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Problems in Aero-Medical Evaluation : Sick Sinus Syndrome

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Sick Sinus Syndrome (SSS) is an important condition in the context of sudden incapacitation in air vis-a-vis aircrew duties. Advances in electrocardiology and its clinical application that have led to changing concepts and greater understanding of SSS are briefly reviewed. Three cases of SSS are presented highlighting aero-medical aspects.

HIGH index of suspicion to seek and exclude conditions potentially capable of causing sudden incapacitation in air is an inseparable part of aircrew medical evaluation. Two preeminent organ-systems involved in this aspect are cardiovascular system (CVS) and central nervous system. Sick Sinus Syndrome is one such CVS clinical entity and needs to be reviewed afresh. The concepts and diagnostic parameters of this syndrome have changed over the decade, reflecting progressive sophistication and application of electrophysiological studies (EPS) in clinical cardiology. These are summarised.

Three cases of SSS in aircrew came to our attention recently and presented features of clinical interest and aeromedical importance. These cases are briefly discussed.

Sick Sinus Syndrome

SSS refers to a complex of symptoms, signs and electro-cardiography (ECG) features attributed to Sinus Node (SN) dysfunction[4] characterised by intrinsic inadequacy of SN to perform its pacemaking function and/or failure of SN impulse to activate rest of atrium[8].

Ultrastructurally,[3,10,15,19] two types of cells are distinguished in the specialised conduction system of heart. P-Cell (Pale-staining) is found in SN, atrioventricular node (AVN) and internodal pathways, being more numerous in SN. T-cell (Transitional Cell) is also found within the two nodes, extending considerable distance into the perinodal zone. Presumably, P-Cell has pacemaker function and T-Cell organises the distribution of

impulses originating from P-Cell. The basic disorder responsible for SSS seems to be a 'generator failure' and a 'transmission failure'[9] resulting in disordered impulse formation and conduction. The term SSS, thus embraces disease of any or all of SN, AVN and its appendages.

Electrophysiologically, pacemaker function is marked by spontaneous cellular depolarisation (Phase IV) [4],[6].SN is susceptible to overdrive suppression by rapid right atrial pacing (AP). 'Sick' SN is abnormally susceptible to the suppressive influence of ectopic atrial activity and remains quiescent for uncontrollably long periods.[5] The time measured from the last paced P wave to the first spontaneous sinus P wave gives Sinus Node Recovery Time (SNRT) and its prolongation indicates SSS. Corrected Sinus Node Recovery Time (CSNRT) increases the reliability of SNRT in the diagnosis of SSS. It is defined as recovery time in excess of basal sinus cycle length (SNRT—PP interval). In normal subjects CSNRT upto 500 msec is considered within limits,[1],[4] and is unaffected by rate and duration of AP. CSNRT is prolonged in SSS. Whereas CSNRT tests the pacemaking function of SN, measurement of Sino-Atrial Conduction Time (SACT) following premature atrial stimulation (PAS) tests the conduction properties of SN and perinodal zone[4],[8].Employing PAS, SACT—(A2-A3)—(A1-A2); where A2-A3 is return cycle length and A1-A2 is test length. SACT is considered abnormal if more than 120 msec[1],[4] and is prolonged in SSS. Close correlation between prolonged SACT and SSS has been demonstrated[4] and SACT inferred to be a more sensitive index as diagnostic criterion compared to CSNRT[1]. High incidence of distal conduction disease in patients with SSS[4],[9] also makes His-Bundle Electrogram (HBE) study mandatory in this condition.

Neurohormonally, autonomic nervous system (ANS) influences intrinsic SN function[10]. Parasympathetic stimulation causes sinus bradycardia by hypopolarisation and decreased rate of phase IV[4]; sympathetic stimulation has opposite effects. Intrinsic Heart Rate (IHR) can be determined after total autonomic blockade (TAB) by administration of propranolol and atropine[10]. A subnormal response consists of more than 10% decrease in age predicated heart rate (HR)[4]. Extrinsic influence of ANS on 'sick' Sinus is complex and sometimes paradoxical. Normalisation of CSNRT following atropine does not exclude SSS.[4] Determining IHR, CSNRT and SACT after TAB aids to define the operative extrinsic influences of ANS and guide medical therapy in subsets of SSS[11].

Electrocardiographically, a wide spectrum of arrhythmias[6] (bradyarrhythmias, tachyarrhythmias, tachy-bradyarrhythmia) are well known to be manifest.

Aetiologically, SSS does not have a single cause and pathogenesis [13], [17], however, it is often obscure[4]. Being located a mm or less beneath epicardium, SN and perinodal zone can be heir to many diseases (eg., inflammatory, vascular, sclerodegenerative, infiltrative) of myocardium and pericardium. Genetic factor may be involved in SSS of young[7].

Clinically, SSS is more frequent in the elderly, with peak incidence between sixth and seventh decades[4]. But no age is exempt. Studies in young people [7], [20] and infants [12], [15] have shown SSS. Symptomatology is unpredictable, bizarre, intermittent and include syncope, black-out, and giddiness from low cardiac output. Physical examination may be non-contributory or reveal arrhythmias. Therefore, a high degree of suspicion and systematic approach is essential for diagnosing SSS. Detailed history and exclusion of other metabolic/neurologic causes of episodic unconsciousness are indispensable. Methodical evaluation by means ot scalar ECG, EPS and dynamic ECG with Holter monitor and telemetry will lead to and clinch the diagnosis.

All patients of SSS need not be treated straightaway. Treatment is on sound pharmacotherapeutic principles or appropriate pacemaker implantation, both based on EPS.

Case Report

Case-1. (Figure-1)— Thirty years old test pilot (1400 hrs flying-Fighters) was being evaluated for a special project requiring exacting medical standards. Bicycle ergometer test

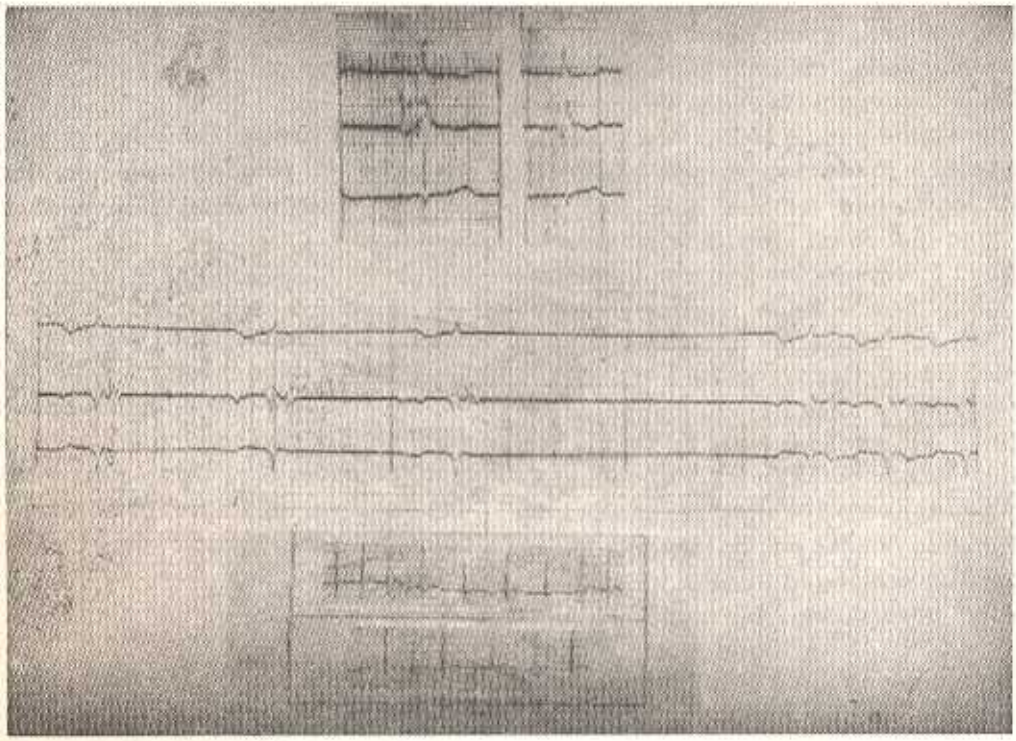

Fig. 1. *Case No. 1—Top strips show HBE. Middle strip shows prolonged SNRT after AP. Bottom strips show Telemetry ECG showing Sinus pause and a junctional ectopic.*

led to peak HR 184 bpm and 'J' depression 1.5-2.0 mm. Recovery induced non-specific 'T' changes. HR varied between 54 to 84 bpm during Tilt-Table studies. Serial ECG revealed sinus bradycardia, atrial ectopics, nonspecific 'T' changes L3 aVF and post-exercise junctional escape rhythm with 'J' depression. EPS revealed the following : basal cycle length 1280 msec; PA 10 msec, AH 70 msec, HV 40 msec. After atropine a very short period of sinus mechanism of cycle length 800 msec was noticed. AP prior to atropine resulted in the occurrence of Wenckebach block at lower rate of 100 bpm. SNRT varied from 1720 to 2900 msec; CSNRT also remained elevated at 900 to 1000 msec. Atropine blockade recorded junctional rhythm. Telemetry ECG recorded once showed sinus pauses and junctional ectopics.

Case-2. 25-years old transport pilot (1000 flying hours) was detected to have Pulmonary Tuberculosis in Oct 79. Anti-Tuberculosis treatment (ATT) was completed in Apr 81 and the course was uneventful. Review showed clinically and radiologically normal lungs, but he developed atrial premature beats (APB) which were present at rest, disappeared immediately after exercise and reemerged as HR slowed down. Following up periodically, APB was seen to persist. Considered benign, in Jun' 81 he was reflighted in restricted temporary A3 medical category. During subsequent review in Jul-Aug 82, serial ECGs revealed APB, junctional escape beats and sino-atrial block. Master's Double Two-Step exercise (DMT) produced tachybradyarrhythmia and stress testing induced increased atrial ectopic activity. EPS revealed the following : SNRT normal; mildly deranged AVN function; 2 : 1 Wenckebach block at HR beyond 120/min.

Case-3. Twenty six years old fighter pilot (634 flying hours) took off for air-test sortie in Type-75 aircraft. He carried out the sortie during which he spent 20min around 3km altitude. He returned to base and because of fuel state, decided to do one circuit for which he made overhead pass at 2.3 km altitude and turned right. At that time he felt a sense of suffocation, disbalance, hard breathing, weakness and some pain left arm. He gave a call on RT "I am blacking out" and simultaneously put auto-pilot on. Following this though ATC tried to contact him, no response was forthcoming. Witnesses, however, saw the aircraft flying straight and level. It has subsequently been possible to ascertain that the pilot was unconscious/unable to respond for a period of 13 min 25 sec. Thereafter, he again came on RT and transmitted "Where am I ?". Subsequently, he could recognise a prominent ground feature and also realise he was low on fuel. He was guided to land safely on another air-field He felt weak although he could walk out of cockpit unaided.

On later interrogation, he disclosed, for the first time that he had experienced in the past similar symptoms six or seven times (approximately once a year) since onset in 1976. He denied loss of consciousness accompanying these episodes ever before, but admitted that the duration of each successive episode appeared to be lengthening. One of the attacks had occurred while riding motorcycle singly and he had to stop for two to three minutes for the symptoms to subside prior to continuing journey.

Thorough evaluation of aircraft systems, flying clothing etc., revealed no fault. Comprehensive examination and investigations ruled out any neurological, ENT, psychiatric or biochemical abnormality. ECG revealed sinus bradycardia with intermittent sinus arrest. EPS revealed the following: basic sinus cycle length 1090 msec, AH 80 msec, HV50-55 msec; SNRT 1520 to 2140 msec; CSNRT 480 to 1050 msec; SACT 387 msec; AH 230 msec at AP 90/min and 280 msec at AP100/min with AH block beyond; maximum HR 94/min but normalised SNRT and CSNRT after atropine: AH Wenckebach block at 165/min.

Discussion

It is clear that flight safety hazard is omnipresent in SSS because of inherent potential to cause sudden incapacitation in air. Pertinently, in a survey of three cardiac centres conducted by Mackintosh[7], nine patients under the age of 15 were diagnosed with SN dysfunction and all were males. All our three cases were also 25 to 30 years old in the group of operational fliers.

Case-1 has been totally asymptomatic and otherwise clinically normal. SSS of obscure aetiology has been discovered incidental to more exacting evaluation than customarily in force to determine medical fitness for flying. There is no evidence of associated AVN disease at this stage and lower pacemaker appears quite adequate. There is no indication for active treatment presently. The problem is that, the natural history of SSS in this age group bearing on the rate and extent of disease progress is not known. A number of mild and asymptomatic forms of this condition may gradually worsen in adolescence or adulthood[14]. Once diagnosed, the possibility of unpredictable, serious, episodic manifestation cannot be ruled out. In the utmost interests of flight safety, this state becomes incompatible with aircrew duties in general and sole control of aircraft in particular. While determining flying status in any individual aircrew, the unique circumstances of SSS diagnosis, as in this case, need to be carefully weighed against the disease profile, flight safety hazard, restrictions on aerial duties warranted and service requirements.

In case 2, secondary SSS aetiologically related to Tuberculosis (TB) infection and cardiac involvement deserves consideration. TB of heart may be due to involvement of myocardium, pericardium or coronary arteries.Kinare[5] studied conduction system in cases of TB heart and concluded that, in pericarditis SAN is likely to be involved while in myocarditis AVN and its appendages. The subject case had antecedent treated pulmonary TB. He deve loped cardiac arrhythmias initially masquerading as being APB. Deterioration over a year into manifest SSS with AVN dysfunction is evident. The prognosis is uncertain. Since cardiac arrhythmia developed

in the wake of full completed ATT and no disease activity, SSS may be the residual effect of healed TB heart that coexisted with pulmonary lesion. Resolution of pathology and reversibility of dysfunction appear unlikely. On the other hand, progressive dysfunction is possible and aircrew duties would be precluded in the interests of flight safety.

Case-3 glaringly exemplifies the flight safety hazard inherent to SSS, the unpredictable manifestations of the disorder and the diagnostic difficulties peculiar to reticent aircrew population. This instance, though concluded miraculously without mishap, nevertheless, does raise the paramount question of preventing recurrence of this nature. Detection of such cases is renderedtmore difficult by low incidence (especially because of pre-selection screening) without a high index of suspicion at the peripheral level manning general medical care of aircrew. Arrhythmias, especially bradyarryhthmias, should never be ignored or presumed physiological or being without any further consideration. Inadequate acceleration of HR or aberrant response to exercise should always be suspect, qualifying for further inquiry and investigation. Bizarre symptomatology and episodic manifestations of SSS dictate obtaining thorough history. Detailed cardiovascular examination and-methodical investigations, if and when indicated, ought not be stinted on extraneous considerations.

Conclusion

SSS is a potential hazard to flight safety and the age group of active aircrew population is not exempt from this disorder. Recent advances in electrocardiology and its clinical application have given us greater understanding of this disorder to appreciate its importance in aviation medicine Bizarre symptoms and intermittent manifestations coupled with reticence on the part of aircrew make the diagnosis difficult. High index of suspicion is demanded and cardiac arrhythmias should not be dismissed lightly unless established benign.

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