A systematic review of the effects of Bhramari pranayama on the central and autonomic nervous system

A dissertation submitted in partial completion of a BHealSc Honours by

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HEAL805

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Abstract

This systematic review is an investigation into the effects of Bhramari pranayama (Bhramari), a yogic breathing practice, and its effects on the central and autonomic nervous systems. To be eligible for inclusion a study must have reported using a variant of Bhramari, which at minimum must have included nasal breathing and humming, any studies combining these two aspects but not defining them as Bhramari were also eligible. Further, the studies must have included one or more markers or tests of autonomic nervous system activity, central nervous system activity, and/or cognitive function. Studies with treatment groups combining Bhramari with other interventions were excluded. ScienceDirect, Scopus, Web of Science, ClinicalKey, EBSCO Health Databases and MEDLINE were last searched on the 8th of December using the term “Bhramari” and eighteen studies were deemed fit for inclusion. The first author extracted results from the included studies into extensive excel tables, which were then summarized to create summary tables, and a risk of bias assessment using the Cochrane risk-of-bias tool for randomized trials (RoB 2). The studies reviewed revealed a modest but consistent, shift to parasympathetic dominance in ANS metrics, increase in paroxysmal gamma waves and alteration of wave power and expression in EEG reading, and increased concentration and accuracy in cognitive assessments. This was present over the domains of autonomic functioning, central nervous system function and cognitive testing. However, the risk of bias assessment resulted in a high risk of bias result for the majority of studies assessed. This is reflective of the large heterogeneity of studies reviewed with heterogeneity being present as far as Bhramari implementation. Therefore, further research should focus on verifying existing findings with more rigorous publications along with methods designed to better deduce mechanism of effect.
Introduction

Breathing is a highly integrated process that has profound physiological effects, yet understanding remains limited. Many traditions across the world see breathing and the use of breathing techniques as integral to mental and physical health. Among these are yogic pranayama breathing practices which have a deep significance and storied history of use to the cultures of the East (Ushamohan et al., 2020). This is consistent with extensive evidence linking neural-circuitry with respiration. Maladaptive nervous system outcomes such as anxiety have on several accounts been linked to respiration. Goodwin and Buka (2008) noted that respiratory issues as early as the first year of life had a significant positive correlation with the need for later treatment for an anxiety disorder. In adult populations there also exists a correlation between depression and anxiety scores and nasal obstruction in chronic rhinosinusitis (Campbell et al., 2017; Schlosser et al., 2016; Tomoum et al., 2015). Further, anxiety disorders have a high comorbidity with chronic obstructive pulmonary disease and asthma (Kunik et al., 2005; Mikkelsen et al., 2004; Nascimento et al., 2002). Specific associations have also been suggested linking olfactory deficits and neuropsychiatric symptoms including cognitive decline in the early stages of certain neurological disorders like Parkinson’s (Bohnen et al., 2010; Cramer et al., 2010; Morley & Duda, 2011; Postuma & Gagnon, 2010). Interestingly, general relaxation oriented breathing techniques have, over diverse populations, been found to decrease symptoms of post-traumatic stress disorder and anxiety (Bryant et al., 2005; Clark et al., 1985; Nardi et al., 2009; Tweeddale et al., 1994). This same point is true for yogic pranayama-based practices, which have shown similar effects in depression and anxiety (Bhimani et al., 2011; Nemati, 2013). It should be noted here that simple breathing augmentations such as slow breathing and nasal breathing have independently also been shown to yield positive nervous system health outcomes (Russo et al., 2017).
Although medical systems have over time become quite capable of treating acute injury and illness, chronic physical and mental health issues remain challenging. Therefore, if effective, the possibilities of a small daily practice could make a meaningful difference in several lives, the investigation and study of which will simultaneously help generate much needed understanding about breathing as well.

**Enquiry statements**

- What, if any, are the effects of Bhramari on the autonomic nervous system?
- What, if any, are the effects of Bhramari on cognition and the central nervous system?

This will therefore also afford an opportunity to explore some general systematic integrations between breathing and the nervous system. However, to do this a base understanding of breathing, it’s commentary and the nervous system itself is necessary.

**Introduction to the nervous system and the physiology of breathing**

The regulation of breathing involves several parts of the nervous system, hence a brief overview of the nervous system is provided here. A brief summary of these same mechanisms can be found in Table 1. The nervous system operates as an integrated unit (Urfy & Suarez, 2014). Regardless, some organizational distinctions can be made, the broadest being that between the central nervous system (CNS) and the peripheral nervous system (PNS). The CNS consists of the brain and spinal cord, and is responsible for processing, perception, comprehension and response. The higher regions of the brain, particularly the cortex, are responsible for conscious perception and response under volition. The PNS is organised into an afferent division and an efferent division. The afferent or ‘sensory’) division generates signals as a response to stimuli and transmits these to the CNS. The
efferent or ‘motor’ division, carries from the CNS, and consists of the somatic and autonomic nervous system (ANS). The ANS can be further segregated into a sympathetic division and a parasympathetic division (Thau et al., 2021). While at any given point both divisions are contributing to functioning at least to some degree, in a moment, one could be said to be in parasympathetic or sympathetic dominance. Parasympathetic dominance is characterized by a calm state where non-immediate (the order of minutes) essential processes for living are carried out such as digestion, and homeostasis is optimised. Conversely, sympathetic dominance is characterised by the shutting down of non-essential functions and other processes take effect in order to prepare the body for rapid response such as the excretion of adrenaline and increased conversion of glycogen into glucose. Although brief bouts in sympathetic dominance is adaptive as it always is for a rapid response to danger, persistent sympathetic dominance is affiliated with chronic stress. Because of this, encouraging parasympathetic dominance is considered a positive health outcome as this puts one in a relatively relaxed state (Urfy & Suarez, 2014).

Table 1

**Overview of the componentry of breathing and its relationship to the nervous system.**

<p>| | |</p>
<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>Respiration rate/depth</td>
<td>Respiration rate/depth has been observed as having several physiological outcomes with some key effects on ventilation efficiency, cardiorespiratory coupling and sympthovagal balance (Russo et al., 2017).</td>
</tr>
<tr>
<td>Oral breathing vs nasal breathing</td>
<td>Nasal breathing allows air to be filtered, warmed and humidified so as to both protect the lower airway and increase ventilation efficiency (Wolf et al., 2004).</td>
</tr>
<tr>
<td>Inhalation/exhalation ratio</td>
<td>Because of its relationship with respiratory sinus arrhythmia, breathing inhalation/exhalation can have a direct impact on cardiac vagal activity (Laborde et al., 2021).</td>
</tr>
</tbody>
</table>
Musculature

Diaphragmatic locomotion in particular has been linked to a range of direct phenomena associated with the CNS such as neural oscillation and intracranial pressures, as well as indirectly affecting the rest of the body with associations to other ANS controlled mechanisms such as gastrointestinal tract functioning (Bordoni et al., 2018).

The Vagus nerve is also critical for proper cardiac functioning (Pagel & Freed, 2018). Although the electrical signalling for the actual cardiac cycle comes from the cardiac nerves, PNS signals via the Vagus nerve modulate beat-to-beat control while also having further effects on the atrioventricular (AV) node and the myocardium directly (Bibevski & Dunlap, 2011). The cardiac cycle itself however, refers to the sequence of events that occurs when the heart beats (Pagel & Freed, 2018).

Both the central and the peripheral nervous systems are integral to the coordination of breathing (Webster & Karan, 2020). The neuronal signalling for respiration comes from the pons and the medulla, both of which work together to create inhalation and exhalation (Russo et al., 2017). This signal is referred to as respiratory drive. The cerebral cortex by its influence over the pons and medulla also creates the conscientious component of breathing, which involves both passive (such as speech) and active (such as wilful breath holds) breathing modulation (Webster & Karan, 2020). Sensory inputs also provides feedback to regulate respiration. These can be identified as the afferent mechanoreceptors, metaboreceptors and chemoreceptors in the CNS and PNS (Webster & Karan, 2020). Mechanoreceptors respond to mechanical stimuli, such as stretch, in key organs such as the trachea and the lungs. Critically, this information is integral for determining breathing rate and though its sensitivity to lung gas volume, also affects tidal volume (Russo et al., 2017). Metaboreceptors conversely respond to metabolic by-products and are located in skeletal muscle. This creates an increase in ventilation when exercising. The chemoreceptors located
in the PNS monitor bloodborne chemicals and respond to aortic blood states such as hypoxia (low O$_2$) and hypercapnia (high CO$_2$). These afferent signals are carried to the medullary neurons. Chemoreceptors located in the CNS itself are also located on the surface of the medulla and retrotrapezoid nucleus. Similar to those in the PNS, these respond to pH levels in the cerebral spinal fluid (Siesjö, 1972). This pH level is largely determined by the levels of hydrogen ions (H+) present in the fluid which are dependent on levels of the CO$_2$ in blood due to the bicarbonate carbonic acid buffer system (Benner et al., 2020). Therefore, high acidity in the fluid will stimulate an increase in ventilation in order to expel CO$_2$ from the bloodstream. Of all the afferent inputs that modulate breathing, this one in particular is thought to have primary control over respiratory drive (Benner et al., 2020). This is why CO$_2$ is important for breath regulation. Additional pertinent components of the pulmonary system are some of the actual organs used in ventilation and the musculature which is mechanically responsible for it.

The nose is also important to consider as it is an organ dedicated to bringing air into the body. It (nasal sinus) can be separated into two parts, the olfactory segment and the respiratory segment. For the purposes of this paper the respiratory segment is significant. This involves the canals that allow for actual ventilation. The nose serves several key functions, including the humidification of ventilated air and its filtration by the Concha (Wolf et al., 2004). This process is critical in protecting the lower airway and ensuring healthy pulmonary functioning in the long term. Nasal breathing has also been suggested to be integral to slow breathing (Zaccaro et al., 2018). A minute ventilation rate is defined as the respiratory rate multiplied by tidal volume and due to respiratory homeostasis the body attempts to keep this rate constant (Russo et al., 2017). This means that if an individual is to breathe slowly (decreasing their respiratory rate) then in order to preserve respiratory homeostasis, breaths deepen (increase tidal volume). At its optimum level, a breath rate of 6
per minute has been found to decrease the natural chemoreflex response to hypoxia and hypercapnia (Russo et al., 2017). There is also the factor of physiological dead space to consider (Russo et al., 2017). Physiological dead space is the sum of air that has poorly entered alveoli or not entered at all plus anatomical dead space (Russo et al., 2017). An increase in respiratory rate does not increase ventilation efficiency because it also increases physiological dead space, however, decreasing respiration rate and increasing tidal volume does increase ventilation efficiency because it increases alveolus recruitment and eliminates dead space (Russo et al., 2017). These phenomena collectively mean that nasal breathing is integral for ventilation efficiency and proper healthy respiration.

NO also must be considered here. NO is a colourless water-soluble and lipid-soluble gas that in the body acts as a signalling molecule having a range of functions. NO has an integrated relationship with the brain and neurons generally acting as a retrograde neurotransmitter as well as modulating brain blood flow (Picon-Pages et al., 2019). NO also has a critical role in healthy cardiovascular functioning being noted as preventing cardiovascular diseases, including by helping regulate blood pressure to healthy levels (Naseem, 2005). Respiration is also directly affected by NO as it increases arterial oxygen tension thereby facilitating necessary gaseous exchange between cells (Dzik, 2011).

Additional functions of NO also include adaptive immune system outcomes and possible advantageous cognitive effects. Regarding NO and the nose however, it is endogenously occurring in large concentrations in the paranasal sinuses, particularly the maxillary sinus (Lundberg & Weitzberg, 1999). Although air in this part of the sinuses is usually static, humming creates gaseous exchange between this static air and respiratored air bringing more of this NO into the breath (Eby, 2006; Lundberg & Weitzberg, 1999; Weitzberg & Lundberg, 2002). It should be noted however, that most individuals in the modern day, habitually mouth breathe due to a host of reasons including palate deformation (Indiarti et al., 2017). This
means the aforementioned advantages of breathing through the nose and NO are simply not awarded to them.

Posture also influences respiratory function (Jung et al., 2016). Changes in posture can result in changes in the shape and positioning of respiratory muscles and thus can adversely affect breathing. Notably, individuals’ ability to maximise their inspiration and expiration is greater in a supine/upright posture (Costa et al., 2015). This is also closely linked to diaphragmatic position which is relevant to the topic at hand.

Cardiorespiratory coupling is also a concept of importance and this is best exemplified in the phenomena of respiratory sinus arrhythmia (RSA). Cardiorespiratory coupling simply refers to the relationship between, heart rate, and by proxy blood pressure, and respiration (Russo et al., 2017). Fundamentally, there is believed to be overlap between the neural networks that govern both HR and respiration (Garcia et al., 2013). In particular both the pons and structures in the medulla including the pre-Bötzingler complex and the nucleus ambiguous have been noted as having overlapping contributions to the phenomena in question. Activity in these parts of the brain demonstrates a kind of synchronicity which could imply a cross communication and relationship important for adaptive functioning (Garcia et al., 2013). RSA itself is HRV synchronized with respiratory rhythm. (Russo et al., 2017). This is manifested as an increase in HR at inspiration and a decrease in HR at expiration, which constitutes what is believed to be a kind of healthy HRV. Importantly, this is why inhalation/exhalation ratios are of interest. Additionally, as already mentioned, HRV and respiration both independently and coupled together have a relationship with the Vagus nerve. So, it is also common to refer to the activation of the nerve regarding these concepts and besides as the behaviour of its tone (Russo et al., 2017). Vagal tone then simply refers to this nerve’s attempt to maintain parasympathetic dominance and is the input attempt it makes to maintain this constant beyond normal base Vagal inputs. This is once again usually
assessed through HRV interpretation (Bibevski & Dunlap, 2011). Furthermore, when referring to Vagal tone in a more specific context, for example in regards particularly to the cardiac component of the Vagal nerve’s modulation, the term cardiac Vagal tone is sometimes employed (Bibevski & Dunlap, 2011).

The neurovisceral integration model is a model that posits that cardiac vagal tone (HRV) can be used to indicate expected activity in higher regions of the brain, specifically those in the cortex affiliated with emotional and cognitive processing (Park & Thayer, 2014). Some findings relating to this model have suggested that a lower HRV at rest is affiliated with a kind of hypervigilance and maladaptive emotional processing, whereas a higher HRV is affiliated with the converse (Park & Thayer, 2014). This is pertinent here, because it represents a clear, consistent and measurable correlation breathing has with higher brain functions, specifically those in the cortex. This is also why cognitive assessments may be of interest. Moreover, further research has also supported the idea that breathing has some sort of relationship with neural activity above the brainstem (Herrero et al., 2018). In particular, this research has suggested that neural oscillations and signals from certain regions of the brain appear to become more stable and clearer in relationship to the inhalation/exhalation cycle. As RSA is conventionally linked only to the ANS and this same inhalation/exhalation can reasonably be thought to be driving the phenomena in question, it may be appropriate to organize these higher order neural patterns as an additional kind of respiratory induced oscillations.

Finally, high frequency/low frequency (HF/LF) ratio should be touched on. This ratio used as a metric to assess HRV and relies on classifying the normal heart rate signal oscillations into preordained low and high frequency (LF and HF) (Shaffer & Ginsberg, 2017). At its simplest level, this is thought to reflect which branch of the ANS is assuming dominance at a given point, with the LF category referring to sympathetic dominance and HF
category referring to parasympathetic dominance. As such HRV is often interpreted as a proxy of ANS activity (Shaffer & Ginsberg, 2017). Furthermore, this relationship between the two branches of the ANS are sometimes referred to as the Sympathovagal balance and as such, HRV and the LF/HF ratio in particular is thought of as reflective of this as well (Russo et al., 2017).

Methods

Search protocol

On the 8th of December 2021 ScienceDirect, Scopus, Web of Science, ClinicalKey, EBSCO Health Databases and MEDLINE databases were searched simply for the term “bhramari”. This iteration of the search protocol was used in order to be as inclusive as possible. The key word was looked for in both title and abstract and a flowchart of the exact steps of the search is represented in Figure 1. The first point of exclusion was solely based on the assessment of titles. Exclusion at this step was based on duplication of articles, obvious books, reviews and non-scientific articles or articles where the full text was not available where also excluded here. The second point of exclusion was based on an assessment of the abstract and exclusion was based on similar grounds to the aforementioned step. The third and final point of exclusion was based on an assessment of the study itself. Aside from satisfying their aforementioned points, the only other requirement for inclusion into this review was that neurally and pulmonary healthy individuals were used. The aim of this review is to make an assessment of the basic effects of Bhr on an individual. If any effect is present at all, it can be reasonably expected that these components are integral to the mechanisms of such an effect, as such any deviation of normal in these mechanisms may result in an alteration of the effect of interest and so only studies using individuals with
healthy neural and pulmonary functioning can be used. Additional studies for inclusion were identified by cross checking appropriate references of included studies.

Figure 1

*Search flow diagram*
Note. The full search protocol in sequence carried out to identify studies for extraction.

*Indicates that these studies will be mentioned in the main text of this review to note why they were excluded.

**Intervention assessed**

Bhramari itself is an ancient form of yogic breathing meditation. Its roots can be traced to some of earliest manuals on the subject such as Hatha Yoga Pradeepika of Swami Swatmarama and the Gheranda Samhita of Sage Gheranda and it has a deep cultural significance to parts pf the east (Ushamohan et al., 2020). As is the case with many ancient texts, several translations exist and in order do justice to the intricacy of Bhramari a more extensive knowledge base then one that can be offered here, however good publications targeted to the lay audience do exist such as Ushamohan et al. (2020). This provides a readily accessible exploration of the different permutations of Bhramari by several different yoga texts which reference it, as well as providing a general introduction to the philosophical ideas underlining it.

This study also provides a detailed description on the particulars of practice involved in Bhramari. An individual is expected to sit in a neutral posture with eyes closed and the tragus is depressed using the thumb or index finger. At inhalation and exhalation, a humming sound is expected to be produced; however, there are permutations of the technique which involve the production of humming only on exhalation. Notably, no chant or involvement of the mouth is necessary or outlined by classical texts. A mudra, the placement of a particular hand posture, may also be used. Typically, this is the Shanmukhi mudra. This involves the depression of the tragus with the thumbs, the placement of the pointer fingers directly above the eyebrows, the placement of the index fingers on the closed eyelids, the placement of the ring fingers at the corners of the nostrils and the placement of the little fingers at the corners.
of the mouth. For the purposes of this review this basic definition of Bhramari will be employed. As such no Bhramari interaction using any mouth compontry such as the use of chants will be allowed for and while Mudras will be noted, they are not necessary for inclusion outright. The key Bhramari features looked for here will be the humming sound produced (on the exhalation in particular) and the depression of the tragus while nasal breathing.

**Outcome measures**

*Autonomic nervous system measures*

The ANS studies use a range of instrumentation an overview of which can be found in Table 2. The outcome measures generated by each of these instruments will now be explored.

Table 2

*Instruments of measurement found in ANS studies.*

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Description</th>
</tr>
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<tbody>
<tr>
<td>Electrocardiogram (ECG)</td>
<td>An ECG is a test which involves the placing of electrodes on the skin in order to detect and evaluate cardiac activity by measuring the electrical activity generated by the heart depolarizing and repolarizing (Serhani et al., 2020).</td>
</tr>
<tr>
<td>Sphygmanomanometer</td>
<td>A sphygmanomanometer or blood pressure monitor is a device designed to measure blood pressure, particularly arterial pressure, usually by way of a wearable cuff (Bakris &amp; Sorrentino, 2018).</td>
</tr>
<tr>
<td>Salivary cortisol assessment</td>
<td>A test of the level of cortisol in saliva done by using enzyme immunoassay (also called EIA or ELISA) usually with an existing kit (Thomsson et al., 2014).</td>
</tr>
</tbody>
</table>
Heart rate and heart rate variability

HR itself is a straightforward metric to understand so no further explanation of it is required here however HRV should be touched on. HRV is simply a metric that refers to the change in an individual’s heart rate (Ernst, 2017). As such, it is considered to be adaptive to have a higher HRV as this indicates a good ability for the body to respond quickly to a changing environment (Ernst, 2017). Notably however, this is only within reasonable parameters. As HRV is highly dependent on age and gender, exact figures are difficult to provide however a normal HRV figure could be anywhere between 20 to 200 ms depending on the individual (Kristal-Boneh et al., 2000). When outside of a normal HRV range, the anomalous HRV figure could be reflective of possible arrhythmia. HRV is usually measured using an electrocardiogram (ECG/EKG) (Serhani et al., 2020).

At its simplest, HRV is a measurement of the change in HR, a simple metric for it is RR. This is the space between the R peak in the QRS complex, the largest spike present in an ECG, to its neighbouring R peak. This is the most basic of HRV measurements, it is also referred to as the interbeat interval (IBI). NN is closely related to RR but is a slightly more refined variant. RR can be thought of as a raw measurement with no refinements, as such, artifacts could be present that might result in skewing of figures if this metric is used. As such NN is defined as the interval between normal R peaks and therefore it attempts to account for this possibility. NN50 then is defined as the number of normal peaks that have a difference greater than 50 ms between them. Another HRV metric to be aware of is the root mean squared of the normal beat to beats or RMSSD. This is defined essentially as the standard interval deviation of successive heartbeats:
RMSSD is a metric that brings in a continuous component to assessing HRV which is unique to the metrics discussed. With the exception of NN50 which is a simple number, all these metrics are expressed as ms figures. Finally, similar to the organizational principles used in EEG data, Fast Fourier Transformation (FFT) or autoregressive (AR) can be used to separate HRV into its component bands (Serhani et al., 2020; Shaffer & Ginsberg, 2017). Of interest here is the high frequency (HF) band and the low frequency (LF) band. The HF band is defined as between 0.15 - 0.4 Hz and the LF band is defined as 0.04 - 0.15 Hz. The ratio between these two bands, HF/LF ratio, is used to define sympathovagal balance and therefore it can also be used to directly establish ANS dominance preference for an individual. NN, RMSSD and HF/LF ratio all share similar mathematical componentry and are all unsurprisingly strongly correlated (Shaffer & Ginsberg, 2017).

**Blood pressure**

A brief note on blood pressure monitoring should also be made. This is usually done using a sphygmomanometer. Readings of BP are relatively straightforward as are demonstrated by the studies extracted for but Ambulatory blood pressure monitoring is somewhat different in this regard. Unlike traditional readings this type of reading involves readings over a prolonged period of time, usually 24 hours (O’Brien et al., 2018). Essentially the same instrumentation is used however readings are generated by a device continuously worn that takes a measurement at least once every 30 minutes if not more frequently and should make up at least 20 waking readings and 7 sleeping readings. Although classically ambulatory blood pressure monitors (ABPM) where/are large belt worn devices with arm
cuffs, today smaller and newer digital devices mean that even more discrete iterations of the required instrumentation are being prototyped (O'Brien et al., 2018).

**Salivary cortical assessment**

This outcome is once again a simple metric to understand as it provides a measure of the free and active cortical in the saliva which reflects plasma cortisol (Thomsson et al., 2014). It is usually expressed in millimoles per litre (mmol/L) or milligrams per decilitre (mg/dL).

**Electroencephalogram analyses**

Electroencephalography (EEG) is another instrument prominently used through the studies reviewed. It consists of a number of electrodes (as many as 128) being placed on the scalp in order to detect the faint electrical signals produced by neuronal activity (Jackson & Bolger, 2014). This particular variant of the EEG is a scalp EEG or sEEG and although others exist such as an intracranial EEG, no type of EEG other than a sEEG was found to be used by the reviewed studies. Electrodes can sample at a number of different rates and data is usually represented graphically. The interpretation of this data can be done in a number of different ways but usually through the segregation of electrical signals into waves. These waves are Delta 0.5 - 4 Hz, Theda 4 - 8 Hz, Alpha 8 - 12 Hz, Beta 12 - 35 Hz and Gamma >35 Hz (Abhang et al., 2016). Furthermore, additional wave information is also pertinent such as wave amplitude which constitutes wave power and wave location referring to the suspected source (area of the brain) the wave originated from. The combination of all these factors allows for an interpretation of EEG.

EEG analysis and interpretation often involves several steps and the use of multiple techniques. This is simply because the sheer volume of locations, influencers on and possible causes of electrical signals in the brain are vast (Lopez Rincon & Shimoda, 2016). As such, a
simple analysis of EEG data can be difficult. The EEG studies reviewed used several
different types of cleaning and analysis protocols, sometimes more than one in the same
study. One of the most basic is that of Relative Spectral Power (RSP). As an EEG signal is
often separated into waves or “bands”, this metric is an account of band spectral power with
respect to total spectral power (Abhang et al., 2016). Represented mathematically it is:

\[ RSP_i = \frac{BSP_i}{TSP}, \quad i \in \{\delta, \theta, \alpha, \beta, \gamma\}, \]

(Prasad et al., 2006). It should be noted that the term spectral and spectrum here are derived
from Fourier analysis which proposes that any signal can be constituted as a number of
discrete frequencies and as such, this analytic concept is the cornerstone of all EEG analysis
done using brainwaves (Jackson & Bolger, 2014). This concept is also very closely related to
fast Fourier transform (FFT). A Fourier transformation is a reference used to the
mathematical process used to convert a waveform data from the time domain into the
frequency domain (Bracewell, 1978). Once again this is an integral component of classical
EEG analysis and readily used to constitute a captured EEG signal into its composite
waveforms. It should be noted here that the use of FFT is usually done with the application of
a window, this is a predefined period of time the signal in question is broken into so the
necessary calculation may be carried out. The windows used are usually defined by the total
duration of the signal and should theoretically provide an approximation to infinity. Although
there are other variations of FFT that eliminate the need of windows such as discrete Fourier
transform (DFT), none were used in the studies reviewed (Ponsen, 1979). Additionally, all
the aforementioned concepts also have a close relationship to power spectral density (PSD).
PSD is simply defined as the power of a signal expressed as a function of its frequency, in per unit frequency (Abhang et al., 2016).

A slightly more advanced EEG analytic technique is low-resolution electromagnetic tomography (LORETA). This technique is essentially a computerized answer to the EEG inversion problem, which essentially is the issue of trying to discern information about distribution of electrical charges from EEG (Sherlin, 2009). This problem remains a fundamental issue in EEG neuroscience research. Put simply, LORETA analysis uses sets of algorithms to compute EEG data and produce what are referred to as low resolution functional images or voxels of the brain (Dattola et al., 2020). For Standardized LORETA (sLORETA) this resolution is estimated to be 5 mm cubed (Dattola et al., 2020). It should be noted that the term low resolution comes from this resolution’s comparison with other, more direct/anatomical brain imaging techniques such as magnetic resonance imaging or functional magnetic resonance imaging (MRI or fMRI) which can field resolutions close to 1 mm cubed or even smaller (Blazejewska et al., 2019). Additionally, as there are a range of permutations of LORETA analysis, such as the aforementioned sLORETA or exact LORETA (eLORETA), LORETA’s are better thought of as a family of analysis rather than simply one standardized function.

Stochastic event Synchrony is a type of EEG analysis based on the concept of bump modelling (Ghanbari & Moradi, 2021). It breaks down a signal, which must be time-frequency in nature, by predefined groups and then continuing to represent the signal over a time domain, it compares bumps over multiple signals thus assessing for synchronicity. This final comparison is done with respect to and an assessment of a range of factors such as time delay and ratio of spurious events. Finally, a brief mention should also be given to brain electrical source analysis (BESA). Although not an analytic technique in itself, it is a pre-existing program/tool box for the analysis of EEG data (Miltner et al., 1994). Most notable
here though is that one service offered by this tool is the detection of dipoles in EEG readings. A dipole is defined as two opposite charges separated by a small distance (Shipton, 1991). These are often constituted as phenomena of interest onto themselves and can be affiliated with both pathology or normal functioning.

**Cognitive assessments**

Cognitive outcomes are largely bespoke to the instrument used to assess cognition itself so a simple understand of the tool used is all that is required. A list of relevant instruments can be found in table 3.

Table 3

<table>
<thead>
<tr>
<th>Instruments of measurement found in cognitive studies</th>
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<tbody>
<tr>
<td><strong>Letter cancellation test</strong></td>
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<tr>
<td><strong>Stop signal task</strong></td>
</tr>
<tr>
<td><strong>The stroop color and word test</strong></td>
</tr>
<tr>
<td><strong>A mental health scale by Kamlesh Sharma</strong></td>
</tr>
</tbody>
</table>

**Risk of bias tool**

Finally, in keeping with best practice and PRISMA 2020, an assessment of quality will also be made of the studies extracted for (Page et al., 2021). This will be done using
TOOL (Sterne et al., 2019). With this assessment, comments can be made on the possible presence of bias after a systematic inquiry into it as well as providing for a generally demonstration of the quality of studies already existing in the field. Heterogeneity has made appropriate tool selection however this tool was selected as the majority of studies identified by the search conducted can be best classified as randomized control trials or versions of such trials.

**Results**

A total number of 18 studies, including 933 participants, were deemed appropriate for inclusion in this review. Heterogeneity between studies was extremely high, in design, reporting convention, instrumentation and outcome measures. This has meant that meta-analysis was not appropriate. Furthermore, generally whatever naming convention and terms were used internally by a paper were also used in our reporting here in order to best reflect the work of the study at hand. Only slight deviations from this were made when overt errors were corrected for studies and this will be clearly indicated when applicable.

**Effects of Bhramari on markers of autonomic activity**

In total the autonomic studies collectively made up 8 studies, 5 longitudinal and 3 acute, covered 8 key outcomes, all 8 covered in the longitudinal studies and 6 over the acute studies, over 748 participants, 645 in longitudinal studies and 103 over acute studies. A full accounting of autonomic results by study can be found in Table 4 and Table 5, generally studies reported a small but consistent shift to parasympathetic dominance.

**Longitudinal effects of Bhramari on autonomic state and function**

Studies examining the longitudinal effects of Bhramari on autonomic activity are summarised in Table 4. Across the 5 studies, Kuppusamy et al. (2020) and Maheshkumar et al. (2021) were the only randomized, control, single blind studies. Of the others both Rampalliwar et al. (2013) and Jain et al. (2011), could not be randomized or employ a control
group simply by the nature of their inquiry. For both of these studies, generally very little was
provided about their design and methodology. The same can be said about Goyal et al.
(2014), which did employ a control group however reported no randomization technique or
blinding measures. Additionally, Kuppusamy et al. (2020) also provided HF and LF metrics.
They reported a change of $-0.21 \pm 0.02$ in the intervention group, a significant finding.
Pertaining to RMSSD, Kuppusamy et al. (2020) reported a small increase of 2.1 ms which
was statistically significant, and regarding LF/HF ratio, saw a drop of 0.2, also significant.

Change in heart rate (HR) for the longitudinal studies ranged between -6 reported by
Goyal et al. (2014) in mild hypertensives, to -7.8 bpm, which was reported in healthy
(although sedentary) individuals’ response to the cold pressor test by Jain et al. (2011). It
should be noted then for comparison’s sake -6.5 was also reported for a healthy population by
both Kuppusamy et al. (2020) and Rampalliwar et al. (2013). All these figures were also
found to be significant in between group comparisons and in a within group comparison for
Rampalliwar et al. (2013). BP change ranged in the longitudinal studies ranged between SBP
-3.5 mmHg and DBP -3.1 mmHg to SBP -5.2 mmHg and DBP -2.1 mmHg, with both these
figures reported for hyper-reactive individuals to the cold pressor test (Jain et al., 2011;
Rampalliwar et al., 2013). It should be noted however that Goyal et al. (2014) reported
finding outside of this range at -8 but these will be further touched on in the risk of bias
section.

**Acute effects of Bhramari on autonomic state**

Of the three acute studies, one could be constituted as a conventional randomized
control trial. Ghati et al. (2020) followed a protocol conducive to this design both in
randomization and by use of a control group but made no mention of blinding. However, in
this instance it is reasonable to expect after inspecting the information provided that at least
some level of blinding (single blind) did occur. It Should also be mentioned that the control
group in this study used a “SSSS” vocalization. As this does not affect the intervention parameters as described for the purposes of this review this does not exclude this study from inclusion, however this does mean that in this particular case, intergroup analysis will not be appropriate here. Both other studies were single-group in nature. Nivethitha et al. (2021), conducted an investigation over four different pranayama’s, the sequence of which was randomized per participant and again made no mention of blinding. Nivethitha et al. (2017), by nature could not randomize in any capacity and also provided no information on blinding. Ghati et al. (2020) and Nivethitha et al. (2017) also provided LF and HF figures. Ghati et al. (2020) reported that at baseline LF was 87.09±2.88 and HF was 36.3±3.01, during LF was 316.22±2.81 and HF was 38.01, post LF was 74.13 ± 3.09 and HF was 38.9±3.38. Of particular interest here would be LF/HF ratios for baseline and post reading which can be calculated to be LF/HF baseline of 2.40 and LF/HF of 1.91. These numbers have been calculated outside of the study in this review and were not provided in the study report. What the study did report was a log LF/HF and was noted as not being significant between control and experiment condition. Nivethitha et al. (2017) more straightforwardly reported a LF/HF ratio at baseline of 3.05±3.07, during 4.33±8.52 and post intervention of 5.18±4.83.

Finally, HR changes for acute studies ranged from -1.3 to -2 bpm (Nivethitha et al., 2021, Nivethitha et al., 2017). BP change spanned from SBP 0 and DBP 0.9 to SBP -5 and DBP -4.5, only the latter being significant (Goyal et al., 2014, Nivethitha et al., 2017). Regarding RMSSD only Ghati et al. (2020) reported the metric and saw an increase of 1.5 ms which was not significant. Additionally, Ghati et al. (2020) saw a similar drop of 0.5 and Nivethitha et al. (2017) saw an increase of 2.13 in LF/HF, the latter was not significant and the former by nature cannot be verified as significant.
### Table 4

<table>
<thead>
<tr>
<th>Reference</th>
<th>Participant information</th>
<th>Intervention</th>
<th>Data collection protocol</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kuppusamy et al., 2020</td>
<td>Experiment group: 127 males &amp; 109 females. No experience, all healthy. 13 – 18 years (mean n/r).</td>
<td>3 to 6 breaths per minute, then 2 minutes rest for 5 rounds, 5 times a week for 6 months, inhalation/exhalation ratio n/r. Eyes closed, tragus depressed.</td>
<td>ECG, sampling frequency n/r. Testing conducted in the morning after 15 minutes of ‘supine rest seated on a couch’, ECG then recorded for 5 minutes.</td>
<td>Intervention relative to control #: ∆HR; - 6.5 bpm†, ∆R-R; 43.1 ms†††, ∆NN50; 4†, ∆RMSDD; 2.1 ms†.</td>
</tr>
<tr>
<td>Control group: 141 males &amp; 101 females. No experience, all healthy. 14 – 18 years (mean n/r).</td>
<td>Non-treatment group used as control, performed only their usual routine.</td>
<td></td>
<td></td>
<td>Intervention group: HR pre; 74.3±4.3 bpm, HR post; 69.7±5.3*** bpm, ∆HR; -4.6±1.4† bpm, RR pre; 739.1±53.2 msec, RR post; 779±61.6*** msec, ∆RR; 39.9±8.3††† msec, NN50 pre; 26.3±10.2, NN50 post; 29.7±11***, ∆NN50; 3.4±0.5†, RMSDD pre; 60±13.5 ms, RMSDD post; 62.1±16.9 ms***, ∆RMSDD; 2.1±0.3 ms†.</td>
</tr>
</tbody>
</table>

Control group: HR pre; 73.2±6.1 bpm, HR post; 75.2±5.7 bpm, ∆HR; 2±1.5† bpm, RR pre; 773.8±49 ms, RR post; 750±57.5 ms, ∆RR; -3.2±0.9 ms†††, NN50 pre; 27.3±10.6, NN50 post; 26±11.1, ∆NN50; -0.6±0.9†, RMSDD pre; 58.8±14.3 ms, RMSDD post; 58.1 ± 13 ms, ∆RMSDD; -0.7±1.2 ms†.
Goyal et al., 2014

Experiment group: 16 males & 9 females. No experience, mild hypertensives. 40–50 years (mean n/r).

10 breath rounds per session, inhalation/exhalation ratio n/r and four fingers over each eye and thumb blocking each ear. Duration of 6 weeks. Treated with antihypertension medication.

Control group: 15 males & 10 females. No experience, mild hypertensives. 35–50 years (mean n/r).

Intervention relative to control #: ∆RPP; -22.1†.

Rampalliwarr et al., 2013

50 pregnant females, 28 hyper-reactor to cold pressor. No experience, 20–28 years (mean n/r).

Preformed 10 minutes per session twic a day, inhalation/exhalation ratio n/r and hand placement/Mudra n/r. Duration of 15 days of training then 2 months of practice. Self-control.

Sphygmomanometer, cold pressor test, 3°-4° C, hand submerged to the wrist. Elevation over of systolic pressure over 20 mm Hg or diastolic over 15 mm Hg was considered hyper-reactive.

Intervention group: HR pre; 95±1.8 bpm, HR post; 76±2*** bpm, SBP pre; 148±1.6 mmHg, SBP post; 127±2.4*** Hg, RPP pre; 140.6±3.2, RPP post; 96.7±4.1***†, ∆RPP; -43.8#.

Control group: HR pre; 95±2.7 bpm, HR post; 80±2.4*** bpm, SBP pre 144±1.4 mmHg, SBP post; 141±2.4*** mmHg, RPP pre; 136.6±3.2, RPP post; 114.7±5.3***, ∆RPP; -21.9#.

Findings of interest: ∆Post Systolic; -5.2*** Hg, ∆Post Diastolic; -2.1*** Hg, ∆HR; -6.5*** bpm.

∆Basal Systolic; 3.1*** Hg, ∆Basal Diastolic; 4.8*** Hg, ∆During Systolic; 9.1*** Hg, ∆During Diastolic; 6.9*** Hg, ∆Post Systolic; 5.2*** Hg, ∆Post Diastolic; 2.1*** Hg, ∆HR; 6.5*** bpm. Pre Basal Systolic; 146.4±5.9 Hg, Pre Basal Diastolic; 93.2±3.2 Hg, Pre during Systolic; 167.9±6 Hg, Pre during Diastolic 106.8±4.1 Hg, Pre rise in Systolic; 21.5±4.45 Hg, Pre rise in Diastolic; 13.5±2.6 Hg, Pre HR; 80±3.4 bpm. Post Basal Systolic; 143.2±4.7 Hg, Post Basal Diastolic; 88.4±2.1 Hg, Post During Systolic; 158.7±6.32 Hg, Post During Diastolic; 99.9±3.4 Hg, Post HR; 73.5±2.8 bmp. Post rise in Systolic; 15.5±2.9 Hg, Post rise in Diastolic; 11.4±2.8 Hg.
<table>
<thead>
<tr>
<th>Jain et al., 2011</th>
<th>Jain et al., 2011</th>
</tr>
</thead>
<tbody>
<tr>
<td>22 males &amp; 32 females. Experience n/r, 9 male and 12 female hyper-reactors, 13 male and 20 female hypo-reactors to cold pressor test. 18 -24 years (mean n/r).</td>
<td>Preformed for 15 minutes in the morning, 15 minutes in the evening, inhalation/exhalation ratio n/r and hand placement/Mudra n/r. Duration of 90 days. Self-control.</td>
</tr>
<tr>
<td>Sphygmomanometer, cold pressor test, 3°-4°C hand submerged to the wrist. Elevation over of systolic pressure over 20 mmHg or diastolic over 15 mmHg was considered hyper-reactive.</td>
<td>Findings of interest: post intervention systolic hyper-reactors down from 14 to 3, post intervention diastolic; hyper-reactors down from 7 to 1</td>
</tr>
<tr>
<td>Base hypo-reactors Systolic; 114.7±6.7, base hypo-reactors Diastolic; 74.7±5.9, base hyper-reactors; 119±6.3, base hyper-reactors Diastolic; 76.4±5.7. Hypo-reactors post test rise Systolic; 13.2, Hypo-reactors post test rise Diastolic; 9.1. Hyper-reactors post test rise Systolic; 19.2, Hyper-reactors post test rise Diastolic; 14.7. Hyper-reactors post intervention rise Systolic; 15.7±2.9*** (T value 6.4), hyper-reactors post intervention rise Diastolic; 11.6±1.9*** (T value 5.6). Hyper-reactor HR pre; 79.1±5.2, Hyper-reactor HR post; 71.3±5.4**. Hyper-reactors respiratory rate pre; 20.4±2.2, Hyper-reactors respiratory rate post; 16.5±1.4**.</td>
<td>Base hypo-reactors Systolic; 114.7±6.7, base hypo-reactors Diastolic; 74.7±5.9, base hyper-reactors; 119±6.3, base hyper-reactors Diastolic; 76.4±5.7. Hypo-reactors post test rise Systolic; 13.2, Hypo-reactors post test rise Diastolic; 9.1. Hyper-reactors post test rise Systolic; 19.2, Hyper-reactors post test rise Diastolic; 14.7. Hyper-reactors post intervention rise Systolic; 15.7±2.9*** (T value 6.4), hyper-reactors post intervention rise Diastolic; 11.6±1.9*** (T value 5.6). Hyper-reactor HR pre; 79.1±5.2, Hyper-reactor HR post; 71.3±5.4**. Hyper-reactors respiratory rate pre; 20.4±2.2, Hyper-reactors respiratory rate post; 16.5±1.4**.</td>
</tr>
</tbody>
</table>
Maheshkumar et al., 2021

Experiment group: 6 males & 7 females. Experience n/r, all healthy. 11 – 19 years (13.8 ± 0.5).

Preformed for 5 minutes then 2 minutes of rest, inhalation/exhalation ratio n/r and eyes closed and tragus depressed. Duration 5 times a week for 6 months.

Salivary cortisol measured. Cold pressor test, 10° C with right hand up to the forearm. Experiment preformed in the morning between 10 am to 12:30 pm. Account made for the proliferative phase of menstruation for female participants.

Findings of interest: 20 min post; 0.73±0.12*† mg/dl, 60 min post; 0.22±0.03***††† mg/dl.

Intervention group: CPT pre; 0.25±0.04 mg/dl, CPT post; 0.26±0.04 mg/dl, 20 min pre; 0.64±0.11 mg/dl, 20 min post; 0.73±0.12*† mg/dl, 40 min pre; 0.44±0.03 mg/dl, 40 min post; 0.41±0.03 mg/dl, 60 min pre; 0.28±0.04 mg/dl, 60 min post; 0.22±0.03***††† mg/dl.

Control group: 5 males & 8 females. Experience n/r, all healthy. 11 – 19 years (14.5 ± 0.9).

Non treatment group used as control, performed no other activity other then their usual routine.

Findings of interest: 20 min post; 0.21±0.07 mg/dl, CPT post; 0.23±0.02 mg/dl, 20 min pre; 0.63±0.08 mg/dl, 20 min post; 0.66±0.10 mg/dl, 40 min pre; 0.47±0.09 mg/dl, 40 min post; 0.46±0.07 mg/dl, 60 min pre; 0.24±0.07 mg/dl, 60 min post; 0.25±0.04 mg/dl

Note. *p = <.05, **p = <0.01, ***p = 0.001. †Between group = <0.05, ††between group = <0.01, †††between group = <0.001.

Table 5

Studies examining the acute effects of Bhramari on markers of autonomic function

<table>
<thead>
<tr>
<th>Reference</th>
<th>Participant information</th>
<th>Intervention particulars</th>
<th>Data collection protocol</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
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</tbody>
</table>
Experiment group: 17 males & 15 females. No experience, all hypertensive. 30 – 70 years (mean n/r).

Preformed for 5 minutes, eyes closed, 6 to 4 bpm, inhalation/exhalation ratio n/r and hand placement/Mudra n/r.

Ambulatory blood pressure monitor. Digital Holter recorder, low frequency band 0.04-0.15 Hz and high frequency band 0.15-0.4 Hz. 5 mins of resting, 5 mins of intervention, then recover (duration n/r), readings taken throughout all phases.

Control group: 19 males & 16 females. No experience, all hypertensive. 30 – 70 years (mean n/r).

Slow breathing used as control. Preformed for 5 minutes, eyes closed, 6 to 4 bpm, inhalation/exhalation ratio n/r and hand placement/Mudra n/r, “sss” used instead of “mmm”.

Preformed for 5 minutes, inhalation/exhalation ratio n/r and hand placement/Mudra n/r. 4 experiment interventions, only one Bhramari.

Nivethitha et al., 2021

20, sex n/r, experience n/r, all healthy. Age range n/r (23.4 ± 3.1).

Preformed 4 interventions, 4 experiment conditions used, 4 groups of 5 randomized to perform interventions in varying orders with 5 min between intervention conditions.

Sphygmomanometer. 4 intervention conditions used. Systolic baseline; 115±14.5 Hg, Systolic during; 119.8±15.2 Hg, Systolic post; 112±13.8 Hg, Diastolic baseline; 70.8±9.2 Hg, Diastolic during; 78.4±10.7 Hg, Diastolic post; 72.1±8.8 Hg

Findings of interest: RMSDD during; 21.4±1.6* ms

Intervention group: HR n/r, N-N n/r, R-R n/r, RMSDD pre; 20.9±1.7 sec, RMSDD during; 21.4±1.6* sec, RMSDD post; 22.4±1.7 ms, Systolic pre; 131.7±9.6 Hg, Systolic during; 131.7±12.8 Hg, Systolic post; 131.7±10.9 Hg, Diastolic pre; 91.4±7.7 Hg, Diastolic during; 94.5±8.8 Hg, Diastolic post; 92.3±8.7 Hg.

Control group: HR n/r, N-N n/r, R-R n/r, RMSDD pre; 23.4±1.7 sec, RMSDD during; 28.9±1.7 sec, RMSDD post; 21.4±1.6 sec, Systolic pre; 127.8±12.9 Hg, Systolic during; 127.1±16.3 Hg, Systolic post; 125.8±12.6 Hg, Diastolic pre; 88.1±9.8 Hg, Diastolic during; 87.4±11.3 Hg, Diastolic post; 88.3±9.2 Hg

Findings of interest: HR during; 92.4±11.3* bpm, Diastolic during; 78.4±10.7* Hg.

HR baseline; 84±10.9 bpm, HR during; 92.4±11.3* bpm, HR post; 86.5±12.5 bpm, Systolic baseline; 115±14.5 Hg, Systolic during; 119.8±15.2 Hg, Systolic post; 112±13.8 Hg, Diastolic baseline; 70.8±9.2 Hg, Diastolic during; 78.4±10.7* Hg, Diastolic post; 72.1±8.8 Hg
Nivethitha et al., 2017

9 males & 7 females. One year of yoga experience, all healthy. Age range n/r (23.5 ± 3.0).

Preformed for 5 minutes, 6 bpm, inhalation/exhalation ratio n/r and hand placement/Mudra n/r.

ECG, sphygmomanometer, electrodes placed as standard limb lead II configuration, sampling rate of 1024Hz. HRV recordings taken for 5 mins at baseline, 5 min during and 5 mins post-intervention.

Findings of interest #: ΔHR during; 4.17 bpm*, ΔSBP post; -5 Hg*, ΔSDP post; -4.5 Hg*.

HR pre; 83.1±8 bpm, HR during; 87.4± 6.4* bpm, HR post; 81.8± 6.6 bpm, R-R pre; 736.3±81.4 msec, R-R during; 727.2±82.1 msec, R-R post; 745.2±64.7 msec, N-N pre; 61.8±47.3, N-N during; 51.6±28.7, N-N post; 57.7±52.8, RMSDD pre; 39.3±16.9 sec, RMSDD during; 37.4±19 sec, RMSDD post; 40.6±13.1 sec. Systolic pre; 115.5±7.8 Hg, Systolic post; 110.5±8.6* Hg, Diastolic pre; 76.9±6.3 Hg, Diastolic post; 72.4±5.9* Hg

Note. *p = <.05, **p = <0.01, ***p = 0.001. †Between group = <0.05, ††between group = <0.01, †††between group = <0.001.
Effects of Bhramari on brain waves

A summary of EEG results by study can be found in Table 6. Studies showed increased paroxysmal gamma waves, symmetrical activation particularly in the temporal regions and an augmentation of wave power and expression. Across the six EEG studies a total of 10 participants were observed. This was over two notable data sets, one of eight individuals, reported in Jin et al. (2014), Vazquez et al. (2013), and Vialatte et al. (2009), and one of two individuals reported in Prasad et al. (2006), Prasad and Matsuno (2007) and Prasad et al. (2007). The set of eight is listed as all men and while the sex of the set of two was not reported. For performance of Bhramari Mudra specifically both Jin et al. (2014) and Vazquez et al. (2013) cited Vialatte et al. (2009), which used the Shanmukhi Mudra. The studies were quasi-experimental with EEG assessed comparing control conditions to experiment conditions with no mention of blinding. Each of the reviewed studies used different primary approaches to analyse the EEG data. Of particular note on this point is Vazquez et al. (2013), who conducted a novel analysis along with more traditional ones. This processing consisted of first principal component analysis, then blind signal separation, followed by feeding into a proximal gamma waves detector and Source localisation before back projection in order to reconstruct the full 128 channel EEG. Findings of this novel analysis were essentially the same as conventional analysis and reflected in table 6 as well.
<table>
<thead>
<tr>
<th>Reference</th>
<th>Participant information</th>
<th>Intervention particulars</th>
<th>Technical and Evaluatory specifics</th>
<th>Results</th>
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</thead>
<tbody>
<tr>
<td>Jin et al., 2014</td>
<td>1 beginner*, 1 intermediate*, 1 expert*, sex n/r, Ages n/r, handedness n/r</td>
<td>20 rounds preformed, inhalation/exhalation ratio n/r, hand placement Mudra n/r</td>
<td>128 channel EEG at a sampling frequency of 2048 Hz. Stochastic Events Synchrony.</td>
<td>Stable symmetrical PGW events detected in temporal lobe for all participants. Signal particularly stable at exaltation.</td>
</tr>
<tr>
<td>Prasad et al., 2007</td>
<td>1 four months of experience, 1 participant with no experience. Sex n/r. 26 and 35 years, handedness n/r</td>
<td>&quot;Planned for&quot; 15 - 20 rounds, inhalation/exhalation ratio n/r, hand placement/Mudra n/r. False BP used as control where all physical positioning was the same but the humming sound was not produced.</td>
<td>128 channel EEG sampling frequency of 256 Hz. 21 channels represented in results as raw data. Brain Electrical Source Analysis, LORETA and RSA.</td>
<td>Intervention resulted in an increase of low frequency (&lt; 35 Hz) dipole events in the prefrontal cortex. No such interpretations of high frequency dipole events can be made from the data set. Relative spectral power across all bands is also affected by intervention.</td>
</tr>
<tr>
<td>Study</td>
<td>Experience</td>
<td>Participants</td>
<td>Age</td>
<td>Handedness</td>
</tr>
<tr>
<td>-------------------------------</td>
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</tr>
<tr>
<td>Prasad &amp; Matsuno, 2007</td>
<td>1 month</td>
<td>1 participant with no experience. Sex n/r. 26 and 35 years, handedness n/r</td>
<td>10 rounds preformed, inhalation/exhalation ratio n/r hand placement/Mudra n/r. False BP used as control where all physical positioning was the same but the humming sound was not produced.</td>
<td>128 channel EEG sampling frequency of 256 Hz. Detrended fluctuation analysis.</td>
</tr>
<tr>
<td>Parsad et al., 2006</td>
<td>1 month</td>
<td>1 participant with no experience. Sex n/r. 26 and 35 years, handedness n/r</td>
<td>15 - 20 rounds preformed, inhalation/exhalation ratio n/r, hand placement n/r.</td>
<td>128 channel EEG at sampling frequency of 1024 Hz. Brain Electrical Source Analysis and RSA.</td>
</tr>
<tr>
<td>Vazquez et al., 2013</td>
<td>Beginner*, Intermediate*, Expert*</td>
<td>Sex n/r, ages n/r, handedness n/r</td>
<td>20 rounds preformed, inhalation/exhalation ratio n/r, hand placement n/r.</td>
<td>128 channel EEG at sampling frequency of 2048 Hz. 3 PGW detection methods used, one novel proposed.</td>
</tr>
</tbody>
</table>
Vialatte et al., 2009

1 beginner*, 6 intermediate*, 1 expert*. All male, all right-handed, ages n/r.

20 rounds preformed, respiratory rate n/r, inhalation/exhalation ratio n/r, modified Shanmukhi Mudra. False BP used as control where all physical positioning was the same but the humming sound was not produced, participants attempted to mimic respiratory rate of intervention condition.

128 channel EEG at sampling frequency of 2048 Hz. Independent component analysis. FFT-based power spectrum analysis and LORETA.

Power drop in low frequency waves, significant in Theta and not so in Alpha. At exhalation, a large increase in high frequency waves, this effect was not observed in the control condition, this effect was largely affiliated to the left temporal region.

Note. Beginner* defined as; never practiced Bhraimari, intermediate* defined as; having practiced for two sessions per day for 31 - 34 days, experienced* defined as; having practiced for two sessions per day for 4 months.
Effects of Bhramari on cognitive function

The design, participant information, and results for studies examining the effect of Bhramari can be found in Table 7 and table 8. Studies showed participants performing cognitive tasks with greater accuracy and increased concentration. A total of 4 cognitive studies were extracted for which covered 175 Individuals in total. 125 over the longitudinal studies and 50 on the acute.

Longitudinal effects of Bhramari on tests of cognitive function

Of the Longitudinal studies neither reported any kind of blinding particulars. Srivastava et al. (2017), used a self as control design but had no control group so no randomization could occur. This is the same with Shankari (2012). Both of these studies reported very little regarding their design and method.

Acute effects of Bhramari on tests of cognitive function

Regarding the Acute studies, once again no blinding was reported for either. Pradhan et al. (2018) used a self as control design but had a dedicated control condition as well as an experiment condition which participants were randomized to. This was the same for Rajesh et al. (2014).
Table 7

Longitudinal studies examining the effects of Bhramari on aspects of cognitive performance

<table>
<thead>
<tr>
<th>Reference</th>
<th>Participant information</th>
<th>Bhr P. Intervention particulars</th>
<th>Technical and Evaluatory specifics</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Srivastava et al., 2017</td>
<td>60 total participants, sex and experience level n/r, 16 - 20 years</td>
<td>Intervention performed for 15 minutes once a day for 20 days, inhalation/exhalation ratio n/r and hand placement/Mudra n/r.</td>
<td>Mental health scale by Kamlesh Sharma (1996), 60 total items, 30 positive, 30 negative.</td>
<td>Pre; 74.26 ± 10.25. Post; 94.46 ± 10.92. T-value 5.61*</td>
</tr>
<tr>
<td>Shankari, 2012</td>
<td>65 total participants, all described as having low attention and academic performance, sex and experience level n/r, age 14 - 15 years</td>
<td>Intervention duration n/r, preformed for 7 days, inhalation/exhalation ratio n/r and hand placement/Mudra n/r.</td>
<td>Stroop word test, scores out of 100.</td>
<td>Pre; 23.44. Post; 32.63*</td>
</tr>
</tbody>
</table>

Note. *p = <.05, **p = <0.01, ***p = 0.001.

Table 8
<table>
<thead>
<tr>
<th>Reference</th>
<th>Participant information</th>
<th>Intervention particulars</th>
<th>Technical and Evaluatory specifics</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pradhan et al., 2018</td>
<td>8 male, 11 female, all constituted as visually impaired with no yogic experience. 15 - 19 years, 15.9 ± 1.6</td>
<td>Bh. P preformed for 10 minutes, preformed while seated cross legged and with eyes closed. Modified Shanmukhi Mudra † used, inhalation/exhalation ratio n/r.</td>
<td>Modified Braille version of six-letter cancellation test. Test alterations developed with the collaboration of 7 visually impaired teachers at school.</td>
<td>Experiment. Pre; 21 ± 7.43, Post; 31.53 ± 9.93***, Δ 50%. Pre wrong; 0.95 ± 1.22, Post wrong; 1.00 ± 1.41, Δ 5%. Control. Pre; 18.84 ± 6.27, Post; 33.37 ± 9.06***, Δ 77%. Pre wrong; 0.95 ± 1.35, Post wrong; 1.58 ± 1.43*, Δ 66%.</td>
</tr>
<tr>
<td>Rajesh et al., 2014</td>
<td>31 male, experience level ranging from 6 months to 5 years. 19 - 31 years, 23.9 ± 3.5</td>
<td>Bh. P. performed for 10 minutes, inhalation/exhalation ratio n/r and hand placement/Mudra n/r</td>
<td>Stop signal task used, stop signal delay initially set at 250 ms with increase and reductions of 50 ms keep probability of successful inhibition approaching 50%.</td>
<td>Experiment. SSRT Pre; 243.75 ± 40.16 ms, Post 232.67 ± 43.82 ms*. Go RT Pre; 732.24 ± 184.10 ms, Post; 732.24 ± 193.40 ms**. Control. SSRT Pre; 239.44 ±</td>
</tr>
</tbody>
</table>
Deep breathing preformed for 10 minutes, inhalation/exhalation ratio n/r and hand placement/Mudra n/r.

20.59 ms, Prost 237.35 ± 38.40 ms. Go RT Pre; 733.88 ± 170.89 ms, Post; 751.81 ± 182.16 ms.

Note. *p = <.05, **p = <0.01, ***p = 0.001.
Risk of bias assessment

The Revised Cochrane risk-of-bias tool for randomized trials (RoB 2) tool was used to assess for risk of bias across all studies (Sterne et al., 2019). Due to the large heterogeneity between studies, which were not always in the form of randomised controlled trials, the application of this tool was a complex and questionable procedure. As a result, the utmost generosity was taken when processing studies and a full account by study can be found in Table 9. Even so some extra points on risk-of-bias factors need mentioning, such as Goyal et al. (2014) reporting of BP. In the study itself this was only referred to as BP however upon inspection of the figures, it became evident that these numbers were much more in line with SBP, so the change was made but such lapse in reporting precision is not confidence inspiring with regards to the quality of a stud. Indeed, a lack of clarity in reporting or outright lack of reporting was generally the primary limiting factor of the studies at hand. Furthermore, due to their nature it would be inappropriate to apply the RoB 2 tool to the EEG studies so some statements assessing their risk of bias/quality must be made as well. Generally, these studies are fairly robust for what they were although again often not reporting extensively on study design or experiment protocol, rather choosing to focus on outlining EEG processing techniques applied. This is not unusual in this type of work however it does limit their usefulness in determining finer traits of the present effect.
<table>
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<tr>
<th>Author</th>
<th>risk of bias arising from the randomization process</th>
<th>risk of bias due to deviations from the intended interventions. (effect of assignment to intervention)</th>
<th>Risk of bias due to deviations from the intended interventions. (effect of adhering to intervention)</th>
<th>Risk of bias due to missing outcome data</th>
<th>Risk of bias in measurement of the outcome</th>
<th>risk of bias in selection of the reported result</th>
<th>Overall risk-of-bias judgement</th>
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<td>Shankari, 2012</td>
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Note. The Cochrane risk-of-bias tool for randomized trials (RoB 2) tool used to assess quality of the studies extracted for. EEG studies were excluded from this table as they could not fairly be constituted as any kind of randomized trial.
Discussion

The review conducted here was, to the best of the authors’ knowledge, the broadest, though and inclusive of its kind, inspecting the effects of Bhramari using as domains of outcomes as possible. Inspection of the extracted data therefore reveals a consistent, although arguably modest effect as a result of Bhramari. Beginning with the ANS studies, all reported at least some level of effect. When the ranges for the effect in question are considered, some interesting patterns become apparent. Namely, that both size of the effect and statistical significance appear to be more present in the longitudinal studies as opposed to the acute studies. This is true across changes in HR, RMSSD and LH/HF figures with only longitudinal studies reporting any kind of significance (Goyal et al., 2014; Jain et al., 2011; Kuppusamy et al., 2020; Nivethitha et al., 2017; Nivethitha et al., 2021). It is also essentially true for BP with only a single acute study reporting significance at its time scale as opposed to both longitudinal studies reporting the same metric (Jain et al., 2011; Nivethitha et al., 2017; Rampalliwar et al., 2013). However, as mentioned, although the finds of these acute studies may not be overtly significant, they too move in the same direction as the longitudinal studies. This could be suggestive of the fact that the modest effects of Bhramari compound over time, becoming more notable (significant) and suggest parasympathetic dominance. This though is not to discredit the acute effects which do seem present as well.

Exacerbating the importance of this point then are the EEG studies which were all performed on an acute time scale and all demonstrated at least some level of consistent effect regardless of experience level. Firstly, of note is the alternation of spectral power and basic symmetry. Prasad et al. (2006) found that although every wave band experienced some level of alteration, due to confounding factors accounted for by the study at hand, not all wave types could be equally inspected. Importantly though Theta and Gamma waves could, which
are associated with the relaxed meditative state and a focused, willful directing of consciousness respectively. Both participants in this study showed an offset of Theta waves with a new wave pattern (spikes not present in the control condition) and an increased gamma wave power initially before falling below control gamma wave power levels and then maintaining a constancy. This was also the case regardless of hemisphere, however other studies using more targeted analytics showed patterns of lateralization.

Jin et al. (2014), Vazquez et al. (2013) and Vialatte et al. (2009) all suggest that activity was largely focused in the temporal lobes. In particular the work of Vialatte et al. (2009) should be noted who used a LORETA to determine location, which placed activity on the left temporal lobe, this location was also specified by Vazquez et al. (2013). These findings however, are only pertinent to PGW activity and relatively little can be said about Theda symmetry. Although regarding Theda waves, the results of Vialatte et al. (2009) also found the same dropping in power effect for both wave types observed by Prasad et al., 2006. Activity in the frontal cortex was also detected by Prasad et al., 2007. This investigation of dipoles showed that at “low” frequencies, a number (5) of frontal/prefrontal cortex dipoles were detected as opposed to none at baseline readings. The most overt limitation with this finding though is that the study constituted anything under 35 Hz as “low”, which can encompass the whole spectrum of named brainwaves. In contrast the observation that no such pattern could be detected for “high” frequency waves may be somewhat unsurprising, but regardless this does show that Bhramari is resulting in some key frontal/prefrontal cortical activity. However, a very similar dipole spread was found in the control (fales Bhramari) condition, which mimicked all of Bhramari except for the humming, this is juxtaposed by the finds of Prasad and Matsuno (2007) who suggested that humming was integral to the phenomena. As such, although all studies at hand showed some level of measurable and
consistent effect, limitations in study design have meant that it is difficult to determine much past this point, in terms of mechanics particulars, with the data at hand.

Indeed, regarding the limitations of the EEG studies, most overtly the issues of sample size and cohort should be mentioned. As best as can be determined here, the six studies covered ten individuals in total and all of them are likely male. This added with the fact that every study used for the most part a different analytic technique, even the reporting of data from simply one study to the other is different which exacerbates the difficulty of cross-checking study results in order to find meta conclusions. Additionally, some of these techniques of analysis are somewhat antiquated now and the lack of convention with even brainwave ranges, for example high and low frequency waves as defined by Prasad et al, 2007 further adds to this aforementioned point.

Interestingly though, a final point of consistency between Vialatte et al. (2009) and Vazquez et al. (2013) was the fact that the signal detected was overtly affected by the inhalation/exhalation cycle. Although not the total focus of either study, both reported that at exhalation the signal detected became clearer and more stable as compared to inhalation. These studies were Investigations of chiefly PGW patterns so this phenomenon is consistent over them as observed in the cortex. Vialatte et al. (2009) provide some metrics on this occurrence and reported that for four (B1, I2 - 5) of its eight participants, the PGW signature itself of interest was actually only present at exhalation. For the other participants it was present at inhalation also however was as mentioned, weaker and more unstable. But a latency figure can be derived from the data on the first four individuals which was 1.2±0.1 sec. Again, although the combination of these two studies is too small and limited to provide for a wide scale assessment or confirmation of such an effect, these findings indicate the presence of a consistent measurable effect inhalation and exhalation has on the cortex.
What furthers this line of thinking is work done more recently such as Herrero et al. (2018). This study builds on previous work done on rodents to determine higher order neural phenomena associated with breathing (Ito et al., 2014; Nguyen Chi et al., 2016). In these models these oscillations were identified to be present in the Hippocampus and somatosensory cortex. In Herrero et al. (2018), similar effects were found to be present in humans as well in the same regions. This is significant as this type of respiratory-locked activity is not unlike that which is seen in the extracted studies, particularly in the same temporal region. Furthermore, briefly respiratory sinus arrhythmia (RSA) should also be mentioned as this is another example of a very similar phenomena. As such, what the collective of these phenomena and datasets may be suggesting is that just as RSA is a kind of quantifiable neural phenomena operating as a function of breathing below the brainstem, so too may there be a very similar phenomena occurring above it.

Building on this neural work are the finds of cognitive studies. However, particularly in the longitudinal studies several limitations raise serious concerns with interpretation of data. Shankari (2012) made no accounting for the learning effect present from repeatedly asking participants to undergo the same testing over consecutive days, and Srivastava et al. (2017) essentially used a subjective measure reporting feeling. Although not defective in itself, the results reported do not fit the profound claims the studies makes. This is unfortunate as it means no meaningful investigation can be made at the longitudinal cognitive effects which are of interest as ANS findings suggesting the building of an effect overtime. However, EEG studies in contrast do clearly demonstrate a meaningful effect operating on the short term so this at least should be briefly touched on.

In contrast to the longitudinal studies, acute studies provided good methodological reporting and appropriate design including making accounts for the learning effect. However, this does not mean that this reporting is without critique. For example, Rajesh et al. (2014),
data suggest on the surface that there was a faster response time in the post intervention group which was significant (11.08 ms faster) and that go signal response was slower, also significant (30.57 ms slower). The study proposed that this meant a higher attention and concerted strategy was being used after the intervention and suggested that this was due to it. But these findings could simply have been the result of participants becoming more comfortable taking the test at hand, something that a basic learning effect analysis would not necessarily detect. This is furthered by the fact that the control group (deep breathing) saw movement in the same direction, albeit not significant (2.09 ms faster at the stop signal, 17.93 ms slower on the go signal). Not only was this limitation not touched on, but no real limitations at all, save a single sentence, were mentioned making it difficult to trust authors' interpretations of their results. Nevertheless interesting though, is that these results are somewhat in keep with the findings of Prasad et al. (2007) dipole assessment in the frontal/prefrontal cortex, which were are of the most direct EEG metrics reported that are also linked to possible cognitive function (Shipton, 1991). A better task of reporting was done by Pradhan et al., 2018, who saw similar results in their braille six letter cancelation test but mentioned that the control group (breath awareness) and intervention group both saw significant improvement in its test. What is of interest here though is that only the Bhramari group did not see a significant rise in wrong answers as well with its increase. This then is the only meaningful suggestion in the cognitive studies reviewed that Bhramari specifically is having a novel effect not analogous to other yogic breathing techniques, but it is still highly tentative at best.

As is highly apparent though in the aforementioned and table 8, risk of bias and issues of design were extremely high in the studies reviewed. The tool used to generate these figures was the Cochrane risk-of-bias tool for randomized trials (RoB 2) (Sterne et al., 2019). This may not be the ideal tool of the task at hand however and if not inspected closely, it may lead
a reader to inappropriate conclusions. This is due to the large heterogeneity in the studies reviewed. Although this is an issue for other reasons as well, it has also made the blanket application of a tool designed clearly for pharmaceuticals randomized trials across so many domains of study difficult. For example, the algorithms of the tool itself, would sometimes lead to instant high-risk classifications of certain studies. The EEG studies for instance did not mention randomization simply because of the nature of their investigation, in this tool that has meant that all are immediately high risk however that does not necessarily mean that they were poorly conducted. They were largely in keeping with how other EEG studies of their kind are reported. Furthermore, the same algorithms did not detect phenomena that they perhaps should. For example, disparities in authors' conclusions/interpretations of results as compared to the results reported. Rajesh et al. (2014) for instance in the cognitive studies gave an exceedingly generous and arguably inappropriate interpretation of their results while not mentioning their overt limitations. There were also instance of author out right commenting on parameters not reported in their results such as Goyal et al. (2014) in the ANS studies, who only provided BP (we assumed it to be SBP) and then in their discussion comment on both SBP and DBP levels. Instances such as these were simply not detected by the algorithms of the tool but certainly can be thought of as speaking to some underlying bias or lack of quality on the part of the study. These points are not to suggest that the studies at hand did not have severe issues and should be considered of high risk of bias, but rather that the assessment of risk of bias in these studies is nuanced and not all the results at hand should be considered with outright suspension.

As such the consistent effect alluded to by studies of Bhramari should at least be considered present if nothing else and some possible underlying mechanisms for it can be considered. These mechanisms are due primarily because of the breath component itself and the mudra/s applied in Bhramari. In order to gain an understanding of these the cranial nerves
must be touched on first. The twelve cranial nerves protrude out directly from the brain and are therefore considered as part of the PNS. Regarding nerves, a few are of key importance here, the Trigeminal nerve (5) and the Vagus nerve (10) (Urfy & Suarez, 2014). Both of these nerves are considered to be mixed, meaning they possess afferent and efferent pathways. The Trigeminal nerve (the largest of the cranial nerves) has three branches, the largest of these branches is the Mandibular nerve and that in turn has another branch that makes up the auriculotemporal nerve located in the upper ear (Greenberg & Breiner, 2021). This provides afferent input for a number of locations on the side of the head. The Vagus nerve is the longest of the cranial nerves and spans from several nuclei located in the medulla oblongata (Bordoni et al., 2018). This nerve is particularly relevant to ANS, considered to be one of its chief drivers and is critical for the functioning of organs all throughout the thoracic cavity (Bordoni et al., 2018). It should also be noted that there is a branch of the Vagus nerve, the Auricular Branch of the Vagus Nerve (ABVN), that is located in the central area of the external ear (Murray et al., 2016).

In terms of stimulation, a neuron responds to stimulation by producing an electrochemical pulse (an action potential) down its axon (Thau et al., 2021). Because of this it is possible to stimulate a neuron artificially and traditionally this has been done by the use of low voltage electrical stimuli presented at or near the site of nerves (Yap et al., 2020). As this is normally done through the skin, this type of stimulation is referred to as transcutaneous. With respect to the aforementioned nerves, there is a president for transcutaneous stimulation of adaptive health outcomes (Yap et al., 2020). These have all focused previously on ABVS transcutaneous electrical stimulation though and little literature exists on other forms of stimulation. A study by Addorisio et al. (2019) however, investigated mechanical (using a small oscillating device) transcutaneous stimulation of the ABVS. They found that this did result in reduced markers of inflammatory responses in its participants and
therefore is an example of specifically mechanical transcutaneous stimulation, via the ABVS, having an autonomic effect. This later point is also supported by additional studies (Bretherton et al., 2019). This then provides a course to think that by depressing the tragus with the fingers in Bhramari and producing a resonance (humming) may produce some type of mechanical transcutaneous stimulation resulting in some of the phenomena observed in the studies at hand.

To understand possible mechanisms linked to breath diaphragmatic movement and neurology must be understood. The diaphragm itself is primarily innervated by the phrenic nerve c. The phrenic nerve is a mixed nerve originating from the C-3 to C-5. It tracks down from this location as a left and right branch passing down the neck, into the thoracic cavity innervating the two domes of the diaphragm. The right branch has a direct route to the diaphragm, while the left along its way also receives sensory input from pericardium tissue. It can be organized at least in part as a musculatory branch of the cervical plexus. It should be noted that this is also where the greater auricular nerve (a cutaneous nerve) also originates from and maybe contributing to possible mechanisms of effect. Respiratory drive for this nerve can be traced to the preBötzinger complex (a neural network located in the medulla oblongata) and retrotrapezoid parafacial complex (another neural network also located in the medulla but superior to the preBötzinger complex) (Bordoni et al., 2018). the Vagus nerve itself originates from the medulla as well with its specific nuclei being primarily, the dorsal motor nucleus of the Vagus nerve, nucleus ambiguous and the solitary nucleus (Bordoni et al., 2018). The significance of this is that it is the same region of the brain responsible for the generation of respiratory drive (Zaccaro et al., 2018). Vagal nerve activity and phrenic nerve activity are also believed to have a direct relationship due to an anastomosis in the crura (Bordoni & Zanier, 2013). The Vagus nerve passes through this part of the diaphragm and innervates it as it follows with the oesophagus through the oesophageal hiatus. As such it is
often constituted that the Vagus nerve provides the sympathetic component of signalling to the diaphragm (Hamasaki, 2020). This means that there is an intimate relationship between ANS functioning, the vagus nerve and diaphragmatic activity (both by the mussel and its associated structures. All of this the unique cadence of Bhramari may also be employing to create its effect, in fact, this may be one of the mechanisms underpinning the general adaptive effect controlled breathing conditions (deep breathing/awareness of breathing controls) appeared to have.

Briefly, the fact that all respiration was done by dedicated nasal breathing would also contribute to these mechanisms of effect for a number of these general breath awareness controls. As mentioned elsewhere nasal breathing has a significant positive effect on general bodily functioning and requesting all participants to breathe nasally for any condition, intervention or control, would capitalize on this. However, only the Bhramari condition is likely to capitalize on the nose’s relationship to NO due to its humming element. Therefore, NO could also be contributing to the mechanisms responsible for the subtle disparity of effect observed across a number of the reviewed studies.

Regardless of mechanism however, as mentioned some effect does seem apparent. This effect appears to be more notable over time however a consistent acute effect also appears present. The clinical significance of this effect and its properties are difficult to comment on due to poor quality of studies at hand but in comparison to exercise interventions, the effect present is analogues to approximately 30 minutes of light activity in a sedentary population (Wheeler et al., 2019). However continued exercise can be suspected to continue to yield increasingly positive health outcomes for participants until outright healthy functioning is reached. In contrast it cannot be determined here where the compounding element of the Bhramari effect caps. As such Bhramari may be a good adjust to
conventional interventions but perhaps not notably more than most other breath awareness meditations/techniques.

Further research suggestions:

- EEG studies exploring the respirative-oscillatory effect of breathing.

- Mudra mechanical contribution to the effect of Bhramari

- Cognitive studies both confirm the suspected effect of Bhramari on attention as well as on other elements of cognition.

- Repeat studies across all domains in order to bring up the quality of work in the field, expand population qualities and confirm suggested effects.
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