Clinical Study

Neckties and Cerebrovascular Reactivity in Young Healthy Males: A Pilot Randomised Crossover Trial

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Background. A necktie may elevate intracranial pressure through compression of venous return. We hypothesised that a tight necktie would deleteriously alter cerebrovascular reactivity.

Materials and Methods. A necktie was simulated using bespoke apparatus comprising pneumatic inner-tube with aneroid pressure-gauge. Using a randomised crossover design, cerebrovascular reactivity was measured with the "pseudo-tie" worn inflated or deflated for 5 minutes (simulating tight/loose necktie resp.). Reactivity was calculated using breath hold index (BHI) and paired "t" testing used for comparative analysis. Results. We enrolled 40 healthy male volunteers. There was a reduction in cerebrovascular reactivity of 0.23 units with "tight" pseudo-tie (BHI loose 1.44 (SD 0.48); BHI tight 1.21 (SD 0.38) P < .001). Conclusion. Impairment in cerebrovascular reactivity was found with inflated pseudo-tie. However, mean BHI is still within a range of considered normal. The situation may differ in patients with vascular risk factors, and confirmatory work is recommended.

1. Introduction

Pathogenesis and treatment of stroke in young adults remain poorly understood. Management strategies are often based on evidence from studies of older patient cohorts however risk factors and prognosis are not equivalent for these two groups. Certain risk factors, for example, patent foramen ovale or illicit drug use, may be pertinent in younger populations, but even for these conditions there is no consensus on relative importance or optimal management [1–4]. Thus identification of novel risk factors for cerebrovascular disease in young adults remains an important area of study. Based on basic anatomy and physiology, extrapolation of data from other medical disciplines, and a degree of “lateral” thinking, we sought to characterise one such novel risk factor.

Many young professionals, including doctors, wear neckties. Recent media coverage has focussed on potential bacterial transmission via a necktie vector. The risk of neckties may not be confined to patients—a further possible “danger” of this sartorial habit is suggested by reports of pathologically increased intraocular pressures in individuals wearing tight neckties, the postulated mechanism being impairment of ocular venous drainage [5, 6]. If neckties cause haemodynamic effects in retinal vasculature, it seems reasonable to suppose that impairment of venous drainage could occur in other supracervical vascular beds including the cerebral vessels. Although an effect of circumferential neck pressure on stroke risk has been previously hypothesised [7], a “real-time” demonstration of the cerebrovascular effect of a tight necktie has not previously been demonstrated.

We hypothesized that, via jugular venous compression, a tight necktie may elevate intracranial venous pressure and impair reactivity in the cerebral circulation—a surrogate marker of cerebrovascular risk. We performed a randomised crossover study to describe the effect of wearing a tight necktie on cerebrovascular reactivity.

2. Materials and Methods

The study was conducted in the cerebrovascular investigation laboratory of our university hospital. The Local Research and Ethics Committee granted ethical approval prior to study.
commencing. All subjects gave informed consent and were allowed to withdraw from the study at any time. As this was a pilot study and as we were interested in novel risk factors, we invited healthy men with no known history of vascular disease and on no regular medication to participate.

For our primary outcome, we used a noninvasive surrogate measure of cerebrovascular reactivity—the Breath Hold Index (BHI). BHI is a validated and commonly used measure, which has been described in detail elsewhere [8]. In brief, breath holding yields a hypercapnic stimulus, which causes vasodilatation of cerebral resistance vessels. This in turn increases blood mean flow velocity in the middle cerebral artery (MCAv). To calculate BHI, the MCAv was measured at rest and after 30 seconds of breath holding. The change in MCAv was then used to calculate BHI using the formula:

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BHI = \frac{[\text{MCAv}(\text{end of BH}) - \text{MCAv}(\text{rest})]}{\text{MCAv}(\text{rest})} \times \frac{100}{\text{Breath Hold (sec)}}.
\]

To simulate and quantify the circumferential pressure applied by a necktie, a novel, purpose built apparatus was used, consisting of a pneumatic inner tube and aneroid pressure gauge (Figure 1).

All patients rested for five minutes in a quiet, temperature-controlled room prior to initial seated measurements. The middle cerebral artery (MCA) was insonated using a transtemporal approach. A Spencer M100 TCD machine (Spencer Technologies, Seattle USA) with 2 MHz transducer was used for the study. End tidal carbon dioxide concentration was measured to ensure that breath holds were adequate. To avoid valsalva, breath holding was undertaken following normal inspiration.

Participants were randomly allocated to begin wearing the purpose-built necktie inflated or deflated (to simulate a tight and loose necktie, resp.). Randomisation was performed using a simple coin tossing procedure. Inflation pressure was determined by the volunteer’s perception of how a “tight” but not excessively uncomfortable necktie would feel. BHI was calculated after 5-minute exposure to tight/loose necktie, with 5-minute “washout” between measurements. This was achieved by measuring the change in MCAv over a 30-second breath hold and calculated using the stated BHI formula. The pressure gauge was concealed from the participant throughout the study, and the same operator performed all measures.

Our primary outcome measure was changed in BHI. Initial descriptive statistics suggested a reasonable parametric distribution, thus a paired t-test was used to compare BHI with necktie inflated and deflated. A sample size of 40 volunteers was deemed necessary to detect a difference in mean BHI of 0.20 units, with 90% power. Post hoc analyses of relationship between BHI (tight) and age; body mass index (BMI), and diastolic blood pressure (mmHg) were performed using chi-square or Pearson correlation as appropriate. All statistics were performed using Minitab software (version 14.0, StateA Inc, PA, USA).

3. Results

We enrolled 40 healthy males; all subjects approached consented to the study with no “drop-outs” by study completion. Mean age was 31.5 (SD 10.5, range 20–59) years. Mean inflation pressure with the tight necktie was 63.5 (SD 12.3) mmHg. There were no adverse events from use of the necktie. MCAv at rest was similar with the necktie inflated (45.72 cm/s) and deflated (45.00 cm/s). The mean increase in MCAv during the breath hold was 16.49 cm/s with the necktie inflated compared to 19.27 cm/s with the necktie deflated. Cerebrovascular reactivity as measured by the BHI was reduced during necktie inflation (BHI = 1.44 with necktie deflated versus 1.21 when necktie inflated, P < .001) (Figure 2). Comparing older (age > 30 years; n = 20) and younger volunteers revealed a nonsignificant trend towards reduction in cerebrovascular reactivity (difference 0.07; 95% CI: −0.25 to 0.13) between the groups. Correlation coefficients calculated for blood pressure and BMI were \( r = 0.23 \) and \( r = 0.003 \), respectively.
4. Discussion

We hypothesised that wearing a tight collar or tie may compromise the venous drainage of the brain and thus impair cerebrovascular reactivity. Using a novel necktie apparatus we confirmed a significant reduction in cerebrovascular reactivity in healthy individuals wearing a tight necktie.

It is, of course, important to determine the clinical significance of this novel observation. The reduction in BHI seen when wearing the tight necktie (0.23 units) was clinically modest. Even with the necktie fully inflated, observed BHI values lay within values accepted as normal. This suggests that the changes seen are of little clinical significance in young healthy males and are unlikely to contribute to stroke risk in younger cohorts.

However, minor changes in cerebrovascular reactivity may be of importance in populations with higher baseline risk. Several cohorts with coexistent cerebrovascular risk factors and tight tie exposure can be postulated—for example, the cardiovascular risk of the obesity-hypoventilation phenotype is well recognised; with their increased neck girth this population may further increase their risk through wearing a tight necktie [9, 10]. The prevalence of tight neckties/collars should not be underestimated; a recent American study suggested that 70% of middle aged men wear a shirt collar at least one size too small [11].

In a cohort of healthy volunteers, analysis of differential effects of necktie pressure by classical vascular risk factors is not possible. We performed a post-hoc analysis to determine possible effects of certain factors. For age, the most powerful vascular risk marker, a trend towards increased pressure of necktie and greater reduction in reactivity was observed. Statistical significance was not achieved, a reflection of the small numbers in each group, but still an intriguing possibility of greater vascular risk with necktie exposure in older age is suggested. Positive correlation between increasing blood pressure and decreasing reactivity was small, and there was no significant relationship between body mass index and reactivity. Again interpretation of these data must be cautious due to low numbers, “healthy” subjects, and the post hoc nature of these analyses.

We accept that these are preliminary data and this initial hypothesis generating study had certain methodological weaknesses. Our choice of outcome measure is open to criticism. BHI is a surrogate marker of future cerebrovascular disease; a definitive statement on neckties and risk would require a more robust outcome. However, noninvasive measures of cerebrovascular reactivity have been shown to be strongly predictive of future stroke risk and as such are suitable as surrogate endpoint, especially in younger cohorts [12, 13]. Other measures of BHI are described but involve further intervention, for example, administration of a vasoactive substrate [14]; for this initial “pilot” study we felt BHI was a suitable compromise.

BHI measurements were performed after only 5 minutes of necktie exposure. Cerebral vasculature may autocorrect if a tie is worn over a more prolonged period, such as a working day, and the significance of a transiently induced reduction in reactivity is unknown. With a single “dose exposure”, our study was unable to define the cumulative effect of repeatedly wearing a necktie. Thus it could be argued that in our study, we have demonstrated the cerebral vascular effects of attempted asphyxiation rather than daily necktie exposure. The pressure defined as “tight” was subjective, with inflation pressures of the “tight” necktie varying by as much as 60 mmHg between participants. This suggests that in a cohort of necktie wearers, some may routinely wear the tie at pressures that may interfere with cerebrovascular reactivity. As discussed, a more informative study may have been to measure cerebrovascular effects in a cohort with prevalent vascular risk factors; for safety reasons a healthy male population was considered for this preliminary analysis.

5. Conclusions

Our data suggest a detrimental effect of tight neckties on the cerebral vasculature. The clinical significance remains to be determined, and it seems unlikely that circumferential neck pressure from a necktie or similar garment is an important contributor to stroke risk in young males. In fact any clinical effect of neckties may be seen only in older patients with concomitant cardiovascular risk factors. The search for important, novel risk factors for stroke in young adults continues. These initial “neutral” findings give some scientific credence to wearing neckties “loose”—if at all.

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