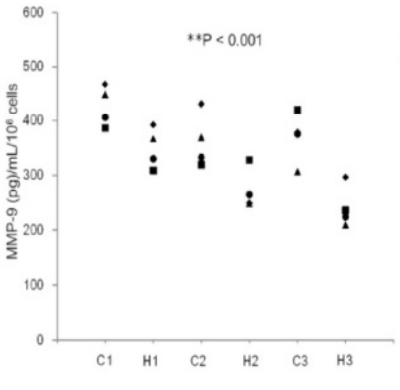




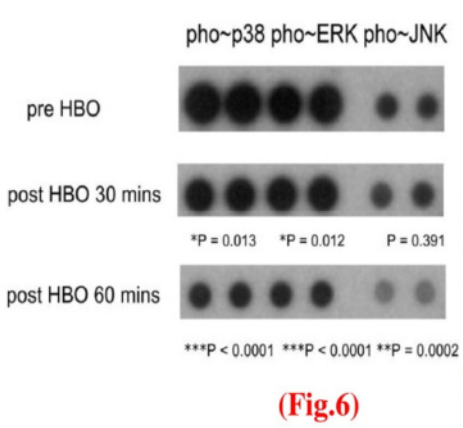
(Fig.4)



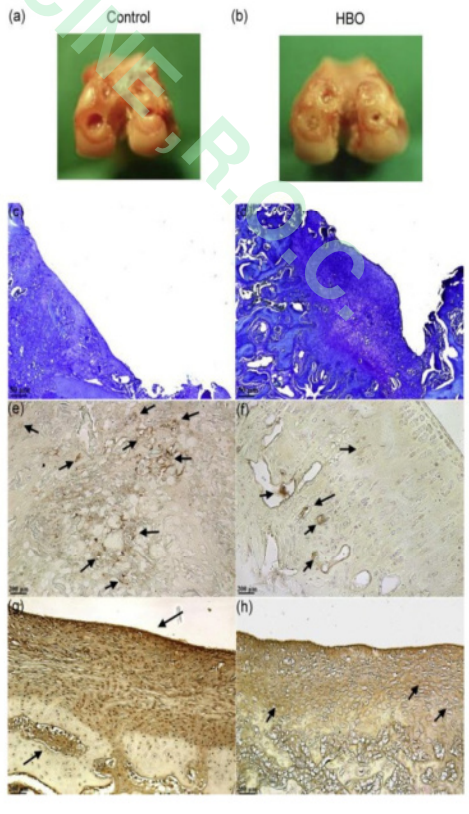
(Fig.5)



(Fig.7)



(Fig.6)



(Fig.8)

編號 25

左側大腦梗塞性中風併鼻淚管阻塞高壓氧治療經驗分享---病例報告

Experience Sharing of Hyperbaric Oxygen Therapy for Left Cerebral Infarction Stroke and Nasolacrimal Duct Obstruction---Case Report

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個案狀況

一位80歲女性，有糖尿病病史和鼻淚管阻塞三年，曾施行鼻淚管探針貫穿手術，仍沒有改善。2021年01月30日，右側肢體無力、無法言語，於他院診斷左側大腦梗塞性中風，住院一個禮拜，出院後持續於他院復健，病情仍無明顯改善，在親友建議之下，至本院接受高壓氧治療。2021年02月22日，開始第一次高壓氧治療（每天一次2ATA，120分鐘），於2021年02月25日第四次高壓氧治療後，評估病患言語講得比較多字，肢體站立的較穩，可以多走幾步路。2021年03月09日高壓氧治療第12次，病患鼻淚管阻塞3年獲得改善，原本溢淚嚴重，眼淚流不停，現今症狀痊癒，也不需要一天點四次眼藥水了；病患左側大腦梗塞性中風發作後，無法自行洗澡沐浴，需要依賴家人或居家照顧服務員協助沐浴，現在可以自行洗澡了。2021年03月26日高壓氧治療結束第24次，言語表達字句較完整，可以多說3~4句話，之前右側無力，無法行走，需坐輪椅，現在走路比較穩定，可以自己步行走路，不需要旁人攙扶行走。

討論

個案罹患左側大腦梗塞性中風一個月內，安排高壓氧治療，共治療24次，原本無法言語、右側肢體無力、日常生活無法自理，高壓氧治療結束後，臨床症狀顯著的進步，言語表達字句較完整、右側肌力評估（Muscle power）分數2分進步到4分、日常生活可以自理、鼻淚管阻塞都獲得良好的改善。高壓氧治療梗塞性中風可以改善腦組織缺血、減輕腦水腫、促進栓塞後腦組織修復、腦部微血管新生及血管側支循環的建立、使血液黏稠度降低、有效預防及治療血栓性疾病、加強藥物對腦神經系統療效。鼻淚管阻塞主要是鼻淚管造成慢性的發炎，使得原本暢通的鼻淚管阻塞而產生溢淚，高壓氧治療意外使得病患罹患鼻淚管阻塞3年獲得良好的改善，高壓氧治療針對鼻淚管阻塞有提高白血球殺菌能力、抗發炎和降低感染處水腫效果。本院的治療經驗，大腦梗塞性中風早期診斷出來，生命徵象穩定，把握適當的黃金治療期，立即給與適當的高壓氧治療，依病人的嚴重度而定，治療療程約十次至三十次不等，配合神經科藥物，病人在接受治療後，可提升病患復原情況，將傷害減到最低，避免永久的傷害，縮短大腦梗塞性中風後的復健療程，增加病患自我的照顧能力，使病患回復到正常生活功能，減輕家屬照護的壓力，雖然沒有辦法保證恢復到正常狀態，卻可以減少後遺症出現，高壓氧治療仍是輔助又有效的治療方式，將是病患、醫療、照顧者的一大福音。

編號 26

接受高壓氧治療患者合併突發性及慢性耳聾的成功個案報告

Successful Complete Recovery of Combined sudden and chronic sensorineural hearing loss Receiving Hyperbaric Oxygen Therapy

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個案

彭先生現年36歲，無特殊疾病史，從事機械性高噪音工作10年多，造成左耳多年長期聽力減退，但不以為意。110/02/23早上起床發現，右耳突然聽不見，伴隨著輕微的耳鳴；急忙前來醫院就診。當日立即住院，聽力檢查顯示右耳RE:52dB及左耳LE:30dB；治療給予靜脈施打類固醇(Solumedro750mg)及耳內類固醇(Decadron)藥物注射，之後大量口服類固醇來抑制內耳發炎，並配合血漿擴張劑(Dextran)及血管擴張劑來幫助增加內耳的血液循環。於110/02/26聽力檢查顯示右耳RE:50dB及左耳LE:43dB，但效果緩慢。110/03/04會診高壓氧治療科，經高壓氧醫師評估後進行高壓氧治療，每次120分鐘，壓力2.0~2.5ATA，總共接受12次高壓氧治療；治療第5次後，110/03/08聽力檢查顯示右耳RE:37dB及左耳LE:37dB，已有明顯改善；出院後繼續門診追蹤，並續做門診高壓氧治療，總共接受12次高壓氧治療；110/03/22再次聽力檢查顯示右耳RE:23 dB及左耳LE:22dB；個案左耳及右耳聽力皆已恢復正常，並重回工作崗位上班。

討論

突發性耳聾(sudden sensorineural hearing loss，SSNHL)的診斷，需要三個要素，在3天內出現連續3個頻率的聽力下降超過30分貝，好發年齡在40到60歲，多發生於單耳，很少雙耳同時發生；目前約只有10 ~ 15%找得到確切原因，例如自體免疫疾病、感染、外傷、耳毒性藥物、內分泌問題、腫瘤等，大部分突發性耳聾目前仍不清楚確切原因。早在1960年代，高壓氧治療已被歐洲學者使用於聽力障礙的輔助治療；文獻中指出：高壓氧治療後，聽力進步的機會約有25%，尤其是對較年輕患者（小於50歲）、較早接受治療（症狀發生後兩週至三個月內）、聽力受損較嚴重的患者；高壓氧治療除了對突發性聽力障礙有良好的療效，對梅尼爾氏症伴有耳鳴、暈眩、暫時性之聽力喪失，高壓氧治療可以降低迷走神經興奮性，解除血管痙攣現象，提昇耳內組織含氧量，改善耳蝸半規管淋巴液流動，進而達到治療效果。

個案因職場噪音造成左耳長期性聽力障礙，僅靠右耳還可以聽到聲音，所以輕忽聽力重要性；直到右耳突發性耳聾，才積極就醫治療，經高壓氧治療後，右耳聽力恢復正常，並意外發現左耳持續多年的慢性耳聾竟然也恢復正常。本院於最近半年內，意外發現至少三位病患，經過高壓氧治療後，原本持續多年的慢性耳聾恢復正常。高壓氧治療對於突發性耳聾，在臨床有相當成效；至於慢性耳聾仍有改善恢復可能，仍需更多個案與研究証實其療效。

顏面神經麻痺患者之高壓氧治療經驗-病例報告

Experiences in Hyperbaric Oxygen Therapy for Bell's palsy-A Case Report

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個案情況

個案為41歲職業婦女，有B肝、高血脂、耳中風病史，因某天起床發現頭、頸、左上背部異常疼痛，臉歪嘴斜還無法閉眼，2021年2月至神經科求診，經醫師評估後診斷為特發性顏面神經麻痺-貝爾氏麻痺，個案經過抗病毒、類固醇藥物治療，期間有至中醫針灸，並同時配合復健，仍常覺得身體發熱疼痛不適，影響睡眠品質，眼睛稍可閉眼但虛弱且慢於正常，左臉法令紋、抬頭紋及酒窩消失，講話漏風不清晰，吃東西易有噎到感，影響進食；經朋友建議可試試高壓氧治療加速復原時間及狀況。

討論

此個案於2021/02/10至2021/02/22接受高壓氧治療，每次120分鐘，壓力2.5ATA，評估個案在第一次治療後就有感於身體明顯放鬆，疼痛指數8分下降為3分，睡眠也因此改善；適逢過年，年後再做一次高壓氧治療，神經科安排臉部神經傳導檢查，顯示左面部神經病變幅度降低58.6%，陸續再做3次治療，評估觀察個案臉部原有紋路回復，臉部歪斜歸正，疼痛也不復見，主訴精神氣爽多了，只有單邊酒窩較淡，其餘皆已恢復；之後因感冒耳痛且經濟考量故暫停治療。由此得知高壓氧治療對於貝爾氏麻痺是有改善之效果，目前持續配合復健及針灸和門診追蹤，漸漸恢復原本的生活，心情也隨著清秀外貌再現而擺脫不安情緒及擔憂，經過幾周的追蹤觀察，無再惡化之表現。

UHMS於1997年曾發表一篇文章:高壓氧治療對於貝爾氏麻痺患者有明顯治療效果； 高壓氧治療可提供高濃度氧氣於面部神經改善缺氧，抑制並降低發炎反應，緩解壓迫減少神經緊張、受損，促進神經細胞修復及再生，並可舒緩肌肉及肌腱張力，降低身體疼痛。雖然無實體數據證實神經細胞能恢復到正常狀態，卻已明顯改善疾病造成外觀面貌改變及後遺症，若能在初期症狀出現時，儘速接受高壓氧治療，相信更可縮短復原時間及減少不適症狀，看到個案一掃陰霾露出開心釋懷的笑容，展現原有的自信，著實替他感到開心振奮。

編號 28

連續監測重症嬰幼兒高壓氧治療之生命參數

Continuous vital sign monitoring in critically ill pediatric patients under hyperbaric oxygen therapy

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Introduction

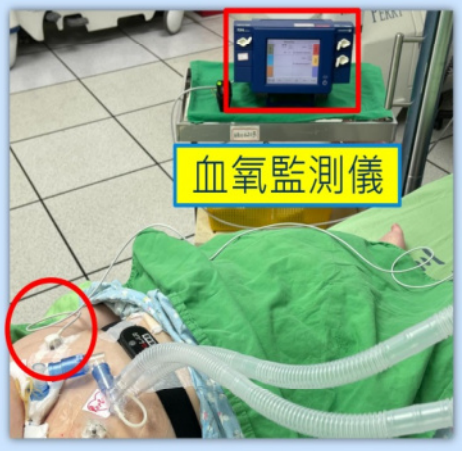
臨床上重症幼兒在進行高壓氧治療時，常見的問題是無法於高壓艙內及時監測患者的生命參數。一般血壓監測儀器不適合單人艙使用，而專用血壓監測系統既不適合嬰幼兒，又多已停產。因此我們希望透過心率傳感器及經皮血氧監測儀的介入，使重症嬰幼兒能安全進行治療。

Method

1. 治療前將心率傳感器置於幼兒胸前靠近心臟位置，並配對連接於手錶，於艙外即時監測心跳數值，紀錄後進行心率變異度分析(圖一)。
2. 治療前將經皮血氧監測儀之電極頭貼於病人胸前，使用非侵入性的皮膚加溫，促使微血管擴張，以測量皮下組織血氧分壓。根據皮下組織血氧高低，進而了解病人血液循環的情形(圖二)。

Figure

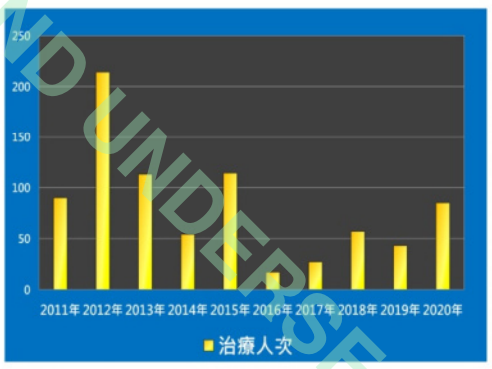
圖一、使用心率傳感器監測重症嬰幼兒治療時的心跳數值



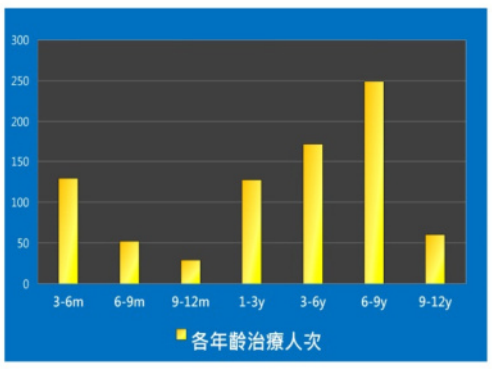
圖二、使用經皮血氧監測儀測量皮下組織血中氧氣分壓

Result

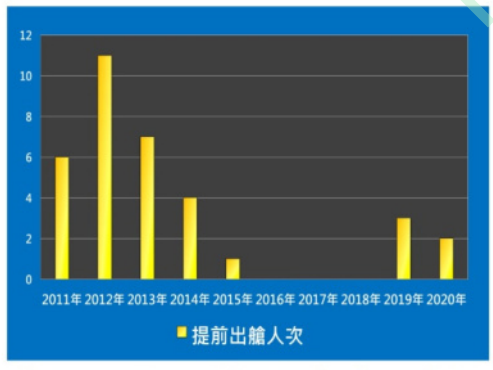
本單位使用心率傳感器及經皮血氧監測儀監測重症嬰幼兒高壓氧治療時的生命參數。於2011至2020年共治療缺氧性腦病變之重症嬰幼兒814人次(圖三)，年齡介於3個月至12歲(圖四)。治療時需提前出艙共計34人次(圖五)，其中以監測時發現心率及血氧異常而提前出艙之患者共18人次(圖六)，出艙經適切處置後，無危及生命之事件發生。



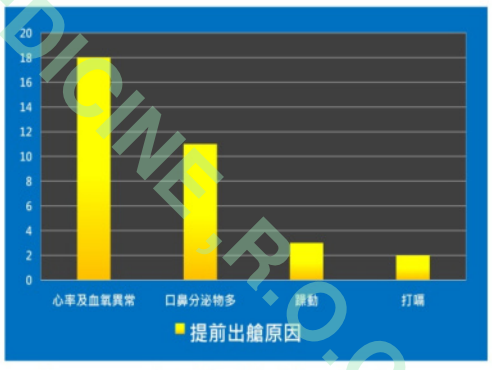
圖三、2011至2020年共治療重症嬰幼兒814人次



圖四、2011至2020年治療重症嬰幼兒年齡分佈



圖五、病人終止治療而提前出艙共34人次



圖六、提前出艙原因以心率及血氧異常最高

Conclusion

此方法可即時監測重症嬰幼兒於高壓艙內的心率及血氧變化，協助操艙技術員觀察及判斷患者治療進行順利與否，提升嬰幼兒高壓氧治療過程之安全性。

編號 29

如何降低高壓氧治療的患者提前出艙之發生討論

How to reduce The Incidence of Patients Early Interruption out of Chamber Undergoing Hyperbaric Oxygen Therapy

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Wholehearted, Holistic Care

全心三總・全人照護

Introduction

高壓氧治療在內、外科醫學當中扮演重要輔助治療之角色，而接受高壓氧治療之患者與日俱增。讓病人順利完成治療為高壓氧技術員之重要職責，而工作當中發現，病患常因各種狀況導致治療中斷，需要提前出艙。提前出艙除了牽涉病人安全問題和降低治療效果，也會間接影響高壓氧的排程，甚至影響單位服務績效，本次研究主要透過回朔2020年8月至2021年4月中進行臨床統計後分析，討論如何降低病患提前出艙機率，以提高服務品質、提高病人治療之成效。

Method

回朔分析2020年8月至2021年4月期間，高壓氧治療異常提前終止治療原因進行統計分析，總共治療**5079**人次。當中**67**人次，因異常原因終止治療(平均發生機率:**1.44%**、每月平均7.4人次)。其中異常原因分為患者治療前**禁食、血壓異常、耳擠壓傷、躁動、焦慮、氧氣中毒**(0%)、**生命徵象異常、痰液多呼吸喘、其他原因**，等問題共9類，異常時紀錄下原因、治療時間、病人主訴等問題，進行原因分析。

Result

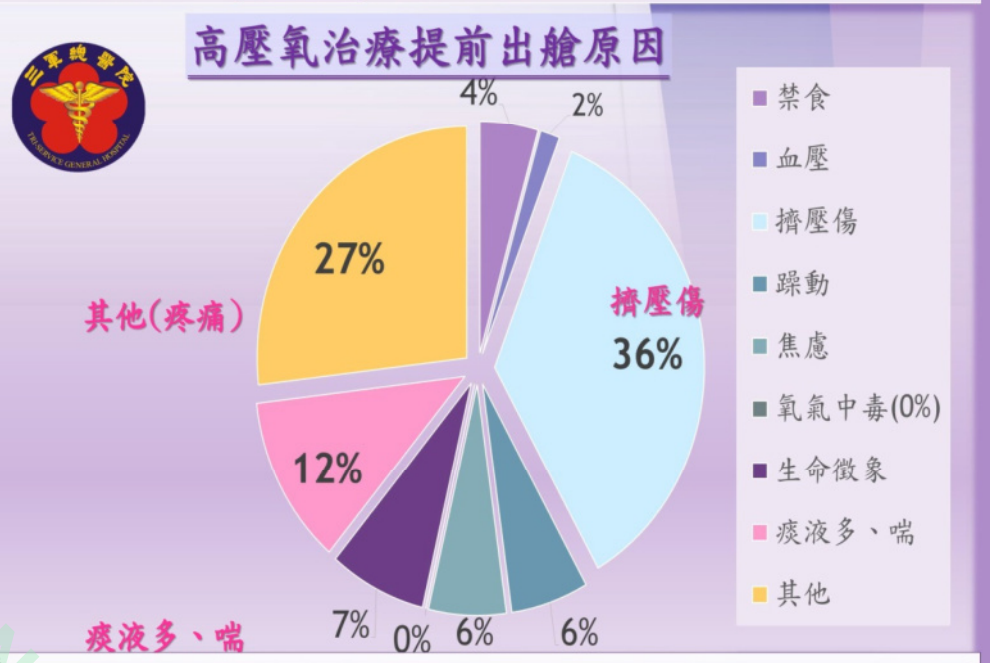
分析統計各項異常原因機率後，其中以耳擠壓傷:35.8%、其他原因(疼痛問題):26.8%，痰液多呼吸喘:11.9%為大宗，個別予以分析:

一. 耳擠壓傷大多發生在第一次進行高壓氧病人，而發生耳擠壓傷病人年齡>65歲長者超過6成。

二. 其他原因分析(疼痛問題)為一名診斷放射線治療後出血性膀胱炎患者，治療過程中曾同時多次進行膀胱鏡檢查及止血手術、連續性膀胱灌洗、玻尿酸膀胱灌注法，造成病人間斷性脹痛、膀胱壓迫感等問題，伴隨情緒緊張、焦慮問題，故予以提前出艙。

Result

三. 放置氣管內管、氣切或重症患者使用呼吸機，因呼吸道清除功能失效，唾液口水、痰液容易造成嗆咳、呼吸喘或費力情形，故予以提前出艙。



Conclusion

透過對策模擬並提出護理行為之改善方法:

一. 重新與病患教導高壓氧加壓期衛教內容，溝通內容減少艱澀專業用詞，與病人進行回覆示教。新病人進艙前、後使用耳視鏡，觀察病人耳膜狀況。

二. 放置氣管內管、氣切患者，進艙前評估呼吸狀態，聽診雙肺呼吸音，協助抽痰及唾液抽吸，維持呼吸道暢通。

三. 老年患者或意識木僵患者，治療前與病房護理師、主要照顧者討論，是否會診耳鼻喉科行耳膜穿刺術，以減少耳擠壓傷發生可能。

四. 進艙前評估病人有疼痛相關問題，進艙前確實予以疼痛指數評估，與本科醫療團隊確認疼痛況狀。提前與病房護理師討論是否給予PRN止痛藥物、協助採適當臥位維持舒適，並評估止痛藥使用狀況，以利調整藥物使用。

編號 30

電控式並聯單人艙治療經驗分享

Treatment experience sharing of electronically controlled monoplace chambers in parallel

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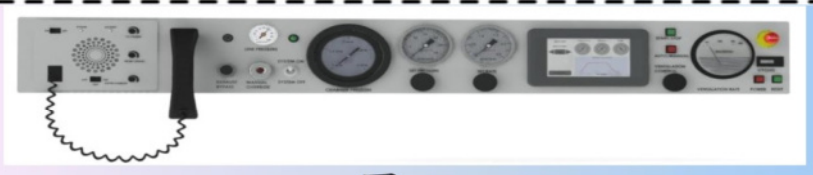
背景與目的

高壓氧單人艙治療每次的治療時間為60~90鐘，每次僅1人治療。在有限的醫療服務資源下，常常有病人要等待多時才能開始治療，等到開始治療又遇到準備出院的問題，無法即時且完整的進行治療。由於醫療需求不斷增加，本中心必須尋求提升服務品質以及服務量的方法。若能一面增加服務量，另一面善加利用有限的醫療資源，節省醫療人力成本，將可提高病人治療成效及服務滿意度。

材料與方法

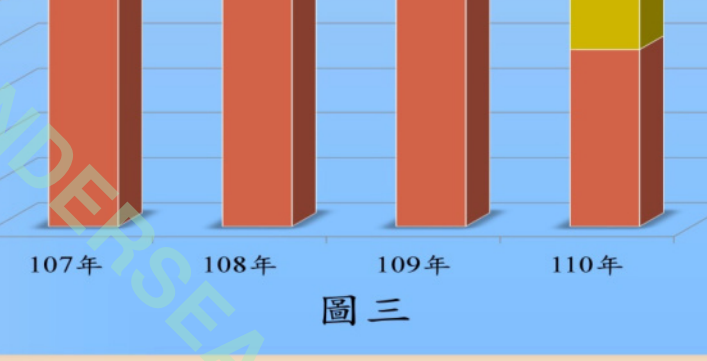
本中心建置3台電控式並聯單人艙(圖一)，在執行教育訓練以及實機操作演練後正式使用於病人治療。



圖一

經過實際操作及討論過後，扣除每艙進出、病人轉移時間，每一單艙每日安排6~7人次治療。病人進出治療艙時需要一位護佐人員協助完成其餘時間則只需一位護理技術員操作並巡視病人治療情形(圖二)。

圖二-1



圖二-2

結果

在正式使用於病人治療後，本中心每月總治療人次達到750人次，較之去年同期治療人次上升38%，新艙治療人次達356人次，占本中心總治療人次47%，大幅提升本中心服務量，也大幅降低病人等待治療時間（圖三）。

圖三

結論

高壓氧治療需受過訓練之專業醫療人員執行、操作。本中心在未增加工作人員及人力成本的狀態下，藉由引進先進治療艙操作模式，大幅提升服務人次，除了能及時給予病人所需之治療、增加服務量、提高病人治療成效之外，也提高醫院營收，更增進病人及醫師的滿意度。

在此次疫情期間，因多人艙無法保持合適之安全距離，此時單人艙的使用可保持安全空間，以及每次使用後消毒，可維持病人安全，此次疫情期間更顯此電控併聯單人艙可提升病人治療成效及服務滿意度。

編號 31

台灣突發性耳聾之發生率在南台灣與北台灣差異研究

Differences between Northern Taiwan and Southern Taiwan in Incidence of Sudden Sensorineural Hearing Loss (2000-2015)

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目的

突發性耳聾為耳鼻喉科之急症，也是高壓氧治療之適應症，但是其發生原因，大部分尚不清楚，也尚未列入台灣健康保險給付之治療項目，因此，大部分之患者未能接受高壓氧之治療。本研究為探討突發性耳聾在北台灣與南台灣之差異。

方法

利用全民健保資料庫200萬抽樣歸人檔（ICD-9-CM代碼388.2），選取2000年至2015年因為突發性聽障(Sudden hearing loss, unspecified)而就醫的患者。

結果

2000-2015年台灣地區有12,497人因突發性耳聾就醫，若以性別來看，男性占53.78%(發生率41.26/10^5)，女性占46.22%(發生率36.17/10^5)，男女性發生率均逐年上升。若以年齡層區分，≦14、15-24、25-44、45-64、≧65歲個案數分別占1.42%、6.71%、26.42%、45.62%、19.74%，發生率逐年上升。若以「地區別」來看，北與南台灣個案數分別占44.68%、20.26%，但是發生率分別為41.13/10^5、49.45/10^5，兩地區發生率均逐年上升，自2001-2015年，分別增加60.27%、85.26%。進一步分析環保署資料，發現$PM_{2.5}$累積量($PM_{2.5}$-year)自2000-2015年逐年上升(雲嘉南與高屏地區高於北部地區)，而本研究顯示，台灣突發性耳聾發生率逐年上升(整體增加13.66%)，以南部地區發生率最高(49.45/10^5)，推測可能與$PM_{2.5}$累積量有相關。

結論與建議

1.男性發生率較高(41.26/10^5)、女性(發生率36.17/10^5)，45-64歲中年族群、南部地區之突發性耳聾發生率最高。
2.是否與空氣污染PM2.5有關，有賴進一步研究。
3.同時，面對持續上升之發生率，需持續改善空氣汙染狀況。
4.民眾教育方面，應注意室內室外空氣汙染狀況，考量民眾之健康與生產力，應早期接受高壓氧治療。

Table 1. Demographic characteristics for patients with sudden hearing loss (ICD-9-CM 388.2) in Northern Taiwan and in Sothern Taiwan (2000-2015).

Location	Northern Taiwan		Southern Taiwan		*P*
Variables	n	%	n	%	
Overall	5,584	44.68	2,532	20.26	
Gender					0.910
Male	2,991	53.56	1,354	53.48	
Female	2,593	46.44	1,178	46.52	
Age (years)	50.94 ± 16.62		50.70 ± 15.86		<0.001
Age group (yrs)					<0.001
<5	8	0.14	1	0.04	
5-14	84	1.50	43	1.70	
15-24	322	5.77	153	6.04	
25-44	1,527	27.35	621	24.53	
45-64	2,472	44.27	1,250	49.37	
≧65	1,171	20.97	464	18.33	
CCI	0.23 ± 0.70		0.26 ± 0.71		0.340
Season					0.797
Spring (Mar-May)	1,422	25.47	646	25.51	
Summer (Jun-Aug)	1,341	24.02	659	26.03	
Autumn (Sep-Nov)	1,493	26.74	668	26.38	
Winter (Dec-Feb)	1,328	23.78	559	22.08	
Urbanization					<0.001
1 (The highest)	2,687	48.12	1,201	47.43	
2	2,733	48.94	1,061	41.90	
3	50	0.90	62	2.45	
4 (The Lowest)	114	2.04	208	8.21	
Level of care					<0.001
Hospital center	3,286	58.85	1,422	56.16	
Regional hospital	2,145	38.41	874	34.52	
Local hospital	153	2.74	236	9.32	

CCI: Charlson Comorbidity Index; P: Chi-square / Fisher exact test on category variables and one-way ANOVA with Scheffe post hoc on continue variables.
Northern Taiwan: Keelung City, Taipei City, New Taipei City, Taoyuan City, Hsinchu County, Hsinchu City
Southern Taiwan: Tainan City, Kaohsiung City, Pingtung County

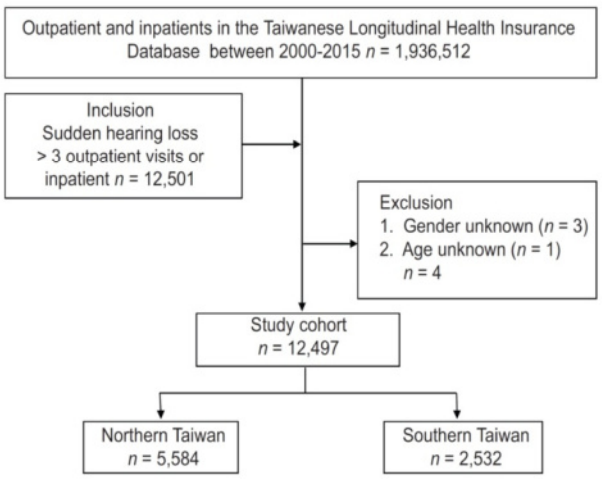

Figure 1: Flowchart of selection of the cohort or patients with sudden sensorineural hearing loss from the National Health Insurance Research Database in Taiwan. Northern Taiwan: Keelung City, Taipei City, New Taipei City, Taoyuan City, Hsinchu County, Hsinchu City. Southern Taiwan: Tainan City, Kaohsiung City, Pingtung County

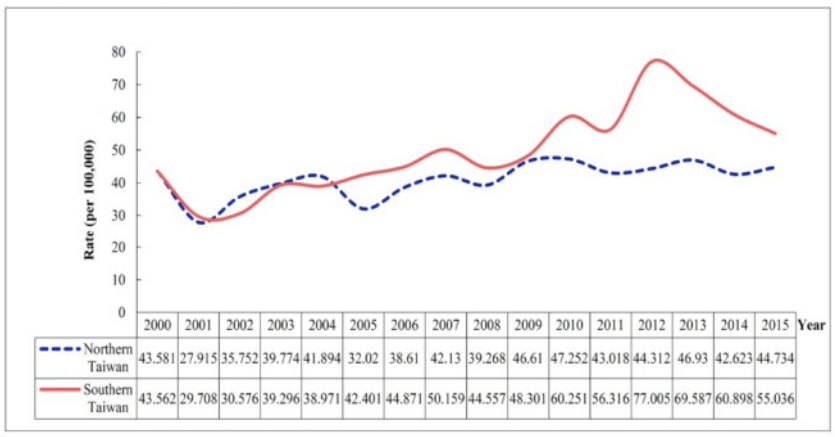

Figure 2.The crude SSNHL incidence from 2000 to 2015 in Northern Taiwan and in Southern Taiwan.

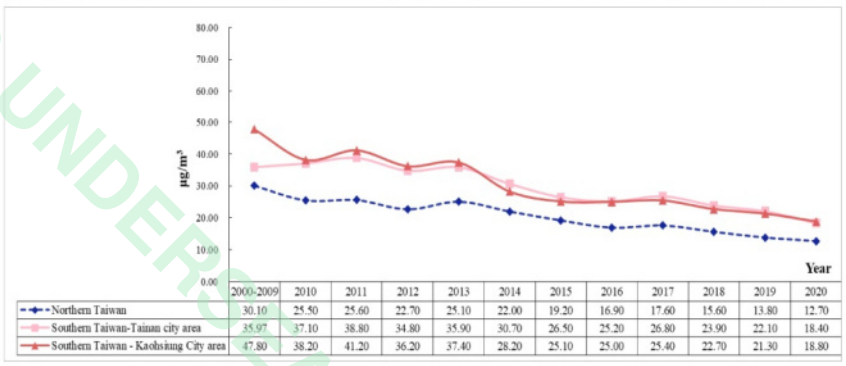

Figure 3. Average concentrations of $PM_{2.5}$ during 2000–2020 in Northern Taiwan and Southern Taiwan.

Figure 4. Cumulative exposure to $PM_{2.5}$ ($PM_{2.5}$-year) during 2000–2015 in Northern Taiwan and Southern Taiwan

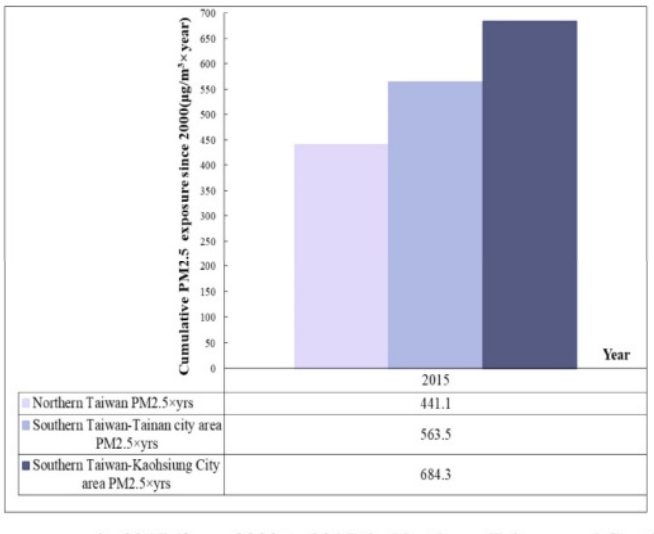

Figure 5. Cumulative $PM_{2.5}$ exposure in 2015 (from 2000 to 2015) in Northern Taiwan and Southern Taiwan

編號 32

以高壓氧輔助治療放射線損傷導致嚴重疼痛-病例報告

ADJUVANT HYPERBARIC OXYGEN THERAPY FOR A RADIATION INJURY PATIENT WITH SEVERE PAIN – A CASE REPORT

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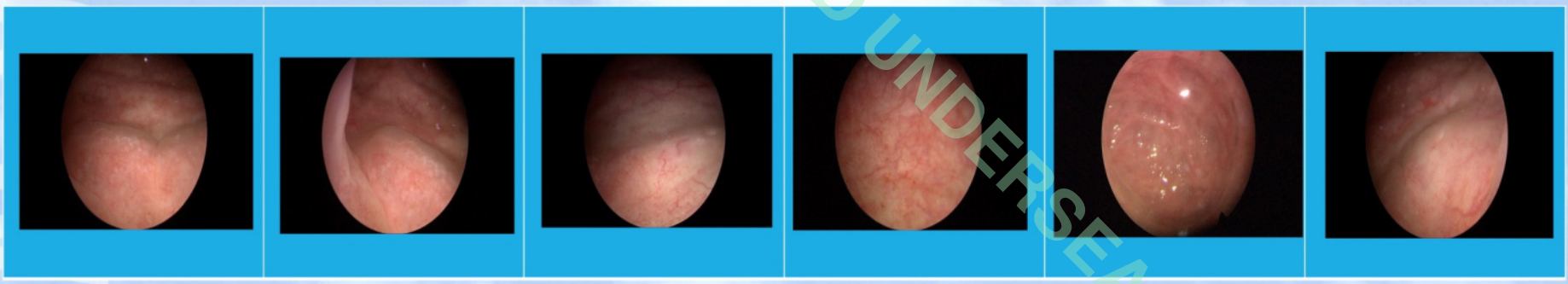
前言

高壓氧治療運用於放射線治療損傷在臨床上已屬常規治療，健保給付含放射線性骨壞死，放射線性膀胱炎或放射性腸炎。但在臨床上放射線治療後遺症應該不只上述三項，很多病人受害於放射線治療後的副作用，無法有效改善其症狀進而影響睡眠及生活品質。藉分享高壓氧治療有益於放射線治療疼痛症狀改善的成功案例，讓高壓氧治療在此領域運用範圍更寬廣。

病例報告

六十四歲家庭主婦102年診斷子宮內膜癌(Endometrial endometrioid adenocarcinoma stage IA, grade 1-2/3)經機器人輔助腹腔鏡(Robot-assisted laparoscopic)，行機器手臂子宮切除術。108年3月到9月及109年01月到7月因子宮內膜癌復發進行放射線治療(External beam radiation therapy, EBRT)。放射線治療後排尿困難、小便疼痛持續困擾著病人，109年07月09日排尿困難至婦科門診求治予止痛藥及抗生素治療，109年8月4日因**嚴重下腹疼痛(pubic area pain)至急診求治**，經止痛藥(kerolac and morphine) 治療後疼痛緩解出院，但8月6日疼痛加重，**無法久坐**，求治於疼痛科行腰椎硬脊膜外神經阻斷術，8月13日回診表示疼痛沒有明顯改善，8月24日小便疼痛及完全性尿失禁（Total incontinence）安排腹部電腦斷層(abdominal CT)確認無子宮內膜癌復發及膀胱壁增厚。8月31日膀胱纖維鏡檢查(Fiberocystoscopy)顯示尿道狹窄伴擦傷(如下圖)。藥物治療仍無法改善疼痛，9月1日至本院高壓氧門診評估，懷疑是放射線損傷安排自費高壓氧治療。9月2日開始高壓氧治療，2.0-2.5大氣壓(ATA)，80分鐘，每日一次，每周5次，病人疼痛逐步改善，從因疼痛無法久坐，進艙須躺床治療到全程可採坐姿治療。高壓氧治療從一周五次漸進式改一周一次維持治療，病人現**已無疼痛問題**，生活品質提升，睡眠、作息正常體重也恢復正常。



討論

高壓氧治療放射線性傷害之效益：1.提高組織氧氣分壓，促進纖維母細胞增生、膠原蛋白及血管新生，加速放射性組織潰瘍之癒合。2.提升組織氧氣分壓，有利於受傷神經的修復及再生，避免肌肉萎縮或肢體癱瘓。3.促使放射線性骨骨內新生血管形成，治療骨壞死。4.提升人體免疫功能，增強白血球功能，避免感染發生。

癌細胞屬缺氧狀態，高壓氧可提升癌細胞的含氧量，增加癌細胞對放射線的敏感性，增強放射線治療癌症的效果及減少正常組織器官傷害後遺症。如何運用高壓氧治療於放射治療，提升治療效果減少放射治療的後遺症，是臨床上值得深入研究探討的議題。本案例屬非典型放射性治療損傷，但病人在求助無門下介入**高壓氧治療，得以緩解疼痛改善生活品質**。所以臨床上高壓氧治療的確是一項有助益又相對安全的輔助治療選項。

文獻

1. 肖平田。高壓氧治療學。中國。北京：人民衛生出版社。2009；P213
2. Jain KK: Textbook of Hyperbaric Medicine. 6th. Cambridge, MA, Hogrefe & Huber Publishers, 2017；P328-330

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編號 33

高壓氧對於臉部美容填充物併發症的治療成效
The efficacy of Hyperbaric Oxygen Therapy as An Adjuvant Therapy in the complications after facial dermal filler injection

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BACKGROUNDS

This real-world study evaluated the effectiveness of hyperbaric oxygen (HBO) for the complications after facial filler injection. We try to figure out what kinds of filler injections and patients' characteristics are prone to complication after the surgery. We also evaluated the effectiveness of HBO in the improvement of complications after dermal filler injection.

Materials and Methods

We conducted this retrospective study at a single medical center from September 2012 to December2019. Patients who were treated with HBO due to complications after facial plastic surgery with dermal filler injection are enrolled.

Date	2012.09~2019.12
Patients	29
Age	20~52 (mean age: 34.9)
Gender	Female: 27, Male: 2
HBO	2-20 secessions (average: 8.2)

Table 1: Patient characteristics

RESULTS

A total 29 patients are treated with HBO for complications after filler injection. Most of patients are female (n=27, 93%), the mead age was 34.9. The fillers category are variable; hyaluronic acid in 10 patients, radiesse in 14 cases , one with sculptra , material were unknown in the rest 4 cases. The complications are categorized into 4 groups. 1. Injection site reaction, without antibiotic treatment (n=4) 2. Infection, such as cellulitis (n=15), all the patients also received antibiotic treatment for infection control. 3. skin necrosis (n=5), 4. Vision loss or cerebrovascular accident (CVA) (n=5).
Patients are treated with HBO from 2 to 20 secessions; the average is 8.2 secessions. Patients complicated with visual impairment or CVA are treated more secessions than other groups. The outcome of HBO therapy is promising. Most of the patients recover well, only 1 patients received flap reconstruction due to skin necrosis, and one patient had permeant visual loss.

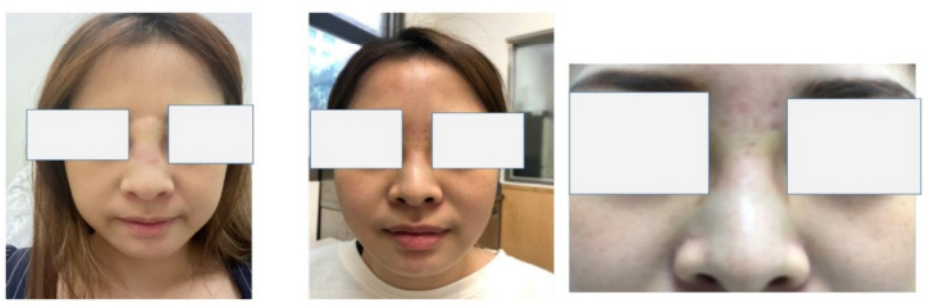

Figure 1: 31F, local site swelling post hyaluronic acid (HA) injection

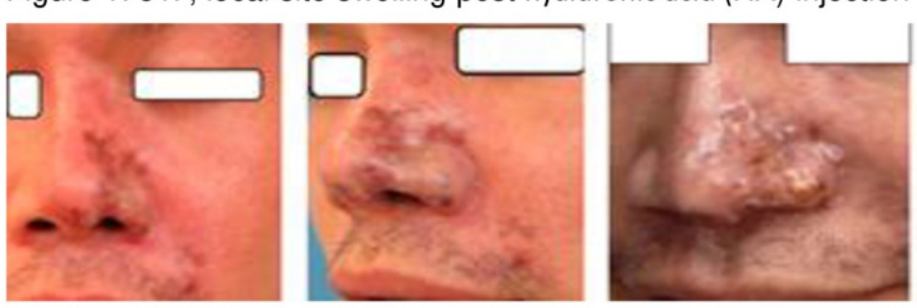

Figure 2: 45M, cellulitis post HA injection

CONCLUSIONS

1. HBOT is beneficial for the complications of facial filler injection
2. Vascular occlusion disease may need more sessions
3. The study need more case number for further evaluation the outcome for HBOT

編號 34

高壓氧治療淋巴水腫病患之病例報告

Case Report - Hyperbaric oxygen therapy in patients with lymphedema

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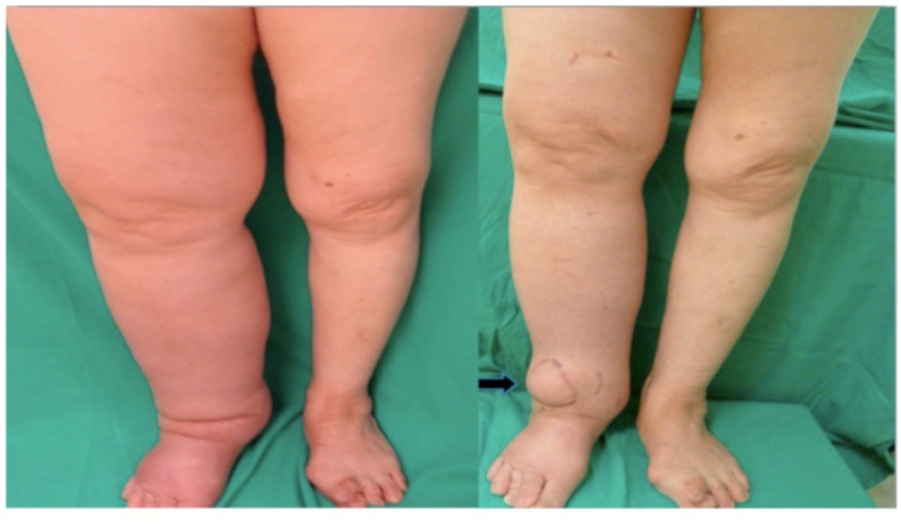
前言

淋巴水腫是指淋巴的回流變慢或阻塞使身體組織淤積過多的淋巴液而產生腫脹。原因分原發性及次發性，原發性原因不明，可能是胚胎時期淋巴管的發育不全，而次發性易發生在放射線治療及感染或是靜脈病變等因素，淋巴水腫檢查包括電腦斷層(CT)、核磁共振(MRI)、都卜勒超音波(Duplex)、近紅外線(NIR)、淋巴核子攝影(lymphoscintigraphy)等。目前淋巴水腫的治療方式有保守治療及手術兩大類。保守治療主要是以運動及壓迫治療為主 (如按摩、彈性襪、彈性繃帶、間歇性壓力裝置等)。手術治療方式包括組織切除、淋巴結移植(lymph node transfer)、淋巴-淋巴管吻合(lymphatic -lymphatic anastomosis)、淋巴靜脈吻合(lymphatic venous- anastomosis) 及淋巴管移植。

在臨床經驗上使用高壓氧做為淋巴水腫病患之輔助治療，是利用高氧濃度使血管收縮而使水腫減退，因水腫減少使微血管循環改善，而使氧氣之輸送更有效率，進而減少局部缺血、組織水腫。維繫組織微循環、保留細胞 ATP 與完整性，並提升淋巴管移植存活率。且高壓氧治療尚可透過加強纖維母細胞製造膠原蛋白，新血管生成及使動靜脈分流(AV Shunts)關閉而使表皮及皮瓣成功率提高。



結果

本院統計2019年至2021共有12名淋巴水腫病例術後輔助高壓氧治療，男性計2人，女性計10人，其中有2名為原發性淋巴水腫(慢性下肢淋巴水腫俗稱「象腿症」)，10名為次發性淋巴水腫，治療的protocol為2.0-2.5ATA每次90分鐘每天1次，治療期間約為10-20天，結果兩側圍徑測量四個點有效改善率是令人滿意 (12名7名改善) 。

	性別	年齡	病 史	水腫部位	治療前	治療後
1	女	79	子宮頸癌	左下肢	CirR/L 40.5/52 32/37. 23/22 23/23.51	CirR/L 41/51.5 32/34.5 19/20.5 23/23
2	女	51	子宮頸癌	右下肢	CirR/L:49.5/52 38/39.7 26/26.7 20.5/21	CirR/L 49.8/49 38/35.8 26.5/26.5 21.5/21
3	女	55	子宮頸癌	右下肢	cirR/L: 49.8/49, 38/35.8, 26.5/26.5 21.5/21	Cir R/L: 52.5/50 38/37 25/23.5 22/20.5
4	女	68	子宮內膜癌	左下肢	cirR/L 41.8/47.3 32/38.5 22/25 21/22.5	Cir R/L44/40 28.2/32.5 20/22.7 20.5/20.5
5	女	35	淋巴絲蟲病	左下肢	cirR/L 42.5/45.8 32.5/29.5 21/17.6 22/20	CirR/L39.7/43.6 29.8/30 20.1/18 22.2/20.5
6	男	34	創傷	左下肢	CirR/L67/72. 51/57.3 29.5/31 30/28.5	CirR/L:69/72.3 50.5/51.8 29/33.5 28.5/29.6
7	女	60	子宮頸癌	左下肢	CirR/L:37/46 27.2/36 17.5/23.5 19.5/24.5	CirR/L:37/44 26/35 17.5/24.1 18/19.6

結論

雖然高壓氧對治療淋巴水腫證據力仍然不足，但可提供未來輔助治療的新選擇。

A Brain Air Embolism in A Post-operation Patient treated by Hyperbaric Oxygen Therapy

以高壓氧治療一位術後腦部急性氣體栓塞病人—病例報告

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Introduction

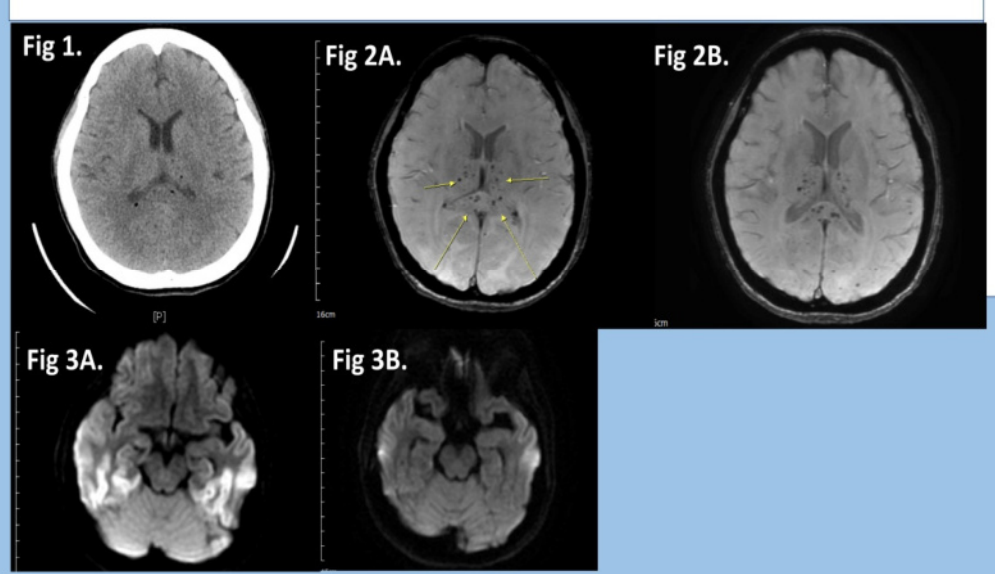
Hyperbaric oxygen therapy (HBOT) is one of the choices for emergent medical treatments in which gas bubbles are released in the tissues leading to profound disturbances of physiological processes and tissue injury. There are two major pathways for gas bubble disease, including decreasing in ambient pressure (known as decompression illness, DCI) and intravascular entrainment of gas (usually of iatrogenic origin). However, whether DCI or iatrogenic arterial gas emboli, hyperbaric oxygen is the most important treatment. This therapy causes not only a mechanical diminution of the gas bubble by raising the ambient pressure, but in application of Henry's law, it creates systemic hyperoxia. This hyperoxia produces an enormous diffusion gradient for oxygen into the gas bubble as well as for egress of the gas from the bubble.

We present a case with a satisfying clinical outcome of patient who accepted HBOT after the brain air embolism.

Case report

The 47-year-old female with a history of type A aortic dissection with severe aortic regurgitation status post Bentall procedure presented to our emergent department (ED) because of progressive shortness of breath for one day.

She received Bentall procedure, ascending aorta and total arch replacement, and frozen elephant trunk one month ago. At ED, the chest radiography showed pleural effusion, and pericardial effusion was found by sonography. Due to the cardiac tamponade with shock, the pericardiectomy was performed subsequently. On the first day after operation, endotracheal tube was weaned smoothly. She suffered from conscious change with nystagmus after removed endobronchial tube. Emergent brain CT showed stippled intense hypodense spots suspected brain air embolism. (Figure 1.) Due to the air embolism, we performed hyperbaric oxygen therapy for her (2.5 ATA, 90 minutes). On the post-operative two day, the patient was recovered with better conscious and stable vital signs in the following few days. In addition, the brain MRI follow-up one week and three weeks respectively after the hyperbaric oxygen therapy revealed decrease of the initial air embolism, and much disappear of previous restricted distribution. (Figure 2A&B. were SWAN, Figure 3A&B. were DWI) Due to the improvement of both clinical condition and image, she then received hyperbaric oxygen therapy ten dives. The patient finally was discharged with well condition and appetite, and regularly followed at outpatient department.



Discussion

Many treatments have been reported for the air embolism, and giving the immediate treatment which aims to interrupt the intervention that caused the embolic event is essential. In comatose patients, the cardiopulmonary resuscitation and endotracheal intubation should also be performed as soon as possible to maintain adequate oxygenation and ventilation. Besides, hyperbaric oxygen (HBO) has been advocated as a therapy for brain air embolism by Peirce in 1980, Tibbles & Edelsberg in 1996, and Hampson in 1999. However, there has been a few criticisms about the use of HBO due to the lack of prospective studies in humans confirming its efficacy, and only a few animal studies have shown the benefits of HBO therapy in brain air embolism.

In 2013, Wondwossen G. Tekle and his colleagues reviewed medical records and neuroimaging of 36 consecutive cerebral arterial gas embolism (CAGE) patients treated with HBO at a state referral hyperbaric facility over a 22-year period. They analyzed the effect of demographics, source of intra-arterial gas, signs and symptoms, results of imaging studies, time between event and HBO treatment, and response to HBO treatment in 36 consecutive patients. A high proportion (72%) of CAGE patients treated with HBO had favorable outcomes which was defined by complete resolution or improvement of CAGE signs and symptoms at 24 h after HBO treatment. Time-to-HBO ≦ 6 hours increased the odds of favorable outcome, whereas the presence of infarct/edema on CT/MRI scan before HBO reduced the odds of a favorable outcome. The article showed timely diagnosis and differentiation from thrombo-embolic ischemic events appeared to be an important determinant of successful HBO treatment.

Therefore, in our case, the patient accepted HBO as soon as possible after the brain air embolism was diagnosed, and she was recovered with better conscious and stable vital signs. She finally received ten dives of HBOT, and was discharged with well condition and appetite, and regularly followed at outpatient department.

Conclusion

As more and more surgical and minimal invasive procedures were executed, we should be aware of the brain air embolism, especially in patients who develop sudden neurological symptoms. A prospective study which was mentioned above showed patients treated with hyperbaric oxygen less than 6 hours had a better outcome than those treated later. Another animal studied showed that even 48 hours after induction of brain gas embolism, the outcome improved with hyperbaric oxygen therapy.

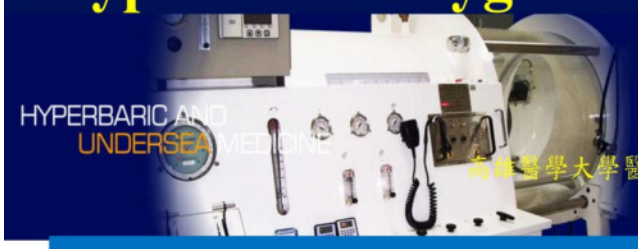
Therefore, from the experience of our case, we suggest that the diagnosis of brain air embolism in patients who received invasive medical procedures should be kept in mind, and execute hyperbaric oxygen as soon as possible after the air embolic event happened.

中國醫藥大學附設醫院
China Medical University Hospital

編號 36

Hyperbaric Oxygen Therapy Attenuates Muscle Atrophy Following Burn

高壓氧治療減輕燙傷後肌肉萎縮



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Introduction

Neuronal inflammation and apoptosis in spinal ventral horn contribute to muscle atrophy after burn. Hyperbaric oxygen therapy (HBOT) exerts anti-inflammatory and neuroprotective effect. Furthermore, hypoxia-inducible factor (HIF)-1α has been reported to promote inflammation and trigger apoptosis. We investigated the therapeutic potential of HBOT on burn-induced muscle atrophy and the role of HIF-1α in burn-induced muscle atrophy.

Materials and Methods

1. Grouping

18 SD rats were divided into three groups (each n=6) randomly at D-1.

- Control group: the rats received sham burn and sham treatment.
- Untreated burn group: the rats received sham HBOT post-burn.
- HBOT group: the rats received two weeks HBOT on days 28 to 41 after burn.

2. Burn-induced rat model

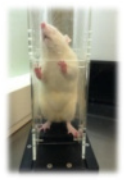
Untreated burn group and HBOT group received burn injury at D0. The right hind paw of the rats were placed on the iron plate with a heated circulating water bath at a temperature of 75 ± 0.5 ℃ while giving 100-grams-weight pressure on to hold the contact for 10 seconds.

3. HBOT

- The rats in the HBOT group were placed in a hyperbaric chamber (Genmall Biotechnology Co., Ltd., Taiwan) and received HBOT (100% oxygen at 2.5 ATA for 90 mins) on days 28 to 41 after burn.
- The chamber gradually pressurized to 2.5 absolute atmospheres (ATA) at the rate of 0.125 ATA/min, being decompressed to 1 ATA at the same rate after finishing 90 minutes treatment.

4. Behavior test by incapacitance meter (Singa Technology, Taipei, Taiwan)

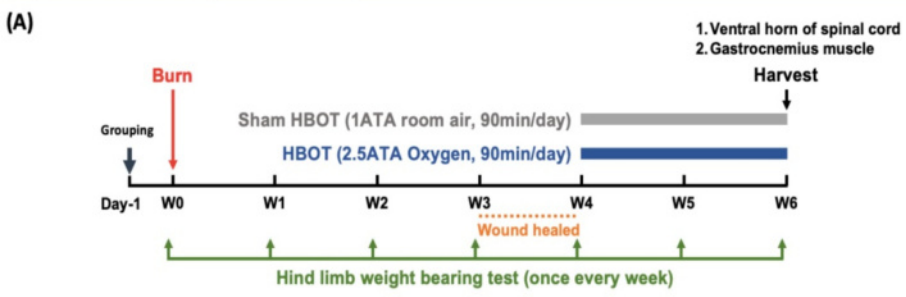
A incapacitance meter was used to measure the ratio of weight distributed between an injured and non-injured hindpaw, while normal rats distribute weight 50-50. The weight balance tests were performed over a 5-s period for 3 measurements, and the change in hindpaw weight distribution was calculated.

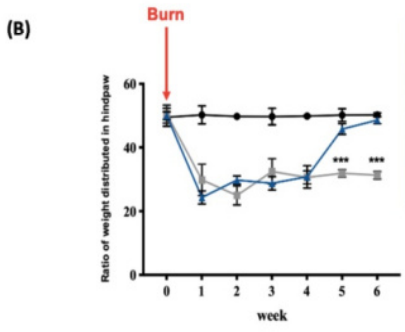


5. Histological examination

Lumbar spinal cord (L3-L4) ventral horn and gastrocnemius muscle were analyzed at six weeks (W6) post-burn.

Figure 1: Experimental design and behavior test results.
(A) Time course of experimental design





(B) Incapacitance test results of each group at indicated time points. Ratio of weight distributed between an injured and non-injured hindpaw was measured. HBOT significantly improved the weight bearing of right hindpaw on week 5 and week 6 post-burn. Data are displayed as mean ± standard deviation. ***p < 0.001. (n=6 rats per group).

Result

1. HBOT attenuated burn-induced muscle atrophy.

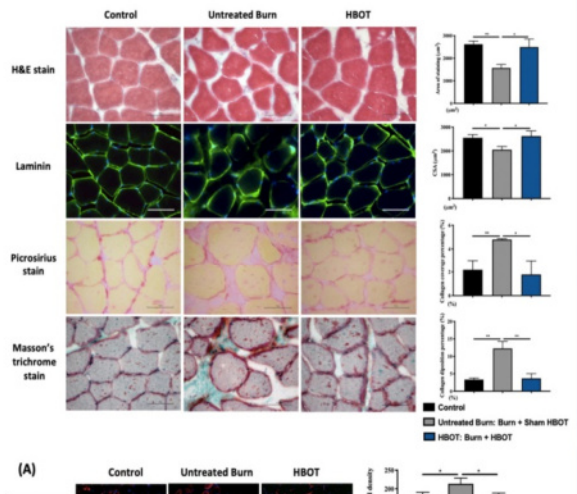


Figure 2: HBOT improves burn-induced muscle atrophy and fibrosis by histological examination.(A)Hematoxylin and Eosin (H&E) stain and (B) Immunofluorescences of Laminin of gastrocnemius muscle cross-sections for each group at days 42 post-burn. HBOT improves the decrease of myofiber cross-sectional area post-burn. (C) Picrosirius red and (D) Masson's trichrome stain of gastrocnemius muscle section to display total collagen content. Results are shown as percentage area of collagen in gastrocnemius muscle section. There is a significant increase of collagen deposition in gastrocnemius muscle in untreated burn groups compared with control and HBOT groups. Original magnificationsx400. Scale bars, 50 μm. *p < 0.05, **p < 0.01 compared with indicated group.

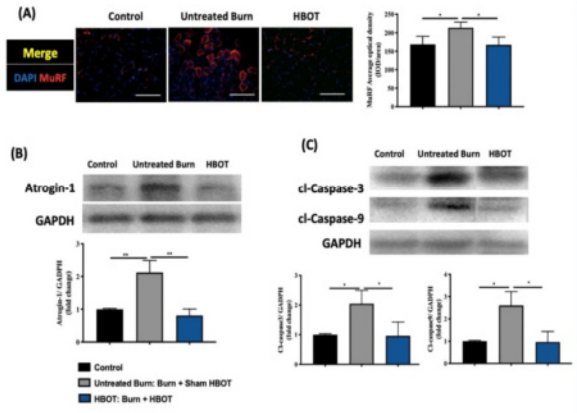


Figure 3: HBOT attenuates burn-induced muscle wasting by immunofluorescence and western blot (A)Immunofluorescence of muscle ring finger-1 (MuF-1) (red) in gastrocnemius muscle. MuF-1 which participates in skeletal muscle atrophy, was up-regulated in untreated burn group and HBOT attenuated the phenomenon. Nuclei of the cells in the muscle sections were counterstained with DAPI (blue) (B) Western blot analysis of atrogin-1, an important regulators of ubiquitin-mediated protein degradation in skeletal muscle. HBOT attenuated a significant decrease of atrogin-1 following burn. (C) Western blot analysis of cl-caspase-3 and -9. HBOT also improve the up-regulation of caspase cascades post-burn. Scale bars, 50 μm. *p < 0.05, **p < 0.01 compared with indicated group.

2. HBOT attenuated burn-induced motor neuroinflammation in ventral horn.

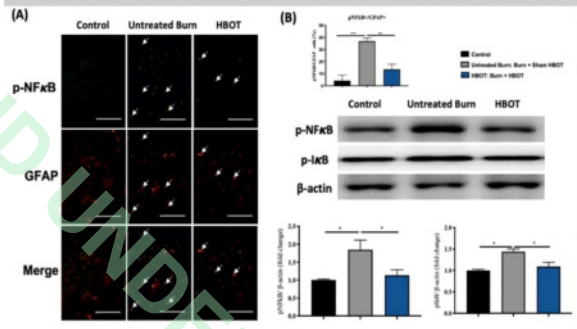


Figure 4: HBOT inhibits burn-induced neuroinflammation in the ventral horn of spinal cord
(A)Representative immunofluorescence images of GFAP (red) and NFkB (green) is the ventral horn of spinal cord. HBOT decreases NFkB-mediated astrocyte activation post-burn. (Scale bars, 50 μm). (B) Western blot analysis of p-NFkB and p-IkB in the ventral horn of spinal cord. HBOT attenuated the increase of p-NFkB and p-IkB following burn. *p < 0.05, **p < 0.01, ***p < 0.001 compared with indicated group.

2. HBOT attenuated burn-induced motor neuron apoptosis by modulating AKT/mTOR/ HIF-1α.

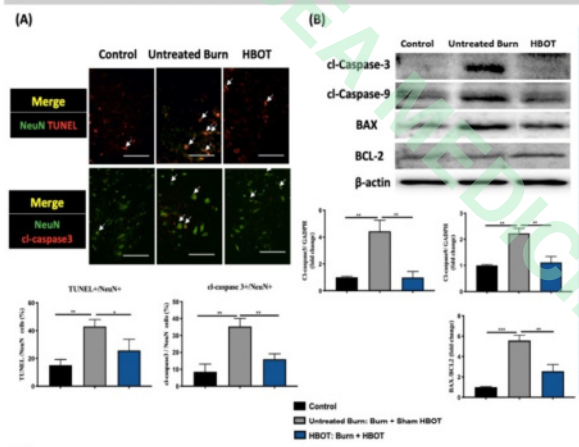


Figure 5: HBOT attenuates burn-induced neuronal apoptosis in the ventral horn of spinal cord. (A)Merged images of TUNEL assay (red) and NeuN (green). Decreased TUNEL-positive cells (double stained, arrows) in HBOT group. (B) Images of cleaved(cl)-caspase-3 (red)/NeuN (green) double immunofluorescence shows that HBOT decreases the number of caspase-3 positive neuron post-burn (double stained, arrows). (Scale bars, 100 μm) (C)Western blot analysis of cl-caspase-3, cl-caspase-9, BAX and BCL-2. A decrease of caspase cascades and BAX/BCL-2 ratio in HBOT group. *p < 0.05, **p < 0.01, ***p < 0.001 compared with indicated group.

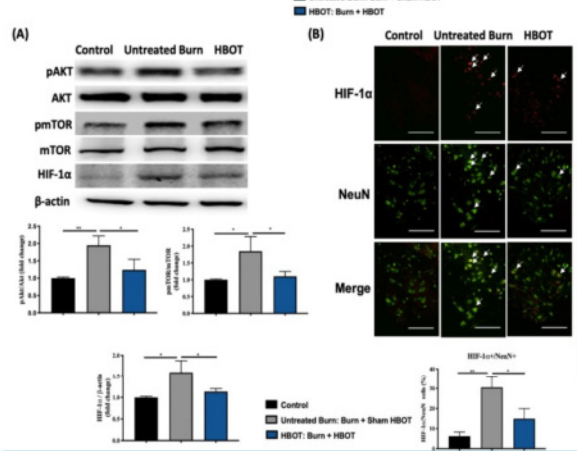


Figure 6: HBOT attenuates burn-induced neuronal apoptosis by modulating AKT/mTOR/ HIF-1α.
(A) Western blot analysis of AKT, mTOR and HIF-1α. HBOT decreases the expression of AKT, mTOR and HIF-1α post-burn. (B) Immunofluorescence of HIF-1α (red) and NeuN (green). Images of HIF-1α (red)/NeuN (green) double immunofluorescence shows that HBOT decreases the number of HIF-1α positive neuron post-burn (double stained, arrows). (Scale bars, 100 μm). *p < 0.05, **p < 0.01 compared with indicated group.

Conclusion

Our study suggests that HBOT mitigates burn-induced neuronal apoptosis in ventral horn post-burn by modulating HIF-1α signaling. HBOT further attenuated denervated gastrocnemius muscle atrophy and fibrotic changes after burn injury.

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