Risk for intracranial pressure increase related to enclosed air in post-craniotomy patients during air ambulance transport: a retrospective cohort study with simulation

Helge Brändström1*, Anna Sundelin1, Daniela Hoseason1, Nina Sundström2, Richard Birgander3, Göran Johansson1, Ola Winsö1, Lars-Owe Koskinen4 and Michael Haney1

Abstract

Background: Post-craniotomy intracranial air can be present in patients scheduled for air ambulance transport to their home hospital. We aimed to assess risk for in-flight intracranial pressure (ICP) increases related to observed intracranial air volumes, hypothetical sea level pre-transport ICP, and different potential flight levels and cabin pressures.

Methods: A cohort of consecutive subdural hematoma evacuation patients from one University Medical Centre was assessed with post-operative intracranial air volume measurements by computed tomography. Intracranial pressure changes related to estimated intracranial air volume effects of changing atmospheric pressure (simulating flight and cabin pressure changes up to 8000 ft) were simulated using an established model for intracranial pressure and volume relations.

Results: Approximately one third of the cohort had post-operative intracranial air. Of these, approximately one third had intracranial air volumes less than 11 ml. The simulation estimated that the expected changes in intracranial pressure during ‘flight’ would not result in intracranial hypertension. For intracranial air volumes above 11 ml, the simulation suggested that it was possible that intracranial hypertension could develop ‘inflight’ related to cabin pressure drop. Depending on the pre-flight intracranial pressure and air volume, this could occur quite early during the assent phase in the flight profile.

Discussion: These findings support the idea that there should be radiographic verification of the presence or absence of intracranial air after craniotomy for patients planned for long distance air transport.

Conclusions: Very small amounts of air are clinically inconsequential. Otherwise, air transport with maintained ground-level cabin pressure should be a priority for these patients.

Keywords: Air ambulance, Pneumocephalus, Intracranial pressure

* Correspondence: helge.brandstrom@vll.se
1Anesthesiology and Intensive Care Medicine, Umeå University, Umeå, Sweden
Full list of author information is available at the end of the article

© The Author(s), 2017. Open Access. This article is distributed under the terms of the Creative Commons Attribution 4.0 International License (http://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons license, and indicate if changes were made. The Creative Commons Public Domain Dedication waiver (http://creativecommons.org/publicdomain/zero/1.0/) applies to the data made available in this article, unless otherwise stated.
Background
Air that occurs in body spaces that normally do not contain air, or cannot eliminate the air directly by some form of ventilation, normally will be resorbed over time and disappear. If intracranial air is present post-operatively [1, 2], for example after the surgical evacuation of an intracranial expansive lesion, it will also be resorbed over time, over weeks [3, 4]. In modern, centralized, specialty care, patients receiving neurosurgical operative interventions (commonly bort hole or mini-craniotomy) at a tertiary care hospital may be transported back to their home hospital early after their operation accompanied by critical care personnel [5, 6], where post-operative intracranial air (if present) has not yet been resorbed. This study concerns assessment of risk for adverse medical consequences related to expansion of post-operative intracranial air. This is a clinical risk for patients who are transported post-operatively between hospitals by fixed wing air ambulance, where cabin pressures change in-flight.

When inside the cabin of an aircraft gaining altitude with the cabin pressure valve in the usual operating position, cabin pressure will decrease down to 0.74 atm, and air in closed body spaces will expand according to Boyle's law (a fixed relationship between gas volume, pressure, and temperature) [7]. In theory, if the flight altitude change occurs more slowly, and likewise the cabin pressure change also occurs more slowly, then it is thought that ICP changes might not be as dramatic, related to cerebrospinal fluid flow out of the cranium over time [8].

The intracranial volume-pressure relationship shows that at higher levels of ICP intracranial compliance decreases dramatically [9], meaning that small changes in intracranial air volume can potentially bring about clinically important changes in ICP. When ICP levels increase above capillary pressures, compromise of cerebral perfusion can occur.

It is not known at which amount of pneumocephalus and at which starting ICP there would develop injurious ICP elevations when patients are exposed to reduced ambient atmospheric pressure. Patients with ICP at the high limits of normal, and with large amounts of intracranial air on the ground and before air transport, would clearly be at risk [10–17]. Patients with low ICP and very small amounts of intracranial air on the ground before air transport can be assumed to be at low risk. There is much uncertainty about patients with moderate ICP at rest and moderate amounts of pneumocephalus. An additional complexity in this clinical assessment is that it is not clinically feasible to measure resting ICP in patients who do not have an implanted intracranial manometer or fluid-filled catheter for this purpose. Consequently, resting ICP in this setting must be estimated based on indirect means.

With commercial flights, where patients sometimes are transported over long distances, there are standard flight profiles which include efficient cruising altitudes, and also reduced cabin pressures during flight [18–20]. Air ambulances with pressurized cabins can offer the possibility of pressurizing the cabin to approximately sea level, provided that they can be allowed a flight profile with quite low and inefficient cruising altitudes. If a clinician knew with confidence how much effect the lower atmospheric pressure in an air ambulance cabin at cruising level would have on ICP, then they could make an informed decision and chose a special ‘ambulance’ flight profile, with cabin pressure at sea level. Alternatively, since flying longer distances with sea-level cabin pressures is more costly, slower, and often more turbulent, this should be avoided if not indicated for patient (or crew) safety.

The first specific hypothesis was that intracranial air was common in the immediate post-operative days after evacuation of subdural hematoma. The second hypothesis was that combinations of resting ICP and environmental (cabin) pressure with corresponding intracranial air volume increases can be identified which define ranges, below which high ICP would not be expected during air transport. Also, conversely, ranges of pre-transport ICP and intracranial air volume could be identified, above which high ICP would be expected during air transport. We aimed to test this in connection with a cohort of post-craniectomy patients who in our region are frequently transferred by air ambulances from the tertiary hospital back to their distant referring hospital. We also include analysis related to cabin pressures typical of long distance air ambulance transfers where there is no option for maintaining sea level cabin pressure. We aimed to test these hypotheses in a regional cohort of subdural hematoma evacuation patients. An established model for estimating ICP changes was used, which incorporates starting ICP and intracranial air volume inputs to transform observed patient intracranial air volumes to an expected ICP during a theoretical flight and cabin pressure change.

Methods
Study design
This was a retrospective observational cohort analysis, which began with post-operative intracranial air volume observations. These were used to generate estimates for intracranial air volume expansion and ICP at different aircraft cabin pressures and rate of change of cabin altitude in flight. An established model [8] for nonlinear transformation of air volume expansion into intracranial pressure was applied.
Subjects
The medical records and radiographic images were identified and examined for consecutive subdural hematoma with burr hole or mini-craniotomy and hematoma evacuation patients admitted to the regional tertiary hospital (University Hospital of Umeå, Sweden) during the calendar year 2014.

Measurements
Pre-operative and last (before discharge or transfer) postoperative computed tomographic (CT) image of the head for this cohort were identified. All scans were performed as spirals with 3 mm reconstructions in axial (HyFa-line), coronal (parallel to dorsal brainstem) and sagittal orientations. Volume calculation of subdural hematomas, as well as subdural air collections and epidural hematomas, was performed. Ellipsoid approximation was used, \( V = \frac{4}{3}\pi r_1 r_2 r_3 \), where \( r \) was defined as the longest continuous diameter in the three perpendicular directions divided by 2. The three diameters were defined as follows: first, parallel to the calvarium in the axial plane; second, parallel to the calvarium in the coronal plane; and third, perpendicular to the calvarium, most commonly in the coronal plane. If the collection was located anteriorly or posteriorly, the axial or sagittal plane was used. When collections were crescent shaped, the diameter was divided into a number of continuous parts (2 to 7), not intersecting the adjacent parenchyma. All radiological evaluations and volume calculations were performed by a single experienced neuroradiologist.

There was no measurement of post-operative ICP at the time of the CT demonstrating intracranial air.

The model for transformation of starting ICP and air volume to final ICP result
During decrease in ambient external pressure (as in an aircraft cabin during ascent in flight) and given unchanged temperature, intracranial gas volume will increase. As intracranial gas volume increases, this will also lead to an increase in ICP, as long as the dura mater and/or calvarium is intact. The model [20] is used to simulate ICP and intracranial air volumes during ascent from sea level (1 ATM) to 8000 ft (0.74 ATM). The parameters used for ICP estimation at different air volumes were as follows: cabin altitude rate of change = 2.54 m/s (500 ft/min), outflow resistance of the cerebrospinal fluid system = \( 1.29e^{11} \) Pa/(m\(^3\) s) and pressure volume index (PVI) of the cerebrospinal fluid system = 12.6 ml. The highest level (8000 ft altitude) (see Fig. 1) represents the cabin pressure at cruising altitude in a typical long haul commercial jet, as well as most executive or business aircrafts with pressurized cabins, flying at the most efficient cruising altitude. The relatively higher ‘cabin pressures’ represent levels that can be achieved if these aircrafts choose to fly at lower altitudes, with intentional increases in cabin pressure. Starting ICPs for the simulations were chosen as low/normal (5 mmHg), normal (10 mmHg), and high/normal (15 mmHg).

Analysis
Descriptive statistics were used to describe the relative frequency and amounts of post-operative intracranial air in the subdural hematoma cohort. For the different simulated air ambulance cabin pressures, ranges including representative point intervals for starting ICP and intracranial air volumes were chosen to include the observed start intracranial air volumes in the post-craniectomy cohort.

Results
There were 119 patients included in the study, with demographics shown in Table 1. In all these, the SDH was treated operatively with craniectomy (burr hole) or mini-craniotomy and hematoma evacuation. Subdural passive drains are standard during 12–24 h postoperatively. All of these patients received their operation in the same Department. They were all anesthetized using the same neuroanesthesia practice routines, which include using propofol, remifentanil and sevoflurane as anesthetics. No nitrous oxide was administered to these patients. The surgical management after the operation was to close the scalp incision to prevent leakage (or air suction), and the incision area was covered with a bandage, minimizing the risk for any air passage across the incision. The patient was treated without head elevation until the subdural drains were removed and a suture applied to close the skin stoma.

Of the 34 patients that had intracranial air post-operatively, 30 required no post-operative ventilator support. The remaining 4 were transferred from the operating room to the intensive care unit and briefly received ventilator support, though all were discharged from the intensive care within 1 day. These 4 had their post-operative head CT done while on the ventilator. All patients were treated initially after their operation with supplemental oxygen, titrated to an oxyhemoglobin saturation of 92–96%.

Review of the post-operative head CT images showed that of these 119, 108 had at least one post-operative head CT examination. Fifty of the 108 had more than one post-operative head CT, and for these, the last CT image was the one included in the analysis. Of the 108 subjects, 34 had post-operative intracranial air on the head CT. Where there were multiple post-operative head CT studies done for an individual patient, and where there was intracranial air detected, it was the study with the largest intracranial air volume that was used in the analysis.
Air volume on the post-operative head CT was derived from the multiplane reconstructions. From these estimations, 28/34 had intracranial air volumes less than 40 ml total (range 6.5–37.2 ml), and 6/34 had intracranial air volume more than 90 ml (range 93.9–230.9 ml) (Table 2).

Table 2 presents IC air volumes observed on CT in ascending order. The final air volume estimation at ‘8000 ft. altitude’ is presented with each initial volume observation. Then, the simulation results for final ICP estimation are presented for each of the 3 hypothetical starting ICPs and the observed starting IC air volume. Resulting ICPs at 8000 ft. altitude estimates show that ICP is expected to remain under 20 mmHg for all IC volumes 11 ml or lower, and this includes those with hypothetical starting ICP 15 mmHg. For the ICP 5 mmHg at start, the final ICP at 8000 ft. was estimated to exceed 20 mmHg for those starting with IC air volumes (sea level) over approximately 27 ml air. For the simulation with starting ICP 15 mmHg, ICP was expected to exceed 20 mmHg for those with starting IC air volume (sea level) of only 14 ml.

Continuous estimations of ICP resulting from the different observed starting IC air volumes along with different starting ICP levels demonstrate the ranges of the potential ICP events related cabin pressure change (Fig. 1). Intracranial air volumes increase as cabin pressure decreases, and these are shown in the third column of Table 1, as well as in Fig. 1. The model demonstrates relatively minor changes in IC air volumes which accompany quite large ICP changes during simulated cabin pressure decrease. As an example, when the subject with 37.2 ml IC air (sea level) goes to simulated cabin pressure 8000 ft, the air volume is expected to increase to 50.1 ml, or approximately 25%. At the same time, if starting ICP was on the higher side (15 mmHg) at start, and closer to the less...
A third finding was that for subjects with more intracranial air, or if starting at higher sea level ICP, the increase in ICP during flight ascent and cabin pressure decrease occurred relative rapidly (Fig. 1). Thereby, signs of ICP increase probably would be detectable early in the ascent phase of the flight plan for patients with either large volumes of IC air, a high starting ICP, or both. This has a clinical implication that where IC air and higher ICP might be suspected (but not confirmed), medical air ambulance personnel should be aware that dramatic ICP increases may occur very early.
in the flight plan ascent. It is not only cruising altitudes and corresponding cabin pressure decreased levels which are dangerous for these patients.

Clinical implications from these findings are several. First, in patients with intra-cranial surgery that need to be transferred over distances requiring air ambulance involvement, clinicians should recognize that IC air can be present in unpredictable amounts. Only a post-operative head CT scan, close in time to the transport date, can identify this. Neuroradiologists can easily approximate air volumes from head CT studies. Without this diagnostic information, it would be prudent to assume that there is IC air since it is not uncommon in a clinical cohort like the one in this study. Assuming that there is IC air which is over a minimal amount (11 ml in this study) means that transporting patients with sea-level air pressure in the cabin is safe. Still, if a post-operative head CT can show that there is air but only a minimal amount (11 ml or less), then these findings suggest that this air by itself, along with a routine flight plan and normal cabin pressure, should not be a danger to this patient. Conversely, most patients would be expected to need sea level cabin pressure for air ambulance flights.

For some transports, for patient travel or repatriation over long distance, private air ambulance where cabin pressure can be regulated may not be a readily available option. Commercial air travel may be preferred, even for patient transport with health care personnel. One must recognize that cabin air pressure in commercial long haul aircraft is normally between 6000 and 8000 ft depending on the airframe type and age, though cabin pressures in commercial passenger airframes have been reported to be as 'high' as 9000 ft [18, 19]. It is prudent to confirm the absence of intracranial air in any post-craniotomy (recent) patient scheduled for air transport on a commercial aircraft, rather than risk an unplanned diversion of a jumbo-jet due to symptoms developing during ascent related to reasonably anticipated IC air expansion.

Sea level cabin pressure is indicated in an air ambulance transport setting when the medical crew has any suspicion that IC air can lead to a change or worsening in a patient's neurological condition. This includes the

| Subject number | IC air tot vol sea level (ml) | Final air vol at 8000 ft | Final ICP (mm Hg) at 8000 ft with ICP5 at start | Final ICP (mm Hg) at 8000 ft with ICP10 at start | Final ICP (mm Hg) at 8000 ft with ICP15 at start |
|----------------|-----------------------------|-------------------------|-----------------------------------------------|-----------------------------------------------|-----------------------------------------------|
| 41             | 6.5                        | 8.8                     | 6.5                                          | 12.2                                         | 17.3                                         |
| 53             | 6.6                        | 8.8                     | 6.6                                          | 12.1                                         | 17.4                                         |
| 113            | 6.7                        | 9.0                     | 6.6                                          | 12.2                                         | 17.4                                         |
| 29             | 6.9                        | 9.3                     | 6.6                                          | 12.2                                         | 17.5                                         |
| 16             | 7.1                        | 9.5                     | 6.7                                          | 12.3                                         | 17.5                                         |
| 54             | 7.9                        | 10.7                    | 6.9                                          | 12.6                                         | 17.9                                         |
| 26             | 8.6                        | 11.6                    | 7.1                                          | 12.8                                         | 18.1                                         |
| 38             | 8.7                        | 11.7                    | 7.1                                          | 12.9                                         | 18.1                                         |
| 88             | 9.7                        | 13.0                    | 7.4                                          | 13.2                                         | 18.5                                         |
| 66             | 10.9                       | 14.7                    | 7.7                                          | 13.6                                         | 19.0                                         |
| 73             | 11.0                       | 14.8                    | 7.8                                          | 13.6                                         | 19.0                                         |

Table 2 Total post-operative intracranial air volumes, intracranial pressure at sea level and estimated at final cruising altitude
situation where there is suspicion of IC air, but where this has not been quantified as in patients that are not awake, and when changes in neurological status or condition may be difficult to detect.

Conclusions
In the absence of clear clinical information about post-operative IC air, medical crews must assume that a post craniectomy/craniotomy patient can have IC air that will expand during air transport with usual cabin pressurisation. Multiple combinations of starting ICP and IC air volumes lead to ICP over 20 mmHg as ‘cabin pressure’ decrease as it would in flight. It is important to perform a post-operative head CT and estimate IC air volumes for post-intracranial surgery patients if there is a possibility that they will be transported with an air ambulance.

Acknowledgements
None.

Funding
This study was funded by a multi-year research grant to the Aeromedical section, Intensive Care Medicine Department, Umeå University Hospital, Umeå, from the County Council of Västerbotten, Sweden. This study also received support from Umeå University, Umeå, Sweden.

Availability of data and materials
All results and data are kept in the section for Anesthesiology and Intensive Care Medicine, Department of Surgical and Perioperative Science, Umeå University. These will be made available from the corresponding author on reasonable request.

Authors’ contributions
All authors contributed to the definition of the study question and study design. All authors contributed to the analysis of the results and the writing of the manuscript. RB performed the radiographic measurement of intracranial air. NS performed the simulation of intracranial air and pressure change during decreases in atmospheric pressure. All authors read an approved the final manuscript.

Competing interests
The authors declare that they have no competing interests.

Consent for publication
Since no individual patient identities or consent were part of the data collection from journal reviews, no specific consent for publication was needed.

Ethics approval and consent to participate
Ethical approval for this study was provided by the Umeå (Sweden) Regional Committee for ethical vetting of human research, diary number 2014/347/31. Data was de-identified, after collection, and therefore there was no consent required from individual patients, according to the ethical approval.

Publisher’s Note
Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Author details
1Anesthesiology and Intensive Care Medicine, Umeå University, Umeå, Sweden. 2Biomedical Engineering, Medical Radiation Sciences, Umeå University, Umeå, Sweden. 3Radiology, Medical Radiation Sciences, Umeå University, Umeå, Sweden. 4Neurosurgery, Pharmacology and Clinical Neurosciences, Umeå University, Umeå, Sweden.

References
1. Reasoner DK, Todd MM, Scaman FL, Warner DS. The incidence of pneumocephalus after supratentorial craniotomy. Observations on the disappearance of intracranial air. Anesthesiology. 1994;80:1008–12.
2. Bouzarth WF, Hash CJ, Lindemuth JR. Tension pneumocephalus following surgery for subdural hematoma. J Trauma. 1980;20:460–3.
3. Amato-Watkins A, Rao VM, Leach P. Air travel after intracranial surgery: a survey of advice given to patients by consultant neurosurgeons in the UK. Br J Neurosurg. 2013;27:9–11.
4. Daboub CB, Salas G, Ebn S, Daboub CF. Review of the management of pneumocephalus. Surg Neurol Int. 2015;6:155.
5. Krüger AJ, Lossius HW, Mikkelsen S, Kurola J, Castrén M, Skogvoll E. Pre-hospital critical care by anaesthesiologist-staffed pre-hospital services in Scandinavia: a prospective population-based study. Acta Anaesthesiol Scand. 2013;57:175–85.
6. Rehn M, Hylöstö PK, Magnusson V, Kurola J, Kongstad P, Rognás L, Juvet UK, Sandberg M. Scandinavian SSAI clinical practice guideline on pre-hospital airway management. Acta Anaesthesiol Scand. 2016;60:852–64.
7. Gradwell DP. The Earth’s Atmosphere. In: Gradwell DP, Rainford DJ, editors. Ernsting’s Aviation and Space Medicine. 5th ed. Boca Raton: CRC Press; 2016. p. 3–12.
8. Andersson N, Grip H, Lindvall P, Koskinen LO, Brandstrom H, Malm J, Eklund A. Air transport of patients with intracranial air: computer model of pressure effects. Aviat Space Environ Med. 2003;74:138–44.
9. Donovan DJ, Iskandar JJ, Dunn CJ, King JA. Aeromedical evacuation of patients with pneumocephalus: outcomes in 21 cases. Aviat Space Environ Med. 2008;79:30–5.
10. Sivri S, Senthilkumaran S, Balamurugan N, Thirumalaikolundusubramanian P. Tension pneumocephalus: A case report with review of literature. Emerg Radiol. 2013;20:573–8.
11. Schimert CM, Heilman CB, Bhardwaj A. Pneumocephalus: case illustrations and review. Neuroradiol. 2010;13:152–8.
12. Shaikh N, Masood J, Hansiens Y, Louou A, Hafz A. Tension pneumocephalus as complication of burst-hole drainage of chronic subdural hematoma: A case report. Surg Neurol Int. 2010;1:27.
13. Ihabi Z. Pneumocephalus after surgical evacuation of chronic subdural hematoma: Is it a serious complication? Asian J Neurosurg. 2012;7:66–74.
14. Willson TJ, Grady C, Braxton E, Weitzel E. Air travel with known pneumocephalus following outpatient sinus surgery. Aviat Space Environ Med. 2014;85:75–7.
15. Seth R, Mir S, Dhir JS, Cheeseman C, Singh J. Fitness to fly post craniotomy — a survey of medical advice from long-haul airline carriers. Br J Neurosurg. 2009;23:184–7.
16. Beda RD, Khot SP, Manning T, Walker M. Airhead: intraparenchymal pneumocephalus after commercial air travel. Surg Neurol. 2007;68:648–9.
17. Jensen MB, Adams HP. Pneumocephalus after air travel. Neurology. 2004;63:400–1.
18. Hampson NB, Kregenow DA, Mahoney AM, Kimland SH, Horan KL, Holm JR, Gerbino AJ. Altitude exposures during commercial flight: a reappraisal. Aviat Space Environ Med. 2013;84:27–31.
19. Kelly PT, Seccombe LM, Rogers PG, Peters MJ. Directly measured cabin pressure conditions during Boeing 747-400 commercial aircraft flights. Respilology. 2007;12:511–5.
20. Aerospace Medical Association, Aviation Safety Committee, Civil Aviation Subcommittee. Cabin cruising altitudes for regular transport aircraft. Aviat Space Environ Med. 2008;79:433–9.