CHAPTER-7

Discussion

OVERVIEW:

Breath regulation has been an essential component of yogabased breathing (Pranayama) practiceswhich would include either uninostril breathing or high frequency yoga breathing. It is evident from the present literature that the practice of Pranayama produces specific, yet differential physiological responses (Madanmohan et al., 2005). Brain is one of the most highly perfused organs in whichacute hypoxia and/or hypercapnia were reported to produce marked dilation of cerebral arteries and arterioles and increase blood flow, while hypocapnia showed to causes constriction and decrease blood flow (Cipolla, 2009). TCD is the technique used to monitor CBF through the large vessels within the Circle of Willis (Yang et al., 2015). Though several yoga based breathing techniques are being practiced by healthy individuals as well as people with various pathological conditions, theavailable evidence is reasonably small to demonstrate the scientific validity of the claimed results (Johnson et al, 2004). It is very much essential to understand the underlying physiological mechanisms before applying them as therapeutic agents in pathological conditions. Hence this study was conducted (i)to understand the difference that exist in cerebrovascular and cardiovascular functions between yoga and non-yoga practitioners and (ii) to find the specific changesin cerebrovascular and cardiovascular functions during the practice of two breath retention techniques -*Antar Kumbhaka* & *Bahir Kumbhaka*, two pranayama techniques -*Bhastrika* (bellows breath) &*Bhramari* (humming bee breath) and a yogic kriya- *Kapalbhati* (frontal brain cleansing breath).In the first phase (which was a cross sectional study) we recruited 15 yoga practitioners and 15 non-yoga practitioners. Assessments included measures of Cerebrovascular hemodynamics and cardiovascular functions which were assessed at one point of time without any intervention.In the second phase (which followed a self as control design), 20 healthy volunteers with experience in yoga were recruited. Assessments were made at baseline, during and immediately after the practice of each yoga based breathing technique. Accordingly, the discussion is presented in 2 parts. Part 1: Cerebrovascular hemodynamics and cardio-respiratory functions in yoga & non-yoga practitioners. Part 2: Cerebrovascular hemodynamics and cardiovascular functions during various pranayama techniques & a yogic kriya.

Part 1:

7.1. Cerebrovascular Hemodynamics and Cardiovascular Functions in Yoga and Non-YogaPractitioners:

Present study showed no significant difference in all the demographic variables between Yoga and non-Yoga practitioners (table-1) which shows that recruited non-Yoga practitioners were matched with Yoga practitioners. Cardiovascular variables such as SBP, PP and MAP were significantly low in Yoga practitioners compared to non-Yoga practitioners. The reasons for lower SBP in Yoga-practitioners could be due to low level of SV, CO & TPR in Yoga-practitioners as observed in this study (table 1).And/or better strengthening of the baroreceptor mechanism, or more responsive norepinephrine transporter inhibition as reported by the previous study (Muralikrishnan, 2012).Significant reduction in PP might be due to decreased SV, left ventricular ejection fraction, or reduced arterial stiffness (Sesso et al., 2000.). A significant reduction in MAP might be due to reduction in PP and/or reduction in vascular resistant (sesso et al., 2000.).

Though DBP was low in Yoga-practitioners,but was not statistically significant in the present study.But a previous study reported a significantly low DBP in Isha Yoga practitioners in which BP was measured using sphygmomanometer in supine position (Muralikrishnan, 2012).

Higher level of SBP was reported to reflect the progressive stiffening of the arterial wall, changes in the vascular structure, and the development of atherosclerosis; Higher levels of PP was reported to be associated with carotid stenosis, left ventricular hypertrophy, myocardial infarction, CVD death, and congestive heart failure in both normotensive and hypertensive populations (Sesso et al., 2000). MAP is the steady flow of blood through the aorta and its arteries and it reflects the left ventricular contractility, HR, and vascular resistance (Sesso et al., 2000).

Since there is a lowered level of SBP, PP, and MAP in yoga practitioners compared to non-Yoga practitioners, regular yoga practices might be useful to prevent cardiovascular diseases in future.Results of this study showed no statistical significant difference in cerebrovascular hemodynamics such as PSV (the flow velocity that present at the time of systole), EDV (the flow velocity that present at the time of diastole), MFV (calculated as EDV plus one-third of the difference between PSV and EDV) and PI (A flow resistance, calculated by subtracting EDV from PSV and dividing the value by MFV) between yoga and non-yoga practitioners. It indicates that the normal cerebrovascular hemodynamics were maintained both in Yoga and non-Yoga practitioners. Further understanding is possible only when individuals with more than five or ten years of experience in Yoga are compared with non-yoga practitioners.

7.2. Breath Holding Time between Yoga and Non-YogaPractitioners:

BHT is one of the most important variables used to measure the respiratory function (Karthik et al., 2014). Respiratory variable i.e. BHT was significantly high in Yoga practitioners compared to non-Yoga practitioners. This could be possibly through Yoga training which was reported to improve the strength of expiratory and inspiratory muscles due to which chest and lungs inflate and deflate to fullest possible extent and muscles are made to work to maximal extent (Karthik et al., 2014); Yoga participants could also possibly inhale more amount of air, thus making a greater amount of oxygen available to the system, which could help in holding the breath for a longer duration (Pal et al., 2013); and regular inspiration and expiration for longer duration would lead to acclimatization of central and peripheral chemoreceptors for both hypercapnoea and hypoxia causes an increase in the voluntary BHT and decrease urge to breathe while doing so; Along with this, the training of the stretch receptors in the respiratory muscles, chest wall and also walls of the alveoli support the breath holding (Karthik et al., 2014).

Part 2:

7.3. Cerebrovascular Hemodynamicsduring Various Pranayama Techniques &a Yogic Kriya

The present study shows that Bhastrika, Kapalbhati and Kumbhaka practices produce considerable effects on cerebral hemodynamic parameters. Cerebral auto-regulation maintains CBF within a narrow range. This is regulated by the autonomic nervous system by altering the tone of arteriolar sphincters. Various factors that regulate the arteriolar sphincteric tone include partial pressure of arterial carbon dioxide ($PaCO_2$), mean arterial pressure (MAP), cerebral metabolism and the autonomic nervous system. $PaCO_2$ is the strongest regulator of arteriolar tone and an increase of one millimetre of Mercury (Hg) increases CBF by 3-6% while a 1 mmHg reduction decreases CBF by 1-3% (Willie et al., 2014).

We did not observe any significant change in the cerebral hemodynamic parameters during normal breathing sessions, probably related to the maintenance of PaCO2, MAP, cerebral metabolism and the balanced state of ANS within a very narrow and stable range (Battisti-Charbonney et al., 2011).

Practice of high frequency breathing i.e. Bhastrika and Kapalbhati could produce an effect similar to hyperventilation which was shown toreduce the end tidal partial pressure of CO_2 by 15.5 ± 1.9 mmHgby washing-out CO_2 (Willie et al., 2014). The resultant hypocapnea increases cerebrovascular resistance and decreases the CBF (Battisti-Charbonney et al., 2011). Our hypothesis is supported by a previous study on Bhastrika and Kapalbhati, where the sympathetic activation was reported indirectly by means of the increase in HR, RPP and double product (Madanmohan et al., 2005). Perhaps, the excessive sympathetic activation through Bhastrika and Kapalbhati might have contributed towards the

development or the progression of cerebrovascular resistance and impaired CBF along with hypocapnea. The cerebral hemodynamic parameters gradually returned to their baseline values within 1 minute of cessation of Bhastrika and Kapalbhati, most probably related to the normalization of $PaCO_2$ during the normal breathing.

During Kumbhaka, significant increase in PSV, EDV and MFV was observed during the 30-60 seconds period, accompanied by significant reduction in PI. It indicates reduced cerebrovascular resistance during the practice of Kumbhaka. Kumbhaka leads to the development of hypercapnia and hypoxia. This phenomenon has been reported previously as a reduction of end tidal partial pressure of oxygen below its normal level of ~100 mmHg while end-tidal $PaCO_2$ rises above its normal level of ~40 mmHg during breath holding maneuver. The cerebral auto-regulation might be impaired during hypercapnia (Battisti- Charbonney et al., 2011) as evidenced by a significant increase in CBF and reduction in cerebrovascular resistance only beyond 30 seconds of breath holding, while no significant change occurred during the first 15 seconds of the maneuver. We speculate that the increase in $PaCO_2$ during Kumbhaka practice might have been within auto-regulatory range during the first 15 seconds and the auto-regulatory mechanism might be impaired between 15-30 seconds. This phenomenon might have been responsible for an increase in CBF by reducing the cerebrovascular resistance (Willie et al., 2014).

The mechanism by which hypercapnia and hypoxia affects cerebrovascular resistance remains poorly understood. Cerebrovascular smooth muscles are believed to be sensitive to changes in $PaCO_2$ and hydrogen ion (H^+) concentration. Increase in CO_2 (Battisti-Charbonney et al., 2011) and hypoxia (Willie CK et al., 2014) leads to increased H^+concentration, which activates voltage gated potassium (K^+) channels. The resultant hyper- polarization of endothelial cells reduces intracellular calcium, leading to cerebrovascular relaxation and vasodilatation (Willie et al., 2014 & Battisti-Charbonney et

al., 2011). Other proposed mechanisms include the release of vasoactive agents such as nitric oxide and prostaglandins in response to the velocity induced shear stress (Battisti-Charbonney et al., 2011) and Adenosine induced vasodilation (Willie et al., 2014).

During Bhramari Pranayama there were no significant changes in the cerebrovascular hemodynamics. It indicates that practice of 5 minutes of Bhramari pranayama might not have observable influencesin cerebrovascular hemodynamics.

7.4. Cardiovascular Functions during Various Pranayama Techniques&a Yogic Kriya

Result of this study showed a significant increase in SBP during the practice of Kapalbhati and Kumbhaka (Kumbhaka>Kapalbhati). In Kapalbhati the significant increase in SBP might be due to the significant increase in HR, and CO during the practice. Whereas, in Kumbhaka the significant increase in SBP might be due to the significant increase in TPR during the practice. Because SBP = CO × peripheral resistance (PR) (Muralikrishnan et al., 2012). Hence, Kapalbhati increases the SBP by CO and Kumbhaka increase the SBP through increasingTPR.

Though there was a significant increase in DBP during all breathing techniques (Kumbhaka>Bhramari>Kapalbhati>Bhastrika) it was sustained in the recovery period only after Bhastika> Kapalbhati and revert back to normal in Kumbhaka and Bhramari. Here, even though there is an increase in DBP during all breathing practices, the contributing factors are different in each breathing technique. For example the increased DBP during the high frequency breathing techniques such as Bhastrika and Kapalbhati might be due to a significant increase in HR and CO; during Bhramari,it might be due to a significant increase in HR and TPR; and during Kumbhaka, it might be due to a significant increase in TPR. The sustained increase in DBP even after the practice of Kapalbhati and Bhastrika could be due to the sustained higher levels of HR following thesepractices.

A significant increase in MAP during all breathing techniques (Kumbhaka > Kapalbhati > Bhramari > Bhastrika) can be attributedto a significant increase in DBP during these practices. MAP Revert back to normal following Bhramari and Kapalbhati and this could possibly be due to the reduction in the TPR after Bhramari and CO afterKapalbhati.

A significant increase in HR and reduction in PI were observed in Bhastrika > Kapalbhati > Bhramari and no such significant changes were observed during Kumbhaka and it was sustained after Bhastrika>Kapalbhati and revert back to normal after Bhramari. It might be due to the increased breath rate during the practice of Bhastrika and Kapalbhati. Whereas, the significant increase in HR and reduction in PI during Bhramari is unclear.

During Bhramari and Kumbhaka, a significant reduction in SV (Bhramari>Kumbhaka) and CO (Kumbhaka>Bhramari) were observed but the reduction was sustained only after Bhramari and revert back to normal after Kumbhaka. The reduction in the CO might be due to the significant reduction in the stroke volume but the reduction in the stroke volume during these practices is unclear and should be studied in the future. In contrast to Bhramari and Kumbhaka, there is a significant increase in CO during Kapalbhati and Bhastrika. This effect could be due to the significant increase in HR during thesepractices.

During Bhramari and Kumbhaka, a significant increase in TPR (Kumbhaka>Bhramari) was observed and revert back to normal after the practice. The reason for this change needs to be explored.